The Birmingham (Lucas) pacemaker *A follow-up with particular reference to dependence and parasystole*

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The results of 58 patients who left hospital having been paced by the Birmingham (Lucas) system are described. This system is asynchronous and has an external generator. Reversion to sinus rhythm or lesser degrees of heart block was seen in many patients. Nevertheless, pacemaker dependence was present at least some of the time in 54 per cent of patients. This dependence was related to Adams-Stokes attacks sustained after pacemaker insertion but was not related to death. Parasystole occurred in 18 patients, of whom 5 are dead. There appears to be no relation between parasystole and death. Coil separation measurements were made at pacemaker insertion and at review. When reviewed after discharge from hospital, effective pacing required a closer approximation of the coils than was needed at insertion of the system, but such measurement was of no value in predicting future pacemaker failure.

1) A system using external controls and leads from them exposes the patient to an increased risk of Adams-Stokes attacks after pacemaking. This disadvantage must be offset against the lesser need for reoperation than with totally implanted units.

2) Parasystole in patients with chronic heart block who have left hospital after pacemaker insertion is not as dangerous as previously considered.

Use of the Birmingham (Lucas) type inductive pacemaker for the treatment of chronic heart block was first described by Abrams, Hudson, and Lightwood (1960). Its merits and disadvantages as compared with the more widely used totally implanted systems have been discussed by Pilcher and Healy (1971). Unlike totally implanted systems the generator is external. This difference allows the study of pacemaker dependence. The paper describes the results of follow-up of patients leaving hospital having been paced by the Lucas system, particular attention having been paid to pacemaker dependence, parasystole, and death.

Description of unit

Current is produced from an external generator powered by a U2 torch battery. This generator has two power settings and is normally always used on low power. The lead to the primary (external) coil is connected to the generator by a push-pull fit. The external coil overlies the subcutaneously implanted secondary (internal) coil, energy being induced across the intact skin. In-

ternal coils are of two types: either they are provided with two electrodes (for epicardial implantation) or with two short leads (for catheter connexion).

Pulse width delivered by the system is one millisecond, but this can be altered by changing the number of turns in the external coil. Voltage wave-form is in the shape of a biphasic sine wave. Delivered voltage is determined by two factors: the position of the low-high power switch on the generator and the lateral or vertical separation of the two coils. At one centimetre coil separation and on low power, peak voltage delivered into a 180 Ohm load is approximately 8 volts. This voltage is doubled on high power. The system is asynchronous on either power.

Patients and methods

There were 58 patients who were seen by us between 1968 and 1971. Of these, 27 were women and 31 were men whose mean age was 66.2 years at pacemaker insertion (range 34 to 86). Of these patients, 26 were paced by thoracotomy and epicardial wires. Thirty-four patients have been given endocardial catheter systems, this figure including 2 patients in whom the original epicardial pacemaker failed. Patients with endocardial systems had a record made at operation

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of the pacing threshold (volts r.m.s.) and of the maximum permissible vertical coil separation before skin closure.

Fifty-seven patients have been seen at least twice since discharge from hospital after pacemaker insertion. One patient died after a single visit. At review patients were asked if they had had 'blackouts' since insertion of the equipment. They were then tested for the following.

a) *Pacemaker dependence* This was determined by an electrocardiographic tracing run for 5 seconds with the pacemaker off, this time being considered to be the limit of safety. Should no independent ventricular systole appear within this time patients were classed as pacemaker dependent. If not pacemaker-dependent rhythm was determined by a longer time with the pacemaker switched off and this rhythm compared with that before pacemaker insertion.

b) *Parasystole* This was determined by an electrocardiographic tracing run for 30 seconds with the pacemaker on. (This test preceded that for pacemaker dependence.)

c) *Maximum permissible vertical coil separation before pacing ceased*—skin thickness was recorded subsequently by calipers.

Patients and their relatives were asked to return external units in case of fault and more particularly should these units be no longer needed. This euphemism did result in the return of most units of the patients who died.

Results

The 58 patients have been paced for a mean of 24.6 months (range 2–59, SD 15.9) until either last seen or until death; 45 patients are still living and 13 are dead. Living patients have been paced a mean of 26.3 months (range 3–59) and dead patients 18.7 months (range 2–57). Of the 13 dead patients, 9 died at home, 7 deaths being sudden and 2 from unrelated causes. Pacemaker units were returned for 6 of these patients. In 5 the external unit worn at death was functioning normally, but in one it had failed (open-circuit). Four patients died in hospital. These deaths were caused by exit block proceeding to asystole once, wire fracture once, congestive cardiac failure once, and probable internal unit failure (exact nature unknown) in the remaining patient.

'Blackouts' since insertion Eighteen patients stated that they had had further blackouts since pacemaker insertion. It is assumed that these were Adams-Stokes in type since all 18 had sustained such attacks before being paced. In 6 patients these 'blackouts' were caused by internal unit failure and reoperation was required. In the remaining 12 the fault was in the external equipment, the commonest cause being breakage or displacement

of the lead connecting the external coil to the pulse generator. In general these 'external' blackouts occurred within the first 3 months after patients had left hospital.

Testing of equipment: (a) pacemaker dependence This has been classed as complete if asystole was observed on each occasion tested, variable if this was observed on some occasions but not others, and non-dependent if never seen. Dependence has then been compared both with Adams-Stokes attacks sustained since pacemaker insertion and with subsequent death (Tables 1 and 2).

TABLE 1 *Adams-Stokes attacks*

<i>Dependence</i>	<i>Adams-Stokes attacks</i>	<i>No. of attacks</i>
Complete	7	10
Variable	8	6
Non-dependent	3	24

There is a significant relation between pacemaker dependence (complete and variable) and subsequent Adams-Stokes attacks ($P < 0.01$).

TABLE 2 *Death*

<i>Dependence</i>	<i>Deaths</i>	<i>Survivors</i>
Complete	4	13
Variable	1	13
Non-dependent	8	19

There is no relation between pacemaker dependence and subsequent death ($P > 0.10$). Finally, heart rhythm at pacemaker insertion has been compared with that at review (Table 3).

TABLE 3 *Heart rhythm at pacemaker insertion and at review*

	<i>At insertion</i>	<i>At review</i>
Sinus rhythm or first-degree block	1	8
Varying second- or third-degree block	6	14
Stable third-degree block (by electrocardiogram)	51	19
Pacemaker dependence (complete)	0	17

In 4 of the 8 patients with sinus rhythm at review this rhythm was unstable, the rhythm varying between sinus and various degrees of heart block.

b) Parasystole This was seen in 18 patients, of whom 5 are dead. By comparison, parasystole was not observed in 40 patients, of whom 8 are dead. Testing by χ^2 does not show parasystole to be associated with death ($P > 0.10$). Mean duration of parasystole (assuming it to have been present since pacemaker insertion) was 21.2 months (range 11–45) in those parasystolic patients who are now dead. By comparison it has been to date 31.2 months (range 6–59) in the 13 parasystolic patients still living. Treatment of parasystole (increasing the rate setting of the pacemaker with or without β -blockade) has been considered necessary in 4 patients. Three of these 4 are still alive and parasystole has been suppressed in 2. In the patient who died parasystole persisted despite treatment for 18 months.

c) Permissible vertical coil separation Sixty-four readings have been made on 48 patients. All readings were made on the low-power setting of the external generator. Mean permissible coil separation (including skin thickness) at review was 2.75 cm (range 1.25–5.5). In 28 patients with endocardial systems it has been possible to make more detailed comparisons. Their permissible coil separation at pacemaker insertion was 5.5 cm (range 3–7.5, SD 0.6) and their mean threshold pacing voltage 1.5 volts r.m.s. (range 0.4–3.0, SD 0.75). There is an inverse association between pacing threshold and coil separation at pacemaker (insertion ($r = 0.45$, $P < 0.05$).

Thirty-seven readings of permissible coil separation have been made at review in these 28 patients. Mean separation at review was 3 cm (range 1.25–5.3, SD 0.5). This distance is significantly less than permissible coil separation at pacemaker insertion ($P < 0.001$).

The 37 readings were taken at varying times from pacemaker insertion (between 2 and 35 months, mean 16.1) and it was wondered whether permissible coil separation had become less as the months went by. Accordingly the time elapsed between insertion and review was compared with the difference in permissible coil separation between these times. There appears to be no relation between duration of pacing and this coil separation difference ($r = 0.19$, $P > 0.10$). Indeed, individual separation figures were relatively constant over the times of observations.

Permissible coil separation was measured, at review, in 4 patients within 6 weeks of their death. The readings were not unusual. Neither were the separation readings unusual in 3 patients obtained within 6 weeks of internal unit failure requiring reoperation (catheter displacement $\times 2$, insulation leakage $\times 1$).

The figures suggest that measurement of permissible coil separation at review is of no prognostic value.

Discussion

This study has shown that pacemaker dependence is present, at least some of the time, in 54 per cent of the patients tested. There is an association between pacemaker dependence and Adams-Stokes attacks after pacemaker insertion, these being sustained by 31 per cent of patients. This figure may seem high but must be considered in the light of the type of pacemaker used. Reoperation was required in 6 patients only of this series (10%) who sustained Adams-Stokes attacks. The other 12 patients of the series had Adams-Stokes attacks as a result of problems with the external unit. Such attacks usually occurred fairly soon after pacemaker insertion and it can be assumed that increasing familiarity with the apparatus is associated with a decreased incidence. However, Adams-Stokes attacks are dangerous, and this is exemplified by the one patient of the series who died as a result of external unit failure. In many ways it is advantageous to have the power source and major electronic components of a pace-making system outside the body where they can be readily replaced. However, accessibility is not entirely advantageous. A particular weakness of the Lucas system is the junction between the lead from the external coil and the pulse generator. This is not difficult to pull out, thus disconnecting the pacemaker from its induction coil. In addition, fractures of the lead at this site, due to repeated flexion, were not infrequent. Faults of this kind have been the major cause of Adams-Stokes attacks in this series. However, the makers are well aware of this and have incorporated an audible warning signal from their earliest models. They have also continuously been introducing other modifications at this point in the equipment to improve the situation. Most recently they have designed a chest-worn external unit without leads to fracture or come loose. We have little experience of this new unit.

The incidence of Adams-Stokes attacks after pacing is not quoted by other authors who have used permanent pacemakers with external controls. Their incidence after insertion of totally implanted units is difficult to determine. Pacemaker failure requiring reoperation is more common with totally implanted units than with the Lucas system (Pilcher and Healy, 1971). Indeed, most implanted pacemakers must be changed within 2½ years (Furman, Escher, and Parker, 1971).

Adams-Stokes attacks are, of course, a manifestation of such pacemaker failure but the purpose of pacemaker clinics is to detect impending failure before attacks occur. Most authors are concerned with pacemaker failure as such and not with its manifestations. However, Donmoyer, DeSanctis, and Austen (1967) reported Adams-Stokes attacks to have occurred in 9 of 44 patients followed for an average of 25 months, an incidence of 10 per cent a year. All these patients required reoperation. Scheppokat *et al.* (1969) reported an incidence of 23 per cent in 421 patients paced for 19.8 months. This represents an incidence of 14 per cent per year but the authors were not certain that Adams-Stokes attacks were the cause of loss of consciousness on each occasion. By contrast, Furman, Escher, and Solomon (1969) reported attacks to have occurred in only 16 of 265 patients followed for more than 3 years, an incidence of less than 2 per cent per year. These figures compare with a combined incidence of 15.5 per cent per year in patients of this series whether or not reoperation was required.

Unfortunately comparison is complicated by the possibility that original indications for pacemaker insertion were not the same between authors. In particular it seems likely that fewer patients of Furman *et al.* had sustained Adams-Stokes attacks before pacemaker insertion than had those of this series. This is shown by comparison of reoperation figures. In this series 7 patients required reoperation and attacks were sustained by 6. In the other series reoperation for established pacemaker failure was required by no fewer than 52 patients though attacks were sustained by a mere 16. Nevertheless, it seems that an increased incidence of Adams-Stokes attacks after pacing is a hazard of a system using external controls. This disadvantage must be offset against the lesser need for reoperation.

Though pacemaker dependence was associated with Adams-Stokes attacks it was not associated with survival or death after pacing. It is interesting how variable such pacemaker dependence appeared to be. In no fewer than 14 patients (i.e. 24% of the series) did pacemaker dependence appear to be complete at one visit, only to be found not to be present at another. This difference was not related to the time such patients had been paced. Three other patients, in whom pacemaker dependence was never observed, sustained Adams-Stokes attacks.

Measurement of pacemaker dependence has confirmed the frequency with which patients with heart block revert either to sinus rhythm or to lesser degrees of block after being paced.

The figures are in agreement with those quoted by Chardack *et al.* (1969) who state that a return to sinus or a competitive rhythm is to be expected in some 20 to 50 per cent of patients long-term paced by any system.

This reversion of paced patients to sinus rhythm or to lesser degrees of block is one of the problems of long-term pacemaking. To discontinue pacing because of reversion to a rhythm that appears to provide an adequate ventricular rate is not safe. Such patients must continue to be paced. However, these patients then become at risk from competition between their own and their artificial pacemaker. This has been thought to be dangerous because of electrical impulses falling into the vulnerable period of diastole (Sowton, 1965).

However, the situation is complex. The current strength required to induce ventricular fibrillation in normal myocardium is about 20 times greater than that required to induce pacemaking (Hoffman and Crane, 1964). In general, current strengths required to induce ventricular fibrillation in normal hearts by brief stimuli are beyond the output capabilities of implantable pacemakers. The Lucas pacemaker on low power is no exception. However, it is likely that the myocardium is not normal in patients with heart block, and it is known that many factors can reduce this safety ratio between fibrillation and pacing thresholds. In particular, it is reduced after coronary occlusion (Wiggers, Wegria, and Pinera, 1940).

This concept led to the evolution of the second-generation pacemakers in which competition was avoided by some or other form of sensing device preventing the pacemaker discharging into the vulnerable period. Their use has been strongly advocated by some authors (e.g. Bilitch, 1969) for use in all forms of heart block. Unfortunately such pacemakers are not only more expensive but have more circuitry to fail. In the acute heart block of myocardial infarction, in which the safety factor between pacemaking and fibrillation thresholds is low, in which the likely need for pacing is short-term, and in which the external generator and sensing device are external (and thus readily replaced), there is no doubt but that this so-called demand pacing is greatly preferable to asynchronous pacing. For long-term pacing the situation is not the same and opinion is not unanimous about how this should best be managed.

Proponents of demand pacing for chronic heart block (e.g. Sowton, 1965; Bilitch, 1969) point out the difference in arrhythmic mortality between patients paced by demand and by asynchronous systems. However, this

mortality difference was in the acute post-operative period during which many of the factors known to lower the fibrillation threshold are operative. The patients of this series did not show an increased mortality if parasystolic, but it will have been noted that they were studied after they had left hospital by which time such provocative factors had returned to normal. A further factor increasing safety is the rise in pacing threshold known to occur two to three weeks after continuous pacing and due to deposition of fibrous tissue around the electrode. This rise in pacing threshold has been indirectly confirmed by this study. Pacing threshold was inversely related to permissible coil separation, and this separation was noted to be less at review than at insertion, thereby indicating a raised threshold. Deposition of such fibrous tissue will diminish electrical energy reaching the myocardium.

It seems that there may be a case for demand pacing to cover the acute postoperative period after pacemaker insertion for chronic heart block. After this period is over the safety ratio between thresholds for pacing and ventricular fibrillation increases. The results of this study suggest that asynchronous pacing is then no longer dangerous despite parasystole.

This is in agreement with the conclusions of Furman *et al.* (1969) and also with those of Siddons and Sowton (1967) who state that, 'Our current view is that there is very little danger from pacemaker-induced ventricular fibrillation in patients with sinus rhythm treated with most commercially available pacemakers and matching electrodes'.

These conclusions are given support by patients of this study exhibiting pacemaker dependence. If parasystole were dangerous it might be expected that pacemaker dependence – in which parasystole presumably does not occur – might protect against death. This it manifestly did not do – indeed, pacemaker dependence did not appear to be a factor of any prognostic importance with regard to death or survival. More importantly, extensive long-term follow-up studies are now available on patients paced with asynchronous pacemakers (Chardack *et al.*, 1969) and show that the life expectancy of such patients comes close to that of a normal population of similar age. Since a return to sinus rhythm or lesser

degree of heart block occurs in a considerable proportion of these patients, a substantial unexpected mortality should now be evident if parasystole were a real hazard – particularly since it has been calculated that during electrical parasystole approximately 3000 stimuli will fall into the vulnerable period over each 24 hours (Sowton, 1965). This unexpected mortality has not been observed.

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References

- Abrams, L. D., Hudson, W. A., and Lightwood, R. (1960). A surgical approach to the management of heart-block using an inductive coupled artificial cardiac pacemaker. *Lancet*, **1**, 1372.
- Bilitch, M. (1969). Ventricular fibrillation and pacing. *Annals of the New York Academy of Sciences*, **167**, art. 2, 934.
- Chardack, W. M., Ishikawa, H., Fochler, F. J., Souther, S., and Gage, A. A. (1969). Pacing and ventricular fibrillation. *Annals of the New York Academy of Sciences*, **167**, art. 2, 919.
- Donmoyer, T. L., DeSanctis, R. W., and Austen, W. G. (1967). Experience with implantable pacemakers using myocardial electrodes in the management of heart block. *Annals of Thoracic Surgery*, **3**, 218.
- Furman, S., Escher, D. J. W., and Parker, B. (1971). The failure of triggered pacemakers. *American Heart Journal*, **82**, 28.
- Furman, S., Escher, D. J. W., and Solomon, N. (1969). Experiences with myocardial and transvenous implanted cardiac pacemakers. *American Journal of Cardiology*, **23**, 66.
- Hoffman, B. F., and Cranefield, P. F. (1964). The physiological basis of cardiac arrhythmias. *American Journal of Medicine*, **37**, 670.
- Pilcher, J., and Healy, M. K. (1971). The Birmingham (Lucas) pacemaker: an appraisal of its use. *British Heart Journal*, **33**, 375.
- Scheppokat, K. D., Bantz, P. M., Giebel, O., Hauch, H. H., Kalmar, P., Kirsch, U., Rodewald, G., Saborowski, F., Tilsner, V., Voss, H., and Westermann, K. W. (1969). Comparison of asynchronous and demand pacing. *Annals of the New York Academy of Sciences*, **167**, art. 2, 968.
- Siddons, H., and Sowton, E. (1967). *Cardiac Pacemakers*, p. 112. Charles C. Thomas, Springfield, Illinois.
- Sowton, E. (1965). Artificial pacemaking and sinus rhythm. *British Heart Journal*, **27**, 311.
- Wiggers, C. J., Wegria, R., and Pinera, B. (1940). The effects of myocardial ischemia on the fibrillation threshold. *American Journal of Physiology*, **131**, 309.

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