Atrioventricular Nodal Reentry Tachycardia
Chameleon in Disguise

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In this segment of Teaching Rounds in Cardiac Electrophysiology, Hayashi et al.1 present a patient with atypical atrioventricular (AV) nodal reentry tachycardia (AVNRT) with an unusual initiation and mechanism for maintaining arrhythmia. Their astute observations, which they present as excellent teaching points, led to successful ablation at the earliest site of retrograde slow pathway activation. The student of electrophysiology will rarely see a similar case in his or her practice. However, the magnitude of the teaching value of such a description lies in the generalizability of the findings and the deductive process that led to a successful outcome. Exceptional cases teach us how we use information available from any case to achieve conceptual understanding and in most cases success.

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The information available to the interventional electrophysiologist that requires analysis and interpretation includes measured intervals, responses to maneuvers, the activation sequence, and information that allows anatomic-physiological correlation.

Intervals, Pseudointervals, and Bystander Intervals

In sinus rhythm, when one calculates the atrium to His bundle (A–H [His bundle to ventricle]), H–V, or P–R (P wave to QRS onset) intervals, the measured value represents a true conduction time from one site to another. However, during tachycardia, a given interval may simply represent the relative activation times between 2 locations and not the actual time required for a propagating wavefront to go from the first to the second location, referred to as a pseudointerval. The disparity between true and pseudointervals and the critical need to appreciate what is actually being measured is best exemplified with AVNRT.

V–A Interval

Conduction time from the ventricle to the atrium during AVNRT is clearly a pseudointerval representing the relative conduction times from a common turnaround site in the AVNRT circuit down to the ventricle and back to the atrium. Thus, the V–A (ventricle to atrium) interval may be negative if conduction to the atrium from the common point is faster than the conduction to the ventricle.

H–A Interval

Although the His bundle is relatively closer to the arrhythmia circuit than a more distal site in the ventricle, it also is not within the circuit, and thus the H–A (His bundle to atrium) interval is a pseudointerval. A negative H–A interval would be favored by rapid conduction from the turnaround point to the atrium, slow conduction from the turnaround point to the His bundle, and by the presence of a lower common pathway.

Of note, because these intervals are not true intervals, they have no a priori relationship with whether retrograde activation is via the slow or fast pathways. Thus, in atypical AVNRT with retrograde slow pathway and a long, lower common pathway, the V–A interval will be short or negative.

A–H Interval

Although most electrophysiologists appreciate that the V–A and H–A intervals are pseudointervals and that they may vary tremendously even during a stable circuit of AVNRT, surely the antegrade A–H interval is true conduction interval?

This would be true but for the presence of a lower common pathway connecting a common turnaround point in the AVNRT circuit and the AV node. For example, in slow–slow AVNRT, if the 2 slow pathways meet outside the AV node and connect to it through a common pathway, then conduction may proceed from the retrograde atrial exit site to the AV node, either through the culprit slow pathway (true interval) or through another bystander slow pathway or a bystander fast pathway or the fast pathway. In the latter 2 instances, the A–H interval does not represent a conduction interval germane to the AVNRT circuit. This possibility is most starkly manifest when the lower common pathway blocks and AVNRT continues with either AV block (more As than Vs) or with a shorter A–H interval from bystander antegrade fast pathway conduction.

A–A Interval

Because the critical components of the AVNRT circuit and indeed our target site for ablation involves the supraventricular tissues, surely the A–A (atrium to atrium) interval cannot be a bystander or pseudointerval? The above would be true but
for the possible presence of an upper common pathway. Presumably, the 2 pathways responsible for AVNRT maintenance may connect with each other at a distinct point from which activation proceeds to the rest of the atrium. Thus, upper common pathway (UCP) decrement delays or decrement may produce varying A–A intervals despite the continuance of a single, stable AVNRT. In fact, with this construct, there may be complete AV block with more Vs than As with upper common pathway block.

**Maneuvers**

**V–A–V Sequence**
Hayashi et al. found that ventricular extrastimuli initiated tachycardia with a V–A–V (ventricle to atrium to ventricle) sequence. In general, an atrial tachycardia would be expected to start with ventricular stimulation only when retrograde conduction is present and the retrograde A starts tachycardia, creating a V–A–V–A (ventricle to atrium to atrium to ventricle) sequence. However, single beats of atypical AVNRT (retrograde fast, antegrade slow, or vice versa) are common during electrophysiology study, and a single atypical AVNRT beat producing a V–A–V sequence may initiate an atrial tachycardia. Similarly, in the absence of a lower common pathway or multiple retrograde slow pathways, a single ventricular extrastimulus may produce 2 atrial activations, one of which constitutes a limb of the induced AVNRT circuit and therefore a V–A–A–V sequence may result when initiating tachycardia. Finally, when an upper common pathway is present and blocks during a PVC-initiated retrograde sequence and tachycardia induction, a V–(no A)–V or V–H–V (ventricle to His bundle to ventricle) sequence may be seen. The student of electrophysiology needs to be aware of these possibilities, albeit rare, so that the differential diagnosis for an arrhythmia is not too quickly restricted from a single maneuver.

Importantly, as emphasized by Hayashi et al., for most ventricular pacing maneuvers used for supraventricular tachycardia diagnostics during tachycardia, careful inspection is performed to ensure that the paced wavefronts are reaching the atrium (typically with acceleration of electrograms to the paced rate) and whether the activation sequence is the same during pacing and tachycardia.

**Comparators**
Maneuvers have been developed to try to determine whether a measured interval represents an actual conduction time or not. For example, the H–A interval (measured from the end of the His to the earliest A assuming stable activation sequence) during tachycardia and ventricular pacing has been used to distinguish the various types of AVNRT and between His bundle tachycardia and AVNRT. In a His bundle tachycardia, the H–A interval is a true conduction time and would be similar to the H–A interval measured during ventricular pacing. On the contrary, the H–A interval may be shorter or longer during AVNRT when compared with ventricular pacing. Because of the vagaries of conduction velocity and wavefront curvature around turnaround points, as well as autonomic tone, experimental evidence rather than deductive reasoning alone is needed to make arrhythmia differential diagnostic determinations. At present, such evidence for complex perinodal tachycardias is scarce.

**Activation Sequence**
Regardless of whether an arrhythmia is from an automatic/focal source or reentrant, the activation sequence is an important determinant of the target site for ablation. In general, the earliest site of activation has little value for defining a reentrant arrhythmia; however, AVNRT is an exception to this generalization. In fact, Hayashi et al. ablated at the earliest atrial activation site to solve this patient’s complex problem. This approach has value only for atypical AVNRT. With typical AVNRT, the earliest site of activation is behind the tendon of Todaro at the fast pathway exit site and anatomically too close to the compact AV node to allow consistently safe and successful ablation. However, with retrograde slow pathway tachycardia, the anatomic distance from the compact AV node for right and left-sided slow pathways do allow for successful ablation, and the typical minimal risk of AV block associated with anatomic slow pathway ablation. Thus, as Hayashi et al. demonstrate, when the arrhythmia is complex, and even careful analysis does not allow exact explanations for all the observed phenomena, if the earliest site of activation is in the vicinity of the slow pathway and not in the anatomic fast pathway site, then mapping of the atrial breakout site and ablation in this location is potentially curative.

**Anatomy and Physiology Correlation**
A major source of difficulty with AVNRT variants is our lack of understanding in terms of anatomic–physiological correlation of critical components of the circuit. This is primarily because we do not have a reliable method of recording compact AV node, transitional zone, or extensions of the AV node in humans. There is no certain way of distinguishing slow pathway atrial input signals, slow pathway extensions of the AV node, the AV node itself, or bystander abnormally conducting atrial sites.

A partial result of this imprecision is the absence of anatomic knowledge of whether the lower common pathway is simply part of the AV node distal to the AVNRT turnaround site or is an extranodal connection to the confluence of ≥2 slow pathways. This distinction is important as noted above because an extranodal lobe common pathway may block with continued tachycardia and paradoxical shortening of the A–H interval because of bystander antegrade fast pathway conduction. The anatomic basis for the proposed upper common pathway is even more nebulous with at present no clear histological or electroanatomically mapped structure that may connect the AVNRT circuit to the atrium.

**Summary**
Hayashi et al. teach us a sequential approach to question the possibilities that explain observed unusual phenomena and demonstrate the importance of finding a reasonable ablation target that is safe although the explanation of all phenomenon is incomplete. Perhaps future developments of mapping systems and novel signal processing presently meant for identifying atrial fibrillation and ventricular fibrillation substrate may
allow us to record nodal and perinodal electrograms to better understand these complex arrhythmias in individual patients.

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References

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