The “Slow Pathway” Potential: Fact or Fiction?
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The exact electroanatomic circuit responsible for atrioventricular nodal reentrant tachycardia (AVNRT) remains poorly understood. Initially, the pathological substrate was thought to be a small reentry circuit within the compact AV node (AVN). However, through a series of detailed histological studies, computer modeling, optical mapping, and uniquely insightful observations of tachycardia behavior, with the introduction of critically timed and placed premature atrial complexes, the determinative role played by the atrial inputs to the AVN has been established.

Defining the Anatomy
Pandozi et al derive their primary conclusions from mapping of Koch’s triangle, the critical area for AVNRT that houses the compact AVN and slow pathway. How does one know that the mapping catheter is actually in Koch’s triangle? Koch’s triangle is bounded by the insertion of the septal leaflet of the tricuspid valve, the tendon of Todaro (TT), and the coronary sinus. Each of these boundaries is difficult to define in the electrophysiological laboratory. If one uses electrogram criteria (loss of atrial electrogram and large ventricular electrogram) to define the insertion site of the septal tricuspid valve leaflet, errors may occur. The tricuspid valve is apically displaced relative to the mitral valve. As a result, the atrioventricular septum is formed wherein a relatively thin layer of atrial myocardium overlays the ventricular myocardium constituting the basal ventricular septum. This atrial myocardium is technically within Koch’s triangle. However, when mapping this region, because of the very large ventricular electrograms that are seen, it is probably assumed that the tricuspid valve already has been crossed and these signals and regions not included in a complete Koch’s triangle map. Similarly, the coronary sinus itself is an irregularly shaped, sloping, funnel-like structure. Should electrograms identified when mapping on this indistinct coronary sinus musculature near the roof be considered to be derived from Koch’s triangle or not? Finally, the TT is a histologically described structure. The authors used the Eustachian ridge as a surrogate for this structure; however, the Eustachian ridge is much larger and the exact site of the TT is unlikely to have been defined. This lack of anatomic precision inherent in any study of Koch’s triangle may give rise to erroneous results. For example, if the fast pathway inputs technically behind the TT are inadvertently included in the Koch’s triangle map, then a collision of wave fronts, rather than block, would be seen. On the other hand, if one electrophysiologically defined the tendon as the site of block or slow conduction between the anterior and posterior inputs to the AVN, then a circular argument where the electrogram and anatomic data are self-supporting results.

High-Density Mapping
For accurate detailed mapping of an area like Koch’s triangle, small electrode tips and interelectrode spacing, contact of the relevant tissue with the mapping bipole, and avoidance of overlapping signal interpretation (same location mapped twice and taken as 2 different points) is essential. Pandozi et al took 79 ± 21 points in Koch’s triangle. However, they determined the area of Koch’s triangle to be 34.4 ± 8.9 mm², given that a 4-mm-tip electrode with 2-mm interelectrode spacing was used for the map. Even if accurate contact was always present, at least an 8-mm region was being mapped with each point, thus making it impossible to avoid most of
the points yielding overlapping signals and consequently making definition of the wave front of activation extremely difficult and improbable.

**Taking and Annotating Individual Points**

Even if a small electrode with ideal contact and without significant cardiac and respiratory motion are assured, annotating the actual moment of local activation based on the retrieved bipolar electrogram is far from straightforward. The authors used the maximum amplitude of the bipolar electrogram and correlated this with the minimum amplitude on the unipolar electrogram to define activation time. However, in areas of slow conduction, diseased tissue, or near-conduction block, these arbitrary points can be inaccurate. For example, if the actual local tissue was diseased but neighboring tissue is normal, the highest amplitude signal retrieved will be that of activation of the neighboring tissue. Similarly, the onset (rather than the maximum negative) of the negative deflection on unipolar recordings may better estimate local activation time, especially in diseased tissue.

The utmost diligence is required when annotating double potentials and fragmented signals for activation time. For example, when double potentials are on either side, a zone of slow conduction or block is recorded. If maximum amplitude electrogram is recorded in both instances, the same activation time (far-field in one instance and near-field in the other) on either side of the line may be taken. Even with carefully executed pacing maneuvers, the actual point of activation in the midst of a fragmented signal may be impossible to ascertain.

**Determining Conduction Velocity**

Pandozi et al took several precautions to minimize errors in defining conduction velocity by including taking “curvilinear measures orthogonal to the wave front of activation” when calculating the propagation distance. Even with such measures, however, conduction velocity is difficult to define given the complex anatomy and multiple wave fronts in the right atrium in sinus rhythm. When looking at the color-coded maps with electroanatomic mapping, multiple color shifts within a short propagation distance defines slow conduction, and the actual velocity can be computed. However, there is no prior reason why multiple colors in proximity represent true conduction and may in fact actually signify relative timing of a wave front propagating elsewhere reaching those local sites. For example, if a wave front splits on either side of the Eustachian ridge but the conduction velocity on either side is markedly different, the appearance of conduction across the Eustachian ridge can result.

**Defining Conduction Block**

Distinguishing conduction block from slowed conduction is no easy task in the daily practice of invasive electrophysiology. The authors defined conduction block as conduction time exceeding 30 ms. However, both slow conduction and “pseudo conduction” from simultaneously activating wave fronts may result in the appearance of block when not actually present. To be certain of block, the wave front must be known to be propagating orthogonal to the line of presumed block, and multiple distinct points of activation showing a complete and transmural reversed activation on the distal side of the line of block must be demonstrated. Even in a relatively straightforward region such as the cavotricuspid isthmus, such determinations are not easy, and given the complex electrograms within the small anatomic area of Koch’s triangle, these difficulties are compounded.

**“Slow” – “Jackman” – “Haissaguerre” – “Intermediate”…What Are These Potentials?**

The complexity of the electrograms in the region of Koch’s triangle has been well recognized but interpreted differently by various electrophysiologists. Pandozi et al defined an early far-field signal followed by a late relatively high-amplitude near-field signal as a “Jackman” potential, whereas an early high-amplitude near-field signal followed by a far-field component as a “Haissaguerre” potential. They also describe hybrid electrograms with characteristics of both these signals. The exact interpretation of what these signals represent mandates perfect assessment of the anatomic location of the TT. If one uses the construct that the fast pathway exit is behind the TT whereas the slow pathway and compact AVN are in front of (closer to the tricuspid valve) the TT, and if the line of block or slow conduction is the TT, then when a catheter is placed in front of the TT, in sinus rhythm, there will be delayed activation of this site representing slow pathway conduction. However, for that same beat, if the catheter is now moved behind the TT, then a near-field signal will time with the early far-field signal seen in front of the TT. Thus, both “Jackman” and “Haissaguerre” potentials may represent the same activation, but the electrogram recorded on either side of the TT. Given the difficulties with defining anatomy, high-density mapping, and annotating points mentioned above, extreme caution is needed when interpreting the results of electroanatomic maps in this region before reaching a definitive conclusion on the nature of these signals.

**Clinical Significance**

What do the student and practitioner of invasive electrophysiology learn from the insights provided by Pandozi et al’s interesting study? What is clear is that complex electrograms are found in the region of the TT in Koch’s triangle. The significance of appreciating this is 2-fold. First, the electrophysiologist must not be lulled into believing that targeting such signals is always safe, because they simply signify location of the ablation catheter in Koch’s triangle where the compact AVN is also located. However, the absence of recording such signals should alert the operator that the catheter may not be located in or making contact with Koch’s triangle tissue, and therefore, unlikely to be targeting the slow pathway input to the AVN.

**Further Study**

Because we are so often successful in eliminating AVNRT, do we need such studies or precision in understanding the electrophysiology of this region? An appreciation of the difficulty and seeking solutions to define these electrograms and arrhythmia are not peculiar to AVNRT, but they are also present for our understanding of the slow zones of conduc-
tion responsible for other reentrant tachyarrhythmia, and in the future when we define the roles of autonomic ganglia signaling, Cajal-like cells, and possibly the integration of cells attempting regenerative therapy and new conduction tissue.

We must congratulate Pandozi et al for venturing to define activation sequences and conduction characteristics in this complex region, given the limitations of the tools and technology we use. Will we ever know what exactly the slow pathway is or what its definitively associated electrogram characteristics are? Further studies probably will require very small electrode tips, techniques to minimize or entirely ameliorate cardiac and respiratory motion, and advanced conjunctive imaging to precisely define the boundaries of Koch’s triangle. In turn, such precisely recorded electrograms will have to be correlated prospectively with sites of early activation with retrograde conduction of the slow pathway and sites that give rise to slow junctional rhythms characteristic of successful ablation locations for AVNRT. The present study reported in this issue by Pandozi et al sheds a small but significant electrophysiological light on this complex area and gives the opportunity for the reader to reflect on the nature of these recorded signals and the arrhythmia—AVNRT—itself.

Disclosures

None.

References


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