Surgical cure of the Wolff-Parkinson-White syndrome — a comparison of two techniques

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Abstract **Curative arrhythmia surgery for patients with symptomatic Wolff-Parkinson-White syndrome (WPW) was first performed in South Africa in November 1987. Pre-operatively all patients were symptomatic despite medical therapy, and 32% were assessed as being at risk for sudden death.**

The first 9 patients (November 1987 to December 1989) underwent either epicardial or localised endocardial surgical dissections, and a cure was obtained in 66%. Aberrant atrioventricular conduction recurred in 2 patients, 3° atrioventricular heart block occurred in 2 patients, and there was 1 postoperative death in a patient who had undergone simultaneous coronary artery bypass grafting. In contrast, a standardised endocardial technique was used in the subsequent 10 patients. Surgical cure was obtained in all 10 patients ($P < 0,01$). However, 1 patient required reoperation 24 hours after the first procedure because of early postoperative recurrence due to initial incorrect pathway localisation. This was successful. There were no deaths, and no patient developed atrioventricular heart block.

In view of the excellent surgical results, arrhythmia surgery should be considered in select WPW patients who either have refractory symptoms or are at risk for sudden death. Furthermore, this reliable surgical technique provides an essential back-up should alternative interventional procedures such as percutaneous radiofrequency ablation fail.

S Afr Med J 1994; **84:** 204-207.

The Wolff-Parkinson-White (WPW) syndrome is a congenital heart disorder caused by aberrant strands of muscle, which are remnants of embryonic myocardium that connected the atrium and ventricle.[1] These strands of muscle allow early ventricular activation, which is usually seen on an electrocardiogram as a 'delta wave' (an early deflection preceding the QRS complex) in conjunction with a short PR interval. Patients with WPW syndrome often only present with paroxysmal supraventricular tachycardias later in life and frequently only after the fourth decade. Pharmacological control of WPW paroxysmal tachycardias is not routinely successful and certain drugs, e.g. digoxin and verapamil, can potentiate the tachycardia. Furthermore, poor control of chronic supraventricular tachycardias can result in progressive ventricular dysfunction that may mimic a cardiomyopathy which is, however, reversible if the tachycardia is cured.[2] Catheter or surgical ablation of aberrant atrioventricular pathways is therefore indicated in WPW patients in whom medical therapy fails to control symptoms. Cure by means of surgery or percutaneous catheter ablation has also become an attractive option for young patients faced with a potential lifetime of anti-arrhythmic medication.[3,4]

WPW arrhythmia surgery was first performed in South Africa in November 1987, although the first successful surgical division of a WPW accessory atrioventricular pathway was accomplished by Sealy and associates[5] in 1968 at Duke University Medical Center, USA. Two primary surgical techniques have been developed; an epicardial and an endocardial approach,[3] and modifications (most notably the addition of cryosurgical methods) have increased the surgical success rate to 100% in major centres in the USA,[4,6] with a 0% incidence of peri-operative heartblock. This study was undertaken to assess and compare the results of the initial group of WPW patients treated surgically at Groote Schuur Hospital[7] with those of the patients operated upon after institution of the standardised endocardial technique advised by Cox and Ferguson.[3] In addition, we outline the rationale for future indications for surgery for the WPW syndrome, now that percutaneous catheter ablation is an available therapeutic option.

Patient population and methods

We retrospectively reviewed 19 consecutive non-randomised WPW patients treated surgically at Groote Schuur Hospital during the 5-year period from November 1987. The characteristics of the initial 9 patients operated on between 1987 and 1989 have already been reported,[7] and are compared with those of the 10 patients who underwent surgery between 1990 and 1992 (Table I). All patients were symptomatic despite medical therapy because of poor compliance, inadequate suppression of arrhythmias or intolerable drug side-effects, and 32% were assessed as being at risk for sudden death.

TABLE I.
Characteristics of WPW patients referred for surgical ablation of their aberrant atrioventricular pathways

	1987 - 1989	1990 - 1992
Number of patients	9	10
Age (years)		
Range	6 - 54	19 - 67
Mean	28 ± 5	37 ± 6
Female	56%	50%
Pre-operative characteristics		
At risk of sudden death	2	4
Young age (less than 25 years)	4	3
Atrial fibrillation	3	5
Other abnormalities present		
Valvular heart disease	2	–
Ischaemic heart disease	1	–
Ebstein's anomaly	–	2
Accessory pathways		
Left free wall	3 (+ 1*)	4
Posterior septum	4	3
Right free wall	2	4
Anterior septum	–	1

* Intra-operative electrophysiological mapping suggested a second pathway.

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Accepted 22 Oct 1992.

Two patients in the second group had WPW syndrome associated with Ebstein's anomaly of the tricuspid valve. In addition, 3 patients had been referred for surgery following unsuccessful percutaneous catheter ablation of their atrioventricular pathways; 2 patients had left free wall pathways and the other had both a posterior septal and a right free wall pathway.

Electrophysiological mapping

All patients underwent both pre-operative and intra-operative electrophysiological studies to determine whether the anatomical site of the accessory atrioventricular pathway was the anterior septal, right free wall, posterior septal or left free wall. Two patients had multiple pathways diagnosed pre-operatively, and both were a combination of right free wall plus posterior septal pathways. An association between posterior septal and additional right free wall pathways has previously been described.[8] The anatomical sites of the pathways in our series in decreasing order of frequency were left free wall (8 pathways), posterior septal (7 pathways), right free wall (6 pathways), and anterior septal (1 pathway) (Table I).

In the initial 2-year period (1987 - 1989) intra-operative mapping was done on normothermic cardiopulmonary bypass with a hand-held probe positioned sequentially around the atrial side of the atrioventricular groove, during induced paroxysmal tachycardia or ventricular pacing. Intra-operative mapping suggested the possibility of a previously unsuspected second pathway in 1 patient — a combination of a left free wall plus posterior septal pathway.

In contrast, in the second period (1990 - 1992) intra-operative mapping was done with a band electrode[3] before significant manipulation of the heart and cardiopulmonary bypass. Both atrial and ventricular sides of the atrioventricular groove were mapped routinely, and endocardial mapping around the tricuspid valve was also used in select patients. Intra-operative mapping influenced the surgical approach in 2 patients; a posterior septal pathway was found to be positioned in the left paraseptal region and therefore both left free wall and posterior septal areas were dissected, and the patient with an anterior septal pathway was thought to have an additional posterior septal pathway and both these spaces and the right free wall were therefore dissected. In retrospect, this anterior septal 'parahisian' pathway was possibly similar to the 'intermediate septal pathway' described by Bolling *et al.*[9]

Surgical techniques

The surgical techniques used between 1987 and 1989 varied. Localised epicardial dissections were done in the first 2 patients (Fig. 1). However, these failed to ablate the aberrant pathways and additional localised endocardial dissections were therefore also performed. The surgical techniques used in the remaining patients during this period were localised endocardial dissections at the sites of earliest activation, as determined by intra-operative mapping.

Standardised endocardial dissections as described by Cox *et al.*[3,4] were used in all patients from January 1990. Right free wall, anteroseptal and posteroseptal pathways were approached via a right atriotomy, while left free wall pathways required a left atriotomy. An endocardial incision was made approximately 1 cm from the annulus of the appropriate atrioventricular valve, and the atrioventricular fat pad was then dissected off the ventricular myocardium until the epicardial reflection was seen (Fig. 1). This dissection divides the ventricular end of the accessory pathway. In addition, the dissection was not localised to the mapped site but extended circumferentially around the valve annulus to include the entire anatomical area in which the pathway was located. Both adjacent anatomical areas were dissected if pathways were mapped to a region between two adjacent areas, i.e. paraseptal pathways. Furthermore, routine cyrosurgical methods were used in all patients to 'square off' the endocardial dissections[3,4] and to ablate tissue close to the atrioventricular node. Repeat mapping was routinely performed after discontinuation of cardiopulmonary bypass to confirm successful division of the aberrant WPW pathway.

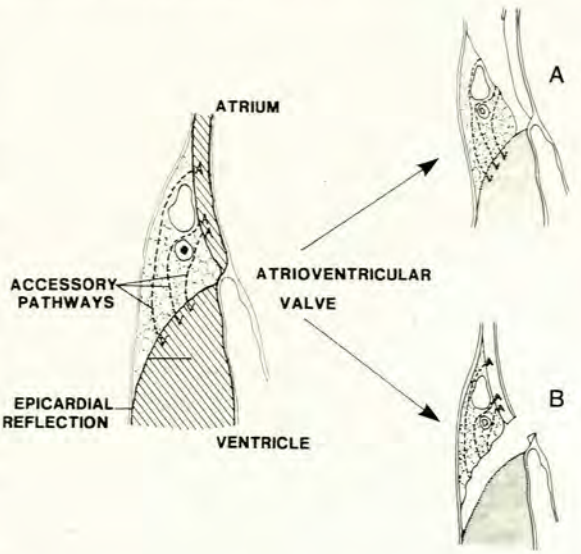

FIG. 1.
Illustration of the surgical techniques used to interrupt anomalous accessory pathways causing the Wolff-Parkinson-White syndrome. These aberrant strands of muscle connect the atrium and ventricle and may be found anywhere in the atrioventricular groove. The epicardial surgical technique (A) divides the atrial insertion of these pathways, whereas the endocardial technique (B) divides the ventricular insertion. (Modified from Cox and Ferguson.[3])

Follow-up

Electrophysiological evaluation was repeated within 1 week of surgery by means of temporary atrial and ventricular epicardial pacing wires inserted at the time of surgery. Late follow-up entailed repeat postoperative electrophysiological testing at 1 - 3 months. However, this was not done in 5 patients in the later period; asymptomatic patients declined and geographical reasons prevented others from undergoing repeat invasive testing. Successful outcome was thus determined either electrophysiologically or by the absence of symptomatic tachycardias during follow-up.

Statistics

Chi-square or Fisher's exact tests were used for comparative purposes; a P value of less then 0,05 was considered significant.

Results

In the earlier period, 1987 - 1989, there was 1 death: a patient who required coronary artery bypass graft surgery as well as division of WPW accessory pathway died unexpectedly on the tenth postoperative day. Complete heart block occurred in 2 patients who had posterior septal dissections, and 2 patients had recurrent

activity of their aberrant atrioventricular pathways (Table II). Nevertheless, 6 patients are now asymptomatic on no anti-arrhythmic medication, and 1 patient is dependent on a dual chamber pacemaker.[7]

TABLE II.
Major complications and results of WPW arrhythmia surgery

	1987 - 1989		1990 - 1992	
	No.	Complications	No.	Complications
Patients	9		10	
Pathways				
Anterior septum	0	—	1	Recurrence (1)*
Right free wall	2	Recurrence (1)	4	None
Posterior septum	4	3° heart block (2) Recurrence (1) Death (1)	3	None†
Left free wall	4	None	4	None
Surgical cure		66%		100%‡

* Recurred 6 hours postoperatively and a previously incorrectly located parahisian pathway was successfully cryo-ablated at immediate reoperation.
† $P < 0,05$ compared with complications associated with posterior septal pathways in earlier time period.
‡ $P < 0,01$ compared with 1987 - 1989 time period.

Following the introduction of standardised endocardial dissections and cryosurgical techniques[3] in 1990, the incidence of surgical complications, recurrence and heart block have diminished. No patient sustained heart block, and only one anterior septal pathway recurred. This single recurrence could be attributed to our failure to recognise the parahisian location of this pathway at the initial intra-operative mapping and not to the surgical technique used. Successful division of this pathway was obtained at immediate reoperation by means of cryosurgical ablation of the tissue between the previous dissection and the atrioventricular node. The cure rate for surgically treated WPW patients has therefore increased from 66% to 100% ($P < 0,01$).

In addition, the high incidence of complications associated with posterior septal dissections in the earlier group of patients has decreased to 0% ($P < 0,05$).

The association between WPW syndrome and Ebstein's anomaly of the tricuspid valve has previously been described.[1,10] Two patients in the later series underwent concomitant repair of Ebstein's anomaly of the tricuspid valve, according to the technique described by Danielson and Fuster.[11] This consisted of horizontal plication of the free wall of the atrialised portion of the right ventricle and posterior tricuspid annuloplasty. However, both patients had residual moderate tricuspid regurgitation postoperatively.

Postoperative complications

Additional postoperative complications noted in this series of patients included 2 cases of postcardiotomy syndrome (Dressler's syndrome) (11%). The postcardiotomy syndrome was noted in 6,7% of patients reported by Cox *et al.*,[4] and was associated predominantly with left free wall pathway dissections. However, no such association was noted in our patients.

Postoperative palpitations or an awareness of an irregular heart beat without recurrence of pre-excitation was noted in 22% of our patients, as in other series.[12] Paroxysmal atrial fibrillation was documented in only 2 patients postoperatively (11%); however, this incidence was lower than that of preoperative atrial fibrillation (44%; $P < 0,05$). Postoperative atrial fibrillation was easily controlled pharmacologically in 1 patient, but was incapacitating and necessitated late percutaneous atrioventricular node ablation with insertion of a rate-adaptive ventricular pacemaker in the other.

Discussion

The primary indication for arrhythmia surgery in WPW patients is failure of medical therapy. However, the risk of sudden death in symptomatic WPW patients is significant; 9% of WPW patients in the series of Iwa *et al.*[13] who did not undergo surgery either because of mild symptoms or refusal to do so, died suddenly at home, and 15% of WPW patients referred for surgery in the series of Cox *et al.*[4] had previously been successfully resuscitated from an episode of 'sudden death'.[4] The 32% prevalence of patients at risk for sudden death in our series was similar to that in the series of Sealy *et al.*,[1] but lower than the figure of 50% reported by Iwa *et al.*[13] WPW patients at risk of sudden death include those with a history of malignant tachycardias, or those in whom electrophysiological studies demonstrate a rapid 1:1 ventricular response with induced atrial fibrillation as a result of a short accessory pathway refractory period. All WPW patients should therefore be assessed for their 'risk of sudden death', and patients at risk should be referred for ablation of their aberrant atrioventricular pathways.

The governing principle for the surgical techniques developed to interrupt WPW accessory pathways is that the accessory pathway must connect the atrium (somewhere between the valve annulus and the epicardial reflection off the atrium) to the ventricle (somewhere between the valve annulus and the epicardial reflection off the ventricle). Division of these anomalous pathways in the atrioventricular groove anywhere between the atrium and ventricle will result in cure (Fig. 1).The epicardial approach divides the atrial end of the accessory pathway with a dissection between the atrioventricular groove fat pad and the atrium.[6,14] This epicardial approach was not successful in the 2 patients in whom it was attempted in our early experience. The reported initial failure rate necessitating reoperation for the epicardial approach remains at 4 - 5% even in major centres experienced with this technique,[6,14] possibly because the plane between the atrioventricular fat pad and atrial wall is not easily defined. In contrast, with experience the endocardial approach[3] is now associated with reoperation rates approaching 0%.[15]

The endocardial approach is performed via a supra-annular endocardial incision and involves dissection in the easily defined plane between the atrioventricular groove fat pad and the ventricular myocardium,[3,4] thereby dividing the ventricular end of the accessory pathway. Three additional principles were recommended by Cox and Ferguson;[3] accessory atrioventricular pathways are often multiple, may be broad pathways and may occur adjacent to the atrioventricular valve annulus. Therefore, the entire anatomical space in which the pathway occurred and both adjacent spaces in the case of paraseptal pathway locations were dissected, and the endocardial incision was 'squared off' by cryosurgical means to ensure interruption of para-annular pathways.[4] Cryosurgery was used because it is the only known means of creating either 'reversible' or permanent heart block depending upon the duration of the applied cryolesion.[3] Parahisian pathways can thus be ablated safely without the risk of creating permanent heart block. The improved surgical results of our later series — a surgical cure rate of 100% with an incidence of heart block of 0% — supports the routine use of the principles outlined by Cox *et al.*[3,4]

WPW patients with Ebstein's anomaly of the tricuspid valve have a high incidence of coexisting multiple right free wall as well as posterior septal pathways, and characteristically these pathways lead into the atrialised portion of the right ventricle.[10,16] Therefore, both these areas were dissected routinely in patients with Ebstein's anomaly, regardless of whether only one pathway had

been mapped. However, the technique used to repair the tricuspid valve is controversial — either the technique advised by Danielson and Fuster[11] or that of Carpentier *et al.*[17] may be used. The former technique was used in the 2 patients in this series with limited success.

Coexistent paroxysmal atrial fibrillation was noted pre-operatively in 44% of our patients, a finding similar to that in the series reported by Sharma *et al.*[18] Atrial fibrillation results in a rapid ventricular response if the accessory pathway has a short refractory period, and these patients may then develop ventricular fibrillation.[19] However, episodes of postoperative atrial flutter or fibrillation were noted in only 2 of our patients (11%). Therefore, our findings support the concept that surgical ablation of WPW accessory pathways assists in the prevention of further episodes of atrial fibrillation.[12,18]

WPW syndrome patients in whom surgical division of their aberrant atrioventricular pathways is indicated now have an alternative therapeutic option available — percutaneous catheter ablation.[20] Catheter ablation should, however, be by means of radiofrequency shocks and not direct-current shocks which are associated with increased complications.[21] Nevertheless, catheter ablation is associated with a failure rate of 10 - 26%[9,22] despite up to one-third of patients undergoing more than one catheter ablation session;[23] 3 of our surgically treated patients suffered failed radiofrequency catheter ablation. In addition, major complications have been reported in 3,5 - 20% of patients undergoing catheter ablation:[9,22,23] heart block, occlusion of coronary arteries, valve perforation, cardiac tamponade and cerebrovascular accidents. Multiple radiofrequency lesions may also make subsequent surgical intervention more difficult and potentially dangerous (personal communication — J. Cox), and we would therefore caution against the administration of prolonged multiple shocks before referral for surgical cure. Nonetheless, good results are being obtained with catheter ablation techniques as experience is gained, and percutaneous radiofrequency ablation is now the interventional procedure of choice in most patients. Catheterisation with multiple large catheters is not, however, feasible in infants and small children, in whom surgical ablation can safely be accomplished.[24] Furthermore, the distorted atrioventricular anatomy in Ebstein's anomaly may make successful catheter ablation in these patients difficult and potentially hazardous. The incidence of failure of catheter ablation is also higher if the pathway is located in either a paranodal site (33 - 50% failure rate)[9,22] or in the right free wall (75% failure rate).[22] Arrhythmia surgery will still have a definite role in the treatment of patients with WPW, both as a reliable back-up for failed radiofrequency ablation and as the primary intervention in patients in whom the percutaneous approach is not suitable.

The excellent results now obtainable with modern surgical techniques for division of WPW accessory pathways — 100% cure and an incidence of heart block of 0% — are the standard against which emerging alternatives such as catheter ablation must be measured.

The expert assistance provided by Professor Ozinsky and his colleagues in the Department of Anaesthesiology is much appreciated. The financial assistance provided by the Harry Crossley Foundation that enabled one of the authors (U.V.O.) to obtain updated training in arrhythmia surgery in 1990 from Professor J. Cox, Washington University School of Medicine, St Louis, USA, is also gratefully acknowledged.

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