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Change in VA interval by a single atrial premature depolarization - What is the mechanism?

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ABSTRACT

A long VA tachycardia during a typical atrioventricular nodal reentrant tachycardia (AVNRT) can be a concomitant atypical AVNRT, atrial tachycardia or rarely atrio-ventricular reentrant tachycardia (AVRT). There are reported associations of AVNRT with other tachycardia substrates. Maneuvers are useful for differentiating the mechanism of the second tachycardia. Atrial tachycardia (AT) is one common association. When the AT originates from the lower triangle of Koch/near coronary sinus ostium, it can mimic slow-slow/fast-slow AVNRT. We encountered an interesting case where a longer VA tachycardia got reproducibly induced when a critically timed atrial premature depolarisation was delivered on typical AVNRT. It was proved to be an AT. A slow pathway modification in the lower TOK was successful to eliminate both the tachycardia substrate.

1. Case

A 35 year-old-lady underwent EP study for recurrent narrow QRS tachycardia (Appendix 1A) terminating with adenosine. Baseline ECG showed no pre-excitation (Appendix 1B). EP study was performed with decapolar catheter in coronary sinus (CS), quadripolar catheters placed in HRA (high right atrium) and His; and one roving catheter in right ventricle (RV). Baseline intervals showed that AH interval was 76 ms. The HV interval was 34 ms. During antegrade study AH jump, AV node duality and intermittent rate related aberrancy with unusual axis (RBBB with right axis deviation, HV 34 ms) was noted. Retrograde study revealed the VA conduction was concentric and decremental (VAERP = 270 ms) with no VA jump. No sustained tachycardia could be induced by standard protocols at baseline. On isoprenaline, a short VA tachycardia with near simultaneous A and V activation [HV = 34 ms, tachycardia cycle length (TCL) = 305 ms, Appendix 1C] was induced with atrial premature depolarization (APD) and ventricular premature depolarization (VPD) with similar rate related aberrancy (Appendix 1C and 1D). Maneuvers confirmed it to be slow-fast atrioventricular nodal reentrant tachycardia (AVNRT) (VAV response, SA-VA = 160 ms, cPPI-TCL = 151 ms). During programmed APDs from CS to differentiate it from junctional tachycardia (JT), the VA interval got abruptly prolonged (Fig 1A and B). The TCL was 260 ms after initial wobble with a septal VA of 140 ms. What could be the mechanism of this changeover with a single APD?

2. Commentary

The possibilities were:

A. It changed to atypical AVNRT (slow-slow or fast-slow) due to conduction via another slow pathway (SP).
B. It could be a different tachycardia - atrial tachycardia (AT) or orthodromic atrioventricular reentrant tachycardia (AVRT).

During the longer VA tachycardia (TCL of 260 ms), the earliest A-EGM was near CS ostium (PCS), hence, slow-fast AVNRT was unlikely. Atypical AVNRT (fast-slow) was however possible. Maneuvers were performed to confirm the diagnosis. But, interestingly this tachycardia could not be entrained by RV pacing, during which the atrium repeatedly got dissociated. This essentially ruled out

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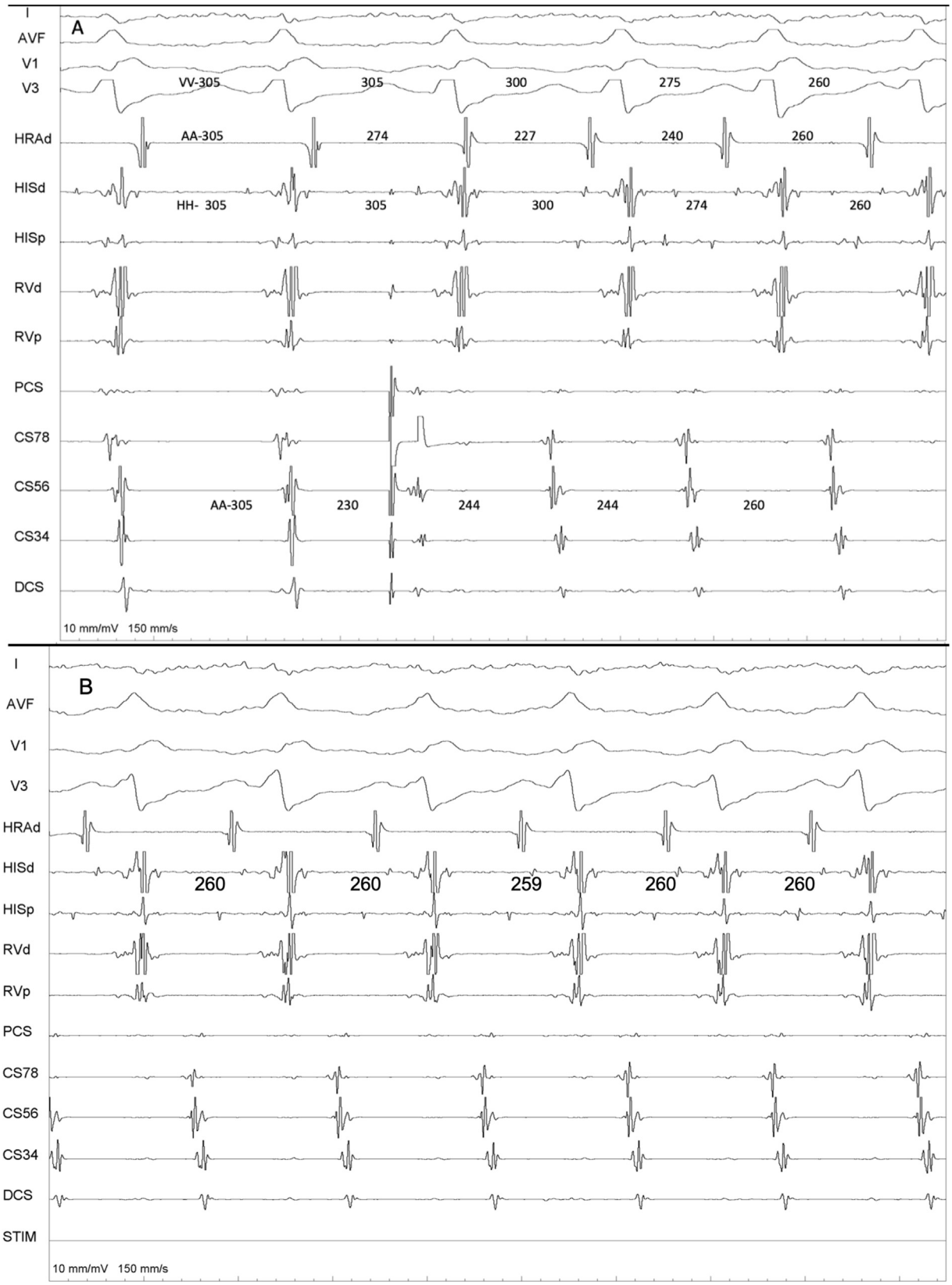

Fig. 1. A: Critically timed APD produced abrupt prolongation of the VA interval. The TCL became shorter (260 ms). The septal VA time was 140 ms.
B: Continued tracings during the longer VA tachycardia.

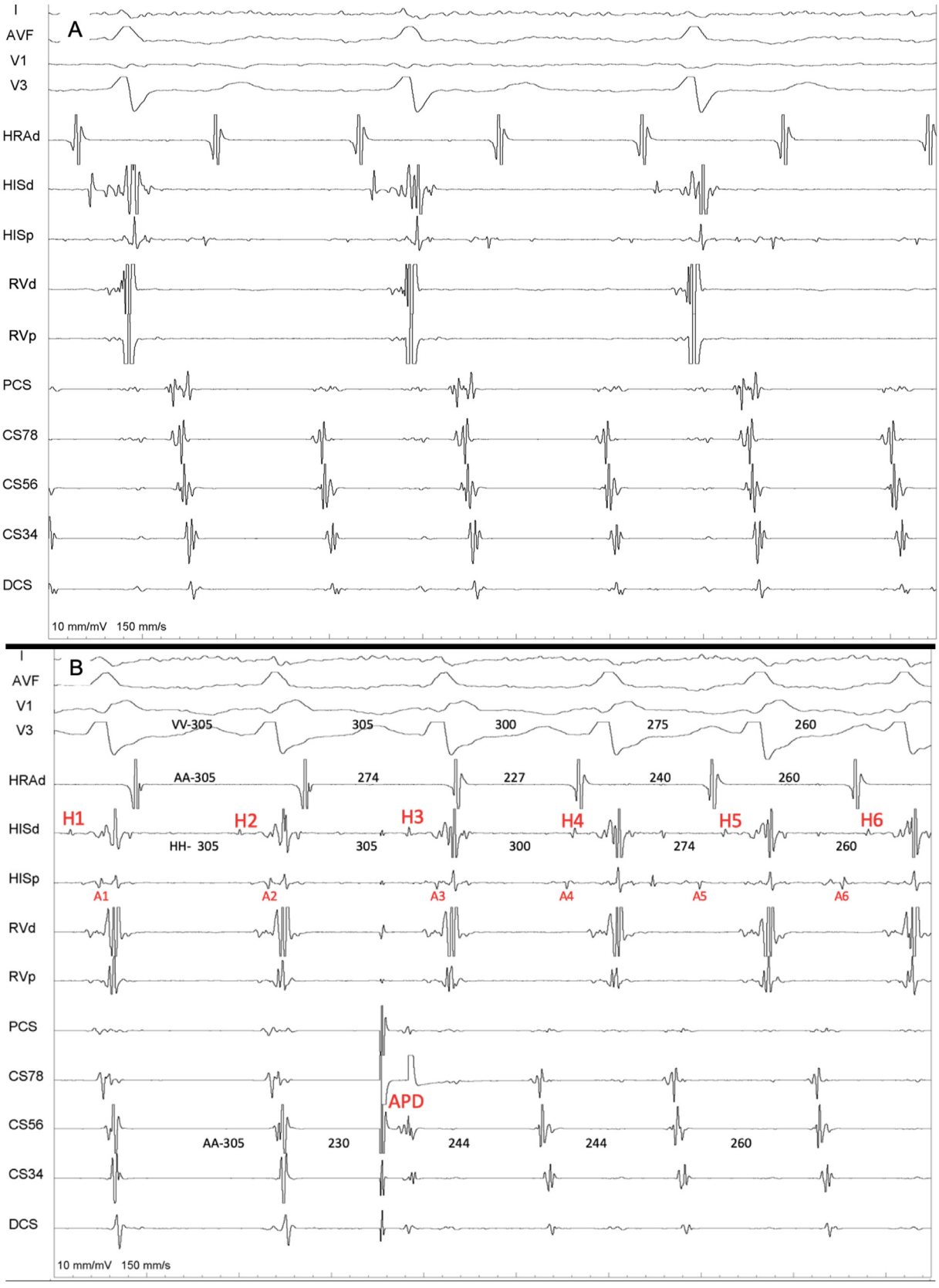

Fig. 2. A: A:V ratio of 2:1 during SVT2 after a an attempt of ventricular entrainment (atrial CL- 255–260 ms)
B: Annotated Fig. 1A showing that the APD did not engage the slow pathway as it was refractory. The H3 and next RR was unperturbed. There was a retrograde conduction *locally (A3)* via FP which could not produce any evident A-EGM in CS as it was prematurely captured by the APD. The same produced another beat of AVNRT (H4) beat via antegrade SP conduction. Hence the timing of next H (H4) was unperturbed. The second AT beat (A4) could capture the fast pathway and terminated the AVNRT after which AT completely took over. Over next few beats the wobble stabilised.

AVRT. To demonstrate VA linking HRA pacing was performed at 20 ms shorter cycle length (CL), but repeatedly terminated the tachycardia. At times, the A:V ratio after VOP (ventricular overdrive pacing) became 2:1 (supra-hisian), with same atrial CL of 255–260 ms (Fig. 2A). Although it can happen with both AT and AVNRT, AT becomes much more likely. Finally, to differentiate between an AT and a difficult-to-entrain AVNRT, injection adenosine (12 mg) was administered which demonstrated complete AV block during a continued tachycardia. This confirmed the diagnosis as AT. Henceforth, we labelled it as SVT2 for the rest of the discussion for easier understanding. Furthermore, few crucial observations were recorded during the transition from SVT1 to SVT2. The TCL during SVT2 became stable (TCL = 260 ms) after initial wobble. The AA intervals were noted to have the first change amongst all the intervals. Among them the sequence was CS-AA→HRA-AA→ HH/VV during the wobble (Figs. 1B and 2B). The atrial electrograms were also different in morphology. During an episode of SVT2, a spontaneous APD terminated SVT2 without perturbing the next His. All these further supported the diagnosis of AT.

An intriguing question here would be when did the AVNRT terminate and AT start during the changeover? Whether it terminated *immediately after the APD or did the AVNRT continue for a few more beats?* The initial wobble rendered important insights to these (Fig. 2B). It is noted that when the APD was delivered @ 230 ms coupling interval, the immediate H (H3) was not perturbed as it was committed to previous SP conduction. Also, the timing of local A-EGM (A3) in proximal His was not altered. In fact, the proximal His-A (A3) occurred after H3 with same local HA interval demonstrating retrograde conduction up the same fast pathway. The unperturbed H4 proves that AVNRT continued locally until this beat. The next AT beat (A4) took place before H4 and finally could enter the fast pathway (FP) and terminate the AVNRT. The next AT beat (A5) conducted antegradely via FP pathway to the ventricle and thenceforward the RR interval equated to the AA/HH interval of 260 ms.

Another unique finding of this case was reproducible induction of the AT with APD only from SVT1 (slow-fast AVNRT), but never from sinus rhythm. The probable explanation could be that the fast pathway ERP (effective refractory period) was reached earlier and the conduction over SP induced typical slow-fast AVNRT. The critical coupling interval (APD @ 230 ms, Fig. 1) required for induction of her AT (likely micro-reentrant) could not be reached as the AVNRT got easily induced with APDs with much longer coupling intervals. Interestingly, entrainment of SVT2 from RV apex was difficult despite good VA conduction (up to 270 ms) in sinus rhythm in contrast to the easily entrainable SVT1. We hypothesize that ERP of FP was reached during PCL @ 240 ms during SVT2 than during slower SVT1. In addition, we speculate that the FP was conducting antegradely during AT, hence was unavailable for retrograde conduction during attempted entrainment from RV.

For AT ablation, the earliest A was mapped and found to be near CS ostium/SP region. Although this AT could have been ablated during the tachycardia (SVT2), there was an apprehension of catheter drift at termination of tachycardia leading to AV nodal injury. Hence, it was decided to perform SP modification in sinus rhythm which might cure both, followed by an attempt of AT induction. RF energy (40 W, 60-degree, medium curve, non-irrigation tip catheter) was delivered in sinus rhythm which promptly resulted in accelerated junctional beats with intact VA conduction. After 90 s lesion, reinduction was attempted. Neither tachycardia could be induced with and without isoprenaline after this.

There are reported associations of AVNRT with other tachycardia substrates. In a retrospective study, Schernthaner et al. reported focal AT in 8% patients in a cohort of 493 patients after AVNRT ablation [1]. An earlier study had reported 15% subjects had inducible AT recorded during AVNRT ablation. Majority of them were however isoprenaline dependent. Moreover, only a minority (7%) with inducible AT, actually developed clinical AT on follow up after only SP modification [2]. When AT is associated with AVNRT, the site of origin can be at the CS ostium/Koch's triangle. P wave morphology and earliest A-EGM can localize the same [3]. The exact mechanism of the coexistence of AVNRT with AT especially originating from Koch's triangle remains indeterminable, although an interaction between AV nodal tissue and perinodal tissue has been postulated [4]. There is only one similar report of AT and AVNRT ablated successfully with SP modification [5].

Declaration of competing interest

None.
Consent has been taken from patient.

Acknowledgment

None.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ipej.2021.11.002.

Conflict of interest

We have no conflict of interest.

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None.

Data availability statement

I have included a statement that the study was approved by an Investigational Review Board (Human Studies Committee or Ethics Committee or Animal Care and Use Committee).

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