THE ROLE OF THE ACCESSORY PATHWAY IN THE ONSET OF ATRIAL FIBRILLATION IN WOLFF-PARKINSON-WHITE SYNDROME —Electrophysiological Examination Before and After Surgical Ablation —

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To determine the role of the accessory pathway in the pathogenesis of atrial fibrillation, we compared electrophysiological findings in 17 patients (44.7 ± 10.2 years) with a history of atrial fibrillation before and after surgical ablation of the accessory pathway. The PA interval was shortened, and the atrial refractory periods and the potential minimal wavelength of an atrial impulse (FRPa/PA) were significantly increased, after surgery. Fragmented atrial activity (an increase of 150% or more in the duration of the high right atrial electrogram) was observed in 80% of the patients before surgery and in 25% after surgery. Its zone was significantly decreased after surgery. Repetitive atrial firing was defined as the occurrence of 3 or more successive atrial electrograms induced by a premature stimulation. This was observed in 60% of the patients before surgery, but in none after surgery. Atrial fibrillation was induced in 16 patients during the preoperative study, but in only 1 patient postoperatively. In conclusion, these results suggest that accessory pathways affect atrial vulnerability and play an important role in the onset of atrial fibrillation in WPW syndrome.

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ATRIAL fibrillation in patients with Wolff-Parkinson-White (WPW) syndrome is a potentially life-threatening arrhythmia because it may lead to a rapid ventricular response! However, the mechanism by which atrial fibrillation is initiated in the presence of the accessory pathway is not fully understood. It has been shown that preceding atrioventricular reciprocating tachycardia plays an important role in its genesis!—4 On the other hand, it has been demonstrated that the accessory pathway itself may initiate atrial fibrillation by virtue of its architecture!–6 There are reports of differences in several electrophysiologic parameters between patients with and without atrial fibrillation in WPW syndrome. Induction of atrial fibrillation has been shown to be reduced after surgical ablation of accessory pathways. However, there has been no

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report concerning atrial properties and/or atrial vulnerabilities after surgical ablation. To determine the role of the accessory pathway in the pathogenesis of atrial fibrillation, we compared the electrophysiological findings in patients with a history of atrial fibrillation before and after surgical ablation of the accessory pathway.

METHODS

Patients
Seventeen patients with WPW syndrome who had a history of paroxysmal atrial fibrillation and who underwent surgical ablation of accessory pathways were examined. The mean age was 44.7 ± 10.2 years. Twelve patients were male and 5 were female. The surgery was performed with cryoablation of the accessory pathways by the endocardial approach.

Electrophysiological Studies
Preoperative electrophysiological studies were performed after discontinuing antiarrhythmic drugs. Two bipolar catheters were positioned at the high right atrium and the right ventricular apex for stimulations. Two quadripolar catheters were positioned at the high lateral right atrium and the His bundle, and an octopolar catheter was positioned in the coronary sinus (CS) for electrogram recordings and stimuli. High right atrial, right ventricular and CS incremental pacing and extrastimulation were performed at a cycle length of 600 msec. The location of accessory pathways was determined by the sequence of retrograde atrial activation during orthodromic atioventricular reciprocating tachycardia and during ventricular pacing. Postoperative electrophysiological studies were performed about 1 month after surgery using the same protocol.

Measurements and Definitions
At a cycle length of 600 msec, intraatrial conduction time (PA interval) and interatrial conduction time (P-CS distal interval) were measured from the stimulus artifact at the high right atrium to the beginning of the atrial electrograms at the His bundle electrogram and at the distal portion of the CS area, respectively. Intraatrial conduction delay and interatrial conduction delay were defined as an increase of 20 msec or more in the conduction time as a result of premature stimulation. The effective refractory period and the functional refractory period of the atrium (ERP_A, FRP_A) were measured. The potential minimal wavelength of an atrial impulse was defined as FRP_A/PA^8. Fragmented atrial activity was defined as an increase of 150% or more in the duration of the high lateral atrial electrogram as a result of premature stimulation^9. Repetitive atrial firing was defined as the occurrence of 3 or more successive atrial electrograms induced by a premature stimulus. Intraatrial and interatrial conduction delay zones, the fragmented atrial activity zone and repetitive atrial firing zone were defined as the resulting range of S1-S2 intervals, respectively. During electrophysiological studies, we examined whether atrial fibrillation was induced or not, and if so, the onset of atrial fibrillation was analyzed by atrial electrograms at various regions.

Statistics
Results were expressed as the mean ± standard deviation. Statistical analysis was performed with the Wilcoxon signed rank test to compare preoperative and postoperative data. Categorical data were compared using the χ^2 test.

RESULTS

Patient Characteristics Before Surgery
Evidence of underlying organic heart disease was found in only 1 patient who had mitral regurgitation due to infectious bacterial endocarditis. Sixteen patients had manifest WPW syndrome, while in 1 patient this condition was concealed. Accessory pathways were located in the left lateral position in 12 patients, the posteroseptal region in 4, and the right lateral region in 3. One patient had multiple accessory pathways and 1 had dual AV nodal pathways. The shortest preexcited RR interval during atrial fibrillation was 276 ± 61 msec.

Electrophysiological Studies
After surgical ablation, the PA interval at a cycle length of 600 msec was significantly decreased while the P-CS distal interval was unchanged. Intraatrial conduction delay and
interatral conduction delay were not significantly changed. ERP_A and FRP_A were significantly increased after surgery. FRP_A/PA was significantly increased (Table I). Fragmented atrial activity was observed in 80% of the patients before surgery and in 24% after surgery. The fragmented atrial activity zone was significantly decreased after surgery. Repetitive atrial firing was observed in 60% of the patients in a zone of 18±22 msec before surgery, but in none of the patients after surgery (Tables I and II).

Atrial fibrillation was induced during the preoperative electrophysiologic study in all but 1 patient. Atrial fibrillation was initiated during right atrial stimulation in 9 patients, right ventricular stimulation in 2, reciprocating tachycardia in 4 and spontaneously during catheter positioning in 2. Earliest atrial activation during the initiating tachycardia occurred at a high lateral right atrial site in 4 patients, at the electrode closest to the accessory pathway in 6 and at an unknown site in 6. However, atrial fibrillation was induced in only 1 patient with mitral regurgitation after surgery.

**DISCUSSION**

Atrial fibrillation is a common finding in WPW syndrome and is observed in up to 32% of patients with or without organic heart disease. In the absence of overt cardiac disease and atrial enlargement, potential causative factors include preceding atrioventricular reciprocating tachycardia, intrinsic atrial properties predisposing to atrial fibrillation or the accessory pathway itself or its atrial/ventricular interface. However, it has been unclear whether recurrent atrial fibrillation is purely a function of the properties of the accessory pathway or whether there is a primary atrial vulnerability to the development of atrial fibrillation.

In previous studies of WPW syndrome, it was reported that the PA interval was significantly longer in a group with atrial flutter or fibrillation than in a group without this condition. ERP_A was similar in the 2 groups and FRP_A was either shorter or similar in the atrial fibrillation group. Allessie et al showed that the potential minimal wavelength of an atrial impulse...
(product of conduction velocity and functional refractory period of the atrium) was shorter in a group with atrial flutter or fibrillation than in a group without it in a dog model. Fujimura et al reported that a comparable measure of the potential minimal wavelength (FRPA/PA) was shorter in a group of atrial fibrillation than in a group without it in WPW syndrome patients. It has also been reported that the zones of fragmented atrial activity, interatrial conduction delay and repetitive atrial firing were wider in patients with atrial fibrillation than in patients without it, and that these parameters showed atrial vulnerability. In the present study, fragmented atrial activity and repetitive atrial firing were observed in 80% and 60% of the patients, respectively, and atrial fibrillation started at the high right atrium in some patients before surgery. These findings suggest that atrial factors may play an important role in the occurrence of atrial fibrillation. There are no reports regarding atrial properties before and after surgical ablation of accessory pathways. In our present study, the PA interval was shortened, ERP_A, FRP_A and FRPA/PA were increased, and the zones of fragmented atrial activity and repetitive atrial firing became narrower after surgical ablation. These changes may be related to a reduced occurrence of atrial fibrillation after surgery.

The above atrial properties (ERP_A, FRP_A and FRPA/PA, fragmented atrial activity and repetitive atrial firing) were related to the occurrence of atrial fibrillation. Some patients had atrial fibrillation while others did not, even though they had accessory pathways. Some episodes of atrial fibrillation began in the high right atrium regardless of the location of the accessory pathway. These data suggest that atrial abnormalities play an important role in the development of atrial fibrillation. However, there is evidence that the accessory pathway itself may also be important in the occurrence of atrial fibrillation. First, operative correction of preexcitation generally results in the cure of recurrent atrial fibrillation, as well as reciprocating tachycardia. Second, Jackman et al postulated that microreentry with a branching framework of the accessory pathway contributed to atrial fibrillation. Third, Iwa and Iwase reported that the accessory atrioventricular connections were thicker at their atrial origin and their myocardial cells appeared hypertrophic and disarrayed. These findings were related to atrial fibrillation. Fourth, some episodes of atrial fibrillation apparently begin near the accessory pathway. Fifth, although atrial fibrillation is rarely induced in normal patients, it is frequently induced in patients with WPW syndrome during catheter placement or extra-stimulus testing. Sixth, if atrial fibrillation is due entirely to the presence of accessory pathways, ablation of the accessory pathways should prevent atrial fibrillation. In contrast, if there is primary atrial vulnerability, atrial fibrillation may continue to occur after surgery. Based on the dramatic reduction in atrial vulnerability and atrial fibrillation after surgery, we considered that the accessory pathway was of prime importance in the induction of spontaneous atrial fibrillation and that intraatrial abnormalities were of secondary importance. However, the results of this study did not clarify whether the accessory pathway itself or its atrial/ventricular interface affect atrial fibrillation, since incision and cryoablation by surgery were performed within the range of the atrioventricular connection at the atrial site. In a recent study of catheter ablation of accessory pathways induced atrial fibrillation was still observed, although somewhat reduced in intensity, in half of the patients after catheter ablation. Thus, the atrial/ventricular interface and/or branching networks of the accessory pathways and histological abnormalities of the connections may play an important role in the induction of atrial fibrillation.

In conclusion, the present data suggest that accessory pathways affect atrial vulnerability and play an important role in the onset of atrial fibrillation in WPW syndrome.

REFERENCES

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