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SPOTLIGHT

Retrograde ventriculoatrial jump—What is the mechanism?

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CASE

During an EP study in a 35-year-old man having narrow QRS tachycardia with no preexcitation at baseline, a short VA tachycardia with concentric atrial activation was induced (Figure 1). Maneuvers during tachycardia like ventricular overdrive pacing (VOP), His synchronous PVC (HSP) could not be performed because of the ill-sustained nature of the SVT even on isoprenaline. Ventricular extra-stimuli (VES, S1–S2) protocol was performed starting @ 500/400 ms and coming down by 10 ms on S2. There was no decrement till 500/270 ms (Figure 2A). At VES @ 500/260 ms there was an abrupt VA jump as noted in Figure 2B. The mechanism of VA jump and probable SVT could be one of the following: (i) jump from FP (fast pathway) to SP (slow pathway)—hence likely AVNRT; (ii) jump from accessory pathway (AP) to AV node—hence likely orthodromic AVRT; or (iii) jump from AV node to AP—hence orthodromic AVRT.

Although the retrograde atrial activation pattern remained similar, the most prominent finding was the relation of His to atrial signal in the second beat (S2) in the upper panel (Figure 3A). At first glance, the last signal in HisD bipole after the second RV-paced beat (VES, 500/270 ms) could be thought of as 'A'-EGM (electrogram) unless the next drive train (shown in Figure 3B) is followed up. The same EGM remains constant on timing and vector directionality (in the next VES of 500/260 ms) proving it to be 'His' signal, followed by the atrial EGM. This proves that in the earlier VES (500/270 ms), the VA conduction was via both AV node and AP; where the retrograde H (via AV node) could appear after atrial EGM (via AP). The His signal appeared after the A-EGM, ruling out a nodal VA conduction and confirming the presence of an AP. The H signal was delayed due to retrograde RBBB. The atrium was already depolarized via AP and the VA traversing via LB-His-node got blocked with a H signal. In the lower panel (Figure 3B) with VES of 500/260 ms the retrograde AP conduction reached effective refractory period (ERP) and nodal VA conduction took place along with retrograde RBBB. Unless this is promptly recognized, an increment in VA time might masquerade as nodal conduction.[1] Among cases with non-sustained tachycardia, the retrograde route can also be delineated by the administration of intravenous adenosine during RV pacing. After confirmation of the diagnosis, the AP was successfully ablated just above the coronary sinus ostium near the right lower mid-septal region. The reproducibly inducible but non-sustained AVRT became non-inducible.

FUNDING INFORMATION

None.

CONFLICT OF INTEREST STATEMENT

Authors declare no conflict of interests for this article.

DATA AVAILABILITY STATEMENT

All raw data and recording during the case are available for review.

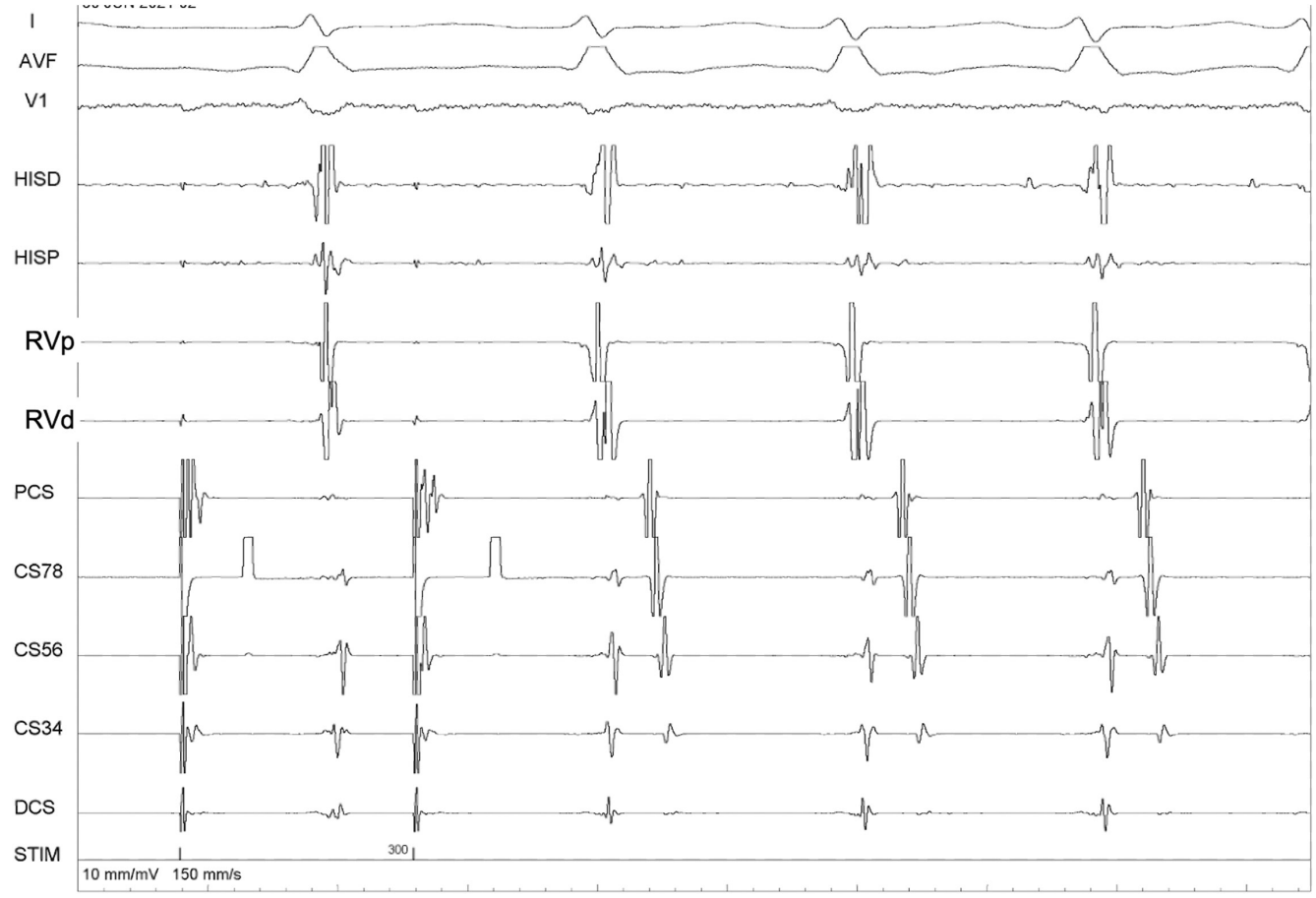

FIGURE 1 Induction of the short VA tachycardia with atrial extra-stimuli. The tachycardia was easily inducible but nonsustained even on isoprenaline.

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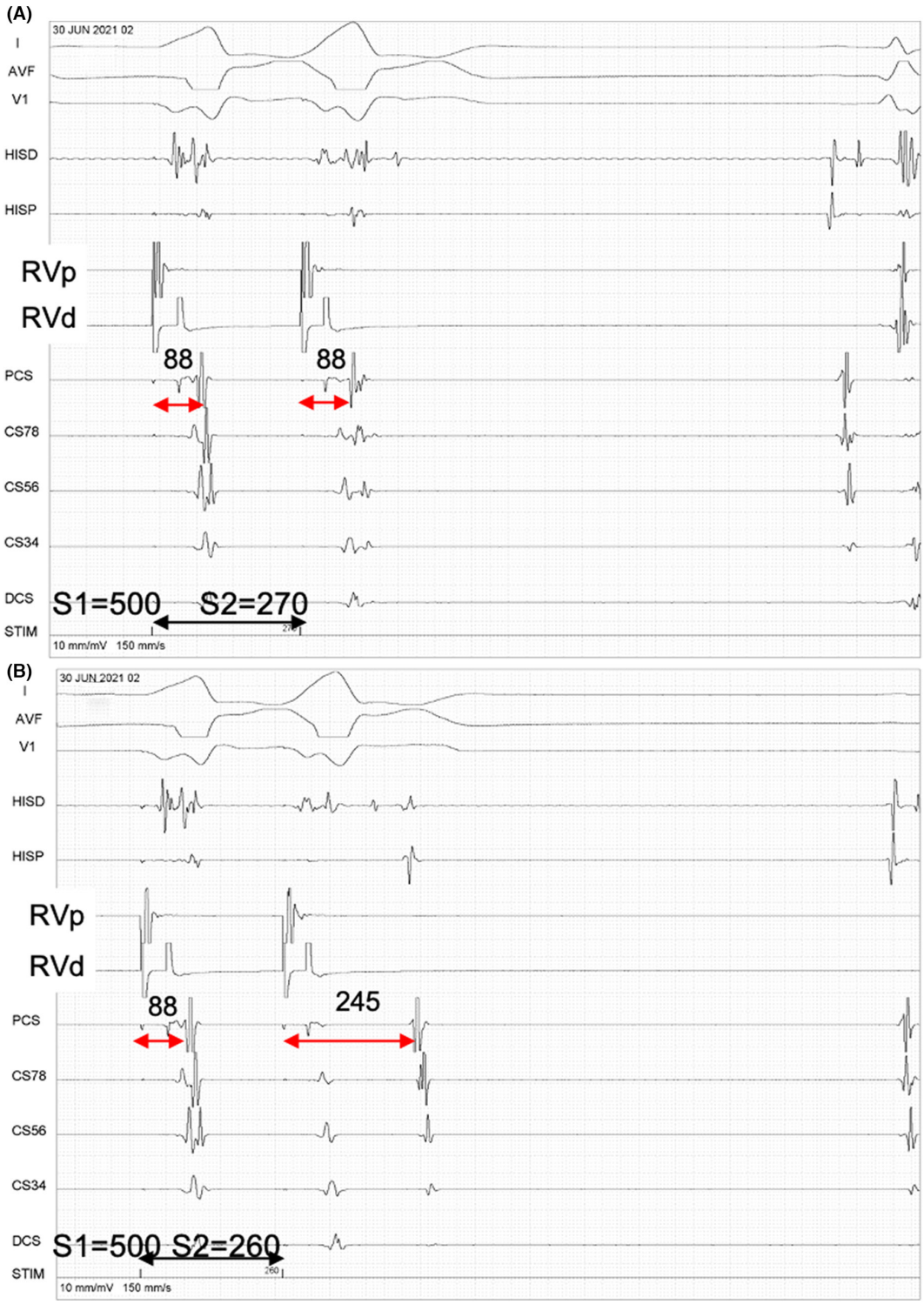

FIGURE 2 (A) The image shows nondecremental VA during VES (500/270 ms). The retrograde His signal is seen after the A-EGM in the second beat which is likely delayed due to retrograde RBBB. (B) During the VES of 500/260 ms the VA is decremental and the A-EGM is after retrograde His. VA conduction in this second beat is conducted via the AV node in contrast to the previous beat (via accessory pathway).

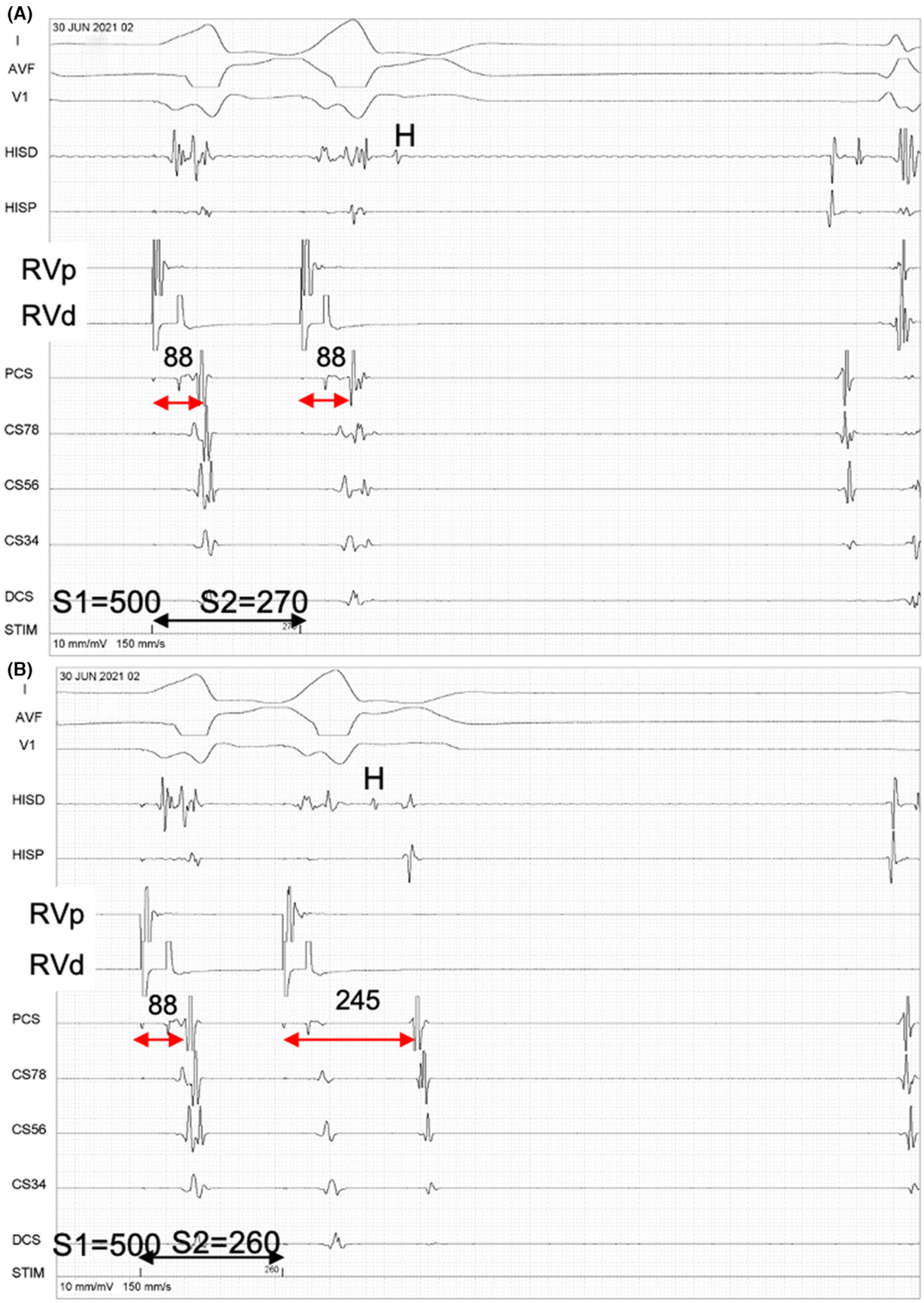

FIGURE 3 (A) and (B) are annotated figures of Figure 2A,B, respectively.