ARRHYTHMIA OF THE MONTH

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Paroxysmal Supraventricular Tachycardia in a Patient with Ventriculoatrial Dissociation in the Baseline State: What is the Tachycardia Mechanism?

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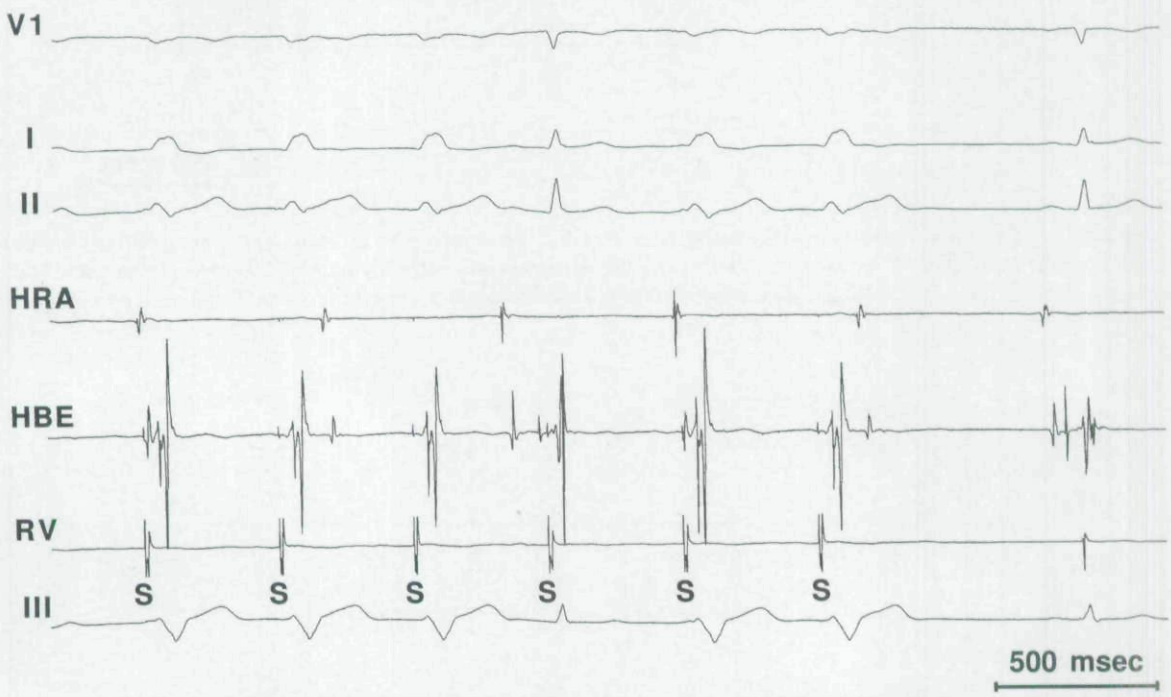

Figure 1. *Right ventricular overdrive pacing atrial tachycardia, cycle length 600 msec, in the baseline state. There is no ventriculoatrial conduction. Shown are leads V1, I, and II, high right atrial electrogram (HRA), His-bundle electrogram (HBE), right ventricular electrogram (RV), and lead III. S = pacing stimulus.*

Case Presentation

A 27-year-old man underwent an electrophysiologic procedure because of a 5-year history of recurrent episodes of paroxysmal supraventricular tachycardia. The baseline sinus cycle length, atrial-His (AH), and His-ventricular (HV) intervals were 810, 70, and 40 msec, respectively. There was no demonstrable ventriculoatrial conduction (Fig. 1), and tachycardia was not inducible with atrial or ventricular pacing. During infusion of 2 μg/min isoproterenol, ventricular pacing at a cycle length of 320 msec resulted in 1:1 ventriculoatrial conduction and the induction of a narrow QRS tachycardia with a cycle length of 290 msec, AH in-

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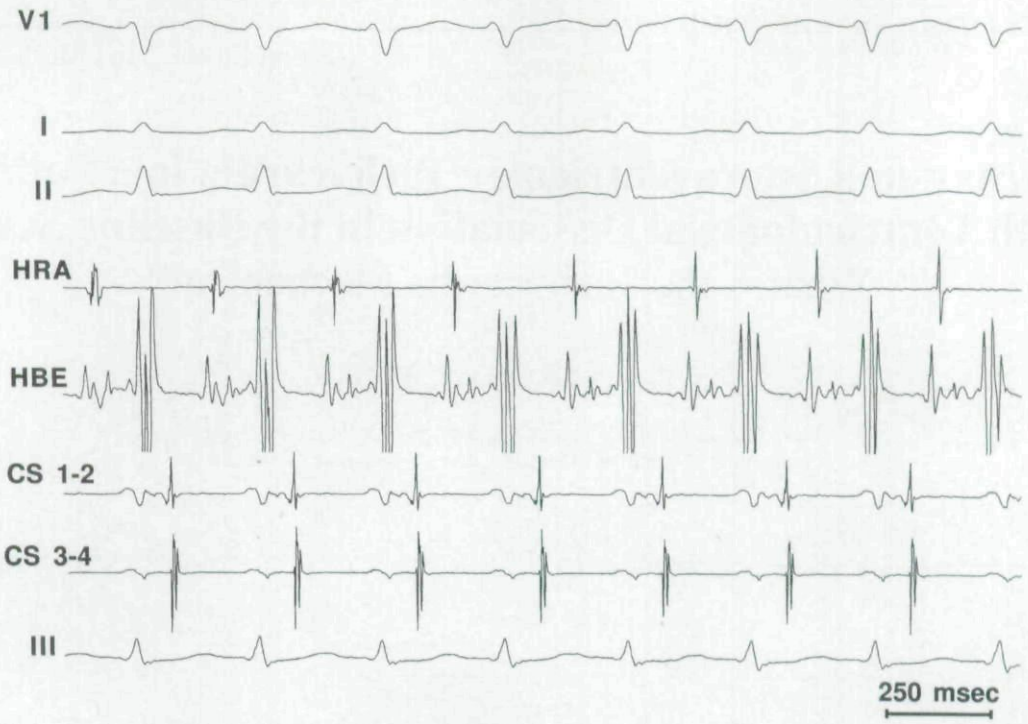

Figure 2. *Paroxysmal supraventricular tachycardia, cycle length 290 msec, induced by ventricular pacing during the infusion of isoproterenol. There is eccentric atrial activation, and the shortest ventriculoatrial interval (110 msec) is recorded with the distal pair of electrodes of the coronary sinus catheter (CS 1–2) positioned in the region of the posterolateral mitral annulus.*

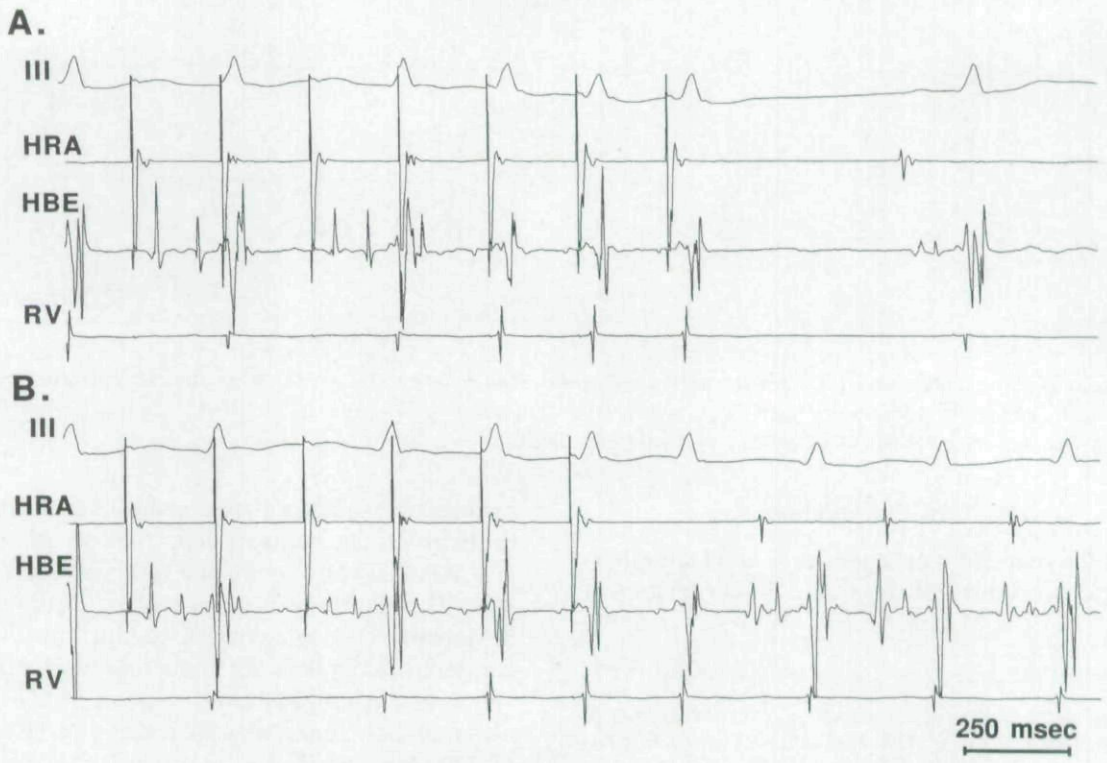

Figure 3. *Atrial overdrive pacing at a cycle length of 210 msec during tachycardia. (A) An example of termination of tachycardia by pacing. (B) An example of resumption of tachycardia after cessation of pacing.*

terval of 60 msec, and His-atrial (HA) interval of 230 msec. There was eccentric atrial activation during the tachycardia, with the shortest ventriculoatrial interval recorded in the coronary sinus, adjacent to the posterolateral mitral annulus (Fig. 2). A ventricular depolarization introduced during tachycardia and coincident with His-bundle refractoriness had no effect on the atrial cycle length. Atrial pacing at a cycle length of 210 msec sometimes terminated the tachycardia (Fig. 3). What is the tachycardia mechanism?

Commentary

During the tachycardia, the AH interval is much longer than the HA interval, consistent with atypical (fast-slow) AV nodal reentrant tachycardia, atrial tachycardia, or orthodromic reciprocating tachycardia. The fact that earliest retrograde atrial activation during the tachycardia was recorded adjacent to the posterolateral mitral annulus makes the first possibility very unlikely, since earliest atrial activation during atypical AV nodal reentrant tachycardia is expected to occur in the region of the coronary sinus ostium. The differential diagnosis therefore is quickly narrowed down to a left atrial tachycardia and orthodromic reciprocating tachycardia utilizing a concealed left posterolateral accessory pathway.

The presence of ventriculoatrial dissociation before the infusion of isoproterenol might be considered to favor a left atrial tachycardia, since it is unusual for concealed accessory pathways to be completely dormant in the baseline state. Furthermore, the inability to advance the atrial electrogram during tachycardia with a ventricular depolarization that is coincident with His-bundle refractoriness is consistent with an atrial tachycardia; however, it should be kept in mind that it is not unusual for a ventricular depolarization introduced in the right ventricle when the His bundle is refractory during orthodromic reciprocating tachycardia utilizing a left-sided accessory pathway not to be able to advance the atrial electrogram.

The most definitive finding in this case is provided by the response to atrial pacing at a cycle length of 220 msec during the tachycardia. There was a consistent relationship between the occurrence of AV block following the last stimulus of the drive train and the termination of tachycardia. Because there would be no reason for termination of an atrial tachycardia to be linked to the occurrence of AV block, it is clear that this is an orthodromic reciprocating tachycardia. Other findings that also could have distinguished orthodromic reciprocating tachycardia from an atrial tachycardia include the presence of a different atrial activation sequence during ventricular pacing than during tachycardia, and the ability to dissociate the ventricular electrograms from the tachycardia by overdrive ventricular pacing (both of which would have favored an atrial tachycardia), and the ability to terminate the tachycardia with a ventricular depolarization coincident with His-bundle refractoriness (which would have ruled out an atrial tachycardia).

This case illustrates a common situation in the differential diagnosis of tachycardia mechanism. Not infrequently, some findings may favor a particular mechanism, while other findings may favor another mechanism. The correct diagnosis depends on the ability to identify which lines of evidence are the most definitive. In the present case, the association between termination of tachycardia and AV block was more compelling than the presence of ventriculoatrial dissociation in the baseline state or the inability to advance the atrial electrogram during tachycardia with a ventricular depolarization.