Pacing in children

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SUMMARY Fourteen children had a permanent pacemaker implanted between May 1967 and July 1983. Postoperative complete heart block was the indication in nine cases, congenital complete heart block in three, and sick sinus syndrome in two. Two patients died, one suddenly and one after aortic valve replacement. A total of 48 pulse generators were implanted; five patients were given an isotopic pacemaker. Twelve patients had epicardial leads implanted initially, and two received a transvenous endocardial system. The lead system implanted initially remained without malfunction in only seven patients. In the other seven patients 20 lead malfunctions occurred. Psychological maturity and physical development seemed to be normal in all 14 children. Improvement in equipment and technique will improve the outlook for paced children in the future.

Paediatric patients form only a minority of the population in a pacemaker clinic. Nevertheless, they account for a substantial proportion of the complications. Postoperative heart block is the most common indication for permanent pacing in children.¹⁻³ Congenital or idiopathic complete heart block are the other important indications. In children the sick sinus syndrome is now also recognised as an arrhythmia with a potentially fatal outcome.⁴⁻⁶ The improved reliability and long life of present day cardiac pulse generators justify pacing in symptomatic patients and in those with potentially life threatening conditions.

Patients and methods

From May 1967 to July 1983 a permanent cardiac pacemaker was implanted in 14 children (seven girls and seven boys). They were aged between 27 and 152 (mean age 79, SD 35) months. Their most important clinical features are given in Table 1.

Nine patients developed complete heart block after surgical repair of a congenital heart defect. Three children had congenital complete heart block, one of whom had a ventricular septal defect and valvar pulmonary stenosis. Two children (cases 1 and 2) had sick sinus syndrome without structural heart disease and have been reported elsewhere.⁶ Two patients also

had severe ventricular arrhythmias (cases 2 and 13).

Adams-Stokes attacks before pacemaker implantation or at the time of pacing malfunction occurred in eight patients (cases 2, 5, 6-9, 11, and 14), whereas no symptoms were related to conduction disturbance in six (cases 1, 3, 4, 10, 12, and 13). One patient (case 1) had sinoatrial disease, and bradycardia had been noticed before birth. This child received a pacemaker because of extreme bradycardia and long ventricular pauses. During the night the ventricular rate varied between 15 and 45 beats/minute. One pause of 4.8, one of 4.6, and numerous pauses of >3 s were recorded. One child (case 3) had asymptomatic congenital complete heart block. Since birth there had been an escape rhythm with narrow QRS. Ambulatory monitoring showed episodes of ventricular asystole lasting up to 4 s and ventricular rates up to 20 beats/min. The second child (case 4) with asymptomatic congenital complete heart block had a nodal (narrow QRS) rhythm slowing down to below 30 beats/min with ventricular pauses lasting up to 5.16 s. Three patients (cases 10, 12, and 13) had postoperative complete heart block. One (case 10) initially received cortisone treatment for atrioventricular block, but this treatment had no effect. A permanent pacemaker was implanted 30 days after surgical repair of his congenital heart defect. In the second patient (case 12) a permanent pacemaker was implanted 35 days after the first surgical intervention. In the third patient (case 13) there was a delay of 21 days between the first operation and pacemaker implantation.

All patients were given ventricular demand or ven-

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Table 1 Clinical features of 14 paced paediatric patients

Case No	Sex	Age (yr)*	Associated heart disease	Indication for pacing	Duration† of follow up (mnh)‡	Total No of pulse generators	Present pacemaker type
1	F	4	—	SS	70	3	Coratomic C101
2	M	5	—	SS	82	2	Medtronic 5985
3	F	7	—	CHB (congenital)	23	1	Medtronic 5985
4	F	12	—	CHB (congenital)	13	1	Coratomic C101
5	F	4	VSD, PS(V)	CHB (congenital)	73	1	Cordis 190A
6	F	2	VSD, PDA	CHB (postoperative)	148	8	Coratomic C101
7	M	7	VSD, PS(I)§	CHB (postoperative)	207	8	Coratomic C101
8	M	4	VSD, PS(I)	CHB (postoperative)	87	2	CPI 523
9	F	6	VSD	CHB (postoperative)	160	4	CPI 505
10	M	7	ASD	CHB (postoperative)	135	3	CPI 504
11	F	5	AC, SAS	CHB (postoperative)	47	1	CPI 505
12	M	4	VSD, PS(I)	CHB (postoperative)	177	6	Coratomic C101
13	M	11	TF	CHB (postoperative)	54 (sudden death)	3	
14	M	8	SAS	CHB (postoperative)	84 (postoperative death after AVR)	5	

*At the time of pacemaker implantation.

†Until 31 August, 1984.

‡After first pacemaker implantation.

§Absence of pulmonary valve.

||Ostium primum type with cleft mitral and cleft tricuspid valve.

AC, aortic coarctation; ASD, atrial septal defect; AVR, aortic valve replacement; CHB, complete heart block; PDA, persistent ductus arteriosus; PS(I), infundibular pulmonary stenosis; PS(V), valvar pulmonary stenosis; SAS, subvalvar aortic stenosis; SS, sick sinus syndrome; TF, tetralogy of Fallot; VSD, ventricular septal defect.

tricular fixed rate pacemakers. Gradually, we had started to prefer atomic pacemaker implantation in children, and so five patients had the small size Coratomic C101 atomic pacemaker. Paediatric suturable epicardial electrodes (Cordis 323-543 and Medtronic 5815A) were preferred. Only two patients received a General Electric bipolar epicardial pacing system as the initial implant. A transvenous endocardial pacemaker system was used as the initial implant in only two children. In 13 patients initial implantations and generator replacements were performed under general anaesthesia. In only one patient (case 4) was a transvenous pacing system implanted under local anaesthesia.

During the follow up of these patients, special attention was given to the presence or absence of escape rhythms occurring after inhibition of the ventricular demand pacemaker by chest wall stimulation. The technique of chest wall stimulation and its application in the pacemaker clinic has been reported elsewhere.⁷

Results

Follow up after implantation of a permanent pacemaker varied between 13 and 207 (mean 97.1, SD 59) months. Two patients died. Sudden death occurred in one boy (case 13), who was known to have severe ventricular arrhythmias, and one (case 14) died postoperatively after aortic valve replacement seven years after his first operation for subvalvar aortic stenosis. One patient (case 2), who was known to have asymptomatic sinus bradycardia and sinuatrial block,

collapsed while playing at school. He was given mouth to mouth respiration and arrived at the emergency department within 10 minutes. Repeated episodes of ventricular tachycardia alternated with periods of atrial and ventricular standstill. After the implantation of a permanent pacemaker he recovered completely.

In the 14 children we implanted a total of 48 pulse generators: 14 initial implants and 34 replacements. The number of generators per patient varied between 1 and 8. Four children still had their initial implant after 13-73 months. The generator was changed for prophylactic reasons in 14 of the 34 replacements. Sudden generator failure occurred in four pacemakers. Generator change without lead change was required because of extrusion in two instances and infection in one. Competitive pacing of a ventricular fixed rate pacemaker was the reason for generator change in one case. In 12 cases non-generator related complications led to pacemaker replacement: (a) nine prophylactic replacements at the time of lead failure; (b) generator plus lead change because of infection in two instances; and (c) exit block treated by the insertion of a high output pacemaker in one case. Table 2 shows these data together with the length of life of the replaced pacemakers. The use of special paediatric pacemakers with only two mercury-zinc batteries was the reason for the early prophylactic changes and failures.

Cordis 323-543 leads were implanted in five patients (cases 1, 2, 3, 5, and 11) and Medtronic 5815A leads in five (cases 7, 8, 10, 12, and 13). Two patients (cases 6 and 14) were initially given a bipolar

Table 2 Indications for pacemaker replacement and life of generators (in months). Figures are number of pacemakers

	<30	30-39	40-49	>50	Total
Prophylactic change	4	1	3	6	14
Sudden failure	2	1	1	—	4
Lead problem	8	2	—	—	10
Extrusion	2	—	—	—	2
Infection	3	—	—	—	3
Competitive pacing	1	—	—	—	1
Total	20	4	4	6	34

General Electric epicardial electrode system, but subsequently these leads were replaced by Medtronic 5815A leads. In three patients it was necessary to replace the epicardial system by a transvenous one because of extrusion (case 6), exit block (case 7), and infection (case 14). In one patient (case 7) the system had to be changed back to an epicardial one after exit block of the transvenous system had occurred. In this instance, epicardial leads were implanted as well as a Hancock pulmonary valve prosthesis, as pulmonary insufficiency caused a high right ventricular stroke volume making adequate positioning of an endocardial lead impossible. In two patients (cases 4 and 9) transvenous endocardial leads were implanted as the initial system.

The initial lead system remained without malfunction in only seven patients. In the remaining seven patients 20 lead malfunctions occurred: lead fractures (6), insulation defects (3), exit blocks of epicardial leads (6), infection-extrusion (2), and exit blocks of endocardial leads (3). Lead fracture required a new lead implantation in three instances and could be overcome by the use of an additional lead on two occasions. One fracture occurred in a spare lead. Insulation defects required lead replacement in two cases, and on only one occasion could the defect be repaired. Because of exit block the epicardial leads had to be replaced in four instances; the pacemaker was replaced by a high output unit on one occasion. In one case the bipolar pacing was changed to the unipolar mode. Because of exit block two transvenous leads had to be repositioned, and in one patient the transvenous system was replaced by an epicardial one. In the two cases of extrusion-infection the old epicardial pacing system was completely removed and replaced by a transvenous one. A prepectoral position was chosen for the pulse generator in one case and an epigastric position in the other. Table 3 summarises the most important features of the complex history of these 14 children.

A spontaneous heart rhythm occasionally inhibiting the ventricular demand pacemaker was seen in only one patient (case 13). Absence of escape rhythm during pacemaker inhibition by chest wall stimulation occurred in six patients (cases 6, 7, 8, 9, 11, and 14). All these six patients had experienced Adams-Stokes

attacks. Two patients with a previous history of Adams-Stokes attacks always had an adequate underlying heart rhythm (cases 2 and 5). During chest wall stimulation all patients who were asymptomatic before pacemaker implantation proved to have a reliable escape rhythm on all occasions (cases 1, 3, 4, 10, 12, and 13).

All these 14 children led a normal physical life and were psychologically well adjusted for their age and intellectual capacity. Excellent results at school were obtained by eight patients (cases 2, 4, 6, 8, 9, 11, 12, and 14). In one patient (case 7), who had recently started an administrative job, the presence of an isotopic pacemaker added to his confidence and that of his parents. In the children with an isotopic pacemaker in particular, the psychological effects of a pacemaker tended to disappear completely.

Discussion

Postoperative complete atrioventricular block was the major indication for cardiac pacing in our patients. We also treated three patients with congenital complete heart block and two with sick sinus syndrome. One patient with congenital heart block also had congenital heart disease. Similar cases are reported by other authors. In a French multicenter study Vanetti *et al* studied 241 children with permanently implanted pacemakers.² Congenital complete heart block was found in 66 cases and was associated with congenital heart disease in a further 32, and 136 cases had postoperative heart block and seven acquired heart block. Young reviewed 448 pacemaker implantations in children.³ In 284 cases pacemaker implantation was required as a result of intracardiac surgery; in 270 cases pacemaker implantation was required because of postoperative heart block which was not necessarily symptomatic and in 14 because of postoperative symptomatic sinus nodal dysfunction. Permanent pacing was undertaken in 137 patients with symptomatic heart block and in 27 with symptomatic sinus nodal dysfunction of idiopathic, congenital, or acquired (non-surgical) aetiology.

Numerous reports discuss the therapeutic dilemma in the case of congenital complete heart block. A benign prognosis has been reported by Campbell and

Table 3 Surgical procedures

Case No	Year of first implantation	Surgical procedures (time between procedures in months*)
1	1978	Initial implant of 2 epicardial leads—(7)—reintervention and use of reserve lead and elective generator replacement—(63)—generator replacement
2	1977	Initial implant of 2 epicardial leads—(58)—generator replacement—(24)
3	1982	Initial implant of 2 epicardial leads—(16)—reintervention and use of reserve lead—(7)
4	1983	Implantation of transvenous lead—(13)
5	1978	Initial implant of 2 epicardial leads—(73)
6	1972	Initial implant of General Electric epicardial lead system—(2)—elective generator replacement at time of lead complication—(14)—subsequent reintervention for implantation of 2 epicardial leads and replacement of generator—(1)—generator replacement—(4)—generator replacement—(8)—implantation of transvenous lead and replacement of generator—(1)—repositioning of transvenous lead—(26)—generator replacement—(50)—generator replacement—(41)—implantation of transvenous lead—(1)
7	1967	Initial implant of 3 epicardial leads—(10)—reintervention for implantation of 1 epicardial lead and replacement of generator—(30)—generator replacement—(38)—elective replacement of generator—(8)—reintervention and use of reserve lead—(4)—implantation of transvenous lead and replacement of generator—(20)—generator replacement—(1)—reintervention for implantation of 2 epicardial leads and generator replacement—(46)—generator replacement—(50)
8	1977	Initial implant of 2 epicardial leads—(50)—generator replacement—(37)
9	1971	Implantation of transvenous lead—(30)—repositioning of transvenous lead and generator replacement—(26)—generator replacement—(54)—generator replacement—(60)
10	1973	Initial implant of 2 epicardial leads—(31)—generator replacement—(44)—generator replacement—(50)
11	1980	Initial implant of 1 epicardial lead—(47)
12	1969	Initial implant of 3 epicardial leads—(13)—reintervention and use of reserve lead and generator replacement—(23)—generator replacement—(5)—generator replacement—(8)—reintervention and use of reserve lead—(33)—generator replacement—(58)—generator replacement—(37)
13	1968	Initial implant of 3 epicardial leads—(26)—generator replacement—(26)—generator replacement—(2)—death
14	1970	Initial implant of General Electric epicardial lead system—(11)—generator replacement—(4)—reintervention for implantation of 1 epicardial lead—(4)—reintervention for implantation of 2 epicardial leads and generator replacement—(5)—implantation of transvenous lead and generator replacement—(40)—generator replacement—(20)—death

*Until 31 August 1984.

Thorne.⁸ They followed seven patients for 25 years and only one died. Only one had an enlarged heart and evidence of aortic regurgitation. Corne and Mathewson also emphasised the excellent prognosis in congenital complete heart block, provided that there is no associated congenital structural heart disease, that the QRS complexes are supraventricular in configuration, and that the condition is not familial.⁹ Molthan *et al*, Ayers *et al*, and Nakamura and Nadas challenged the general belief that complete heart block has a good prognosis.^{10–12} Molthan *et al* have reported three cases of fatal Adams-Stokes attacks in a series of 35 children with congenital complete heart block examined between 1953 and 1961.¹⁰ Nakamura and Nadas found an unexpectedly high mortality of 21.3% (13 cases).¹² The severe disturbance in atrioventricular conduction, causing Adams-Stokes attacks, could clearly be indicated as the cause of death in only three patients. None of these patients died of the first attack; in fact, they all had at least three attacks. These workers emphasised that complete heart block not only causes death by Adams-Stokes attacks but may also worsen the prognosis of associated congenital heart disease. The patients who have slower rates, wide QRS complexes, previous Adams-Stokes attacks, and a poor response to exercise or drugs are those most likely to have a fatal outcome. Ayers *et al*¹¹ agreed with Nakamura that in any child

with complete heart block who has had two or more Adams-Stokes attacks, permanent pacemaker treatment should be seriously considered. Karpawich *et al* suggested that the simple procedure of measuring heart rate at rest appeared to be the best current predictor of bradycardia induced syncope in young children with congenital complete heart block.¹³ No child with a heart rate at rest of 50 beats/min or more had syncope, whereas seven of the 11 children with a lower heart rate had syncope. Karpawich *et al* recommended cardiac pacing in any child with symptoms attributable to Adams-Stokes attacks or congestive heart failure caused by a low heart rate. The greatest risk of death for the child with congenital heart block is in early infancy.¹⁴

In our patient group we had three cases of congenital complete heart block. All three had a pacemaker implanted. Two patients (cases 3 and 4) were asymptomatic but showed pronounced bradycardia and long ventricular pauses. Adams-Stokes attacks had occurred in one patient (case 5). In our view the indication for permanent cardiac pacing in these three patients was clear.

Postoperative complete heart block is generally considered to be a serious and life threatening condition. Murphy *et al* studied eight patients with postoperative complete heart block who were not given a pacemaker.¹⁵ During a mean observation period of

two years all were asymptomatic and none died. The follow up period of two years was possibly too short to allow a valid clinical judgement. Lillehei *et al*, McGoon *et al*, Sayed, and Liu *et al* have favoured the use of cardiac pacemakers for the long term management of postoperative complete heart block because of the high mortality rate in patients given medical treatment alone.¹⁶⁻¹⁹ Hurwitz *et al* observed 13 children with chronic postoperative complete heart block; of these, 10 were not given a pacemaker and three of the 10 died.²⁰ The survivors were followed up for periods of eight months to nine years, but only three of the seven survivors did not experience Adams-Stokes attacks. Hofschire *et al* reported postoperative complete heart block in 64 children, some of whom were treated with cardiac pacing and some who were not.¹ Five of the seven patients with complete heart block who received medical treatment died after discharge from hospital. The authors proposed a policy of implanting a permanent pacemaker when complete heart block persisted for 4-6 weeks after operation. Forty seven of their patients with complete heart block survived the immediate postoperative period. In 23 (50%) regular sinus rhythm returned within four weeks after the operation.

Postoperative complete heart block accounted for nine cases in our series. All patients except cases 10, 12, and 13 experienced Adams-Stokes attacks. In the three asymptomatic patients we waited between 21 and 35 days before implanting a permanent pacemaker. In view of the poor prognosis of postoperative complete heart block we consider that a permanent cardiac pacemaker should be implanted in patients with complete heart block after intracardiac surgery when there is no restoration of normal atrioventricular conduction within a few weeks after the occurrence of complete heart block. The three asymptomatic patients with postoperative complete heart block always had a reliable underlying heart rhythm when their pacemaker was inhibited by chest wall stimulation.

The two children with sick sinus syndrome are the two paced patients of a series of five paediatric patients who have been extensively reported elsewhere.⁶ One child survived sudden death and the other had extreme bradycardia and prolonged ventricular pauses.

Complications in a population of paced children are common. Premature battery failure, fracture of leads, infection, change in pacing threshold, arrhythmia, and pulse generator failure are reported in numerous series.^{1-3 21-25} In our series only two patients received a transvenous pacing system as the initial implant. In the study of Vanetti *et al* 93.4% of the children had myocardial leads implanted.² Hayes *et al* reported that in their patients 13% of those with

transvenous endocardial leads required a second operation compared with 35% of those with epicardial pacing.²² In the patients with epicardial pacing reoperation involved a thoracotomy in 18%. We preferred to use epicardial leads in children because (a) we wanted to save the transvenous access for later life and (b) recovery after a small thoracotomy was always uneventful in children.

Gillette *et al* established clear criteria for transvenous pacing in children: they were (a) aged 4 years or more; (b) weighed 15 kg or more; (c) had no right to left shunt; (d) had adequate communication of a superior vena cava with at least part of an atrium; and (e) were not undergoing a simultaneous cardiac operation.²⁶

Growth and lead stretch were not a major cause of failure in either transvenous or epicardial leads. One patient (case 9) to date has been paced by the same transvenous lead for 130 months. The persistent small size of this child is the reason for the absence of lead complications. In only one patient (case 6) was lead stretch the apparent cause of fracture in the transvenous lead. Fracture occurred after 118 months when the child had grown very tall.

Fracture of epicardial leads occurred after seven months (case 1), 16 months (case 3), 13 months (case 12), and four months (case 14). Lack of surgical skill and technical defects rather than growth and lead stretch are the most likely causes of failure after so short a period of time. To date well implanted epicardial leads have been functioning for 63 (case 1), 82 (case 2), 73 (case 5), 76 (case 7), 96 (case 7), 87 (case 8), 135 (case 10), 128 (case 12), and 54 (case 13) months.

Repositioning of the endocardial lead was necessary in two out of five patients who had received a transvenous lead (two initial and three subsequent implants). The complication of exit block or dislocation or both would probably have been prevented by the use of leads with active or passive fixation. To achieve maximum reliability and longevity of cardiac pulse generators, we now prefer to use isotopic pacemakers in young people. The benefits of the increased longevity of the lithium battery, which was initially very promising, are outweighed by the initial demands for a smaller size and later for more complex circuits. The longevity and reliability of the isotopic pacemaker have been found to be superior to pacemakers powered by chemical batteries.²⁷ Parsonnet *et al* reported on 151 atomic units implanted in 131 patients.²⁸ The survival of pacemakers after 10 years was 92%. This high reliability and superior performance suggest that the continued use of a radioisotopic power source is justified.

The short life of the pacemakers used in the first

decade of paediatric pacing is shown in Table 2. Only six of the 34 replaced pacemakers functioned for 50 months or more. The limited life of mercury-zinc batteries together with the large size of their generators has played an important role in the complication rate. Attempts to manufacture small pacemakers with only one or two mercury-zinc batteries resulted in further shortening of longevity and a reduction in reliability.

Despite the high complication rate, pacing in children is a challenge that must be accepted by a pacemaker clinic. The long term follow up is a rewarding experience which is echoed by others.^{24 25} Despite frequent admissions to hospital and pacemaker replacements paced children have normal physical development and psychological maturation. The important advances in equipment and techniques will largely improve the outlook for the future of paced children.

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