Case Presentation No. 1

A 52-year-old man was referred for recurrent palpitations after 2 prior electrophysiology studies and ablations at an outside institution targeting right anteroseptal and left posterolateral accessory pathways (AP) for orthodromic reentrant tachycardia (ORT). Baseline ECG revealed sinus rhythm without preexcitation and an incomplete right bundle branch block. An ECG from his most recent tachycardia episode demonstrated a long RP tachycardia, with an incomplete right bundle branch block pattern at a rate of 135 beats per minute. Prior echocardiogram showed no evidence of structural heart disease.

At electrophysiology study, the baseline atrial-His (AH) and His-ventricular (HV) intervals were both 58 ms. There was no preexcitation with incremental atrial pacing. There were spontaneous echo beats, which had a ventriculoatrial (VA) interval of 230 ms with concentric-appearing retrograde activation. Tachycardia with a cycle length (CL) of 460 ms was reproducibly initiated with programmed ventricular stimulation after a retrograde VA jump. Entrainment from the right ventricle (RV; mid-septum) showed a V-A-V response and a postpacing interval (PPI) of 583 ms without significant AH prolongation (Figure 1). Stim-A minus V-A time was 97 ms. Premature ventricular complexes (PVCs) from the septum during tachycardia advanced the atrium only if they were delivered ≥ 45 ms before the His deflection. Differential pacing was performed with entrainment from the RV apex yielding a PPI of 605 ms, whereas RV basal pacing resulted in a PPI of 601 ms. PPI from the proximal coronary sinus (CS) was markedly prolonged (>50 ms over tachycardia CL) and was even longer from the mid CS (Figure 2). Mapping of the annular septum showed earliest retrograde activation at the mid right atrial (RA) septum. PPI with entrainment from here was 20 ms >tachycardia CL. What is the mechanism of the tachycardia, and what is the appropriate next step?

Discussion

Initial electrophysiology study demonstrated no evidence of preexcitation at baseline or with incremental pacing, excluding antegrade AP conduction. The tachycardia demonstrated a long VA time, with a V-A-V entrainment response consistent with atypical atrioventricular nodal reentrant tachycardia (AVNRT) versus ORT. The tachycardia initiation with a
retrograde jump during ventricular pacing, as well as with ventricular extrastimuli, and the V-A-V response to entrainment effectively excluded atrial tachycardia.

The patient had prior reported radiofrequency ablation of a right anteroseptal AP, as well as a left posterolateral AP. The retrograde activation sequence showing proximal-to-distal CS activation and activation of both the His and RA before the CS was not consistent with recurrent conduction over the left posterolateral pathway. Recurrence of the right septal pathway might be possible; however, the findings are inconsistent.

Entrainment from the RV resulted in a relatively prolonged PPI and stimulus-atrial-V A, potentially suggesting atypical A VNRT. The atrium did not advance with His synchronous PVCs and advanced only with PVCs that were 45 ms before the His and 103 ms before ventricular activation. This also reset the tachycardia. Atrial advancement occurred without advancement of the His potential from the PVC. These findings would not be expected with atypical AVNRT. ORT using a septal pathway would also be less likely because septal pathways reset with PVCs timed at <75 ms prematurity relative to the tachycardia CL. Para-Hisian pacing was performed at rates where the retrograde fast atroventricular node did not conduct well, producing a long VA interval and atrial sequence as in tachycardia. This was then repeated during supraventricular tachycardia (ie, para-Hisian entrainment) and in both cases was consistent with an extranodal response suggestive of AP conduction. The His catheter atrial electrograms did not clearly occur before the P wave, and the P-wave morphology (although confounded by the terminal T wave) was not clearly consistent with AVNRT. In addition, the minimal difference in PPI with entrainment from the RV base versus apex would also not be expected with atypical AVNRT, and reports vary on whether it is consistent with AVNRT using a septal pathway.

Detailed RA septum mapping showed earlier activation in the mid RA septum at least a centimeter away from the annulus with the PPI equal to the tachycardia CL. A hint of this could be seen on the retrograde His, in which the atrial electrogram on the proximal His is slightly before the distal His activation. The combination of these findings all suggested ORT using a left-sided pathway and the need for further mapping of the left side.

**Figure 2.** Markedly prolonged postpacing interval after entrainment from the mid-coronary sinus.

**Figure 3.** Electrograms demonstrating early fractionated atrial activation at anterolateral left atrial annulus (arrow). Although tachycardia termination occurred due to a blocked premature atrial complex several seconds into the radiofrequency lesion (not shown), the pathway was eliminated and tachycardia was no longer inducible.
atrium, despite the proximal-to-distal delayed CS activation sequence and long PPIs from the CS.

After transseptal puncture, earlier atrial electrograms and shorter VA times were found at the anterolateral mitral annulus (Figures 3 and 4), significantly preceding those on the His catheter. Entrainment from the atrial and ventricular sides at the earliest atrial deflection demonstrated that the PPI was equal to the tachycardia CL, confirming ORT using a left anterolateral pathway. The atrial and ventricular signals at this site were fractionated, possibly related to the prior ablation. Ablation of the atrial insertion eliminated tachycardia and retrograde AP conduction.

**Case Presentation No. 2**

A 20-year-old woman with a long history of palpitations presented with a left bundle branch block tachycardia that terminated with adenosine. Baseline ECG showed a narrow QRS without preexcitation. The ejection fraction was 60% as shown by echocardiogram. During the electrophysiology study, baseline AH and HV intervals were 88 ms and 35 ms, respectively. There was no change in the HV interval with incremental atrial pacing. Retrograde atrial activation with ventricular pacing was earliest at the posterolateral mitral annulus. Tachycardia was consistent with ORT, with the earliest retrograde atrial activation bracketed between CS 5,6 and CS 3,4 with an HA time of 155 ms (Figure 5). Ablation was performed during ventricular pacing and directed at the posterolateral mitral annulus. After ablation, retrograde atrial activation with ventricular pacing became earliest at the His followed by proximal-to-distal CS activation, consistent with the elimination of the pathway. However, tachycardia with a left bundle branch block morphology was again induced, and similar to ventricular pacing, retrograde activation was now earliest at the His. The HA timing in the posterior CS was 266 ms, and the posterior CS timing was later than the most distal and proximal CS recordings. Repetitive attempts at ventricular entrainment terminated the tachycardia (Figure 6). Based on the above findings, what is the mechanism of the tachycardia?

**Discussion**

The lack of baseline preexcitation, along with the inability to demonstrate HV changes with atrial pacing, excluded antegrade conduction over an AP. The initial presence of a left posterolateral AP was confirmed by the retrograde atrial activation that was bracketed between CS 5,6 and 3,4 in the posterior CS. After ablation of the left posterolateral AP, retrograde atrial activation with ventricular pacing was earliest at the His and latest in the posterior CS. A left bundle branch block morphology tachycardia was induced with retrograde activation similar to ventricular pacing that was earliest at the His, with an HA interval of 266 ms in the posterior CS. With the CS catheter at the CS ostium, retrograde activation remained earliest at the His, rendering both a posteroseptal or left posterolateral AP and retrograde slow pathways unlikely. Repetitive attempts at ventricular entrainment terminated the tachycardia without affecting the atrial activation, excluding an atrial tachycardia. Although there was no advancement of the atrial electrogram during the transition zone (V pacing with fusion during tachycardia), tachycardia termination because of retrograde block occurred at the end of this zone, likely with the first completely captured beat. Careful inspection of the transition zone (Figure 6) shows that the atrial electrogram was not perturbed without capture of much of the left ventricle with RV pacing (on the third beat), but only terminated tachycardia when the
distal CS ventricular electrograms were also advanced (fourth beat). Tachycardia termination with the first fully captured beat (or late in the transition zone) is indicative of an AP.5–7 The requirement to capture the lateral left ventricle (advancing the distal and not just proximal CS) to affect the tachycardia should suggest the presence of a left lateral pathway, despite the atrial activation sequence on the CS catheter.

A deflectable decapolar catheter was then inserted beyond the existing CS catheter, with the distal tip near the anterior interventricular vein–great cardiac vein junction. Earliest retrograde atrial activation (in tachycardia and with RV pacing) was then found to be at the anterolateral mitral annulus just inferior to the base of the left atrial appendage (Figure 7). The late appearing CS electrograms posteriorly, which activated from proximal to lateral, were related to mitral isthmus block from the earlier ablation of the posterolateral pathway, which then masked the presence of the anterolateral pathway. Ablation was performed at that site during ventricular pacing, and loss of VA conduction was seen with ablation of the pathway. No retrograde nodal activation was present at baseline or after ablation. No further tachycardias were induced.

Summary

These cases display that the presence of a left-sided pathway cannot be excluded based on concentric-appearing retrograde CS activation only and illustrate the potential benefit of more distal evaluation of the CS when possible, particularly after prior ablation in the mitral isthmus region, which may result in significant conduction delay or block. In both cases, delayed and concentric-appearing retrograde activation in the CS, despite the participation of an anterolateral mitral annulus AP, was related to mitral isthmus block from the prior left posterolateral AP ablation.

The atrial activation pattern was related to counterclockwise conduction along the mitral annulus from the insertion site, with resultant proximal-to-distal CS activation after the septal atrial electrograms, initially suggesting a possible septal pathway. If distal CS catheter placement cannot be obtained, PPI mapping in the right atrium may provide a clue for the presence of a left-sided AP before transseptal puncture, but should be interpreted cautiously, because the right-sided breakthrough may be within the macroreentrant circuit. Furthermore, careful examination of the transition...
zone during ventricular entrainment is useful for the diagnosis of an AP.

Disclosures
None.

References

Key words: catheter, ablation ▪ arrhythmias, cardiac ▪ tachycardia, supraventricular

EDITOR'S PERSPECTIVE
In this issue’s submission for the “Teaching Rounds in Cardiac Electrophysiology” series, Wright et al present instructive examples of supraventricular tachycardia where the activation sequence in the coronary sinus could have been misinterpreted and led the unwary away from the correct diagnosis. In addition to the excellent tips provided by the authors, the cases illustrate some general concepts of educational value, the first being that eccentric activation can occur in the coronary sinus despite earliest activation site being on the septum.

AV Nodal Reentrant Tachycardia (AVNRT)
Many electrophysiologists fall prey to the trap of misdiagnosing AV node reentry as a freewall accessory pathway based on the coronary sinus (CS) activation sequence. In AVNRT, retrograde activation is either through the fast AV nodal pathway or a slow pathway. Each of these involves early activation, either on the true interatrial septum (fast pathway) or the proximal coronary sinus (right or left slow pathway). When the fast pathway region is activated, conduction typically occurs from proximal to distal in the coronary sinus. The fast pathway is located behind the tendon of Todaro, and activation can proceed across the tendon and Eustachian ridge to activate the proximal coronary sinus musculature and produce this sequence. In some cases, however, the Eustachian ridge and tendon represent a line of block and delay, and the wavefront will then need to propagate around the Eustachian ridge between it and the crista terminalis to activate the CS, but this still produces the familiar concentric activation sequence.

In a few patients, however, the Eustachian ridge and crista terminalis are confluent and represent a second line of block. Now conduction needs to proceed to the left atrium in order to complete the AVNRT circuit. Once the left atrium is required, the vagaries of left atrial activation, as well as the site and number of left atrium to CS connections, will determine the activation sequence in the CS. For instance, left atrial activation may cause course superior to the posterior wall along Bachmann’s bundle and then enter the coronary sinus via a relatively distal LA-CS connection and produce distal to proximal activation of the CS.

The key finding, however, is that a recording catheter placed at the fast pathway (just behind the tendon of Todaro in the right atrium) will record earlier activation than the earliest electrogram in the CS. Thus, although the CS activation is eccentric, when all electrograms are considered, the true early site of atrial activation is on the septum.

Accessory Pathways
Theoretically retrograde conduction over a septal accessory pathway could produce similarly misleading retrograde atrial activation sequence. A more common difficulty with accessory pathways, however, is distinguishing AVNRT with an eccentric activation sequence from AVNRT with a bystander left freewall accessory pathway. The key finding is to note whether the atrial activation sequence changes when PVCs during tachycardia advance all or some of the atrial electrograms. If the sequence does not change, there is a single mechanism for atrial activation (only AVNRT or only a septal pathway, etc.). On the other hand, if the sequence changes, fusion is present and by analyzing which electrogram is advanced and whether or not the His bundle electrogram is advanced retrogradely or anterogradely, the mechanism of tachycardia and the presence of a bystander can be determined.4

Misleading Concentric Coronary Sinus Activation
As also pointed out by Wright et al,1 a left-sided accessory pathway may present with concentric coronary sinus activation.5 If the accessory pathway is located on the lateral mitral annulus ablation inferior to the pathway between the annulus and the left inferior pulmonary vein may result in block across the left inferior pulmonary vein-mitral annular isthmus. Then, activation from the early left lateral site proceeds in a counterclockwise direction around the anterior superior annulus and results in proximal to distal coronary sinus activation. Here again, the key is to find the true site of early activation. Maneuvers to prove that the tachycardia is still due to AV reentry and careful mapping lateral to the ablation area will reveal the appropriate site to target for ablation.

The Coronary Sinus Is Not the Left Atrium
The coronary sinus is a cardiac chamber in its own right, with electrically active myocardium and venous components similar to the right or left atria. Although the coronary sinus catheter is used as a surrogate for left atrial activation, in reality it records electrograms from the left atrium as well as the coronary sinus myocardium itself. In some patients, distinct and limited myocardial connections between the CS and LA exist.6 If these connections are ablated, then the CS catheter no longer serves as a reliable surrogate for left atrial activation. This can cause misleading activation sequences for accessory pathways as well as some focal atrial tachycardias. In it can also complicate assessment of mitral isthmus block following ablation.7 Assessing the CS electrograms alone may suggest block when in fact endocardial conduction in the left atrium is actually present.

Circ Arrhythm Electrophysiol June 2013
Similarly, CS-based ablation either for an accessory pathway or an atrial tachycardia may produce misleading activation patterns within the CS that are not entirely reflective of endocardial left atrial activation.

**Site of Stimulation, Number of Electrodes, and Spacing of Electrodes: Getting it Right**

Clarity is even harder to achieve in complex cases when there are a limited number of electrodes, widely-spaced multielectrode catheters are used, or when care is not taken to determine the best pacing and recording sites for executing the diagnostic maneuver. For example, Wright et al note, “PVCs from the septum during tachycardia advanced the atrium only if they were delivered ≥45 ms before the His deflection.” While assessment of PVC timing in relation to the effect on tachycardia can be useful, it is not absolutely reliable because of the idiosyncrasies of intraventricular conduction and the possibility of slow conduction in an accessory pathway that may have been damaged by ablation. If, however, the PVCs are placed from the base relatively close to the His bundle, then the retrograde His deflection can usually be visualized allowing assessment of the relative advancement of the retrograde His and atrium to yield the true diagnosis. For instance, if retrograde His is advanced by 10 ms but the retrograde A is advanced to a greater degree, by 50 ms, then an accessory pathway is present.

Not only is it important to have electrodes mapping key sites, such as the true fast pathway, the typical sites of early activation of the retrograde right and left slow pathways, and the anterolateral region of the mitral annulus, an appreciation of pitfalls in interpreting the activation sequence is required to avoid a wrong diagnosis. The authors note, “A hint of this could be seen on the retrograde His, in which the atrial electrogram on the proximal His is slightly before the distal His activation.” Identifying the pattern of activation of the His bundle electrograms or the atrial electrograms on the His catheter can be very difficult. In general, ventricular activation on the His bundle catheter during sinus rhythm shows earlier ventricular electrograms from the distal His than the proximal His bundle region. This is because the His bundle itself is insulated, and initial ventricular activation occurs closer to the apex from where it proceeds towards the base of the heart. Similarly, retrograde atrial activation from the AV nodal fast pathway is earlier behind the tendon of Todaro (proximal His) than in from of the tendon (where the distal or mid electrodes of the His catheter are typically located). With retrograde conduction over a septal accessory pathway, however, the annular atrial myocardium will be activated before the more proximal septum that is further from the annulus, behind the tendon of Todaro. However, to meaningfully interpret or use these constructs, closely spaced electrodes and utmost care in placing the catheter perpendicular to the axis of the annulus is required.

**Conclusion**

The cases presented by Wright et al instructively draw our attention to the need of careful and nuanced interpretation of retrograde atrial activation sequences, in general, and more particularly in the coronary sinus region to correctly identify the presence and location of accessory pathways, particularly after ablation has altered conduction in the region.

**References**

Two Cases of Supraventricular Tachycardia After Accessory Pathway Ablation
Jennifer M. Wright, Dalip Singh, Adam Price and Peter A. Santucci

Circ Arrhythm Electrophysiol. 2013;6:e26-e31
doi: 10.1161/CIRCEP.113.000268

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circep.ahajournals.org/content/6/3/e26