# Surgical Treatment of Adams-Stokes Syndrome Using Long-term Inductive Coupled Coil Pacemaking: \*

Experience with 30 Patients

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NEARLY 150 years ago the Surgeon, Robert Adams<sup>3</sup> and the Physician, William Stokes <sup>28</sup> gave the first complete account of the syndrome which bears their names although Marcus Gerbezius (1719), Morgagni in De Sedibus (1761), and Thomas Spens (1793) had made prior complete descriptions. Four years after Adams' report was published in Dublin, Michael Faraday elucidated the principle of electromagnetic induction (1831), but almost 100 years passed until the principle was employed to stimulate living tissue, Loucks (1933), Light and Chaffee (1934), and Fender (1936) and these efforts were directed primarily to neural tissue stimulation. Utilization of the principle of electromagnetic induction in the treatment of Adams-Stokes

\* Submitted for publication March 26, 1963.

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Supported in part by The Endowment Fund, United Birmingham Hospitals, The Queen Elizabeth Hospital, Department of Surgery, University of Birmingham, Edgbaston, Birmingham, England, Joseph Lucas, Limited, Birmingham, England, and the USPHS Grant HF-17, 767. syndrome was first reported in  $1960^{1}$  by Abrams *et al.* and later by the same investigators in 1961.<sup>2</sup> This report presents the subsequent experience of this endeavor involving the surgical management of 30 patients with complete heart block employing electromagnetic induction coupled coil pacemakers.

#### History

John Aldini, nephew of Galvani, reported on the conceivable use of electric shock as a means of resuscitation in 1819,<sup>4</sup> apparently having applied an electrode directly to the heart without eliciting contractions in a criminal two hours dead (1802). Vassali in the same era was able to elicit contractions under similar circumstances. In 1932, Hyman<sup>11</sup> made a plea for the use of an artificial pacemaker for the arrested heart and quoted 65 papers concerning electrical pacemaking from 1862 onward.

### Background

Callaghan and Bigelow<sup>5</sup> reported, in 1951, on artificial electrical pacemaking for cardiac standstill and in 1952, Zoll<sup>32</sup> described successful treatment of ventricular arrest complicating complete heart block employing an external pacemaker. After the advent of open-heart surgery with total cardiopulmonary bypass and resultant *surgical* heart block following repair of ventricular septal defects, Weirich, Gott and

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Fig. 1. Inductive Coupled Coil Pacemaker System. The implanted secondary coil consists of 1,000 turns of No. 38 standard wire gauge copper wire flat wound, ring shape air cored, with an inner aperture of 2.0 cm. The coil is fitted with braided 26-gauge stainless steel output leads swedged onto curved atraumatic needles for myocardial insertion (resistance 95 ohms). The coil and its leads are encapsulated in a silicone-rubber compound. This secondary coil measures 3.0 cm. in diameter, 0.5 cm. in thickness and can be autoclaved. The external primary coil consists of 50 turns of No. 36 standard wire gauge enamelled copper wire flat wound, ring shaped, air cored, potted in epoxy resin with twin leads of high grade flex wire for connection to the pulse generator (resistance 2 ohm). The inductive system pulse generator. Three types of pulse generators have been employed, each evolving from the prior, with increasing miniaturization of components, compactness, and portability (Fig. 2–4).

Lillehei demonstrated, in 1958, that such heart block could be successfully treated with suture electrodes in the ventricular muscle brought out through the chest wall to a pacemaker. The same year, Thevenet *et al.*<sup>29</sup> described the percutaneous insertion of a myocardial electrode and in 1959, Furman and Schwedel<sup>9</sup> described the transvenous passage of an endocardial electrode in the form of a modified Cournand cardiac catheter.

#### Development of the Method

Complete heart block may be symptomless, disabling because of low cardiac output, or a threat to life because of repeated Adams-Stokes attacks. In the review of 251 cases of complete heart block by Penton, Miller and Levine,<sup>24</sup> the average duration of life after the first syncopal attack was approximately three years and after the first appearance of complete heart block approximately two years. Thus it became apparent that if a satisfactory system with long-term pacemaking could be evolved, a group of patients would be greatly helped.

In our early experience employing a modified external indirect method of Zoll,<sup>\*</sup> it was found that repeated shocks in the range of 65 volts necessary for pacemaking were both painful and irritating because of secondary skeletal muscular contractions and consequently unacceptable as a system for long-term stimulation. This type of stimulation has been used continuously, however, for 14 days, and intermittently for eight months (Zoll and Linenthal, 1960<sup>33</sup>).

The use of the direct method with myocardial electrodes implanted at thoracotomy and brought out through the chest

<sup>•</sup> The application of metal plate electrodes to the skin of the intact chest over the apex of the left ventricle and the area over the second interspace just to the left of the sternum with a D.C. pulse of 10 milliseconds duration variable between 0 and 180 volts.



# The relay operated pacemaker

Fig. 2. (Prototype Relay Pulse Generator, Mark I). In this version, the double wound 5 milliampere relay is employed as a blocking oscillator transformer in conjunction with the transistor T1. The pulse rate is established by the values of R1, R2 and C. The pulse width, that period during which the relay contacts S1 close, depends on the electrical and mechanical characteristics of the relay. The output transistor T2 serves as a heavy current switch, the D.C. resistance of which is controlled by modifying the bias current with adjustment of the power control R4. The inductive voltage swing, generated as the field of the load coil collapses, momentarily exceeds the specified maximum collector voltage of the transistor. In practice no difficulties arose from this. In view of the low average power, no heat sink is required on T2.

wall is limited in regard to long-term pacing because of the possibility of infection traveling from skin to myocardium. Lillehei has, however, reported continued success at a 15-month interval.

Use of the percutaneously inserted direct myocardial wire has been limited because the dangers accompanying insertion without direct vision (bleeding, injury of a coronary artery or vein, and the possibility of a free-moving, unanchored wire in the myocardium).

The employment of an endocardial modified cardiac *catheter* electrode for longterm pacemaking is undesirable because of the constant danger of loss of pacemaking due to shifting of the catheter tip in the right ventricle or perforation resulting in loss of pacemaking with or without hemorrhage. The necessity of long-term anticoagulation with its inherent risks is a second factor.

Thus it became apparent any system employed should satisfy certain simple criteria: 1) no wire leads extending through the integument and chest wall which could act as a portal of entry for infection; 2) simplicity of electronic design with a minimum number of components located internally; 3) complete control of pulse stimulus (rate, duration and strength); and 4) compactness. The inductive coupled coil pacemaker system satisfies these criteria.

#### Characteristics of Induction as Employed

Our experience has shown that allowing for a 2.5–3 cm. separation of the external primary and internal secondary coils with some unavoidable lateral co-axial movement, pulse power in the range of 30–50 watts is needed from the pulse generator to the external coil. Greater instantaneous power than this appears for a brief period due to the oscillatory inductive swing at the trailing edge of the applied square wave. This inductive pulse communicated by transformer action stimulates the myocardium.

Myocardial impedance has been measured and estimated to be in the range of 140–200 ohms. This is in agreement with the findings of Lillehei. The pulse width of the square wave of voltage applied across the load coil is not critical and stimulation is effectual at these powers from  $\frac{1}{2}$  to approximately 5 milliseconds. The pulse duration need only be of sufficient length to store adequate energy in the magnetic field. The system, therefore, is basically a transformer. The pulse wave at the myocardium is always of an alternating form and theoretically this should minimize polarization of the myocardial electrodes and in turn minimize an increasing power requirement with time. There are indications with this system at this juncture that after approximately 15-18 months of inductive coupled coil pacemaking the power demands are increased by an estimated 15 per cent.



The non-relay version of the pacemaker

FIG. 3. (Prototype Non-relay Pulse Generator, Mark II). In this commercial<sup>®</sup> version Transistor T1 with the transformer L1 and L2 form a blocking oscillator, the rate of which is controlled by manual adjustment of the variable resistor R2. The output P-N-P transistor, of very low impedance when bottomed, is normally biased off and lifted into full conduction by the controlling pulse derived from the tertiary winding L3. The power source is a six volt nickel alkali battery giving 48 hours of operation on a 12 hour charge. An audible click generated in the crystal microphone insert can be switched off.

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# Management of Patients

The symptom complex consisting of sudden loss of consciousness and convulsions with prior and subsequent bradycardia has been loosely referred to as Adams-Stokes syndrome with attacks. Actually, sustained runs of multiple ectopic ventricular beats, ventricular tachycardia, fibrillation, standstill, or sudden onset of auricular dysrhythmias with a drop in cerebral blood flow may result in a seizure (Parkinson et al. 1941<sup>23</sup> and Clark and White, 1952). Each of the 30 patients in this current series has shown an electrocardiographic confirmation of complete (third degree) heart block except for one who was in sinus rhythm between Adams-Stokes attacks.

In managing a patient with complete heart block and a history of Adams-Stokes attacks, it is important to realize that any single episode can be fatal. In the care of such cases without pacemaker control this should be continually borne in mind.

All patients after arrival by ambulance or helicopter are admitted directly to an intensive therapy unit or cardiac surgery recovery room where facilities for intermittent and continual electrocardiographic monitoring are available. Frequently associated congestive cardiac failure, emphysema, bronchitis, diabetes, benign prostatic hypertrophy in this group may require concomitant therapy.

By history, physical examination and electrocardiographic analyses, exactly how the patient fits into the schema is determined before medical management is undertaken.\*

Sympathicomimetic amines are relatively contraindicated if attacks of circulatory arrest are due to any of the forms of ventricular hyperirritability. In comparison, sympathicomimetic amines are effective in the management of symptomatic complete heart block with circulatory arrest secondary to ventricular asystole. Isopropylnorepinephrine (Isoproterenol, Isuprel, Isoprenaline) sublingually in the range of 10–20 mg. every three hours is employed. Some patients at this stage have reverted to sinus rhythm and demonstrated an underlying prolonged P-R interval with left or right bundle branch block pattern.

Management of an Adams-Stokes Attack. Should an attack occur after admission, its exact underlying cause and duration is determined and confirmed with electrocardiographic analysis. It is most likely that ventricular asystole will last only a few seconds. If asystole (rather than ventricular *flutter*, tachycardia or fibrillation as de-





#### The current Shockley Diode Pacemaker

FIG. 4. (Shockley Diode Type Pulse Generator, Mark III). In this commercial version <sup>•</sup> a charging resistor supplies an exponentially rising voltage to a 20 microfarad paper-type capacitor. The resistor is variable and controls the rate. A four-layer diode, capable of passing 30 ampere pulses is connected across the capacitor. When the capacitor is charged to the "switch on" voltage of the diode, the former is discharged through a load coil which functions as the primary coil of the pacemaker. Immediately following this discharge the capacitor voltage rises again exponentially. A crystal-type sounder can be optionally switched into the circuit to indicate the pacemaking rate.

Specifications: 1. Pulse character, single oscillatory swing of bidirectional voltage. 2. Pulse width, in the order of  $\frac{1}{2}$  millisecond, (500 microseconds) at its base line at 8 volts. 3. Power delivered by 1° coil, in the order of 500 watts. 4. Pulse repetition rate, 30–150/min. 5. Primary power source, compact 671/2 volt high tension battery. 6. Battery life, approximately two weeks.

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termined by monitoring equipment) lasts more than 45 seconds a sharp blow across the precordium repeated several times may restore ventricular contractions. If a ventricular response is not achieved after one minute and asystole is seen to persist, intracardiac Isuprel 0.02 mg, or Adrenalin 5 cc. of 1:10,000 is injected and the patient placed on the floor for external cardiac massage (Jude *et al.*<sup>12</sup>). Our experience with external pacemaking through the intact chest wall (Zoll<sup>32</sup>) although successful in one instance has in the main been somewhat disappointing. The percutaneous modified electrode (Thevenet et al.29) has been effected on only one occasion. It is now our practice to pass an intracardiac pacemaking catheter electrode (Furman and Schwedel<sup>9</sup>) on an emergency basis. This can be done, preferably via the right external jugular vein, in 90 seconds; simultaneously an intratracheal tube is inserted for the insufflation of 100 per cent oxygen delivered at intermittent positive pressure. Satisfactory pacemaking has been achieved with the tip of the catheter in the right atrium employing a 10 millisecond pulse of 60 volts intensity.

When Should Long-term Inductive Coupled Coil Pacemaking be Established? The insertion of a permanent pacemaker is not undertaken until it has been agreed by cardiologist, internist and surgeon that the patient with multiple Adams-Stokes attacks is refractory to medical therapy. If permanent insertion is tentatively considered, ideally the patient undergoes cardiac catheterization with simultaneous intracardiac catheter electrode pacemaking to determine the hemodynamic effect of permanent pacemaking, as reflected by cardiac output studies at idioventricular rhythm and pacemade rhythm. If cardiac output is not improved and the Adams-Stokes attacks can be controlled with medical measures, insertion of a permanent pacemaker is not undertaken. The converse findings are indications for permanent in-

Short Term Intracardiac Catheter Electrode Pacemaking. During the period of intracardiac catheter electrode pacemaking, we have employed a tetracycline cover (250 mg. p.o. four times a day). Anticoagulants have not been employed. Intracardiac catheter electrodes have been left in place, ideally wedged into the tip of the right ventricle or, less ideally, in "L" position in the right ventricular outflow tract, for nine days. Advantages of this short-term intracardiac pacemaking are five-fold:

1. The patient is under external control and further Adams-Stokes attacks can be avoided.

2. The hemodynamic effects of intracardiac pacemaking can be extrapolated to the anticipated result of the proposed permanent inductive coil pacemaker.

3. During this interval those patients who are incapable of increasing their cardiac output at a pacemade rhythm come to the fore and, if Isoprenaline can control the number and severity of Adams-Stokes seizures, are excluded from operative intervention.

4. Associated congestive cardiac failure can be improved in the preoperative period with intracardiac catheter electrode pacemaking and the risks associated with operative intervention decreased.

5. Intracardiac catheter electrode pacemaking is employed as a means of external control throughout this period and, if operation is undertaken, during the precarious interval of anesthetic induction.

Following insertion of the pacemaking catheter, the decision for using permanent inductive coil pacemaking can be made more leisurely. The reappearance of idioventricular rhythm with or without Adams-Stokes attacks with a pacemaking catheter in situ is indicative of a shift of the catheter tip within the right ventricle or perforation of the right ventricle with resultant loss of pacemaking. Right ventricular perforation has occurred on three instanceslater confirmed at thoracotomy without any evidence of intrapericardial bleeding (None of these patients had been given anticoagulants during this period). In two instances pacemaking was re-established without manipulation of the perforating catheter tip by increasing the strength and duration of the stimulating pulse from 9 volts to 20 volts and 5 to 10 milliseconds.

# Electronic Systems Utilized During Procedure

In addition to the usual preparations for a left thoracotomy, seven electronic systems are simultaneously and intermittently employed during the procedure:

1. Continual electrocardiographic monitoring with visual cardioscope.

2. Photo-electric pulse indicator.

3. Intracardiac pacemaking electrode with its pulse generator.

4. Direct pacemaker electrodes for application through the open chest with their pulse generator.

5. The inductive coupled coil system with its pulse generator.

6. The direct myocardial wire system with leads to be brought out through the chest wall and its pulse generator.

7. Defibrillator electrodes with their pulse generator.

System 1. Of conventional design and essential throughout.

Sustem 2. Comprised of a light source directed through a suitable peripheral tissue into a photo-transistor. The output from the light cell is amplified by a threestage amplifier with filters to minimize artifacts. Both a meter and an audible indicator are arranged in the output. The audible note is produced by a biased-off multivibrator which the amplified pulse lifts into conduction to produce 900-cycle audible notes. The unit is powered from a rechargeable 6-volt battery and can be applied to a digit, ear lobe or cheek. Unlike the electrocardioscope it monitors both satisfactory ventricular systole and ejection.

System 3. Intracardiac pacemaking catheter electrode consisting of a number 6F Cournand catheter with a wire core and continuous metal tip. The second electrode is applied to the skin to complete the circuit. The unit delivers a 5 millisecond pulse SURGICAL TREATMENT OF ADAMS-STOKES SYNDROME

adjustable from 0-9 volts with a variable rate between 30 and 150 pulse per minute.

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System 4. Direct stimulating electrodes manually applied to the surface of the epicardium consisting of a bi-polar electrode assembly terminating in 1.5 cm. silver discs. The unit delivers a 5 millisecond pulse adjustable from 0-9 volts with a variable rate between 30 and 150 per minute.

System 5. The inductive coupled coil system previously described (Fig. 1-4).

System 6. The short-term myocardial lead system consisting of No. 32 gauge braided stainless steel electrodes with swedged-on needles and nylon insulation. The Direct Pacemaking\* power source delivers a 5 millisecond pulse adjustable between 0-9 volts with a variable rate between 30 and 150 pulses per minute.

System 7. Sterile defibrillator electrodes of conventional design for direct application to the myocardium delivering D.C. shocks over a range of 120–400 volts of 10– 200 millisecond duration and an alternative standard A.C. shock of 200 volt intensity set at 200-millisecond duration.

System 1 is essential throughout the inductive phase of anesthesia and the operative procedure. System 2 monitors satisfactory ventricular systole *and* injection. System 3 is employed both diagnostically and therapeutically during the preoperative period and is maintained until during the

<sup>•</sup> Direct Pacemaking Unit. The direct connection pacemaker serves as the pulse generator for Systems 3—Intracardiac pacemaking catheter electrode; 4—Direct epicardial contact electrodes and 6—Short-term direct myocardial lead system; and consists of a transistor multivibrator square wave generator with adjustable time constants and output voltage controls. A crystal microphone insert is employed as a *sounder*. Rate and amplitude of stimulation are manually variable. Specifications:

A.	Pulse	width
	_	

В.	Pulse	repetition	rate
-			-

- C. Ohmic output impedance
- D. Primary power source
- E. Battery life
- F. Output voltage
- 5 milliseconds 30–150/minute 1,000 ohms four 9-volt dry batteries 14 days 0–9 volts

procedure when it can be supplanted by System 4, or after the procedure when it can be supplanted by Systems 5 and 6. The maintenance of System 3 is particularly critical during the inductive period of anesthesia wherein minimal anoxia without pacemaker control can result in severe Adams-Stokes attacks. System 4 can be utilized once the chest and pericardium are opened if intracardiac catheter electrode pacemaking is unsatisfactory. This is readily assessed when the myocardium is exposed. System 5 is permanent. System 6 is employed for control during the immediate postoperative phase while edema of the operative site subsides and the patient is familiarized with the inductive coupled coil management. System 7 is kept at hand as a precautionary measure. It is not uncommon to be confronted with ventricular fibrillation just after opening the chest and before opening the pericardium.

# **Operative** Procedure

Endotracheal anesthetic induction is effected with continued intracardiac electrode pacemaking and simultaneous electrocardioscopic and photo-electric cell pulse monitoring. This is a critical period requiring extreme vigilance. The neutral electrode of the intracardiac catheter system is positioned well clear of the operative field, ideally just cephalad to the left second costal cartilage.

In the supine position with a support beneath the left scapula, the left arm is abducted to a right angle with the trunk and the photo-electric cell pulse monitor affixed to an exposed digit (Fig. 5).

A left anterolateral incision is made over the fourth intercostal space along the fifth rib in order to decrease the proximity of the incision to the implanted coil yet afford maximum exposure of the epicardium of the left and right ventricles (Fig. 6). The extents of this initial incision are the left lateral border of the sternum medially and the anterior axillary line laterally. At the



FIGURE 5.

level of the pectoral fascia (assuming satisfactory pacemaking continues), the dissection is carried superiorly to a level just above the second costal cartilage and the lateral border of the sternum medially to create a recess for the implanted coil over the second and third interspaces to the left of the sternum (Fig. 7). Inasmuch as the critical distance between the external primary and internal secondary coils is approximately 2.5 cm., in excessively obese patients placement of the coil over the



FIGURE 6.

sternum centrally and a little more cephalad minimizes the possibility of exceeding the critical distance of co-axial coil separation and renders the implanted secondary coil more easily palpable for subsequent co-axial alignment with the external coil.

In the event of unsatisfactory catheter pacemaking at any time, dissection of the internal coil recess is delayed, the chest opened immediately along with the pericardium and direct contact electrodes applied for pacemaking under visual control. Precluding this turn of events, the pectoralis major is divided transversely just above its origin from the fifth rib along with the underlying intercostal musculature, endothoracic fascia and pleura; the internal mammary artery and vein are ligated and the pericardium exposed. The pericardium is widely opened in linear fashion medial to the left phrenic nerve. At this juncture the myocardium should be examined with regard to color, consistency and form of contraction. Greatest attention is paid to the possibility of the pacemaking catheter having penetrated the tip of the right ventricle inferiorly or the outflow tract anteriorly.

The internal coil is tested by applying its needle electrodes to the epicardium 3 or 4 cm. apart and placing the primary coil wrapped in a sterile towel over the implanted secondary coil in co-axial alignment. The pulse generator to the catheter electrode or direct electrodes is switched off and pacemaking with the inductive coupled system effected. Continued satisfactory pacemaking with up to 3 cm. co-axial separation of the primary external and internal-secondary coils is essential. When this system is proved to be satisfactory, suitable placement of the internal coil is undertaken. Ideally the internal coil is placed over the pectoral muscle in such a manner that the depth of tissue overlying it is minimal consistent with continued intervening tissue viability and over that

portion of the muscle which contracts least on movement of the arm and shoulder.

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After the internal coil has been suitably tested, an aperture larger than the combined cross-sectional area of the two inductive leads is fashioned through the tissues overlying the second intercostal space into the pleural cavity and the inductive leads pulled through. The coil is placed on the pectoral muscle (or over the sternum in the obese) and the inductive leads are arranged in semi-circumferential fashion about the coil before being turned down through the chest wall aperture avoiding all sharp angulations throughout (Fig. 7). Avascular areas of the right and left ventricle are sought. The right ventricular inductive lead is implanted initially, care being taken to ensure adequate lead length to result in a gentle sweeping curve from the point of entry into the pleural cavity to the point of myocardial implantation. It is desirable that the direction of systolic and diastolic movement of the heart be observed at this juncture so that the lead can be implanted at right angles to the direction of this movement. The silicone rubber insulation is removed from the redundant portion of the lead, a rectangle of  $\frac{1}{12}$ -inch Teflon felt passed over the swedged-on needle and the needle passed through the epicardium and superficial myocardium three times to fashion an almost completely buried "Z" stitch limited to a 1 cm.<sup>2</sup> area. With the insulation abutting on the epicardium, the redundant needle and wire are cut; the Teflon rectangle is drawn down snugly over the epicardial surface above insulation and lead and secured in place with interrupted arterial suture of 5-0 black silk (Fig. 8, 9).

The inductive coil lead to the left ventricle is inserted in a similar fashion approximately 3 cm. to the left of the right inductive coil lead at the same level. At this stage the inductive lead to the left ventricle may be so close to the phrenic nerve that diaphragmatic contractions result with



FIGURE 7.



FIGURE 8.



FIGURE 9.

each inductive pulse wave. This can be avoided by stripping the phrenic nerve away from the pericardium, dislocating it more posteriorly and securing it with interrupted sutures of 4-0 arterial silk. After testing the inductive coil once again, the temporary postoperative direct connection pacemaking leads are inserted (Fig 8, 9). These are arranged in staggered fashion cephalad to the inductive coil leads so that the right direct ventricular lead is caudad and to the right of the right inductive lead: the left direct ventricular lead is caudad and between the right and left inductive leads (Fig. 8, 9). By staggering the four leads in this fashion the influence of one pacemaking circuit over the other is minimized in regard to ohmic conductive paths. Care must be taken to strip back the insulation of all leads to ensure contact of bare wire with the myocardium. Subsequent percutaneous removal of the direct leads in the late postoperative period is difficult if too tortuous an insertion or too secure a fixation has been made.

The direct pacemaking leads are brought out through the lower chest wall into the epigastrium through separate apertures. The points of exit should be far enough away from the thoracotomy to minimize the possibility of cross infection by proximity from either sites, and to allow for separate dressings. The direct leads are brought through and subsequently half-hitched over short lengths of 1/8-inch rubber tubing and the tubing sutured to the skin for additional support (Fig. 9). A flex lead then links these leads to a direct pacemaker. The pericardium is closed after a second check for diaphragmatic contracture in phase with a trial of inductive coil pacemaking. The play in the inductive coil leads between subcutaneous coil and the site of myocardial implantation is checked for adequacy and placed outside the pericardial sac. Hemostasis about the subcutaneous recess of the implanted coil is checked, the coil anchored with interrupted sutures of 4-0 black silk and the incision closed with intercostal drainage.

### Immediate Postoperative Phase

The external coil is secured to the integument overlying the implanted coil with waterproof adhesive and again tested after appropriate connection to the inductive pacemaking pulse generator. The direct connection leads are then connected to a twin lead which is linked to a direct pacemaking pulse generator. The direct leads are separately dressed from the thoracotomy incision. When it is established that both inductive and direct pacemaking systems are functioning satisfactorily, the intracardiac catheter pacemaking electrode is withdrawn from its point of insertion in the left cephalic or right external jugular vein.

During the immediate postoperative phase, control is effected with the direct lead pacemaking system. Only when the patient is able to take an active interest

in the inductive coil system (7-10 days following operation) is a trial run with the latter undertaken. Any difficulties prior to this phase with direct pacemaking may make an earlier transfer to the inductive system mandatory. There is no objection to both direct and inductive systems being utilized simultaneously inasmuch as the inductive pacemaker delivers a pulse of sufficient power to control the multivibrator direct type in current use.

After the inductive system has been employed for progressively longer intervals and a succession of nights, the direct connection pacemaker is disconnected from its pulse generator and the myocardial leads withdrawn with gentle but firm continued traction approximately one week later.

Any attempt to reduce the rate following a period of artificial pacemaking of any type is undertaken with caution, the rate being decreased in increments of five per minute every two to three minutes. A sudden decrease over larger increments is followed by an Adams-Stokes attack or its prodromata.

Clinical Summary. During the last 36month interval between January 1960 and January 1963, 30 patients have undergone operation with insertion of long-term inductive coil pacemaking system.

Age-Sex Incidence. Of this series, the incidence of operative intervention among men has been more than twice that in women (22 men and 8 women). The voungest patient was a 33-year-old man with complete heart block, an auricular rate of 85 and a ventricular rate of 26 with the onset of Adams-Stokes attacks of increasing frequency and severity over the ten months prior to admission. The oldest patient was a 76-year-old man. With two patients in the third decade, three in the fourth, eight in the fifth, nine in the sixth and eight in the seventh decades, the preponderance in the sixth and seventh decades is apparent. The average age of the

 TABLE 1. Age and Sex Distribution of Thirty Patients

 with Inductive Coupled Coil Pacemaking Systems

Age	Men	Women	Totals
30–39	2	0	2
40-49	2	1	3
50-59	5	3	8
60-69	8	1	9
70–79	6	2	8
Average	61.0	55.8	59.7

total series was 59.7. The average age of men was 61.0 and that of women 55.8 (Table 1).

# **Duration of Symptoms**

Duration of symptoms varied from ten years to one day, and ran the gamut between asymptomatic idioventricular rhythm of long duration progressing to Adams-Stokes attacks of increasing frequency and severity to an initial severe attack with prolonged asystole resulting in coma. All patients had exhibited Adams-Stokes attacks with varving periods of asystole confirmed by monitoring or direct-writing electrocardiography except for one 67year-old man with complete A-V block, an auricular rate of 60 and a ventricular rate of 27, left bundle branch block and a four-year symptom complex of orthopnea, dyspnea and peripheral edema due to low cardiac output. This patient underwent insertion of an inductive coil pacemaking system in April 1960, two days after cardiac output studies during intracardiac catheter pacemaking demonstrated that a substantial increase of cardiac output could be effected. He underwent successful prostatectomy for benign prostatic hypertrophy in September 1961. In December 1961, insertion of a second internal coil with connection to the former inductive leads became necessary. He is currently being pacemade satisfactorily with the inductive coil system 33 months following the first procedure.

# Etiology

Virtually all patients have shown varying degrees of systolic hypertension and lowered diastolic levels with increases in pulse pressures, and an initial ill-conceived impression may suggest that hypertensive arteriosclerotic heart disease has caused complete atrioventricular dissociation and Adams-Stokes attacks. However, with continued intracardiac, direct and ultimately inductive coupled coil pacemaking, systolic pressures routinely fell, diastolic pressures rose, and pulse pressures decreased, often times to normal levels. The hemodynamic effect of long-term inductive coil pacemaking on this series is being reported (Segel et al.<sup>26</sup>). In none of this current series could a congenital, luetic or traumatic basis for complete heart block be established. A rheumatic basis was implicated in three instances. One 42-year-old man with a three-year history of Adams-Stokes attacks, complete A-V block, an auricular rate of 60, a ventricular rate of 28 with right bundle branch block and left axis deviation (blood pressure 200/70) gave a history of diphtheria during the fall of 1948. This patient underwent insertion of an inductive coil system in March 1961 one day after cardiac output studies during intracardiac pacemaking demonstrated an increase in cardiac output from 4.9 L./min. at idioventricular rhythm (28/min.) to 7.93 L/min. at pacemade (90/min.) rhythm. Although the removal of the implanted coil became necessary because of infection seven months later, he continues to be pacemade on a direct myocardial wire system 22 months after the first procedure and 15 months after the second. He is scheduled for readmission for reconversion to an inductive system.

There has been a paucity of findings at autopsy to pinpoint the etiology of heartblock in the material presented for examination. In only one specimen were minute areas of fibrosis found on serial sectioning of the atrioventricular Bundle of His.

Electrocardiogram. All 30 patients exhibited ventricular rates varying between 15/min. and 36/min. with complete atrioventricular dissociation between periods of asystole, except for one 60-year-old man who was admitted with a six-day history of multiple Adams-Stokes attacks, whose electrocardiographic analysis between attacks demonstrated only sinus tachycardia with no evidence of atrioventricular dissociation. He underwent insertion of an inductive coil pacemaking system in April 1960, and was pacemade for four months without recurrence of attacks until expiration from subacute bacterial endocarditis with vegetations on a congenital bicuspid aortic value with terminal pulmonary infarction.

Timing of Operation. All operative procedures have been undertaken on an elective or semi-elective basis save for the first, wherein a 58-year-old woman with complete heart block sustained a prolonged  $(4\frac{1}{3})$  minutes) period of asystole resulting in coma and convulsions. Asystole occurred subsequently during induction of anesthesia necessitating insertion of a perpacemaking cutaneous electrode. She gradually recovered consciousness 72 hours following the procedure and although she subsequently required the insertion of a second inductive coil, she was pacemade until demise from carcinoma of the urinary bladder 32 months after the initial procedure.

Use of Intracardiac Pacemaking Catheter Electrode. Twenty of this series of 30 patients have undergone passage of an intracardiac pacemaking electrode catheter with determining of cardiac outputs and indices at idioventricular and pacemade rhythms (50, 60, 70, 80 and 90/min.). The use of this technic for simultaneous control of subsequent Adams-Stokes attacks, observation of anticipated effect with longterm inductive coil pacemaking and as an adjunct in preoperative preparation by "pacemaking the patient out of congestive

failure" is to be commended (Muller and Bellet<sup>21</sup>). Moreover, in 40 per cent of the 251 cases analyzed by Penton et al.24 congestive heart failure preceded, accompanied or followed the onset of complete heart block. Nonetheless, this is an extremely precarious period and close surveillance with continued monitoring essential. In three patients perforation of the right ventricular myocardium has occurred and although no evidence of intrapericardial bleeding was found at subsequent thoracotomy when the bare tip of the catheter was seen protruding from the myocardium after opening the pericardium, loss of optimum pacemaking control producing a potentially untenable therapeutic position made thoracotomy an urgent matter. One patient, a 72-year-old man with a one-day history of sudden onset of Adams-Stokes attacks every 60 to 90 seconds demonstrated complete atrioventricular dissociation with a ventricular rate of 36, an auricular rate of 85 and left bundle branch block. An intracardiac pacemaking catheter electrode was passed on the day of admission and he was pacemade for five days satisfactorily before demise, presumably secondary to another Adams-Stokes uncontrolled by the catheter which had probably shifted its position in the right ventricle. Operation was to have been undertaken the following day. (This myocardium at autopsy demonstrated small areas of fibrosis in the upper portion of the

interventricular septum.) Reversion to Sinus Rhythm with Intracardiac Pacemaking Catheter. One patient, a 43-year-old woman, was transferred by helicopter with an 18-month history of rapidly recurring Adams-Stokes attacks of recent increasing frequency and severity. Eye witness accounts suggested that she first became quite pale, then suddenly unconscious with no evidence of cardiac action. She then resumed a regular cardiac rhythm at about 40/min. associated with disorientation in the recovery period which lasted about three minutes. Flushing had not been noticed. There was a history of rheumatic fever at age eight. On physical examination she demonstrated an aortic ejection systolic and early diastolic murmur. Although presumably in sinus rhythm on admission the initial electrocardiogram showed complete heart block with a ventricular rate of 36 and right bundle branch block complexes. She had no further Adams-Stokes attacks but insertion of an intracardiac pacemaking catheter electrode was elected as a precautionary measure in view of the very frequent attacks she had so recently sustained. This was effected on October 15, 1961. Catheter pacemaking resulted in return to sinus rhythm with a rate of 88/min. and a normal p-r interval with only an occasional response to the pacemaker set at 72. The catheter was removed after 72 hours, and she remained in sinus rhythm for two months. Recurrence of Adams-Stokes attacks subsequently led to insertion of an inductive coil system on December 27, 1961, and she continues to be pacemade to date with no untoward effects.

Complications and Mortality. Of this group of 30 patients, there are currently 15 long-term survivors pacemaking without difficulties. Four of these 15 have required re-operation. Eleven have undergone no subsequent operative procedures other than the initial insertion. One of these has a small persistent sinus.

*Re-operations:* One male patient has undergone insertion of a new inductive coil attached to the previous inductive leads because of a broken inductive lead at the point of exit from the coil due to sharp angulation and subsequently is being pacemade satisfactorily at 33 months. A second male patient underwent re-operation and replacement of inductive coil system because of a broken inductive coil lead at the myocardial teflon interface and is pacemaking satisfactorily at eight months. A third male patient is pacemaking satisfactorily at 22 months on inductive leads converted to direct leads at 20 months after infection about the inductive coil necessitated its removal after insertion. Finally, one female patient is pacemaking at 22 months after retained direct leads "shorted out" the inductive coil leads necessitating re-operation, removal of retained direct leads and insertion of a second inductive coil system.

Mortality. Of this group of 30 patients 15 have died over the subsequent three-year period (Table 2).

# Summary of Failures and Complications

In the five instances where the cause of death was unknown, postmortem examination of the electronic components along with gross and microscopic analyses of the myocardial tissues gave no indication of the primary cause of failure. By exclusion, however, the inductive coil system should be implicated—although no means are available to prove this.

A prototype pulse generator has failed in one instance resulting in death. The responsible component was the charging capacitor. Subsequent capacitors of greater reliability were utilized.

Ingress of extracellular and interstitial fluid through the encapsulating teflon coating of a prototype internal coil resulted in the coil going *high resistance*, unsatisfactory pacemaking and subsequent death in one instance.

Infection about the inductive coil has necessitated removal in one instance and will probably result in removal and replacement in another. The importance of scrupulous hemostasis, meticulous technic and obliteration of dead space about the internal coil are apparent.

The technic of removal of the direct pacemaking leads in the late postoperative period has been described under "Operative Procedure." In one instance wherein this could not be effected, there resulted an effective *shorting out* of the inductive leads. This resulted from improper implantation of a direct lead *between* the two inductive leads in the myocardium. The retained direct lead functioned as a low resistance impedence with subsequent dissipation of stimulating inductive energy. Re-operation became necessary because of increasing power demands. The retained direct leads were removed and a new inductive coil system inserted.

Breakage of inductive coil leads has occurred in three instances. One occurred at the point of exit from the internal coil prior to passage through the intercostal tissues and turning down into the left pleura space. This was believed to be secondary to sharp angulation of the inductive lead. Insertion of a new internal coil attached \* to the old inductive leads became necessary. A second inductive coil lead broke at the Teflon rectangle-Teflon insulation interface and required insertion of a new inductive coil system. The third inductive coil lead broke at the Teflon rectangle-Teflon insulation interface and resulted in death. (No lead breakage has occurred after modification of the technic of implantation as depicted in Fig. 9 wherein the Teflon insulation extends beneath the re-inforcing Teflon rectangle and abutts on epicardium.)

### Discussion

The inductive coupled coil pacemaking system with external control of pulse rate, width, amplitude and therefore total energy is one method for the control of complete atrioventricular dissociation and the

<sup>&</sup>lt;sup>o</sup> The break was located on lateral and oblique chest films. At re-operation, the wire strands of the two leads of the new coil were inter-twined with those of the old inductive leads and suture re-enforced with number 35 stainless steel. A polyethylene sleeve was pulled over the joint and dental cement impacted down the sleeve. Silicone compound was painted over the entire joint and entrance into the pleural cavity avoided.

	Age	Sex	Date of Death	Primary Cause
1.	72	М	1 day before operation	Malpositioning of intracardiac pace-making catheter
2.	76	М	1 day	Myocardial infarction.
3.	68	М	2 days	Sudden total collapse of left lung.
4.	72	М	2 days	Respiratory insufficiency and bilateral bronchopneumonia.
5.	71	F	3 days	Undetermined.
6.	67	м	6 days	Undetermined.
7.	59	М	7 days	Ventricular tachycardia with pacemaker set a 50/minute.
8.	71	М	2 weeks	Undetermined.
9.	61	F	3 months	Broken inductive lead at teflon epicardial interface.
10.	60	М	4 months	Pulmonary infarct with S.B.E.
11.	33	М	4 months	Mechanical failur of charging capacitor in pulse generator.
12.	55	М	5 months	Undetermined.
13.	57	М	11 months	Undetermined.
14.	41	М	16 months	High resistance of internal coil secondary to ingress o extracellular and interstitial fluid.
15.	60	F	32 months	Carcinoma of bladder. (Pacemaking until demise.)

TABLE 2. Non-survivors

prevention of Adams-Stokes attacks. The stimulating power source is immediately available for examination and alteration. The radio-frequency methods employ essentially the same principles. Totally implantable systems (Senning 1959, Chardack *et al.*<sup>7</sup> and Zoll *et al.*<sup>35</sup>) while presenting the advantage of no external apparatus and obviating the possibility of loss of co-axial alignment between external and internal units with subsequent loss of pacemaking relinquish external immediate control of the aforementioned parameters.

The problem of lead breakage can be lessened by employing electrodes of sufficient tensile strength to withstand flexure attendant with cardiac motion over the years. The recently reported platinumiridium helical spring electrodes (Chardack *et al.*<sup>7</sup>) seem most promising. It is conceivable that the more recently reported method of radiofrequency stimulation employing a receiver unit attached directly to the myocardium without electrodes by Cammilli *et al.*<sup>6</sup> may prove to be the ultimate method with maintenance of external control. Further evaluation of the clinical applications of these innovations will be of interest.

We have observed no instance in this series of a "runaway" pacemaker nor any instance wherein the pacemaker has been implicated in the production of ventricular fibrillation (Zoll *et al.*<sup>35</sup>).

Asynchrony of auricular and ventricular systoles resulting from artificial pacemaking has not seemed to result in any untoward manifestations.

In certain patients of this series it has become apparent that after 15 to 18 months of continual pacemaking, the power demand at the myocardium for continual satisfactory pacemaking increases approximately 15 per cent with an apparent increase in myocardial threshold. The exact reason for this increase has not yet been definitely determined. We have postulated that this may be a manifestation either of fibrosis about the electrodes or of polarization of the electrodes. If the latter proves to be partially or totally responsible and the biphasic voltage induced at the myocardium is indeed critical in regard to the polarity of the leading edge (rising positively or rising negatively), it is conceivable that this postulated cause and observed effect resulting in an increase in myocardial threshold can be reversed—simply by turning the external coil over or reversing the two connections at the pulse generator at regular intervals.

# Summary

Experience with 30 patients undergoing long-term inductive coupled coil pacemaking for complete atrioventricular dissociation with Adams-Stokes attacks is presented.

History, evolution of the method, rationale of the therapeutic approach and the characteristics of induction as employed are presented.

The inductive coupled coil system with its pulse generators and their modifications are described.

Management of patients with Adams-Stokes attacks and considerations involved in the selection for insertion of long-term inductive coupled coil pacemakers are discussed.

Electronics systems utilized before, during and after the operative procedure are enumerated and described.

The operative procedure is described.

Clinical analyses including age and sex distribution, etiology, duration of symptoms, electrocardiographic findings, use of the intracardiac pacemaking catheter electrode and timing of operation are presented.

Analyses of morbidity and mortality are presented.

Advantages and disadvantages of currently available pacemaking systems, the problem of broken leads and the observed increase in myocardial threshold after longterm pacemaking with postulated explanations are discussed.

### Acknowledgments

The authors wish to acknowledge their appreciation to Professor A. L. d'Abreu, Professor of Cardiac Surgery and Dean of the Medical School, Professor W. Melville Arnott, William Withering Professor of Medicine, Dr. A. Brian Taylor, Consultant Physician, United Birmingham Hospitals, Mr. J. Leigh Collis, Consultant Thoracic Surgeon, United Birmingham Hospitals, Dr. N. Segel, Lecturer in Medicine, Dr. Trevor Bayley, Medical Registrar, Mr. John Shah, Thoracic Surgical Registrar, Mr. W. J. Pardoe, Medical Illustrator, Department of Anatomy, and Mr. T. F. Dee, Clinical Photographer.

### References

- Abrams, L. D., W. A. Hudson and R. Lightwood: A Surgical Approach to the Management of Heart Block Using an Inductive Coupled Artificial Cardiac Pacemaker. Lancet, ii:1372, 1960.
- Abrams, L. D. and W. A. Hudson: The Treatment of Complete Heart Block, Postgrad. Med. J., 37:240, 1961.
- 3. Adams, R.: Cases of Diseases of the Heart, Accompanied with Pathological Observations. Dublin Hospital Reports, 4: 353, 1827.
- Aldini, J.: General Views on the Application of Galvanism to Medical Purposes: Principally in Cases of Suspended Animation. London J., 1819.
- Callaghan, J. C. and W. G. Bigelow: Electrical Artificial Pacemaker for Standstill of Heart. Ann. Surg., 134:8, 1951.
- Cammilli, L., R. Pozzi and C. Drago: Remote Heart Stimulation by Radio-frequency for Permanent Rhythm Control in the Morgagni-Adams-Stokes Syndrome. Surgery, 52:765, 1962.
- Chardack, W. M., A. A. Gage and W. Greatbach: Correction of Complete Heart Block by a Self-contained and Subcutaneously Implanted Pacemaker. Clinical Experience with 15 Patients. J. Thor. Surg., 42:814, 1961.
- Fender, F. A.: Method for Prolonged Stimulation of Nervous System. Am. J. Physiol., 116:47, 1936.
- 9. Furman, S. and J. B. Schwedel: An Intracardiac Pacemaker for Stokes-Adams Seizures. New Engl. J. Med., 261:943, 1959.
- Glenn, W. W. L., A. Mauro, E. Longo, P. H. Lavietes and F. J. Mackay: Remote Stimulation of the Heart by Radiofrequency Transmission. Clinical Application to a Patient with Stokes-Adams Syndrome. New Engl. J. Med., 261:948, 1959.
- Hyman, A. S.: Resuscitation of Stopped Heart by Intracardiac Therapy: Experimental Use of an Artificial Pacemaker. Arch. Int. Med., 50:283, 1932.

- Jude, J. R., W. B. Kouwenhoven and G. G. Knickerbocker: A New Approach to Cardiac Resuscitation. Ann. Surg., 154:311, 1961.
- Kantrowitz, A., R. Cohen, H. Raillard, J. Schmidt and D. S. Feldman: The Treatment of Complete Heart Block with an Implanted, Controllable Pacemaker. Surg. Gynec. & Obst., 115:415, 1962.
- Levitsky, S., W. W. L. Glenn, A. Mauro, L. Eisenberg and P. W. Smith: Long-term Stimulation of the Heart with Radiofrequency Transmission. Surgery, 52:64, 1962.
- Light, R. U. and E. L. Chaffee: Electrical Excitation of Nervous System—Introducing New Principle; Remote Control: Preliminary Report. Science, 79:299, 1934.
- Lightwood, R.: Electronics in Surgery. J. Soc. Cardiolog. Technicians of Great Britain, 4: 33, 1959.
- Lillehei, C. W., V. L. Gott, P. C. Hodges, D. M. Long and E. E. Bakken: Transistor Pacemaker for Treatment of Complete Atrioventricular Dissociation. J.A.M.A., 172:2006, 1956.
- Loucks, R. B.: Preliminary Report of Technique for Stimulation or Destruction of Tissues Beneath Integument and Establishing of Conditioned Reactions with Faradization of Cerebral Cortex. J. Comp. Physiol. Psychol., 16:439, 1933.
- Kouwenhoven, W. B., W. R. Miler, G. G. Knickerbocker and W. R. Chestnut: Close Chest Defibrillation of the Heart. Surgery, 42:550, 1957.
- Major, R. H.: Classic Descriptions of Disease. Springfield, Ill., 1932, Charles C Thomas.
- Muller, O. F. and S. Bellet: Treatment of Intractable Heart Failure in the Presence of Complete Atrioventricular Block by the Use of the Internal Cardiac Pacemaker. New Engl. J. Med., 265:769, 1961.
- 22. Nathanson, M. H. and H. Miller: The Action of Norepinephrine, Epinephrine and Isopropylnorepinephrine on the Rythmic Function of the Heart. Circulation, **6**:238, 1952 (1962).
- Parkinson, J., C. Papp and W. Evans: The Electrocardiogram of the Stokes-Adams Attack. Brit. Heart J., 3:171, 1941.

- Penton, G. B., H. Miller and S. A. Levine: Some Clinical Features of Complete Heart Block. Circulation, 13:801, 1956.
- Schwedel, J. B., S. Furman and D. J. W. Escher: Use of an Intracardiac Pacemaker in the Treatment of Stokes-Adams Seizures. Prog. Cardiov. Dis., 3:170, 1960.
- 26. Segel, N., W. A. Hudson, J. M. Bishop and P. Harris: The Effects of Varying Heart Rate upon the Systemic and Pulmonary Circulations in Patients with Complete Heart Block at Rest and During Exercise. In press.
- 27. Stack, M. F., B. Rader, B. J. Sobol, S. J. Farber and L. W. Eichna: Cardiovascular Haemodynamic Functions in Complete Heart Block and the Effect of Isopropylnorepinephrine. Circulation 17:526, 1958.
- Stokes, W.: Observations on Some Cases of Permanently Slow Pulse. Dublin Quarterly J. Med. Sc., 2:73, 1846.
- 29. Thevenet, A., P. C. Hodges and C. W. Lillehei: The Use of a Myocardial Electrode Inserted Percutaneously for Control of Complete Atrioventricular Block by an Artificial Pacemaker. Dis. Chest, 34:621, 1958.
- 30. Weirich, W. L., V. L. Gott and C. W. Lillehei: The Treatment of Complete Heart Block by the Combined Use of a Myocardial Electrode and an Artificial Pacemaker. S. Forum, 8:360, 1953.
- Willius, F. and T. E. Keys: Cardiac Classics, St. Louis, 1941. C. V. Mosby.
- Zoll, P. M.: Resuscitation of the Heart in Ventricular Standstill by External Electric Stimulation. New Engl. J. Med., 247:768, 1952.
- 33. Zoll., P. M., M. H. Paul, A. L. Linenthal, L. R. Norman and W. Gibson: The Effects of External Electric Currents on the Heart. Control of Cardiac Rhythm and Induction and Termination of Cardiac Arrhythmias. Circulation, 14:745, 1956.
- Zoll, P. M.: Anesthesia in Patients with Atrioventricular Block. Anesthesia & Analgesia, 37:148, 1958.
- 35. Zoll, P. M., H. A. Frank, L. R. N. Zarsky, A. L. Linenthal and A. H. Belgard: Longterm Electric Stimulation of the Heart for Stokes-Adams Disease. Ann. Surg., 154:330, 1961.