Tachycardias that are successfully ablated in the vicinity of the atrioventricular (AV) node are common, and although frequently successfully treated, are among the most difficult arrhythmias to understand in cardiac electrophysiology. Even AV node reentrant tachycardia (AVNRT), despite near universal cure, is complex with the anatomic constructs for typical, atypical forms, left-sided AVNRT, upper common pathway block, and lower common pathway block, not readily apparent. Even less understood arrhythmias in this group are junctional tachycardias and peri–AV nodal “atrial” tachycardias that may or may not be reentrant and may or may not involve AV node–like tissue.

One unique member of this group of peri–AV nodal tachycardias is verapamil and adenosine-sensitive “atrial” tachycardia described to arise from the apex of the triangle of Koch’s and considered reentrant mechanism involving the AV node or AV node–like transitional tissue.

In this issue of the Circulation: Arrhythmia and Electrophysiology, Yamabe et al provide novel insights into the mechanism of this arrhythmia. They analyzed characteristics by placing late-coupled atrial extrastimuli at 9 predetermined locations in the region of the triangle of Koch’s and the coronary sinus ostium and concluded that the AV node, His bundle, and atrial tissue within the triangle of Koch’s are not involved in the tachycardia circuit. The importance of their study far exceeds the frequency with which we encounter this uncommon arrhythmia. Their findings highlight the incredible complexity of the electroanatomy and electrophysiology of the peri–AV nodal region, brings to focus the difficulties in defining mechanism of arrhythmia, and perhaps, most importantly, forces us to ask the question, “What is AVNRT?” It is perhaps easier to gain appreciation of these broader concepts by first acknowledging the difficulties in answering seemingly simpler questions.

Is the Tachycardia Mechanism Reentry?

Entrainment and Reset

Reentry as a mechanism of tachycardia is classically determined with entrainment maneuvers. When pacing at a cycle length shorter than that of tachycardia, continuous resetting of the tachycardia occurs such that on cessation of pacing, the last paced beat is entrained but not fused—the so-called first criterion of entrainment. In this study, the authors studied reset responses by varying the site of late-coupled atrial extrastimulus during a stable tachycardia. If a noncompensatory pause occurred after a captured atrial extrastimulus, they diagnosed reset, then analyzed the ease of reset (lateness of the coupling interval), and then further used the return cycle length and similarity to tachycardia cycle length (TCL) as evidence of pacing location within the tachycardia circuit. There are several limitations with this simple technique. With reset, the next atrial beat may be delayed, as evidenced in several of their examples. When such postexcitation occurs, continuous resetting is difficult to demonstrate, yet, in the only 2 patients that the authors attempted classic entrainment, they shortened the TCL.

Manifest and Concealed

Manifest versus concealed entrainment is frequently used to define where in the tachycardia circuit the pacing catheter is located with concealed entrainment seen when pacing in the slow zone or close to the exit site of tachycardia. Manif est entrainment may be seen at sites not in the tachycardia circuit, as well as sites in the circuit but proximal to the slow zone (entrance site). Further distinction between these latter 2 situations is done by measuring the difference between the return electrogram on the pacing catheter (postpacing interval) at cessation of pacing and the tachycardia cycle length. In this study, the authors used a limited number of electrodes in a minority of their patients to determine whether the activation sequence changed with attempted entrainment. Further, they used, in part, the bipolar electrogram morphology to determine whether activation at a particular site was antidromic or orthodromic to the tachycardia circuit. Whereas unipolar electrode-derived morphology accurately identifies wave front change, closely spaced bipolar electrodes do not do so as reliably.

Mapping the Circuit

When detailed mapping of automatic tachycardias is performed, the time from earliest to latest activation is a small percentage of the TCL; however, with reentry, because there is continuous electric activity throughout the TCL (circuit), the mapped cycle length should approximate the TCL. The authors report data in only 2 of their 10 patients, and in these,
a very small (18% and 25%) percentage of the TCL was mappable. Although this discrepancy is easy to understand if the mechanism of tachycardia was abnormal automaticity or triggered activity, reentry may still be evoked if, as previously reported, a large percentage of the cycle length is taken up by fractionated electrograms that are not included in the activation map. Yamabe et al, however, did not find fractionated electrograms in their defined circuit or site of early activation. Can there be such a significant region of slow conduction that takes up 80% of the TCL without discernible fractionated electrograms? AV nodal tissue with presently used filtering of signals shows no discernible electrogram (normal or fractionated) and is a region of very slow conduction. In another study, fractionated electrograms were not found and a large part of the TCL was also unmappable. However, single extrastimuli from the slow pathway input region to the AV node reset the tachycardia with the site of earliest activation in the region of the fast pathway (similar location to the apex of the triangle of Koch’s, as reported by Yamabe et al).

Mapping the Earliest Site of Activation
With automatic tachycardias including triggered automaticity, the earliest site of activation is critical to map and is the site of successful ablation. With reentry, however, “early” sites are of limited, if any, relevance. There is always an earlier site to any electrogram found during reentrant tachycardia. Perhaps the authors suggest in these patients that early site mapping was relevant and successful for ablation despite reentry being the mechanism of tachycardia because the slow zone could not be mapped and the exit site closest to the slow zone gave the impression of an early electrogram in a situation akin to an automatic tachycardia (breakout site). Why, then, was the neighboring slow zone unmappable? Why were no electrograms (fractionated or otherwise) found at sites adjacent to the exit/early activation site? Perhaps worth noting is that with typical AV node reentry, the site of earliest activation is in the region just behind the tendon of Todaro, close to the apex of the triangle of Koch’s, and represents the atrial myocardial input into the AV node and transitional tissue.

Was the His Bundle/AV Node Part of the Circuit?
Determining whether the His bundle is part of a tachycardia circuit is not straightforward. Ideally, direct His bundle capture with high output pacing clearly capturing the ventricle from near the base such that the retrograde His bundle electrogram is clearly seen to be advanced and then assessing the effect on tachycardia. The authors used pacing to capture the atrium near the His bundle and assessed the reset response. This technique has limitations since the true His bundle is located in the membranous portion of the interventricular septum, and over this site, atrial tissue is not present.

Although His bundle participation in tachycardia is difficult to determine, compact AV nodal participation is nearly impossible to directly determine. This is because there is no definable electrogram with standard mapping attributable to the compact AV node. Therefore, AV node’s or the AV nodal extension’s participation in tachycardia must be inferred. Criteria often used in practice to make this inference include adenosine or verapamil sensitivity, junctional rhythm during successful ablation, and reset (with preexcitation or postexcitation) of the tachycardia when pacing in the slow pathway region (antegrade posterior input during tachycardia) with the exit site located at a distance near the fast pathway in the vicinity of the apex of the triangle of Koch’s (behind the tendon of Todaro).

With the AV Node Nearby, Can We Use Standard Maneuvers to Assess Tachycardia Mechanism?
To identify a region of slow conduction distinct from the AV node, yet in close proximity to the AV node, is a tall order. Pacing in this region may result in reset of the tachycardia only with delay in activation because of the decremental nature of the intervening tissue. Thus, sites may well be within the tachycardia circuit, but late-coupled extrastimuli may find it very difficult to penetrate the circuit when they are “upstream” (antegrade) to the AV node or other decremental AV node-like structures. Further, inducing cycle length variability as the AV node adapts when attempting continuous resetting or entrainment is a major obstacle to using these classic maneuvers in this unique environment. Interpreting data is also difficult. For example, in Figure 5 of the Yamabe article, an extrastimulus delivered in the inferior coronary sinus ostium advances the next His electrogram by 10 ms when the premature beat is 40 ms early and by 20 ms when the premature beat is 90 ms early. It becomes difficult to understand why the AV node located between the inferior coronary sinus ostium and the His bundle did not show decrement in this situation. Further, because the slow pathway inputs to the AV node may join proximal to the compact AV node (lower common pathway), delay to the His may be seen simultaneous with preexcitation of the next atrial electrogram despite this arrhythmia being called AVNRT. Given the vagaries of entrainment and reset being performed in this region, the authors obtained incredibly consistent data with picture-perfect graphs without wobble during the reset maneuvers and near identical return cycle length to tachycardia cycle length with premature beats. To obtain such results in analyzing a complex arrhythmia in this quagmire-like terrain is too good and probably is a testament to the investigators’ care in including only stable periods of tachycardia with little change in autonomic tone and avoiding early coupled premature beats.

Was This an Atrial Tachycardia?
In both the prior publications describing verapamil-sensitive atrial tachycardia and the present study, the authorized operators used several criteria in attempting to establish that this studied arrhythmia was indeed an atrial tachycardia. The use of these multiple criteria and even with these, the limitations noted below are evidence of the extreme difficulty in distinguishing atrial tachycardia aris-
ing from the peri–AV nodal area and AVNRT. Perpetuation of tachycardia was independent of AV block suggesting atrial tachycardia. However, AV node reentry may occur with AV block or dissociation and the level of block being at, above, or below the His bundle region.15–18 The authors noted that retrograde atrial activation sequence with ventricular pacing was different from during tachycardia. The retrograde limb of AVNRT may be different than that seen with ventricular pacing, and AVNRT may occur in the absence of V-A conduction with pacing altogether.20–23 During ventricular pacing, the authors noted some instances of AV dissociation without affecting the TCL. More specifically, it has been observed with ventricular pacing during tachycardia or ventricular extrastimulation during tachycardia that if the retrograde His has been advanced significantly (more than 15 ms) with no change in the tachycardia cycle length, most variants of AVNRT are excluded.24 This finding is not with ventricular pacing altogether.20–23 During ventricular pacing, the authors noted some instances of AV dissociation without affecting the TCL. More specifically, it has been observed with ventricular pacing during tachycardia or ventricular extrastimulation during tachycardia that if the retrograde His has been advanced significantly (more than 15 ms) with no change in the tachycardia cycle length, most variants of AVNRT are excluded.24 This finding is not reported in the present study. Although clearly suggestive of atrial tachycardia, this finding is well described and frequently observed with AVNRT as well. Also noted was that changes in the A-A interval during tachycardia resulted in subsequent changes in the V-V interval. This finding occurs in both atrial tachycardia and AVNRT. More specifically, however, if a change in the H-A interval reliably and reproducibly produces a change in the subsequent A-A interval, AV node reentry was more likely. These intervals were not specifically reported. Finally, the authors noted that with cessation of ventricular pacing, presumably entraining the tachycardia, a V-A-A-V activation sequence resulted. Although highly suggestive of atrial tachycardia, when long delays and decrement via the AV node are seen with ventricular pacing, multiple (more than 2) atrial inputs to the AV node are present, or when retrograde 1:2 ventriculoatrial conduction occurs with pacing, V-A-A-V responses can be seen in AVNRT as well.

Is This Junctional Tachycardia? Another arrhythmia with protean manifestations, evasive of exact diagnosis, and near impossibility to distinguish from some forms of AVNRT is junctional tachycardia.25–28 Junctional tachycardia is a catch-all term that includes such varied tachycardias as automatic and reentrant His bundle tachycardias, automatic compact AV nodal tachycardias, and tachycardias of unknown mechanism arising from the extensions of the AV node frequently ablated in the slow pathway region. Distinguishing between an automatic tachycardia in an extension of the AV node in the fast pathway region from an atrial tachycardia arising from the myocardial input to the AV node at the tendon of Todaro may be impossible except that one may expect verapamil or adenosine sensitivity of the arrhythmia if arising from the horn or transitional region.

Why Did the Ablation Work? In all patients, ablation at the site of earliest activation sometimes with extremely low energy (10 to 15 W) was curative as noted above. The “earliest” site of activation has little specific meaning with reentrant arrhythmia, and with most reentrant arrhythmias, ablation beyond a single application of energy, often linear lesions anchored to electrically inert structures is required for cure. An exception is AVNRT when ablation is performed relatively close to the extensions of the AV node. Finally, in 7 of the 10 patients with successful ablation, accelerated junctional rhythm was noted. Did this play a role in their success with ablation?29 As the authors point out, in some instances, ablation may also have targeted myocardium similar to that ablated from the juxtaposed coronary cusps of the aortic valve.30–32

If It Quacks Like a Duck and Walks Like a Duck . . .

The arrhythmia in the patients described by Yamabe et al7 was sensitive to verapamil and adenosine. The earliest site of activation was in the vicinity of the fast pathway input to the AV node. Reset characteristics did suggest reentry; however, the circuit could not be mapped, and an unexplained silent slow zone had to be postulated. The possibility of AV node–like tissue along the AV annulus playing a role in this arrhythmia was suggested, and with successful ablation, the majority of patients had an accelerated junctional rhythm.33 All these features are also routinely seen in AVNRT. . . But What is the Duck? In prior descriptions, the unique features of verapamil-sensitive atrial tachycardia have been stressed and further highlighted in the present report.7 Although possible and well described, easily demonstrated AV dissociation during arrhythmia and the absence of dual AV nodal physiology is uncommon with AVNRT. What, then, is the defining characteristic that distinguishes peri–AV nodal tachycardias from AVNRT?34,35 The reason for difficulty in accurate delineation and nomenclature of these arrhythmias lies in the fact that despite our great success with ablating AVNRT, we know little about the fundamental anatomy and electrophysiology that demarcates this arrhythmia. The border zone between AVNRT and junctional tachycardia, transitional cell tachycardias, and atrial tachycardias arising very close to the extensions of the AV node are fuzzy at best and perhaps impossible to distinguish.3,4 When there are several features of AVNRT but some do not fit, do we call it another arrhythmia or another variant of AVNRT? Is this a rare duck with striped feathers, or is this a new bird altogether? Does it Matter? Safety and Semantics One may ask, because junctional tachycardia, AVNRT, and peri–nodal AV tachycardias share a large and inscrutable border zone, is it essential to distinguish these arrhythmias in practice? The finding that revolutionized management of AVNRT was that premature beats could reset the tachycardia at significant distances from the AV node at or near the slow pathway inputs to the AV node and its posterior extensions. Thus, with typical AVNRT, although the site of earliest activation is just behind the tendon of Todaro near its apex, ablation is performed in the region of the coronary sinus floor with near universal success and safety. The authors correctly point out the risk to AV conduction damage when ablating at
the apex of the tendon of Todaro, and they used great caution and suggest the use of cryoablation, if in fact some patients with verapamil-sensitive atrial tachycardia have a rare manifestation of AVNRT, ablation may be successful and safe in the slow pathway region. We do not know from this study whether complete slow pathway ablation (anatomic) would have eliminated arrhythmia inducibility in at least some patients.

Summary

Yamabe et al, in both their elegant prior reports and in the present study, draw attention to the unique features of verapamil-sensitive atrial tachycardia. They admirably delve headlong into the complexity of defining arrhythmia mechanism in the AV junction vicinity. In doing so, not only do they enlighten us on this arrhythmia, but they bring into sharp focus our incomplete understanding of reset and entrainment near the AV node, the difficulty with identifying junctional tachycardia, and, most importantly, our lack of basic understanding of AVNRT.

Future studies that may include attempts at slow pathway ablation before targeting the earliest site of activation, complete right atrial, left atrial, coronary sinus, and aortic cusp activation mapping, and entrainment mapping may shed further light on how we can best distinguish these closely related arrhythmias. Before we can make a judgment on whether data presented to us define what is not AVNRT, we need further study to appreciate what is AVNRT.

Disclosures

None.

References


**KEY WORDS:** ablation ■ atrioventricular node ■ tachycardia ■ verapamil-sensitive ■ Editorials
Tachycardia and the AV Nodal Region: Guilt by Association?
Frank C. Chen and Samuel J. Asirvatham

Circ Arrhythm Electrophysiol. 2010;3:2-6
doi: 10.1161/CIRCEP.110.937144
Circulation: Arrhythmia and Electrophysiology is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2010 American Heart Association, Inc. All rights reserved.
Print ISSN: 1941-3149. Online ISSN: 1941-3084

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/3/1/2

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation: Arrhythmia and Electrophysiology can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation: Arrhythmia and Electrophysiology is online at:
http://circep.ahajournals.org//subscriptions/