Ventricular tachycardia arising from the aortomitral continuity in structural heart disease: Characteristics and therapeutical considerations for an anatomically challenging area of origin

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AMC – aortomitral continuity
CRT – cardiac resynchronization therapy
ECG – electrocardiogram
GCV – great cardiac vein
LCC – left coronary cusp
LVOT – left ventricular outflow tract
NCC – non coronary cusp
PVC – premature ventricular complex
RCC – right coronary cusp
RF – radiofrequency
TCEA – transcoronary ethanol ablation
VT – ventricular tachycardia
Abstract

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 two patients underwent cryosurgical ablation. Our first catheter ablation procedure was less often successful (66.7%) for peri-AMC VTs as compared to 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 one 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 to VTs originating from the free LV wall. This region provides unique challenges for RF ablation, but cryosurgery and transcoronary alcohol ablation appear feasible in some cases.

Key words: ablation; alcohol; angiography; catheter ablation; tachyarrhythmia
Introduction

Ventricular tachycardias (VT) with a QRS morphology consistent with a left ventricular outflow tract (LVOT) origin may arise from various anatomical sites surrounding the LVOT. 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). 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. In the present study we report VT 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.

Methods

Patients

A total of 21 patients with recurrent VT originating at the peri-AMC region were identified from retrospective review of 550 consecutive patients undergoing VT ablation between May 2000 and May 2008. 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. 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. 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, WI). Nonfluoroscopic electroanatomic mapping was performed using a three-dimensional mapping system (CARTO®, Biosense-Webster, Inc, Diamond Bar, CA). 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. 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. When VT was unstable for mapping, exit sites were targeted on the basis of the QRS morphology during pacing. The following ablation catheters were used: a non-irrigated 4-mm tip 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, MA) 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.
Following ablation, programmed right ventricular stimulation was performed with up to three extrastimuli and burst pacing and during administration of isoproterenol in selected cases. The acute success rates during the first procedure were compared to 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) gender 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) RF 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 post-pacing interval) or later activation. VT storm was defined as occurrence of > 3 VT episodes in a 24 hour period.

Coronary angiograms

After recognizing that RF 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 two 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 ± standard deviation (SD), or as median and interquartile range if appropriate. For comparing VTs arising from the LV free wall and the peri-AMC region, the $\chi^2$ test for categorical and t-test for continuous data were used where appropriate. Tests were performed two-tailed, and p-values < 0.05 were considered statistically significant. The statistical analysis was performed using SPSS 16.0 for Macintosh (SPSS Inc., IL).

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 ICD implantation, of which 5 (31.3%) had a CRT-D device for biventricular pacing. Twelve of the patients were receiving beta blockers. Patients were receiving a mean of 1.6 ± 0.8 antiarrhythmic drugs at the time of the procedure (Table 1).

The reason for referral was electrical storm in 6 (28.6%) patients, repetitive ICD 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 0-5) VT episodes within the last 7 days prior to 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 (Figure 2 and 3), and these had a mean cycle length of 395 ± 88 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]).

At the successful ablation site, the earliest local activation preceded the QRS onset by a mean of 48 ms (range 23-157 ms) and showed a mean electrogram amplitude of 0.73 mV (0.12- 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 (2-52 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 three 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 in all VTs arising from the AMC consistent with its superior location in the basal LV. The QRS duration was 165 ± 47 ms (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 1-3) procedures. Endocardial mapping was initially performed in all patients. Fourteen patients (66.7%) had a second procedure either due to late recurrence or initial failure to successfully ablate the AMC VT. In 6 (28.6%) of those a repeat endocardial and in another 6 (28.6%) an epicardial RF ablation was performed. Two (9.5%) patients underwent a transcoronary ethanol ablation during their second procedure. In four (19.1%) patients a third procedure was performed, consisting of endocardial RF ablation in one, transcoronary ethanol ablation in one and cryothermal surgical ablation in two patients.

Endocardial RF ablation alone was acutely successful in 11 patients (52.4%). The success rate after a single procedure was 33.3% (n=7). RF 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 RF ablation

In nine 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 six patients in two of whom the VT was successfully abolished from this site. In the other four patients earliest activation in the GCV lead to mapping using a subxiphoidal epicardial access with successful ablation in three patients.

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

Transcoronary ethanol ablation
In three 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 RF ablation attempts underwent cryosurgery. In one patient no branch supplying the peri-AMC area was identified and one 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 were performed along the right ventricular outflow tract extended towards the septum targeting the area of presystolic electrical activity underneath the left anterior descending coronary artery rendered the VT non-inducible.

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

Thus, in all 21 patients VT was acutely abolished by RF ablation in 16, ethanol ablation in 3 and 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 0-6.8) years, 12 (57.4%) patients remained VT free after the last ablation procedure. In 5 patients, antitachycardia
pacing effectively terminated recurrent VTs, while 3 patients received further ICD shocks. Seven out of the 8 recurrences arose from the AMC based on QRS morphology.

Three patients died during the follow-up period. In one 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 one patient. One patient underwent orthotopic heart transplantation for worsening heart failure, but died shortly thereafter. The other two patients died of uncertain causes 6 and 17 months post ablation, respectively.

Evaluation of normal coronary angiograms

In the present series, transcoronary 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 predominantly from the circumflex artery (in one 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 to 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,\textsuperscript{18} and it has been speculated that these cells may contribute to some idiopathic VTs.\textsuperscript{1, 5, 7}

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.\textsuperscript{19} 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 anatomical 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 transcoronary 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 endo- 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 RF 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 post-pacing interval during entrainment from the RVOT and then the great cardiac vein 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.¹⁵ In
our population, similar to that of Kumagai et al., the qR pattern was not sensitive;¹
only 5 out 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 as compared
to 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.

Since anatomic challenges might be encountered during attempts of RF ablation
for VTs arising from the peri-AMC region, we considered transcoronary ethanol
ablation as an alternative approach after failed endo- and epicardial RF catheter
ablation.¹⁷ 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
likely has risks of artery perforation, atrioventricular block and mechanical
dissynchrony, 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 more difficult to control and ablate arrhythmias. 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 as well transcoronary ethanol ablation are feasible in some patients. Better technologies are needed to target and ablate VTs from this challenging area.

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References


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<tr>
<td>Patients – number</td>
<td>21</td>
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<tr>
<td>Age – years</td>
<td>62.7 ± 11</td>
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<td>Males – number (%)</td>
<td>17 (81)</td>
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<td>Total Procedures – number</td>
<td>39</td>
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<td>VTs induced – number</td>
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<tr>
<td>AMC VT cycle length – ms</td>
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<td>Left ventricular ejection fraction – %</td>
<td>43.6 ± 17</td>
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<td>Enddiastolic left LV diameter – cm</td>
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<td>Interventricular septum thickness – cm</td>
<td>1.1 ± .3</td>
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<td>Structural heart disease – number (%)</td>
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<tr>
<td>- Ischemic cardiomyopathy</td>
<td>7 (33.3)</td>
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<tr>
<td>- Idiopathic cardiomyopathy</td>
<td>10 (47.6)</td>
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<td>- Valvular disease</td>
<td>4 (19)</td>
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<td>- Anterobasal</td>
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<tr>
<td>- Inferior</td>
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<tr>
<td>Procedure duration – minutes</td>
<td>230 ± 62.4</td>
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<td>Fluoroscopy – minutes</td>
<td>42.1 ± 19.3</td>
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<td>- other - number</td>
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<td>- Amiodarone</td>
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**Table 1:** Patients’ and procedural characteristics
**ECG characteristics of VTs originating from the AMC**

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<td>TCL – ms</td>
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<td>Amplitude lead II – mV</td>
<td>1.6 ± .72</td>
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<td>Amplitude lead III – mV</td>
<td>1.59 ± .85</td>
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<td>Lead II / III amplitude ratio</td>
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<td>Precordial Transition</td>
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<tr>
<td>&gt;V2 - number (%)</td>
<td>2 (9.5)</td>
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<tr>
<td>&lt;V2 - number (%)</td>
<td>19 (90.5)</td>
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<td>QRS duration – ms</td>
<td>165 ± 50</td>
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**Table 2: ECG characteristics of VTs originating from the AMC**

![Circulation Arrhythmia and Electrophysiology](image)

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<table>
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<th>AMC group (n= 21)</th>
<th>Comparison group (n= 246)</th>
<th>p-value</th>
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<td>Age -years</td>
<td>58.3 ± 15.3</td>
<td>62.2 ± 10.7</td>
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<td>Gender -male (%)</td>
<td>17 (81)</td>
<td>200 (81.3)</td>
<td>1</td>
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<tr>
<td>LV function -%</td>
<td>42.6 ± 16.8</td>
<td>37.3 ± 15.6</td>
<td>.18</td>
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<td>Acute success -number (%)</td>
<td>14 (66.7)</td>
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<tr>
<td>• other</td>
<td>0</td>
<td>52 (19.3)</td>
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P-value comparing etiology of structural heart disease reflects 2x2 chi square test between ischemic and non-ischemic cardiomyopathy.

Table 3: Comparison of acute success between patients with VTs arising from the AMC as compared to patients with VTs from the free LV wall.
Figures Legend

Figure 1: Gross anatomy of the heart: The left panel (a) demonstrates a view from the atria towards the valvular apparatus showing the anatomical relationship between aortic (AV), mitral (MV), pulmonary (PV) tricuspid valve (TV). The asterisks indicate the left and the rights atrial appendage, respectively. The white triangle represents the area of the AMC corresponding to Figure 2. The right panel (b) shows a view onto 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. The arrow indicates the left main artery (LCC= left coronary cusp, NCC= noncoronary cusp, RCC= right coronary cusp).

Figure 2: The figure shows the LVOT as viewed from its ventricular aspect. The triangular shaped peri-AMC region between the left coronary cusp (LCC) and the anterior leaflet of the mitral valve is shown. It can be appreciated that strands of fibrous tissue (white) extend from the annulus of the valvular apparatus towards the base of the trigone.

Figure 3: Catheter position during ablation of an AMC related VT (left panel (a) LAO 45°, right panel (b) RAO 30°). The map catheter (1) is placed on the AMC using a retrograde transaortic approach. (Catheter position: 2= Coronary sinus, 3= Right ventricular outflow tract, 4= Right atrium, 5= Right ventricle, 6= Ultrasound probe positioned in the right atrium) This patient had idiopathic VT, but images demonstrate the anatomy.

Figure 4: Schematic delineation of the region adjacent to the AMC (ventricular view): The figure demonstrates the anatomical relation of aortic, mitral and tricuspid valve. The shaded gray triangle represents the peri-AMC region.
The asterisks represent the approximate locations of the successful ablation sites of each VT.

Figure 5: Examples for QRS morphologies found in this study cohort. A II/III ratio > 1 was found in all patients as well as a positive concordance from V2 through V6. A variation in QRS morphologies was noted throughout the patients with AMC VT in the setting of structural heart disease.

Figure 6: Coronary angiogram (left panel (a) LAO 34°, CAU 34°, right panel (b) RAO 22°, CRAN 22°): The figure shows a normal coronary angiogram of the left system. The valvular apparatus is indicated with dotted lines (AV= aortic valve, MV= mitral valve). The asterisk indicates a branch from the circumflex artery potentially suitable for transcoronary ethanol ablation for VTs arising from the AMC.
Ventricular Tachycardia Arising from the Aortomitral Continuity in Structural Heart Disease: Characteristics and Therapeutical Considerations for an Anatomically Challenging Area of Origin

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