

Editor's Perspective: Similia Similibus Curantur
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Similia Similibus Curantur

Samuel J. Asirvatham, MD; William G. Stevenson, MD

In the present installment of the Teaching Rounds in Electrophysiology series, Atienza et al¹ draw our attention to the need for an integrated and innovative approach to help patients with ventricular arrhythmias that involve the summit of the heart. They superbly outline the anatomic basis for both the difficulties encountered when mapping and ablating in this region, as well as the therapeutic options. The anatomic dissection presented in their article illustrates why there is a need for a nuanced and meticulous approach to ablation of arrhythmias in this region.

Article see p e80

Anatomic Summit of the Heart

The highest point of the left ventricular myocardium, sometimes termed the summit, lies on the anterior mitral annulus epicardially just lateral and superior to the aortic annulus. The left coronary cusp is the most cranial of the aortic cusps, and abutting on the cusp superiorly, laterally, and anteriorly is typically thick myocardium constituting the summit. Because the ostium of the left main coronary artery also arises cranial to the cusp, the summit lies in immediate relationship to the 2 principal branches of the left main coronary artery: the left anterior descending and left circumflex arteries. The detailed and highly informative cardiac dissection presented by Atienza et al¹ deserves a close study by electrophysiology students. One will note the arteriovenous triangle (Brocq and Mouchet)² formed by the left anterior descending, left circumflex artery, and the anterior interventricular vein from its confluence with the great cardiac vein as it slopes toward the left border of the right ventricular outflow tract. The summit region can be mapped or potentially ablated through these vessels or via epicardial access. It is important to note, however, that the structures that overlay the summit that need to be reflected away to visualize this region include the pulmonary artery and the left atrial appendage. Thus, potential epicardial access routes to the summit include direct epicardial access, the arteriovenous system, the left atrial appendage, and the posterior suprapulmonic valve region of the pulmonary artery. Regardless of the access route, epicardial fat is frequently found in this region, which makes energy delivery to deep myocardial substrate challenging.

The opinions expressed in this editorial are not necessarily those of the American Heart Association.

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ECG Differential Diagnosis

QRS morphology of the ventricular arrhythmia, specifically the electric axis of activation, when appropriately correlated with imaging and a detailed understanding of the anatomy, can be valuable in guiding mapping and ablation approaches. However, the ECG helps us localize the point of exit from which activation from a reentry circuit or focus spreads across the ventricles, to a region rather than a site where ablation will necessarily be successful. For example, in the case presented by Atienza et al, one can deduce that the origin is high (cranial) and to the left of the body.¹ When understanding the anatomy of the region,^{3,4} we know that structures that require detailed mapping will likely include the distal right ventricular outflow tract and proximal pulmonary artery, the left coronary cusp, the anterior anteromedial mitral annulus, the epimyocardium of the summit, the great cardiac vein and anterior interventricular vein, and associated arterial vessels. Beyond this information, directing energy delivery and identifying the best site for ablation will depend on detailed mapping of each of these sites and understanding the mechanism of arrhythmia.

Mechanism of Arrhythmia

Atienza et al¹ point out that arrhythmias in this region are commonly assumed to be due to a focus of triggered activity, which is identified as the earliest site of activation and where ablation will be curative. However, this case illustrates the importance of identifying substrate abnormalities, because the mechanism may be a focal area of reentry. As with reentry anywhere in the heart, early activation has little meaning, because activation occurs during the entire cycle length of arrhythmia (or coupling interval in the case of reentrant premature ventricular contractions [PVCs]). In such instances, linear ablation or anchoring ablation at a slow area of conduction to an anatomic obstacle may be required. In such cases, apparently paradoxical success may be obtained when ablating at a site where the pace map does not resemble the arrhythmia and activation may not be characterized as presystolic.

An electrocardiographic clue for automaticity as the mechanism is a similar morphology for the first beat of tachycardia, isolated PVCs, and sustained monomorphic tachycardia, as seen in this case. However, in the cul-de-sacs of the heart (summit, supravalar myocardium, etc), reentry may cause PVCs as a result of variable exit to the myocardium. Electrocardiography alone cannot identify the arrhythmia; however, varying coupling intervals despite similar cycle lengths, sometimes with slightly varying QRS morphologies during different heart rates, may point to reentry in 1 of these regions.

Like Cures Like!

The authors of this insightful contribution to the Teaching Points series note the wisdom of Hippocrates of Kos: sometimes, uniquely problematic arrhythmias can be treated if we understand

the pathology that created the uniqueness. In this instance, the patient had ischemia and likely heterogenous scar from previous injury that contributed to the arrhythmogenic substrate. By using the arteries that may course through the arteriovenous triangle of the summit (intermediate ramus,⁵ high diagonal, proximal obtuse margin, or a separate venous connecting vessel draining this region), an innovative and curative option was found, thankfully avoiding transplantation for this patient.

We congratulate the authors on their persistent and exact correlation of information from imaging, understanding of anatomy, and appreciation of why previous mapping and treatment options, including surgery, had failed. With this demonstration, the student of electrophysiology should be encouraged to use similar approaches for the management of arrhythmia elsewhere in the heart.

Disclosures

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W.G.S. is coholder of a patent on needle ablation that is consigned to Brigham and Women's Hospital.

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