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Narrow-QRS-Complex Tachycardia: What Is the Mechanism?

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A 30 year old female with history of atrio-ventricular nodal reentrant tachycardia (AVNRT) who underwent slow pathway modification at age of 12 years presented with recurrent palpitations and presyncope for one year. She was found to have recurrent supraventricular tachycardia sensitive to adenosine and was referred for electrophysiology study and catheter ablation. Baseline electrocardiogram showed sinus rhythm, RBBB, with no evidence of preexcitation. Electrophysiology study showed an AH 73ms, HV 48 ms, and RR 876 ms. Atrial extra-stimulus pacing showed no evidence of dual AV node physiology. Ventricular extra-stimulus pacing showed midline decremental conduction, figure 1A, and no change in the retrograde atrial activation sequence. Supraventricular tachycardia was consistently induced with single atrial extra-stimuli without an AH jump. Ventricular extra-stimuli never induced tachycardia. At baseline, the tachycardia had a cycle length of 450 ms, which decreased to 320–330 ms on isoproterenol, and occasionally, showed cycle length variability, figure 1B. Tachycardia always terminated with ventricular activation and the response to adenosine showed no retrograde VA conduction. Ventricular overdrive pacing at a cycle length of 310 ms when tachycardia had a CL of 330 ms is shown in figure 2A, and the response to atrial pacing at a cycle length of 310 ms, when tachycardia cycle length was otherwise stable at 320 ms, is shown in figure 2B. Entrainment from the ventricle repeatedly terminated tachycardia. Ventricular overdrive pacing at a CL much faster than the tachycardia lead to VA dissociation. Single His bundle refractory ventricular extra-stimuli reproducibly showed the response in figure 3 and on one occasion, terminated tachycardia. What is the mechanism of tachycardia?

DISCUSSION

This tachycardia had several unusual features. The retrograde atrial activation sequence during ventricular pacing showed the earliest atrial activation at the proximal His bundle recording site, consistent with fast pathway activation, figure 1A. Ventricular pacing also showed no change in the retrograde atrial activation sequence with no evidence of retrograde AH jump. However, during tachycardia, the earliest atrial activation was seen in mid CS (CS 5,6). AVNRT with left sided atrial extension has been previously reported.^{1, 2} In addition, the different atrial activation sequence during tachycardia compared to ventricular pacing could potentially be explained by anisotropic conduction, allowing preferential conduction up the fast pathway during ventricular pacing without easy access to the damaged/modified slow pathway. On the other hand, during tachycardia, anisotropic conduction could lead to

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preferential activation of the slow pathway with a left sided extension, showing earliest atrial activation in CS 5,6 (mid CS).

The circuit of this tachycardia was further remarkable in that neither atrial tachycardia nor AVNRT typically show spontaneous oscillation in cycle length without evidence of decremental conduction. In this case, the longer VA intervals seen during tachycardia were not preceded by short VA intervals, while the short VA intervals were not preceded by long His-His or VA intervals. Therefore, the changes in cycle length could not be explained by “Wenkebach-type” behavior. It is possible that a damaged slow pathway with longitudinal dissociation³ or two anatomically close slow pathways could explain the oscillation. During ventricular overdrive pacing, figure 2A, tachycardia continued with the same atrial activation despite full ventricular capture and then terminates without advancing the atrium. This finding ruled out atrial tachycardia.

Of note, overdrive pacing of the high right atrium at a cycle length slightly faster than the tachycardia cycle length, figure 2B, showed capture of the His bundle for at least 2 beats prior to capture of the coronary sinus. The response to high right atrial overdrive pacing pointed to the fact that the tachycardia circuit did not involve the His bundle, lower common pathway, or ventricle, and may only involve part of the AV node and peri-nodal atrial tissue at the coronary sinus. This finding clearly ruled out AV reentrant tachycardia utilizing an accessory pathway and also a nodoventricular/nodofascicular (NV/NF) reentrant tachycardia. His bundle refractory PVC's during tachycardia showed evidence of post-excitation with the same retrograde atrial activation sequence as that seen in during tachycardia, figure 3. Furthermore, on one occasion, a His bundle refractory ventricular extra-stimulus terminated tachycardia without atrial activation (figure not shown). This finding further ruled out atrial tachycardia. Although post-excitation with a His bundle refractory ventricular extra-stimulus typically indicates presence and participation of a decremental atrio-ventricular pathway, in this case, it most likely represented the presence of a bystander (NV/NF) pathway with access to the AV nodal/perinodal circuit.⁴ The His bundle refractory ventricular extra-stimulus resulted in decremental conduction in the AV region of the circuit and then conducted to the atrium with the same atrial activation sequence as the tachycardia.

In this patient ablation at the anterior lip of the CS ostium at the usual slow pathway location made the tachycardia non-inducible. This area *did not* represent the earliest atrial activation, and is consistent with previously reported study of AVNRT with left sided extension.¹

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REFERENCES

1. Hwang C, Martin DJ, Goodman JS, et al. Atypical atrioventricular node reciprocating tachycardia masquerading as tachycardia using a left-sided accessory pathway. *J Am Coll Cardiol.* 1997; 30:218–225. [PubMed: 9207645]
2. Yano K, Horikawa T, Hirao K, et al. Slow-fast form of atrioventricular nodal reentrant tachycardia with eccentric retrograde left-sided activation. *Jpn Heart J.* 1999; 40:655–664. [PubMed: 10888385]
3. Atié J, Brugada P, Brugada J. Longitudinal dissociation of atrioventricular accessory pathways. *J Am Coll Cardiol.* 1991; 17:161–166. [PubMed: 1987221]
4. Evans SJ, Blumberg SM, Scheinman MM. A nodoventricular fiber associated with dual AV nodal conduction, AV nodal reentrant tachycardia, and anterior location of the slow AV nodal pathway. *Pacing Clin Electrophysiol.* 1999; 22:119–122. [PubMed: 9990610]

Figure 1.

Panel A shows the response to ventricular extra-stimuli at 400/230 ms prior to ventricular effective refractory period. Panel B shows occasional oscillation in cycle lengths noted during tachycardia. HRA = high right atrium, His p = His bundle proximal, His m = His bundle mid, His d = His bundle distal, CS = coronary sinus, RV = right ventricle, CS 9,10 was located at the coronary sinus ostium.

Figure 2.

Panel A shows the response to ventricular over drive pacing at cycle length of 310 ms during tachycardia (tachycardia CL was 330 ms on isoproterenol). The arrow points to the first captured ventricular beat that leads to a change in the atrial activation sequence. Cessation of overdrive pacing showed resumption of sinus rhythm indicating termination of tachycardia during ventricular pacing. Panel B shows initiation of pacing from the high right atrium at a cycle length of 310 ms during tachycardia, which was stable prior to initiation of pacing at a cycle length of 320 ms. HRA = high right atrium, His p = His bundle proximal, His m = His bundle mid, His d = His bundle distal, CS = coronary sinus, RV = right ventricle, CS 9,10 was located at the coronary sinus ostium.

Figure 3.

The response to a His bundle refractory ventricular extra-stimulus at a time of stable tachycardia cycle length of 450 ms is shown. HRA = high right atrium, His p = His bundle proximal, His m = His bundle mid, His d = His bundle distal, CS = coronary sinus, RV = right ventricle.