ARRHYTHMIA OF THE MONTH

Section Editor: Fred Morady, M.D.

A Preexcited Left Bundle Branch Block Tachycardia: What is the Tachycardia Mechanism?

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Case Presentation

A 52-year-old woman underwent an electrophysiologic procedure because of a 30-year history of recurrent episodes of paroxysmal tachycardia. The baseline sinus cycle length was 600 msec and the QRS complexes had a left bundle branch block configuration, with a QRS duration of 120 msec. The atrial-His (AH) and His-ventricular (HV) intervals were 90 and 20 msec, respectively. Programmed atrial stimulation with a single extrastimulus demonstrated progressively greater degrees of ventricular preexcitation, with the HV interval shortening to –15 msec as the AV interval increased from 110 to 200 msec. Programmed atrial stimulation with two extrastimuli resulted in the induction of a tachycardia that had a cycle length of 310 msec and a left bundle branch block configuration (Fig. 1), identical to the QRS complexes generated in response to

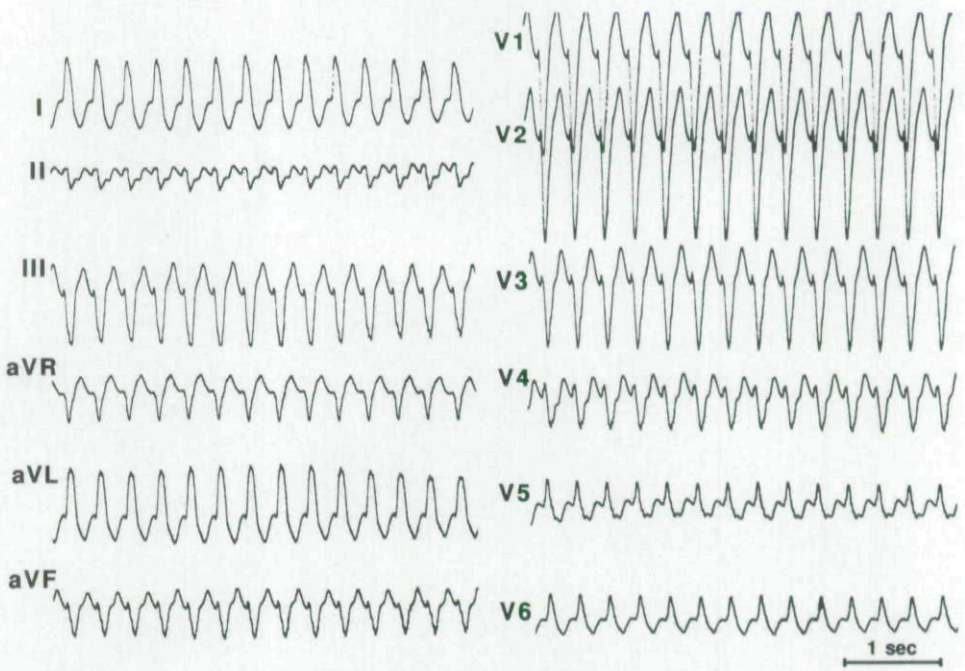

Figure 1. *Tachycardia having a left bundle branch block configuration and a cycle length of 310 msec.*

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programmed atrial stimulation. The HV interval during the tachycardia was –15 msec (Fig. 2). What is the tachycardia mechanism?

Commentary

The baseline HV interval of 20 msec indicates that there is ventricular preexcitation during sinus rhythm, and the results of electrophysiologic testing indicated the presence of a bypass tract that had decremental properties. Given the fact that the QRS complexes during tachycardia had the same configuration as the preexcited QRS complexes generated in response to atrial pacing, the tachycardia shown in Figure 1 can be presumed to be preexcited. A left bundle branch block configuration during preexcitation may occur with a right-sided accessory AV connection, atriofascicular bypass tract, or nodoventricular/nodofascicular pathway. With right-sided accessory AV connections, the precordial transition from a small to large R wave typically occurs between leads V_2 and V_4, whereas a precordial transition between leads V_4 and V_5, as seen in Figure 1, is typical of tachycardias involving an atriofascicular or nodoventricular/nodofascicular pathway.

The presence of an atriofascicular or nodoventricular/nodofascicular pathway is consistent with some of the findings in Figure 2, namely, substantial lengthening of the AV and AH intervals in association with shortening of the HV interval from 20 to –15 msec and progressive preexcitation of the QRS complex (Fig. 3). In addition, the right ventricular apex electrogram is coincident with the ventricular depolarization in the His-bundle electrogram when there is not preexcitation (as occurs following S3), but precedes the ventricular depolarization in the His-bundle electrogram by 40 to 50 msec when there is preexcitation. This is also consistent with an atriofascicular or nodoventricular/nodofascicular pathway. However, the fact that the tachycardia continues despite VA Wenckebach block (Fig. 3) rules out involvement of the atrium, thereby also ruling out the possibility of an atriofascicular bypass tract. Therefore, ventricular activation during the tachycardia is via a nodoventricular or nodofascicular pathway.

There are two mechanisms of tachycardia that could account for ventricular activation over a nodoventricular or nodofascicular pathway in association with VA block. The first is an antidromic reentry circuit in which the nodoventricular or nodofascicular pathway serves as the anterograde limb and the AV node–His-Purkinje axis as the retrograde limb. The second is AV nodal reentrant tachycardia in which the nodoventricular or nodofascicular pathway is an "inno-

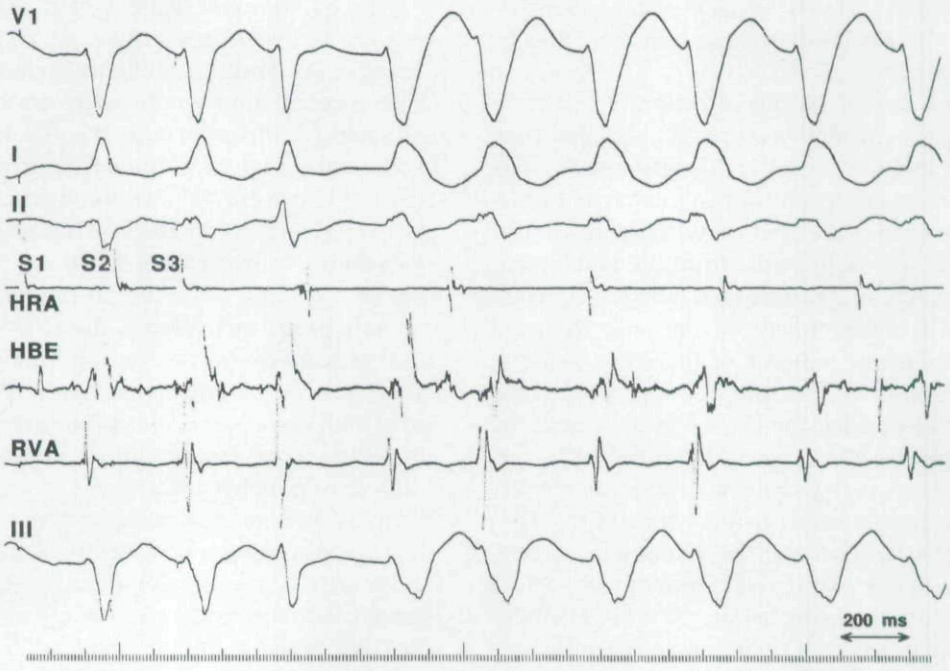

Figure 2. *Induction of the tachycardia by programmed atrial stimulation. Shown are leads V1, I, and II, the high right atrial (HRA) electrogram, the His-bundle electrogram (HBE), the right ventricular apex (RVA) electrogram, and lead III.*

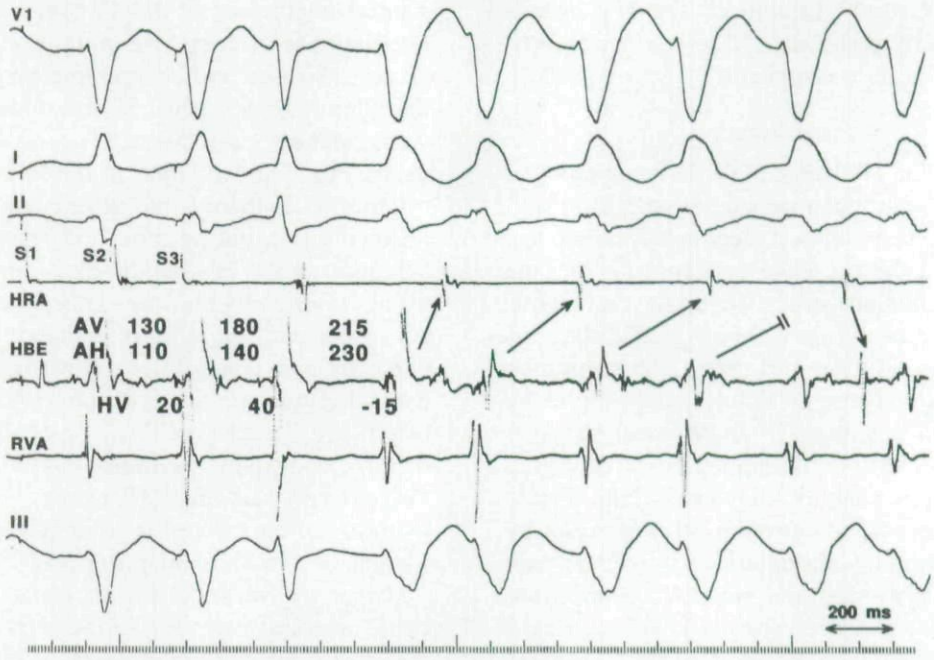

Figure 3. *The same tracing as in Figure 2, with atrioventricular (AV), atrial-His (AH), and His-ventricular (HV) intervals designated in milliseconds. The second atrial extrastimulus (S3) induces an atrial echo that initiates the tachycardia. Note that the first atrial extrastimulus (S2) is followed by a QRS complex that is partially preexcited, with an HV interval of 20 ms. When there is maximal preexcitation, as in the first QRS complex of the tachycardia, the HV interval shortens to –15 ms in association with lengthening of the AV interval from 130 to 215 ms. This is typical of an atriofascicular or nodoventricular/nodofascicular bypass tract. The arrows depict the pattern of VA conduction. The fact that the tachycardia continues despite VA block rules out an atriofascicular bypass tract.*

cent bystander." The issue in this case then becomes how to distinguish these two mechanisms of tachycardia.

VA block during AV nodal reentrant tachycardia probably is a rare occurrence, and this may be a point in favor of an antidromic reentry circuit. There are other types of evidence, not present in this case, that also could help to distinguish antidromic tachycardia from AV nodal reentrant tachycardia in a patient with a nodoventricular or nodofascicular pathway: (1) because the right ventricle is an integral part of the reentry circuit in an antidromic tachycardia but not in AV nodal reentrant tachycardia, the ability to dissociate the right ventricular electrogram from the tachycardia with ventricular pacing would favor the latter; (2) a variable relationship between the His-bundle depolarization and the ventricular electrogram would favor AV nodal reentrant tachycardia over an antidromic tachycardia; (3) if the His-bundle potential precedes a right bundle potential during the tachycardia, this would imply anterograde activation of the His-Purkinje system and would be definitive evidence of AV nodal reentrant tachycardia; (4) variable degrees of preexcitation during the tachycardia also would be definitive evidence of AV nodal reentrant tachycardia, since variable preexcitation would necessitate anterograde conduction through both the AV node and the nodoventricular or nodofascicular pathway; and (5) if there were 1:1 VA conduction during the tachycardia, a comparison of the His-atrial intervals during tachycardia and during ventricular pacing at the same cycle length might be useful. In an antidromic tachycardia, the His-atrial intervals during tachycardia and during ventricular pacing should be very similar, whereas in AV nodal reentrant tachycardia, the His-atrial interval would be expected to be longer during ventricular pacing than during tachycardia.

In the present case, it was felt that an antidromic tachycardia using a nodoventricular or nodofascicular bypass tract as the anterograde limb of the reentry circuit was the most likely mechanism, but that AV nodal reentrant tachycardia could not be ruled out. Attempts to ablate the bypass tract at its ventricular insertion were unsuccessful. In the hope that the AV nodal insertion of the pathway might

be in the posterior portion of the AV node, and because the possibility of AV nodal reentrant tachycardia had not been ruled out, several applications of radiofrequency energy were delivered in the posterior septum, near the ostium of the coronary sinus, at typical slow pathway ablation sites, but this also was ineffective. The failure to eliminate the tachycardia by slow pathway ablation might be additional evidence that the tachycardia mechanism was not AV nodal reentry. The patient was treated with flecainide and has had no further episodes of symptomatic tachycardia.