Teaching Rounds in Cardiac Electrophysiology

Biatrial Tachycardia
Distinguishing Between Active and Passive Activation

James E. Ip, MD; Jim W. Cheung, MD; Christopher F. Liu, MD; George Thomas, MD; Steven M. Markowitz, MD; Bruce B. Lerman, MD

The approach to successful ablation of arrhythmias is based on elucidating the underlying mechanism and relative dimensions of the tachycardia circuit. Activation mapping readily distinguishes macroreentry from focal arrhythmias, the latter of which have centrifugal spread. When macroreentrant atrial tachycardia is suspected, entrainment maneuvers with atrial overdrive pacing at various anatomic sites help differentiate right-sided from left-sided circuits. Based on the difference between the postpacing interval (PPI) and tachycardia cycle length (TCL), the proximity of the pacing site to the reentrant circuit can be assessed. However, even in a reentrant tachycardia, a short PPI–TCL interval (<30 ms) may not always reliably identify whether the pacing site is within the tachycardia circuit, particularly when widely disparate locations seem to be “in the circuit”. In these cases, it is important to return to the fundamental principles of entrainment and to recognize the subtle changes in antidromic and orthodromic wavefronts in response to overdrive pacing that help to correctly identify the tachycardia circuit.

See Editor’s Perspective by Asirvatham and Stevenson

Case Presentation

A 57-year-old man with a history of hypertension, hyperlipidemia, coronary artery disease, rheumatic heart disease, and 5 previous surgeries for mechanical aortic and mitral valve replacements and coronary artery bypass grafting presented with 6 months of worsening dyspnea on exertion that correlated with the onset of persistent atrial tachycardia (Figure 1). In the electrophysiology laboratory, the patient’s clinical atrial TCL was 360 ms. Diagnostic catheters were placed to encircle the tricuspid annulus within the coronary sinus (CS) and at the superior margin of the tricuspid annulus to record the His bundle (Figure 2A). Entrainment mapping demonstrated a short PPI–TCL from the cavo-tricuspid isthmus (CTI; Figure 2B), the proximal CS (Figure 2C), and the superior tricuspid annulus (Figure 2D). Entrainment from the distal CS also showed a PPI–TCL of 0 ms (Figure 2E). Based on the activation sequence during tachycardia and standard entrainment responses that showed a PPI–TCL <30 ms at widely disparate locations in the right atria (RA) and left atria (LA), the circuit of the tachycardia remained indeterminate.

Discussion

Typical atrial flutter is a right atrial macroreentrant tachycardia that funnels the activation wavefront through the CTI. It is diagnosed by activation mapping, demonstrating counterclockwise propagation of reentry around the tricuspid annulus and by entrainment mapping. During entrainment mapping, the return cycle length after cessation of overdrive atrial pacing is typically short (PPI–TCL <30 ms) from locations within or contiguous to the macroreentrant circuit (ie, high RA, CTI, and proximal CS) and long (PPI–TCL >50 ms) from locations distant from the circuit, such as the distal CS. In our case, not only were short PPI–TCL intervals measured from the proximal CS and 2 locations in the RA, but the distal CS also showed a PPI–TCL of 0 ms during multiple atrial overdrive pacing sequences, an unexpected finding with typical atrial flutter. A short PPI–TCL from the distal CS is typically seen with left-sided circuits, particularly mitral annular flutter or roof-dependent flutter. However, such tachycardias are not expected to also have short PPI–TCL from the lateral RA.

One hallmark of entrainment is the observation of fusion (collision of the antidromic pacing wavefront with the orthodromically conducted tachycardia beat) with concurrent reset (the orthodromically conducted pacing beat advances the TCL to the pacing cycle length). Because the typical atrial flutter circuit is confined to the RA and the left atrium is passively activated, CS activation proceeds in a proximal to distal direction. Overdrive pacing from the distal CS during atrial flutter typically results in an antidromic wavefront that activates the CS eccentrically (distal to proximal sequence) and a long PPI–TCL interval after cessation of pacing because the pacing site is remote from the tachycardia circuit (Figure 3).

In the case presented, results from entrainment mapping from the proximal CS, superior tricuspid annulus, and CTI were consistent with typical RA flutter, but the interpretation of the circuit topology was confounded by a short PPI–TCL interval from the distal CS. Therefore, based on our findings, the differential diagnosis includes biatrial tachycardia with dual loop reentry (figure-of-8) around the tricuspid and mitral annuli, biatrial tachycardia with single loop reentry around the tricuspid and mitral annuli, or RA flutter with the left atrium participating as a passive bystander during entrainment.
Additional observations during tachycardia as well as overdrive pacing from the distal and proximal CS serve to elucidate the mechanism. Because CS activation during tachycardia is concentric (proximal to distal), biatrial dual loop reentry with figure-of-8 activation around the annuli is excluded (counterclockwise

Figure 1. Twelve-lead ECG of atrial tachycardia.

Figure 2. Entrainment mapping during tachycardia. A, Catheter positions of intracardiac catheters in left anterior oblique projection. B, Surface leads 1, aVF, and V1 are shown, as well as intracardiac recordings from the right atrium (RA), His bundle (His), and coronary sinus (CS) during overdrive atrial pacing from multiple right and left atrial locations. A entrainment mapping from the cavotricuspid isthmus (CTI). The PPI–TCL was 0 ms. C, Entrainment mapping from the proximal CS. The PPI–TCL was 0 ms. D, Entrainment mapping from the superior tricuspid annulus. The PPI–TCL was 18 ms. E, Entrainment mapping from the distal CS. The PPI–TCL was 0 ms. CSd indicates distal coronary sinus catheter; CSp, proximal coronary sinus; d, distal; p, proximal; PL, posterolateral; PS, posteroseptal; PPI, postpacing interval; RA (PL), posterolateral right atrial catheter; RA (PS), posteroseptal right atrial catheter; and TCL, tachycardia cycle length.
Entrainment of Biatrial Flutter

Figure 3. A, Usual response to entrainment from the distal coronary sinus (CS) during typical atrial flutter (not from patient presented in this study). Overdrive pacing results in fixed fusion along the CS and a long postpacing interval (PPI)–tachycardia cycle length (TCL) of 95 ms. B, Schematic showing entrainment of typical counterclockwise flutter circuit. Entrainment from distal CS leads to long postpacing interval with antidromic activation (blue arrow) of the coronary sinus (distal to proximal) during pacing and orthodromic activation of the coronary sinus (proximal to distal) after cessation of pacing (green arrow). Orange bar represents collision of antidromic (blue arrow) and orthodromic (red arrow) wavefronts during entrainment. CSd indicates distal coronary sinus catheter; CSp, proximal coronary sinus; d, distal; MA, mitral annulus; p, proximal; and TA, tricuspid annulus.

The second observation is that the last entrained beat at the most proximal CS electrogram does not originate from the immediate beat after the pacing stimulus but from the following beat (Figure 4A, asterisk) because that electrogram is accelerated to the pacing cycle length (320 ms). In order for this phenomenon to occur, there has to be delayed interatrial conduction and prolonged refractoriness within the CS. Prolonged conduction to the anterosheetum (detected by the His catheter and proximal poles of the RA catheter) allows the pacing impulse, via retrograde conduction over Bachmann’s bundle, to orthodromically activate the CTI and the proximal CS. In the case of active LA participation (single loop reentry) circuit (Figure 4B) or in RA flutter with the left atrium participating as a bystander with delayed septal activation (Figure 4C). In the latter case, for the PPI to equal the TCL, the activation time from pacing the distal CS to Bachmann’s bundle to the proximal CS and back to the distal CS has to be equivalent to the activation time of the reentrant circuit around the tricuspid annulus. Moreover, this scenario requires the presence of longitudinal dissociation along the septum in the form of a protected channel, which would obviate collision of the antidromic and orthodromic wavefronts, which would otherwise terminate tachycardia. Although such a scenario is unlikely, definitive differentiation between active and passive activation of the left atrium can be made by the presence or absence of biatrial linking during entrainment from the proximal CS during tachycardia.

In the case of biatrial flutter with active participation of the left atrium, the distal CS should be linked to the RA, that is, the activation interval between the left and RA are fixed during tachycardia and during entrainment. This is a similar concept to the maneuver used to determine whether a supraventricular tachycardia is dependent on a fixed ventricular-to-atrial relationship (ventriculoatrial linking). The ventriculoatrial interval of the return cycle after atrial overdrive pacing during supraventricular tachycardia is expected to be within 10 ms of the ventriculoatrial interval during tachycardia in the case of orthodromic reciprocating tachycardia because the timing of atrial activation is dependent on ventricular activation. Similarly, it is informative to compare the interval between distal CS atrial activation (CSd-A) and septal atrial activation (as recorded on the His bundle electrogram [His-A]) during tachycardia and entrainment when pacing from the proximal CS. In the case of active LA participation (single loop reentry), the CSd-A and the His-A are activated in series during tachycardia and should show the same sequential relationship during proximal CS entrainment. Furthermore, because they are linked, the CSd-A to His-A interval during proximal CS overdrive pacing should be similar to that during tachycardia. In contrast, in RA flutter with passive LA participation, the distal CS and right anterosheetum are activated in parallel.
Therefore, although the CS and right anterosheetum are also activated in parallel during CS overdrive pacing, the Csd-A to His-A interval will differ slightly between pacing and tachycardia because the sites are activated independently and are not linked. As shown in Figure 2C, the Csd-A to His-A activation sequence and timing are identical during entrainment and tachycardia (red bars, 135 ms), suggesting that the tachycardia mechanism is a counterclockwise reentry around both tricuspid and mitral annuli with the left atrium participating in a large circuit (single loop reentry). Because of scar (grey rectangle) along the septum. Of note, the CSd-A to His-A interval is much shorter during pacing (135 ms; Figure 2C), thus confirming the diagnosis of biatrial tachycardia because the sites are activated independently and are not linked. During CS pacing, the CSd-A to His-A interval should be greater during tachycardia than during proximal CS pacing. In contrast, in RA flutter with passive LA participation, the Csd-A and His-A are activated in parallel during both tachycardia and proximal CS pacing. Therefore, the Csd-A to His-A interval in tachycardia would be similar to Csd-A to His-A interval during proximal CS pacing. As shown in Figure 5B, the Csd-A to His-A interval is much shorter during pacing than during tachycardia (135 ms; Figure 2C), thus confirming the diagnosis of single loop reentry. Although pacing was performed at a longer cycle length (500 ms) than the tachycardia (360 ms), it is unlikely that this accounted for the large differential in activation time between the Csd-A and His-A (Figure 5B). However, it is important to recognize this potential limitation and to avoid any misinterpretation of this maneuver by pacing at the same cycle length as the tachycardia.

After termination of tachycardia, no arrhythmias were inducible with programmed stimulation. PACing the high RA showed eccentric activation along the CS with significant interatrial delay (Figure 5C). During subsequent mapping, significant scar was observed along the right posterior atrial septum as evidenced by the absence of electrograms along the posteroseptal poles of the RA catheter (Figure 2B through 2E). PACing from the high RA is delayed or blocked in the low septum, leading to preferential conduction over Bachmann’s bundle, resulting in distal to proximal activation of the CS (Figure 5D). This scar
was likely the result of this patient’s 5 previous cardiac surgeries involving right and left atriotomies and provided the myopathic substrate for the electrophysiological phenomena and macro-reentrant biatrial tachycardia observed in our case.

To our knowledge, this is the first reported case of biatrial reentrant tachycardia involving both tricuspid and mitral annuli and a single circuit. Although there has been a recent report of tachycardias involving the left atria and RA after linear ablation from the anterior mitral annulus to the right superior pulmonary veins or to a roof line, these tachycardias were not CTI-dependent.

Although the PPI–TCL is considered a critical interval for determining whether a pacing site is within a reentrant circuit, there are occasionally exceptions to the rule. For example, a long PPI–TCL can be observed with entrainment of typical atrial flutter from the CTI. This may occur if the pacing cycle length is significantly shorter than the TCL and produces rate-dependent slowing of conduction or alterations of conduction pathways. Furthermore, despite a pacing cycle length within 20 ms of the TCL, 18% of patients can have a misleadingly pronged PPI–TCL because of local pacing latency. A PPI that is shorter than the TCL (negative PPI–TCL) after cessation of overdrive pacing can also be observed and be misleading. This can occur because of erroneous measurement (ie, determining PPI when multiple local electrograms are present on the bipolar signal) or pacing that results in capture of far-field tissue across a conduction barrier adjacent to the pacing site, for example, Eustachian ridge.

### Table. Summary of Observations and Maneuvers to Diagnose Biatrial Tachycardia

<table>
<thead>
<tr>
<th>Observations/Maneuvers</th>
<th>Typical Atrial Flutter</th>
<th>Biatrial Tachycardia With Active LA Participation in Figure-of-8 Dual Loop Reentry</th>
<th>Biatrial Tachycardia With LA as Bystander</th>
<th>Biatrial Tachycardia With Active LA Participation in Single Loop Reentry</th>
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<tr>
<td>Entrainment from CTI and CSp showing PPI–TCL &lt;30 ms</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
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<tr>
<td>Entrainment from dCS showing PPI–TCL &lt;30 ms</td>
<td>No</td>
<td>Yes</td>
<td>Possible</td>
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<tr>
<td>CS activation during tachycardia</td>
<td>Concentric</td>
<td>Eccentric</td>
<td>Concentric</td>
<td>Concentric</td>
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<tr>
<td>CSd-A to His-A timing during CSp entrainment vs tachycardia</td>
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<td>Linked</td>
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<tr>
<td>Termination with ablation along CTI</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
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<tr>
<td>(CSd-His A)$<em>{tcl}$/CSd-His A$</em>{pacing}$</td>
<td>$\approx 1$</td>
<td>$&gt;1$</td>
<td>$\approx 1$</td>
<td>$&gt;1$</td>
</tr>
</tbody>
</table>

CSD indicates distal coronary sinus; CSp, proximal coronary sinus; CTI, cavotricuspid isthmus; His, His bundle; LA, left atria; PPI, postpacing interval; SVT, supraventricular tachycardia; and TCL, tachycardia cycle length.

Figure 5. A, Termination of tachycardia during ablation along the cavotricuspid isthmus. B, Pacing proximal coronary sinus (CS) during sinus rhythm shows a much shorter activation interval between the distal CS and right anteroseptum (His-A) because of parallel activation (14 ms) compared with serial activation during tachycardia (135 ms; Figure 2C). C, Pacing the high right atrium revealed eccentric activation along the CS with significant interatrial delay. D, Schematic showing mechanism of eccentric activation of the coronary sinus (distal $\rightarrow$ proximal) is because of presence of septal scar (grey rectangle) and preferential activation of the left atrium over Bachman’s bundle. Blue bars represent areas of conduction block. CSd indicates distal coronary sinus catheter; CSp, proximal coronary sinus; d, distal; and p, proximal.
Returning to the first principles of entrainment and being attentive to the subtle changes in antidromic and orthodromic wavefronts in response to overdrive pacing, as well as to comparisons of conduction intervals during pacing (Table), are essential in elucidating the critical components of a macroreentrant circuit.

Disclosures
None.

References

Key Words: atrial flutter ■ biatrial tachycardia ■ cardiac arrhythmias ■ entrainment ■ supraventricular tachycardia
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