Localization of the Fast and Slow Pathways in Atrioventricular Nodal Reentrant Tachycardia by Intraoperative Ice Mapping

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Background. Atrioventricular (AV) nodal reentrant tachycardia is classically described as a reentrant rhythm entirely contained within the compact AV node. Although the concepts of longitudinal dissociation of two intranodal pathways and a distal common pathway are accepted, the proximal portion of the circuit remains undefined. Current reports suggest that the two pathways may be separable by atrial tissue and not contained entirely within the compact node.

Methods and Results. We used an ice mapping method to demonstrate the slow and fast pathways of the reentrant circuit and their relation to the atrial septum around the AV node. Six patients with the usual form (slow-fast) of AV nodal reentrant tachycardia were mapped during surgery. In most patients, antegrade slow pathway localization was posterior and inferior to the compact AV node along the tricuspid annulus; in two patients, it was superior along the tendon of Todaro. Retrograde fast pathway localization was anterior or superior to the compact AV node in all patients. In all patients, anatomic distinction was made between the two pathways and the compact node.

Conclusions. We conclude that no upper common pathway exists within the compact AV node in the usual type of nodal reentrant tachycardia and that the perinodal atrial tissue is a requisite part of the tachycardia circuit. (Circulation 1992;86:919–925)

Key Words • tachycardia, atrioventricular nodal reentrant • mapping • tachycardia, supraventricular

Atrioventricular (AV) nodal reentrant tachycardia is classically described as a circus rhythm using functionally distinct antegrade and retrograde pathways within the AV node.1–4 Ventricular activation occurs via a distal turnaround in the NH region and the His-Purkinje system, the so-called distal common pathway. It is less clear whether atrial activation occurs independent of reentry or as an integral part of the tachycardia circuit. Atrial involvement in the proximal portion of the tachycardia circuit was proposed by Mendez and Moe5 and subsequently by others.6–10 However, current information does not exclude a model of tachycardia circuit contained entirely within the compact AV node.11 Retrograde atrial activation during tachycardia would then occur via an upper common pathway, as frequently depicted, and atrial tissue would not be required to complete the tachycardia circuit. The boundaries of the reentrant circuit and the extent of atrial involvement have important implications regarding the physiological characteristics of tachycardia and therapeutic alternatives.

In the present study, a nondestructive mapping method was used to distinguish the antegrade from the retrograde pathway in AV nodal reentrant tachycardia. This allowed determination as to whether the circuit was contained entirely within the compact AV node or inclusive of perinodal atrial tissue. Cooling of atrial tissue—ice mapping—temporarily rendered discrete areas unable to conduct sufficiently to sustain a tachycardia. When the tissue is cooled, its functional participation in the reentrant circuit can be determined from the characteristic pattern of tachycardia termination. It can then be classified as part of the antegrade or retrograde pathway or as nonessential to the tachycardia circuit. Ice mapping of the atrial septum was performed in six patients. The present report describes the observations made during intraoperative mapping.

Methods

Six patients with clinical tachycardia that was not adequately controlled with medication were referred for AV nodal modification surgery. Standard diagnostic electrophysiological studies were performed. All antiarrhythmic medications were stopped for more than five half-lives. All tachycardias were the common type of AV nodal reentry in which the antegrade limb conducted slowly and the retrograde limb conducted rapidly as indicated by AH intervals being greater than HA intervals. All patients demonstrated discontinuous A1A2/H1H2 curves during premature atrial stimulation. The interval from ventricular activation to earliest atrial activation during tachycardia was less than 70 msec. No

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patient had demonstrable retrograde slow pathway conduction or the uncommon form of AV nodal reentrant tachycardia. All six patients had accessory pathways excluded using standard techniques.

The patients underwent general anesthesia and median sternotomy. Reference plaque electrodes for pacing and recording cardiac signals were placed on the left atrium and right ventricular outflow tract. Antegrade and retrograde conduction times were determined. AV nodal reentrant tachycardia was induced with atrial pacing. Atrial epicardial mapping around the AV groove was performed to exclude accessory pathways. The patients were then placed on normothermic cardiopulmonary bypass, and a right atriotomy was made to expose the atrial septum.

A mapping template was designed to map the atrial septum in reference to reproducible atrial landmarks (Figure 1). The epicenter of the template was the compact AV node. The site was identified visually and confirmed by cooling the site with a 5-mm cryoprobe (Frigitronics, Shelton, Conn.) set to 0°C. The discrete area where complete AV block resulted from such cooling was considered the site of the compact node. This area was generally proximal to the central fibrous body and the intersection of the tendon of Todaro with the AV ring. Four arrays of mapping points emanated from this center. Array C passed inferiorly along the tricuspid annulus from the compact node to the coronary sinus. The terminus of the C array was placed immediately anterior to the coronary sinus os. Array T trekked along the tendon of Todaro. Array A passed anteriorly and superiorly along the annulus. The M array divided the sector defined by A and T. Each array was composed of five equidistant points; the distance from each point was defined by anatomic constraints. There was no anatomic overlap between adjacent points. This mapping template did not include the small amount of atrial tissue between the compact AV node and the tricuspid annulus; this small area did not appear to be physiologically important.

Ice mapping was performed in all patients during AV nodal reentrant tachycardia. Array mapping was arbitrarily chosen to begin with C and continue with T, M, and A. The 5-mm cryoprobe was cooled to 0°C. The tip of the probe was then placed on one of the defined points of atrial septum, starting at the most peripheral point in the array. This tissue then was reversibly cooled to create localized conduction delay.

If no change was seen in the tachycardia cycle length after approximately 5 seconds, the probe was removed and advanced to the next point along the array in a centripetal fashion. If the tachycardia cycle length significantly increased in a sustained fashion or the tachycardia terminated, cooling was stopped, and the tissue was allowed to recover. All points of the array were mapped. If termination of the tachycardia was observed at a peripheral point, termination was identical in all of the more central points. Site specificity and reproducibility of tachycardia slowing and termination were confirmed with multiple coolings at a particular point. Cooling at immediately adjacent sites was repeated if alterations of tachycardia were noted. In all patients, separate areas were identified that permitted termination of the tachycardia in either the retrograde or the antegrade limb. Block in the antegrade limb was identified by atrial activation, followed by the absence of ventricular activation. Block in the retrograde limb was identified by ventricular activation, followed by the absence of atrial activity. Antegrade conduction time was the interval between the atrial electrogram and the ventricular electrogram. Retrograde conduction time was the interval between the ventricular electrogram and the atrial electrogram. Cooling of the compact node was the only site that created both antegrade and retrograde AV block during tachycardia and in sinus rhythm. The site may have represented some His bundle area.

![Figure 1](http://circ.ahajournals.org/doi/10.1161/01.CIR.86.3.920)
fibers but because of its effect on the tachycardia included the distal AV node and the tachycardia circuit.

In three patients, activation mapping of the atrial septum during tachycardia was performed using the same template. Atrial electrograms recorded from a bipolar probe (1-mm spacing) from all 25 points were recorded at 100 mm/sec on strip chart paper (Electronics for Medicine) for analysis. Isochrones were constructed, and the site of earliest retrograde atrial activation was determined.

**Results**

The patient population consisted of four women and two men (age range, 11–70 years; mean age, 42.6 years). All had structurally normal hearts except patient 4, who had suffered a previous myocardial infarction. All had normal resting ECGs. No patient had an additional inducible arrhythmia. The mean cycle length of the induced tachycardia was 378±114 msec.

**Localization of Slow Pathway**

Ice mapping identified discrete areas of atrial tissue that, when cooled, terminated the tachycardia in the antegrade or retrograde limbs (Table 1). In four patients, the area of slow pathway block occurred during cooling inferiorly and posteriorly along the tricuspid annulus, the C array. In two other patients, the area of antegrade slow conduction block occurred during cooling superior to the compact node. Cooling at points more centrally located along the array also yielded block in the slow pathway. None of the more centrally located points along these arrays yielded block in the fast pathway until the compact node was reached. Frequently, there was slowing of the tachycardia cycle length with cooling of a site more peripheral to a site that initiated block, but this finding was not consistent.

Block in the slow pathway was very characteristic (Figure 2). There was significant cycle length slowing of the tachycardia attributable exclusively to delay in the atrial-to-ventricular conduction interval (antegrade slow pathway) before termination of the tachycardia. Termination was always associated with retrograde atrial activation without subsequent reentrant ventricular activation. The first sinusal beat after termination of tachycardia was not associated with any prolongation of the PR interval. Thus, antegrade conduction through the “normal” node or, perhaps, the fast pathway did not appear to be affected.

**Localization of Fast Pathway**

Tissue associated with block in the retrograde fast conduction pathway during cooling was identified in all patients (Table 1). The location tended to be more central along the tendon of Todaro. In two patients, central points on dissimilar arrays caused block in the retrograde fast pathway. This may have represented a tangential pathway coursing across the artificial template or a fast pathway exit from the compact AV node that was interposed between the two mapping sites and was cooled sufficiently from either site to be affected. The area of fast pathway block was always distinguishable from the area of slow conduction by several millimeters.

Cooling in the areas of the fast retrograde pathway had a minimal effect on the overall cycle length of the tachycardia, even though there was some slowing of conduction in the pathway (i.e., prolongation of VA interval) (Figure 3). Termination was associated with little or minimal change in the ventriculoatrial conduction interval. It was always manifested as ventricular activation followed by the absence of reentrant atrial activation (Figure 3). The PR interval was only minimally prolonged after tachycardia termination. Cooling the point of fast pathway conduction during sinus rhythm slightly prolonged the PR interval. This could indicate that although antegrade conduction occurred

**FIGURE 2.** Identification of atrial septal tissue involved in the antegrade slow pathway demonstrating the characteristic termination of tachycardia in the slow pathway when an area of critical conduction (area C4 in this case) is cooled. Note the increment in tachycardia cycle length before termination that is due entirely to prolongation of the atrial to the right ventricular (antegrade slow conduction pathway) interval. The tachycardia terminates with block in the slow pathway as noted by an A with no reentrant V. Return of atrial activity shows no prolongation of the AV conduction interval. I, II, and V1 are surface ECG leads. A, left atrial electrogram; RV, right ventricular electrogram. Time intervals between electrograms are given in msec.
via this pathway during normal sinus rhythm, it may not represent the "normal" antegrade pathway. Alternatively, this could have represented activation through the slow pathway; however, there was only a minimal increase in the AV interval.

Mapping of retrograde atrial activation during tachycardia correlated with the location of the retrograde fast pathway as determined by ice mapping in two patients. The area of earliest activation was the T1 region in these two patients. In the one discordant case, the earliest area of atrial activation, the M1 point, was bracketed by areas of functional fast pathway conduction at T1 and A1.

Selective Ablation of Pathways

The first four patients underwent AV nodal modification as described by Cox et al12; the AV node was encircled by ablative cryolesions. This resulted in minimal PR interval prolongation and no retrograde ventriculoatrial conduction. In patients 5 and 6, the areas of antegrade slow and retrograde fast pathways, as determined by mapping, were selectively ablated. In both, the areas of slow conduction were posterior (C4 and C5) and were ablated first. Cryolesions were initially placed along the entire C array, after which tachycardia was no longer inducible. Retrograde fast conduction was still present. There was no change in the PR interval in sinus rhythm. Cryolesions were then placed in the region of fast conduction along the T array. This represented the retrograde fast pathway in both patients and resulted in a 20- and 30-msec increase in the PR interval, which was still normal in duration. Retrograde conduction could not be demonstrated after fast pathway ablation.

Cryoablation then was performed along the A and M arrays. Ablation of the other arrays made no appreciable change in the conduction characteristics except for further prolongation of the PR interval. Because surgical subselective AV nodal modification has not been well characterized, all points located circumferentially around the compact AV node were eventually ablated in all patients, as described by Cox et al.12

Discussion

Longitudinal dissociation of separate pathways within the AV node was first proposed by Mendez and Moe.5 This has been confirmed by many investigators in both animals and humans.2,3,7,9 Eloquent mapping studies in animals have delineated the areas of fast and slow conduction as well as a distal common pathway.8,9 Characterization of the proximal tachycardia circuit has been elusive. Investigations in animals have resulted in mixed conclusions regarding the presence or absence of

<table>
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Letters and numbers signify the array and points of the mapping scheme as shown in Figure 1.

FIGURE 3. Identification of atrial septal tissue involved in the retrograde fast pathway of the circuit. A point 1 cm away from the compact node (area T2 in this case) was cooled. The tachycardia terminated in the retrograde fast limb preceded by minimal change in the cycle length. The termination is manifested as a ventricular electrogram followed by the absence of reentrant atrial activation. In the first two beats after tachycardia termination, there is a mild increase in the AV interval over the baseline value of 150 msec. This did not progress to more significant prolongation, even after the area underwent cryoablation.
an intranodal reentry circuit with an upper common pathway.\textsuperscript{9,10} Constraints of conduction characteristics in smaller animal hearts may necessitate the involvement of atrial tissue. Thus, the tachycardia circuit in animals may not be entirely representative of the situation in humans.

The mammalian AV node is a complex structure, and the anatomic limits are not easily defined.\textsuperscript{6-7,13} The distal region of the AV node is discretely organized and is contiguous with the His bundle. The proximal portion of the AV node, the so-called transitional node, is a heterogenous mixture of atrial myocytes and AV node cells. The nodal boundary with the atrium is not clearly defined. This irregular anatomic arrangement of AV nodal and atrial fibers in the proximal transitional zone node may explain the asymmetry of the AV node in regard to atrial input.\textsuperscript{14} However, this tissue inhomogeneity may allow anisotropy and could be the substrate for reentry.

Clinical observations in humans have been useful but, like animal studies, have been inconclusive regarding the upper turnaround of the tachycardia circuit during AV nodal reentry. Both catheter\textsuperscript{15} and intraoperative mapping studies\textsuperscript{16} have demonstrated multiple-exit sites of retrograde atrial activation from the AV node in patients with dual AV nodal physiology and no extranodal atrioventricular accessory pathways. These findings are consistent with but do not prove the absence of a proximal common pathway. These findings could not rule out varying atrial exit sites proximal to a common pathway. Although activation mapping can localize the retrograde fast pathway by identifying the earliest site of atrial activation, such mapping cannot identify the site of the slow pathway. Without localization of a distinct antegrade slow pathway, an upper common pathway cannot be localized.

It has not been feasible in humans to routinely perform detailed mapping studies; therefore, much of the information available about the human AV nodal reentrant tachycardia circuit has been derived from pacing studies. Josephson and Kastor\textsuperscript{17} demonstrated with an extrastimulus technique that in humans the bulk of the atrium was not required to sustain AV nodal reentrant tachycardia. They were unable to exclude a rim of perinodal atrial tissue as part of the reentrant circuit.

There have been rare instances of 2:1 retrograde block occurring during AV nodal reentrant tachycardia,\textsuperscript{18} but these may represent intra-atrial conduction block.\textsuperscript{19} Jackman et al\textsuperscript{20} recently described a technique in which premature atrial stimuli were delivered during tachycardia in the posterior region of the atrial septum or proximal coronary sinus, and this reset the tachycardia. This suggests that at least part of the atrium constitutes a portion of the reentrant circuit or at least has preferential input into the antegrade slow pathway. McGuire et al\textsuperscript{21} recently reported three patients who had both usual and unusual forms of AV nodal reentrant tachycardia. Although these represent unusual patients, their findings of varying retrograde atrial activation sequences, ventriculoatrial intervals, and tachycardia cycle lengths were best explained by a model of AV junctional reentry inclusive of perinodal atrial tissue without a proximal common pathway.\textsuperscript{21}

In this report, ice mapping localized discrete areas of atrial septum critical to the function of antegrade and retrograde conducting fibers during tachycardia. These were visibly different in location, functionally distinguishable, and separated by tissue that was not critical to maintaining the tachycardia. It is not clear whether the terminology of "pathway" is correct, and it appears unlikely that a discrete bundle, as with accessory pathways, truly exists outside of the compact node. Our data indicate that AV nodal fibers are arranged in an eccentric fashion. Fibers that conduct at a faster rate are directed superiorly toward the tendon of Todaro and the foramen ovale, and slower conducting fibers are directed inferiorly along the tricuspid annulus toward the coronary sinus os. The ice mapping data also indicate that retrograde insertion of fast fibers into atrial tissue occurs just outside the compact AV node as cooling of atrial septal tissue with ice mapping must be close to the compact node to terminate the tachycardia. This insertion site is consistent with data from atrial activation mapping. Conduction slowing and block induced by cooling further away from this atrial insertion could be bypassed by intra-atrial reentry, and the tachycardia cycle length would be virtually unchanged.

The sites at which ice mapping caused block in the slow pathway during tachycardia would indicate that atrial insertion sites of these critical slow fibers are more peripheral from the AV node and run along the tricuspid annulus. They appear to have a broader insertion site, ranging from areas several millimeters posterior to the compact AV node to sites next to the coronary sinus os. Such locations of insertion sites would also explain why cooling at sites closer to the AV node causes block in the slow pathway. Although these fibers appear to play a critical role during tachycardia, they do not appear to be necessary for normal antegrade conduction. Cooling and cryoablation of these pathways had no impact on the PR interval during sinus rhythm.

The data suggest a physiological organization to AV nodal fibers, at least in patients with AV nodal reentrant tachycardia. The so-called fast and slow "pathways" probably represent the fastest of the fast fibers and the slowest of the slow fibers, respectively, which are likely to run at opposite edges of the AV node. The fast ones are oriented superiorly, whereas the slow ones are oriented inferiorly. The fibers in-between these two pathways most likely participate in normal antegrade conduction through the node but do not contribute to sustenance of tachycardia. This speculation is supported by the observation that cooling at points between the critical fast and slow pathways did not alter tachycardia. The tachycardia circuit probably circumvents these "intermediate" fibers. Although the connection between the two fiber sets remains unclear, we speculate that this connection is a dynamic process that is determined by anatomic constraints and conduction characteristics of the atrial septum. The absence of a discrete upper turnaround pathway is supported by our inability to terminate tachycardia at sites intermediate between the critical slow and the fast pathways. Cooling of these intermediate sites merely required that the retrograde wavefront in the septum enter the slow pathways via a slightly more circuitous route, making virtually no difference in tachycardia cycle length. Whether this wavefront entering the slow pathway actually propagates through atrial muscle or is transmitted transversely across transitional nodal fibers may be difficult to dis-
tistinguish. As the actual separation of transitional nodal fibers from atrial tissues may not be clear cut, this distinction may have no clinical or physiological importance.

The above description of the reentry process suggests a specific model for AV nodal reentrant tachycardia, which is illustrated in Figure 4. AV nodal fibers emanate from the compact node in a fanlike fashion that spreads from the tricuspid annulus beyond the tendon of Todaro. It is unclear how much further superior these fibers may extend. The lower turnaround of the tachycardia circuit occurs at the compact node. The impulse then propagates retrogradely via the fast pathways located superiorly along the compact node and exists into the atrial septum near the tendon of Todaro. The septal wavefront cannot enter the slow pathways at sites near the compact node due either to anatomic absence of connection or to functional refractoriness of these fibers. The wavefront enters more distally into the slow pathway located along the tricuspid annulus near the coronary sinus os and propagates along the pathway into the compact AV node. This model is consistent with all the findings of the present report as well as with those previously reported by others in AV nodal reentrant tachycardia.

Ice mapping has relevance to the current therapeutic strategies of catheter ablation with radiofrequency current. The proximity of fast fiber atrial insertion site to the compact AV node may explain why radiofrequency current lesions placed in this area are associated with an increased incidence of heart block and PR prolongation. Radiofrequency current lesions directed at ablating the slow fiber more posteriorly would be expected to have a lower incidence of AV nodal conduction abnormalities as they can be placed well away from the compact AV node. Ice mapping is able to terminate the tachycardia in both the fast and slow fibers without significantly altering antegrade AV conduction. It is possible that limited lesions could be made to interrupt fast pathway conduction but leave a relatively normal PR interval. Successful ablation probably is associated with selective damage of AV nodal fibers of either set. Which of these fibers constitutes the normal antegrade AV nodal pathway remains unknown.

Conclusions

Ice mapping of the reentrant circuit in AV nodal reentrant tachycardia demonstrates that slowly conducting fibers critical to maintaining reentry are situated away from the fast conducting fibers. The atrial insertion of these slow fibers is more remote from the compact node than is the insertion of the fast pathways. The identification of nonessential atrial septal tissue interposed between the two critical areas of conduction suggests that the proximal turnaround is a dynamic rather than fixed circuit and argues against the concept of a proximal common pathway.

References

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