

Wide QRS Tachycardia with Atrioventricular Dissociation and an HV Interval of 60 msec: What Is the Tachycardia Mechanism?

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Case Presentation

A 63-year-old man had an episode of unexplained syncope. He had triple-vessel coronary artery disease, a history of an anterior and inferior myocardial infarction, and had undergone a coronary artery bypass operation 3 years earlier. Cardiac catheterization demonstrated a left ventricular ejection fraction of 0.17, and patency of all three saphenous vein grafts. An electrophysiologic test was performed. The baseline sinus cycle length was 760 msec, the QRS duration was 110 msec, and the atrial-His and His-ventricular (HV) intervals were 120 and 55 msec, respectively. Sinus node function was normal, and there was no carotid hypersensitivity, infranodal block during atrial pacing, or evidence of dual AV nodal physiology. The AV block cycle length was 370 msec. Tachycardia was not inducible by atrial pacing. A wide QRS complex tachycardia having a cycle length of 210 msec was induced by programmed ventricular stimulation (Fig. 1). The tachycardia had a left bundle branch block configuration (Fig. 2). When sustained, the tachycardia resulted in severe hypotension and required electrical cardioversion. Sometimes the tachycardia was nonsustained (Fig. 3). What is the tachycardia mechanism?

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Commentary

The intracardiac electrograms recorded during this wide QRS complex tachycardia demonstrate AV dissociation and a His-bundle depolarization preceding each QRS complex, with an HV interval during tachycardia of 60 msec. AV dissociation rules out the possibility of atrial tachycardia, orthodromic reciprocating tachycardia, and antidromic tachycardia using an AV or atriofascicular accessory pathway. Furthermore, the HV interval of 60 msec indicates that the ventricle is not being activated through a nodoventricular bypass tract.

Theoretically, there are six tachycardia mechanisms that could account for a wide QRS complex tachycardia that has AV dissociation and an HV interval of 60 msec: (1) ventricular tachycardia, with retrograde activation of the His bundle; (2) bundle branch reentry tachycardia; (3) automatic junctional tachycardia; (4) AV nodal reentrant tachycardia, with retrograde upper common pathway block; (5) His-bundle reentry; and (6) a reentrant tachycardia using the AV conduction system as the anterograde limb and a concealed nodoventricular bypass tract as the retrograde limb of the reentry circuit. From the standpoint of clinical practice, the first two possibilities are the most likely, the third and fourth possibilities are unusual but occur often enough to warrant serious consideration, and the fifth and sixth possibilities are probably extremely rare or nonexistent and mostly of academic interest.

The list of possible tachycardia mechanisms can be quickly narrowed down by consideration of

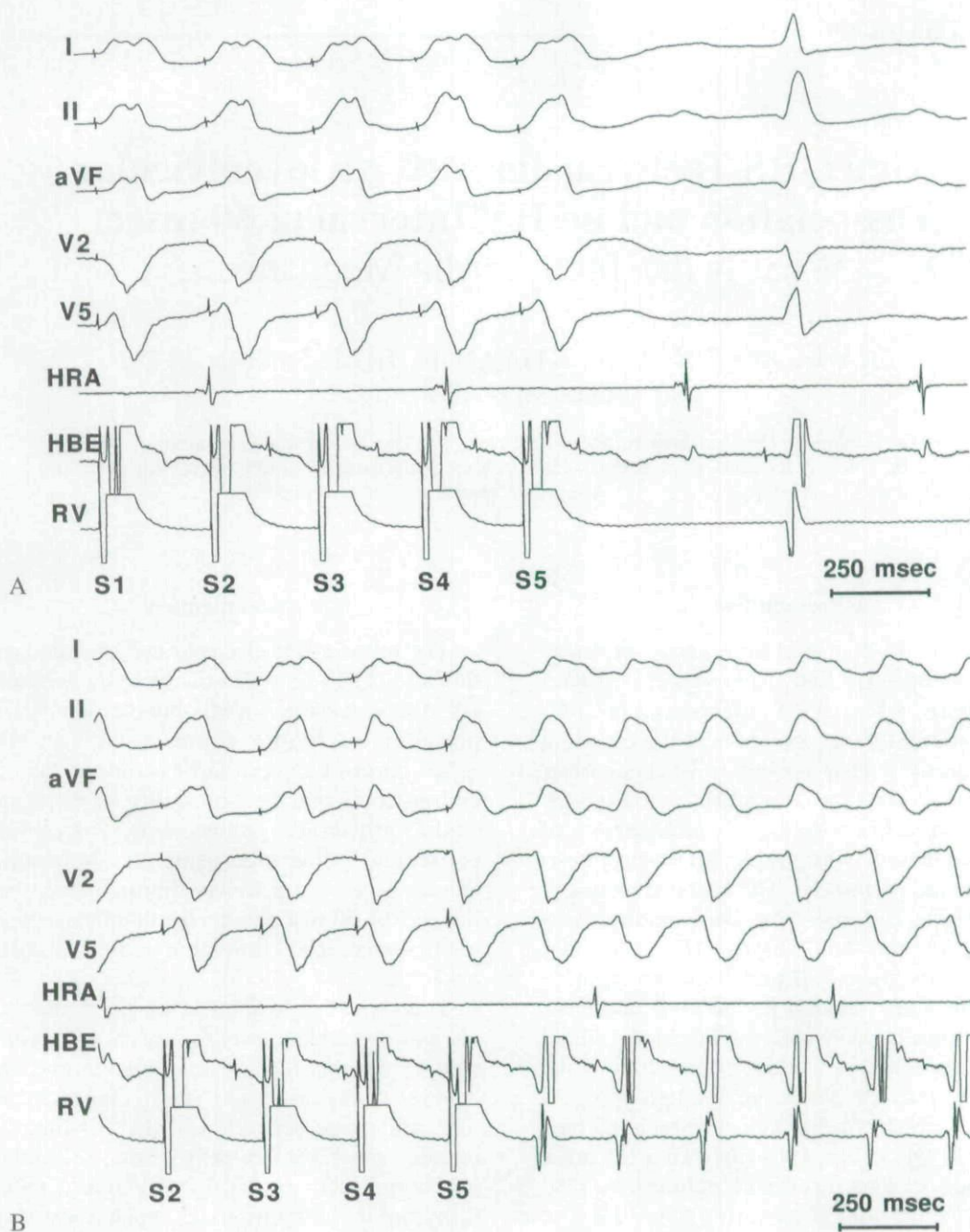


Figure 1. Induction of tachycardia by programmed ventricular stimulation. (A) Programmed ventricular stimulation with four extrastimuli (S2, S3, S4, S5) at coupling intervals of 290, 280, 270, and 260 msec does not induce tachycardia. There is retrograde activation of the His bundle after each paced ventricular complex, and the ventricular-His interval after S5 is 130 msec. Shown are leads I, II, aVF, V2, and V5, the high right atrial electrogram (HRA), His-bundle electrogram (HBE), and right ventricular electrogram (RV). (B) When the coupling intervals of S2, S3, S4, and S5 were shortened to 270, 260, 250, and 240 msec, the ventricular-His interval after S4 was 170 msec, and tachycardia was induced. Abbreviations as in panel A.

some of the basic findings of the electrophysiologic study. The fact that the tachycardia was inducible only by programmed ventricular stimulation makes automatic junctional tachycardia unlikely. The fact that the AV block cycle length was

370 msec, which was much longer than the tachycardia cycle length of 210 msec, suggests that the AV node was not part of the tachycardia circuit, making AV nodal reentrant tachycardia and a tachycardia using a concealed nodoventricular bypass

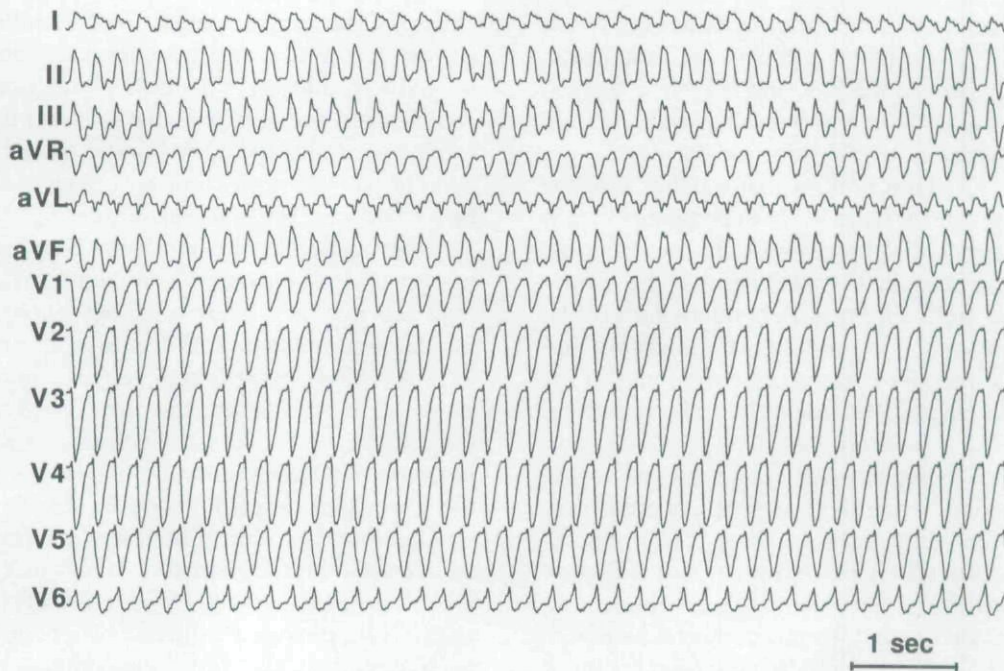


Figure 2. Twelve-lead ECG of the wide QRS tachycardia. The cycle length is 210 msec.

tract unlikely. When reentry occurs within the His bundle, two His-bundle potentials should be present in diastole; in this case, only one His-bundle potential is seen in diastole, making His-bundle reentry unlikely. Therefore, the two most likely possibilities are ventricular tachycardia and bundle branch reentry.

When there is spontaneous variability of the tachycardia cycle length, as occurs in Figure 1B, analysis of the R-R and His-His intervals may be helpful in distinguishing ventricular tachycardia from bundle branch reentry. In bundle branch reentry, changes in the His-His interval should precede changes in the R-R interval, and the opposite should

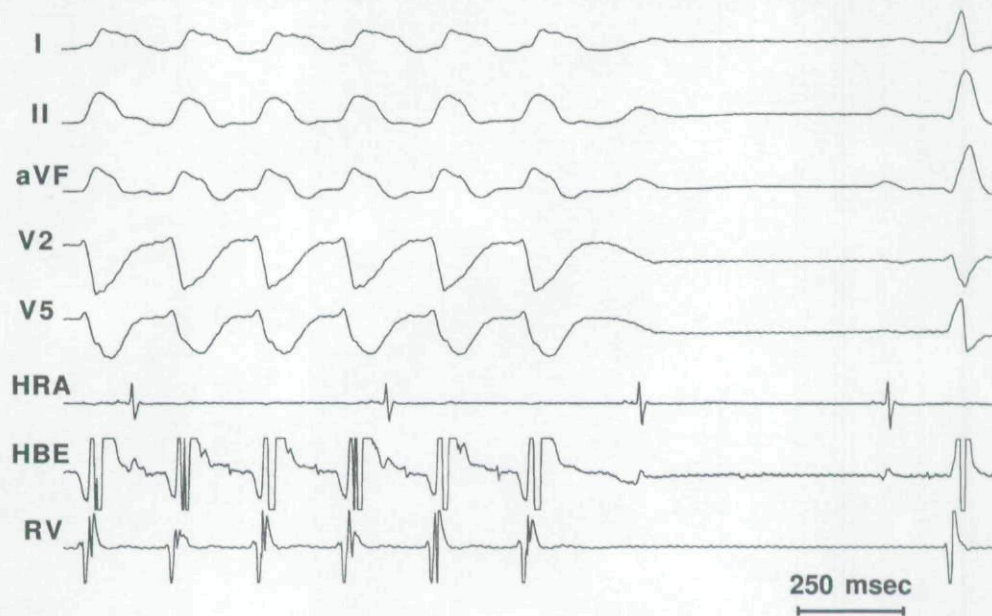


Figure 3. Spontaneous termination of the wide QRS tachycardia. Abbreviations as in Figure 1A.

be true in ventricular tachycardia. However, in the present case, there is not a consistent relationship between changes in the R-R and His-His intervals, and analysis of these intervals does not provide any diagnostically useful information.

Two findings are present that indicate that the tachycardia is much more likely to be bundle branch reentry tachycardia than ventricular tachycardia. The first, as seen in Figure 1, is that the induction of the tachycardia is dependent on critical lengthening of the ventricular-His interval, suggesting that the His-Purkinje system is part of the tachycardia circuit. This not only makes ventricular tachycardia unlikely, but also provides additional evidence against AV nodal reentrant tachycardia. Second, when there is spontaneous termination of the tachycardia, as shown in Figure 3, the last ventricular electrogram is not followed by a His-bundle electrogram. If this were ventricular tachycardia with 1:1 retrograde conduction to the His bundle, there would be no reason for block between the ventricle and the His bundle to be associated with termination of the tachycardia. On the other hand, in bundle branch reentry block in the retrograde limb of the circuit, in this case the left bundle, would result both in the absence of a subsequent His-bundle depolarization and also in the termination of tachycardia.

Radiofrequency ablation of the right bundle was performed. The tachycardia was no longer inducible, either immediately after ablation of the right bundle or 1 week later, providing confirmation that bundle branch reentry was the correct di-

agnosis. The typical patient with bundle branch reentry has a dilated cardiomyopathy and a prolonged HV interval. This case points out that exceptions are possible and that it is worthwhile to think about and look for bundle branch reentry even in patients with coronary artery disease who have a normal HV interval.

From an academic standpoint, it must be admitted that a concealed nodoventricular bypass tract was not definitively ruled out before the right bundle was ablated. It is true that the relatively long AV block cycle length suggests noninvolvement of the AV node, but this could be countered by arguing that the block occurs above the insertion of a nodoventricular bypass tract and that the lower portion of the node is capable of rapid conduction. The ability to reset or terminate the tachycardia with a premature ventricular depolarization coincident with His-bundle refractoriness might have provided evidence of a nodoventricular bypass tract, but this maneuver was not feasible because of hemodynamic instability. Had a right bundle potential been recorded, the relationship between the His- and right bundle potentials during tachycardia compared to sinus rhythm also might have been helpful in the differential diagnosis. Nevertheless, because termination of the tachycardia in association with block between the ventricle and His bundle was very suggestive of bundle branch reentry and because concealed nodoventricular bypass tracts probably are extremely rare, the decision to ablate the right bundle was clinically justified.

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