Efficacy and safety of transcatheter ablation of posteroseptal accessory pathways

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ABSTRACT Eight patients with a posteroseptal accessory pathway and symptomatic atrial fibrillation and/or orthodromic reciprocating tachycardia underwent attempted transcatheter ablation of the accessory pathway. A quadrupolar electrode catheter was positioned within the coronary sinus such that the proximal pair of electrodes straddled the os. The proximal pair of electrodes was made electrically common and connected to the cathodal output of a defibrillator. A patch electrode placed over the midthoracic spine was connected to the anodal sink of the defibrillator. Two to three transcatheter shocks were delivered, with a cumulative energy of 600 to 900 J. Immediately after the shocks were delivered, retrograde accessory pathway conduction was absent in each patient. Anterograde conduction through the posteroseptal accessory pathway was absent in six patients and could not be assessed in two patients who each had a second right-sided accessory pathway. Long-term results were assessed in seven patients 4 to 11 months after delivery of the shocks. Anterograde and retrograde conduction through the posteroseptal accessory pathway was absent in five patients. In one patient, retrograde accessory pathway conduction was absent and anterograde conduction was present but was slower than at baseline. In this patient, orthodromic tachycardia was no longer inducible and the ventricular rate during induced atrial fibrillation was 150 beats/min, compared with 220 beats/min before the attempted ablation. He has remained asymptomatic without antiarrhythmic drug therapy for 18 months. In one patient, the transcatheter shocks had no long-term effect on accessory pathway conduction. The shocks delivered at the os of the coronary sinus were well tolerated. There was no long-term effect on anterograde conduction through the atrioventricular junction. Retrograde conduction through the atrioventricular junction was impaired in three patients. In four patients who underwent direct or angiographic visualization of the coronary sinus, no abnormalities were seen. Transcatheter ablation of posteroseptal accessory pathways has a long-term success rate of at least 75% and a low morbidity rate. Closed-chest catheter ablation of posteroseptal accessory pathways is an attractive alternative to surgical ablation of these pathways.


EXPERIENCE in more than 150 patients with supraventricular tachycardia refractory to pharmacologic treatment has indicated that catheter ablation of the atrioventricular junction results in good or excellent arrhythmia control in approximately 90% of patients.1,2 Catheter ablation of accessory pathways has also been attempted, but with less overall success. Attempts at ablation of left-sided accessory pathways by shocks delivered within the coronary sinus have generally been unsuccessful and, moreover, have resulted in perforation of the coronary sinus.3 Catheter ablation of right-sided accessory pathways has been limited by difficulty in precisely localizing these pathways.4–6 Attempts at transcatheter ablation of posteroseptal accessory pathways have met with some degree of success but, to date, the total published experience with this technique has consisted of only six patients.7–10 The posteroseptal accessory pathway was successfully ablated in four of these six patients, and no complications were reported. However, only two cases in which the procedure was successful were reported in detail.7–10 Although encouraging, the results reported to date are clearly too preliminary to allow valid conclusions regarding the efficacy or safety of the catheter ablation technique in the treatment of patients who have a posteroseptal accessory pathway.

The purpose of this report is to present the results of
attempted catheter ablation of a posteroseptal accessory pathway in a series of eight patients to help define the efficacy and safety of this new alternative to surgical ablation of posteroseptal accessory pathways.

Methods

**Patient characteristics.** The subjects of this report were five men and three women who had recurrent, symptomatic orthodromic reciprocating tachycardia. Their mean age was 31 ± 10 years (mean ± SD). Seven patients had the Wolff-Parkinson-White syndrome and a documented history of between one and six episodes of atrial fibrillation at an average rate varying between 220 and 300 beats/min. One patient (No. 4) had a concealed bypass tract and no history of atrial fibrillation. Seven patients had been treated with a mean of 2.3 ± 1.4 antiarrhythmic drugs that were either ineffective or not tolerated because of side effects. One patient did not undergo a clinical trial of antiarrhythmic drug treatment before the attempt at catheter ablation. Patient 1 was the subject of a prior report.7

**Baseline electrophysiological study.** Before catheter ablation, each patient underwent an electrophysiologic study while in the fasting, unsedated state, at least four half-lives after discontinuation of antiarrhythmic drugs. Quadripolar electrode catheters were introduced through a femoral or subclavian vein and positioned within the right atrium, right ventricle, coronary sinus, and across the tricuspid valve for recording the His bundle electrogram. The baseline accessory pathway conduction properties and refractory periods are listed in table 1. When determination of the anterograde or retrograde effective refractory period of the accessory pathway was limited by atrial or ventricular refractoriness, the accessory pathway effective refractory period was considered to be equal to or less than the atrial functional refractory period or the ventricular effective refractory period.

Orthodromic reciprocating tachycardia was induced in each

**TABLE 1**

Accessory pathway characteristics before catheter ablation

<table>
<thead>
<tr>
<th>No.</th>
<th>ERP (msec)</th>
<th>Conduction (msec)</th>
<th>Retrograde ERP (msec)</th>
<th>VA at os of CS (msec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1:1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>275</td>
<td>≤320</td>
<td>≥200</td>
<td>90</td>
</tr>
<tr>
<td>2</td>
<td>300</td>
<td>≥280</td>
<td>290</td>
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<td>3</td>
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<td>225</td>
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</tr>
<tr>
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<td>CBT</td>
<td>CBT</td>
<td>260</td>
<td>85</td>
</tr>
<tr>
<td>5</td>
<td>250</td>
<td>250</td>
<td>≥230</td>
<td>77</td>
</tr>
<tr>
<td>6</td>
<td>275</td>
<td>≤250</td>
<td>300</td>
<td>115</td>
</tr>
</tbody>
</table>

**Patients with a single posteroseptal accessory pathway**

| 7   | 275        | 260              | 300                  | ND                   |
| 8   | 400        | 330              | 250                  | 130                  |

ERP = effective refractory period; VA = ventriculoatrial interval; CS = coronary sinus; CBT = concealed bypass tract; ND = not determined.

*Minimum atrial paced cycle length associated with 1:1 anterograde accessory pathway conduction.

*Minimum ventricular paced cycle length associated with 1:1 retrograde accessory pathway conduction.

*Ventriculoatrial interval recorded at the os of the coronary sinus during orthodromic reciprocating tachycardia.

**THERAPY AND PREVENTION–ARRHYTHMIA**

In five patients, 1 to 1.5 g of intravenous procainamide (mean serum level 8.2 ± 3.1 mg/ml) was administered and found not to have any appreciable effects on accessory pathway conduction or refractoriness. Electropharmacologic testing was not performed in patient 3 or 4.

**Catheter ablation protocol.** The patients were offered the options of treatment with an investigational or, if appropriate, a conventional antiarrhythmic drug, surgical accessory pathway ablation, or catheter ablation. The two patients who had dual accessory pathways chose to undergo an attempt at catheter ablation of the posteroseptal accessory pathway in hopes of improving symptoms or as a prelude to surgical ablation of the right anterior accessory pathway. Before undergoing the catheter ablation procedure, each patient provided informed consent under a protocol approved by the Committee on Human Research at the University of California, San Francisco, or the University of Michigan.

Patients were brought to the electrophysiology laboratory while they were in the fasting, unsedated state. Blood pressure was monitored with a No. 5F cannula inserted into a femoral artery. An electrode catheter was inserted into a femoral vein and positioned against the right ventricular apex for pacing, if needed.

A No. 6F or 7F central lumen catheter was inserted into a subclavian vein and positioned in the coronary sinus under fluoroscopic guidance. After injection of contrast material to visualize the location of the os of the coronary sinus, this catheter was removed and replaced with a No. 6F or 7F quadripolar electrode catheter with 1 cm interelectrode distance (USCI, Billerica, MA). This catheter was positioned in the coronary sinus such that the proximal pair of electrodes straddled the os (figure 1). This proximal pair of electrodes was made electrically common and connected to the cathodal output of a defibrillator (Physio-Control, Redmond, WA). A 16 cm patch electrode (R-2 Corp., Skokie, IL) was placed over the midthoracic spine and connected to the anodal sink of the defibrillator. The patients were then anesthetized with sodium thiopental or sodium methohexital, and two or three 200 to 400 J discharges synchronized to the QRS were delivered within a 30 min period.

Atrial and ventricular stimulation were performed 5 to 10 min after each shock to assess their short-term effects on the accessory pathway.

After the ablation procedure, serial creatine kinase and creatine kinase–MB fraction levels were measured and the patients underwent continuous electrocardiographic monitoring for at least 4 days. A technetium pyrophosphate scintigram was obtained in each of five patients. A 24 hr continuous ambulatory electrocardiographic recording was obtained 4 to 7 days after delivery of the shocks.

A follow-up electrophysiologic study was performed 4 to 8 months after the attempt at catheter ablation in each of the six
patients who had an isolated posteroseptal accessory pathway. In patient 8, the effect of the intracardiac shocks on the posteroseptal accessory pathway was assessed by direct epicardial mapping 11 months later, at the time of surgical ablation of the right anterior accessory pathway. Patient 7 has not undergone follow-up electrophysiologic testing.

In three patients (Nos. 1, 5, and 6) coronary angiography was performed and the coronary sinus was visualized during the venous phase 4 months after the catheter ablation procedure. In patient 8, the coronary sinus was inspected directly at the time of surgical ablation of a right-sided accessory pathway 11 months after the catheter ablation procedure.

Results

Immediate results (table 2). The initial shock was either 300 or 400 J in strength. The immediate effect of this initial shock was to abolish anterograde conduction through the posteroseptal accessory pathway in four of the seven patients in whom the bypass tract was not concealed. In patient 6, anterograde conduction was not abolished after an initial shock of 300 J, but was abolished after a second shock of 400 J. In the two patients with dual accessory pathways (patients 7 and 8), the short-term effect of the shocks on anterograde conduction through the posteroseptal accessory pathway was indeterminate because of the presence of ventricular preexcitation via the right anterior accessory pathway. No change in the delta wave pattern during sinus rhythm or incremental atrial pacing was noted in these two patients.

In seven patients, retrograde conduction through the posteroseptal accessory pathway was abolished after the first shock; in patient 6, retrograde conduction over the accessory pathway was abolished only after the second shock.

After anterograde and/or retrograde conduction was abolished by the first or second shock, each patient received an additional 200 to 400 J discharge. The cumulative stored energy was 600 to 900 J (mean 762 ± 106 J).

Late results (table 2). The long-term effects of the transcatheter shocks on conduction through the posteroseptal accessory pathway were assessed after 4 to 11 months of follow-up in seven patients. In five patients, there was no evidence of anterograde or retrograde conduction through the posteroseptal accessory pathway (figures 2 and 3). Each of these patients has remained asymptomatic over a follow-up period of 5 to 22 months (mean 11.4 ± 7.3 months).

Delta waves reappeared on the electrocardiogram between 1 and 6 weeks after delivery of the transcatheter shocks in patient 2. He remained asymptomatic without antiarrhythmic medications, and an electrophysiologic study 8 months later demonstrated one-to-one anterograde accessory pathway conduction to a minimum atrial paced cycle length of 350 msec, and an anterograde accessory pathway effective refractory period of less than 210 msec. However, there was no evidence of retrograde accessory pathway conduction,

### TABLE 2

**Effects of transcatheter shocks on posteroseptal accessory pathway conduction**

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>No. of shocks</th>
<th>Cumulative energy (J)</th>
<th>Ant cond</th>
<th>Retro cond</th>
<th>Ant cond</th>
<th>Retro cond</th>
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<tbody>
<tr>
<td>1</td>
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<td>800</td>
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<tr>
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<td>+</td>
<td>+</td>
</tr>
<tr>
<td>4</td>
<td>2</td>
<td>600</td>
<td>CBT</td>
<td>0</td>
<td>CBT</td>
<td>0</td>
</tr>
<tr>
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<td>600</td>
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<td>0</td>
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<td>0</td>
</tr>
<tr>
<td>6</td>
<td>3</td>
<td>900</td>
<td>0</td>
<td>0</td>
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<td>7</td>
<td>2</td>
<td>800</td>
<td>Indet*</td>
<td>0</td>
<td>ND</td>
<td>ND</td>
</tr>
<tr>
<td>8</td>
<td>2</td>
<td>800</td>
<td>Indet*</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

0 = No conduction; + = conduction present; Ant = anterograde; cond = conduction; CBT = concealed bypass tract; Indet = indeterminate; ND = not determined; Retro = retrograde.

*Conduction present, but markedly diminished from baseline.

*Effect on conduction indeterminate because of ventricular preexcitation via right anterior accessory pathway.
FIGURE 2. Results of electrophysiologic testing before and 4 months after transcatheter ablation of a posteroseptal accessory pathway in patient 1. A, Baseline His bundle electrogram (HBE) demonstrated ventricular preexcitation, with an HV of -10 msec and an atrioventricular interval (AV) of 100 msec. A = atrial electrogram; H = His bundle depolarization; HRA = high right atrial electrogram. Time lines in this and subsequent figures represent 1 sec intervals. B, Atrial pacing at a cycle length of 300 msec resulted in 1:1 conduction through the accessory pathway. The AV interval remained constant at 100 msec. CS = coronary sinus electrogram. C, During orthodromic reciprocating tachycardia, the shortest ventriculoatrial (VA) interval (90 msec) was recorded at the os of the coronary sinus (CS_s), consistent with retrograde conduction over a posteroseptal accessory pathway during the tachycardia. The number over each electrogram refers to the VA interval in msec recorded in that electrogram. CS_{prox} = proximal coronary sinus. D, Four months after the delivery of two 400 J shocks at the os of the coronary sinus, there is no ventricular preexcitation during sinus rhythm. The HV has increased to 50 msec, and the AV to 150 msec. E, Atrial pacing at a cycle length of 300 msec resulted in 2:1 atrioventricular nodal block, with an HV of 45 msec and no ventricular preexcitation. S = stimulus. F, Ventricular pacing at a cycle length of 600 msec resulted in 2:1 retrograde conduction through the atrioventricular junction. Note that the low septal right atrial electrogram (recorded in the HBE) now precedes the atrial electrogram recorded at the coronary sinus os. In this patient, anterograde and retrograde conduction over the posteroseptal accessory pathway were completely eliminated by the transcatheter shocks.
FIGURE 3. Electrocardiograms before and 4 months after transcatheter ablation of a posteroseptal accessory pathway in patient 5. A, The baseline electrocardiogram demonstrated inverted delta waves in leads II, III, and aVF, and upright delta waves in leads I, aVL, and V1-V6. This pattern of delta waves and the transition from a small to large R wave between V1 and V2 is characteristic of ventricular preexcitation over a posteroseptal accessory pathway. B, Four months after the delivery of two 300 J shocks at the coronary sinus os, there is a normal PR interval and delta waves are not present. Electrophysiologic testing demonstrated the absence of anterograde or retrograde accessory pathway conduction.
and orthodromic reciprocating tachycardia was not inducible. When atrial fibrillation was induced, the average ventricular rate was 150 beats/min, and the shortest preexcited RR interval was 320 msec (vs 220 beats/min and 200 msec, respectively, at baseline). This patient has remained asymptomatic without drug treatment over a follow-up period of 18 months.

In patient 3, delta waves reappeared on the electrocardiogram on the sixth day after delivery of the transcatheter shocks. An electrophysiologic study 1 day later demonstrated that both anterograde and retrograde accessory pathway conduction were present, without a change from baseline. He was treated with quinidine and nadolol and experienced no symptomatic arrhythmia. After 9 months, drug therapy was temporarily discontinued and an electrophysiologic study demonstrated that the conduction and refractoriness properties of the accessory pathway were not changed from baseline. He was treated with procainamide and has remained asymptomatic over a total follow-up period of 17 months.

Effects on the atrioventricular junction. A transient junctional rhythm lasting several seconds to several minutes occurred in each patient immediately after delivery of the transcatheter shocks. Atrioventricular nodal Wenckebach block occurred for several minutes in one patient. Continuous ambulatory electrocardiographic monitoring 4 to 7 days after delivery of the shocks demonstrated sinus rhythm without atrioventricular block in all patients.

The effects of the transcatheter shocks on the atrioventricular junction were assessed in six patients during a follow-up electrophysiologic study 4 to 9 months after delivery of the transcatheter shocks (table 3). The atrioventricular nodal conduction time (AH) was 75 to 100 msec (mean 87 ± 11 msec) and in each patient was within 5 msec of the baseline AH (mean 88 ± 12, p > .05). The infranodal conduction time (HV) was 40 to 50 msec (mean 45 ± 4 msec) in the absence of ventricular preexcitation during sinus rhythm (patients 1, 4 to 6), after 700 mg of intravenous procainamide (patient 2), and during orthodromic reciprocating tachycardia (patient 3).

During incremental atrial pacing there was one-to-one conduction through the atrioventricular junction to a minimum paced cycle length of 275 to 400 msec (mean 380 ± 54 msec). In patient 3, the presence of ventricular preexcitation during atrial pacing prevented assessment of conduction through the atrioventricular junction. The mean atrioventricular nodal effective refractory period was 305 ± 54 msec (range 240 to 370 msec).

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>AH (msec)</th>
<th>HV (msec)</th>
<th>1:1 anterograde cond (msec)</th>
<th>Ant AVN ERP (msec)</th>
<th>1:1 retro cond (msec)</th>
<th>Retro ERP (msec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
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</tr>
<tr>
<td>2</td>
<td>100</td>
<td>50</td>
<td>400</td>
<td>370</td>
<td>VAD</td>
<td>—</td>
</tr>
<tr>
<td>3</td>
<td>85</td>
<td>45</td>
<td>Preex</td>
<td>240</td>
<td>ND</td>
<td>ND</td>
</tr>
<tr>
<td>4</td>
<td>80</td>
<td>40</td>
<td>400</td>
<td>370</td>
<td>VAD</td>
<td>—</td>
</tr>
<tr>
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<td>45</td>
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<td>300</td>
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<td>6</td>
<td>80</td>
<td>40</td>
<td>375</td>
<td>270</td>
<td>350</td>
<td>260</td>
</tr>
</tbody>
</table>

Ant = anterograde; cond = conduction; ERP = effective refractory period; Preex = preexcited; VAD = ventriculoatrial dissociation.

aIn the absence of ventricular preexcitation.

bMinimum paced atrial cycle length associated with 1:1 conduction.

cMinimum paced ventricular cycle length associated with 1:1 conduction.

dNot determined, because of retrograde accessory pathway conduction.

Incremental ventricular pacing demonstrated that ventriculoatrial dissociation was present in two patients (Nos. 2 and 4). One-to-one retrograde conduction through the atrioventricular node was present to a minimum paced cycle length of between 350 and 700 msec (mean 467 ± 202 msec) in four patients. The retrograde effective refractory period could be determined in four patients and varied between 220 and 340 msec (mean 273 ± 61). In patient 3, the presence of retrograde accessory pathway conduction prevented the measurement of retrograde conduction or refractoriness in the atrioventricular junction.

Other effects of the transcatheter shocks. The mean peak creatine kinase serum level after delivery of the shocks was 364 ± 360 IU/liter, and the mean peak MB fraction was 31 ± 28 IU/liter. A technetium pyrophosphate scintigram showed no myocardial uptake in four patients and focal lateral ventricular uptake suggestive of a myocardial infarction in one patient.

Occasional multiform ventricular premature depolarizations were noted in patient 3 during the first 4 days after the catheter ablation procedure. Ventricular or supraventricular tachycardia was not observed in any patient.

In three patients (Nos. 1, 5, and 6) coronary angiography demonstrated a normal left and right coronary artery and a normal coronary sinus. In patient 8, the coronary sinus and right atrium appeared grossly normal on direct visual inspection at the time of surgical ablation of a right-sided accessory pathway 11 months after delivery of the transcatheter shocks.
Discussion

The results presented herein suggest that the delivery of transcatheter shocks at the os of the coronary sinus is an effective and safe technique by which posteroseptal accessory pathways can be ablated on a long-term basis. Evaluation at 4 or more months of follow-up demonstrated that accessory pathway conduction was completely eliminated, or at least modified so that potentially life-threatening tachyarrhythmias no longer occurred, in a minimum of six of eight patients (75%). Because the ablation procedure may have been effective in patient 7 (who did not undergo a follow-up electrophysiologic study) the success rate may have been as high as 88% (seven of eight patients). The catheter ablation technique therefore appears to be an attractive alternative to surgical bypass tract ablation in patients with a posteroseptal accessory pathway who are appropriate candidates for nonpharmacologic therapy. The catheter ablation technique obviates the need for a thoracotomy and is associated with less expense, less discomfort, and a considerably shorter convalescence period than is surgical accessory pathway ablation.

There have been few detailed histologic studies of posteroseptal accessory pathways. In a recent report by Critelli et al.12 postmortem histologic analysis in a patient who had a posteroseptal accessory pathway demonstrated that the fibers of the accessory pathway originated from the lower rim of the coronary sinus. The close proximity of posteroseptal accessory pathways to the os of the coronary sinus provides the anatomic basis for the effectiveness of the technique used in the present report, in which transcatheter shocks are applied at the coronary sinus os. Although the reason that the ablation procedure was not successful in patient 3 is unclear, it may be that the posteroseptal accessory pathways do not always originate at the rim of the coronary sinus os.

A prior study on the effects of transcatheter shocks delivered within the coronary sinus in patients with left-sided accessory pathways demonstrated that perforation of the coronary sinus and cardiac tamponade was possible with two shocks of 100 and 150 J.3 Although the shocks used in the present report were as high as 400 J in strength, no patient developed signs of coronary sinus perforation or cardiac tamponade. However, great care was taken to deliver these high-energy shocks at the os, where there is atrial myocardium, instead of within the coronary sinus, which is a thin-walled vein. The risk of cardiac tamponade induced by transcatheter shocks delivered within the coronary sinus emphasizes the importance of visualizing the os by contrast injection and positioning the electrode catheter such that the proximal two electrodes straddle the os and are not actually within the coronary sinus when the transcatheter shocks are delivered.

Because posteroseptal accessory pathways may lie in close proximity to the atrioventricular node–His bundle axis, surgical ablation of these pathways is associated with approximately a 10% risk of creating high-degree atrioventricular block.13 Our results indicate that although transcatheter shocks at the os of the coronary sinus may temporarily impair atrioventricular conduction, persistent atrioventricular block is unlikely to occur. Although transient junctional rhythm or atrioventricular nodal block occurred in several patients, persistent high-degree atrioventricular nodal block was never induced by the transcatheter shocks. The only long-term effect on the atrioventricular junction observed was impaired retrograde conduction in three patients. A study on the effects of transcatheter shocks on the atrioventricular junction in dogs demonstrated that even when shocks are delivered in close proximity to the atrioventricular node, histologic damage to the atrioventricular junction may be associated with only minimal changes in atrioventricular conduction14; this observation suggests that there is a large safety margin for atrioventricular conduction. It is therefore unlikely that high-degree atrioventricular block would occur from shocks applied at the coronary sinus os. However, because of the possibility of inducing transient atrioventricular block, a temporary ventricular pacemaker should be in place before catheter ablation of a posteroseptal accessory pathway is attempted.

Prior reports have demonstrated an association between posteroseptal and right-sided accessory pathways in patients with and those without Ebstein’s anomaly.15,16 Accordingly, two patients in this report (Nos. 7 and 8) had a posteroseptal and right-sided accessory pathway. Although an effect of the transcatheter shocks on retrograde conduction through the posteroseptal accessory pathway was apparent based on a dramatic change in the atrial activation sequence during orthodromic reciprocating tachycardia, the effect on anterograde conduction was obscured by the presence of ventricular preexcitation via the right-sided accessory pathway. It is interesting to note that both patients had inverted delta waves in the inferior leads on their baseline electrocardiograms, consistent with ventricular activation through a posteroseptal accessory pathway. In neither patient was there a change in the delta wave pattern after the transcatheter shocks had
been delivered. Although this suggested that there was persisting anterograde conduction over the posteroseptal accessory pathway, direct epicardial mapping at the time of surgical ablation of the right-sided accessory pathway in patient 8 demonstrated no evidence of anterograde or retrograde conduction through a posteroseptal accessory pathway. The reason that the delta wave pattern did not change despite complete conduction block in the posteroseptal accessory pathway is unclear.

As would be expected, transcatheter shocks of 200 to 400 J resulted in some degree of myocardial injury, as evidenced by release of creatine–kinase MB fraction. However, technetium pyrophosphate scintigrams demonstrated either no myocardial uptake or a small area of focal uptake, suggesting that the injury was very localized. Although ventricular premature depolarizations occurred transiently in one patient, no patient experienced a malignant ventricular arrhythmia as a complication of the transcatheter shocks.

Coronary angiography in three patients demonstrated that the transcatheter shocks did not cause any damage to the coronary arteries. Whereas shocks within the coronary sinus frequently result in coronary sinus obstruction or stenosis, no coronary sinus abnormalities were observed as a result of transcatheter shocks to the coronary sinus os in the four patients in the present series in whom the coronary sinus was visualized either directly or during angiography.

Approximately one-third of accessory atrioventricular connections are posteroseptal in location. In the past, if the patient with a posteroseptal pathway was an appropriate candidate for nonpharmacologic therapy, the definitive form of treatment was surgical ablation of the accessory pathway. However, even in the best of hands, surgical ablation of posteroseptal accessory pathways is more difficult than ablation of free-wall accessory pathways, and is associated with approximately a 10% risk of complete atrioventricular block. Our results suggest that catheter ablation of posteroseptal accessory pathways may be a viable alternative to surgical ablation. Although confirmation in a larger number of patients is needed, the long-term success rate of the catheter ablation technique described herein appears to be at least 75% and the morbidity rate appears to be acceptably small.

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References

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F Morady, M M Scheinman, S A Winston, L A DiCarlo, Jr, J C Davis, J C Griffin, M Ruder, J A Abbott and M Eldar

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