Value of pacing in cardiac failure associated with chronic atrioventricular block

M. R. HETZEL¹, W. R. GINKS, A. J. PICKERSGILL², AND A. LEATHAM

From the Cardiac Department, St. George's Hospital, London

SUMMARY The effect of permanent pacing on chronic complete atrioventricular block complicated by cardiac failure was studied in 6 patients by measurement of indirect left atrial pressure 15 minutes after institution of pacing and again 3 to 12 months later. In addition, 21 patients with complete heart block and clinical plus radiological evidence of cardiac failure at the time of pacing 3 to 6 years earlier were also reviewed.

Only 1 of 6 patients studied haemodynamically improved and 1 died in cardiac failure. Of 21 patients assessed clinically, 10 had improved and 8 had died after a mean follow-up of 53 months.

In the absence of syncope, pacing was of little symptomatic benefit but still may be justified to prolong survival. Both studies indicated a particularly poor prognosis for patients known to have coronary artery disease. No reliable means were found of determining the prognosis in the individual patient with cardiac failure before pacing.

While improvement in expectation of life from long-term pacing for the treatment of chronic atrioventricular block with syncope is well established (Siddons, 1974), there is controversy as to whether or not pacing is beneficial to patients with additional heart failure (Bernstein *et al.*, 1971; Davidson *et al.*, 1972; *British Medical Journal*, 1975). Many patients complain of shortness of breath on exertion which improves after pacing, but only a few patients present with objective evidence of cardiac failure. Recognising the morbidity of pacemaker implantation, it is particularly difficult to decide if pacing is the correct treatment for the elderly patient with chronic atrioventricular block who presents in cardiac failure without syncope.

We have, therefore, studied the immediate and long-term effects of pacing in a group of patients in chronic atrioventricular block who were in cardiac failure when referred for pacing. In a prospective haemodynamic study the changes in indirect left atrial pressure were measured when permanent pacing was instituted and were repeated

3 to 12 months later. A long-term view of the value of pacing in the treatment of cardiac failure was obtained by a retrospective clinical study of similar patients who had been managed by permanent pacing for more than 3 years.

Patients and methods

In both studies the criteria for inclusion were cardiac failure of more than 1 month's duration in the presence of chronic complete atrioventricular block at the time of referral for pacing. Cardiac failure was defined as a rise in jugular venous pressure of more than 5 cm above the sternal angle and/or a chest x-ray film compatible with left ventricular failure, in the opinion of a cardiac radiologist. All patients were subsequently paced with fixed rate pacemakers and transvenous electrodes implanted in the right ventricle. Patients with sinoatrial disease, intermittent heart block, acute heart block after myocardial infarction, and congenital and surgically induced block were excluded from the studies.

HAEMODYNAMIC STUDY

Six patients satisfied these criteria and presented between 1975 and 1976. They gave informed consent to haemodynamic studies during the installation of a permanent pacing system. Right

¹Present address: Department of Thoracic Medicine, St. James' Hospital, Sarsfield Road, SW12.

²Present address: Department of Medicine, Stanford University Medical Center, U.S.A.

heart catheterisation was performed in each patient. The mid-chest level from the angle of Louis was used as the zero reference. An oscilloscopic photographic recorder was used for recording pressures measured with strain gauge transducers (Bell and Howell).

When a stable electrode position had been obtained at the time of pacemaker implantation, a Swan Ganz catheter was advanced via the right antecubital vein, and unpaced resting indirect left atrial pressure was measured. In 3 patients additional measurement of indirect left atrial pressure was then made after a 3-minute exercise period of supine straight leg raising. The endocardial electrode was then attached to an external pulse generator and all patients were paced at 70 beats per minute for 15 minutes. Indirect left atrial pressure was then recorded again in the resting state while pacing continued. In the same 3 cases who had exercised before pacing was instituted, the exercise test was subsequently repeated while pacing continued and indirect left atrial pressure was again measured at the end of 3 minutes of straight leg raising. Finally the catheter was removed and the endocardial electrode was attached to a fixed rate pacemaker implanted subcutaneously in the right axilla.

Swan Ganz right heart catheterisation was repeated after continued pacing from the permanent pacing system for 3 months in 2 patients and 12 months in the remainder. Clinical and radiological examinations were repeated at the same time. Only paced indirect left atrial pressure could now be measured as the permanent system continued to function. Resting measurements were made in all patients and the exercise procedure was then repeated in the same 3 patients who had exercised in the first study.

In all cases comparisons were then made between the original unpaced results, the results after 15 minutes pacing, and the long-term response to continued pacing for 3 to 12 months.

CLINICAL STUDY

While the haemodynamic study was in progress, we retrospectively reviewed the records of another group of patients consisting of all patients paced for chronic atrioventricular block between January 1971 and December 1973 to select those who satisfied our criteria for cardiac failure at presentation. These patients have now all been followed up for a minimum of 3 years after pacemaker implantation. Survivors were reviewed by clinical and radiological methods for evidence of improvement in cardiac failure. Information on early progress was obtained from pacing clinic notes made 1 month and then

at yearly intervals after pacemaker implantation. Deaths and necropsy details were reviewed from hospital records.

Results

HAEMODYNAMIC STUDY

Patients' mean age was 76 years (range 70 to 81 years). There were 5 men and 1 woman. Two patients showed features of coronary artery disease which consisted of a history of angina in 1 and old anterior myocardial infarction confirmed by the pre-pacing electrocardiogram in the other. The aetiology of the atrioventricular block in the other patients was unknown. All patients were clinically in severe failure, defined as a gross rise in jugular venous pressure and/or distinct x-ray features of left ventricular failure. Two patients had syncope. Five were treated with diuretics during the study and 1 took digoxin. No alterations were made to drug regimens between the initial and final catheterisations.

Only 1 patient (JM male aged 72) showed distinct clinical and radiological improvement when reviewed at 12 months. Slight improvement was seen after continued pacing in 4 patients but one of them (CO male aged 81), who had evidence of previous myocardial infarction and was reviewed at 3 months, subsequently died in intractable failure after 12 months. Chest x-rays showed persistent cardiac enlargement in 4 patients. Of 2 patients with normal cardiac diameter on x-ray at presentation, only 1 (JM) improved.

The early response to pacing is shown in Fig. 1 where indirect left atrial pressure before and after a 15-minute period of fixed rate pacing are compared in each patient. The mean resting unpaced pressure for all 6 patients fell from 20 mmHg at an average idioventricular rate of 38 beats per minute to 18 mmHg at 70 beats per minute. This slight improvement was not statistically significant (Student's paired *t* test; $P > 0.1$). In the 3 patients who also exercised, indirect left atrial pressure rose in spite of the increase in ventricular rate from an unpaced mean of 27 mmHg to 33 mmHg after pacing. No statistical conclusions can be drawn from results in only 3 patients but an adverse trend is indicated.

The long-term effect of pacing on indirect left atrial pressure is shown in Fig. 2. Comparison is made between the initial unpaced indirect left atrial pressures, which have already been shown in Fig. 1, and paced measurements recorded during continued fixed rate pacing 3 months later in 2 cases and 12 months later in the other 4. The mean resting pressure after prolonged pacing

Fig. 1 Early response of indirect left atrial pressure to electrical pacing. Individual and mean results for indirect left atrial pressures are shown, related to prevailing ventricular rate, at rest (6 patients) and after 3 minutes exercise (3 patients); unpaced and then after 15 minutes pacing.

Fig. 2 Long-term response of indirect left atrial pressure to electrical pacing. Comparison of prepacing left atrial pressure at rest (6 patients) and on exercise (3 patients) with paced left atrial pressure 3 to 12 months later.

in all patients was 21 mmHg and showed no significant improvement on the original unpaced mean of 20 mmHg ($P > 0.05$). Exercise data are

for the same 3 patients as in Fig. 1. In 2 patients pronounced rises in exercising, indirect, left atrial pressure of 30 to 40 mmHg and 25 to 35 mmHg were seen between the original unpaced study and the final measurements after prolonged continued pacing. The third patient (case JM), however, showed an eventual fall from 25 to 16 mmHg; thus the mean pressures for these 3 patients only rose from 27 mmHg to 31 mmHg.

Haemodynamic improvement after prolonged pacing was seen only in case JM who was reviewed at 12 months when clinical improvement was also impressive. His indirect left atrial pressure at rest eventually fell from 21 to 7 mmHg and post-exercise results improved from 25 to 16 mmHg. However, no appreciable improvement was seen after 15 minutes of pacing in the first study in this patient.

CLINICAL STUDY

Three hundred and twenty-four patients with chronic atrioventricular block were paced between 1971 and 1973. Twenty-one (6.5%) satisfied our criteria for cardiac failure and were the subject of the clinical study. There were 14 men and 7 women, the mean age was 72.9 years (range 44 to 87 years). Nine were in severe cardiac failure before pacing as defined by a gross rise in venous pressure and/or pronounced x-ray features of left ventricular failure. Twelve patients had syncope.

All patients had improved when discharged from hospital but follow-up after an average of 53 months (range 38 to 72 months) disclosed 3 groups of patients:

Group A: 8 patients who deteriorated and died in cardiac failure.

Group B: 3 patients who showed no real improvement.

Group C: 10 patients who maintained a definite improvement.

These results were evident at the first annual follow-up except for 2 patients in group A who improved during the first year after pacing.

The long-term prognosis of these patients was not influenced by age, idioventricular rate, or cardiac size before pacing. The mean age of the survivors was 72 years (range 44 to 87 years) and 74 years (range 68 to 87 years) in those who died. The average idioventricular rates were 37 per minute (range 25 to 50 per minute) in those who improved and 39 beats per minute (range 30 to 60 per minute) in those who died. Mean transverse cardiac diameter before pacing was 17.0 cm (range 14.0 to 21.0 cm) in those who improved and 16.5 cm (range 15.0 to 18.0 cm) in patients who died. Five patients, including 2 who died in cardiac failure, showed

decreases of more than 1.0 cm in transverse diameter within one month of pacing.

The clinical findings and medical treatment are outlined in the Table. In group A, 5 of the 8 patients who died had coronary artery disease on the basis of electrocardiographic evidence of infarction in 2 and necropsy data in 3. Five patients were in severe cardiac failure before pacing; 3 were treated with digoxin and diuretics before pacing and 2 continued full treatment afterwards. Three deaths occurred in patients who had less severe cardiac failure and were treated with diuretics only before pacing, but with digoxin plus diuretics afterwards. Evidence of cardiac failure increased in the first year and was a major factor in all deaths. There was no evidence that pacemaker malfunction contributed to any deaths.

Table Results of pacing in 21 patients with cardiac failure and complete heart block

	Total	Increasing cardiac failure and death Group A	No change Group B	Improved Group C
Result of pacing after mean follow-up of 53 months	21	8	3	10
Patients with evidence of coronary artery disease	8	5	1	2
Patients in severe cardiac failure before pacing	9	5	0	4
Patients on full treatment (digoxin and diuretics) before pacing	3	3	0	0
Patients on full treatment 1 year after pacing	9	5	1	3

In group B, the 3 patients in whom the signs and x-ray films remained unchanged were not in severe cardiac failure before pacing. One had diuretics before pacing, and 1 had diuretics and digoxin afterwards. One patient had mild angina.

In group C, 2 of the 10 patients had a history of angina. Four presented in severe heart failure; 3 had diuretics before pacing and 3 were also treated with digoxin after pacing. Of the remaining 6 patients, 3 had diuretics before pacing and 2 had diuretics plus digoxin afterwards. Three of the younger patients (ages 44 to 68 years) had no evidence of coronary disease and discontinued medication within 1 year of pacing.

Fig. 3 shows the survival curve of these 21 patients paced for cardiac failure in comparison with curves for 649 paced and 113 unpaced patients

Fig. 3 Effect of pacing on survival of patients with chronic atrioventricular block who present in cardiac failure. Survival curve of present study is compared with curves of 649 paced and 113 unpaced patients who did not present in cardiac failure (Siddons, 1974) and 26 patients in cardiac failure who were not paced (compiled from Eraut, 1975).

who were not in cardiac failure at presentation (Siddons, 1974) and 26 patients who presented in cardiac failure but were not paced (compiled from Eraut, 1975). Statistical analysis of the effect of pacing on survival of cases complicated by cardiac failure is most valid at 2 years as this is the maximum period of follow-up completed by all Eraut's patients. The 2-year survival of patients in cardiac failure is apparently improved from 42 to 69 per cent but this difference is only significant at the 10 per cent level (χ^2 test with Yates' correction; $P = 0.11$).

Discussion

The haemodynamic effects of pacing in chronic atrioventricular block and in particular the effect on cardiac output have been examined in a number of studies (Samet *et al.*, 1964; Sowton, 1964; Bevegård *et al.*, 1967). Such studies of patients with uncomplicated atrioventricular block show a variable change in cardiac output which may frequently increase by more than 50 per cent with pacing. The change in cardiac output, however, is dependent

on the pacing rate. Judge *et al.* (1964) showed a negligible increase in cardiac output with increases in pacing rate between 60 to 110/minute. It has also been seen that in patients with myocardial disease and complete heart block, changes in cardiac output are critically dependent upon the pacing rate, the optimum rate for maximal increase in cardiac output and decrease in venous pressure being as low as 55/minute in some cases (Sowton, 1964). In retrospect it seems likely that the patients studied in this series, who had severe heart failure, were perhaps paced at a rate in excess of the optimum for a maximum reduction in filling pressure and rise in cardiac output.

Little information is available on the haemodynamic effects of pacing in patients with overt heart failure. Gobel *et al.* (1969) described 9 patients with resting unpaced pulmonary artery wedge pressures averaging 13.8 mmHg which fell to 9.9 mmHg mean after 15 minutes pacing. Very little change occurred in cardiac output. The long-term effect of pacing was not studied. In the present haemodynamic study patients were selected with signs of severe cardiac failure; the mean unpaced indirect left atrial pressure was 20 mmHg and only minimal improvement with a fall to 18 mmHg was seen on short-term pacing. This lack of response to pacing presumably reflects advanced myocardial disease.

Our haemodynamic results imply that the majority of patients in whom cardiac failure is severe enough to be the principal indication for pacing show little immediate benefit from pacing and further decompensation occurs as permanent pacing continues. Moreover, the immediate haemodynamic response to pacing seems to have little prognostic value since one patient (JM) who did eventually show considerable benefit with pacing displayed no greater improvement on 15 minutes' pacing than the other patients.

The clinical study showed that 50 per cent (10 of 21 patients) with cardiac failure of varying severity had sustained some improvement. Our experience may be compared with a study by Bernstein *et al.* (1971) which showed that 9 of 22 patients with cardiac failure had died within 1 year of pacing and none of the survivors had improved. Davidson *et al.* (1972) reported a group of 20 patients paced with cardiac failure and followed for 2 years; 13 improved and 3 deaths occurred.

We looked for criteria at the time of pacing which might indicate the prognosis in any individual paced with cardiac failure. Clinical assessment of the severity of cardiac failure, radiological measurement of heart size, and the idioventricular rate were not relevant. In the haemodynamic study

the immediate response to pacing was also of limited prognostic value. Both studies suggest a particularly poor prognosis for patients known to have coronary artery disease. An early improvement after pacing, though often seen, may not be maintained. This phenomenon probably reflects the beneficial effect of the period of bed rest involved in pacemaker implantation. Results after 1 year of pacing appear to be a guide to future progress.

Since only 50 per cent of patients can be expected to show improvement after pacing and these cannot be readily identified, we have studied the influence of pacing on survival. Unfortunately a satisfactory control series of unpaced patients with cardiac failure is difficult to find. Studies before the pacing era such as those of Penton *et al.* (1956) and Johansson (1966) indicate that the unpaced mortality of chronic atrioventricular block may reach 50 per cent in the first year after diagnosis and up to 40 per cent of patients may have signs of cardiac failure at some time (Penton *et al.*, 1956). However, the specific mortality of cases with cardiac failure was not described.

We, therefore, chose for comparison a series described by Eraut (1975) who studied the incidence of chronic atrioventricular block in the general population. There were 31 patients (9% of the patients studied) with cardiac failure. Five of these patients were paced and 22 followed for 3 years.

Survival curves compared in Fig. 3 suggest that the mortality of atrioventricular block is adversely affected by cardiac failure. Survival was apparently improved by pacing but the numbers of patients studied were small and this improvement was not significant.

It is difficult to define the role of digoxin and diuretics as there was no set procedure for their prescription; furthermore both are potent agents and are liable to produce toxic side effects in the elderly. Nevertheless, it seems unwise to deny such a patient the possible benefit of continued treatment with both digoxin and diuretics.

We conclude that pacing is of limited value in the treatment of cardiac failure which complicates chronic complete atrioventricular block. We found no reliable means of selecting patients likely to benefit from pacing. The response to pacing and survival are poor in patients known to have coronary artery disease. Though pacing may have little effect on the quality of life, it may be justified by improving the duration of survival.

We thank Dr K. Jefferson for reviewing the chest x-rays.

References

- Bernstein, V., Rotem, C. E., and Peretez, D. I. (1971). Permanent pacemakers. 8-year follow-up study. Incidence and management of congestive cardiac failure and perforations. *Annals of Internal Medicine*, **74**, 361-369.
- Bevegård, S., Jonsson, B., Karlöf, I., Lagergren, H., and Sowton, E. (1967). Effect of changes in ventricular rate on cardiac output and central pressures at rest and during exercise in patients with artificial pacemakers. *Cardiovascular Research*, **1**, 21-33.
- British Medical Journal* (1975). Editorial. Why so few pacemakers? **2**, 3-4.
- Davidson, D. M., Braak, C. A., Preston, T. A., and Judge, R. D. (1972). Permanent ventricular pacing. Effect on long term survival, congestive heart failure and subsequent myocardial infarction and stroke. *Annals of Internal Medicine*, **77**, 345-351.
- Eraut, D. (1975). The prevalence, incidence, morbidity and mortality of heart block. MD thesis, Cambridge, pp. 86-87.
- Gobel, E. L., Medina, J. R., Guenter, C. A., and Wang, Y. (1969). Immediate hemodynamic response of patients with atrioventricular block and cardiac failure to transvenous pacing. *Circulation*, **39**, 64-71.
- Johansson, B. W. (1966). Complete heart block. Clinical, haemodynamic and pharmacological study in patients with and without an artificial pacemaker. *Acta Medica Scandinavica*, **180**, Suppl. 451, p. 41.
- Judge, R. D., Wilson, W. S., and Siegel, J. H. (1964). Hemodynamic studies in patients with implanted cardiac pacemakers. *New England Journal of Medicine*, **270**, 1391-1395.
- Penton, G. B., Miller, H., and Levine, S. A. (1956). Some clinical features of complete heart block. *Circulation*, **13**, 801-824.
- Samet, P., Bernstein, W. H., Medow, A., and Nathan, D. A. (1964). Effect of alterations in ventricular rate on cardiac output in complete heart block. *American Journal of Cardiology*, **14**, 477-482.
- Siddons, H. (1974). Deaths in long term paced patients. *British Heart Journal*, **36**, 1201-1209.
- Sowton, E. (1964). Haemodynamic studies in patients with artificial pacemakers. *British Heart Journal*, **26**, 737-746.
- Requests for reprints to Dr M. R. Hetzel, Department of Thoracic Medicine, St. James' Hospital, Sarsfeld Road, London SW12 8HW.