Teaching Rounds in Cardiac Electrophysiology

Innocent Bystander or the Heel of Achilles

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Case Presentation
A 29-year-old male presented with palpitations without a documented tachycardia. ECG showed minimal preexcitation (Figure 1A), and echo confirmed a normal heart. At electrophysiological study in the drug-free state, the HV interval was 13 ms in sinus rhythm, and ventricular (V) activation in the right para-Hisian region preceded the surface delta wave (Figure 2A). Programmed stimulation from the septal right atrium (A) increased preexcitation at shorter S1–S2 intervals (below 600–360 ms), as evidenced by a subtle loss of physiological left-to-right ventricular (RV) septal activation (loss of r wave in V1 and q waves in I, aVL) and an increasingly negative HV interval. The S2–δ interval also prolonged with progressive preexcitation. Figures 1B and 2B show increased preexcitation elicited at an S1–S2 interval of 220 ms. Atriovenous (AV) effective refractory period was shorter than 600–220 ms, and dual AV nodal physiology was absent. These features along with the existence of escape junctional preexcited beat (Figure 1C and 2C) supported the presence of a right-sided manifest nodoventricular (NV) pathway bypassing a portion of the AV node, and ruled against typical anterograde AV bypass tracts, Mahaim fibers from AV ring, and fasciculoventricular pathways.

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Retrograde conduction was through a concealed left lateral accessory pathway (AP) that had slow decremental conduction and a long ventriculoatrial (VA) effective refractory period of 600–340 ms. Despite multiple atrial and ventricular programmed or burst stimulation attempts, no tachycardia was induced. However, coincidental with decrement in retrograde AP conduction (VA interval increasing from 112 to 136 ms), single reciprocating ventricular echo beats were easily reproducible, even during fixed RV pacing at rates moderately faster than the sinus rate (Figure 3). After intravenous adenosine 12-mg, VA block developed during RV pacing (cycle length: 550 ms) consistent with the decremental nature of VA conduction and the lack of typical retrograde bypass tracts (Figure 1A in the Data Supplement). Soon after recovery of VA block, during ongoing RV pacing, a nonsustained 1:1 VA reciprocating tachycardia (cycle length: 310–345 ms) was induced (lasting 35 s) with an atrial activation pattern identical to that of the ventricular echo beats (Figure 4A; Figure 1B in the Data Supplement). An His-refractory RV pacing stimulus advanced the subsequent A with resetting of the tachycardia without change in atrial activation (Figure 4B), thereby confirming participation of the left lateral AP in the tachycardia. The QRS morphology of the ventricular echo beats, the tachycardia and the beat subsequent to the atrial advancement brought by the His-refractory extrastimulus represented variably fused anterograde NV fiber and AV nodal conduction (Figures 3 and 4).

In light of these features: (1) Why was the induction and maintenance of tachycardia so difficult, despite easily provoked ventricular echo beats? (2) What was the role of the NV pathway in the tachycardia mechanism?

Considerations
Nature of Anterograde Preexcitation
The intranodal origin of the para-Hisian right-sided pathway, and therefore its interpretation as a NV fiber and exclusion of alternative preexcitation mechanisms, was well supported in various respects: Mahaim fibres from the AV ring (long atriofascicular or short AV types) exhibit increased preexcitation with pacing closer to the atrial insertion of the AP.1,2 As seen in Figures 1 and 2, the degree of preexcitation did not change during S1–S1 pacing from septal right atrium (close to the AP atrial insertion). Only programmed atrial stimulation at shorter S1–S2 intervals could dissociate the anterograde conduction over the AP and AV node and consequently demonstrate the change in preexcitation degree, strongly supporting the presence of a NV fiber.1,3 Besides, RV apical activation did not precede δ-wave onset, ruling out an atriofascicular type Mahaim fiber.2 A preexcited junctional beat excluded all AV bypass tracts, including atriofascicular pathways. However, Mahaim automaticity from the AV ring can be mistaken as a junctional beat. Their differentiation, however, lied in the degree of manifest preexcitation. Whereas the preexcitation is maximally expressed during Mahaim automaticity, surface preexcitation with fusion, and HV interval identical to that in sinus rhythm favored a junctional origin of the beat and origin of the AP from a portion distal in the AV junction.1 Finally, unlike a fasciculoventricular pathway, preexcitation was not fixed, but dependent on the S1–S2 interval.

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Tachycardia Discourse

Role of Excitable Gap
A fundamental prerequisite for maintaining reentry is an electric wavelength ($\lambda_e$) shorter than the anatomic wavelength ($\lambda_a$), defined as the excitable gap ($\lambda_a - \lambda_e$). In the persistent form of junctional reciprocating tachycardia, 2 reciprocal limbs (AV node and concealed AP) manifest decrement in their conduction velocity and electric wavelengths ($\lambda = \text{conduction velocity} \times \text{effective refractory period}$) at faster rates. Therefore, $\lambda_e$ can be considerably shorter than $\lambda_a$, which gives rise to a large excitable gap. These features make persistent form of junctional reciprocating tachycardia readily inducible (often after slight variation in the sinus rate), relatively incessant, and also susceptible to premature activations.

Single Persistent Form of Junctional Reciprocating Tachycardia Beats
The patient's reciprocating ventricular excitation was unusual. Single ventricular echo beats were readily inducible with decrement in retrograde AP conduction even during RV pacing slightly faster than the sinus rate, but tachycardia was not sustained. This can be explained by the accompanying NV preexcitation. After the echo beat, the preexcited component of the anterograde ventricular wavefront preempted normal AV conduction time and penetrated the left lateral AP during its effective refractory period, blocking further reentry.

Effect of Adenosine
A nonsustained reciprocating tachycardia induced shortly after adenosine suggested that tachycardia initiation was dependent on adenosine-mediated conduction delay. As VA and AV nodal conduction recovered during adenosine withdrawal, a vital degree of anterograde conduction delay persisted in the NV fiber. This prevented premature penetration of the left lateral AP during the excitable gap, thereby allowing tachycardia initiation. Several seconds later, once the effect of adenosine on NV fiber conduction delay also weaned off, anterograde ventricular preexcitation, bypassing the AV node, resulted in tachycardia termination.

The distinctly different time course of adenosine-induced block in the AV node and the NV fiber analogous to the present observations has been previously described. Block occurs initially both in the NV fiber and in the AV node, with subsequent recovery of conduction over the AV node followed several seconds later by the resumption of conduction over the NV fiber. It is likely that the time course of block in the concealed left lateral AP matched that of the AV node.

Intracardiac Ventricular Activation Transition
The aforesaid bearings of NV fiber conduction on the tachycardia induction and maintenance were surmised through a focal transition in ventricular activation observed before tachycardia termination. There was reversal in the dominant

![Figure 1. 12-lead ECGs during sinus rhythm (A), programmed extrastimulation from septal right atrium at S1–S1=600 ms and S1–S2=220 ms (B), and a junctional beat (C).](http://circ.ahajournals.org)
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polarity of activation of the local V electrogram in the HISd channel and its shift ahead of the beginning of surface ventricular depolarization before tachycardia termination (Figure 5A and 5B). The local V onset to surface δ interval increased from 26 to 30 ms. With the HIS channels mapping the putative ventricular insertion of the NV fiber in the right para-Hisian region, the change from positive to negative polarity in the initial component of the local V electrogram marked a switch from apicobasal activation coming off the conduction system to a basoapical activation originating from the NV fiber. This preexcited wavefront beat-by-beat progressively paved its way locally through the para-Hisian myocardium into the tachycardia circuit and eventually led to its termination in the left lateral AP. The additional myocardial tissue activated during this expansion of NV preexcitation wavefront was probably too small to visibly alter the surface QRS, which thus remained latent on the surface ECG. Furthermore, there was no change in the tachycardia cycle length before its termination.

Despite a lack of change in manifest surface preexcitation, the local ventricular activation transition was deservedly an indicator of increased anterograde preexcitation. Indeed, a reversal in the initial polarity of the HISd ventricular electrogram was also noted in parallel with a prominence in the surface δ wave of the QRS that followed atrial advancement brought by the His-refractory extrastimulus during tachycardia (Figure 4B). This single beat of premature right para-Hisian NV activation, however, did not have the merit to penetrate and disrupt a primarily left-sided tachycardia circuit, especially when the natural NV conduction was still impaired under adenosine.

Role of NV Fiber

The temporal association of tachycardia initiation with adenosine, and ventricular excitation transition before tachycardia termination, practically resolved the bystander nature of the NV fiber in the tachycardia mechanism. Maintenance of the tachycardia was dependent on the engagement of the AV nodal conduction and a partial disengagement from NV preexcitation (Figure 6). Although the concealed left lateral pathway was the obvious culprit for tachycardia, the NV fiber was not stationed just as an innocuous bystander. It lent negating consequences on what could otherwise be an incessant orthodromic reciprocating tachycardia.

Management

Cognizant of these results, rationalizing patient’s palpitations to the ventricular echo beats or short runs of tachycardia, we...
decided catheter ablation of the concealed left AP. This was successfully achieved transeptally along the mitral annulus 2 o'clock position. Post ablation, there was no VA conduction and no ventricular echo beats or additional tachycardias were inducible. Ablation of the NV pathway was, therefore, not attempted. Patient has remained free of palpitations without drugs at 6 months of follow-up.

Bystander NV pathway coexisting with another AV node-dependent tachycardia has been reported previously, but our case uniquely involved a decremental left-sided concealed AP. It is essential to analyze the mechanism critical for tachycardia operation because targeting the NV pathway may not only fail but also unexpectedly engineer a more incessant tachycardia as conceivable from the present case.

**Figure 3.** A, Intracardiac electrograms during pacing (S1–S1=460 ms) from right ventricular apex (RVA). Depending on the decrement in the retrograde accessory pathway conduction (VA interval increased from 112 to 136 ms), single reciprocating ventricular echo beats were reproducible. B, 12-lead ECG of a ventricular echo beat elicited during pacing from RVA. CS indicates coronary sinus; and RA, right atrium.

**Figure 4.** A, Intracardiac electrograms during tachycardia induced during adenosine washout. B, Atrial advancement in response to a ventricular stimulus (S1) falling in the His-refractory phase during tachycardia while on pacing from the right ventricular apex (RVA). The subsequent QRS has increased surface preexcitation (best evident in leads II and V2), which is accompanied by a change in morphology of the local ventricular electrogram in the HISd channel (*). The next S1 stimulus is in the ventricular refractory period and fails to capture. C, 12-lead ECG of the tachycardia. CS indicates coronary sinus; and RA, right atrium.
Conclusions

In aggregate, these findings demonstrated an uncommon pairing of multiple decremental APs, which paradoxically cannot support a persistent form of AV reciprocating tachycardia. This was validated by certain impromptu but specific electropharmacological responses. The time course of adenosine-induced

Figure 5. A, Intracardiac electrograms during tachycardia before its spontaneous termination. Compared with the beginning tachycardia beats (represented by first 5 beats; blue label), the morphology of local ventricular electrogram in the Hisd channel changes in the 7 beats before tachycardia termination (orange label). There was no appreciable change in the surface QRS morphology, and the tachycardia cycle length (345 ms) remained consistent during this transition until termination. B, The transition period shown in A is displayed at a faster recording speed of 200 mm/s. The dominant polarity of activation in the initial part of the local ventricular electrogram in the Hisd channel is reversed (*). The local V onset to surface δ interval increased from 26 to 30 ms, indicating expanded anterograde preexcitation wavefront. CS indicates coronary sinus; RA, right atrium; and RVA, right ventricular apex.
block in the NV fiber differed from that of AV nodal and left lateral AP block and became decisive in defining tachycardia components. Preexcitation from the NV pathway reduced the reciprocating tachycardia’s excitable gap, thus becoming an Achilles heel to what would otherwise be considered a safe circumrotation.

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None.

References

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SUPPLEMENTAL MATERIAL

Supplementary Figure 1. Response to intravenous adenosine 12mg during RV pacing (S1-S1=550ms).

A. Initial response. Sinus-rate slowing and VA dissociation.

B. Later response. Sinus acceleration, recovery of VA conduction (as seen after two premature ventricular complexes), and tachycardia initiation. Decrement in VA interval after the second premature ventricular complex precedes tachycardia onset. Channels arrangement and abbreviations are as in Figure 2.
Supplementary Figure 1

A

B