In this segment of the Teaching Rounds in Electrophysiology, Chen et al. 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. Often the highest educational value in the electrophysiology laboratory when teaching a rule is to recognize and analyze an exception. Chen et al. 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. 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. 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 refractori-
ness advances the atrial activation of a supraventricular tachy-
cardia, we typically diagnosis 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 arrhym-
ia, 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 conduc-
tion (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, how-
ever, may be successfully eliminated with energy delivered to
the anatomic slow pathway region.

**AVNRT**
With typical AVNRT, anatomic slow pathway ablation elimi-
nates 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 tri-
gle 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 activa-
tion 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 sep-
tum 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 elimi-
nated with anatomic slow pathway modification.

**Orthodromic Nodoventricular Tachycardia**
For unclear reasons, nodoventricular tachycardia can be elimi-
nated with slow pathway ablation. Initially, it was thought that
nodoventricular tachycardia would require ablation in the ven-
tricle where the pathway was thought to exist. Presumably, in
some instances, the nodoventricular 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 antero-
posterior 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 path-
way 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 anmu-
lus but rather within the atrial myocardium. Thus, the atrial
insertion of a posteroseptal accessory pathway may be modi-
ified without elimination of the pathway.

**Orthodromic Nodoventricular Tachycardia**
Some forms of nodoventricular tachycardia do not necessar-
ily 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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