Surgery for the Wolff-Parkinson-White syndrome

The Groote Schuur Hospital experience

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Summary

Surgical division of accessory atrioventricular (AV) connections has been performed on 9 patients with the Wolff-Parkinson-White (WPW) syndrome at Groote Schuur Hospital. All patients had symptomatic paroxysmal tachycardia. The indication for surgery in 5 patients was poor control on anti-arrhythmic drugs. Surgery was performed on a 15-year-old boy to prevent lifelong dependence on drugs, although his atrial fibrillation (ventricular rate > 300/min) was controllable with sotalol 1 280 mg daily. The remaining 3 patients required cardiac surgery for other indications and therefore their accessory pathways (APs) were divided concurrently. The AP was localised by pre-operative endocardial mapping and intra-operative epicardial mapping. There were 4 posteroseptal, 3 left free-wall and 2 right free-wall pathways. An endocardial approach was used to divide the pathways.

All 5 free-wall APs were successfully divided without complications or recurrence. However, 1 patient with paroxysmal atrial fibrillation and severe unstable angina due to coronary artery disease died unexpectedly 10 days after 4-vessel coronary bypass grafting and division of a posteroseptal AP. Postoperative complications occurred in a further 2 patients with posteroseptal APs. One patient developed complete heart block and is now asymptomatic with a DDD pacemaker, while the other had recurrence of retrograde bypass conduction postoperatively, but is now successfully controlled on sotalol. Therefore 7 of the 8 survivors are free of recurrence of tachycardia on no anti-arrhythmic drugs after a mean follow-up of 14,3 months. New insights into the surgical technique, particularly for division of posteroseptal pathways, can be expected to improve the outlook.

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Surgical division of the accessory atrioventricular (AV) connections in patients with the Wolff-Parkinson-White (WPW) syndrome was pioneered by Sealy and his colleagues from Duke University in North Carolina and reported in 1968.[1] That institution has remained in the forefront of surgical treatment of this condition, but such surgery is now widely practised throughout the world. Before 1981, the failure rate of WPW surgery was 14 - 30%.[2] However, with experience and modified techniques, the success rate has now increased to 100% in some centres.[3]

The WPW syndrome is an uncommon condition (1 - 3 cases/1 000 population) and surgery has been undertaken mainly in those patients with severe symptoms, inadequate response to drug treatment or potentially life-threatening arrhythmias. It may also be considered in a young patient in order to avoid lifelong dependence on anti-arrhythmic drugs. This is becoming an increasingly common indication for surgery in view of the excellent results now obtained in major centres.[3,4] However, appropriate surgical experience is essential and, as the number of such cases requiring surgery is limited, surgery should only be offered at a few major centres. This article reports on the first 9 such patients operated on at Groote Schuur Hospital, and outlines the rationale for such treatment.

The WPW syndrome

The association of paroxysmal tachycardias in otherwise healthy young people with the characteristic ECG pattern of a short P-R interval and a wide QRS complex with slurred upstroke (delta wave) (Fig. 1) was described in 1930 by the authors whose names are now indelibly attached to this syndrome.[5] It has fascinated electrophysiologists over the years because it is the best understood clinical example of the phenomenon of re-entry. An abnormal bridge of muscular tissue crossing the AV groove, capable of conducting impulses more rapidly than the AV node, causes early activation of the ventricle and accounts for the delta wave. The wide QRS complex is due to fusion of the impulse activating the ventricle via the accessory pathway (AP) with the normal impulse arriving later via the AV node.

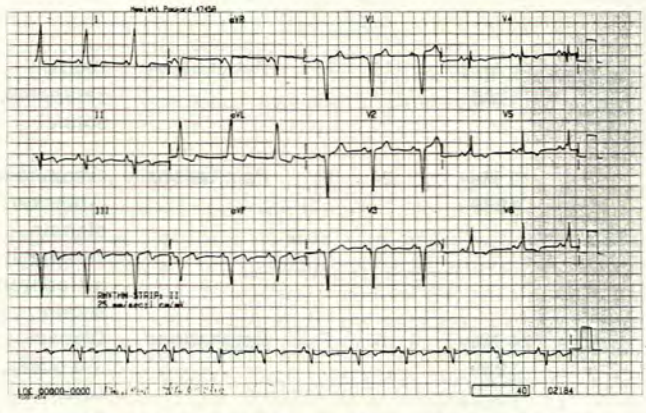

Fig. 1. ECG of patient 5 taken during sinus rhythm demonstrating the typical features of the WPW pattern, namely a short P-R interval (0,12 s) and a widened (0,16 s) QRS complex. The widening is due to the slurred initial upstroke (delta wave) resulting from fusion of the atrial impulse conducted via the AP that depolarises part of the ventricle prematurely, with that of the normal impulse conducted via the AV node. The negative delta waves in the inferior leads give the false impression of inferior myocardial infarction.

Under certain conditions an impulse may fail to conduct via the AP and the impulse conducted via the AV node may then enter the AP retrogradely from below, thus re-exciting the

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atrium and a circus movement tachycardia results. Conduction via the AP is usually in the retrograde direction during the tachycardia, hence activation of the ventricle will be normal and the delta wave is therefore not visible *during the tachycardia*. These tachycardias are characteristically sudden in onset, may be very rapid and may stop abruptly, either spontaneously or as a result of vagal stimulation that interrupts the impulse in the AV node. If the tachycardia persists it will be necessary to terminate it either by synchronised DC cardioversion or administration of an anti-arrhythmic agent.

The other major and potentially more serious complication of the WPW syndrome is the occurrence of atrial fibrillation (AF). This occurs in about 30% of symptomatic patients even in the absence of other heart disease.[6] The reason for this is poorly understood. However, in the presence of AF the ventricular rate may be extremely rapid because the protective filter of the AV node is bypassed, and a high proportion of the impulses may be rapidly conducted via the AP to the ventricle (Fig. 2). In certain instances this may result in ventricular fibrillation. Although the incidence of sudden death in this syndrome appears to be low, it it more likely if the refractory period of the AP is short and if the shortest R-R interval during atrial fibrillation is less than 205 ms.[7] Such patients may be identified by means of electrophysiological studies

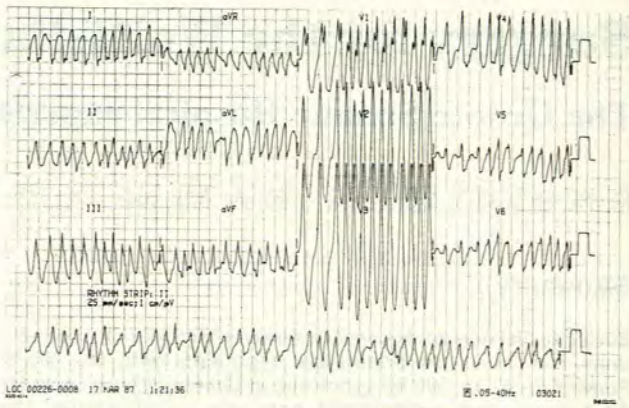

Fig. 2. The ECG at initial presentation of patient 4 showing the extremely rapid (300/min), irregular, bizarre wide QRS complex tachycardia. This was due to AF conducting predominantly via the AP. The shortest R-R interval was 140 ms.

(EPS), which consist in recording intracardiac ECG signals via electrode catheters and studying the effects of programmed stimulation delivered by specially constructed external pace-

TABLE I. PATIENT CHARACTERISTICS

Patient	Date of surgery	Age (yrs)	Sex	Symptoms	Previous drug therapy	Arrhythmia	Other disease	Indications for surgery
1	17 Nov. 1987	32	M	Palpitations, chest pain, dyspnoea	Disopyramide	AVRT	Mixed aortic valve	Aortic valve replacement
2	18 Nov. 1987	46	M	Palpitations, syncope	Disopyramide, amiodarone, sotalol	AVRT AF	None	AF with rapid ventricular rate (260/min); poor response and/or intolerance to drugs
3	4 Oct. 1988	10	F	Palpitations, dyspnoea	None	AVRT	Primum ASD repair 1982; severe mitral regurgitation	Mitral valve replacement
4	13 April 1988	15	M	Palpitations	Digoxin, verapamil, sotalol	AF AVRT	None	Risk of ventricular fibrillation; to avoid dependence on anti-arrhythmic drugs
5	12 May 1989	24	F	Palpitations	Disopyramide, β-blocker, digoxin, verapamil, amiodarone, sotalol	AVRT	None	Poor control with drugs; lack of availability of drugs; to avoid drugs during future pregnancies
6*	24 May 1989	6	F	Palpitations	Digoxin, β-blocker, sotalol	AVRT	None	Poor control with drugs
7*	4 Aug. 1989	30	F	Palpitations, chest pain	Digoxin, verapamil, amiodarone	AVRT	None	Poor control with drugs
8	23 Aug. 1989	54	M	Angina, dizziness	Sotalol, disopyramide, disopyramide + verapamil	AF	IHD	Coronary bypass graft for severe angina
9	19 Oct. 1989	37	F	Palpitations, dizziness	Digoxin, β-blocker, verapamil, amiodarone	AVRT	None	Poor control with drugs

* Concealed accessory pathway; IHD = ischaemic heart disease; ASD = atrial septal defeat.

makers.[6] Such studies are used to characterise the AP and to assess the response to treatment. If surgery is considered, EPS is essential in order to map the location of the bypass tract. In addition, if surgical division of the pathway is undertaken it is localised intra-operatively by electrical mapping as the pathways are invisible to the surgeon. A high degree of co-operation between the cardiac surgeon and the electrophysiologist is therefore essential.

Patients and methods

The characteristics of the 9 patients are listed in Table I. There were 4 male subjects and 5 female subjects, ranging in age from 6 years to 54 years (mean 28,2 years). All had symptoms of paroxysmal tachycardias. Delta waves were present in 7 patients, whereas in 2 (patients 6 and 7), the APs were concealed (no delta waves), but conducted retrogradely during paroxysmal tachycardia.

Five patients remained symptomatic despite 3 - 6 drug trials (mean 3,8) including amiodarone in 4 and sotalol in the other. Three required cardiac surgery for other reasons (valve replacement in 2- and 4-vessel coronary bypass grafting in 1), and so the opportunity was used to divide the APs at the same time. Finally, patient 4, a 15-year-old boy, presented in atrial fibrillation with an extremely rapid ventricular response (over 300/min) and a shortest pre-excited R-R interval of 140 ms (Fig. 2). It was possible to prevent induction of atrioventricular re-entry tachycardia (AVRT) and rapid AF with large doses of sotalol (1 280 mg daily). However, we recommended surgery in his case to avoid a possible lifetime risk of sudden death and dependence on anti-arrhythmic drugs. The location of the AP was posteroseptal in 4 patients, left free wall in 3, and right free wall in 2 (Fig. 3).

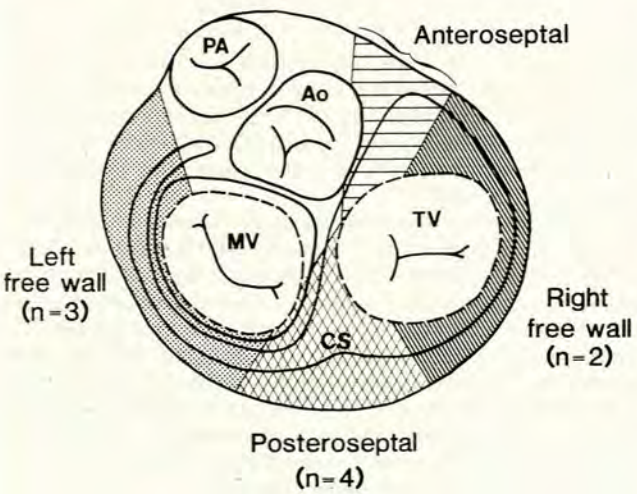

Fig. 3. Diagrammatic representation of a transverse section of the heart above the AV valves, to show the four main areas in which APs occur. The numbers refer to the distribution of APs in our patients (PA = pulmonary artery; AO = aorta; MV = mitral valve; CS = coronary sinus; TV = tricuspid valve).

Electrophysiological testing was done using standard techniques.[8] Briefly, multiple electrode catheters were inserted via the femoral and subclavian veins and positioned in the heart to record electrograms from the His bundle, right ventricle, right atrium and left atrium via the coronary sinus. Programmed electrical stimulation was used to determine refractory periods and to induce reciprocating tachycardia and AF.

The location of the accessory pathway was mapped at the time of EPS via the electrode catheters by recording local retrograde atrial activation times at various positions in the coronary sinus and around the tricuspid valve ring during induced AVRT or right ventricular pacing. Biplane cine films were taken to assist in localisation of the electrodes at each recording site in an attempt to correlate the activation times as accurately as possible with the cardiac anatomy.

At operation, mapping was repeated on cardiopulmonary bypass while at normothermia. A hand-held probe was positioned sequentially around the atrial side of the AV groove during AVRT or right ventricular pacing. The site where the earliest atrial activation was recorded was taken to be the site of insertion of the AP. After analysing the traces, the surgeon proceeded with the dissection of the defined area. An endocardial surgical approach[4] was used with the heart arrested with cold crystalloid cardioplegia. Valve replacement and coronary bypass grafting was done before AP dissection in the 3 patients undergoing these procedures. On completion of the surgical procedure, after the heart was re-warmed and once beating at normothermia, mapping was repeated to confirm successful interruption of the accessory pathway.

A limited EPS was done within 1 week of the operation using temporary atrial and ventricular epicardial pacing wires (inserted at the time of surgery). A complete EPS using electrode catheters was repeated 1 - 3 months postoperatively in all except the 1 patient who died and the patient who remained in complete heart block.

Results (Table II)

Immediate

In one patient with a concealed free-wall AP (patient 7) there was no evidence of accessory pathway function intra-operatively. The surgical dissection was therefore carried out on the basis of the pre-operative endocardial mapping. There has been no evidence of return of AP conduction and she has been free of tachycardia for 8 months on no therapy, having had frequent episodes before surgery despite anti-arrhythmic drugs. In the other 8 patients, the AP was initially mapped intra-operatively and no AP conduction could be demonstrated after surgical division of these sites. However, AP conduction returned within 24 hours with recurrence of tachycardia in 1 patient (No. 6) who had a concealed posteroseptal pathway.

Complications

Complete heart block was initially present at the end of the operation in 3 of the 4 patients with posteroseptal pathways. However, it recovered spontaneously in 1 patient (No. 5), and normal AV nodal refractoriness and conduction during rapid atrial pacing was found at follow-up EPS. Patient 8 died unexpectedly on the 10th postoperative day from a probable myocardial infarction, after saphenous vein grafting of the coronary arteries and division of a posteroseptal AP. Post-operatively, this patient was paced satisfactorily because of atrial fibrillation with complete heart block and a ventricular escape rhythm of 70/min. Patient 2 remained in complete heart block with intermittent recovery of AV conduction (without delta waves) and a permanent DDD pacemaker was implanted. Patient 3, in whom a right-sided AP was divided in addition to a mitral valve replacement, required repeat thoracotomy for bleeding. Her postoperative recovery was slow because of right ventricular dysfunction and tricuspid regurgitation, but she subsequently did well.

These early results are similar to the first reported results from major centres[2] where, having developed surgical expertise, a success rate of 100% is now reported.[4]

TABLE II. RESULTS

Patient	Site of AP	Surgery	Complications	AP function: Early	AP function: Late	Arrhythmia	Symptoms	Treatment	Follow-up
1	R free wall	Aortic valve replacement, AP dissection	None	None	Retrograde only	None	None	Warfarin	Well at 30 mo.
2	Posteroseptal	AP dissection	CHB	None	None	CHB, intermittent AF	None	DDD pacemaker	Well at 25 mo.
3	R free wall	Mitral valve replacement; AP dissection	Postoperative haemorrhage	None	None	None	None	Warfarin, diuretic	Well at 1 mo., lives in Botswana and has not returned for follow-up
4	L free wall	AP dissection	None	None	None	None	None	None	Well at 24 mo.
5	Posteroseptal	AP dissection	Transient CHB	None	None	None	None	None	Well at 23 mo.
6	Posteroseptal	AP dissection	None	Present	Present	AVRT	Palpitations	Sotalol	Well at 13 mo.
7	L free wall	AP dissection	None	None	None	None	None	None	Well at 8 mo.
8	Posteroseptal	Coronary artery bypass graft, AP dissection	CHB, died 10th post-operative day; myocardial infarction	None	—	CHB		Temporary pacemaker	Died 10th day
9	L free wall	AP dissection	None	None	None	None	None	None	Well at 3 mo.

CHB = complete heart block.

Late

The 8 survivors have been followed up for 1 - 30 months (mean 14,3 months). Patient 3 lives in Botswana and has not yet returned for follow-up.

Five patients have been free of arrhythmias on no anti-arrhythmic drugs. Patient 2 has a permanent DDD pacemaker and has had several episodes of atrial fibrillation (his predominant pre-operative arrhythmia) in the first few months after surgery. No conduction to the ventricles occurred but the pacemaker rate increased because of sensed atrial activity. He is therefore on a small dose of β-blockers. However, his quality of life is markedly improved compared to the frequent pre-operative palpitations and syncope, which did not respond to drugs.

Recurrence of AVRT has occurred in 1 patient (No. 6) as a result of the return of retrograde conduction through her concealed, posteroseptal AP. However, the arrhythmias are now easily controlled on sotalol.

Discussion

This small series is the first report of surgical treatment of the WPW syndrome in South Africa. The results are encouraging, particularly in the patients with free-wall APs, since no major complications or recurrences occurred in this group.

We encountered an unusually high proportion of patients with posteroseptal APs. Successful division of APs in this location is more difficult and requires extensive dissection in the triangle of Koch, adjacent to the AV node, and carries a higher risk of heart block.[2] Experience and new insights into the surgical techniques should help to improve these results.

The decision to divide the AP surgically in a patient with the WPW syndrome depends on a number of factors:

1. Severe symptoms not adequately controlled by carefully chosen drug therapy.

The choice of anti-arrhythmic agents should be guided by the functional characteristics of the bypass tract and the ability of the chosen agent to suppress inducible tachycardias. If no available drug adequately suppresses symptomatic tachycardias or is not tolerated because of side-effects, surgery should be seriously considered. Five of our 9 patients fell into this category. In these cases, surgery offers relief of symptoms that medical treatment cannot. In severe cases, such as patient 2, even long-term dependence on a pacemaker is preferable to the incapacitating symptoms of the WPW syndrome.

2. Potentially life-threatening arrhythmias.

Patient 4 presented with AF (Fig. 2) accompanied by an extremely rapid ventricular response. The shortest R-R interval of 140 ms indicates a significant risk of the arrhythmia degenerating into ventricular fibrillation.[7] Although we were able to demonstrate that large doses of sotalol could alter the AP refractoriness sufficiently to render this much less likely,[9] we felt that surgery offered a better long-term alternative by eliminating the risk and removing the need for long-term drug treatment in an otherwise healthy young boy.

3. Life-long dependence on anti-arrhythmic drugs.

Successful surgery will eliminate the need for anti-arrhythmic drugs and is likely to be cost-efficient, particularly in young people with otherwise normal hearts. It should be considered particularly in young women before they have

children. Patient 5 illustrates this indication as, in addition to poor control with the drugs that had been tried, she lives in a neighbouring country where many such agents are not available. She is also keen to have children and would prefer not to be on any drugs during pregnancy.

4. As an adjunct to other necessary open heart surgery.

Although patients 1 and 3 could have been treated medically for their arrhythmias, both had easily accessible APs that could be divided with little additional risk at the time of valve replacement. Patient 8 had severe coronary artery disease with disabling angina, which was aggravated by very rapid heart rates during AF. His postoperative death was myocardial in nature rather than arrhythmic, and it is unlikely that the additional cardiopulmonary bypass and ischaemic cross-clamp times required for the AP dissection contributed to his death, since he had been recovering well before his unexpected death on the 10th postoperative day.

5. The availability of the necessary equipment and expertise.

Surgery for arrhythmias can only be undertaken in a unit staffed by personnel with experience in electrophysiology and equipped to do detailed endocardial mapping.[10] This is essential, since the decision to operate will be influenced by the characteristics of the pathway, its location and the possibility of more than one AP that may be present in up to 20% of patients.[11] In addition to the necessary surgical skills, the surgeon must also be familiar with the electrophysiological techniques involved and be prepared to undertake the detailed intra-operative mapping necessary to locate the AP and to confirm that it has been successfully divided. This requires a great deal of co-operation between surgeon, electrophysiologist and technologist.

Excellent results are being obtained in units doing large numbers of procedures[12] but are also satisfactory in those doing smaller numbers,[13] although there clearly is a learning curve.[7] It has recently become clear, since one of us (U.V.O.) visited Dr James Cox's unit in St Louis, Mo., USA, that there have been major advances both in intra-operative mapping and in surgical technique in the last few years, which can be expected to markedly improve the results of surgery for the WPW syndrome. These include computerised mapping that allows for instantaneous analysis of a single beat (thus improving intra-operative mapping), dissection of the entire anatomical space in which the AP is located (because of the high incidence of multiple pathways), and the use of cryosurgery.[14]

A start has been made in offering WPW surgery in South Africa. In the future, with the application of these new techniques, we hope to emulate the outstanding results obtained in leading centres.

Inevitably, when reports of techniques such as this are presented at this stage of the evolution of health care resources in this country, questions of cost-benefit will be raised. All patients with symptomatic WPW syndrome should undergo EPS to assess risk and the response to anti-arrhythmic drugs. Those young patients that require lifelong drug treatment with all the risks of side-effects of these expensive, relatively toxic agents, should be offered surgery. The benefits to the health system are considerable, since curative surgery will avoid the costs of many (perhaps 30) years of anti-arrhythmic therapy, multiple re-admissions to hospital for the treatment of breakthrough arrhythmias and drug failures. In addition, the risks of sudden death, estimated to be as high as 15% by J. Cox *et al.* (personal communication), are avoided. However, if we are to obtain results similar to those reported internationally, much of our custom-built equipment will need to be replaced by commercially available computerised equipment to facilitate pre- and intra-operative mapping.

Mr L. W. Piller provided invaluable expert technical assistance, including the manufacture of the probes used for intra-operative mapping. We thank the Medical Superintendent of Groote Schuur Hospital for permission to publish.

Addendum

Since submission of this manuscript, a further 5 APs have been successfully divided without complications in 4 patients (1 right free wall and posteroseptal with Ebstein's anomaly, 1 anteroseptal, 1 right free wall and 1 left free wall).

REFERENCES

1. Cobb FR, Blumenschein SD, Sealy WC *et al.* Successful surgical interruption of the bundle of Kent in a patient with Wolff-Parkinson-White syndrome. *Circulation* 1968; **38**: 1018-1029.
2. Gallagher JJ, Sealy WC, Cox JL *et al.* Results of surgery for pre-excitation caused by accessory atrioventricular pathways in 267 consecutive cases. In: Josephson ME, Wellens HJJ, eds. *Tachycardias: Mechanism, Diagnosis and Treatment*. Philadelphia: Lea & Febiger, 1984: 259-269.
3. Gallagher JJ, Sealy WC, Cox JL, Kasell J. Results of surgery for pre-excitation in 200 cases (Abstract). *Circulation* 1981; **64**: suppl. IV, 146.
4. Cox JL, Gallagher JJ, Cain ME. Experience with 118 consecutive patients undergoing operation for the Wolff-Parkinson-White syndrome. *J Thorac Cardiovasc Surg* 1985; **90**: 490-501.
5. Wolff L, Parkinson J, White PD. Bundle branch block with short P-R interval in healthy young people prone to paroxysmal tachycardia. *Am Heart J* 1930; **5**: 685-704.
6. Campbell RWF, Smith RA, Gallagher JJ, Pritchett ELC, Wallace AG. Atrial fibrillation in the pre-excitation syndrome. *Am J Cardiol* 1977; **40**: 514-520.
7. Gallagher JJ, Pritchett ELC, Sealy WC, Kasell J, Wallace AG. The pre-excitation syndromes. *Prog Cardiovasc Dis* 1978; **20**: 285-327.
8. Ward DE, Camm AJ. *Clinical Electrophysiology of the Heart*. London: Edward Arnold, 1987.
9. Scott Millar RN. Clinical and electrophysiological effects of sotalol in Wolff-Parkinson-White syndrome. In: Belhassen B, Feldman S, Copperman Y, eds. Tel Aviv, Israel: *Cardiac Pacing and Electrophysiology: Proceedings of the VIIIth World Symposium on Cardiac Pacing and Electrophysiology*. R + L: Creative Communications, 1987: 373-379.
10. Gallagher JJ, Sealy WC, Kasell J. Intraoperative mapping studies in Wolff-Parkinson-White syndrome. *Pace* 1979; **2**: 523-537.
11. Gallagher JJ, Sealy WC, Kasell J, Wallace AG. Multiple accessory pathways in patients with pre-excitation syndrome. *Circulation* 1976; **54**: 571-591.
12. Guiraudon GM, Klein GJ, Sharma AD, Yee R, McLellan DG. Surgical treatment of supraventricular tachycardia: a five year experience. *Pace* 1986; **9**: 1376-1379.
13. Rowland E, Robinson K, Edmondson S, Krikler DM, Bentall HH. Cryoablation of the accessory pathway in Wolff-Parkinson-White syndrome: initial results and long-term follow-up. *Br Heart J* 1988; **59**: 453-457.
14. Cox JL, Ferguson TB: Cardiac arrhythmia surgery. *Curr Probl Surg* 1989; **26**: 206-232.