Mechanism of a Wide QRS Complex Tachycardia With Variable Atrial, His, and Ventricular Relationships

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Case Presentation
A 57-year-old man with a structurally normal heart and normal baseline ECG (Figure 1) underwent pulmonary vein isolation for atrial fibrillation. An electrophysiology study was then undertaken with an octopolar catheter positioned at the His bundle and an ablation catheter at the mid-right atrium.

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Discussion

At baseline, the sinus cycle length was 890 ms, the AH interval 48 ms, the HV interval 80 ms, and the QRS duration 80 ms. During extrastimulus atrial pacing, the HV interval shortened and the QRS complex widened with a left bundle branch block morphology (Figure 2). Atrial burst pacing at cycle length 330 ms demonstrates progressive shortening of the HV interval and widening of the QRS complex with left bundle branch block morphology (Figure 3A and 3B). On the final 3 beats in the figure, a His bundle electrogram seems to precede the QRS complex. On termination of pacing, a wide QRS complex tachycardia of identical morphology is noted (Figure 4). The atrial (A), His (H), and ventricular (V) electrograms are labeled. What is the mechanism of the tachycardia?

Although the baseline ECG is normal, the presence of a slowly conducting accessory pathway is apparent from atrial extrastimulus pacing demonstrating HV interval shortening, QRS widening, and a long A–V interval (Figure 2). Further inferences of the nature of the accessory pathway may also be made. The retrograde right bundle (RB) and His activation preceding the preexcited ventricular complex with left bundle branch block morphology is most consistent with an accessory pathway connection directly into the right bundle branch (atriofascicular pathway or nodo-fascicular pathway).

Similar changes are noted at the onset of atrial burst pacing (Figure 3A). There is evidence of a slowly conducting accessory pathway with shortening of HV interval and a long A–V interval. The His electrogram precedes the preexcited ventricular complex. Because only the distal electrode records an His electrogram, retrograde versus antegrade His activation cannot be discerned on this tracing. The H and V relationship changes further on the sixth paced beat when the His bundle electrogram occurs after the ventricular electrogram. Having established the presence of an atrio-fascicular (or nodo-fascicular) pathway, the only explanation for the late His is the development of retrograde block in the RB. Most likely, the His activation is still retrograde via transseptal activation of the left bundle branch. This is represented schematically in Figure 3B. Less likely, retrograde block in the RB allows for antegrade conduction over an AV node slow pathway and antegrade His activation.

At termination of the atrial pacing, a left bundle branch block morphology wide QRS tachycardia is initiated (Figure 4). The tachycardia morphology is identical to the pre-excited QRS complex. The His activation is not seen on the first beat of the tachycardia but occurs just before the onset of the QRS for the second and third beats. The His recording is seen after the ventricular signal for the last 2 complexes. Changes in the H–H intervals precede changes in the V–V intervals as well as the subsequent A–A intervals, although the relationship is not precise (Figure 5A). The H–A interval shortens on the last 2 beats and accounts for the inexact relationship.

The differential diagnosis for the wide QRS complex tachycardia includes ventricular tachycardia, supraventricular tachycardia (SVT) with aberrant conduction, SVT with bystander accessory pathway conduction and preexcited (antidromic) tachycardia. Bundle branch reentry ventricular tachycardia and SVT with aberration are excluded by the short HV interval. Myocardial ventricular tachycardia with retrograde His activation is also excluded by the variable H and V relationships with changes in the H–H interval predicting changes in the V–V intervals.

Two possibilities remain: antidromic tachycardia and SVT (AV node reentry tachycardia or atrial tachycardia) with bystander accessory pathway conduction. The most probable mechanism is antidromic atrioventricular reentry tachycardia (AVRT). Development of retrograde block in the RB and transseptal conduction enlarges the circuit; increases the tachycardia cycle length; and—because of simultaneous conduction down the RB and up the His and AV node—shortens the RB–A interval (Figure 5B and 5C). The tachycardia cycle length prolongation during retrograde RB block is diagnostic of antidromic AVRT as it provides evidence for participation of the ventricle in the tachycardia. However, the possibility of SVT occurring simultaneously with antidromic AVRT is not excluded. During the first 2 beats of tachycardia, antidromic AVRT with its faster cycle length could entrain SVT. In the

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third and fourth beat, retrograde block in the RB prolongs the AVRT cycle length beyond that of an underlying SVT, allowing it to manifest (Figure 5D). In this scenario, the H–A interval (or RB–A interval) is longer during the first 2 AVRT beats because of the sequential retrograde conduction system activation and the HA interval shortens as expected for the third and fourth SVT (AV node reentry tachycardia) beats because of simultaneous conduction down the His bundle and up the AV node fast pathway.

Proof of an atriofascicular as opposed to nodofascicular accessory pathway mediated antidromic tachycardia was confirmed by delivery of a paced premature atrial complex at a time of atrial septal refractoriness during the long V–H tachycardia.\(^1,2\) This advanced ventricular and subsequent atrial activation equally (Figure 6). A discrete accessory pathway potential was identified at the lateral tricuspid valve annulus (Figure 7) and a single 40 W radiofrequency energy application eliminated preexcitation and the tachycardia. This case illustrates the diagnostic opportunities and challenges with simultaneous variations in multiple limbs of a reentrant rhythm.

Disclosures

None.

References


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**Figure 1.** Baseline 12 lead ECG.
Figure 2. Atrial extrastimulus pacing. Following S2, the QRS widens, the HV interval shortens and the His and right bundle activation sequence reverses. Dotted line and arrows provide reference for the anterograde to retrograde activation change. Pre-excitation with a long AV interval, left bundle branch block morphology, and right bundle activation preceding ventricular activation suggest an atriofascicular accessory pathway. His 7 to 8 through 1 to 2 His bundle catheter recordings are proximal to distal; pacing intervals in milliseconds are marked. H indicates His; RA, right atrium; S1, drive train stimulus; and S2, extrastimulus. Legends are same in all figures.

Figure 3. A. Right atrial pacing at 330 ms. A, H, and V electrograms are labeled. Note shortening of HV interval and fusion of QRS on the third-paced beat, retrograde His activation via right bundle branch on the fourth- and fifth-paced beats and sudden jump to a V–H relationship on the sixth-paced beat indicating retrograde block in the right bundle branch and conduction via left bundle branch. B. Schematic diagram of the mechanism of changes A, H, and V relationships seen in the last 3 beats in A. Atrial pacing results in anterograde block in AV node, anterograde conduction via atriofascicular pathway, and retrograde RB block. His 7 to 8 through 1 to 2 His bundle catheter recordings are proximal to distal; pacing intervals in milliseconds are marked. H indicates His; RA, right atrium; S1, drive train stimulus; and S2, extrastimulus. Legends are same in all figures.

Figure 4. Atrial pacing initiates tachycardia with left bundle branch block morphology, variable cycle lengths and variable relationships between the A, H, and V signals. A indicates atrial; H, His; RA, right atrium; and V, ventricular.
Figure 5. A. Electrogram intervals of tachycardia in Figure 4. All intervals are in milliseconds. B–D, Schematic representation of tachycardia circuit changes in A. B, First 3 beats of the tachycardia. With cessation of atrial pacing anterograde conduction continues down the atriofascicular pathway with retrograde conduction via right bundle branch completing the circuit. C, Fourth and fifth tachycardia beats where retrograde block occurs in the right bundle branch, and transseptal conduction and left bundle branch activation forms the retrograde tachycardia limb. D, Alternative possibility of dual AV node physiology during AVNRT with atriofascicular pathway conduction as a bystander. A indicates atrial; AFP, atriofascicular pathway; AVN, atrioventricular node; AVNRT, AV node reentry tachycardia; H, His; LB, left bundle branch; RA, right atrium; RB, right bundle branch; and V, ventricular.

Figure 6. A premature atrial complex (S₂) introduced at the time of atrial septal refractoriness during the long V–H tachycardia advances the ventricular and atrial activation. A indicates atrial; H, His; RA, right atrium; and V, ventricular.

Figure 7. Atriofascicular pathway potential (M) is recorded from the ablation catheter (ABL) placed at the lateral tricuspid annulus. Ablation here eliminated preexcitation. Note the short HV intervals on H1–2 electrodes. H indicates His.
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