Disabling Supraventricular Tachycardia of Wolff-Parkinson-White Syndrome (Type A) Controlled by Surgical A-V Block and a Demand Pacemaker after Epicardial Mapping Studies

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SUMMARY
A patient with W-P-W syndrome (type A) and disabling supraventricular tachycardia was studied with epicardial mapping prior to surgical interruption of the circus pathway. Analysis of the delta vector and results of the epicardial mapping strongly suggested aberrant pathway located very posteriorly and crossing the atrioventricular sulcus in or adjacent to the interventricular septum. A circus movement responsible for the supraventricular tachycardia was felt to be a mechanism of antegrade A-V conduction and retrograde accessory bundle conduction. Attempts to interrupt the aberrant pathway primarily were unsuccessful, and surgical A-V block was performed. An epicardial demand pacemaker was inserted as a safety feature. Since the operation the patient has been free of supraventricular tachycardia. The postoperative electrocardiograms demonstrated antegrade accessory pathway conduction, but neither antegrade nor retrograde A-V conduction. Failure to interrupt the accessory pathway after incision of the entire right posterior A-V sulcus supported a location with, or to the left of, the atrioventricular septum for the pathway. This case, with the recent results of others, adds to the further understanding of accessory pathway location in W-P-W syndrome (type A).

Additional Indexing Words:
Accessory bundle       Circus pathway

SUPRAVENTRICULAR tachycardia (SVT) associated with Wolff-Parkinson-White syndrome (W-P-W) usually responds to varying combinations of antiarrhythmic and digitalis drugs. The occasional patient with hypotensive or cerebral ischemic symptoms has required DC shock. Iatrogenic myxedema has been sparingly used in resistant cases. Recently, three different surgical technics have been described for patients with disabling SVT accompanying W-P-W. These include (1) the use of a pacemaker with external rate control to stop the SVT, (2) surgical interruption of the accessory A-V pathway, and (3) surgical A-V block followed by use of a pacemaker.

Recent surgical approaches and studies with epicardial mapping have helped to identify the location of the accessory pathway in the various types of the W-P-W syndrome. Epicardial mapping followed by interruption of the accessory pathway has so far been accomplished only in types B and AB of the
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W-P-W syndrome, but has not been reported in type A in the human.

This report describes a patient with W-P-W syndrome (type A) and disabling SVT in whom epicardial mapping demonstrated the posterior location of the accessory pathway. Surgical A-V block and implantation of an epicardial demand pacemaker were performed after attempts to section the accessory pathway failed.

Report of Case

A 38-year-old Caucasian female had a history of SVT since her teens. In the early years the SVT was short-lived and of minor concern. In October 1966, she experienced a 30-min episode of documented VT with a rate of 160/min. Following that attack, her episodes of SVT lasted progressively longer and became more severe. She began to have SVT with the slightest provocation, such as emotional stress, exposure of her body to warm bath water, turning in bed, and swallowing. Her SVT would last from 10 min to 48 hours and would be accompanied by diaphoresis, palor, and weakness.

During the first 6 months of 1967, varying combinations of procainamide (Pronestyl), quinidine, atropine, diphenylhydantoin (Dilantin), and digitalis were tried without benefit. Propranolol was tried but its use had to be discontinued because of symptoms of orthostatic hypotension and fainting.

On January 31, 1968, when the patient was first seen in the Emory University Clinic, she was having SVT intermittently throughout each day. She experienced extreme fatigue as well as occasional substernal pain during the SVT. A family history revealed that her mother had intermittent W-P-W but was asymptomatic. Her twin sister and two brothers also had W-P-W but no SVT. One sister had had episodes of SVT but had never had evidence of W-P-W in the ECG. One maternal aunt, four maternal uncles, and her maternal grandmother had died suddenly.

Cardiac examination was normal except for a loud S1 attributed to a short P-R interval. Posteroanterior and lateral chest roentgenograms were normal. An ECG showed evidence of typical type A W-P-W, the mean QRS vector being markedly superior and anterior (fig. 1).

In June 1968, after the other drugs had failed, methimazole (Tapazole), 40 mg daily, was given without improvement. In September 1968, reserpine and guanethidine (Ismelin) were tried, but symptomatic hypotension prevented their continued use. The patient was admitted to Emory University Hospital where iatrogenic myxedema was produced with 131I without altering the SVT. Thyroid extract was given and she became euthyroid again.

By March 1969 she was in SVT most of the time. During these attacks she was severely limited because of marked dyspnea on exertion and fatigue, and the only successful method of conversion to sinus rhythm was the intravenous use of edrophonium (Tensilon).

Analysis of the ECG suggested a location of the preexcitation and a probable circus mechanism accounting for the SVT. The delta vector was

Figure 1

Standard 12-lead electrocardiogram before surgery.
directed leftward and anteriorly and this suggested a posterior location for an anomalous pathway. The RBBB with left axis deviation (LAD) pattern during normal sinus rhythm indicated early invasion of the posterior fascicle of the left bundle simulating RBBB with left anterior hemiblock. The normal QRS complex during SVT supported an antegrade conduction through the normal A-V node and His bundle. Finally, the retrograde P wave followed the QRS complex during SVT by a time interval (R-P interval) equal to the P-R interval during the W-P-W conduction. This conduction supported a mechanism of retrograde accessory bundle conduction during paroxysmal atrial tachycardia and antegrade accessory conduction during W-P-W. The circus movement, namely retrograde conduction through the accessory bundle and antegrade conduction through the A-V node and His bundle, was, therefore, the most likely mechanism for the tachycardia (fig. 2). It appeared likely that if this circus mechanism could be interrupted at some point in its course, the tachycardia should be prevented.

**Surgical Procedure**

On March 21, 1969, the patient was taken to the operating room and placed on cardiopulmonary bypass. Through a median sternotomy, epicardial mapping was performed by using an exploring bipolar electrode to determine the area of earliest ventricular depolarization. The entire surface of the right ventricle was mapped, but only the most anterior portions of the left ventricle could be checked. As expected, an area of very early depolarization was found posteriorly below the atrioventricular groove adjacent to the interventricular septum. It could not be determined whether the earliest breakthrough lay to the right or left of the septum, since the posterior part of the left ventricular epicardium was not approachable through the median sternal incision. The right coronary artery was dissected free from the atrioventricular groove and the right atrium was opened. A right ventriculotomy was made, beginning anterolaterally beneath and parallel to the atrioventricular groove and extending posteriorly to the ventricular septum by the technic described by Cobb and associates from Duke University.2 The W-P-W pattern persisted. Subendocardial injections of lidocaine were made in an attempt to interrupt an aberrant pathway in the atrial wall or interatrial septum, but this was also unsuccessful. It was felt that the aberrant pathway of earliest ventricular activation either traversed in, or to the left side of, the interventricular septum. In either case, it was surgically inaccessible. A Medtronic epicardial demand pacemaker set at 80 beats/min was sewed in place. Surgical heart block was created with two layers of no. 2-0 black silk sutures through the area of the A-V node and common His bundle which had been extensively cauterized.

**Postoperative Course**

Postoperatively there were no complications, and the pacemaker functioned constantly during her convalescence. In June 1969, her resting ECG showed pacemaker rhythm (fig. 3). An ECG taken after activity showed sinus tachycardia of 110/min and W-P-W conduction to the ventricles (fig. 4). She had returned to work and had experienced no episodes of SVT. In March 1971, 2 years after the operation, she was working and symptom-free without medication except thyroid replacement. Accessory pathway conduction has continued without SVT.

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

*Figure 2*

A 12-lead electrocardiogram before surgery during episode of supraventricular tachycardia.
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Figure 3
Postoperative 12-lead electrocardiogram at rest demonstrating pacemaker control of ventricular depolarization.

Figure 4
Postoperative 12-lead electrocardiogram after activity demonstrating sinus control of ventricular depolarization through the accessory pathway.

Discussion
The first attempt to map the epicardial surface, find an anomalous atrioventricular pathway, and then interrupt the pathway in a patient with W-P-W was reported in 1967 by Burchell and associates. Their patient had type B W-P-W syndrome. The interruption was only temporary, and this result was thought due to a too meager incision. Cobb and associates reported another case of type B
W-P-W in which epicardial mapping pinpointed an anomalous pathway that was interrupted successfully.\textsuperscript{2} Then Dreifus,\textsuperscript{3} and Edmonds,\textsuperscript{4} and their associates described patients with type A W-P-W who were treated successfully with surgical A-V block and pacemakers. Neither group included epicardial mapping or any attempt to interrupt an anomalous pathway in the procedure. Several recent discussions of the evidence for circus movements, anomalous pathways, and the variations of preexcitation have been published.\textsuperscript{8-10} James\textsuperscript{11-13} and Sherf and James\textsuperscript{14} have recently discussed the evidence for what is known and what is unknown about pathways and mechanisms for the SVT of W-P-W. James' thorough reviews point out the inconsistency with which Kent bundles have been found in hearts with known W-P-W syndromes and the occasional occurrence of accessory A-V connections in normal hearts. Lev\textsuperscript{15} and Ferrer\textsuperscript{16} have summarized the combinations of atrial bypass and Mahaim fibers which could potentially give circus movement tachycardias and preexcitation ECGs. Evidence for circus movement producing the SVT in W-P-W has been alluded to in our case. All of the present literature is either anatomic or physiologic, and very few investigators have shown anatomic structures when electrical events have been mapped. One exception is Boineau and Moore\textsuperscript{6} who recently reported on epicardial mapping in a dog with type A W-P-W. An area of early breakthrough was found posteriorly across the right atrioventricular groove, and sections through this area showed fibers consistent with a Kent type anomalous bundle.

Most patients with type B W-P-W who have been studied with epicardial mapping have demonstrated early ventricular activation on the right side some distance from the interventricular septum. The two successful surgical procedures on patients with type B support this probability. It appears from vector analysis, epicardial mapping, and some anatomic evidence, that patients with type A W-P-W have early ventricular activation pathways posteriorly either within or to the right or the left of the interventricular septum. Epicardial mapping in our patient (fig. 5) supports a posterior location for the pathway. This finding could not distinguish between an anomalous pathway on either side or within the ventricular septum. Harris and associates\textsuperscript{17} showed that stimulation of the left atrium posteriorly near the septum would produce a P vector identical to that seen in our patient when in SVT. They also showed that atrial stimulation slightly to the right of the septum could produce the same P vector directed superiorly and to the right.

Preoperatively we presumed that the anomalous pathway in our patient was posterior. The delta and mean QRS vectors producing the pattern of right bundle-branch block with left axis deviation suggested that the anomalous pathway entered the posterior division of the left bundle. The P-wave vector during SVT indicated retrograde activation of the atrium posteriorly near the septum. It was

\textbf{Figure 5}

Sequential ventricular activation from epicardial mapping during surgery. Initial activation begins posteriorly and on the right.
postulated that a circus-type conduction traveled antegrade through the normal A-V tissues and retrograde through the anomalous pathway producing SVT. Evidence for retrograde conductions through the anomalous pathway was the equality of the P-R interval during sinus rhythm and the QRS-P interval (R-P interval) during SVT (figs. 1 and 2). If the anomalous pathway were posterior but to the right of the septum as in the dog described by Boineau and Moore, we hoped to interrupt it surgically. Failure to ablate the anomalous A-V conduction with a generous incision below the right A-V groove supports a more leftward location for the pathway in our case. Postoperative demonstration of preexcitation of the ventricles with absence of A-V nodal conduction lends further support to the concept of circus propagation of the SVT of W-P-W (fig. 3). It seems that interruption of this circuit at either the A-V node or the anomalous pathway is equally effective if complete.

James has outlined a concept in which impulse delay and early activation pathways within the interventricular septum explain the preexcitation syndrome. This “longitudinal dissociation” theory does not require accessory pathways outside the His bundle to explain the variations of the W-P-W syndrome. We feel that our report is strong evidence against such a unified concept. We accomplished generous destruction of the A-V nodal and His bundle areas to the point where no further normal A-V or V-A conduction has been observed or suspected. Accessory pathway activation has been unhampered, however, and type A W-P-W is still evident by electrocardiogram. Finally, we have shown that A-V and His bundle destruction does not necessarily make a patient permanently dependent on a pacemaker; if A-V activation continues by an accessory pathway, a relatively normal life can be enjoyed. We feel the added insurance of a standby pacemaker is mandatory in this type of procedure. It seems clear that no unified theory for preexcitation can explain the location of all accessory A-V communication(s) in the W-P-W syndrome. For the present we would agree with Burchell that “... tentative categorization of patients dependent on the predicted site of individualistic pathway, allowing premature excitation of a portion of one or the other ventricle, seems warranted.”

All the evidence from the surgical cases reported indicates that type B W-P-W is very likely to have an anomalous A-V pathway which lies some distance from the septum and can be surgically divided. It appears that type A W-P-W is less likely to have a pathway which can be severed by right ventricular incisions and may lie variably within or to the left or the right of the interventricular septum. Surgical A-V block will be required in most cases. In the occasional patient in whom symptoms are incapacitating and medical treatment has failed, a surgical approach may be effective. We, like others, stress the rarity with which operations of this type will be necessary. We agree with James that these experimental operations should continue to be undertaken at medical centers where epicardial mapping can be performed and where each operation can add to the growing knowledge about W-P-W.

References


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