Atrial fibrillation in patients with Wolff-Parkinson-White syndrome: incidence after surgical ablation of the accessory pathway

A. D. Sharma, M.D., G. J. Klein, M.D., G. M. Guiraudon, M.D., and S. Milstein, M.D.

ABSTRACT The effect of surgical ablation of atroventricular accessory pathways on the incidence of atrial fibrillation in patients with Wolff-Parkinson-White syndrome was examined and the results of preoperative electrophysiologic testing were studied to determine factors predictive of outcome. Among 50 consecutive surgical cases, 19 patients were identified with a past history of at least one episode of spontaneous atrial fibrillation documented by electrocardiogram before surgery. The mean number of episodes of atrial fibrillation was 1.97/patient/year during a mean symptomatic period of 6.9 years before surgery. These patients were compared with 19 consecutive patients undergoing surgery during the same time period who had a history of only reciprocating tachycardia. Patients with atrial fibrillation had a significantly shorter antegrade accessory pathway effective refractory period (270 ± 39 vs 330 ± 107 msec; p < .05) and much faster ventricular rates during induced atrial fibrillation (shortest RR interval 219 ± 73 vs 294 ± 60 msec, p < .005; average RR interval 324 ± 109 vs 405 ± 127 msec, p < .01). Patients with atrial fibrillation also had longer PA intervals (47 ± 13 vs 37 ± 7 msec; p < .02). At preoperative electrophysiologic testing, 18 patients with atrial fibrillation had atrial fibrillation induced and 14 sustained the arrhythmia for longer than 10 min. In contrast, atrial fibrillation, although induced in 14 of 19 patients with reciprocating tachycardia, was not sustained in any. Thus electrophysiologic testing suggested that both accessory pathway properties and atrial vulnerability may predispose to atrial fibrillation. There were five episodes of atrial fibrillation in three patients (0.3/patient) during the first month after surgery. During a mean follow-up of 1.9 years, only one patient with an associated cardiomyopathy has had recurrent atrial fibrillation. We conclude that surgical ablation of the accessory pathway in patients without organic heart disease prevents further atrial fibrillation. This suggests that reciprocating tachycardia or ectopy mediated by the accessory pathway is the mechanism of induction of spontaneous atrial fibrillation in the majority of patients with Wolff-Parkinson-White syndrome.


ATRIAL FIBRILLATION occurring in patients with Wolff-Parkinson-White syndrome is a potentially life-threatening arrhythmia because it may lead to ventricular fibrillation.1 Accordingly, the pathogenesis of atrial fibrillation in these patients has been the subject of a number of investigations. Electrophysiologic testing of patients has revealed that atrioventricular (AV) reciprocating tachycardia can be induced in the majority of patients who have a past history of atrial fibrillation.2,3 Furthermore, the spontaneous degeneration of AV reciprocating tachycardia to atrial fibrillation has been observed during electrophysiologic testing.2,4,6 These findings suggest that atrial fibrillation can occur secondary to AV reentry by means of the accessory pathway and that the accessory pathway directly participates in the induction of atrial fibrillation.

However, only a minority of patients with a past history of atrial fibrillation are observed to have spontaneous degeneration of AV reciprocating tachycardia to atrial fibrillation during electrophysiologic testing.2,4,7 In patients in whom extrastimulus techniques have been required to induce atrial fibrillation,2,7,8 the induction of atrial fibrillation by a single extrastimulus has suggested primary atrial vulnerability to fibrillation.7 In these patients the accessory pathway would not be an obligatory link in the initiation of atrial fibril-
lation, and atrial fibrillation would be expected to recur even after the surgical ablation of the accessory pathway.

To determine the role of the accessory pathway in the pathogenesis of atrial fibrillation, we first compared the electrophysiologic properties of patients with atrial fibrillation to those with only reciprocating tachycardia. Second, we studied the effects of surgical ablation of the accessory pathway on the incidence of atrial fibrillation in patients with a past history of atrial fibrillation.

Methods

Patient selection. The records of 50 consecutive patients undergoing surgical ablation of an atrioventricular accessory pathway between August 1981 and February 1984 were examined. Indications for surgery included prior cardiac arrest, arrhythmias refractory to pharmacologic therapy, or intolerable side effects from pharmacologic therapy. Nineteen patients were identified with a past history of at least one episode of atrial fibrillation that had been documented by 12-lead electrocardiograms. Although a number of patients described irregular palpitations suggestive of atrial fibrillation, for the purposes of this study only electrocardiographically documented episodes of atrial fibrillation were included. Efforts were made to obtain all electrocardiographic tracings from referring physicians. This study group was compared with 19 consecutive patients undergoing surgical ablation of an accessory pathway during the same period who had no past history of atrial fibrillation documented by electrocardiogram.

Clinical evaluation. All patients provided their medical history and underwent physical examination, chest roentgenography, 12-lead electrocardiography, 24 hr Holter monitoring, exercise testing, and two-dimensional echocardiography. Male patients over the age of 40 and patients with clinical evidence of significant organic heart disease underwent coronary angiography and left ventriculography.

Study protocol. All patients underwent a standard protocol that consisted of a preoperative electrophysiologic study, intraoperative epicardial AV ring mapping, and a limited electrophysiologic study carried out 1 week after surgery using epicardial wires implanted during the operation. Written informed consent was obtained for each of these procedures. All patients underwent continuous electrocardiographic monitoring for 24 to 48 hr immediately after surgery. Patients were reviewed 6 weeks after surgery and a 12-lead electrocardiogram was obtained at that time. All patients were contacted at the end of the study period and any patients with symptoms of palpitation or documented irregularity of pulse on physical examination were asked to forward copies of electrocardiograms.

Electrophysiologic testing. Electrophysiologic evaluation was carried out with patients in the postabsorptive, nonsedated state after all antiarrhythmic medications had been discontinued for five half-lives. The protocol for the preoperative electrophysiologic study has been previously described. Briefly, this consists of the recording of basic intervals, sinoatrial conduction time, sinus node recovery time, atrial extrastimulus testing at cycle lengths 400 and 600 msec, antegrade incremental pacing to AV block, ventricular refractory periods at cycle length 400 and 600 msec, and ventricular incremental pacing to ventricular block. In all patients a quadripolar catheter was positioned in the coronary sinus. In cases where there was evidence to suggest a right-sided accessory pathway, the right atrial catheter was replaced by a bipolar atrial mapping catheter for tricuspid AV ring mapping. Standard criteria for the participation of an accessory pathway in atrioventricular reciprocating tachycardia were used. In patients in whom atrial fibrillation was not induced before this, rapid atrial pacing at rates of 400 to 500 beats/min was performed until atrial fibrillation was induced. The pacing stimuli used throughout the study were square-wave pulses of 2 msec duration at an amplitude two times diastolic threshold.

Surgical technique. The surgical approach to the ablation of accessory pathways has been previously described. During the study period the technique varied and included both open and closed techniques for ablating left-sided accessory pathways. Intraoperatively, ventricular activation at the AV ring was determined during right atrial pacing. Retrograde atrial activation at the AV ring was determined during ventricular pacing or reciprocating tachycardia. In each case the findings of the previous electrophysiologic study were confirmed. At the conclusion of the procedure, epicardial wires were sutured to the right atrium and right ventricle for use during postoperative electrophysiologic testing.

Postoperative electrophysiologic testing. One week after surgery, all patients underwent repeat electrophysiologic testing. This included atrial incremental pacing to the point of AV block and ventricular incremental pacing to the point of ventriculoatrial block. When necessary, atrial and ventricular extra-stimulus testing were used to clarify the absence of conduction characteristic of accessory pathways.

Statistical analysis was performed with the Student t test for unpaired data, chi square analysis, and the Wilcoxon signed-rank test to compare preoperative and postoperative incidence of atrial fibrillation.

Results

Patient characteristics. Nineteen patients were identified with a past history of atrial fibrillation among the 50 consecutive patients who underwent surgical ablation of an AV accessory pathway. The mean age was 28.9 years (range 12 to 58). Fourteen patients were male and five were female. Sixteen patients had no evidence of cardiac disease other than Wolff-Parkinson-White syndrome, and one had Ebstein’s anomaly, one mitral valve prolapse, and one patient an idiopathic congestive cardiomyopathy. There was no evidence for coronary artery disease in this group of patients. Two of these patients had a past history of cardiac arrest. The onset of atrial fibrillation was related to various factors, including exercise (n = 8), sleep (n = 2), emotion (n = 1), alcohol ingestion (n = 1), and no known factors (n = 7).

The study group was compared with 19 consecutive patients who underwent surgical ablation of an accessory pathway during the same period. None of these patients with only reciprocating tachycardia had organic heart disease. Patient characteristics are summarized in table 1.

Electrophysiologic testing. At the initial electrophysiologic study, both groups of patients were characterized in terms of accessory pathway location and properties, functional properties of the normal AV conduction
system, and ventricular response to atrial fibrillation.  

Accessory pathway characteristics. In patients with paroxysmal atrial fibrillation, accessory pathways were located in the left lateral position in 11, the posteroseptal region in nine, and the right anteroseptal location in six. Seven of 19 patients had multiple accessory pathways, five had dual AV nodal pathways, and one had a nodoventricular Maheim pathway. Thus, 11 of 19 patients had either dual AV nodal pathways or multiple accessory pathways (58%) that could contribute to multiple sites for retrograde atrial activation. Among the patients without atrial fibrillation, significantly fewer subjects (n = 4) had only single accessory pathway (n = 2) and/or dual AV nodal pathways (n = 3) (p < .025). No other differences were noted in location of accessory pathways. However, all patients with atrial fibrillation had ventricular preexcitation, whereas only 14 patients without atrial fibrillation had antegrade accessory pathway conduction (p < .05). That is, no patient with a concealed accessory pathway had atrial fibrillation.

The results of electrophysiologic testing are summarized in table 2. Patients with paroxysmal atrial fibrillation had significantly shorter antegrade accessory pathway effective refractory period (drive cycle length 600 msec) (APERP₀₋₀ 270 ± 39 msec) compared with patients with only reciprocating tachycardia (330 ± 107 msec) (p < .05). Consistent with this observation, when atrial fibrillation was induced during electrophysiologic testing, patients with paroxysmal atrial fibrillation had more rapid ventricular rates. The group mean of the shortest interval between consecutive preexcited beats (SRR) was 219 ± 73 msec, and the mean of the average interval between preexcited beats (ARR) was 334 ± 109 msec. In patients with only reciprocating tachycardia, the mean SRR was 294 ± 60 msec (p < .005) and the mean ARR was 405 ± 127 msec (p < .01). The differences between the two patient groups were greater than the means indicated because one patient with atrial fibrillation and a cardiomyopathy had a very slow ventricular rate during atrial fibrillation. In the remaining 18 patients with atrial fibrillation, the mean SRR was 205 ± 45 msec.

AF reciprocating tachycardia of the orthodromic type was induced in 37 patients. In addition, five patients had antidromic tachycardia and two patients had AV nodal reentrant tachycardia. No differences were observed between the two groups of patients in the ease of initiation of reciprocating tachycardia, tachycardia mechanism, or tachycardia cycle length. There were no differences in retrograde effective refractory period of the accessory pathway.

**Functional properties.** No differences were observed between the two patient groups in spontaneous sinus node cycle length, AH interval, sinus node recovery time, sinoatrial conduction time, or atrial and ventricular functional and effective refractory periods. The PA interval was significantly longer in patients with atrial fibrillation (47 ± 13 msec) compared with patients with only reciprocating tachycardia (37 ± 7 msec; p < .02). Ten patients with atrial fibrillation had a PA greater than 45 msec, and in eight of these patients the left atrium was adequately visualized echocardiographically. The left atrial dimensions were normal.

<table>
<thead>
<tr>
<th>TABLE 2</th>
<th>Electrophysiologic parameters in patients with atrial fibrillation compared with patients with only reciprocating tachycardia</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>AF (n = 19)</td>
</tr>
<tr>
<td>AERP₀₋₀ (msec ± SD)</td>
<td>270 ± 39</td>
</tr>
<tr>
<td>AERP₀₋₀, (retrograde)</td>
<td>268 ± 40</td>
</tr>
<tr>
<td>SRR (AF)</td>
<td>219 ± 73</td>
</tr>
<tr>
<td>ARR (AF)</td>
<td>324 ± 109</td>
</tr>
<tr>
<td>RTCL</td>
<td>307 ± 61</td>
</tr>
<tr>
<td>AERP₀₋₀</td>
<td>199 ± 36</td>
</tr>
<tr>
<td>PA interval</td>
<td>47 ± 13</td>
</tr>
<tr>
<td>AH interval</td>
<td>65 ± 35</td>
</tr>
<tr>
<td>WCL (postop)</td>
<td>290 ± 61</td>
</tr>
<tr>
<td>Multiple AP or dual AVN</td>
<td>11</td>
</tr>
<tr>
<td>Antegrade preexcitation</td>
<td>19</td>
</tr>
</tbody>
</table>

APERP = accessory pathway effective refractory period at designated cycle length; SRR = shortest RR interval in atrial fibrillation; ARR = average interval during atrial fibrillation; RTCL = cycle length of AV reciprocating tachycardia; AERP = atrial effective refractory period; WCL = Wenckebach cycle length; AP = accessory pathway; AVN = AV node; other abbreviations as in table 1.
(<40 mm) in seven, with one patient (with cardiomyopathy) having an enlarged left atrium. Thus slowing of intra-atrial conduction was rarely associated with structural abnormality in these patients. AV nodal conduction could be assessed only in the minority of patients, but antegrade (254 ± 41 vs 262 ± 43 msec) and retrograde AV nodal effective refractory periods (322 ± 117 vs 371 ± 145 msec) did not differ in those patients where it could be measured. We therefore compared the AV nodal Wenckebach cycle length in all patients postoperatively and observed no differences between the two groups of patients. However, among patients with paroxysmal atrial fibrillation, nine of 19 patients had an AH interval less than or equal to 60 msec and a postoperative Wenckebach cycle length less than 300 msec, consistent with ‘enhanced’ AV nodal conduction. Only two patients with reciprocating tachycardia had similarly rapid AV nodal conduction.

**Atrial fibrillation during electrophysiologic testing.** Induction of atrial fibrillation was attempted during the preoperative electrophysiologic study in all but one patient. This patient had a documented past history of rapid atrial fibrillation and refused to be studied off medication. The modes of induction of atrial fibrillation are described in table 3. All patients with a past history of atrial fibrillation had atrial fibrillation induced only during electrophysiologic testing and the atrial fibrillation was sustained more than 10 min in 14 of 18. When clinically indicated, these patients were given disopyramide (n = 9) or procainamide (n = 3) intravenously, and if atrial fibrillation failed to terminate, a transthoracic cardioversion shock was used to restore sinus rhythm (n = 4). In contrast, atrial fibrillation could be induced only in 14 of the patients who had no past history of atrial fibrillation, and in only one of these patients was atrial fibrillation sustained more than 10 min.

The modes of induction of atrial fibrillation suggested that the arrhythmia was easier to induce in those patients who had documented spontaneous episodes of atrial fibrillation. These patients frequently developed atrial fibrillation inadvertently during atrial or coronary sinus catheter placement (n = 6) or after a single atrial extrastimulus (n = 3) (figure 1). In the latter episodes, the onset of atrial fibrillation was not associated with AV reentry. The preceding modes of induction were rarely observed in patients with a past history of only reciprocating tachycardia. The latter patients usually required rapid atrial pacing (n = 10) to initiate a brief period of atrial fibrillation (figure 2). Among all 38 patients, only four (10.5%) were observed to have spontaneous degeneration of reciprocating tachycardia to atrial fibrillation (figure 3). One patient with a history of only reciprocating tachycardia was observed to rapidly revert to sinus rhythm after reciprocating tachycardia degenerated to atrial fibrillation. This rapid, spontaneous cardioversion could have accounted for the lack of documentation of spontaneous atrial fibrillation.

Some of the modes of onset of atrial fibrillation are illustrated in figures 1 to 4. In these examples, earliest spontaneous atrial activity was observed to occur at a site removed from the accessory pathway, usually the high right atrium. This suggests that onset of atrial fibrillation was related to intra-atrial factors rather than to an abnormality of the atrium at the site of the atrial insertion of the accessory pathway. The induction of atrial fibrillation during ventricular pacing (figure 4) indicates that patients with concealed accessory pathways may potentially be at risk for the development of atrial fibrillation.

**Incidence of atrial fibrillation (table 4).** A past history of atrial fibrillation was determined by electrocardiographic documentation. An arbitrary upper limit of 10 episodes of atrial fibrillation was allowed per patient for purposes of analysis. This was done to prevent the results of a few patients with many episodes from skewing the data. In addition, as the episodes of atrial fibrillation became more frequent, it was increasingly difficult to obtain accurate histories and electrocardiographic documentation. All patients had an episode of atrial fibrillation during the year before surgery and this frequently precipitated referral for surgery. During a mean preoperative symptomatic period of 6.9 ± 5.8 years there were 89 episodes of atrial fibrillation in 19 patients. The incidence of atrial fibrillation in each patient was determined with a resultant average incidence of 1.97 ± 2.8/patient/year. This is undoubtedly

### Table 3

**Induction of atrial fibrillation during electrophysiologic testing**

<table>
<thead>
<tr>
<th>Method of induction</th>
<th>AF (n = 19)</th>
<th>RT (n = 19)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Induction of AF</td>
<td>18/18(^a)</td>
<td>14/19</td>
</tr>
<tr>
<td>AF sustained 10 min</td>
<td>14/18</td>
<td>0/14(^b)</td>
</tr>
<tr>
<td>Method of induction</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Single extrastimulus</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>RT to AF</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>Rapid atrial pacing</td>
<td>6</td>
<td>10</td>
</tr>
<tr>
<td>Mechanical stimulation</td>
<td>6</td>
<td>0</td>
</tr>
</tbody>
</table>

\(^a\)Atrial fibrillation induction not attempted in one patient with documented rapid atrial fibrillation.

\(^b\)p < .001.
**FIGURE 1.** Induction of atrial flutter by a single atrial extrastimulus. After a drive of 8 beats at cycle length 400 msec, a premature stimulus at coupling interval 190 msec induces atrial flutter with 2:1 antegrade conduction via an accessory pathway. In this patient with a single left lateral atrioventricular accessory pathway, earliest spontaneous atrial activity is observed in the His bundle recording (HBE), at a site distant from the atrial insertion of the accessory pathway. The onset of atrial flutter is not related to atrioventricular reentry. Atrial flutter rapidly degenerated to atrial fibrillation. HRA = high right atrium; RV = right ventricle; CS_p = proximal coronary sinus; CS_d = distal coronary sinus.

**FIGURE 2.** Induction of atrial fibrillation during atrial incremental pacing. After antegrade block in the accessory pathway, an alteration is observed in the high right atrial electrogram (first arrow). On the second cycle after block, activity in the high right atrium is observed to precede the stimulus artifact (second arrow). Organized repetitive atrial activity arising in the high right atrium continues for a further six cycles before degenerating to atrial fibrillation, which was sustained when the pacing was terminated. Abbreviations as in figure 1.
FIGURE 3. Spontaneous degeneration of AV reciprocating tachycardia to atrial fibrillation. Orthodromic AV reciprocating tachycardia used a left lateral accessory pathway in this patient. At the point indicated by the first arrow, spontaneous premature activation is observed in the high right atrium. Repetitive atrial activity follows. However, reciprocating tachycardia continues for a further two cycles before atrial activation at the coronary sinus is altered. After this, rapid antegrade conduction via the accessory pathway is observed during atrial fibrillation. Abbreviations as in figure 1.

a conservative estimate because of the requirement for documentation, and the upper limit of fibrillation episodes set per patient. Two patients had only one episode of atrial fibrillation.

Surgical results in this group of 38 patients include successful ablation of accessory pathways in all but two patients, no mortality, and one patient with heart block. Surgical failures occurred with the use of the open heart technique in patients with multiple accessory pathways. During the first month after surgery there were five episodes of atrial fibrillation in three patients (0.3/patient). These early episodes of atrial fibrillation may have been related to postoperative inflammation. No episodes of atrial fibrillation were induced at the 1 week postoperative electrophysiologic study. During a mean long-term follow-up of 1.9 ± 0.7 years (range 0.8 to 3.2), only one patient has had recurrent atrial fibrillation. This particular patient, with a history of preoperative slow atrial fibrillation, has a cardiomyopathy. His ventricular response is well controlled and he has been maintained without medication. With the following exceptions, all patients have been maintained without medication. One patient with unsuccessful surgical ablation of the accessory pathway has been treated with amiodarone successfully. One patient had AV nodal reentrant tachycardia 1 year after surgery. This patient has been treated with propafenone and has had no further episodes of tachycardia. Preoperatively, this patient had reciprocating tachycardia and atrial fibrillation using an accessory pathway despite medical therapy.

Discussion

Recurrent paroxysmal atrial fibrillation has been reported to occur in up to 32% of patients with Wolff-Parkinson-White syndrome. Atrial fibrillation in these patients may be potentially life threatening when it leads to rapid ventricular rates that may ultimately result in ventricular fibrillation. When pharmacologic management fails to control these arrhythmias, surgical ablation of the accessory pathway is a suitable alternative. However, it is not known whether surgical

### TABLE 4
Incidence of atrial fibrillation

<table>
<thead>
<tr>
<th></th>
<th>Duration of symptoms (yr)</th>
<th>Episodes</th>
<th>Patients</th>
<th>Incidence (/patient/yr)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preoperative</td>
<td>6.9 ± 5.8</td>
<td>89</td>
<td>19</td>
<td>1.97 ± 2.54&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>1 month postop</td>
<td>0.08</td>
<td>5</td>
<td>3</td>
<td>—</td>
</tr>
<tr>
<td>Follow-up</td>
<td>1.9 ± 0.7</td>
<td>5</td>
<td>1</td>
<td>0.098 ± 0.42&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

<sup>a</sup>p < .01.
ablation of the accessory pathway will merely result in continued atrial fibrillation without preexcitation or whether it will actually alter the incidence of atrial fibrillation. Our study attempted to determine the effect of surgical ablation of the accessory pathway on the incidence of recurrent atrial fibrillation and to determine whether there are certain electrophysiologic parameters that identify patients in whom surgery is more likely to prevent recurrent atrial fibrillation.

Patients with a past history of recurrent, spontaneous atrial fibrillation are characterized by the presence of antegrade preexcitation, the frequent presence of multiple accessory pathways, and the presence of an accessory pathway with a relatively short antegrade effective refractory period. This combination of factors contributes to the excessively rapid ventricular rates that may occur in this group of patients when atrial fibrillation develops.\(^1\)\(^,\)\(^2\) A major difference observed between patients with a past history of atrial fibrillation and those with only reciprocating tachycardia was that the atrial fibrillation tended to be sustained when it was induced in the laboratory. In contrast, patients with reciprocating tachycardia had nonsustained atrial fibrillation in the electrophysiology laboratory and even this required more aggressive stimulation techniques. Two potential differences between these groups of patients may have contributed to atrial fibrillation being sustained. First, rapid ventricular responses to atrial fibrillation may result in hemodynamic deterioration and hypotension,\(^1\)\(^3\) with a resultant reflex increase in sympathetic neural activity.\(^1\)\(^6\) This increased sympathetic neural activity may predispose to the maintenance of atrial fibrillation and the development of more rapid ventricular response rates. This phenomenon has been observed in the electrophysiology laboratory in response to isoproterenol.\(^1\)\(^7\) Second, the PA interval was observed to be significantly longer in patients with a past history of atrial fibrillation. This measurement may provide an index of intra-atrial conduction time.\(^1\)\(^8\) Thus, in patients with potentially slower right intra-atrial conduction, there may be a predisposition to developing sustained atrial fibrillation. Therefore the results of the electrophysiologic study suggested a multifactorial etiology for sustained atrial fibrillation, including factors related to both the accessory pathway and slow conduction in the atrium.

The mode of initiation of atrial fibrillation in the laboratory more frequently suggested the presence of primary atrial vulnerability to atrial fibrillation. We observed spontaneous degeneration of AV reciprocating tachycardia to atrial fibrillation in only 16% of our patients, which is similar to the 26% observed by

---

**FIGURE 4.** Induction of atrial flutter during ventricular incremental pacing. The arrow points to the first complex of atrial flutter. This patient had two atrioventricular accessory pathways, in the posteroseptal and left lateral region, both capable of conducting retrogradely. The presence of multiple pathways may lead to atrial activation from multiple sites during spontaneous ventricular ectopy. A collision of multiple atrial activation fronts may have provided the substrate for intra-atrial reentry. Atrial flutter rapidly degenerated to sustained atrial fibrillation in this case.
Bauernfeind et al. and 19% observed by Sung et al. It is not clear to what extent this mode of induction of atrial fibrillation reflects primary atrial vulnerability. In the majority of patients, extrastimulus techniques or mechanically induced extrastimuli induced atrial fibrillation. Examination of the onset of atrial flutter and fibrillation by these modes did not indicate that the accessory pathway participated. We and others have observed that sustained atrial fibrillation is rarely induced in normal patients during catheter placement or extrastimulus testing. This suggests that patients with atrial fibrillation induced by catheter placement or extrastimulus testing very likely have a primary atrial vulnerability to the development of atrial fibrillation.

On the basis of results of electrophysiologic testing, we could not determine whether recurrent atrial fibrillation was purely a function of the accessory pathway properties or whether there was indeed primary atrial vulnerability to the development of atrial fibrillation. We hypothesized that if recurrent atrial fibrillation is due entirely to the presence of the accessory pathway, then surgical ablation of the accessory pathway should prevent recurrent atrial fibrillation. In contrast, if there is primary atrial vulnerability then atrial fibrillation may continue to occur after surgery. Indeed, one might expect atrial fibrillation to occur more frequently in the immediate postoperative phase.

A total of 89 episodes of atrial fibrillation had been documented in 19 patients during a mean preoperative period of 6.9 years. This produced a conservative estimate of 1.97 episodes of atrial fibrillation per patient/year. During a mean follow-up of 1.9 years, only one patient had recurrent atrial fibrillation (p < .01). On the basis of this dramatic reduction in the recurrence of atrial fibrillation, we conclude that the accessory pathway is of prime importance in the induction of spontaneous atrial fibrillation and that any intratrial abnormalities are of secondary importance and may contribute only to sustaining atrial fibrillation once initiated. This finding is of considerable significance because it allows us to maintain this group of patients without antiarrhythmic drug therapy after surgery. Recurrent atrial fibrillation after surgery could still be potentially dangerous in a high percentage of our patients. We observed that nine of 19 patients had AV nodal properties consistent with enhanced AV nodal conduction and might be able to sustain relatively rapid ventricular rates in the event of recurrent atrial fibrillation. Thus it is of considerable importance that atrial fibrillation does not recur after surgery. It will be of importance to determine whether this group of young patients with apparent atrial vulnerability will develop a high incidence of atrial fibrillation as they age.

It might be argued that the duration of follow-up is inadequate to determine the precise effects of surgery. However, among our patients, all had had at least one episode of atrial fibrillation during the year before surgery. In addition, only two patients had had a single episode of atrial fibrillation. Thus the great majority of our patients had multiple episodes of atrial fibrillation during their preoperative period. In addition, most patients showed a trend toward increasing frequency and severity of episodes of atrial fibrillation despite pharmacologic management. Only two of our patients required pharmacologic therapy after surgery, and thus, in the absence of antiarrhythmic drug therapy, we might have expected to have seen an even greater incidence of recurrent atrial fibrillation in the absence of a surgical effect.

Certain insights were provided by the single patient who continued to have recurrent atrial fibrillation. This patient was markedly different from the remaining population of patients with atrial fibrillation. This particular patient had a cardiomyopathy and thus was one of the few patients with organic heart disease in our patient population. In addition, the shortest RR interval during induced atrial fibrillation in this patient was 450 msec. This patient was therefore distinctly different in that his ventricular rate during atrial fibrillation was generally slow even in the absence of pharmacologic therapy. The observation that the occurrence of atrial fibrillation in patients with Wolff-Parkinson-White syndrome and organic heart disease may have different substrates than patients without heart disease is consistent with previous findings. Bauernfeind et al. demonstrated that a subgroup of patients with Wolff-Parkinson-White syndrome without inducible AV reentrant tachycardia frequently had organic heart disease (86%). Thus organic heart disease may lead to atrial fibrillation even in the absence of AV reciprocating tachycardia. Our patient with a cardiomyopathy did have AV reciprocating tachycardia, but one would expect that this would only make the patient more likely to have atrial fibrillation in the presence of organic heart disease.

We conclude that patients with Wolff-Parkinson-White syndrome who have no organic heart disease but have antegrade preexcitation and develop rapid ventricular response rates during atrial fibrillation are not likely to have recurrent atrial fibrillation after surgical ablation of their accessory pathways. In contrast, patients with a history of paroxysmal atrial fibrillation who have organic heart disease, absent antegrade con-
duction via an accessory pathway, or slow ventricular rates during induced atrial fibrillation may be likely to have recurrent atrial fibrillation after ablation of their accessory pathways. This latter group may require continued antiarrhythmic therapy after surgery.

We express our appreciation to A. MacDonald, R.N., and N. Smith, R.N., for assistance in the electrophysiology laboratory, and to Mrs. D. Vigna for secretarial work.

References
Atrial fibrillation in patients with Wolff-Parkinson-White syndrome: incidence after surgical ablation of the accessory pathway.
A D Sharma, G J Klein, G M Guiraudon and S Milstein

Circulation. 1985;72:161-169
doi: 10.1161/01.CIR.72.1.161

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1985 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/72/1/161

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/