Atrioventricular nodal reentrant tachycardia and the dilemma of reentry circuit components: A proof of concept

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Introduction
Atrioventricular nodal reentrant tachycardia (AVNRT) is one of the most common supraventricular tachycardias, and uncommon manifestations present a challenging diagnosis and can lead to misdiagnosis, subjecting patients to unnecessary risks, making the correct diagnosis of utmost importance. Uncommon AVNRT manifestations can mimic other supraventricular tachycardias such as atrial fibrillation, atrial tachycardia (AT), junctional tachycardia (JT), or some supraventricular arrhythmias mediated by rare or common bypass tracts.

Case report
A 42-year-old woman was referred for electrophysiological study owing to palpitations refractory to beta-blockers. An electrocardiogram (ECG) recorded during symptoms revealed a supraventricular tachycardia (SVT). The patient complained of recurrent palpitations since adolescence frequently interrupted by 6 mg adenosine bolus. No comorbidity was referred. The cardiologic evaluation revealed normal left ventricular function, sinus resting ECG, and no sustained arrhythmias in 24-hour Holter monitoring.

Under conscious sedation and local anesthesia, 2 right femoral vein and 1 right jugular vein punctures were performed to insert catheters in the right chambers (Supplemental Figure 1S): 1 quadripolar catheter in the right atrium / right ventricle, 1 quadripolar catheter in His bundle, and a decapolar catheter in coronary sinus floor, respectively. Baseline ventriculoatrial (VA) conduction was evaluated by incremental ventricular pacing and a 1:1 VA relation (earlier atrium in proximal coronary sinus) up to 600 ms cycle length was observed.

Incremental atrial pacing (high right atrium and coronary sinus) demonstrated atrioventricular (AV) anterograde Wenckebach point at 380 ms cycle length. No sign of ventricular pre-excitation on the surface ECG was detected and the HV interval remained within normal range. High atrium programmed stimulation induced an SVT with critical AH interval (AHi) owing to AV node “jump” (Supplemental Figure 2S). A progressive RP interval prolongation followed by a P blocked wave, resembling a Wenckebach cycle, was observed (Figure 1 and Supplemental Figure 3S). Subsequently, a spontaneous atrial contraction anticipates the next beat, resetting the circuit (Supplemental Figure 4S). Tachycardia cycle length (TCL) was prolonged and the atria were activated at each every other cycle (2:1 VA block, Figure 2) with upper-axis P waves in the frontal plane (Supplemental Figure 5S). A few seconds later, a 2:1 VA block alternates with a 2:1 AV infra-Hisian block (Figure 3). After that, 1:1 AV conduction resumes with left bundle branch morphology along with a 2:1 VA conduction (Supplemental Figure 6S) and further up, lone His bundle

KEY TEACHING POINTS

- Atrioventricular nodal reentrant tachycardia (AVRNT) can manifest itself as unusual electrocardiographic and electrophysiological findings.
- Attentive appraisal to electrical spontaneous events during tachycardia is essential to the understanding of its mechanism.
- At least in some patients with AVNRT, the upper and lower common pathways are present and responsible for some unusual findings.

KEYWORDS
Atrioventricular nodal reentrant tachycardia; Atrioventricular node; Catheter ablation; Supraventricular tachycardia; Tachycardia (Heart Rhythm Case Reports 2021;7:439–441)

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depolarizations during tachycardia suggests a “concealed” reentry circuit within or under the AV node (Supplemental Figure 7S). Sinus rhythm was restored and during atrial pacing, a different 1:2 A/V ratio tachycardia with longer RP interval was induced (Supplemental Figure 8S). A slow-slow AVNRT was diagnosed and the slow pathway mapped and successfully ablated in the triangle of Koch. After ablation, programmed and decremental atrial pacing was performed with and without isoprenaline effect and no evidence of slow pathway was found, nor was tachycardia induced. The patient remained asymptomatic after a 12-month follow-up.

Discussion
We present a case of an SVT induced with a critical AHi, a variable septal VA interval, and different AV/VA relations in a patient with palpitation refractory to beta-blocker therapy. As in other SVTs, differential diagnosis of AT, accessory AV reentrant tachycardia, AVNRT, and JT was imposed. The lack of 1:1 AV relation during tachycardia excludes AV reentrant tachycardia, and a AV relation < 1 makes AT extremely unlikely. Differential diagnosis of JT and AVNRT became imperative. A spontaneously atrial contraction anticipating the next tachycardia beat suggests the diagnosis of AVNRT (Supplemental Figure 4S), albeit the rare situation of a patient with a focal JT and a “bystander” anterograde slow pathway over which conduction can proceed cannot be excluded.1

Interestingly, in a matter of minutes tachycardia presented differently, making diagnosis challenging. Despite all controversy, the uncommon AVNRT behavior in the presented case can be best explained by the presence of upper common pathway (UCP). A body of evidence suggests that atrial

Figure 1  Upper common pathway (UCP) Wenckebach phenomenon during atrioventricular nodal reentrant tachycardia. A: Wenckebach cycle to the atria at UCP. Surface electrocardiography shows a progressive increase in RP interval (black arrows) until a P wave blocks in the UCP. B: Wenckebach cycle illustration in UCP: green arrows indicate conduction by the fast pathway and red irregular arrows conduction by the slow pathway. Straight black arrows represent depolarization of UCP and wavy lines represent impulse block. AVN = atrioventricular node; HB = His bundle; LCP = lower common pathway.

Figure 2  Upper common pathway 2:1 block during atrioventricular nodal reentrant tachycardia. A: After an increase in tachycardia cycle length, the impulse blocks in the upper common pathway (UCP) every other cycle (fixed AH interval, septal VA interval, and HA interval) in a 2:1 VA fashion. B: Upper-axis P waves in frontal plane every other QRS complex corresponding to a 2:1 VA block in the UCP. Green and red arrows: conduction by the fast and slow pathways, respectively. Straight black arrows represent the impulse through the UCP and the wavy lines the impulse block. AVN = atrioventricular node; HB = His bundle; LCP = lower common pathway.
dissociation by fast atrial pacing without disturbing tachycardia, and a difference in AHi during pacing and during AVNRT, could indicate the presence of UCP. These strategies, however, are limited because they are based on the principle that active and bystander circuit limbs have the same anterogradely and retrogradely electrophysiological properties. Difficulty in atrial tissue capture owing to TCL irregularity or to atrial refractoriness are also limitations. Spontaneous recordings suggesting UCP, although rare, contribute to a better understanding of the reentrant circuit by allowing conclusions free of intrinsic bias related to the methodologies of these stimulation studies. Despite its being rare, and considering the absence of ventricular pre-excitation in decremental and programmed atrial stimulations, a concealed nodofascicular reentrant tachycardia could be considered. The ΔAH, ΔHA, and ΔVA pacing maneuvers along with ventricular extrastimulus in His-refractory could provide more elements for the diagnosis, but variations in TCL made pacing maneuvers impossible to perform. Concealed nodofascicular reentrant tachycardia, however, manifests itself as a long RP tachycardia (RP > PR) and from a mechanistic point of view it is not compatible with an A/V ratio > 1.

**Conclusion**

The AVNRT reentrant circuit is complex and electrophysiological manifestation can be heterogeneous. Our finding strongly suggests the existence of a UCP, at least in some patients with AVNRT.

**Appendix**

**Supplementary data**

Supplementary data associated with this article can be found in the online version at https://doi.org/10.1016/j.hrcr.2021.03.022.

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