Catheter Ablation of Accessory Pathways With a Direct Approach
Results in 35 Patients

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Thirty-five consecutive patients with an overt accessory pathway, all but two suffering from
arrhythmia (atrial fibrillation, reciprocating tachycardia, or both), underwent attempted
transcatheter ablation (fulguration) of their accessory pathway. Thirty-three patients had been
treated with a mean of 2.3 ± 1.4 antiarrhythmic drugs. A standard bipolar catheter was
positioned on the internal surface of the right or left atrioventricular anulus with 1) a subclavian
approach of the right cardiac cavities in 29 patients with right-sided accessory pathway (n = 27)
or left posteroseptal accessory pathway (n = 2), 2) a patent foramen ovale in five patients (two
with a left posterolateral accessory pathway and three with a left parietal accessory pathway),
and a transseptal catheter (one patient with a left parietal accessory pathway). Cathodic
shocks (mean, 4.3 shocks/patient) with a mean cumulative energy of 690 J enabled the ablation
(disappearance of both anterograde and retrograde conduction) of the accessory pathway in 32
patients with a follow-up ranging from 1 to 32 months (mean, 10 ± 8 months). Two of the
remaining three accessory pathways were impaired: one pathway became intermittent, the
anterograde effective refractory period of the second pathway increased from 260 to 410 msec,
and the third pathway was slightly impaired. This latter patient is the only one who still requires
therapy, with a single antiarrhythmic drug. All others are free of arrhythmias and require no
therapy. Not using coronary sinus catheterism inclusive of its os has led to only a few, benign
side effects. Only one third-degree atrioventricular block occurred in a posteroseptal accessory
pathway ablation. Three cases of patients with incessant reciprocating tachycardia involving a
further successful ablation occurred at the beginning of our experience. The best area for
ablation is, in our opinion, the recording site for the Kent-bundle activity (18 of 35 patients), but
a meticulous mapping of the atrioventricular anulus during orthodromic reciprocating tachycardia
makes ablation possible when the shortest ventriculoatrial time (V-A') can be recorded with
reliability (mean, 85 ± 18 msec). Such a procedure is an alternative to surgical ablation
regardless of the location of the accessory pathway—not only posteroseptally. (Circulation
1988;78:800–815)

Patients with accessory pathways are often prone to serious, sometimes disabling,
arrhythmias. Some are immediately at risk when suffering from syncope and paroxysmal atrial
fibrillation with short RR intervals. Others with less-alarming symptoms are still at risk, but nonin-
vasive investigations and, especially, electrophysiological studies make it possible to decide on those
particularly at risk, because of the great permeabil-

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Received October 13, 1987; revision accepted May 26, 1988.
suggested a procedure suitable only for the posteroseptal accessory pathways with a long-term success rate of 75% and a low morbidity. Even in this particular location, less convincing results\textsuperscript{8,9} or serious adverse effects\textsuperscript{10,11} have been reported.

The purpose of this study is to present the results of fulguration with a new method for any location of the accessory pathway with standard catheters currently available. This procedure consists of delivering shocks directly to the atrioventricular anulus (at the site where the accessory pathway crosses over the atrioventricular groove), without catheterism of the coronary sinus inclusive of its os.\textsuperscript{12}

**Patients and Methods**

**Patient Characteristics**

Of the 35 patients participating in the study, 33 were suffering from spontaneous symptomatic orthodromic reciprocating tachycardia, paroxysmal atrial fibrillation, or both. Two were asymptomatic. The ages of the 24 males and 11 females were 32 ± 13 (mean ± SD) years old (range, 14–65 years).

The location of the accessory pathway was right anteroseptal (n = 7), right parietal (n = 5), right posteroseptal (n = 15), left posteroseptal (n = 2), left posterolateral (n = 2), and left parietal (n = 4).

Right or left posteroseptal and posterolateral location of the accessory pathway was established not only by the pattern of major preexcitation in standard leads but also with an electrophysiological study: the QRS configuration during a major preexcitation accurately localizes the ventricular insertion of the accessory pathway but not its atrial one. Thus, a right or left approach for ablation cannot be decided without an electrophysiological study.

All patients except two were referred to our hospital because their arrhythmia was unresponsive to usual therapy (often given before admission), including amiodarone, quinidine, propafenone, and/or flecainide [2.3 ± 1.4 antiarrhythmic drugs]. These drugs were either ineffective or poorly tolerated because of their side effects.

Fulguration was then performed when the risk of sudden death was obvious (syncopy and atrial fibrillation with very short RR) and when patient acceptance was not obtained (n = 2). In other cases, fulguration was performed because of unsuitable results with antiarrhythmic therapy (n = 31). Finally, fulguration was carried out in two asymptomatic patients.

![Figure 1](http://circ.ahajournals.org/lookup/suppl/doi:10.1161/CIRCULATIONAHA.117.035402/-/DC1/1.png)

**Figure 1.** Panel A: Electrocardiogram of right parietal accessory pathway (RP AP) (patient 2). Panel B: Recording of a potential (K) likely with the AP activity during sinus rhythm (SR) identifying the ablation site (ABL. SITE). Panel C: Induced orthodromic reciprocating tachycardia (ORT) with a potential (K') likely with retrograde activation of the AP. Panel D: Electrocardiogram 32 months after ablation. Right bundle branch block was recorded during electrophysiological study when preexcitation disappeared after atrial premature beats—during ORT—after ablation. RA, electrogram recording the high right atrium activity; AV, electrogram recording the His bundle potential.

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Owing to potentially threatening arrhythmia: athletes in whom long-lasting atrial fibrillation with short RR intervals (<220 msec) was induced during the electrophysiological study by only one atrial extrastimulus, thus disclosing an atrial vulnerability.

Informed consent was required from all patients; many referred for refractory arrhythmia were advised that the electrophysiological study could lead to an ablation procedure.

**Baseline Electrophysiological Study**

Each patient underwent an electrophysiological study while in an unsedated state at least five half-lives after interruption of antiarrhythmic drugs (except amiodarone).

Electrograms were recorded with a Polygraph VR12 (Electronics for Medicine, Honeywell, Inc) with filtering 30–500 Hz and amplification 0.1–0.5 mV/cm (mean, 0.2 mV/cm). The electrophysiological study was performed with a programmable electrical stimulator (SAVITA CSO, VPA Medical, Paris) delivering impulses of 1 msec at twice diastolic threshold. A defibrillator (Defigard M Robert et Carrière, Paris) delivered electric shocks synchronized with the QRS. A switching device allowed the catheter to be switched quickly from the recording to the cardioversion mode.

Two standard bipolar electrode catheters (USCI [Billera, Massachusetts] or CORDIS, 1-cm interelectrode) were introduced through the femoral vein. The first was positioned in the right ventricular apex, and the other was positioned across the tricuspid valve for recording the His-bundle activity. In some instances, additional catheters could be inserted. Another bipolar (1-cm interelectrode) or multipolar catheter (0.5-cm interelectrode) was introduced through a subclavian vein to record the earliest atrial activation during an orthodromic reciprocating tachycardia rather than during a right ventricular pacing or to record as often as possible the specific Kent-bundle activity by moving the electrode slightly around the former site. This potential may be clearly identified only at the atrioventricular anulus, where both atrial and ventricular activity may be recorded but with very different ratios (Figures 1 and 2).

Before endorsing the specificity of such a potential, we tried to dismiss both a His-bundle activation and a late atrial or early ventricular potential.

A His-bundle activity was easily ruled out in most cases by the location of the catheter. Elsewhere (in anteroseptal accessory pathways), this was ruled out by the steadiness between this potential and the onset of the delta wave during an increasing atrial rate (Figure 3).

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**FIGURE 2.** Panel A: Electrocardiogram of right anteroseptal accessory pathways (RAS AP) (patient 13). Panel B: Atrial fibrillation (AF) with short RR (240 msec). Panel C: Recording at the tricuspid anulus (ANN) of a potential (K) consistent with the Kent-bundle activity during sinus rhythm. Panel D: Electrocardiogram 13 months after ablation.
FIGURE 3. Electrogram of use of rapid right atrial pacing to rule out a possible His-bundle potential recorded at the tricuspid anulus (ANN): the assumed K potential remains 30 msec before the onset of the delta wave, whatever the pacing rate (cycle length, 590 or 450 msec).

FIGURE 4. Electrogram of right anteroseptal accessory pathway (RAS AP) (patient 13). Use of an atrial premature beat (APB) to exclude a late atrial potential. The loss of the AP potential (K) associated with the loss of ventricular preexcitation, without change of atrial activation, provides strong evidence that this cannot be an atrial activity. His-bundle activity is also recorded by the same catheter: EL2, which was not used for ablation (see Figure 14).
A late atrial potential was ruled out if an atrial premature beat or an increasing atrial pacing rate induced both the disappearance of preexcitation and the loss of this potential without a change in atrial activation (block at the atrial-Kent bundle interface) (Figure 4) or if one or several ventricular premature beats inhibited the anterograde “AP potential” by retrograde activation of the accessory pathway without affecting the atrial activation (Figures 5 and 6).

A late atrial potential was excluded in all 18 patients.

An early ventricular potential is more difficult to exclude. This would imply that after exclusion of a late atrial potential by evidence of a proximal block (at the atrial-Kent bundle interface), it would be possible to rule out an early ventricular activity by producing a block distal to the accessory pathway (at the Kent bundle-ventricle interface). Obviously such a block at two different sites is implausible in the same patient, and we have never observed such a phenomenon.

Indeed, in three patients during atrial pacing, we recorded the occurrence of a block distal to the assumed accessory pathway potential; this was sufficient evidence that it didn’t represent activation of the ventricular myocardium. However, as expected in these patients, we were unable to induce a proximal block (at the atrial-accessory pathway interface). So, a late atrial potential could not be excluded (Figure 7).

In two patients, a late atrial potential had already been excluded, and a ventricular pacing allowed the recording of the retrograde activation of the Kent bundle. Ventricular premature beats induced a retrograde ventriculoatrial block, and the loss of the accessory pathway potential provided strong evidence that this potential was not attributable to activation of the ventricular myocardium (block at the ventricle-accessory pathway interface). However, owing to the low resolution power of standard catheters, the retrograde depolarization of the accessory pathway was exceptionally recorded in our study.

In one patient who received ajmaline during orthodromic reciprocating tachycardia, we noticed an increment of both VK’ and K’A’ intervals, which suggests that the accessory pathway potential was neither atrial nor ventricular.

Finally, we postulated that an extra-atrial potential recorded during a sinus rhythm 25 msec or more before the onset of the QRS complex in standard leads was unlikely with a ventricular activity.
Figure 6. Electrogram of right anteroseptal accessory pathway (RAS AP). Panel A: After three ventricular premature beats (VPB: $S_1$, $S_2$, $S_3$), the ventricular preexcitation becomes intermittent. Panel B: A potential likely with a Kent-bundle activity (arrow) is seen only before the preexcited beats, without another change in atrial activation. A late atrial potential can therefore be ruled out and ablation was performed at this site (ABL. SITE).

In summary, positioning of the ablation catheter was mainly performed (after recording of the earliest atrial activity during orthodromic tachycardia) with electrograms recorded at the atroventricular anulus and, in most cases, with anterior x-ray control only. All attention was paid to recording both atrial and ventricular activities during sinus rhythm, with very early ventricular potentials ahead of or at least synchronous with the onset of the delta wave in standard leads. When a quasicontinuous electrical activity was recorded, a Kent-bundle activity had been searched and validated as previously described.

This specific potential was recorded in six of seven right anteroseptal, two of five right parietal, eight of 15 right posteroseptal, none of two left posteroseptal, none of two left posterolateral, and two of four left parietal accessory pathways.

The main electrophysiological data in our 35 patients with an overt accessory pathway are shown in Table 1.

Catheter Ablation Protocol

Ablation was performed in 18 patients at the recording site of the Kent-bundle activity, according to the criteria described above. In 25 patients (Table 1), orthodromic reciprocating tachycardia could be induced, thus allowing the measurement of the shortest ventriculoatrial time ($VA'$). In 10 patients, atrial pacing induced atrial fibrillation but not orthodromic tachycardia, and so ablation was performed: in three patients at the site where the shortest $VA'$ time was measured by ventricular pacing and in seven patients where a potential likely with Kent-bundle activity was recorded. Of course, when Kent-bundle activity was recorded, this elective site corresponded to that in which the shortest $VA'$ was recorded. In six patients, after recording the probable potential with the accessory pathway activation or the localization of the earliest atrial activity during orthodromic reciprocating tachycardia, we observed the disappearance of preexcitation, which was probably attributable to the pressure of the catheter on the accessory pathway itself (as previously described). Indeed, in these patients, the delta wave did not disappear after exercise of ajmaline testing. By pacing this elective site, we always obtained the perfect replication of a major preexcitation without a measurable delay between the spike and the QRS.
Efficacious anticoagulation was used in every case with subcutaneous injection of calcium heparinate twice a day during hospitalization.

After a short anesthesia (250 mg sodium thiopental), we delivered two successive cathodic shocks of 160 J at the elective site with only the distal electrode (Figure 8), except in the event of a sustained atrioventricular block or a displacement of the catheter. The anode was a large skin electrode (66 cm²) positioned over the left scapula in posteroseptal, posterolateral, and left parietal accessory pathways; over the left anterolateral chest wall in anteroseptal accessory pathways; and over the right anterolateral chest wall in right parietal accessory pathways. Depending on the ablation results, the shocks were repeated in the same manner: 1) during the same session after a new localization of the accessory pathway and a new brief anesthesia if ablation was unsuccessful; 2) immediately after the occurrence of an incessant orthodromic reciprocating tachycardia when only anterograde conduction was ablated; 3) the next day when preexcitation was resumed after a first session; and 4) 1 month later when a third procedure was necessary. With one exception, a fourth ablation was required the next day to obtain a perfect result. Thus, 1–12 (mean, 4.3) cathodic shocks were delivered with a mean cumulative energy of 690 J.

The electrophysiological study, the location of the best ablation site, and fulguration were performed in 3–4 hours, except in our first cases in which we tried to record too much data with unsuitable catheters. Nevertheless, we never had to abort the procedure of ablation.

After the procedure, serial creatinine–kinase and creatinine-MB fraction levels were measured, and the patients underwent a continuous electrocardiographic recording for 3 days. In all cases, echocardiographic controls were performed. Before discharge (day 6), anterograde and retrograde conductions were reassessed and an additional electrophysiological study was performed 4–8 months later in 13 patients.

Exercise testings were planned 6 and 12 months after ablation.

No coronary angiogram was performed before the procedure but before discharge in four patients: one in whom we observed a transient ST elevation (right parietal accessory pathway) and three who underwent two ablation procedures. Late coronary controls were performed in six patients 1 year after fulguration.

No antiarrhythmic drug was given after discharge.

Results

Immediate Results

Electrophysiological data. In 18 patients, we recorded the Kent-bundle activity 10–40 msec (mean, 28±9 msec) before the onset of the QRS complexes in standard leads. Amplitude ranged from 0.2 to 0.8 mV (mean, 0.5 mV). During ortho-
dromic reciprocating tachycardia, the shortest VA' ranged from 60 to 110 msec (Table 1).

**Immediate results of ablation.** After fulguration, both anterograde and retrograde conduction were abolished in 32 patients (Figures 1 and 2). In three patients, the anterograde conduction was damaged, but retrograde conduction remained, and permanent reciprocating tachycardia occurred within 1–
48 hours (Figure 9A). An immediate additional ablation at the same site provided a definitive cure in all patients. In nine patients, preexcitation resumed within 24 hours, and the next day a second attempt was successful in six patients but only temporarily so in the others (recurrence also within 24 hours). Thus, a third session in two patients (patients 16 and 23) and even a fourth (patient 32) with 1-month intervals was necessary for obtaining full ablation. In one patient (patient 25), only one shock induced a complete atrioventricular block for 15 minutes, and preexcitation resumed 3 days later. Because of the obvious risk of atrioventricular block, no other attempt was made.

Therefore, 34 of 35 accessory pathways were ablated in one session (n = 22), two (n = 9), three (n = 2), or four (n = 1).

The creatine-phosphokinase MB fraction ranged from 6 to 99 IU/l (normal values, 0–16 IU/l). Echographic controls after ablation and before discharge revealed no pericardial effusion. Continuous electrocardiographic recordings only disclosed unsteady ephemeral premature ventricular beats, which were isolated or in short salvos and did not require any antiarrhythmic treatment. The electrophysiological study performed before discharge con-

firmed in all 34 cases the disappearance of both anterograde and retrograde conduction in the ablated accessory pathway. The four early coronary angiograms showed no damage.

Late Results

The preexcitation returned in two patients after discharge (10–30 days): one was spontaneously intermittent (patient 8) and in another (patient 24), preexcitation disappeared during exercise testing or atrial pacing (125/min) and no tachycardia could be induced during the electrophysiological study, even after isoproterenol infusion. This electrophysiological study showed that the properties of the accessory pathway were deeply modified with an effective anterograde refractory period increasing from 260 to 410 msec and effective retrograde refractory period from 260 to 380 msec. Both of these patients are free of arrhythmia with no medication. The only patient requiring preventive treatment is patient 25, in whom only one shock of 160 J was given because of the observed risk of a permanent atrioventricular block (preexcitation resuming 3rd day).

Among our 35 patients with an overt accessory pathway, standard electrograms remained normal in 31. Indeed, one patient (patient 14) had two
different left-sided accessory pathways. One of these was ablated with transeptal catheterism (left parietal accessory pathway), but another unknown preexcitation became obvious and with a very different pattern (posteroseptal accessory pathway); this ablation has not yet been attempted because of its lower permeability. The other three patients are those described above.

In 13 patients, reassessment 4–8 months after ablation confirmed the disappearance of both anterograde and retrograde conduction over the accessory pathway (Table 2). No recurrence of tachycardia occurred without preventive treatment, with a follow-up ranging from 1 to 32 months (10±8 months). Late electrophysiological results are summarized in Table 2.

In six patients, a late coronary angiogram was performed 11–15 months after ablation: the location of the accessory pathway was right parietal in two patients (two sessions, cumulative energy = 640 and 960 J, respectively), right posteroseptal in two patients (two sessions, cumulative energy = 640 and 960 J, respectively), left posteroseptal in one patient (one session, cumulative energy = 640 J), and left parietal in one patient (four sessions, cumulative energy = 1,600 J). Coronary arteries were normal in all patients as were the coronary sinus and great cardiac vein.

**Side Effects**

With this procedure, some side effects were observed: three ventricular fibrillations reduced by electric shock; one pericardial rub without pericardial effusion; one transient ST elevation; and six third-degree (nodal) atrioventricular blocks. Five of the latter were transient and disappeared within several hours to 17 days. The accessory pathway was right posteroseptal in three patients and right anteroseptal in two patients. Only one third-degree (complete) atrioventricular block remained 3 months after the ablation of a right posteroseptal accessory pathway. Four of five patients in whom apparently normal conduction recurred were reassessed (Table 2); conduction through the atrioventricular node appeared to be normal on exercise testing and electrophysiological study, which showed AH, HV, and a Wenckebach point within normal limits, especially in our first patient, with a follow-up of 32 months. The sixth patient has a reliable junctional rhythm without alarming bradycardia during sev-

**Figure 9.** Panel A: Electrocardiogram of a right posteroseptal accessory pathway (RPS AP) (patient 10). Panel B: Anterior, right anterior oblique, left anterior oblique, and profile views of catheter ablation location (arrow). It was introduced through the left subclavian vein. Its tip (arrow) is very posterior: at the foot of the interatrial septum and obviously outside the coronary sinus. The two other catheters are located in the high right atrium and in the right ventricular apex. Panel C: Electrogram recorded at the ablation site (ABL. SITE) with a potential consistent with a Kent-bundle activity (arrow) and very early ventricular potentials.
eral continuous electrocardiographic recordings and achieves good performance during exercise testing (240 W). Thus, none of our patients required a permanent pacemaker.

**Discussion**

These results suggest that transcatheter shocks delivered to the site where the accessory pathway crosses over the atrioventricular groove is an effective and safe technique. In our experience, fulguration by this direct approach to the atrioventricular anulus is efficient, regardless of the location of the accessory pathway and despite the difficulty of obtaining good stability of the catheter without using the catheterism of the coronary sinus. We have never used this latter site because of the potential risk of hemopericardium, not only when fulguration is performed inside the coronary sinus but also at its ostium.

Stabilizing and positioning the catheter pose various difficulties. Displacement of the catheter is very frequent in the right parietal accessory pathways. In posteroseptal accessory pathways, whatever the ventricular insertion of the accessory pathway, ablation is feasible with a right approach and without catheterism of the coronary sinus. Therefore, not only may the right posteroseptal accessory pathways (Figure 9) be attained but also the left posteroseptal accessory pathways (Figure 10). In left posteroseptal accessory pathways, fulguration may be successful for various reasons: 1) the discrimination between right and left posteroseptal accessory pathways is perhaps ill-founded—the atrial insertion of all these accessory pathways can be septal, near the coronary sinus ostium; 2) fulguration can have a transmural effect, or 3) fulguration can be delivered to the right atrium—left ventricle sulcus. Indeed, in these cases of posteroseptal accessory pathways, the shortest VA’ time during orthodromic reciprocating tachycardia is not always in the os but near the os of the coronary sinus (Figure 10). Ablation may thus be performed with lower risk. Ablation of posterolateral or left parietal accessory pathways requires access to the left cavities, thus implying a patent foramen ovale (found in 20% of patients [Figure 11]) or a transseptal catheterism (Figure 12). In posterolateral accessory pathways, mapping the coronary sinus during an orthodromic reciprocating tachycardia is useful with a multielectrode catheter: the electrodes recording the earliest atrial activity or even the Kent-bundle activity become the radiological target, which in turn identifies the elective site where the tip of the ablation catheter is to be directed (Figure 11). Thus, the catheter used by Jackman with closely spaced electrodes aligned across the circumference of the catheter can be useful.

In our experience, a third of all patients need two or more procedures. Furthermore, as emphasized by Becker and Anderson, left-sided accessory pathways are consistently found in the subepicardial fatpad connecting the epicardial surfaces of the left atrium and the left ventricle. Therefore, high energy would be needed, or possibly the inflation of a small balloon straddling the interatrial septum and producing a pseudoforamen ovale (but not a real

### Table 2. Patients Reassessed 4–8 Months After Ablation

<table>
<thead>
<tr>
<th>Patient</th>
<th>Location of the accessory pathway</th>
<th>Result of ablation</th>
<th>Atrioventricular block after ablation</th>
<th>AH (msec)</th>
<th>HV (msec)</th>
<th>1:1 ANT COND (msec)</th>
<th>1:1 RETRO COND (msec)</th>
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AH, interval between atrial electrogram and His-bundle depolarization in the His-bundle; HV, interval between the His-bundle depolarization and the earliest onset of ventricular activation; RAS, right anteroseptal accessory pathway (AP); RP, right parietal AP; RPS, right posteroseptal AP; LPS, left posteroseptal AP; LPL, left posterolateral AP; VA’ during ORT, interval between the onset of QRS in standard leads and intrinscose deflection or the earliest atrial activation during ORT. In some cases, AF was induced but not ORT, and when Kent-bundle activity was recorded with sufficient reliability, shocks were delivered at this elective site. In other cases, right ventricular pacing was used.

1:1 ANT COND, minimum paced atrial cycle associated with 1:1 conduction; 1:1 RETRO COND, minimum paced ventricular cycle associated with 1:1 conduction; VA block, no retrograde conduction.
atrial septal defect). This would eliminate the need of another transseptal catheterism (so simplifying the subsequent procedure). Until now, we have not had recourse to this technique.

In our opinion, the elective area for ablation is the recording site of the Kent-bundle activity (already recorded exceptionally during an electrophysiological study, but this component is only useful in one patient out of two. Fulguration can be efficient with meticulous mapping of the atrioventricular anulus during orthodromic reciprocating tachycardia, yet it is always difficult to be certain of having recorded the real earliest atrial potential. As shown in Figures 9–13, electric shocks were always delivered where the earliest ventricular potentials were recorded before, or simultaneously with, the onset of the delta wave in standard leads. For us, this is a good additional criterion for identifying the site of ablation.

Similarly, we think it more important during validation of the Kent-bundle activity to exclude a late atrial potential, which has no relation with the preexcitation, than to exclude an early ventricular activity, which is always associated with it.

Damage to the atrioventricular node or the His bundle could be feared particularly in anteroseptal accessory pathways because of the vicinity of the two pathways. In our seven patients, we never observed long-term effects on anterograde conduction through the atrioventricular junction (Table 2).

In four patients out of seven, no atrioventricular block occurred. In two patients, atrioventricular block did not appear directly after ablation, but surprisingly within 24 hours, and this lasted 5 and 17 days, respectively (patients 10 and 12), probably because of an edematous or hemorrhagic reaction. Further electrophysiological study as well as exercise testing confirmed that the atrioventricular junction returned to normal. The third atrioventricular block lasted only 18 hours, but this patient refused any reassessment, thinking it unnecessary. In the anteroseptal accessory pathway, this risk of damaging the His bundle can be assessed by recording its activity with another catheter. In two of our patients, the same catheter introduced either by a femoral or a subclavian vein recorded both accessory pathway and His-bundle activities (Figure 13).
In these patients, a slight displacement of the catheter used for ablation made it possible to find a site at which the His-bundle activity was not clearly visible, without significantly modifying the shortest VA' time during orthodromic reciprocating tachycardia. A successful ablation was achieved without damage of the nodo-hissian pathway (Figure 13). In only one patient with right postero-septal accessory pathway (patient 15) did permanent atrioventricular block occur. In this patient, the accessory pathway activity was easily recorded. After a first shock (160 J), the preexcitation disappeared and atrioventricular conduction was normal. The next shock induced a complete atrioventricular block, and we now tend to not deliver a systematic second shock when the Kent bundle is initially recorded with reliability. As far as damage to the nodo-hissian pathway is concerned, fulguration seems to be as reliable as surgical ablation, which can be associated with atrioventricular block in 10% of all patients.\(^\text{17}\)

A 24-hour time lapse seems critical before any success can be expected. If preexcitation returns afterward, the properties of the accessory pathway are impaired in proportion to the time lapse of the recurrence. In one of our patients, preexcitation reappeared 1 month after ablation, but electrophysiological study confirmed sufficient impairment to prevent any recurrence of tachycardia even after isoproterenol infusion (patient 24).

When ablation is unsuccessful, it is difficult, even impossible, to distinguish inefficacy caused by inaccurate mapping from inefficacy caused by inadequate energy. However, when the most indicative criteria are considered together (short VA' time, very early first ventricular potentials, and even recording the Kent-bundle activity), we speculate that the second hypothesis is more convincing. Indeed, in the left parietal accessory pathways, we are at present successfully using more important cumulative energies (with shocks of 240 J) without obvious side effects.

The lack of serious adverse effects seems to be related to the fact that a coronary sinus catheterism is never used to obtain good stability of the catheter.
FIGURE 12. Panel A: Left parietal accessory pathway (LP AP) ablated by transseptal catheterization. One multielectrode (0.5-cm interelectrode) is introduced via a subclavian vein into the coronary sinus (DCS, distal coronary sinus). One catheter is inside the left ventricle after withdrawal of the pigtail catheter needed for the transseptal catheterization. It allowed ventricular pacing. The ablation catheter (arrow) is introduced via a femoral vein. During an orthodromic reciprocating tachycardia (ORT), it records at the ablation site (ABL. SITE) a VA' time (80 msec) shorter than that recorded in the DCS. During sinus rhythm (SR) or after an atrial premature beat (APB), ventricular potentials are synchronous with the onset of the delta wave.

Catheterization of the coronary sinus vein may be sufficient to produce damage, which may develop into a rupture or dissection because of the super-pressure related to ablation. The atrial side of the atrioventricular anulus may also be frail, but ablation of the atrial foci18,19 has produced evidence of the safety of such a procedure, as evidenced by experimental studies.20 Surrounding structures such as coronary arteries that are proximal to left or right free-wall accessory pathway may react to fulguration with acute spasm21 or chronic fibrosis.22 However, in only one patient with a right parietal accessory pathway did we observe transient but significant ST elevation, without any increase of CPK MB, alteration of his electrogram, or any injury to the coronary arteries or to ventricular function. In this respect, we performed four early coronary controls after ablation of a right parietal accessory pathway (one patient) or a posteroseptal right accessory pathway (three patients) without discovering any damage to the coronary arteries. However, an arterial consequence might become apparent only after long-term follow-up.22 This implies close surveillance by exercise testing and scintigraphic or angiographic controls. Nevertheless, we emphasize that no coronary damage was disclosed 1 year after fulguration in six patients, including one who underwent four ablation sessions with high cumulative energy (1,600 J). This potential side effect justifies further investigations but is not to be regarded as redhibitory.

We have not observed additional cases of ventricular or atrial arrhythmia other than after atrioventricular junction ablation for refractory supraven-
tricular tachycardia. As for rhythm disorders, we have observed three cases of ventricular fibrillation despite synchronized shocks. This could be explained by the high level of catecholamines in unsedated patients during electrophysiological study or by previous infusion of isoproterenol for inducing sustained and repetitive orthodromic reciprocating tachycardia, when precisely locating the elective site for ablation. In some cases, we recorded sinus pauses up to 22 seconds immediately after ablation without further evidence of sinus dysfunction. The most serious rhythm disorder related to ablation was in our first cases: the occurrence of an incessant reciprocating tachycardia resulting from an elective impairment of anterograde conduction. Another immediate ablation was successful, despite alteration of local atrial electrograms that made mapping more difficult. This side effect occurred at the beginning of our experience. Now, with better localization of the accessory pathway, this side effect is no longer observed.

As often as possible, we perform atrioventricular anulus mapping during orthodromic reciprocating tachycardia instead of during right ventricular pacing (except when only atrial fibrillation can be induced) to ablate the accessory pathway involved by the clinical tachycardia.

In our series, three patients had two different accessory pathways. One patient had a left parietal and a left posteroseptal accessory pathway (patient 18). The first was ablated with a transseptal approach; the second is not currently responsible for frequent or fast enough tachycardia to justify ablation. Another patient (patient 18) had a right posteroseptal accessory pathway easily ablated. During control electrophysiological study, we discovered an anteroseptal accessory pathway that was functional only during heart rates ranging from 90 to 110/min. We easily recorded the Kent-bundle activity, and a phase 3 and phase 4 block phenomenon proximal to the Kent-bundle activity at the atrial–accessory pathway interface. In this patient, no retrograde ventriculoatrial conduction could be observed because of a block at the same site. No ablation was attempted because no tachycardia could be induced. The last patient (patient 12) had a right anteroseptal accessory pathway ablated and experienced no recurrence of tachycardia because of his left parietal one. Moreover, in seven patients, electrophysiological study revealed reciprocating tachycardia or

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**FIGURE 13.** Panel A: Electrograms of right anteroseptal accessory pathway (RAS AP) (patient 13). Three catheters are introduced via femoral veins. One is in the right atrium, one is in the right ventricle, and one (EL2) is in the His-bundle region. This catheter records both His and AP potentials (left Electrogram). A successful ablation was performed with a fourth catheter (EL1) introduced through the left subclavian vein. It was positioned slightly above the former one and so does not record the His potential but does record a small Kent-bundle activity (ABL. SITE in right Electrogram).
echos related to an intranodal reentry, and some patients also had an enhanced retrograde conduction.

Conclusion

When an active therapy is justified, we believe fulguration is the first-line treatment instead of surgery in all right-sided accessory pathways; in left posteroseptal accessory pathways that can be ablated by shocks delivered by a right approach, outside the coronary sinus, and when the earliest activation potential can be recorded with a favorable VA time during orthodromic reciprocating tachycardia; and in left lateral and posterolateral accessory pathways, when the foramen ovale is patent.

When these requirements are not fulfilled, only a transseptal catheterism allows ablation. At present, however, this technique is still being assessed.

If the reliability and safety of this method are confirmed, its indications could be extended to symptomatic patients to avoid chronic therapy and asymptomatic patients with high risk of threatening arrhythmia.

In our opinion, most patients with the Wolff-Parkinson-White syndrome could be treated with this new method, which should modify the treatment strategy because today's choice is now no longer a question of either antiarrhythmic drugs or surgery.

Fulguration appears to be safe on condition that the coronary sinus is not used even with its proximal segment. Indeed, serious adverse effects have been described only with such a technique and never when ablation was performed elsewhere, such as in the atrium. Nor have we observed any damage to structures surrounding the atrioventricular anulus, but care is nevertheless to be taken here. Therefore, in our opinion, fulguration with this direct approach to the atrioventricular anulus is the first-line treatment for ablatting accessory pathways in patients at risk.

Acknowledgments

We are grateful for the excellent secretarial assistance and patience of Ms. M.H. Cina and for the contribution of R. Cook.

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KEY WORDS • Wolff-Parkinson-White syndrome • fulguration
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Circulation. 1988;78:800-815
doi: 10.1161/01.CIR.78.4.800

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