ELECTRICAL PACING OF THE HEART IN ATRIOVENTRICULAR BLOCK

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The high mortality of atrioventricular block is largely due to Stokes-Adams attacks.[1] These attacks are associated either with periods of ventricular standstill or ventricular fibrillation (Figs. 1 and 2). Emotional disturbances, particularly excitement, may precipitate attacks,[33] but in general it is not possible to predict their onset or severity, and any attack may end fatally. With complete atrioventricular dissociation the ventricular rate is often so slow that the cardiac output is inadequate and angina pectoris or cardiac failure may supervene.[2]

Treatment aims either at relieving the block or increasing the ventricular rate. In certain cases of incomplete block, atropine, by depressing vagal impulses, may improve atrioventricular conduction and thus quicken the pulse. It is of no value in complete dissociation. Ephedrine, adrenaline, and various synthetic amines, notably isoprenaline, have for many years been the mainstay of therapy, but their value is limited. Isoprenaline is said to be more effective when administered rectally than when given sublingually.[5] Recently Linenthal and Zoll[4] have shown that adrenaline and

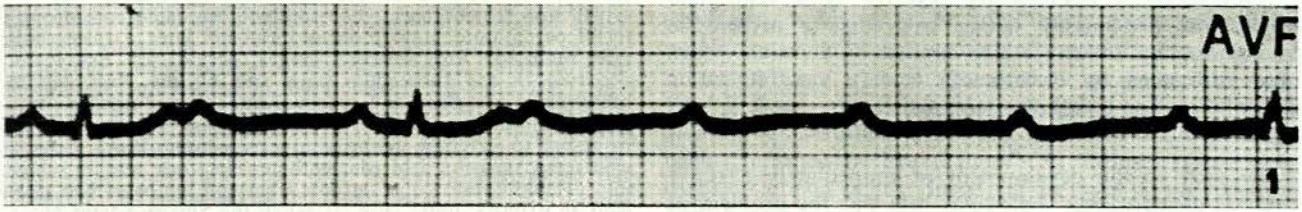

Fig. 1. ECG showing ventricular asystole lasting 3½ seconds in case 4.

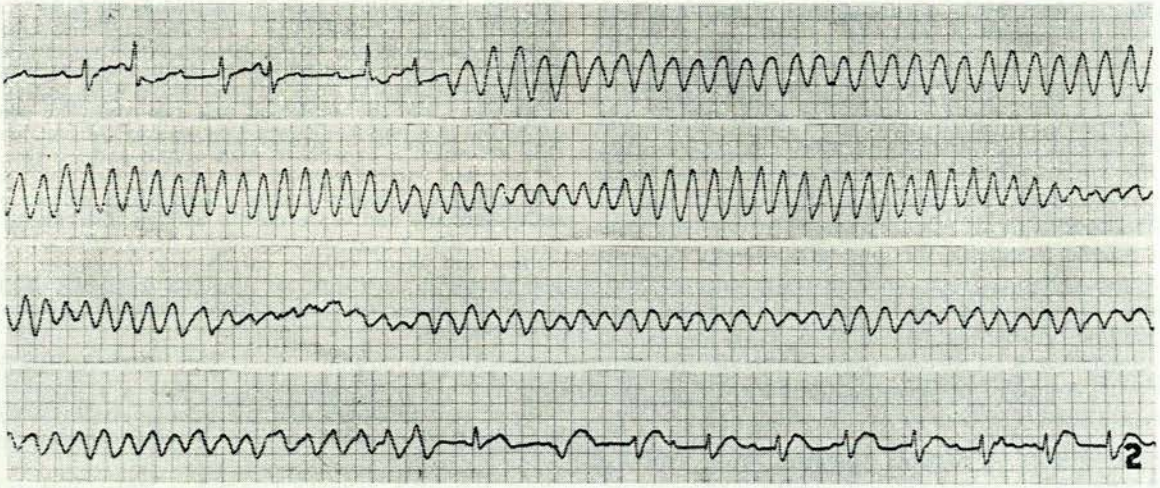

Fig. 2. ECG (continuous strip of standard lead 1), recorded during Stokes-Adams attack, showing ventricular fibrillation and spontaneous reversion to idioventricular rhythm (case 3).

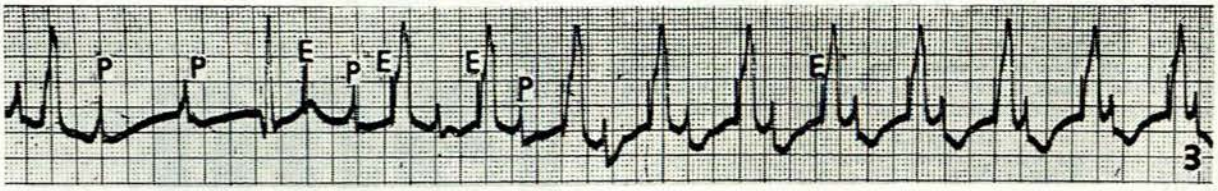

Fig. 3. ECG (oesophageal lead) in case 1 showing, at the left of the tracing, atrioventricular block. Thereafter, pacing via a catheter electrode with ventricular complexes follows immediately on the pacemaker impulse (E). The very first impulse has not produced ventricular excitation. Dissociation between the paced ventricular rhythm and the patient's own atrial rhythm is demonstrated.

isoprenaline are more effective when given by intravenous infusion than by other routes. When administered intravenously to patients with inadequate intrinsic ventricular activity these drugs arouse ventricular pacemakers that accelerate the heart and increase the force of ventricular contraction. This therapy effectively controls Stokes-Adams attacks whether caused by ventricular standstill or ventricular fibrillation.[4] The action of these drugs in preventing ventricular fibrillation is unexpected in view of the known effect of adrenaline in predisposing the heart to ventricular fibrillation. However, it appears that in the presence of heart block adrenaline produces a more 'stable' rhythm. Good results have also recently been reported with a sustained-action oral isoprenaline preparation,[34] but this drug is barely out of the experimental stage and it is as yet too early to judge its long-term effectiveness. Sodium lactate facilitates atrioventricular conduction but its accelerating action is minimal[3] and it is potentially dangerous.[6] Chlorothiazide is occasionally effective as a ventricular accelerator, presumably acting by reducing myocardial potassium, but its use is also limited,[1, 7] Steroids, which were introduced by Prinzmetal,[8] have a variable action and, whereas some authors claim satisfactory results,[1, 7, 9-11] others maintain that these drugs are ineffective.[4, 7]

The spur to the development of immediately effective methods of increasing ventricular rate was the problem of heart block encountered by cardiac surgeons when they first closed ventricular septal defects. The bundle of His is vulnerable to direct injury during closure of such a defect. Weirich *et al.*[15] conceived the idea of suturing electrodes into the myocardium in these cases and directly stimulating the heart with an external pacemaker. It was then but a step to attempt long-term control of heart block, but problems related to electrolysis of steel electrodes, infection of the wire tracks, and fracture of the electrodes, made it necessary to refine the technique. Zoll *et al.*[12] had, in 1954, introduced external electrical pacing of the heart, but this had proved unsuitable for long-term therapy. Various modifications of operative techniques and pacing methods have now been evolved and progress continues. The perfect method of electrical pacing of the heart has not yet been evolved, but it is already possible greatly to assist patients with complete atrioventricular dissociation, whether they are subject to Stokes-Adams attacks or not.

We have treated 4 patients suffering from repeated Stokes-Adams attacks with long-term electrical pacemaking, and the results of this experience are reported here. Two cases (1 and 2) are presented in detail, and the clinical features and treatment of the other two (3 and 4) are summarized:

Case 1

A 55-year-old European male sustained a myocardial infarction in June 1962. Atrioventricular block soon followed, initially varying from 2-to-1 to complete block and eventually remaining complete. He experienced a number of syncopal attacks which, it was observed, were due to ventricular standstill. Treatment with dexamethasone, sublingual isoprenaline, ephedrine, and hypodermic adrenaline, resulted in some improvement. On this regime he remained confined to his home for the following year, during which time he had a number of Stokes-Adams attacks, at first associated with ventricular standstill, and later with both standstill and ventricular fibrillation. He also developed a marked decrease of exercise tolerance and orthopnoea. In July 1963 a unipolar catheter electrode was passed from the right external jugular vein into the right ventricle and the heart was paced from an external unit. The pacing, however, was not entirely successful in that it tended to be intermittent. Later the same day an internal pacemaker* was implanted, which stimulated the heart via platinum leads sewn to the surface of the left ventricle. Pacing was now satisfactory for 32 days, when suddenly his pulse rate dropped to 40 per minute, though some moments later a faster pulse rate returned. Progressively longer periods of bradycardia occurred in the succeeding days and by the 36th day the bradycardia was fixed and syncopal attacks, due now to ventricular fibrillation, occurred. It was necessary on two occasions to resort to external defibrillation by counter-shock.[31] As an emergency measure a bipolar catheter electrode was introduced into the right ventricular outflow tract and the heart was paced with an external unit† (Fig. 3), when the Stokes-Adams attacks were immediately controlled. The next day the implanted pacemaker was exposed by surgical operation, and it was found that the heart could be paced via the existing wires with an external unit. It was apparent that high resistance had developed at the implanted electrodes, for effective stimulation was produced only by a current larger than could be supplied by the implanted battery unit. The abdominal wires were therefore exteriorized and the implanted unit removed. The wires were connected to a portable external pacemaker‡ of our own design (Fig. 4), which is capable of high output and variable control of rate and current voltage. The patient has since remained well and free of Stokes-Adams attacks. He is able to live relatively normally and can even run without discomfort. On one occasion, after a near motor-collision, he experienced symptoms of fear and anger without palpitations (pacemaker set at 80 per minute at the time). No ill-effects were experienced as a result of the absence of 'normal' tachycardia under these circumstances.

Case 2

At the age of 66 years this patient, a European woman living at Port Elizabeth, first had fainting attacks. A year later she sought medical advice and Stokes-Adams disease with

*Elema Implantable Pacemaker, supplied by Protea Holdings Limited, Johannesburg.
†Electrodyne, supplied by Medical Distributors (Pty) Limited, Johannesburg.
‡Supplied by Medical Ideas & Equipment Co., Johannesburg.

complete heart block was diagnosed. The frequency and severity of the attacks rapidly increased despite medical treatment, and the patient was virtually confined to bed. At times

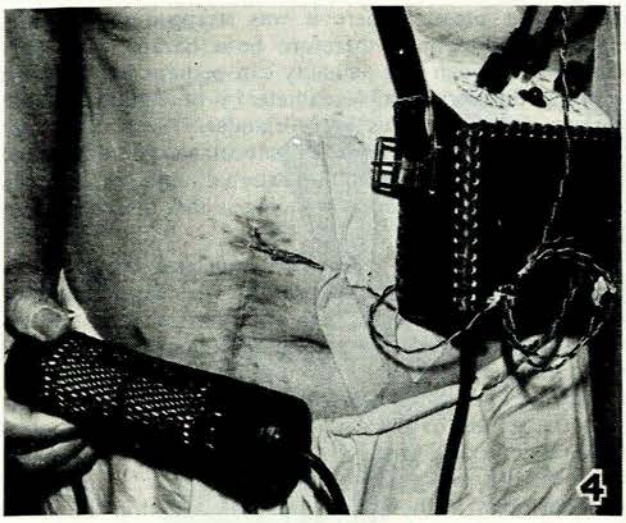

Fig. 4. Case 1. Our external pacing unit is seen in its holster. The pacemaker puts out a square wave of 2 milliseconds' duration. The current output can be varied from 15 ma to 60 ma by the current control switch on the patient's left of the control panel. The rate control switch gives a range of 60 - 120 impulses per minute. A neon lamp flasher on the control panel indicates operation. The instrument measures $5\frac{3}{8} \times 4\frac{1}{4} \times 2$ inches and weighs $1\frac{1}{4}$ lb. complete with battery. The portable battery charger can be seen in the patient's right hand. This plugs into AC mains. An indicator for the state of charge of the batteries is supplied.

the syncopal episodes were so frequent that she would have no recollection of an entire day. Treatment with various drugs, including adrenaline, digoxin, steroids, and sublingual isoprenaline, was ineffective, and emergency measures were taken to transfer her to hospital in Johannesburg in June 1963. Just before being flown from Port Elizabeth she was experiencing up to 10 Stokes-Adams attacks a day. ECG on arrival showed complete atrioventricular block with frequent runs of multifocal ectopic ventricular beats (Fig. 5). Paroxysmal ventricular fibrillation was suspected as the likely cause of her attacks and the following day this was confirmed by ECG when numerous episodes of unconsciousness occurred. The ventricular fibrillation was partially controlled by continuous intravenous isoprenaline, and later the same day a bipolar catheter electrode was passed into the right ventricle. The effect of direct cardiac pacing was dramatic, and all syncopal attacks ceased. Pacing by this route was maintained for 12 weeks, after which an internal pacemaker¶ was implanted. She has remained in stable rhythm for 5 months and has returned home fully ambulant. She has recently written that she is able to do gardening again and that she feels very well.

¶Medtronic Implantable Pacemaker, supplied by Eric Mance (Pty) Limited, Johannesburg.

Case 3

European female aged 51. Presented with syncopal attacks and marked decrease in exercise tolerance. Basic rhythm—atrioventricular block from 2 - 1 to complete. Stokes-Adams attacks with ventricular fibrillation. Medical therapy—potassium chloride, sublingual isoprenaline, digoxin, and sedation. Method of pacing—bipolar catheter electrode; direct cardiac electrodes and implanted battery unit. At time of writing—no attacks for 5 months; stable ventricular rate.

Case 4

European female aged 69. Presented with syncopal attacks. Basic rhythm—sinus rhythm; prolonged P-R interval; atrioventricular block from 2 - 1 to complete. Stokes-Adams attacks with ventricular standstill. Medical therapy—sublingual isoprenaline, hypodermic adrenaline, atropine, prednisolone, and ephedrine. Method of pacing—external pacing; bipolar catheter electrode; direct cardiac electrodes and implanted battery unit. At time of writing—no attacks for 5 months; stable ventricular rate; subsequent left ventricular failure (?myocardial infarction) but with stable pacing.

METHODS OF ELECTRICAL PACING

The following are the various methods available:

(A) *External pacing.* Since the introduction of this technique by Zoll *et al.*,[12-14, 30] pacing of the heart with metal electrodes on the chest has been used extensively. It has proved effective and often life-saving. Two or three electrodes, one of them an earthing connection, are strapped to the left pectoral region, with suitable electrode paste on the skin to diminish resistance. High voltages (50 - 150 v.) are required, which cause severe pain, and heavy sedation is therefore necessary. Skin ulceration may also occur,[16] as happened in our case 4. Pericardial and epicardial injuries have been reported.[17] External pacing is therefore only an emergency measure to be used while arrangements for initiating a more effective method are being made.

(B) *Catheter pacing.* The method of pacing the heart via an intracardiac electrode was introduced by Furman and Schwedel[18] in 1959. The effectiveness of this route has been confirmed by Portal *et al.*[2] A platinum-tipped solid cardiac catheter is introduced via an external vein into the right ventricle until the tip engages on ventricular muscle (Fig. 6.) The use of an external jugular vein is preferable to that of an arm vein, because it allows the patient greater mobility. A unipolar intracardiac electrode may be used with the indifferent electrode placed on the skin. Contact of the indifferent electrode may vary, however, and this was perhaps responsible for the intermittency of pacing noted in our case 1 before surgery. We thus prefer a bipolar electrode,** and this has been used effectively in

**Supplied by U.S. Catheter and Instrument Company through Protea Holdings Limited, Johannesburg.

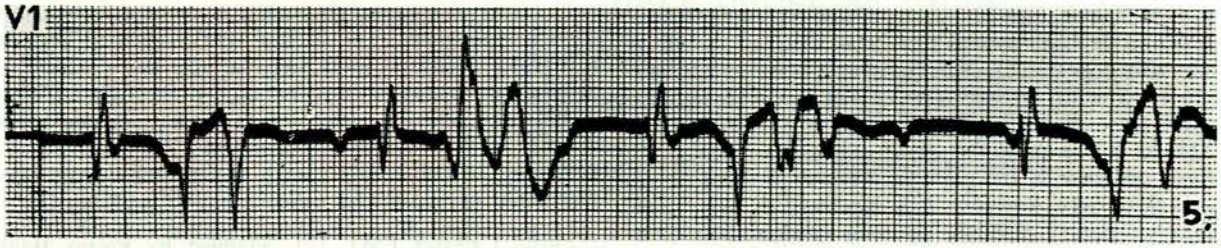

Fig. 5. ECG (lead VI) showing multiple foci of ventricular activity (case 2).

all 4 cases for periods varying from 2 to 12 weeks. In all our cases the catheter was inserted through the right external jugular vein, and no difficulty was experienced in placing the tip in the right ventricle. However, the heart should be viewed with the fluoroscope in the lateral as well as the antero-posterior position, for in the latter the catheter may appear to be in the right ventricle when actually it is in the coronary sinus. This happened in case 3, where it was found that the heart could be paced from this site, but only with a large current (about 12·5 volts) and the patient experienced some retrosternal pain. This hazard is best avoided by first passing the catheter into the pulmonary artery and then withdrawing it into the right ventricle. The patient is unaware of the pacing stimulus with a correctly positioned catheter, since as little as 1 volt will pace, though 2·5 volts is generally used to provide a margin of safety. With a portable transistorized unit patients are able to walk about. To avoid movement of the catheter, it is tied to the vein with black silk and fixed to the skin with similar material, and finally a large loop of catheter is strapped to the neck. Dangers of the method are perforation of the heart, infection, and thrombus formation.[2] We have experienced none of these complications when using a No. 5 rather than a No. 6 catheter and maintaining the patients on prophylactic penicillin. We have not used anticoagulants and pulmonary embolus has not occurred. Stable rhythm has been attained in all 4 patients, with cessation of Stokes-Adams attacks, whether owing to ventricular asystole (cases 1 and 4) or ventricular fibrillation (cases 1, 2 and 3). Release from the fear of these attacks has engendered great confidence among all concerned, not least in the patients themselves. Because of the theoretical risks, however, this method has been used only as a temporary measure pending implantation of a pacemaker. We have found it so satisfactory that we consider that for this purpose it should always be used. With a suitable external unit, it is possible that the route could be used indefinitely. However, in case 4, when the catheter was removed after 15 days, a fine crack in the nylon coating was present where it was strapped to the skin. Electrode failure may therefore be a hazard with long-term use, although this difficulty can perhaps be overcome by using the very flexible catheter†† of certain Swedish workers described to us by Norlander.[19] Such a catheter can be connected either to a subcutaneously implanted battery unit or to a suitable external unit. An obvious advantage of this method of pacing is that a thoracotomy is not required.

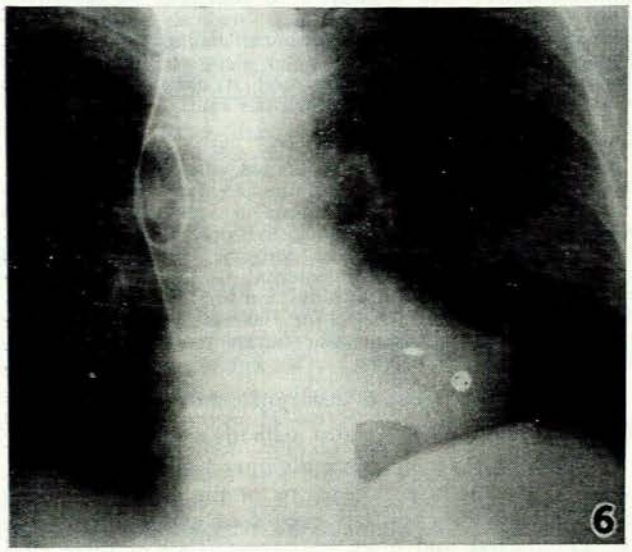

Fig. 6. Postero-anterior roentgenogram in case 1, showing a catheter electrode in the right ventricular outflow tract and myocardial electrodes sewn onto the surface of the left ventricle.

(C) *Direct cardiac electrodes connected by wires to implantable battery units.* Credit for the development of this method of pacing is due to Chardack and his co-workers.[20-22] Their introduction of platinum electrodes has solved many of the problems of this technique by providing low initial resistance with minimal increase of resistance from the effects of electrolysis. Two electrodes are sewn onto, or embedded under, the epicardium and are joined by wires to a miniature battery unit, which is implanted subcutaneously in the abdominal wall or within the rectus sheath. The wires are insulated with 'teflon' and the unit is embedded in silastic or epoxy resin to prevent corrosion. A major early problem was the frequent breakage of the connecting wires, but this complication has been minimized both by using helical coiled wires, made preferably of platinum-iridium alloy,[22] and also by looping the wires. With implanted pacemaker units the rate of pacing is pre-set or can be adjusted by a special needle. It is not possible for the patients themselves to alter the rate in order to adjust for the demands of fever or exercise, and changes in cardiac output must therefore depend solely on changes in stroke volume. Implantable pacemakers were used in all our patients and pacing has been effective and uneventful for up to 5 months in 3 of them (cases 2, 3 and 4).

(D) *Direct cardiac electrodes connected by wires to external pacing units.* With external units, provision can be made for adjustment of heart rates by the patient. The chief disadvantage is the risk of infection along the wires. This risk may be lowered by exteriorizing the wires low down in the abdominal wall,[23] and we used this system in one instance (case 1).

Surgical Technique of Inserting Implantable Pacemaker Unit

In view of the risk of Stokes-Adams attacks during anaesthesia we established a stable rhythm with a catheter electrode pacemaker before thoracotomy was performed.

The heart is exposed through a short anterior incision in the 5th interspace. The pericardium is opened only sufficiently to expose a small area of the left ventricle. The pacemaker battery unit is introduced through an incision in the left abdominal wall into a pocket formed subcutaneously at the level of the umbilicus. Alternatively it can be placed in the rectus sheath behind the rectus muscle. The wires are passed by subcutaneous tunnelling from the

††Elema Catheter EMT 570. Supplied by Protea Holdings Limited, Johannesburg.

site of pacemaker implantation to the pericardial sac. The electrodes are sewn onto the surface of the left ventricle, or are embedded into tiny stab wounds made in the surface of the left ventricle (Fig. 6). It is advisable both to sew a square of 'ivalon', $\frac{1}{8}$th of an inch thick, over both electrodes, and also to close the pericardium over the ivalon in order to maintain firm contact of the electrodes with the myocardium. The wires are made to form an upward loop within the pericardium to allow for longitudinal mobility, and a second loop close to the pacemaker unit is made for the same purpose and to facilitate reconnection in the event of its being afterwards necessary to replace the unit. The chest is drained and closed in the routine manner. When the unit is placed subcutaneously it is advisable to drain the pocket and thus decrease the possibility of serous accumulation. The patient is kept under antibiotic cover for about 10 days after the operation.

FURTHER DISCUSSION

The management of Stokes-Adams attacks consists of two basic procedures. Firstly, the restoration of an effective heart-beat and the maintenance of adequate cardiorespiratory function until this is accomplished and, secondly, the prevention of further episodes of cardiac syncope.

During any attack that does not rapidly and spontaneously terminate, sharp blows to the chest followed if necessary by external cardiac massage[29] must be performed. Direct intrathoracic cardiac massage may be necessary if the external method fails.[32]

Methods of long-term electrical pacing of the heart have proved a great advance in the management of the complications of atrioventricular block. It is now possible to salvage many patients and return them to a useful life. Heart block may occur in patients with relatively healthy hearts,[2] and for them there is now the possibility of a normal lifespan.

We agree with Portal *et al.*[2] that catheter electrode pacing is ideal for emergency therapy. Confidence is gained by the patient after this very minor procedure, and adequate time becomes available to plan the future management. This method of treatment should now be applied to most patients experiencing Stokes-Adams attacks. It can even be used for patients with acute myocardial infarction who develop Stokes-Adams attacks.[28]

At the present time direct epicardial electrodes should be used when the heart block is permanent. In 3 of our 4 cases, implantable battery units have been successful for periods of up to 5 months to the date of writing. The actual life of these units cannot be accurately determined, although the shelf life is known. The battery life is affected by the rate of stimulation and by 'current drain', which will vary with rise of threshold at the electrodes, but it is anticipated that the batteries will last from 2 to 5 years. The units are so constructed that the timing device is affected by battery failure before the pacing circuit. The consequent rise in pulse rate should give adequate warning of impending pacing failure.

An external unit has several advantages over the implanted variety. Battery failure is avoided by charging from the mains. (The unit developed in our Cardiovascular Research Unit incorporates a meter that registers the amount of charging required each day.) Battery replacement is simple and requires no surgical procedure. Altering the rate of pacing is also easy and can be done by the patient himself. In addition to the risk of infection, however, disadvantages of an external unit are its inconvenience and the danger of breakage of the external wires.

The question of the optimum rate of pacing is not yet settled. Chardack[22] favours a rate of 60 per minute or less. He states that if pacing fails the time of return of idioventricular rhythm will be longer after more rapid rates of pacing and he believes that a rate of 60 per minute is adequate for most purposes. Zoll,[7] however, considers the optimal rate to be 72 per minute. In case 4 in our series we were able to vary the rate of pacing via the catheter electrode and we found that pacing at about 80 per minute produced to the least extent the irregularity that is caused by dissociation between the paced rhythm and the patient's own sinus rhythm (parasystole). Our patient with the external adjustable control (case 1) has found that he is most comfortable at a rate of 80 per minute.

Kantrowitz *et al.*[35] have described the use of direct surface electrodes connected to an implanted induction coil activated by an external unit via radio frequency currents. The heart rate can thus be adjusted by the patient and the external unit can be carried in any convenient way, e.g. in a pocket or handbag. The disadvantages and risks of exteriorized wires are thus avoided.

Much experimental work is still in progress and the future will no doubt bring many improvements. A major shortcoming of the methods we have described is the incoordination of atrial and ventricular contraction. This has been shown to result in a lower cardiac output compared to coordinated contraction at the same rate.[25] To overcome this disadvantage, Nathan *et al.*[26] have devised a pacemaker that is triggered by the P wave of the ECG via an atrial electrode. Special safety devices are incorporated to deal with atrial arrhythmias or atrial asystole. This type of pacemaker has been successfully used in 2 patients.[27] The problem of the optimal rate of pacing does not apply with a synchronized pacemaker.

To avoid the need for connecting wires, a micromodule implanted on the heart surface has been devised by Stoeckle and Schuder.[24] This method is still in the experimental stage.

These lines of research will, we believe, form the basis of future developments in the treatment of complete atrioventricular block.

SUMMARY

Four patients with heart block and Stokes-Adams disease have been treated by internal electrical pacing. This was accomplished first by catheter electrodes and later by myocardial electrodes with implantable battery units. In one case a rise in electrical resistance at the terminals necessitated the use of an external pacing unit. Various methods of internal pacing and possible lines of future development are discussed.

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REFERENCES

1. Zion, M. M. and Bradlow, B. A. (1964): S. Afr. Med. J., **38**, 144.
2. Portal, R. W., Davis, J. A., Leatham, A. and Siddos, A. H. A. (1962): Lancet, **2**, 1362.
3. Zoll, P. M., Linenthal, A. J., Gibson, W., Paul, M. H. and Norman, L. R. (1958): Circulation, **17**, 325.
4. Linenthal, A. J. and Zoll, P. M. (1963): *Ibid.*, **27**, 5.
5. Lillehei, W. C., Bilgutay, A. M., Varco, R. L., Long, D. M., Bakken, E. E. and Sellers, R. D. (1962): *Fetschrift George E. Fahr* Minneapolis: Lancet Publications.
6. Murray, J. F. and Boyer, S. H. (1957): Circulation, **15**, 547.
7. Dimond, E. G., Bay, E. H., Chardack, W. M., Dack, S. and Zoll, P. M. (1963): Dis. Chest, **43**, 456.
8. Prinzmetal, M. and Kennamer, R. (1954): J. Amer. Med. Assoc., **154**, 1049.
9. Gilchrist, A. R. (1960): Brit. Med. J., **1**, 215.
10. Aber, C. P. and Wynn Jones, E. (1960): Brit. Heart J., **22**, 723.
11. Doll, J. L. and Buchanan, J. (1962): Lancet, **2**, 8.
12. Zoll, P. M., Linenthal, A. J. and Norman, L. R. (1954): Circulation, **9**, 482.
13. Zoll, P. M., Linenthal, A. J. and Gibson, W. (1955): Arch. Intern. Med., **96**, 639.
14. Zoll, P. M., Paul, M. H., Linenthal, A. J., Norman, L. R. and Gibson, W. (1956): Circulation, **14**, 745.
15. Weirich, W. L., Gott, U. L. and Lillehei, C. W. (1958): Surg. Forum, **8**, 360.
16. Levowitz, B. S., Ford, W. B. and Smith, J. W. jnr. (1960): J. Thorac. Cardiovasc. Surg., **40**, 283.
17. Reiff, T. R., Oppenheimer, J. and Ferguson, G. (1957): Amer. Heart J., **54**, 437.
18. Furman, S. and Schwedel, J. B. (1959): New Engl. J. Med., **261**, 943.
19. Norlander, O. P. (1963): Personal communication.
20. Chardack, W. M., Gage, A. A. and Greatbatch, W. I. (1960): Surgery, **48**, 643.
21. *Idem* (1961): J. Thorac. Cardiovasc. Surg., **42**, 814.
22. Chardack, W. M. (1963): Dis. Chest., **43**, 225.
23. Elmquist, R., Landegren, J., Pettersson, S. O., Senning, A. and William-Olsson, G. (1963): Amer. Heart J., **65**, 731.
24. Stoeckle, H. and Schuder, J. C. (1963): Circulation, **27**, 676.
25. Mitchell, J. H., Gilmore, J. P. and Sarnoff, S. J. (1962): Amer. J. Cardiol., **9**, 237.
26. Nathan, D. A., Center, S., Chang-You-Wu and Keller, W. (1963): *Ibid.*, **11**, 362.
27. *Idem* (1963): Circulation, **27**, 682.
28. Samet, P., Jacobs, W. and Bernstein, W. H. (1963): Amer. J. Cardiol., **11**, 379.
29. Kouwenhoven, W. B., Jude, J. R. and Knickerbocker, G. G. (1960): J. Amer. Med. Assoc., **193**, 1064.
30. Zoll, P. M. (1952): New Engl. J. Med., **247**, 768.
31. Zoll, P. M., Linenthal, A. J., Gibson, W., Paul, M. H. and Norman, L. R. (1956): *Ibid.*, **254**, 727.
32. Zoll, P. M. and Linenthal, A. J. (1963): Circulation, **27**, 1.
33. Meinhardt, K. and Robinson, H. A. (1962): Psychosom. Med., **25**, 325.
34. Fleming, H. A. and Mirams, J. A. (1963): Lancet, **2**, 214.
35. Kantrowitz, A., Cohen, R., Raillard, H. and Schmidt, J. (1961): Circulation, **24**, 967.