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Arrhythmia (D Spragg, Section Editor)

Ablation of Outflow Tract Ventricular Tachycardia

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Opinion statement

Ventricular tachycardia in patients with structurally normal hearts is most frequently due to adenosine-sensitive, triggered activity. The most common sites of origin are the right and left ventricular outflow tracts. Patients may present with symptoms such as palpitations, or less commonly cardiomyopathy. Treatment options include beta blockers, calcium channel blockers, sodium channel blockers, potassium channel blockers, and catheter ablation. Catheter ablation is highly effective and when performed by a skilled electrophysiologist, can be considered first-line treatment. Knowledge of outflow tract and surrounding anatomy is vital to optimizing results. In this review, we discuss outflow tract anatomy and electrocardiographic morphology, as well as techniques for optimizing ablation outcomes.

Introduction

Ventricular tachycardia (VT) most often occurs in the setting of structural heart disease, but in 10 % of patients, it occurs in structurally normal hearts and is termed idiopathic [1]. Idiopathic VT most commonly originates from the right and left ventricular outflow tracts (RVOT and LVOT). The anatomic relationships of the RVOT and LVOT to each other and to their surrounding structures are complex. With a detailed understanding of this anatomy, the 12-lead electrocardiogram (ECG) can be used to localize the arrhythmia site of origin and ablation outcomes can be optimized.

Mechanism of outflow tract VT

The mechanism of outflow tract VT is triggered activity resulting from delayed afterdepolarizations, often occurring in high catecholamine states, such as exercise. Burst pacing and isoproterenol infusion are useful techniques for inducing outflow tract VT in the electrophysiology laboratory. The delayed afterdepolarizations result from intracellular calcium release, which is blunted by adenosine [2]. These arrhythmias often terminate with administration of adenosine or calcium channel-blockers.

Clinical presentation

Outflow tract VT classically manifests in younger patients, usually in the third to fifth decade of life [3, 4]. Specific arrhythmia manifestations include premature ventricular contractions (PVCs), salvos of nonsustained VT and sustained VT. Common symptoms include palpitations, chest discomfort, and lightheadedness. Syncope is infrequent and should raise suspicion of structural heart disease or an additional arrhythmia process. Sudden death is exceedingly rare.

High PVC burdens can cause left ventricular dysfunction [5]. Risk for development of cardiomyopathy is generally thought to begin at 20 %, though cardiomyopathy less frequently occurs with lower burdens [6]. Additional risk factors for the development of PVC-induced cardiomyopathy include wider PVC QRS duration, greater coupling interval dispersion, as well as epicardial, RV (as opposed to LV), and non-OT (as opposed to OT) sites of origin [7–10]. The efficacy of catheter ablation is superior to antiarrhythmic medications in the treatment of PVC-induced cardiomyopathy [11•]. Elimination of PVCs typically results in improved LV dimensions, ejection fraction, and mitral regurgitation within several months [12]. However, full recovery has been reported to occur several years after ablation [13]. Although complete elimination of PVCs is optimal, we have found that significant (>80 %) reduction in PVC burden is usually sufficient to lead to recovery [14].

Sudden death risk and distinction from arrhythmogenic right ventricular cardiomyopathy

Sudden death associated with idiopathic outflow tract VT is very rare. However, it is important to differentiate patients with true idiopathic VT from those with scar-based outflow tract VT, such as arrhythmogenic right ventricular cardiomyopathy (ARVC) because the latter patients are at increased risk for lethal arrhythmias and may therefore benefit from implantable cardioverter-defibrillator implantation. In both substrates, VT commonly originates from the RVOT, with a left bundle configuration in lead V1 and inferior axis. A number of ECG signs have been described to distinguish ARVC from idiopathic RVOT VT, including a wider, more notched QRS complex with later precordial transition [15•, 16, 17]. Even when these signs are absent, scar-based VT should be suspected when multiple different morphologies are observed. Cardiac magnetic resonance imaging and signal-averaged ECG are useful for detecting structural and electrical abnormalities respectively [18, 19]. Voltage mapping is usually definitive.

Anatomic and electrocardiographic characteristics of the ventricular outflow tracts

Anatomic orientation of the outflow tracts

Normally, the RVOT lies anterior to the LVOT as it courses superiorly and leftward to connect to the pulmonary artery. The LVOT is located posteriorly and courses rightward to connect to the aortic root. The pulmonic valve is superior, anterior, and leftward to the aortic valve. The aortic root is centrally located and in direct contact with both atria, the interatrial septum, the mitral valve, and the posterior RVOT (Fig. 1) [20]. It has been well described that myocardial sleeves can extend above the pulmonic and aortic valves into the great vessels, and may be the source of arrhythmias [21]. In nearly three-fourths of patients, sleeves of myocardium extend above the pulmonic valve into the

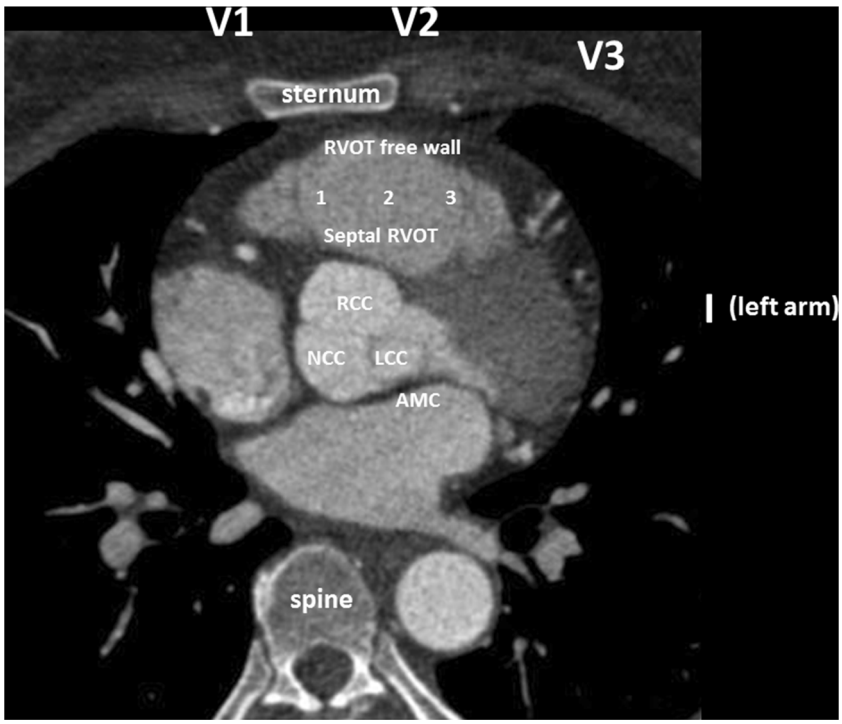

Fig. 1. Anatomy of the ventricular outflow tracts and relationship to electrocardiogram morphology. The free wall of the RVOT lies anteriorly within the chest, just beneath the sternum and precordial ECG leads. The electrocardiographic forces generated by ventricular tachycardia from the free wall of the RVOT are negative through much of the precordium, as the activation wavefront moves away from the anterior chest wall. As one progresses posteriorly toward the spine, from the septal RVOT to the right coronary cusp, left coronary cusp, aortomitral continuity, and superior mitral annulus, the activation wavefront moves increasingly toward the precordial leads, generating a greater degree of positivity in lead V1 as well as an earlier precordial transition. We divide the RVOT into sites 1 (*most rightward*), 2, and 3 (*most leftward*). Site 1 of the RVOT generates a positive complex in limb lead I, as the wavefront moves toward the left arm. By site 3 of the RVOT, the wavefront moves away from the left arm, generating a negative complex in lead I. *RVOT* right ventricular outflow tract, *RCC* right coronary cusp, *LCC* left coronary cusp, *NCC* noncoronary cusp, *AMC* aortomitral continuity.

pulmonary artery [22]. Myocardial sleeves similarly extend above the aortic valve into the aortic root in over one-half of patients, most commonly in the right (55 %) and left (24 %) coronary cusps, rarely in the noncoronary cusp (<1 %).

Electrocardiographic localization of outflow tract VT

As the outflow tracts are superiorly located, positivity in the inferior ECG leads (II, III, and aVF) is the rule. The RVOT free wall is situated most anteriorly within the chest (Fig. 1), just beneath the sternum and precordial ECG leads. As one progresses posteriorly, the septal RVOT is encountered next, followed by the right coronary cusp, left coronary cusp, aortomitral continuity, and superior mitral annulus. The RVOT free wall is therefore the most negative in the precordium, with a left bundle configuration in lead V1 and precordial transition at V4 or later. As one moves posteriorly toward the spine, greater degrees of positivity are observed in lead V1 and the precordium transitions earlier. The precordial transition is typically V3 or V4 for the septal RVOT, V2 or V3 for the right coronary cusp, V2 or right bundle configuration in lead V1 for the left coronary cusp, qR configuration in lead V1 for the aortomitral continuity and positivity throughout the precordium for VT originating from the superior mitral annulus.

These criteria assume accurate positioning of the ECG leads. We have shown that placing leads V1 and V2 too high on the chest results in excess negativity, while placing them too low results in excess positivity [23].

Distinguishing RVOT from LVOT sites of origin

The ability to accurately distinguish RVOT from LVOT sites of origin is desirable to counsel patients about risk of systemic embolism and to decide whether to insert an arterial sheath. VTs with precordial transition at V3 can originate from the septal RVOT or right coronary cusp. We have found that adjusting the VT precordial transition for the sinus rhythm precordial transition is useful for distinguishing these two sites of origin. We do this by calculating the V2 transition ratio, dividing the R/(R+S) during VT by the R/(R+S) during sinus rhythm [24•]. A V2 transition ratio >0.6 is highly specific (91 %) and sensitive (100 %) for an LVOT site origin. Interestingly, the V3 transition ratio was not useful for distinguishing RVOT from LVOT sites of origin for VT transitioning at V3. Others have proposed different algorithms for distinguishing RVOT from LVOT sites of origin, all of which are based on greater degrees of precordial positivity for LVOT sites of origin [25, 26].

In our experience, VT often originates between the septal RVOT and right coronary cusp and can be eliminated with ablation from either side. Alternatively, ablation may be required from both sides to eliminate VT.

RVOT VT

The borders of the RVOT are the pulmonic valve (superiorly), tricuspid valve (inferiorly), interventricular septum (medially), and RV free wall (laterally). The majority (>80 %) of RVOT tachycardias originate along the septum, just beneath the pulmonic valve [27]. We divide the RVOT into free wall and septal aspects, and describe the most rightward extent as site 1 (positive in lead I, Fig. 1 and

Table 1. Electrocardiographic morphology of outflow tract ventricular tachycardias

Site of Origin	Lead V1	Precordial Transition	Lead I	Additional
RVOT free wall	–	≥V4	Site 1 + Site 2 +/– Site 3 –	Notching in inferior leads
Septal RVOT	–	V3 or V4	Site 1 + Site 2 +/– Site 3 –	
Right coronary cusp	–	V2 or V3	+	Notching in downstroke of V1 suggests junction of RCC and LCC
Left ventricular summit	–	V2 or V3	– or +/–	Pattern break in V2 with more net negativity than V1 or V3
Left coronary cusp	+/–	+ throughout or V2	–	M or W configuration common in V1
Aortomitral continuity	qR	+ throughout	+/–	
Superior mitral annulus	+	+ throughout	Septal + Lateral –	

All outflow tract ventricular tachycardias are positive in the inferior leads (II, III, and aVF)
+positive; +/–isoelectric or biphasic; –negative

Table 1), the middle as site 2 (biphasic or isoelectric in lead I), and the most leftward extent as site 3 (negative in lead I). Beneath sites 1, 2, and 3 are sites 4, 5, and 6 [28]. VT originating from the RVOT free wall has notching in the inferior leads and a later precordial transition than VT originating from the septal RVOT.

LVOT VT

LVOT VT can originate from the left ventricular endocardium, the aortic root, and the epicardial LV summit. As one moves along the superior mitral annulus from the septum to lateral wall, the QRS complex widens, with lead V1 becoming more positive and lead I more negative [29].

VT commonly originates from the right and left coronary cusps. The right coronary cusp is in contact with the top of the left ventricular septum. A wide range of ECG morphologies can be observed from the right coronary cusp, though typically the axis is leftward and inferior. As patients age, the aorta may uncoil and contact the septum more inferiorly, leading to less positivity in the inferior leads. The precordial transition is earlier in the left coronary cusp, often with a W or M shaped pattern in lead V1 [30]. VT frequently originates from the commissure between the right and left coronary cusps [31]. The electrocardiographic signature is notching in the downstroke of V1.

Very rarely, VT may originate from the noncoronary cusp. In one series of 90 consecutive patients undergoing ablation of VT in the aortic root, six VTs (7 %) originated from the noncoronary cusp [32]. These patients were younger with narrower VT QRS complexes and smaller III/II ratios than those with VT from

the right coronary cusp.

VT can also originate from the LV summit, the superior most portion of the epicardium, near the bifurcation of the left main coronary artery. This location is best accessed through the coronary venous system. Epicardial mapping and ablation are frequently limited by overlying fat. LV summit VT typically has a left bundle configuration in lead V1, with early precordial transition and a pattern break in V2, with more net negativity than lead V1 or V3. Criteria have been proposed to distinguish VT that is accessible for ablation from VT that originates too close to the bifurcation of the left main coronary artery to be safely targeted [33, 34]. As one moves laterally from the anterior interventricular vein to the great cardiac vein, lead V1 develops a right bundle configuration and lead I a qS pattern.

Treatment of outflow tract VT

Medications

Treatment of outflow tract VT is aimed at alleviating symptoms or reversing PVC-induced cardiomyopathy. Because the underlying mechanism of arrhythmogenesis involves triggered activity from delayed afterdepolarizations, which is mediated by cAMP and intracellular calcium, medications that alter the cAMP-mediated calcium influx such as adenosine, calcium channel blockers, and beta-blockers are often effective in acutely terminating VT. For long-term VT suppression, beta-blockers and calcium channel blockers are most commonly prescribed, with modest efficacy in the 25 %–50 % range [35, 36]. Sodium channel blockers are slightly more effective, with 55 % success rate in one comparative study [37]. Potassium channel blockers are used less frequently.

Catheter ablation

Catheter ablation is generally a safe and effective method to permanently cure outflow tract tachycardias and should be considered a reasonable first-line treatment [38•]. When performed by an experienced operator, success rates are in excess of 90 %, with an acceptably low rate of serious complications (approximately 1 %) [39].

To enhance the likelihood of successful ablation, it is important to maximize PVC frequency during the ablation. We typically discontinue beta blockers, calcium channel blockers, and other antiarrhythmic medications several days before ablation. Amiodarone is ideally discontinued several weeks beforehand. As idiopathic VT is sensitive to catecholamine state, we limit the use of propofol and benzodiazepines. Instead, we favor remifentanyl, which is rapidly eliminated following discontinuation and has fewer antiarrhythmic properties [40]. Isoproterenol infusion and burst pacing are used when VT remains infrequent.

We carefully examine the ECG to anticipate the site of VT origin. A three-dimensional electroanatomic map of that chamber is created, under fluoroscopic and intracardiac echocardiographic (ICE) guidance. We have found ICE

to be extremely valuable for understanding anatomic relationships, guiding catheter position, and avoiding/detecting complications. In addition to guiding ablation, voltage mapping identifies myocardial scarring, differentiating idiopathic VT from scar based arrhythmias such as ARVC [41].

When PVCs or VT are frequent enough, we perform activation mapping to identify the site of earliest activation, which should be well before the onset of the QRS complex. The unipolar electrogram should have a qS morphology and simultaneous onset with the bipolar electrogram. When a diffuse area of earliest activation is encountered, one should suspect breakthrough from another chamber, such as the epicardium or contralateral outflow tract [39].

When VT is not frequent enough to activation map, pacemapping is performed at threshold output with the same coupling interval as the spontaneous PVC or cycle length as spontaneous VT. A perfect 12/12 match should be sought. Ablation at sites without perfect 12/12 matches are less likely to be successful [42]. Pacemapping alone results in success rates which are inferior to activation mapping [43]. We have found pacemapping to be highly accurate within the RVOT, but less so within the aortic root, where high pacing outputs are frequently required.

Although either irrigated or nonirrigated ablation is usually sufficient within the RVOT, we exclusively use irrigated ablation within the aortic root and coronary venous system to allow sufficient energy delivery. When ablating within the right or left coronary cusp, care must be taken to define the location of the coronary ostia, either by ICE or angiography, to ensure adequate separation. Coronary injury is uncommon ablating at the bottom of a coronary cusp [44]. Outflow tract VT has rarely been reported to arise from the coronary arteries themselves, and isolation of the coronary ostia has been performed to eliminate these arrhythmia without injuring the artery [45]. The His bundle penetrates the central fibrous body, which is inferior to the noncoronary cusp, so care must be taken when ablating near the junction of the right and noncoronary cusps to avoid damaging the conduction system [46].

Left coronary angiography must be performed prior to targeting LV summit VT from the anterior interventricular vein, to ensure adequate separation. When the VT site of origin is too close, rather than risking injury to the left anterior descending coronary artery, ablation should be attempted from nearby structures including the LV endocardium, left coronary cusp, or septal site 3 of the RVOT [47, 48]. Of note, septal site 3 of the RVOT just beneath the pulmonic valve is close not only to the anterior interventricular vein, but also to the left anterior descending coronary artery, and thus, energy should be delivered judiciously in this location.

If VT is suppressed during ablation but recurs shortly thereafter, the ECG morphology should be reexamined to determine whether the VT exit has changed. For example, after ablating in site 1 of the septal RVOT, the VT exit site may shift to the right coronary cusp. If indeed the ECG morphology has changed, mapping should be repeated, including in adjacent structures. When ablating within a coronary cusp results in suppression and then return of VT, we are often successful ablating the LV endocardium just beneath that

cusp. We consider a procedure successful when VT cannot be induced with isoproterenol infusion and burst pacing at least one hour following the last ablation lesion.

Conclusions

Idiopathic outflow tract VT can cause symptoms or cardiomyopathy. Treatment options include medications and catheter ablation. With a detailed understanding of outflow tract and surrounding anatomy, the ECG can be used to predict the site of VT origin. Catheter ablation is a highly successful and generally safe procedure for eliminating outflow tract VT.

Compliance with Ethics Guidelines

Conflict of Interest
Jackson J. Liang, Yuchi Y. Han, and David S. Frankel all declare that they have no conflicts of interest.

Human and Animal Rights and Informed Consent
This article does not contain any studies with human or animal subjects performed by any of the authors.

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