Teaching Rounds in Cardiac Electrophysiology

Identical Atrio–His Interval and A–A Intervals During Long RP Tachycardia
What Is the Mechanism?

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Case Presentation
A 46-year-old woman with frequent palpitations underwent electrophysiological study. The findings of transthoracic echocardiography were normal, and a 12-lead ECG showed no ventricular pre-excitation. Four catheters were placed in standard locations: high right atrium, His bundle, coronary sinus (CS), and right ventricular apex. All the recorded baseline intervals were within normal limits. Retrograde ventriculoatrial (VA) conduction showed decremental and dual pathways during ventricular pacing and single extrastimulus testing. Antegrade atrioventricular (AV) nodal conduction showed decremental and triple pathways during atrial single extrastimulus testing. Narrow-complex supraventricular tachycardia (SVT) was reliably induced with a V–A–V sequence by a single ventricular extrastimulus (Figure 1). Ventricular premature depolarization (VPD) during tachycardia, when the His bundle was refractory, did not reset the atrial cycle. Retrograde Wenckebach periodicity occurred during right ventricular overdrive pacing at a cycle length (CL) of 500 ms. The tachycardia was terminated with atrio–His (A–H) and VA block during atrial and ventricular entrainment pacing. Figure 2 shows progressive prolongation and abrupt shortening of the A–A interval with block during SVT. In addition, the A–H interval was identical to the A–A interval during SVT. On the basis of these findings, what is the mechanism of the tachycardia?

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Commentary
The differential diagnoses of long RP interval tachycardia, with the earliest atrial activation near the ostium of the CS, are atypical AV nodal reentrant tachycardia (AVNRT), orthodromic reciprocating tachycardia with a slowly conducting and decremental accessory pathway and postero-septal atrial tachycardia (AT). Because of the presence of the His-refractory VPD without the reset phenomenon as well as sustained SVT during A–H block, the possibility of orthodromic reciprocating tachycardia was excluded. Moreover, automatic AT with initiating AV nodal echo was not completely excluded by the V–A–V sequence on tachycardia initiation. However, AT was less likely because of the termination of tachycardia with A–H/V–A block during atrial/ventricular entrainment pacing and the direct correlation between the A–A interval and the A–H interval during SVT. Therefore, on the basis of these findings, atypical AVNRT was the most likely diagnosis.

In Figure 2, the first 3 A–A intervals were 450 ms, whereas the A–H intervals were gradually prolonged. This observation could be explained by the presence of a lower common pathway (LCP, which is defined as the conduction pathway between the tachycardia circuit and the His bundle) with decremental conduction. The presence of an LCP was proven by the fact that the retrograde Wenckebach CL (500 ms) during ventricular pacing was longer than the tachycardia CL (450 ms). Subsequently, the next A–A intervals were progressively prolonged to 455–480 ms and further to 540 ms and abruptly shortened to 440 ms with an A–H block. Interestingly, the fifth and sixth A–H intervals were identical to the A–A interval. There are several potential mechanisms that can explain these findings. First, a simple hypothesis was that the A–H intervals became progressively longer because of the decremental conduction of an LCP, which was much stronger than that of the antegrade AV nodal pathway, thereby causing a block of the LCP that was reflected as an A–H block. However, this hypothesis does not explain the abrupt shortening of the A–A intervals with an A–H block. The second hypothesis was that the fifth and sixth As switched to a slow pathway, with a slow LCP resulting in the retrograde A preceding the His bundle. Subsequently, the seventh and eighth As are the result of a double fire from the upper turn around point above the LCP and a lower turn around point (LTP) below the LCP. However, during the first 4 A–A intervals, the conduction of the LCP was progressively prolonged, whereas the conduction of the antegrade AV nodal pathway was only 5 ms. Therefore, in the subsequent As, the hypothesis of an LCP block was more
likely. Finally, the most likely mechanism may be the dual ventricular response with an LCP block, that is, the antegrade impulse of the tachycardia circuit traveled down a fast pathway, which was followed by a turn to a retrograde slow pathway via the LTP1 with LCP block before the His bundle (Figure 3). Although, at the same time, the antegrade impulse showed a double ventricular response (Figure 3, asterisk), an antegrade slow pathway was conducted to LTP2 but did not turn to a retrograde slow pathway because of the effective refractory period. The next beat also showed a double ventricular response. First, the antegrade fast pathway was conducted to LTP1 and turned to the retrograde slow pathway, while at the same time, an antegrade slow pathway was conducted to LTP2 with decremental conduction and turned to the retrograde slow pathway, which was characterized by recovered excitability and resumed conduction because of the greater prolongation of the A–A interval. However, the impulse from the retrograde slow pathway, which was turned from the antegrade fast pathway, did not travel down the both antegrade fast and slow pathways because of the effective refractory period.

![Figure 1. Intracardiac electrogram showing that narrow-complex supraventricular tachycardia was induced with a V–A–V sequence by a single ventricular extrastimulus (600/370 ms). CS indicates coronary sinus; HBE, His bundle electrogram; HRA, high right atrium; and RVA, right ventricular apex.](image)

![Figure 2. Intracardiac electrogram showing progressive prolongation and abrupt shortening of the A–A interval during supraventricular tachycardia. *The atrial and His bundle potentials were fused. CS indicates coronary sinus; HBE, His bundle electrogram; HRA, high right atrium; and RVA, right ventricular apex.](image)
refractory period. As a result of these conduction pathways, the A–A interval was shortened to 440 ms with an A–H block.

Long RP tachycardia with variable A–H intervals is usually diagnosed as AT. However, atypical AVNRT with variable A–H intervals has been reported previously. Long RP tachycardia with variable A–H intervals during atypical AVNRT were progressively prolonged compared with the prolongation of A–A intervals. This observation was evidenced by the presence of an LCP, as an out-of-tachycardia circuit. In addition, a dual ventricular response can be sustained, producing a 1:2 tachycardia, which is otherwise known as a nonreentrant SVT. Nonreentrant SVT typically showed that a single atrial impulse conducts both dual AV nodal pathways, and this is followed by excitation of both the His bundle and the ventricles. The main point of interest in the present case is that the A–A interval were identical to the A–H interval during AVNRT. These observations could be explained by a dual ventricular response with an LCP block. To our knowledge, this is the first case in which atypical AVNRT was sustained via a dual ventricular response. Radiofrequency catheter ablation was applied at the earliest retrograde slow pathway lesion at the posteroseptum, with successful elimination and the absence of inducible tachycardia.

Disclosures

None.

References


Key Words: atrioventricular block □ atrioventricular node □ cardiac electrophysiology □ catheter ablation
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