Radiofrequency Catheter Ablation of Atriofascicular and Nodoventricular Mahaim Tracts

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Background Several mechanisms have been proposed to explain the pathogenesis of tachycardia in patients with Mahaim tracts. The tachycardia may involve antegrade conduction over an atriofascicular pathway with decremental properties or a nodoventricular pathway.

Methods and Results We report six patients with recurrent episodes of preexcited tachycardia with findings consistent with "Mahaim tract" conduction. All patients exhibited decremental antegrade preexcited conduction with atrial pacing and a preexcited tachycardia with initial activation of the proximal right bundle branch. In four patients (group 1), atrial premature complexes (APCs) induced at the tricuspid annulus just after the inscription of the septal atrial electrogram and during left bundle branch block preexcited tachycardia advanced the next preexcited ventricular complex. In these patients, discrete Mahaim potentials were observed over the right anterolateral or lateral tricuspid annulus. Two patients (group 2) had evidence of dual atrioventricular nodal conduction. APCs during left bundle branch block tachycardia just after inscription of the septal atrial electrogram failed to advance the next ventricular complex with similar preexcited morphology, and no Mahaim potentials could be recorded from the tricuspid annulus. In group 1 patients, application of radiofrequency energy to sites recording the Mahaim potentials resulted in tachycardia cure. For patients in group 2, selective slow atrioventricular nodal pathway ablation in the midseptal region resulted in complete ablation of both the slow atrioventricular nodal pathway and Mahaim conduction in two patients.

Conclusions Mahaim tachycardia can be due to atriofascicular pathways, which may be ablated over the right tricuspid annulus, or to septal pathways, which may arise from the slow atrioventricular nodal pathway in patients with dual atrioventricular nodal physiology. In the latter circumstance, successful ablation is achieved by placing the lesion in the midseptal region. (Circulation. 1994;90:272-281.)

Key Words • radiofrequency • ablation • Mahaim tracts • catheters • tachycardia

In 1937, Mahaim and Winston1 described tracts connecting the atrioventricular node and ventricular myocardium as well as discrete connections from the fascicles to the ventricles. Mahaim conduction is characterized by gradual increases in the atrioventricular interval simultaneous with the development of a left bundle branch block and shortening of the HV interval in response to atrial overdrive pacing. In addition, the right bundle branch potential precedes the His deflection during preexcited complexes. Preexcited tachycardia is manifested by antegrade conduction over the pathway to the right bundle branch with retrograde activation of the His bundle, atrioventricular node, and atrium.

Controversy exists relative to the precise anatomic counterparts of patients with "Mahaim physiology." Earlier studies were interpreted as showing a nodoventricular origin of these pathways with insertion into either the right bundle branch or the right ventricle.2,3,4 More recently, evidence from electrophysiological studies, surgery, and catheter ablation has definitely established that right atriofascicular fibers crossing the tricuspid annulus serve as the basis for typical Mahaim conduction in most, if not all, of these patients.4,5,6,7 The purpose of the present report was to review our experience with radiofrequency catheter ablation as a cure of patients with Mahaim tracts. In addition, we demonstrate for the first time evidence of the existence of septal (probably nodoventricular) bypass tracts with Mahaim physiology using radiofrequency catheter techniques.

Methods

Patients

Over 2 years, six patients with Mahaim tachycardia were studied. All patients had symptomatic, documented wide complex tachycardia. They ranged in age from 27 to 55 years (Table). One or more antiarrhythmic drugs were ineffective in all patients. Echocardiograms revealed normal hearts in four of six patients. Patient 2 had Ebstein's anomaly and an atrial septal defect, and patient 3 had Ebstein's anomaly.

See p 636

Electrophysiological Testing

All patients underwent electrophysiological testing while in the fasting state after informed written consent was obtained according to the guidelines of our institutional review board. Patients were sedated with midazolam and fentanyl. Quadrupolar electrode catheters were placed in the right ventricular apex, the His bundle position, the high right atrium, and the coronary sinus. Signals were filtered using a band-pass of 30 to 250 Hz. Programmed electrical stimulation was performed using atrial and ventricular overdrive and extrastimulus pacing and premature atrial stimulation from the right atrial free wall during tachycardia. Retrograde atrial conduction during both
ventricular pacing and tachycardia was assessed by mapping the atrial activation sequence over the tricuspid annulus.

Mapping was performed using either an 8F EP Technologies or a Mansfield steerable catheter. The position of the catheter tip was confirmed in the left anterior oblique and right anterior oblique projections. The tricuspid annulus was carefully mapped for the presence of a Mahaim potential in sinus rhythm and during tachycardia. If a discrete potential could be found from the femoral vein approach (mapping both the atrial and the ventricular sides of the tricuspid annulus), then an attempt was made to ablate this area. Failing successful ablation from the femoral vein, ablation was attempted from the right subclavian vein (to approach the ventricular side of the tricuspid annulus). Atrial pace-mapping over the tricuspid annulus was not performed. Ablation was accomplished using unmodulated radiofrequency energy (500 kHz, 40 to 50 W) delivered via a custom-designed generator (EP Technologies). Radiofrequency energy was delivered between the distal electrode of the ablation catheter and a large-diameter skin patch electrode. Postablation electrophysiological testing was performed with both programmed atrial and ventricular as well as overdrive pacing before and during isoproterenol infusion.

Criteria for the presence of Mahaim fiber conduction included (1) baseline antegrade preexcitation with left bundle branch block morphology or normal conduction, (2) increasing ventricular preexcitation associated with an increasing AV interval with a short HV interval with atrial pacing at increasing rates (in patient 6, preexcitation remained fixed despite progressive increases in the AV interval), and (3) a reciprocating tachycardia with the same left bundle branch block preexcitation morphology and the right bundle branch electrogram preceding His bundle activation during antegrade preexcitation. The presence of dual atrioventricular nodal physiology was supported by an increase of 50 milliseconds in the AH interval in response to a 10-millisecond decrement in atrial extrastimuli during atrial pacing. This occurred in the absence of Mahaim conduction in patient 5 and in the presence of constant minimal preexcitation in patient 6.

Results

Group 1 Patients

Four patients (group 1) demonstrated evidence of an atriofascicular pathway. All four showed distinct Mahaim potentials along the tricuspid annulus (Fig 1). In addition, right atrial extrastimuli induced after inscription of the septal atrial electrogram always advanced the next preexcited ventricular electrogram and subsequent atrial electrogram with the same retrograde activation sequence (Fig 2). This finding suggests that the mechanism of the tachycardia is unlikely to be due to atrioventricular nodal reentry. In addition to a Mahaim
Fig 2. Tracings of insertion of premature atrial complex during wide complex tachycardia. Right atrial extrastimulus induced after inscription of septal atrial electrogram advances following QRS complex with the same preexcited morphology and subsequent retrograde atrial activation sequence. (See text for discussion.) Surface leads are V₁, I, and aVF. Intracardiac leads are the coronary sinus distal and proximal (CSd and CSp), His bundle electrograms, and right ventricular apex (RV). a indicates septal atrial electrogram; v, ventricular electrogram.

Fig 3. Tracings of initiation of preexcited left bundle branch block tachycardia in patient 5. Premature atrial extrastimulus conducts over the slow atrioventricular nodal pathway and is associated with a typical atrioventricular nodal echocardiographic (Aₚ) complex without preexcitation. The next complex is preexcited and may either represent antegrade conduction over the Mahaim with retrograde conduction over the atrioventricular node or result from bystander Mahaim conduction and atrioventricular nodal reentry. Surface leads are V₁, I, and aVF. Intracardiac leads are the high right atrium (HRA); coronary sinus distal and proximal (CSd and CSp); His bundle distal, mid, and proximal (His d, His m, and His p); and right ventricle (RV). a indicates atrial echocardiographic beat from atrioventricular nodal reentry.
pathway, patient 3 had a right posteroseptal accessory pathway. This patient had both an antidromic tachycardia using the Mahaim in the antegrade direction and the posteroseptal pathway in the retrograde direction and a narrow complex tachycardia using the atrioventricular node in the antegrade direction and the posteroseptal pathway in the retrograde direction. After successful ablation of the posteroseptal pathway, the patient exhibited the features associated with a typical atriofascicular Mahaim fiber.

In all group 1 patients, a discrete Mahaim potential was recorded at the tricuspid annulus. The location of these Mahaim potentials was anterolateral in patients 1 and 2 and lateral in patients 3 and 4. The ablation was performed over the atrial side of the tricuspid annulus in patients 1 and 2, the ventricular side of the tricuspid annulus in patient 3, and at the ventricular insertion in patient 4. Loss of preexcitation resulted in a right bundle branch block in 1 patient (patient 1) that later resolved. During application of radiofrequency energy over the area of Mahaim potential, episodes of an irregular Mahaim or atrial tachycardia were inscribed before tachycardia termination in patient 1. The Mahaim tract was successfully ablated in all patients at the site where the Mahaim potential was recorded.

**Group 2 Patients**

Two patients (group 2) demonstrated evidence of nodoventricular pathways, and no discrete Mahaim potential could be found along the tricuspid annulus. During sinus rhythm, these patients were minimally preexcited. These patients demonstrated dual atrioventricular nodal conduction in the antegrade direction.

Representative examples of dual AV nodal physiology are shown for patients 5 and 6. Patient 5 displayed evidence of typical dual atrioventricular nodal physiology. Fig 3 shows induction of a typical atrioventricular nodal echo complex followed by initiation of preexcited tachycardia. The His bundle deflection is not clearly recorded during preexcited tachycardia, and the retrograde VA interval is 110 milliseconds. During the preexcited tachycardia, critically timed atrial premature complexes (APCs) resulted in a switch from the preexcited tachycardia to typical atrioventricular nodal reentrant tachycardia. In Fig 4, during preexcited tachycardia (first three complexes), the VA interval increased (120 milliseconds) and a retrograde His deflection is clearly inscribed after ventricular activation. The retrograde HA interval during preexcited tachycardia is longer (40 versus 25 milliseconds) than the HA interval during narrow complex tachycardia. In addition, the
narrow complex tachycardia is associated with a shorter cycle length (380 versus 410 milliseconds) compared with the wide complex tachycardia. These observations suggest that the His-Purkinje system is involved in the tachycardia circuit during the wide complex tachycardia. If the mechanism of tachycardia was atrioventricular nodal reentry with a bystander Mahaim tract, then the retrograde HA interval and cycle length should be identical for narrow and preexcited tachycardia. In Fig 3, the cycle length of the wide complex tachycardia is 380 milliseconds, similar to the narrow complex tachycardia recorded in Fig 4. Thus, this wide complex tachycardia may represent atrioventricular nodal reentry with Mahaim bystander conduction. Fig 5 shows that an APC induced just after inscription of the septal atrial electrogram fails to advance the next ventricular complex. The VA interval (Fig 5) was greater than in previous wide complex tachycardias, which may represent retrograde block in the right bundle branch and transeptal activation of the left bundle branch. This explanation is supported by the finding of transient right bundle branch block after ablation (see Fig 6). In addition, no Mahaim potential was recorded along the tricuspid annulus. Delivery of radiofrequency energy to the midseptal region resulted in ablation of both the slow pathway and Mahaim conduction (see Fig 6). The bundle branch block was transient.

Patient 6

Fig 7 shows evidence of dual atrioventricular nodal conduction in patient 6. Critically timed APCs result in echo beats or tachycardia. Note that minimal preexcitation was present both during sinus rhythm and during pacing at an atrial cycle length of 600 milliseconds. In Fig 7B, a critically timed APC is associated with a marked increase in the AH interval with no change in the preexcited ventricular complex. These findings are most consistent with a Mahaim tract originating from the distal common pathway of an atrioventricular nodal reentry circuit. Either an atriofascicular or a nodofascicular Mahaim tract originating from the slow atrioventricular nodal pathway would be expected to show varying degrees of fusion in response to atrial extrastimuli. An interesting pattern of response was discerned in response to atrial pacing during the atrial diastolic cycle (Fig 8). Critically timed APCs induced after inscription of the septal atrial electrogram resulted in advancement of the next electrogram with conduction block in the Mahaim pathway (Fig 8). Earlier APCs resulted in prolongation of conduction over the slow pathway with retention of Mahaim pathway conduction. In addition, the HA interval during preexcited complexes is identical to that for normal complexes. These findings support atrioventricular nodal reentry with the Mahaim as a bystander. Initial attempts were made to ablate the fast
atrioventricular nodal pathway. One radiofrequency application just inferior to the His bundle recording site revealed PR prolongation and persistent preexcitation (Fig 9B). There was no evidence of retrograde VA conduction, and no tachycardia could be induced. The anteroseptal site was chosen because early in our experience with ablation of atrioventricular nodal reentrant tachycardia, the anteroseptal region (fast pathway) was the initial target used for ablative lesions. In this patient, after continued observation in the laboratory, the PR interval returned toward baseline (Fig 9A) and an additional radiofrequency lesion was placed in the midseptal area, resulting in prompt and complete loss of preexcitation (Fig 9C). These findings were interpreted as resulting from damage to both fast and slow atrioventricular nodal pathways.

This patient showed a pattern of preexcitation that is a slight variant of Mahaim conduction since the putative Mahaim pathway arises from the node distal to the final common pathway. Fast pathway ablation prolonged the PR interval but preserved the preexcited pattern. Only after application of radiofrequency

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**Fig 6.** Tracing of atrial extrastimulation after catheter ablation showing atrioventricular nodal block without slow pathway conduction or Mahaim conduction. Right bundle branch block pattern proved to be transient in this patient. A indicates atrial paced complex. Surface leads are I, II, and aVF. Intracardiac leads are the coronary sinus distal and proximal (CSd and CSP), high right atrium (HRA), and His bundle proximal (His p).

**Fig 7.** Tracings of dual atrioventricular nodal physiology in patient 6. At a drive cycle length of 600 milliseconds, shortening the atrial extrastimulus by 20 milliseconds (7A, 290 milliseconds; 7B, 270 milliseconds) results in switch from fast to slow pathway conduction with a similar degree of preexcitation. (See text for discussion.) Surface leads are V1, I, II, and aVF. Intracardiac leads are the coronary sinus distal and proximal (CSd and CSP); high right atrium (HRA); and His bundle distal, mid, and proximal (HBd, HBm, and HBp).
energy to the midseptal region did preexcitation completely disappear.

Discussion

Main Findings

The precise origin and cardiac course of “Mahaim” fibers remain controversial. Earlier studies were interpreted to show that these fibers arose from the atrioventricular node and inserted into or near the right bundle branch. More recent observations have suggested that the majority, if not all, of Mahaim tracts are atriofascicular. In our study, we provide evidence that the origin of the Mahaim tracts may be from either the slow atrioventricular nodal pathway or the distal final common pathway in some patients with atrioventricular nodal reentrant tachycardia and Mahaim tracts.

In our studies, we described two patients (patients 5 and 6) with both atrioventricular nodal reentry and Mahaim tract conduction properties. Both patients showed classic dual atrioventricular nodal conduction and evidence of atrioventricular nodal reentry. In one patient (patient 5), premature atrial or ventricular stimulation readily changed narrow complex tachycardia into preexcited tachycardia, and vice versa. In addition, the HA interval and tachycardia cycle length were shorter during narrow complex tachycardia compared with preexcited tachycardia. A single application of radiofrequency energy resulted in simultaneous ablation of both slow pathway and Mahaim conduction. We believe that these observations are best explained by the following schema (Fig 10, top). The Mahaim tract originates from the slow pathway responsible for atrioventricular node reentry. During wide complex preexcited tachycardia, the circuit involves antegrade conduction over the Mahaim tract and right bundle branch with retrograde conduction over the His bundle and atrioventricular node. The narrow complex tachycardia is due to typical atrioventricular nodal reentry.

Another patient showed atrioventricular nodal reentry and a variant form of Mahaim tract conduction that was manifested by progressive prolongation of the AV interval without change in the pattern of preexcitation. Typical atrioventricular nodal echos, without preexcitation complexes, could be achieved by APCs during preexcited tachycardia. Because the HA was identical between narrow complex and preexcited complexes, it is believed that in this patient the tachycardia mechanism is atrioventricular node reentry with a bystander Mahaim. Of great interest was the finding that radiofrequency application over the anterior septum (fast path-
FIG 9. ECGs from patient 6 in preexcited baseline state (A), after ablation in anteroseptal space (fast pathway) (B) showing a preexcited QRS complex with a longer PR interval, and after midseptal ablation (C), which shows a long PR interval but normal QRS complex. (See text for discussion.)
way) resulted in PR prolongation but with retention of the preexcited pattern. Radiofrequency lesions applied to the midseptum after spontaneous return of the baseline pattern abolished preexcitation. We believe that the most reasonable explanation of these findings is described in Fig 10, bottom. Our findings suggest that the Mahaim tract originates from the final common pathway of the atrioventricular nodal reentrant circuit or from the His bundle with insertion into the proximal right bundle branch. This schema explains the relatively narrow preexcited QRS as well as the lack of change in preexcited QRS in response to atrial overdrive or premature stimulation. Moreover, a selective lesion placed in the midseptal region, which abolished preexcitation, suggests that the Mahaim tract courses from the atrioventricular node rather than the His bundle through the central fibrous body and connects to the proximal right bundle branch by coursing over the midseptal area.

Observations of Others

Our hypothesis is consistent with the observations of Gallagher.\(^3\) He described three patients with Mahaim tracts who were cured by dissection of the posteroseptal space. In contrast, Gillette and coworkers\(^4\) suggested that Mahaim tracts were due to decrementally conducting right-sided accessory pathways. Furthermore, Klein and coworkers\(^5\) reported two patients with Mahaim conduction who had right free wall atrioventricular pathways with decremental conduction during cardiac surgery. One of their patients had a narrow complex atrioventricular nodal reentrant tachycardia that converted to a left bundle branch block Mahaim tachycardia with a premature ventricular complex (PVC). They hypothesized that during atrioventricular nodal reentry, concealed retrograde penetration of the accessory pathway prevented bystander participation. The PVCs presumably penetrated the fast pathway during atrioventricular nodal reentry and preexcited the atrium and antegrade slow pathway, terminating atrioventricular nodal reentry but initiating antidromic tachycardia antegradely via the Mahaim pathway. The cycle length slowed with the development of the wide complex tachycardia. This is similar to patient 5, in whom there was an increase in cycle length when there was a change from narrow complex to wide complex tachycardia, indicating His-Purkinje involvement in the tachycardia circuit. However, we were able to ablate the Mahaim pathway in the posteroseptal region (slow atrioventricular nodal pathway), not on the tricuspid annulus.

Tchou et al\(^14\) added greatly to our understanding of Mahaim physiology. They stressed the importance of preferential right versus left atrial inputs into the Mahaim tracts. They showed that right atrial stimuli introduced after insertion of the septal atrial electrogram advance the next preexcited QRS. These findings demonstrate that the Mahaim tract is a component of the tachycardia independent of atrioventricular nodal reentry. In patient 5, with typical Mahaim physiology (and atrioventricular nodal reentry), right atrial premature stimuli failed to advance the preexcited tachycardia. Conceivably, stimulation from the region of the coronary sinus could have advanced the tachycardia.\(^5\) In patient 6, right atrial stimuli advanced the QRS but abolished the preexcited complex. This finding is explained on the basis of atrioventricular nodal reentry with a bystander Mahaim tract.

More recently, information gained during catheter ablation has contributed to our understanding of Mahaim pathway function. Haissaguerre and coworkers\(^6\) reported three patients whose Mahaim fibers were ablated with DC energy. They used ventricular pace-mapping and activation times during tachycardia to ablate the ventricular insertion of the pathway. Klein and coworkers\(^7\) reported using radiofrequency energy to ablate Mahaim pathways in four patients. Three patients had an atriofascicular connection, and one had an atrioventricular connection. The atrial insertion sites were right lateral (one), right posterolateral (two), and right posterior (one), whereas the ventricular insertion sites were into the distal right bundle branch (three) and posterolateral right ventricle (the patient with an atrioventricular connection). One patient in their study had evidence of atrioventricular nodal reentry, but none of the four patients had bystander Mahaim participation as the mechanism of tachycardia. Atriofascicular pathway mapping was done by delivering premature atrial stimuli during the tachycardia to advance the ventricular activation and stimulus to delta wave mapping but not by recording discrete Mahaim potentials. We were
able to record a Mahaim potential in the four patients with atriofascicular pathways and used this as a target for ablation.

Conclusions

Our observations show that patients with Mahaim tracts may have atriofascicular pathways that traverse the tricuspid annulus. These pathways may be best localized by finding discrete Mahaim potentials. We also showed that patients with Mahaim physiology may have Mahaim tracts that arise from either the slow atrioventricular nodal pathway or the atrioventricular node proper. Distinction between right free wall atriofascicular and nodofascicular pathways during study is of paramount importance. We found that the following features suggest the presence of a nodovenetric Mahaim tract: (1) presence of dual atrioventricular nodal conduction with ready interchange between normal and preexcited complexes; (2) premature extrastimuli delivered from the anterior or lateral right atrial wall near the tricuspid annulus during inscription of the septal atrial electrogram that fail to advance the next preexcited complex; and (3) the inability to record discrete Mahaim potentials over the tricuspid annulus (this finding may also reflect technical problems in successfully recording this potential). These findings suggest the presence of dual atrioventricular nodal physiology with the Mahaim tract originating from the slow pathway (or atrioventricular node) and suggest initial application of radiofrequency lesions in the mid-septal region.

References

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