Supraventricular tachycardia with abrupt onset and termination: What is the mechanism?

Mukund A Prabhu^{1,2} MD DM PDF, Robert D. Anderson¹ MBBS, Paul B Sparks MBBS, FRACP, Ph.D.

1. Department of Cardiology, Royal Melbourne Hospital and University of Melbourne Grattan Street, Parkville, VIC 3050, Australia

2. Kasturba Medical College, Manipal academy of higher education (Manipal University), Manipal, Karnataka 576104, India

Corresponding author

Dr. Paul Sparks MBBS, FRACP, PhD

Director of Heart Rhythm Services, Royal Melbourne Hospital

Postal Address: Department of Cardiology
Royal Melbourne Hospital, 300 Grattan Street
Parkville VIC 3050

Telephone: 61409951330

Fax: 613 93495411

Email: paul.sparks@mh.org.au

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Case report

A 44-year old woman had undergone a slow pathway ablation for atrioventricular re-entrant tachycardia in 2016. She had symptomatic relief for about a year. However, she had recurrence of palpitation recently, and she could distinguish that the episodes were slower and shorter compared to her original ones. Her arrhythmia was captured on a Holter tracing (Fig:1) and she underwent an electrophysiological study (Fig:2 &3). The baseline intervals were normal, and there was retrograde dual AV nodal physiology with VA (retrograde H was not seen to measure HA) jump and echo for a 40ms window of S2. There was no antegrade dual AV nodal physiology (even with high dose of Isoprenaline), and the AV block CL was 470ms. The tachycardia was induced with difficulty by atrial burst pacing at 340ms while on Isoprenaline at 8mcg/min infusion. The tachycardia was of abrupt onset, with CL of 420ms,

earliest A in the coronary sinus proximal (CSP) electrode, and a ventriculoatrial (VA) interval of 0ms. The entrainment showed a VAHV response with a long post-pacing interval (PPI-tachycardia CL=140ms), and the difference between Stim-A and the VA intervals was 110s. The effect of atrial ectopics during the tachycardia is shown in **Fig:2 and 3**, recorded during the electrophysiological study (EPS). What is the likely mechanism of the tachycardia, and did the atrial ectopics reset the tachycardia?

Discussion

The Holter strip (Fig:1) shows the onset of the tachycardia, which is abrupt. More strikingly, the onset is with a QRS complex, and the RP interval was very short with the P wave immediately following the QRS. This suggests the possibility of an atrioventricular nodal reentrant tachycardia (AVNRT) being triggered by a 1:2 response, or a Junctional tachycardia (JT). The cycle length (CL) of the tachycardia was stable, and it terminated abruptly but with a change in the P wave axis and the RP relation. The abruptness of the onset and the stability in the CL suggests the possibility of an AVNRT, as these are the features of a reentrant mechanism. However, the termination of the tachycardia shows a striking change in the P wave axis, which progressively becomes isoelectric before getting upright and marching out of the QRS, and preceding it. Presumably, the sinus node must have accelerated and overdriven the tachycardia as seen in the latter part of the strip that shows sinus tachycardia. Such a response suggests an isoarrhythmic atrioventricular (AV) dissociation. Though an AV dissociation is possible in AVNRT, it is more commonly a feature of JT.

The atrial extrastimulus did not advance the His though it occurred before the His deflection (**Fig:2**). However, there was a spontaneous premature atrial complex (PAC) following this that nullifies the intended maneuver. But, the PAC did advance the His potential. The returning cycle commenced with a His potential with a fully compensatory

pause. This continuation of the tachycardia is against a typical AVNRT, which would have terminated with advancement of the His. Moreover, the VA is not 'linked' in the few cycles after the PAC and this is also more consistent with JT. The fully compensatory pause speaks against the rare possibility of a re-initiation by 1:2 response through a slow pathway that would arguably have a different returning cycle. Both the ectopics probably did not reset the tachycardia, resulting in a fully compensatory pause ($HH' + HH = 2HH$, **Fig:2**). The compensatory pause (i.e. no reset) would also suggest a relatively protected H focus.

The response to atrial extra is useful in differentiating JT and AVNRT(1). The early atrial extras the advancement of His will engage the fast pathway orthodromically and thus terminate the AVNRT, whereas JT can continue with advancement, delay, or no change in the next beat. In contrast, the His-committed atrial extra doesn't affect JT, whereas it can advance or delay the next beat in AVNRT as it can engage the slow pathway that may conduct with or without a decrement. The His-committed atrial extra did not affect the tachycardia in the present case (**Fig:3**), whereas as early atrial ectopic, it advanced the His without terminating it. This latter response excludes a typical AVNRT that will be terminated by an atrial extra that advances the His, since this will render the fast pathway refractory. The His-committed APC should be interpreted with caution as an atrial extrastimulus can penetrate into the tachycardia focus without affecting the timing of the His. The refractoriness of His at the time of atrial extra can only be assured by ensuring that the local A in the His catheter follows the His potential(1). This is important because the atrial extra penetrating the tachycardia focus can evoke any of the responses- advancement, delay or lack of any affect in the case of JT. In the present case it is likely that though the local His was recorded after the His potential, the tachycardia was not reset as revealed by the unperturbed HH interval despite the shortened AA' interval and the fully compensatory pause ($AA' + AA = 2 \times AA$, Fig)

There are a few maneuvers to differentiate between AVNRT and JT (2) (3), which are summarized in table:1. The patient was planned for medical management in view of difficulty in induction, and higher risk of AV block associated with the ablation of JT.

Legends

Figure:1 **A** The Holter tracing shows the onset of the tachycardia with a QRS complex (5th complex) that is coincident on the P wave. The first 2 beats of the tachycardia lack as P wave after the QRS, whereas the subsequent beats have a P wave immediately following the QRS. **Panel B** shows the termination of the tachycardia as recorded by the channel-1 of the Holter. The P wave axis changes to isoelectric, and then to positive. The RP relation alters as the P wave that followed the QRS disappears inside the QRS before marching out to precede it suggesting an isoarrhythmic AV dissociation. Ch1-3 represents the three Holter channels

Figure:2 The EPS tracing at 100mm/sec paper speed. The channels represent (from top to bottom), the standard electrocardiographic leads I, II, V1, V6, followed by the His distal and proximal electrodes (HisD and HisP, respectively), followed by coronary sinus electrodes presented in distal to proximal order (CSD to CSP, respectively) in this as well as the next figure.

The HH intervals are shown in bold. The atrial extrastimulus which did not advance the H. This is followed by a spontaneous atrial ectopic. The resultant His (H') is advanced by 100ms. The subsequent pause is fully compensatory ($HH' + H'H = 410 + 510 = 2 \times HH = 820$). The tachycardia continues at the initial rate thereafter. The intervals are in milliseconds.

Figure:3 The HH intervals and the AA intervals are depicted in bold. The atrial extrastimulus is committed to the His. Note that the local A recorded in the His occurs after the His. The HH interval is unperturbed, whereas the AA is shortened to 410ms. The subsequent pause is 470ms and is fully compensatory ($410+470=2 \times 410=880\text{ms}$). Note the that the tachycardia is slightly slower in the current tracing, likely due to the effect of sedation.

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Table:1 Summarizes the various maneuvers that help to differentiate AVNRT and JT and the precautions to interpretation.

Abbreviations- AVNRT-Atrioventricular nodal reentrant tachycardia. FP- Fast pathway, JT- Junctional tachycardia,

Manoeuvre	AVNRT	JT	Comment
Early Atrial extra	Resets the tachycardia (advancement of His with continuation of tachycardia) ¹	Advance the His and terminates the tachycardia ² , or Resets it with a prolonged AH interval (delays the H) ²	1 Specific for JT with the exception being double fire 2 Nonspecific responses as the may occur in JT as well
His refractory Atrial extra	Doesn't affect the tachycardia ³	Can advance, delay or terminate the tachycardia ⁴	3 Nonspecific response 4 Specific for AVNRT
V pacing at TCL- Delta HA interval	Close to 0(or positive)	Negative	Serial HA activation in JT as opposed to parallel activation in

			<p>AVNRT</p> <p>HA interval in AVNRT is a pseudo interval</p>
<p>Atrial entrainment – returning cycle</p>	<p>AHHA response</p>	<p>AHA response</p>	<p>Double fire is a potential exception (AVNRT mimics JT)</p> <p>Diseased FP with bystander dual AVN– JT may potentially mimic AVNRT</p>

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Author/s:

Prabhu, MA;Anderson, RD;Sparks, PB

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