A 23-year-old woman underwent electrophysiological study because of a 9-year history of frequent episodes of supraventricular tachycardia. The patient had a normal baseline ECG and no structural heart disease. The baseline AH and HV intervals were 70 and 40 msec, respectively. Incremental atrial pacing and ventricular pacing reproducibly induced a narrow QRS complex tachycardia (cycle length 344 ms) with negative P-wave polarity in inferior leads and positive in V1 lead. During the tachycardia the earliest atrial activation was recorded at the ostium of the coronary sinus, where the AH and HA intervals were 95 and 237 ms, respectively (Fig. 1). What is the mechanism of this tachycardia?

The differential diagnosis of this tachycardia with an HA interval greater than the AH interval and an AH interval less than 200 ms includes an atrial tachycardia (AT), atypical fast-slow atrioventricular nodal reentrant tachycardia (AVNRT), and orthodromic reciprocating tachycardia using a slowly conducting accessory pathway (AVRT). The occurrence of AV block during the tachycardia ruled out the AVRT narrowing the differential diagnosis to AVNRT and AT. Following termination of the overdrive ventricular pacing during the tachycardia, the tachycardia returns with a V-A-V pattern (Fig. 2). This essentially rules out atrial tachycardia which typically demonstrates an A-A-V pattern upon discontinuation of ventricular pacing. Furthermore, the difference between the postpacing interval and tachycardia cycle length > 115 ms as well the difference between the stimulus-A and VA intervals > 85 ms favoured the diagnosis of AVNRT. During pacing from the base of the right ventricle with intermittent ventricular and His bundle capture, alternated with atrial capture, the tachycardia reinitiated with 2:1 AV block at the level of AV node (Fig. 3). This finding excludes the AVRT tachycardia and also demonstrates that the infranodal structures are not required for maintenance of the tachycardia.

AVNRT with eccentric retrograde activation sequences in the coronary sinus electrodes is well described and anatomic slow pathway ablation in the right atrium near the coronary sinus ostium has been reported to successfully eliminate the tachycardia (3). In the present case, ablation of the slow pathway was performed where junctional rhythm was noted. Atrial and ventricular pacing with up to three atrial premature beats with and without isoprenaline (10 μg/min) did not induce the tachycardia.
REFERENCE

