ARRHYTHMIA OF THE MONTH

Section Editor: Fred Morady, M.D.

An Unusual Induction of Tachycardia: What Is the Tachycardia Mechanism?—V

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Case Presentation

A 48-year-old woman underwent an electrophysiologic procedure because of a 20-year history of paroxysmal supraventricular tachycardia. In the baseline state, the sinus cycle length was 720 msec, the atrial-His (AH) and His-ventricular (HV) intervals were 110 and 50 msec, respectively, and tachycardia was not inducible by atrial or ventricular pacing. During infusion of 1 μg/min of isoproterenol, the sinus cycle length was 520 msec, and the AH and HV intervals were 90 and 50 msec, respectively. There was decremental VA conduction, with a VA block cycle length of 300 msec. Programmed atrial stimulation with a single extrastimulus at coupling intervals of 220 to 250 msec reproducibly induced a narrow-QRS tachycardia, which had a cycle length of 330 to 340

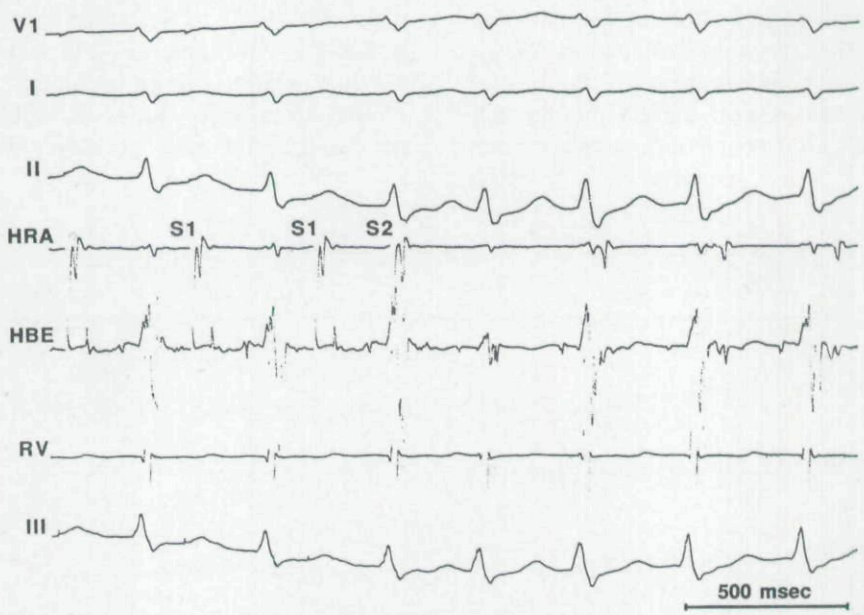

Figure 1. *Induction of a narrow-QRS tachycardia by programmed atrial stimulation with an S1S2 interval of 230 msec during infusion of isoproterenol. The tachycardia cycle length is 330 to 340 msec. HRA = high right atrial electrogram; HBE = His-bundle electrogram; S1 = atrial basic drive train stimulus; S2 = atrial extrastimulus.*

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msec. A typical induction of tachycardia is shown in Figure 1. The tachycardia could be entrained with rapid atrial pacing. A ventricular depolarization introduced when the His bundle was refractory did not preexcite the atrium. Ventricular pacing at a cycle length of 280 msec terminated the tachycardia without premature depolarization of the atria. How was the tachycardia induced? What is the tachycardia mechanism?

Commentary

Figure 1 demonstrates that a single atrial extrastimulus results in two ventricular depolarizations. This uncommon "double fire" or "two-for-one" phenomenon is most readily explained by conduction of a single atrial depolarization to the ventricle simultaneously through a fast and slow AV nodal pathway. An alternative explanation might be that the atrial extrastimulus initiates AV nodal reentrant tachycardia in which there is an initial, single instance of retrograde block to the atrium. This possibility is unlikely because the initiation of typical AV nodal reentrant tachycardia requires anterograde conduction through a slow AV nodal pathway, whereas the A2H2 interval in this case is only 100 msec, indicative of fast pathway conduction. Another possible explanation for the apparent "double fire" phenomenon is that the atrial extrastimulus conducts to the ventricle through only a single AV nodal pathway and also initiates a junctional tachycardia caused by either intra-Hisian reentry or triggered activity. However, intra-Hisian reentry is unlikely in the absence of a prolonged HV interval, and the fact that the tachycardia could be entrained makes triggered activity very unlikely.

The tachycardia is a narrow-QRS tachycardia with a concentric pattern of atrial activation and with a His-atrial interval that is 50 msec shorter than the AH interval. The first VA interval during the tachycardia is shorter than the subsequent VA intervals. The differential diagnosis includes atrial tachycardia, orthodromic tachycardia utilizing an accessory pathway for retrograde conduction to the atrium, and AV nodal reentrant tachycardia. Although it is likely that the tachycardia is initiated by a "double fire" through dual AV nodal pathways, this does not establish the mechanism as being AV nodal reentry. It is possible that the atrial extrastimulus resulted in anterograde conduction through a fast and slow AV nodal pathway, followed by an echo due to retrograde conduction through the fast pathway, and that this echo initiated either an atrial or orthodromic tachycardia.

There is, in fact, no way to exclude the possibility of atrial or orthodromic tachycardia based only on the information provided in Figure 1. However, other findings described above rule out these possibilities. Termination of the tachycardia by ventricular pacing without premature depolarization of the atrium rules out an atrial tachycardia, and the decremental and concentric pattern of VA con-

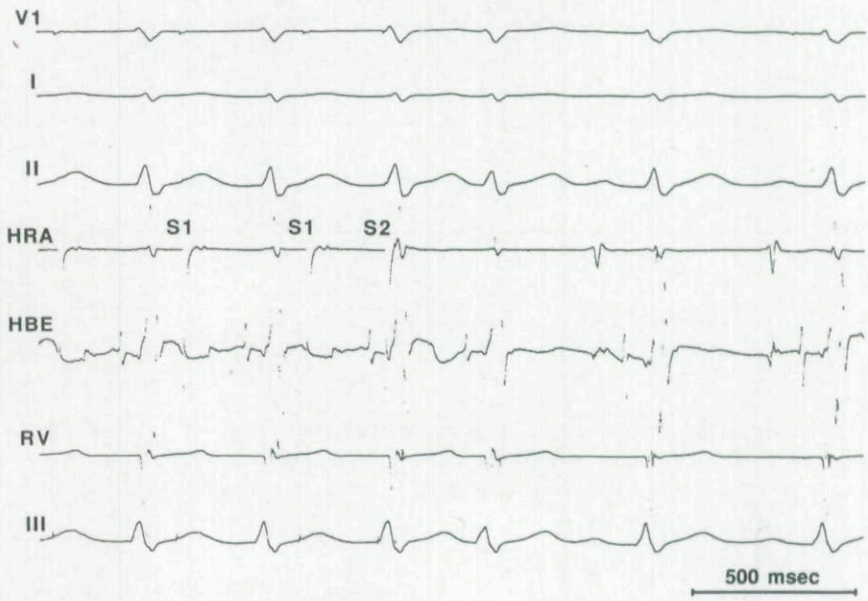

Figure 2. *Programmed atrial stimulation with an S1S2 interval of 230 msec during infusion of isoproterenol, after radiofrequency ablation of the slow pathway. Tachycardia was no longer inducible.*

duction along with the inability to preexcite the atrium during tachycardia when the His bundle was refractory make orthodromic tachycardia unlikely. By exclusion, AV nodal reentrant tachycardia is the most likely mechanism of this tachycardia. Slow pathway ablation was performed by delivering radiofrequency energy in the posteroseptal right atrium, near the ostium of the coronary sinus, and this eliminated both the "double fire" phenomenon and the tachycardia (Fig. 2).

The occurrence of two different VA intervals during the tachycardia raises the question of whether there was more than one pathway available for retrograde conduction. This possibility cannot be ruled out, but the findings can be explained by a single retrograde pathway in which there is a rate-related conduction delay once the tachycardia is established.

Also noteworthy is that the AH interval of 200 msec during the tachycardia is very different than the AH intervals of 130 and 440 msec during the "double fire," raising the possibility of three different pathways capable of anterograde conduction. Again, this possibility cannot be ruled out, but the anterograde pathway during tachycardia could be the same slow pathway used in the "double fire," with the longer AH interval being explained by the A1A2 interval being 100 msec shorter than the tachycardia cycle length or by an electrotonic interaction between the fast and slow pathways. The fact that all evidence of slow pathway function was eliminated by radiofrequency energy application at a single site may make it more likely that there was only one slow pathway.