INNOVATIVE COLLECTIONS

COMPLEX CASE STUDY

Intra-atrial Conduction Variability during Supraventricular Tachycardia Mimicking Orthodromic Reciprocating Tachycardia Involving Multiple Accessory Pathways

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ABSTRACT. A 24-year-old man underwent electrophysiology testing for evaluation of recurrent tachycardia. A supraventricular tachycardia was initiated with ventricular pacing. Mapping and the response to ventricular pacing entrainment confirmed orthodromic reciprocating tachycardia involving a left-sided accessory pathway. The tachycardia had a regular pattern of VA interval and cycle length variability associated with a change in the atrial activation sequence, indicating that a second accessory pathway was likely present. Mapping and ablation however documented only a single accessory pathway. Post-ablation atrial pacing at the ablation site demonstrated that the findings during tachycardia were the result of intra-atrial conduction delay or block between the left and right atrium.

KEYWORDS. intra-atrial conduction, orthodromic reciprocating tachycardia, PAROXYSMAL supraventricular tachycardia.

Case Report

A 24-year-old man was referred to the electrophysiology laboratory for evaluation of recurrent tachycardia. The baseline electrophysiology study revealed sinus cycle length, AH and HV intervals of 600 ms, 60 ms, and 40 ms respectively and no evidence of ventricular pre-excitation. A supraventricular tachycardia (SVT) was induced with ventricular pacing (Figure 1). A coronary sinus catheter was unable to be placed. During tachycardia, oscillations in VA timing (150 and 120 ms) and tachycardia cycle length (TCL) (315 and 295 ms) occurred in a regular pattern, with the VA interval and TCL shortening on every third beat. The shorter TCL was always preceded by shortening of the VA interval measured on the His bundle catheter. During changes in VA interval timing it was noted that there were also changes in the atrial activation pattern. With the long His VA interval high right atrium (HRA) activation occurred before the His A, and with the short His VA interval the His A occurred before HRA activation (Figure 2). Ventricular pacing at a cycle length of 260 ms during tachycardia resulted in entrainment of the tachycardia. There was an atrial–ventricular response after pacing with a post-pacing interval (PPI) of 470 ms and a corrected PPI–TCL interval difference of 135 ms. The atrial–ventricular response after ventricular pacing entrainment ruled out an atrial tachycardia and the fact that HRA activation at times preceded His atrial activation is inconsistent with AV node re-entry (AVNRT); therefore, by exclusion orthodromic reciprocating tachycardia (ORT) was felt to be the most likely diagnosis. The long corrected PPI–TCL interval after ventricular pacing entrainment during ORT effectively ruled out a septal or right-sided accessory pathway (AP) and is most consistent with involvement of a left lateral AP. The fact that there was variability in the VA interval associated with a change in the atrial activation pattern and TCL during ORT was felt to be strong evidence for the presence of a second AP, although the existence of a
dual tachycardia (i.e. ORT and AVNRT) could not be completely excluded.

Mapping of the mitral annulus via a transseptal approach demonstrated a single site of earliest atrial activation along the anterolateral annulus with a short and fixed VA conduction time (Figure 2). A single radiofrequency application at this site resulted in accessory pathway block and tachycardia termination. Post-ablation ventricular pacing demonstrated VA dissociation confirming the presence of only a single AP. Atrial pacing at close to the TCL interval (300 ms) along the mitral annulus at the site of ablation demonstrated variability of the SA interval at the His (60–100 ms), with changes in the right atrial activation sequence replicating what was seen during tachycardia (Figure 3).

Discussion

This case demonstrates that the presence of unrecognized intra-atrial conduction variability can lead to diagnostic confusion when attempting to characterize a SVT. The difficulty is due to the fact that the presence of intra-atrial conduction variability invalidates many of the rules that are used to confirm a tachycardia mechanism. The behavior of a tachycardia during periods of TCL variability has been shown to be an important clue to the mechanism of a tachycardia. TCL variability of 15 ms or more has been demonstrated to occur in approximately 75% of cases of paroxysmal SVT induced in the electrophysiology laboratory. When changes in the atrial cycle length precede changes in the ventricular cycle length, as was seen in this case, this has been shown to be indicative of either an atrial tachycardia or atypical AVNRT with a reported sensitivity and specificity of 83% and 100% respectively. Both of these mechanisms as the sole cause of the tachycardia however were eliminated based on the response to ventricular pacing entrainment and the atrial activation sequence during portions of the tachycardia. Thus by exclusion ORT had to account for at least a portion of the tachycardia despite the strong evidence against this as the mechanism based on the criteria listed above.

After a diagnosis of ORT was confirmed the atrial timing changes were felt to be best explained by the presence of a second accessory pathway. When variability in the VA time is associated with a change in the atrial activation sequence during ORT, this is felt to be
nearly pathognomonic for participation of a second AP. The reason for this is that during ORT, AP, and intra-atrial conduction are usually fixed and therefore any change in VA timing associated with a change in the atrial activation sequence indicates that the site of initial atrial activation has shifted. If the change in atrial timing and activation sequence is associated with a change in the TCL, this implies that the second AP participates in the tachycardia and is not just a bystander AP. In this case however mapping and ablation demonstrated only a single AP. Post-ablation pacing along the mitral annulus replicated the atrial timing changes seen during tachycardia, confirming that intra-atrial conduction variability was the cause of these changes.

Left-to-right atrial conduction has been demonstrated to occur preferentially at certain areas along the atrial septum. These areas include the region of Bachman’s bundle, the fossa ovalis, musculature of the coronary sinus ostium and some posterior intercaval connections. During a tachycardia with left-to-right atrial activation, conduction breakthrough to the right atrium can occur at one area alone, or may occur nearly simultaneously at multiple areas. The area of earliest right atrial activation is primarily determined by the location of the earliest left atrial activation since different left atrial sites are associated with preferential patterns of left-to-right atrial conduction. In our patient, the VA interval on the HRA recording was fixed, indicating that there was stable and persistent conduction at the level of Bachman’s bundle during tachycardia. The shortening of the VA interval on the His bundle recording was most likely the result of intermittent conduction across either the coronary sinus ostium or fossa ovalis. The fact that the TCL shortened along with shortening of the VA interval on the His recording indicates that the net effect of this intermittent conduction breakthrough was to effectively reduce the size of the tachycardia circuit.

In summary, this case demonstrates that variations in intra-atrial conduction during ORT involving a left-sided AP can affect measured conduction times and patterns of atrial activation in the right atrium. This conduction variability can lead to diagnostic confusion regarding the tachycardia mechanism and can mimic the involvement of multiple APs during ORT. In this case, variability in left-to-right atrial conduction occurred in a patient without apparent heart disease or evidence of intra-atrial conduction delay, indicating that this phenomenon needs to be considered in the differential in anyone who

Figure 2: Tachycardia mapping along the anterolateral mitral annulus. The surface and intracardiac recordings shown are the same as in Figure 1 with the addition of the ablation catheter distal electrode pair (ABd). The ablation catheter had to be positioned close to the ventricle along the mitral annulus to allow for catheter stability at the site of the earliest atrial activation. This location resulted in large ventricular (V) and small atrial (A) electrogram recordings during tachycardia. The timing on the ablation catheter demonstrates continuous electrical activity between the ventricular and atrial electrograms. Note, that despite a fixed VA interval on the ablation catheter there is still variability in the VA interval on the His recordings and variability in the tachycardia cycle length (TCL). With the long His VA interval the onset of the high right atrium (HRA) activation occurred before the onset of the His A (first dashed line) and with the short His VA interval the HRA activation was later than the His A (second dashed line). A single radiofrequency application at this ablation site terminated the tachycardia and eliminated VA conduction.
displays variable VA timing during ORT. We feel that in this case it was simply the AP pathway location which facilitated conduction breakthrough at more than one right atrial site rather than any true abnormality of intra-atrial conduction. A firm understanding of the possible variations of left-to-right atrial conduction can help avoid some of this diagnostic confusion and streamline the mapping and ablation process.

References


Figure 3. This figure demonstrates the effects of pacing in the left atrium at the ablation site. The figured is labeled as in Figure 2 with the addition of the proximal ablation electrode pair (ABp). Pacing at close to the tachycardia cycle length (TCL) (300 ms) resulted in a variable stimulus to atrial (S-A) response on the His recordings (60–100 ms) with changes in the right atrial activation sequence replicating the atrial timing changes seen during tachycardia.