LONG-TERM ARTIFICIAL CARDIAC PACING: EXPERIENCE IN ADULTS WITH HEART BLOCK

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by

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ATRIO-VENTRICULAR DISSOCIATION, or complete heart block, occurs in three groups of patients:

(1) The newborn as a congenital defect,
(2) as a result of operations for congenital cardiac defects, and
(3) in the adult as an acquired phenomenon.

It is with this last group that I am concerned, though the techniques we use are also applicable to the second group. The ventricle when independent of the atrium contracts at its inherent rate, usually from 25 to 45 beats a minute, and this slow rate may result in cardiac failure. Only limited physical activity is possible and even this may be associated with dizziness and transient unconsciousness. Attacks of unconsciousness in patients with heart block, however, are more commonly due to periods of asystole, or to various types of ineffective ventricular activity. For some years it has been realized that a suitable electrical impulse brought to the ventricular muscle in these periods of arrest will result in an effective beat, and that a slow heart can be speeded up to a more efficient rate by stimulation at suitable intervals. This lecture is concerned with the development of a suitable apparatus and technique for achieving such cardiac pacing on a long-term basis.

The idea of the use of electricity in the treatment of syncope is not new. As early as 1819 Aldini, in a lecture to the Royal Humane Society, after describing various experiments, said, " These considerations lead me to recommend the administration of galvanism in cases of syncope ". It appears to have been first attempted by Duchenne of Boulogne in 1870 for a patient with a slow pulse in an attack of diphtheria. With one electrode on the patient's skin he held the other electrode in his hand and passed it rhythmically over the praecordium to avert cardiac arrest. He claimed success though the patient died a few weeks later. In 1889, McWilliam reported experiments on isolated cats' hearts from which he concluded that direct excitation of the heart would be beneficial for a heart suddenly arrested in diastole caused by a disease of a transient nature. The arrested heart in an intact animal was successfully stimulated into action by Hyman in 1932, using two myocardial needle electrodes in dogs anaesthetized to the stage of cardiac arrest. It was not until 1952 that the

technique was first used with success in a patient when Zoll, using a subcutaneous needle as an electrode near the apex of the heart, resuscitated a patient in cardiac arrest.

This basic work and his subsequent successes led my medical colleague at St. George's Hospital, Dr. Leatham, and myself to make a special study of this subject. In reporting our experience it must be emphasized that the patients with heart block referred to us for possible electrical pacing had been carefully selected and cannot be taken as representative of all cases with the condition. We have, however, learnt much of interest from the 26 patients whom we have attempted to pace by various techniques. Details of these cases are set out in Table I.

Aetiology of heart block

Though it is customary to attribute most cases of acquired heart block to ischaemia of the conducting tissue as a result of coronary atherosclerosis with or without hypertension, this widespread teaching requires careful appraisal. Complete heart block does occur occasionally in cardiac infarction, but only in serious cases and often as a terminal event. The patients who have been referred to us for pacing, on the contrary, very rarely give any history suggestive of cardiac infarction. In only four of our 26 patients did we obtain a history of anginal pain. Their electrocardiographic tracings were, owing to the block, difficult to interpret, but in 17 of them there was nothing to suggest cardiac ischaemia, and in only a few of the remainder was there firm evidence of it.

Some had high systolic and very wide pulse pressures with their exceptionally slow pulse rates. Once we had paced such patients at normal rates the blood pressure often reverted to normal; in only three did the diastolic pressure remain over 100 mm. Hg.

It has been recognized for many years that calcific aortic valve disease may be associated with heart block, and it was present in two of our cases. Only one patient gave a history of rheumatic fever, but it may be significant that four had had diphtheria even though the heart block did not date from the diphtheritic attack.

In 13 of the 26 cases the aetiology was obscure, there being no hypertension, no history of cardiac pain, no electrocardiographic changes suggestive of ischaemia, no associated valve disease, and no history of rheumatic fever or of diphtheria.

Four of these 13 patients with heart block of unknown aetiology have died, and we have been fortunate in being able to examine fully at autopsy the hearts of three of them. My colleague, Dr. Dexter, has been especially interested in studying coronary arteries by means of injections of radio-opaque substance (Crawford *et al.*, 1961), but in spite of examining these hearts by this injection technique, as well as by routine macroscopic and

TAB

DETAILS OF 26 PATIENTS ON

No.	Age	Sex	Aetiology, etc. (footnote)	E.C.G. evidence ischaemia	Attacks unconsciousness	Heart failure	Rhythm during attack (footnote)
1	65	M		+	+	–	A
2	78	M	D	–	+	+	A
3	68	F	S	–	+	+	A
4	62	M		–	+	–	
5	74	F		–	+	+	
6	55	F	D	–	+	–	A
7	74	F		+	+	–	VT
8	66	F		+	+	–	A & VT
9	36	M		–	+	–	A & VT
10	70	F		+	+	+	A
11	76	F	HDA	+	–	+	
12	58	M		+	+	+	
13	55	M		–	+	–	A
14	27	F	DA	–	+	–	VT
15	76	F		–	+	+	VT
16	60	M		–	+	–	A
17	66	F		–	+	–	VT
18	56	M	H	–	+	–	
19	72	M	A	+	+	–	
20	66	M		–	+	–	
21	58	F		–	+	–	
22	54	M		–	+	+	A
23	66	M		–	+	+	A

LONG TERM PACING
Cases Nos. 1 and 7

1	65	M		+	+	–	A
7	74	F		+	+	–	VT
A	58	M	S	+	+	+	A
B	81	M	HA	–	+	+	
C	76	M		+	+	–	

Foctnotes: Aetiology, etc.: Key—D=diphtheria. A=angina. H=hypertension. S=aortic stenosis.
Rhythm during attack: Key—A=asystole. VT=Ventricular tachycardia.

LE 1

WHOM PACING WAS ATTEMPTED

Date implant	*Pacemaker type: months used (footnote)*		*Complications, etc.*	*S = Satisfactory pacing now D = Death*
31st March 1960	Sw	10 months	Pacemaker failure.	
6th February 1961	E, I	11 months	Sepsis around cardiac electrode. Wire fracture.	
21st December 1961	V	9 months	(see also below)	S
24th October 1960	E	23 months		S
30th January 1961	I	7 days	Death in V.F. 8th day	D
20th March 1961	E	17 months	Wire fracture repaired.	S
27th April 1961	E	11 days	Death. Renal failure Emboli. 12th day.	D
22nd June 1961	E	14 months	Type changed at patient's request.	S
16th August 1962	M	1 month	Threshold rise	
6th July 1961	V	3 weeks	Septicaemia.	S
	E	14 months	(See also below)	
2nd November 1961	M	5 months	Wire + connection	
9th April 1962	M	6 months	failures. Electrolysis from pacemaker fault.	S
14th December 1961	M, I	1 month	Connection failure repaired. Pacemaker failure—death.	D
21st December 1961	M	9 months	Hemiplegia at 7 months	S
18th January 1962	M	9 months	Connection repair at 3 months. Recurrence heart failure.	S
21st February 1962	M	2 weeks	Pacemaker fault	
	E	6 months		S
15th March 1962	2M	2 days	Sudden death in V.F.	D
12th April 1962	M	5 months		S
19th April 1962	Dble M	5 months	Connection failure repaired.	S
3rd May 1962	M	5 months		S
17th May 1962	M	4 months		S
29th May 1962	M	4 months		S
31st May 1962	M	2 days	Sudden death.	D
12th July 1962	M	2 months		S
19th July 1962	M	2 months	Threshold rise.	S
2nd August 1962	M	1 month		S
21st August 1962	M	1 month	Threshold rise.	S

INTRAVENOUS ROUTE
see also above

21st December 1961	9 months		
18th June 1961	3 weeks	Septicaemia: cured by antibiotics. Paced other route.	
10th November 1962	3 weeks	Septicaemia: cured by antibiotics. Died 10 weeks after pacing. Infarct + emboli.	D
13th June 1962	4 months		S
14th July 1962	3 months		S

Pacemaker type: Key—E=external wires. V=intravenous. Sw=Swedish chargeable. I=induction type. M=mercury cell implanted (various models).

microscopic methods, we failed to find any evidence of ischaemia of the conducting bundle or surrounding tissue. Indeed the coronary vessels appeared to be remarkably good (Fig. 1).

To summarize, although we are unable to put forward any alternative aetiology, we feel it unjustifiable to assume that in the absence of any apparent cause the conduction defect is due to ischaemia. The absence of

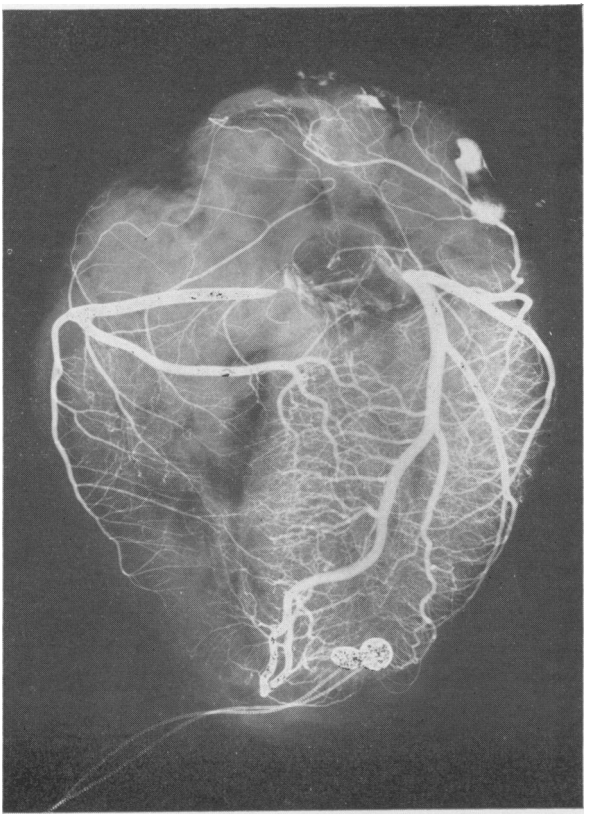

Fig. 1. Coronary arteries injected. Elmqvist electrodes on ventricle.

demonstrable cardiac disease in half the patients is extremely encouraging as the prognosis should be good if the heart can be satisfactorily paced.

Clinical features of heart block

Attacks of unconsciousness in heart block follow a fairly clear pattern. Before the onset there may be a momentary awareness that something is going to happen. No pulse can be felt and the pupils dilate widely. Convulsive movements may occur in the more severe cases. With return of the heart beat consciousness is regained immediately, and is usually

associated with flushing. A patient in bed may be unaware of the incident excepting for the flushing.

In some the attacks are taken for epilepsy, and one of our patients (Case 13) had been treated for four years with anti-convulsants before the true nature of his illness was realized. This difficulty does not arise when the heart block persists between attacks, the slow pulse at once suggesting the diagnosis. In the rarer case not showing persistent heart block between attacks diagnosis is much more difficult unless an attack is observed.

We have watched the electrocardiograph during a number of attacks. Complete ventricular asystole is the commonest finding. Multiple ventricular extrasystoles, sometimes amounting to ventricular tachycardia, were also seen, and in at least one patient transient ventricular fibrillation.

Treatment

My medical colleagues and I have reported more fully elsewhere (Portal *et al.*, 1962) on the short-term management of these cases; briefly, medical treatment consists of routine measures for heart failure and the use of drugs which may increase the heart rate, particularly sympatho-mimetic compounds like ephedrine, adrenaline and isoprenaline. Atropine, procaineamide, intravenous molar lactate and steroids have also been used. If these measures fail to control heart failure and to prevent attacks of unconsciousness, or if the attacks are rapidly repeated and threaten life, then we resort to artificial pacing.

Pacemaker methods

With the exception of a Swedish type (Elmqvist and Senning, 1960) used in two patients, the artificial pacemakers used by us have all been designed at St. George's Hospital by my technician colleague, Mr. J. G. Davies. In the course of development we have employed a considerable number of quite different methods, and now have accumulated some evidence on which to judge their relative merits.

EXTERNAL PACEMAKER

Skin electrodes
Needle to heart
Intravenous wire to right ventricle
Wires through skin to ventricular muscle

INTERNAL PACEMAKER

Induction with power externally
Cells chargeable by induction } No external
Mercury powered cells } apparatus.

In our first attempt at pacing, in 1955, we used electrodes planted on the skin of the chest. We still use this method in extreme emergency but, as

150–200 volts are required when given through the intact skin, stimuli result in strong painful twitches of the pectoral and other muscles (Fig. 2). Pain can be controlled adequately by morphia or pethidine for a few hours whilst pacing by the intravenous method, as described below, is instituted. For urgent pacing some have used needles passed through the intact skin into the myocardium (Ross and Hawkins, 1959); this has the advantage of allowing a much smaller current to be used than when pacing through the intact skin, but we have not tried this technique, as experience in other conditions has shown us that there is a serious risk of the needle damaging the heart and producing haemopericardium and tamponade.

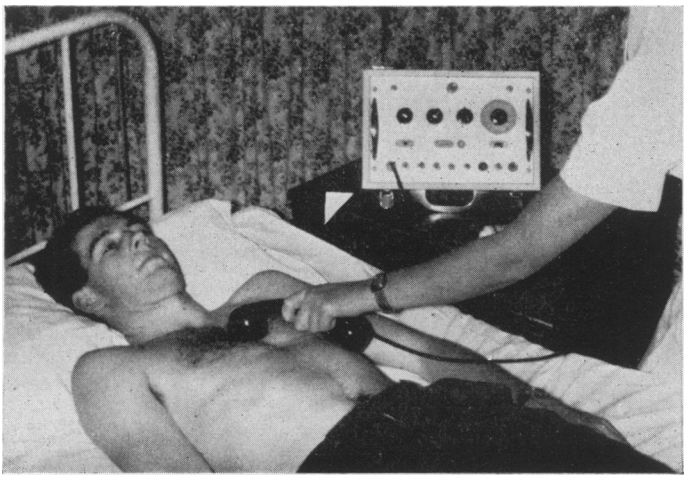

Fig. 2. Demonstration of external pacing. The electrodes are built into the telephone.

Intravenous method

In less urgent circumstances, we pass an electrode (similar to a cardiac catheter) intravenously to the right ventricle. When the electrode connected to the negative pole of the pacemaker is in contact with the endocardium of the ventricle, only very small currents are required. In none of our 26 cases was the threshold over 2 volts. If, however, the electrode is within the atrium, a voltage of 20 times greater or more is necessary to stimulate the ventricle (Davies *et al.*, 1959). Though it might be hoped that a few hours or days of pacing by this route might tide the patient over a danger period, enabling the pacemaker to be switched off, the circumstances in which such short-term pacing is required do not often arise. We have used such treatment for cardiac failure without attacks of unconsciousness, but continued pacing is usually necessary. If pacing is to be discontinued, one must not switch off the pacemaker abruptly as this may

result in a long period of asystole. If, however, the rate is gradually slowed it can be observed whether the heart will take over adequately.

If the electrode is passed via an arm vein, great care must be taken that the arm is not moved, for in at least one of our cases we have evidence that the rather rigid catheter had penetrated the myocardium, allowing the tip to lie in the pericardial sac. The heart failed to respond to the pacemaker and the catheter had to be urgently withdrawn to the ventricle. Though a small haemopericardium developed, the degree of tamponade was not severe and the patient survived. We have heard of others having this complication, but it is unlikely to occur with the softer catheters which are now in use, namely, No. 5, size C 50 catheters with a platinum tip.*

Schwedel *et al.* (1960) have recommended that the electrode be passed via the external jugular vein rather than the arm vein, and we now use this route as it is less hindrance to the patient, the electrode is less likely to become displaced from the ventricle and the jugular vein seems to be immune from the spreading phlebitis so common with indwelling intravenous catheters in the arm.

For short-term intravenous pacing the positive lead of the pacemaker is connected to a skin plate electrode of the type used for electrocardiograms. For long-term pacing we prefer to insert a subcutaneous electrode in the neck near the entry of the jugular lead, as the skin beneath a plate electrode becomes sore after a time, especially in patients sensitive to the conducting jelly which must be used.

The intravenous route has been used on a long-term basis by others (Furman *et al.*, 1961; Hurwitt, 1961) for as much as 22 months despite the risks of sepsis and thrombo-embolism. Our policy has been to use this method as a preliminary to placing epicardial electrodes by operation, which can then be done in safe conditions without the fear of cardiac arrest, which has been recorded as a serious risk during induction in patients with heart block (Zoll *et al.*, 1961). We have employed pacing by the intravenous route for periods of a few days in all 26 of the cases considered in this paper, in some on more than one occasion. In five of them we considered the risk of operating to place epicardial electrodes to be great, and elected to try longer-term intravenous pacing. In one of these five patients sepsis had already tracked down an external wire involving the pericardium, so that he could no longer be paced with epicardial electrodes. The other four were of advanced age or in very poor general condition. Two developed staphylococcal septicaemia after about three weeks of intravenous pacing. Antibiotics and the removal of the electrode cleared the infection, and one has been paced since by alternative methods. The other died two months later without further pacing. In the other three

* Supplied by the United States Catheter Corporation as Pacemaker Catheters.

patients we have used anti-coagulants but not continuous antibiotic cover and are still successfully pacing them after nine, four and three months respectively.

Epicardial electrodes with wires through the skin to an external pacemaker. Weirich *et al.* (1958) developed this technique and used it clinically, their patients being those in whom the conducting bundle had been damaged

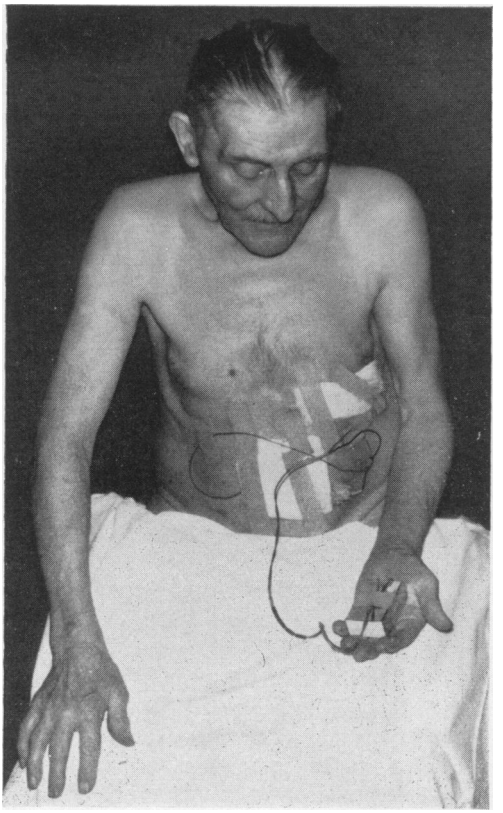

Fig. 3. Patient holding a transistorized unit in his hand.

during open heart surgery. It has since been used by various workers for periods up to about six months (Friese and Dittmar, 1961). The threshold stimulus required for pacing in this way is usually 3 or 4 volts, which is greater than that required by the intravenous route. Using this technique we have paced seven patients; six of them for more than a month and these six are alive. However, sepsis interfered with pacing in two of them; in one the use of a spare lead implanted at the initial operation has enabled it to be continued for 18 months, and in the other after 11

months with external wires we have had to resort to the intravenous route. The remaining four patients have been paced satisfactorily without sepsis for 23, 15, 13 and 7 months respectively. The seventh patient, who died, will be referred to below.

The apparatus we use for pacing with external wires passed either by a vein or through the skin direct to the heart is of two types. At first a battery-powered box capable of giving up to 20 volts incorporating a control for altering the pulse rate is used. It is essential that such apparatus be run on batteries, thus avoiding the risk of electrocuting the patient from the mains (Schwedel *et al.*, 1960; Noordijk *et al.*, 1961; Pengelly and Klassen, 1961; Mody and Richings, 1962). This apparatus is approximately 6 in. × 6 in. × 2 in. and is carried about by the patient. It is, however, inconveniently large, and once the threshold voltage required for an individual patient is determined, we make up a transistorized unit, which is about the size of a packet of cigarettes (Fig. 3). It contains batteries with a life of many months and is set to give impulses at a fixed rate, though it is possible to alter this by a minor adjustment.

The day when it becomes practical to pick up the P wave from the atrium and trigger from this a stimulus to the ventricle may not be far distant. This has been achieved in animals (Battye and Weale, 1960; Folkman and Watkins, 1958; Stephenson *et al.*, 1959), but necessitates an apparatus of considerable size and complexity, which must be capable of meeting various eventualities such as auricular fibrillation, flutter or asystole, and be able to distinguish the auricular complex from its own and from the ventricular complex. We have not worked on these lines, wishing to develop first an efficient technique using artificial rates.

Internal pacemakers

To avoid the risk of sepsis tracking down a wire, which is inherent in any method with an external pacemaker, we turned to internal pacemakers. Of the three main types we first used *Elmqvist's*, which is buried in the tissues and derives its electrical power from a cadmium-nickel cell which could be charged by induction from outside at intervals of one to two weeks. Senning (1959) has reported its use and we also have implanted two of these pacemakers. One of them worked successfully for 11 months and then developed an electrical fault. The other failed to pace the heart after 22 hours, as a result of a defect in the material covering the pacemaker which allowed tissue fluid to penetrate. Others have had similar difficulties. We therefore sought to achieve our aim by a different technique, that of inducing a current from without into an implanted receiver.

Induction pacemaker with external power. Pacing by this principle has been successfully achieved independently at three centres—by Glenn *et al.* (1959) in the U.S.A., by Abrams *et al.* (1960) in Birmingham, as well as at St. George's.

In the method used by Glenn *et al.* (1959) and Eisenberg *et al.* (1961), a series of radio frequency oscillations are transmitted between two coils placed near to each other in such a way that the centres of the coils are coincidental. These pulses are then rectified by an implanted unit to give monophasic square waves which are fed to the heart by wires. The system that we have used is similar in principle except that the two coils are arranged to be approximately on the same plane with the transmitting coil surrounding the receiver. This is achieved by having one coil around the waist of the patient and the receiver placed in the rectus sheath. Another approach to the problem, reported by Abrams, is to use two coils centred closely over each other as in Glenn's method, but to pass a large current into the transmitting coil at a physiological heart rate. This produces a damped oscillating current in the secondary coil and it is this signal that is

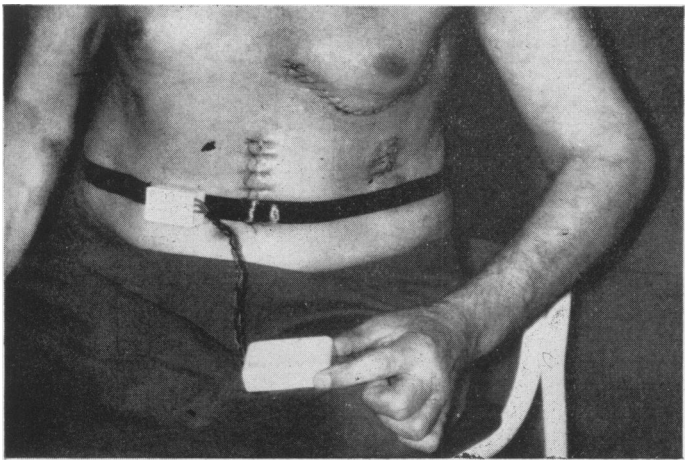

Fig. 4. Transistor transmitter via black belt to implanted induction pacemaker.

fed to the heart by wires. All three methods give control over rate and voltage, but the electrical pulse duration is varied only in Glenn's and our own techniques.

We have successfully paced a few patients by induction for up to three months, with only minor difficulties concerned with maintaining the transmission belt in the correct relationship with the receiver within the rectus sheath (Fig. 4). However, psychologically the arrangement is not ideal, as patients find it hard to forget their condition and are apt to keep on fingering the small transmitter and belt.

Internal pacemaker powered by mercury cells. In order to rid the patient of any external apparatus we turned to a completely indwelling transistorized apparatus powered by its own mercury cells, and a pacemaker of this type we now use as our method of choice. Including our

own model we know of five mercury cell type internal pacemakers available commercially. Table II gives some particulars about them. The most important factor in determining the size and weight of the various models is the number of mercury cells used, since the rest of the components can be miniaturized to take up only a fraction of the unit. Battery life cannot be foretold, but 3–5 years is anticipated as a reasonable estimate for all of the five models, which are so designed that a change in the fixed pulse rate warns of impending battery failure. When this occurs the whole pacemaker is replaced by a relatively minor procedure. The G.E.C. model differs from the other four in that it can be speeded up by an induction method from outside. In the course of developing our model we have tried remote control and have implanted pacemakers which could be speeded up, slowed down, stopped and started. Although these pacemakers have been no bigger than our present model we are not using these more complicated devices because we wish to keep the unit down to the simplest possible workable form, and because we have found so little

TABLE II

MERCURY CELL PACEMAKERS AVAILABLE COMMERCIALLY

	Manufacturer	*Country*	*Volume*	*Weight*	
			c.c.	*gm.*	
Elmqvist	Elema	Sweden	66	170	Constant voltage
Zoll	Electrodyne	U.S.A.	66	170	Constant current
Chardack	Medtronic	U.S.A.	32	125	Constant current
Kantrowitz	G.E.C.	U.S.A.	60	140	Constant power
Siddons	Devices	G.B.	40	95	Constant voltage

clinical use for controls. The fixed pulse rate does not seem to handicap our patients. The G.E.C. model on being speeded up has a smaller impulse output, and it is suggested that this could be used as a means of testing whether a patient's threshold requirement is rising and approaching the pacemaker's output. There are now a number of clinical reports regarding mercury-celled units manufactured in the U.S.A. (Chardack *et al.*, 1960; Lillehei *et al.*, 1962; Simpson *et al.*, 1962; Zoll *et al.*, 1961), and our experience with our design of this type now covers 14 patients. It is clear from the reports of others and from our own experience that there are a number of practical difficulties yet to be surmounted.

Rising threshold

Several workers in this field (Lillehei *et al.*, 1962; Zoll *et al.*, 1961) have recorded that their greatest practical difficulty in achieving prolonged pacing with myocardial or epicardial electrodes has come from the rising threshold of current required to produce a heart beat. This phenomenon has not been recorded using the intravenous route with an endocardial electrode. Up to a month ago we appeared to have avoided this difficulty except in the two patients mentioned above in whom sepsis tracked down the wire from the skin to the epicardial electrode. In the five patients who had wires coming externally we were able to measure this threshold from

time to time, and any rise has always been explicable by sepsis or a broken wire (Fig. 5 and Table III). In patients with internal pacemakers it is not possible to measure the threshold, and in the first ten cases using our standard mercury-celled pacemaker, we had no failure unless a gross electrical fault developed. However, in three of the last four patients, after a few weeks of pacing, the heart has intermittently failed to follow the pacemaker, and we have some evidence that this is due to a rise in threshold. We are at present gathering evidence to explain these failures, and this

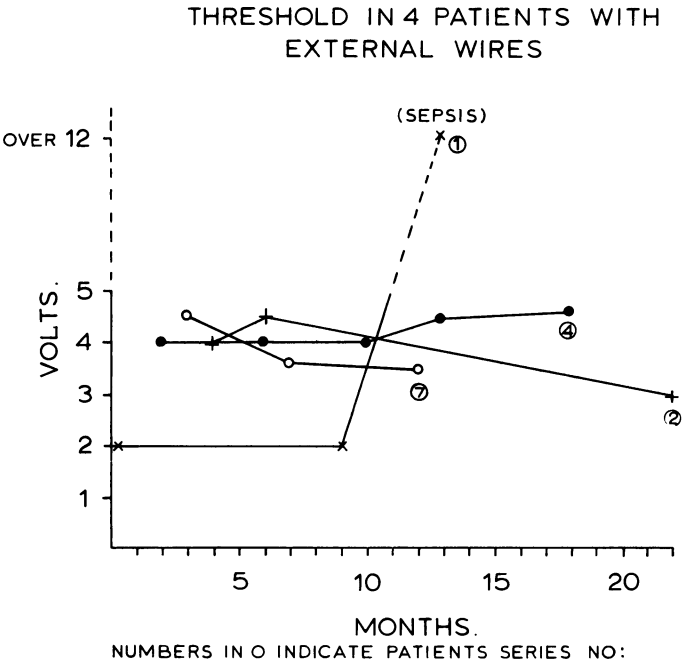

Fig. 5.

seems to incriminate an unusually gross inflammatory reaction around the electrodes, which may be due to bacterial contamination or to irritant materials.

As our pacemaker is of the constant voltage type, it is possible to increase the current output by increasing the size of the indifferent electrode, which can be done by a very minor procedure under local anaesthesia. At the time of writing this manoeuvre has enabled us to gain control of the heart beat in all three of these patients. We hope, however, to track down the cause of these three incidents and avoid a complication which might prove very serious.

Electrodes

The stimulating current can be applied either to two electrodes on the ventricle or to one ventricular electrode and a second remote electrode placed anywhere else in the body using the tissues between as a conductor. We have tried both methods and find a single electrode on the heart just as satisfactory as the double cardiac electrode, providing that the negative pole is joined to the cardiac electrode and the positive to the remote electrode. This polarity is important because if these poles are reversed, the voltage required is in some cases six or seven times as great. It has been reported that the use of two cardiac electrodes (Chardack *et al.*, 1960; Hunter *et al.*, 1959; Levowitz *et al.*, 1960) results in lower thresholds. We have not been able to test this out, but the threshold has, with the exception of the cases referred to above, been below the 5 volts which our pacemaker produces, so that any further lowering in threshold is immaterial.

TABLE III

THRESHOLD AND RESISTANCE

Patient No.	*Month*	*Volts*	*Ohms.*
2	4	4.0	400
	22	3.0	200
4	10	4.0	140
	13	4.5	156
6	13	3.0	440
7	7	3.6	118
	12	3.5	176
12	1	2.5	375

These figures show that threshold is not entirely dependent on resistance.

It is desirable to have the electrode and the wire leading to it as one unit devoid of any join and thus the wire material itself must be suitable as an electrode. Since with a heart rate of 70 the wire leading to the heart is subjected to a flexing movement 38 million times a year the liability to fatigue fracture is great. We have selected a wire wound in a loose helical spiral as the best design to stand up to this, and in our present model the cardiac wire is looped back on itself with the middle portion free of insulation to act as electrode. This produces, in effect, two wires with half the electrical impedance of a single wire, and does not increase the surface area of the electrode, which would otherwise be larger and thus demand greater voltage to produce the required current density. The stainless steel (EN 58 J) which we use at present has proved acceptable so far, but there are theoretical reasons for believing that platinum or some platinum alloys might be better, providing sufficient safety against fracture, and having suitable electrical properties, especially in respect of resistance, electrolysis and polarization.

Our pacemakers have been designed to put out a current in a wave form which will create as little electrolysis as possible. On the only occasion in which there has been trouble from electrolysis or polarization, it was found to be due to a component failure in the pacemaker, one of the only two component failures we have encountered in pacemakers of our own design.

Our remote or tissue electrode is also a stainless steel wire, but shorter and more robust, insulated for the first few inches from the pacemaker so that its bare area can be placed at a site selected with a view to avoiding skeletal muscle twitching at the site of implantation.

Pacemaker details

The internal pacemaker which we now use is approximately 5 × 4 × 2 cm. weighing 95 gm. or 3½ oz., and its electrical and technical details have been described elsewhere by the designer (Davies, 1962).

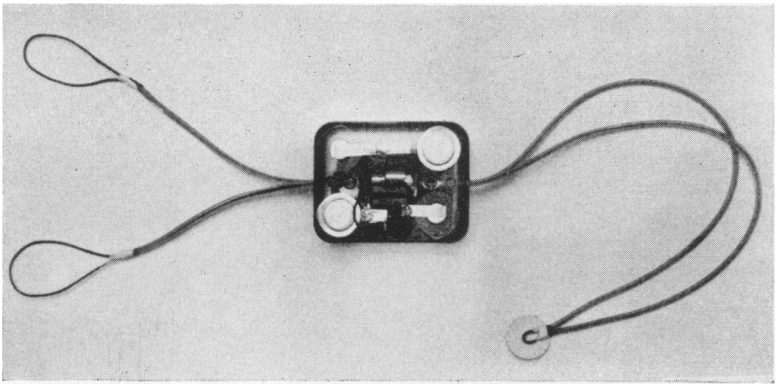

Fig. 6. Photograph of pacemaker.

Almost all the pacemakers we have used have been made up in the physics department of St. George's Hospital, but they are now being manufactured commercially according to our design* (Fig. 6).

The electrical apparatus is " potted " in epoxy resin and though we have tried coating it with other inert plastics this seems to be unnecessary. The myocardial wire leads are insulated with silicone rubber (Dow-Corning RTV 502) and the joints so arranged that in the event of battery failure, or a fault developing, the lead can be unscrewed and reinserted into the new pacemaker. We estimate the current drain is such that the battery life will be three to five years. Silicone rubber insulation has the advantage over polythene of being more flexible and easier to seal at the joins with the pacemaker. The whole apparatus has been sterilized with antiseptics, and to date we have had no proven sepsis from totally implanted apparatus,

* By Messrs. Devices Ltd., Welwyn Garden City, Herts.

though we are investigating the possibility that low grade sepsis is the cause of the increasing threshold in the three recent cases mentioned above. We have not grown pathogenic organisms from them.

Safeguards against electrical failure

Electrical apparatus implanted within the body must be reliable. It may seem surprising that we have had greater difficulty in devising suitable wires and methods of connecting these to the pacemaker than we have had with the pacemaker itself. The most obvious safety measure, which we have adopted with every patient, is to lay, in addition to the double wire between pacemaker and cardiac electrode already described, a spare wire electrode to the myocardium. We also duplicate the remote tissue electrode using two separate wires. All wires must be laid deeply as they tend to ulcerate through the skin unless they are well buried.

Electrical failure within pacemakers of our design has occurred twice and after considering various safety measures to cover such failures we have tried three different methods.

Our first technique was to implant two independent pacemakers, one with mercury cells and as spare a second of the induction type into which we could transmit when required by means of a waistbelt. This patient's pacemaker was one that failed, unfortunately whilst he was at home, and cessation of pacing was followed by sudden death, before the induction method could be started (Case No. 9). This tragedy emphasized the necessity of having an alternative which would take over automatically in the event of failure.

A second safety method was applied in a patient in whom we particularly feared pacemaker failure as he had alarmingly long attacks of unconsciousness prior to initial emergency pacing and it was observed that stopping the pacemaker was always followed by circulatory arrest. The most certain method of guarding against failure of the pacemaker seemed to be to implant two complete units entirely independent of one another. As the two independent pacemakers could not be synchronized an irregular pulse like an atrial fibrillation would result. We had some initial doubts as to the safety of this procedure as experimental animal work by Wiggers and Wegria (1940) had suggested that stimuli reaching the heart in certain phases of the cardiac cycle can create ventricular fibrillation. On the contrary, others (Maaske and Bromberger-Barnes, 1958) hold that this does not apply to a normal heart, and we decided to accept this theoretical risk as we had already paced a number of patients in whom the natural pacemaker competed against the artificial one. In these patients the artificial stimulus had come at any phase in the cardiac cycle and had produced no ill-effects other than the irregular pulse. The patient (Case No. 13) with the two artificial pacemakers did, in fact, die suddenly in ventricular fibrillation on the second post-operative day. This, however, may have borne no relation to the technique of pacing.

Our third method of safeguarding against the effects of electrical failure was to implant two pacemakers connected together in such a way that one or other could work, but not both simultaneously. We have had partial success with this in one patient (Case No. 15), but further work must be done before we can recommend this method as practical.

Routine of treatment

Our practice now is as follows: If the patient requires very urgent pacing owing to attacks of unconsciousness threatening life, we do this with external electrodes on the chest wall. In all other cases, and in those in whom control has been gained by external pacing, we pass an intravenous electrode into the right ventricle and pace by this route until the patient is deemed fit for surgery. In some patients in heart failure intravenous pacing may be required for several weeks before the failure is relieved sufficiently for safe surgery. During this period of intravenous pacing the optimum heart rate for the individual is determined by trial and error.

Operative technique

The heart is exposed by a small left anterior thoracotomy through the bed of the fifth or sixth rib. Electrode sites are selected on either right or left ventricle in areas free from epicardial fat and away from the phrenic nerve, to avoid diaphragmatic twitching. On occasion the phrenic nerve may have to be displaced to avoid this complication. We have not buried the electrode within the cardiac muscle as appears to be the practice of most others. At times we have sewn down patches of silicone rubber overlying the electrode, and similarly when using the platinum disc electrodes designed by Elmqvist we sutured his epoxy resin cap to a patch of nylon mesh which in turn could be easily sutured to the ventricle. It is of interest that the presence of these materials in the pericardium has apparently not interfered with pacing. On one occasion an area of heart muscle which appeared fibrotic had to be avoided as the threshold current required over it was greater than elsewhere. We have also observed this phenomenon with the electrode within the ventricle.

Through a separate abdominal incision the pacemaker is placed in the rectus sheath behind the muscle. This seems to be a suitable site, the wires to the heart being easily led through the diaphragm in preference to bringing them up subcutaneously which would increase the risk already mentioned of ulceration through the skin. The remote or tissue electrode is placed outside the rectus sheath, selecting by trial and error a site where skeletal muscle twitching is not produced. It must be remembered that relaxant drugs used by the anaesthetist may mask twitching.

The spare myocardial electrode is led to a site well away from the heart so that should it have to be picked up later for external stimulation the length of the route to the heart will make it unlikely that sepsis will track down it. The wounds are not drained.

Results

Patients who have very slow pulses show marked clinical improvement on pacing. Several patients were very slow and lethargic prior to operation, spending much of their time dozing and sleeping. The dramatic awakening which occurred directly they were paced at normal heart rates was surprising both to doctor and patient. They immediately became transformed to active beings; and those whom one might have classed as senile became once more capable of work or at least of active life. Patients with established heart failure were much slower to respond, but in every case there was marked improvement on pacing. The patients themselves find the freedom from attacks of unconsciousness the most gratifying of all the results.

Deaths

Six of the 26 cases have died (four of them having been referred to above.) Four were sudden cardiac deaths, of which two occurred in proven ventricular fibrillation on the second and eighth days after operation; a third occurring on the second day was probably similar in mechanism, since shortly before death a bout of ventricular fibrillation had been observed. The fourth occurred as a result of pacemaker failure after 32 days. The fifth death occurred in a patient who three months before had been paced by the intravenous route and who had developed septicaemia. Death in this case was due to a cardiac infarct and not directly related to the previous pacing. The sixth occurred after 11 days' pacing with external wires traversing the skin, in a patient who was, when operated on, in uraemic heart failure (blood urea 117 mg./100 ml.). Death was found to be due to renal failure with evidence of both systemic and pulmonary emboli. We would now pace such a patient intravenously until the general condition was greatly improved before operating.

The remaining 20 patients continue to be paced artificially. The first has now been paced for two-and-a-half years. He, like some of the others, has had his vicissitudes. For the first 11 months he came to hospital for one night each week to have his implanted pacemaker recharged (Siddons and Humphries, 1961). On one visit his pulse was found to be running at 150 instead of 70, and he was therefore paced intravenously whilst the pacemaker was removed and his epicardial lead exteriorized and connected to an external pacemaker. A few months later sepsis tracked down the transcutaneous lead so that the electrode on the heart became useless, and had to be replaced. After a further period a sudden and intermittent increase in threshold occurred, probably as a result of fracture of the wire leading to the heart. After one year and nine months' pacing, the epicardial route had to be abandoned and for the last nine months he has run smoothly with a transjugular endocardial electrode lying in the right ventricle. Other patients fortunately have had much less stormy courses. We now have three (including this first patient) on long-term

intravenous pacing, five with wires from epicardial electrodes to external pacemakers (one of these also has an internal pacemaker), and 12 running on entirely implanted apparatus. We started using our internal model powered by mercury cells only in December 1961, and thus have had only nine months' experience of this method. Various difficulties have been encountered in the 14 patients in whom we have implanted our standard mercury cell type pacemaker. The 14 include one of the sudden deaths just mentioned. In one of the early models we had a component failure, and in another a wire connection fault. In three recent patients pacing became intermittent after a few weeks, and we suspect an inflammatory reaction greater than we have met previously. In spite of these difficulties, 13 of the 14 patients are alive; we have achieved control and are pacing all of them, though in one by resorting to the external wire method. In eight pacing has proceeded without incident for an average of five months.

Looking to the future there is much to learn and develop. The aetiology of half the cases reported is unknown. Such apparently simple matters as making suitable wires and devising methods of connecting them to the pacemaker still offer scope for improvement. Further, since threshold currents of only 1–2 volts are required when the electrode lies on the endocardium, there should be a practical means of using this site without having to bring the lead to the surface with all the attendant disadvantages. We are at present considering using the endocardial electrode with an implanted pacemaker.

Summary and conclusions

Complete heart block with attacks of unconsciousness is not a common condition, but a large proportion of those who do suffer from it have hearts that are capable of years of efficient life if they can be paced. There are available suitable artificial pacemakers reliable enough to be practical and small enough to be embedded in the tissues, though further improvements in both these two aspects are desirable. At present the most suitable source of power appears to be a mercury cell the size of which prevents much further miniaturization of the pacemaker. When a less bulky power source is available a smaller pacemaker will be evolved and this will probably be best placed on the heart itself, so avoiding the use of one source of potential failure, the wires leading to the heart. When such an apparatus is available it may find a wider use than in heart block, since it might prove of value in other more common disorders of rhythm.

Mr. President and members of Council, you will appreciate that this work has been the work of a team. I cannot express my gratitude individually to everyone who has contributed. I wish, however, to acknowledge the technical advice that has come from some organizations outside St. George's Hospital, in particular the Imperial College of Science and Technology, the National Research Development Corporation, and the British Insulated Callender's Cables Company. The cost of the work has been borne by Hunter's hospital, namely St. George's.

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