ECG OF THE MONTH

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What Is the Tachycardia Mechanism?—II

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Case Presentation

A 34-year-old man with a 10-year history of paroxysmal supraventricular tachycardia underwent an electrophysiologic procedure. A narrow-QRS complex tachycardia with a cycle length of 260 to 280 msec reproducibly was induced by right atrial overdrive pacing. At times there was a 1:1 relationship between the atrium and ventricle (Fig. 1), and at other times there was Mobitz I second-degree AV block during the tachycardia (Fig. 2). The atrial electrogram recorded by the His-bundle catheter preceded the high right atrial electrogram. At a time when the tachycardia cycle length was 280 msec and there was a 1:1 AV relationship, ventricular overdrive pacing was performed at a cycle length of 270 msec (Fig. 3). What is the mechanism of this tachycardia?

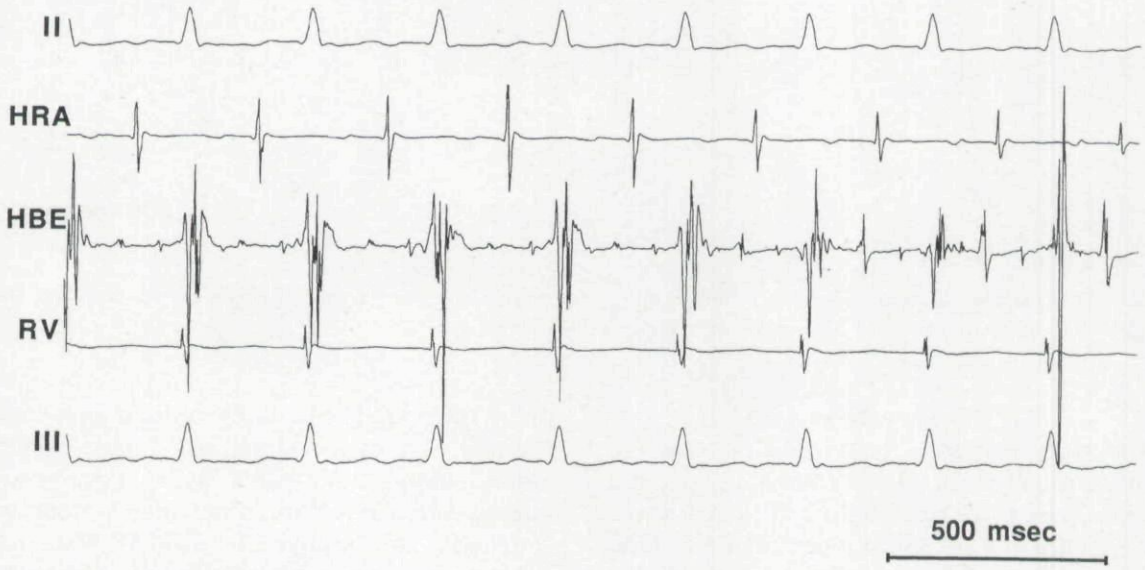

Figure 1. *A long-RP narrow-QRS tachycardia with an RR cycle length of 280 msec. The tachycardia has a 1:1 AV relationship. HBE = His-bundle electrogram; HRA = high right atrial electrogram; RV = right ventricular electrogram.*

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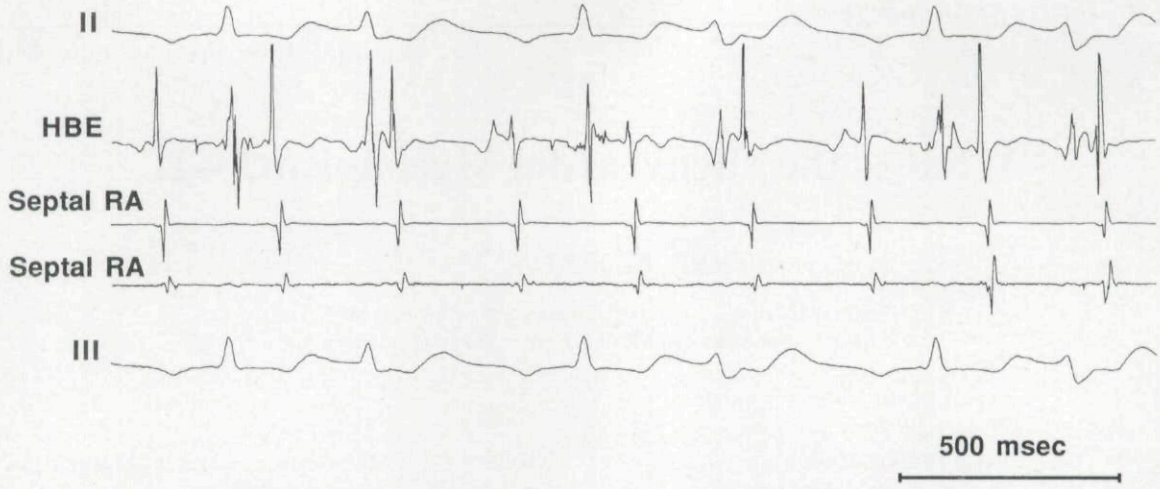

Figure 2. *The tachycardia cycle length has shortened to 260 msec, and there is now a 3:2 pattern of Mobitz I (Wenckebach) AV block during the tachycardia. RA = right atrium; other abbreviations as in Figure 1.*

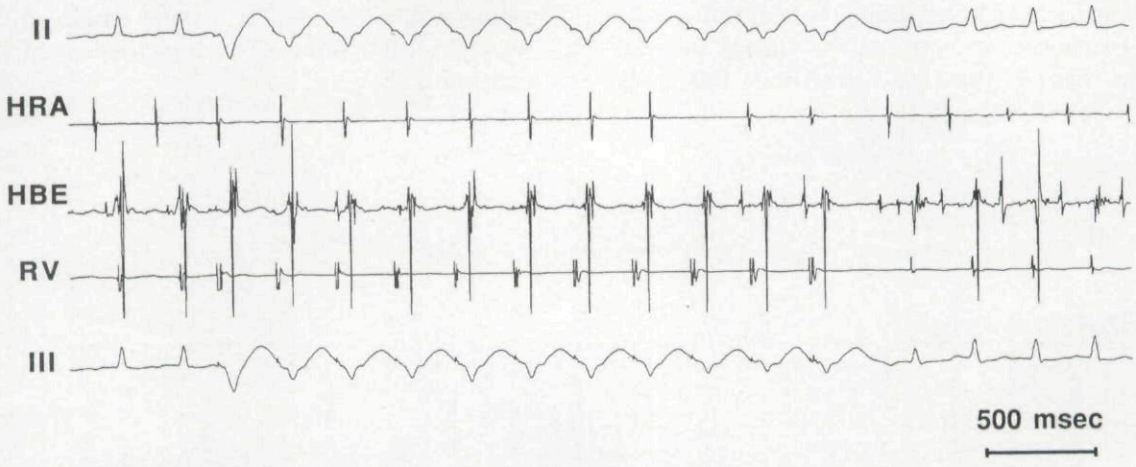

Figure 3. *The response to right ventricular pacing at a cycle length of 270 msec, at a time when the tachycardia cycle length was 280 msec and there was a 1:1 AV relationship. Abbreviations as in Figure 1.*

Commentary

The supraventricular tachycardia presented in this case usually had a 1:1 AV relationship, but a slight decrease in the tachycardia cycle length sometimes resulted in a Wenckebach pattern of AV block, with continuation of the tachycardia. When the tachycardia had a 1:1 AV relationship, the His-atrial interval was longer than the atrial-His interval, consistent with a long-RP tachycardia.

The differential diagnosis of a long-RP tachycardia consists of orthodromic tachycardia utilizing a relatively slowly conducting accessory pathway, atrial tachycardia, and atypical ("short-long") AV nodal reentrant tachycardia. The first possibility is easily excluded, since it is impossible for orthodromic tachycardia to continue in the presence of AV block. Although a Wenckebach pattern of AV block during a paroxysmal supraventricular tachycardia is much more common with atrial tachycardia than AV nodal reentrant tachycardia, it should be kept in mind that a Mobitz I or Mobitz II pattern of AV block at times may occur because of block distal to the lower turnaround point of an AV nodal reentry circuit. Therefore, it cannot be assumed that the tachycardia is atrial, and diagnostic maneuvers are necessary to differentiate atrial from AV nodal reentrant tachycardia.

The response to ventricular pacing provided the key evidence in this case. Ventricular overdrive

pacing during the tachycardia at first had no effect on the tachycardia, as evidenced by the unperturbed atrial electrograms recorded in the high right atrium. However, coincident with the ninth ventricular complex of the pacing train, there is abrupt termination of the tachycardia, manifest as a sudden lengthening of the atrial cycle length. The ability of ventricular overdrive pacing to terminate the tachycardia without premature depolarization of the atrium indicates that the tachycardia was not being generated in the atrium and demonstrates that the tachycardia must have been due to AV nodal reentry.

After termination of the tachycardia, the last three paced ventricular complexes resulted in conduction to the atrium and initiation of the tachycardia, but this should not obscure the important observation that the tachycardia was interrupted by ventricular pacing. Ventricular pacing presumably resulted in penetration of the AV node and some portion of the AV nodal reentry circuit, which interfered with the continuation of reentry and caused an abrupt loss of the atrial electrogram. In contrast, if the tachycardia had been atrial, its termination by ventricular pacing would have had to have been preceded by premature depolarization of the atrium.

Note that in Figure 2, a His-bundle depolarization is not present in front of every ventricular electrogram, probably because of instability of the His-bundle catheter. Despite the absence of a His-bundle depolarization in front of every ventricular complex, it is likely that the Wenckebach block was occurring above the level of the His bundle. Accordingly, the mild degree of aberration in the QRS complexes that follows the short RR intervals is explained by a "long-short" phenomenon in the His-Purkinje system.

Radiofrequency catheter ablation of the slow pathway was performed during sinus rhythm and was effective in eliminating the tachycardia.