


The transition of the tachycardia from narrow to wide by a spontaneous atrial premature beat: What is the mechanism?

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Keywords: Ashman phenomenon, atrial premature beat, atrioventricular nodal reentrant tachycardia, coumel sign, slow-fast AVNRT
41-year-old woman underwent radiofrequency catheter ablation of narrow QRS tachycardia (NQT). Twelve-lead ECG was normal during sinus rhythm. The electrophysiological study showed normal AH and HV intervals during sinus rhythm. Ventricular pacing showed decremental ventriculoatrial (VA) conduction with concentric atrial activation. Parahisian pacing showed VA prolongation with the loss of His capture consistent with nodal conduction. Atrial burst pacing reproducibly induced an NQT with near-simultaneous atrial and ventricular activation patterns. A spontaneous premature atrial extra stimulus (AES) has resulted in the transition of the tachycardia from narrow to wide. What is the mechanism of tachycardia?

The differential diagnosis of a short RP NQT with the earliest atrial activation in the His bundle region includes atrial tachycardia (AT) originating from near the AV node, orthodromic reciprocating tachycardia (ORT) using a septal accessory pathway, typical (slow-fast form) atrioventricular nodal reentrant tachycardia (AVNRT), junctional tachycardia (JT), and orthodromic nodofascicular (NF) or nodoventricular (NV) reentrant tachycardia (NFRT/NVRT).¹ A on V tachycardia (with a short septal VA interval of <70 ms) easily excludes ORT.¹

Whereas the principal unique maneuver in wide QRS tachycardia diagnosis is the placement of sensed AES during tachycardia; it is also a useful method to identify AVNRT from JT.^{2,3} It might ease the changes of QRS morphology into wide configuration by long-short sequence (Ashman phenomenon). Indeed, the transition from one tachycardia into another is very uncommon. It could occur due to a

spontaneous or induced AES, conduction block at one limb of tachycardia, or tachycardia-induced tachycardia.

The recognition of His bundle potential is important for differentiation. In our patient, the His bundle potential could not be detected during tachycardia, and a sharp His-like potential preceding the QRS complex [as of right bundle (RB) potential] was found in the ventricular septum (Figure 1). The RB potential nicely also demonstrates long-short sequence in current tracing. When an AES is timed to His refractoriness, any perturbation (advancement, delay, or termination of tachycardia) of the next His indicates that anterograde AV nodal slow pathway conduction is present during the tachycardia.² This finding would be true only in the absence of simultaneous anterograde fast and slow pathway conduction.³ However, when an AES (irrespective of early or late coupled) perturbs the next His, this confirms the diagnosis of AVNRT and allowing for differentiation from JT.³ In the current case, even if His potential is not clear; there was a delay in the subsequent RB and QRS by 120 msec confirming engagement of slow AV nodal pathway.³ There was also a fixed VA linking.⁴ These responses exclude JT and AT, and leaves only AVNRT and the much less commonly observed NV/NFRT.⁵ There was no change in the ventriculoatrial interval with the development of the RB branch block suggesting the diagnosis of typical AVNRT compared to NF/NV pathways.⁵ Therefore, this was a typical AVNRT during which a premature atrial beat causes a long-short sequence resulting in RB aberration. Radiofrequency ablation delivered in the slow pathway region elicited junctional beats and resulted in tachycardia noninducibility.

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FIGURE 1 The transition of the tachycardia from narrow to wide by a spontaneous atrial premature contraction is seen. The beat following the narrow QRS beat shows RBBB



CONFLICT OF INTEREST

None.

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How to cite this article: Kara M, Korkmaz A, Deveci B, et al.

The transition of the tachycardia from narrow to wide by a spontaneous atrial premature beat: What is the mechanism?. *J Arrhythmia*. 2021;37:462–463. <https://doi.org/10.1002/joa3.12499>