Diagnostic Perturbations

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An electrophysiologist may find a procedure difficult because he or she is not familiar with the diagnostic or therapeutic maneuver needed to address an unusual, unexpected finding. However, perhaps the most challenging case we confront is the typical case, that is, one where we need to think simply without unnecessary complication with high expectation of success with ablation. The primary purpose of the series Teaching Rounds in Cardiac Electrophysiology is to present students of invasive electrophysiology and device management that bridge between these extremes and demonstrate the way an expert reasons in a straightforward manner but with the background plethora of multiple complex and involved possibilities.

See Article by Bagga et al

In this issue of *Circulation: Arrhythmia and Electrophysiology*, Bagga et al[1] provide an outstanding example of how to combine practicality with erudition. They not only provide excellent teaching points and unique insights but openly showcase the problem of incomplete data and indeed explain how, despite the lack of an exact diagnosis, a successful outcome for their patient was enabled. The authors provide an elegant discussion of some of the cardinal rules and their exceptions when using diagnostic maneuvers to perturb an unknown circuit. A few of these principles are worth reemphasizing.

The Concept of Pseudointervals

We assume that the H-V interval is because of conduction traveling from the His bundle to a ventricular exit, and this interval represents the conduction time from the H to the V. However, in many instances such as during bundle branch reentry, the H-V interval represents a pseudointerval, that is, an interval created by the difference in conduction time from a common turnaround point (left bundle/distal His junction) to the recorded His and to the ventricular exit. Similarly, the H-A interval during atrioventricular node reentry or the H-V interval when both atrioventricular node conduction and an antegrade conducting pathway are present during sinus rhythm and atrioventricular conduction are pseudointervals.

It is imperative in approaching complex arrhythmias to distinguish between true conduction intervals and pseudointervals. During bundle branch reentry, the H-V interval may be shorter than the H-V interval during sinus rhythm or, if right bundle conduction is extremely delayed, longer or equal to the H-V interval seen in normal rhythm. On the other hand, the right bundle to ventricular exit interval will be similar during typical bundle branch reentry and atrial pacing with intact atrioventricular nodal conduction at a rate similar to tachycardia.

Similarly, with slow/fast atrioventricular nodal reentrant tachycardia, the H-A interval may be negative, resulting in a long RP tachycardia if antegrade conduction from the turnaround point of the tachycardia circuit to the ventricle is long (infra-Hisian conduction delay), and retrograde conduction via the fast pathway is rapid.

Early or Late?

In some instances, the conduction interval may exist but is inverted. For example, in interfascicular tachycardia, diseased infra-Hisian conduction and multiple bystander loops involving the conduction system and ventricular myocardium, late bystander activation of a fascicle, may be recorded closer to the subsequent ventricular electrogram giving the mistaken impression that the candidate signal is early rather than late with regard to the QRS onset. Similarly, during antidromic tachycardia with an antegrade nodofascicular or atriofascicular tract, retrograde activation of the His may occur before the antegrade ventricular exit creating a negative H-V interval giving the mistaken impression of antegrade conduction via the normal conduction system access. The electrophysiologist must be alert to these possibilities and indeed suspects that a pseudointerval is operative whenever there is a marked difference in measured conduction time between normal rhythm and tachycardia.[2]

His Refractoriness

The classic maneuver to identify a retrograde extranodal connection to the atrium from the ventricle is placing His refractory premature ventricular complexes (PVCs) during tachycardia and observing whether the atrium can be preexcited and subsequently reset the tachycardia. It is assumed that if an antegrade His is recorded at about the time of the PVC during tachycardia, then retrograde conduction cannot occur via the atrioventricular node. This, in turn, supposes that the only connection to the atrioventricular node from the ventricle other than an atrioventricular bypass tract is the His bundle and further assumes that the His will be engaged immediately or soon after the paced PVC.

Bagga et al[1] point out important exceptions where these assumptions cannot be made. For example, although the ventricular pacing stimulus may be delivered when the His is

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refractory, the actual paced ventricular wavefront of activation may reach the His much later. For instance, when there is retrograde right bundle branch block, the time for transseptal conduction and possible retrograde left bundle conduction delay, the paced wavefront may still engage the His retrograde even though the pacing stimulus was indeed placed at a time the His should be refractory. Late arrival of the stimulated wavefront could also occur when long capture latency, exit delay, or intraventricular block delay engagement of the retrograde conduction system from the pacing stimulus site.

The His bundle and an atrioventricular bypass tract may not be the only ways for a stimulated ventricular activation wavefront to reach the compact atrioventricular node. Rarely, with retrograde conducting nodoventricular and nodofascicular tracts when present and not directly related to the primary tachycardia (bystander), a PVC can preexcite atrioventricular node reentry even when the pacing stimulus was delivered at a time of true His refractoriness.

Catheter Position and Activation Sequence

Using maneuvers to elucidate the mechanism of a complex tachycardia requires knowledge of exact recording catheter position and activation sequence with high spatial and temporal resolution. For example, we rarely need to differentiate between a recording electrode in the distal His bundle or the proximal right bundle branch. However, in typical bundle branch reentrant tachycardia,[3,4] the right bundle is part of the circuit, whereas the His bundle is not. Thus, a catheter recording the His bundle, which is a bystandermay, shows the Purkinje potential to show dissociation, lack of reset, and not anticipate oscillations in the tachycardia cycle length, whereas a right bundle branch recording, which is from part of the circuit, will show changes demonstrating that it is linked to the tachycardia.

Activation sequence determination with closely spaced recording electrodes or multiple activation points would show retrograde activation of the His bundle in bundle branch reentry but antegrade activation of the right bundle branch. Indeed, if longitudinal continuous structures such as the His bundle/right bundle axis show a fusion of activation or 2 separate wave fronts, then the 2 portions of this axis both cannot be in the tachycardia circuit. Similarly, if one knows that the recording closely spaced electrodes are on the His bundle only, then retrograde activation of the His will exclude supraventricular tachycardia with antegrade bundle branch block as a mechanism for a wide complex tachycardia (eg, atrioventricular nodal reentrant tachycardia with bundle branch block). Similarly, antegrade or fused activation of the right bundle but retrograde activation of the His bundle in a wide complex tachycardia may be seen with antidromic atriofascicular tachycardia with retrograde right bundle branch block.

Pacing Output

The differential diagnosis in the case presented by Bagga et al[1] included the need to define whether the ventricular myocardium or the conduction system was an essential part of a reentrant circuit. A variant technique of classical entrainment for reset maneuvers is to vary the pacing output while maintaining a similar pacing position and rate. For example, para-Hisian pacing during a reentrant tachycardia performed at high output (with His bundle capture) versus low output (without His bundle capture), although checking for reset and maintenance of the activation sequence (concealment) can define whether the His bundle itself is an essential component of the reentrant circuit. Similarly, high- and low-output pacing–induced PVCs near the right bundle or left fascicles during a wide complex reentrant tachycardia can define whether the conduction system is critical to the circuit as with bundle branch reentry or macroreentrant fascicular reentry. Once again, careful mapping with closely spaced multielectrode catheters placed over the suspected conduction system limbs of the tachycardia circuit will help define whether the reset occurred with concealment or not.[5]

When Entrainment Fails

Experienced electrophysiologists are familiar with the nuances and techniques to interpret entrainment maneuvers and define a critical and targetable ablation limb of a reentrant circuit. As in the case described by Bagga et al,[1] at times, entrainment fails.

Unable to Entrain

Pacing at shorter cycle lengths than the tachycardia may fail to entrain, that is, reset with fusion the tachycardia. Instead, overdrive suppression or simply the inability to enter a protected circuit may be present. When overdrive suppression is seen, as in an automatic tachycardia, mapping to identify the earliest site of activation may be sufficient to ablate the arrhythmia without exact knowledge of the underlying substrate, etc. Inability to enter the critical portions of a reentrant circuit (entrance block) indicates that the pacing site is not a reasonable target for ablation.

Termination of Tachycardia

When tachycardia terminates repeatedly while attempting entrainment, useful information can be obtained. Here, when detailed activation sequences along with the surface QRS and p wave are available, one can determine which beat in the entrainment cycle terminated the tachycardia. Further analysis can show whether fusion was required to terminate the tachycardia and if so, in which locations of the heart was this necessary. Fusion before termination suggests a reentrant mechanism and regions where fusion produced an antidromic wavefront that reproducibly terminates the tachycardia strongly suggest participation of those sites in the tachycardia.

Subthreshold Stimulation

Pacing at outputs that do not reveal manifest global ventricular or atrial capture but yet either delay or terminate the tachycardia represents strong evidence of those sites being necessary for tachycardia maintenance and thus a potential target for ablation.

Summary

A careful study of cases such as the one presented by Bagga et al[1] presents valuable opportunities to learn the scientific and logical basis for diagnostic maneuvers, balanced with practicality that defines the art of invasive electrophysiology.

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, Elsevier, Zoll. Dr Stevenson is coholder of a patent on needle ablation that is consigned to Brigham and Women's Hospital.

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