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*CARDIAC PACEMAKERS*

PARKLAND MEMORIAL HOSPITAL

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# CARDIAC PACEMAKERS

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I. Temporary Pacing

A. Indications for emergency pacing

In the event of cardiac arrest, the first mode of therapy is that of external cardiac massage and artificial respiration rather than an attempt to immediately insert a cardiac pacemaker. The first maneuver following restoration of circulation with closed-chest cardiac massage and artificial respiration is to determine what type of cardiac arrest is present. If the patient is in ventricular fibrillation, the obvious means of therapy is immediate defibrillation. If the patient is in an asystolic phase and is not responsive to external massage, an attempt to restore a sinus rhythm is mandatory. The first mode of therapy in an asystolic heart is to hit the anterior chest wall in an attempt to restart the heart. If the normal electrical pacemaker focus in the heart does not respond, then external massage should be instituted while drug therapy is started. The second mode of therapy is the use of drugs such as isuprel or if necessary aramine or norepinephrine (Levophed) or epinephrine (Adrenalin) in an attempt to stimulate the normal pacemaker centers in the heart. If adequate control of the heart rate is not forthcoming with the drug therapy, then an attempt should be made to insert an electrical pacemaker to supply the heart with a pacemaking system. In an emergency there is probably little indication for institution of electrical pacemaking without previously attempting to control the heart rate with drug therapy.

*DRUG THERAPY IN ACUTE HEART BLOCK*

1. Isuprel infusion
2. Atropine - for sinus bradycardia
3. Steroids, ephedrine, chlorothiazides of little value

The indications for the use of temporary pacing are listed below.

*INDICATIONS FOR TEMPORARY PACING*

1. As an emergency measure in Stokes-Adams attack
2. In acute myocardial infarction with block, or certain arrhythmias
3. To improve clinical state before implanting a permanent unit, *i.e.*, to increase cardiac output
4. To cover risk of cardiac arrest during operation for implanting unit
5. In patient particularly susceptible to cardiac arrest while undergoing anesthesia or operation
6. As a trial to see if improvement occurs in cerebral function or other symptoms, possibly due to low cardiac output
7. To suppress ventricular ectopic activity, *i.e.*, multiple PVC's or runs of ventricular tachycardia

B. Methods of temporary pacing

1. External pacing

The first recorded success with external pacing in the treatment of cardiac asystole by electrical impulses was in 1952 by Zoll (1). This report was followed by a number of others (2-5). These early records disclosed that asystolic episodes in Stokes-Adams attacks could be successfully treated by external cardiac pacing and could be life saving. However, limitations soon became evident when it was discovered that this form of pacing was of no value in patients with ventricular tachycardia, ventricular fibrillation, and in patients who were conscious. The voltages required for external stimulation are in the range of 500-200 volts with a 2 msec duration of pulse. This voltage was quite painful to patients who are conscious and often, in fact, produced electrode burns at the site of pacing electrodes (6).

This method of pacing is now used only in emergency situations when other forms of pacemaker methods are unavailable.

2. Percutaneous direct myocardial pacing

This mode of temporary pacemaking is accomplished by means of insertion of a needle directly through the chest wall into the heart through which an insulated wire with a platinum tip is inserted into the heart chamber and direct pacing is carried out through this wire. Thevenet first described this technique using a needle which was passed through the fourth or fifth left intercostal space directly into the right ventricular cavity (7). Lillehei (8, 9), Roe (10) and Ross and Hoffman (11) have all used forms of this direct percutaneous myocardial pacing using different techniques, following such procedures as anesthetic arrest and postsurgical arrest.

Currently at this hospital we are using an Ele-Cath bipolar pacing wire which is inserted through a #18 gauge long needle through the third or fourth intercostal space directly into the left ventricular chamber. An illustration of the anatomic location of insertion of this needle is seen in the appendix on page 1 (Fig. 1). This illustration is taken from Bellet (12). The needle is inserted through the skin with a stylet in place at the third or fourth intercostal space at the left sternal border. When it is felt that the needle point has passed through the rib cage, the stylet is removed and a syringe is placed on the end of the needle and continuous aspiration is accomplished while the needle is inserted into the right ventricular chamber. Evidence of arriving in the chamber is indicated when dark blood is rapidly withdrawn into the syringe. At this point the syringe is removed and the pacing wire is inserted to the second white mark on the wire which indicates that the electrode tip is 1 cm in the cardiac chamber. The needle is removed over the wire being careful not to extract the wire while removing the needle. A pacemaker generator is then attached to the wire by means of alligator clips to the

most distal end of the pacing wire and the wire manipulated in the chest wall until capture is obtained. This method is the most rapid means of inserting a pacemaker into the heart in an emergency situation when this arises. No real complications have arisen from the use of this pacemaker. However, very few patient survivals have been credited to its use primarily due to the fact that this pacemaker is a last resort method of attempt at cardiac resuscitation. This emergency pacemaker equipment is available at Parkland in the emergency room, the medical intensive care unit and coronary care unit, and the catheterization laboratory for immediate use.

3. Transvenous catheter pacing

This method of insertion of a pacemaker into the heart is the technique of choice at this hospital and was originally described at Montefiore Hospital in New York (13). A pacemaking electrode catheter is passed through a vein under direct fluoroscopic vision into the right atrium and then manipulated through the tricuspid valve into the right ventricle. The electrode wire is impacted into the right ventricular apex among the trabeculae so that it will remain stably located in that position. The right ventricle is then captured by means of a pacemaking battery generator box. This may be either a demand or standard fixed-rate pacemaker, described in more detail under permanent pacing.

a. Selection of entry site

Many different sites have been utilized for the insertion of the transvenous pacemaker: the cubital and antecubital vein in either arm, the external jugular and occasionally the internal jugular, the femoral vein, the subclavian vein, and even retrograde from a brachial artery in the left ventricle.

There are advantages and disadvantages of the various entry sites. The external jugular vein is one of the most commonly used veins. However, it frequently is obstructed as it passes into the thoracic cavity or its course is tortuous when it connects with the subclavian vein, and for these reasons it is frequently difficult to pass the catheter through this vein by direct cutdown technique. The internal jugular tends to bleed prolifically and is a rather deep-lying vein and difficult to use. The arm veins are quite adequate; however, we find that the catheters frequently become dislodged when using the arms simply because the patient displaces the catheter from his heart chamber when he moves his arm, and therefore we refrain from using the arm except when other routes are inaccessible. The femoral vein tends to be uncomfortable for the patient because of its location.

Currently at Parkland we are using a percutaneous direct subclavian vein insertion of a semifloating bipolar pacemaking electrode. An illustration of this technique may be seen in the appendix on page 1 (Fig. 2). A #14 Jelco needle is inserted under

local anesthesia at the junction of the proximal and middle third of the clavicle at its inferior margin. A syringe is attached to the Jelco needle, and the needle is progressed slowly beneath the clavicle toward the xiphoid notch. Close observation will reveal the internal jugular vein progressing beneath the sternocleidomastoid and passing into the jugular notch. This is used as a landmark for directing the needle. The angle of insertion anteriorly-posteriorly is about 45° (14). This technique is entirely on the outside of the thoracic cavity, since the subclavian vein runs beneath the clavicle on top of the rib cage. When venous blood is aspirated into the syringe, the syringe and needle are removed from the polyethylene outer sheath and then a bipolar pacing wire is passed through the sheath into the subclavian vein and under direct fluoroscopic vision into the superior vena cava, right atrium, and right ventricle. We have had few complications from this technique, and this entry site has been quite satisfactory in management of the patient on the wards since 1) it seems to be comfortable for the patient, and 2) dislodgement is uncommon since this point of insertion is relatively immobile.

b. Positioning and threshold measurement

Different sites for pacing capture of the heart can be selected. Atrial pacing can be accomplished by insertion of the pacemaker into the right atrial appendage or at the junction of the superior vena cava and right atrium. Atrial capture is of value in patients with sinus bradycardia or sinus rhythm but is of *no value in patients with AV block.* The catheter is easily dislodged from the atrium and therefore not usually a satisfactory site for pacing.

The right ventricle is the most commonly selected chamber for pacing. Capture can be obtained from anywhere in the right ventricular chamber or the pulmonary outflow tract. We have found through experience that the site which is most easily entered and the one from which the pacemaker tends not to be dislodged is the right ventricular apex.

Clinically, threshold is the minimum amperage required to produce a contraction and is considered in more detail under that section. A threshold measurement of the milliamperage necessary to capture the heart is recorded. If the milliamperage required to pace the heart is below 1.5, we consider this a satisfactory site for pacing. Occasionally, by necessity, we must settle for a 2 ma threshold. Most of the threshold measurements are in the range of .5-.7 ma. The pacing amperage then is set 1-2 ma above the minimum threshold and pacing is instituted.

A chest x-ray is always taken after a pacemaker insertion of the pacemaker to determine correct positioning and for future reference in case the pacemaker becomes dislodged.

c. Unipolar versus bipolar pacemakers

Stimulation of the heart can be carried out either by use of two electrodes placed either in or upon the heart (endocardial versus epicardial) or with one electrode in or upon the heart and the other situated elsewhere on the patient. If both electrodes are in or upon the heart, this is a bipolar system, and if one is in or upon the heart, this is a unipolar system. A bipolar pacemaker catheter simply means that two wires are present in the catheter, and in a unipolar pacemaker catheter only one wire is present in the catheter. When a unipolar catheter is used, then a second electrode must be placed in the skin. The site chosen is usually somewhere close to the entry site of the catheter and the electrode is a stainless steel wire which is sutured to the skin, insulated, and connected to the positive pole of the pacemaking box. This electrode is termed the "indifferent" electrode. It is important to always connect the negative pole of the pacemaking apparatus to the intracardiac catheter in a unipolar system. Some disadvantages are present with a unipolar system. It is difficult to capture a heart when the unipolar catheter becomes free floating in the chamber of the heart, for the current density is not always sufficient to capture in the intracavitary position. Furthermore, it is almost impossible to use this pacemaker with pacing milliamperages above 5 ma since the patient begins to complain of muscle twitching and pain at the site of the "indifferent" electrode.

The bipolar electrode has the advantage of being able to capture the ventricle when the catheter tip is free floating in the chamber. This is of considerable value in emergency situations. High milliamperages (15-25 ma) can be used with this catheter before the patient begins to complain of any muscle twitching in the anterior chest wall.

Another advantage of the bipolar system is that if wire breakage occurs in one wire electrode, then this catheter can simply be used as a unipolar pacing catheter. This necessitates the reversing of polarity of the bipolar systems every 12 to 24 hours to prevent an abnormal rise of threshold. The bipolar system does act on the blood like an electrophoretic system, and small fibrous clots may form at the tip resulting in a rise in threshold. Fortunately, major clotting has not been a problem.

In clinical practice either method is quite satisfactory. However, the bipolar system perhaps has a few advantages and this is the system usually utilized in this hospital.

d. Blind positioning of electrodes

In emergency situations it is frequently impossible to move the patient into a room where fluoroscopy is available for placement of the endocardial electrode. Kimball (15) and Harris (16) have described a method of blind insertion of a pacemaking wire into the right ventricle for pacing. The same technique is used as

described above for insertion under fluoroscopy except that electrocardiographic monitoring is used to determine the level fo the catheter tip. After insertion of the bipolar pacing catheter through the Jelco needle into the subclavian vein the catheter is connected by a sterile alligator clip and wire to the V lead of an electrocardiogram. Care is always taken to use a separate ground wire to ground the electrocardiogram to the patient to eliminate any possibility of cross-current flow through the heart via the electrocardiographic machine. Under direct electrocardiographic monitoring the catheter is passed down the superior vena cava into the right atrium where typical intracavitary right atrial P wave is obtained and then the catheter is manipulated to the tricuspid valve and into the right ventricle where characteristically large voltage QRS intracavitary potentials are obtained. A typical set of these electrocardiograms is illustrated on page 1 of the appendix (Fig. 3).

e. Transesophageal pacing

Two reports of pacing of the heart via a transesophageal cardiac pacemaker have been reported. Shafiroff and Linder (17) attempted pacing by means of electrodes in the esophagus. The threshold proved to be 30-50 volts and at that level the tendency to muscle contractions and local tissue burning made prolonged pacing impractical. However, Burack and Furman (18) were successful in pacing the heart of a patient at a fixed rate and on the demand mode for over two days with no adverse effects to the esophagus. This method of cardiac pacing requires further clinical study but certainly holds exciting possitilities.

II. Permanent Pacemakers

A. Indications

The indications for long-term pacing are listed below (19, 20).

*INDICATIONS FOR PERMANENT PACEMAKER*

1. Stokes-Adams attack
2. Sinus bradycardia with CHF or angina
3. Low cardiac output improved with temporary pacing
4. Acquired permanent second or third degree block
5. Sinus arrest
6. Congenital third degree heart block if symptomatic
7. Overdrive for arrhythmia control

If the symptoms of Stokes-Adams attacks, congestive heart failure or cerebral symptoms, or decreased renal output are in doubt, then these patients receive a trial of temporary pacing up to three to four weeks to see if improvement occurs before inserting a permanent pacemaker.

Almost all permanent pacemakers which are inserted at this hospital are of the demand type, Medtronics #5841 and electrodes #5816. Of interest is the cost of these permanent pacemakers, which is around $1,000 for the pacemaking unit alone not including any hospitalization cost. This becomes important when you consider that the average life expectancy of the power unit in a permanent pacemaker is around 24 months. Several different companies market implantable pacemakers: Medtronics, American Optical, Cordis, Devices Limited in England, General Electric, Elema.

B. Transvenous versus epicardial leads

Two types of electrodes are available for connection to a permanent pacemaking unit. These are 1) transvenous endocardial catheters and 2) epicardial-sutured electrodes. The latter require a thoracotomy for insertion, whereas the former can be done under local anesthesia in the cardiac catheterization laboratory. Currently we are using almost exclusively the transvenous endocardial pacemaking electrodes. The advantages and disadvantages of each are listed below.

*TRANSVENOUS PACING*

1. Advantages
   a. Ease of installation
   b. Minimal surgical risk
   c. Ideally suited for elderly, does not require thoracotomy
2. Disadvantages
   a. Perforation with and without tamponade
   b. Diaphragm pacing
   c. Dislodgement of catheter
   d. Higher threshold
   e. Less permanent

*EPICARDIAL LEADS*

1. Advantages
   a. More permanent
   b. Lower threshold
   c. Increased reliability
2. Disadvantages
   a. Requires thoracotomy
   b. Broken electrodes

The implantable pacemaker units are now prepared with a circuitry design powered by mercury cell batteries and covered by either silicone rubber or epoxy resin. These can be sterilized by ethylene oxide and are received from the manufacturer presterilized. The unit is implanted under strictly sterile conditions in an operating room environment to prevent sepsis from occurring around the foreign body.

C. Types of units

There are several types of pacemakers available.

1. Standard or asynchronous

This is a fixed rate pacemaker in which the rate is preset at the factory and cannot be varied at time of implantation. Various rates can be obtained from 60 to 80 beats/min or higher if specially ordered. These pacemakers were the first to be designed and are used much less commonly now than previously for reasons that are discussed below. A typical EKG from a patient with this type of pacemaker can be seen on page 2 of the appendix (Fig. 4).

2. Ventricular synchronous

Ventrocor is at all times synchronized to the R wave of ventricular contraction, and no delay occurs between the onset of a QRS complex and emission of the stimulating impulse. A typical electrocardiogram is shown in the appendix, page 2 (Fig. 5). In the presence of spontaneous ventricular activity, the pacemaker impulse continually falls into the absolute refractory period of ventricular contraction (in the QRS) and no response is produced at normal or rapid ventricular rates. However, should the spontaneous ventricular rate fall below the preset 70 electrical contractions per minute, the pacemaker will spontaneously fire the ventricle at the preset rate of 70. It then acts like an asynchronous or standard type pacemaker unless the spontaneous heart rate again exceeds the 70 per minute which results in the impulse again falling into the refractory period of the heart's normal QRS. A refractory period of 400 msec follows each pacemaker emission. A premature ventricular contraction falling in this refractory period will not be detected by the pacemaker and can result in a pacemaker artifact occurring in the vulnerable period of the T wave which could potentially produce ventricular fibrillation.

3. Atrial synchronous or Atricor

This is an implantable pacemaker requiring a thoracotomy. Two pairs of leads are used; one pair is sutured to the atrium and the other is sutured on the ventricular wall. These are attached to the pacemaking unit which senses the P wave and then after a suitable delay (P-R interval) the pacemaker then triggers the impulse into the ventricle. This has the obvious physiologic advantage of allowing atrial contraction to contribute to ventricular filling. The most important advantage is that of variation in the heart rate and therefore the cardiac output according to the patient's exercise demands. As the atrial rate increases with exercise or excitement, the ventricular rate can correspondingly increase. This pacemaker has a lower limit of 60 beats/minute and an upper limit of 120 beats/minute. As the normal atrial rate exceeds or falls below these two levels, then the pacemaker will automatically begin pacing at 60 beats/minute. The disadvantages

in this pacemaker are that it requires a thoracotomy and frequently has difficulty sensing the P wave because of low voltage or atrial arrhythmias, and consequently may act as a demand pacemaker at 60 beats/minute resulting in a competitive rhythm. Also when the atrial rate exceeds 120 with exercise, there may be a sudden reversal of the ventricular rate to 60/minute producing symptoms of bradycardia in the patient. This is probably the pacemaker of choice in young patients with symptomatic congenital block or in young patients with acquired block and essentially no atrial disease or atrial arrhythmias. An example of an EKG with atrial synchronous pacing is found in the appendix, page 3 (Fig. 6).

4. Demand pacemaker

This pacemaker has a sensing circuit which detects the heart's own spontaneous electrical activity. This circuit is designed so that it turns the pacemaker off when the spontaneous heart rate is above that which was previously set in the pacer. It turns on when the heart rate drops below the previously set rate of the pacemaker. In other words, these pacemakers detect spontaneous ventricular activity and do not produce a stimulus unless the R-R interval exceeds a preset limit. An example is found on page 3 of the appendix (Fig. 7).

The demand unit is the one used almost exclusively in this hospital and in the majority of other hospitals around the country. Its advantage is that it does not produce a competitive rhythm with the heart's own intrinsic activity. This is especially important since more than a quarter of all patients requiring pacing for Stokes-Adams attacks are in sinus rhythm and require ventricular stimulation when an attack of complete block supervenes (21). The pacemaking requirements for this group of patients are ideal for a demand-type of pacemaker which will automatically turn itself on and off as needed.

The disadvantage of this type of pacemaker is that it is ineffective in the treatment of overdriving an abnormal focus in the heart. If a ventricular tachycardia or atrial tachycardia supervenes, the pacemaker simply shuts itself off. In addition, if a patient is in normal sinus rhythm above the rate of the preset level of the pacemaker the electrocardiogram does not reveal any activity and it is difficult to find if the pacemaker is working satisfactorily. Carotid sinus massage or injection of 1-2 mg Tensilon can sometimes be used to slow the heart rate sufficiently for the demand unit to supervene and allow one to determine its functional rate (21). Some demand units can be activated by external magnets for purposes of checking their pacing rate and battery charge level.

5. Radiofrequency induction type

This pacemaker utilizes either epicardial leads or pervenous catheter leads which are attached to an induction coil implanted

subcutaneously. The power source is outside the body and operates through a second induction coil which is simply taped to the skin above the primary coil. This type of unit has the advantage that the rate and power can be controlled easily to compensate for necessary increases or decreases in rate with activity or any changes in threshold which might arise. It also has the advantage in that the batteries can be easily changed without a second operation.

The disadvantages of this type of apparatus are those of any external type of apparatus, namely, that of keeping the external coil in contact with the internal coil in a perpendicular fashion and frequent battery changes because of the large energy required in the external transmitter. The danger is that of the loss of pacing if the external coil becomes dislodged from the skin. This method has not been widely used at this time.

6. Power supplies

All of the pacemaker units are dependent upon a form of chemical to electric energy for their power supply. In present usage are the popular mercury-mercury chloride batteries which have a relatively long lifespan. The average life expectancies for the different pacemaking units are listed at the end of this section.

The Atomic Energy Commission is now investigating a nuclear power supply, plutonium-238, which can be used as a continuous source of power and will essentially produce a lifetime battery. This unit is under investigation but is rather large and bulky because of the shielding required. However, recent advances have indicated that a workable unit will soon be available experimentally.

A bioelectric form of power is being developed in which large electrodes are planted beneath the skin and use the redox potential between the plates and the tissues to form a chemical reaction which in turn produces an electrical source of power. These electrodes are too large to be of practical value at this time. However, they do have potential for the future.

C. Clinical guidelines for selection of pacemaker type

It is important to realize that no mode of pacing duplicates completely the electrophysiologic and hemodynamic flexibility that characterizes the normal beating heart. It is imperative that each patient be evaluated carefully and the type of pacing that best meets his needs be selected. A few guidelines may be helpful for selection of the individual pacemakers.

A fixed rate ventricular pacemaker is indicated for the treatment of permanent complete heart block. The difficulty arises here in determining if the heart block is in fact permanent. Another use is for the suppression of tachycardias. This pacemaker allows the use of the overdrive mechanism described in another section to control tachyarrhythmias. The antiarrhythmic agents may be of value in conjunction with this type of pacemaker

in suppressing any competitive rhythms or parasystoles which may develop.

Synchronized atrial pacing is employed in patients in whom the contribution of a properly-timed atrial systole and maintenance of cardiac output are desirable and a changing rate may be of assistance in day-to-day activities. This should be used in young patients with no known atrial arrhythmias.

A demand ventricular pacemaker is most useful for those who have normal rhythm interrupted by periods of AV block or of sinus arrest. This pacemaker has an advantage of not developing a competitive rhythm with the heart's own intrinsic pacemaker.

The ventricular synchronous pacemaker can be used with the same indications as the demand pacemaker.

*INDICATIONS FOR PERMANENT PACEMAKER TYPES*

1. Atrial synchronous
   a. Young patients with permanent third degree block
   b. When atrial intracavitary lead is above 1.5 mv
   c. When no atrial arrhythmias or atrial disease are found
   d. When thoracotomy is not contraindicated
2. Standard asynchronous
   a. In recurrent ventricular tachycardia
   b. In *permanent* third degree block
3. Ventricular synchronous (refractory period)
4. Demand
   a. Elderly patients with transient blocks
   b. In patients with atrial disease, arrhythmias, sinus arrest
   c. In all cases not listed above
      Most frequently used now

*FOLLOW-UP OF PATIENTS WITH PERMANENT PACEMAKERS*

1. Heart rate
2. Chest film, position of electrode
3. Films of battery
4. Electrocardiogram
5. Impulse display
6. Card to say patient has pacemaker, type, and date of insertion

| *TYPES OF PACEMAKERS* | *AVERAGE REPLACEMENT TIME* | *IMPLANTED* |
|---|---|---|
| 1. Atrial synchronous | 15 mo | Thoracotomy |
| 2. Ventricular synchronous | 18 mo | Transvenous |
| 3. Demand | 21 mo | Transvenous |
| 4. Asynchronous | 24 mo | Transvenous |
| 5. Radiofrequency | - | - |
| 6. Nuclear powered | - | - |
| 7. Bioelectric powered | - | - |

III. Pacing in Acute Myocardial Infarction

A. Incidence and mortality of heart block with myocardial infarction

The occurrence of second degree and complete heart block in acute myocardial infarction has been considered generally a poor prognostic sign. The mortality reaches at least 50% with complete heart block in acute myocardial infarction (22). However, of the survivors only 10% or less retain a permanent block for more than two weeks (22-24). The long-term mortality among patients returning to sinus rhythm is comparable to patients who never developed block (25).

**INCIDENCE OF HEART BLOCK AND SINUS BRADYCARDIA IN AMI (26)*

| | |
|---|---|
| First degree | 9.7% |
| Second degree | 7.0% |
| Third degree | 5.6% |
| Sinus bradycardia | 12.6% |

* Average incidence in 360 patients in literature survey.

Therefore 35% of patients with acute myocardial infarction might benefit from pacing.

**MORTALITY ASSOCIATED WITH HEART BLOCK IN AMI (26)*

| | | |
|---|---|---|
| First degree | 26% (15-45) | (anticipated) |
| Second degree | 44% (30-60) | |
| Third degree | 58% (25-100) | |

* Survey of literature (nonmonitored patients).

The mortality with first degree block is similar to the anticipated mortality in myocardial infarction without block. The almost doubled mortality with second and third degree block *should* be improved by ventricular pacing. No good reports on the mortality associated with sinus bradycardia and myocardial infarction have appeared. However, many patients with slow heart rates are seen to hemodynamically improve significantly, *i.e.*, progress from shock to a stable blood pressure, with pacing (27-29).

Paulk and Hurst (30) reported 1400 patients with infarction and found 47 (3.3%) who developed complete block. Forty-one of these 47 were paced with 22 survivors, 5 of whom developed permanent block. Nineteen of the 41 had cardiac arrest with resuscitation before pacing with a mortality of 63%. Twenty-two of the 41 had complete block but no major cardiac arrest with a mortality of 31%.

Cosby (31) and Courter (32), however, found little difference (39%) in mortality between paced and nonpaced patients. More encouragingly, Bruce (33) reported only a 23% mortality with pacing, and Sowton (21) reported 11% mortality with pacing. Harris and Bluestone reported a 36% mortality (34).

In a recent review of the literature (35) of 125 cases paced with acute myocardial infarction and heart block, 60% recovered, with 9% remaining in permanent block. Close scrutiny of the cases and remembering the high complication rate with transvenous pacing in its infancy (*i.e.*, 19% incidence of ventricular fibrillation with pacer insertions (30)), *artificial pacing probably halves the mortality in patients who do not have a major episode of cardiac arrest.*

B. Indications for pacing

Below is a comprehensive list of two hospitals' indications for pacemaker insertion.

*REASONS FOR INSERTION OF TEMPORARY TRANSVENOUS PACEMAKERS IN ACUTE MYOCARDIAL INFARCTION (26)*

| | Kimbell & Killip | Miltzer & Dalla |
|---|---|---|
| % of total | 84/242 (35%) | 51/200 (25%) |
| First degree | 4% | 2% |
| Second degree | 18% | 12% |
| Third degree | 29% | 32% |
| Sinus bradycardia | 42% | 20% |
| Ventricular fibrillation | 7% | 6% |
| Overdrive | - | 28% |
| Pacemaker used | 37/84 (44%) | 21/51 (40%) |

The indications used for pacing in acute myocardial infarction at Parkland Memorial Hospital are listed at the end of this section.

Hemodynamically slow rates below 60/min tend to decrease cardiac output significantly and therefore rates should be maintained above 60. However, rates above 60-70 only increase resting cardiac output by no more than 10-15% but do appreciably increase the work of the heart (and probably myocardial oxygen consumption) as measured by the time-tension index (26); see appendix, page 3 (Fig. 8). Clinically, the slowest rate that maintains the blood pressure is the proper rate selection.

C. Location of infarction and block

Anatomically it is well established that the artery supplying the AV node arises from the right coronary artery 80-90% of the time (36). The right coronary artery is usually involved in inferior (posterior) myocardial infarctions. Therefore, it is of interest to analyze the mortality with pacing associated with anterior and inferior myocardial infarctions. Julian *et al.* (37) analyzed 81 patients with acute myocardial infarction with third degree block and pacing and found the following results:

*JULIAN - 81 PATIENTS PACED WITH ACUTE MYOCARDIAL INFARCTION*

| | Mortality | Benefited |
|---|---|---|
| Inferior infarction | 26% | 67% |
| Anterior infarction | 86% | 9% |

Stock and Macken (38) continuously monitored the electrocardiogram of 24 patients with acute myocardial infarction who developed complete heart block. They found the patients with posterior infarction usually progressed through first degree and Mobitz type I and rarely II second degree block but not BBB before developing complete heart block, while those patients with anterior infarction and complete block suddenly developed complete block or passed through BBB or Mobitz type II block first, indicating septal involvement with the anterior infarction and block and an AV nodal lesion in posterior infarction (39).

The explanation for this discrepancy in mortality between anterior and inferior myocardial infarctions with block may well be explained by pathological findings of Sutton and Davies (40). They found upon a detailed histological examination of the conduction system in 29 cases with acute myocardial infarction with block that in the 24 cases with posterior (inferior) infarction the coronary artery supplying the AV node was occluded, whereas in the 5 cases with anterior infarction the AV nodal artery was patent but massive damage to the conduction system (both bundles) was present. This may well explain the high mortality with anterior infarction and block plus the transient nature of the block with posterior infarction.

*INDICATIONS FOR PACING IN MYOCARDIAL INFARCTION*

1. Second degree heart block
2. Third degree heart block
3. Sinus bradycardia with rate below 60/min unresponsive to drugs
4. Runs of ventricular tachycardia
5. Frequent PVC's with bradycardia
6. Prior to suppressive therapy for ventricular arrhythmias when heart block or sinus arrest might occur

IV. Pacing for Control of Arrhythmias

A. "Overdrive" pacing for tachyarrhythmias

"Overdrive" pacing may be defined as pacing at a rate in excess of the heart's own intrinsic rate (which may be either atrial or ventricular) with the purpose of suppression of the heart's own impulse focus. The most common use is ventricular pacing at a rate higher than the normal sinus rhythm in order to suppress an irritable focus which may be precipitating paroxysmal ventricular tachycardia or frequent PVC's.

The primary purpose of pacing is to prevent life-threatening arrhythmias such as ventricular asystole and ventricular fibrillation. However, the use of pacing has been expanded to control refractory tachycardias, atrial and ventricular, which are hemodynamically compromising but not always emergencies. In addition,

pacing will permit the use of antiarrhythmic drugs without fear of producing an increase in heart block or suppression of an impulse center.

In 1964 Sowton, Leatham, and Carson (41) used artificial pacing to treat two patients having normal AV conduction who developed rapid ventricular arrhythmias refractory to conventional drug therapy. In these patients the arrhythmias were suppressed by pacing the right ventricle with a temporary transvenous catheter at rates of 120 beats/min. Heiman and Helwig (42) also used intracardiac pacing to control ventricular tachycardia and fibrillation; in one patient stimulation of the atrium at a rate of 150 beats/min was employed while in the other patient the ventricle was paced at a rate of 130 beats/min.

Kastor, DeSanctis, Harthorne, and Schwartz (43) reported two patients in whom transvenous atrial pacing at rates of 110 beats/min was used to treat refractory ventricular arrhythmias, and Cohen, Buccino, Morrow, and Braunwald recorded the use of a radiofrequency pacemaker attached to both the left atrium and ventricle in conjunction with propranolol to prevent attacks of recurrent ventricular tachycardia in an otherwise healthy young girl (44). Several other reports of similar "overdrive" pacing by both temporary and permanent pacemakers have been successfully documented (45-49). Two cases of recurrent ventricular fibrillation, unresponsive to drug therapy but suppressed by ventricular pacing associated with hypomagnesemia, have been reported by Loeb and co-workers (50).

Thioridazine (Mellaril), a psychotropic agent, has been reported to cause electrocardiographic repolarization abnormalities, supraventricular arrhythmias, first degree heart block, intraventricular conduction delays, and nonconducted premature atrial beats (51). In addition, several episodes of fatal ventricular tachycardia have been reported (52). Schoonmaker *et al.* (52) recently reported a successful control of a patient with recurrent ventricular tachycardia induced by Mellaril with ventricular pacing ("overdrive") previously unresponsive to drug therapy.

B. Postoperative pacing

Recently surgeons have begun to routinely insert pacing wires in the atria or ventricle during heart operations to be used if needed in the postoperative period to suppress irritable foci in the heart (53, 54).

These reports document that it is feasible to suppress episodes of ventricular tachycardia and fibrillation and atrial tachycardia in the presence of normal AV conduction by pacing either the atrium or ventricle at a suitable rate.

C. Mechanisms of action

The mechanisms by which pacing, either atrial or ventricular, successfully prevents recurrent tachycardias are uncertain. Some data are available and applicable: 1) Slow rates predispose to

ectopic beating, possibly because diastole is prolonged. During slow rates the recovery of excitability is prolonged, and the likelihood of reentrant excitation is enhanced. Rapid rates decrease the time of recovery of excitability and make repolarization more homogeneous throughout the ventricle. 2) Depolarization wave fronts produced by exogenously applied stimuli may make reentry pathways refractory or cancel reentry wave fronts and therefore suppress ectopic foci (55).

Physiological factors which may also play a role in the suppression of pacemakers by "overdriving" include 1) the release of acetylcholine and other autonomic mediators by the pacing stimulus and 2) shifts in potassium concentrations in the intra- and extracellular milieu (54).

Ventricular pacing has been successfully used to control atrial tachycardia despite the fact that it should not be expected to affect the atrial ectopic activity in atrial tachycardias. Two explanations can be offered: 1) Improvement in atrial function through improved coronary circulation consequent to restoration of effective regular ventricular rhythm may abolish the atrial tachycardia by favorably affecting the stimulation and fibrillation threshold of the atria. 2) Ventricular pacing causes constant retrograde depolarization of the atrioventricular node and the atria and thus suppresses the nodal or atrial pacemakers.

D. Problems

1. Atrial pacing

Catheter placement is the most difficult problem encountered with transvenous atrial pacing. These catheters become dislodged or are free-floating and frequently atrial capture is lost or else the current required for capture is a discomfort to the patient. The patient may develop atrial flutter or fibrillation and then the atrial pacing is of no value. However, recent cardioversions of atrial flutter with atrial pacing (atrial "overdrive") with a return to normal sinus rhythm have been reported (56).

2. Ventricular pacing

The use of ventricular pacing in the presence of normal atrioventricular conduction results in two distinct problems: first, the risk of pacemaker-induced ventricular fibrillation caused by the stimuli falling in the vulnerable period of the cardiac cycle, and, second, the hemodynamic problems associated with asynchronous pacing (49). In the majority of patients who manifest pacemaker parasystole the pacemaker stimulus intensity does not reach the threshold for provocation of ventricular fibrillation, but under certain circumstances the fibrillation threshold of the heart can become low enough for a pacemaker impulse to provoke repetitive responses (ventricular tachycardia) or ventricular fibrillation. The second problem involves consideration of the hemodynamically unfavorable effects of loss of atrial contribution to ventricular

filling with asynchronous ventricular pacing and is discussed in more detail in a later section.

The primary therapy of ventricular arrhythmias in the absence of AV block is the use of myocardial depressant drugs such as quinidine, procainamide, lidocaine, diphenylhydantoin, and propranolol. However, drugs of this type, usually administered to *depress* ectopic activity, have been shown under some circumstances to *increase* paradoxically the ventricular arrhythmias (54). When such is the case, rapid pacing may be life-saving. Furthermore, after adequate pacing has been established, these antiarrhythmic drugs may then be concomitantly employed with less risk and more efficacy should they be necessary.

V. Results of Permanent Pacing in Heart Block

Indications for permanent pacing include complete heart block with Stokes-Adams attacks; sinus bradycardia with congestive heart failure, angina, syncope, or low cardiac output improved with temporary pacing; periods of sinus arrest; overdrive for control of persistent ventricular arrhythmias such as bigeminy, multifocal PVC's, runs of ventricular tachycardia resistant to the usual forms of drug therapy; congenital complete heart block with symptoms of angina, syncope, congestive heart failure, or progressive cardiac enlargement.

The most common indication for permanent pacing with implanted pacemakers is complete heart block, and therefore the incidence, etiology, and mortality will be reviewed.

A. Congenital block

Complete heart block occurs in 1/10,000 to 20,000 live births (21, 57). Over half the cases are associated with other congenital cardiac abnormalities (58); the more common are corrected transposition and ostium primum defects.

Prognosis is good in uncomplicated block (59, 60). On exercise there is a considerable increase in the ventricular rate and cardiac output with the capacity for physical exercise near normal (61, 62). Stokes-Adams attacks may occur but are rare, and the risk for an individual patient has been estimated at 10% (63-65).

With this low incidence of syncope and relatively normal hemodynamics, we reserve pacing in congenital heart block for those patients that become symptomatic, *i.e.*, Stokes-Adams attacks, congestive heart failure, angina.

B. Postsurgical block

Heart block is not uncommon after heart operations for the repair of lesions close to or involving the conduction system, *i.e.*, ventricular septal defect, tetralogy of Fallot, aortic stenosis, ostium primum defect. This complication may be as high as 10% (66, 67). The prognosis for postoperative heart block is poor, with

about an 80% mortality (9). The ventricles can be paced during and in the immediate postoperative course with a temporary unit and later replaced with a permanent unit if the block remains. The mortality is reduced to 14% with pacing in these postoperative patients (66).

C. Acquired heart block

The incidence of complete heart block in the general population ranges from 26 to 63 per million population per year, with an average estimated at 5/100,000 (68-70). The etiology may be extremely variable as seen from the following list:

| *ETIOLOGY OF HEART BLOCK* | *INFECTIONS CAUSING HEART BLOCK* | |
|---|---|---|
| Primary heart block | Chagas' disease | Tuberculosis |
| Arteriosclerotic heart disease | Scarlet fever | Typhoid |
| Hypertension | Pneumonia | Typhus |
| Cardiomyopathies | Influenza | Mumps |
| Diphtheria | Measles | Rubella |
| Syphilis | Malaria | Amebic hepatitis |
| Diabetes mellitus | Pertussis | |
| Rheumatic fever | | |
| Rheumatoid arthritis | | |
| Nonspecific myocarditis | | |
| Myxedema | | |
| Progressive muscular dystrophy | *DRUGS CAUSING HEART BLOCK* | |
| Hemachromatosis | Digitalis | Streptomycin |
| Cardiac lymphangitis | Potassium | Prostigmin |
| Paget's disease | Quinidine | Pilocarpine |
| Nonpenetrating chest wounds | Mellaril | Procaine amide |

The most common etiology for acquired heart block for years was thought to be arteriosclerotic heart disease. However, recent reviews of continuously recorded electrocardiographic monitoring systems and detailed pathological examinations have disclosed this not to be the most common etiology. In postmortem studies of patients with acquired complete block, less than 50% were due to coronary artery disease (74, 75). The most common finding in patients with complete heart block is focal fibrosis of the conduction system (74), and this has been substantiated clinically using coronary angiography (76, 77). The absence of myocardial disease or significant coronary artery disease in the majority of these patients makes them ideal candidates for treatment with artificial pacemakers (21).

Complete block complicates only about 5% of all acute myocardial infarcts with 90% returning to normal sinus rhythm within two weeks (22, 71-73).

Stokes-Adams attacks occur on an average in 67% of patients with acquired third degree heart block (78, 79).

Since Stokes-Adams attacks are the primary presenting symptom, it is important to determine the etiology of the cardiac arrhythmia producing them. Harris *et al.* (81) analyzed the monitored electrocardiograms in 100 patients during their Stokes-Adams attacks and found 77% had attacks of asystole alone, 7% ventricular tachycardia alone, and 16% with asystole and ventricular tachycardia. Thus 93% of Stokes-Adams attacks are associated with at least short periods of ventricular standstill and could thus be controlled by pacing. Sowton has emphasized that 25% of "permanent" complete heart block periodically returns to normal sinus rhythm, therefore making the selection of a demand pacemaker mandatory (21).

Other presenting symptoms which may occur in patients with complete heart block are congestive heart failure, angina, signs of cerebral ischemia, or low cardiac output which responds to increasing the heart rate with a pacemaker. It is important to point out that Friedberg reported heart block alone does not produce congestive failure in man, although it is a significant contributory cause. He claimed that coexistent myocardial or renal disease is needed to develop heart and renal failure (80).

*CLINICAL COMPLICATIONS OF COMPLETE AV BLOCK*

1. Severe bradycardia or asystole
2. Slow idioventricular rhythm contributing to ventricular tachycardia or fibrillation
3. Stokes-Adams attacks
4. Congestive heart failure

*MORTALITY IN ACQUIRED COMPLETE HEART BLOCK*

Without pacing - 50% in one year
With pacing - 10-20% in one year

*MORTALITY IN PERMANENT PACING*

1st year - 10%
2nd year - 16%
3rd year - 37%

Acquired "permanent" complete heart block probably should always be an indication for artificial pacing since the risk of fatal ventricular escape rhythms and asystole are forever present. The following discussion of mortality with and without pacing in acquired heart block is evidence to substantiate this.

Friedberg reviewed a series of 100 cases of acquired complete heart block with Stokes-Adams attacks and found a 50% survival for one year (72). Johansson reviewed 42 cases and also found a 50% survival for one year (64). Most of these deaths were sudden.

Cosby's (31) analysis of 100 unselected patients with complete heart block is particularly pertinent, as 50 were seen prior to

and 50 after the advent of permanent pacing. In patients without myocardial infarction the mortality was 60-80% before and 10% with pacing for one year.

Chardack (26) compared his results in 113 patients surviving operation for endocardial leads or those with transvenous leads and found 89% alive at one year with a normal expectancy in this age group of 96%. He compared these with Friedberg's patients who were untreated and this is shown in graph form in the appendix, page 3 (Fig. 9).

Morris (26) reports a similar finding and is shown in the appendix, page 3 (Fig. 10).

Harthorne (82) reported a series of 129 patients from the Mass General with permanent pacemakers Medtronics 5816 and 5818 between July 1965 and August 1968 with a mortality of 10% for 12 months, 16% for 24 months, and 37% for 36 months.

Siddons and Sowton reviewed the total published experience with all types of permanent pacemakers throughout the world (21). There were 1499 patients with 112 (7.5%) postimplant deaths and 163 (10.9%) late deaths.

These results indicate *at least* a 30% decrease in mortality in chronic complete heart block with pacing irrespective of the improvement these patients have in their activity levels due to improved hemodynamics.

## VI. Hemodynamics With Pacing

The changes in hemodynamics with an increase in heart rate with pacing in patients with heart block are of vital interest in the desirable rate setting, type of pacemaker selected (*i.e.*, atrial synchronous versus ventricular, demand versus asynchronous).

### A. Cardiac output

Changes in cardiac output with ventricular rate have been studied by many investigators (83-88). It has been firmly established that low cardiac output with complete heart block is augmented by increasing the rate with a pacemaker. A maximum resting cardiac output is reached in the range of 70-90/min (86, 89, 90) above which no increase is observed. See appendix, page 4 (Fig. 11), for a typical cardiac output rate response with a permanent implanted pacer.

Ross (91) and others have evidence to show that in normal subjects and in pacemaker patients with good myocardial function the cardiac output is not dependent upon pacing rates above 50-60/min but is adjusted largely by changes in stroke volume. However, in hearts with depressed myocardial function the stroke volume is relatively fixed and cardiac output becomes dependent upon the heart rate (21, 92).

B. Rate selection

The selection of an optimal heart rate is of importance and may require individualization in each patient. Each patient will have a rate at which he reaches maximum cardiac output and above which cardiac output will not change until the rate becomes fairly rapid and then cardiac output will again fall. Since myocardial oxygen consumption is dependent upon rate, the lowest pacemaker rate which gives a maximum cardiac output at rest should be selected. As can be seen by the example in the appendix, page 4 (Fig. 11), the cardiac output may become less rate dependent with chronic pacing. However, the rate at which the maximum cardiac output occurs does not change. This may indicate improvement of myocardial function with long-term pacing (21, 93).

Several studies are reported (28, 84, 94-97) with variable rates and cardiac output determinations to find the ideal pacemaker rate. These range from 50 to 105 beats/min with an average between 70 and 80 beats/min.

A method of determining the optimal heart rate may be based on a chart prepared from work by Sjöstrand (98) and found in the appendix, page 4 (Fig. 12), using the height and hemoglobin of the patient.

C. Atrial synchronous

The effects of atrial systole are important in evaluating a patient for an atrial synchronous pacemaker. Snyder and Wood (99) found an ineffective atrial systole resulted in a 10% decrease in cardiac output, while Mitchell (100) found that the influence of atrial systole in determining stroke volume was greater at higher heart rates. These results are confirmed in man with atrial synchronous pacemakers (101, 102) especially during exercise (103). However, in most patients at rest the cardiac output is essentially the same after a few minutes of stabilization with either ventricular or atrial pacing indicating that atrial-triggered pacers are of value with exercise but have little to offer in the basal state in most patients.

VII. Threshold

A. Definition

For purposes of clinical cardiac pacing, *threshold* is the minimem electrical impulse necessary to stimulate the heart. Regardless of the make or model pacemaker used, every implanted pacemaker system will have a threshold, below which the stimulus is too weak to excite the heart.

B. Exit block

Exit block is a clinical condition in which a normal pacemaker stimulus does not pace the heart because of abnormally high threshold.

C. Factors affecting threshold

Threshold is determined by a number of factors, including electrode position in the heart, electrode composition and size, pulse waveform, and electrode-tissue interface (104). The power output of the unit must exceed any threshold clinically encountered to prevent exit block from occurring.

Through experience, we know the threshold of stimulation should be less than 1 ma and below 2 ma is probably an absolute requirement for permanent pacing. Several reinsertions of the pacing electrode may be required before an area is found with an acceptable low threshold.

Studies of the changes in myocardial threshold have shown a rapid continuous rise in threshold during the first week with some slow increase occasionally occurring late (105, 106). This change may be of the order of magnitude of 3-8 times the initial threshold determination. Since most permanent pacemakers use stimuli in the 9-12 ma range, it is essential to select a pacing site with a minimum of ma requirement initially (*i.e.*, < 1.5 ma). In temporary pacing, it is often necessary to gradually increase the pacing stimulus (ma) during the first few hours or days following its insertion to allow for the increase in threshold.

Determinations of threshold after implantation of pacemakers have demonstrated that myocardial threshold in man is not a static quantity. On the contrary, it is highly variable, subject to spontaneous changes with physiologic and pharmacologic interventions. Some of the commonly encountered states in cardiac patients and the threshold changes encountered are listed in the following table (104-110).

*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

It becomes apparent why many patients with temporary pacemakers set at minimum pacing stimulus may only intermittently capture (develop exit block) since any number of factors may elevate the myocardial threshold to stimulation.

The reasons for the change in threshold are multiple. The excitation of a myocardium is a function of 1) the exciting stimulus, 2) the resting membrane potential, 3) membrane resistance, and 4) cellular threshold potential. For excitation to occur, the exciting stimulus must reduce the resting membrane potential to the level of the cellular threshold potential to activate the action potential. Any reduction in resting membrane potential of the myocardial cells will reduce the threshold requirements and an increase in cellular resting and threshold membrane potentials would increase the myocardial threshold (107-109).

The glucocorticoids such as prednisone have been useful in management of exit block in permanent pacemakers when it occurs. These drugs often result in an increase in responsiveness to the pacemaker which may continue after the steroid is discontinued (104, 110).

The mode of action of the steroids is uncertain but perhaps one or all of the following could be responsible: 1) changes in cell membrane permeability, 2) interferance with the sodium-potassium pump across the cell membrane, 3) potentiation of catecholamines (104).

Alterations in threshold are clinically useful not only in managing exit block but also in hearts manifesting irritability (coupled rhythms, etc) during the immediate postpacemaker implant period, in early power unit failure, in the choice of anesthetics, and during intravenous or other medical therapy during cardiac pacing.

D. Fibrillation threshold

A threshold for fibrillation also exists in the heart. Any current above this level results in fibrillation. This is usually 10-30 times the stimulation threshold in normal hearts; however, ischemic or diseased hearts or hearts subjected to drugs may have fibrillation thresholds approximating the stimulation threshold (26). Thus the pacing stimulus is usually kept to a minimum to avoid the fibrillation threshold. A graph of the relationship of fibrillation to stimulation threshold is illustrated on page 4 of the appendix (Fig. 13).

VIII. Complications of Pacemakers

A. Dislodgement of electrodes

A summary of the complications of pacemakers is given at the end of this section and on page 5 of the appendix (Fig. 17). The most commonly encountered complication of temporary pacemakers is the dislodgement of the electrode catheter from the right ventri-

cle. This usually is found to be retracted into the right atrium and atrial pacing may ensue; however, this is of no value in complete heart block. An example is seen on page 4 of the appendix (Fig. 15). Occasionally the catheter tip has floated into the pulmonary artery at which time ventricular capture is no longer present. The simple maneuver of attaching the catheter to the V lead electrocardiogram and obtaining an intracavitary tracing will allow one to determine if the catheter is lodged in the right atrium with the resultant right atrial intracavitary electrocardiogram or if it is in the pulmonary artery for then no intracavitary electrocardiogram will be obtained. Under the latter circumstances the catheter can simply be withdrawn an inch or two into the right ventricle and capture will result. Occasionally the pacing catheter has become dislodged and lies in the inferior vena cava resulting in diaphragm pacing. The patient complains of "hiccups" at 70-80 times per minute.

B. Shorting of electrode wires

The second most commonly encountered problem is a short-circuiting of the pacemaking electrodes at the site of attachment to the extension wire which leads into the pacing box. If the end of the catheter is allowed to become moist or the alligator clips are allowed to touch each other, a short-circuit will result and no electrical stimulus will reach the heart. It is simple to check for this particular problem by attaching an electrocardiogram to the patient to see if an electrical potential from the pacemaker is recorded. If a short has occurred somewhere before the pacing catheter enters through the skin, then no pacing spike will be obtained on the electrocardiogram. However, if the distal end of the catheter is simply dislodged from one of the cardiac chambers or one of the wires is broken in the pacemaker internally, then a pacing spike will still develop but no capture will follow.

C. Pneumothorax, hemothorax

Pneumothorax and hemothorax have occurred following the subclavian puncture technique but are unusual. Septicemia and infections from pacemakers are rare but may be fatal. This is rarely seen if proper sterile precautions are undertaken during insertion. Ventricular fibrillation can develop during insertion of pacemakers especially in patients with acute myocardial infarctions. These patients are almost always successfully defibrillated provided the proper equipment is in readiness. Ventricular perforation is not an uncommon finding (81, 111-116), and cardiac tamponade has been reported (117-119) but is rare. Electrode wire breakage is relatively uncommon with the new pacing electrodes but was not unusual in the older systems.

D. Battery failure

Battery failure in the current pacemaking units is heralded by a decrease in pacing rate by 5 beats/min with each battery component failure. Therefore, it is essential to obtain an electrocardiogram on follow-up visits from all pacemaker patients in order to determine the pacemaker rate to rule out any evidence of battery failure. The usual life of the power unit is about two years.

E. External electrical interference

External electrical interference can be a real problem and the patient should be warned to stay away from the electrical systems of automobiles, airplanes, lawn mowers, and other electrical apparatus (120). Special warning should be given against any diathermy treatments or riding on motorcycles or coming in close proximity to powerful radio or radar stations.

F. Asystole with discontinuation of pacing

Asystole following the cessation of artificial pacing must be emphasized. If the ventricles with complete AV block are paced at a rapid rate and pacing then ceases suddenly, the idioventricular pacemaker is depressed and is unable to institute a spontaneous rhythm until a latent period has elapsed (121, 122). The length of the ventricular asystole is often dependent upon the preceding pacing rate, being longer when pacing ceases from a rapid rate. When it is desired to remove a functioning pacemaker from a patient, it must be remembered that the pacing rate must be slowly decreased to allow the patient's own intrinsic pacemaker to function prior to the sudden cessation of the electrical impulse from the pacemaker.

G. Competitive rhythm

Wiggers and Wegria (123) reported and characterized the presence of a vulnerable period, during which short electrical shocks delivered to the ventricular muscle will result in ventricular arrhythmias including ventricular fibrillation. This period occurs near the end of systole at the time when the T wave of the electrocardiogram is being inscribed. An illustration of the vulnerable period in the electrocardiogram is found in the appendix, page 4 (Fig. 14). In fixed rate pacing a competitive rhythm between the heart's own basic intrinsic rhythm and that of the pacemaker rhythm may result in an occasional stimulation during the vulnerable period of the T wave. This is referred to as a competitive rhythm. Sowton (21) compared mortality rates between patients with competitive and noncompetitive rhythms and found the rate five times higher in the former. Individual case reports (122, 124-126) have documented the onset of ventricular fibrillation, tachycardia, or both at the moment of inscription of the pacemaker impulse. To avoid the vulnerable period in the cardiac cycle, most pacemakers now are of the demand variety with a very short

refractory period allowing them to sense any ectopic beats which may occur in the heart and shut the pacemaker off preventing any aberrant stimuli occurring during the T wave. An example of a pacemaker-triggered ventricular fibrillation may be seen in the appendix, page 5 (Fig. 16). This must be kept in mind when selecting any permanent pacemaker for a patient with third degree heart block. Sowton (21) has noted a 23% incidence of intermittent return to sinus rhythm in patients with cardiac pacemakers. Many of these patients were termed permanent heart blocks prior to insertion of the pacemaker. For this reason, we utilize the demand pacemaker almost exclusively in permanent pacing.

*COMPLICATIONS OF PACEMAKERS*

1. Power failure (battery or component)
   a. Acute
   b. Premature
   c. Runaway
2. Wire breakage
3. Electrical problems - dislodgement, diaphragm pacing
4. Increased threshold, especially atrial (exit block)
5. Rhythm complications
   a. Atrial pacing
   b. Ventricular synchronous - refractory
   c. Demand
6. Surgical
   a. Arrhythmia
   b. Infection
   c. Erosion through skin
   d. Postpericardectomy syndrome
   e. Hematoma
   f. Pneumothorax
7. Thrombosis
8. External electrical interference
   a. Auto, airplane, lawn mower, spark plugs, coils, etc
   b. Diathermy
   c. Powerful radio stations
   d. Motor cycle riding
   e. Electrosurgery
9. Infarction
10. Perforation and rarely tamponade

*FACTORS TENDING TO INCREASE RISK OF ARRHYTHMIAS DURING PACING*

Abnormal myocardium
Low threshold for ventricular fibrillation - particularly due to
- ischemia
- hypoxia
- sympathomimetic and other drugs
- anesthetic agents
- postsurgical inflammatory reaction

Parasystole (5x)
Multiple ectopic (nonpaced) beats
Stimulus intensity many times threshold for contraction, i.e. x10
Long impulse duration (over 5 msec)
Two simultaneous stimulation sites
Electrode in an ischemic area

*FACTORS ASSOCIATED WITH LOW RISK OF ARRHYTHMIAS DURING PACING*

Normal myocardium
Stimulus intensity just above threshold for contraction
Absence of nonpaced ventricular activity
Short impulse duration (less than 5 msec)
Small contact area between electrode and myocardium

## IX. Parkland Memorial Hospital Statistics

*PACEMAKER INSERTIONS AT PARKLAND MEMORIAL HOSPITAL*

| | |
|---|---|
| 1965 | 4 |
| 1966 | 13 |
| 1967 | 25 |
| 1968 | 74 |
| 1969 (Sept) | 66 |
| Total | 182 |

An indication of broadened clinical uses for pacemakers with a relatively low complication rate.

PACING WITH MYOCARDIAL INFARCTION AT PMH, 1967-69

*GENERAL*

| | | | | |
|---|---|---|---|---|
| Males | 15 | average age 66 | 6 deaths - | 40% |
| Females | 13 | average age 54 | 7 deaths - | 54% |
| Total | 28 | | 13 | 46% |

*LOCATION OF INFARCTION RELATED TO RHYTHM AND MORTALITY*

| EKG Location of Infarction | # | % | Type of Block 3° | % | 2° | % | Sinus Brady | % | Mortality Deaths | % |
|---|---|---|---|---|---|---|---|---|---|---|
| Posterior (inferior) | 21 | 75 | 12 | 57 | 5 | 24 | 4 | 19 | 8 | 38 |
| Anterior | 6 | 21 | 3 | 50 | - | - | 3 | 50 | 4 | 66 |
| Anterior & posterior | 1 | 4 | 1 | 100 | - | - | - | - | 1 | 100 |
| TOTAL | | | 17 | 60 | 5 | 18 | 7 | 25 | | |

Five patients with 2° block, 4 Wenckebach and 1 unknown.

*MORTALITY RELATED TO RHYTHM*

| | 3° | 2° | Sinus Brady |
|---|---|---|---|
| Anterior | 3 | | 1 |
| Posterior | 7 | 1 | |
| Anterior & posterior | 1 | | |
| PER CENT | 85% | 7.5% | 7.5% |

*DURATION OF PACING*

Duration of pacing until return to nsr = 6.1 days, range 2-20 days
Number requiring permanent pacemakers - 2 = 7%
Duration of pacing prior to death - 4 days, range 1 hr - 20 days

*CHF AND SHOCK*

| | Lived # | Lived % | Died # | Died % |
|---|---|---|---|---|
| CHF | 5 | 33 | 13 | 100 |
| Shock | 0 | 0 | 8 | 61 |

## PERMANENT PACEMAKERS AT PMH, 1966-69

*GENERAL*

Males 14, average age 71
Females 16, average age 68
TOTAL 30

| | # of Patients | % |
|---|---|---|
| *INDICATIONS* | | |
| Syncope | 23 | 77 |
| Low CO with bradycardia | 6 | 20 |
| Angina | 1 | 3 |
| CHF | 1 | 3 |
| *DIAGNOSIS* | | |
| ASHD | 15 | 50 |
| Postacute MI | 2 | 7 |
| Idiopathic | 9 | 30 |
| Congenital | 3 | 10 |
| Mellaril? | 1 | 3 |

*TEMPORARY PACING*

15 for an average of 11 days, range 3-28 days

| *RHYTHM* | | | Intermittently Return to NSR |
|---|---|---|---|
| Complete heart block | 22 | 73 | 5+(1)* |
| Second degree block | 4 | 13 | 3 |
| Sinus bradycardia | 2 | 7 | 2 |
| AF with block | 2 | 7 | 0 |

* - One returned to permanent nsr.

*COMPLICATIONS*

Battery failure - 6, average 22.7 mo, range 6-39 mo
Sepsis - 2
Hematoma - 3
Catheter tip dislodged - 6
Skin erosion by pacer box - 3

*DEATHS*

Sepsis - 1
Stroke - 1
MI - 1

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APPENDIX

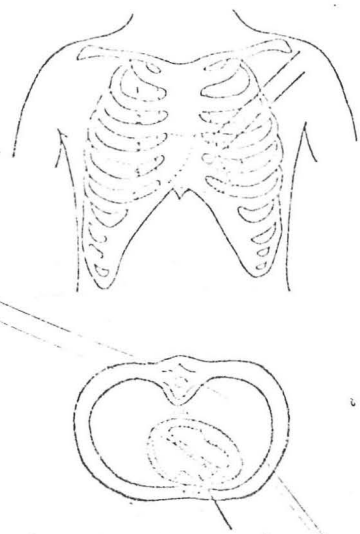


Fig. 1. Anatomical sites of insertion of percutaneous myocardial electrode for emergency pacing (12).

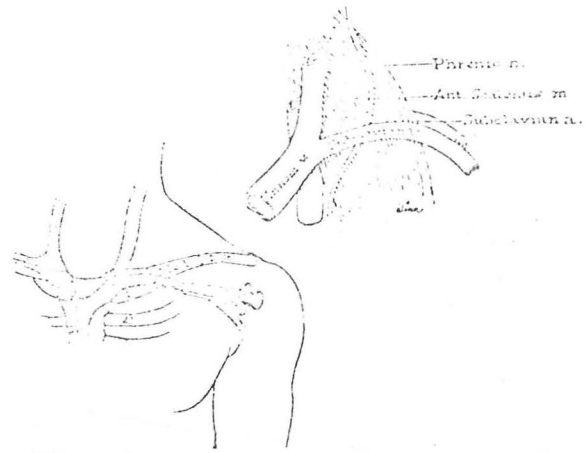


Fig. 2. Anatomical landmarks for percutaneous subclavian vein pacemaker insertion; either left or right may be used.

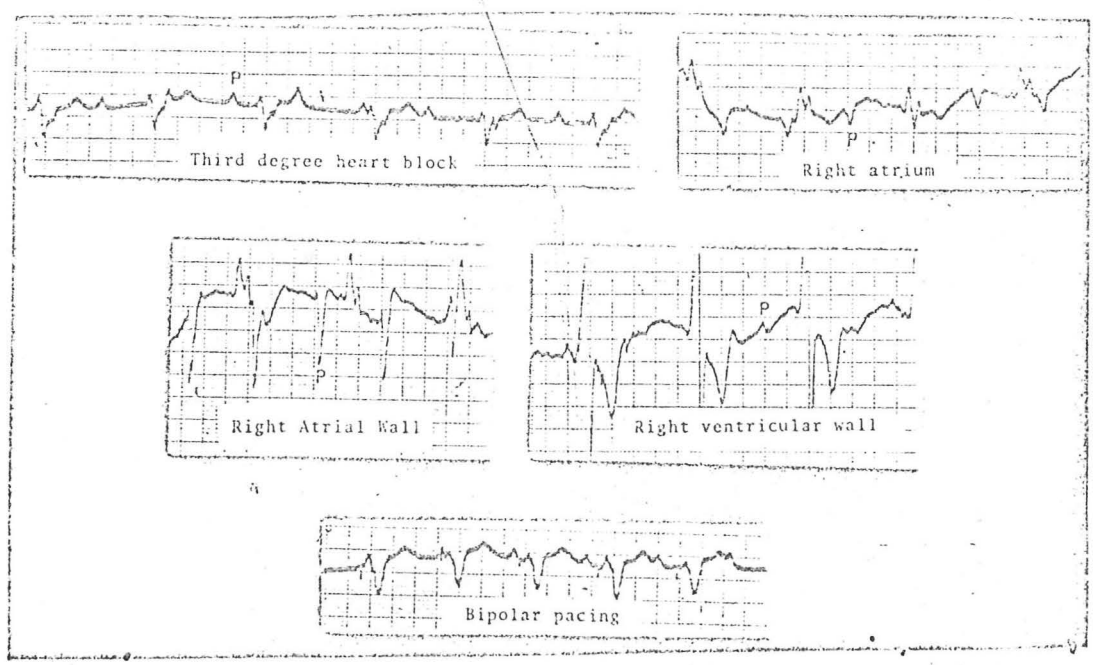



Fig. 3. Intracavitary EKG taken from the unipolar lead during blind insertion of a pacemaker.

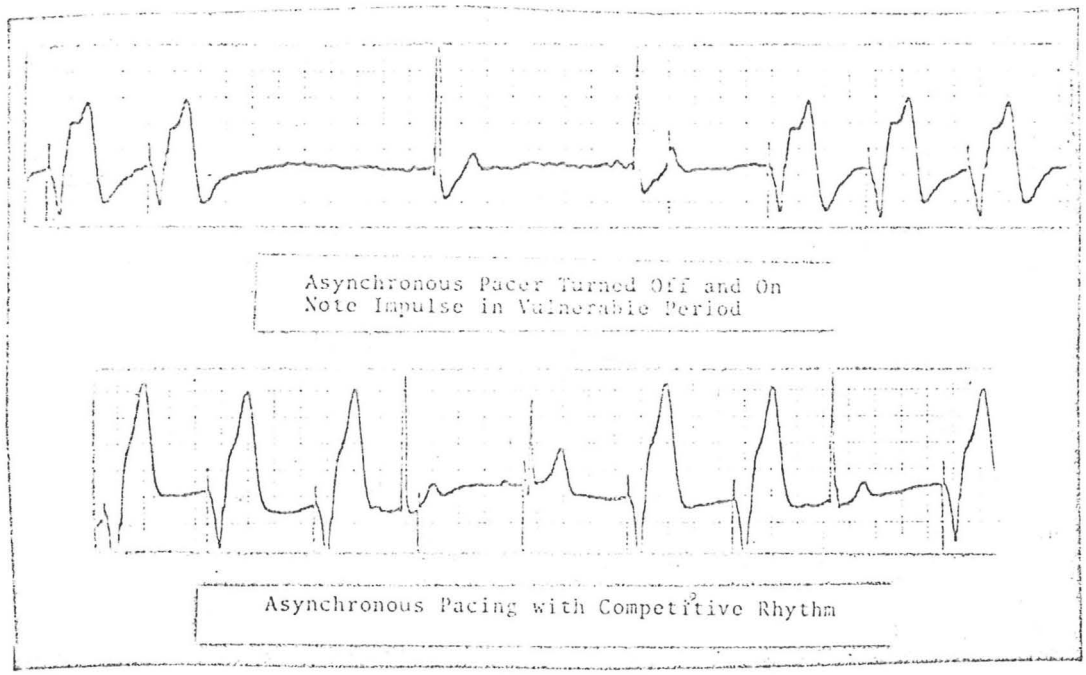
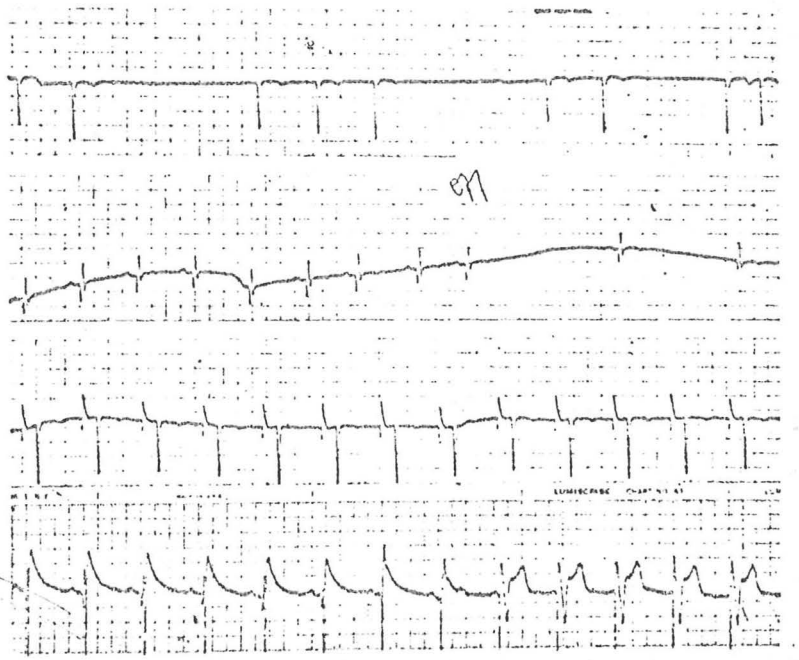



Fig. 4. Standard or fixed rate pacemaker rhythm.



The upper two tracings are of sinoatrial arrest with long periods of asystole. The third of pacing of the atrium with ventricular response. The lower tracing is of ventricular synchronous pacing.

Fig. 5

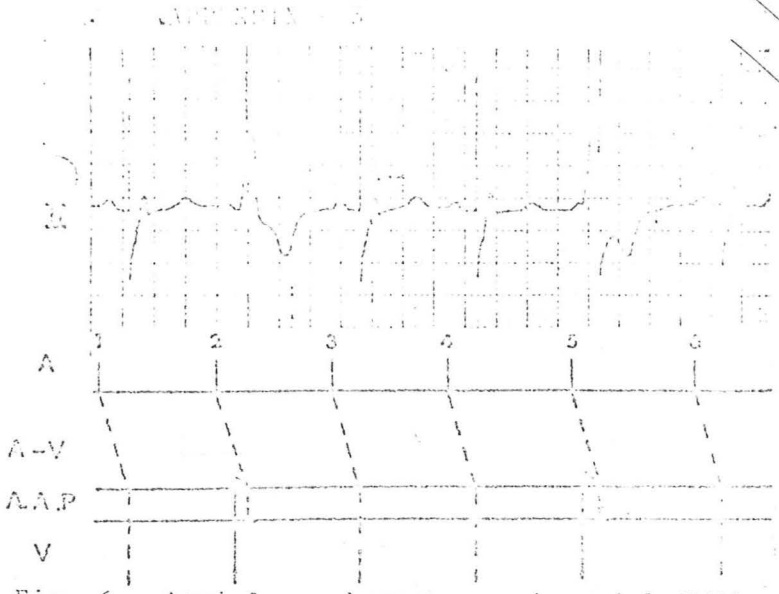



Fig. 6. Atrial synchronous pacing with PVC's.

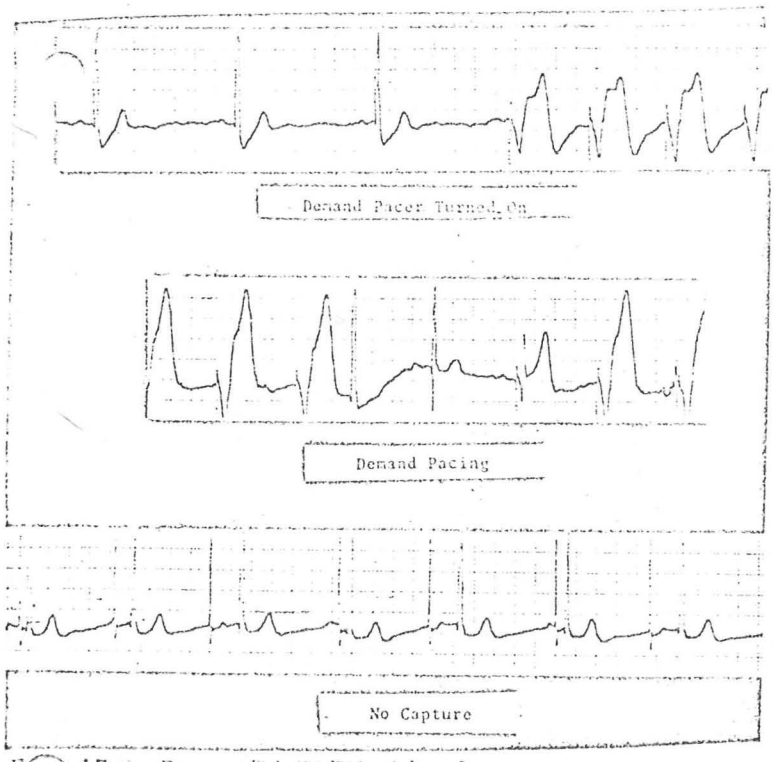



Fig. 7. Demand pacing rhythm.

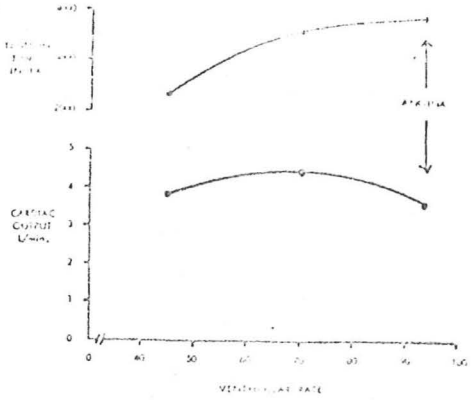


Fig. 8. Relationship of cardia output and TTI with pacing rate in acute myocardial infarction (26).

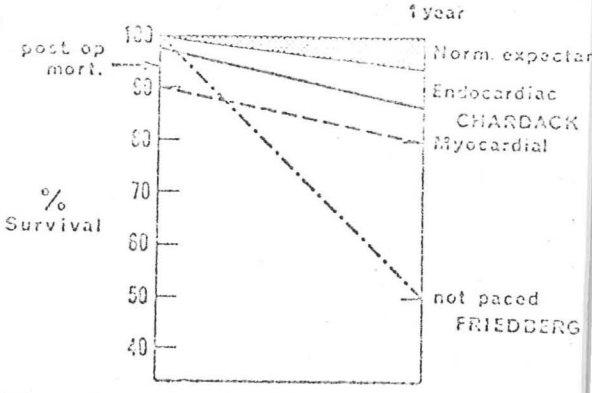



Fig. 9. Chardack's results of surv val in patients with myocardial and endocardial pacemakers with complet heart block compared with Friedberg patients (26).

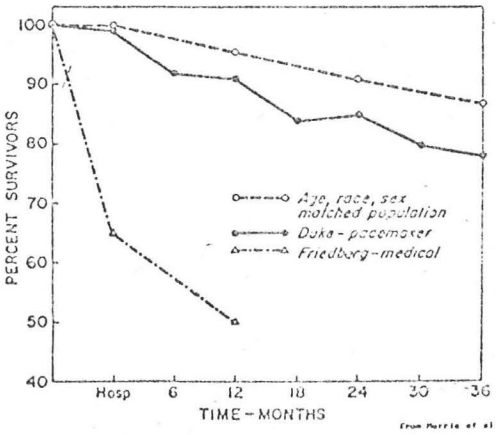



Fig. 10. Comparison of survive in patients paced with patients treated medically in complete heart block.

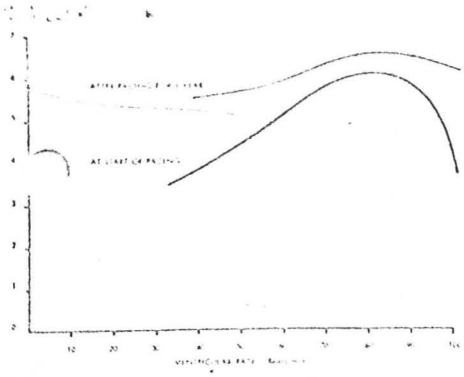


Fig. 11 (26).

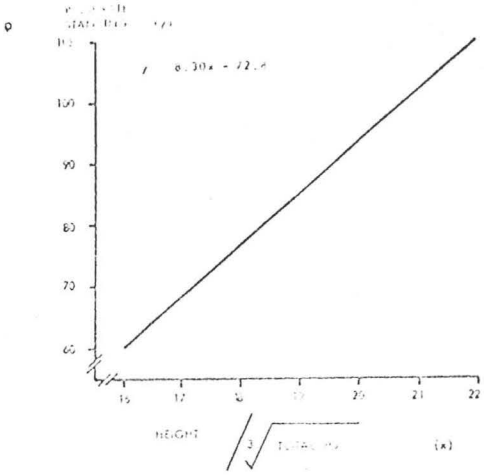



Fig. 12 (98).

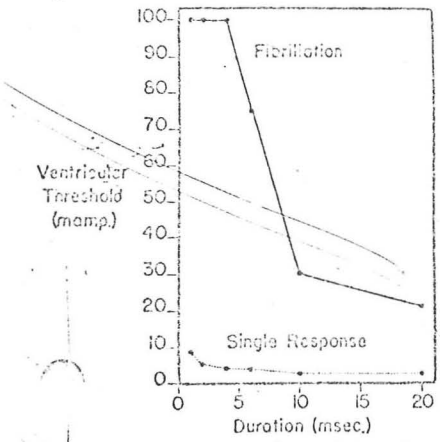



Fig. 13. Relationship between stimulation and fibrillation threshold (26).

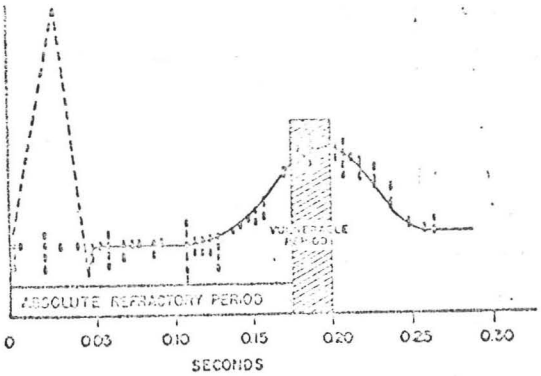



Fig. 14. EKG cycle illustrating vulnerable period when the fibrillation threshold is lowest.

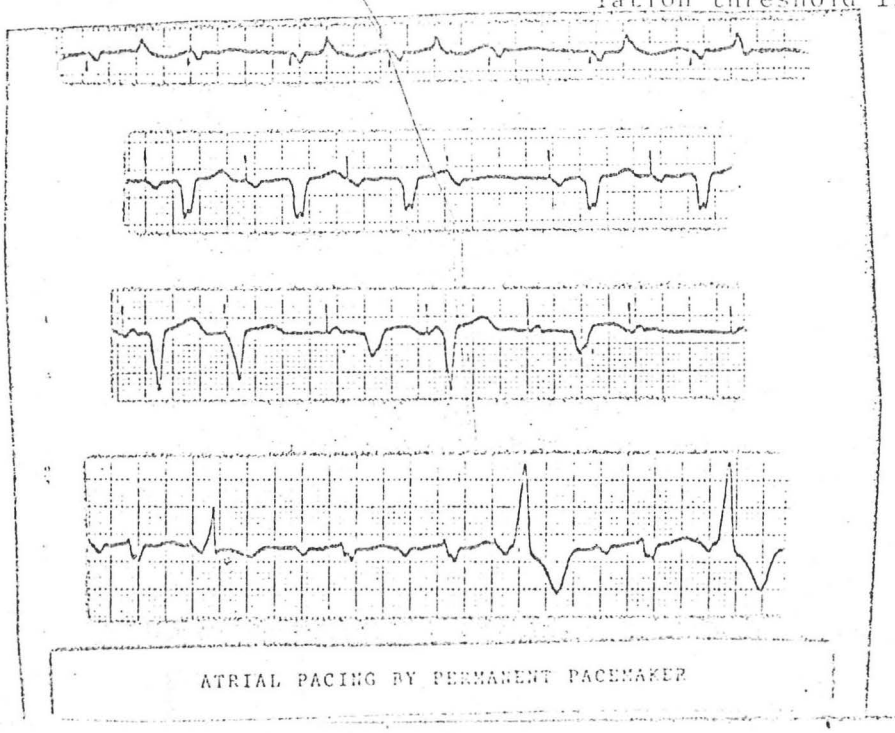
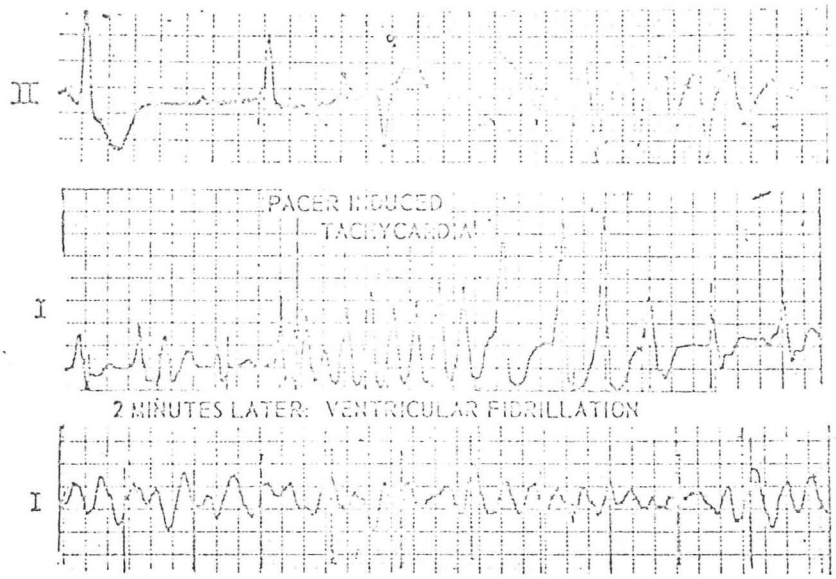
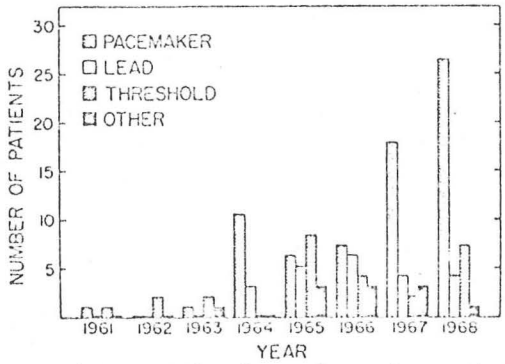



Fig. 15.




Pacer-induced tachycardia (top two tracings). Ventricular fibrillation after pacer-induced ventricular tachycardia (bottom tracing).

Fig. 16.




Graph demonstrating major causes for pacemaker reoperations. Pacemaker failure has increased as the cause for reoperation.

Fig. 17.