Subtle Changes in Electrogram Morphology During Para-Hisian Pacing Performed on IV Adenosine: A Role in Diagnosis of Accessory Pathways with Prolonged Retrograde Conduction Times

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ABSTRACT. Para-Hisian pacing is a valuable electrophysiologic maneuver devised to differentiate between retrograde conduction over atrioventricular (AV) nodal and accessory pathways. This case highlights the utility of this important technique. Furthermore, it emphasizes the significance of recognizing subtle changes in the electrogram morphology as a complementary tool when assessing for the presence of accessory pathway versus AV nodal conduction. It also offers a role for modified para-Hisian pacing in the presence of adenosine to evaluate for the existence of unidirectional accessory pathways with retrograde conduction times longer than that of the AV node.

KEYWORDS. accessory pathway, adenosine, electrogram morphology, para-Hisian pacing.

Case presentation

A 47-year-old woman with a history of lifelong symptomatic supraventricular tachycardia underwent an electrophysiologic study. At the onset of the procedure, pacing from the right ventricular apex (RVA) yielded a “flat” but eccentric atrial activation pattern, with the earliest activation noted on the distal coronary sinus (CS), consistent with retrograde conduction over a left lateral or a left posterior accessory pathway (Figure 1a). The CS catheter was repositioned within the small-caliber CS, and a long RP narrow-complex tachycardia with a cycle length of 410 ms was subsequently inducible (Figure 1b). Entrainment of the tachycardia from the RVA yielded a VAV response most consistent with an orthodromic atrioventricular re-entrant tachycardia (AVRT). During tachycardia, the earliest atrial activation was again seen on the distal CS, consistent with retrograde conduction over a left lateral or a left posterior accessory pathway (Figure 1c). Next, the site of the earliest atrial activation was carefully mapped via a transseptal approach while pacing from the RVA (600/300 ms). The presence of a left posterior accessory pathway was confirmed, and the accessory pathway was successfully ablated using radio-frequency (RF) energy.

Post-ablation, atrioventricular (AV) nodal physiology was found to be single with midline and decremental AV conduction. VA conduction was also intact and decremental. However, VA conduction again yielded a mildly eccentric atrial activation pattern, with the earliest atrial activation now recorded on CS 7,8. In
order to exclude extranodal pathways, intravenous (IV) adenosine was administered, which clearly resulted in transient third-degree AV block, consistent with anterograde conduction over AV nodal pathways (Figure 2a). But when pacing from the RVA at 600 ms, 1-to-1 VA conduction was still present with prolonged VA conduction times, with CS 7,8 as the earliest site of atrial activation (Figure 2b). Although adenosine has been shown to exert differential effects on anterograde and retrograde AV nodal pathways, the above finding coupled with a mildly eccentric atrial activation seemed to suggest the possible presence of a second accessory pathway.

To evaluate this, para-Hisian pacing was performed next. During His capture the stim-A measured 64 ms. Following loss of His capture (during local ventricular capture), stim-A was 131 ms (Figure 2c). Although at first glance this might seem suggestive of normal physiology with an AV node–AV node response, careful assessment of the intracardiac recordings seemed to suggest otherwise. Firstly, the earliest atrial activation during His capture was clearly evident on CS 9,10. On the other hand, the site of the earliest atrial activation during loss of His capture had noticeably shifted towards CS 7,8. In addition, the atrial electrogram morphologies on CS had distinctly changed during the presence and absence of His capture (most clearly seen on CS 3,4, CS 5,6, and CS 7,8), implying the presence of different pathways for atrial activation.

To further investigate these findings, it was decided to repeat para-Hisian pacing following administration of IV adenosine (Figure 2d). Para-Hisian pacing performed immediately following administration of adenosine yielded a stim-A interval of 152 ms during both His capture and during loss of His capture. In addition, retrograde atrial activation was distinctly more eccentric, with the earliest activation noted consistently on CS 7,8 during both presence and absence of His capture (Figure 2d). Furthermore, the morphology of the atrial electrograms recorded during both His capture and local ventricular capture remained unchanged. But both were appreciably different from the morphologies recorded during para-Hisian pacing off adenosine. These observations were consistent with an accessory pathway–accessory pathway response involving a unidirectional, retrogradely conducting, left posteroseptal accessory pathway with a VA time significantly longer than that of the AV node. The presence of the accessory pathway could further be substantiated through careful assessments of the local VA and HA times to deduce the degree of retrograde AV nodal participation, and also by differential pacing from apical and posterobasal right ventricle (RV). The retrograde effective refractory period (ERP) of this accessory pathway could not be determined, since it too was longer than the AV nodal ERP (600/240 ms).

In the meantime, with repeated induction attempts a second tachycardia consistent with an orthodromic AVRT using a left posteroseptal accessory pathway was eventually inducible (Figure 3a). The earliest atrial activation during tachycardia was recorded on CS 7,8 and the VA time was prolonged, measuring 162 ms. Next, the site of the earliest atrial activation during RVA pacing following administration of IV adenosine, was ablated using RF energy. Adenosine proved equally beneficial during mapping and ablation of the
accessory pathway through pharmacologic suppression of the AV node. Post ablation, IV adenosine was once again administered during RVA pacing. This time, absent retrograde (VA) conduction, with the earliest activation noted on CS 7,8 (emphasized by the dotted arrow). This maneuver poses the possibility of conduction over a posteroseptal accessory pathway. An alternative explanation might propose that VA block was not achieved successfully by the administered dose of adenosine. However, this would be unlikely since the maneuver was reproducible, even at higher doses of adenosine (up to 18 mg). A para-Hisian pacing maneuver during both His and local ventricular capture. As seen here, the stim-A measures 64 ms during His capture and is significantly prolonged (131 ms) following loss of His capture. This might initially suggest an AV node–AV node response. However, further examination of the atrial activation pattern on the CS catheter shows subtle changes. During His capture, the earliest atrial activation is seen on CS 9,10. On the other hand, during local ventricular capture the site of the earliest atrial activation transitions to CS 7,8, suggesting that conduction may be fusing over both the AV node and an accessory pathway. In addition, the atrial electrogram morphologies distinctly differ between the two events, most obviously on CS 3,4, CS 5,6 and CS 7,8. (d) Modified para-Hisian pacing following administration of intravenous adenosine. This time, stim-A intervals during presence and absence of His capture were both the same at 152 ms confirming the presence of an accessory pathway. Moreover, the atrial activation is now noticeably more eccentric, with the earliest activation localized to CS 7,8. Once again, the electrogram morphologies also differ from those seen during His capture off adenosine. In fact, further assessment of the electrogram morphologies would suggest 1) conduction occurring predominantly over the AV node during His capture off adenosine; 2) conduction likely fusing over the AV node and the accessory pathway during loss of His capture off adenosine; and 3) conduction occurring exclusively over the accessory pathway during modified para-Hisian pacing performed on adenosine. These observations and findings are indeed consistent with the presence of a unidirectional, retrograde and slowly conducting, left posteroseptal accessory pathway with a VA conduction time significantly longer than that of the AV node.

**Commentary**

Para-Hisian pacing is a valuable and essential electrophysiologic maneuver devised to differentiate between retrograde conduction over AV nodal or accessory pathways.\(^3\) This case highlights the utility of this important technique. Furthermore, it emphasizes the significance of recognizing changes in the electrogram
morphology as a complementary tool when assessing for presence of accessory pathway versus AV nodal conduction. It also offers a role for modified para-Hisian pacing in presence of adenosine to evaluate for existence of unidirectional accessory pathways with retrograde conduction times longer than that of the AV node.

The above observations and maneuvers proved exceptionally useful in the treatment of our patient since the VA time during retrograde conduction of her second accessory pathway was significantly longer than retrograde conduction over her AV node. In addition, the ERP of the accessory pathway was longer than that of the AV node. Therefore, retrograde conduction always occurred preferentially over the AV node, constantly masking conduction over the accessory pathway. This obviously explains the observation of decremental VA conduction following ablation of the first accessory pathway. It also provides a clear explanation of why para-Hisian pacing yielded a shorter stim-A interval in presence versus absence of His capture. Careful assessment of the electrogram morphology recorded during para-Hisian pacing performed before and after adenosine clearly suggests fusion in the atrial activation when performed off adenosine (between the AV node and the accessory pathway). On the other hand, during modified para-Hisian pacing performed on adenosine, the retrograde limb of the AV node was transiently inhibited, thereby unmasking conduction exclusively over the accessory pathway. This accounts for the striking differences noted in the atrial electrogram morphologies following administration of adenosine.

In this case, para-Hisian pacing demonstrated subtle changes in the atrial activation patterns with marked electrogram morphological changes, but without the classical response normally seen in patients with retrogradely conducting accessory pathways. The abnormal findings from para-Hisian pacing were further validated by recognizing the notable changes in the electrogram morphology and satisfactorily reconfirmed following administration of IV adenosine, which demonstrated the clear presence of a second accessory pathway in our patient. By this approach, a modified para-Hisian pacing maneuver yielded the expected “classical” response that is normally seen in patients with retrogradely conducting accessory pathways. Moreover, administration of adenosine proved highly valuable during ablation of the second accessory pathway by allowing for more precise mapping and ablation of the earliest atrial activation site, through pharmacologic suppression of the retrograde AV nodal inputs.

In summary, change in the electrogram morphology noted during para-Hisian pacing is a valuable marker and may suggest the presence of multiple conduction pathways. In rare instances when the retrograde accessory pathway conduction time is longer than that of the AV node, assessment of electrogram morphology coupled with modified para-Hisian pacing performed immediately following administration of IV adenosine can be effectively utilized to diagnose and unmask accessory pathway conduction by the AV node.

References