INNOVATIVE COLLECTIONS

COMPLEX CASE STUDY

Differentiation of Anterior Tricuspid Annular Atrial Tachycardia from Slow–Fast AV Node Re-entry: The Confounding Effect of First-Degree AV Nodal Block

SETH J. LESSNER, MD, STEVEN M. MARKOWITZ, JAMES IP, MD, CHRIS LIU, MD, JIM CHEUNG, MD, GEORGE THOMAS, MD and BRUCE B. LERMAN, MD

Department of Medicine, Division of Cardiology, Cornell University Medical Center, New York, NY

KEYWORDS. atrial tachycardia, atrioventricular nodal re-entry, high right atrium, supraventricular tachycardia, ventriculoatrial.

Introduction

Short RP’ supraventricular tachycardia (SVT) is usually due to the slow–fast form of atrioventricular node re-entry (AVNR), atrioventricular (AV) reciprocating tachycardia, or, less commonly, junctional tachycardia. Mechanistic differentiation between these arrhythmias is usually unambiguous; however, atrial tachycardia (AT), in the setting of pre-existing first-degree AV block or dual AV nodal pathways, can also present with a short RP’ tachycardia and pose a diagnostic dilemma in distinguishing it from AVNR tachycardia when the AT is associated with septal atrial activation. Both arrhythmias result in a ventriculoatrial (VA) interval <70 ms and concentric midline atrial activation, and, although, standard electrophysiologic maneuvers and the response to adenosine are usually informative in differentiating between the different subtypes of SVT,1–3 the results are not always unequivocal. Additional maneuvers such as evaluating the VA return interval at multiple atrial pacing cycle lengths4 or comparing ΔVA intervals of the last entrained beat during atrial overdrive pacing from right atrial and coronary sinus pacing sites are often dispositive;5 however, these maneuvers are limited by modest positive and negative predictive values,4 or by an inability to either to entrain the ventricle during atrial overdrive pacing or by reproducible termination of the tachycardia during pacing. Observing whether changes in the A–A interval precede changes in the V–V interval or vice versa can be contributory, but is dependent on the presence of a sufficient degree of cycle length oscillation.6

In this report, we present a diagnostic approach to short RP’ tachycardia with concentric midline atrial activation, in the context of preexisting first-degree AV block.

Case report

A 68-year-old man with a history of aortic stenosis and bioprosthetic aortic valve replacement 6 years earlier, presented with recurrent palpitations for the previous 2 years. His episodes recently increased in frequency despite treatment with β-blockers, leading to numerous emergency room visits where a short RP’ narrow complex tachycardia with rates up to 180 bpm was documented and was similar in morphology to the induced tachycardia (Figure 1a). The arrhythmia reproducibly terminated with adenosine. The baseline 12-lead electrocardiogram showed sinus rhythm without pre-excitation and a prolonged PR interval of 250 ms. The patient was referred to our institution for invasive electrophysiology study and catheter ablation.
The Confounding Effect of First-Degree AV Nodal Block

A.

B.

C.
Three catheters were advanced from the femoral veins to the high right atrium (HRA), His bundle, and right ventricular apex (RVA). A decapolar catheter was inserted transvenously in the right internal jugular vein and advanced into the coronary sinus (CS). Dual AV node physiology was demonstrated with atrial

Figure 1: (a) Twelve-lead electrocardiogram (ECG) of the supraventricular tachycardia (SVT) induced during invasive electrophysiology study. (b) Dual atrioventricular (AV) nodal physiology demonstrated during concurrent isoproterenol infusion. Surface ECG leads I, aVF and V1, are shown, as well as intracardiac electrograms from the high right atrium (HRA), His bundle (HBE), coronary sinus (CS), and right ventricular apex (RVA). Single extrastimuli (S2) are delivered following a drive train of 400 ms from the coronary sinus. As the S2 is shortened from 300 ms to 290 ms, the effective refractory period of the AV nodal fast pathway is reached with resulting “jump” with conduction now down the AV nodal slow pathway. (c) Induction of short RP' tachycardia. Surface and intracardiac electrograms are displayed as denoted above. Tachycardia is induced with atrial extrastimulus from the HRA during isoproterenol infusion. Initiation appears to be based on anterograde conduction delay in the AV node. There is near simultaneous atrial and ventricular activation during tachycardia. Note that the PR interval of the last paced atrial beat is greater than the corresponding RR interval.

Figure 2: (a) Concentric retrograde atrial activation during ventricular pacing. (b) Spontaneous atrioventricular (AV) block during tachycardia. Owing to an unstable His bundle recording, the level of block could not be discerned: intranodal versus infra-Hisian. This finding eliminated AV reciprocating tachycardia (AVRT) as a consideration but was consistent with either atrial tachycardia (AT) or AV node re-entry tachycardia (AVNRT). Surface and intracardiac electrograms are displayed as in Figure 1.
extrastimuli (Figure 1b). Tachycardia was initially non-inducible in the basal state. During isoproterenol infusion, SVT was induced with atrial extrastimuli (cycle length 320–380 ms). Induction appeared to be dependent on a critical prolongation of the AH interval and was associated with a short septal VA interval (<70 ms) and concentric midline atrial activation (Figure 1c). Further support for evidence of anterograde slow AV nodal conduction during tachycardia was a PR/RR interval >1.0 during rapid atrial pacing.7

Pacing from the RVA demonstrated concentric atrial activation (Figure 2a), similar to that observed during tachycardia (Figure 1c). VA block in response to adenosine during concurrent ventricular pacing and isoproterenol infusion suggested the absence of a retrograde concealed accessory pathway. During tachycardia, spontaneous AV block had no effect on atrial cycle length (Figure 2b), confirming the absence of atrial cycle length. Atrial activation, although obscured by ventricular activation during tachycardia when AV concordance was 11, con-

Figure 3: (a) Comparison of AH intervals during tachycardia (upper left) and atrial pacing at tachycardia cycle length (upper right). The AH intervals are nearly identical under both conditions. Surface and intracardiac electrograms are displayed as in previous figures. (b) Ladder diagram showing the relationship between the AH interval during atrial pacing/atrial tachycardia and that observed during AV node re-entry.
firmed midline atrial activation during spontaneous AV block (Figure 2b). These findings narrowed the differential diagnosis to AVNR tachycardia or septal AT. Since ventricular extrastimuli and overdrive pacing failed to penetrate the tachycardia circuit and therefore could not entrain or terminate the arrhythmia, standard diagnostic responses to ventricular stimulation could not be assessed. Atrial overdrive pacing during tachycardia from the HRA and CS ostium was also non-diagnostic, because pacing stimuli failed penetrate the circuit, thereby precluding evaluation of the return VA relationship. Since the tachycardia showed minimal cycle length variability, dependence of the tachycardia on an atrial or AV nodal driver was also not identifiable.

Finally, in order to assess whether there was an upper common upper pathway comprising AV node-like tissue interposed between the atrium and tachycardia circuit, the HRA was paced during sinus rhythm at the tachycardia cycle length. At this stage of the study, the tachycardia was no longer dependent on isoproterenol for induction and the cycle length independent of catecholamine stimulation was 420 ms. The AH intervals during atrial pacing and tachycardia were nearly identical (Figure 3a,b), providing evidence that the tachycardia circuit originated above the upper common pathway and was therefore atrial in origin. Also consistent with this diagnosis is that the Wenckebach cycle length (370 ms) was less than the tachycardia cycle length of 420 ms. Three-dimensional activation mapping was consistent with a focal tachycardia with earliest activation recorded in the anterior/superior region of the tricuspid annulus (Figure 4). A single radiofrequency lesion at the site of earliest activation terminated tachycardia after 4.1 s. SVT was non-inducible with programmed stimulation with and without isoproterenol following a 60-min waiting period.

Discussion
Differentiation between typical AVNR tachycardia and anterior/superior tricuspid annular atrial tachycardia presents a unique set of challenges. Retrograde activation during pacing and tachycardia may be nearly identical; however, the RP’ or VA interval often differentiates between the two arrhythmias, with a long RP’ observed in AT. Nevertheless, in the presence of slow AV nodal conduction, as observed in our patient, this distinction is no longer useful since the RP’ interval is short regardless of the mechanism as is the associated VA interval (<70 ms). Other confounding factors included the presence of dual AV nodal pathways, a PR-RR during atrial pacing, and the impression that tachycardia initiation was dependent on conduction delay within the AV node. Furthermore, the inability to capture the atria during tachycardia with ventricular extrastimuli or ventricular pacing precluded evaluation of a V–A–A–V versus V–A–V response. Other maneu-

Figure 4: (a) Electroanatomical display anatomy of the right atrium in the left anterior oblique (LAO) projection. The site of earliest activation and successful ablation is tagged with an orange dot. Areas where His bundle electrogroms were recorded are tagged in brown and labeled. (b) Surface electrocardiogram leads from leads I, aVF and V1 are displayed with the bipolar electrogram recorded at the site of earliest activation on the superior tricuspid annulus. Atrial and ventricular electrograms are noted on the bipolar recording, consistent with an annular locatio.
vers that should reliably distinguish between the two arrhythmias, like the presence or absence of VA linking, could not be evaluated due to the absence of AV conduct during atrial overdrive pacing. The response to adenosine was also uninformative since adenosine is known to terminate both focal AT and AVNR tachycardia (see Table 1 for complete summary of maneuvers).

In the presence of confounding and contradictory data, the finding that clarified the diagnosis was the comparison of AH intervals during atrial pacing and tachycardia. Appropriate interpretation of this maneuver assumes that anterograde conduction over the AV node occurs over the same pathway during both tachycardia and atrial pacing. In our patient, this was considered likely due to the presence of underlying first-degree AV block. Interpretation of this pacing maneuver also assumes that there is an upper common pathway, superior to the circuit for AV node re-entry. Therefore, during atrial pacing in our patient, the AH interval can be considered to comprise an upper common pathway (UCP) and the AV node (slow pathway/conduction); that is, \( \text{AH}_{\text{PACING}} = \text{UCP} + \text{AV node} \) (slow pathway) (Figure 3b). This is the same course traveled by the impulse during AT. Therefore, pacing at a cycle length similar to AT should result in a similar AH interval. In contrast, during AVNR tachycardia, the UCP is activated retrogradely from the fast pathway at the same time the anterograde slow pathway is activated in parallel. As a result, during AVNR tachycardia, the AH interval reflects a shorter anterograde course (\( \text{AH}_{\text{SVT}} = [-] \) UCP + AV node, slow pathway), and thus should be shorter during tachycardia than atrial pacing. Our finding that the AH intervals during pacing and AT are nearly identical is thus consistent with the diagnosis AT. Under ideal circumstances, the AH interval during atrial pacing would have been determined during tachycardia, coincident with entrainment of the ventricles with atrial pacing. This would have confirmed that comparison of AH intervals during SVT and pacing comprised the same anterograde slow AV nodal pathway. However, our inability to entrain the tachycardia, because of AV block and/or termination, precluded this maneuver. Parenthetically, even if different anterograde AV nodal pathways were used during tachycardia and atrial pacing, that is, a slow pathway and fast pathway, respectively, the AH interval during pacing would have been less than the AH during SVT, findings contrary to our results. Also supportive of the diagnosis of AT is that the cycle length for pacing-induced AV node Wenckebach was less than the tachycardia cycle length, suggesting an absence of an UCP above the tachycardia circuit like in AV node re-entrant tachycardia.9

It is important to reliably distinguish between the two tachycardia mechanisms in order to avoid inappropriate slow pathway ablation and unintended AV block. This case demonstrates an approach for making the correct diagnosis of SVT when neither conventional ventricular nor atrial pacing maneuvers lead to diagnostic tachycardia responses and when atypical tachycardia features are present.

Table 1: Summary of Electrophysiologic Results

<table>
<thead>
<tr>
<th>Feature</th>
<th>AVNR tachycardia</th>
<th>Superior annular AT</th>
<th>Patient findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>PR &gt; RR during AP</td>
<td>+</td>
<td>–</td>
<td>+</td>
</tr>
<tr>
<td>VA&lt;70 ms</td>
<td>+</td>
<td>–</td>
<td>+</td>
</tr>
<tr>
<td>Dual AV nodal pathways</td>
<td>+</td>
<td>–</td>
<td>+</td>
</tr>
<tr>
<td>SVT initiation dependent on critical AH prolongation</td>
<td>+</td>
<td>–</td>
<td>+</td>
</tr>
<tr>
<td>Termination with adenosine</td>
<td>+</td>
<td>+</td>
<td>–</td>
</tr>
<tr>
<td>Concentric retrograde atrial activation during SVT and VP</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Right and left atrial activity dissociated from SVT and V</td>
<td>+</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>SVT initiation from the V with V–A–A–V sequence</td>
<td>-</td>
<td>+</td>
<td>–</td>
</tr>
<tr>
<td>V–A–V response following ventricular entrainment</td>
<td>+</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>V–A–A–V response following ventricular entrainment</td>
<td>-</td>
<td>+</td>
<td>–</td>
</tr>
<tr>
<td>SVT termination with VES without atrial advancement</td>
<td>+</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>AV block without affecting SVT cycle length</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>( \Delta A = A ) interval during SVT precedes ( \Delta V = V )</td>
<td>-</td>
<td>+</td>
<td>No oscillation in SVT cycle length</td>
</tr>
<tr>
<td>VA linking</td>
<td>+</td>
<td>–</td>
<td>Indeterminate due to inability to entrain the V from the atrium</td>
</tr>
<tr>
<td>( \Delta V )A interval during differential atrial pacing &gt;14 ms</td>
<td>–</td>
<td>+</td>
<td>Indeterminate due to inability to entrain the V from the atrium</td>
</tr>
<tr>
<td>( \Delta \text{AH}<em>{\text{AP}} = \text{AH}</em>{\text{SVT}} &lt; 20 ) ms</td>
<td>–</td>
<td>–</td>
<td>+</td>
</tr>
</tbody>
</table>

AP: atrial pacing; VA: ventriculoatrial; SVT: supraventricular tachycardia; VP: ventricular pacing; AV: atrioventricular; VES: ventricular extrastimulus.

*Initiation was coincident with AH prolongation but was not dependent on it.

References

1. Knight BP, Ebinger M, Oral H, et al. Diagnostic value of tachycardia features and pacing maneuvers during paroxysmal...


