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A narrow QRS tachycardia with changing rate: What is the mechanism(s)?

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Case presentation

A 15-year-old girl presents with frequent daily palpitations and a structurally normal heart on clinical examination and echocardiography. Her 12-lead electrogram was normal and the Holter monitoring is shown **Fig:1 and Fig:2**. What is the likely mechanism(s) of the tachycardia?

Discussion

There are a few key phenomena that deserve critical attention. The onset of the tachycardia(s) is always triggered by an atrial ectopic beat which is conducted with a long PR interval suggesting dual AV node physiology with slow pathway conduction. There are two types of RP relationships. The tachycardia in **Fig:1A** is a short RP tachycardia with 1:1 VA relation with P waves seen just after the QRS complexes and is characteristic of an orthodromic AVRT though at this stage other mechanisms such as AVNRT or AT cannot be

categorically excluded. The tachycardia in **Fig:1B** has such a P wave seen only in the first beat. Thereafter, the P waves disappear (likely hidden within the QRS). This variation in the RP relationship would be consistent either with variable retrograde conduction during AVNRT or with 2 different tachycardia mechanisms. During ongoing tachycardia (**Fig: 1B**), the ventricular rate suddenly halves and P waves regularly occur mid-way between the QRS complexes. (ventricular rate drops from 174bpm approximately to 87bpm approximately, **Fig:2B**). However, P waves continue to fall at places where QRS complexes were expected during the faster tachycardia. This continues till the ventricular rate abruptly doubles again as seen in **Fig:2**. This is consistent with ongoing AVNRT with 2:1 conduction to the ventricle. Conduction intermittently recovers with wide QRS beats (arrows, **Fig:2A**) probably as a result of a transient bundle branch block subsequent to a sudden increase in the rate. The consistency of the pattern in which this occurs suggests aberrancy rather than ventricular ectopy as the likely mechanism of these wide-QRS beats. In due course, block recovers and the tachycardia continues with P waves merged within the QRS complexes (**Fig:2B**), characteristic of an AVNRT which spontaneously terminates with a QRS complex (**Fig:2C**). The consistent onset of this tachycardia with abrupt prolongation of the PR interval together with the positioning of the P wave exactly midway between QRS complexes during 2:1 conduction is most consistent with AVNRT rather than an AT.

This patient underwent an electrophysiological study. Baseline intervals were normal, with no preexcitation. Ventricular pacing showed eccentric non-decremental VA conduction with the earliest A in the coronary sinus (CS)-5,6 dipole. The tachycardia was induced by ventricular pacing and followed an eccentric VA activation. The entrainment responses were consistent with AVRT. A His-committed ventricular extra stimulus (**Fig:3**) advanced the

subsequent A consistent with an AVRT. However, thereafter the atrial activation pattern changed with the earliest A now in the His catheter (**Fig:3**, double arrow) with a shorter VA interval consistent with typical AVNRT which spontaneously terminated in 2 beats with a an "A" (this occurred repeatedly ruling out atrial tachycardia) (**Fig: 3**, double arrow). The accessory pathway was first ablated during the tachycardia at the posterior mitral annulus and subsequently, a slow-pathway ablation was performed. No tachycardia was inducible after the ablations.

The occurrence of the block in the LCP in the setting of induced AVNRT in the electrophysiology laboratory was estimated at approximately 10%¹. The block is usually functional and transient. The incidence of this phenomenon in the setting of spontaneous AVNRT is unknown. Such instances may be missed as the phenomenon is transient and it is possible that this is more common than previously suspected. In the current case, 2:1 AVNRT was misinterpreted as a sinus rhythm with first degree AV block and the patient incorrectly told that slow pathway ablation would create a risk of AV block.

References

1. Man KC, Brinkman K, Bogun F, Knight B, Bahu M, Weiss R, Goyal R, Harvey M, Daoud EG, Strickberger SA, Morady F. 2:1 Atrioventricular Block During Atrioventricular Node Reentrant Tachycardia. *J Am Coll Cardiol Elsevier Masson SAS*; 1996;**28**:1770–1774.

Legends to figures

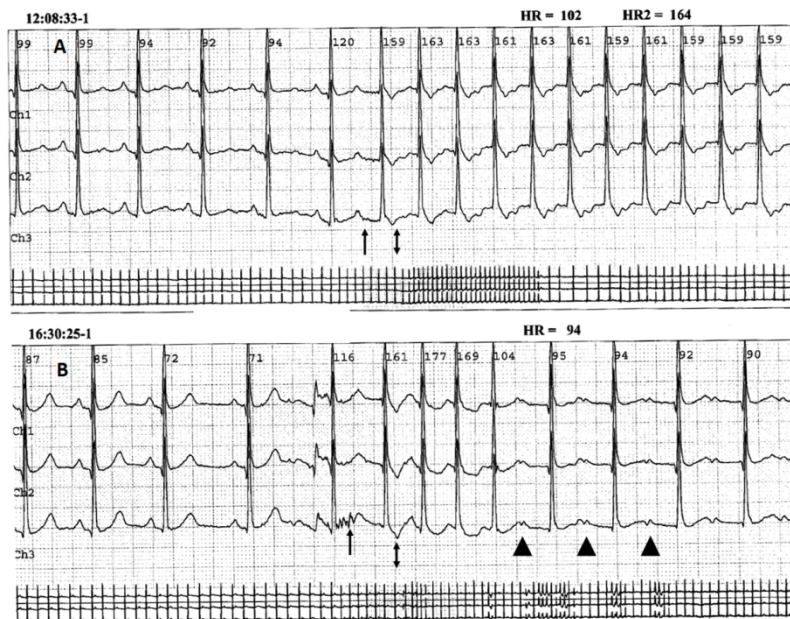


Fig1: The Holter tracing shows tachycardia onset which is triggered by an atrial ectopic beat (single arrow, panelA). The double-arrow marks the P wave of this short-RP tachycardia. The next panel (B) shows another instance of a tachycardia triggered by an atrial ectopic (single-arrow). The double-arrow in the panel B represents the P wave following the QRS of the first tachycardia beat. The P wave is not seen during the next two beats and thereafter the tachycardia halves in rate and the P waves are seen mid-way between the QRS complexes (arrow-heads). Note that the two panels are recorded at different times. See text for further details.

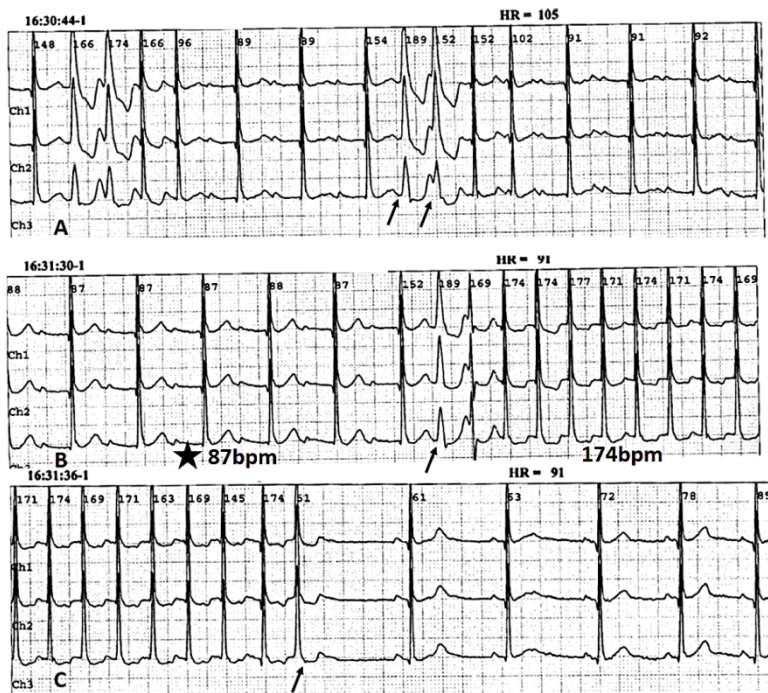


Fig2: The Holter tracing shows the continuation of the tachycardia shown in Figure 1B. Panel A: Shows the P waves falling midway between the QRS complexes, and wide-QRS beats when the rate abruptly increases due to rate-related aberrancy Panel B: There is a sudden doubling of the ventricular rate in the second half of the panel to 174bpm (asterisk shows a rate of 84bpm in the initial half of the panel) which again in a single aberrantly conducted beat (arrow) followed by continuation of the narrow-QRS tachycardia with P waves probably occurring within the QRS complexes. Panel C: The tachycardia terminates with a QRS complex (arrow). See text for further details.

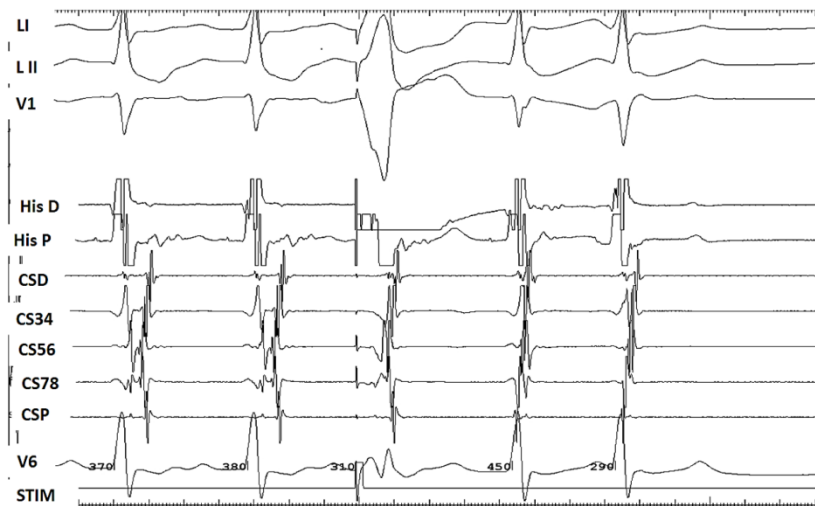


Fig3: The intracardiac recording during the electrophysiological study is shown at 100mm/sec recording speed and 0.5 voltage gain. The first three channels represent surface ECG leads I, II and V1, respectively. This is followed by the recordings from the distal and proximal His catheter bipoles. The five bipoles from the coronary sinus(CS) catheter are shown in the distal to proximal order are shown next. Finally, the lead V4 and the stimulation channel are shown. The first two beats are during the tachycardia and show eccentric atrial activation with earliest A in the CS-5,6. The third beat is a His-committed ventricular extra-stimulus delivered from the His-distal catheter to capture the right ventricle. This advances the A which has the same activation as that of the tachycardia. After this, the last two beats represent another tachycardia that has a shorter VA interval and a different atrial activation pattern with the earliest A now being in the His region. This terminated spontaneously with an A.