HANDS ON

How to map and ablate left ventricular summit arrhythmias

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Introduction
Catheter ablation of idiopathic ventricular arrhythmias (VAs) is highly successful, with overall cure rates >90%, and is accepted as a first-line therapy by current guidelines. However, despite the advances in mapping and ablation techniques, there is a percentage of patients in whom successful ablation cannot be achieved because of anatomic limitations. In this regard, one of the most challenging clinical problems that electrophysiologists may face in the laboratory is the approach to VAs arising from the summit of the left ventricle (LV). This region is the highest portion of the LV epicardium, near the bifurcation of the left main coronary artery (LMCA), and accounts for up to 14.5% of LV VAs. The complex relationships between the left ventricular summit (LVS) and surrounding structures underscore the importance of understanding the anatomy of this region and the value of imaging techniques for detailed mapping and safe ablation. In this article, we review the anatomy of the LVS and our approach to mapping and ablating arrhythmias originating from this region.

Anatomic definition
According to the original work of McAlpine, the LVS is defined as the highest point of the LV epicardium, located above both the upper end of the anterior interventricular sulcus and the aortic portion of the LV ostium (Figure 1). It is a triangular region bounded by the bifurcation between the left anterior descending (LAD) and the left circumflex (LCx) coronary arteries and is bisected laterally by the great cardiac vein (GCV) resulting in 2 regions: a medial and more superior region, close to the apex of the triangle, which is inaccessible to catheter ablation because of close proximity to the major coronary vessels and the presence of epicardial fat (inaccessible area); and a more lateral and inferior region, toward the base of the triangle, which may be suitable for catheter ablation (accessible area). Below this epicardial landmark lies the basal portion of the LV ostium next to the septum and in direct contact with the left coronary cusp (LCC). Arrhythmias that originate from this area of myocardium may be targeted from the LVS, the LV endocardium below it, or the LCC. Furthermore, the septal branches of the LAD and the septal veins draining into the anterior interventricular vein (AIV) could be used for mapping and ablation of these arrhythmias.

Anatomic relationships
A detailed understanding of the left ventricular outflow tract (LVOT) anatomy and its relationships is essential for mapping and ablation of LVS arrhythmias. At the base of the heart, the elliptical opening of the LV, also known as LV ostium, provides attachment to the aortic and mitral valves. It is covered by a large fibrous structure, the aortoventricular membrane, which is perforated by the aortic valve anteriorly and the mitral valve (MV) posteriorly. The aortic root is the continuation of the LVOT and occupies a central position within the heart, located to the right and posterior relative to the subpulmonary infundibulum. Its components include the aortic sinuses (sinuses of Valsalva), the valve leaflets or cusps, and the interleaflet triangles. The right coronary cusp (RCC) is the most anterior cusp and lies immediately posterior to the infundibular portion of the right ventricular outflow tract (RVOT). The LCC is posterior and leftward, whereas the noncoronary cusp (NCC) is posterior and rightward, in close proximity to both the left and right atria.

The ventriculo–aortic junction is composed of a fibrous portion and a muscular portion. The muscular portion, which is more extensive, corresponds to the interventricular septum and is under the right coronary sinus and the anterior half of the left coronary sinus. The fibrous portion corresponds to the aorto–mitral continuity, a curtain of fibrous tissue that extends between the anterior leaflet of the MV and...
the noncoronary and left coronary leaflets of the aortic valve. At either end, the aorto–mitral continuity is attached by fibrous expansions, the left and right fibrous trigones, to the ventricular myocardium. The right trigone is continuous with the membranous septum, the combined entity being known as the central fibrous body. The membranous ventricular septum is located just beneath the junction between the RCC and NCC and is the site of the His bundle.

The AIV originates at the lower or middle third of the anterior interventricular groove, follows the groove adjacent to the LAD, and angulates laterally toward the base of the heart to form the GCV. The relationships between the GCV–AIV and the coronary arteries are variable between patients. In the majority of cases, the AIV originates left to the LAD and is adjacent to this vessel, whereas in other cases the AIV originates right to the LAD and then crosses the LAD either superficially or deeply. For its part, the GCV crosses the LCx, forming the triangle of Brocq and Mouchet, composed by the intersection between the LAD, LCx, and GCV. The intersection between the GCV and LCx also is variable, occurring either distally or proximally in the atrioventricular groove, and with the vein crossing superficial or deep to the arterial branch.

The anatomic relationships of the LVS relevant for catheter ablation can be appreciated in a sagittal section of the heart at the level of the muscular portion of the ventriculo-aortic junction (Figure 1). The LCC forms the lateral and posterolateral attachment of the aorta to the LV ostium and is in close proximity to the intramural component of the LVS. The LMCA ostium is located 15 to 20 mm above the nadir of the cusp, and this artery courses laterally between the base of the pulmonary trunk and the left atrial appendage (LAA) before branching into the LAD and LCx. The posterior portion of the LCC is not in direct contact with LV myocardium but apposes the left fibrous trigone, a membranous region joining the LCC to the left side of the anterior MV leaflet. On the other side, the anteroseptal aspect of the RVOT is also adjacent to the LCC, in close proximity to the LAD and AIV, whereas its posteroseptal aspect is adjacent to the RCC.
ECG characteristics

LVS VAs typically have a right bundle branch block pattern with inferior axis and larger R waves in lead III compared to lead II but a left bundle branch block pattern with inferior axis; early transition (V2 or V3) also can be seen.2,6,7 Epicardial origin is suggested by slurring of the initial portion of the QRS complex,8 as reflected by several indexes: (1) time to earliest rapid deflection in precordial leads (pseudo-delta wave) ≥34 ms, (2) interval to peak of R wave in lead V2 (intrinsiod deflection time) ≥85 ms, (3) shortest interval to maximal positive or negative deflection divided by QRS duration (maximum deflection index) ≥0.55 ms, and (4) time to earliest QRS nadir in precordial leads (shortest RS complex) ≥121 ms. A QS pattern in lead I is observed in 30% of patients. In the absence of these classic findings, another particular ECG pattern is the precordial “pattern break” in V2, consisting of an abrupt loss of R wave in lead V2 compared to V1 and V3. This suggests an origin from the septal aspect of the LVS (anatomically opposite to lead V2), usually in close proximity to the LAD9 and less likely to be eliminated from the epicardial aspect.

In patients who undergo percutaneous epicardial mapping after failed ablation from the coronary venous system and LV/RV endocardium, ECG predictors of successful epicardial ablation include Q-wave ratio > 1.85 in aVL/aVR, R/S ratio > 2 in V1, and absence of q waves in V1. All of these ECG features point to a more lateral origin, farther from the more medially located coronary arteries.

Mapping and ablation

The LVS can be accessed directly via percutaneous epicardial puncture, but this approach usually is limited by the proximity to major coronary arteries and the presence of a thick layer of epicardial fat. Rather, LVS VAs are more often targeted from the coronary venous system (AIV/GCV), and because the origin could be intramural below the epicardial LVS, they can also be ablated from adjacent structures such as the LCC, LVOT endocardium, or septal RVOT. Anecdotally, ablation of an LVS ventricular tachycardia (VT) from the LAA has also been reported.10

We propose a comprehensive approach for mapping and ablation of LVS VAs (see Online Supplementary Figure 1). The first step is to suspect an LVOT origin based on careful analysis of the 12-lead ECGs of the clinical VT/premature ventricular complex (PVC). If an LVS origin is suspected, the second step is to define the anatomy of the area of interest, including both outflow tracts as well as the coronary venous circulation. Because the anatomic relationships are highly variable from patient to patient, we believe that detailed characterization of the individual anatomy is essential. By obtaining multiple tomographic views from the right atrium and the right ventricle, a 3-dimensional (3D) anatomic map of the ventricles and outflow tracts is constructed using intracardiac echocardiography (ICE) and displayed with the CartoSound module (Biosense Webster, Diamond Bar, CA). This has the advantage over computed tomography or magnetic resonance imaging in that it is performed online with the patient on the procedure table and immediately integrated into the mapping system, limiting integration resolution errors. In addition, the LMCA ostium and the course of the LAD are located with Doppler flow and reconstructed on the 3D map.

Ideally, the preferred mapping method is activation mapping during spontaneous arrhythmia. In the case of infrequent PVCs, induction of VT or PVCs is attempted with isoproterenol infusion and ventricular or atrial burst pacing. Pace-mapping may be limited in this area because of preferential conduction and the close proximity of multiple structures; thus, the pace-map could be poor even at sites of very early activation or successful ablation. When pace-mapping is used, it should be done at threshold pacing looking for an identical match (>95%).

First step: Mapping the coronary sinus and GCV/AIV

A 4Fr quadripolar catheter (Inquiry, St. Jude Medical, St. Paul, MN) should be placed as distally as possible to compare the activation time of the GCV/AIV region with other sites within the LVOT. We typically start by obtaining access into the coronary sinus with a long deflectable sheath over a guidewire (Agilis or Lamp 90, St. Jude Medical). The sheath allows placement of mapping diagnostic catheters and wires into the venous circulation and then, if needed, interchange of these with ablation catheters. We always perform a venogram using a balloon tip catheter if necessary to delineate the course and caliber of the coronary venous circulation and, of particular interest, the GCV and the AIV with its septal branches.

The intramural component of the LV septum can be mapped by advancing a guidewire into a septal venous perforator branch. We use a 4Fr hydrophilic coated catheter (Glidectah, Terumo Somerset, NJ) to selectively cannulate and perform a venogram of the AIV branches. If a septal perforator branch suitable for mapping is identified, a straight 0.14-inch, 175-cm wire (Visionwire, Biotronik, Berlin, Germany) is placed in this branch and connected in a unipolar fashion to obtain activation information as well as perform pace-mapping (see Online Supplementary Figure 2).

An epicardial LVS origin is suggested by a ventricular activation time at the distal GCV or proximal AIV earlier than in other sites within the LVOT or RVOT, usually with a good pace-map match. Mapping of the septal perforator vein allows one to distinguish VT/PVCs originating in the epicardium (earlier in GCV/AIV) from intramural foci (earlier in septal perforator) and efficiently direct the next mapping efforts accordingly.

Second step: Mapping the coronary cusps

The coronary cusps are mapped next. The mapping catheter is inserted in the right femoral artery and advanced to the aortic root in a retrograde fashion. Intravenous heparin is administered to maintain an activated clotting time >300
seconds. By advancing the ICE catheter into the RVOT base, followed by clockwise rotation, a short-axis image of the aortic cusp region with its characteristic trileaflet appearance is obtained. Activation mapping is performed during spontaneous or induced VAs from the base of the RCC, LCC, and RCC–LCC junction.

**Third step: Mapping the LV endocardium below the LCC**

Subsequently, the ablation catheter is advanced across the aortic valve to map the LV subvalvular endocardium below the LCC. In case of earliest activation in the GCV/AIV, the mapping catheter is placed opposite the best epicardial site marked by a mapping catheter or wire. Use of contact force sensing catheters allows one to monitor optimal contact and adequate orientation of the catheter tip vector. We routinely use a long SL1 or SL0 sheath (St. Jude Medical) in the proximal ascending aorta, which provides excellent catheter control and limits the catheter “pop out” of the LV that occurs frequently when mapping this challenging area. The left anterior oblique (LAO) fluoroscopic view is used to orient the catheter tip toward the lateral base, and the subaortic position is confirmed on ICE by counterclocking the probe in the same position as in step 2 when visualizing the LCC. This opens the LVOT below the LCC and confirms catheter location.

**Fourth step: Mapping the RVOT**

The septal RVOT should also be mapped before radiofrequency (RF) delivery is attempted, especially when very early activation (<20 ms presystolic) has not been found during the previous steps. This is the initial step in cases in which the 12-lead ECG suggests an RVOT origin (left bundle branch block pattern and transition after V3), but this is uncommon in LVS arrhythmias. The anatomic separation between the leftmost aspect of the septal RVOT and the AIV is 10 mm or less, and our group reported 2 cases of between the leftmost aspect of the septal RVOT and the AIV.

**Fifth step: Ablation**

After detailed mapping, a decision is made to ablate the earliest activation anatomic site first; however, not infrequently all the sites are found equally “early” (on time or barely pre-QRS), which suggests an intramural origin.

**Complications**

Procedural complications are infrequent but may be serious. Ablation from the AIV/GCV may result in acute coronary artery injury and thrombosis requiring immediate intervention, or vein perforation resulting in pericardial effusion and
Figure 2  Left ventricular summit premature ventricular complex (PVC). Earliest activation is recorded in the anterior interventricular vein (AIV) (left panel) and ablation is not limited by coronary anatomy (right panel). Radiofrequency (RF) delivery within the AIV eliminated the PVC. As a general rule, RF ablation should be avoided within 5 mm of a coronary artery visualized in at least 2 fluoroscopic projections. LAO = left anterior oblique; RAO = right anterior oblique.

Figure 3  Successful ablation of left ventricular summit (LVS) premature ventricular complex (PVC) from the basal left ventricular endocardium. Earliest activation was recorded at a unipolar wire (arrowhead) advanced into a septal venous perforator (earlier than great cardiac vein/anterior interventricular vein and any endocardial site), suggesting an intramural origin. The ablation catheter was positioned at the left ventricular endocardium, opposite to the site of earliest activation, and radiofrequency delivery successfully eliminated the PVC. Panel A shows the initial CS venogram. In panels B and C we see the position of the catheters in right anterior oblique and left anterior oblique fluoroscopic views, respectively. Panel D shows the CARTO 3-D reconstruction with the ablation catheter and lesion set. Panel E shows the electrograms recorded from the unipolar wire and the ablation catheter, and panel F shows the pacemap obtained by pacing from the unipolar wire.
cardiac tamponade. It should be noted that injury to the LAD may also occur with ablation from the most anterior aspect of the RVOT or above the pulmonic valve. Other potential complications include aortic valve damage resulting from mechanical trauma or RF delivered to the valvular tissue, and embolic events (thrombus or air), which are avoided by careful attention to catheter irrigation and periprocedural anticoagulation.
Summary

The LVS is a common source of idiopathic VAs, and catheter ablation is challenging. The origin of these arrhythmias may be the LVOT epicardium or the intramural myocardium next to the basal septum, and mapping of the proximal septal venous perforator can help to make this differentiation. LVS VAs can be eliminated by ablation from the coronary venous system or from adjacent endocardial structures, including the LCC, basal LV endocardium, or septal RVOT. Ablation from the endocardium is preferred when an intramural origin is suspected or when ablation from the GCV/AIV is unsafe because of close proximity to coronary vessel or not technically feasible because of size or branching.

Appendix

Supplementary data

Supplementary data associated with this article can be found in the online version at http://dx.doi.org/10.1016/j.hrthm.2016.09.018.

References


