Artificial Pacemakers

Indications and Management

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SUMMARY
The development of artificial pacemakers for the electrical control of the cardiac rhythm has greatly enhanced the physician's ability to treat cardiac dysrhythmias. Pacemakers have been useful in treating Stokes-Adams syndrome and symptomatic bradycardias; they have helped control the occurrence of tachyarrhythmias and have played an important role in the management of arrhythmias accompanying myocardial infarctions. With their more frequent use, pacemakers have contributed to our knowledge of underlying conduction and natural pacemaker disorders. As new indications for artificial pacemaking have been elucidated, more complex pulse generators have been developed, and newer technics found for their insertion. In spite of recent developments, the pulse generators in general use have a limited useful lifetime.

This paper reviews the indications for pacemaker insertion that are commonly employed. In addition, an approach to the problem of pulse generator replacement is presented.

Additional Indexing Words:
Heart block    Bradycardia    Stokes-Adams syndrome    Myocardial infarction

ZOLL DESCRIBED an effective means of supporting patients with failure of cardiac pacemaker activity and/or conducting tissue for the first time in 1952.1 Pacing of the heart was accomplished by subcutaneous electrodes, but could be maintained for only short periods of time. In 1957 complete heart block, resulting as a complication of open-heart surgery, was successfully treated by means of electrodes attached directly to the myocardium.2 These electrodes were directed externally through the chest wall and attached to a lightweight transistorized pacemaker. Although the patient could be completely mobile, he was constantly vulnerable to infection along the electrode tract; furthermore, the electrodes were fragile and easily broken. It was clear from these early but limited experiences that electrical failure of the heart could be controlled. These early observations prompted Chardack, Gage, and Greathatch3 to develop a self-contained, totally implantable pacemaker.

A number of laboratories are engaged in the design and testing of power sources that hopefully will function longer than the 24-30-month period that is currently thought to be safe for most pulse generators. The recent development of atomic power sources seems to be the most promising for long-term function.

As the use of pacing of the heart was extended, efforts were directed toward the development of means for implanting the electrodes in the heart without a thoracotomy. This was especially desirable in the elderly patient. Furman in 1959 reported the use of catheters that could be implanted in the right ventricle by the venous route.4 Several reports attest to the reliability of this technic.5-7 Until recently fluoroscopy and skill in catheter manipulation were essential for the catheter placement even for temporary pacing. With the advent of semiflow-directed catheters, the electrodes can be easily introduced so as to accomplish effective pacing by the percutaneous route without fluoroscopic visualization.8 Whereas electrical control of the heart was formidable and highly technical procedure, it now can be initiated by physicians and paramedical personnel with limited experience. Although electrical pacing of the heart is a commonplace procedure, it is not without hazard, and a great deal of judgment is necessary to determine when pacing is to be initiated.

Types of Pacemakers
Pacemakers may be used to stimulate the atrium, the ventricle, or both in succession, thus bypassing the blocked A-V node. On occasion pacemakers may be employed to suppress ectopic foci by
“overriding,” or to interrupt a tachyarrhythmia maintained by a reentry mechanism. These functions are accomplished by one of two types of pulse generators: fixed rate of asynchronous and the noncompetitive.

Fixed-rate pacemakers discharge at a regular predetermined rate regardless of intrinsic cardiac activity. The resultant electrical output duplicates the electrocardiographic phenomena of a parasympathetic focus with entrance block. As a result, there is a potential hazard of the impulse occurring during the vulnerable period of ventricular depolarization and causing ventricular fibrillation (fig. 1). The incidence of the occurrence is unknown, but is most frequently recognized after acute myocardial infarctions when the fibrillatory threshold is at its lowest.

The risk of inducing ventricular fibrillation can be obviated by using the noncompetitive type. There are two types of noncompetitive pacemakers: demand and standby. The demand pacemaker senses any intrinsic ventricular activity of the heart and inhibits the pulse-generator discharge (R-wave inhibition). Standby or ventricular-triggered pacemakers sense each intrinsic ventricular beat, whether it is normally conducted or ectopic in origin, and discharges into the refractory period of the electrical cycle. Most such units have a refractory period after discharge of 0.4 sec during which they do not discharge. This interval is well beyond the vulnerable period.

The demand pacemaker possesses the inherent capability of sensing extrinsic electric sources thus being inhibited. Theoretically, the patient is at risk when around electrical cautery, radar sources, electric shavers, and microwave grills. The risk depends on the frequency of the wave, strength of the current, and distance of the patient from the power source. The standby pacemaker cannot be inhibited by external currents; it theoretically could be discharged up to every 0.4 sec or 150 times per min if the extrinsic current possessed the proper characteristics. Standby pacemakers discharge synchronously into the beginning of the QRS complex, whether normally or ectopically conducted, and interfere with the electrocardiographic diagnosis of myocardial infarction.

These basic types of pacemakers have been used primarily to pace the ventricles, occasionally the atrium, when there is no recognized interference of A-V conduction and the benefit of the atrial systole is desired. The noncompetitive type has been modified so as to afford an effective atrial systole even in the presence of an A-V conduction defect. A bifocal demand pacemaker senses atrial depolarization, then stimulates the ventricle approximately 0.2 sec later.

The rate of discharge may be fixed at the factory; it may be fixed but have the capacity to be altered in the laboratory by a small screwdriverlike instrument; or it may have the capability of having the rate changed between two levels, for example 60 and 90 beats/min, by an external magnet.

There is some theoretic advantage to being able to adjust the rate. Occasionally a patient will remain in cardiac decompensation at a rate of 60 beats/min. This may be improved if the rate is increased to 76 beats/min. Finally, the patient with a fixed rate, for example, 60 beats/min, may develop shock if he bleeds or has an elevated temperature and is unable to compensate by an increase in heart rate.

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Figure 1
Asynchronous ventricular pacemaker illustrating the consequences of stimulus discharge into the vulnerable period. The second pacemaker stimulus fires during the T wave producing a short run of ventricular tachycardia. In the second panel, the third pacing stimulus produces a longer run of ventricular tachycardia. (Reproduced by permission.)

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Long reliability of a pulse generator is a major consideration in selecting a specific type for a given patient. As a general rule, the more versatile the generator, the more components required; the more components required, the more likely the batteries will be drained prematurely.

One last consideration of the pulse generator is the size. This is of little consequence when the generator is placed subcutaneously in the abdomen. However, it can be a problem in thin, somewhat cachectic persons when placed on the infraclavicular fossa. It can, unless well fixed to and by the fasiae, descend down into the atrophic breast. It should be remembered that a pulse generator placed in the infraclavicular fossa may interfere with the swing of the golf club and thus remove one last remaining pleasure for the elderly.

Electrodes

With the development of reliable pulse generators, the prime limiting factor to successful cardiac pacing has been the wire electrode. Wires that have low output resistance but high resistance to electrolysis from body fluids, fatigue, stress corrosion, and stress fatigue have been developed. Certain metals or alloys of metal are better conductors than others, but may prove more vulnerable to breakage. The type of pole for which an electrode is to be used also determines its resistance to electrolysis and physical stresses. Elgiloy alloy and platinum iridium appear to be the best substances for endurance although both possess rather high electric impedance.16

Unipolar Systems and Bipolar Systems

Theoretically there is little difference between the unipolar and bipolar systems.17 Bipolar electrodes in general are larger than unipolar since two wires are necessary and have a greater electric impedance. Unipolar systems, however, are more sensitive to external electric sources and therefore not to be recommended for demand systems. On the other hand, if a wire breaks, the bipolar system can be easily converted to a unipolar system. Due to the size of the electric field produced by the unipolar system, one can usually identify the type of electrode system from the routine electrocardiogram. The pacemaker stimulus with the unipolar system is often eight to 10 times the size of the bipolar stimulus.

Techines for Electrode Insertion

Temporary Pacing

The most rapid, yet least desirable method to insert pacing electrodes, is the transthoracic approach. Direct puncture of the chest wall and apex of the heart is done with a long needle through which a bipolar electrode is threaded when the ventricular cavity has been entered. This approach is used in extreme emergencies and is followed by transvenous approaches. Pacing has also been accomplished by electrodes positioned in the esophagus opposite the left ventricle.

More conventional pacing is accomplished by inserting pacing electrodes transvenously via the median cephalic, saphenous, subclavian, and external jugular veins. The electrodes are advanced ideally to the apex of the right ventricle or to the outflow tract. In the past it was necessary to employ fluoroscopic control for electrode positioning, but newer catheters that are more pliable and partially flow-directed can be positioned with the aid of electrocardiographic monitoring from the electrode tip.8

Permanent Pacing

The direct application of electrodes to the myocardium has been the most certain method for electrode placement. Traditionally this has required general anesthesia and thoracotomy. Technics have recently been described for the limited thoracotomy and may even be done under local anesthesia, thus affording the opportunity of utilizing this approach in the elderly and more infirm patient.18

The transvenous approach is very reliable when done properly.19, 20 The cephalic vein or the subclavicular approach to the external jugular are desired routes for insertion. Once the electrode tip is in position, it is imperative to avoid acute angles of the catheter and provide a gentle sweep so that points of extramechanical stress are not created. This approach is employed with the aid of local anesthesia and is well tolerated by the patient.

Other approaches have been tried for the insertion of electrodes including mediastinoscopy for positioning atrial electrodes to pace the heart synchronously.21 In addition, subxyphoid routes have been attempted. These latter two approaches have not been employed as extensively as the transvenous and transthoracic.

Indications for Pacing

Bradyarrhythmias

As the heart slows below 50 or 60 beats/min, the cardiac output may decrease. Compensation for the slowing of the rate is not always accomplished by an increase in stroke output. Depending on the...
heart rate and the needs of the body for blood, the oxygen-carrying capacity of the blood and the state of the vascular bed, in relation to the minute output of the heart, symptoms may occur. These symptoms include primarily those of heart failure, mental confusion, or angina pectoris.

Bradyarrhythmias may result from a slow sinus mechanism, sinus arrest, atrial fibrillation or flutter with a slow ventricular response, A-V junctional rhythm, and second- (usually Mobitz Type II) or third-degree A-V block. The type and cause of the slow rhythm must be determined.

Sinus bradycardia may occur normally in well-trained athletes or in the elderly. It may accompany myxedema, obstructive jaundice, malnutrition, or may occur during convalescence from certain infections. It may result from excessive vagal tone due to stimulation of the vagal nerve anywhere along its course. Eye surgery, meningitis, intracranial tumors, cervical and mediastinal tumors may be accompanied by sinus bradycardia. It frequently occurs in patients with diaphragmatic or posterior myocardial infarctions.

Certain types of bradycardia may be due to a number of drugs including digitalis, quinidine, or beta blockers. In such cases withdrawal of the medication will usually result in an increase in heart rate and an improvement in cardiac function. If the patient has significant impairment of the circulation as a result of the slow rate, pacing should be considered. If the patient is on a bradytonic agent, then temporary pacing is all that is indicated. If sufficient time has elapsed since the drug was discontinued to establish that the bradycardia is likely to persist, then permanent pacing should be instituted. With the increase in rate, one can expect an improvement in the circulation. Follow-up studies, however, have demonstrated that the increase in cardiac output is not dramatic. Electroencephalographic abnormalities are often reversed indicating an increase in cerebral blood flow. The cerebral effects are not, however, always demonstrated immediately and may take several weeks to months before there is complete resolution of abnormalities.

Since many patients with supraventricular bradyarrhythmias also have disruption of normal conduction through the A-V node when pacing is indicated, the electrodes should usually be placed on the ventricle. Patients with sinus bradycardia or sinus arrest may be successfully paced from the atrium although this decision must be made only after atrial pacing at higher rates has been performed.

Often marked A-V block is demonstrated with the atrial pacing. Atrial fibrillation is a definite contraindication to atrial pacing.

If atrial pacing is to be permanent, we have preferred to suture the electrodes directly to the atria. Transvenous and mediastinal approaches have been attempted by others. Since the arrhythmia is often intermittent, we prefer the demand type of pulse generator.

Established complete heart block can either be congenital or acquired. Congenital heart block is frequently associated with other congenital defects in the heart; however, we are particularly interested in the otherwise normal group. In such cases, the QRS complex is of normal or only slightly prolonged duration. The rate tends to be faster than in acquiring heart block, and the rate may increase slightly in response to exercise or atropine. Such patients rarely are symptomatic. In one series of 61 patients, only three eventually developed the Stokes-Adams syndrome. Symptoms are more common when the duration of QRS complex is prolonged. As block is lower in the conduction system, the intrinsic rate of the functioning pacemaker is slower. Such patients should be considered to have a more serious prognosis, and permanent pacing may be indicated.

**Stokes-Adams Syndrome**

Stokes-Adams syndrome is defined as the occurrence of cerebral symptoms, usually syncope, in patients as a result of a rhythm disturbance. During the attack the electrocardiogram may reveal either ventricular standstill, ventricular fibrillation, ventricular tachycardia, or the slowing of the idioventricular impulse below a critical rate. Marked sinus bradycardia or atrial fibrillation with high degree of A-V block may also be implicated. Lepeschkin and Rosenbaum described several clues that may be seen in the QRS morphology which suggest that interference in normal conduction has occurred. Lenegre has pointed out that the majority of patients with complete heart block that he studied did not indeed have disease of the A-V node. He found that the interruption of conduction occurred primarily within the fascicles of the bundle branches. In his experience he found the occurrence of ischemic heart disease to be an uncommon etiologic factor in the development of chronic heart block.

The conducting system distal to the common bundle consists of three fascicles: the right bundle, the left anterior branch of the left bundle, and the
Acute Myocardial Infarction

Although it was appreciated that the development of the Stokes-Adams syndrome in the presence of myocardial infarction was associated with a high mortality rate, it was not until continuous monitoring was developed that the seriousness of heart block without syncope was appreciated.30 Despite widespread use, it is not clear whether pacemakers in all or in selected patients with heart block have resulted in a reduction in the mortality.

Bradyarrhythmias with heart block in the patient with myocardial infarction have also been reported to be between 45 and 60, it is most frequently less than 50.40 As the cycle length (R-R interval) becomes longer, diastolic depolarization is likely to occur and thus lower the fibrillatory threshold which is already apparently lowered in myocardial infarctions.41 In addition, the slow heart rate has important hemodynamic consequences. Because of the decrease in compliance associated with the infarction, there is less of an increase in stroke volume to compensate for the slow rate, and thus the cardiac output falls.42

The incidence of complete heart block has been reported to occur in between 5 and 7% of patients suffering a myocardial infarction.38, 43 Second-degree heart block occurs a bit more frequently. The mortality rate of advanced heart block (second and third degree) has been recorded as about 40%.43 It has been anticipated that treatment of this

![Figure 2](image-url)

A portion of a continuously tape-recorded electrocardiogram (Holter monitor) from a 72-year-old woman with a 1-year history of syncopal episodes. Previous 12-lead electrocardiograms were normal and she was suspected of having cerebral vascular insufficiency. The recording demonstrates sinus arrest with a marked period of asystole before an escape beat occurs. Her episodes were controlled with ventricular pacing.
complication would dramatically lower the mortality rates for myocardial infarctions, but the evidence is still not available. Nonetheless, the control of heart rate by pacemakers still plays an important role in the management of patients admitted to CCU’s. Many of the discrepancies with comparative results have occurred because of the different courses in patients with inferior and anterior infarctions.43–46

Complete heart block is more common in inferior myocardial infarctions than in anterior infarctions. Inferior myocardial infarctions are caused in the majority of cases by occlusion of the right coronary artery. In 90% of human hearts, the artery to the A-V node arises from the right coronary artery.47 With progressive occlusion of the right coronary artery, collaterals developed from the left coronary artery to the distal right. With the occurrence of complete occlusion, there is frequently ischemia without infarction of the A-V node. The blood supply to the A-V node is frequently maintained, if only marginally, by the left coronary system.

Heart block in patients with an inferior myocardial infarction usually occurs during the first day of the infarct, rarely later than the fourth day. In such patients, complete heart block rarely occurs suddenly; it is usually preceded by the progression of first-degree block to various stages of second-degree block. It has been observed, however, to occasionally develop more rapidly after the administration of morphine.48 Rarely is the complete block permanent.

The ventricular complex in complete heart block complicating inferior myocardial infarction is usually of short duration; the rate is above 50 beats/min.49 Even if the duration of the QRS complex is prolonged, the prognosis does not appear worsened. In the absence of heart failure or hypotension, the presence of heart block does not appear to increase the mortality.43

Patients with anterior infarctions have a higher mortality rate than those with an inferior infarction. Anterior infarctions are secondary to occlusion of the left anterior descending coronary artery.43 Since the left anterior coronary artery does not normally supply blood to the A-V node, heart block is usually not due to ischemia of conducting tissue. When heart block occurs in the presence of anterior infarction, the area of infarction is usually large; the heart block results from destruction of the peripheral branches of the conducting system. The right bundle which traverses the intraventricular septum is particularly prone to disruption. Heart block in this situation usually develops suddenly, without progression from first-degree block and lesser degrees of second-degree block.48 The development of a right bundle-branch block with left-axis deviation is a premonitory sign of probable heart block.50 The QRS complex may be broad and bizarre; the conduction defect is characterized by instability and a tendency to sudden arrest.

From the studies made on the natural history of heartblock occurring in myocardial infarction, it is clear that not all patients developing first- or second-degree atroventricular block will progress to complete heart block.44 Likewise, there is ample clinical evidence to indicate that pacemakers may help to avoid hemodynamic embarrassment as well as sudden cardiac arrest if complete heart block does occur.42 Furthermore, based on the above considerations, it is obvious that pacemaker management varies for patients with inferior and anterior myocardial infarctions.

There is little debate regarding patients presenting in complete heart block in the presence of anterior myocardial infarctions, or patients developing Stokes-Adams attacks in either type of infarction. These patients require immediate institution of ventricular pacing.51–53 Patients with other bradyarrhythmias, likewise, who have a low cardiac output or are rapidly deteriorating, should be paced.51

Although some consider the use of pacemakers in the presence of first-degree heart block, this appears to be warranted only in patients with anterior infarctions. Because first-degree block usually progresses to Wenckebach in inferior myocardial infarctions, these patients should be treated with atropine to reverse the conduction defect. Especially if sinus bradycardia is present, atropine or isoproterenol is indicated. These drugs are frequently successful in reversing the block, but careful monitoring must be maintained since an elevation of the atrial rate may lead to higher degrees of block.54 If ventricular irritability is not present or there are no hemodynamic alterations, pacemaker insertion is not indicated.51 Once second-degree block is present, either as Wenckebach or high degrees of block, then it is imperative to try to reverse the block. If drug therapy fails, ventricular pacing should be performed. In the past this decision was modified because of the necessity of specialized radiographic technics. Currently in our coronary care unit, the insertion of flexible pacing...
wires via the left subclavian vein with a preformed bipolar electrode has been very successful and can be done with little discomfort to the patient. Once the pacing has been initiated, the rate should be maintained in the range of 70–80 beats/min. A demand-type pacemaker should be utilized so that recognition of the patient’s return to normal conduction can be easily determined. The demand function also lessens the risk of ventricular fibrillation due to the impulse being fired into the vulnerable period of ventricular repolarization.53 The pacemaker should be left in place for at least 3 days after normal conduction returns, although longer periods have been recommended to avoid late cardiac arrest.54 At least in inferior myocardial infarction, the likelihood of late cardiac arrest is rare, and the insertion of a pacemaker for long periods of time may unnecessarily restrict the patient during the rehabilitation period.48 The results of ventricular pacing in inferior myocardial infarction are not conclusive,43 but in one study appear to favorably influence the outcome.52

Anterior myocardial infarctions present a far more difficult decision, since, as Friedberg has pointed out, the mortality rate is apparently little altered by pacing.43 However, when one is dealing with an individual patient, the statistics are hard to utilize and the clinician is hard pressed to watch helplessly without instituting possible beneficial therapy. First-degree block in anterior infarctions is an indication for pacing since complete block may occur suddenly. The development of right bundle-branch block and left-axis deviation is probably the most frequent premonitory sign. In survivors, pacing is usually necessary for only a short time, although there appears to be little chance of reversion to normal rhythm if complete heart block is present on admission.55

Prevention and Treatment of Ventricular and Atrial Tachyarrhythmias

Among the specialized uses of pacemakers have been the prevention of dangerous arrhythmias and the termination of others. This overdrive method has been utilized principally in the treatment of acute myocardial infarction, but is predicated on well-known physiologic concepts that were noted long before their clinical application. Ventricular premature beats have been shown to have an inhibitory effect on the sinus node and to alter its rhythmicity.56 This effect is similar to that of asystole noted to occur when pacemakers are suddenly stopped. There usually is an escape beat from a lower ectopic focus before the sinus node takes over pacemaker activity. The faster the heart rate, the shorter is the refractory period with consequent elevation of vulnerable period threshold. This is particularly effective in preventing reentrant arrhythmias and ventricular fibrillation.

The use of overdrive is especially useful to suppress ventricular tachycardia and fibrillation in the presence of myocardial infarctions without heart block.57 If drug therapy fails to suppress the ectopic activity, pacing to increase the ventricular rate 15–20 beats above the intrinsic heart rate is often effective. Pharmacologic depressants may be used with pacing and may become effective with the more rapid heart rate.58 Ventricular rates per minute may be accompanied by a decrease in cardiac output, and therefore should not be maintained for long periods of time.42

When overdrive is utilized in myocardial infarction, the duration is usually for relatively short periods of time, rarely exceeding 48 hours. Once the necessity for overdrive has ceased, the pacemaker should be removed since its presence in the right ventricle can stimulate ectopic beats.

The right ventricle has been the site most commonly used for pacing when suppression of a ventricular arrhythmia is desired. Zipes et al. reported a case that was successfully controlled only by atrial pacing at a rate of 100 beats/min.50 Atrial pacing has also been employed to control certain atrial arrhythmias. There have been several reports of converting atrial flutter to normal sinus rhythm by rapid atrial pacing.29, 60 Junctional tachycardias have also been converted to normal rhythm, and pacing may be the preferred treatment especially if digitalis intoxication is suspected as the etiology. Certain tachyarrhythmias associated with the Wolff-Parkinson-White syndrome respond to atrial pacing.

The development of atrioventricular block with atrial pacing at rapid rates is a complication that must be considered. Davidson et al.,61 however, utilized this phenomenon to control the ventricular rate in a patient with refractory supraventricular tachycardia. Their patient maintained a resting ventricular rate below 100 and was able to decrease the block with exercise to achieve a near-normal cardiac response.

The main complication of atrial pacing is the conversion of the rhythm to atrial fibrillation when the atrium is stimulated.82 Since most atrial arrhythmias respond well to conventional pharmacologic therapy, pacemaker insertion is still a secondary therapeutic approach.
Postoperative Management of Open-Heart Surgery

The use of pacemakers has contributed greatly to the management of patients who have recently undergone open-heart surgery. Since many procedures are done with ischemic cardiac arrest, it is often impossible to determine whether or not damage or interruption of the conducting system has occurred. We therefore recommend the insertion of an epicardial wire for all patients undergoing either aortic valve surgery or repair of congenital intracardiac defects. Certain patients who have had the mitral valve replaced, especially if they are slow in developing spontaneous cardiac activity, should have pacemaker electrodes inserted before the chest is closed. As has been pointed out, in tricuspid valve replacement, it is imperative to suture electrodes to the heart since there is obvious difficulty in initiating transvenous pacing. Although the major consideration for pacemaker use has been the prevention of heart block and cardiac arrest, the electrodes are very useful in management of tachyarrhythmias occurring postoperatively.

Miscellaneous Uses of Pacemakers

Evaluation of Coronary Artery Disease

Atrial pacing is particularly advantageous where other forms of well-controlled stress testing are not available, but probably offers no significant advantage over the latter in deciding upon the significance of lesions. For a research tool, especially in the study of myocardial metabolism, it has an obvious use. A limitation to the use of atrial pacing to produce angina is the development of atrioventricular block. If block develops before angina occurs, then the test is of little clinical significance. Treadmills or bicycle ergometers often can produce much higher rates, and therefore are more apt to induce angina.

Arrhythmia Interpretation

Frequently, the standard electrocardiogram is abnormal, but the mechanism of the arrhythmia is obscured because of the inability to identify P waves. Attaching a pacemaker to the precordial lead of a well-grounded electrocardiogram and recording within the right atrial cavity enhances accurate detection of the P wave.

Atrial pacing has proven to be an important adjunct in understanding the genesis of arrhythmias. When other means of provoking or demonstrating heart block have failed, paced atrial rates may demonstrate the development of various forms of second-degree block. The combination of atrial pacing and the His bundle electrogram are also useful in the presence of sinus node disease. Determining the presence of block in the common bundle or lower is important when consideration for a permanent atrial pacemaker is being made. Demonstration of any form of block dictates the use of ventricular pacing.

Pulse Generator Replacement

Once permanent pacemaking has been instituted, the physician is faced with a long-term responsibility to his patient. Any malfunction of the pacemaking system or deterioration of the pulse generator has the potential for serious consequences. Although the reliability of the output from the pulse generator is stated by the manufacturer, the confidence limits are not absolute and therefore careful observation of the patient's clinical status is essential.

Because of the complexities of pacemaking, it would appear that regional clinics would afford the best care for patients. However, since there are only a few such clinics available, most patients are followed by their personal physicians. Routine replacement of pulse generators after a certain period of time is often recommended, and in many instances when access to medical facilities capable of inserting generators is limited, this represents a sound conservative approach. Unfortunately, the patient still needs close surveillance. Analysis of the pacemaker impulse by an oscilloscope and interval counter is the most sophisticated method for evaluating pulse generators but is not a readily available method.

Patients should be examined at regular intervals once the pacemaker is inserted. The intervals may vary, but should become shorter as the generator life approaches the manufacturer's predicted lifetime. Accurate counting of the impulse rate should be done by a well-serviced electrocardiogram. The asynchronous pacemaker stimulus interval can easily be measured. With demand or standby units, carotid massage should be performed if the intrinsic rate exceeds the predicted pacemaker rate. Certain units can be temporarily converted to asynchronous units by placing a strong magnet over the pulse generator, thus affording the opportunity to measure the output rate. Most units today manifest battery deterioration by decreasing the rate of stimulus discharge. Older pacemakers had the potential to "run away," i.e. increase their rate significantly.
In addition to rate, variation in the size of the stimulus artifact should be looked for since this often represents a variable generator output. Cordis pacemakers may lose their ability to synchronize, and thus revert to a fixed rate as the pacemaker fails. Demand pacemakers likewise will lose their ability to "sense."

Periodically X-rays of the chest and pulse generator should be performed. Any displacement or breakage in the electrodes should be sought. It has been suggested that the status of the batteries might be determined by X-rays of the generator.21 This has not been helpful in our hands.

At present the most useful criterion for pulse generator replacement is a change in discharge rate, especially a decrease.22 A 0.01-sec decrease in discharge is probably significant, although the cardiac function at that time may not be impaired. In order to detect such changes, the paper drive of the electrocardiographic machine must have a uniform and properly adjusted rate. Experience, however, has shown that once a decrease in rate begins, the rate of generator failures begins to increase rapidly. We recommend replacement of the pulse generator once a significant rate change has been detected. Others have noted that variation in impulse amplitude has not been as reliable a sign. We also feel that if the pacemaker is not functioning properly in the demand or standby mode that the pulse generator should be replaced. The failure of the component responsible for the demand capabilities may place an added drain on the batteries.

The long-term management of patients in whom pacemakers have been implanted becomes progressively more complex. Because of the greater mobility exercised by society in general, patients frequently are far from their personal physician or medical center when problems develop. In addition, modifications are frequently made in the various pacemaker components which are peculiar to that lot. A national registry of pacemakers seems desirable to provide necessary information to any physician about the pacemaker status of the patient being attended. Data concerning lot numbers, discharge rates, and indications for pacemaker insertion could be readily available from the central registry. A program for follow-up visits could be easily developed, and obviate the need for the patient to return to the original center. Until such programs are established, each physician must diligently outline a follow-up program for his patient which assures him of having a functioning pacemaker. No matter how many patients with pacemakers the physician has under his care, the task is formidable.

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