The Nodoventricular Mahaim Pathway: An Endangered Concept?

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It is unusual, if not unprecedented, to have three articles on the same topic in two consecutive issues of Circulation, especially relating to a relatively obscure and infrequently observed electrophysiological problem. This can only be a reflection of the intense interest and passion that students of arrhythmias have always had for the preexcitation syndromes and their variants. Our understanding of clinical arrhythmias has been greatly enhanced by both anatomic-pathological observations and electrophysiological observations. Nonetheless, the correlation between anatomic and electrophysiological observations is frequently problematic. The electrophysiologist will speculate on the variable anatomic structures that could be invoked to explain the observed phenomena, whereas the anatomist ponders the function of the structures observed under the microscope.

Mahaim et al.2,3 initially described anatomic connections between the normal atrioventricular (AV) conduction system and the ventricles, and others subsequently verified and expanded on these observations.5-7 Wellens5 was the first to invoke the concept of the nodoventricular fiber to explain intracardiac electrophysiological phenomena, and others subsequently followed suit.9-13 The essence of this issue was as follows. The “typical” accessory AV pathway has a relatively short conduction time and exhibits “all or none” conduction when stressed by progressively premature extrastimuli or rapid rates. Some accessory pathways, however, have impressively long conduction times and exhibit rate-dependent prolongation (“decremental”) conduction that greatly resembles conduction observed over the normal AV node. It was imminently logical to assume that these atypical pathways derived their unusual characteristics by virtue of originating from the normal AV node. This explanation was so elegant and simple that the anatomic observations of Mahaim became linked with the electrophysiological observations of Wellens and many others to become firmly established in the electrophysiological literature. Anderson and coworkers2 subsequently suggested a classification of Mahaim fibers based on anatomic insertion sites with nodoventricular pathways connecting AV node to ventricle, nodo-fascicular pathways joining AV node to the fascicular system, and fasciculoventricular pathways linking the distal conduction system to ventricle.

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These concepts went largely unchallenged until the era of correctional interventions, when the acid test of the concept, namely, to localize and ablate the offending pathway, was carried out. Gillette et al.20 initially described a group of patients with clinical features suggesting nodoventricular conduction that were ultimately related to an accessory pathway ablated operatively at the right parietal AV groove. Others subsequently verified intraoperatively that patients with clinical features thought to be typical of nodoventricular Mahaim conduction usually had atriofascicular pathways linking the atrium at the parietal tricuspid ring to the distal conduction system of the right ventricle or, less frequently, AV pathways at the parietal tricuspid AV groove with decremental properties.21-24 The inertia of the well-established concept was reflected by the difficulty in getting some of these publications25,26 through the review process in the face of skeptical reviewers (P. Gillette, personal communication). The impact of these entrenched concepts extended into the catheter ablation era, which essentially began with the use of high-energy DC shock applied to the AV node region to ablate this structure.27-31 Two groups of investigators independently reported that AV node ablation cured tachycardia in patients with “nodoventricular” connections.27,28 After AV node ablation, these investigators observed that patients were left with a fully preexcited QRS with a long conduction time, decremental conduction, and no residual retrograde conduction. Interestingly, they concluded that this relatively destructive lesion resulted in loss of AV node conduction but spared the portion of the node harboring the nodo-fascicular fiber as opposed to the more probable but heretical conclusion that the AV node and the accessory pathway were in fact anatomically distinct.

Two articles in this issue of Circulation and one in the June issue describe the experience of large, well-established groups with radiofrequency ablation of “nodoventricular” pathways.32-34 Although the emphasis in each article is somewhat different, all essentially confirm that in the large majority of patients, a clinical and electrophysiological presentation previously ascribed to nodoventricular Mahaim pathways is related to atriofascicular connections. These extend from a variable location on the parietal tricuspid AV ring to insert more apically via a long and electrically insulated tract to the apical right ventricular region near or at the distal right bundle branch. These pathways could be ablated at any point along their length but were generally ablated more...
readily at the AV ring near their origin. The unique characteristics of atriofascicular pathways make them more difficult to localize by the usual techniques used in mapping of conventional AV pathways. Since the distal insertion site is not at the AV ring, a search for the earliest ventricular activation during preexcitation at the ring is inappropriate and futile. Atrial “pace mapping” at the tricuspid ring searching for a site providing the shortest A-to-delta interval was shown to be technically difficult and relatively imprecise. Mapping the atrial insertion site during retrograde conduction is not applicable since most (all?) of these pathways are incapable of retrograde conduction. The original observation of McClelland et al.\(^1\) that a potential similar to a His potential can be recorded at the tricuspid AV ring (and followed through to the insertion site in the ventricle) provides the best “target” for ablation. This atriofascicular potential can invariably be located at the tricuspid ring even if preexcitation is not evident on the surface ECG.\(^3\) Cappato et al.\(^5\) emphasize a unique mode of mapping in which a deliberate attempt is made to obtund conduction over the pathway by catheter manipulation (“bump mapping”) to verify the successful ablation site. This is a novel approach, especially since most investigators try to avoid injuring the pathway during mapping for fear of losing the target for ablation for prolonged periods. The limitations of this approach are reasonably expressed by these investigators, and the ultimate utility of this technique remains to be verified.

The time has come to abandon the vague and inappropriate jargon applied to entities involving accessory pathways with long conduction times and rate-dependent conduction, including “Mahaim tract” and “Mahaim physiology.” Atriofascicular pathways are associated with the following characteristics: \(^2\)-\(^4\), \(^2\)-\(^9\), \(^3\)-\(^1\):

1. Preexcitation on the routine 12-lead ECG is absent to minimal.
2. The preexcited pattern elicited by pacing or during preexcited tachycardia has left bundle branch block morphology and generally a leftward axis. This is not surprising, since the distal insertion site is at or near the right bundle branch terminus.
3. Pac ing from right atrial sites provides more marked preexcitation and a shorter stimulus-to-QRS interval than pacing from left atrial sites at comparable cycle lengths.
4. The pathway exhibits rate-dependent or “decremental” conduction with prolongation of the A-to-delta interval with atrial extrastimuli or incremental atrial pacing.
5. Retrograde conduction over the pathway is usually (always?) absent.
6. The most commonly observed clinical tachycardia is antidromic tachycardia proceeding antegrade over the atriofascicular pathway and retrogradely over the normal AV conduction system.
7. Associated AV node reentry and other accessory AV pathways are observed relatively frequently.
8. During intracardiac recording, earliest ventricular activation during preexcitation is recorded at the right ventricular apical catheter with activation at or before the onset of the QRS on the surface ECG.

“AV” pathways in the right parietal tricuspid region with decremental properties may have characteristics similar to the above and are observed less frequently. The essential difference is the insertion of the pathway into the right ventricle at the level of the annulus, as is observed for conventional AV pathways. This is readily appreciated during electrophysiological testing, since the right ventricular apical electrogram during maximal preexcitation occurs well past the onset of the QRS on the surface ECG.

Is there a clinical correlate of the true nodoventricular pathway as described by Mahaim and others? A convincing electrophysiological demonstration of such a structure would involve the observation of preexcited tachycardia using the pathway without participation of the atria, an observation infrequently made in the literature.\(^1\),\(^3\) Gmeiner and coworkers\(^1\) described a case with electrophysiological findings suggestive of a nodoventricular pathway with the patient subsequently coming to autopsy. A nodoventricular structure was observed that was postulated to be the anatomic substrate for the findings seen. A similar case was described by Motte et al.\(^1\) However, accessory pathways around the AV ring can be very difficult to find if the anatomist is not alerted to the general vicinity of the pathway, and it is conceivable that such a pathway may have been missed. The electrophysiological data presented by Grogin et al.\(^1\) to support the existence of nodofascicular fibers are not without controversy. In their case 5, loss of preexcitation simultaneously with the slow pathway by ablation at a midseptal site supports the existence of the nodofascicular structure. However, inspection of their Fig 4 shows the earliest ventricular activation during preexcited tachycardia to be at the His bundle site and not at the right ventricular apex. This clearly cannot be a nodofascicular pathway, for which the reverse would be true. It may be a nodoventricular pathway inserting into the ventricular summit, but the alternative explanation of a midseptal accessory pathway with decremental properties is not disproved with the known potential for such pathways to be near the AV node. In addition, their case 6 is substantially unique in that significant decrement is not observed, the His-ventricular interval is short and constant, there is little variance in a minimally preexcited pattern, and the pathway does not participate actively in clinical tachycardia. This is more typical of the fasciculoventricular pathway described by Gallagher et al.\(^1\) On the basis of evidence to date, it is not possible to be definitive about the existence of functional nodofascicular fibers. However, it is certainly accurate to say that the great majority of patients presenting with decremental pathways with a long conduction time have either atriofascicular or AV pathways originating in the parietal tricuspid ring.

It is interesting to speculate on the anatomic substrate of the atriofascicular pathway. The initial suggestion of Gallagher et al.\(^1\) that the structure, if present, may represent a secondary or congenitally displaced AV node and AV conduction system has considerable appeal. As ably discussed by McClelland et al.,\(^2\) there is reasonable anatomic evidence for the existence of AV node-like structures in the parietal tricuspid AV ring. The elegant electrophysiological observations of McClelland et al. suggest a structure in the tricuspid AV ring with slow, rate-dependent conduction, intrinsic automaticity, and connection to a rapidly conducting insulated tract generating a “His-like” potential. This
nodelike structure blocks with adenosine and is relatively superficial (endocardial), since it readily loses conduction with catheter pressure. It is only a matter of time before definitive anatomic assessment of this hypothesis will be available.

References


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