

Termination of a Narrow QRS-Complex Tachycardia: What is the Mechanism?

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Case Presentation

A 27-year-old man with a 10-year history of recurrent episodes of paroxysmal supraventricular tachycardia underwent an electrophysiologic procedure. The baseline sinus cycle length was 860 msec, and the atrial-His (AH) and His-ventricular (HV) intervals were 80 and 45 msec, respectively. A narrow QRS-complex tachycardia was induced by atrial overdrive pacing. The cycle length of the tachycardia varied between 280 and 350 msec. A single ventricular depolar-

ization introduced at the right ventricular apex during the tachycardia simultaneous with the His-bundle depolarization terminated the tachycardia (Fig. 1). This response was reproducible. What is the mechanism of the tachycardia, and what is the mechanism of termination?

Commentary

The cycle length of the tachycardia fluctuated between 280 and 305 msec, but the atrial cycle length enveloping the premature ventricular depolarization was 255 msec, indicating that the atrial electrograms were advanced by the ventricular depolarization (Fig. 2). Because the His bundle was refractory at the time the ventricular depolarization was introduced, this response demonstrates the presence of an extranodal pathway between the ventricular and atrium. The presence of an accessory pathway by itself does not prove that the tachycardia was orthodromic reciprocating tachycardia. However, the fact that the atrial activation sequence

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Figure 1. Introduction of a ventricular depolarization during a narrow QRS-complex tachycardia. HBE_d, HBE_m, HBE_p = distal, medial, and proximal His-bundle electrograms, respectively; HRA = high right atrium; RVA = right ventricular apex; S = stimulus; Stim = stimulus channel.



Figure 2. Same recording as in Figure 1, with atrial cycle lengths indicated in milliseconds.

was unaffected by the premature ventricular depolarization makes orthodromic reciprocating tachycardia far more likely than AV nodal reentrant tachycardia or atrial tachycardia in combination with a bystander accessory pathway.

When a ventricular premature depolarization simultaneous with the His-bundle depolarization terminates orthodromic reciprocating tachycardia, this usually occurs immediately after the premature depolarization. In the present case, termination was delayed and occurred one cycle later, with block between the ventricle and atrium. This type of response to the premature depolarization was reproducible, ruling out a random event.

A basic electrophysiologic property of accessory pathways may provide the best explanation for why this tachycardia terminated. As in the His-Purkinje system and in ventricular muscle, the refractory period of an accessory pathway varies in direct fashion with the duration of the prior cycle length. Figure 2 demonstrates that advancement of the atrial electrogram by the premature ventricular depolarization resulted in prolongation of the next AH interval, attributable to the AV node being in its relative refractory period. Because the AH interval prolonged to a greater degree than the atrial electrograms were advanced, the next atrial cycle length was lengthened to 380 msec. This set up a “long-short” sequence in the accessory pathway, such that the next retrograde wavefront found the accessory pathway refractory (Fig. 3).

As is often the case in clinical electrophysiology, this proposed mechanism of termination is presumptive and cannot

be proven. Why then is this the most likely explanation for termination of the tachycardia? Because it makes sense physiologically, and because there is no other mechanism that explains the events as well.

Mapping demonstrated the presence of a posteroseptal accessory pathway that was successfully ablated. Tachycardia no longer was inducible after catheter ablation of the accessory pathway.

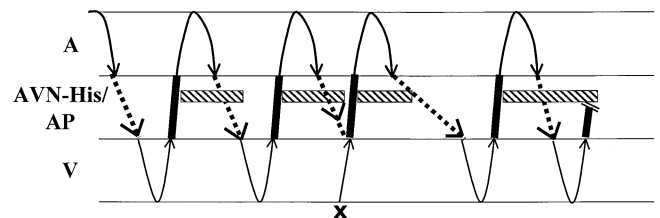


Figure 3. Schematic diagram of the orthodromic reciprocating tachycardia circuit, with conduction from the atrium (A) to ventricle (V) over the AV node/His-Purkinje system (AVN-His, dashed arrows) and from the ventricle back to the atrium over an accessory pathway (AP, solid bars). The striped bars signify the accessory pathway effective refractory period, and the “x” signifies a premature ventricular depolarization introduced during His-bundle refractoriness. Note that the accessory pathway effective refractory period is proportional to the prior cycle length. The tachycardia terminated because a relatively long cycle length after the ventricular premature depolarization resulted in lengthening of the accessory pathway effective refractory period one cycle length later.