Surgical Correction
of Anomalous Left Ventricular Pre-excitation:
Wolff-Parkinson-White (Type A)

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SUMMARY
This report describes two patients with the Wolff-Parkinson-White syndrome including episodes of supraventricular tachycardia and atrial fibrillation. Both patients had Type A electrocardiograms. Electrophysiological studies demonstrated pre-excitation and evidence that the site of pre-excitation involved the left ventricle. The effective refractory periods of the accessory pathway during atrioventricular conduction were 240 and 220 msec respectively. Epicardial mapping at the time of surgery showed that anomalous excitation began adjacent to the annulus of the mitral valve near a marginal branch of the left circumflex coronary artery. An incision which separated the atrial muscle from the annulus of the mitral valve at the region of anomalous excitation abolished the delta wave. Epicardial maps after surgery showed normal ventricular activation and follow-up studies have shown normal electrocardiograms and no arrhythmias.

Additional Indexing Words:
Kent bundle Electrophysiology Arrhythmias Heart surgery

In 1930 Wolff, Parkinson, and White described a syndrome consisting of a short P-R interval and a wide QRS complex on the electrocardiogram in healthy individuals who were prone to paroxysms of tachycardia. Holzman and Scherf first postulated that one or more muscular bridges between the atrium and ventricle, similar to those described by Paladino in 1876 and by Kent in 1893 might account for the Wolff-Parkinson-White (WPW) syndrome; and direct evidence for such a bridge in a patient with WPW was provided by Wood, Wolferth, and Geckler. In 1967 Durrer and Burchell reported epicardial mapping studies in patients with Type B Wolff-Parkinson-White. Those studies revealed early anomalous ventricular excitation at the right lateral margin of the heart. Stimulated by those reports, we carried out epicardial mapping in a patient with Type B Wolff-Parkinson-White and recurrent episodes of paroxysmal atrial tachycardia. After mapping, the atrioventricular ring was divided at the area of anomalous excitation and both the delta wave and episodes of atrial tachycardia were abolished. Subsequent to that report we have evaluated a larger group of patients with the Wolff-Parkinson-White syndrome and have operated successfully on a selected subgroup of these patients. The purpose of this report is to describe two patients with left ventricular pre-excitation and to demonstrate the feasibility of complete surgical correction of Type A Wolff-Parkinson-White syndrome. To our knowledge, this is the first report of the successful surgical treatment of patients with Type A Wolff-Parkinson-White syndrome.

Case Reports
Case #1
L. S. is a 51 year old, white, married female who was referred to Duke University Medical Center on February 19, 1972. The patient was in excellent health and gave no history of heart disease prior to the age of 28. In 1949 she first noted the onset of episodic attacks of palpitation. These were sudden in onset, lasted 10-15 minutes and usually stopped spontaneously. The attacks occurred four to five
times per year. In 1969 she had an episode which was more prolonged and required hospitalization. An electrocardiogram confirmed the diagnosis of supraventricular tachycardia with a regular ventricular rate of 200 beats/min. She was converted to sinus rhythm with intravenous Cedilanid and the postconversion electrocardiogram revealed the Wolff-Parkinson-White abnormality, Type A. In 1969 the attacks of paroxysmal rapid heart action began to occur more frequently, lasted longer and were associated with episodes of severe substernal pain. On several occasions the ventricular rate was documented to be approximately 240 beats/min, and electrocardiograms confirmed the clinical impression of atrial fibrillation with a rapid ventricular response. From 1970 to 1972 she was tried on a variety of medications singly and in various combinations. These included digoxin 0.25 mg/day, procainamide 250 mg q 6 h, quinidine 400 mg q 6 h, and inderal 40 mg q 6 h. She continued to have attacks despite these medications. The dosage of propranolol had to be lowered because of sinus bradycardia, and procainamide treatment had to be terminated because of a lupus-like reaction. Because of these recurring difficulties, she was referred to Duke for further evaluation.

On physical examination the patient appeared well. Her blood pressure was 130/180 and the pulse was 74. The general aspects of the physical examination were unremarkable. The jugular venous pressure and the venous pulse were normal. The examination of the heart revealed that the PMI was located 7 cm to the left of the midsternal line in the fifth intercostal space and was unremarkable. Intermittently, a midsystolic click and late systolic musical murmur could be heard. The chest X-ray and cardiac fluoroscopy were normal. An echocardiogram revealed the characteristic abnormalities of a balloon mitral valve. The electrocardiogram showed Type A Wolff-Parkinson-White syndrome (fig. 1, upper panel). The vectorcardiogram confirmed that the initial slurred forces were directed slightly to the right, anteriorly and inferiorly.

The patient was taken to the catheterization laboratory and an electrophysiologic study was performed. Recordings from the region of the bundle of His revealed a P–H interval of 90 msec and an H–delta interval of 20 msec. When the heart rate was increased by high right atrial pacing, the P–H interval increased with no change in the P–delta so that the His complex was observed to merge into the QRS complex. During right atrial pacing at a heart rate of 90 beats/min the effective refractory period of the Kent bundle was defined as 240 msec.

Figure 1

Electrocardiograms from Patient #1. Standard leads I through V6. Before = preoperative; After = 1 year postoperative.
Recordings from the distal coronary sinus revealed that the interval between left atrial activity and activity from the base of the left ventricle was approximately 30 msec. Left ventricular activity was recorded coincident with the onset of the delta wave on the surface electrocardiogram. During left atrial pacing from the coronary sinus, the degree of pre-excitation on the surface ECG was greater than with right atrial pacing at comparable heart rates. Episodes of supraventricular tachycardia could be induced by premature atrial stimulation, and during these episodes the left atrial activity occurred coincident with the end of the QRS complex and preceded right atrial activity by 90 msec.

On March 8, 1972, the patient was taken to the operating room where the heart was exposed through a left thoracotomy. Mapping the sequence of excitation of the two ventricles was performed. During sinus rhythm with anomalous excitation, the earliest area of ventricular activity occurred coincident with the delta wave. This area of early activity was located on the left ventricle in the region of the A-V groove. The patient was then placed on cardiopulmonary bypass and the left atrium was opened. Both mitral leaflets were seen to be grossly deformed and to balloon into the left atrium during systole. The left atrium was incised at its insertion into the annulus of the mitral valve and this incision was extended approximately 1-½ cm posterior and anterior to the region of early excitation. The atrial wall was then sutured to the annulus, the atrium was closed and the patient was taken off of cardiopulmonary bypass. Epicardial mapping after completion of the surgery showed no evidence of anomalous excitation. Postoperative electrocardiograms revealed a normal P–R interval and no evidence of the Wolff-Parkinson-White abnormality (fig. 1, lower panel). She was discharged from the hospital and during 18 months of follow-up has had no episodes of paroxysmal tachycardia. On March 4, 1973, she returned to the Clinical Research Unit. Electrocardiograms were all normal. She gave no history of paroxysmal tachycardia. A His bundle study was repeated and revealed a P–H interval of 80 msec and an H–V interval of 40 msec. No evidence of pre-excitation was obtained and no episodes of tachycardia were induced.

**Case #2**

N. S. is a 24-year-old tobacco farmer who was referred to Duke Hospital on May 13, 1973. The patient was in excellent health until the age of 15. At that time he noted the onset of episodic palpitation which occurred at a frequency of two to three times per week. These episodes were abrupt in onset, lasted anywhere from five minutes to several hours and stopped abruptly. Usually, the episodes were very short and could be terminated when the patient did his Valsalva maneuver. During these episodes he noted weakness, but no pain or syncope. On the twelfth of May, 1973, he was awakened by a violent episode of palpitation. This episode was different from any previous one and was associated with chest pain, diaphoresis,

![Figure 2](http://circ.ahajournals.org/)

*Figure 2*

Electrocardiograms from patient #2. Standard leads I through V₆. Before = preoperative; After = 3 months postoperative.
and extreme weakness. After several hours of unsuccessful attempts to terminate the episode, he was hospitalized. An electrocardiogram obtained at that hospital revealed atrial fibrillation, anomalous excitation, and a ventricular response which varied between 240-360 beats/min. He was treated with intravenous cedilanid 1.6 mg and within approximately 30 min his rhythm degenerated into ventricular fibrillation which required defibrillation. He was referred to Duke Hospital for further evaluation.

On physical examination the patient looked well. His blood pressure was 110/70 and the pulse rate was 62 and regular. The general aspects of the physical examination were unremarkable. The venous pressure and the jugular venous pulse were normal. The PMI was located 10 cm to the left of the midsternal line in the fifth intercostal space. There were no murmurs, gallops, or clicks. The remainder of the physical examination was entirely normal.

The chest X-ray and fluoroscopic examinations of the heart were both normal. The electrocardiogram revealed Type A Wolff-Parkinson-White abnormality (fig. 2, upper panel). The vectorcardiogram confirmed that the initial forces were directed to the right, inferiorly and slightly anteriorly.

Two days after admission the patient underwent an electrophysiologic study. Recordings from the bundle of His revealed a P-H interval of 90 msec. Even during sinus rhythm at a rate of 65 beats/min the H complex followed the onset of the delta wave on the surface electrocardiogram. With right atrial pacing the P-H interval increased and the H complex merged into the QRS. While pacing the right atrium at a heart rate of 80 beats/min premature atrial depolarizations were induced. All premature beats propagated to the ventricle with WPW-type conduction until a coupling interval of 220 msec was reached at which point the atrium was refractory. Thus, the effective refractory period of the accessory pathway seemed to be limited by atrial refractoriness and was approximately 230 msec. The recording catheter was then advanced into the coronary sinus. Left atrial activity occurred 50 msec before the onset of left ventricular activity. The left ventricular activity recorded with this catheter occurred within the first 10 msec of the delta wave on the surface ECG. During left atrial pacing from the distal coronary sinus, the degree of pre-excitation on the surface electrocardiogram was greater than at comparable heart rates with right atrial pacing. During supraventricular tachycardia with retrograde conduction, left atrial activity occurred at the end of the QRS complex and preceded right atrial activity by 60 msec. Atrial pacing at a cycle length of 240 msec induced spontaneous atrial flutter with a 1:1 ventricular response and with anomalous excitation on all beats. Often, atrial flutter degenerated into atrial fibrillation with a ventricular rate of approximately 280 beats/min. These transient episodes of atrial fibrillation stopped spontaneously in a matter of seconds.

On July 6, 1973, the patient was taken to the operating room and the heart was exposed through a left thoracotomy. During anesthesia, he developed atrial fibrillation which degenerated into ventricular fibrillation and required defibrillation. Following this episode, anomalous excitation disappeared, but with left atrial pacing the pattern was easily restored. During left atrial pacing with anomalous excitation, epicardial mapping was carried out. The mapping studies revealed early excitation of the left ventricle near a large marginal branch of the left circumflex coronary artery (fig. 3 and 4). The patient was then placed on cardiopul-

Figure 3
Lateral and superior view of the heart which depicts the sites of ventricular pre-excitation in patients 1 and 2. Note that the areas of earliest ventricular activation were adjacent to the annulus of the mitral valve and just posterior to a marginal branch of the circumflex coronary artery.
monary bypass and the left atrium was opened. The left atrium was incised and its attachments to the annulus of the mitral ring were divided for a distance of 1-2 cm on both sides of the region of anomalous excitation. The atrial wall was then sutured back to the annulus. After cardiopulmonary bypass, the delta wave on the electrocardiogram was abolished and a repeat epicardial map revealed a normal sequence of ventricular excitation (fig. 4).

The postoperative course was uncomplicated and daily electrocardiograms revealed a normal P-R interval and no evidence of a delta wave. At the time of this report he has returned to his work and has had no arrhythmias during two months of follow-up. His electrocardiogram continues to be normal (fig. 2, lower panel).

Discussion

It seems clear that the most tenable hypothesis for the basis of the Wolff-Parkinson-White abnormality is an anatomic bridge or bridges of accessory tissue connecting the atrial and ventricular chambers. These bridges are termed "accessory" because they lie outside of the normal A-V node and bundle of His and because they offer an additional pathway or pathways over which the electrical impulse may be transmitted from the atrium to the ventricle or from the ventricle to the atrium. In patients with the Wolff-Parkinson-White syndrome accessory muscle bridges have now been described joining the right atrium and right ventricle, the left atrium and left ventricle, and the atrial septum and ventricular septum. The variable location and number of accessory bridges undoubtedly contribute to the complexity which surrounds the analysis of the Wolff-Parkinson-White syndrome but do not detract from the validity and usefulness of the general hypothesis stated above regarding the anatomic basis of the syndrome.

One criticism of this hypothesis is that muscular bridges across the A-V groove have not been discovered on careful anatomic dissection in all cases of Wolff-Parkinson-White syndrome, and alternative anatomic basis have been suggested. A second observation of considerable importance is the possibility that patients who have anomalous excitation over an accessory muscular bridge may also have episodes of tachycardia which do not involve the accessory bridge in a re-entrant circuit.

Successful surgical treatment of patients with the Wolff-Parkinson-White syndrome depends upon the presence of an accessory muscular bridge, a demonstration that it is accessible to surgical division, and finally that the accessory bridge participates in the tachycardia. Elegant electrophysiologic studies by Durrer, Puech, Castellanos, Wellens, Coumel, and Roelandt form the basis for the current studies which allow one to characterize patients with the Wolff-Parkinson-White syndrome, to predict the location of the accessory muscular bridge and its participation in the arrhythmias. The essence of the requirements which must be met to conclude that the WPW syndrome is due to anomalous pre-excitation and that the accessory bridge participates in producing
the tachycardia are the following: First, using catheter recording techniques, it is necessary to demonstrate a His complex and to demonstrate that the delta wave occurs coincident with or before the His bundle electrogram. Second, it is essential to demonstrate, either during antegrade or retrograde conduction, that a gap or "window" exists between the effective refractory periods of the accessory muscular bridge and the normal A-V node. When premature beats are introduced during this window, the pathways from atrium to ventricle or vice versa can be dissociated and appropriately timed premature beats can then initiate either single or repetitive cycles of re-entrant tachycardia. In the usual circumstance these episodes of tachycardia involve antegrade conduction from atrium to ventricle over the normal A-V conducting system and a return path over the accessory bridge. It is usually possible to demonstrate that the return path over the accessory bridge differs from the normal A-V node in the following respects: a) the conduction time from ventricle to atrium is shorter than conduction time over the normal pathway and b) the conduction time over the accessory bridge is usually not modified by cycle length or coupling interval over a range which would be expected to alter conduction through the A-V node. Finally, it is important to determine the site of pre-excitation. The two patients presented in this report with left lateral pre-excitation had Type A electrocardiograms with upright initial forces in lead V1, and also easily discernible Q waves in lead aVL. The findings in these two patients are also consistent with the observation noted by Wellens that with left ventricular pre-excitation the degree of pre-excitation is greater during left atrial pacing than it is during right atrial pacing at comparable heart rates. Presumably, this is a consequence of pacing from a site closer to the accessory bridge. An additional point to be derived from these two cases is that bipolar recordings from the distal coronary sinus revealed ventricular excitation at the onset of the delta wave and in close temporal proximity to the adjacent left atrial activity.

In both of our patients left lateral pre-excitation was anticipated preoperatively, and the patients were explored through a left thoracotomy which provided optimal exposure for mapping of the left ventricle. In both patients the earliest epicardial activity was recorded from the left ventricle just beneath the A-V ring and at a position close to a large marginal branch of the circumflex artery (fig. 3 and 4). In both patients the operation was carried out with direct visualization of the mitral annulus through a left atriotomy. The procedure involved incising the atrium at the site of anomalous excitation and for a distance of 1-2 cm on both sides of the site. The incision was made so that the left atrial wall was disarticulated from the annulus and then sutured back to the annulus. The operation was performed in such a way that any bridge which passed beneath or through a defect in the annulus would have been divided and replaced by subsequent scar tissue.

Subsequent to the operation, both of our patients have demonstrated normal electrocardiograms with a normal P-R interval and no evidence of a delta wave. Both patients have been free of arrhythmias subsequent to the surgical intervention. One of the patients has undergone repeat electrophysiologic studies. During the latter, it was demonstrated that the interval between the His complex and the onset of ventricular excitation was normal and there was no evidence of pre-excitation during either right or left atrial stimulation. The diastolic interval was scanned with premature stimuli and failed to initiate episodes of arrhythmia. Both patients are doing well at the time of this report.

In 1967 Burchell and his colleagues reported temporary ablation of the delta wave in a patient with an atrial septal defect and Type B Wolff-Parkinson-White abnormality. At the time of surgery epicardial mapping revealed anomalous excitation at the lateral margin of the right ventricle and the delta wave was abolished by the injection of procaine into this region. In 1968, Cobb and associates from this medical center reported the first permanent correction of Type B Wolff-Parkinson-White abnormality in a 38-year-old man. Anomalous excitation of the right ventricle was demonstrated at surgery and the delta wave and arrhythmias were abolished by division of the tricuspid annulus at the site of pre-excitation. In subsequent reports in 1970, Sealy and other members of our group reported two additional patients with Type B Wolff-Parkinson-White in whom surgical intervention corrected the electrocardiographic abnormality and abolished arrhythmias. In one of these patients anomalous excitation was found at the lateral margin of the right ventricle and in the other at the inferior margin of the right atrioventricular groove. In 1970 Iwa reported the successful abolishment of Type B Wolff-Parkinson-White in a patient with right lateral pre-excitation and Fountaine and Tajik have each reported similar successful experiences.

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There have been failures to abolish the delta wave by surgical intervention in patients with Type B Wolff-Parkinson-White syndrome. Cole and associates reported two such instances. In one of their patients, who had apparent pre-excitation of the right ventricle, incisions below and above the area of pre-excitation failed to abolish the delta wave. Curiously, however, the patient did not suffer arrhythmias subsequent to the surgical intervention. Unfortunately, their second patient died after a lengthy surgical procedure in which there was intraoperative evidence and subsequent anatomic evidence that the accessory muscular bridge connected the atrial septum to the ventricular septum anterior to the His bundle. Dr. Durrer pointed out that the Type B electrocardiogram might be the consequence of septal pre-excitation. It seems likely that one of the cases reported by Cole was such an instance. Very recently, we had such a case in which the delta wave was abolished by disarticulating the atrial septum from the A-V ring anterior to the His bundle. The postoperative studies on that patient have demonstrated the absence of anomalous pre-excitation.

To our knowledge, this is the first report of successful division of a communication between the left atrium and left ventricle resulting in Type A Wolff-Parkinson-White abnormality. Dreifus, Latour, and Edmond and Coumel have reported four patients with Type A Wolff-Parkinson-White and recurrent episodes of tachycardia in whom the bundle of His was divided in an effort to terminate episodes of tachycardia. In the report by Dreifus this intervention was successful although a permanent pacemaker was required because of heart block and only intermittent conduction over the accessory pathway. In the case by Latour episodes of supraventricular tachycardia have been abolished, but atrial fibrillation with conduction over the accessory pathway continues to present a problem in patient management. While division of the bundle of His is a feasible approach to patients with Wolff-Parkinson-White syndrome and re-entrant tachycardias, division of the accessory muscular bridge seems to us a more desirable approach. The latter does not involve the creation of heart block and, therefore, avoids the necessity for the implantation of a pacemaker. Furthermore, for reasons which are not clear to us, it appears that patients with Type A Wolff-Parkinson-White abnormality have a high incidence of atrial flutter and fibrillation with a rapid ventricular response. When the refractory period of the accessory bridge is short, division of the bundle of His fails to protect such patients against rapid and sometimes catastrophic ventricular response to atrial fibrillation.

In conclusion, this report describes two patients with Type A Wolff-Parkinson-White abnormality in whom surgical division of the left atrioventricular ring abolished the delta wave and arrhythmias. These patients had preoperative evidence of anomalous excitation of the left ventricle. This evidence included an enhanced degree of pre-excitation with left atrial pacing when compared to right atrial pacing, early left ventricular activity coincident with the delta wave recorded from the distal coronary sinus, delta waves which produced negative deflections in lead aVL, and vectorcardiograms which demonstrated initial forces directed to the right, inferiorly and anteriorly. Detailed epicardial mapping at the time of surgery confirmed that the earliest area of epicardial activity was coincident with the onset of the delta wave and occurred adjacent to the lateral aspects of the mitral annulus. The operative intervention divided any potential muscular bridges between the left atrial wall and the left ventricular wall in the region of pre-excitation. Postoperative electrograms show no evidence of anomalous excitation and there have been no episodes of spontaneous or induced arrhythmias during the follow-up.

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