Wenckebach During Supraventricular Tachycardia

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In case 1, a 71-year-old man with a history of coronary artery disease presented with recurrent paroxysmal supraventricular tachycardia (SVT) despite treatment with β-blockers. During electrophysiology study, the baseline sinus cycle length was 895 ms, atrial-His (AH) interval was 120 ms, and His-ventricular interval was 60 ms.

A narrow complex SVT with tachycardia cycle length of 480 ms was initiated with atrial overdrive pacing at 400 ms, as well as with single atrial extrastimuli. The tachycardia showed a long RP interval with regular concentric atrial activation pattern and atrioventricular Wenckebach conduction (Figure 1). Ventricular overdrive pacing during tachycardia repeatedly terminated the tachycardia without activation of the atrium (Figure 2).

In case 2, a 32-year-old woman with a history of recurrent, debilitating palpitations for more than 5 years, despite medical therapy, was referred after 4 electrophysiology studies and unsuccessful ablations.

At electrophysiology study, multipolar catheters were placed in the right atrium, His-bundle region, right ventricle, and coronary sinus. At baseline, the patient was found to be in normal sinus rhythm, without evidence of preexcitation, and she had normal AH and His-ventricular intervals. During catheter placement, an irregular narrow-complex SVT occurred spontaneously (Figure 3). Attempts to entrain the short RP tachycardia with ventricular overdrive pacing resulted in ventriculoatrial dissociation. Multiple late atrial premature beats (APBs) were introduced during tachycardia and did not influence the tachycardia cycle length (Figure 4). In addition, the AH interval during atrial pacing in sinus rhythm, at the tachycardia cycle length, was 40 ms greater than the AH interval during tachycardia.

Figure 1. Inducible SVT with Wenckeback block above the His (arrows) in patient 1 (ECG leads I, III, V1, and V6). His d indicates distal His; His p, proximal His; CS 1,2, distal coronary sinus; CS 7,8, proximal coronary sinus; and RVa, right ventricular apex.
Commentary

SVT with Wenckebach essentially ruled out an atrioventricular reentrant tachycardia in both cases. The differential diagnosis included atrioventricular nodal tachycardia (AVNRT) with lower common pathway Wenckebach, atrial tachycardia (AT), or junctional tachycardia (JT). In case 1, termination of tachycardia by ventricular pacing without exciting the atrium excluded AT. For case 2, Figure 4 demonstrates a late atrial premature beat that preexcited the atrial electrogram (A) but had no effect on the His-His (H-H) interval; hence, the A-A interval did not predict the subsequent H-H interval, which excluded AT. Furthermore, when the AH interval during atrial pacing in sinus rhythm was 40 ms greater than the AH interval during tachycardia, this favored AVNRT over AT. Dual atrioventricular node physiology was demonstrated in both cases. In addition, to differentiate AVNRT from JT, a late APB was inserted during tachycardia when the septal atrial electrogram was committed; this late APB terminated tachycardia, which is diagnostic for AVNRT and ruled out JT. AVNRT with lower common pathway Wenckebach is uncommon but can occur in the elderly and in patients who have had previous ablations.

Once the diagnosis was made, ablation was performed in the usual slow pathway region at the inferior triangle of Koch and elicited junctional automaticity in both cases. However, tachycardia was still inducible in both cases. By introducing APBs that reset the tachycardia, we targeted possible leftward extensions of the slow pathway. In case 1, successful ablation was performed at the roof of the mid coronary sinus. In case 2, ablation in the coronary sinus was unsuccessful; hence, we proceeded to the left inferoseptum along the mitral annulus (accessed via the patent foramen ovale). Ablation at this site (Figure 5) elicited junctional automaticity and rendered the tachycardia noninducible. Both patients have remained free of tachycardia. Although rare, unsuccessful ablation in the slow pathway region for AVNRT with lower common pathway block should prompt consideration of left-sided extensions, which have been shown to be integral in certain cases of AVNRT. An association between AVNRT with lower common pathway block and left-sided extensions has been previously noted and is further substantiated by these case reports, but the mechanism for this is unknown. Interestingly, these previously reported cases of AVNRT and left-sided...
extension all had eccentric coronary sinus activation, whereas our 2 cases had concentric coronary sinus activation.

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References


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