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A V-A-V response during induction of supraventricular tachycardia: What is the mechanism?

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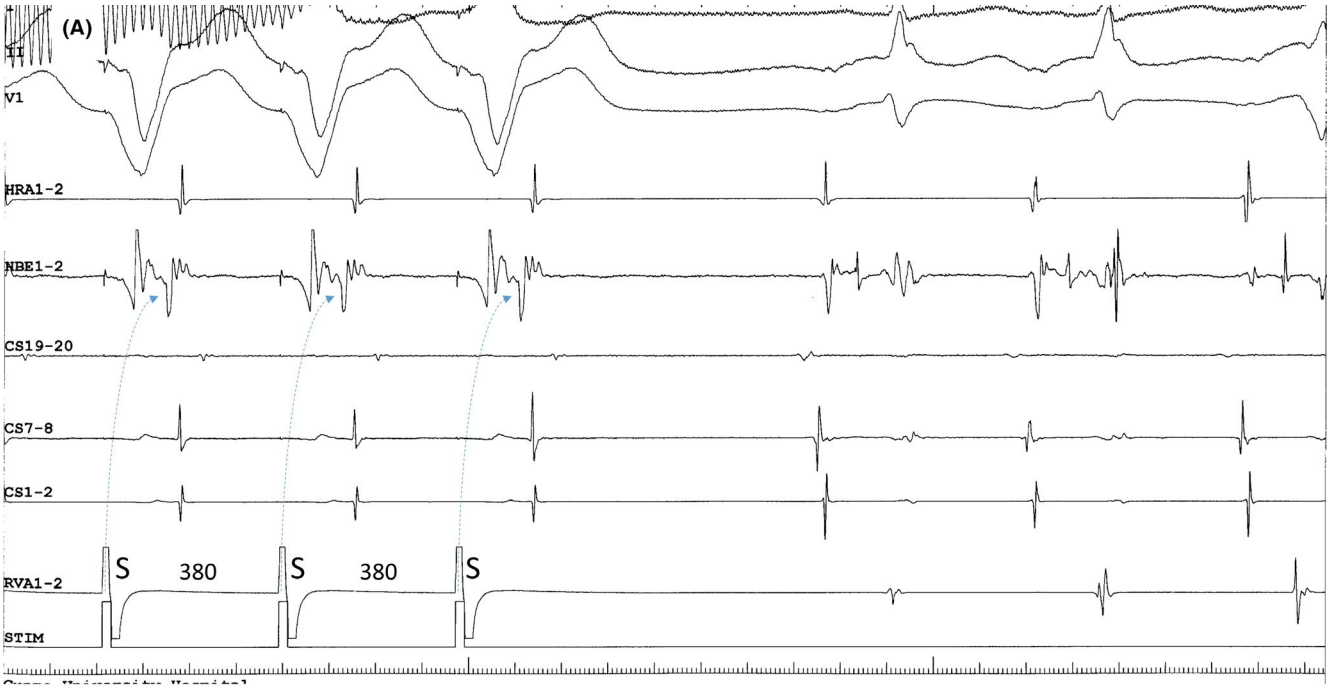
1 | CASE PRESENTATION

A 53-year-old man with a history of multiple episodes of paroxysmal supraventricular tachycardia underwent electrophysiological studies and a catheter ablation procedure. The 12 lead electrocardiogram during tachycardia showed a long RP tachycardia and negative P waves in inferior leads. At baseline, ventriculoatrial (VA) conduction was observed, with the earliest atrial activation site in the His bundle (HB) region, consistent with retrograde conduction via a fast pathway (FP) of the atrioventricular (AV) node. The tachycardia with an initial V-A-A-V activation sequence was reproducibly induced by right ventricular (RV) burst pacing (Figure 1A). During tachycardia, the earliest atrial activation was recorded at the posterolateral right atrium (RA) along the tricuspid annulus (Figure 1C,D). A 6 mg bolus injection of adenosine triphosphate (ATP) reproducibly terminated the tachycardia. Interestingly, a V-A-V activation sequence was reproducibly observed during the induction of the tachycardia with RV burst pacing (Figure 1B). What is the mechanism of the tachycardia, and the V-A-A-V and V-A-V activation sequences?

2 | COMMENTARY

The differential diagnosis of long RP tachycardia with the earliest site of atrial activation at the posterolateral RA and ATP sensitivity includes ATP-sensitive atrial tachycardia (AT) originating from the tricuspid annulus (TA),[1] fast-slow AV nodal reentrant tachycardia (AVNRT) using a slow pathway extending posterolateral RA (SP),[2] and AV reentrant tachycardia using a slowly conducting accessory pathway (AP). We meticulously analyzed the intracardiac electrograms immediately after ventricular entrainment of the tachycardia and observed a transient intraatrial fusion of a progressively centrifugal propagation in the atria resulting from retrograde conduction over the FP, with the atrial activation preserving the tachycardia cycles, compatible with a cyclic activation originating from the atrial focus rather than retrograde activation over SP or AP during overdrive pacing (Figure 2). This phenomenon excludes the diagnosis of AVNRT and AVRT and confirms the diagnosis of AT. The phenomenon may be observed when the origin of AT is farther away from the retrograde breakthrough of the FP. Successful ablation was attained at the earliest site of atrial activation.

In the present case, the mechanism responsible for the V-A-A-V activation sequence was determined to result from an initiation of AT triggered by the retrograde atrial activation after the last ventricular stimulus (Figure 1A). The feasibility of induction and termination of tachycardia with pacing suggested reentry as the mechanism. Based on the principle of reentry, we assumed that during ventricular overdrive pacing, the orthodromic wave-front provoked by the $(N\text{-}1)^{th}$ stimulus collides with the antidromic wave-front provoked by the N^{th} stimulus within the reentry circuit of the tachycardia. However, during ventricular burst pacing followed by a V-A-V response, because retrograde atrial activation after the last ventricular stimulus was absent owing to a frequency-dependent block at the FP, the orthodromic wave-front provoked by the next to last ventricular stimulus broke through the exit of the reentry circuit because of a lack of an antidromic wave-front into the circuit. Consequently, AT was initiated after


(A)
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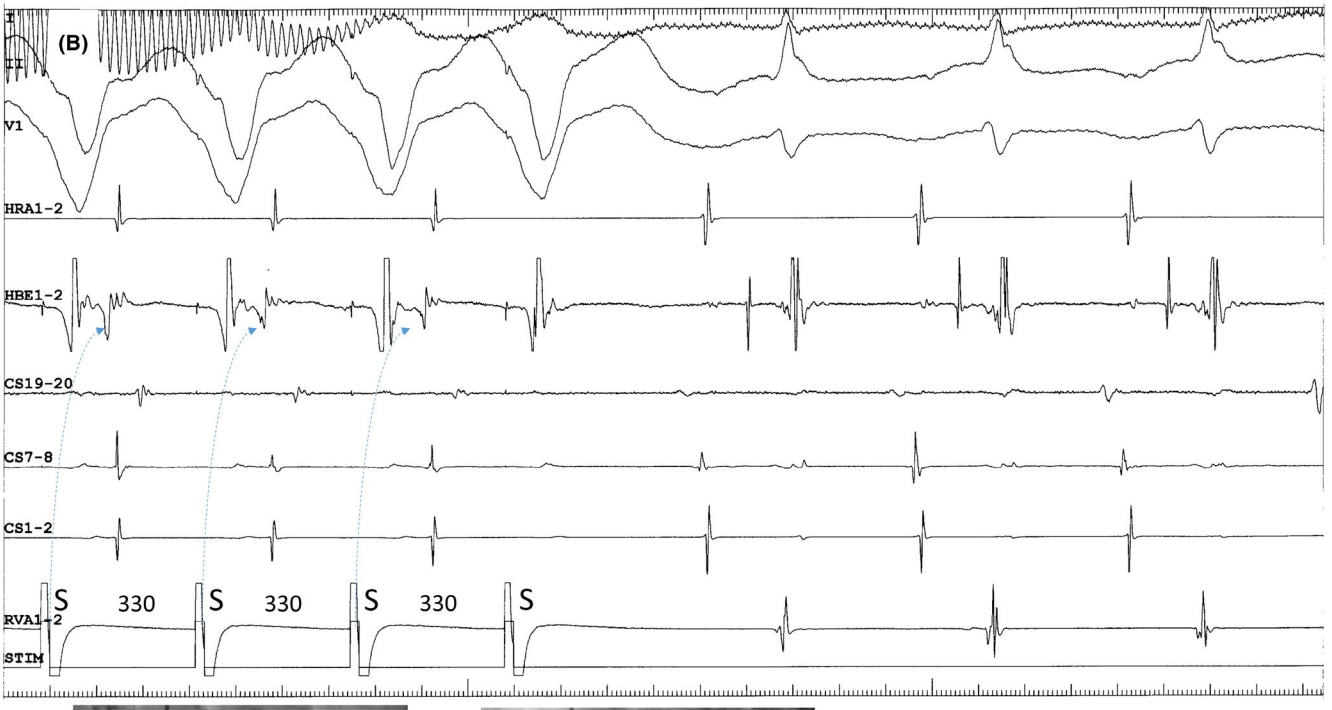
(B)
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CS7-8
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RVA1-2
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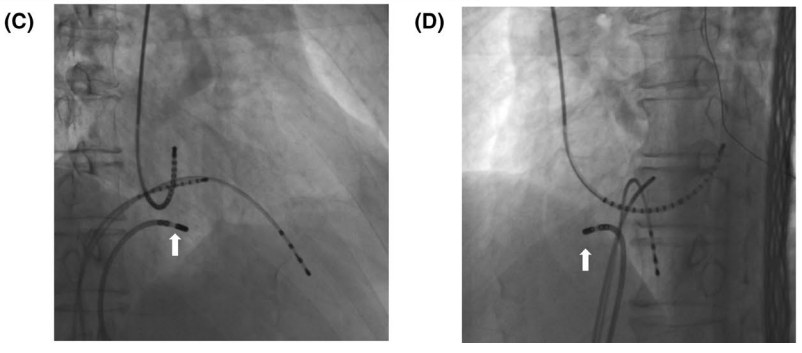
(C)
(D)

FIGURE 1 Induction of tachycardia with an initial V-A-A-V activation sequence with RV apical (RVA) overdrive pacing at an S-S cycle length of 440 ms, and with an initial V-A-V activation sequence with RVA overdrive pacing at an S-S cycle length of 330 ms (B), and fluoroscopic views of the position of the catheters in the RAO and LAO projections (C and D), respectively, showing the site of successful ablation (white arrows). The tachycardia was initiated after retrograde conduction over the fast pathway (dot arrows) in A, but was initiated after the last stimulus was blocked at the fast pathway in B. The earliest atrial activation during tachycardia is recorded as far-field signals at CS19-20 adjacent to the posterolateral RA. HRA = distal high right atrium; HBE1-2 and 3-4 = distal to the proximal HB region; CS19-20 to 1-2 = proximal to the distal CS recording

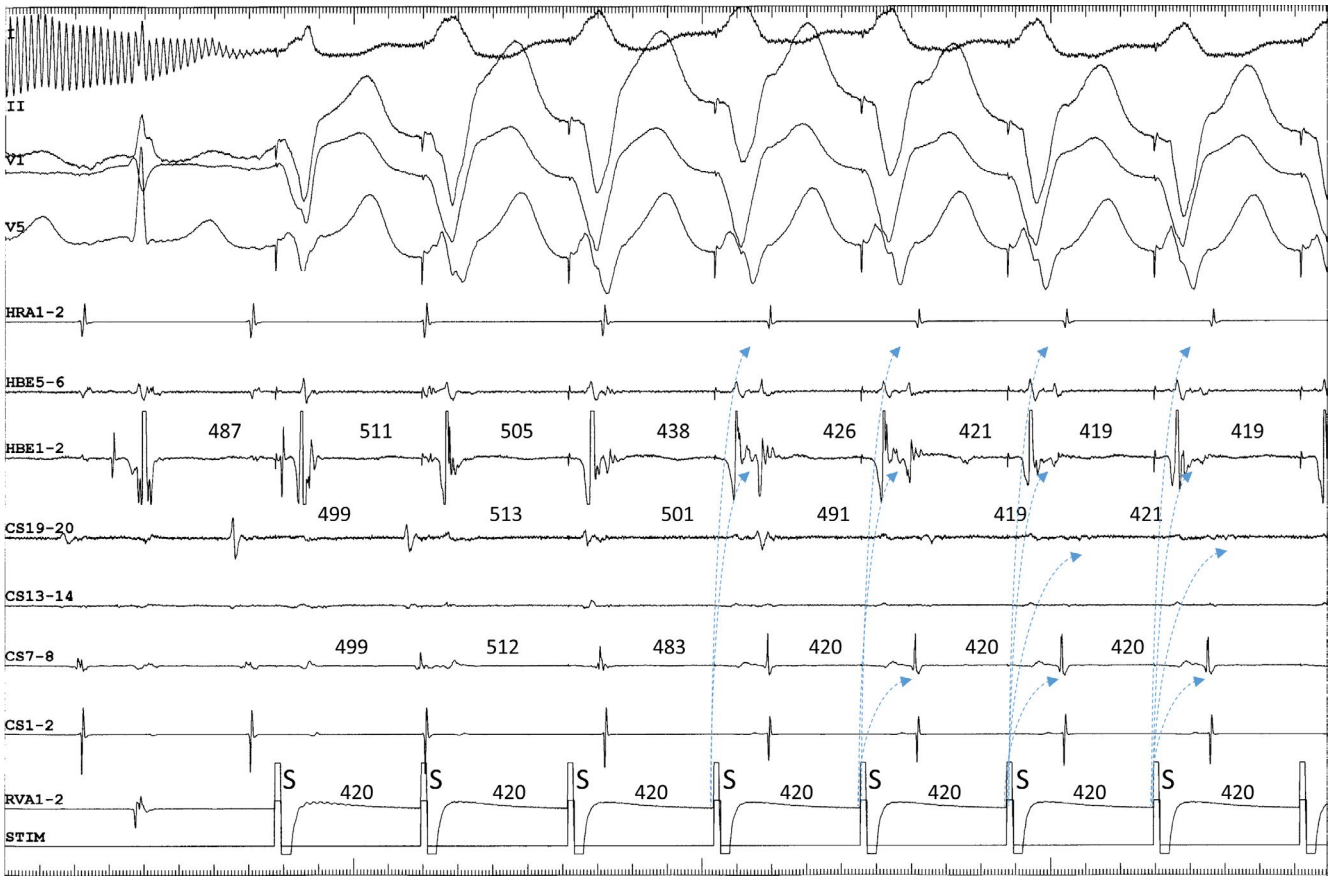

FIGURE 2 An intra-atrial fusion during RV overdrive pacing of the tachycardia at an S-S CL of 420 ms. The first, second, and third stimuli do not perturb the tachycardia cycle at all of the recording sites. However, the fourth stimulus advances the atrial deflection at HBE1-2 only and the fifth stimulus advances the atrial deflections at not only HBE but also HRA and CS7-8 and 1-2 (other than CS19-20) (dot arrows), dissociated with the atrial cycles of the tachycardia at the other recording sites. Finally, the sixth stimulus captures the atrial deflections at all of the recording sites. The numbers labeled on HBE1-2, CS19-20, and CS7-8 indicate the atrial CL in each recording site in ms. The other abbreviations are as in Figure 1

the last ventricular stimulus, representing the apparent (so-called pseudo) V-A-V activation sequence (Figure 1B). The V-A-V response to ventricular induction is a powerful indicator for excluding a diagnosis of AT, but this is based on the assumption that the first atrial activation of tachycardia during ventricular induction is truly attributable to a retrograde atrial activation by the last ventricular stimulus in contrast to ventricular entrainment capturing the atria during which V-A causal relationship is objectively obvious.[3] This case illustrates the importance of confirming this V-A causal relationship during ventricular induction for the diagnosis of supraventricular tachycardia. The pseudo V-A-V response may occur especially when the rate of ventricular burst pacing is rapid enough to provoke V-A conduction block.

CONFLICT OF INTEREST

Authors declare no conflict of interests for this article.

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