

THE MANAGEMENT OF PATIENTS WITH PACEMAKERS

MEDICAL GRAND ROUNDS

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In 1952, Zoll published an original article on resuscitation of the heart in ventricular standstill. In that paper, he described the use of external electrical stimulation to provide a new mode of therapy for asystole (1). This early device provided physicians of that day with a new and dramatic therapy for complete heart block. A commentary on the efficacy of the conventional treatment for heart block at that time can be provided by a textbook of medicine from that period.

"The treatment of the more advanced states of heart block is unsatisfactory.....As a heroic emergency measure when death seems almost certain, it has been advocated that aqueous epinephrine be injected directly into the cardiac wall..... Success has been reported occasionally with other drugs, but in general they have proved disappointing and can be disregarded." (2)

(Harrison's textbook of medicine)

In 1957 complete heart block complicating open-heart surgery was successfully treated using electrodes directly attached to the myocardium (3). These electrodes were attached through the chest wall to an external transistorized pacemaker.

These early experiences indicated that electrical failure of the heart could be controlled. However, patients remained vulnerable to infections from the entry site of the electrodes and the electrodes themselves were fragile and easily broken. Spurred by these shortcomings, Chardack, Gage, and Greatbatch developed and reported the clinical utility of a self-contained, totally

implantable pacemaker (4). While these units were implantable they required a thoracotomy for attachment of the leads to the myocardium.

As the use of the cardiac pacing was extended, efforts were directed toward the development of means for implanting pacing electrodes without a thoracotomy, and in 1959 Furman (at the time a Chief Resident in Surgery at Montefiore Hospital in New York) reported the use of catheters that could be implanted in the right ventricle by way of the per venous route (5). These early experiences were soon confirmed by others (6,7,8). As a result, the electrical control of the heart - a previously formidable technical procedure - can now be initiated by physicians and paramedical personnel with limited experience.

It has been estimated that over 200,000 patients presently have implanted pacemakers (9). The present sales of pacemakers is now in excess of 80,000 per year. Although electrical pacing of the heart is a commonplace procedure, it is not without hazard, and with the rapid changes that have occurred in pacemaker technology, it may prove worthwhile to review some of the concepts that underlie pacemaker utilization and operation.

When the subject of pacemaking was last discussed at these proceedings in 1969 (10), major emphasis was placed upon the indications for cardiac pacing. In the ten years that have elapsed, the indications for cardiac pacing have expanded and the literature has grown. This review will focus on the pacemaker as a therapeutic agent with emphasis on the mechanics of the system, and follow-up.

The pacemaker system consists of two components:

- 1) a pulse generator with its component parts (battery or

power source, sensing and pacing circuits, and adjustment controls in some models) and,

- 2) the lead or wires which connect the pulse generator to the heart.

In a temporary system, the pulse generator is external and the lead passes transvenously to the endocardium, or after open heart surgery, the lead passes through the chest wall to the myocardium. In permanent systems, the pulse generator is implanted subcutaneously in the pectoral region and the electrode placed in the right ventricular apex. An alternate approach is to implant the pulse generator in the abdominal wall with the electrode passing transthoracically to the ventricular or atrial myocardium.

TERMINOLOGY:

Types of pulse generators

When only one or two types of pacemakers were available, the nomenclature was relatively simple. After 1964, however, with the rapid development of new kinds of pulse generators with various sensing and pacing modes, existing terminology and proprietary names lead to increasing confusion. It is the recommendation of the Inter-Society Commission for Heart Disease Resources that a uniform code be utilized to designate the operation of pacemaker pulse generators (11).

TABLE I

THREE LETTER IDENTIFICATION CODE

1st Letter	2nd Letter	3rd Letter
Chamber Paced	Chamber Sensed	Mode of Response
V - VENTRICLE		I - INHIBITED
A - ATRIUM		T - TRIGGERED
D - DOUBLE CHAMBER		O - NOT APPLICABLE

First letter: The paced chamber is identified by V for ventricle, A for atrium or D for double - both atrium and ventricle.

Second letter: The sensed chamber, if either, is again V for ventricle, A for atrium.

Third letter: The mode of response, if any, is either:

I for inhibited, a pacemaker whose output is *blocked* by a sensed signal, or

T for triggered, a unit whose output is *discharged* by a sensed signal.

The letter "O" indicates that a specific comment is not applicable.

Table II indicates the combinations of pacing and sensing sites with mode of response that are presently available along with mode of response that are presently available along with previously used designations.

TABLE II

Chamber paced	Chamber sensed	Mode of response	Generic description	Previously used designation
V	O	O	Ventricular pacing; no sensing function	Asynchronous; fixed rate; set rate
A	O	O	Atrial pacing; no sensing function	Atrial fixed rate; atrial asynchronous
D	O	O	Atrioventricular pacing; no sensing function	AV sequential fixed rate (asynchronous)
V	V	I	Ventricular pacing and sensing, inhibited mode	Ventricular inhibited; R inhibited; R blocking; R suppressed; non-competitive inhibited; demand; standby
V	V	T	Ventricular pacing and sensing, triggered mode	Ventricular triggered; R triggered; R wave stimulated; non-competitive triggered; following; R synchronous; demand; standby
A	A	I	Atrial pacing and sensing; inhibited mode	Atrial inhibited; P inhibited; P blocking; P suppressed
A	A	T	Atrial pacing and sensing; triggered mode	Atrial triggered; P triggered; P stimulated; P synchronous
V	A	T	Ventricular pacing, atrial sensing, triggered mode	Atrial synchronous, atrial synchronized, AV synchronous
D	V	I	Atrioventricular pacing, ventricular sensing, inhibited mode	Bifocal sequential demand, AV sequential

The most commonly utilized pulse generators today are VVI (ventricular inhibited, R wave inhibited). The coding system simply states that the ventricle is the chamber paced, and that if an

intrinsic QRS complex is sensed in the ventricle the pulse generator will be inhibited.

The original implantable pacemakers were of the V00 type (asynchronous, fixed rate, set rate). The only chamber paced was the ventricle and there was no sensing circuit. Since there was no sensing circuit, mode of inhibition was not applicable. This type of pacemaker was largely abandoned in the late 1960's because of reports of pacemaker induced ventricular tachycardia and ventricular fibrillation (12) when a pacer spike fell on the patient's intrinsic T wave. It is worth noting that this type of pacing has now reappeared in the form of newer programmable pacemakers and will be discussed later.

Subsequent pacemakers included the ventricular "triggered" pacemaker generator (VVT). The pacing site was the ventricle and that chamber was sensed to detect intrinsic ventricular activity. The "T" designation indicates that the pacemaker will discharge or trigger if an appropriate escape interval elapses or if an intrinsic beat appears. Hence a pacemaker spike will appear in an intrinsic beat as well as when a sufficient period of time elapses with no spontaneous ventricular activity. The pacing spike usually does not alter ventricular activation when it is fired into an intrinsic beat, but will often "deform" the resulting QRS complex. An example of such a pacing system is shown in Figure 1 and 2.

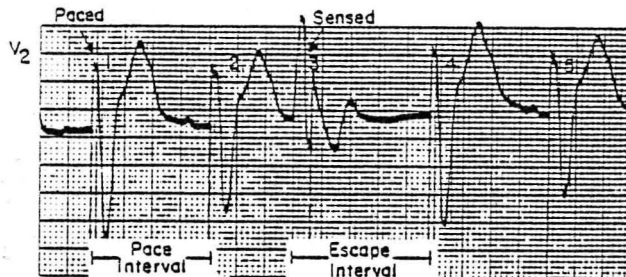


FIGURE 1

Normal R-wave synchronous (VVT) pacer function: Lead V_2 from a right ventricular endocardial R-wave synchronous pacer. All beats are paced except 3 which is sensed. The instant of sensing is late in the QRS complex (spike that deforms the S-wave) presumably because of delay in right ventricular activation as suggested by the right bundle branch block pattern. The pacer escape interval following the sensed beat and the pace interval are equal.

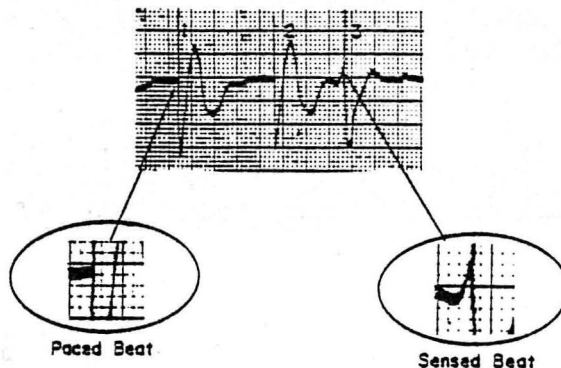


FIGURE 2

Pacing and Sensing with R-wave Synchronous (VVT) pacers: The first QRS is a paced beat. Baseline joins the pacer spike at a 90° angle (left inset). Beat 3 is sensed, the QRS begins with a positive deflection and activates the pacemaker which discharges a moment later (right inset). Careful attention must be paid to the earliest moment of depolarization when differentiating between paced and sensed beats. Observe also that the sensed beat (QRS #3) occurs "prematurely" and that its T wave is different. Differences in the appearance of QRS when visible may also be used to distinguish paced from sensed beats.

There are two common pacing systems which involve the atrium. The Cordis Atricor series (VAT) or "atrial synchronous" pulse generators sense the atrial depolarization through one electrode and after an appropriate delay, the ventricle is paced through a separate electrode. Thereby, the normal sequence of atrial and ventricular contraction is restored and the pacemaker can respond to physiologic needs by increasing its rate. Using this system assumes that the atrial rate is adequate (if it is not, the pacemaker will pace the ventricle at a preset rate).

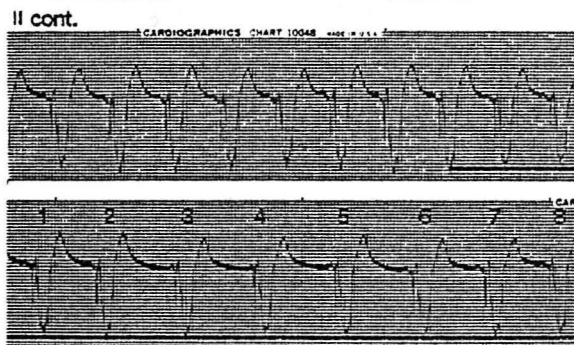


FIGURE 3. Continuous electrocardiographic strips of an atrial synchronous pacemaker (VAT). Top strip sinus rate 86/min. Each p wave is followed at 0.12 sec by a pacemaker spike stimulating the ventricle. Carotid sinus massage instituted at right of top strip results in slowing of sinus mechanism with concomitant slowing of ventricular pacing rate. As sinus rate drops below 60 beats/min., the pacemaker no longer behaves as atrial synchronous and reverts to a fixed mode ventricular pacing (V00). This property serves as a protective mechanism in case of lack of p wave sensing, sinus arrest or development of atrial fibrillation.

Another atrial pacing system is the Medtronic 5951 AD and 5995 AP series (both are AAI) or "atrial inhibited". Atrial pacing with this system can be accomplished only in patients with an intact conduction system since only the atrium is paced.

While the atrial pacing systems have their appeal from a physiologic point of view, they have limited usefulness. The AAI system requires an intact AV node, and the atrial lead is difficult to place in a reliable and stable location making a thoracotomy necessary in many instances (13).

Perhaps the most physiologic but also most complex system is the DVI ("bifocal demand", "A-V sequential") pacemaker. This system requires a pacing wire in both the atrium and the ventricle and can respond in any of the following sequences: (Figure 4).

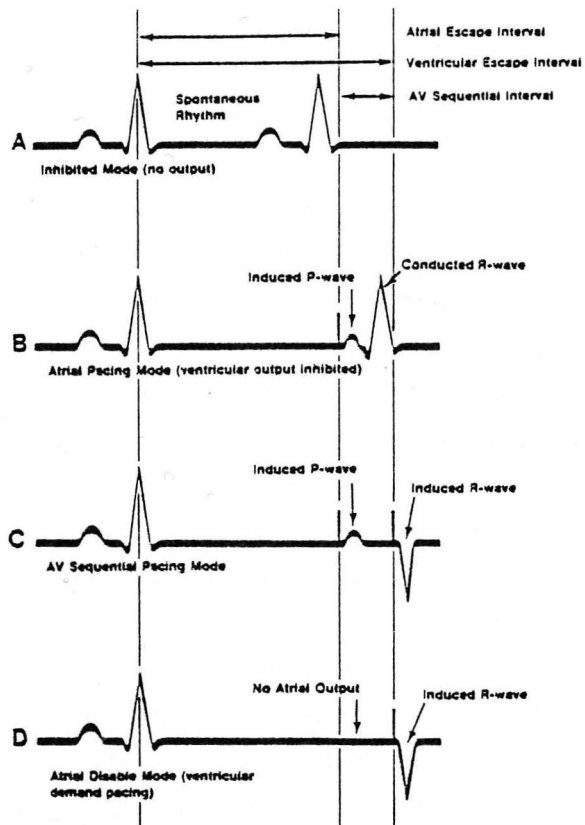


FIGURE 4

Unfortunately, catheter placement is difficult with this unit, and battery life is short (13).

UNIPOLAR AND BIPOLAR PACEMAKERS:

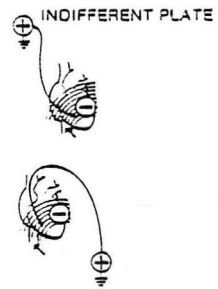
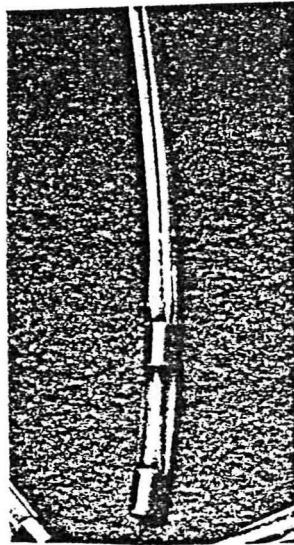
All pulse generators have both negative and positive outputs, and both must be connected to the body in order to stimulate the heart. The terms unipolar and bipolar refer to the electrode stimulating the heart and whether both positive and negative inputs lie within the ventricle or myocardium (bipolar system). The unipolar system utilizes a negative terminal to stimulate the heart with a positive terminal elsewhere in the body.

The original pacemakers used bipolar leads with external pulse generators. The present external "temporary" pacemakers are the result of years of refinement of those earlier models. Almost all temporary leads are bipolar. Permanent pacemakers are about equally divided between unipolar and bipolar systems. The unipolar and bipolar pacemakers each have their advantages.

In a bipolar lead system, a current is generated across a small distance of myocardium. (Figure 5 (a). In this circumstance the distal electrode ("tip") is the cathode or negative pole. The proximal electrode ("ring") is the anode or positive pole.

In a unipolar lead system the pacemaker electrode in contact with the myocardium is the negative pole (cathode), and the positive or indifferent electrode (anode) is located outside the heart (either as a metal plate near the pulse generator or as a direct connection to the pulse generator case). (Figure 5 (b). While it is possible to use unipolar pacing on a temporary basis, the anode would have to be placed somewhere on or under the skin surface where it would have a tenuous stability.

Figure 5



(a) Bipolar pacing electrode
(*larger than actual size*)

(b) Unipolar pacing electrode

In the bipolar system current is generated at the end of the pacing electrode where the positive and negative wires terminate.

In the unipolar pacing system, a single electrode (cathode) is in contact with the myocardium.

The stated advantages of the bipolar system include:

1. There is lower likelihood for producing unwanted stimulation of non-cardiac muscle. With a unipolar system, the indifferent electrode (anode) is usually the pacemaker case or a plate located near the pacemaker. Since the pulse generator is usually placed in the pectoral region, the proximity of the anode to the musculature of the chest wall may result in muscle stimulation which is annoying to the patient.

2. There is less chance for interference from muscle potentials and electromechanical interference (EMI). The large distance between the anode and cathode of the unipolar electrode makes it as much as 10 times as sensitive to extraneous electrical stimuli such as electrically "noisy" motors, microwave ovens, and other forms of EMI (14-16). In practice, however, the design of unipolar pacemakers has incorporated circuitry which effectively screen out "unwanted" electrical stimulation (EMI) and either rejects the signal or will cause the pulse generator to revert to an asynchronous (VOO) mode rather than be inhibited by the unwanted signal.

3. Since the bipolar electrode has two electrodes in contact with the myocardium, if one electrode becomes dysfunctional (wire breakage) the system can be converted to a bipolar system. This may be of importance in the long term management of a permanent pacemaker system.

The stated advantages for the unipolar system include:

1. There is less chance for initiating ventricular irritability (ventricular tachycardia or ventricular fibrillation)

if the pacemaker is operating in an asynchronous mode (V00) (12).

2. Although the unipolar system may be more sensitive to electromechanical interference, it is also more sensitive to the electrical signals from the heart. In a unipolar system the single electrode in the heart records the depolarization wave. Since there are no cardiac cells around the indifferent electrode, it will act as a zero potential or ground reference.

3. Voltage thresholds may be lower than bipolar units.

4. It can be argued that if one of the wires in a bipolar lead breaks, the likelihood of both wires breaking is high.

The similarities of the two systems include:

1. The threshold of stimulation is about the same for both electrodes.

2. Both can be used for asynchronous, triggered, or inhibited systems.

Competitive pacemakers and ventricular fibrillation

Electrical stimuli such as those emitted by cardiac pacemakers can cause ventricular fibrillation if they fall on the downslope of the T wave (vulnerable period, "R or T", "spike on T"). In 1973, Preston (12) reviewed all reported instances of ventricular tachycardia or fibrillation occurring in patients with artificial ventricular pacemakers. All 21 documented cases occurred in patients with bipolar pacing electrode systems and all were patients with fixed rate systems (V00). The arrhythmia occurred only when the pacing spike fell on the T wave. Preston concluded that pacemaker induced ventricular tachycardia/

fibrillation in humans was unlikely to occur in unipolar ventricular triggered or inhibited pacemakers (VVT or VVI). Subsequently Vera has reported unipolar induction of ventricular tachycardia in two patients during asynchronous pacing (72).

The distinction between unipolar and bipolar systems with reference to the likelihood of inducing ventricular tachycardia seems moot in stable patients since most pacemakers are VVI (R wave inhibited) and T wave stimulation is unlikely. However, if a programmable pacemaker is converted from VVI to VOO mode (now possible with some units) pacemaker induced ventricular tachycardia may be a potential problem again.

THRESHOLD AND THRESHOLD DETERMINATION:

The threshold is defined as "the minimum stimulation level consistently producing propagated cardiac depolarizations" (16). Threshold, therefore, is the smallest amplitude of a pacing stimulus that consistently evokes cardiac excitation.

The most common practice in testing the threshold for a temporary pacemaker is to slowly reduce the current (in milliamps or mA) until capture is lost or the lower limit of the temporary pacing system is reached (0.1 mA). The result is recorded (less than 1 mA is acceptable and less than 0.5 mA is desirable) and the pacing unit is reset to a higher threshold output to insure continued capture. This method measures the threshold current and is the only way to test threshold without using more elaborate equipment.

However, a voltage threshold can also be measured which can provide more information (and is routinely measured when a permanent pacemaker is installed). Since current (mA) is the result of input voltage (V) divided by the resistance or impedance, the recording of the current alone may give erroneous information as to the ability of a given pacemaker pulse generator to provide a predictable and dependable cardiac excitation. The voltage threshold is of particular importance when pulse generator replacement is planned and the old lead will be reutilized.

According to Ohms Law

$$I = V/R \qquad R = V/I \qquad V = R \times I$$

where I = current (mA) R = resistance (ohms) and V = voltage
Voltage loss across the interface between the pacing tip and

myocardium is a function of the electrode surface area (inversely proportional) (17), pulse width (increases for wider pulse width) and current amplitude (proportionately greater for lower current). In other words, the varying impedance (resistance) during delivery of the pacemaker pulse means that voltage and current cannot maintain a constant linear relation when there is a change in any one of the variables of the stimulating output pulse (18). For this reason, threshold measurements in volts cannot be compared with measurements in current. This is particularly important if pulse width changes. Also, in comparing one electrode system to another, impedance levels may vary so widely that a standard 5.4 volt pulse generator may deliver 15 mA to one electrode system but only 5 mA to another electrode system. Thus, a current threshold measurement of 1 mA may be very good for one system but very poor for another.

At the time of pacemaker implantation, a voltage threshold is preferable to the current threshold for several reasons: 1) the proper voltage threshold at a given pulse duration is relatively unaffected by electrode surface area; 2) the voltage output of the implanted pacemaker generator is generally fixed and as such the nominal output (usually about five volts) is what will be delivered to the electrodes. In contrast, the current (mA) of a pacemaker is usually measured at the factory across a 500 ohm resistor. ($R = V/I$; or $I = V/R$; or $I = 5/500$). Therefore $I = .01$ amps or 10 mA across the 500 ohm resistor.

On the other hand, if there were a fractured lead or break in the lead insulation the actual resistance (impedance) might be 5000 ohms which would yield a current at the endocardium of 1 mA which would not suffice for long term pacing.

In the setting of a temporary pacemaker, an increasing current (mA) requirement for capture usually means that exit block is developing for which little can be done save reposition the electrode.

It has been recommended that the acute voltage threshold for ventricular pacing be 15 percent or less of the voltage output delivered by the implanted pulse generator. Since most pulse generators have an output of about 5 volts, the acute threshold should be about .75 to .80 volts or less before being deemed acceptable. (The resistance will increase with time as the lead becomes fibrosed into its permanent position and voltage requirements will increase ($V = I \times R$).

For pacemaker generator replacement where the same lead will be reutilized (i.e. a chronic stable electrode), the chronic threshold should be less than 70 percent of the voltage output of the replacement pulse generator or about 3.5 volts or less for most current generator systems (16).

Electrode threshold during the implantation procedure is termed acute. Once threshold has stabilized, several weeks to months after implant, it is chronic. During the intervening interval, as the electrode matures, threshold is labile - tending to rise to a peak about once a week after implant and then declining progressively to a stable level which is about two to four times the acute threshold.

The use of small surface area electrodes has been utilized to reduce the impedance of lead systems and while smaller surface areas tend to result in larger increases in thresholds with maturation, the much smaller acute electrode threshold of the small (8mm^2) electrode tips tend to leave a much more acceptable chronic threshold.

In a study of 120 patients by Luceri (19), threshold behavior was evaluated for a minimum of five years after the initial pacemaker implantation (range 5 - 11.3 years). The threshold remained stable in 81 percent of the patients. Only 19 percent or 23 patients showed an increase in threshold. These data as well as other studies have been utilized to provide support for the use of newer lower output pulse generators with increased longevity. In addition, they indicate that threshold remains fairly stable and can be utilized in choosing future energy requirements when the pacemaker generator requires replacement. Starke (20) has reported similar data. These data were obtained during steady state conditions.

Interestingly even after the pacing electrode has "matured" the pacing threshold will not remain a static quantity. The list below relates some of the changes that occur with common events and with commonly used drugs (21-29).

PACEMAKER THRESHOLD CHANGES

1. Exercise ↓
2. Erect position ↑
3. Fasting ↑
4. Eating ↑
5. Sleeping ↑
6. ↑ Blood sugar ↑
7. "Polarizing solution" ↑
8. KCl ↓
9. Isuprel ↑ then > ↑
10. Other sympathomimetic amines ↑
11. Glucocorticoids (prednisone) ↑
12. Minerolocorticoids (aldosterone) ↑
13. Most anesthetic agents ↑
14. Ca⁺⁺ gluconate, atropine, digitalis, lidocaine, propranolol, procaine amide - little or no change in usual dosages.
(Propranolol, procaine amide, and quinidine may cause ↑ in high or toxic doses)

Sleeping will increase the threshold an average of 36 percent (range 30 to 41 percent); eating raises the threshold an average of 24 percent (0 - 42 percent); and exercise can decrease the threshold (-25 percent; range -11 to -37 percent).

Thus there are multiple reasons for a patient with a temporary pacemaker to develop intermittent failure to capture (exit block) if the stimulation threshold (mA) is held at the bare minimum. Since the ventricular fibrillation threshold of most hearts is some ten to thirty times the stimulation threshold most temporary pacing units will not generate enough current to incite fibrillation unless the stimulus falls on the vulnerable position of the T wave. However, in patients with badly diseased or ischemic hearts may have much lower fibrillation thresholds (29).

The use of the electrogram in inserting and troubleshooting temporary pacemaker problems

Most of us are familiar with the technique of temporary pacemaker insertion utilizing electrocardiographic monitoring. This technique can be lifesaving in a patient with an intrinsic QRS but an inadequate heart rate. It is the only safe method for "blind insertion" (without fluoroscopic control) of a pacemaker. Figure 7 illustrates the changes in the electrocardiographic trace during pacemaker positioning.

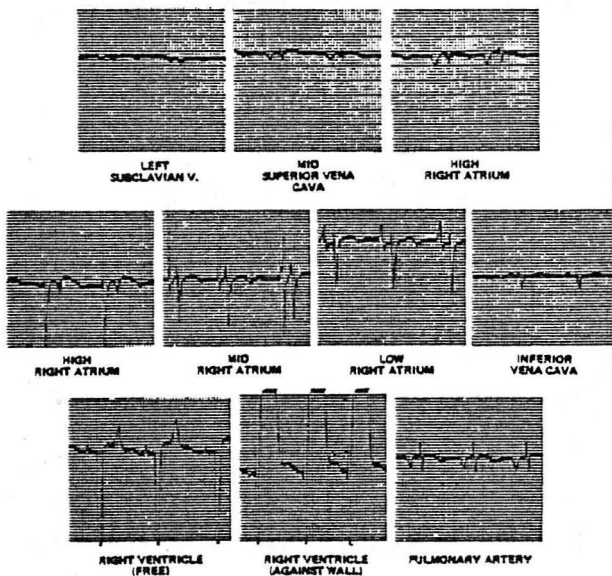


FIGURE 7

Note the large injury current when the pacemaker is in its proper position against the wall of the right ventricle. This is a normal intracardiac electrogram.

An electrogram is an endocardial ECG recording of the patient's intrinsic rhythm and QRS complex. For diagnostic purposes the electrogram should be taken in the same configuration as the pulse generator (unipolar or bipolar). Temporary pacemakers are almost always bipolar, therefore, it is imperative that the electrogram be obtained in that fashion (as well as in a unipolar configuration if desired).

Figure 8 illustrates how an electrogram is obtained.

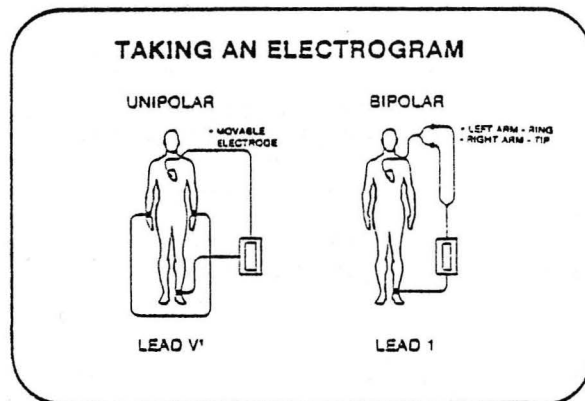


FIGURE 8

For a unipolar electrogram, the distal ("tip" or -) lead or the proximal ("ring" or +) lead is attached to the V lead of the ECG machine and the limb leads attached in the usual fashion. The V lead selector setting will then record the electrogram.

For a bipolar electrogram the left leg lead is attached to the patient, the positive electrode (anode) is attached to the left arm lead and the negative electrode (cathode) or tip electrode fastened to the right arm electrode. The lead I configuration will provide the bipolar electrogram.

A word of caution, the ECG machine must be well grounded and the exposed portions of the pacing wire must not come in contact with any potential source of electrical current.

Typical unipolar electrograms are shown in Figure 9.

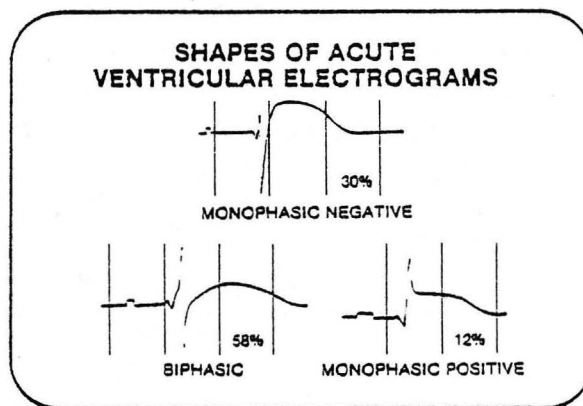


FIGURE 9

TYPICAL UNIPOLAR ELECTROGRAMS

Two comments regarding the recording of the tracing: 1) the standardization must be included since many of the complexes will be quite large; 2) the complex recorded will have no relationship to the configuration of the surface ECG since it indicates the electrical activity over a very small area of the heart.

The characteristics of the complexes recorded will be slightly different with bipolar and unipolar electrograms, although the basic morphology will be about the same.

The electrogram can indicate whether the lead position is appropriate for adequate pacemaker sensing. Figure 10 illustrates the optimal and worst lead position for a bipolar sensing system.

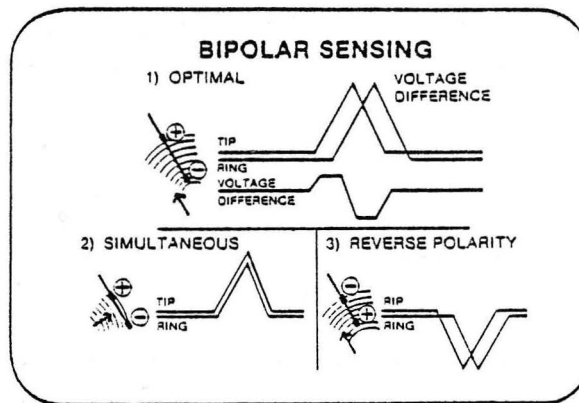


FIGURE 10

For optimal sensing, the intrinsic cardiac depolarization wave travels parallel to the direction the lead is pointing. The depolarization wave hits one electrode first, and then the other electrode. This

configuration would maximize the amplitude of the resultant R wave "seen" by the pulse generator. If the lead is positioned so that the depolarization wave hits both electrodes simultaneously, the sensing situation is poor since there is no significant voltage difference between the two electrodes.

In the unipolar system, the single electrode in the heart records the depolarization wave. Any potential difference between the voltage in the heart and the indifferent plate will be recorded. (Figure 11)

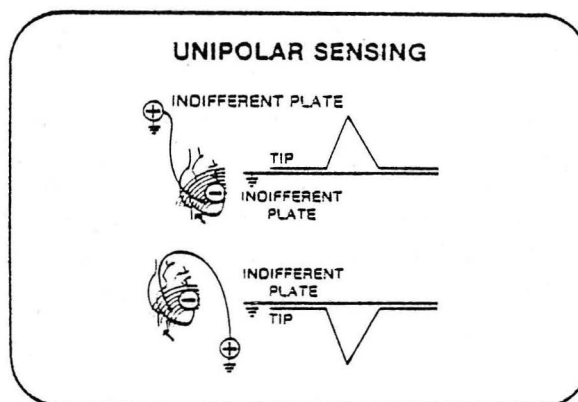


FIGURE 11

Decaprio (21) has described the characteristics of unipolar and bipolar electrograms obtained from the same catheter. These data are summarized as follows:

	Unipolar	Bipolar
R wave voltage	same	same
R wave slew rate	same	same
R wave duration		28% less
T wave voltage		34% less
ST segment elevation		37% less

The amplitude of the R wave will determine whether or not the pulse generator will be able to sense a given QRS complex. While a bipolar lead system may at times be positioned so that R wave amplitude is poor, it will tend to provide a T and P wave amplitude that are disproportionately smaller than a unipolar lead system, thereby reducing the possibility of false sensing of those complexes.

The electrogram can be useful in troubleshooting a newly installed temporary pacemaker. The sensitivity dial on the temporary pacemaker can be set by guess work (by turning the dial counterclockwise until sensing is lost). By measuring the actual voltage of the electrogram, a more accurate method for determining an optimal sensing setting can be obtained. For example, a 10 mV R wave would provide very good sensing. A 1 or 2 mV R wave would provide a very poor sensing signal and would suggest that reposition of the lead be accomplished early. An example of the latter circumstance is illustrated in Figure 12.

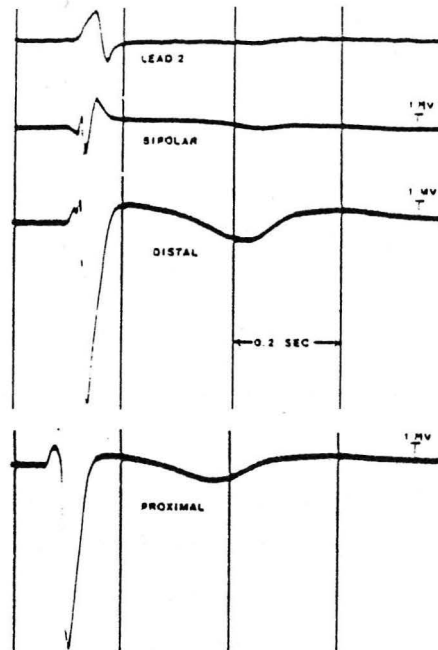


FIGURE 12

Two large unipolar electrograms on the electrode tip (distal) and ring (proximal) are oriented so that the resultant bipolar electrogram is too small to trigger the pacemaker. Correction is accomplished by conversion to a unipolar assembly (for a chronically implanted lead) or by repositioning in the case of a temporary bipolar lead.

The combination of a small R wave and a large T wave could lead to T wave sensing. This can be determined from the electrogram.

The intracardiac electrogram will change with time (Figure 13). Thus, if failure to sense becomes a problem with a temporary pacemaker, the electrogram may be helpful in determining whether a change in the sensitivity setting will be helpful or whether a change in lead position will be necessary.

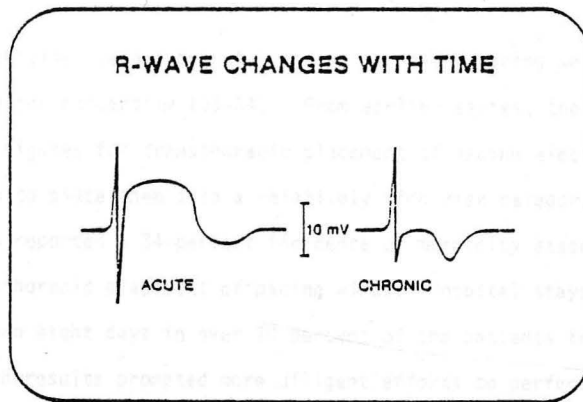


FIGURE 13

With permanent pacemakers, the data contained in the electrogram is vital for defining appropriate conditions for unioctar or bipolar pacing and sensing. Some of these functions can be performed with currently available threshold measuring devices (e.g. Medtronic Pacing System Analyzer).

Initial reports of permanent permanent pacing suggested that lead dislodgment was a common sequelae; dislodgment rates of 20 percent have been reported (17). The present acceptable upper limit for lead dislodgment and dislodgment rates is five percent. Manufacturers have suggested that newer "ring" leads have further reduced the dislodgment rate of permanent leads, but there are few data in the literature to support or refute this claim.

LEAD TYPES:

The original lead types utilized in permanent pacing were sutured to the epicardium (33-34). From earlier series, the morbidity figures for transthoracic placement of pacing electrodes would seem to place them into a relatively high risk category. The Duke group reported a 34 percent incidence of morbidity associated with transthoracic placement of pacing wires. Hospital stays were greater than eight days in over 70 percent of the patients treated (35). Such results prompted more diligent efforts to perfect the transvenous pacing wires (36-37) and prompted some to question the wisdom of the transthoracic approach to cardiac pacing.

As surgical techniques changed and new leads appeared, the morbidity rate associated with transthoracic leads has been markedly reduced (38-42). Nonetheless, the pervenous approach for permanent pacemaker placement is now the preferred approach for lead placement. Exceptions include patients in whom endocardial placement of electrodes cannot be accomplished or result in an inordinately high endocardial threshold and the prophylactic or therapeutic placement of pacing wires at the time of open heart surgery.

Initial reports of pervenous permanent pacing suggested that lead dislodgement was a common sequelae; dislodgement rates of 20 percent have been reported (37). The present acceptable upper limit for improper lead placement/lead dislodgement rates is five percent. Manufacturers have suggested that newer "tine" leads have further reduced the dislodgement rate of pervenous leads, but there are few data in the literature to support or refute this claim.

Typical leads are shown.

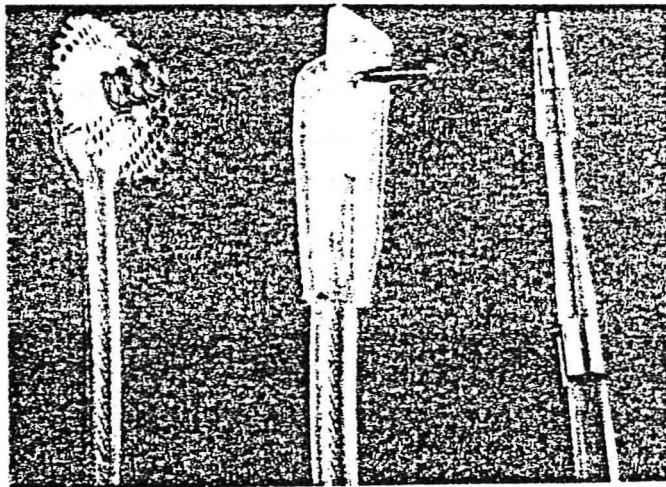


Figure 14

Electrodes types. Left, sutureless (Medtronic 6917); center, sutured epicardial (Medtronic 6913); right, transvenous (Medtronic 6901).

COMPLICATIONS:

The insertion of a pacemaker involves some surgical risk. The epicardial approach requires direct exposure of the heart, with the consequent complications expected in an elderly and often debilitated patient. The overall postoperative mortality after epicardial electrode insertion was reported to average 7.5 percent when reviewed in 1970 by Lown and Kosowsky (13). Intraoperative myocardial infarction has frequently been observed among those who have died.

Transvenous insertion of endocardial electrodes avoids many of the problems associated with thoracotomy. The transvenous route is associated with a hospital mortality rate of less than one percent and is attended by a lower postoperative morbidity.

Infection and phlebitis related to permanent pacemaker implantation are known complications of the procedure. The rate of infection is, of course, extremely dependent upon the surgeon/internist inserting the device. Rates of infection of up to 4.2 percent (23/546) have been reported as recently as 1975 (45). However, the overall rate of infection is probably much lower (46).

The occurrence of sepsis is an unusual but dreaded complication of pacemaker therapy. Occasional reports have suggested that intensive medical therapy and local I and D can suffice (47). If septicemia associated with pacemaker insertion persists, the lead and pulse generator must be removed. Since a fibrous reaction forms at the tip of the pacing electrode in the transvenous position, removal is difficult. Gentle traction has been utilized to

successfully remove imbedded pacemaker tips, but if this proves unsuccessful, thoracotomy with cardiopulmonary bypass has occasionally been necessary to remove the infected foreign body (48-49).

Pulmonary embolism and superior vena cava thrombosis were reported early in the use of pacemakers. These events have proved to be a very rare complication of transvenous permanent pacing (50-53). This was somewhat surprising in view of the association of superior vena cava thrombosis with CVP lines.

The actual incidence of upper extremity venous thrombosis in permanent transvenous pacemakers is unknown (54-55). However, Stoney, et al (56) reported a series of patients who were prospectively venogrammed at the time of elective pulse generator change. Of the 32 patients studied, 44 percent were noted to have severe (50-90%) occlusion of the innominate or subclavian vein and 21 percent had total occlusion of the vein. Of interest, however, was the observation that clinical symptoms and physical findings were rare. One patient had been hospitalized for the sudden onset of pain and edema of the left arm after four years of uneventful pacing and became asymptomatic with elevation of the extremity and heparinization. No other signs or symptoms were noted in the remaining patients. A recent review of upper extremity venous thrombosis (57) made no mention of pacemaker lead induced thrombosis. Recommendations for symptomatic deep venous thrombosis of the upper extremity include elevation, seven days of heparin therapy, starting coumadin therapy concomitantly with heparin and continuing if the patient remains symptomatic.

Cardiac perforation with tamponade is an extremely rare complication of pacemaker therapy in skilled hands (58) but is included as a reminder of the potentially lethal hazards associated with temporary and permanent pacemaker insertion.

The scope of cardiac pacing has expanded greatly since the original patients with complete heart block were described. As a result, a few unusual but nonetheless important complications have been reported. The return of A-V nodal conduction is not uncommon after the development of heart block in the face of a myocardial infarction. In these patients it is not unusual for ventriculoatrial conduction to occur. When ventricular pacing occurs in patients with an intact AV node, retrograde P wave may form. Given the appropriate timing of the impulse and integrity of the atria, ventriculoatrial conduction may cause cannon A waves in the left atrium creating high pulmonary wedge pressures (this will mimic the pulse tracing of severe mitral regurgitation at cardiac cath). Also, important tricuspid regurgitation may occur (59-60). Hence, it becomes important that patients who are to be paced be carefully evaluated. The presence or absence of any murmurs or neck vein pulsations must be carefully documented both before and after the insertion of the pacemaker. In the cases reported, this complication was successfully treated by reduction in the pacemaker rate by installing a new pulse generator with a pacing rate less than the sinus rate. Had the complication been noted earlier, a programmable unit could have been implanted in the first place.

One lethal complication of pacing related to the pulse generator itself is the so-called "runaway" pacemaker. Earlier model pulse generators did not incorporate any rate limiting circuitry, and as such could potentially pace at rates as high as 1000 if internal component failure occurred (61-63). Modern pacemakers do not (or should not) carry this hazard. All presently marketed pacemakers incorporate a rate limiting circuit so that even with component failure, the pulse generator should not pace faster than about 120 beats per minute.

New programmable pacemakers can be programmed deliberately to produce rates up to 120 beats per minute. Hence, the differential diagnosis of a pacemaker with a rapid rate in today's clinical setting would be: 1) a pacemaker programmed to pace rapidly, or 2) "runaway". The former is far and away the more likely of the two diagnoses. (Examples of programmable pacemakers include: Cordis "Omni" series of pacemakers; the Medtronic model numbers 5954, 5955, 5995, and 5994; the Intermedics models 251 and 252, and the CPI models 0505 and 0605).

Runaway pacemaker malfunction has not been reported in the newer lithium powered pacemakers. Most of the case reports of pacemaker runaway date from the early 1970's suggesting that the presently implanted pacing systems are more reliable in this regard. Two disturbing reports have appeared wherein mercury battery powered pacemakers of fairly recent vintage have demonstrated "runaway" phenomena (64-65). These pacemakers had normal rates of 72 beats per minute and "runaway" rates of 92 and 98 beats per minute. They were successfully treated with expedient replacement of the pulse generator.

If a true "runaway" is found, the first maneuver which can be attempted to terminate the rapid rate is place a magnet over the pulse generator thereby converting it to a fixed rate (V00) mode. Since the magnetic rate is generated by bypassing the sensing circuit, this occasionally will terminate a disasterously rapid rate. If the magnet mode fails to terminate the runaway, and the patient is sufficiently symptomatic the faulty pulse generator must be removed. If this is done, a temporary pacemaker must be available to replace the function of the disabled unit.

PACEMAKER LONGEVITY:

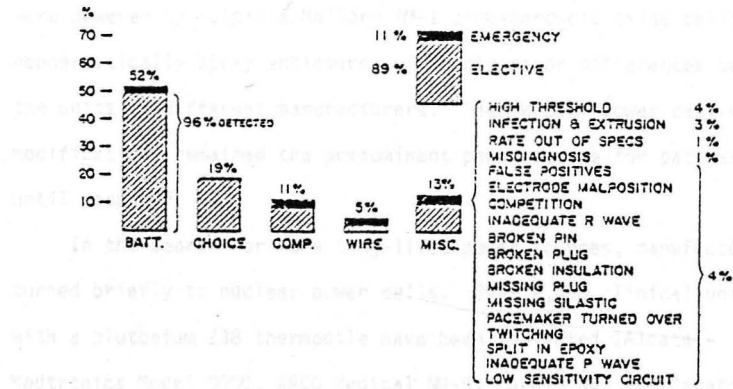
Lown in 1970 concluded his review of pacemakers with the comment that,

"Cardiac pacing constitutes a valuable lifesaving medical advance. It has spurred development of a new bioelectronic technology and has brought many electrophysiologic insights to the bedside. Pacemakers in current use, as yet, are inadequate and unreliable devices. Frequent breakdowns and far too short life-spans are characteristic of all pacer systems.....further development and innovation. With further development and innovation these limitations, no doubt, will be overcome." (13)

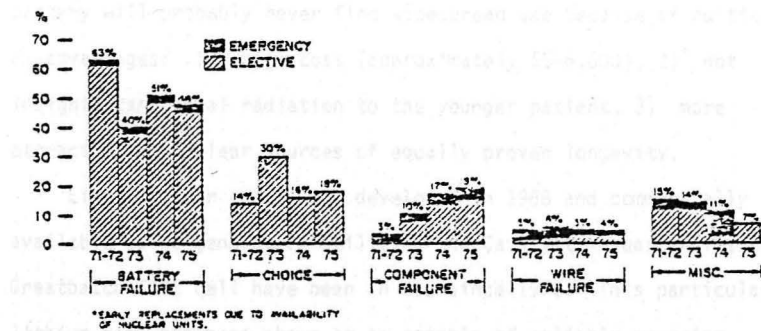
Since Lown wrote his review, many changes in pacemaker and lead technology have occurred. A brief review of the literature from the mid to late 1960's (66-70) clearly indicates that Dr. Lown was appropriately pessimistic about the state of the art at that time. Pacemaker battery life was short with most units lasting less than 20 months, and lead fractures were commonplace.

More reliable pacemaker circuitry and power sources are now available and should offer greater pulse generator life expectancy. For the next year or two, continued concern must be focused on battery depletion for many of the last generation of mercury powered pulse generators are still implanted and will be reaching the end of useful life.

Parsonnet's experience from 1971 to 1975 (43) indicates that careful follow-up of patients with pacemakers can reduce the number of emergency replacements to the 11 percent range (Figure 15). Of particular note is the 96 percent rate of detection of battery depletion. The mean life expectancy of the mercury-zinc power cells available during this period was less than 36 months and usually less than 30 months (43, 71, 72).



Reasons for pulse generator replacement at the Newark Beth Israel Medical Center from 1971 to 1975 (all causes; 669 cases)
 BATT. = battery; COMP. = component; SPECS = specifications.



Reasons for pulse generator replacement at the Newark Beth Israel Medical Center from 1971 to 1975, classified by year (all causes; 669 cases). The increase in percent of component failure was related to only one model of pacemaker, used frequently at this institution.

From 1960 through 1969 essentially all implanted pacemakers were powered by multiple Mallory RM-1 zinc-mercuric oxide cells in nonhermetically epoxy enclosures with only minor differences between the units of different manufacturers. The mercury power cell with modifications remained the predominant power source for pacemakers until recently.

In the search for more long-lived power sources, manufacturers turned briefly to nuclear power cells. Over 2,000 clinical units with a plutonium 238 thermopile have been implanted (Alcatel-Medtronics Model 9000, ARCO Medical NU-5, Curdis Nu, and Coratomic C100 and C101). To date no battery failures have been reported despite the use of nuclear power sources since 1973 (73). A continuing argument between the proponents and opponents of nuclear pacers revolves around the question of radiation safety. Levels of "safe" exposure have been continually reduced. The nuclear powered battery will probably never find widespread use because of multiple disadvantages: 1) high cost (approximately \$5-6,000), 2) not insignificant local radiation to the younger patient, 3) more attractive nonnuclear sources of equally proven longevity.

Lithium power cells were developed in 1968 and commercially available pulse generators utilizing the Catalyst Research-Wilson Greatbatch 702E cell have been in use since 1972. This particular lithium cell has been shown to be capable of reliably powering pacemakers for at least six years.

It is hoped that the various lithium power cells being marketed in today's pacemakers will continue to exhibit this remarkable

reliability and longevity. There are variations in the projected longevity advertised by the various pacemaker manufacturers which are related to various construction techniques and cell capacities. Pacemaker warranties now range from three to four years to the life of the patient. Parsonnet has suggested that 10 years of pacing from a single pulse generator may now be a realizable goal (74).

A concise review of pulse generator power sources has been published by Tyers and Brownlee (73).

PACEMAKER TROUBLESHOOTING AND FOLLOW-UP:

Acute Problems

Whether the pacemaker system is temporary or permanent, acute problems tend to fall into the categories of:

1. No pacemaker complexes noted on ECG
2. Pacemaker artifacts on ECG without capture
3. Failure of the pacemaker to sense
4. Oversensing
5. Erratic rhythm

A very concise troubleshooting guide for pacemaker malfunction has been devised by Dr. Kirk Lipscomb at the VA Medical Center (75) and is included in the following table.

No Pacemaker Complexes on ECG	Pacemaker Artifacts without Capture	Failure to Sense	Oversensing	Erratic Rhythm
Slow spontaneous heart rate	Loss of capture only when artifact in ventricular refractory period—normal for fixed rate. See "Failure to Sense" if demand	Spontaneous QRS complex occurring shortly after pacemaker impulse not sensed—normal with ventricular synchronous due to pacemaker refractory period (approximately 400 msec).	Sensing of P and T waves—decrease sensitivity or convert to fixed rate pacing	Pacemaker artifacts buried in QRS complex rather than slightly preceding it—primary cardiac arrhythmia with ventricular synchronous pacemaker. Treat arrhythmia
Pacemaker output low or zero—faulty pulse generator	Pacemaker rate change—generator failure. Replace	Chest wall stimulation	Pacemaker inhibited during arm movement, artifacts on pacemaker wire electrogram during arm movement—skeletal muscle inhibition. If serious, convert to bipolar	Escape interval (sensed spontaneous beat to pacemaker impulse) greater than automatic interval (pacemaker impulse to pacemaker impulse)—hysteresis circuit, normal in some pacemakers
Unipolar electrogram zero—broken wire. Replace. If bipolar, convert to unipolar using other wire	Pacemaker wire displacement on chest x-ray—reposition	No suppression—faulty pulse generator or wire, confirmed by pacemaker wire electrograms and/or change in magnet rate	Broken electrode on chest x-ray, abrupt changes in pacemaker wire electrogram baseline—intermittent apposition of broken pacemaker wire stimulating QRS	Extrinsic electromagnetic interference—usually due to microwave ovens; close proximity to strong radar, TV, or radio antenna; or electro-surgery
Unipolar electrograms normal, bipolar electrogram zero—shorted bipolar wires	ECG complex changed from LBBB to RBBB—perforation or coronary sinus position. Pull back and/or reposition	Suppression	Extrinsic interference—usually due to microwave oven; very close radio, TV, or radar antenna; or electro-cautery	Magnet rate changed from previously—faulty pulse generator
Rapid spontaneous heart rate	Threshold increased	Pacemaker wire electrogram less than 5 mv restored to over 5 mv by positioning—wire dislocation		
Fixed rate pacing when magnet over pacemaker—normally suppressed pacemaker	Restored to normal by repositioning—pacemaker wire dislocation	Pacemaker wire electrogram less than 5 mv and not restored by positioning—decreased myocardial potential. Convert from bipolar to unipolar or fixed rate pacing		
No pacemaker artifact when magnet over pacemaker—See "slow spontaneous heart rate"	Not restored to normal by multiple repositioning—Elevated endocardial threshold. Change to higher output pacemaker or epicardial position			

Patients who are completely paced may present a problem to the clinician if an acute myocardial infarction is suspected since the paced rhythm will mask any changes in the underlying ECG. Also, in some circumstances it may be desirable to ascertain the patient's underlying rhythm.

For these situations the pacemaker can be inhibited by either "overdriving" or "underdriving" the implanted unit with an external temporary pacing unit.

1. An ECG electrode (paste-on or suction cups) is positioned over or near the implanted pacemaker and another electrode near the implanted electrode tip. Some experimentation may be necessary since the purpose of the external pacing is to generate a current that is parallel to the path taken by the current of the implanted pacing system.

2. The ECG electrodes are connected to the external pacer.

3. The sensitivity control of the external pacer is set at a fixed rate or the asynchronous position.

4. The rate of the external pacer is set above the rate of the implanted unit (for overdrive suppression); or the rate of the external pacer is set at a rate 30 to 50 percent lower than the rate of the implanted unit (for underdrive suppression).

5. The amplitude of the external pacer is set at 5 mA or higher. The amplitude may be increased until the implanted pacer is inhibited, although currents higher than 15 mA may cause patient discomfort.

Either method can be used to assess the underlying ECG, but in

either case the external unit must be immediately disconnected if the patient has a very slow or absent underlying rhythm. For example, overdriving the implanted pacemaker will completely inhibit a VVI pacemaker. If the ventricle in complete AV block is paced at a rapid rate and pacing ceases suddenly, the idioventricular pacemaker is depressed and prolonged asystole may occur before an idioventricular rhythm appears.

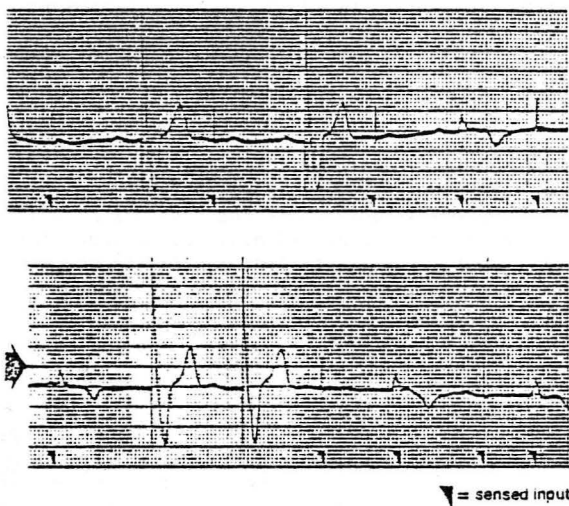


Figure 16

An example of "underdrive" pacing where each triangle represents a sensed impulse (either from the external pacemaker or from the intrinsic QRS).

LONG TERM FOLLOW-UP

Evaluation of pacemakers requires the knowledge of the particular pacemaker brand, model, and often, the engineering series of the unit in question. This information must be obtained to properly assess the operating characteristics of the pulse generator.

1. What brand and model is implanted? This problem frequently arises when a pacemaker has been installed at an institution other than where the pacemaker follow-up is being performed?

- a. Hopefully this information can be obtained from hospital or office records.
- b. All pacemaker companies supply patients with pacemaker data cards (Figure 17) which will provide the relevant data. The pacemaker manufacturer can then be contacted to obtain the specifications that apply to the pacemaker in question.

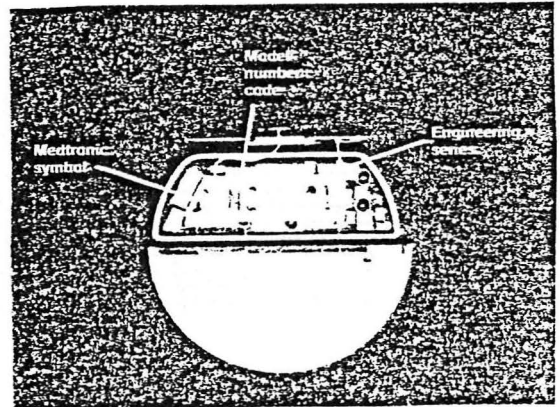
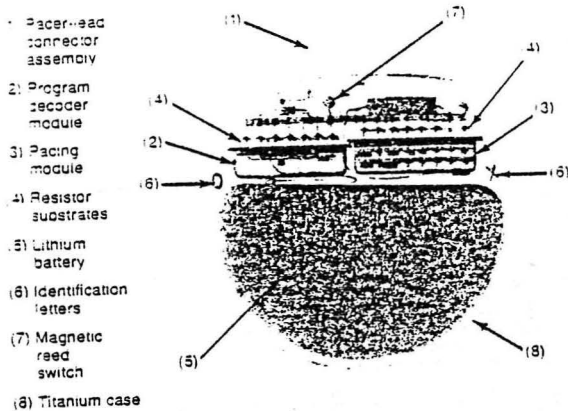
**CARDIAC PACEMAKER
IDENTIFICATION CARD**
Intermedics Inc.
P.O. Box 617 • Freeport, Texas 77541

Name	John Doe	Rate	72 BPM
Address	1611 Taft Street		
	Taylor, Indiana	46410	
Phone	219-292-4137	Model	225
Type of Device:	R Wave Inhibited	Serial Number	1234
		Type of Leads	Endocardial
		Date of Implant	5-14-77

IN CASE OF EMERGENCY CONTACT PHYSICIAN
IDENTIFIED ON REVERSE OF CARD

FIGURE 17

- c. Most pacemaker companies now incorporate X-ray identification symbols into the pacemaker case. An overpenetrated PA chest X-ray can usually be used to find this information and the pacemaker company can be contacted. Examples of the pacemaker identification code for the Medtronic and Cordis pacemakers are shown in Figure 18.



A listing of the identification codes for the more recent Medtronic and Cordis pacemakers is provided in Appendix A. The CPI pacemakers manufactured after 1976 will carry an identification number which can be seen on X-ray. Following the letters "CPI", a three digit number refers to the model designation.

2. Does the pacemaker sense and capture properly?

This information can be determined from the routine ECG. Holding a magnet over the pulse generator will convert most pacemakers to fixed rate (VVO) mode which can be used to check the ability of the pacemaker to capture in patients who have an intrinsic heart rate greater than the pacing rate of a demand (VVI) pacemaker. Figure 19 depicts normal sensing in the upper tracing. Normal capture is depicted in the lower two tracings which are continuous. The pacing spike will fail to capture when it falls during the refractory period of the ventricle (middle strip and the first two pacing spikes in the third strip).

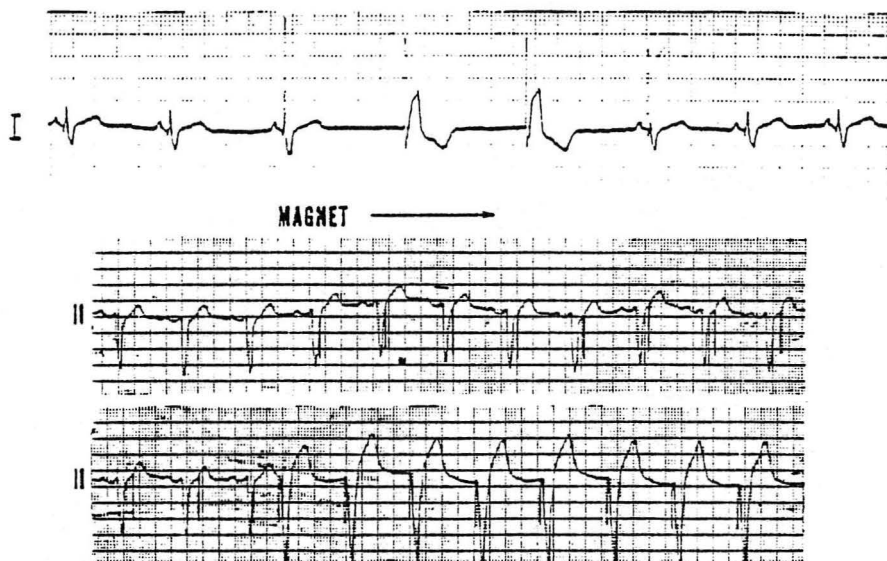


FIGURE 19

3. Does the pacemaker meet its design standards?

a. Is the magnet mode operational?

The magnet rate may be different from the automatic rate.

b. Are the pacing rate and pulse width (pulse duration) within the design specifications?

Appendix B gives some of the specifications for the measurable parameters of some recent models of pacemakers. As is obvious, there are many different specifications for each model and brand. However, pulse rate and pulse interval or duration are the important measurable values.

c. Is the pacemaker exhibiting end-of-life (EOL)/elective replacement time characteristics.

For the most recent generation of mercury-zinc powered pacemakers, a rate decrease is indicative of battery depletion/exhaustion. For example, the Medtronic 5950 and 5951 ("Xytron") series pacemakers have an automatic rate of 72 and a pulse width of .80 msec and .50 msec respectively (the values with magnet application are the same). These values remain constant through the pacemaker's useful battery life. At the end of battery's useful life, the pacing rate will fall and the pulse width will increase (the latter function is to provide a constant current as the voltage declines and maintain the pacemaker's ability to capture the ventricle).

In the newer lithium-iodide pulse generators (Medtronic 5972 and 5973 "Xyrel") the automatic rate can vary slightly ($\pm 3\%$) and the normal magnet rate will be 0 to $2\frac{1}{2}$ beats per minute higher than the automatic rate. The magnet rate is recommended for use as an end of battery life indicator. When the 5972 (bipolar) pulse generator battery has become depleted (voltage has dropped from 5.6 to 4.0), the magnetic rate will fall 5 to 8 beats indicating the need for replacement. To compensate for the voltage decline, the pulse width will increase from .80 msec to 1.60 msec.

Methods for establishing the heart rate accurately from a standard ECG have been described (76, 77), but for the most part are too cumbersome to be of value. The detection of small but potentially important changes in pacing rate requires more accurate measurement than is usually available from a standard ECG machine. Standard ECG machines operate at 25 mm/sec paper speed and even if the speed is doubled, the accuracy of the paper speed may be inadequate for accurate measurement of the pacing rate. The design specifications for the automatic rate of the Medtronic Model 5972 (Xyrel) within bipolar pacemakers is: basic rate $\pm 3\%$ or 72 ± 2.16 .

Pacing rates in seconds and milliseconds are listed.

Pacing rate beats/min	Pulse intervals	
	msec	seconds
74	811	.811
73	822	
72	833	.833
71	845	
70	857	.857
69	870	
68	882	
67	896	
66		
65	923	

At a nominal rate of 72, a pacemaker could have a pulse interval from .811 sec to .857 seconds and remain within design specifications. Bear in mind that the smallest time line on an ECG run at standard speed is .04 seconds or 40 milliseconds. Hence, on a beat to beat basis, the difference in one "box" on the ECG trace is the difference between the entire range of "normal" for the Medtronics 5972 pacemaker. The specifications for most other presently implanted pacemakers are roughly similar. Admittedly a patient can monitor his own heart rate by taking his pulse (the most simple expedient) however, the accuracy of this method for detecting small changes in rate (like the standard ECG) may have significant shortcomings.

Currently available pacemaker monitoring systems are available to assess pulse width to $\pm .02$ msec, heart rate to .1 pulse/minute, and pulse interval to 1 msec. Units the size of a cigarette pack are available for approximately \$200.00.

4. When should elective replacement of a pulse generator be accomplished?

- a. If malfunction is noted (failure to sense or capture, a rate increase beyond design parameters) the pulse generator, lead or both should obviously be replaced.
- b. If testing as indicated in section 3 suggests the battery is reaching its end of life, elective replacement is indicated.
- c. The pulse generator may be prophylactically replaced due to known propensity for failure. This data can be obtained from manufacturers (e. g. pacemakers that have been "recalled" or have a "product advisory" suggesting a higher than normal expectancy for failure). Also, both pacemaker manufacturers and some multicenter pacemaker follow-up clinics are able to provide actuarial data on the life expectancy of some pulse generators. Dr. Bilitch at the University of Southern California is publishing the combined experience of three major pacemaker clinics under an FDA contract. Those data appear in each issue of a new journal called Pacing and Clinical Electrophysiology (73).

5. How often should patients be followed?

Presently, the recommendations of the Inter-Society Commission for Heart Disease resources are (11):

Following transthoracic implantation, or transvenous implantation, the patient should have continuous monitoring up to 48 hours (the time interval may be reduced depending upon the pre-existing rhythm, reliability of the operating team, and the extent to which the patient is pacemaker dependent).

At a minimum there should be daily evaluation of pacemaker function either by ECG or by oscilloscopic monitoring during the patient's hospital stay. A post-op overpenetrated PA and Lateral chest X-ray should be taken prior to discharge to serve as a baseline for subsequent clinical evaluations and to confirm the proper electrode position. Less stringent follow-up is suggested for routine pulse generator replacement.

Optimally, patients should be seen within one month after discharge from the hospital to detect early problems. They should then be seen at least three times during the first year and at least every two months thereafter until the anticipated end-of-life of the pacemaker is approached. Approximately six months before anticipated end-of-life, the patient should begin to receive closer surveillance either with monthly visits, weekly transtelephone monitoring, or both.

The efficacy of telephone surveillance as a follow-up system has been adequately demonstrated by Parsonnet and others and offers a simple means for frequent pacemaker follow-up (79-85). Examples of pertinent data provided from such monitoring systems are included in APPENDIX C.

PROGRAMMABLE PACEMAKERS:

In 1972, externally programmable pacemakers were introduced to help solve the problems of battery longevity. Systems which had variable: current output, pulse width, or rate became available. Since pacemaker energy output can be represented by the product of voltage, current, and pulse duration, energy conservation can in theory be affected by reducing any one of those parameters. An extension in battery life has not been clearly shown by using such techniques. In fact, one report suggests that there is no difference between programmable and nonprogrammable pacemaker longevity (72). If the lithium power source lives up to its projections, pacemaker longevity may be more dependent upon lead integrity than the pulse generator (85).

The non-invasive programmable pacemakers presently being marketed all have a rate adjustability feature which can have practical advantages for the patient with a pacemaker. Programming is accomplished in many systems by using an electromagnetic programmer which emits a coded series of electromagnetic impulses to "reprogram" the circuitry of the pulse generator. The signal, in the case of a rate change, will "instruct" the circuitry of the pulse generator to increase or decrease the pulse rate. (The Cordis "Omni" series of programmable pacemakers can be set at 60, 65, 70, 90, or 100 for the standard range and 50, 54, 60, 65, 70, 81, 100, or 120 beats/minute for the extended range units. The Medtronic lithium powered rate programmable units (5994 and 5995) can be adjusted for rates of 30, 50, 60, 70, 80, 90, or 100. The CPI programmable units (505 and 605) will program any rate from 50 to 119.

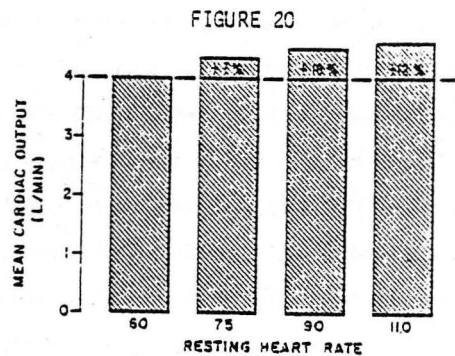
Possible reasons for decreasing the rate from 72 (the setting at the factory) to a lower rate

1. Potentially therapeutic in a patient with angina where an attempt is being made to reduce heart rate (as with propranolol).
2. In patients who have regained an intrinsic heart rhythm. This maneuver would potentially increase cardiac efficiency by restoring the atrial kick (86).
3. For evaluating the underlying rhythm and QRS morphology.

Possible reasons for increasing the pacing rate from 72

1. Overdrive therapy for suppression of arrhythmias.
2. Potentially increase the cardiac output in patients with severe cardiac dysfunction and congestive heart failure.

There are numerous studies (37-39) which suggest that if the myocardium is reasonably normal, there is little or no change in resting cardiac output by increasing the rate above 70 (Figure 20). Whether patients with advanced myocardial disease can have their resting and/or exercise cardiac output "optimized" by changing the rate will await further testing (perhaps with non-invasive methods for determining cardiac output).



Mean Effect of Heart Rate on Cardiac Output in 12 Patients (38).

In the near future, several companies will market programmable pacemakers which will enable the physician to program a large number of parameters in one pacemaker. Some of these programmable features include:

- rate
- pulse duration
- sensitivity
- pacemaker refractory period
- hysteresis
- mode of stimulation (V00, VVT, VVI)
- test lead integrity

An interesting paper has recently been published supporting the use of these "smart" pacemakers and I would refer you to that review (90). Many of the Cordis "Omni" series of programmable pacemakers which are already implanted have an "unused" chip in the circuitry which will respond to a new programming device and convert VVI units to V00 mode (asynchronous).

IT IS IMPORTANT TO NOTE THAT THE PROGRAMMER FOR ONE BRAND OF PACEMAKER WILL NOT CORRECTLY PROGRAM A PULSE GENERATOR MANUFACTURED BY ANOTHER COMPANY. In fact, the new multiple programmable pacemakers will have entirely different programmers than the units currently in use.

The use of the incorrect programmer can result in incorrect programming of a pulse generator. "Phantom" or inadvertent programming has already occurred but is presently a rare occurrence (91). However, the widespread use of programmable pacemakers and the plethora of different programming systems will undoubtedly result in an increasing number of such events. Likewise, pacemaker follow-up will become increasingly complex as rates and other pacing parameters become easily changed.

APPENDIX A

Hydrolytic Enzyme Classification Codes

Enzyme Code Enzyme Name Enzyme Code Enzyme Name

(Acidophylic powered)

5044	(70-75)		
5045	(70-75)		
5046	(70-75)		
5047	(70-75)		
5048	(70-75)		
5049	(70-75)		
5050	(70-75)		
5051	(70-75)		
5052	(70-75)		
5053	(70-75)		

(Alkaliphilic powered)

5054	(70-75)		
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APPENDIX A

(Alkaliphilic powered)

5055	(70-75)		
5056	(70-75)		
5057	(70-75)		
5058	(70-75)		
5059	(70-75)		
5060	(70-75)		
5061	(70-75)		
5062	(70-75)		
5063	(70-75)		
5064	(70-75)		
5065	(70-75)		
5066	(70-75)		
5067	(70-75)		
5068	(70-75)		
5069	(70-75)		
5070	(70-75)		
5071	(70-75)		
5072	(70-75)		
5073	(70-75)		
5074	(70-75)		
5075	(70-75)		
5076	(70-75)		
5077	(70-75)		
5078	(70-75)		
5079	(70-75)		
5080	(70-75)		
5081	(70-75)		
5082	(70-75)		
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5089	(70-75)		
5090	(70-75)		
5091	(70-75)		
5092	(70-75)		
5093	(70-75)		
5094	(70-75)		
5095	(70-75)		
5096	(70-75)		
5097	(70-75)		
5098	(70-75)		
5099	(70-75)		
5100	(70-75)		

* Models ending in an even number are bipolar; those ending with an odd number are unipolar.

* Approximate year of deposition

APPENDIX A

Medtronic pacemaker identification codes:

<u>Model #</u>	<u>Trade Name</u>	<u>Programmable</u>	<u>IAA Code</u>	<u>X-Ray ID Code</u>
* (mercury-zinc powered)				
5944 (70-73)		-	VVI	
5945 (70-73)		-	VVI	
5912 (73-77)			V00	N
5950 (73-77)	Xytron	-	VVI	P
5951 (73-77)	Xytron	-	VVI	Y
5954 (73-77)	Xytron	+	VVI	F
5955 (73-77)	Xytron	+	VVI	U
(nuclear powered)				
9000 (70-78)		-	VVI	
(lithium powered)				
5972 (76-)	Xyrel	-	VVI	MA
5973 (76-)	Xyrel	-	VVI	MD
5994 (78-)	Xyrel	+	VVI	MU
5995 (78-)	Xyrel	+	VVI	MK
5926 (78-)	Xyrel	-	VVI	-
5927 (78-)	Xyrel	-	VVI	-
5988 (79-)	Mirel	-	VVI	ED
5989 (79-)	Mirel	-	VVI	EE
5982 (79-)	Mirel	-	VVI	NB
5983 (79-)	Mirel	-	VVI	NC
5996 (79-)	Xyrel-HT	+	VVI	CT
5997 (79-)	Xyrel-HT	+	VVI	CM

* Models ending in an even number are bipolar; those ending with an odd number are unipolar

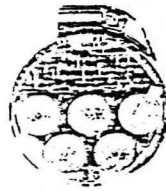
() = approximate year of production

Cordis pacemaker identification codes:

<u>Model #</u>	<u>Trade Name</u>	<u>Programmable</u>	<u>X-Ray ID Code</u>
(mercury-zinc powered)			
162 C	Omni-Stanikor	+	DA
171 C7	Stanikor "kappa"	-	MF
171 D7	Stanikor "kappa"	-	MK
(lithium powered)			
190 A	Omni- tanikor "lambda"	+	DX
206 A	Omni-Stanikor "lambda" (has narrower pulse duration)	+	PF
190 F	Omni-Stanikor "lambda"	+	PO
188 D7	Stanikor "lambda"	-	MM
221 D7	Stanikor "theta"	-	MD

Subsequent information is reproduced from material supplied by
Cordis Corporation

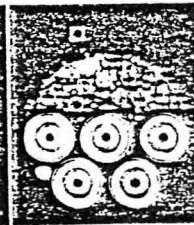
Nonprogrammable Implantable Pacers Stanicor, R-wave inhibited (WVI)



Model 143E



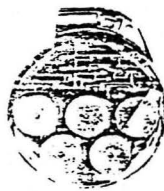
Model 143A



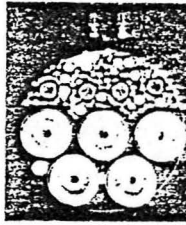
Model 143E
(model shown prior to
introduction of radio-
graphic identification)

Model, series	Radiopaque code	Automatic (fixed) rate	Features
143 A6	---	58-64	Fixed rate decreases with battery depletion prior to loss of R-wave inhibited function. Magnetic switch for converting from R-wave inhibited to fixed-rate mode. Pulse duration 1.5 msec. Sensitivity 2.0 mv. Output current 9 ma. Refractory 0.24 sec. Superseded by 143G.
A7	---	65-74	
A8	---	75-84	
A9	---	85-94	
143 C6	---	58-64	Rate decrease, magnetic switch, pulse duration, sensitivity, output current, refractory as in 143A. Superseded by 143E.
C7	---	65-74	
C8	---	75-84	
C9	---	85-94	
143 E6	SA	58-64	Rate decrease, magnetic switch, pulse duration, sensitivity, output current, refractory as in 143A. Integral connector. Superseded by 143J.
E7	SB	65-74	
E8	SC	75-84	
E9	SD	85-94	
143 G6	SF	58-64	Rate decrease, magnetic switch, sensitivity, refractory as in 143A. Integral connector. Pulse duration 1.0 msec. Output current 7 ma. Superseded by 143L.
G7	SG	65-74	
G8	SH	75-84	
G9	SI	85-94	
143 J6	SP	58-64	Rate decrease, magnetic switch, pulse duration, output current, refractory, integral connector as in 143E. Sensitivity 1.5 mv. Standard-output model.
J7	SQ	65-74	
J8	SR	75-84	
J9	SS	85-94	
143 L6	SZ	58-64	Rate decrease, magnetic switch, pulse duration, output current, refractory, integral connector as in 143G. Sensitivity 1.5 mv. Reduced-output model.
L7	TB	65-74	
L8	TC	75-84	
L9	TD	85-94	

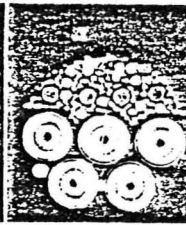
Ectocor, R-wave synchronous (VVT)



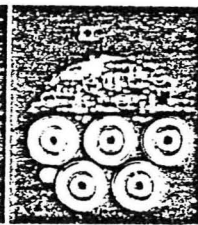
Model 144C



Model 129E



Model 129L

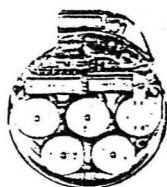


Model 144C
(model shown prior to
introduction of radio-
graphic identification)

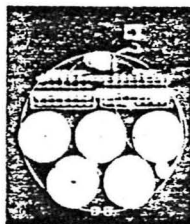
Model, series	Radiopaque code	Automatic (fixed) rate	Maximum rate	Features
129 A	—	70	145	Pulse duration 1.5 msec. Sensitivity 2 mv. Output current 8-12 ma. Refractory 0.4 sec. Fixed rate can be specified. Superseded by 129B.
129 B	—	70	145	Pulse duration, sensitivity, output current, refractory, rate specification as in 129A. Fixed rate decreases with battery depletion. Magnetic switch for converting from R-wave synchronous to fixed-rate mode. Superseded by 129C.
129 C	—	70	125	Same as 129B except: Refractory 0.5 sec. Superseded by 129E.
129 E7	—	68-74	143-157	Same as 129C except: Refractory 0.38-0.42 sec, and fixed rate can be specified as E6 (60), E7 (70), or E8 (80). Superseded by 144E.
129 L7	—	68-74	143-157	Same as 129E except: Integral connector. Superseded by 144C.
144 A7	—	68-74	143-157	Same as 129E except: Fixed rate decreases with battery depletion prior to loss of R-wave synchrony. Superseded by 144B.
144 B7	—	68-74	143-157	Rate decrease, magnetic switch, rate specification, output current, pulse duration, sensitivity as in 144A. Superseded by 144C.
144 C6	EA	58-64	143-157	Rate decrease, magnetic switch, rate specification, integral connector as in 129L7. Output current, pulse duration, sensitivity as in 144B. Superseded by 144G.
C7	EB	68-74	143-157	
C8	EC	78-84	143-157	
144 E6	EE	58-64	143-157	Rate decrease, magnetic switch, rate specification, sensitivity as in 144B. Integral connector. Output current 7 ma. Pulse duration 1.0 msec. Superseded by 144J.
E7	EF	68-74	143-157	
E8	EG	78-84	143-157	
144 G6	EK	58-64	143-157	Rate decrease, magnetic switch, rate specification, integral connector, output current, pulse duration as in 144C. Sensitivity 1.5 mv. Standard-output model.
G7	EL	68-74	143-157	
G8	EM	78-84	143-157	
144 J6	ES	58-64	143-157	Rate decrease, magnetic switch, rate specification, integral connector, output current, pulse duration as in 144E. Sensitivity 1.5 mv. Reduced-output model.
J7	ET	68-74	143-157	
J8	EU	78-84	143-157	

Programmable Implantable Pacers

Three of the four pacers in the externally programmable Cordis Omnitor System have the same physical appearance and can be distinguished on X-ray photographs only by their radiopaque code. The exception is Omni-Atricator, which can be distinguished by its atrial sensing lead as well as by its radiopaque code. The photograph and X-ray of Omni-Ectocor shown below thus are generally typical of the Omnitor pacers as a group.



Model 163A



Model 163A

Model, series	Radiopaque code	Programmable fixed rates	Refractory, msec.	Pulse duration, msec.	Features	
Omni-Stanitor, R-wave inhibited programmable pacer (VVI)						
162 C	DA	60	375	2.0	Output current can be programmed at 9, 6, 4, or 2.3 ma. Sensitivity 1.5 mv. Integral connector.	
		65	346	1.3		
		70	322	1.7		
		80	281	1.5		
		90	250	1.3		
		100	225	1.2		
Omni-Ectocor, R-wave synchronous programmable pacer (VVT)						
163 A	DB	60	375	2.0	Output current can be programmed at 9, 6, 4, or 2.3 ma. Sensitivity 1.5 mv. Integral connector.	
		65	346	1.8		
		70	322	1.7		
		80	281	1.5		
Omni-Ventricor, asynchronous programmable pacer (V00)						
167 A	DD	60	—	2.0	Output current can be programmed at 9, 6, 4, or 2.3 ma. Integral connector.	
		65		1.8		
		70		1.7		
		80		1.5		
		90		1.3		
		100		1.2		
Model, series	Radiopaque code	Programmable fixed rates	Refractory, msec.	AV delay, msec.	Pulse duration, msec.	Features
Omni-Atricor, P-wave synchronized programmable pacer (VAT)						
164	DC	60	500	125	2.0	Output current can be programmed at 9, 6, 4, or 2.3 ma. Sensitivity 1.0 mv. Integral connector.
		65	461	115	1.8	
		70	429	107	1.7	
		80	375	94	1.5	

	<u>Medtronics</u>		<u>Medtronics</u>		<u>CPI</u>
Model #	5950	5951	5973	608	
Basic Rate (PPM) (Nominal)	72	72	72	72	
Magnet rate (PPM)	72	72	+ 0 - 2½ PPM over basic		+ 20% over basic
Pulse duration (msec)	.80	.50	.80	1.0	
Amplitude (volts)	4.9	5.1	5.1	5.0	
(mA)	9.8	10.2	10.0	10.0	
Refractory period (msec)	325 + 55	same	same	330	
Sensitivity (mV)	1.25 - 3.25	1.6 - 3.6	2.0 - 3.7		
End of Life Indicators:					
Rate	+ 8 - 16% of basic	+ 8 - 16% of basic	+ 8 + 3 PPM from magnetic rate	+ 6 PPM from basic or magnet rate	
Pulse width increase	1.2 msec	1.3 msec	0.7 msec		

APPENDIX C

Cardiocare Testing Corporation

425 East 51st St. • New York • NY 10021 • (212) 751-8601

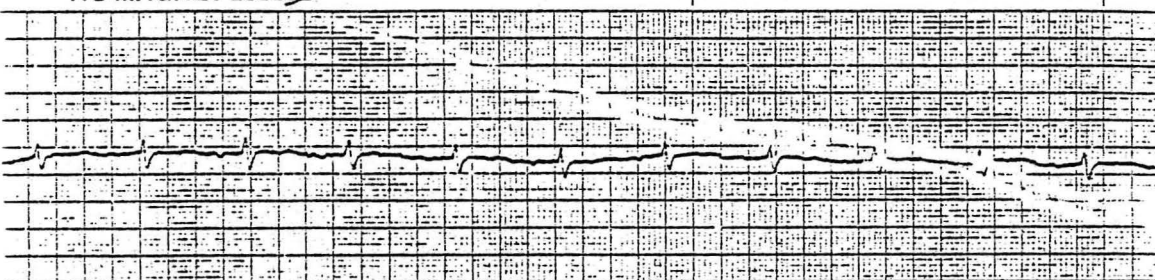
APPENDIX C

-59-

APPENDIX C patient 1

Patient Name [REDACTED] Patient # [REDACTED] Sex M
 Manufacture Medtronic Serial # [REDACTED] Date of Implant 4/30/76
 Model # 5950 Type VVI Rate of Implant 71
 Underlying Rhythm 3⁰ A-V Block Catheter Model # 6901-58 Date on Service 10/30/78
 (02970MC)

NO MAGNET Lead II



No. ECG 100

MAGNET Lead II



No. ECG 100

Rate: No Magnet bpm Magnet 71.57 ppm

Comments: **100% Spontaneous Activity Without Magnet.**

Spontaneous Rate Approximately 80 BPM.

Magnetic Switch Operative.

Evaluation:	Sensing—	Response to Pacer Stimuli—	Interpretation—
	Normal	Normal	Normal Function

Date MAR 20 1979 Dr. Signature LAWRENCE J. COHN, M.D.

Physician

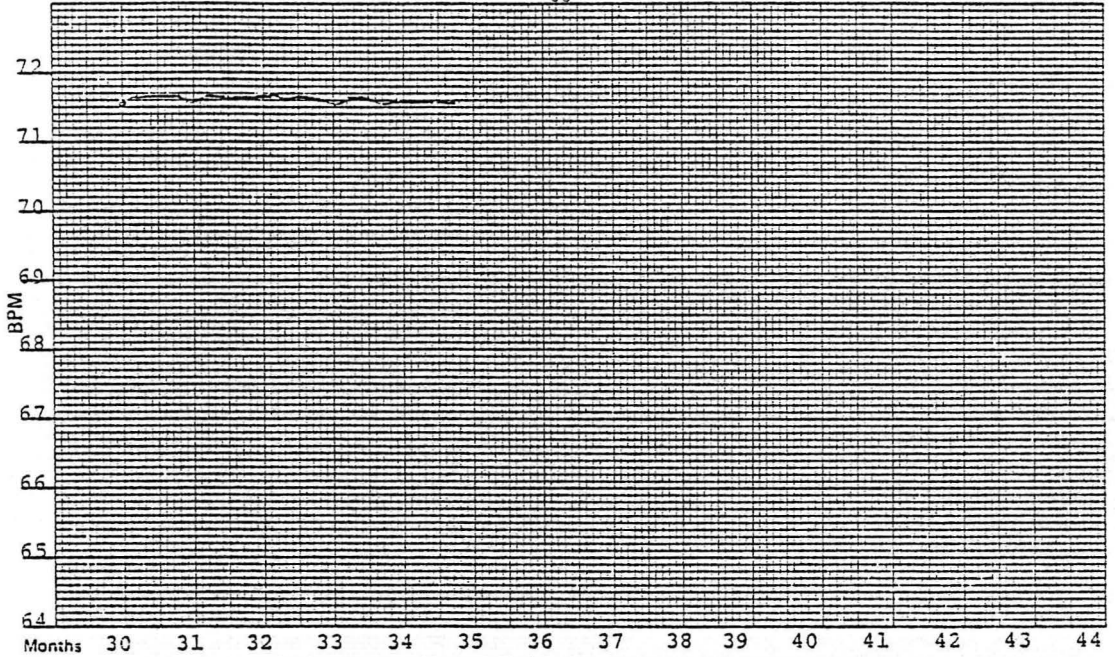
[] Dou Blettell, P.A. (111H)
 Hypertension Clinic
 VA Medical Center
 4500 South Lancaster Road
 Dallas, TX 75216

Physician

[] Kenneth A. Narahara, M.D. (111A)
 VA Medical Center
 4500 South Lancaster Road
 Dallas, TX 75216

-APPENDIX C

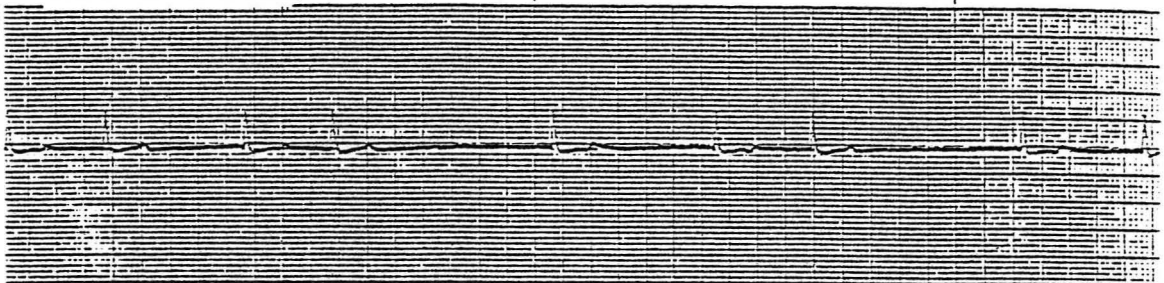
Patient Name	<u>[REDACTED]</u>	Patient #	<u>[REDACTED]</u>	Date of Implant	<u>4/30/76</u>
Manufacture	<u>Medtronic</u>	Serial #	<u>[REDACTED]</u>	Rate of Implant	<u>71</u>
Model #	<u>5950</u>	Type	<u>VVI</u>	Dr.	<u>Narahara</u>

[illegible]

425 East 51st St. • New York • NY 10021 • (212) 751-6901

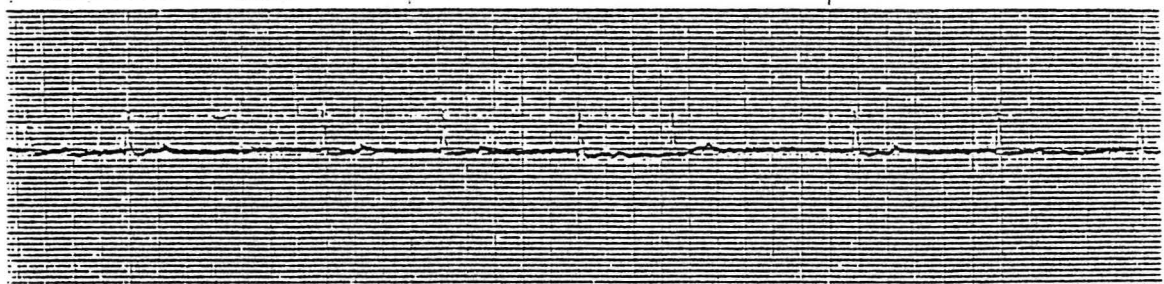
Patient Name [REDACTED] Patient # [REDACTED] APPENDIX C patient 2
 Sex [REDACTED]
 Manufacture Medtronic Serial # [REDACTED] Date of Implant 9/2/76
 Model # 5950 Type VVI Rate of Implant 72
 Underlying Rhythm Brady-Tachy Catheter Model # 6901-58 Date on Service 1/15/79

NO MAGNET Lead 1



☐ Cardiocare

NO MAGNET Lead 2



- 100

Rate: No Magnet 58 bpm Magnet 58 ppm

Comments: NO EVIDENCE OF PACER ACTIVITY OBSERVED WITH OR WITHOUT MAGNET. THIS IS POSSIBLY DUE TO HIGH CURRENT DRAIN. SPONTANEOUS RATE APPROXIMATELY 58 BPM'S. DOCTOR NOTIFIED.

This patient had a small fracture in his pacing lead. The pacemaker pulse generator was functioning normally the week prior to this evaluation. The patient remained asymptomatic and was unaware of any change in his pulse. His pacemaker was replaced without incident.

REFERENCES

1. Zoll PM: Resuscitation of the heart in ventricular standstill by external electrical stimulation. *New England Journal of Medicine* 247:768-711, 1952.
2. Principles of Internal Medicine, edited by T. R. Harrison. Philadelphia and Toronto, Blakeston, 1959, p. 1261.
3. Weirich WL, Gott VL, Lillehei CW: The treatment of complete heart block by the combined use of a myocardial electrode and artificial pacemaker. *Surgical Forum* 8:360, 1957.
4. Chardack WM, Gage AA, and Greatbatch W: A transistorized, self-contained, implantable pacemaker for the long-term correction of complete heart block. *Surgery* 48:643-654, 1960.
5. Furman S and Schwedel JB: An intracardiac pacemaker for Stokes-Adams seizures. *New England Journal of Medicine* 261:943-951.
6. Zoll PM, Frank HA, Zarsky LRN, Linenthal AJ, and Belgard AH: Long-term electric stimulation of the heart for Stokes-Adams disease. *Annals of Surgery* 154:330-346, 1961.
7. Chardack WM, Gage AA, and Greatbatch W: Correction of complete heart block by a self-contained and subcutaneously implanted pacemaker. *Journal of Thoracic and Cardiovascular Surgery* 42:814-830, 1961.
8. Parsonnet V, Gilbert L, Zucker IR, and Asa MM: A plan for the treatment of complete heart block and Stokes-Adams syndrome with an intracardiac dipolar electrode and a permanent implantable pacemaker. *Angiology* 14:343-348, 1963.
9. Furman S: Editorial: Why a new journal? Why PACE? *Pacing and Clinical Electrophysiology* 1:1, 1978.
10. Mullins C: Cardiac pacemakers. *PMH Grand Rounds*, October 10, 1969.
11. Parsonnet V, Furman S and Smyth NPD: Implantable cardiac pacemakers status report and resource guideline. *Circulation* 50:A21-A35, 1974.
12. Preston TA: Anodal stimulation as a cause of pacemaker-induced ventricular fibrillation. *American Heart Journal* 86:366-372, 1973.
13. Lown B and Kosowsky BD: Artificial cardiac pacemakers. *New England Journal of Medicine* 283:907-916, 971-977, 1023-1031, 1970.

14. Piller LW and Kennelly BM: Myopotential inhibition of demand pacemakers. *Chest* 66:418-420, 1974.
15. Ohm OJ, Bruland H, Pedersen OM and Waerness E: Interference effect of myopotentials on function of unipolar demand pacemakers. *British Heart Journal* 36:77-84, 1973.
16. Preston TA and Barold SS: Problems in measuring threshold for cardiac pacing. *The American Journal of Cardiology* 40:658-660, 1970.
17. Smyth NPD, Tarjan PP, Chernoff E and Baker N: The significance of electrode surface area and stimulating thresholds in permanent cardiac pacing. *The Journal of Thoracic and Cardiovascular Surgery* 71:559-565, 1976.
18. Furman S, Hurzeler P and Mehra R: Cardiac pacing and pacemakers IV. Threshold of cardiac stimulation. *American Heart Journal* 94:115-124, 1977.
19. Luceri RM, Furman S, Hurzeler P and Escher DJW: Threshold behavior of electrodes in long-term ventricular pacing. *The American Journal of Cardiology* 40:184-188, 1977.
20. Starke ID: Long-term follow up of cardiac pacing threshold using a noninvasive method of measurement. *British Heart Journal* 40:530-533, 1978.
21. DeCaprio V, Hurzeler P and Furman S: A comparison of unipolar and bipolar electrograms for cardiac pacemaker sensing. *Circulation* 56:750-755, 1977.
22. Preston TA, Fletcher RD, Lucchesi BR and Judge RD: Changes in myocardial threshold. Physiologic and pharmacologic factors in patients with implanted pacemakers. *American Heart Journal* 74:235, 1967.
23. Sowton E and Davies JG: Investigations of failure of artificial pacing. *British Medical Journal* 1:1470, 1964.
24. Davies JF and Sowton GE: Electrical threshold of the human heart. *British Medical Journal* 28:231, 1966.
25. Preston RA, Judge RD, Lucchesi BR and Bowers DL: Myocardial threshold in patients with artificial pacemakers. *American Journal of Cardiology* 18:83, 1966.
26. Gay RJ and Brown DF: Pacemaker failure due to procainamide toxicity. *The American Journal of Cardiology* 34:728-732, 1974.

27. Hughes HC, Jr., Tyers FO and Torman HA: Effects of acid-base imbalance on myocardial pacing thresholds. *The Journal of Thoracic and Cardiovascular Surgery* 69:743-746, 1975.
28. Al-Abdulla HM and Lulu DJ: Hypokalemia and pacemaker failure. *The American Surgeon*, April, 1974, pp. 234-236.
29. O'Reilly MV, Murnaghan DP and Williams MB: Transvenous pacemaker failure induced by hyperkalemia. *JAMA* 228:336-337, 1974.
30. Preston TA and Judge RD: Alteration of pacemaker threshold by drug and physiological factors. *Annals New York Academia of Sciences* 686-692.
31. Furman S, Escher DJW and Solomon N: Experiences with myocardial and transvenous implanted cardiac pacemakers. *The American Journal of Cardiology* 23:66-72, 1969.
32. Dixon SH, Jr., Perryman RA, Morris JJ, Jr., and Young WG, Jr: Transmediastinal permanent ventricular pacing. *The Annals of Thoracic Surgery* 14:206-213, 1972.
33. Brenner AS, Wagner GS, Anderson ST, Rosati RA and Morris JJ, Jr: Transvenous, transmediastinal, and transthoracic ventricular pacing: A comparison after complete two-year follow-up. *Circulation* 49:407-414, 1974.
34. Parsonnet V, Gilbert L and Zucker IR: The natural history of pacemaker wires. *The Journal of Thoracic and Cardiovascular Surgery* 65:315-322, 1973.
35. Imparato AM and Kim GE: Electrode complications in patients with permanent cardiac pacemakers. *Archives of Surgery* 105:705-710, 1972.
36. Magilligan DJ, Jr., Hakimi M and Davila JC: The sutureless electrode: Comparison with transvenous and sutured epicardial electrode placement for permanent pacing. *The Annals of Thoracic Surgery* 22:80-86, 1976.
37. Stewart S: Placement of the sutureless epicardial pacemaker lead by the subxiphoid approach. *The Annals of Thoracic Surgery* 18:308-313, 1974.
38. Stewart S, Cohen J and Murphy G: Sutureless epicardial pacemaker lead: A satisfactory preliminary experience. *Chest* 67:564-567, 1975.
39. Mansour CA, Fleming WH and Hatcher CR, Jr: Initial experience with a sutureless, screw-in electrode for cardiac pacing. *The Annals of Thoracic Surgery* 16:127-135, 1973.

42. Furman S, Garvey J and Hurzeler P: Pulse duration variation and electrode size as factors in pacemaker longevity. *The Journal of Thoracic and Cardiovascular Surgery* 69:382-389, 1975.
43. Parsonnet V and Manhardt M: Permanent pacing of the heart: 1952 to 1976. *The American Journal of Cardiology* 39:250-256, 1977.
45. Lemire GG, Morin JE and Dobell ARC: Pacemaker infections: A 12-year review. *The Canadian Journal of Surgery* 18:181-184, 1975.
46. Grögler FM, Frank G, Greven G, Dragojevic D, Oelert H, Leitz K, Dalichau H, Brinke U, Löhlein D, Rogge D, Hetzer R, Hannersdorf G, and Borst HG: Complications of permanent transvenous cardiac pacing. *The Journal of Thoracic and Cardiovascular Surgery* 69:895-904, 1975.
47. Dieter RA, Jr., Asselmeier GH, McCray RM, Grissom JH and Zimmerman RC: An alternative to removal of an infected pacemaker. *Geriatrics*, February 1973, pp. 78-80.
48. Kennelly BM and Piller LW: Management of infected transvenous permanent pacemakers. *British Heart Journal* 36:1133-1140, 1974.
49. Yarnoz MD, Attai LA and Furman S: Infection of pacemaker electrode and removal with cardiopulmonary bypass. *The Journal of Thoracic and Cardiovascular Surgery* 68:43-46, 1974.
50. Kaulbach MG and Krukoniis EE: Pacemaker electrode-induced thrombosis in the superior vena cava with pulmonary embolization. *The American Journal of Cardiology* 26:205-207, 1970.
51. Edelstein JM: Pulmonary emboli associated with transvenous pacemaker. *American Heart Journal* 87:808-809, 1974.
52. Williams DR and Demos NJ: Thrombosis of superior vena cava caused by pacemaker wire and managed with streptokinase. *The Journal of Thoracic and Cardiovascular Surgery* 68:134-137, 1974.
53. Wertheimer M, Hughes RK and Castle H: Superior vena cava syndrome. *JAMA* 224:1172-1173, 1973.
54. Sethi GK, Bhayana JN and Scott SM: Innominate venous thrombosis: A rare complication of transvenous pacemaker electrodes. *American Heart Journal* 87:770-772, 1974.
55. Friedman SA, Berger N, Cerruit MM and Kosmoski J: Venous thrombosis and permanent cardiac pacing. *American Heart Journal* 85:531-533, 1973.

56. Stoney WS, Addlestone RB, Alford WC, Jr., Burrus GR, Frist RA and Thomas CS, Jr: The incidence of venous thrombosis following long-term transvenous pacing. *The Annals of Thoracic Surgery* 22:166-170, 1976.
57. Prescott SM and Tikoff G: Deep venous thrombosis of the upper extremity: A reappraisal. *Circulation* 59:350-355, 1979.
58. Kalloor GJ: Cardiac tamponade: Report of a case after insertion of transvenous endocardial electrode. *American Heart Journal* 88:88-89, 1974.
59. Werres R, Parsonnet V, Gilbert L, and Zucker IR: Symptomatic unilateral cannon "a" waves in a patient with a ventricular pacemaker. *Chest* 73:539-542, 1978.
60. Johnson AD, Laiken SL, Engler RL: Hemodynamic compromise associated with ventriculoatrial conduction following transvenous pacemaker placement. *The American Journal of Medicine* 65:75-79, 1978.
61. Wallace WA, Abelman WH, Norman JC: Runaway demand pacemaker; report, invitro reproduction, and review. *Annals of Thoracic Surgery* 9:209-220, 1970.
62. Nasrallah A, Hall RJ, Garcia E, Kyger ER, Hallman GL and Cooley DA: Runaway pacemaker in seven patients: A persisting problem. *The Journal of Thoracic and Cardiovascular Surgery* 69:365-368, 1975.
63. Bramowitz AD, Smith JW, Leslie ME, Berens SC, Bilitch M and Grechko M: Runaway pacemaker: A persisting problem. *JAMA* 228:340-341, 1974.
64. Asato H, Bowyer AF, Jain AC, Marshall RJ and Tarnay TJ: Runaway pacemaker: Diagnosis and management. *The West Virginia Medical Journal* 73:73-76, 1977.
65. Lee HJ, Berman GM, and Ozolins AE: Runaway demand pacemakers in two asymptomatic patients. *Cardiovascular Medicine* 4:85-86, 1979.
66. Morris JD, Judge RD, Leininger BJ, and Vontz FK: Clinical experience and problems encountered with an implantable pacemaker. *Journal of Thoracic and Cardiovascular Surgery* 50:849-856, 1965.
67. Trimble AS: The implantable cardiac pacemaker: Late failures and their management. *Journal of Thoracic and Cardiovascular Surgery* 50:707-709, 1965.
68. Gadboys HL, Lukban S and Litwak RS: Long-term follow-up of patients with cardiac pacemakers. *The American Journal of Cardiology* 21:55-59, 1968.

69. Norman JC, Lightwood R and Abrams LD: Surgical treatment of Adams-Stokes syndrome using long-term inductive coupled coil pacemaking: Experience with 30 patients. *Annals of Surgery* 159:344-361, 1964.
70. Parsonnet V, Gilbert L, Zucker IR, and Asa MM: Complications of the implanted pacemaker: A scheme for determining the cause of the defect and methods for correction. *Journal of Thoracic and Cardiovascular Surgery* 45:801-812, 1963.
71. Parker B: Obituary: A vindication of the zinc-mercury pacemaker battery. *Pacing and Clinical Electrophysiology* 1:148-149, 1978.
72. Vera A, Janzen D and Mason DT: Longevity of programmable energy output pacemakers: Early results and experiences. *British Heart Journal* 39:1364-1373, 1977.
73. Tyers GFO, and Brownlee RR: Current status of pacemaker power sources. *The Annals of Thoracic Surgery* 25:571-587, 1978.
74. Parsonnet V: Cardiac pacing and pacemakers VII. Power sources for implantable pacemakers. Part II. *American Heart Journal* 94:658-664, 1977.
75. Lipscomb K: Cardiac pacing in clinical cardiology. Edited by J. T. Willerson and C. A. Sanders. Grune and Stratton, 1977. p. 234.
76. Kostis JB and Fearn W: Accurate electrocardiographic measurement of the rate of artificial pacemakers. *Annals of Internal Medicine* 78:711-713, 1973.
77. Gordon AJ: Pacemaker followup with transistor radio and stopwatch. *Chest* 66:557-559, 1974.
78. Bilitch M (ed): Performance of cardiac pacemaker pulse generators. *Pacing and Clinical Electrophysiology* 1:157-159, 1978.
79. Parsonnet V, Myert GH, Gilbert L and Zucker IR: Prediction of impending pacemaker failure in a pacemaker clinic. *The American Journal of Cardiology* 25:311-319, 1970.
80. Furman S and Escher DJW: Transtelephone pacemaker monitoring: Five years later. *The Annals of Thoracic Surgery* 20:326-338, 1975.
81. Grunkemeier GL, Dobbs JL and Starr A: Statistical analysis of pacemaker follow-up data: Rate stability and reliability. *Circulation* 53:241-244, 1976.

82. Parsonnet V, Myers GH, Zucker IR and Shilling E: Follow-up of implanted pacemakers. *American Heart Journal* 87:642-653, 1974.
83. Furman S: Cardiac pacing and pacemakers VIII. The pacemaker follow-up clinic. *American Heart Journal* 94:795-804, 1977.
84. MacGregor DC, Noble EJ, Morrow JD, Scully HE, Covvey HD, and Goldman BS: Management of a pacemaker recall. *The Journal of Thoracic and Cardiovascular Surgery* 74:657-667, 1977.
85. Mantini EL, Majors RK, Kennedy JR and Lebo GR: A recommended protocol for pacemaker follow-up: Analysis of 1,705 implanted pacemakers. *The Annals of Thoracic Surgery* 24:62-67, 1977.
86. Mitchell JH, Gupta DN, and Payne RM: Influence of atrial systole on effective ventricular stroke volume. *Circulation Research* 42:11-18, 1965.
87. Ross J, Jr., Linhart JW, and Braunwald E: Effects of changing heart rate in man by electrical stimulation of the right atrium: Studies at rest, during exercise, and with isoproterenol. *Circulation* 32:549-558, 1965.
88. Judge RD, Wilson WS and Siegel JH: Hemodynamic studies in patients with implanted cardiac pacemakers. *New England Journal of Medicine* 270:1391-1395, 1964.
89. Adolph RJ, Holmes JC, and Fukusumi H: Hemodynamic studies in patients with chronically implanted pacemakers. *American Heart Journal* 76:829-838, 1968.
90. Vera A, Klein RC, and Mason DT: Recent advances in programmable pacemakers: Consideration of advantages, longevity and future expectations. *The American Journal of Medicine* 66:473-483, 1979.
91. Fieldman A and Dobrow RJ: Phantom pacemaker programming. *Pacing and Clinical Electrophysiology* 1:166-171, 1978.