The initial model of atrioventricular nodal tachycardia (AVNRT) as a small circuit of reentry within the AV node precluded a catheter-based cure for this arrhythmia without the occurrence of AV conduction block. Landmark observations that the atria were part of the circuit of AVNRT and that the arrhythmia could be reset with paced beats placed at sites anatomically distant from the compact AV node set the stage for the present expectation of curative ablation without AV block. Despite this understanding of the AVNRT mechanism and the large accrued operator experience during the past 3 decades when ablating this arrhythmia, AV block still occurs in ≈1% to 2.3% as a complication of ablation. See Article p 739

In this installment of teaching rounds, Chen et al share with us what we can learn from a series of patients with permanent or transient AV block noted during and soon after radiofrequency energy delivery. The authors provide an honest, transparent, and instructive view of their 5 cases.

The fact that we are successful ≈99% of the time without complication suggests that the present methods for maintaining safety, including monitoring junctional rhythm during ablation and relating catheter position to the fluoroscopic anatomic approximation of the AV node location are mostly successful. The student of electrophysiology must, however, be aware of how these precautions can fail us.

Anatomy
Even closely spaced bipolar electrodes placed over the compact AV node are not able to record electrograms from the node itself. Because we cannot map the structure we need to avoid, we must estimate anatomically its location and correlate this information with our standard fluoroscopic views.

Utility
The compact AV node is located within Koch’s triangle, approximately in its mid and central position. Using the His bundle as a surrogate for the superior vertex and the floor of the coronary sinus (CS) as its base, we visualize the mid-septal–located AV node and place our ablation lesions inferior to this approximated site while maintaining contact on the septum and position within the triangle.

Pitfalls
The His bundle recording catheter is often relatively ventricular in position and recording a right bundle electrogram, which may lead the electrophysiologist to overestimate the distance from the ablation catheter inferior to the His catheter to the compact node, with consequent energy delivery closer than anticipated to the compact AV node.

The CS catheter is used as the surrogate to estimate the location of the floor of the CS. The CS itself is a 3-dimensional structure and catheters placed within this vein may be located anywhere from its roof to its floor. For example, an operator used to placing a multielectrode CS catheter from superior access (eg, an internal jugular vein) may be accustomed to these electrodes lying on the CS floor, but when the CS catheter is placed in the femoral vein the electrodes may be pushed up against its roof. In the latter case, using the CS catheter as a guide results in placing lesions higher in the triangle of Koch, and, as pointed out by Chen et al, potentially close to the usual anatomic location of the compact AV node.

Junctional Beats
Because slow pathway ablation is essentially anatomic, real-time assessment of adequacy and completeness of lesion creation can be challenging. The most commonly used marker for the correct ablation location is the presence of a relatively slow stable, junctional rhythm with consistent stable retrograde atrial conduction during radiofrequency application.

Utility
Although the actual mechanism for junctional rhythm and ectopy during slow pathway ablation is unclear, presumably the atrial to slow pathway extension of the AV node is being heated and because retrograde fast pathway conduction remains present, the compact AV node can be assumed to be intact.

Pitfalls
Junctional rhythm or tachycardia will also be present when ablating the fast pathway or the compact AV node. Usually the junctional beats are faster and often irregular and thus alert...
the operator to stop delivering energy. The distinction between safe and unsafe junctional beats is not always straightforward.

Rapid junctional rhythm may have 2:1 block to the atrium and the ventricle, giving the impression of a slower and presumed safer rhythm. Furthermore, if prior ablation was done relatively more superior to a present site of energy delivery, junctional rhythm may not be seen despite adequate slow pathway ablation, and the operator may be inclined to ablate at more superior locations unnecessarily.

During junctional rhythm, the retrograde atrial conduction may change slightly and represent retrograde conduction via another source such as a left slow pathway during inadvertent ablation of the fast pathway or AV node. This may have been the cause for difficulty in case 1 in Chen’s series; however, for junctional beats to conduct retrogradely through the accessory pathway would have required conduction to the ventricle first and an abrupt change in V-A timing for retrograde conduction could have occurred.

Finally, if the fast pathway has already been injured, stable, slow pathway ablation-related junctional beats may be seen and appropriately not trigger cessation of ablation. However, with completion of the lesion set, now complete heart block or junctional beats may remain.

Junctional Beats Absent

Utility

The absence of junctional beats typically tells us that we are ablating away from the compact AV node, and although we may not be certain of success, we are often assured of safety and continue to deliver energy.

Pitfalls

Although it is well recognized, particularly with the linear slow pathway ablation done at the level of the floor of the CS, that junctional beats may be absent and yet the slow pathway inputs to the AV node completely eliminated, Chen et al describe a patient with complete AV block post ablation, yet without junctional beats being seen. Junctional beats may be mistaken for atrial ectopy, particularly if the junctional beats arise from the fast pathway input or the peripheral regions of the AV node. Here, because conduction to the atrium occurs much earlier than eventual conduction to the His bundle and ventricle, the electrophysiologist may diagnose these beats as atrial ectopy and continue ablation with AV block resulting without prior junctional beats. Junctional beats may be mistaken for atrial ectopy, particularly if the junctional beats arise from the fast pathway input or the peripheral regions of the AV node. Here, because conduction to the atrium occurs much earlier than eventual conduction to the His bundle and ventricle, the electrophysiologist may diagnose these beats as atrial ectopy and continue ablation with AV block resulting without prior junctional beats. If the sinus rate is rapid (as in the presence of isoproterenol), it may be difficult to appreciate junctional beats, which may be evident only as an apparent shortening of the P-R and A-H intervals.

Rapid junctional tachycardia sometimes exhibits block to the atrium or ventricle or both. If there is block to the His but conduction to the atrium, it would be difficult to know that highly malignant junctional beats and not benign atrial ectopy is occurring. If there is block of a rapid junctional tachycardia to both A and His, then no junctional beats or His bundle electrograms will be seen and the only clue will be a normally occurring sinus beat has unexpected prolongation of atrioventricular conduction. As Chen et al discuss, pacing the atrium may help if done faster than the rate of the typically noted junctional beats and with close monitoring of atrioventricular conduction. A similar approach is useful when there is no retrograde conduction via the fast pathway or with ventricular pacing, and thus both safe and unsafe junctional beats have no retrograde conduction during ablation.

Occasionally ablating on the floor of the CS may elicit an autonomic response, causing transient vagally mediated AV block. No junctional beats would be expected before this event, but the phenomenon is reversible, and ablation can typically be continued.

Mapping

Unlike most other cardiac arrhythmias, we do not map or ablate the earliest site of atrial activation during AVNRT because this location is often at the fast pathway exit site and ablation here has a high risk of AV block.

Utility

Mapping may be useful with atypical AVNRT and retrograde right or left slow pathway exit to the atrium, in which case the site of earliest atrial activation is at the slow pathway that is used retrogradely in the circuit. Ablation targeting the early A is often successful and obviates the need for a pure anatomic ablation.

Even with an anatomic approach, we do use the electrograms recorded from the ablation catheter, avoiding ablation at sites where a large atrial electrogram is present because this is seen over the compact AV node, which is an atrial structure. Some operators will also map to look for the so-called slow pathway (see below).

Pitfalls

If atypical AVNRT is present and ablation targets the earliest atrial activation using a map-guided targeted ablation approach, caution must still be exercised as slow pathway exit sites can be close to the compact AV node, particularly if the anatomy is distorted by a large CS or if a left retrograde slow pathway is being used. One must still use fluoroscopic imaging and monitor the type of junctional rhythm and retrograde atrial conduction.

Bipolar mapping anterior to the tendon of Todaro typically shows a characteristic electrogram with an early far-field atrial signal followed by a sharper late signal. This is a result of the bipole being in front of the tendon, recording the earlier activation from tissue behind the tendon (far-field signal), near the fast pathway, and then later activation in Koch’s triangle and the proximal CS region, producing the late sharp signal. Successful sites of ablation on the slow pathway often show this type of signal; however, similar electrograms may be found in most regions in Koch’s triangle including or near the compact AV node. Thus, although the presumed slow pathway potential may represent a safe effective site, when used without appropriate cognizance of the relative anatomic landmarks can lead to energy delivery unnecessarily close or on the compact AV node. Furthermore, the atrial electrograms in a large dilated CS may be small, and ablation here near the CS roof could damage the AV node and ablation more anteriorly could potentially disturb the arterial supply to the compact AV node.
Energy Delivery
Chen et al\(^4\) discuss how a steam pop during radiofrequency application may have caused damage distant from the catheter location.

Utility
Most operators avoid irrigated catheters, ablation during tachycardia or isoproterenol infusion, or use of large surface area catheters (eg, 8-mm electrodes) and high power when ablating the slow pathway. Large lesions and unstable contact, as during tachycardia, can simultaneously reduce the likelihood of success and increase the potential for compact AV node ablation.

Cryoablation, not used in this series, is often the energy of choice, particularly in children. However, because of possible higher rates of recurrence, most adult electrophysiologists use radiofrequency energy.

Pitfalls
Any energy source, including cryoablation, or small surface area, nonirrigated radiofrequency ablation can cause AV block if the appropriate precautions are not followed. Furthermore, as pointed out\(^4\) even with solid tip ablation electrodes, cooling from CS flow and possible tricuspid regurgitation can lead to deep lesions and potential steam formation and pops at lower electrode temperatures than might be anticipated for the solid tip ablation electrode.

Assumptions and Needs
In recommending or using any approach that putatively reduces the risk of AV block, we assume that we have made the right diagnosis, the compact AV node is reliably located around the middle of the triangle of Koch. We recognize that in certain congenital heart disease such as ccTGA (congenitally corrected transposition of the great arteries),\(^7,8\) AV node anatomy can vary significantly and include either a second or abnormally placed posterior AV node. We know less about variations in the normal heart.

The primary reason for the remaining uncertainty when we ablate the slow pathway and the continued occurrence of heart block as a complication, despite our best efforts, is a result of our inability to record signals from the AV node itself. Now 40 years from the day when the His bundle electrogram was first recorded,\(^9,10\) and with remarkable technology advances in signal processing and recording, we still are unable to reliably detect what most certainly exists, that is, electric conduction through the AV node. Until such recording is possible, we must continue to assume that in all but the rarest of instances, a thoughtful correlative approach that combines precise anatomic localization, checking to see that our surrogates for the boundaries of triangle of Koch are intact, real-time assessment of the type of junctional rhythm and retrograde conduction, and using appropriate energy sources will minimize the risk of AV block during ablation for AVNRT.

Disclosures
Dr Asirvatham receives no significant honoraria and is a consultant with Abiomed, Atisecure, Biosense Webster, Biotronik, Boston Scientific, Medtronic, Spectranetics, St. Jude, Sanofi-Aventis, Wolters Kluwer, and Elsevier. Dr Stevenson is coholder of a patent on needle ablation that is consigned to Brigham and Women’s Hospital.

References

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Samuel J. Asirvatham and William G. Stevenson

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