Reappraisal of resetting response to locate the antegrade pathway in slow-fast atrio-ventricular nodal re-entrant tachycardia

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ABSTRACT

Typical atrio-ventricular nodal re-entrant tachycardia (AVNRT) can occasionally remain easily inducible after slow pathway (SP) modification in lower Triangle of Koch (TOK). Analysis of resetting response by delivering atrial premature depolarizations (APD) from various sites (TOK, right atrium, coronary sinus and left atrium) can pin-point the culprit SP serving as the antegrade limb of the tachycardia circuit. However, the maneuver is under-utilized by most centers. We describe a case where anatomical SP modification in TOK failed to cure the arrhythmia. The resetting maneuver performed subsequently, helped us to rule out leftward inferior extension of SP and suggested right inferior SP as essential part of AVNRT circuit. Further ablation was performed at M1-M2 region (on the right side) to achieve success.

1. Case

A 50-year-old gentleman underwent electrophysiology (EP) study and radiofrequency ablation (RFA) for typical AVNRT. The diagnosis was confirmed and RF energy (40 W, 60 °C, non-irrigation Biosense Webster catheter) at lower part of TOK [1,2] (P1–P2 and P2-M1 region) produced abundant irregular junctional beats (over 90 secs) with intact VA conduction, but the tachycardia remained very easily inducible. Maneuvers (ventricular overdrive pacing, His-sync PVC) reconﬁrmed the supraventricular tachycardia (SVT) as slow-fast AVNRT. Hence, left sided extension of slow pathway (SP) was considered and atrial premature depolarizations (APDs) from various places (TOK, right atrium, inside coronary sinus) were delivered at 10 ms decrement starting 80 ms after His signal timing to analyze resetting response. It was much easier to reset tachycardia from right side (mid TOK, M1-M2 region than left side i.e from 2 to 3 cm inside CS ostium) [1,2]. From mid-TOK, the latest APD which could reset the next His-EGM and tachycardia, was 42 ms after H-signal (Fig. 1A) which delayed the next His-EGM by 15 ms. Progressively more premature APDs could reset the His by greater magnitude. More premature APDs could reproducibly terminate the AVNRT, a deﬁnite sign for tachycardia reset (Fig. 1B). On the contrary, to deliver APD within CS, the CS catheter was pushed inside and APDs were delivered from CS-78 pole, which was ﬂuoroscopically 2–3 cm inside CS OS. Several early APDs (even as early as 20 ms prior to His) could capture atrium, but failed to perturb the tachycardia circuit even a little (Fig. 1C and D). Hence, leftward inferior extension of SP was essentially ruled out as part of AVNRT circuit. Left lateral extension of SP was although not excluded which requires timed APD delivered from lateral left atrium, as the maneuver had already suggested a right sided circuit. Therefore, ablation was attempted at the mid-TOK (M1-M2) from where resetting was demonstrated easily. Three more RF energies were delivered producing accelerated junctional beats. RF energy was also delivered just outside CS ostium and SP conduction as well the tachycardia could be eliminated after this.

2. Discussion

Though the rightward inferior extension is by far the commonest SP involved; not uncommonly the leftward inferior
extension and rarely left lateral extension of SP can be the actual slow pathway participating in the AVNRT circuit [3]. Analysis of resetting response by APDs from various places can localize the SP serving as antegrade limb of the AVNRT [4–7]. The practical challenge faced while performing the maneuver is to ensure atrial capture especially during a faster tachycardia.

Our case highlights that, this simple yet useful maneuver can rule out leftward inferior extension of SP. The site from where the tachycardia could be most easily reset can be a good target for SP modification. This has been emphasized by Prof. Warren Jackman from his experiences at University of Oklahoma Health Sciences Center (USA). He also demonstrated that this maneuver is not time consuming when performed in trigger sweep mode. Rather it saves considerable lab time and radiation when the operator is aware of the culprit SP from beginning.

Fig. 1A: APD from lower TOK. The latest APD which could reset AVNRT was 42 ms later than His signal. It delayed the next His by 15 ms. (the red and green vertical line marks the tachycardia cycle length of 378–379 ms, in all the diagrams).

Fig. 1B: More premature APD (exactly simultaneous to His timing) terminated the AVNRT (Most convincing way to prove the tachycardia reset).

Fig. 1C and D: APD delivered from 2 to 3 cm inside CS ostium. No APD could RESET (pull/push/perturb next His signal, HH remained constant 380 ms) the AVNRT.

In Fig. 1D, even more premature APD (20 ms earlier than His) could not RESET the tachycardia.

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Declaration of competing interest
This is to declare that all of us are authors of the following manuscript titled ‘Reappraisal of resetting response to locate the antegrade pathway in slow-fast atrio-ventricular nodal re-entrant tachycardia’ and w23e have no conflict of interest.

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