Treatment of tachycardias associated with the Wolff-Parkinson-White syndrome by transvenous electrical ablation of accessory pathways

DAVID E WARD,* A JOHN CAMM†

From the *Cardiology Department, St George's Hospital, and the †Department of Cardiovascular Medicine, St Bartholomew's Hospital, London

SUMMARY Three patients with tachycardias associated with the Wolff-Parkinson-White syndrome had failed to respond to antiarrhythmic drugs and underwent transvenous ablation of accessory pathways. Intracardiac studies located the site of accessory pathway to the septum in two patients and mid-posterobasal left atrioventricular junction in one. Ablation was performed by positioning an electrode lead as close as possible to the accessory tract and delivering shocks of 50 to 100J using a conventional defibrillator. In all patients the accessory pathway was abolished after the first three shocks. In two patients followed for four and nine months there was no recurrence of tachycardia or pre-excitation. The other patient developed pre-excitation again three weeks later and repeat ablation was performed. This patient has been followed for six months with no evidence of a recurrence of pre-excitation.

This method may provide a valuable alternative to pacemaker and surgical treatment in selected patients with drug resistant arrhythmias associated with accessory atrioventricular connexions.

Transvenous pacing and ablation techniques^{1,2} are emerging as important alternatives to drug and surgical treatment for the control of recurrent tachycardias. Pacemakers are usually considered in patients who have failed to respond to drugs or cannot tolerate them for various reasons. There are, however, some situations where pacemaker treatment is not appropriate or the patient is unwilling to consider implantation of a device. Until recently, ablation could be performed only by surgical intervention.³ Transvenous ablation of atrioventricular nodal pathways has been successfully used to control a variety of atrial and junctional arrhythmias by several investigators⁴⁻⁶ and it is likely that this technique will supplant surgical ablation of atrioventricular pathways. Fisher and colleagues have modified this technique to ablate accessory pathways in patients with the tachycardias associated with the Wolff-Parkinson-White syndrome.⁷ In this report we describe a similar method which was successfully used in three patients with this syndrome.

Patients and methods

The three patients were referred for investigation and treatment of tachycardias associated with the Wolff-Parkinson-White syndrome. The Table summarises the clinical details. Transvenous ablation was considered because drugs had either failed or produced intolerable side effects.

ELECTROPHYSIOLOGICAL STUDIES

Standard electrophysiological techniques were used as described previously.⁸ Bipolar or multipolar electrodes with 1 cm interelectrode distances were used for stimulation and recording. The clinical arrhythmia was initiated in each patient. In two patients (cases 2 and 3) mapping of the atrial activation sequence was performed during atrioventricular re-entrant tachycardia. In both cases, earliest atrial activation was found in the coronary sinus region indicating a left direct atrioventricular connexion.⁹ In the patient in case 2 the pathway was localised to the mid-coronary sinus and that in case 3 to the septal region. In the patient in case 1, the accessory pathway conducted only anterogradely and localisation was

Requests for reprints to Dr D E Ward, Cardiology Department, St George's Hospital, Blackshaw Road, London SW17 0QT.

Accepted for publication 13 September 1984

Ablation of anomalous conduction

Table Clinical details and results

Case No	Age (yr)/ sex	ECG pattern	Symptoms	Drugs	Side effects	Details of shocks	Peak CPK activity	Follow up
1	33 M	WPW type A	Palpitation Syncope	Disopyramide Flecainide	Ineffective Rash	50, 80, 80, 100 J 50, 80×5 J (at second attempt)	260 IU/l 304 IU/l (MB 29, 16)	3 Weeks 6 Months
2	26 M	WPW type A	Palpitation	Disopyramide Digoxin Propranolol Verapamil Flecainide	Ineffective Ineffective Ineffective Ineffective Ineffective	50, 80, 80, 100×3 J	1024 IU/l	9 Months
3	31 F	WPW type A	Palpitation	Amiodarone Amiodarone Flecainide Sotalol	Thyrotoxicosis Photosensitivity Ineffective Ineffective	80, 80, 50×8 J	Not done	4 Months

WPW, Wolff-Parkinson-White; CPK, creatine phosphokinase.

achieved by finding the shortest interval between atrial stimulation and ventricular activation while pacing at various sites in the coronary sinus.¹⁰ In this patient, the pathway was located to the septal region.

CORONARY ARTERIOGRAPHY

After detailed electrophysiological studies, selective left coronary arteriography using the Judkins' method¹¹ was performed to define the anatomy of the

left circumflex coronary artery and its branches and the coronary sinus and the relation of these structures particularly in the region of the accessory pathway. In all patients the left coronary artery was non-dominant and the marginal branches were remote from the coronary sinus at the location of the accessory pathway. The coronary sinus itself was of reasonable size (about 1 cm in diameter) as estimated by using the interelectrode distance as a guide.

Fig. 1 Electrogram and electrocardiogram showing effect of 50 W s shock to the distal pole (case 1). During atrial pacing ventricular pre-excitation is evident. Immediately after the shock normal QRS complexes appear. The PR interval is prolonged but returned to normal within several minutes. There is obvious ST segment depression and T wave inversion in lead aVF. This recording was taken at the first attempt at ablation. AV, atrioventricular; RA, right atrium; PCS, proximal coronary sinus; HRA, high right atrium; CS, coronary sinus; HB, His bundle region; RV, right ventricular apex; I, aVF, V1, V6, surface ECG leads.

Fig. 2 Electrogram and electrocardiograms recorded during atrial flutter or fibrillation before and after ablation of accessory pathways (case 1). Before ablation, atrial flutter results in 1:1 atrioventricular conduction and a rapid rate. Atrial fibrillation results in a mean rate of 165 beats/min with a minimum RR interval of 210 ms (not shown). After ablation, atrial fibrillation conducts over the normal pathway with a controlled ventricular response. AP, accessory pathway; CS, coronary sinus; HB, His bundle region; HR, heart rate; I, aVF, V1, V6, surface ECG leads.

ABLATION TECHNIQUE

Ablation of accessory pathways was performed using an adaptation of the technique previously described for ablation of the atrioventricular node-His bundle.⁶ A new 6F or 7F (USCI) bipolar or quadripolar electrode was positioned in the coronary sinus as close as possible to the accessory pathway. Either the distal or the next most proximal pole was attached to a standard defibrillator using an adaptor, and between 4 and 10 synchronised shocks of 50 to 100 J were delivered with the anode positioned at various sites on the anterior chest wall. The positioning of the anode was arbitrary but attempts were made to cover a wide area on the assumption that a vectorial element of the discharge (electrical or mechanical) might be essential to the success of the method. The position of the electrode lead was kept more or less constant for all the shocks; some movement during the shock was, of necessity, unavoidable.

Results

In two patients (cases 1 and 2) pre-excitation disappeared after the first shock (Fig. 1) and in the third patient after the third shock. After the procedure, atrial pacing failed to show ventricular pre-excitation even when combined with isoprenaline infusion. Atrial fibrillation was reinitiated in the patient in case 1, and there was no evidence of pre-excitation (Fig. 2). In no patient could re-entrant tachycardia be started and retrograde conduction was absent. In two patients serum was taken for creatine phosphokinase estimations within one hour and again 10 hours later; the MB fraction was measured in one patient. Repeat studies before discharge failed to elicit tachycardia in the two patients with atroventricular re-entry, and in no patient was there any evidence of pre-excitation.

The patients were followed from four to nine months (Table). Both patients who presented with

re-entrant tachycardia were free of attacks without treatment. The patient with atrial fibrillation (case 1) had a recurrence of attacks at three weeks, and on the electrocardiogram pre-excitation was found to have recurred. At restudy, ventricular pre-excitation was confirmed, and the minimum RR interval as a result of accessory conduction was 290 ms during atrial pacing compared with 190 ms during atrial fibrillation or atrial pacing before ablation. Atrial fibrillation was not induced at the second study. In view of the continued potential for rapid ventricular rates, a second attempt at ablation was undertaken using six shocks (Table). This was successful and symptoms have not returned over six months' follow up.

In all patients subsequent electrocardiograms and 24 hour tape recordings showed normal QRS complexes. Inferior T wave inversion noted immediately after ablation in one patient (case 1) (Fig. 1) gradually disappeared. No adverse effects were detected. None of the patients required antiarrhythmic drug treatment.

Discussion

Transvenous ablation of atrioventricular pathways has now been successfully applied by several groups.⁴⁻⁶ Most of these patients have had refractory atrial or junctional tachycardias, and the aim has been to induce complete atrioventricular block to prevent a rapid ventricular response. In not all cases was this achieved permanently, but the anatomical substrate for tachycardia was damaged and tachycardia did not recur despite resumption of sinus rhythm. In one such patient the tachycardia was intranodal re-entrant in type.⁶ After ablation there was first degree atrioventricular block, but no tachycardias could be induced. In other patients, an accessory pathway was inadvertently ablated during attempted ablation of atrioventricular conduction,^{4,12} and the patients remained free of tachycardias despite continued atrioventricular conduction. Thus selective ablation of the tachycardia mechanism avoiding induction of atrioventricular block is possible. This is preferable because it obviates the need for permanent pacing. In patients with refractory atrioventricular tachycardias associated with the Wolff-Parkinson-White syndrome ablation of the accessory pathway rather than the atrioventricular non-His bundle is the procedure of choice.¹³ The ability to abolish accessory conduction by a transvenous method would, therefore, represent a considerable advance. This possibility was explored by Brodman and Fisher.¹⁴ They delivered low energy shocks of between 35 and 45 J in the coronary sinus of dogs and subsequently found localised histological changes in the atrial wall and atrioventricular groove. They speculated that such damage might be sufficient

to ablate conduction in direct left atrioventricular accessory connexions. They also presented preliminary data on the use of this technique in four patients.⁷ Energy levels of 60-80 J were delivered via an electrode in the coronary sinus. Complete block of accessory conduction was not achieved in any patient but a symptomatic improvement was noted in all. In some patients they found that the accessory pathway became more sensitive to antiarrhythmic drugs.

The present report further confirms the feasibility of this technique for ablation of left sided accessory pathways near the ostium of the coronary sinus. We have been reluctant, however, to apply shocks to the distal coronary sinus region especially when the diameter of the sinus is not much greater than that of the electrode. We also have reservations about delivering shocks in the coronary sinus if the circumflex artery is in close approximation to the sinus. Brodman and Fisher noted intimal hyperplasia in dogs, but there was no angiographic abnormality of the coronary artery.¹⁴

The energies we used are similar to those applied by Fisher *et al.*⁷ Our higher success rate may have been the result of a more favourable relation between electrode and accessory pathway. Nevertheless, unlike these authors, we chose to use an external anode plate rather than two poles of the electrode. Whether the movement of the anode over the anterior chest wall increases the likelihood of permanently impairing accessory conduction is not known. These differences in technique may account in part for the discrepancy between our results and those of Fisher *et al.*⁷ The evidence of Brodman and Fisher suggests that multiple lower energy shocks are safer and more effective than a single high energy shock.¹⁴ There is as yet no information about the optimum number of shocks that may be given. We have not used energies greater than 100 J because they may rupture the coronary sinus.¹⁴ In a recently published case report accessory conduction was abolished by two shocks of 400 J delivered to the ostium of the coronary sinus.¹⁵ This patient was followed for nine months without recurrence of pre-excitation or tachycardias. As yet, we have no experience of transvenous ablation of right sided pathways. In one such patient, Weber and Schmitz used two shocks of 150 J in the right atrium which caused transient abolition of pre-excitation.¹⁶ Failure to achieve block may have been related to difficulties in accurately localising right atrioventricular accessory pathways.⁹ The ability to record potentials from accessory pathways¹⁷ may enhance the success of this technique. Jackman *et al* did not, however, achieve permanent ablation of accessory pathways despite having recorded potentials from the region of the accessory pathway.¹⁸ The feasibility and utility of this recording method remains to be evalu-

ated.

No side effects were noted. In particular there was no evidence of significant myocardial, arterial, or valvar damage. The electrocardiographic changes observed in the patient in case 1 were present immediately after ablation and are likely to have been caused by longstanding pre-excitation. This phenomenon has been well described by Nicolai *et al.*¹⁹ The small rise in creatine phosphokinase MB activity indicates some myocardial cellular damage, but this was not associated with electrocardiographic changes or clinical effects. The reasons for failure of the initial attempt at ablation in one patient are not apparent. It is possible that shocks may cause a temporary stunning of conduction such as has been noted in patients undergoing His bundle ablation.⁶ The second attempt may have succeeded where the first failed because of cumulative effects. The decrease in the maximum conduction frequency after the first attempt supports this possibility.

In conclusion, transvenous ablation of accessory pathways is a feasible method of treatment of the Wolff-Parkinson-White syndrome in selected patients. At present, we feel it should be limited to patients with refractory arrhythmias associated with accessory pathways on the right or near to the ostium of the coronary sinus. We would like to emphasise that this method is under evaluation and further information is needed before it can be recommended for more widespread use.

References

- 1 Fisher JD, Matos JA, Kim SG. The sparkling joules of internal cardiac stimulation: cardioversion, defibrillation and ablation. *Am Heart J* 1982; 104: 177-81.
- 2 Ward DE. Supraventricular tachycardias. *Br Med J* 1984; 288: 344-5.
- 3 Camm AJ, Ward DE, Spurrell RAJ, Rees GM. Cryothermal mapping and cryoablation in the treatment of refractory cardiac arrhythmias. *Circulation* 1980; 62: 67-74.
- 4 Gallagher JJ, Svenson RH, Kasell JH, *et al.* Catheter technique for closed-chest ablation of the atrioventricular conduction system. A therapeutic alternative for the treatment of refractory supraventricular tachycardia. *N Engl J Med* 1982; 306: 194-200.
- 5 Scheinman MM, Morady F, Hess DS, Gonzalez R. Catheter-induced ablation of the atrioventricular junction to control refractory supraventricular arrhythmias. *JAMA* 1982; 248: 851-5.
- 6 Ward DE, Hellestrand K, Nathan A, Camm AJ. Transvenous ablation of atrioventricular conduction by high energy shock—a new method of tachycardia control. In: Steinbach K, Glogar D, Laszkovics A, Scheibelhofer W, Weber H, eds. *Cardiac pacing. Proceedings of the VII world symposium on cardiac pacing, Vienna*. Darmstadt: Steinkopff Verlag, 1983: 891.
- 7 Fisher JD, Brodman R, Kim SG, Matos JA. Nonsurgical Kent bundle ablation via the coronary sinus in patients with the Wolff-Parkinson-White syndrome [Abstract]. *Circulation* 1982; 66 (suppl II): II-375.
- 8 Ward DE, Camm AJ, Pearce RC, Spurrell RAJ, Rees GM. Incessant atrioventricular tachycardia involving an accessory pathway: preoperative and intraoperative electrophysiologic studies and surgical correction. *Am J Cardiol* 1979; 44: 428-34.
- 9 Gallagher JJ, Pritchett ELC, Benditt DG, *et al.* New catheter techniques for analysis of the sequence of retrograde atrial activation in man. *Eur J Cardiol* 1977; 6: 1-14.
- 10 Denes P, Wyndham CR, Amat-y-Leon F, *et al.* Atrial pacing at multiple sites in the Wolff-Parkinson-White syndrome. *Br Heart J* 1977; 39: 506-14.
- 11 Judkins MP. Selective coronary arteriography. *Radiology* 1974; 89: 815-24.
- 12 Critelli G, Perticone F, Coltorti F, Monda V, Gallagher J. Closed chest modification of atrioventricular conduction system in man for treatment of refractory supraventricular tachycardia. *Br Heart J* 1983; 49: 544-9.
- 13 Gallagher JJ. Surgical treatment of arrhythmias: current status and future directions. *Am J Cardiol* 1978; 41: 1035-44.
- 14 Brodman R, Fisher JD. Evaluation of a catheter technique for ablation of accessory pathways near the coronary sinus using a canine model. *Circulation* 1983; 67: 923-9.
- 15 Morady F, Scheinman M. Transvenous catheter ablation of a posteroseptal accessory pathway in a patient with the Wolff-Parkinson-White syndrome. *N Engl J Med* 1984; 310: 705-7.
- 16 Weber H, Schmitz L. Catheter technique for closed-chest ablation of an accessory atrioventricular pathway [Letter]. *N Engl J Med* 1983; 308: 653-4.
- 17 Prystowsky EN, Browne KF, Zipes DP. Intracardiac recording by catheter electrode of accessory pathway depolarization. *J Am Coll Cardiol* 1983; 2: 468-70.
- 18 Jackman WM, Friday KJ, Scherlag BJ, *et al.* Direct endocardial recording from an accessory atrioventricular pathway: localization of the site of block, effect of antiarrhythmic drugs, and attempt at nonsurgical ablation. *Circulation* 1983; 68: 906-16.
- 19 Nicolai P, Medvedowsky JL, Delaage M, Barnay C, Blache E, Pisapia A. Wolff-Parkinson-White syndrome: T wave abnormalities during normal pathway conduction. *J Electrocardiol* 1981; 14: 295-300.