VIEWPOINT

Electrocardiographic recognition and ablation of outflow tract ventricular tachycardia

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Outflow tract tachycardias comprise a subgroup of idiopathic ventricular tachyarrhythmias (VTs) that occur in the absence of structural heart disease. The arrhythmia mechanism appears to be calcium-dependent triggered activity, and no discrete anatomic abnormalities have been identified. In our experience, most of these outflow tract tachycardias originate from a fairly narrow anatomic zone. These arrhythmias originate in an arc-like fashion from the right ventricular (RV) inflow region to the anteroseptal aspect of the right ventricular outflow tract (RVOT) under the pulmonic valve. The arc then extends leftward to include the cusp region of the aortic valve and the anterior left ventricle (LV) in front of the aortic valve, both endocardially and epicardially, and then further extends toward the aorto-mitral continuity and superior mitral annulus. Most outflow tract tachycardias originate in perivalvular tissue, which may be anatomically predisposed to fiber disruption that enhances arrhythmogenesis. In addition, the proximity of the outflow tract to the epicardial fat pads containing the ganglionated plexuses and the unique response to exercise and hormonal changes suggest that the autonomic nervous system also plays a role in this arrhythmogenesis. Because outflow tract tachycardias tend to occur in the absence of structural heart disease and are focal in origin, the 12-lead ECG recorded during VT is a precise localizing tool. In this review, we highlight the ECG features of outflow tract tachycardias that aid in localization and describe our approach to mapping and ablation.

Important anatomic considerations

The RVOT region is defined superiorly by the pulmonic valve and inferiorly by the RV inflow tract and the top of the tricuspid valve. The lateral aspect of the RVOT region is the RV free wall, and the medial aspect is formed by the interventricular septum at its base of the RVOT and RV musculature opposite the root of the aorta at the region just inferior to the pulmonic valve. From the coronal view above the pulmonic valve, the RVOT region is seen wrapping around the root of the aorta and extending leftward. The top of the RVOT may be convex or crescent shaped, with the posteroseptal region directed rightward and the anteroseptal region directed leftward. Occasionally, an extreme convexity of the superior septal RVOT region creates a leftward direction for the most posterior and anterior aspects of the septal RVOT and results in a net negative QRS complex in lead I from either site. The anteroseptal aspect of the RVOT actually is located in close proximity to the LV epicardium, adjacent to the anterior interventricular vein and in proximity to the left anterior descending coronary artery. The aortic valve cusps sit squarely within the crescent-shaped septal region of the RVOT and are inferior to the pulmonic valve. The posteroseptal aspect of the RVOT is adjacent to the region of the right coronary cusp, and the anterior septal surface is adjacent to the anterior margin of the right coronary cusp or the medial aspect of the left coronary cusp. In young patients, the aortic valve is parallel to the pulmonic valve and perpendicular to the mitral valve. In older patients, the aortic valve may have a more vertical tilt and parallel the mitral valve. In addition, the location, rotation, and horizontal position of the heart in the chest cavity influence surface ECG characteristics. These anatomic considerations are critical in analyzing 12-lead ECG patterns and localizing the site of origin of outflow tract tachycardias.¹

RVOT tachycardias

RVOT tachycardias have a left bundle branch block morphology with a precordial QRS transition that begins no earlier than lead V₃ and more typically occurs in lead V₄. The frontal plane axis, precordial QRS transition, QRS width, and complexity of the QRS in the inferior leads can pinpoint the origin of VT in RVOT. Tachycardias originating from the top of the tricuspid valve in the inflow area of the RVOT, which is lower and to the right in the outflow tract, will have a positive QRS deflection in lead aVL, QRS amplitude in lead II > lead III, and typically a very positive QRS complex in lead I.

A study from our institution further divided the septal and free wall areas into distinct anatomic sites, and pace mapping was performed at each site. The 12-lead ECG QRS

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complex from the pace maps and from spontaneous VT that was successfully localized and ablated were analyzed. The septal and free-wall sites just under the pulmonic valve were designated as site 1 (posterior/right), site 2 (mid), and site 3 (anterior/left) to maintain simplicity with respect to the nomenclature used to describe the anatomic location. The QRS complex changes most notably in lead I when moving from site 3—anterior and leftward (negative QRS in lead I)—to site 1—posterior and rightward (positive QRS in lead I). In contrast to the corresponding septal sites, the free-wall sites demonstrated a later QRS transition, wider QRS duration and notching, and decreased amplitude in the inferior leads and lateral precordial leads.

The most common site of origin for RVOT tachycardias is the left septal side of the outflow tract just underneath the pulmonic valve. These tachycardias produce a characteristic 12-lead ECG appearance with large positive QRS complexes in leads II, III, and aVF and large negative complexes in leads aVR and aVL. The QRS morphology in lead I typically is multiphasic and has a net QRS vector of zero or only modestly positive. Frequently, the VT may originate at the leftward edge of the septal aspect of the outflow tract just under the pulmonic valve. This extreme “leftward” aspect of the RVOT actually produces a negative QRS complex in lead I, consistent with how far this aspect of the RVOT wraps around the aortic root and is located to the left of the apical septum (Figure 1).

Of note, placement of the limb leads on the chest will influence the lead I QRS vector, particularly when VT originates from the right posterior septal RVOT location. Pace maps from this region are positive when the leads are placed on the shoulders and negative when the leads are placed on the chest. This change is likely due to a narrowing of the overall vector of lead I when “limb” leads are placed on the chest, as commonly done with exercise testing. Recognition of the influence of limb lead and precordial lead placement can prevent inaccurate VT localization based on ECG interpretation.

**LVOT tachycardias**

Left bundle morphology VT from the aortic cusp region and top of the LV septum has a precordial transition that is...
earlier than from the RVOT region. The R wave is positive by lead V2 or V3 with VT from the right coronary cusp and by lead V1 or V2 from the left coronary cusp. Ouyang et al noted a broad R-wave duration in lead V1 or V2 in the cusp region vs the RVOT and thus reported an R-wave duration index ≥50% and R/S ratio ≥30% in lead V1 or V2 in their series of seven patients when VT originated from the aortic sinuses. Of note, the noncoronary cusp is notable for atrial capture during pace mapping as it abuts the interatrial septum and may be the source of atrial arrhythmias.

Left coronary cusp VT and VT originating from the endocardium or epicardium just in front of the left coronary cusp is often associated with a W- or M-shaped pattern in lead V1 and thus is difficult to classify as a true left or right bundle branch block pattern. Similar to RVOT tachycardias, VT from the left coronary cusp and usually the right coronary cusp has a tall QRS in the inferior leads if the valve remains parallel to the pulmonic valve. The basis for this tall QRS morphology in the inferior leads probably is the fact that the muscular septum is located below both valve planes and is activated in a similar fashion from both the right and left septal perivalvular structures. As mentioned previously, aging may draw the aortic valve down in a vertical tilt in relation to the pulmonic valve; thus, right coronary cusp tachycardias can exhibit a QRS complex in lead II > lead III and a biphasic (positive/negative) complex in lead aVL. In this situation, the left coronary cusp remains superior and exhibits a positive QRS in the frontal plane axis. Changes in the QRS complex in lead I are important in the aortic cusp region. The left coronary cusp tends to have a QS or rS complex in lead I, whereas the right coronary cusp has greater R-wave amplitude in lead I based on how posterior and rightward the right coronary cusp is positioned. In young patients with a vertical heart, the QRS complex in lead I may be negative in and around both the left coronary cusp and right coronary cusp regions. In patients with a horizontal heart, the area surrounding the aortic valve will be directed rightward relative to the LV apex/lateral wall, and a positive QRS complex in lead I can be seen. Because of the paucity of muscle fibers, pace mapping in the aortic cusp region can be challenging and often requires good contact, stability, high current strength, and placement into

Figure 2  ECG patterns associated with selected outflow tract locations. Twelve-lead ECG pace maps or ventricular tachycardia (VT) from the free wall of the right ventricular outflow tract (RVOT), anteroseptal aspect of the RVOT (typical RVOT VT origin), left coronary cusp, aorto-mitral (A-M) continuity, and superior mitral annulus are shown. As one moves from right to left from the RVOT free wall to the anteroseptum and across the left ventricular outflow tract, it is clear that the precordial transition becomes earlier, with an eventual monophasic R wave in lead V1 from the superior mitral annulus. Note the signature W shape in lead V1 of the left coronary cusp VT and the qR in lead V1 from the aorto-mitral continuity pace map. AV = aortic valve; MV = mitral valve; NC = noncoronary cusp; PV = pulmonic valve.
the base of the sinus. In our experience, pace maps and, as a corollary, VT across a single cusp occasionally vary when one moves right to left for the right coronary cusp and anterior to posterior for the left coronary cusp.

Moving leftward to the aorto–mitral continuity on the endocardium, a qR complex typically is seen in lead V1 and an Rs/rs complex is noted in lead I. With VT originating further leftward across the anterior mitral annulus, the R wave in lead I diminishes and a broad, positive R wave is seen in lead V1 (Figure 2).4

VT from LV epicardial sites of origin, anterior to the aortic valve, is frequently similar to patterns from the left coronary cusp. In a study by Daniels et al.,5 these tachycardias were seen to cluster at perivascular sites at the LV epicardium, with the majority at the junction of the great cardiac vein/anterior interventricular vein and proximal anterior interventricular vein. The majority were notable for a left bundle morphology in lead V1, and all had slurring in the initial portion of the QRS indexed by the maximum deflection index (onset of R or r wave to nadir of S duration in any precordial lead/total QRS duration >55%). In our experience, LV epicardial VT in this region commonly have a QS morphology in lead I and may show a characteristic QRS transition pattern break, with the QRS in lead V2 being less positive or having a smaller R wave than in both lead V1 and V3. This pattern break is noted for VT with a left bundle branch block or slightly more leftward positioned VT with a right bundle branch block QRS morphology (Figure 3).

Successful ablation of outflow tract tachycardias begins with a careful analysis of the 12-lead ECG pattern of the VT, coupled with recognition of the common sites of VT origin. A detailed electroanatomic map can help delineate the anatomic features of the outflow tract (Figures 1 and 3). Pace mapping is performed in regions of interest based on analysis of the 12-lead ECG during VT, with attention to an exact match for all 12 ECG leads. Activation mapping with unipolar and bipolar recordings is used to corroborate the pace map finding when recurrent ventricular premature depolarizations (VPDs) or VT is observed. Intracardiac echo-

Figure 3  Representation of epicardial ventricular tachycardia (VT) from the anterobasal left ventricle (LV). Left: ECG pattern of the epicardial VT from the anterobasal LV. Arrows indicate the typical QS complex in lead I and the characteristic pattern break from leads V1–V3, with relative negativity in lead V3. A delayed or slurred pattern of initial QRS activation is noted. Red arrow identifies the epicardial electrogram recording 40 ms before the QRS. Middle, bottom: Superimposed three-dimensional electroanatomic map of the LV endocardium (ENDO) and epicardium (EPI) shows the earliest activation from the anterobasal epicardium identified by the red color (large black arrow). Middle, top: Fluoroscopic image shows the successful site of VT elimination in proximity to the left anterior descending coronary artery and the catheter tip position corresponding to the exact 12/12 pace map match (right).
cardiography can be used to assess catheter tip position in the aortic cusp region to confirm anatomic location and proximity to coronary anatomy. Prior to ablation in the aortic cusp region and in the LV endocardium/epicardium in front of the aortic valve, angiography usually is performed to assess proximity to the coronary circulation. The area anterior to the aortic valve and next to the proximal anterior interventricular vein is adjacent to the left main/left anterior descending coronary artery bifurcation, and caution must be exercised when ablating in this area (Figure 3). Standard 4-mm-tip radiofrequency ablation is almost uniformly effective if care is paid to precisely localizing the focal origin of the VT for both RVOT and LVOT VTs.

**Conclusion**

ECG recognition of outflow tract tachycardia location allows one to distinguish right from left and epicardial from endocardial VTs. Careful attention to frontal plane axis, precordial QRS transition, QRS width, and complexity of the QRS in the inferior leads can further pinpoint the origin of VT. This analysis is helpful for planning the ablation procedure, counseling patients with regard to risks and anticipated outcomes, and facilitating a nearly uniform successful ablation strategy.

**References**