Long-Term Electric Stimulation of the Heart for Stokes-Adams Disease *

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STOKES-ADAMS disease consists of unpredictable disturbances of ventricular rhythm in patients with atrioventricular block that produce cerebral ischemia manifested by dizziness, unconsciousness, convulsions, or death. The heart block may be transient or permanent, and partial or complete. The cardiac mechanisms causing the attacks may be very slow idioventricular rhythm, ventricular standstill, ventricular tachycardia, or ventricular fibrillation.

Emergency resuscitation from seizures can be accomplished by external electric stimulation and external electric countershock.[22, 23] The major remaining problem in this disease is the prevention of unpredictable recurrent seizures, for which drug therapy is often inadequate. Replacement of an unreliable intrinsic ventricular pacemaker with a reliable electric one that would drive the ventricles continuously and indefinitely would assure complete prevention of seizures, whether due to ventricular standstill or fibrillation.[23] Long-term stimulation can be carried out directly with electrodes placed on or in a ventricle without the pain and skin irritation that make external electric stimulation impractical for this purpose and at current levels sufficiently low to permit the use of portable and implantable power sources.[21]

This paper presents a method for long-term direct electric stimulation of the heart. The rationale and experimental basis of the method are given with technical details of its application and clinical experiences in 14 patients.

Myocardial Electrodes. A rise in the electrical threshold for myocardial stimulation has been the major obstacle to the clinical application of this approach. We have studied this problem experimentally during the past four years. Stimulus threshold was measured in voltage and amperage in repeated tests in 43 dogs with sino-atrial rhythm during periods up to 16 months after implantation of myocardial electrodes. At times the electrode wires were connected to a small electric pacemaker for continuous stimulation; usually, however, they were placed subcutaneously and were aseptically re-exposed for periodic measurements of the threshold.

Many variables in the electrode system have been studied. Electrodes were placed in the ventricular cavities, in the ventricular myocardium, on the epicardium, and on the pericardium at various areas of the heart. Materials used for the electrodes included tantalum, stainless steel, vitallium, a conductive plastic, copper, platinized platinum, and gold and platinum of very

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high purity. The wires from the electrodes were usually of No. 0 braided stainless steel, insulated with polyamide (Nylon), methylmethacrylate, polyethylene, polyvinyl, and the fluorocarbons polytrifluorochloroethylene (Kel-F) and polytrifluoroethylene (Teflon). These materials were sometimes also used as plates to carry the electrodes. Electrodes of various sizes and shapes were affixed to the surface of the heart by sutures or perforated carrying plates. Bare ends or segments up to 1.0 cm. in length were inserted at varying depths into or through the myocardium. Sutures used to hold the wires or plates in place were of silk, Dacron, fine tantalum wire, and glass fiber or fine stainless steel wire coated with Teflon.

The initial thresholds for stimulation depended on electrode location and size but not on the materials used. With point electrodes on the epicardium or in the myocardium, initial values were as low as 0.1 milliampere and 0.3 volt. Initial thresholds with larger epicardial or myocardial electrodes or with electrodes in the ventricular cavity or on the pericardium were higher. The initial value, however, was not reflected in the final level.

Threshold amperage and voltage started to rise within one week after placement of electrodes. At times, three- to 10-fold rises within a month were followed by stabilization at levels below 6 milliamperes or 6 volts, which were considered the practicable limit. In other experiments, thresholds rose excessively, usually within a few weeks but occasionally not for a few months. The thresholds rose or remained stable whether or not stimulation was applied. The rise in threshold appears to result from a reaction of the cardiac tissue to the implanted foreign body and not to injurious effects or polarization due to the electric stimuli. Both voltage and current rose proportionately, indicating that there was no increase in electrical resistance but a decrease in effective current density at the excitable myocardium.

Several materials gave plateau levels that remained satisfactory for four months and longer, the most consistently satisfactory being platinum (Table 1). The longest satisfactory level is 2.0 milliamperes and 1.3 volts in a dog in which loop-shaped platinum electrodes were sutured on the epicardium over 16 months ago. Failure to obtain long-term satisfactory thresholds

TABLE 1. *Stimulus Threshold Experiments*

Electrode Material	No. Experiments	Satisfactory Thresholds* No.	
Platinum	8	5	*Electrode placement*
			Epicardial loops
			Epicardial Teflon plates
			In myocardium
Platinized platinum	1	1	*Sutures*
			Teflon-coated stainless steel
			Silk
Gold	5	1	Tantalum
			Teflon-coated glass fiber
			Insulation
Tantalum	12	2	Teflon
			Kel-F
			Silicone rubber
Stainless steel	9	2	
Total	35	11	

* Thresholds remained below 6 milliamperes and 6 volts over 4 months.

when they might have been expected resulted from wire breakage, dislodgement of the electrodes, gross infection, and marked inflammation suggesting a foreign body reaction. Four of the trials with stainless steel were with the electrodes devised by Hunter *et al.*;[9] two gave satisfactory results but two were failures, due to displacement of the electrode or marked tissue reaction at the site of implantation.

It has been suggested[5, 9] that bipolar myocardial electrodes (both in close juxtaposition) permit long-term stimulation at much lower thresholds than do unipolar electrodes (negative on the myocardium and indifferent at a distance). In our experimental and clinical experiences and in those of Lillehei *et al.*,[12] there has been no significant difference between bipolar and unipolar thresholds. Two electrodes in the heart do provide a safety factor: if the threshold rises in one electrode, stimulation may continue through the other. It has also been suggested[5] that the threshold for stimulation is lower in the presence of complete heart block than during sino-atrial rhythm. We found no such differences in repeated tests in a dog with transient complete heart block and in a patient with varying conduction.

On the basis of these experimental studies, for clinical trial we chose bipolar electrodes of platinum wire imbedded in the left ventricular myocardium for maximal mechanical stability, fixed in place with fine Teflon-coated braided stainless steel sutures, and connected to the pacemaker by multistranded stainless steel wire insulated with Teflon. At the time of our first clinical experience, Hunter had maintained successful stimulation in a patient for over a year using bipolar stainless steel electrodes held in a silicone rubber base,[9] and Chardack *et al.*[5] had also used this electrode successfully in several patients. We, therefore, imbedded the Hunter electrodes as well as our own in our first three patients. We stopped doing so when our own electrodes proved satisfactory, easier to insert, and free of the rigidity and foreign body mass of the Hunter electrodes.

Route of Insertion of Electrodes. We accept the need for thoracotomy for myocardial implantation of the electrodes in order to achieve their optimal placement and fixation. Exposure of the heart also permits early control of cardiac rhythm with a temporary epicardial electrode.

It has been suggested that full thoracotomy be avoided by implanting the electrodes in the anterior right ventricular surface under local anesthesia and through a small extrapleural pericardial exposure.[12] This limited exposure restricts the choice of implantation site and does not provide adequate access to the heart for resuscitation in case of cardiac arrest. Furthermore, implantation of the electrodes in the thin right ventricular wall rather than the left carries greater risks of hemorrhage and rupture.

Thoracotomy has been avoided by inserting a wire into the myocardium by blind percutaneous puncture[18] and by passing a venous catheter with a wire into the right ventricular chamber.[7] These approaches appear undesirable for long-term use because of the risks of infection, thrombosis and embolism, hemorrhage, cardiac injury and arrhythmia, and loss of electric contact with slight movement of the wire.

Connections from Electrodes to Pacemaker. Conducting wires from the myocardial electrodes through the skin to externally carried pacemakers, as originally applied by Weirich *et al.*[19] are useful in the transient heart block that may follow cardiac surgery but involve significant risks infection and of breakage and other accidental loss of electric contact. These limitations make this technic unsatisfactory for the usual patients with chronic atrioventricular block and Stokes-Adams disease in whom long-term stimulation is desired. External wires carry the additional risk of ventricular fibrillation when instru-

ments using line power are attached to them. Oscilloscopes, galvanometers, electrocardiographs, and pacemakers, unless battery-powered, should be carefully grounded to prevent leakage of small, but potentially lethal current to the heart.[7, 14, 21]

The passage of wires through the skin may be avoided by the use of an induction circuit or radio-wave transmission across the skin. Early in our work we developed a small, transistorized, battery-powered pulse generator and radio frequency transmitter that transmitted a signal through the skin to a small subcutaneous receiver (a diode detector) from which wires delivered the stimulus to the heart. This approach has three major attractions: the subcutaneously implanted receiver may be very small (0.9 × 2.5 cm.), the external power source may be readily replaced, and the rate of stimulation can be varied. We found this approach feasible experimentally,[23] and others have applied it clinically and experimentally.[1, 8] The primary disadvantage of this technic is the need for constant close apposition of the external and subcutaneous components, for slight, unavoidable displacements interrupt stimulation and carry the risks of recurrent Stokes-Adams attacks.

We therefore prefer to lead the wires from the heart to a pacemaker buried under the skin even though minor surgery will be needed to replace the pacemaker before the batteries fail, at intervals estimated at present to be five years. In addition, the patient is freed of the continuous concern with cardiac action and electrical equipment inescapable in systems that depend upon external units.

The Pacemaker. We and others [5] have built small pacemakers containing long-life batteries for subcutaneous implantation and have found their use feasible in dogs and in man. One may anticipate implanting a rechargeable battery and receiver with the pacemaker; at convenient intervals an external radio-frequency transmitter would be used to send a recharging current through the skin. At the present stage of development rechargeable units are too short-lived to offer any advantage.

We propose to drive the ventricles continuously and indefinitely at a fixed, predetermined rate regardless of the presence or absence of intrinsic ventricular activity. Continual stimulation does not require monitoring or automatic triggering devices; it provides an uncomplicated and reliable means of preventing recurrent Stokes-Adams attacks. It has been suggested that externally carried pacemakers may be turned off and on by the patient as needed.[1, 2] This practice appears unwise because of the unpredictability, abruptness of onset, and hazard of death of Stokes-Adams attacks. Suppression of intrinsic ventricular activity or competition between intrinsic and artificial pacemakers does not create clinical difficulties; some irregularity of rhythm may occur but is not significant; although the stroke volume of ectopic ventricular beats may be somewhat diminished, adequate circulation can be maintained.

Although variability of the rate of stimulation has certain advantages, selection of a fixed rate makes for greater simplicity and reliability, and for smaller size of the pacemaker. For longest battery life it is desirable to set the rate as low as possible compatible with satisfactory cardiac output and prevention of seizures. The usual idioventricular rate of 25 to 45 beats per minute in patients with complete heart block is adequate to prevent cerebral symptoms and is compatible with normal activity for many years. Chardack [5] has therefore set his pacemakers at about 50 per minute. Functionally, however, such rates are insufficient in that cardiac hypertrophy almost invariably occurs, and limitation of exercise tolerance and congestive failure often develop. It would seem, therefore, that driving the heart at a fixed rate between 70 and 90 beats per minute would provide a greater cardiac output with

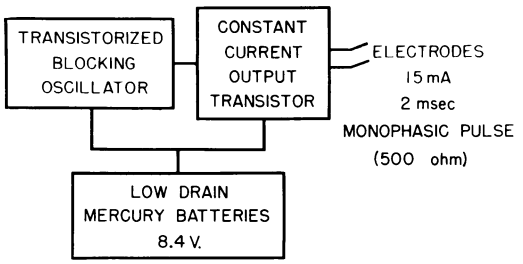

FIG. 1. Block diagram of pacemaker circuit.

smaller stroke volume and would more adequately meet increased demands upon the heart, as with exercise. Rates in this range were also found more reliable in preventing recurrent ventricular tachycardia and fibrillation; in occasional patients, however, rates up to 108 per minute were required.[23]

A number of workers have suggested the desirability of driving the heart at the "natural," physiologically varying sino-atrial rate rather than at an arbitrary fixed rate.[6, 10, 17] Following the lead of Butterworth and Poindexter,[4] they have by artificial electronic means used the atrial impulse to stimulate the ventricle. These artificial atrioventricular conducting systems involved two sets of wires through the chest wall and external electronic amplifiers and other devices. In addition, safeguards were perforce included in the systems to prevent ventricular slowing or tachycardia in response to atrial arrhythmias. It would seem, therefore, that the theoretical advantages of a physiologically varying rate over a fixed one are not sufficient to justify the complicated systems necessary at present.

The Present Pacemaker-Electrode Unit. On the basis of the foregoing considerations a pacemaker * was designed small enough to be implanted subcutaneously (6.5 × 6.0 × 1.7 cm. and 170 Gm.) that continuously and at a fixed rate delivers monophasic impulses two milliseconds in duration, eight volts in intensity, and approximately 15 milliamperes in current flow across the usual resistance (500 ohms) between the electrodes (Fig. 1, 2). The batteries consist of six low-drain mercury cells, each rated at 1,000 milliampere-hours, and are estimated to last approximately five years when the rate of discharge is set at 70 per minute. As the batteries begin to fail, the rate of discharge will slow; furthermore, as the current falls to threshold levels, the ventricles will not respond to all stimuli, so that the ventricular rate will slow still more. Nevertheless, it is recommended that a new pacemaker with fresh batteries be inserted well before failure is expected.

Electrical parts are embedded in a nonconductive epoxy resin ** and enclosed in a steel case coated with Teflon or Kel-F, or, more recently, in a smoothly finished case molded of an epoxy resin known to be tolerated well by human tissues.*** [16] Before implantation, for easy fixation of the pacemaker to the tissues, the instrument is encased in an envelope of Teflon mesh that is sewn together with Teflon-coated stainless steel sutures.†

The electrode wires emerging from the box are of multistranded stainless steel †† insulated with seven to ten layers of Teflon. Fifteen inches from the pacemaker unit the ends of the wires are silver-soldered to 1.0 cm. lengths of uninsulated pure platinum wire 0.025-inch in diameter.††† These platinum segments are the electrodes to be implanted. Another four-inch segment of insulated steel wire is joined to each platinum wire and to a stainless steel ⅜-circle taper point needle 3.1 cm. from tip to hub

* Pacemaker-electrode unit manufactured by Electrodyne Company, Norwood, Massachusetts.

** Scotchcast Resin No. 5, manufactured by Minnesota Mining and Manufacturing Co., St. Paul, Minn.

*** Resinweld No. 620, Medical Grade, manufactured by H. B. Fuller Co., St. Paul, Minn.

† American Silk Sutures, Inc., Roslyn Heights, N. Y.

†† No. 0 New Construction Wire, manufactured by American Cyanamid Co.

††† Reference grade (more than 99.999 per cent pure) annealed platinum wire from Sigmund Cohn Corp., Mt. Vernon, New York.

for introducing the electrodes into the myocardium. The two electrode wires leave the pacemaker unit enclosed in a silicone-rubber tube ¶ $\frac{1}{16}$ inch in internal diameter and $\frac{1}{32}$ inch in wall thickness that is molded into the case of the pacemaker and terminates two inches proximal to the electrode segments.

Because of the occasional foreign body reactions observed in the experimental studies, special care is taken to avoid contamination of the electrodes with lint, dust, oils, and other foreign materials. The ends of the electrode wires and myocardial sutures are boiled in a solution of Ivory Flakes for 15 minutes, and rinsed thoroughly with distilled water.

Since the pacemaker cannot be heated, the pacemaker and electrodes are then sterilized by immersion in beta-propiolactone § for 20 minutes at room temperature.[3] Great care is taken in handling this chemical because of its irritant and necrotizing properties. The instruments are then rinsed thoroughly in sterile saline solution, isopropyl alcohol, and again in saline solution, to hydrolyze the beta-propiolactone to inert products and to remove alcohol-soluble polymers; beta-propiolactone has been reported to be carcinogenic.[15] The Silastic tube containing the electrode wires is punctured with a fine needle near the pacemaker, and its entire length is irrigated thoroughly in this same sequence to ensure sterility of this blind tract. Each electrode is then covered with a protecting length of previously cleansed and sterilized Silastic tube, and the whole unit is wrapped in sterile nonconductive, powder-free plastic film before being packaged for delivery to the operating table.

An insulated cable about nine feet long leading from two spring clips on one end to a plug that fits into the socket of a battery-powered pacemaker §§ is also sterilized with beta-propiolactone. This cable is used to drive the heart directly during the surgery before implantation of the permanent unit.

When the myocardial electrodes and the pacemaker are implanted in separate procedures or the pacemaker is to be replaced, the two sets of connecting wires are joined in a solderless connector * with a crimping tool and sealed with silicone rubber.** The connector and the sealing agent have been tolerated well in tissue. The connectors,

¶ Rway Silatube, Reiss Mfg. Corp., Little Falls, N. J.

§ Supplied by courtesy of Dr. Jules Kleiner, American Cyanamid Co. and Dr. John T. Murphy, Massachusetts General Hospital; may also be procured from Testagar Corp., Detroit, Mich.

§§ TR-3, Electrodyne Co., Norwood, Mass.

* Solderless Krimp Connector No. 34070C, American Pamcor, Inc., Havertown, Penn.

** Medical adhesive type A, Dow Corning Corp., Midland, Mich.

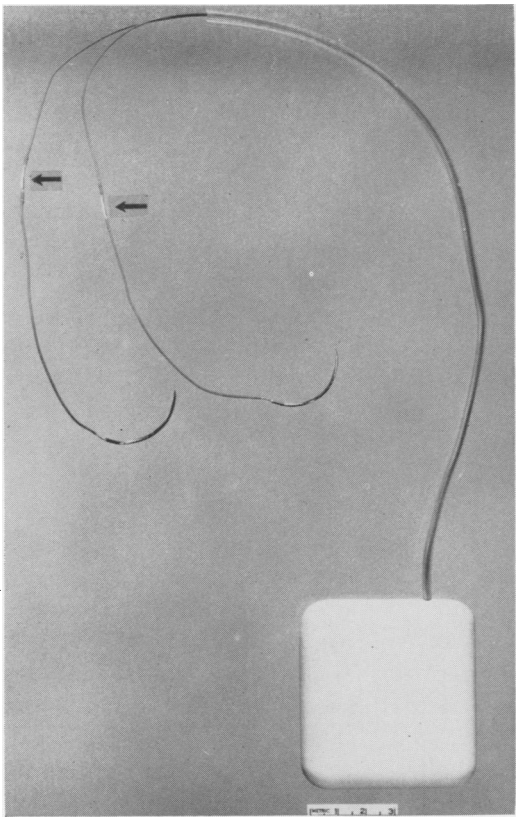

FIG. 2. Photograph of myocardial electrodes (arrows) and implantable pacemaker.

crimping tool, and sealing agent are sterilized by autoclaving.

Surgical Considerations. Ability to control the ventricular rhythm at all times is of major importance in any operation upon a patient with heart block.[20] Until the permanent pacemaker is operating, it is desirable to drive the ventricles continuously at 60 to 90 beats per minute to prevent standstill, to reduce multifocal activity,[23] and to increase cardiac output. Cardiac arrest due to ventricular standstill or ventricular fibrillation may occur abruptly at any stage of the procedure, even from partial block or from normal conduction. Our first patient developed ventricular standstill, and our second patient developed ventricular fibrillation during the induction of anesthesia. We, therefore, do not start anesthesia until the external electric pacemaker and defibrillator are ready for instant use, the surgical table is laid out, and the surgeon is ready to open the chest hastily for cardiac massage, if external measures fail.

Patients with Stokes-Adams disease are monitored continuously during hospitalization with a pacemaker-monitor; *** in the operating room, after the first stage of anesthesia is induced, the electrodes are moved from the precordium, where they interfere with preparation of the surgical field, to the upper arms, where they monitor cardiac action but are not useful for stimulating the heart. Two long subcutaneous needles (3½-inches, 18- or 19-gauge, spinal needles with shafts Teflon-coated to the terminal half inch) are introduced anteriorly at sites away from the line of the proposed incision, and are advanced in the subcutaneous tissue toward the heart with care to avoid deep penetration. The bare hubs of these needle electrodes are attached to the leads of an external pacemaker and the ability to drive the heart externally is established before tracheal intubation. Alternatively, the heart may be driven with an electrode in the esophagus behind the ventricle or in a venous catheter in the right ventricle together with an indifferent surface electrode. When the heart is exposed, a negative wire is clipped to the pericardium or to an epicardial tab and a positive wire is attached to an exposed muscle so that the heart may be driven directly without skeletal contractions until the permanent electrodes are implanted.

The heart is exposed widely through an anterolateral incision in the fifth left intercostal space. The skin incision is kept horizontal instead of following the interspace, to maintain an intact skin and subcutaneous tissue covering for the pacemaker unit, which is placed against the upper anterior medial wall of the axilla, behind the border of the pectoralis major muscle. It is convenient to develop the subcutaneous pocket at the outset, but if there is any difficulty with control of cardiac action, it is better to expose the pericardium at once and attach the clip leads for temporary direct cardiac stimulation. The pacemaker unit is placed loosely in its pocket and the wires are led medially deep to the pectoralis major and into the chest through the third interspace near the sternum. After the completion of electrode implantation the pacemaker is secured in its space by interrupted sutures of Teflon-coated fine surgical steel taken through the meshwork of the Teflon bag and special attention is given to hemostasis and avoidance of dead space. The electrode wires are looped in the pocket to provide adequate length for any subsequent procedure, and are held in place with sutures at the internal and external aspects of the rib cage.

The pericardium is opened over the left ventricle by a long incision anterior to the phrenic nerve, and a site is chosen where the electrodes may be implanted about 1.0 cm. apart without injury to coronary vessels and along the long axis of the heart for minimal flexion with heart beat and respira-

*** PM-65, Electrodyne Co.

tion. To prevent contamination of the electrodes with lint or powder, all intrapericardial procedures are carried out through an aperture in a large plastic sheet covering the field, the surgeons' gloves and the heart are rinsed thoroughly with saline, and Gelfoam pledgets are used as sponges. The protective Silastic tubes are removed from the platinum electrodes, the needles are touched to the myocardium as a final test that the pacemaker is emitting effective stimuli at the desired rate, and the needles are then placed in the myocardium and are pulled through until the bare platinum segments lie deep in the myocardial wall (Fig. 3). The electrode wires are fixed in place at entry and exit points by cross-stitches of fine Teflon-coated stainless steel with swaged-on needles.* The distal wires and needles are cut off, and the uninsulated surfaces of the divided ends are turned against the myocardium to avoid stimulation of the phrenic nerve. The individual wires are led in smooth arcs toward the base of the heart where they emerge from the pericardial sac. The wires in the Silastic tube are held to the pericardium with one or two silk stitches and are then led along the mediastinum in a smooth curve to their point of entry through the chest wall. The pericardial incision is closed after generous posterior counter-incision for drainage into the left pleural cavity.

Clinical Experiences

We have applied this procedure for long-term prevention of Stokes-Adam attacks in 14 patients. Our experiences are summarized in Table 2 and brief reports are presented of six cases.

Case Reports

Case 1. M. S., a 62-year-old physician, was well until April 1959 when he began to have severe Stokes-Adams attacks as often as 4 times a day. There was frequent variation from normal

* American Silk Suture Co., Roslyn Heights, N. Y.

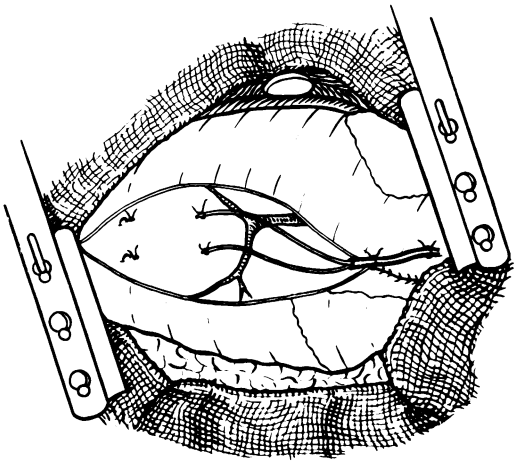

FIG. 3. Diagram of implantation procedure.

atrioventricular conduction to complete block; slow idioventricular rates (Fig. 4) were associated with weakness and sweating but the mechanism of the seizures was not documented. He was completely incapacitated by the attacks despite treatment with epinephrine, isoproterenol, ephedrine, chlorothiazide, corticosteroids, thyroid, and atropine. Although we were not sure that our technic was entirely ready for clinical application, he urged us to operate without delay because of his desperate state.

On July 20, 1960, two sets of electrodes, our own platinum and the stainless steel ones of Hunter, were implanted in the myocardium in this first case. During intubation (after induction of anesthesia with sodium thiopental and ether) ventricular standstill occurred abruptly, but prompt thoracotomy and cardiac massage restored circulation. After implantation the thresholds for stimulation with both sets of electrodes were about 1 volt. The two sets of wire were carried through the skin at separate points and the ones from the platinum electrodes were attached to a small, externally worn pacemaker.

Three months later, on October 27, the thresholds for stimulation had stabilized at 5 milliamperes and 2 volts. Consequently, on the next day, a pacemaker discharging at 65 per minute was joined to the wires from the platinum electrodes and implanted subcutaneously. The second set of wires was also placed subcutaneously. This first pacemaker was larger and more sharply edged than those constructed thereafter, and a corner of the instrument eroded through the skin in the course of several months. Subsequent attempts to close the wound and to implant another pacemaker failed because of recurrent infection. The heart is

TABLE 2. *Summary of 14 Cases*

Case No. and Initials	Age Sex	Degree of A-V Block	Duration of Stokes-Adams Disease	Ventricular Rhythms Causing Attacks		Date of Implantation	Thresholds*			Complications		Present Status
				Standstill	Tachycardia or Fibrillation		Time After Operation	Levels Milliamps	Levels Volts	Operative	Postoperative	
1. M.S.	62 M	N**, 3	17 mos.	?	?	7-20-60	0 3 mos. 8 mos.	— 5.0 4.5	1.0 2.0 2.5	Ventricular standstill	Pacemaker erosion	No Stokes-Adams attacks Effective stimulation with temporary unit worn externally
2. F.B.	60 M	1, 3	18 mos.	+	—	8-9-60	0 3 mos. 9 mos. (1)* 9 mos. (2)	— 4.0 2.8 —	1.5 1.8 3.7 >15.0	Ventricular fibrillation	Broken electrode Transient Stokes-Adams attack	No Stokes-Adams attacks Effective stimulation with implanted unit
3. A.K.	69 M	N, 2, 3	8 mos.	+	—	10-4-60	0 5 wks.	— 2.2	1.0 1.5	None	Temporary anasarca	No Stokes-Adams attacks Effective stimulation with implanted unit No water retention
4. L.G. CMC***	8 M	3	(none)			11-10-60	5½ mos.	—	5.0	None	Pacemaker failure with local inflammation	No Stokes-Adams attacks Effective stimulation with implanted unit
5. E.V.	56 F	2, 3	23 mos.	+	—	12-16-60	—	—	—	None	None	No Stokes-Adams attacks Effective stimulation with implanted unit
6. M.C. PBBH	39 F	N, 3	3 mos.	+	+	2-21-61	—	—	—	Ventricular standstill (Mitral valvuloplasty in same operation)	None	No Stokes-Adams attacks Effective stimulation with implanted unit

* The lowest monopolar or bipolar values are recorded. When the thresholds of the 2 electrodes differ widely, they are recorded separately.

** N normal atrioventricular conduction.

*** CMC Children's Medical Center, Boston. PBBH Peter Bent Brigham Hospital, Boston. NBIH Beth Israel Hospital, Newark, N. J.

TABLE 2—*Continued*

Case No. and Initials	Age Sex	Degree of A-V Block	Duration of Stokes-Adams Disease	Ventricular Rhythms Causing Attacks		Date of Implantation	Thresholds*			Complications		Present Status
				Standstill	Tachycardia or Fibrillation		Time After Operation	Levels: Milliamps	Levels: Volts	Operative	Postoperative	
7. O.S.	65 M	N, 3	9 yrs.	+	—	3-9-61	6 wks. (1) 6 wks. (2)	— —	12.0 2.0	Cardiac tamponade	Pacemaker failure with local inflammation and sepsis Ineffective electrode	No Stokes-Adams attacks Effective stimulation with temporary unit worn externally Persistent congestive failure
8. M.B.	41 F	N, 2, 3	3 mos.	—	+	3-30-61	—	—	—	None	Pacemaker sinus	No Stokes-Adams attacks Effective stimulation with implanted unit
9. L.T.	42 M	N, 2, 3	23 mos.	+	—	4-4-61	—	—	—	Ventricular standstill	Atelectasis	No Stokes-Adams attacks Effective stimulation with implanted unit
10. G.S. NBIH	60 M	3	2 wks.	—	+	4-7-61	2 wks. (1) 2 wks. (2) 7 wks. (2)	— — 2.5	9.0 2.0 3.0	None	Ineffective electrode Pacemaker failure	No Stokes-Adams attacks Effective stimulation with temporary unit worn externally
11. L.Th.	61 M	N, 3	8 mos.	+	+	4-11-61	—	—	—	None	None	No Stokes-Adams attacks Effective stimulation with implanted unit
12. J.W.	69 M	N, 2, 3	2 yrs.	+	—	4-14-61	6 wks.	3.0	1.5	None	Pneumonia Pacemaker failure	No Stokes-Adams attacks Effective stimulation with temporary unit worn externally Progressive cerebrovascular disease
13. A.B.	52 F	2, 3	2¾ yrs.	+	—	4-18-61	—	—	—	None	Pleural effusion	No Stokes-Adams attacks Effective stimulation with implanted unit
14. L.M.	65 M	2, 3	2½ yrs.	+	—	5-2-61	—	—	—	None	None	No Stokes-Adams attacks Effective stimulation with implanted unit

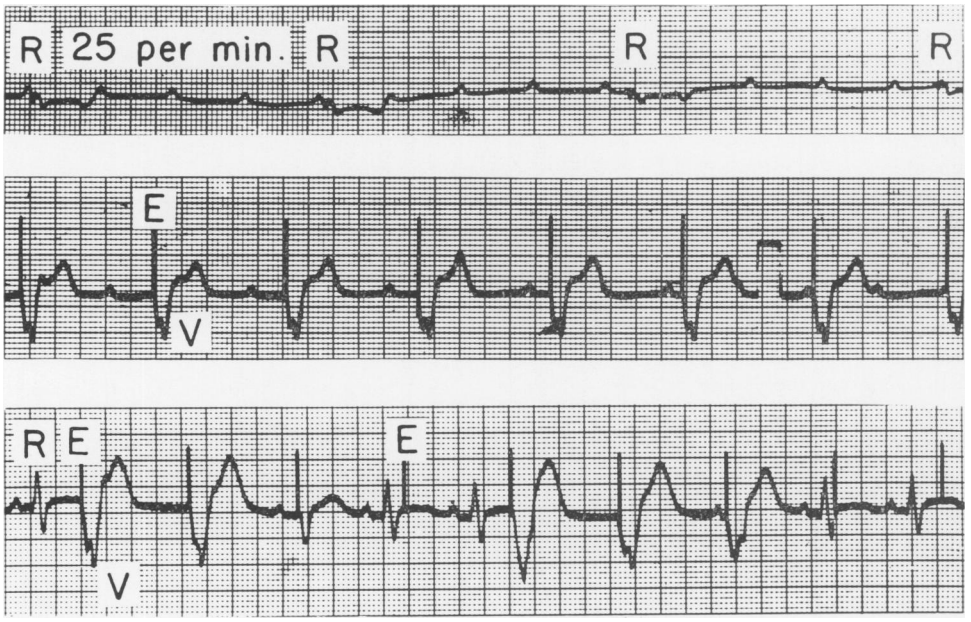

FIG. 4. Electrocardiograms, Case 1. *Top:* Complete heart block and slow idioventricular pacemaker (R) before surgery. *Middle:* Electric stimuli (E) from the artificial pacemaker produce regular ventricular responses (V). *Bottom:* Competition between conducted sino-atrial beats (R) and the artificial pacemaker. A fusion beat follows the third electric stimulus.

now being driven by an externally worn pacemaker attached to wires through the skin. When wound healing is completed, we intend to implant the pacemaker subcutaneously. The last measurements of thresholds, 8 months after implantation of the electrodes, showed no rise from the levels 5 months earlier, being 4 to 4.5 milliamperes and 2 to 2.5 volts, well below the output of the pacemaker.

The patient has not had any Stokes-Adams attacks in 10 months. At times the pulse is regular, the ventricles being driven by the electric pacemaker alone; presumable, complete atrioventricular block is present at these times. At other times the pulse is irregular because normal conduction is present and the sino-atrial and electric pacemakers compete for control of the ventricular response (Fig. 4).

Case 2. F. B., a 60-year-old man with right bundle-branch block and diabetes mellitus for years, started having repeated severe Stokes-Adams attacks in February 1959 and was hospitalized 7 times in the next year and one half. Epinephrine, isoproterenol, ephedrine, chlorothiazide, and atropine gave only temporary improvement. His conduction varied from first degree to complete block and several seizures were observed to be due to ventricular standstill.

On August 9, 1960, sets of platinum and stainless steel Hunter electrodes were implanted, the wires from the platinum electrodes were connected to a small, externally worn pacemaker, and the other wires were placed subcutaneously. Shortly after induction of anesthesia and after intravenous succinylcholine, ventricular fibrillation occurred. Prompt thoracotomy and cardiac massage restored circulation and the fibrillation stopped within a few minutes. Cardiac activity was then maintained by direct electric stimulation.

The initial thresholds of the platinum electrodes were about 1.5 volts. On November 7 the values were 4 milliamperes and 1.8 volts. Accordingly, a small pacemaker with a stimulus interval of 0.84 second, a rate of 71 per minute, was connected to the platinum electrode wires and was implanted subcutaneously.

The patient was well until May 5, 1961, when seizures recurred due to ineffective stimulation. They were stopped by stimulation through an endocardial catheter.* The pacemaker was found to be only intermittently effective and discharging at an interval of 0.75 second, or 80 per minute.

* The emergency catheterization and stimulation were performed by Drs. Claude R. Joyner and Charles Kirby, University Hospital, Philadelphia.

An x-ray showed a break in one of the platinum electrodes. On May 12, under local anesthesia the wires from the 2 sets of electrodes were exposed and the connections were removed between the platinum electrode wires and the pacemaker; no tissue reaction was found. The threshold of one platinum electrode was low, at 2.8 milliamperes and 3.7 volts, as it had been 6 months before; the threshold of the other platinum electrode varied intermittently, from 4.2 milliamperes and 3 volts to above 15 volts. The output of the pacemaker was measured across a 1,000-ohm resistance and was found to be satisfactory. These observations suggested that the broken platinum electrode caused the high threshold with resultant ineffective stimulation. The high resistance in the circuit due to the broken electrode caused the faster rate of the pacemaker. Intermittently better contacts of the broken segments gave lower thresholds and effective stimulation. The thresholds of the Hunter electrodes were high, stimulation being ineffective at 15 volts. Consequently the good platinum electrode was connected to the negative wire from the implanted pacemaker, and a bare braided stainless steel wire was sewn into an exposed muscle and connected to the other pacemaker wire to complete the circuit. Effective stimulation returned immediately at the original rate of 71 beats per minute.

Case 3. A. K., a 69-year-old man with atrioventricular conduction varying from normal to complete block, spent most of the time in hospitals from February to October 1960 because of frequent and severe Stokes-Adams attacks. His condition was so desperate that he attempted suicide. The seizures were observed to be due to ventricular standstill, which at times occurred abruptly after sino-atrial rhythm (Fig. 5). Epinephrine, isoproterenol, and atropine did not control the seizures.

On October 4, 1960, platinum and stainless steel electrodes were implanted. The wires from the platinum electrodes were connected to a small, externally worn pacemaker and the other wires were implanted subcutaneously. The thresholds for stimulation were about 1 volt. Two weeks later, however, he developed dyspnea, peripheral edema, venous distension, ascites, and pleural effusions, which did not respond to the usual treatment for congestive heart failure but cleared promptly after therapy with corticosteroids.

On November 10, 5 weeks after operation, the threshold for stimulation was 2.2 milliamperes and

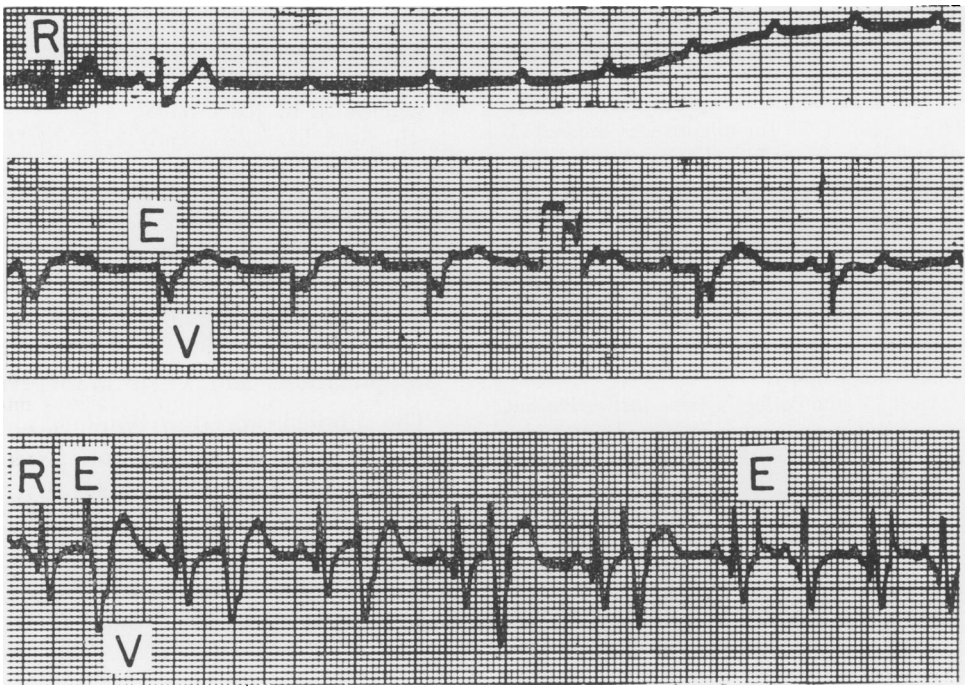

FIG. 5. Electrocardiograms, Case 3. *Top:* Ventricular standstill following two normally conducted sino-atrial beats. *Middle:* Electric stimuli (E) from the artificial pacemaker produce regular ventricular responses (V). *Bottom:* Competition between conducted sino-atrial beats (R) and the artificial pacemaker.

1.5 volts. Four days later, a small pacemaker was attached to the platinum wires and implanted. Thereafter, the patient has been well and happy, with no Stokes-Adams attacks and no limitation of activity. At times his pulse is regular with only the electric pacemaker driving the ventricles; at other times the pulse is irregular with competition between the sino-atrial and electric pacemakers (Fig. 5).

Case 5. E. V., a 56-year-old woman with heart block varying between 2 : 1 and complete, had 17 hospital admissions from January 1959 to December 1960 because of repeated Stokes-Adams attacks due to ventricular standstill that were not controlled medically.

On December 16, 1960, platinum electrodes were planted in the myocardium and a pacemaker discharging at a rate of 72 per minute was placed subcutaneously. She has been well since, without further attacks and with a regular, artificially paced rate of 72 beats per minute.

Case 7. O. S., a 65-year-old man, had occasional complete heart block with seizures for 9 years. In October 1960 the block became fixed, seizures due to ventricular standstill increased in frequency up to 20 a day and became more severe, and congestive failure developed. Digitoxin, chlorothiazide, isoproterenol, and corticosteroids were given; congestive failure improved somewhat but the seizures continued.

On March 9, 1961, a pacemaker-electrode unit with a rate of 72 per minute was implanted. The operation was complicated by pericardial tamponade from inadvertent cardiac puncture with the long needle used to drive the heart externally before its exposure. The postoperative course was complicated by severe local tissue reaction around the pacemaker followed by staphylococcal infection, azotemia, and congestive failure. Marked improvement followed local drainage and sodium methicillin (Staphcillin).

On April 17 stimulation became ineffective but an idioventricular pacemaker appeared promptly; the subcutaneous pacemaker was then removed under local anesthesia. Perforations were found through the Teflon-coated steel case and beneath them was a dead battery. These findings suggested that tiny imperfections in the Teflon coat had permitted tissue fluid to reach a chemically active layer binding the Teflon to the steel with consequent perforation of the case and battery, and marked tissue reaction. The thresholds for stimulation of the two electrodes were about 2 and 12 volts. A small externally worn pacemaker discharging at 74 per minute was attached to the good electrode wire and to a subcutaneously placed ground wire. The heart has been driven regularly by the pacemaker since then except for occasional spontaneous ventricular beats. When wound healing is complete, we plan to implant a new pacemaker subcutaneously.

Case 10. G. S., a 60-year-old man, was in good health except for right bundle-branch block until he suddenly developed complete heart block and repeated seizures due to ventricular standstill or fibrillation for which he was admitted to the Beth Israel Hospital, Newark, N. J. and resuscitated by external electric stimulation or countershock. After two weeks of seizures despite drug therapy, on April 7, 1961, a pair of electrodes and an epoxy encased pacemaker discharging at 60 per minute were implanted. Two weeks later stimulation became ineffective but an idioventricular pacemaker appeared without delay. On April 22, under local anesthesia, the wires were exposed and cut near the pacemaker. The threshold of 1 electrode was at a satisfactory level of 2 volts but the other was high at 9 volts. Effective stimulation was re-established by reconnecting the pacemaker to the good electrode wire and placing an indifferent electrode in an adjacent muscle.

On April 29 the heart rate was observed to rise slowly but progressively. After a few hours, when it was 110 per minute, the small wires were again re-exposed and divided (Drs. L. Gilbert and V. Parsonnet), and connected to an external pacemaker. A short circuit apparently had developed within the pacemaker, presumably due to permeation by tissue fluids.

His heart has subsequently been driven regularly at 62 beats per minute by an externally worn unit, and he has had no further seizures due to either ventricular standstill or fibrillation. On May 29, 7 weeks after the implantation, the threshold for stimulation of the functioning platinum electrode was 2.5 milliamperes and 3 volts. We intend to replace the implanted pacemaker.*

Complications and Their Management

The surgical procedure employed for the implantation of electrodes and pacemaker in itself produced little morbidity. Convalescence was delayed in one patient each by left lower lobe atelectasis (Case 9), left pleural effusion (Case 13), renal and electrolyte disturbance, confusion, and pneumonia (Case 12), and anasarca that cleared after corticosteroid therapy (Case 3).

* The functioning electrode became displaced by accidental traction on the wire. Therefore, new myocardial electrodes as well as a new pacemaker were implanted on June 16, 1961.

The pacemaker has been exteriorized because of local tissue reaction in three patients. A corner of the first unit implanted eroded through the skin in the course of four months (Case 1); we have since reduced the thickness and rounded the contours of the outer casing. Two early units enclosed in Teflon-coated steel boxes (Cases 4, 7) gave rise to severe local inflammation associated with perforation of the Teflon and erosion of the metal. There has been one instance of low-grade staphylococcal infection at the site of pacemaker implantation (Case 8); this has not required removal of the unit. Since shifting to an epoxy case, we have found no evidence of tissue irritation. The pacemakers in their retropectoral locations have produced little deformity, discomfort, or interference with function of thorax or arm (Fig. 6).

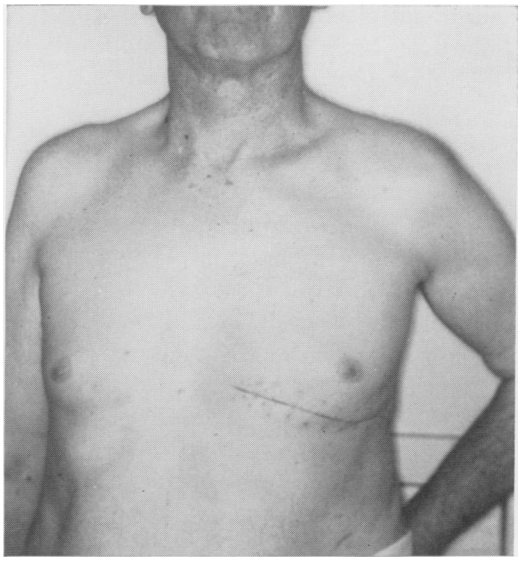

FIG. 6. Photograph of a patient with implanted electrodes and pacemaker two weeks after operation (Case 11).

Electrical difficulty with the pacemaker has occurred in four patients, manifested by ineffective stimulation in three (Cases 4, 7, 12), and by progressive increase in rate of discharge in one (Case 10). In Cases 4 and 7 erosion of the steel pacemaker cases was associated with battery failure; such units have not been used subsequently. In Case 10, the progressive increase in rate of discharge almost certainly indicated a shorting of the timing circuit probably due to permeation of the epoxy case by tissue fluid. In Case 12 the battery failure was probably also due to permeation of the epoxy casing. It is expected that this source of pacemaker failure can be avoided by improved insulation of the internal components and curing of the epoxy case.

Difficulty with an electrode has been observed in three patients (Cases 2, 7, 10). In Case 2 the platinum electrode was seen by x-ray to be broken, probably due to the use of an unusually long platinum segment. The resulting increase in electrical resistance was manifested by a stable 12 per cent increase in the rate of the pacemaker and by only intermittently effective stimulation. In Cases 7 and 10, in addition to the pacemaker failures noted above, single electrodes were found to have high thresholds for stimulation.

In watching for electrical complications single-lead electrocardiograms are taken periodically to permit accurate measurement of the interval between stimuli and to demonstrate constancy of ventricular response. Progressive increase in the rate of discharge of more than 10 per cent, or ineffective stimuli, call for immediate measures to prevent severe tachycardia or ventricular fibrillation on the one hand,* or recurrent Stokes-Adams attacks on the other. An increasing rate demands immediate division of the wires in their subcutaneous course and their connection to a properly functioning pacemaker. Ineffective stimulation also calls for division of the wires; the threshold of each electrode is tested with a variable-output battery-powered pacemaker and an indifferent electrode. If both thresholds are low, the pacemaker requires replacement. If only one electrode provides effective stimulation, its wire may be reconnected to the pacemaker, the other lead of which is attached to an

* In one patient treated elsewhere ventricular fibrillation did occur in this way.

indifferent electrode in the chest wall. The failure of both electrodes would call for re-implantation in the myocardium.

Discussion

The successful experiences in all these patients demonstrate that Stokes-Adams attacks can be completely prevented by continuous, long-term, direct electric stimulation of the heart. The artificial electric pacemaker with a rate fixed at about 70 per minute provides a cardiac output sufficient for ordinary activity and prevents seizures due to ventricular standstill.* It also prevents seizures due to ventricular tachycardia or fibrillation (Cases 6, 10); occasional patients may require a faster pacemaker.[23] In patients with complete heart block the electric pacemaker provides a regular ventricular rhythm (Cases 2, 4, 5, 7, 10, 12, 13, 14). In patients wtih intermittent atrioventricular conduction competition occurs between the sino-atrial and artificial pacemakers (Cases 1, 3, 6, 8, 9, 11); the resultant irregularity is of no clinical consequence.

Surgical implantation of cardiac pacemakers was undertaken in these patients only because seizures continued with desperate frequency and severity despite ephedrine, isoproterenol, atropine, corticosteroids, and chlorothiazide. When such drugs prevent seizures completely, surgical intervention is not necessary. In the one patient without Stokes-Adams disease (Case 4), the poor prognosis associated with postoperative heart block[11] was the indication for the implantation.

It would seem desirable to have ready at all open-heart operations a pair of myocardial electrodes, such as our platinum ones, with which long-term stimulation may be carried out in case of heart block. An externally worn pacemaker can be attached to the wires to drive the heart for a few weeks. If atrioventricular conduction returns, the wires may be cut and placed under the skin. If heart block persists, however, a second thoracotomy is not necessary, but a long-term pacemaker need only be attached to the wires and inserted subcutaneously.

More difficult is the decision about surgery in the intermediate group of patients with attacks that are relatively infrequent or apparently mild. In these cases the risks of surgery must be balanced against the risk of recurrent attacks, an evaluation that is made difficult by the characteristic unpredictability of the frequency and severity of Stokes-Adams attacks. If our experience continues favorable as the period of observation lengthens, it may well be appropriate to consider surgical treatment in every patient with Stokes-Adams disease no matter how mild.

Congestive heart failure in patients with complete heart block even in the absence of Stokes-Adams attacks constitutes another indication for surgery. Implantation of an artificial pacemaker at a rate fast enough to provide a more adequate cardiac output would improve congestive failure, increase exercise tolerance, and decrease cardiac size. One such patient has already been treated successfully.[13]

The technical aspects of the procedure will, undoubtedly, be modified with further experience. The surgical approach seems satisfactory and the morbidity due to thoracotomy is not excessive. At least one electrode has remained effective in every patient thus far. The initial stimulation threshold of our cylindrical platinum wire electrode is somewhat higher than that of point electrodes, but is well within the acceptable range. It appears to provide good stability in implantation, a safer, larger area of contact with the myocardium, and little intrapericardial foreign material.

A smaller pacemaker would be desirable, especially for use in children. Until new developments in battery construction permit the packaging of equivalent amounts of electrical energy in smaller space, the size

* Case 1 has subsequently undergone prostatectomy and Case 14 cholecyestectomy without difficulty.

of the pacemaker cannot be reduced significantly without reducing the magnitude or duration of the power output. Our 2-millisecond, 15 milliampere pulse is of optimal duration and provides a three to five-fold margin of safety for unusual rises in threshold. Reduction of duration or amplitude of the stimulus, of rate of stimulation, or of life of the unit seems undesirable. If a new electrode system were found that provided stable thresholds of 2 milliamperes or less, a satisfactory margin of safety might then be attained with smaller batteries.

Summary

We have developed a technic for implantation of a pacemaker-electrode system for long-term direct electric stimulation of the heart. It has been applied successfully in 14 patients for periods up to 12 months to prevent Stokes-Adams attacks due to ventricular standstill or fibrillation. Two important precautions in the procedure are to maintain continuous control of cardiac rhythm and to avoid contamination of the electrodes and of the site of implantation with foreign material of any kind.

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Discussion

Dr. Elliott S. Hurwitt: The authors are to be congratulated on their many worthwhile contributions to this important problem, and particularly on the development of this present device. The hazard of infection associated with wires emerging from the skin is obviated by the various implantable pacemakers.

The apparatus developed by Mauro and Glenn at Yale possesses the additional advantage of radio beam controlled variability in rate and intensity as the clinical situation changes, rather than constantly driving the heart along a predetermined course. The ultimate in artificial pacing should include these criteria, while avoiding the necessity for formal thoracotomy, and anesthesia, and the risk of wire breakage anywhere in the circuit.

Since July 1958, 25 patients have been treated by the intracardiac catheter pacemaker developed by Furman at Montefiore. This experience is described in detail in the January, 1961, issue of *Surgery*. The catheter is threaded through the external jugular vein and the tip positioned in the outflow track of the right ventricle, as in a conventional right-heart catheterization.

No significant increases in current have been required in periods of intermittent pacing up to 22 months, to date. The patients are fully ambulatory and some have returned to work. The equipment is under direct control at all times, and when pacing is no longer required—as may occasionally occur—removal of the apparatus may be accomplished without an operation.

Heart block produced during open heart surgery has also responded to this technic. Disadvantages include the need for long-term anticoagulant therapy, and the risk of infection at the site of catheter emergence for which prophylactic antibiotics may not always be effective.

A word of caution and surgical restraint may be in order. The vast majority of the large number of people with heart block, including most of those subject to Stokes-Adams seizures, will respond to the skillful manipulation of a variety of medical measures. The ready availability and ease of insertion of electronic pacing devices could lead to their indiscriminate and unnecessary use in vulnerable cardiac patients inappropriately selected.

Dr. Howard A. Frank: [Closing] We have followed Dr. Hurwitt's reports with great interest, and I think he has presented clearly the separate advantages of the two methods. We have used the intracardiac catheter electrode successfully to tide over several instances of emergency arrest, and also to maintain ventricular action during the early stages of the implantation procedure.

We like the implantation procedure in part, as I have said, because the patients are free of external wires and equipment and, as time goes by, begin to forget their pulse rates and hearts. This is undoubtedly a secondary advantage, but a real one nonetheless. We also, as I have said, do not like to put our patients in charge of their own pulse rates, so to speak, although Mr. Abrams of Birmingham tells me his patients like to be able to speed their hearts when they are going to be active, and slow them for sleep. He prefers to stop pacing during periods of returned conduction. We have given our arguments against this. Of course, our procedure depends upon a thoroughly reliable implanted pacemaker. We are given reason to believe that the pacemaker difficulties described can be fully overcome. Otherwise the argument would become stronger for not implanting the power source and timing circuit.

In infants and children, it is hard to find a proper place for the implantation of our relatively large pacemaker. One solution, described in the text, would be to reduce the battery size and expect to change the unit every year or two as the child grows. However, a safe margin must be maintained to deal with the occasional rise in stimulus threshold, and the threshold change is likely to occur within the first three to six weeks after electrode implantation. Implanting just the electrodes and a secondary coil, and stimulating by induction, would be an answer to the problem of limited space for implantation in the child. I do not know how difficult or otherwise disadvantageous it would prove to maintain the external components of the induction circuit in proper position upon an infant or child.

As one who has come only lately to the problem of Stokes-Adams disease, I must say that I have become aware how very distressing an illness it is, and how bad its prognosis despite good medical management. Although people may tolerate heart-block without apparent difficulty for long periods, very few survive five years once Stokes-Adams seizures begin. The threat of sudden unconsciousness or death is always present whether the interval between seizures is long or short. Although the electrical stimulation methods have not been in use long enough to show an effect upon survival rates, it is clear that the life of the patient is greatly improved and may become normal.