

A 24-year-old patient presented with history of recurrent palpitation and was diagnosed as wide QRS tachycardia which was cardioverted. The sinus rhythm ECG and the tachycardia ECG are shown in Figure 1. During the electrophysiological study, 2 morphologies of tachycardia were inducible, 1 with right bundle branch block (RBBB) morphology (Figure 2A) and another with left bundle branch block (LBBB) morphology (Figure 2B), which was her clinical tachycardia. The LBBB type wide QRS tachycardia was faster (Figure 2B). During the RBBB morphology tachycardia, ventricular entrainment showed a V–A–V response and a His refractory ventricular extrastimulation advanced the retrograde atrial activation, resetting the tachycardia.

The LBBB morphology tachycardia showed variation in cycle length with a constant Ventriculo–Atrial (VA) interval. Atrial entrainment did not change the QRS morphology or VA relationship. An early premature atrial extrastimulation from the lateral right atrium showed an interesting finding (Figure 3). Very late atrial extrastimulus from the same location did not advance the V without affecting the septal A. What are the mechanisms of the two tachycardia?

Commentary

The baseline ECG showed features typical of an accessory pathway, in the right posterior location. The clinical tachycardia was a regular wide QRS tachycardia (Figure 1B), with LBBB morphology and left axis deviation. The differential diagnosis of a regular wide QRS tachycardia in a patient with baseline pre-excitation includes (1) orthodromic reentrant tachycardia with aberrancy; (2) atrial flutter or atrial tachycardia with ventricular pre-excitation; (3) classical antidromic tachycardia with retrograde conduction through bundle branch–His–atrioventricular (AV) node axis; (4) pre-excited tachycardia due to pathway-to-pathway conduction; (5) atrioventricular nodal reentrant tachycardia (AVNRT) with bystander accessory pathway conduction; (6) orthodromic reentrant tachycardia with bystander activation of ventricles using another pathway; (7) ventricular tachycardia; (8) junctional tachycardia with aberrancy or fasciculoventricular connection; and (9) nodoventricular tachycardia (pathway arising proximal to the distal node).

The 12-lead ECG of the tachycardia gives certain clues to the underlying pathways. The typical antidromic tachycardia would have resulted in a wider QRS and predominantly positive complexes in the lateral precordial leads because of ventricular activation solely through the accessory pathway. Although atrial flutter or tachycardia are common causes of such a presentation, we would expect a more wide predominantly positive QRS complexes in the lateral precordial leads because of activation through the nondecremental accessory pathway. A fast conducting AV–His bundle may, however, result in relatively narrower QRS. When the tachycardia morphology does not correspond to the pre-excitation pattern in sinus rhythm, multiple pathways should be considered. The wide QRS tachycardia shows left axis deviation and LBBB and has a sharp rapid component of the initial part and has no precordial transition. These features are more typical of an atriofascicular (Mahaim) tachycardia, although the sinus rhythm ECG is not typical for a Mahaim fiber.¹² Most differential diagnoses, including ventricular tachycardia, should however be considered during the intracardiac study.

Intracardiac recording in Figure 2 shows an orthodromic tachycardia ([ORT] with RBBB aberrancy) with antegrade conduction through the His bundle, and retrograde conduction through a right-sided pathway (Figure 2A); earliest atrial activity was noted in the distal HALO electrodes in the posterolateral tricuspid annulus. An atrial tachycardia with aberrancy was ruled out by a VAV response to ventricular entrainment. A short postspacing interval and atrial pre-excitation by a His refractory ventricular stimulus confirmed the diagnosis.

The wide QRS tachycardia with LBBB morphology (Figure 2B) has a shorter cycle length but has the same eccentric retrograde activation sequence. It was initiated by catheter-induced ventricular ectopics during the ORT. The HV was negative (ventricular–His [VH]=80) showing that it is a truly pre-excited long VH tachycardia. At this point, it can be assumed that the H is a retrograde H (further confirmed by observations mentioned below). Two additional points should be noted in Figure 2B: (1) the retrograde activation sequence is same as during the ORT (tachycardia 1) and (2) the cycle length is considerably shorter during the wide QRS LBBB morphology rhythm.¹ These essentially rule out...
a classic antidromic tachycardia mediated by the same AV connection. However, the presence of RBBB or incorporation of the slow pathway during the ORT alone may cause it to be slower than antidromic tachycardia. Orthodromic reentry with aberrancy is ruled out by the negative HV. AVNRT with bystander accessory pathway is excluded because of eccentric atrial activation and because the retrograde activation did not change between the tachycardias. Further, in AVNRT with bystander activation, the VH is usually short and the H recorded is an antegrade H. A late atrial extrastimulus during the tachycardia when the AV junction is refractory can be used to diagnose AVNRT with bystander pathway4,5 (see below). A His refractory ventricular stimulus can also exclude AVNRT if it resets the tachycardia with the same activation sequence in atria—this was demonstrated in this case. The possibility then we have to consider is that the second tachycardia is because of a pathway-to-pathway conduction—antegrade conduction through a right-sided accessory pathway (LBBB, left axis), with a negative VH as noted in the figure and retrograde conduction through the same right posterolateral pathway mediating the ORT, with earliest atrial activation in HALO 1, 2. Pathway-to-pathway tachycardia are usually possible only when the pathways are at a considerable distance anatomically.3 When 2 pathways are close by, like in this case, tachycardia can be sustained more easily if at least one of the pathways has decremental conduction property. In such a scenario, the AVN can conduct retrogradely and activate part of the atria. The rare possibility of ORT with bystander pathway was previously reported; however, they are associated with minimal QRS widening and a short rather than negative HV.6

Atrial entrainment showed resetting of the tachycardia without alteration of the QRS morphology or VH interval, thus confirming atria as necessary part of the circuit and also making AVNRT or ORT with bystander pathway unlikely. If the atriofascicular pathway was merely a bystander, QRS morphology and the VH relationship would change with resetting of the tachycardia. This also rules out a myocardial VT. Coupled atrial premature stimuli also showed advancing of the V. A relatively early coupled atrial stimulus produced an interesting finding as noted in Figure 3, which shows a coupled

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**Figure 1.** Twelve-lead electrocardiograms of the patient in sinus rhythm (A) and during clinical tachycardia (B).
atrial extrastimuli during the pre-excited rhythm, which delayed the ventricular activation through the pathway and reset the tachycardia without changing the VH or VA interval. This demonstrates the decremental nature of the anterograde pathway during tachycardia. It also demonstrates that the H is a retrograde H related to V. If His bundle activation contributed

![Figure 2](image1.png)

**Figure 2.** Surface leads I, III, and V1 and intracardiac electrograms from right atrium (HALO 1, 2 at lateral right atrium to HALO 17, 18), His bundle (His bundle distal [HBED] and His bundle middle [HBEm]), the coronary sinus (coronary sinus distal [CSD] and coronary sinus proximal [CSP]) and right ventricular apex (RVA). A, The orthodromic tachycardia with right bundle branch block aberrancy. B, The left bundle branch block tachycardia of shorter cycle length and long ventricular–His of 80 ms with the same retrograde eccentric atrial activation as in the initial tachycardia.

![Figure 3](image2.png)

**Figure 3.** Surface leads I, III, and V1 and intracardiac electrograms from right atrium (HALO 1, 2 at lateral right atrium to HALO 17,18), His bundle (His bundle distal [HBED] and His bundle middle [HBEm]), the coronary sinus (coronary sinus distal [CSD] and coronary sinus proximal [CSP]) and right ventricular apex (RVA). The effect of an early coupled atrial stimulus (asterisk) during tachycardia is shown. CI indicates coupling interval of atrial stimulus; TCL, tachycardia cycle length; and V–V′, interval between surface QRS following the extrastimulus.
to activation of septal atrium, this resetting would have been expected to at least slightly alter the atrial activation pattern. Even very late atrial extrastimuli (delivered from the presumed site of antegrade pathway) advanced the septal A making it likely that atrium in the vicinity of the His is activated through the retrograde accessory pathway and there is H to A block during the LBBB tachycardia. Further, the atrial activation during both the ORT and the pre-excited tachycardia is the same showing that there is no retrograde activation of atrium through the AVN. During cycle length changes in the LBBB tachycardia the VH and VA interval remained constant. Thus, a diagnosis of pathway-to-pathway tachycardia, mediated by a Mahaim fiber and a closely located nondecremental pathway was made. The rare possibility of a pre-excited tachycardia because of a nodofascicular tachycardia could not be excluded at this stage; 2 potentially useful maneuvers—(1) demonstration of AV dissociation during tachycardia and (2) ventricular pre-excitation by atrial premature beat when septal atrium is refractory—could not be demonstrated, essentially because of the additional bypass tract involved.7

Patient subsequently developed an atrial tachycardia with 2:1 AV conduction through both accessory AV connection

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**Figure 4.** Fluoroscopic left anterior oblique projections of location and electrograms from the following catheters: the duo-decapolar catheter along the tricuspid annulus (HALO 1, 2 to 17, 18), the decapolar coronary sinus (CS), the mapping and ablation catheter (shown with asterisk, RFd and RFp) and quadripolar catheters in the His (His bundle distal [HBED], His bundle middle [HBEm], and His bundle proximal [HBEp]) and right ventricular apex (RVA). A, Atrial tachycardia with 2:1 conduction and M potentials at the ablation catheter at the site of successful ablation of the Mahaim fiber. Note the short M-Local V interval and simultaneous activation of the RVA and ventricular annulus (RFd) because of antegrade conduction through both pathways. B, The recording during orthodromic tachycardia, ablation at the posterolateral tricuspid annulus results in local separation of VA and termination of tachycardia (solid down arrow).
Ebstein’s anomaly of the tricuspid valve.

there was persistent RBBB and no VA conduction at 500 ms annulus region (Figure 4B). Post ablation of both pathways during ORT and successfully ablated at the posterior tricuspid
Successful ablation of the Mahaim fiber resulted in change in potential at 9 o’clock tricuspid location with a short M to (Figure 4A). Figure 4A shows the characteristic Mahaim proximate input and output elements for the AV node, namely the septal atrial myocardium near the slow and fast pathways, and the His bundle not reliably possible in the clinical electrophysiology laboratory, and AV nodal activation must be inferred from assessment of activation near the direction of its activation. Although it has been many years since the first description of the His bundle recording, direct AV nodal recording is still (AV) nodal activation.

and the reason for their careful analysis and execution of maneuvers to uncover the true diagnoses is our inability to directly record atrioventricular (AV) node). Most of the individual differential diagnostic possibilities can be excluded if we know when the compact AV node is being activated and the direction of its activation. Although it has been many years since the first description of the His bundle recording, direct AV nodal recording is still not reliably possible in the clinical electrophysiology laboratory, and AV nodal activation must be inferred from assessment of activation near the proximate input and output elements for the AV node, namely the septal atrial myocardium near the slow and fast pathways, and the His bundle and the His–Purkinje system. Because We Cannot Directly Measure…We Must Deduce…

In this installment of the Teaching Points in Cardiac Electrophysiology series of our journal, Thajudeen et al.2 describe interesting deductions of educational value from a patient with multiple potential mechanisms for arrhythmia. They step us through their thought process as the case unfolded, and how they included or excluded considerations that were relevant to the findings at EP study. Implicit in the difficulties that faced these operators and the reason for their careful analysis and execution of maneuvers to uncover the true diagnoses is our inability to directly record atrioventricular (AV) nodal activation.

key Words: accessory AV connection • Mahaim fibers • pre-excitation syndrome • wide QRS tachycardia

Disclosures

None.

References


EDITOR’S PERSPECTIVE

Because We Cannot Directly Measure…We Must Deduce…

Septal Refractoriness

Because the septal atrial myocardium (left or right) needs to be activated before antegrade compact AV nodal activation, I surrogates used to determine whether the AV node is responsible for conduction antegrade to the ventricle is the septal atrial electrograms. The authors emphasize the importance of a critically timed atrial extrastimulus at a site away from the septum (right freewall, and in this instance closer to the accessory pathway), but monitoring the septal atrial electrogram to deduce whether or not the extrastimulus affected ventricular activation via conduction through the AV node. In the absence of change in septal atrial activation, time advancing ventricular activation (QRS and ventricular electrograms) without changing the activation sequence (QRS morphology) and resetting the tachycardia is diagnostic of antegrade activation through an accessory pathway that is participating in tachycardia. Incidentally, the advancement of the septal atrial electrograms for the subsequent beat after the atrial extrastimulus helps in defining the retrograde limb of the tachycardia circuit. If the advanced ventricular activation, in turn, advances the septal atrial electrogram without changing the retrograde activation sequence when resetting the tachycardia and the septal A is earlier than lateral right atrial or coronary sinus atrial electrograms, then another free wall pathway as the retrograde limb is excluded. However, whether the retrograde limb is a septal accessory pathway or the AV conduction system cannot be determined with this maneuver alone, and analysis of another neighbor of the AV node, namely the septal atrial myocardium near the slow and fast pathways, and the His bundle and the His–Purkinje system.

His–Purkinje Refractoriness

Retrograde activation distinctions between the AV node and an accessory pathway are generally easier because the His bundle electrogram is distinct and a reliable marker for whether the AV node itself is being activated retrograde in the tachycardia. Nevertheless, when analyzing complex arrhythmia circuits with multiple pathways, etc, specifically noting the effect on tachycardia when His bundle activation is delayed or early can be helpful and sometimes allows analysis of the entire circuit of tachycardia with a single, critically timed atrial extrastimulus. For example, when during a wide complex tachycardia, if retrograde atrial activation is delayed when retrograde right bundle branch block occurs resulting in increased ventricular–His time and thus delay in retrograde His bundle activation, then the AV conduction system can be ascertained to be the retrograde limb of the circuit. Therefore, a right freewall-placed atrial extrastimulus that does not affect septal activation but advances the next ventricular activation without change in the QRS morphology and resets the tachycardia by advancing the next atrial activation and an increase in the ventricular–His interval that may occur spontaneously during tachycardia (retrograde right bundle branch block) results in delayed atrial activation without changing the atrial activation sequence is diagnostic of antiodromic tachycardia (antegrade limb is the accessory pathway; retrograde limb is the His–Purkinje system and AV node).
**Direction of Activation of the His**

The authors point out that when multipolar recordings of the His bundle are available, simply analyzing whether the His itself is activated antegrade or retrograde should be able to tell us whether the AV node is activated antegrade or retrograde. Although this is generally true, there are important caveats. For example, sometimes the His recording we obtain is actually the proximal and mid right bundle recording. In this case if there is proximal right bundle branch block during tachycardia (eg, AV node reentry), then the right bundle recordings may show distal earlier than proximal (retrograde penetration of the right bundle), falsely suggesting retrograde activation of the AV conduction system and thus possibly misdiagnosing atrioventricular nodal reentrant tachycardia with right bundle branch block as an anterograde tachycardia. Similarly, if retrograde right bundle branch block is present distally, then an appearance of antegrade activation of the His (proximal right bundle earlier than distal right bundle) may be seen during anterograde tachycardia again leading to misdiagnosis.

Analysis of the curves relating atrial extrastimulus timing to the A–H interval also indicates whether the His bundle is being activated retrograde rather than antegrade. For example, as the atrial cycle length increases, if the A–H continues to prolong, we can assume that antegrade activation is occurring through the AV node. When the curve suddenly plateaus, that is the A–H intervals becomes fixed or changes very slightly, then the antegrade AV node is probably blocked, and retrograde activation is occurring through an accessory pathway.

**Nomenclature**

It is important to be aware of the potentially confusing names applied to various types of accessory pathways that have changed over time as our understanding has evolved. For example, Mahaim described anatomic connections between the AV node and ventricle and proximal His–Purkinje system and ventricle. Today, however, the term Mahaim fiber has been commonly applied to atriofascicular or atrioventricular connections with decremental properties because these were initially confused with nodoventricular fibers based on their physiology. Further, the term Mahaim is not usually applied today to fascicularventricular fibers or nodoventricular fibers, despite the fact that they do occur and require recognition in the electrophysiology laboratory. Thajudeen et al illustrate elegantly in their discussion that the electrophysiologist should be most concerned with using observed physiology to define the anatomic sites that require targeted energy delivery rather than deciphering the nuances of confusing nomenclature.

Standard maneuvers with their own nuances of nomenclature have to be applied cautiously in complex situations such as those in this patient involving possible multiple accessory pathway. For example, the authors note that a V–A–V response excluded atrial tachycardia as a mechanism. However, when multiple pathways that can conduct antegrade in addition to the AV node are present, then a V–A–V response may be seen during atrial tachycardia with the retrograde and antegrade limb responsible for V–A and A–V activation are all bystanders. Similarly, the pre-excitation index has little or no value when analyzing the retrograde activation mechanism during ART.

**Leaky Annulus: Then and Now**

The contemporary electrophysiologist trainee may question why such maneuvers and deductive knowledge should be learned in the present era of atrial fibrillation and ventricular tachycardia ablation. However, difficult diagnostic dilemmas remain when the perianular tissue is activated early. We see this propensity in perimembranous and atrioventricular annular ventricular tachycardias (and PVCs), as well as atrial tachycardias with early activation near the annulus sometimes (as possibly in this case) mysteriously coexisting with other causes for supraventricular tachycardia. The ability to appreciate the anatomy of abnormal structures or locations that require ablation with observed physiology remains important.

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