Ventricular Tachycardia Arising From the Aortomitral Continuity in Structural Heart Disease: Characteristics and Therapeutic Considerations for an Anatomically Challenging Area of Origin

Daniel Steven, MD; Kurt C. Roberts-Thomson, MBBS, PhD; Jens Seiler, MD, PhD; Keiichi Inada, MD; Usha B. Tedrow, MD, MSc; Richard N. Mitchell, MD, PhD; Piotr S. Sobieszczuk, MD; Andrew C. Eisenhauer, MD; Gregory S. Couper, MD; William G. Stevenson, MD, FHRS

Background—The aortomitral continuity (AMC) has been described as a site of origin for ventricular tachycardias (VT) in structurally normal hearts. There is a paucity of data on the contribution of this region to VTs in patients with structural heart disease.

Methods and Results—Data from 550 consecutive patients undergoing catheter ablation for VT associated with structural heart disease were reviewed. Twenty-one (3.8%) had a VT involving the peri-AMC region (age, 62.7±11 years; median left ventricular ejection fraction, 43.6±17%). Structural heart disease was ischemic in 7 (33%), dilated cardiomyopathy in 10 (47.6%), and valvular cardiomyopathy in 4 (19%) patients, respectively. After 1.9±0.8 catheter ablation procedures (including 3 transcoronary ethanol ablations) the peri-AMC VT was not inducible in 19 patients. The remaining 2 patients underwent cryosurgical ablation. Our first catheter ablation procedure was less often successful (66.7%) for peri-AMC VTs compared with that for 246 VTs originating from the LV free wall (81.4%, P=0.03). During a mean follow-up of 1.9±2.1 years, 12 (57.1%) patients remained free of VT, peri-AMC VT recurred in 7 patients, and 1 patient had recurrent VT from a remote location. Three patients died. Analysis of 50 normal coronary angiograms demonstrated an early septal branch supplying the peri-AMC area in 58% of cases that is a potential target for ethanol ablation.

Conclusions—VTs involving the peri-AMC region occur in patients with structural heart disease and appear to be more difficult to ablate compared with VTs originating from the free LV wall. This region provides unique challenges for radiofrequency ablation, but cryosurgery and transcoronary alcohol ablation appear feasible in some cases. (Circ Arrhythm Electrophysiol. 2009;2:660–666.)

Key Words: ablation ■ alcohol ■ angiography ■ catheter ablation ■ tachyarrhythmia

Ventricular tachycardias (VTs) with a QRS morphology consistent with a left ventricular outflow tract (LVOT) origin may arise from various anatomic sites surrounding the LVOT.1,2 These sites include (1) the aortic root with its cusps, (2) the mitral annulus, (3) the superior basal septum, (4) the epicardium, and (5) the region around the aortomitral continuity (peri-AMC).3,4 The latter is located at the superior basal portion of the left ventricle and mainly consists of fibrous tissue. It is embedded between the aortic and mitral valve annuli, bordered by the ventricular septum, and the anterior left ventricle (Figure 1). Recently, there have been reports of VTs originating from the peri-AMC region; however, these data are limited to idiopathic VTs.1,5–8 In the present study we report VTs originating from the this particular region in patients with structural heart disease. The VTs in this region were often difficult to control with endocardial catheter ablation and the feasibility of alternative approaches, including transcoronary ethanol ablation and cryosurgery are demonstrated.

Clinical Perspective on p 666

Methods

Patients

A total of 21 patients with recurrent VT originating at the peri-AMC region were identified from retrospective review of 550 consecutive
Two patients with valvular heart disease were included in a previous report. All patients provided written consent for electrophysiological procedures, and the Institutional Review Board of Brigham and Women’s Hospital approved the collection of data.

Electrophysiology Study

Initial mapping and ablation was performed as described previously.10 The left ventricle was accessed either via a retrograde aortic approach or in case of prior aortic valve replacement via a transseptal antegrade mitral approach. In patients who underwent epicardial mapping and ablation, subxiphoidal epicardial access was performed as described previously.11,12 During left ventricular endocardial mapping and ablation, systemic anticoagulation with heparin was maintained. Surface ECG leads and intracardiac electrograms were stored in digital format (Prucka CardioLab EP System, GE Healthcare, Wis). Nonfluoroscopic electroanatomic mapping was performed using a 3-dimensional mapping system (CARTO, Biosense-Webster, Inc, Diamond Bar, Calif). A sinus rhythm voltage map was performed in all 21 patients. An anatomic low-voltage scar was defined as 2 adjacent points within a structural territory with a bipolar voltage ≤1.5 mV.13,14 The mechanism of VT was defined as scar-related reentry when VT was inducible with programmed stimulation, could be entrained, and had an exit site at a low-voltage area consistent with scar. Reentry circuit sites were defined by entrainment mapping and pace mapping as reported previously.13 When VT was unstable for mapping, exit sites were targeted on the basis of the QRS morphology during pacing.10,16 The following ablation catheters were used: a nonirrigated 4-mm-tipped catheter (Navistar, Biosense-Webster, Inc) in 2 patients, an externally irrigated 3.5-mm-tip catheter (Thermocool, Biosense-Webster, Inc) in 19 patients, and a 4-mm internally irrigated electrode (Chilli, Boston Scientific, Inc, Natick, Mass) in 2 patients. In 2 cases, more than 1 ablation catheter was used.

In 3 patients, transcoronary ethanol ablation was attempted by subselectively cannulating arteries that perfused the presumed VT region and assessing whether balloon occlusion and saline infusion terminated tachycardia, followed by injection of 1 to 3 mL of absolute ethanol, as previously described.17 After ablation, programmed right ventricular stimulation was performed with up to 3 extrastimuli and burst pacing and during administration of isoproterenol in selected cases. The acute success rates during the first procedure were compared with a consecutive group of patients who underwent endocardial ablation of VTs arising from the free wall of the LV outside the peri-AMC region. This group was similar with regard to (1) age, (2) sex, and (3) LV function.

Definitions

The peri-AMC region was defined fluoroscopically by its characteristic annular location in the right and left anterior oblique fluoroscopic views. VT was defined as originating from this region if (1) radiofrequency ablation at the site terminated and abolished inducible VT, (2) the peri-AMC region was an exit or isthmus for the VT based on entrainment, or (3) earliest presystolic activation was identified in the region, with entrainment or activation evidence that sites at increasing distance from the peri-AMC region were progressively further from the VT circuit based on entrainment (longer postpacing interval) or later activation. VT storm was defined as occurrence of >3 VT episodes in a 24-hour period.

Coronary Angiograms

After recognizing that radiofrequency catheter ablation in the peri-AMC region was often difficult, we sought to determine the frequency of identifiable coronary arterial perfusion to the area that might be targeted for ethanol ablation. Angiograms from 50 consecutive patients without structural heart disease and no prior history of VT who had undergone coronary angiography for assessment of suspected coronary artery disease were reviewed by 2 angiographers to determine the presence of an early branch deriving from the circumflex or the left anterior descending artery supplying the region adjacent to the AMC.

Statistical Analysis

Continuous variables are reported as mean±SD or as median and interquartile range if appropriate. For comparing VTs arising from the LV free wall and the peri-AMC region, the χ² test for categorical data and t test for continuous data were used where appropriate. Tests were performed as 2-tailed, and probability values <0.05 were considered statistically significant. The statistical analysis was performed using SPSS 16.0 for Macintosh (SPSS, Inc, Chicago, Ill).

Results

Patient Characteristics

AMC VT was identified in 21 of 550 patients (3.8%) with recurrent VT and structural heart disease (Table 1). Patients had a mean age of 62.7±11 years; 19 were male (82.6%). Underlying structural heart disease was nonischemic cardiomyopathy in 10 (47.6%), prior myocardial infarction in 7 (33.3%), and aortic valvular replacement in 4 (19%) patients, respectively. Sixteen (76.2%) patients had undergone prior cardioverter-defibrillator implantation, of which 5 (31.3%) had a device for biventricular pacing. Twelve of the patients were receiving β-blockers. Patients were receiving a mean of 1.6±0.8 antiarrhythmic drugs at the time of the procedure (Table 1).

Figure 1. Gross anatomy of the heart. a, View from the atria toward the valvular apparatus showing the anatomic relationship between aortic valve (AV), mitral valve (MV), pulmonary valve (PV), and tricuspid valve (TV). Asterisks indicate the left and the right atrial appendages, respectively. White triangle represents the area of the aortomitral continuity corresponding to Figure 2. b, View of the anterior leaflet of the mitral valve (asterisk) after opening of the left ventricle and the aortic valve showing the close relation of the aortic valve and the anterior leaflet of the mitral valve. Arrow indicates the left main artery. LCC indicates left coronary cusp; NCC, noncoronary cusp; RCC, right coronary cusp.
The reason for referral was electrical storm in 6 (28.6%) patients, repetitive implantable cardioverter-defibrillator therapies in 9 (42.9%) patients, symptomatic self-terminating VT in 4 (19.0%), and hemodynamically stable sustained VT in 1 patient (4.8%). The patients had a mean of 2.1 (range, 0 to 5) VT episodes within the last 7 days before the procedure.

**Tachycardia and Substrate Characteristics**

In these 21 patients, a total of 39 different VTs were inducible, of which 35 were targeted for ablation. Twenty-one VTs arose from the aortomitral continuity (Figures 2 and 3), and these had a mean cycle length of 395±90.4 ms. The other 18 VTs were related to scar areas remote from the peri-AMC region throughout the left ventricle (lateral free wall, n=6; the anterior free wall, n=4; apical, n=5; and inferior, n=3).

**Table 1. Patients and Procedural Characteristics**

<table>
<thead>
<tr>
<th>Patient Cohort</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients</td>
<td>21</td>
</tr>
<tr>
<td>Age</td>
<td>62.7±11</td>
</tr>
<tr>
<td>Male</td>
<td>17 (81)</td>
</tr>
<tr>
<td>Total procedures</td>
<td>39</td>
</tr>
<tr>
<td>VTs induced</td>
<td>39</td>
</tr>
<tr>
<td>VTs targeted</td>
<td>35</td>
</tr>
<tr>
<td>VTs arising from the AMC</td>
<td>21</td>
</tr>
<tr>
<td>AMC VT cycle length, ms</td>
<td>385±90.4</td>
</tr>
<tr>
<td>LV ejection fraction, %</td>
<td>43.6±17</td>
</tr>
<tr>
<td>End-diastolic LV diameter, cm</td>
<td>5.4±0.8</td>
</tr>
<tr>
<td>Interventricular septum thickness, cm</td>
<td>1.1±0.3</td>
</tr>
</tbody>
</table>

**Location of myocardial infarction**

- Anteroseptal: 2
- Anterobasal: 3
- Inferior: 2

**Procedure duration, min**

- 230±62.4

**Fluoroscopy, min**

- 42.1±19.3

**Ethanol ablation**

- 3

**Surgical cryoablation**

- 2

**VT recurrence (according to 12-lead ECG)**

- From peri-AMC region: 7
- Other: 1

**Antiarrhythmic drugs at index procedure**

- Mexiletine: 6
- Quinidine: 5
- β-Blocker: 12
- Sotalol: 2
- Dofetilide: 1
- Amiodarone: 8

Data are presented as n (%) or mean±SD.

The reason for referral was electrical storm in 6 (28.6%) patients, repetitive implantable cardioverter-defibrillator therapies in 9 (42.9%) patients, symptomatic self-terminating VT in 4 (19.0%), and hemodynamically stable sustained VT in 1 patient (4.8%). The patients had a mean of 2±1.2 (range, 0 to 5) VT episodes within the last 7 days before the procedure.

At the successful ablation site, the earliest local activation precede the QRS onset by a mean of 48 ms (range, 23 to 157 ms) and showed a mean electrogram amplitude of 0.73 mV (0.12 to 2.4 mV). A low-voltage area (≤1.5 mV) consistent with scar was found in 18 patients, all of whom had scar-related reentrant VT with a median scar size of 22 mm² (2 to 52 mm²). At least part of the scar as well as the exit of the VT was adjacent to the aortomitral continuity (Figure 4) in all patients. In 3 patients, according to the electroanatomic map, no area of scar was identified on the endocardium adjacent to the AMC, but the VT arose from that area as indicated by the successful ablation site on the electroanatomic map.

**ECG Characteristics**

The QRS pattern of the VTs arising from the AMC area was right bundle-branch block–like morphology in 19 (90.5%) VTs and left bundle-branch block–like morphology in 2 VTs. Positive concordance was present in the precordial leads from V2 to V6 in all VTs. A qR pattern in V1 was appreciated in 5 (23.8%) patients. The R-wave ratio in leads II and III was 1 (Table 2 and Figure 5).

**Radiofrequency Ablation Procedures**

The procedural data are given in Table 1. The patients underwent a mean number of 1.9±0.75 (range, 1 to 3) procedures. Endocardial mapping was initially performed in all patients. Fourteen patients (66.7%) had a second procedure either because of late recurrence or initial failure to successfully ablate the AMC VT. In 6 (28.6%) of those, a repeat endocardial radiofrequency ablation was performed, and in another 6 (28.6%), an epicardial radiofrequency ablation was performed. Two (9.5%) patients underwent a transcoronary ethanol ablation during their second procedure. In 4 (19.1%) patients, a third procedure was performed, consisting of endocardial radiofrequency ablation in 1 patient.
transcoronary ethanol ablation in 1 patient, and cryothermal surgical ablation in 2 patients.

Endocardial radiofrequency ablation alone was acutely successful in 11 patients (52.4%). The success rate after a single procedure was 33.3% (n=7). Radiofrequency ablation during the first procedure was less often acutely successful (66.7%) than for VTs originating from the free wall of the LV (81.4% [P=0.03]; Table 3).

Epicardial Mapping and Radiofrequency Ablation

In 9 patients, epicardial evaluation of the AMC VT was performed either mapping the great cardiac vein (GCV) or using a subxiphoidal epicardial access. Mapping within the GCV was performed in 6 patients, in 2 of whom the VT was successfully abolished from this site. In the other 4 patients, earliest activation in the GCV led to mapping using a subxiphoidal epicardial access, with successful ablation in 3 patients.

The myocardial thickness between the endocardium and epicardium around the mitral annulus was 23.5±2.2 mm, as evaluated in the electroanatomic map.

Transcoronary Ethanol Ablation

In 3 patients, branches from the circumflex artery supplying the peri-AMC region were suitable for transcoronary ethanol ablation, which was performed with initial success. Two patients with failed endocardial radiofrequency ablation attempts underwent cryosurgery. In 1 patient, no branch supplying the peri-AMC area was identified, and 1 patient had already planned to undergo bypass and aortic valve surgery.

Cryothermal Surgical Ablation

Cryothermal surgical ablation was performed through a median sternotomy in cardioplegia in one and off-pump using a mini-anterior thoracotomy access in the other patient. During the off-pump procedure, VT was inducible. Freezes performed along the right ventricular outflow tract extended toward the septum, targeting the area of presystolic electric activity underneath the left anterior descending coronary artery, rendering the VT noninducible.

In the second patient, simultaneous bypass grafts as well as aortic valve replacement were performed during cardioplegia. Freezes of ~60°C were placed from the aortic annulus along the LVOT toward the septum. Additional freezes were placed from the fibrous trigone of the AMC toward the basal summit of the LV, aiming for the aortic commissures. The freezes were ~2 cm in diameter and showed a substantial overlap when completed.

Thus, in all 21 patients, VT was acutely abolished by radiofrequency ablation in 16, ethanol ablation in 3, and

**Table 2. ECG Characteristics of VTs Originating From the AMC**

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>VTs, No.</td>
<td>21</td>
</tr>
<tr>
<td>Tachycardia cycle length, ms</td>
<td>385±90.4</td>
</tr>
<tr>
<td>Amplitude lead II, mV</td>
<td>1.6±0.72</td>
</tr>
<tr>
<td>Amplitude lead III, mV</td>
<td>1.59±0.85</td>
</tr>
<tr>
<td>Lead II/III amplitude ratio</td>
<td>1.01±0.23</td>
</tr>
<tr>
<td>Precordial transition</td>
<td></td>
</tr>
<tr>
<td>&gt;V₅₆ No. (%)</td>
<td>2 (9.5)</td>
</tr>
<tr>
<td>&lt;V₅₆ No. (%)</td>
<td>19 (90.5)</td>
</tr>
<tr>
<td>QRS duration, ms</td>
<td>165±50</td>
</tr>
</tbody>
</table>

Data are presented as n (%) or mean±SD.
cryothermal surgical ablation in 2 patients, respectively. There were no serious procedural complications.

**Follow-Up**

During a mean follow-up of 1.9±0.8 (range, 0 to 6.8) years, 12 (57.4%) patients remained VT free after the last ablation procedure. In 5 patients, antitachycardia pacing effectively terminated recurrent VTs, whereas 3 patients received further implantable cardioverter-defibrillator shocks. Seven of the 8 recurrences arose from the AMC, based on QRS morphology.

Three patients died during the follow-up period. In 1 of those, no further follow-up of VT recurrence was provided. All patients died had significantly depressed left ventricular function (mean left ventricular ejection fraction, 20.3±5.5%). The underlying disease was dilated cardiomyopathy in 2 patients and coronary artery disease in 1 patient. One patient underwent orthotopic heart transplantation for worsening heart failure but died shortly thereafter. The other 2 patients died of uncertain causes 6 and 17 months after ablation, respectively.

### Table 3. Comparison of Acute Success Between Patients With VTs Arising From the AMC Compared With Patients With VTs From the Free LV Wall

<table>
<thead>
<tr>
<th></th>
<th>AMC Group (n=21)</th>
<th>Comparison Group (n=246)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>58.3±15.3</td>
<td>62.2±10.7</td>
<td>0.24</td>
</tr>
<tr>
<td>Sex, male</td>
<td>17 (81)</td>
<td>200 (81.3)</td>
<td>1</td>
</tr>
<tr>
<td>LV function, %</td>
<td>42.6±16.8</td>
<td>37.3±15.6</td>
<td>0.18</td>
</tr>
<tr>
<td>Acute success</td>
<td>14 (66.7)</td>
<td>201 (81.4)</td>
<td>0.03</td>
</tr>
<tr>
<td>Structural heart disease</td>
<td>21 (100)</td>
<td>246 (100)</td>
<td></td>
</tr>
<tr>
<td>Ischemic cardiomyopathy</td>
<td>7 (33.3)</td>
<td>139 (56)</td>
<td>0.066</td>
</tr>
<tr>
<td>Idiopathic cardiomyopathy</td>
<td>10 (47.6)</td>
<td>42 (16.9)</td>
<td></td>
</tr>
<tr>
<td>Valvular disease</td>
<td>4 (19)</td>
<td>15 (6)</td>
<td></td>
</tr>
<tr>
<td>Other</td>
<td>0</td>
<td>52 (19.3)</td>
<td></td>
</tr>
</tbody>
</table>

Data are presented as n (%) or mean±SD. P value comparing etiology of structural heart disease reflects 2×2 χ² test between ischemic and nonischemic cardiomyopathy.

**Evaluation of Normal Coronary Angiograms**

In the present series, transcorynary ethanol ablation was performed in 3 patients. To evaluate the potential for this procedure, angiograms of 50 patients with normal coronary arteries who underwent coronary angiography in our institution were reviewed. In 29 (58%) studies, branches predominately from the circumflex artery (in 1 case a branch from the left anterior descending coronary artery) were present supplying the peri-AMC region (Figure 6).

**Discussion**

In contrast to patients with idiopathic VT that can arise from the AMC region, all of our patients had structural heart disease, and evidence of scar serving as a potential substrate for reentrant VT was found in 90% of these patients. Ablation was found to be more difficult and less successful compared with VTs arising from the LV free wall. The peri-AMC region was therefore identified as an uncommon but challenging origin of VT in patients with structural heart disease.

The AMC is a recognized, potentially arrhythmogenic area of the heart that can give rise to atrial and ventricular arrhythmias, which was thought to mainly consist of fibrous tissue. McGuire et al found cells histologically and electrophysiologically resembling AV junctional cells in this region, and it has been speculated that these cells may contribute to some idiopathic VTs.

Proximity to the AMC is consonant with reports suggesting that reentry circuits supporting scar related VTs tend to develop adjacent to the valve annuli, as observed at tricuspid, mitral, pulmonic, and now, aortic annular regions. It is conceivable that in the remaining patients without identifiable scar along the peri-AMC region, triggered automaticity plays a role as a VT mechanism, as found in structural normal hearts.

The structural and anatomic particularities of the myocardium surrounding the AMC may explain failures of ablation procedures in patients with structural heart disease, with multiple procedures required in the majority of our patients including epicardial, surgical, and transcorynary ethanol ablation. As demonstrated in the electroanatomic maps, the myocardium in this region can be thick, allowing deep, intramural circuits that are not interrupted by ablation from the endocardium or epicardium. Furthermore, the presence of fibrous tissue in the peri-AMC region may also contribute to protection of the areas of slow conduction responsible for maintenance of the reentry circuit and therefore increase the difficulty of ablation.

Mapping from the GCV can be helpful to identify potential epicardial targets. However, ablation from this side can be limited by restricted flow rates and high temperatures using externally irrigated radiofrequency energy. Furthermore, epicardial ablation may be constrained by the proximity to coronary arteries. Our suggested approach to mapping and ablation of this VT is to assess the timing and postpacing interval during entrainment from the right ventricular outflow tract and then the GCV via the coronary sinus. If the area of the great cardiac vein is in the circuit, ablation may be attempted at that site if it is not in contact with the circumflex coronary artery. Next, mapping of the aortic root and LVOT.
is performed via a retrograde aortic approach. If a desirable and successful ablation target is not identified in those areas, epicardial mapping after percutaneous pericardial access should be considered. However, the use of transcoronary ethanol ablation and surgical mapping and ablation is reserved for patients who failed other approaches and continue to have severe symptoms.

An AMC origin for VT can be suspected based on the QRS morphology, as suggested by studies in structural normal hearts. Using pace-mapping as a guide, Dixit et al. found that a qR pattern in lead V1 was specific for pacing at the AMC. In our population, similar to that of Kumagai et al., the qR pattern was not sensitive; only 5 of 21 (23.8%) VTs showed a qR pattern in the 12-lead ECG. The regional characteristics of the AMC and likely larger size of scar-related reentry compared with the origins of idiopathic VT may make the ECG a less reliable guide to VT arising from this area. The adjacent myocardium encompasses a wide area including septum, anterior and inferior left ventricular wall, with different potential paths for ventricular wave front propagation away from the area. Areas of scar and slow conduction in patients with structural heart disease may further influence ventricular activation and QRS morphology.

Because anatomic challenges might be encountered during attempts of radiofrequency ablation for VTs arising from the peri-AMC region, we considered transcoronary ethanol ablation as an alternative approach after failed endocardial and epicardial radiofrequency catheter ablation. This approach was useful in 3 patients, although VT later recurred in 1 patient. In normal angiograms, we found that the majority of patients have a visible artery supplying the region. Thus, coronary ethanol ablation can be considered, but it probably has risks of artery perforation, atrophicventricular block, and mechanical dysynchrony and the potential for worsening heart failure if a large area of infarction occurs. Better methods to verify that the putative branch supplies the VT circuit would be useful.

**Limitations**

Although the acute success rate was lower than for VTs originating in the free wall of the left ventricle, we cannot exclude the possibility that this is due to other unrecognized confounding factors. It is possible that the referral nature of our patient population introduces bias toward arrhythmias that are more difficult to control and ablate. Failure of ablation may indicate that we did not accurately identify the VT origin. However, we were acutely successful for all VTs, suggesting that the AMC region ablation site was close to the VT origin.

**Conclusion**

The region below the AMC is an uncommon but potentially important location for VT in patients with structural heart disease. Ablation is often acutely successful, but recurrence is common. Cryosurgery and transcoronary ethanol ablation are feasible in some patients. Better technologies are needed to target and ablate VTs from this challenging area.

**Sources of Funding**

Dr Steven is the recipient of a research grant from Biosense-Webster. Dr Seiler is the recipient of a research grant from St Jude Medical (Switzerland). Dr Roberts-Thomson is the recipient of an Overseas Based Clinical Research Fellowship from the National Health and Medical Research Council of Australia (grant 489419).

**Disclosures**

Dr Stevenson received consulting fees from Biosense Webster, Inc, and speaking honoraria from Biosense Webster, Inc, Medtronic, Inc, Boston Scientific, Inc, and St Jude Medical, Inc. Dr Tedrow received speaking honoraria from Medtronic, Boston Scientific, Inc, and St Jude Medical, Inc, and research grants from Biosense Webster and Boston Scientific.

**References**


**CLINICAL PERSPECTIVE**

Ventricular tachycardias (VTs) with a surface ECG morphology suggesting a left ventricular outflow tract origin may successfully be ablated at different locations including the basal septum, the epicardium, the aortic root, and the mitral annulus. More recently, the area along the aortic and mitral valve continuity (peri-AMC region) has been identified as a possible origin of idiopathic VTs arising from the left ventricular outflow tract. The present study identifies VTs arising from this area in patients with structural heart disease. It was found that the surface ECG morphology may show more variation than is reported from patients with idiopathic VTs arising from this area. Therefore, the region around the AMC should be considered as a potential ablation site in VTs showing a left ventricular outflow tract morphology in patients with structural heart disease. The acute success rates for catheter ablation of VTs from the peri-AMC region were found to be lower as compared with VTs originating from the left ventricular free wall. In 5 of 21 patients analyzed, catheter ablation failed and transcoronary ethanol ablation (n = 3) or cryosurgery (n = 2) had to be performed to abolish the peri-AMC VT. During analysis of 50 coronary angiograms of patients without prior ablation and structurally normal hearts, in 29 patients a high septal branch of the circumflex artery was identified, potentially serving as a target vessel for transcoronary ethanol ablation for VTs originating from the peri-AMC region. In patients who fail these approaches, cryosurgery can be considered as a treatment option.
Ventricular Tachycardia Arising From the Aortomitral Continuity in Structural Heart Disease: Characteristics and Therapeutic Considerations for an Anatomically Challenging Area of Origin

Daniel Steven, Kurt C. Roberts-Thomson, Jens Seiler, Keiichi Inada, Usha B. Tedrow, Richard N. Mitchell, Piotr S. Sobieszczyk, Andrew C. Eisenhauer, Gregory S. Couper and William G. Stevenson

Circ Arrhythm Electrophysiol. 2009;2:660-666; originally published online September 10, 2009; doi: 10.1161/CIRCEP.109.853531

Circulation: Arrhythmia and Electrophysiology is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2009 American Heart Association, Inc. All rights reserved.
Print ISSN: 1941-3149. Online ISSN: 1941-3084

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circep.ahajournals.org/content/2/6/660

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation: Arrhythmia and Electrophysiology can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation: Arrhythmia and Electrophysiology is online at:
http://circep.ahajournals.org/subscriptions/