Supraventricular Tachycardia with 2:1 Atrioventricular Block: What is the Mechanism?

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Case Presentation

A 20-year-old woman underwent an electrophysiologic procedure for treatment of paroxysmal supraventricular tachycardia. A tachycardia with 2:1 AV block was induced by programmed atrial stimulation with a single extrastimulus. The response to a single ventricular extrastimulus introduced during the tachycardia is shown in Figure 1A. How does the ventricular premature depolarization convert the 2:1 block to 1:1 conduction? What is the mechanism of the tachycardia?

Commentary

Figure 1A demonstrates a supraventricular tachycardia with an atrial cycle length of 240 msec and 2:1 AV block that is replaced by 1:1 conduction after the introduction of a ventricular premature depolarization. Orthodromic reciprocating tachycardia is immediately ruled out by the presence of 2:1 AV block, leaving atrial tachycardia and AV nodal reentrant tachycardia in the differential diagnosis. The atrial activation sequence and the AV relationship when there is 1:1 conduction are compatible with either possibility.

The resolution of 2:1 AV block on the introduction of a ventricular premature depolarization indicates that the AV block was functional in nature. By retrogradely penetrating the His-Purkinje system, the ventricular premature depolarization eliminates the "long-short" sequence that perpetuates the 2:1 AV block (Fig. 1B). AV nodal block is always a rate-related phenomenon and never resolves after a ventricular premature depolarization. Therefore, the functional block observed in this case is most likely intra-Hisian.¹ The very small Hisbundle depolarizations seen in Figure 1A in association with AV block are consistent with intra-Hisian block.

Functional 2:1 AV block has been reported to occur in a small percentage of AV nodal reentrant tachycardias.¹ In contrast, when 2:1 AV block occurs in association with an atrial tachycardia, the AV block is always rate related and not functional in nature. Therefore, the resolution of AV block in response to a ventricular premature depolarization observed in the present case indicates that the mechanism of tachycardia was AV nodal reentry. The tachycardia was eliminated successfully by radiofrequency ablation of the slow pathway.

The reason that functional 2:1 AV block is much more likely to occur in association with AV nodal reentrant tachycardia than atrial tachycardia is not entirely clear. It is possible that the initiation of AV nodal reentrant tachycardia by conduction down the slow pathway is more likely to create a "long-short" sequence responsible for functional block.

Also noteworthy in Figure 1A is the transient right bundle branch block that occurs on resumption of 1:1 AV block. Although the ventricular premature depolarization resulted in a prior HH interval not long enough to prolong refractoriness to a degree sufficient to cause AV block, there was still enough of a "long-short" sequence to result in block or slowed conduction in the right bundle. The right bundle branch block persists in one additional QRS complex due to a linking phenomenon that then resolves.²

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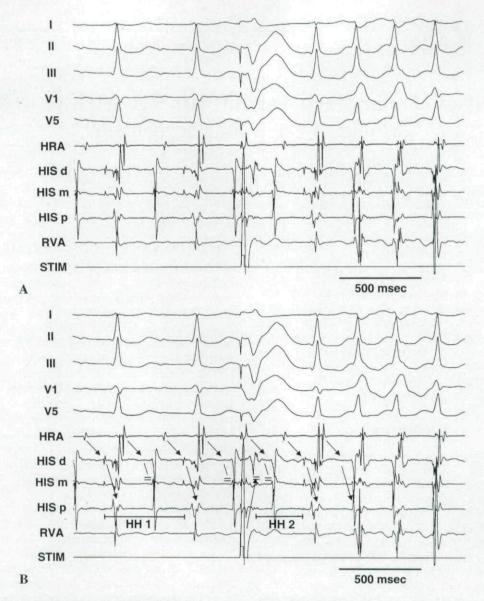


Figure 1. (A) Supraventricular tachycardia with 2:1 AV block that resolves after introduction of a single ventricular extrastimulus. Shown are leads I, II, III, VI, and V5, intracardiac electrograms recorded at the high right atrium (HRA), distal, medial, and proximal His-bundle positions (HIS d, HIS m, and HIS p), and right ventricular apex (RVA), and a stimulus channel (STIM). (B) Presumed mechanism by which a ventricular premature depolarization converts 2:1 AV block to 1:1 conduction. Functional block occurs in the distal portion of the His bundle because of a long prior HH interval (HH 1). By depolarizing the distal portion of the His bundle, the ventricular premature depolarization results in shortening of the HH interval (HH 2). A lesser degree of prolongation in the prior HH interval allows resolution of 2:1 intra-Hisian block.

References

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