Control of Recurrent Tachycardia of Wolff-Parkinson-White Syndrome by Surgical Ligature of the A-V Bundle

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SUMMARY

In a patient with WPW, type A, pharmacological therapy and radioactive iodine failed to control the disabling, life-threatening arrhythmia. Surgical ligation of the A-V bundle was undertaken in view of the rapidly deteriorating clinical course. Failure to identify A-V block after several sutures were placed in the A-V junction and subsequent activation of the ventricles in a heart with known A-V block demonstrates that the accessory A-V bundles may completely activate the ventricles. Complete elimination of the recurrent tachycardias after A-V ligation suggests that the normal A-V transmission system may be a crucial link in the circus pathway of WPW tachycardia.

Additional Indexing Words:
Heart conduction system
Electrocardiography
Heart surgery
Ventricular activation
A-V block
Accessory A-V bundles

THE Wolff-Parkinson-White (WPW) pattern represents an isolated electrocardiographic variation from the normal which does not alter prognosis except in patients with cardiac disease or recurrent supraventricular arrhythmias.1 Several different clinical reports have suggested that from 40 to 80% of the people who have an electrocardiographic WPW pattern experience recurrent paroxysmal atrial tachycardia, atrial fibrillation, or atrial flutter.2,3 Many of the case reports, however, emanate from symptomatic patients who sought medical care for repeated tachycardia. The reported incidence of the WPW pattern, ranging from 0.1 to 3.1 per 1,000 population, suggests that this electrocardiographic pattern occurs frequently in asymptomatic healthy persons.4-7 In a study of 128 Air Force personnel who demonstrated a WPW pattern, only 17 men (13.3%) experienced ectopic rhythms during a follow-up period that ranged from 5 to 28 years.1

In some patients, however, the onset of tachycardia is associated with marked deterioration in cardiovascular hemodynamics.8 When this occurs, neurological signs and sudden death are not uncommon. Pharmacological therapy is frequently inadequate, and precordial electroshock must be used to terminate the rapid mechanism. In several carefully studied cases8-11 muscular bridges known as the bundles of Kent have been demonstrated; in other patients no atrioventricular bridges were found.8 It has also been suggested that many combinations of anomalous pathways, including the fibers of James and Mahaim, could produce electrocardiographic changes indicative of the WPW pattern regardless of the duration of the P-R interval. The possibility of supraventricular...

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Tachycardia exists whether the P-R interval is short, normal, or prolonged. Durrer and Roos have recently reported the pattern of epicardial excitation in a patient with Wolff-Parkinson-White syndrome, type B. Earlier, Burchell had identified pre-excitation in an isolated dog heart. During the closure of an atrial septal defect in a patient with WPW, type B, Burchell and his associates determined activation time by using a unipolar lead to perform an epicardial exploration of the right ventricle below the A-V groove. The injection of procaine, presumably at the site of the Kent bundle, was followed by the disappearance of right ventricular pre-excitation. Burchell and associates also observed that pressure on the right ventricle next to the A-V groove terminated the supraventricular tachycardia on several occasions during the surgical procedure. However, an attempt to interrupt the Kent bundle permanently by a transverse incision in the subendocardium of the right atrium was unsuccessful at that time.

Therapeutic surgical division of the human conduction system to prevent supraventricular tachycardia was first attempted in 1967 by Giannelli and associates. They reported a case in which ligation, followed by severance of the A-V node during aortic and mitral valve replacement, prevented the occurrence of troublesome and life-threatening supraventricular tachycardias. Interruption of the A-V pathway in this case of Wolff-Parkinson-White syndrome prevented the recurrent supraventricular tachy-

![Figure 1](https://circ.ahajournals.org/)

(Upper portion) Twelve-lead electrocardiogram. WPW, type A pattern. P-R interval is 0.12 sec. Delta waves are present in leads II, III, aVF, and V1 through V6. (Lower strip) Lead V1. A supraventricular tachycardia (140/min) was terminated by intravenous administration of 10 mg of edrophonium chloride (Tensilon).
cardia. Several pertinent issues concerning the anatomic-physiological function of anomalous pathways and the requirement of an intact A-V junction for re-entry of the excitation wave and the perpetuation of the tachycardia are discussed in this case report.

Report of Case

A 55-year-old female with a history of recurrent supraventricular tachycardia was seen for the first time in Hahnemann Hospital in April 1966. During the 3 months prior to this admission, she had been hospitalized at Troy Memorial Hospital, Troy, Pennsylvania, on 17 occasions because of rapid heart action. In spite of a prophylactic maintenance dose of quinidine and digitalis, the patient suffered tachycardia with increasing frequency. Several pharmacological programs, including procainamide, diphenylhydantoin, antazoline, and propranolol, alone or in combination, were unsuccessful in controlling or preventing the recurrent arrhythmias.

On examination, the blood pressure was 120/80 mm Hg. Carotid ejection was normal. There were no murmurs. Physiological splitting of the second heart sound was present at the base of the heart.

Results of laboratory studies, including urinary catecholamines, vanilmandelic acid (VMA), and metanephrine, urinalysis, and biochemical profile, were within normal limits. The radioactive iodine \(^{131}I\) uptake at 2 hours was 6.9% and at 24 hours 21.4%, and the liothyronine red cell \(^{131}I\) were administered on May 13, 1966 in an electrocardiogram (fig. 1) showed a classical WPW pattern, type A. Kent fibers are presumably on the left; however, posterior fibers on the right could also give the type A configuration. A temporary termination of the tachycardia, caused by 10 mg of edrophonium chloride (Tensilon) given intravenously, can be seen in the lower strip of V1.

Seventeen millicuries of radioactive iodine \(^{131}I\) were administered on May 13, 1966 in an attempt to inhibit the recurrence of the supraventricular mechanism. The patient was discharged to her home, but tachycardia recurred during the next 4 months and always required precordial shock for control. In February 1967, the patient was readmitted to Hahnemann Hospital. The radioactive iodine uptake was 14.3% after 24 hours, and a second dose of 17 mc of \(^{131}I\) was administered on February 20, 1967. Propranolol, 40 mg, four times daily, quinidine, 400 mg, four times daily, and digoxin, 0.25 mg daily, were ineffectual in preventing recurrent tachycardia. No improvement was discernible after the second dose of \(^{131}I\). In May 1967, the patient was obviously myxedematous with an \(^{131}I\) uptake of 8.4% after 24 hours. The tachycardias became more frequent and required precordial shock two to three times per week for control. At this time, the tachycardias were associated with hypotension and mental confusion.

Surgical ligation of the A-V bundles appeared justified in this case because all pharmacological measures had failed to control recurrent and life-threatening supraventricular tachycardia. The measure was undertaken as an emergency procedure because of the severe hemodynamic penalty associated with the onset of the supraventricular mechanism.

Report of Operation

On May 14, 1967, with the left index finger inserted into the right atrium through a purse-stringed opening in the atrial appendage, sutures were introduced from outside the atrial wall.  

![Figure 2](attachment:image.png)  

**Figure 2**

Sites of surgical ligatures to produce A-V block. The first ligature (arrow 1) was probably sufficient to produce A-V block. However, ventricular activation continued through the bypass. Kent fibers are diagrammatically shown on the right but may be anatomically posterior and may produce pre-excitation of either ventricle. CS = carotid sinus; SVC = superior vena cava.

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LIGATION OF A-V NODE FOR WPW TACHYCARDIA

Figure 3
(Lead I). A-V block produced after several ligatures were placed along the A-V junction. Electrical pacing was required to prevent standstill as the bypass fibers temporarily failed to activate the ventricles.

The needle carrying the suture was guided by the left index finger so that it was passed at a right angle to the A-V ring. The needle was then directed back through the atrial wall. The suture was tied outside the heart to invaginate the right atrial wall down to the region of the A-V node. This ordinarily would cause A-V block by interrupting the common A-V bundle (bundle of His). However, at this point in the operation, it became apparent that electrocardiographic confirmation of A-V block might not be possible because of anomalous atrioventricular conduction. A crucial decision had to be made as to whether the suture was correctly placed, although in our opinion this ligature shown by arrow 1 in figure 2 was probably adequate to produce A-V block.

Open heart surgery was begun immediately using a disc oxygenator with venous return by gravity to the venous reservoir. Oxygenated

Figure 4
(Upper strip) Lead II. Synchronous pacing; beats 1 to 5. Disconnection of pacemaker at right. (Lower strip) Temporary removal of pacemaker. Ventricles activated through bypass fibers.

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blood was introduced through a plastic cannula from the femoral artery; the pump oxygenator was primed with whole blood. As the right A-V junction was exposed, the second suture (fig. 2) was placed to interrupt the A-V bundle. The electrocardiogram failed to reveal A-V block. Several additional sutures (3 to 5, fig. 2) were placed along the A-V junction. Fortunately, transient A-V block ensued (fig. 3). Electrical pacing of the heart was immediately initiated with a synchronous pacemaker (Cordis), which had been positioned subcutaneously in the abdomen with the pacing electrodes on the lateral surface of the left ventricle and the sensing electrodes on the left atrium prior to the ligation (fig. 3). It was thought at this time that the A-V transmission system had been adequately ligated and the surgical procedure was terminated. However, 2 days later, A-V transmission through the anomalous pathway was again noted and has continued to the present time. Synchronous pacing was continued until April 1968, when it was noted that the atrial circuit was functioning intermittently. This allowed sinus rhythm via anomalous atrioventricular conduction to compete with the fixed rate feature of the synchronous pacemaker. Accordingly, the original pacemaker was removed from the abdomen (fig. 4) and replaced with a demand unit (Cordis, Ventricor III) with a refractory period of 0.4 seconds. The patient still receives 60 mg of desiccated thyroid (Proloid) twice daily to control the symptoms of myxedema. In spite of the stormy preoperative clinical course, the patient is presently in good health, and there has been no evidence of tachycardia since the surgical ligation of the A-V transmission system.

**Discussion**

Recently, Ferrer'12 summarized the pertinent concepts relating electrocardiographic patterns of pre-excitation to the possible anatomic pathways. From this discussion it is apparent that the presence of pathways from atria to ventricles can account for various electrocardiographic patterns and different P-R intervals. Lev's17 has accepted the presence of bypass fibers described by James and Mahaim in addition to the fibers of Kent. As early as 1926, DeBoer'18 postulated that a circus movement of the excitatory wave, caused by the presence of an anomalous pathway connecting adjacent parts of the atria and ventricles, was responsible for attacks of supraventricular tachycardia. He argued that refractoriness in the bundle of Kent could permit antegrade conduction only through the normal A-V transmission system. Therefore, normal activation of the ventricles would occur with excitation of the basal part of the ventricles at a time when the Kent bundle was no longer refractory. The atria then could be activated from the atrial end of the Kent bundle (fig. 5C). Hence, a sequence of excitation could be repeated with resulting tachycardia. Carefully reasoned analysis by Holzmann and Scherf,19 as well

![Diagram of Cardiac Pathways](https://example.com/heart_diagram.png)

**Figure 5**

(A) Normal conduction pathways of atria and ventricles. (B) Antegrade Kent conduction with premature ventricular depolarization. (C) Retrograde anomalous atrioventricular conduction resulting in atrial tachycardia. (D) Retrograde A-V bundle (AVB) conduction resulting in atrial (supraventricular) tachycardia and antegrade conduction in Kent. QRS may be wide and bizarre and suggest ventricular tachycardia.
as Wolferth and Wood,²⁰ and direct experimental evidence offered by Butterworth and Poindexter²¹ indicated that this argument was indeed plausible. Later, clinical findings suggesting retrograde conduction via accessory pathways during supraventricular tachycardia were reported by Wolff²² and Harischfeger.²³

Observations by Burchell and associates,¹⁵ as well as Durrer and Roos,¹³ suggested that surgical intervention might be remedial if an aberrant A-V pathway could be identified. Durrer and Roos¹³ indicated that, depending upon the timing of a premature atrial systole and the state of refractoriness of the His and Kent bundles, excitation of the ventricles could occur either predominantly through the A-V nodal system, or predominantly through the Kent bundle, or through one or both conduction systems (fig. 5B). By appropriate stimulation of the atria and ventricles during an attack of supraventricular tachycardia, these authors reported that they could shorten a single cycle length without changing those of the following beats. This finding suggested that a circus movement involving the atria, the normal A-V conduction system, and the Kent bundle was present. If a premature ventricular stimulus was given at a sharply defined delay interval of about 320 msec, the retrograde P wave was followed by supraventricular tachycardia with normal excitation of the ventricles. This sequence of events suggested that the atria were activated in a retrograde way through the Kent bundle (fig. 5C). Durrer and Roos¹³ also suggested that the pre-excitation syndrome could cause a tachycardia compatible with a ventricular mechanism by a circus movement in the reverse direction. Basically, the impulse would enter the normal A-V transmission system in a retrograde fashion to activate the atria and then re-enter the ventricles through the bypass (fig. 5D).

The most direct proof of a circus movement involving both the normal A-V transmission system and the anomalous pathways resulted from studies by Burchell and associates.¹⁵ They reported a case of WPW syndrome, type B, with late activation (0.12 sec) of the epicardium over the outflow tract of the right ventricle during paroxysms of tachycardia. This finding demonstrated that the area was excited much later during the supraventricular tachycardia than when a sinus mechanism was present. Injection of procaine and surgical incision on the inside of the right atrium temporarily interrupted the pre-excitation and gave further support to the viewpoint that the circus pathway associated with the WPW syndrome might be surgically ablated.

Although the surgical treatment in the present case appears to have been an emergency measure, the following arguments were first carefully considered: (1) The tachycardias associated with the WPW syndrome were completely disabling and threatened the life of the patient. (2) All known pharmacological therapy had been tried in a vain attempt to control or prevent the tachycardia. (3) The patient had entered a progressive devolutionary clinical course in the weeks prior to surgery. (4) Therapeutic surgical division of the human conduction system during mitral and aortic valve surgery to prevent recurrent tachycardia had been successful and the procedure was considered feasible.

After several sutures had been placed in the His bundle and along the A-V junction, it became apparent that electrocardiographic evidence of A-V block could never be obtained if all atrial impulses were transmitted to the ventricles via a bypass which appeared to exist between the left atrium and the left ventricle. Fortunately, a transitory period occurred during which the impulse was not transmitted through the accessory pathway and the presence of high-grade A-V block was established. In future procedures of this kind, procaine should be injected into the area of the Kent fibers according to the method of Burchell and associates¹⁵ to prove beyond all doubt that the A-V transmission system has been correctly ligated. The A-V junction is the part of the circus pathway most easily identified, and properly placed.
sutures will consistently and permanently interrupt this pathway as only temporary interruption was achieved even after precise identification and incision of the bypass fibers in the case reported by Burchell and associates.15

The present case further illustrates the function of an intact A-V transmission system as a possible link in the circus pathway for perpetuation of the tachycardias associated with the Wolff-Parkinson-White syndrome. Finally, this case demonstrates that accessory A-V bundles may completely activate the ventricles in the presence of a known A-V bundle block, and their pressure is not merely an anatomic curiosity.

References
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