Arrhythmias after coronary bypass surgery

O J M ORMEROD, C G A McGREGOR, D L STONE, C WISBEY, M C PETCH

From the Regional Cardiac and Cardiothoracic Surgical Units, Papworth Hospital, Cambridge

SUMMARY Ninety patients undergoing coronary bypass surgery were studied prospectively by bedside and subsequent ambulatory electrocardiographic monitoring to investigate the incidence, possible causes, and prevention of atrial fibrillation. Patients with good left ventricular function were divided randomly into a control group or groups treated with digoxin or propranolol. In the control group the incidence of atrial fibrillation was 27% and of significant ventricular extrasystoles 3%. Propranolol reduced the incidence of atrial fibrillation (14·8%), whereas digoxin had no effect and increased the incidence of ventricular extrasystoles. Age, sex, severity of symptoms, cardiomegaly, heart failure, previous myocardial infarction, and number of grafts did not affect the result. The operative myocardial ischaemic time was related to the occurrence of atrial fibrillation. There was also a significant relation between atrial fibrillation and bundle branch block. Atrial fibrillation is common after coronary artery grafting; it may be due to diffuse myocardial ischaemia or hypothermic injury. The incidence may be reduced by beta blockade.

The risks of cardiac surgery have decreased in the past decade. The lower mortality, due largely to the lower incidence of intraoperative myocardial injury,[1][2] has allowed surgical treatment of mildly symptomatic lesions with a view to improving life expectancy. One result of this has been that the morbidity of heart surgery has assumed greater importance. Cardiac arrhythmias, especially atrial fibrillation, are a common cause of early morbidity causing discomfort to the patient and prolonging hospital stay. The aetiology of postoperative atrial fibrillation is not understood; no reliable prophylaxis is known, and treatment is usually aimed at controlling the ventricular response until reversion to sinus rhythm occurs spontaneously.

This investigation of arrhythmias in patients undergoing coronary artery bypass grafting was carried out to assess their incidence, to identify any aetiological factors, and to observe the effects of antiarrhythmic treatment with digoxin and low doses of propranolol. Patients with coronary artery disease were chosen because they represented a homogeneous group with good left ventricular function. We decided to study the effect of digoxin because this drug is used routinely after cardiac surgery in many units in this

country and of propranolol at a low dose to test the hypothesis that early postoperative arrhythmias might be exacerbated by beta blocker withdrawal and prevented by this form of treatment.

Patients and methods

Consecutive patients undergoing elective coronary artery bypass surgery for angina pectoris were studied. They were randomly allocated to three groups: a control group receiving no specific antiarrhythmic agent, a group receiving digoxin (0·25 mg daily), and a group receiving propranolol (15–30 mg a day according to body weight). Treatment was started on the morning after operation as soon as the patient was able to take drugs by mouth. Patients with preoperative ejection fractions of <0·40 and known contraindications to either antiarrhythmic drug (for example, bronchial asthma) were excluded. Patients with postoperative low cardiac output or continued assisted ventilation preventing oral drug treatment were withdrawn.

The preoperative data were acquired from the patients' records at the time of randomisation, usually at the cardiology-surgery case conference the week before. Observations were made on the severity of symptoms graded according to the New York Heart Association classification, previous myocardial infarction, heart size on chest x ray film, left ventricular end diastolic pressure, left ventricular ejection fraction

derived from single plane conventional angiography, and extent of significant coronary artery involvement (lesions of >50%) on coronary arteriography. All operations were performed at this hospital using a method of myocardial protection including systemic hypothermia, single dose cold cardioplegia, and topical hypothermia.[3] The operative and postoperative data were acquired prospectively. Patients were followed by normal ward observations, daily electrocardiograms for one week, with appropriate recording of arrhythmias, and 24 hour ambulatory electrocardiographic monitoring on the fourth postoperative day, which was analysed by a Reynolds Pathfinder analyser with a trend recorder. Significant atrial fibrillation was defined as a prolonged period of irregular atrial tachycardia and significant ventricular arrhythmia as greater than a mean hourly rate of 120 ventricular extrasystoles or two or more occurring consecutively.

Groups were compared using the χ^2 test.

Results

Ninety four consecutive patients who fulfilled the entry criteria were randomised. Four patients were withdrawn after operation, as described above. Ninety patients were entered into the treatment stage of the study. There were 81 men and nine women (mean age 54 (range 38–67) years). The distribution of preoperative and operative factors in each treatment group are shown in Table 1; there are no significant differences. The postoperative treatment had no significant effect on the incidence of atrial fibrillation (Table 1). To determine the preoperative factors affecting this arrhythmia the groups were pooled as shown in Table 2. There was no difference in the

Table 1 *Preoperative data and incidence of postoperative arrhythmias in three randomised groups of patients*

	Groups treated with:		Control group
	Digoxin	Propranolol	
Preoperative data			
Total No of patients	30	27	33
Mean age (yr)	57	54·9	51·8
Male/female	28/2	23/4	30/3
Previous myocardial infarction (No)	13	12	12
Mean vessel involvement	2·6	2·6	2·4
Mean No of grafts inserted†	2·9	3·1	2·9
Postoperative data			
Arrhythmias (No of patients)			
Atrial fibrillation	10	4	9
Ventricular extrasystoles	8	3	1*
Bundle branch block	6	2	6

*p<0·01.
†Including grafts to the major divisions and their subdivisions (for example, left anterior descending and diagonal).

incidence of atrial fibrillation according to the various criteria.

The operative data are also detailed in Table 2. Although there was no relation to the number of grafts inserted, there was a significant (p<0·05) association between the incidence of postoperative atrial fibrillation and the operative ischaemic time. The Figure shows the cumulative percentage of patients having atrial fibrillation as the ischaemic time prolongs and illustrates the sudden increase in frequency after 50–60 minutes.

Although the postoperative treatment had no significant effect on the incidence of atrial fibrillation those patients taking digoxin had more ventricular extrasystoles (Table 1). The incidence of bundle branch block was not significantly different in the three treatment groups, but the overall incidence was high (15·5%). There was a significant association between the occurrence of atrial fibrillation and bundle branch block (Table 2) (p<0·05).

Table 2 *Preoperative and postoperative data in relation to presence of atrial fibrillation*

|	Total No of patients	No with atrial fibrillation
Preoperative data		
Age (yr)		
<60	67	15
≥60	23	8
Sex		
F	9	1
M	81	22
Symptoms (NYHA class)		
I	1	0
II	41	8
III	33	14
IV	15	1
Previous myocardial infarction		
Yes	37	10
No	53	13
Cardiothoracic ratio		
≥50%	3	2
≤49%	87	21
LV end diastolic pressure (mm Hg)		
>12	23	8
≤12	62	15
Extent of coronary artery disease		
Single vessel	8	2
Double vessel	24	4
Triple vessel	58	17
Postoperative data		
No of grafts		
1	8	1
2	17	5
3	38	6
4	26	11
5	1	0
Significance	NS	
Ischaemic time (min)		
0–55	36	5
>56	54	18
Significance	p<0·05	
Bundle branch block		
With	14	7
Without	76	16
Significance	p<0·05	
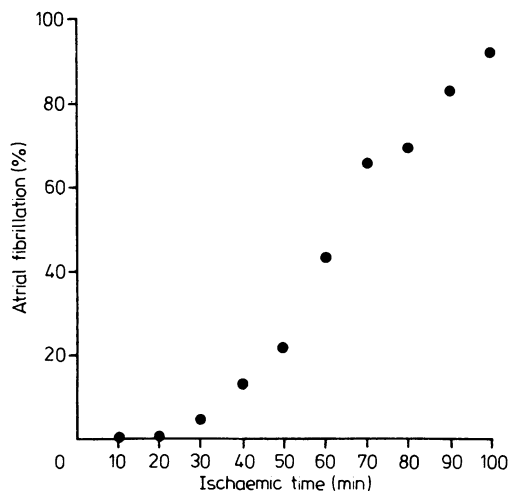
Figure   *Cumulative percentage of incidence of atrial fibrillation in relation to myocardial ischaemic time.*

Discussion

Disturbances of atrioventricular conduction due to direct surgical trauma to the conducting tissue are a well recognised and understandable complication of aortic valve replacement,[4][5] and surgery for congenital defects particularly the repair of a ventricular septal defect.[6-8] Ectopic tachycardias are less easy to understand but seem to be associated with all procedures involving cardiac arrest. Atrial fibrillation and flutter are most common with an incidence of 10–30%,[9] which is in agreement with our findings.

Many factors might provoke atrial arrhythmias after heart surgery. These include pre-existing left ventricular dysfunction with left atrial hypertension and dilatation, direct trauma—for example, from the venous lines—ischaemic damage, biochemical changes associated with the use of cardioplegic solutions, and sudden withdrawal of beta blocking drugs.[10-12] In recent years the incidence of intraoperative myocardial injury has decreased, as judged by enzyme release or ventricular function studies.[13][14] This has been attributed to improvements in myocardial preservation, particularly the change from coronary perfusion to cardioplegia.[13][15][16] Nevertheless, whereas in the past decade the incidence of intraoperative myocardial damage has fallen appreciably, the incidence of atrial fibrillation has remained high. This makes the simpler explanations such as direct trauma to the atria or myocardial injury less likely. Postoperative metabolic causes, such as electrolyte imbalance and pericarditis, have also been investigated and appear to have no effect.[1][17] Furthermore, in our patients the preoperative factors did not appear to influence the incidence of atrial fibrillation.

We cannot assess directly the likelihood of a beta blocker withdrawal phenomenon because almost all of our patients were taking beta adrenergic blocking drugs before operation. It is our experience with others,[17] however, that postoperative atrial fibrillation is equally common in patients after aortic valve replacement who have not received beta blocking drugs. Moreover, low dose propranolol, which can abolish this phenomenon, had no significant effect. Digoxin is a myocardial irritant, and, although it could have been responsible for provoking either atrial or ventricular arrhythmias, we found no effect on the incidence of atrial fibrillation. Although prophylactic digitalisation after cardiac surgery has been proposed,[18][19] our experience indicates that this is unhelpful and may increase ventricular ectopic activity. It has been suggested that increased myocardial sensitivity to digoxin after cardiopulmonary bypass accounts for the occurrence of arrhythmias at therapeutic digoxin concentrations,[20] and this may have been a factor in our patients who experienced ventricular arrhythmias. The routine use of digoxin after cardiac surgery cannot be recommended.

In our study the most important factor determining the incidence of postoperative atrial fibrillation was the myocardial ischaemic time. This supports a similar previous observation.[2] There was a notable increase in the frequency of atrial fibrillation in those patients whose ischaemic time exceeded 50 minutes. Although cardioplegia is theoretically safe for ischaemic times of 120 minutes,[2] the relation between the ischaemic time and the incidence of atrial fibrillation suggests that the arrhythmia is a response to myocardial ischaemia. Despite this, the usual markers of myocardial injury were rarely seen; for example, the incidence of transmural infarction assessed by the development of new Q waves was only 2·2%. Such electrocardiographic changes would, however, be expected with major damage involving occlusion of a large vessel. Perhaps atrial arrhythmias result from more diffuse damage secondary to cardiac arrest and artificial myocardial protection.

Transient bundle branch block is common after cold cardioplegic arrest. Right bundle branch block appears to be benign and may be due to the effects of the technique.[21] Persisting right bundle branch block or left bundle branch block is less common, carries a more serious prognosis, and is more likely to be due to myocardial injury.[22] The incidence of right bundle branch block in our patients (16%) is similar to that in other studies.[21] The association between right bundle branch block and atrial fibrillation in our series suggests a common aetiology for these abnormalities. Transient right bundle branch block is associated with prolongation of the QT interval and J waves early after heart surgery, and it has been suggested

that benign bundle branch block may be a consequence of hypothermia.[21] Thus the hypothermia associated with the cardioplegic solution may be a contributory factor in provoking atrial fibrillation after heart surgery with preferential damage to the conducting tissue as suggested by animal experiments.

References

1 Brewer DL, Bilbro RH, Bartel AG. Myocardial infarction as a complication of coronary bypass surgery. *Circulation* 1973; **47**: 58–64.

2 Engelman RM, Rousou JH, Vertrees RA, Rohrer C, Auvil J. Safety of prolonged ischemic arrest using hypothermic cardioplegia. *J Thorac Cardiovasc Surg* 1980; **79**: 705–12.

3 Wheeldon DR, Bethune DW, Gill RD, English TAH. A simple cooling circuit for establishing and maintaining profound cardiac hypothermia during cardio-pulmonary bypass. *Proc Am Soc Extracorporeal Technology* 1978; **6**: 145–53.

4 Gannon PG, Sellers RD, Kanjuh VI, Edwards JE, Lillehei CW. Complete heart block following replacement of the aortic valve. *Circulation* 1966; **33** (suppl 1): 152–61.

5 Ginks WR, Follath F. Changes in QRS complex after aortic valve replacement [Abstract]. *Br Heart J* 1971; **33**: 613–4.

6 Chesler E, Beck W. Schrire V. Left anterior hemi-block and right bundle branch block before and after surgical repair of tetralogy of Fallot. *Am Heart J* 1972; **84**: 45–52.

7 Gelband H, Waldo AL, Kaiser GA, Bowman FO Jr, Malm JR, Hoffman BF. Etiology of right bundle-branch block in patients undergoing total correction of tetralogy of Fallot. *Circulation* 1971; **44**: 1022–33.

8 Fryda RJ, Kaplan S, Helmsworth JA. Postoperative complete heart block in children. *Br Heart J* 1971; **33**: 456–62.

9 Mills SA, Poole GV, Breyer RH, Hudspeth AS, Johnston FR, Cordell AR. Digoxin and propranolol in the prophylaxis of dysrhythmias after coronary artery bypass surgery [Abstract]. *Circulation* 1982; **66** (suppl II): 220.

10 Miller RR, Olson HG, Amsterdam EA, Mason DT. Propranolol-withdrawal rebound phenomenon: exacerbation of coronary events after abrupt cessation of anti-anginal therapy. *N Engl J Med* 1975; **293**: 416–8.

11 Frishman W, Silverman R, Strom J, Elkayam W, Sonnenblick E. Clinical pharmacology of the new β-adrenergic blocking drugs. Part 4. Adverse effects. Choosing a β-adrenoreceptor blocker. *Am Heart J* 1979; **98**: 256–62.

12 Williams LC, Turney JH, Parsons V. Beta-blocker withdrawal syndrome [Letter]. *Lancet* 1979; i: 494–5.

13 Davids HA, Hermans WTH, Hollaar L, van der Laarse A, Huysmans HA. Extent of myocardial damage after open-heart surgery assessed from serial plasma enzyme levels in either of two periods (1975 and 1980). *Br Heart J* 1982; **47**: 167–72.

14 Espinoza J, Lipski J, Litwak R, Donoso E, Dack S. New Q waves after coronary artery bypass surgery for angina pectoris. *Am J Cardiol* 1974; **33**: 221–4.

15 Craver JM, Sams AB, Hatcher CR Jr. Potassium-induced cardioplegia. *J Thorac Cardiovasc Surg* 1978; **76**: 24–7.

16 Adams PX, Cunningham JN Jr, Trehan NK, Brazier JR, Reed GE, Spencer FC. Clinical experience using potassium-induced cardioplegia with hypothermia in aortic valve replacement. *J Thorac Cardiovasc Surg* 1978; **75**: 564–8.

17 Williams JF Jr, Morrow AG, Braunwald E. The incidence and management of "medical" complication following cardiac operation. *Circulation* 1965; **32**: 608–19.

18 Johnson LW, Dickstein RA, Fruehan T, et al. Prophylactic digitalization for coronary artery bypass surgery. *Circulation* 1976; **53**: 819–22.

19 Csicsko JH, Schatzlein MH, King RD. Immediate postoperative digitalization in the prophylaxis of supraventricular arrhythmias following coronary artery bypass. *J Thorac Cardiovasc Surg* 1981; **81**: 419–22.

20 Morrison J, Killip T. Serum digitalis and arrhythmia in patients undergoing cardiopulmonary bypass. *Circulation* 1973; **47**: 341–52.

21 O'Connell JB, Wallis D, Johnson SA, Pifarre R, Gunnar RM. Transient bundle branch block following use of hypothermic cardioplegia in coronary artery bypass surgery: high incidence without perioperative myocardial infarction. *Am Heart J* 1982; **103**: 85–91.

22 Zeldis SM, Morganroth J, Horowitz LN, et al. Fascicular conduction disturbances after coronary bypass surgery. *Am J Cardiol* 1978; **41**: 860–4.