Causes of Death in Patients with Complete Heart Block and Artificial Pacemakers

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Artificial pacing for chronic heart block has not yet dramatically improved the expectation of life, though the quality of life has undoubtedly been improved. The 1-year survival rate for untreated patients with chronic complete heart block is possibly 60 per cent (Friedberg, Donoso, and Stein, 1964; Johansson, 1966), but an accurate estimation is difficult to determine from the published reports (Siddons and Sowton, 1967). The 1-year survival rate for 108 patients paced at St. George's Hospital is 78 per cent, which is comparable to other centres (Chardack *et al.*, 1965; Elmqvist and Senning, 1960; Nathan *et al.*, 1963; Taylor, 1966; Zoll *et al.*, 1961). In an attempt to find an explanation for this still relatively high mortality (22%), an analysis has been made of the clinical and pathological findings in all patients who came to necropsy following pacing in the past 4 years.

PATIENTS STUDIED

There were 26 patients with the full clinical, post-mortem, and technical data available. Their ages ranged from 53 to 84 years (mean 73) and the indications for pacing were Adams-Stokes attacks in 24 and congestive cardiac failure in 2 others. The methods of pacing were endocardial (Bluestone *et al.*, 1965) in 19 patients (10 with external units and 9 with implanted units) and epicardial (Siddons, 1963) in 7 patients (6 with external units and 1 with an implanted unit). The duration of pacing ranged from a few minutes to 54 months (mean 13.6 months). The units were of the constant voltage type, the no-load voltage being 5 volts, and powered by 3 or 4 mercury batteries giving a square wave impulse of 0.7 to 1 m.sec. at a fixed rate of 75 a minute.

Post-mortem examination has included a special study of the conducting tissue by serial sectioning at 8 μ intervals and multiple blocks have been examined from

all valves and chambers. The coronary arteries were studied by a simple injection technique and serial blocks.

The pacemaker systems were carefully examined for faults in the units, electrodes, and connecting wires.

A number of other patients have died while on artificial pacemakers, but they were excluded from this study because either post-mortem examinations or details of their mode of death were not obtained.

RESULTS

The results are divided into three groups.

Group 1: Technical Failures

There were 11 patients whose deaths could be directly attributed to the method of pacing (Table I). The duration of pacing ranged from 1 day to 54 months (mean 14.3 months).

TABLE I

GROUP 1: TECHNICAL FAILURES—11 PATIENTS

	No.
Displacement of endocardial electrode { Early	4
Late	2
Septicaemia	2
Fractured electrode wire (C.50 No. 5 U.S. Catheter Corporation)	1
Unit failure	2

Six patients died during a recurrence of Adams-Stokes attacks due to displacement of the endocardial electrode resulting in intermittent or a complete failure to pace. In 4 of these patients the endocardial electrode became displaced within 7 days of insertion; in 3 of them the electrode had been inserted into the right ventricle via an arm vein for a period of temporary pacing without careful immobilization of the arm, and the fourth had an endocardial electrode inserted via the external jugular vein and attached to an implanted pace-

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maker for long-term pacing. The other 2 patients died as a result of late displacement of the endocardial electrode. One patient paced satisfactorily for 2 months with a completely implanted endocardial jugular system but died when the electrode became displaced. The displacement resulted from the formation of a loop in the electrode wire from a redundant length of electrode wire left in the right atrium at the time of its insertion. The other patient died after 54 months of satisfactory pacing with an endocardial electrode exteriorized in the neck. During the course of a very serious gastrointestinal infection treated at home the patient's endocardial electrode was pulled out of position and the patient subsequently died from heart failure secondary to a very slow rate.

Two patients died from septicaemia following ulceration of a previously buried loop of electrode wire through the skin with subsequent infection.

Two patients died from a recurrence of Adams-Stokes attacks when their pacemakers failed prematurely. The remaining patient died when pacing abruptly stopped following a fracture of the C.50 No. 5 electrode wire 18 months after insertion. The cardiac histology in these 11 patients is given in Table II and shows that 7 of the 11 patients should have had a good prognosis with pacing since their myocardium was relatively healthy.

Comment. Endocardial pacing from an arm vein is hazardous since displacement from a satisfactory pacing position may occur if the patient moves the arm carrying the transvenous electrode. To reduce this complication the period of temporary pacing via an arm vein should be kept to a minimum and

steps taken to immobilize the arm. A harness has been devised in order to restrain the patient's arm and this has proved satisfactory (Fig. 1).

The late displacement of the endocardial electrode inserted via the external jugular vein occurred due to faulty technique in one patient when a redundant loop of electrode wire was left in the right atrium which later resulted in electrode tip displacement. At the time of insertion of the endocardial wire the patient should be carefully screened in several planes in order that redundant loops should be taken out of the endocardial wire anywhere in its course within the heart (Fig. 2). Confirmation of a satisfactory wedged position of the electrode tip in the right ventricle can be obtained from an electrocardiographic tracing obtained by using the endocardial wire as a cavity lead. An injury pattern, with an increase of the S-T segment of at least 2 to 3 millivolts, is usually obtained when the electrode tip is satisfactorily impacted (Fig. 3).

The other late displacement occurred with an endocardial wire exteriorized in the neck. This method of pacing was used in the early days of endocardial pacing. Although maintenance of pacing in patients with a jugular endocardial wire exteriorized in the neck is relatively easy, displacement of the electrode frequently occurred (Harris *et al.*, 1965) but was reduced by a long subcutaneous course. Displacement has not yet occurred with an entirely buried system provided that pacing during the first 3 days is perfect, indicating a satisfactory site, and no redundant loops of electrode wire are left in the right atrium or ventricle.

Septicaemia occurred in 2 patients following ulceration through the skin of the endocardial wire

FIG. 1.—The patient is being temporarily paced with an endocardial wire inserted via an antecubital fossa vein. During the period of temporary pacing the harness shown restricts the movement of the arm.

FIG. 2.—Lateral chest x-ray film showing the endocardial electrode with implanted axillary pacemaker. The endocardial wire shows a fairly straight course through the heart, free from loops and "wedged" at the apex of the right ventricle.

and infection along the course of the wire in the neck. That this was allowed to occur was a failure of supervision in both these patients. Whenever the area of infection is close to the point of insertion of the endocardial wire into the jugular vein there is a risk of septicaemia, especially if the wire has to be manipulated. It is recommended that the appropriate antibiotics and local treatment should be begun as soon as this occurs and later the system of pacing reimplemented. Once septicaemia has occurred it is almost certain that the endocardial electrode will have to be temporarily removed in order to achieve a cure.

Premature unit failure was responsible for the death of 2 patients. This hazard will always remain, but we hope with the improvement of the design and components, as well as the supervision of the construction of the units, that this will be reduced to a minimum. That only 2 deaths occurred due to unit failure is surprisingly good since more than 300 pacemaker units have been implanted at St. George's Hospital over the past five years.

The problem of pacing failure, due to fractures in the endocardial C.50 No. 5 (U.S. Catheter Corporation) with long-term use in conjunction with an implanted pacemaker (Bluestone *et al.*, 1965), necessitated the development of a more suitable endocardial electrode wire. We no longer use the C.50

FIG. 3.—Endocardial electrocardiographic tracings recorded from the tip of the endocardial wire in the right ventricle. In (A) a large injury current (S-T elevation) is shown when the tip of the electrode wire was in a good "wedged" position. In (B) the "wedged" position was poor and the injury current was not recorded. In (C) contact with the right ventricular wall was intermittent, which results in variations in the height of the injury current. Only "wedged" positions showing the pattern illustrated by tracing (A) are acceptable for long-term endocardial pacing.

No. 5 electrode wire for long-term pacing and a polythene covered stainless steel wire coil with a nylon core and a platinum or stainless steel tip is currently in use, and though more difficult to position than the C.50 No. 5 electrode wire, it is less likely to develop fractures. Experience with the new endocardial electrode has been obtained in 30 patients with a buried system and 21 patients with an external system during the past 9 months, and so far no instance of electrode fracture has occurred.

Group 2: Ventricular Fibrillation While Pacing

There were 8 patients whose death was due to ventricular fibrillation observed during continuous monitoring of the electrocardiogram, with otherwise satisfactory pacing. The duration of pacing ranged from minutes to 36 months (mean 18.3 months). Three patients were being paced with endocardial wires, 2 long-term with the wire inserted via the jugular vein, and 1 temporarily with the endocardial wire passed via an arm vein. Five patients were being paced with epicardial electrodes attached to an external unit. There were several common factors in this group of patients (Table III). All the patients had shown unstable rhythms before and during the time they had been paced. Six patients had short episodes of ventricular tachycardia and 2 had multifocal ventricular ectopic beats. The 5 patients on epicardial pacing had their pacemaker units exteriorized because of sepsis and 4 of them had a rise in the threshold for pacing requiring more powerful units in order that pacing could be continued. In addition, 1 patient had developed pericarditis due to infection tracking to the epicardial electrodes via the epicardial wires which had been exteriorized because of sepsis around the implanted abdominal unit.

The histological findings in this group of patients were of great interest. There was a high incidence of myocardial disease (Table IV). Two patients had unsuspected active rheumatic carditis and had been paced for 36 and 18 months, respectively, for Adams-Stokes attacks. One patient had histological evidence of acute myocarditis, having presented with Adams-Stokes attacks 2 years before death, and she had been paced with an endocardial electrode for 18 months; a terminal feature was multifocal ventricular ectopic beats during the course of normal pacing.

One patient had diffuse fibrosis of the myocardium (collagen disease), and a feature of his 33 months of pacing had been episodes of ventricular tachycardia. These episodes had largely been suppressed by oral quinidine, but 36 hours before death he stopped taking the quinidine.

TABLE II
HISTOLOGY—GROUP 1: TECHNICAL FAILURES—
11 PATIENTS

No myocardial disease	No.	Myocardial disease	No.
Bilateral bundle-branch fibrosis	3	Cardiomyopathy	3
Valve ring calcification	2	Amyloid deposits	1
Congenital heart block	1		
Aneurysm of membranous septum	1		
Total	7		4

TABLE III
GROUP 2: VENTRICULAR FIBRILLATION
8 PATIENTS

	No.
Arrhythmias while pacing { Ventricular tachycardia Multifocal ventricular ectopic beats	6
Method of pacing { Epicardial Endocardial	2 5
Rising pacing threshold	3 5

TABLE IV
HISTOLOGY—GROUP 2:
VENTRICULAR FIBRILLATION—8 PATIENTS

Myocardial disease	No.	No myocardial disease	No.
Rheumatic carditis	2	Bilateral branch fibrosis	1
Acute myocarditis	1	Valve ring calcification	1
Collagen disease	1		
Cardiomyopathy	1		
Coronary artery disease	1		
Total	6		2

One patient who died shortly after admission to hospital had multiple Adams-Stokes attacks due to runs of ventricular tachycardia and ventricular fibrillation as well as episodes of asystole. This patient died a few minutes after pacing with an endocardial wire had been started. Histology of the heart showed typical features of a cardiomyopathy.

One patient in this group had bilateral bundle-branch fibrosis and an otherwise healthy myocardium. She had been paced for 36 months with an endocardial wire and, during her final admission for intermittent pacing due to an unexplained rise of threshold, her endocardial wire was exteriorized in the axilla and attached to a more powerful unit. She paced satisfactorily for 4 days and died suddenly from ventricular fibrillation.

Comment. The majority of patients in this group had presented with unstable ventricular rhythms both before and during pacing. In addition, the threshold for pacing had risen in 5 patients requiring

more power for pacing to be continued. It seems likely that the presence of myocardial disease is more likely to give rise to unstable ventricular rhythms during the course of otherwise satisfactory pacing and may lower the threshold for precipitating pacemaker-induced ventricular fibrillation. In retrospect, perhaps in those 5 patients where the threshold for pacing had risen, a new system of pacing with lower threshold should have been installed instead of using a more powerful unit in order to continue pacing. The only clinical clue to the presence of myocardial disease was the presence of unstable rhythms which continued to occur in spite of satisfactory pacing. In the majority of patients with bilateral bundle-branch block and an otherwise healthy myocardium, ectopic rhythms tend to be suppressed by pacing. Perhaps those patients who continue to have ectopic rhythms while pacing should all be treated with anti-arrhythmic drugs such as propranolol (Harris, 1966) or quinidine and pacing maintained with a ventricular inhibited pacemaker.

Group 3: Miscellaneous Causes of Death

There were 7 patients in this group (Table V) and the duration of pacing ranged from minutes to 54 months (mean 11 months). Three of these patients were admitted to hospital *in extremis* due to multiple Adams-Stokes attacks with prolonged periods of asystole, and died while efforts to resuscitate them by pacing were made. One patient died from carcinomatosis four days after temporary pacing had been started. During this patient's terminal illness frequent Adams-Stokes attacks were causing distress to the patient and for this reason he was temporarily paced. One patient had paced satisfactorily for 54 months when she suddenly died. At necropsy the cause of death was found to be a complete occlusion of the arch of the aorta by thrombus, and her pacing system was functioning perfectly.

Two patients who had been pacing satisfactorily for 12 months and 1 month, respectively, died from no obvious cause. Arrhythmias had not been a

TABLE V
GROUP 3: MISCELLANEOUS CAUSES OF DEATH
—7 PATIENTS

	No.
Moribund at time of pacing	3
Carcinomatosis	1
Thrombosed arch of aorta	1
Cause unknown	2
Method of pacing { Epicardial	2
Endocardial	5

problem in either patient while they were being paced. One patient was paced with an endocardial system which was intact and correctly positioned at necropsy. The other patient was on an epicardial system with an external unit. It is possible that the threshold for pacing had risen in this patient, which might have resulted in intermittent pacing; but out-patient measurements taken shortly before his death did not indicate that this was occurring.

The myocardial histology in this group of patients is shown in Table VI.

TABLE VI
HISTOLOGY—GROUP 3:
MISCELLANEOUS CAUSES OF DEATH—7 PATIENTS

	No.
Bilateral branch fibrosis	6
Cardiomyopathy	1

Comment. A number of patients with chronic heart block will continue to die in the acute phase of recurrent Adams-Stokes attacks unless resuscitation measures are promptly instituted. The first 3 patients were admitted to this unit virtually moribund owing to prolonged periods of cardiac arrest at home. Prevention of this situation lies in the early diagnosis of heart block and the general realization that untreated chronic heart block is associated with a high mortality.

DISCUSSION AND CONCLUSIONS

The aetiology of the complete heart block in the 26 patients was commonly isolated disease of the conducting tissue or myocardial disease. Coronary disease was rare, only occurring in 1 patient in whom it was not the cause of death.

In this series 42 per cent of the deaths were attributed to technical failure, principally related to failure to maintain satisfactory pacing with an endocardial wire. These deaths are liable to occur when endocardial pacing is temporarily carried out from an arm vein or if redundant loops of electrode wire are left in the heart. By immobilizing the arm and reducing the time of temporary pacing to a minimum when using an arm vein and careful x-ray screening to ensure that redundant loops of endocardial wire are not left within the heart, the number of technical failures would be considerably reduced.

The majority of patients in Groups 1 and 3 had a relatively healthy myocardium, the abnormal histological findings being confined to the main bundle or bundle-branches. However, the majority of patients in Group 2, with ventricular fibrillation

despite satisfactory pacing, showed myocardial disease in addition to disease of the main bundle or bundle-branches. A common clinical failure of those patients in Group 2 was the presence of ectopic beats both before and during satisfactory pacing. Perhaps the combination of myocardial disease together with ectopic rhythms may have contributed to the occurrence of ventricular fibrillation when the pacemaker impulse fell during the vulnerable period of an ectopic beat (R on T phenomenon). The presence of unstable rhythms during pacing should raise the possibility that disease is not confined to the conducting tissue, and the use of a ventricular inhibited pacemaker or cardiac depressant drugs should be considered.

SUMMARY

The cause of death in 26 patients while being treated with long-term pacing for chronic heart block have been analysed. The patients were paced by endocardial (19) or epicardial (7) systems for an average of 13.6 months (range—minutes to 54 months) before death occurred.

Necropsy has included a special study of the conducting tissues at 8 μ intervals, and multiple blocks have been examined from all valves and chambers. The coronary arteries were studied by a simple injection technique and serial blocks.

The pacemaker systems were carefully examined for faults in the units, electrodes, and connecting wires.

Eleven patients died as a direct result of technical failure from either displacement of the endocardial electrode resulting in failure to pace (6), septicaemia (2), unit failure (2), or a fractured endocardial electrode wire (1). Histological findings in these 11 patients showed that 7 had a relatively healthy myocardium and should have had a good prognosis with satisfactory pacing. Temporary endocardial pacing from an arm vein can be dangerous unless the arm is carefully immobilized to prevent displacement of the endocardial electrode, and a suitable harness is shown. A new endocardial electrode wire suitable for long-term use is described, which has so far proved to be free from fractures with 9 months' use in 51 patients.

The incidence of disease of the myocardium in addition to disease of the main bundle or branches was 46 per cent in the 26 patients; the incidence of myocardial disease in those patients with ventricular fibrillation despite satisfactory pacing was 75 per

cent. Ventricular fibrillation occurred in 8 patients whose pacing systems were functioning properly. The possible cause for the onset of ventricular fibrillation may be related to the occurrence of unstable cardiac rhythms in competition to the pacemaker, changes in the threshold (power) for pacing, and the underlying myocardial disease.

Three patients died during efforts to resuscitate them with pacemakers during acute Adams-Stokes attacks, and the need for early diagnosis and treatment of chronic heart block is emphasized. Two patients died from unrelated disease and 2 others from no obvious cause.

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