

His Bundle Refractoriness

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Multiple points of instruction discussed practically in the context of a challenging case make for a memorable learning experience. In this installment of Teaching Rounds in Cardiac Electrophysiology, Nayyar et al¹ elucidate the unique findings and analysis in such a manner and take us through to diagnosis and a treatment plan that eliminated arrhythmia without having to ablate all of the abnormal structures identified in their patient. Their case in which 2 unusual arrhythmogenic substrates coexist and interact to result in what appears to be a random and inexplicable series of electrophysiological findings is broken down in logical steps defining each aspect of the arrhythmia.¹ These steps include defining the antegrade and retrograde limbs of the putative tachycardia circuit, defining the mechanism of tachycardia, and coming to a conclusion keeping in mind certain difficult differential diagnostic situations and the importance of interpreting His bundle refractory extrastimuli maneuvers.

See Article by Nayyar et al

Antegrade Limb

When anterograde conduction over the AV node and an accessory pathway is present, the primary maneuver to determine the mechanism of AV conduction is incremental atrial pacing or delivery of coupled atrial extrastimuli. With usual accessory pathway activation, the ventricular beats represent fusion between AV node and accessory pathway conduction, and premature atrial beats progressively delay AV nodal conduction with no significant effect on the pathway. As a result, the stimulus to earliest ventricular activation remains fixed, but the H–V interval becomes progressively negative and the QRS progressively more preexcited.

Decrement in the Pathway

Nayyar et al¹ noted that while subtle but significant increase in preexcitation and a more negative H–V interval did occur with atrial extrastimuli, there was a clearly discernible increment in overall conduction time to the ventricle. This finding may be seen with atriofascicular or AV pathways with AV node-like characteristics (incorrectly referred to as Mahaim fibers), nodoventricular/nodofascicular (NV/NF) tracts, duplicate AV node with or without a Mönckeberg sling, or with a fasciculoventricular tract where >1 tract is present and sequential block is seen.

Differential Site Atrial Pacing

The closer one paces to the atrial insertion of a pathway, the more preexcited the QRS appears. The authors paced near the atrial septum and the free wall of the right atrium at similar cycle lengths and coupling intervals and found no change in the degree of preexcitation. This finding is consistent with NV/NF fibers² because the AV node represents an upper common pathway to the 2 methods of ventricular excitation, atriofascicular, or AV fibers on the septum in close proximity to the AV node, and dual AV nodes with or without a Mönckeberg sling.^{3,4}

Preexcited Beats Without Evidence of Preceding Atrial Activation

Analysis of escape beats or premature beats without previous atrial activation can be revealing as to defining the antegrade limb. With usual AV pathways, junctional beats are devoid of any preexcitation, whereas ectopic beats from the pathway will be fully preexcited. Thus, escape beats will not show QRS fusion. Junctional beats with preexcitation may be seen with NV/NF tracts or fasciculoventricular tracts. Conversely, ectopic beats with fusion may be seen when they originate from an atriofascicular tract or accessory AV node with a connecting fiber (Mönckeberg sling).

The authors concluded that the progressive preexcitation and spontaneous ectopy with near-identical fusion to conducted beats observed in their patient was most consistent with the NV/NF tract.

Retrograde Limb

Nondecremental early atrial activation on the free walls of the atria during ventricular pacing or tachycardia suggests accessory pathway presence, whereas decremental early activation in the region nearer the His bundle but posterior to the tendon of Todaro or in the proximal coronary sinus (CS) is consistent with retrograde AV nodal activation.

When nondecremental early septal activation occurs, additional clarification typically with para-Hisian pacing is needed to distinguish between relatively fast AV nodal conduction or a septal accessory pathway.^{5,6}

Decremental but eccentric (free wall early) atrial activation may be seen with less common decremental accessory pathways, with AV nodal activation, or with unusual variance of AV nodal activation.³ Retrograde AV node fast pathway activation

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may present with an eccentric CS activation sequence, even though the fast pathway site is the first to activate, as a result of trans-Eustachian ridge block that requires the AV node reentrant tachycardia wavefront to enter the left atrium and then propagate via CS myocardium–left atrial connections to return to the AV node. Rarely, a left-sided slow pathway may exit to the left atrial free wall on the mitral annulus producing eccentric activation with decremental properties. Once again, para-Hisian pacing would clarify whether the specific activation sequence was dependent on AV nodal conduction.

For the patient described, para-Hisian pacing was not performed. Indeed, para-Hisian pacing can be difficult to execute or interpret in the presence of retrograde conducting unusual accessory pathways. For example, a fasciculoventricular tract with retrograde conduction is essentially because of a loss of some of the insulating fibrous tissue that usually surrounds the His and proximal right bundles. Thus, either high or low output para-Hisian pacing will result in a narrow QRS complex with both myocardial and the equivalent of direct conduction system capture. If the fasciculoventricular tract is right-sided, para-Hisian pacing performed from the left side may still be of value.

Another intriguing feature seen in the outstanding tracings provided in their submission¹ is the relatively near-simultaneous activation of multiple atrial sites during tachycardia and ventricular pacing before ablation. Although the earliest site was near the distal CS, near-simultaneous activation is seen in the mid and proximal CS, and indeed the right septum is not as late as one would anticipate from a typical far left-side accessory pathway. Multiple early sites suggest that either multiple connections or the true early site is not mapped and is located somewhere between the other relatively early sites. Yet in this patient, a single site of ablation at the 2 o'clock position on the mitral annulus eliminated all ventriculoatrial activation!

Mechanism of Tachycardia

The basic maneuver to define mechanisms of supraventricular tachycardia is whether ventricular extrastimuli can pre-excite the atrium and reset the tachycardia without engaging the His bundle and thus presumably the compact AV node to do so. Tachycardias that can be reset by premature ventricular contraction (PVC) only by preexciting the His bundle are consistent with AV node reentry. An important exception to this premise occurs when a retrograde conducting NV/NF tract is present. Regardless of whether the primary arrhythmia is AV node reentry, orthodromic NV/NF tachycardia, or orthodromic AV reentrant tachycardia of another type, PVCs engage the His even when late-coupled at a time of apparent His refractoriness based on the timing of antegrade His bundle activation.

One of the other insightful contributions from Nayyar et al¹'s submission results from their analysis of why tachycardia was difficult to induce despite slow-conducting antegrade and retrograde limbs. They note a possible interesting role for an anterior nodoventricular fiber to paradoxically make tachycardia induction difficult. For instance, ventricular beats with retrograde conduction up a lateral accessory pathway and relatively early conduction down a nodoventricular fiber may produce a ventricular wavefront that arrives at the retrograde

lateral pathway when it is refractory. Interestingly, however, during atrial extrastimulus testing, early beats tended to block in the presumed nodoventricular fiber (loss of R wave in V1 and septal cues in the lateral leads).

Tough Stuff

Most often, even with complex forms of supraventricular tachycardia, an experienced electrophysiologist carefully performing and interpreting the available repertoire of diagnostic maneuvers can arrive at a definite diagnosis and then plan ablation. Some arrhythmias, however, are difficult to distinguish from each other and may represent points on a spectrum of abnormality where diagnosis may well be a semantic distinction. When confronted by these situations, in which we may not be able to precisely name the arrhythmia, we plan for the safest and most likely successful ablation.

AV Node Reentry With Bystander Retrograde Conducting NV/NF Tract Versus Orthodromic NV/NF Tachycardia

His bundle-refractory late PVCs in these tachycardias pre-excite the atrium suggesting an accessory pathway. However, other findings including simultaneous ventriculoatrial activation are consistent with AV node reentry. The diagnosis may indeed be AV nodal reentrant tachycardia, but with a bystander NV/NF retrograde conducting tract. The AV node can be activated even when antegrade His activation has just occurred, and indeed the tachycardia can be reset. A similar finding is seen with orthodromic NV/NF tachycardia. However, with orthodromic NV/NF tachycardia, the ventricle is part of the circuit, and as a result, the subsequent antegrade His is also necessarily advanced to reset the tachycardia.

Antidromic NV/NF Tachycardia Versus Antidromic Septal Atriofascicular Bypass Tract-Mediated Tachycardia

We generally consider antegrade atriofascicular or shorter AV decremental pathways to occur on the free wall and not on the septum. However, the logic behind this distinction is somewhat circular because of our principle diagnostic maneuver to identify these tracts. Namely, premature atrial contractions placed from the free wall without activating the septal A, resetting the tachycardia by advancing the next V with an identical QRS helps diagnose this entity. However, if the decremental atriofascicular tract is located on the septum, then the septal A is advanced when advancing the V and resetting the tachycardia. When this occurs, we often diagnose a NV/NF causing the tachycardia. Furthermore, the successful ablation site is often on the septum in the vicinity of the usual slow pathway input to the AV node where we may be successful in eliminating several tachycardias including AV nodal reentrant tachycardia, NV/NF-mediated tachycardia, and septal atriofascicular tract, and thus despite our inability to make an exact diagnosis, the arrhythmia may be eliminated.

His Bundle Refractoriness

The cornerstone for defining nodal versus extranodal antegrade or retrograde activation is determining whether the His bundle

is refractory when atrial or ventricular extrastimulation is delivered. This determination, however, is not straightforward and indeed can be impossible with present mapping systems.

Retrograde Activation

Even when a PVC is placed during or just after antegrade activation of the His bundle, the His bundle is bypassed, carrying the ventricular activation wavefront directly to the AV node, and thus AV nodal reentrant tachycardia may be terminated or reset by a PVC delivered when the His was refractory. Furthermore, when retrograde right bundle-branch block is present, PVCs from the ventricular apex may have considerably increased transit time and depolarize the His bundle even though they were delivered at a time where the His would likely be refractory. Previous ablation proximal to the ablation site or scar from other reasons could exaggerate this phenomenon.

Antegrade Conduction

Attempting to use the His as a surrogate for antegrade AV nodal activation is also complex. For example, when antidromic AV reentrant tachycardia is to be distinguished from AV node reentry with bundle-branch block, the recorded His bundle electrogram may represent activation that is either retrograde (via the bypass tract) or antegrade, and thus, the response to a premature atrial contraction delivered soon after a His electrogram is recorded cannot be interpreted without knowing how the His was activated. It is for this reason that in wide QRS tachycardia when PACs are placed, we specifically analyze the response looking at whether the septal atrial electrograms have been advanced rather than the His bundle. However, as mentioned above, the septal A is a poor surrogate for the AV node because there may be unusual inputs to the AV node such as from the left atrium or septal accessory pathways. Particularly those with decremental properties and that connect to the infra-Hisian conduction system can be nearly impossible to distinguish from NV/NF fibers or a duplicate AV node.

The reason for these difficulties is because we cannot record a discernible His electrogram when ventricular or atrial activation overlaps, and more importantly, we presently have no method of directly recording AV nodal conduction. Future mapping systems, some currently under development, aim to

filter and display His and Purkinje signals distinct from the ventricular electrogram and with sufficient temporal resolution and dynamic range may allow reliable recording of AV nodal conduction.⁷ At such time, complex maneuvers that require determining whether the His bundle is refractory and indeed the challenge of avoiding inadvertent AV nodal damage may become obsolete.

Disclosures

Dr Asirvatham received 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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