ARRHYTHMIA OF THE MONTH

Section Editor: Fred Morady, M.D.

What Is the Tachycardia Mechanism?—III

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Case Presentation

A 66-year-old woman with a 24-year history of paroxysmal supraventricular tachycardia underwent an electrophysiologic procedure. The baseline sinus cycle length, atrial-His (AH), and His-ventricular (HV) intervals were 800, 70, and 40 msec, respectively.

Programmed stimulation with a single atrial extrastimulus did not demonstrate dual AV nodal physiology and did not result in the induction of tachycardia. Supraventricular tachycardia with a cycle length of 350 msec was induced by rapid atrial pacing (Fig. 1). A single ventricular extrastimulus introduced during tachycardia coincident with His-bundle refractoriness did not preexcite the atrium. Double ventricular extrastimuli introduced during the tachycardia resulted in advancement of the atrial electrograms. At times, the tachycardia was terminated (Fig. 2); at other times, it was not (Fig. 3). Termination of the tachycardia always was associated with shortening of the AH interval after the double ventricular extrastimuli. What is the mechanism of this tachycardia?

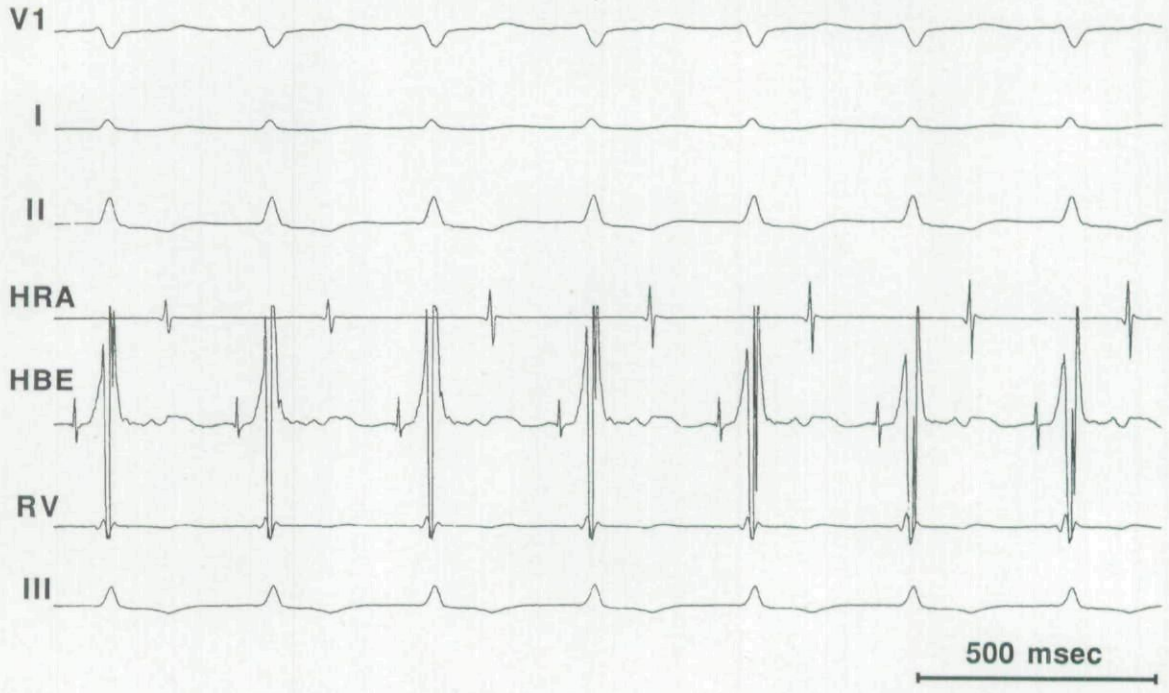

Figure 1. *Supraventricular tachycardia, cycle length 350 msec, which was induced by rapid atrial pacing. From top to bottom are leads V1, I, and II, the high right atrial electrogram (HRA), the His-bundle electrogram (HBE), a right ventricular electrogram (RV), and lead III.*

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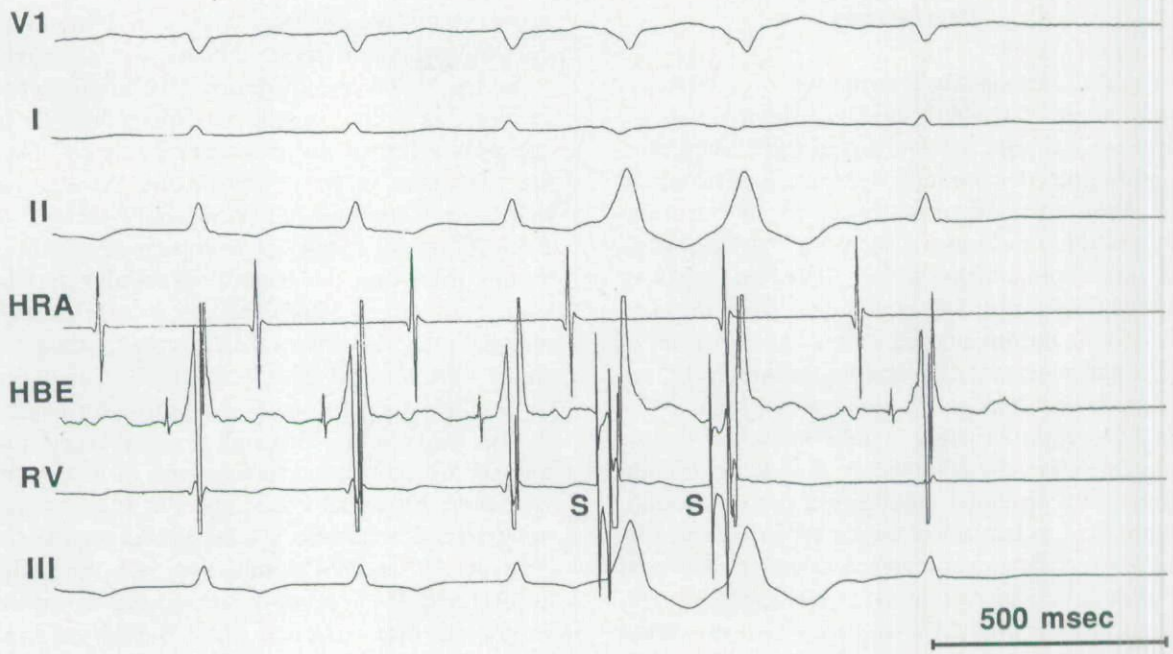

Figure 2. *Termination of the tachycardia after the introduction of two extrastimuli (S) in the right ventricle. Note that there is shortening of the atrial-His (AH) interval after the second ventricular extrastimulus. The association between shortening of the AH interval and termination of the tachycardia was reproducible. Abbreviations as in Figure 1.*

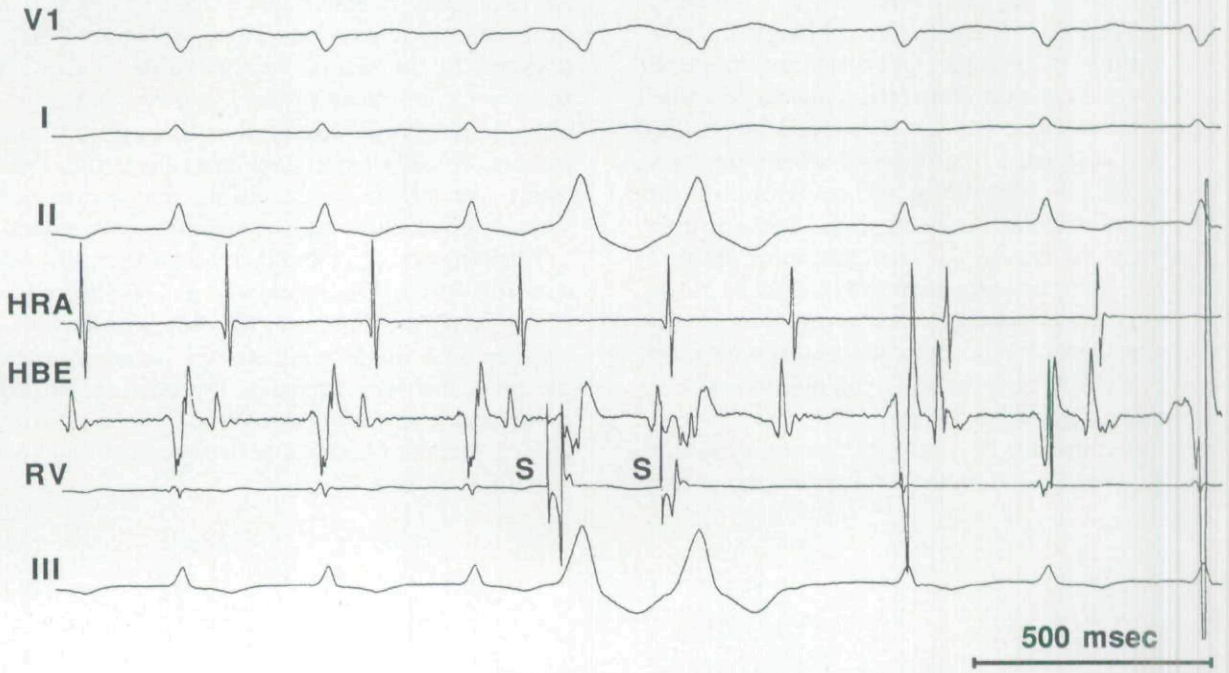

Figure 3. *Continuation of the tachycardia after the introduction of two extrastimuli in the right ventricle. Because of displacement of the His-bundle catheter, a His-bundle depolarization is absent or very small. Note that the AV interval of the complex following the second ventricular depolarization is prolonged, without prolongation of the HV interval. Abbreviations as in Figure 1.*

Commentary

Figure 1 demonstrates a supraventricular tachycardia in which the AH and His-atrial intervals are almost equal, and the low septal right atrial electrogram precedes the high right atrial electrogram. The differential diagnosis based on the information provided in Figure 1 includes atrial tachycardia, orthodromic tachycardia utilizing an accessory pathway for retrograde conduction from the ventricle to the atrium, and the "slow-slow" variant of AV nodal reentrant tachycardia, in which the anterograde and retrograde limbs of the reentry circuit have approximately equal conduction times.

In this case, the response to double ventricular extrastimuli provided information useful in establishing the mechanism of the tachycardia. The second ventricular depolarization advances the next atrial depolarization, indicating that there was retrograde conduction to the atrium. This observation by itself is not helpful in determining the tachycardia mechanism, because analysis of Figure 2 demonstrates that the second ventricular depolarization occurs approximately 160 msec earlier than the next anticipated His-bundle depolarization and, therefore, could have resulted in retrograde conduction to the atrium regardless of the tachycardia mechanism. However, there are two other observations that are helpful in ruling out an atrial tachycardia. The first is that the morphology and relative timing of the high right atrial and low right atrial septal electrograms remain unchanged when the atrial electrograms are advanced, suggesting that the atria are being depolarized via the same retrograde pathway during the tachycardia as after the ventricular depolarization. Second, there would be no reason for termination of an atrial tachycardia to be associated with events occurring in the AV node; the fact that termination of the tachycardia was consistently associated with shortening of the AH interval is strong evidence against an atrial tachycardia.

If a ventricular depolarization introduced during His-bundle refractoriness preexcites the atrium, this is definitive evidence that an extranodal accessory pathway is present; however, the inability to preexcite the atrium during His-bundle refractoriness, as in the present case, does not rule out the possibility of an accessory pathway. There are two other observations in this case that are helpful in distinguishing orthodromic tachycardia from AV nodal reentry. A comparison of AH intervals following the second ventricular depolarization in Figures 2 and 3 reveals paradoxic shortening of the AH interval in Figure 2, despite a shorter VA interval than in Figure 3. This would be unlikely to occur in a straightforward orthodromic tachycardia, although it might be compatible with orthodromic tachycardia in association with dual AV nodal pathways. But Figure 3 also demonstrates that there is a reciprocal relationship between AV and VA conduction; note that when the AH and AV intervals increase after the second ventricular depolarization, the VA interval shortens. Although accessory pathways at times may have decremental conduction properties, this finding is much more common when the anterograde and retrograde limbs of the reentry circuit both involve the AV node.

Based on the information provided in Figures 2 and 3, the most likely diagnosis is the "slow-slow" form of AV nodal reentry. The events in Figure 2 are explained by anterograde block in one of the slow pathways after advancement of the atrial electrograms by the second ventricular depolarization and conduction down a "fast" pathway. In this patient, anterograde conduction through the fast pathway reliably terminated the tachycardia, indicating that, for whatever reason, anterograde fast pathway conduction was incompatible with reentry.

Mapping demonstrated that the earliest atrial activation during tachycardia was in the posteroseptal right atrium, near the ostium of the coronary sinus. A single application of radiofrequency energy at this site eliminated the tachycardia and all evidence of slow pathway conduction, suggesting that the two slow pathways were in close proximity to each other.