Atrioventricular Nodal Reentry
Clinical, Electrophysiological, and Therapeutic Considerations

Masood Akhtar, MD; Mohammad R. Jazayeri, MD; Jasbir Sra, MD; Zalmen Blanck, MD; Sanjay Deshpande, MD; and Anwer Dhala, MD

Background. Atrioventricular (AV) nodal reentry is a relatively common cause of regular, narrow QRS tachycardia. The underlying basis for this arrhythmia is functional (and anatomic) duality of pathways in the region of the AV node, although the exact boundaries of the reentrant circuit have not been convincingly defined. During the more common type of AV nodal reentry (seen in approximately 90% of cases), a slow conducting pathway is used in the anterograde direction, and a fast pathway is operative in the retrograde direction. In the uncommon form, the direction of impulse propagation within the reentrant circuit is reversed. In this article, the clinical, ECG, and electrophysiological features of AV nodal reentry as well as approaches to therapy are discussed.

Methods and Results. Clinical diagnosis may be made from the surface ECG. In the common type of AV nodal reentry, the P wave is obscured by the QRS or may be present in its terminal portion. The P wave in the uncommon form occurs late (i.e., in or after the T wave), producing a pattern of long RP and short PR. Both forms of AV nodal reentry are controllable with various therapeutic modalities. For acute termination, adenosine is probably the ideal agent. Prevention of recurrences can be achieved with several pharmacological agents, including β-blockers, calcium channel blockers, and class Ia, Ic, and III antiarrhythmic agents. Curative therapy is now available with a variety of nonpharmacological methods. However, the most promising therapy at the present time is catheter modification of the AV node by ablation of either the fast or slow pathway, using radiofrequency energy. Ablation of the fast pathway carries a higher risk of second- or third-degree AV block. Slow pathway ablation, by providing a high rate of success and minimal risk of AV block, seems to be a more acceptable initial approach.

Conclusions. AV nodal reentry is a common cause of paroxysmal supraventricular tachycardia, and a precise diagnosis can be made with intracardiac electrophysiological evaluation. Although the arrhythmia responds to a variety of antiarrhythmic agents, curative therapy can now be offered with catheter modification of the AV node using radiofrequency energy. At the time of this writing, it seems that catheter modification of the AV node is rapidly becoming the therapy of initial choice in patients with symptomatic AV nodal reentrant tachycardia requiring treatment. (Circulation 1993;88:282-295)

Key Words: reentry • pharmacology • catheter ablation

Atrioventricular (AV) nodal reentrant tachycardia is a relatively common cause of regular, narrow QRS tachycardia. The existing literature covers reports of this entity under several other terms, including paroxysmal atrial tachycardia, paroxysmal supraventricular tachycardia, and paroxysmal junctional tachycardia. These expressions are either incorrect, such as paroxysmal atrial tachycardia, or nonspecific, because a variety of tachycardia entities could correctly share the same terminology. In this article, specific terms will be used and defined to eliminate any confusion regarding the nature of tachycardia under discussion.

Historical Perspective

The paroxysmal nature of supraventricular tachycardia was recognized as early as 1909 by Lewis. Subsequent to the description of reentry as a mechanism of cardiac arrhythmias by Mines in 1913, Iliescu and Sebastiani postulated that paroxysmal supraventricular tachycardia could be caused by reentry. The AV node as a site for reentry in some forms of paroxysmal supraventricular tachycardia was suggested by Barker et al in 1943. Animal work by Moe et al and Mendez and Moe established functional duality of AV nodal pathways as the basis for AV nodal reentry. Clinical studies using intracardiac recordings and programmed electric stimulation suggested the occurrence of reentrant excitation in humans near the region of the AV node as a cause of paroxysmal supraventricular tachycardia. His bundle recordings allowed Goldreyer and Damato to document that the site of conduction delay was proximal to the His bundle recording site and hence, most likely localized to the area of the AV node. Shortly thereafter, the presence of dual AV nodal pathways was demonstrated in patients with paroxysmal supraventricular tachycardia caused by AV nodal reentrant tachycardia. Elucidation of retrograde conduction characteristics documented the ability of bidirectional conduction of the AV node to sustain this form of reentry.

Clinical Presentation

AV nodal reentrant tachycardia can present at any age, but the usual age at the time of presentation in our
referral center is 43±20 years (range, 9–82 years), and it seems to be more common in women. Depending on age at presentation, concomitant structural heart disease may or may not be present. Our own experience (in managing more than 300 patients with sustained AV nodal tachycardia) does not suggest any particular relation to a specific form of organic heart disease. Its overall prevalence is unknown, but in the absence of ventricular preexcitation, AV nodal reentry is the most common form of regular narrow QRS tachycardia, with the possible exception of atrial flutter. The symptoms are generally mild and are described by patients as rapid heartbeat, palpitations, weakness, dizziness, and presyncope. Some patients with relatively rapid tachycardia, i.e., rates >200 beats per minute, and/or concomitant significant organic heart disease may experience syncope.

**ECG Features**

AV nodal reentrant tachycardia has two main surface ECG patterns. In the so-called common, usual, or typical form of AV nodal reentry seen in the vast majority of patients (approximately 90%), the retrograde P wave occurs in close relation to the QRS complex. The P wave is usually obscured by the QRS complex (Figure 1), but in some cases it may be seen at the terminal part of the QRS, mimicking terminal delay. Even more rare is the onset of retrograde P wave preceding the QRS by a rather short PR interval of <100 msec. In the remaining 10% of the cases, the retrograde P wave occurs late (in or after the T wave), and its polarity is relatively easy to recognize if the onset of the P wave follows the T wave (Figure 2). The P-wave appearance is negative in leads II, III, and aVF. This surface ECG pattern is frequently referred to as the uncommon, unusual, or atypical form of AV nodal reentry and can closely simulate other forms of SVT arising in the AV junction. The reasons for the two distinct ECG appearances of AV nodal reentry and distinction from other types of junctional tachycardias will be presented below.

**Electrophysiology of AV Nodal Reentrant Tachycardia: AV Nodal Reentrant Phenomenon**

It has been customary to visualize AV nodal reentry as a process in which the impulse reciprocates within the region of the AV node. The concept of reentry does require at least two pathways with different conduction and refractory period properties to initiate the process. During sinus rhythm or atrial pacing at slow rates, the electric impulse is expected to reach the His bundle (and the ventricle) preferentially over a faster conducting pathway (Figure 3, panel A). The same impulse is also likely to penetrate the slow pathway anterogradely. However, the effective propagation of impulse to the His bundle through the so-called slow pathway is unlikely when the fast pathway is operative. If a cross-connection exists between the fast pathway and slow pathway in the AV node, retrograde penetration of the slow pathway with subsequent collision of this impulse with oncoming anterograde slow pathway impulse is conceivable. Spontaneous or induced atrial premature beats encounter variable amounts of AV nodal conduction delay, depending on timing of their occurrence. As long as the premature impulse continues to propagate along the fast pathway, the events along the slow pathway remain obscured. A block in the fast pathway shifts the AV nodal propagation to the slow pathway to activate the His bundle, and a jump in AV nodal conduction time (and hence the PR interval) is noted.

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**Figure 1.** Surface ECG in common atrioventricular (AV) nodal reentry. This is a typical 12-lead scalar ECG of the common type of AV nodal reentry. Note the regularity of tachycardia at a rate of 158 beats per minute. No P waves can be identified in any of the leads. However, 1:1 P-to-QRS relation can be suspected because no beat-to-beat variation is seen in the appearance of the ST-T segment. Individual leads are labeled.
(Figure 3, panels B and C). Retrograde penetration and further propagation of impulse can occur along the fast pathway if the conduction delay is sufficient in the anterograde direction to initiate the common type of AV nodal reentry (Figure 3, panel C). Continuous reciprocation from that point onward is a function of recovery of tissue ahead of the reentrant impulse (Figure 3, panel D). In the common or typical type of AV nodal reentry, the slow pathway conducts in the anterograde direction, and when the impulse reaches the lower turnaround point, the conduction times from there to the ventricle (through the His–Purkinje system [HPS], i.e., the HV) and to the atria (via the fast pathway, i.e., the HA) are fairly rapid. It is the difference between these two conduction wave fronts that determines the location of retrograde P wave relative to the QRS complex.

During the uncommon or atypical AV nodal reentry, the direction of impulse propagation is reversed such that the slow pathway is used in the retrograde direction and the fast pathway in the anterograde direction. This results in the onset of P wave relatively late, with a long RP and shorter PR interval on the surface ECG (Figure 2). Because of the slow pathway being used in the retrograde direction, this form of reentry is easiest to set up during ventricular pacing, a method rarely successful in initiating the common variety.11

Intracardiac Electrophysiological Studies
The ability to record the His bundle ECGs and to initiate and terminate the AV nodal reentrant tachycardia has greatly enhanced our understanding of this arrhythmia.16 During intracardiac electrophysiological assessment, pacing studies are performed to replicate the clinical tachycardia, define its precise nature and behavior, and also study the effect of intervention. The typical recordings are at least three surface ECG leads, I, II or aVF, and V1 (more can be used), and intracardiac recordings from the region of the His bundle, right atrium, and at times from the coronary sinus to record the atrial activity primarily around the region of the coronary sinus ostium. Once the recordings are obtained, initiation of the tachycardia is attempted. Programmed stimulation from the right atrium, either with progressive increase in pacing rate (incremental pacing) or during extrastimuli, is used to initiate the arrhythmia.11 Similarly, ventricular pacing studies are done to initiate or terminate the tachycardia, identify the retrograde atrial activation sequence, and also rule out the possibility of atrial tachycardias and accessory pathways. When the arrhythmia cannot be initiated at the baseline, isoproterenol infusion is used to facilitate such an induction.12 In patients with known sustained AV nodal reentrant tachycardia, such maneuvers will succeed in starting the tachycardia in virtually all cases.

The common form of AV nodal reentrant tachycardia as a rule is initiated when a critical degree of AV nodal conduction delay is achieved either by incremental atrial pacing or by delivery of critically timed atrial premature beats (Figure 4). At least two responses may be observed. Many patients will demonstrate a sudden jump in the AV nodal conduction time (atrio-His [AH] interval) at a certain atrial coupling interval (Figure 5, panel B).9,10,17 This finding has been used as a manifestation of dual AV nodal pathway physiology during anterograde propagation of impulses.9,10 The interpretation of these findings is as follows: During sinus rhythm, the atrial impulse preferentially activates the His bundle via the fast pathway. Incremental pacing and/or extrastimulus initially conducts over the fast pathway, with progressive prolongation in the AH interval. When a block is encountered in the fast pathway,
Figure 3. Atrioventricular (AV) nodal reentrant phenomenon. Each panel shows intra-atrial electrogram, surface ECG lead, and schematic representation of intranodal conduction. The two pathways are labeled fast (F) and slow (S). In panel A, during the basic drive beats (S1, A1), the impulse reaches the His bundle (H) via the fast pathway, and concomitant anterograde penetration of slow pathway occurs. However, if the impulse along the fast pathway reaches the lower crossover pathway ahead of the slow pathway impulse, retrograde penetration of the slow pathway is likely. In panel B, a premature beat (S2, A2) blocks in the fast pathway but encounters insufficient delay along the slow pathway to allow recovery of the fast pathway for retrograde propagation. An earlier premature beat depicted in panel C encounters greater conduction delay (note the longer PR interval) and allows more time for recovery of the fast pathway to initiate the AV nodal reentry, and a single atrial echo beat (Ae) occurs. Further anterograde conduction along the slow pathway, if the latter is recovered, could start a sustained reentry (panel D). See text for details. S, stimulus artifact; A, local atrial electrogram.

A sudden prolongation in the AH interval is noted, reflecting a switch of conduction from fast to slow pathway. This phenomenon has been most commonly shown during the atrial extrastimulus method. A shift from fast to slow pathway conduction is usually (but not always) associated with the onset of AV nodal reentry (Figure 5, panel B). This abrupt change in AH may not be seen in the remaining patients with inducible AV nodal reentrant tachycardia (Figure 5, panel A).

Retrograde conduction studies in patients with the common type of AV nodal reentry reveal relatively fast conduction, with the retrograde HA intervals frequently measuring ≤50 msec, although at times they could exceed 100 msec.11 With incremental pacing, a 1:1 ventriculoatrial (VA) response is noted at least up to the cycle length of AV nodal reentrant tachycardia (Figure 4, compare panels A and C). When a change in the VA interval is noted during incremental pacing, it is primarily a result of an increase in the HA component. During ventricular extrastimulus technique, the typical behavior of the retrograde conduction in these patients is that of a progressive increase in the V2A2 interval, but here most of this delay is caused by an increase in the retrograde HPS conduction time (i.e., V2A2), with little or no change in the H2A2 interval (Figure 6). In fact, this behavior is so typical of retrograde conduction over the HPS–AV node axis that a lack of increase in V2A2 at progressively shorter V2V2 should suggest conduction via an accessory pathway, even in the absence of His bundle recording.18 Since one can rarely produce a significant delay and/or block in the AV nodal region during the V2V2 method, induction of common AV nodal reentry is seldom possible. Some increase in the H2A2 can be achieved at shorter paced basic cycle length, because it allows earlier input into the AV node as a result of the decrease in HPS refractoriness.19

When spontaneous or induced changes (by vagal maneuvers or programmed atrial or ventricular stimulation) are noted in the cycle length of tachycardia, they are caused by the alteration in the AH interval, whereas the HA tends to remain fairly constant.11 The spontaneous termination of tachycardia can occur in either the retrograde (H but no A) or anterograde (A but no H) direction, but the latter is more common.
The uncommon form of AV nodal reentry can be initiated with atrial and/or ventricular stimulation (Figure 7). However, as opposed to the common variety, both incremental ventricular pacing and the V,V technique are invariably successful in its induction. During the atrial extrastimulus method, the tachycardia starts with relatively small delays in the AH interval (Figure 7), since the anterograde conduction over the fast pathway must remain intact to initiate the tachycardia with the impulse returning to the atria via the slow pathway. If dual AV nodal physiology is demonstrated in these patients, it is usually during retrograde propagation that a switch from fast to slow pathway may be observed.20,21 This will generally coincide with initiation of uncommon AV nodal reentry. When the retrograde fast pathway conduction is not observed, the tachycardia starts with progressive prolongation in the VA (HA) interval during incremental ventricular pacing and/or progressively shorter V,V intervals.

The pattern of tachycardia is that of a short AH and long HA interval and, hence, the typical surface ECG pattern of long RP. Spontaneous and induced changes in cycle length can be along the anterograde or retrograde direction, but the spontaneous termination is usually in the retrograde direction (H but no A). The location of retrograde atrial deflection depends on many factors (inherent conduction time, autonomic tone, etc.) but is usually within or after the T wave.11

**Anatomic Location of AV Node**

It is important to address the anatomic location of the AV node to fully comprehend the nature of AV nodal reentry, controversy regarding the reentrant circuit, and nonpharmacological approaches to its management. The AV node is located within the triangle of Koch, bounded by the septal leaflet of the tricuspid valve, the tendon of Todoro, and the coronary sinus ostium.22,23 The exact shape, dimensions, and boundaries of the AV node are still not completely clear. However, there appears to be a compact portion in which all of the atrial approaches ultimately converge and from which the AV bundle originates.24 There are fascicles of small nodal fibers that extend into the atrium and interatrial septum (Figure 8). It is unclear whether these nodal projections have definable atrionodular junctures with the atrial approaches.24 At least two atrial approaches seem pertinent to the atrionodal reentrant tachycardia. Anterior/superior approaches provide a more direct and shorter route to the junction of the AV node and the AV bundle. Conversely, the atrial fibers on the inferior part of the node ascend toward the atrionodal junction and provide a longer and more indirect route of conduction.24 Conceivably, the anterior, superior, and inferior nodal projections and the corresponding atrial approaches could participate in the AV nodal reentrant phenomena. A correlation between these morphological observations and physiological behavior needs further clarification. This concern arises in part because these anatomic features have been described in patients without known AV nodal reentrant tachycardia.22,23 It remains to be seen, therefore, whether patients with AV nodal reentry have any different anatomic features.

Although the structure just described, i.e., the compact AV node and its atrial approaches alone, can provide the appropriate substrate for AV nodal reentry, other morphological pathways have been observed in this region of the AV junction. These include atrial fibers inserting in the lower AV node, proximal His, and crest of the ventricular septum and thus partially or
paced conduction should be noted in the genesis of AV nodal reentry, creating reentrant arrhythmias, including atrial fibrillation. Previous workers26 described a direct left atrial to His bundle muscular bridge crossing over the anterior aspect of the interatrial septum. The clinical significance of these connections and their role in the genesis of heart block, including the AV nodal reentry, is uncertain at the present time. Nonetheless, it is important to keep these morphological variants in mind, since it is quite conceivable that some rare forms of AV nodal reentry may use such substrates and create complex electrophysiological patterns.

FIGURE 6. Tracings showing retrograde conduction in patients with atrioventricular (AV) nodal reentry. The basic paced ventricular cycle length (VCL) is 600 msec. At a V1V2 of 360 msec (panel A), the V2A2 measures 115 msec, whereas the V1A1 is 100 msec. When the V1V2 is shortened to 330 msec (panel B), the retrograde His deflection (H2) emerges. It should be noted that most of the V2A2 interval of 190 msec can be accounted for by the retrograde His-Purkinje system conduction time (V2H2=155 msec), whereas the H2A2 measures only 35 msec. Rapid retrograde conduction via the "AV node" is quite typical of patients with common AV nodal reentry. HRA, high right atrium; HB, His bundle.

FIGURE 7. Tracings showing induction of uncommon atrioventricular (AV) nodal reentry. During pacing from the right atrium at a basic cycle length of 700 msec, uncommon AV nodal reentry is initiated. Note the relatively short A2H2 (135 msec) and long H2Ae (290 msec) at the onset and typical RP and PR relation during the tachycardia. Pertinent signals and numbers are labeled. HB, proximal His bundle recording; HBd, distal His bundle recording; HRA, high right atrium.

FIGURE 8. Diagram showing morphology of the human atrioventricular (AV) node. The compact portion of the AV node and approaches to the AV node (X) are depicted. Anatomic landmarks are labeled. LBB, left bundle branch; RBB, right bundle branch; CS, ostium of coronary sinus. Modified with permission.24

Intranodal Versus Partial Extranodal Location of AV Nodal Reentrant Circuits

Extensive experimental and clinical studies have repeatedly revealed that anterograde AV nodal conduction shows decremental properties.5-11 The bulk of this delay can be shown to reside in the so-called N region of the compact AV node.27,28 The atrial area has cells that are considered transitional with some atrial tissue-like properties such as rapid upstroke and conduction. The nodo-His fibers are situated in the lower AV node and also display more rapid conduction. There are, however, no clear anatomic demarcations between these intranodal zones, which otherwise show different functional properties.

Initial clinical studies demonstrated initiation of this clinical arrhythmia with programmed atrial stimulation and also suggested dependence of induction on achievement of a critical degree of AV nodal conduction delays.10 These findings were consistent with the intranodal origin for this interesting form of junctional tachycardia. However, questions were raised in several subsequent publications regarding the true nature of "AV nodal" reentrant pathways. In the following discussion, an attempt is made to delineate some of the reasons for the evolution of our current understanding of the AV nodal reentrant circuits.

A key observation reported by several investigators was that, in patients with common AV nodal reentry, the anterograde pathways demonstrated decremental...
behavior typical of AV node, but in the retrograde direction, the conduction was rapid and seldom exhibited significant conduction delay.\textsuperscript{11,29,30} This raised the possibility of partial or complete bypass of the AV node by some conduction fibers with electrophysiological behavior more typical of accessory pathways. This issue was further complicated by another observation that these so-called retrograde “nodal pathways” showed a depressant effect in response to intravenous procainamide, a drug known to show similar effects on atrial and ventricular myocardium, accessory pathways, and the HPS.\textsuperscript{31} Anterograde AV nodal conduction, conversely, rarely exhibits a depressant effect in response to intravenous procainamide even in patients with AV nodal reentry.\textsuperscript{32}

None of these arguments appeared sufficient to prove or disprove the intranodal versus extranodal nature of the retrograde pathway in patients with common AV nodal reentry, for the following reasons: 1) The rapid retrograde AV nodal conduction in many of these patients did show a progressive increase in conduction time, albeit subtle, but appreciable with rapid ventricular pacing and also in response to ventricular extrastimuli at shorter paced basic cycle lengths, a behavior not typical of accessory pathways.\textsuperscript{11,19} 2) Responses of these retrograde “AV nodal” pathways to antiarrhythmic agents are not drug specific.\textsuperscript{17,32,33} In fact, we have observed depressant behavior of these rapidly conducting retrograde pathways in response to \(\beta\)-blockers (Figure 9), calcium channel blockers, and class I and III antiarrhythmic agents.\textsuperscript{21,32,33} Rapidly conducting accessory pathways, atrial and ventricular myocardium, and HPS seldom show a depressant response to \(\beta\)-blockers or calcium channel blockers.\textsuperscript{34} Therefore, the published data implying the role of partial or complete AV nodal bypass tracts in the common form of AV nodal reentry to date have not been convincing. A different physiological behavior of these pathways in the anterograde and retrograde direction and response to pharmacological agents may be more related to fiber orientation of atrial approaches, geometry of intranodal connections, autonomic tone variations, and perhaps other physiological factors such as anisotropic conduction, current density, and summation and inhibition.\textsuperscript{35–37} To support this viewpoint, one can cite a relatively simple example of patients with ECG-documented AV nodal reentry not inducible in the laboratory and with no demonstrable VA conduction in the unmedicated state.\textsuperscript{38} These patients exhibit a pattern of 1:1 VA conduction after isoproterenol, which is often quite rapid, along with the return of tachycardia inducibility.\textsuperscript{38} This behavior of retrograde conduction is difficult to explain on the basis of anatomic considerations alone.

Nonetheless, intranodal versus partially extranodal origin of so-called AV nodal reentry has intrigued clinical electrophysiologists for some time, and a variety of other arguments have been put forth to suggest intranodal situation of the entire process of reentry. To explain the physiological behavior of common AV nodal reentry being entirely localized to the AV node, the concept of upper and lower common AV nodal pathways (connecting the slow pathway and fast pathway) has been proposed.\textsuperscript{11,39,40} In this model, retrograde impulses can reach the atria only via the upper common pathway, whereas they reach the HPS through the lower common pathway. Since the lower common pathway is less controversial, it will be discussed first.

Evidence suggesting the existence of the lower common pathway in the AV node includes the demonstration that the point of impulse turnaround is proximal to the His bundle recording site. Findings that suggest that this may be the case include 1) a block in the HPS, i.e., bilaterally below the His bundle recording site (Figure 10) without affecting the tachycardia cycle length.\textsuperscript{41} 2) During tachycardia, the interval between the antegrade onset of His bundle deflection and subsequent atrial echo beat is invariably less than the retrograde HA interval during ventricular pacing, particularly when corrected for the rate of tachycardia.\textsuperscript{11} 3) In some cases, transient catheter- and/or pharmacologically induced intranodal block can be demonstrated without interruption of the tachycardia.\textsuperscript{22} 4) Penetration of the His bundle by premature ventricular impulses without affecting the cycle length of AV nodal reentrant tachycardia.\textsuperscript{42} The above findings do indicate nonparticipation of the HPS in the circuit of reentry and tend to suggest
location of turnaround proximal to the His bundle recording site, probably in the distal portion of the AV node.\textsuperscript{11,40-42}

The issue of upper turnaround site is somewhat more complex and difficult to settle.\textsuperscript{39,40,42,43} There is a prevailing belief that an upper common pathway connects the two intranodal pathways of reentry to the right atrium. This type of geometric arrangement would suggest that the entire circuit of intranodal reentry is localized within the AV node and that no part of the atrium is necessary. A set of observations made and consequent arguments have been put forth in an attempt to prove this point. It should be pointed out at the outset that the evidence available on this issue is all indirect and somewhat sketchy and insufficient to prove the point. Nonetheless, the observations, their interpretations, and the logic for the conclusions drawn are discussed as follows.

**Spontaneous VA (HA) Block or VA (HA) Dissociation During AV Nodal Reentrant Tachycardia**

This dissociation would clearly be the strongest evidence to support the lack of atrial participation.\textsuperscript{42,44,45} It is exceedingly rare, however, to see VA block and/or VA dissociation during AV nodal reentrant tachycardia. We have never seen either of these phenomena in our series of more than 300 patients with AV nodal reentrant tachycardia. When such findings are encountered, other AV junctional tachycardias such as intra-His bundle reentry, bundle branch reentry, automatic junctional tachycardia, nonreentrant junctional tachycardia, or sinus rhythm with 1:2 conduction in which atrial impulses conduct over the fast and slow pathways alternately are difficult to rule out, and such possibilities have seldom been convincingly excluded.\textsuperscript{46}

**Premature Atrial Depolarization During AV Nodal Reentry Without Affecting the Tachycardia Cycle Length**

Published demonstration of this effect is limited to one or two cycles in which the cycle length of the tachycardia does not seem to change, whereas both high and low right atria appear to be depolarized ahead of the expected timing.\textsuperscript{39} There are several problems with this form of evidence. 1) In several cases of AV nodal reentrant tachycardia, AV nodal penetration and hence resetting can be demonstrated if the entire diastolic interval is systematically scanned.\textsuperscript{40} At certain coupling intervals, the degree of AV nodal delay after the premature beat may equal the decrease in the atrial coupling interval, giving the false impression of nonpenetration of the tachycardia circuit. 2) Since the slow pathway in common AV nodal reentry is used in the anterograde direction (Figure 11), penetration into the slow pathway may be facilitated by pacing near the coronary sinus ostium. Using that site, it is at times possible to advance the His depolarization even after atrial activation on the His bundle electrogram (i.e., low atrial septal region) has already begun.\textsuperscript{47}

**Shorter Cycle Length of 1:1 Conduction During AV Nodal Reentrant Tachycardia Compared With Atrial Pacing**

It is a frequent observation that during pacing from the high right atrium, the development of the AV nodal Wenckebach phenomenon leads to the onset of sustained tachycardia.\textsuperscript{11,39,40} The cycle length of induced tachycardia is sometimes shorter than the atrial cycle length, which results in the AV nodal Wenckebach phenomenon. One interpretation of this finding is that the reentrant impulse bypasses some portion of the AV node (upper common pathway) needed for AV nodal penetration by the paced atrial impulse. Another related observation is that AH intervals during AV nodal reentry are shorter than at a pacing cycle length identical to the tachycardia cycle length when a comparison of 1:1 conduction is made.\textsuperscript{39,40} Although these arguments superficially appear reasonable, they suffer from at least two flaws. 1) In the absence of autonomic blockade, it is unlikely that one will get stable and accurate assessments of AV nodal conduction time and refractoriness. At the onset of pacing, there is less sympathetic stimulation, which could progressively increase as the rate of pacing increases and also at the onset of tachycardia. Further enhancement of sympathetic tone could follow as the tachycardia continues. Depending on the timing of analysis, the cycle length of tachycardia could be significantly shorter than the paced cycle length, which induces the AV nodal Wenckebach phenomenon and the onset of tachycardia. Similarly, the AH during the tachycardia may be shorter com-

**Figure 10.** Tracings showing continuation of atrioventricular (AV) nodal reentry despite block in the His-Purkinje system (HPS). The initiation of AV nodal reentry occurs after the second atrial premature beat (A₂). At the onset of functional block, there is a 3:2 HPS (HV) Wenckebach and then a 2:1 block in HPS. This, however, has no influence on the cycle length of the tachycardia, suggesting that the lower common pathway is above the level of the His bundle recording site. These findings convincingly excluded the possibility of AV accessory pathway participation in the reentry. HRA, high right atrium; HB, His bundle; T, time lines.
pared with 1:1 AV conduction during pacing if the sympathetic tone is higher during the tachycardia. 2) It is currently realized that the slow AV nodal pathway is located more posteriorly and that the measurement made from elsewhere in the atrium may not be representative of the actual conduction times. Unless the latter are from the area at or near the slow pathway entrance site, such measurements are bound to be misleading (see Figures 11, 13, and 14).

The original observations of Sung et al. that the retrograde breakthroughs of AV nodal reentrant impulse via the fast pathway and slow pathway are clearly different, are now confirmed (Figure 11). The atrial activity on the His bundle trace is earliest during the common AV nodal reentry, suggesting an anterior superior breakthrough. Conversely, a posterior inferior breakthrough of retrograde atrial activation is the rule during the uncommon type of AV nodal reentry, and hence, the atrial activation near the ostium of the coronary sinus precedes that on the His bundle recording.

The retrograde atrial activation sequence during AV nodal reentrant tachycardia and ventricular pacing in these patients is similar. However, it is still unclear whether the anterograde and retrograde conduction occurs over the same pathway in many of these cases. Our preliminary observations suggest the existence of multiple fast and slow pathways in some patients.

**Differential Diagnosis**

Common AV nodal reentry can be confused with several other forms of atrial and AV junctional tachycardias when the P wave cannot be clearly identified on the surface ECG leads. These include unifocal atrial tachycardia, atrial flutter, orthodromic AV reentry using an accessory pathway, and occasionally, automatic and nonreentrant junctional tachycardia. AV nodal reentrant tachycardia with aberrant conduction can also be mistaken for bundle branch reentry; the latter, however, shows AV dissociation in most cases. Distinction from atrial tachycardia and flutter can be readily made by standard maneuvers that, by producing a high degree of AV block, reveal the atrial origin of tachycardia. With similar maneuvers, AV junctional tachycardia (both AV nodal and orthodromic AV reentry) will either terminate, demonstrate slowing, or show no effect. However, a 1:1 P-to-QRS relation is maintained if the tachycardia continues. When the P wave or atrial activity can be identified (via esophageal or right atrial electrogram), simultaneous atrial and ventricular activation is virtually diagnostic for AV nodal reentry (Figures 1, 4, 10, and 11).

In the case of AV reentry, the ventricular activation must occur first, before the impulse can retrogradely engage the accessory pathway and reach the atria, and therefore, the retrograde P wave occurs after the QRS. Occasionally an atrial tachycardia with a long PR and 1:1 P-to-QRS relation may mimic AV nodal reentry, but here the sequence of atrial activation should give the clue, i.e., low interatrial septal activation will precede all other atrial sites in the case of AV nodal reentry. Further distinction from atrial tachycardia can be made by observation that the retrograde atrial activation sequence during ventricular pacing is identical to that during the AV nodal reentrant tachycardia.

Several forms of supraventricular tachycardia can mimic uncommon AV nodal reentry. The most difficult distinction is between uncommon AV nodal reentry and the so-called "permanent" form of junctional reentrant tachycardia (PJRT). In this arrhythmia, which can present as paroxysmal or incessant, the retrograde conduction is via a slow conducting accessory pathway that is not connected to the normal His bundle, yet the retrograde atrial breakthrough is in the posterior septal region. A clear separation between this entity and uncommon AV nodal reentry can be made by advancing the atrial activation with programmed ventricular extra-stimuli delivered at a time when the His bundle is refractory. Atrial tachycardia arising in the region of the low interatrial septum may also be difficult to separate from uncommon AV nodal reentry.

When both common and uncommon AV nodal reentry occur in the same patient, AV nodal origin of both tachycardias can be diagnosed with some degree of confidence by the shear coexistence of the two arrhythmias.

**Management of AV Nodal Reentrant Tachycardia**

**Acute Termination**

Vagal maneuvers such as carotid sinus massage, the Valsalva maneuver, etc. can frequently terminate this arrhythmia, and patients can often be instructed to apply these techniques. Reentry can be readily interrupted by a variety of pharmacological agents. At this time, the ideal agent is adenosine, which has a rather short half-life and can be administered intravenously. Other agents that are also effective include calcium channel blockers, i.e., verapamil and diltiazem, as well
as β-blockers. Although digitalis can be used, it is probably the least effective. All of the agents mentioned generally affect reentry by depressing conduction and by prolonging the refractoriness of the AV node in the anterograde direction. Class I agents such as procainamide can terminate the same arrhythmia by blocking the conduction in the retrograde direction.

**Chronic Management**

**Pharmacological therapy.** Figure 12 depicts the likely site of drug action of various therapeutic agents in the common form of AV nodal reentrant tachycardia. Drugs working on the anterograde slow pathway are often used but seem less effective. Class I agents, particularly Class Ic drugs such as flecaïnine and encainide, are effective in controlling recurrences. Their use may be limited because of the potential for adverse reaction in the presence of structural heart disease. Propafenone may also work in this and other forms of AV junctional reentry. Because of significant systemic side effects, the routine use of amiodarone for treatment of common or uncommon AV nodal reentry is not recommended.

During chronic management, these pharmacological agents may control recurrences not only by direct effects on the reentry circuit but also by suppressing the triggers, i.e., spontaneous premature beats. Chronic therapy is usually recommended in patients with either frequent, disabling, or severely symptomatic tachycardia. Individuals with rare, brief, or mildly symptomatic episodes could be left alone or managed with oral medications taken only at the time of a tachycardia episode as needed.

It should be pointed out that the depressant effect of pharmacological agents in AV nodal reentry is reversible with adrenergic stimulation. This may account for clinical recurrences in patients who seem to have achieved pharmacological control in the laboratory. This is a significant limitation of drug therapy, since a minimal drug effect may be present during physical exertion, anxiety, or mental stress. The addition of β-blockers to the regimen frequently helps to prevent such clinical recurrences by blocking the effect of catecholamines. The description above is based primarily on experience with common AV nodal reentry. The effectiveness of various agents and the location of drug action in uncommon form has seldom been systematically examined.

**Nonpharmacological therapy.** Earlier surgical treatment (surgical ablation) was aimed at interruption of the AV conduction. During one of these surgical procedures, the AV node was fortuitously modified, resulting in abolition of AV nodal reentrant tachycardia without production of AV block. In 1985, map-guided perinodal dissection was introduced as a cure for AV nodal reentry by Ross et al. Either the anterograde or retrograde limb of the circuit could be selectively eliminated by careful dissection. The surgical cure of AV nodal reentry was achieved by Cox et al, who used cryosurgical modification of the AV node. A series of cryolesions were created in the perinodal area. To what extent these cryolesions or perinodal dissection affected the compact portion of the AV node is unclear, but these surgical methods were successful in controlling the tachycardia while AV conduction remained intact. The main limitation of surgical therapy, of course, is that thoracotomy and cardiopulmonary bypass are required, with the potential of significant morbidity.

Catheter ablation has evolved very rapidly from a last-resort treatment in the form of AV junctional ablation with interruption of AV conduction to selective modification of the AV node as the ideal treatment. High-energy DC electric shock was used initially to produce AV block by ablation of the AV junction. Subsequently, modification of DC ablation technique showed selective damage to one of the critical AV nodal pathways. In an earlier report, Haissaguerre et al ablated the fast pathway in 16 of 21 patients and produced AV block in two. Epstein et al delivered a current from the anterior to the posterior region of the AV junction along the line extending from the His bundle to the region of coronary sinus. AV nodal reentry was successfully controlled by ablation of either the fast or slow pathway in six of nine cases. The use of radiofrequency as the energy source in patients with AV nodal reentrant tachycardia was first reported by Goy et al.

Most of the earlier experience with radiofrequency modification of the AV node deals with ablation of the fast pathway. The successful outcome was noted in 50–91% of the cases, but complete AV block occurred in 2–8%. A preliminary report of selective slow pathway ablation by Roman et al. encouraged other investigators to take this approach to eliminate AV nodal reentrant tachycardia. Reported results of slow pathway ablation show a success rate of >90%, with <2.0% incidence of AV block. If slow pathway ablation is tried first and fast pathway ablation is attempted only in the event that the slow pathway ablation fails, a successful result can be achieved in >95% of cases, and one can minimize the risk of AV block.

The techniques of selective fast and slow pathway ablation with radiofrequency energy from different laboratories have been published and will not be detailed here. However, a brief description of the technique at our laboratory is as follows. The radiofrequency energy is delivered between the tip of an ablation catheter and external adhesive patch electrode placed over the chest wall. A conventional electrosurgical generator is the source of radiofrequency current, producing unmodulated bipolar output at 300–750 kHz. The positioning of catheters in specific locations is critical to
achievement of selective ablation of the fast or slow pathway (Figures 13 and 14).

For a fast pathway ablation, the catheter is initially placed along the AV node–His bundle axis to record a bipolar His bundle potential. The catheter is then withdrawn to obtain a large atrial but a small or no His bundle potential. The radiofrequency energy is applied to this region (Figure 14, panel A). The PR interval is carefully monitored, and any prolongation should be followed by testing of the retrograde conduction. The end point is elimination of retrograde conduction over the fast pathway and noninducibility of AV nodal reentry.

Ablation of the slow pathway can be more easily accomplished if retrograde slow pathway conduction can be demonstrated. Recording of slow pathway potential to achieve successful slow pathway ablation has also been recommended. In the majority of patients, retrograde slow pathway conduction cannot be demonstrated; hence, we have taken a more practical approach, which is anatomically guided for ablation of the slow pathway. Since the slow pathway seems to be located posteriorly near the region of the coronary sinus ostium, we start slow pathway ablation in this region first (Figure 14, panel B). After careful catheter placement, a lesion is made on the atrial side of the tricuspid annulus adjacent to the coronary sinus ostium. If the applied lesion is not successful, the catheter is gradually moved more anteriorly (Figure 14, panels C and D). After each lesion, inducibility of tachycardia is tested. Noninducibility of tachycardia is considered a successful result even if a single AV nodal reentrant echo beat is still present. Results of radiofrequency modification of AV node in our series of patients with AV nodal reentry are depicted in Table 1.

Nineteen patients underwent fast pathway ablation, with complete AV block occurring in three (16%). Currently, our initial approach is slow pathway ablation, which has now been applied in 193 consecutive patients as the initial approach. Fast pathway ablation was used only in those cases where slow pathway ablation failed.

Aside from providing the opportunity for a permanent cure, surgical approaches and catheter modification techniques of the AV node have unmasked several electrophysiological phenomena and necessitated the reassessment of the true nature of AV nodal reentrant circuits. Further confirmation of these findings will undoubtedly lead to changes in how this phenomenon is viewed. Some aspects are highlighted below:

1. It seems that the locations of the so-called fast and slow pathways are clearly distinct anatomically. The curative approach is anterior superior for the fast pathway and posterior inferior for the slow pathway (Figures 11, 13, and 14).

2. The magnitude of anatomic separation of the two exits suggests that either the AV node is a larger structure than is believed or, perhaps more likely, that the perinodal tissue and/or the approaches to the AV node are incorporated into the reentry process in most cases. This could mean, in essence, that some degree of atrial participation in the AV nodal reentry circuit may exist. It is also conceivable that there may be sufficient functional and anatomic differences among individual patients that a single, unified concept may not explain all cases of AV nodal reentry. Until these issues are resolved, we suggest that the precise nature of upper turnaround during AV nodal reentry remains uncommitted.

3. The so-called fast or slow pathway may be made up of several groups of fibers (or broad bands), and the same fibers may or may not be used in both directions. In some patients, for example, anterograde fast pathway conduction may remain intact while retrograde fast pathway block occurs and vice versa. Similarly, anterograde slow pathway conduction may persist in some fibers while a critical slow pathway is ablated, eliminating the sustained form of reentry. These findings are
**Table 1. Atrioventricular Nodal Modification in 212 Patients**

<table>
<thead>
<tr>
<th>Fast pathway (n=19)</th>
<th>Slow pathway (n=193)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Successful AV block</td>
<td>Unsuccesful*</td>
</tr>
<tr>
<td>(no AV block)</td>
<td>(5)</td>
</tr>
<tr>
<td>Uncommon AVNR</td>
<td>(3)</td>
</tr>
</tbody>
</table>

**Slow pathway ablation**

<table>
<thead>
<tr>
<th>Fast pathway ablation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Successful AV block</td>
</tr>
<tr>
<td>(no AV block)</td>
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</tbody>
</table>

**Overall success**

<table>
<thead>
<tr>
<th>Speed pathway</th>
<th>AV block</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fast pathway</td>
<td>188/193 (97%)</td>
</tr>
<tr>
<td>Slow pathway</td>
<td>24/24 (100%)</td>
</tr>
</tbody>
</table>

AVNR, atrioventricular nodal reentry.
*There have been no failures or AV block in the last 117 cases with slow pathway ablation.

**Summary**

1. AV nodal reentry is a relatively common cause of supraventricular arrhythmia presenting as a regular, narrow, QRS tachycardia that at times may show aberrant conduction.

2. A correct diagnosis can be suspected from the surface ECG when the P wave is either obscured by the QRS complex or is recognizable at the tail end of it in the common type and from a long RP in the uncommon variety. It is rare to find examples of AV nodal reentry.
in which the surface ECG pattern does not fit the above descriptions.\(^ {21} \)

3. Precise diagnosis, definition of circuits, and assessment of AV nodal properties can be made during intracardiac electrophysiological studies.

4. The basis for a reentry is the functional (and anatomic) duality of conduction pathways, and the so-called fast and slow pathways have a distinct anatomic location. The entire circuit in all probability incorporates atrial approaches to the AV node.

5. Precise boundaries of the AV node, its approaches, and the three-dimensional geometry of AV nodal circuits still need better definition.

6. The arrhythmia can be terminated and recurrences prevented by specific pharmacological agents, and the reason for successful results can usually be demonstrated.

7. Catheter ablation of the slow pathway by radiofrequency energy is the preferred initial approach to curative treatment and is rapidly becoming the therapy of first choice.

8. Finally, AV nodal reentry represents an interesting entity in which focused research and consequent better understanding of the physiology have enabled us to precisely localize the problem area and accurately apply very specific curative therapy, which relieves these patients from life-long suffering.

References


