In the Middle

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The student of electrophysiology necessarily learns classic patterns and clear descriptions of anatomy and electrophysiology correlations to facilitate safe and effective invasive procedures. However, the same aid to learning serves as an obstacle when confronted with unusual arrhythmia manifestation that requires the appreciation of exceptions to the usual more simplistic constructs to succeed. In this segment of Teaching Rounds in Electrophysiology, Chen et al. describe 2 cases of intramural ventricular tachycardia arising from the outflow tract septum. They teach us with their precise approach for mapping and ablation how and when to break the rules we have learned and indeed use the uniqueness of the situation to solve the problem.

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What Is the Septum?
The interventricular septum separates the right and left ventricles. We easily visualize the septum of the ventricular inflow portion, which is indeed central and between the right and left ventricular inflow tracts. Thus, in the left anterior oblique projection, if we have placed a catheter in the right ventricular inflow, then we know that moving the catheter leftward will allow contact on the septum. This construct, however, is not applicable to the septum of the ventricular outflow tracts. This is because the right ventricular outflow tract takes a complex course anteriorly and superficially to the left ventricular outflow tract with the pulmonic valve lying cephalad and leftward of the aortic valve. As a result, a catheter in the right ventricular inflow outflow tract will need to be manipulated posteriorly to reach the ventricular septum, and moving leftward leads one closer to the left free wall portion of the right ventricular outflow tract near the pulmonic valve. The relationship, however, is a gradual change from the relatively midline inflow septum to the oblique and parallel-to-the-chest-wall course of the outflow tract septum. It is nearly impossible with fluoroscopy alone to identify which sites being mapped are actually septal. Intracardiac ultrasound or other imaging in real time is needed to appreciate that the myocardium being mapped or targeted for ablation is between the right and left ventricular chambers.

What Is Intramural?
Correlation among electrocardiography, anatomy, and a planned mapping and ablation approach for ventricular tachycardia presupposes that the right and left ventricles are distinct entities as is endocardial versus epicardial arrhythmogenic substrate. A focus or pathological tissue critical for reentrant tachycardia may reside intramurally and thus defy these clear distinctions. On the free wall of the left ventricle, intramural tissue represents the mid myocardial fibers that may be difficult to access from endovascular or subxiphoid pericardial approaches. On the septum, intramural tissue is complex, representing overlapping and intertwined myocardial fibers that can be traced to the right or left ventricular myocardium, and generally devoid of conduction tissue. Even more complex is the intramural substrate of endocavitary structures, such as the papillary muscle. Being cylindrical and having variably oriented myocardial fibers with a rich admixture of conduction tissue identifying intramural origin within these structures can be particularly challenging.

Intramural Mapping
How do we map tissue that we cannot access? Chen et al. describe a unique technique to use intramural structures to map the surrounding substrate.

Suspecting Intramural Substrate
The first step to map these difficult arrhythmias successfully is to suspect that the intramural myocardium is responsible for a ventricular tachycardia. For septal intramural substrate when mapping the right ventricle, for example, a diffuse area of near-simultaneous activation for focal tachycardias will be noted. The operator will suspect left ventricular origin, which is indeed usually the reason for this pattern of spread. However, when mapping the left ventricular side of the septum, a similar activation pattern of multiple, equally early sites is noted. This should lead to the suspicion of a midportion of the septum housing the focus and requires targeted ablation.

For reentry which involves the mid myocardium, analogous information from entrainment mapping where several sites on either side of the septum show postspacing intervals slightly longer than the tachycardia cycle length and longer postspacing intervals at pacing sites further from the septum. Sites on the septum may even be in the circuit, with a postspacing interval matching the VT cycle length, but with features of an outer

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loop site, or site just outside the VT exit and not within a channel with slow conduction in the circuit.4

Deducing the Intramyocardial Activation
When suspected, careful analysis comparing the bipolar and unipolar electrograms obtained on either side of the septum (or in the case of the free wall endocardial versus epicardial) will suggest that a site distant from the contact site of mapping is the true area of origin for the focus of the arrhythmogenic substrate.5

Ablating Intramural Substrate
A common approach to ablate deep intramural substrate is to create deep lesions from the endocardium on either side of the septum or endocardial and epicardial lesions that reach the underlying targeted tissue. However, even with irrigated or large surface area catheters, maintaining contact and adequate power delivery to create such a deep lesion is challenging actually. A controlled steam pop can be allowed by increasing flow rates and power. A large area of denuded endothe-lium and crater formation, however, may result, which can be potentially a source of thromboembolism particularly from the endocardial left ventricle.

Bipolar ablation with catheters placed on either side of the septum (or endocardially and epicardially) has occasionally been used.6 However, it is as yet unclear whether this will be better than ablating so as to create deep lesions in a monopolar fashion on either side of the intramural substrate. Novel catheters that include combined extraction or needle injection of current or repeated saline may be available in the future to help us target the midmyocardium without collateral damage to the endocardium.

Chen et al1 elucidate a series of teaching points on how to map and then adequately deliver energy to the intramu-ral substrate for ventricular arrhythmia in 2 patients. Their well-constructed schematic sections are designed to be non-conventional views of looking at the regional anatomy of the intramural substrate. When studied along with the discussion of Chen et al.,1 we appreciate several nuances of what the epicardial surface, coronary veins, and arteries offer for mapping and access to this region.

Coronary Veins and the Epicardial Surface
The coronary veins, such as the epicardial coronary arteries, are thought of as structures that course over the pericardial aspect of the myocardium. Although the veins can be and have been used to ablate epicardial tissue,7,8 the veins may also facilitate intramural ablation.9 The epicardial surface, even when accessed via subxiphoid puncture, may not offer an ideal vantage point for deep myocardial or epicardial substrate because of the overlying fat and coronary arteries. Because the veins are embedded within the fat, better epimycocardial contact is often possible through the veins with minimal power delivery sufficient to treat arrhythmia arising from an adjacent location. The deep myocardial veins drain toward the larger epicardial venous system and thus when accessed can serve as a route for mapping as well as energy delivery.

Coronary Veins and Arteries
In general, we visualize the course of the arterial supply to an organ to be nearly identical with that of the draining veins. Perhaps the most important teaching point from the case description of Chen et al1 is that there are important differences in the course of the veins versus the arteries. It is precisely this difference that allows us to use the veins for mapping and ablation without necessarily risking injury to the artery. The arteries themselves may allow access to deep substrates as a route for alcohol delivery, balloon occlusion, or in rare instances energy delivery to create a distal discrete infarc-tion to ablate the intramural substrate possibly.

In summary, Chen et al1 succinctly highlight with their teaching points how we suspect, map, and definitively target deep intramural substrate and thus add to our armamentarium a unique awareness and skillset to manage ventricular arrhythmia.

Disclosures
Dr Asirvatham receives no significant honoraria and is a consultant with Abiomed, Atricure, Biosense Webster, Biotronik, Boston Scientific, Medtronic, Spectranetics, St. Jude, Sanofi-Aventis, Wolters Kluwer, and Elsevier. Dr Stevenson is coholder of a patent on needle ablation that is consigned to Brigham and Women’s Hospital.

References

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