PATHOPHYSIOLOGY AND NATURAL HISTORY
ARRHYTHMIA

Atriofascicular connection or a nodoventricular Mahaim fiber? Electrophysiologic elucidation of the pathway and associated reentrant circuit

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ABSTRACT Accessory pathways showing decremental properties and inserting into the right ventricle have been frequently described as “nodoventricular” or Mahaim pathways. However, conclusive evidence for a nodal origination of such pathways is lacking. The patient in this study had characteristics typical of such a pathway. Antegrade, the pathway showed decremental, nodelike conduction properties. With the aid of right bundle branch recordings, the pathway was demonstrated to insert directly into the right bundle branch. Atrioventricular reciprocating tachycardia could be readily initiated by atrial or ventricular pacing. The QRS morphology was normal during sinus rhythm and demonstrated a left bundle branch block pattern with normal axis during tachycardia. The reentrant circuit involved antegrade conduction over the accessory pathway and retrograde conduction via the bundle branches, His bundle, and the atrioventricular node. More significantly, late atrial stimuli delivered during tachycardia could preexcite the ventricle via the accessory pathway despite their inability to enter the atrioventricular node. Thus, the upper “turn around” of the reentrant circuit involved atrial tissue and the accessory pathway originated directly from the right atrium independent of the atrioventricular node. In view of these new findings and other recent observations during surgical resection of similar pathways, a reassessment of previous descriptions of “nodoventricular” fibers may be necessary. Many of these pathways may actually represent atriofascicular or atrioventricular connections with decremental properties.


ANOMALOUS conduction pathways connecting the atrioventricular node or His-Purkinje system to the ventricle have been described anatomically since the late 1930s.1-2 The possible roles that these pathways may play in human cardiac arrhythmias have been more recently presented.3-4 However, delineation of the reentrant circuit during reciprocating tachycardias can sometimes be difficult. At least two types of tachycardia circuits have been proposed. One used a nodoventricular bypass antegradeley and the His-Purkinje system and the atrioventricular node retrogradely.3-5 Another involved reentry within the atrioventricular node with the bypass tract and the His-Purkinje system serving a bystander role.6 These proposals all suggested that the accessory pathway involved the atrioventricular node. In fact, schematically, these pathways have usually been represented as arising from the node and inserting into the right ventricle.7-8 However, definitive evidence that such pathways do arise from the atrioventricular node is lacking.

In this communication, evidence will be presented that will: (1) describe an accessory pathway that behaved as a typical “nodoventricular” Mahaim fiber, but actually arose directly from the right atrium and inserted into the right bundle branch, and (2) demonstrate the usefulness of a stable right bundle branch recording in addition to the usual His recording in elucidating participation of the His-Purkinje system in reentrant tachycardia involving this pathway.

Methods

The patient was a 16-year-old boy referred for evaluation of recurrent episodes of palpitation. At the onset of these episodes, he would feel dizzy, but he never lost consciousness. During one attack, he was seen in an emergency room where a wide complex tachycardia was recorded and terminated with intravenous verapamil. Otherwise, he was asymptomatic and actively participated in vigorous sporting activities.

An electrophysiologic study was performed with the patient in the nonsedated postabsorptive state after informed consent.
Results

The surface electrocardiogram during sinus rhythm (figure 1, A) was normal except for presence of increased voltages in the right precordial leads. An electrocardiogram obtained during tachycardia showed a ventricular rate of 200 beats/min with a widened QRS that resembled left bundle branch block with a normal axis (figure 1, B). A 12-lead electrocardiogram obtained during atrial pacing at a similar rate showed a QRS morphology identical to that seen during spontaneous tachycardia (figure 1, C).

Observations during incremental atrial pacing. During incremental atrial pacing, there was a progressive

was obtained. One No. 6F quadripolar catheter was inserted via the left antecubital vein and positioned in the coronary sinus. A No. 6F decapolar electrode catheter was inserted through the right antecubital vein and the tip was positioned at the right ventricular apex. On this catheter, a proximal pair of electrodes located in the high right atrium was used to record atrial electrograms. Two No. 6F quadripolar catheters were inserted via the right femoral vein. One was positioned across the tricuspid valve. His bundle electrograms were recorded from the proximal pair of electrodes of this catheter and right bundle branch recordings were obtained from the corresponding distal pair of electrodes. The tip of the second femoral catheter was positioned at the right anterior portion of the low right atrium for pacing. Programmed electrical stimulation was performed from the right atrium, coronary sinus, and right ventricular apex.

During ventriculoatrial conduction, the His to atrial electrogram interval (HA interval) was measured from the onset of His electrogram to the onset of low septal atrial electrogram recorded from the His bundle electrodes. When the initial deflections of low atrial electrograms could not be seen due to overlap with ventricular electrograms, HA intervals were obtained by subtracting intra-atrial conduction time (septal to high right atrial electrogram) from the His to high right atrial interval. Such subtractions were considered to be acceptable since no detectable conduction delays were seen in the atria.

FIGURE 1. Electrocardiograms obtained during sinus rhythm, tachycardia, and rapid atrial pacing. Twelve-lead surface electrocardiograms obtained during sinus rhythm (A), clinical supraventricular tachycardia (B), and atrial pacing at a cycle length close to that of spontaneous supraventricular tachycardia (C) are illustrated. Note the similarities between the QRS morphology during clinical tachycardia and that seen during rapid atrial pacing.

FIGURE 2. Atioventricular conduction intervals during incremental atrial pacing. The intervals were measured from stimulus artifact in the right atrium to the respective His, right bundle, and earliest ventricular electrograms. Note that while there were gradual increases in the intervals with shortening of pacing cycle length, there was a decrease in the difference between SV and S-RB or SH intervals. This decrease was associated with progressive preexcitation of the QRS complexes. At cycle lengths less than 380 msec, the QRS complexes became maximally preexcited in association with a quick rise in S-RB and SH intervals. At the same time, there was a reversal in activation sequence of the ventricle, right bundle, and His bundle. This reversal of activation sequence implied that the right and His bundle were now activated retrogradely.
increase in atrioventricular conduction times as measured by the time interval between the atrial stimulus and the earliest ventricular electrogram (SV interval), as well as the His and right bundle electrograms (SH and S-RB intervals, figure 2). Note that at cycle lengths less than 380 msec, SH intervals were actually longer than SV intervals. Lengthening of atrioventricular conduction time was associated with progressive preexcitation of QRS complexes until at maximal preexcitation (cycle lengths < 500 msec, figure 2), the QRS complexes were identical to those seen during clinical tachycardia. These findings were consistent with behavior of so-called nodoventricular Mahaim fibers. However, data presented later in this report would be inconsistent with origination of this accessory pathway from the atrioventricular node.

Progressive preexcitation was also associated with a shift in the relationship between His and right bundle electrograms. The His to right bundle activation sequence actually reversed at cycle lengths associated with maximal preexcitation, as illustrated in figure 3. This reversed sequence, which was identical to RB-H sequence during ventricular pacing (shown later in figure 6), demonstrated that the right bundle branch and His bundle were activated in a retrograde direction when there was maximal preexcitation of the QRS complex. Without the right bundle recording, it would have been impossible to verify that any portion of the His-Purkinje system was actually activated retrogradely during atrial pacing.

It should also be noted that the right bundle electrogram in figure 3, B, occurred within the first 5 msec of earliest ventricular activation seen on any lead, implying that the pathway of preexcitation inserted directly into the right bundle branch. Otherwise, it would be highly unlikely for these structures to be activated retrogradely so early in relationship to ventricular activation if conduction were to occur through myocardium before entering the His-Purkinje system.

At an atrial pacing cycle length of 300 msec, reciprocating tachycardia was induced. Figure 4 shows the initiation of tachycardia. Atrial activation sequence of the fourth atrial beat (arrow) was different from that of preceding paced beats, indicating an alternate activation of the atria. The RB, H, and atrial activation sequence of the same beat, however, was identical to that seen during the subsequent tachycardia and during ventricular pacing (shown later in figure 6). It appeared that maximal preexcitation during atrial pacing was associated with retrograde activation of the right bundle branch and His bundle, and that tachycardia started when the impulse propagated through the atrioven-

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**FIGURE 3.** Reversal of H-RB activation sequence during rapid atrial pacing. The electrograms shown here and in all subsequent figures are in the same format. Two surface electrocardiographic leads and all six intracardiac leads are shown. The following abbreviations identify the intracardiac leads: HRA = high right atrium; CSp and CSd = proximal and distal coronary sinus respectively; HB and RB = His and right bundles; RV = right ventricular apex. All right atrial pacing in this figure and subsequent ones was from a low anterior right atrial site. A, Right atrial pacing at a cycle length of 550 msec. Atrial stimulation artifacts, H and RB deflections, are indicated by arrows. The QRS morphology showed no preexcitation. The H-RB interval (10 msec) was consistent with antegrade conduction over the right bundle. B, Right atrial pacing at a cycle length of 320 msec. The QRS now shows maximal preexcitation. Note that activation sequences of His and right bundles are reversed (perpendicular lines added to facilitate comparison) and RB-H interval is now 10 msec. The right bundle deflection in B occurred within the first 5 msec of earliest ventricular activation, implying that the pathway of preexcitation inserted directly into the right bundle branch.
tricular node and completed the reentrant loop by reactivating the atria. As explained in the legend to figure 4, after initiation with the fourth beat, the tachycardia continued to be entrained by atrial pacing until the last stimulus.

Comparison of right and left atrial pacing. While preexcitation could be seen during right atrial pacing at cycle lengths as long as 500 msec, pacing from the coronary sinus at cycle lengths as short as 350 msec did not reveal any ventricular preexcitation. A comparison of right atrial and coronary sinus pacing at a cycle length of 350 msec is shown in figure 5, A and B. Note the preexcited QRS morphology in panel A when pacing was performed from the right atrium in contrast to the narrow QRS morphology seen in panel B when pacing was switched to the distal coronary sinus electrodes. During coronary sinus pacing at cycle lengths of 320 msec or below, transition from normal QRS morphology to the preexcited form occurred abruptly (figure 5, C). The first preexcited beat was always maximally preexcited and was followed by initiation of tachycardia. Initiation of tachycardia during coronary sinus pacing always followed the same sequence.

In contrast, right atrial pacing at cycle lengths below 350 msec showed maximal preexcitation but without triggering the tachycardia until pacing cycle length was shortened to 300 msec (figure 4). This difference in right vs left atrial pacing indicated a quicker input from the right atrium into the accessory pathway. Because of this earlier access, pacing from the right atrium resulted in a preexcited QRS morphology at cycle lengths that were longer than those needed during coronary sinus pacing.

On the other hand, preferential input was into the normal, fast-conducting, atrioventricular nodal pathway during coronary sinus pacing and preexcitation only occurred when conduction blocked in this pathway, which then allowed right bundle and ventricular activation via the accessory pathway, leading to reentry. The fact that tachycardia could be initiated at longer cycle lengths during coronary sinus pacing than with right atrial pacing was also consistent with the proposed preferential inputs into those pathways. With any reentrant circuit, one would expect initiation of tachycardia to be easier when pacing was performed nearer the site of unidirectional block, in this case, nearer the normal atrioventricular nodal pathway that had preferential input from the left atrium.

Effects of incremental ventricular pacing. Incremental ventricular pacing was performed from the right ven-

FIGURE 4. Induction of reciprocating tachycardia during right atrial pacing. Reentrant tachycardia was initiated at the fourth beat of this illustration (arrow). Note that stimulus to A (high right atrium recording) and V to A intervals (coronary sinus recording) changed abruptly on that beat, indicating an alternate activation of the atria than that of the preceding beats. This alternate activation was the same as that seen during the subsequent tachycardia. It appeared that maximal preexcitation during rapid atrial pacing was associated with retrograde activation of the right and His bundles (RB-H = 10 msec), and that tachycardia started when this retrograde activation succeeded in traversing the atrioventricular node to complete the reentrant loop. While tachycardia started with the fourth beat, it continued to be entrained by atrial pacing. Such entrainment was evidenced by continued activation of the ventricle at the paced cycle length and presence of fusion, most likely in the right atrium, until the last pacing stimulus. The last stimulus (i.e., the seventh) probably did not capture the atria since the V-V interval following that stimulus increased to the length of the tachycardia cycle length. Alternatively, it is possible that antegrade conduction via the accessory pathway suddenly slowed at that moment. Electrograms are designated as in figure 3.

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tricular apex. Figure 6 shows electrograms during ventricular pacing at a cycle length of 300 msec. Right bundle branch and His bundle electrograms can be seen at the beginnings of the respective ventricular electrograms. The retrograde activation sequences of the right bundle, His, and atrial electrograms and their respective intervals (RB-H, H-A, and intra-atrial conduction intervals) seen here were identical to those seen during tachycardia. Reciprocating tachycardia was initiated during this pacing train (heavy arrow). The first 3 beats of tachycardia revealed QRS fusion, indicating activation of the ventricle from both the paced wavefront and the reentrant wavefront. Stimuli to RB intervals (S-RB) of these beats were foreshortened, implying preexcitation of the right bundle via the accessory bypass. The retrograde activation sequences of RB, H, and A during these fusion beats and during the ensuing tachycardia were identical to those seen during ventricular pacing. This identity strongly suggested that the retrograde limb of reentry involved the right and His bundles. The extent to which the atria may be involved in this circuit, if any, could not be determined from the data presented thus far.

**Effects of premature beats during right and left atrial pacing.** To compare effects of premature atrial beats from the right vs left atria, premature stimuli were
Introduction of tachycardia from the coronary sinus and right atrium during pacing at a basic cycle length of 350 msec. With pacing from the right atrium, QRS complexes following both S1 and S2 were totally preexcited (figure 7, A). Tachycardia could not be initiated at any S1S2 coupling interval and atrioventricular block occurred at a coupling interval of 240 msec.

In contrast, the pattern of conduction was quite different during pacing from the coronary sinus. Ventricular preexcitation was seen neither during the basic drive nor at S1S2 coupling intervals exceeding 280 msec (figure 7, B). With coupling intervals of 280 msec or less, however, V2 showed maximal preexcitation and always initiated the tachycardia (figure 7, C). These observations again suggested a preferential input from the right atrium into a decrementally conducting right atrium to right bundle branch pathway.

It was also interesting to note the events following induction of tachycardia, as illustrated in figure 7 by the arrows indicating positions of the right and His bundle deflections. The first 4 beats of tachycardia showed alternation of cycle length in association with retrograde right bundle branch block or conduction delay. Atrioventricular intervals during these beats remained essentially unchanged. However, retrograde right bundle branch block occurred with the first beat of tachycardia (H and RB followed V), forcing the reentrant impulse to propagate over the left bundle, thus lengthening the tachycardia cycle length (320 msec). The next beat of tachycardia showed normal retrograde right bundle conduction and a shorter cycle length (270 msec).

It is curious, however, that the third beat of tachycardia again demonstrated retrograde right bundle conduction delay, with prolongation of VA interval, movement of H and RB electrograms into the ventricular electrograms, and prolongation of the cycle length (290 msec). Subsequent beats showed normal retrograde right bundle propagation and stable cycle lengths of 275 msec. This phenomenon of alternating conduction delays in the His-Purkinje system, was most likely due to long-shorn-long sequence of ventricular activation, which has been shown to lengthen His-Purkinje system refractoriness.9

It is unusual for His-Purkinje system conduction delays to cause alteration of cycle lengths during reentrant supraventricular tachycardias. Such alternations, usually seen at beginnings of supraventricular tachycardias, have been typically attributed to beat-to-beat changes in atrioventricular nodal conduction.10 In this unique example, atrioventricular and HA intervals did not alternate; only VH intervals did. The associated changes in cycle length confirmed the integral role of the bundle branches in the tachycardia circuit.

Effects of ventricular premature beats during ventricular pacing. Ventricular refractory period determination was
performed from the right ventricular apex at a basic cycle length of 550 msec. At a long S1S2 coupling interval of 400 msec, no retrograde conduction delay was noticed (figure 8, A). As this coupling interval was shortened to 330 msec, retrograde conduction delay (longer S2A2) could be seen (B) concomitant with disappearance of His and right bundle deflections from the beginnings of their respective ventricular electrograms. With further shortening of S1S2 coupling interval to 290 msec (C), retrograde His and right bundle electrograms could be seen to emerge after the ventricular electrograms.

Several noteworthy features of these three panels should be pointed out: (1) With progressively earlier premature beats, the His and right bundle deflections shifted from a position preceding their respective ventricular electrograms to a position following them. This behavior confirmed that these recordings indeed behaved as expected for His and right bundle recordings. (2) While the RB-H sequence during ventricular
pacing was opposite that seen during atrial pacing with no preexcitation, these two deflections were simultaneous when they appeared after the ventricular electrogram of S₂ in panel C. Such changes in activation further confirmed that these recordings were from the His and right bundles. (3) Based on HA intervals measured in panels A through C, retrograde conduction delay seen with shortening of S₁S₂ coupling intervals could be localized to the His-Purkinje system with no significant delay occurring in the atrioventricular node.

With shortening of S₁S₂ coupling interval to 280 msec (figure 8, D), reciprocating tachycardia was initiated. VA intervals of the first and second beats of tachycardia were prolonged, with RB and H deflections visible after their respective ventricular electrograms (arrows indicate RB and H deflections after V₂ and the first 3 beats of tachycardia). The following sequence of events could be deduced regarding the initiation of tachycardia. V₂ blocked within the right bundle branch and conducted retrogradely up the left bundle. The impulse then simultaneously conducted down the right bundle and up the His bundle (RB-H = 0). The impulse conducting down the right bundle blocked before activating the ventricle due to refractoriness persisting in the right bundle from the preceding retrograde penetration by V₂. The impulse, conducting up the His bundle, propagated through the atrioventricular node, and excited the ventricle (V₃) via the accessory con-
connection. $V_3$ also blocked retrogradely within the right bundle as the right and His bundle electrograms again appeared following their respective ventricular electrograms. $V_3$ conducted to the node again via the left bundle and continued the reentrant process.

A similar phenomenon was seen with $V_4$ before resolution of retrograde right bundle branch block with $V_5$. Starting with $V_5$, retrograde impulses propagated to the node via the right bundle. Maintenance of retrograde right bundle branch block for 2 beats after $V_2$ most likely represented a linking-by-interference phenomenon whereby successive impulses traveling in opposite directions repetitively blocked due to residual refractoriness from the preceding blocked impulse. This process of linking was initiated with $V_2$, which blocked retrogradely in the right bundle.

Of note during initiation of tachycardia was that $V_3$-$V_4$ and $V_4$-$V_5$ intervals were longer than the subsequent intervals, despite the fact that the AH intervals after $V_3$ and $V_4$ were shorter than during subsequent beats of the tachycardia. This provided further evidence that the His-Purkinje system was an essential part of the reentrant circuit. Although either bundle branch could be used for the retrograde limb, when the left bundle branch was used, tachycardia cycle length was increased due to addition of transeptal conduction time. The constancy of HA intervals seen in these panels, despite changes in the route of impulse propagation through the bundle branches, firmly established that retrograde atrial activation occurred via the atrioventricular node.

**Effect of early atrial beats introduced during tachycardia.** Atrial stimuli were introduced during tachycardia to the right atrium through the tip of the atrial catheter, which was positioned at the anterior part of the low right atrial wall. Multiple episodes of tachycardia resetting were demonstrated, with atrial stimuli delivered as late as 10 to 40 msec after onset of the low septal atrial electrogram. Figure 9 shows an example of atrial stimulus delivered 40 msec after the onset of the retrograde low septal atrial electrogram, which was nevertheless able to advance ventricular activation during tachycardia.
The ability of such a late right atrial beat to preexcite the ventricle during tachycardia indicated that the accessory pathway originated directly from the right atrium and that the tachycardia circuit used low atrial tissues as the upper “crossover” between the retrograde fast pathway and the antegrade, slower conducting, accessory limb of the circuit. Given this observation, it is highly improbable that this accessory pathway was a “nodoventricular” fiber.

Discussion

Insights gained from right bundle recording. This report illustrated the usefulness of recording right bundle branch electrograms in elucidating the pathways of reentry. During incremental atrial pacing and after premature atrial beats, the presence of right bundle recordings revealed that, with progressive ventricular preexcitation, activation sequence of the right and His bundles became completely reversed before initiation of tachycardia. The observation that right bundle branch potential occurred almost simultaneously with earliest ventricular activation during tachycardia and during rapid atrial pacing excluded the possibility that the bypass tract was a right-sided Kent bundle with decremental properties. For the right bundle to be activated this early, the bypass tract must have inserted directly into the right bundle branch.

Since RB-H intervals during tachycardia were the same as those during ventricular pacing, the site of bypass tract insertion into the right bundle must have been below the RB recording site. The fact that RB-H-A activation sequence was the same during ventricular pacing and during tachycardia also suggested that this pathway was part of the tachycardia circuit. Furthermore, the presence of right bundle and His recordings during every beat of tachycardia provided conclusive evidence that the His-Purkinje system was an integral part of the reentrant circuit by demonstrating occurrence of retrograde right bundle branch block and its effect on cycle length of tachycardia. Lengthening of the VH interval with occurrence of retrograde right bundle branch block (as illustrated in figure 8, D), in association with prolongation of tachycardia cycle length, was a direct indicator of His-Purkinje system involvement in the reentrant pathway.

The upper “turn around” site of the reentrant pathway. Ever since the description of nodoventricular fibers by Mahaim,1,2 the potential that these fibers may be involved in reentrant pathways, or in “bystander” activation of the ventricle during supraventricular tachycardias, has been a subject of intense interest among cardiac electrophysiologists.3-7,12,13 The QRS during the reported tachycardias was typically left bundle branch block in morphology, with or without left-axis deviation.

Antegrade atrioventricular conduction usually showed dual-pathway physiology, with a fast pathway used in the retrograde limb of reentry and a slow pathway with preferential right atrial input associated with the accessory Mahaim fiber. The “atrioventricular nodal” properties of the slow pathway were manifested by progressive prolongation of atrioventricular conduction time with shorter coupling intervals of atrial premature beats or shorter cycle lengths of atrial pacing. The reentrant tachycardia reported here appeared to have all of these characteristics and could easily have been considered to implicate the presence of a “nodoventricular” pathway. Schematic representations of these bypass tracts have depicted them as arising from the right side of the atrioventricular node and connecting to the right ventricle3-8 or to the right bundle branch.12 The suggestion in such reports was that the upper turnaround of the reentrant circuit was within the atrioventricular node, much like those diagrammed in atrioventricular nodal reentrant tachycardias.

However, conclusive evidence demonstrating the intranodal nature of the upper turn around during reentry involving nodoventricular fibers has been lacking. The data presented in this report suggest that the atrial end of the reentrant pathway in this case was quite different from those seen in atrioventricular nodal reentrant tachycardias. Miller et al.14 have suggested that there is atrioventricular nodal tissue above the intranodal reentrant pathway in atrioventricular nodal reentrant tachycardia. The intranodal nature of the reentrant loop would make it difficult to capture the circuit with a single premature atrial beat. In fact, Kerr et al.15 have suggested that single premature atrial beats delivered close to the atrioventricular node during atrioventricular nodal reentrant tachycardia could not be delivered early enough to capture the reentrant circuit.

In this patient, atrial electrograms were never seen to dissociate from the tachycardia. Although this fact by itself did not necessarily imply that the atria were part of the circuit, other data obtained during this study (figure 9) strongly suggested that a portion of the right atrium was a link in the reentrant pathway. The fact that a right atrial stimulus delivered during tachycardia as late as 40 msec after onset of the low septal right atrial electrogram could, nevertheless, advance ventricular activation indicated that right atrial input into the accessory pathway did not involve the atrioventricular node. In addition, there must have been a fair distance between the atrial site of retrograde impulse emergence...
during tachycardia and the site of right atrial entrance into the accessory pathway to allow resetting of the tachycardia with such a late right atrial stimulus. Further evidence confirming that the origination of the accessory pathway was separate from the atrioventricular node can be derived from figure 4. Since retrograde activation of the atria occurred via the atrioventricular node, presence of entrainment with atrial fusion during right atrial pacing implied that the accessory pathway was independent of the node.

Proposed reentrant tachycardia circuit. Figure 10 is a schematic representation of the proposed tachycardia circuit based on data presented here. The atrial origination of the accessory bypass is totally dissociated from the normal atrioventricular node, and arises from the right atrium at a significant distance from the input to the atrioventricular nodal pathway. This accessory pathway has decremental properties, much like an atrioventricular node, and inserts at its lower end into the right bundle branch. The possibility that this accessory pathway also inserts into ventricular muscle cannot be excluded, although such a postulation is not necessary to explain the data reported here. During tachycardia, an atrial impulse propagates antegradely over the accessory pathway and into the right bundle branch. The impulse then bifurcates, with one conducting down the bundle to activate the ventricle and the other traveling up the right and His bundles before penetrating the atrioventricular node and reactivating the atria. If retrograde right bundle branch block occurs, the impulse conducting antegradely down the right bundle may still enter the ventricle. The His bundle is then activated via transeptal and left bundle conduction, resulting in prolongation of ventriculoatrial conduction time and tachycardia cycle length.

Clinical implications. In reported cases of atrioventricular reciprocating tachycardias attributed to the presence of so-called nodoventricular pathways, no definitive evidence implicating true Mahaim fibers in the reentrant circuit has been presented. Decremental conduction properties need not imply atrioventricular nodal origin. While one may argue that such evidence would be difficult to obtain during a clinical electrophysiologic study, the data reported here suggest an alternative anatomic origin that can be verified during electrophysiologic evaluations. Retrograde conduction over such accessory pathways is unusual. Thus, identification of atrial insertion of these pathways is more problematic than it is with accessory atrioventricular connections that have good retrograde conduction. The delivery of programmed premature atrial stimuli during tachycardia near the right atrioventricular ring can potentially discriminate between atrioventricular nodal and extranodal pathways. The ability to preexcite the ventricles during tachycardia, even though the premature atrial stimulus is delivered after retrograde atrial activation has begun, indicates an accessory pathway that is independent of the node, provided that the retrograde pathway can be firmly established to be atrioventricular nodal (as shown in figure 8).

Several reports of surgical resection of Mahaim-like decremental accessory pathways suggest that these pathways have no attachments to the atrioventricular node. Gillette et al. reported the surgical division of such pathways in three patients during anterior dissection of the right atrioventricular groove. In another recent preliminary report, three cases of successful surgical division of nodoventricular bypasses after dissection of the pyramidal space and right atrioventricular groove were described. Klein et al. recently reported two surgical cases of what was believed to be typical nodoventricular pathways during preoperative
electrophysiologic evaluations. Intraoperatively, these pathways were localized endocardially to the right anterior atrium at the atrioventricular groove. Thus, it is quite conceivable that these bypass tracts originated directly from the right atrium independent of the atrioventricular node.

In view of the above, the appropriateness of schematically picturing these accessory pathways as nodoventricular or describing them as Mahaim tracts should be reconsidered. It would appear more likely that such pathways represent atriofascicular or atrioventricular connections with decremental conduction properties but having no direct anatomic association with the atrioventricular node. Further data obtained from electrophysiologic studies or during surgery in such cases would help resolve this question.

References
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P Tchou, M H Lehmann, M Jazayeri and M Akhtar

Circulation. 1988;77:837-848  
doi: 10.1161/01.CIR.77.4.837

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231  
Copyright © 1988 American Heart Association, Inc. All rights reserved.  
Print ISSN: 0009-7322. Online ISSN: 1524-4539

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