In this segment of the Teaching Rounds in Electrophysiology, Chen et al\(^1\) provide an instructive discussion taking us through one of the most difficult diagnostic differentiations in cardiac electrophysiology—AV nodal re-entry tachycardia (AVNRT) versus junctional tachycardia.\(^2\) Often the highest educational value in the electrophysiology laboratory when teaching a rule is to recognize and analyze an exception. Chen et al\(^1\) induced a slower tachycardia with similar activation sequence as an originally induced AVNRT. After slow pathway ablation and the use of isoproterenol, they had to determine whether further ablation is necessary or could potentially be harmful (slower AVNRT versus junctional tachycardia), importantly they found an exceptional response to premature atrial contractions (PACs) that span the cycle length of the arrhythmia and, with their careful analysis and stepwise logical interpretation of the data, concluded correctly that further slow pathway ablation will eliminate the arrhythmia.

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The slow pathway is usually anatomically defined as the myocardium anterior toward the ventricle to the Eustachian ridge and tendon of Todaro, behind the tricuspid annulus septally, and just cranial to the coronary sinus ostium.\(^3\) Although exceptions exist for any diagnostic maneuver in the electrophysiology laboratory, maneuvers related to the AV nodal slow pathway can be particularly difficult because there may be anatomic variants and decrement conduction delay within the circuit. Understanding common causes of difficulty with analyzing the results of maneuvers performed and appreciating the diagnostic effect of success and failure with anatomic slow pathway ablation can be important.

Causes of Difficulty With Interpreting Commonly Executed Diagnostic Maneuvers in the Electrophysiology Laboratory

Cycle Length Variation

Cycle length stability before initiating a maneuver such as introduction premature atrial beats to perturb the circuit is a paramount for accurate diagnostic interpretation. Occasionally, even if the cycle length varies, the maneuver can be executed, as long as the variation is occurring in a predictable manner. For example, orthodromic AVRT with alternating cycle lengths from antegrade fast and slow pathway conduction may still be diagnosed when sense PVCs are placed specifically with the alternating longer or shorter cycle lengths. Particularly problematic is when there is decrement somewhere within the circuit, and unpredictable cycle length variation is occurring. The operator cannot readily use a standard perturbing maneuver, but careful analysis of the sequence of the cycle length variation can lead to a correct diagnosis. For example, if there is decremental conduction in the AV node resulting in cycle length variation but the atrial cycle length is fairly fixed, then atrial tachycardia is likely. On the contrary, if in a similar circumstance, decrease in the V–V interval leads to reciprocal changes in the atrial cycle length, etc, then atrial tachycardia is unlikely. An extension of this problem with cycle length variation in the extreme form is conduction block to parts of the heart during an arrhythmia. For example, in AVNRT, with upper common pathway block, it can be impossible to use the introduction of PVCs or PACs during tachycardia to define the mechanism. Diagnosis may still be made if there are times where the conduction block is not present (1:1 tachycardia) or with specialized maneuvers such as subthreshold stimulation in the region of the slow pathway.

Alternate Conduction Pathways

Interpreting diagnostic maneuvers can be challenging when >1 alternative or bystander conduction pathway is present but is not part of the re-entry circuit. Chen et al\(^1\) describe an example of 2:1 conduction as a possible explanation for the conflicting results from the maneuver performed in their case. In AVNRT, PACs reach the AV node through the slow pathway and as a result can do so only if placed with a relatively short coupling interval (and reset the tachycardia with a long A–H interval) or when placed at a time of proximate His refractoriness can engage the circuit to affect only the subsequent beat. However, if a bystander pathway is present, such as the fast pathway in slow–slow AVNRT or a second bystander slow pathway in typical AVNRT, then the PAC may advance the proximate His in addition to affecting the subsequent beats of tachycardia. Similarly, with junctional tachycardia, PACs can engage the proximal His and either terminate or overdrive suppress the tachycardia in a reproducible fashion. However, if the junctional rhythm arises close to the fast pathway, then antegrade conduction through the fast pathway may be challenging, but if a bystander slow pathway is present, the PAC may seem to affect or reset the subsequent beat of tachycardia.
When a PVC introduced at a time of His bundle refractoriness advances the atrial activation of a supraventricular tachycardia, we typically diagnose AVNRT. However, in a patient with AVNRT, a similar response can be observed if a bystander retrograde-only conducting nodoventricular or nodofascicular tract is present.5,6

**Serendipitous Spontaneous Ectopy**

Spontaneous ectopy that occurs by chance during the execution of a diagnostic maneuver can cause conflicting and difficult to explain results. Similarly, when attempting to induce an arrhythmia, a spontaneous junctional beat may start AVNRT with no discernible relation to the atrial paced beats being used to start the arrhythmia, giving the mistaken impression of junctional tachycardia being the mechanism of the sustained arrhythmia.

**Unusual Effects on Conduction by Introduced Premature Beats**

Occasionally, the response to an introduced premature beat is confusing because of less common effects on conduction. For example, an early coupled PAC may fail to affect the proximate His bundle electrogram, leading to a mistaken suspicion for junctional tachycardia if there is significant intra-atrial delay, a type of gap phenomenon.7 Similarly, premature beats may affect the source–sink relationship between the atrium and ventricle and facilitate either antegrade or retrograde conduction (Prinzmetal and Wedensky phenomenon, respectively).8,9

**Slow Pathway Ablation Is Successful**

When slow pathway ablation successfully eliminates the arrhythmia, important insights into the mechanism of the original tachycardia are present. Several arrhythmias, however, may be successfully eliminated with energy delivered to the anatomic slow pathway region.

**AVNRT**

With typical AVNRT, anatomic slow pathway ablation eliminates the tachycardia in the majority of patients and slow, regular junctional ectopy is typically seen during the ablation.

**Junctional Tachycardia**

Some junctional tachycardias can be successfully eliminated without damage to the compact AV node, presumably because of origin of the automatic focus is near the slow pathway extension of the AV node.

**Atrial Tachycardia From the Coronary Sinus Ostium Region**

Because the anatomic slow pathway is usually within the triangle of Koch, ablation in this region may eliminate an atrial tachycardia arising from the myocardium of the coronary sinus ostium. Particularly confusing may be the elimination of the tachycardia concurrent with initiation of junctional ectopy during the burn.

**Slow Pathway Tachycardia With Retrograde Block**

Tachycardia may arise from the slow pathway input to the AV node but exhibit retrograde block such that atrial activation occurs only via compact AV nodal activation and then retrograde activation via the fast pathway thus mimicking the pattern of AVNRT. Again, this type of arrhythmia could be eliminated when performing anatomic slow pathway ablation.

**Posteroseptal Accessory Pathway-Mediated Tachycardia**

The posterior septum is not a part of the true interatrial septum and includes the pyramidal space, coronary sinus ostium, and the atrial insertion sites of slow pathway inputs to the AV node. A posteroseptal accessory pathway may thus be eliminated with anatomic slow pathway modification.

**Orthodromic Nodoven tricular Tachycardia**

For unclear reasons, nodoven tricular tachycardia can be eliminated with slow pathway ablation. Initially, it was thought that nodoven tricular tachycardia would require ablation in the ventricle where the pathway was thought to exist. Presumably, in some instances, the nodoven tricular tract inserts specifically into the slow pathway rather than the compact AV node and thus can be cured with slow pathway ablation.

**Slow Pathway Ablation Fails to Eliminate the Arrhythmia**

**AV Nodal Re-entry Tachycardia**

Less common variants of AV node re-entry may not involve the right slow pathway input to the AV node. Ablation at the anatomic slow pathway may result in the anticipated junctional rhythm during energy delivery but will not eliminate the tachycardia.

**Junctional Tachycardia**

Junctional tachycardia is a conglomerate disease with varying causes and sites of origin. Automatic junctional tachycardia arising from the His bundle or compact AV node will not be eliminated with slow pathway modification.

**Atrial Tachycardia From the Coronary Sinus Ostium Region**

The site of origin of the CS tachycardia may be at an anteroposterior level or site different from where the slow pathway modification is being targeted.

**Slow Pathway Tachycardia With Retrograde Block**

Anatomic transection of the right slow pathway may be done successfully, but a focus of tachycardia within the slow pathway region may still be present and continue to give rise to arrhythmia despite successful slow pathway modification.

**Posteroseptal Accessory Pathway-Mediated Tachycardia**

Anatomic slow pathway ablation is typically not at the annulus but rather within the atrial myocardium. Thus, the atrial insertion of a posteroseptal accessory pathway may be modified without elimination of the pathway.

**Orthodromic Nodove ntricular Tachycardia**

Some forms of nodoven tricular tachycardia do not necessarily need to involve an insertion site in the right slow pathway, and in these instances, slow pathway modification will not be
successful, perhaps necessitating ventricular or other AV node input site ablation.

The differential diagnosis for both successful and failed anatomic slow pathway ablation is similar. Thus, the failure to eliminate arrhythmia when ablating at the site may simply be because of incomplete ablation or an alternate diagnosis. Therefore, it is critically important to accurately interpret a diagnostic maneuver to define the mechanism of tachycardia before energy delivery. Chen et al elegantly discuss one such maneuver and use a unique response in their case to clarify the electrophysiological basis for this commonly performed diagnostic test.

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References

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