Radiofrequency Catheter Ablation of Mahaim Fibers at the Tricuspid Annulus

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Background. The purpose of this study was to test the feasibility of radiofrequency catheter ablation of Mahaim fibers at the tricuspid annulus.

Methods and Results. Four patients who fulfilled criteria for having Mahaim fibers and preexcited reciprocating tachycardia underwent radiofrequency catheter ablation. Three patients had atriofascicular connections, and one patient had an atrioventricular connection. The mean age was 27 years (age range, 11–48 years). All patients had highly symptomatic tachycardias, producing syncope in one patient and presyncope in the remaining three patients. Symptoms were present for a mean of 13 years (range, 4–23 years). All pathways conducted only anterogradely, and preexcitation resulted in a left bundle branch block QRS morphology. Adenosine caused block in the accessory pathway in the three patients in whom it was tested. The stimulus to delta interval increased by 75 msec (range, 35–90 msec) during rapid atrial pacing. The atrial insertion of the Mahaim fiber was in the right lateral atrium in one patient, right posterolateral atrium in two patients, and right posterior atrium in one patient. The ventricular insertion was in the distal right bundle branch in three patients and in the posterolateral right ventricle near the tricuspid annulus in the patient with an atrioventricular connection. Stimulus to delta wave mapping was used to help localize the atrial insertion of the atriofascicular connections. A mean of 15 radiofrequency pulses (range, 10–19 pulses) delivered to the tricuspid annulus in the posterior to lateral regions eliminated accessory pathway conduction in all patients. No complications occurred. Tachycardia did not recur during a mean follow-up of 8 months (range, 2–15 months).

Conclusions. Radiofrequency current applied to the tricuspid annulus can safely eliminate tachycardia in patients with Mahaim fibers. (Circulation 1993;87:738–747)

KEY WORDS • tachycardia, reciprocating • Mahaim fibers • accessory pathways • ablation, catheter, radiofrequency

The term “Mahaim fiber” has been used to refer to accessory pathways thought to originate from the atrioventricular node and insert at or adjacent to the distal right bundle branch (nodofascicular fiber) or the ventricle near the tricuspid annulus (nodoventricular fiber).1–5 Recently, several studies have demonstrated that these pathways usually do not originate from the atrioventricular node but instead originate from the atrial free wall.6–9 Properties of these unique accessory pathways include conduction only anterogradely, participation as the anterograde limb in antidromic atrioventricular reciprocating tachycardia, decremental conduction (conduction delay at increasing rates), and right-sided locations so that preexcited QRS complexes have a left bundle branch block morphology.

Therapeutic approaches to patients with Mahaim fibers have included pharmacological therapy.10 His bundle ablation,11 surgical ablation of the accessory pathway,8,9 direct current catheter ablation of the ventricular insertion of the accessory pathway,12 and radiofrequency catheter ablation of the atrial insertion of an atriofascicular connection in a single patient.13 In the present report, we describe a series of four patients whose Mahaim fibers were eliminated by radiofrequency catheter ablation targeted to the tricuspid annulus. Three patients had atriofascicular connections, and one patient had an atrioventricular connection.

Methods

Patient Characteristics

Between April 1990 and March 1992, four patients who had atriofascicular or atrioventricular pathways that met the above criteria for Mahaim fibers were referred to our institution for accessory pathway ablation (Table 1 and Figure 1). All patients had recurrent syncope or presyncope during tachycardia despite antiarrhythmic drugs targeting either the atrioventricular node or the accessory pathway.

Electrophysiological Study and Ablation Procedure

The patients were studied in the postabsorptive state at least five half-lives after discontinuation of all antiarrhythmic medications. Patients were sedated with midazolam, fentanyl, and promethazine. Electrode catheters were positioned in the high right atrium, His
bundle region, right ventricular apex, and coronary sinus. Programmed electrical stimulation was performed using atrial and ventricular pacing at increasing rates until atrioventricular or ventriculoatrial block and/or as many as three atrial or two ventricular extrastimuli at three drive cycle lengths (600, 500, and 400 msec) were delivered. Rectangular stimuli were twice-diastolic pacing threshold and of 2-msec duration. Isoproterenol (1–2 μg/min i.v.) was infused in patients 2, 3, and 4 to sustain tachycardia.

The presence and participation of the accessory pathways in the tachycardia were then defined by demonstrating an increase in the interval between the atrial deflection and the delta wave and the merging of the His deflection with the QRS complex during atrial pacing at increasing rates, a reversal of the sequence of His bundle activation during maximal preexcitation and reciprocating tachycardia (see “Results”), lack of retrograde conduction over the accessory pathway, and a left bundle branch block morphology of the preexcited QRS complexes. Accessory pathway conduction properties and refractory periods were determined. Maximally preexcited ECGs during atrial pacing were obtained to confirm that the QRS complexes were identical to those during tachycardia (Figure 2). The sequence of atrial and His-Purkinje activation during tachycardia was determined. Adenosine (12 mg) was administered during tachycardia in three of the four patients to terminate it and determine the location of adenosine-induced block.

Once the diagnosis of a tachycardia mediated by an atriofascicular or atrioventricular fiber was demonstrated by the above criteria, a large-tip deflectable (Polaris) electrode catheter (7F; distal electrode length, 4 mm; surface area, 27 mm²; Mansfield-Webster catheters, Boston Scientific, Watertown, Mass.) was introduced into a femoral vein and advanced to the right atrium or ventricle to map the ventricular insertion of the accessory pathway to allow differentiation of atrioventricular from atriofascicular connections.

The ablation catheter tip location was confirmed using right anterior oblique, left anterior oblique, and anteroposterior radiographic views. Radiofrequency current (unmodulated continuous sine wave, 500 kHz) was generated by a power source (EP Technologies, Inc., Mountainview, Calif., or Valley Laboratory, Boulder, Colo.). Radiofrequency current was delivered at 25–45 W between the large-tip catheter electrode and a standard electrosurgical adhesive patch applied to the chest wall or leg.

Electrophysiological testing was repeated in all patients after the successful radiofrequency pulse was delivered to confirm the absence of accessory pathway conduction. Programmed stimulation techniques included atrial pacing at increasing rates until atrioventricular block, delivery of one to three atrial premature complexes to the point of atrioventricular refractoriness, ventricular pacing at increasing rates to the point of ventriculoatrial block, and delivery of one or two ventricular premature complexes to the point of refractoriness of the ventriculoatrial conduction system. Isoproterenol was administered in all patients, and electrophysiological testing was repeated.

A follow-up electrophysiology study using a similar protocol, with isoproterenol infusion, was performed 6 weeks after the ablation session in all four patients. A two-dimensional Doppler echocardiogram was obtained before ablation and 24 hours after the ablation procedure. Creatine phosphokinase (CPK) and its isoenzymes were obtained immediately after the ablation and at 8- and 16-hour intervals thereafter. Heparin was not administered during the procedure.

Results

Electrophysiological Study and Mapping

None of the accessory pathways in the four patients demonstrated retrograde conduction. Anterograde conduction properties and effective refractory periods are listed in Table 1. The atrial electrogram–to–delta wave interval increased with atrial pacing at shorter cycles in all patients (range, 35–90 msec). After elimination of the accessory pathway, patient 4 demonstrated single atrioventricular nodal reentry but no tachycardia. Atrioventricular nodal reentry was not apparent in this patient before elimination of the accessory pathway.

The tachycardia had a left bundle branch block contour in each patient (Figure 1). The frontal plane axes of tachycardia ranged from −25° to −50° (Table 1) and were not helpful in differentiating the atrioventricular from the atriofascicular pathways. Adenosine, administered during tachycardia in patients 1, 2, and 4, terminated tachycardia via anterograde block in the accessory pathway in all instances (Table 1 and Figure 3).

The cycle length of tachycardia ranged from 300 to 320 msec. The ventricular insertion site was in the region of the distal right bundle branch in patients 1, 3, and 4. The ventricular insertion site was at the tricuspid annulus in patient 2. The atrial insertion site was at the tricuspid annulus in patients 2 and 3, and right posterior location in patient 4.

In no patient was there evidence for ventricular preexcitation during sinus rhythm (Figure 2). During atrial pacing at increasing rates, the His potential merged with the ventricular complexes, which became preexcited. Further, during tachycardia an atriofascicular connection was confirmed by early ventricular activation in the right ventricular apex near the distal insertion of the right bundle branch (Figure 4). Reversal of the His bundle activation sequence during tachycardia, such that the distal His bundle (or right bundle branch) was activated before the proximal His bundle (Figures 4 and 5), confirmed that the tachycardia was truly antidromic and that the His bundle was being activated retrogradely.

In the patient with an atrioventricular accessory pathway (patient 2; Table 1), the tricuspid annulus was mapped during atrial pacing and tachycardia to determine the earliest site of ventricular activation (Figure 6a). Once found, radiofrequency energy was delivered to the adjacent atrial side of the tricuspid annulus at this site.

The other three patients had atriofascicular connections. Mapping ventricular activation during atrial pacing or tachycardia in each of these patients demonstrated late ventricular activation at the tricuspid annulus and early ventricular activation at the right ventricular apex (Figure 7). The atrial insertion site of the accessory pathway was identified in patient 1 by introducing atrial premature complexes at the tricuspid annulus during tachycardia to determine the site from...
TABLE 1. Patient and Pathway Characteristics: Ablation Results

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age/sex</th>
<th>Duration (years)</th>
<th>Syncope or presyncope</th>
<th>AAD</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>31/F</td>
<td>23</td>
<td>Syncope</td>
<td>Verapamil, Digoxin, Propranolol, Atenolol</td>
</tr>
<tr>
<td>2</td>
<td>11/M</td>
<td>6</td>
<td>Presyncope</td>
<td>Propranolol, Atenolol</td>
</tr>
<tr>
<td>3</td>
<td>48/F</td>
<td>18</td>
<td>Presyncope</td>
<td>Encainide, Procainamide, Disopyramide</td>
</tr>
<tr>
<td>4</td>
<td>17/M</td>
<td>4</td>
<td>Presyncope</td>
<td>Procainamide, Flecaainide</td>
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</tbody>
</table>

Pathway Properties

<table>
<thead>
<tr>
<th>1:1 Ant</th>
<th>ERP (PCL)</th>
<th>A-δ ↑</th>
</tr>
</thead>
<tbody>
<tr>
<td>200</td>
<td>&lt;320 (600)</td>
<td>90</td>
</tr>
<tr>
<td>&lt;300</td>
<td>&lt;310 (500)</td>
<td>45</td>
</tr>
<tr>
<td>270</td>
<td>260 (600)</td>
<td>35</td>
</tr>
<tr>
<td>290</td>
<td>300 (500)</td>
<td>40</td>
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</table>

Pathway Insertion

<table>
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<th>Ventricular</th>
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</thead>
<tbody>
<tr>
<td>(R)L</td>
<td>RBB</td>
</tr>
<tr>
<td>(R)PL</td>
<td>V</td>
</tr>
<tr>
<td>(R)PL</td>
<td>RBB</td>
</tr>
<tr>
<td>(R)F</td>
<td>RBB</td>
</tr>
</tbody>
</table>

AAD, previous antiarrhythmic drugs; 1:1 Ant, shortest atrial pacing cycle length maintaining 1:1 atrioventricular conduction over the accessory pathway; ERP, effective refractory period; PCL, cycle length of the pacing drive train; A-δ↑, increase in the atrioventricular interval due to rapid atrial pacing; CL, cycle length; AVNR, atrioventricular nodal reentry; RT, atrioventricular reciprocating tachycardia; TA, tricuspid annulus; CPK, creatine phosphokinase; MB, myocardial band; RF, radiofrequency; AP, accessory pathway; (R), right; L, lateral; P, posterior; PL, posterolateral.

which the latest atrial premature complexes preexcited the ventricle without advancing the atrial electrogram recorded from the His bundle region (Figure 7). This site was presumed to be the atrial insertion site of the accessory pathway.

Radiofrequency energy was then delivered to this region at the tricuspid annulus (Figure 8). In patients 3 and 4, the atrial insertion was localized to the site where the shortest interval between atrial activation during constant atrial pacing (from various regions of the right atrium at the tricuspid annulus) and the onset of the delta wave could be obtained, with radiofrequency energy then targeted to these regions.

Ablation Procedure

In patients 1 and 2, radiofrequency energy was delivered during tachycardia. The efficacy of the radiofrequency pulse delivery was determined by termination of the tachycardia due to anterograde block in the accessory pathway (Figure 8). In patients 3 and 4, radiofrequency current was delivered during sinus rhythm to enhance catheter stability. The success of the radiofrequency catheter ablation procedure was determined subsequently by demonstrating anterograde block over the accessory pathway and failure to initiate tachycardia.

The radiofrequency ablation catheter was positioned on the atrial side of the tricuspid annulus in patients 1,

![Figure 1](http://circ.ahajournals.org/doi/10.1161/01.CIR.87.3.740)

**Figure 1.** Twelve-lead scalar ECGs during reciprocating tachycardia are depicted for the four patients in this report.
Table 1. Continued

<table>
<thead>
<tr>
<th>Tachycardia</th>
<th>Site of block with adenosine (RT)</th>
<th>Ablation site (TA)</th>
<th>No. of RF pulses</th>
<th>Cardiac enzymes</th>
<th>Follow-up (months)</th>
<th>RF success</th>
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<td>CL</td>
<td>Morphology/axis</td>
<td>AVNR</td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>310</td>
<td>LB/−50°</td>
<td>No</td>
<td>AP</td>
<td>10</td>
<td>131</td>
<td>6</td>
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<tr>
<td>300</td>
<td>LB/−50°</td>
<td>No</td>
<td>AP</td>
<td>16</td>
<td>2,038</td>
<td>4</td>
</tr>
<tr>
<td>310</td>
<td>LB/−25°</td>
<td>No</td>
<td>Not tested</td>
<td>14</td>
<td>78</td>
<td>5</td>
</tr>
<tr>
<td>320</td>
<td>LB/−45°</td>
<td>Yes (echoes)</td>
<td>AP</td>
<td>19</td>
<td>309</td>
<td>2</td>
</tr>
</tbody>
</table>

3, and 4 (Figures 9a and 9b). In patient 2, the catheter was initially positioned on the atrial side of the tricuspid annulus (Figure 9c), and the ablation procedure was initially successful. However, the tachycardia recurred spontaneously 90 minutes later. The ablation catheter was then positioned under the tricuspid valve (ventricular side of the tricuspid annulus; Figure 9d) in a right posterolateral location, and radiofrequency energy at that site terminated the tachycardia within 2 seconds. The successful site demonstrated early ventricular activation (35 msec before the onset of the delta wave; Figure 6a). The location of the catheter tip at the ventricular side of the tricuspid valve annulus was confirmed by recording a small atrial electrogram and a large ventricular electrogram during sinus rhythm after delivery of the successful ablation pulse (Figure 6b). Subsequently, the absence of accessory pathway conduction was confirmed by programmed stimulation techniques.

**Follow-up**

The patients were followed for 15, 9, 6, and 2 months, respectively. No patient has had recurrent tachycardia. All patients have had repeat electrophysiological testing.

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**Figure 2.** ECG from patient 3. Panel a: Scalar 12-lead ECG during sinus rhythm (the ECG is normal); panel b: scalar 12-lead ECG during tachycardia using an atriofascicular fiber; panel c: scalar 12-lead ECG during atrial pacing. The morphology of the preexcited complexes is identical to those during tachycardia.
FIGURE 3. ECG from patient 1. Shown are surface leads I, II, III, and V1 and intracardiac recordings from the high right atrium (HRA) and right ventricle (RV). Adenosine was administered during tachycardia and terminated it with anterograde block in the accessory pathway. When sinus rhythm was restored, there was still a high degree of atrioventricular nodal conduction delay, and conduction occurred preferentially down the accessory pathway.

30 minutes and 6 weeks after delivery of the successful radiofrequency pulse, and no patient has had inducible tachycardia or ventricular preexcitation. Patient 4, who had atrioventricular nodal reentrant echo beats at the ablation study, also did not have inducible atrioventricular nodal reentrant tachycardia at follow-up study.

Doppler echocardiograms following the procedure were normal in all four patients. Peak CPK values are listed in Table 1. There was a slight rise in the CPK, but no wall motion abnormality was detectable by echocardiogram in any patient.

Complications

There were no embolic or hemorrhagic complications. Anterograde atrioventricular nodal and His-Purkinje conduction were unchanged after the ablation procedures. Patient 2 had transient right bundle branch block due to catheter trauma.

Discussion

Main Findings

The results from this study suggest that radiofrequency catheter ablation of the atrial insertion of Mahaim (atriofascicular or atrioventricular) accessory connections is feasible and safe, regardless of the location of the ventricular insertion of the accessory pathway. Furthermore, ablation at the ventricular insertion site of an atrioventricular connection can be accomplished. There were no complications in this series, and all patients have remained asymptomatic during follow-up. The atrial insertion of all four pathways was not near the atrioventricular node, and there was no impairment of normal atrioventricular conduction due to the radiofrequency catheter ablation procedure.

Anatomic Substrate of Tachycardia

Several observations in this study provide evidence incompatible with the concept of a “nodoventricular” or “nodosfascicular” accessory pathway as being responsible for Mahaim tachycardias. First, premature atrial extra-stimuli delivered to the atrium during tachycardia failed to preexcite atrial tissue located near the atrioventricular node and recorded in the His bundle electrogram, despite preexciting the His bundle and advancing the tachycardia, i.e., resetting it. Second, all accessory pathways in these four patients were successfully ablated at regions quite remote from the atrioventricular node. These two observations provide strong evidence against the cephalad insertion of the accessory pathways being in the atrioventricular node. Furthermore, despite the fact that only one patient (patient 4) had evidence for atrioventricular nodal reentry, bystander participation of the atrioventricular or atriofascicular accessory pathway during atrioventricular nodal reentry was not the mechanism of tachycardia in any of our four patients. This study also demonstrates that Mahaim tachycardias can be mediated by either atriofascicular or atrioventricular accessory pathways, and whether a pathway inserts into the ventricle at the tricuspid annulus (atrioventricular pathway) or into the distal right bundle branch (atriofascicular pathway) cannot be easily differentiated by the surface ECG. These findings suggest that intracardiac electrophysiological studies are required to differentiate these two types of accessory connections.
Atrioventricular Versus Atriofascicular Connections

Atrioventricular accessory pathways can be quickly differentiated from atriofascicular connections by pacing the atrium to the point of maximal ventricular preexcitation or recording during tachycardia. In patients with atriofascicular connections, ventricular activation begins at the right ventricular apex (RVA) and proceeds retrogradely over the His-Purkinje system. In atrioventricular accessory pathways, activation of the distal His bundle precedes that of the proximal His bundle, and His-Purkinje activation is retrograde, beginning with the distal right bundle branch, and that activation of the ventricular myocardium at the right ventricular apex (RVA) is early.

FIGURE 4. ECG from patient 1. Shown are surface leads I, II, III, and V1 and intracardiac recordings from the high right atrium (HRA), proximal His bundle (HBEp), middle His bundle (HBEp), distal His bundle (HBEd), and proximal and distal right bundle branch (RBBp and RBBd, respectively). During reciprocating tachycardia using the atriofascicular fiber for anterograde conduction and the His-Purkinje system for retrograde conduction, activation of the distal His bundle precedes that of the proximal His bundle. Note that His-Purkinje activation is retrograde, beginning with the distal right bundle branch, and that activation of the ventricular myocardium at the right ventricular apex (RVA) is early.

FIGURE 5. ECG from patient 1. Shown are surface leads I, II, III, and V1 and intracardiac recordings from the high right atrium (HRA), proximal His bundle (HBEp), and distal His bundle (HBEd). As in Figure 4, reciprocating tachycardia conducts retrogradely over the His-Purkinje system as evidenced by activation of the distal His bundle before the proximal His bundle. An atrial premature complex (S2) is introduced during tachycardia and terminates it. A subsequent sinus beat shows normal His-Purkinje activation with the HBEp activated before the HBEd.
SUCCESSFUL SITE

**Panel a:** Recordings from the ablation catheter when the tip was positioned under the tricuspid valve in a right posterolateral location in the right ventricle. Ventricular activation during tachycardia preceded the onset of the QRS complex by 35 msec. Atrial activation is not clearly identified on the recording.

**Panel b:** After elimination of the accessory pathway, an atrial electrogram is recorded, confirming that the ablation catheter tip was at the annulus on the ventricular side. Note that right ventricular activation now is recorded in the terminal portion of the QRS complex, the delay in part due to the right bundle branch block. Right bundle branch conduction subsequently recovered and was therefore thought to be due to catheter trauma.

The presence of an atriofascicular connection without retrograde conduction presents mapping difficulties. A previous study demonstrated that the ventricular insertion could be successfully and safely ablated with direct current shocks. The only complication in that study was the creation of permanent right bundle branch block in one of the three reported patients. However, when applied to the ventricle, direct current energy is potentially arrhythmogenic. Furthermore, ablation in this region also incurs the risk of creating retrograde right bundle branch block without eliminating the ventricular insertion of the atriofascicular connection. Were this to happen, the tachycardia mediated by the atriofascicular fiber could still occur, with the retrograde tachycardia circuit traversing the septum and the left bundle branch. This longer retrograde circuit could allow sufficient time for an incessant tachycardia to occur. Eliminating the atrial insertion of the accessory pathway with radiofrequency current avoids both of these risks.

**Mapping and Ablation Approaches**

An atrioventricular connection with characteristics of a Mahaim fiber (patient 2) also is very amenable to radiofrequency catheter ablation techniques. Such an accessory pathway is similar to other right-sided accessory pathways except that it conducts only anterogradely, participates only in antidromic tachycardia, and...
demonstrates decremental conduction properties. Earliest ventricular activation can therefore be mapped to the base of the heart in the region of the tricuspid annulus, in contrast with the patients who have atriofascicular fibers in which earliest ventricular activation occurs near the right ventricular apex. Thus, in patients with an atrioventricular connection, catheter mapping of the tricuspid annulus during either atrial pacing or tachycardia can localize the ventricular insertion site, and radiofrequency current can be targeted to this region.

For the patient with an atriofascicular connection, three mapping techniques have been proposed: stimulus-to-delta wave mapping, premature atrial stimulation during tachycardia from a catheter on the atrial side of the tricuspid annulus, and recording Mahaim potentials. The second proposal is based on the reasoning that the site from which the latest atrial premature complex advances the QRS (i.e., the QRS occurs earlier than expected), without advancing the atrial electrogram in the His region, presumably occurs from a site adjacent to the atrial insertion of the atriofascicular pathway (Figure 7). The first two of these techniques are technically demanding because it is difficult to position a catheter securely and reliably at the tricuspid annulus (and not in nearby atrium) and

FIGURE 7. ECG from patient 1. Shown are surface leads I, II, III, and V1 and intracardiac recordings from the high right atrium (HRA), proximal His bundle (HBEp), middle His bundle (HBEm), and distal His bundle (HBEd). Also, there is a catheter (with proximal and distal electrode pairs) at the tricuspid annulus (TAp and TAd). Recordings are made during reciprocating tachycardia having a cycle length of 295 msec. An atrial extrastimulus (S2) is delivered at the tricuspid annulus in a right lateral location. The subsequent His deflection occurs 20 msec earlier, but the preceding atrial electrogram recorded from the HBEp is not similarly advanced, accounting for the “apparent” AH shortening recorded from the His bundle region. This confirms that the proximal portion of the accessory pathway inserts in the atrium at a location separate from the atrial insertion of the atroventricular node. Furthermore, this catheter location provided the site at which the latest atrial extrastimulus could advance the His deflection without advancing the atrial electrogram in the His bundle lead, a mapping technique used to identify the atrial insertion of the accessory pathway.

FIGURE 8. ECG from patient 1. Sustained tachycardia using the atriofascicular pathway anterogradely and the ventriculoatrial conduction system retrogradely is present. As radiofrequency current is delivered to the atrial side of the tricuspid annulus at the atrial insertion of the accessory pathway, tachycardia gradually slows and terminates within 3.5 seconds. This radiofrequency pulse eliminated accessory pathway conduction.
deliver atrial extrastimuli, particularly during tachycardia, without the catheter moving. These methods are also confounded by the fact that changes in autonomic tone and heart rate can alter the conduction time in the decrementally conducting atriofascicular fibers, making reliable stimulus–to–delta wave measurements difficult to obtain over the course of a mapping procedure. In addition, the optimal ablation site may not be a location where adequate atrial pacing can be obtained. Although we did not attempt to record Mahaim potentials in these patients, there are two preliminary reports of such recordings.\textsuperscript{14,17} Whether recording electrograms from Mahaim fibers would have shortened the time to achieve ablation success cannot be stated from this study.

An alternative approach to eliminating the atrial insertion of an atriofascicular connection is to empirically deliver short pulses of radiofrequency current at the tricuspid annulus in a region where stimulus–to–delta wave mapping has grossly localized the atrial insertion of the accessory pathway, preferably during ventricular preexcitation (tachycardia or atrial pacing). Loss of preexcitation can then be used as a marker for success. Because stimulus–to–delta wave mapping can be inaccurate, such a technique carries the risk of delivery of unnecessary radiofrequency applications to a potentially larger-than-desired region of myocardium, resulting in a rise in cardiac enzymes. Nevertheless, this technique can be practical and successful, as demonstrated in this series.

\textbf{Atrioventricular Nodal Reentry}

Some\textsuperscript{18–20} have suggested that atrioventricular nodal reentry is often associated with Mahaim connections. In fact, it has been suggested that Mahaim tachycardia in some patients may represent atrioventricular nodal reentry with bystander preexcitation via the Mahaim fiber. Such was clearly not the case in this series. Only one patient had evidence for atrioventricular nodal reentry, and only single echo beats were inducible (patient 4). Furthermore, there was no inducible tachycardia after elimination of the accessory pathway, a finding that would not be expected if the accessory pathway was merely a bystander to atrioventricular nodal reentry. The ablation pulses in this study were not delivered to areas that would have been expected to eliminate slow atrioventricular nodal pathways (i.e., right posteroseptal).

\textbf{Conclusions}

This study demonstrates that radiofrequency catheter ablation techniques can successfully and safely eliminate tachycardias mediated by Mahaim fibers, either atriofascicular or atrioventricular connections. This procedure should be considered as early therapy in symptomatic patients with recurrent tachycardias mediated by Mahaim connections.

\textbf{Acknowledgments}

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