CARDIAC PACING
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Presented by the American College of Cardiology and the Medical College of Virginia.

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Introduction

This issue of the MEDICAL COLLEGE OF VIRGINIA QUARTERLY is an edited form of the proceedings of the Cardiac Pacing symposium held in Williamsburg, Virginia on April 16 and 17, 1971. The meeting was co-sponsored by the American College of Cardiology and the Department of Continuing Education, Medical College of Virginia, Health Sciences Division, Virginia Commonwealth University. The program emphasized the clinical aspects of cardiac pacing, and the format was structured with formal presentations followed by panel discussions. Manuscripts were not requested from the speakers, and with the exception of Dr. Philip Samet who prefers that the reader review the publications listed on p. 173 rather than rely on the transcriptions of his talks, "Hemodynamics and Electrical Studies in Pacing" and "Indications for Temporary Pacing," bibliographic references are not included. The success of this meeting which approximately three hundred attended must be attributed to the outstanding guest faculty whose contributions in the past are largely responsible for the presently employed techniques. Dr. Onkar Narula was an unscheduled speaker who kindly consented to present his experience with His bundle recordings during the session on temporary pacing. The interest aroused by the program reflects the realization by physicians of the growing impact of engineering in clinical medicine.

I would like to express my appreciation to Mr. William Nelligan and Miss Mary Anne McInerny and other members of the staff of the American College of Cardiology who assisted in the organization of this meeting. I am grateful for the support provided by Dr. M. Pinson Neal, Jr, and Miss Erma Blanchard of the Department of Continuing Education as well as Mr. Melvin Shaffer and his staff of the Audio-Visual Department. I would like to acknowledge the work required in the preparation of this manuscript by my secretary, Mrs. Barbara Hendrick, and by the editorial staff of the MEDICAL COLLEGE OF VIRGINIA QUARTERLY, particularly Mrs. Ronnie Madoff and Miss Mary Parke Johnson.

CHARLES L. BAIRD, JR., M.D.
I would like to discuss the development and historical aspects of cardiac pacemaking from a personal point of view, presented as it appeared to me over the years. This approach is biased, selective, and incomplete, but I think as informative as I can make it.

It has long been known, indeed from the early days of Galvani, that electrical currents have effects on the heart. The knowledge that a cardiac contraction may result from an electrical stimulus led to no clinically useful result until relatively recently. Other instances are common perhaps even at the present time in medicine in which basic information is available for advances in therapy but is not being used.

There was not much change in this field until about 1900, when a great deal of research was conducted on the effects of electrical currents on the heart. It was demonstrated in physiological and pharmacological laboratories then that electrical stimuli produced action currents that were conducted along nerve or muscle cells, and produced contractions in skeletal, smooth, and cardiac muscle. Among the important contributors were Adrian, Evans, and John Erlanger. In 1900, Prevost and Batelli demonstrated that electrical currents, both direct-current and alternating-current discharges, would terminate ventricular fibrillation. The shocks were applied directly to the heart, but it took a very long time again before this information was used effectively. In 1933, Hooker, Kouwenhoven, and Langworthy made extensive and valuable laboratory studies on the effects of alternating current in terminating ventricular fibrillation, again the current was applied directly to the exposed heart. This work was extended in 1936 by Ferris, King, Spence, and Williams, who demonstrated that capacitor discharges also terminated ventricular fibrillation. They used sheep, relatively large animals, with hearts similar to man's in size, but again information lay dormant without human, clinical application. In 1946, some Russian workers, Gurvich and Yuniev, recorded very briefly in the English literature that capacitor discharges would terminate ventricular fibrillation. About 1940, Wiggers and Wegrzyna conducted extensive experimental studies outlining the various features of electric shock that were useful in terminating ventricular fibrillation. Wiggers demonstrated that an initial alternating-current shock might produce ventricular fibrillation in a normally beating heart and a second shock of the same nature but perhaps a little stronger might terminate the fibrillation. Wiggers applied the term “countershock” to this procedure, which I like to use and keep alive despite current usage of other terms because of its historical significance. I think we should give him credit for providing us with the sound experimental basis for the technique of defibrillation which has saved so many lives. The first clinically successful termination of ventricular fibrillation in man was done in 1947 by Beck, Fritchard, and Feil, who resuscitated a patient from ventricular fibrillation by performing emergency thoracotomy, direct cardiac “massage,” and application of alternating current to the exposed heart. In this very brief review of direct ventricular defibrillation, I have passed over some instances in 1902 and 1910 of defibrillation that were unsuccessful. In 1947, Claude Beck and his group in Cleveland had established that opening the chest and applying a-c countershock would terminate ventricular fibrillation, this procedure formed the basis for the treatment of cardiac arrest that was widely taught and vigorously practiced in those days. In the presence of cardiac arrest, under any circumstances one was supposed to cut the chest open, “massage,” i.e. squeeze the heart rhythmically to provide an effective output, and if necessary to defibrillate the heart by direct application of alternating-current countershocks.

In 1932, Dr Albert S. Hyman in New York City addressed himself to the parallel problem of ventricular standstill. He attempted to develop an effective means of stimulating the heart in ventricular standstill so as to terminate cardiac arrest. He applied a stimulus from an electric pacemaker to the atrium by passing a long needle or electrode through the chest wall into the atrial musculature. He demonstrated effective atrial stimulation in the rabbit by this technique, but it was never applied successfully in man as far as I know. A major difficulty lay in the maintenance of good contact of the needle electrode in the cardiac.
muscle without displacement or injury. Another problem was in the application of the stimulus to the atrium, in clinical cardiac arrest atrioventricular block is often present so that atrial beats are not conducted and do not arouse ventricular contractions.

In their extensive studies in 1940, Wiggers and Wégbria examined the effects of electric stimuli of varying intensity and duration at varying intervals after a preceding beat, and clearly delineated the various phases of cardiac excitability. They demonstrated and applied the term “vulnerable phase” to the interval in the relative refractory period when a strong (well above threshold for a single response) or long stimulus produces multiple or repetitive beats and even fibrillation. The stimuli used in these studies were 10 milliseconds long, whereas present-day pacemakers provide stimuli usually less than 2 milliseconds long. This explains why they were able to demonstrate repetitive responses and the vulnerable period so readily.

I became interested in electric stimulation of the heart shortly after World War II, after I had observed much of the pioneering cardiac surgery done by Dr. Dwight Harken for the removal of foreign bodies in and about the heart. The heart appeared, indeed, to be a very sensitive organ that responded readily with ventricular contractions to stimuli, arousal from ventricular standstill by appropriate stimulation should, therefore, not be difficult. The other point with which I was impressed is that I should have learned in first-year anatomy—the esophagus lies behind the heart in very close contact with it. Thoracotomy and direct electrical stimulation of the heart seemed to be an inappropriate and excessively traumatic approach to the problem of effective cardiac stimulation that was not much better than the universally accepted program of thoracotomy, cardiac massage, and direct countershock defibrillation. It seemed to me that a stimulus might arouse a ventricular contraction in a patient with cardiac arrest if it were applied in the esophagus close to the heart by way of a long wire electrode passed down the mouth.

I thought about this matter for some time but did not do much about it, I am afraid, for a long while. My background in electricity was inadequate, so I did not know how to build a pacemaker that would provide an appropriate stimulus. In 1950, William C. Callaghan, a Canadian cardiac surgeon, spoke at a Boston Surgical Society meeting and described an approach of passing a wire electrode “catheter” down the jugular vein in a dog, and stimulating the area of the sino-atrial node to provide an effective rhythm controlled by an external pacemaker. This method has not been used clinically but it is very interesting and it contains elements of many of our present-day techniques, especially the idea of applying an electric stimulus by way of a percutaneous endocardial “catheter” wire electrode. He made a great effort to apply it to the sino-atrial node, not an appropriate site for ordinary clinical purposes in patients with A-V block, but one that is being reconsidered for special purposes now.

I found that he used a standard physiologic pacemaker made by the Grass Instrument Company in Quincy, Mass., and within the next year I managed to borrow one from Dr. Otto Kreyer, head of the Pharmacology department of the Harvard Medical School. With a long wire electrode in the esophagus and a second electrode over the precordium in a dog, I was quickly able to demonstrate that electrical stimuli would indeed arouse atrial or ventricular electrical responses and effective muscular contractions with which cardiac arrest could be terminated and the circulation maintained. An extensive laboratory study was undertaken to develop the details of this new technique of external electric stimulation of the heart, to improve and simplify the procedure and the apparatus, to define its clinical applicability and limitations, and to expose any associated hazards. It was soon found that rather large currents were necessary for effective stimulation (30 to 150 volts or 50 to 200 milliamperes) unless the negative electrode actually touched the heart, and that two surface electrodes on each side of the precordium were as effective as an esophageal-precordial pair. Short stimuli, 2-3 milliseconds in duration, were selected when they were found to be almost as effective as longer ones in stimulating single responses and never produced repetitive responses, tachycardia, or fibrillation except in hearts seriously damaged by ischemia, anoxia, or overdoses of digitalis or quinidine.

In 1952, we applied the method of external electric stimulation for the first time in man, and it quickly became established as an effective emergency means of arousing the heart from ventricular standstill. Although most frequently used in patients with Stokes-Adams disease, it was also effective in standstill of any origin, even in the absence of A-V block, so long as cardiac contractility was not overly depressed by prolonged anoxia or drugs. Although external stimulation was occasionally used in desperate circumstances for hours or days to maintain an artificial rhythm, it proved too painful for ordinary long-term use. For the purposes of arousing, accelerating, and maintaining intrinsic ventricular rhythms in patients with high-degree A-V block, Dr. Arthur J. Linenthal and I developed the method of intravenous administration of dilute solutions of sympathomimetic amines, particularly epinephrine and isoproterenol. Combined use of the two methods often enabled us to keep patients alive through intervals of severely unstable rhythm and frequent Stokes-Adams attacks.

We also found at that time that provision of a rapid regular rhythm by external electric stimulation or by acceleration of an intrinsic pacemaker pharmacologically would often stop recurrent ventricular tachycardia and fibrillation. This demonstration lies behind
the concept of overdriving the heart to suppress multiple competing rhythmic foci, which is an important aspect of present-day control of cardiac rhythm with electric pacemakers.

The method of external electric cardiac stimulation is based on the idea of applying a large electric current to the surface of the chest so that a small portion of it may reach the heart and stimulate a response, just as a small electric stimulus applied directly to the exposed heart was known to do. The success of this technique suggested modification of the usual countershock defibrillation after thoracotomy by application of a large countershock externally across the surface of the chest. In 1954, contemporaneously with William Kouwenhoven at Johns Hopkins and Arthur C. Guyton at the University of Mississippi, we developed a technique for external electric countershock defibrillation with large 60-cycle alternating-current shocks in dogs and domestic pigs. The next year we applied it successfully for the first time to resuscitate a patient from ventricular fibrillation. We also demonstrated in the laboratory that external a-c countershock would terminate every type of rapid arrhythmia. atrial tachycardia, atrial fibrillation, ventricular tachycardia, and ventricular fibrillation. In 1955, we terminated several desperate attacks of ventricular tachycardia and in 1957 one of atrial tachycardia in man with external a-c countershock. In 1961, Bernard Lown at the Peter Bent Brigham Hospital greatly modified this approach by applying external direct-current shocks (obtained by capacitor discharge through an inductance) that were synchronized to fall immediately after the R wave so as to avoid the vulnerable phase of the cardiac cycle. He used this method, which he termed "cardioversion," for the elective termination of lesser arrhythmias with the thought that synchronization would prevent the production of ventricular fibrillation. Although some disagreement with this view persists (from myself and other workers as well), the technique of synchronized d-c shock is most generally used today. The initial enthusiasm for cardioversion of minor arrhythmias has subsided considerably because of the frequent early recurrence of the arrhythmia with little clinical benefit.

The development of these two methods of external electric stimulation and of external electric countershock led to great changes in the management of cardiac arrest. With effective emergency means of arousal of the heart from standstill and of defibrillation, the need for thoracotomy, programs of action became simpler, less traumatic, and more successful. The unpredictability of attacks and the limited time available for resuscitation pointed to the need for prompt recognition of the onset of arrest and identification of the arrhythmia. From our early experiences with patients with Stokes-Adams disease, the development of cardiac monitors in 1954 that provide an audible signal of each beat, continuous visual display of the electrocardiogram, an alarm signal of appropriate changes in rate, and prompt, even automatic availability of an external electric pacemaker. In 1960, William Kouwenhoven developed the technique of "external cardiac massage," or external cardiac compression, by which circulation of blood may be restored immediately in the emergency of cardiac arrest and, if necessary, the time interval for more definitive management of arrhythmia may be extended.

In 1963, Hughes Day combined all these techniques and applied them to patients with acute myocardial infarction, the largest group with serious arrhythmias. He placed patients with this condition under continuous monitoring in a coronary care unit where monitors, pacemakers, and defibrillators were concentrated together with personnel expert in their use and trained in a program for management of cardiac arrest. His demonstration in the pioneer unit at Bethany, Kansas, of a major reduction in mortality from acute myocardial infarction led to the widespread acceptance of the concept of the coronary care unit.

It seems unfortunate to me, however, that the idea of continuous cardiac monitoring has not been applied widely to surgical operating rooms where unexpected cardiac arrest still occurs with significant frequency and mortality. In 1963, Drs. Morris Nicholson and Joseph Crehan, anesthesiologists at the New England Deaconess Hospital in Boston, monitored a number of very sick patients throughout anesthesia. In this group they demonstrated a striking improvement in successful resuscitation from cardiac arrest. Despite this convincing experience, continuous cardiac monitoring with automatic alarm has not become a standard part of the management of all patients undergoing anesthesia, as I believe it should.

The final subject of discussion is the development of internal cardiac pacemakers for the long-term provision of a reliable rhythm at any rate desired. The need for a method of prolonged cardiac stimulation was obvious early in our experience with patients with Stokes-Adams disease, when we were able to resuscitate patients from attacks of standstill or fibrillation but were unable to prevent recurrent episodes. Direct cardiac pacing with small, imperceptible electric stimuli by means of electrodes implanted in the myocardium had long been known and used experimentally, but for years long-term pacing in this way was frustrated by a progressive rise in threshold for stimulation. In 1958, Thevenet, Hodges, and Lillehei in Minneapolis attempted to stimulate the heart directly in patients with Stokes-Adams disease with a stainless-steel electrode placed in the myocardium at thoracotomy that was connected to an externally carried pulse generator. Stimulation failed uniformly within 7 weeks, however, because of increasing thres-
HISTORICAL DEVELOPMENT

hold for cardiac response. We finally recognized the problem as a foreign-body tissue reaction to minute, sterile contaminants on the electrode surface and solved it by using inert metals for the active electrode (platinum, gold, or stainless steel) and by boiling it in Ivory soap flakes. Dr Samuel A. Hunter in St. Paul developed the Hunter-Roth electrode and with it was the first in 1959 to achieve long-term pacing in a patient with Stokes-Adams disease. In 1960, Drs. William Chardack, Adrian Kantrowitz, and Howard Frank and I began series of implantations of pacemaker-electrode systems by way of thoracotomy, and adequate long-term management of A-V block and Stokes-Adams disease was finally accomplished.

Many problems arose and many changes were made in long-term cardiac pacing to meet them. Displacement of electrodes, wire breakage, and component failure in the pulse generator have in large measure been corrected, but early battery depletion remains the major difficulty at present. An important modification, which has become widely accepted, has been in the placement of a pervenous endocardial “catheter” electrode under fluoroscopic control rather than by thoracotomy. This approach was first introduced by Drs. Seymour Furman and John Schwede at the Montefiore Hospital in New York City with the electrode passed through an antecubital vein and connected to an externally carried pulse generator. Subsequently, other veins were used and the entire pacemaker system was placed subcutaneously. Although the procedure is relatively minor, it is not entirely satisfactory in that electrode placement is sometimes very difficult and even unsuccessful, and electrode displacement, myocardial perforation, high threshold for stimulation, and wire breakage occur with small but significant frequency. In this regard, we have recently developed an “arrowhead” electrode that may be inserted rather quickly and securely through a small thoracotomy but under direct vision. This technique is an attempt to gain the advantages of both approaches—secure placement of the electrode in the myocardium but with a relatively small, well-tolerated procedure.

In recent years, many types of pacemakers have been developed to avoid competition of an independent fixed-rate pulse generator with intrinsic beats, which do occur often from ectopic ventricular foci or from return of A-V conduction. It appears that competition offers little risk clinically of repetitive response and ventricular fibrillation, unless an electrode is inserted in an area of acute myocardial ischemia or infarction. Nevertheless, variable-rate (“synchronous,” “demand,” or “stand-by”) pacemakers may at times be advantageous in producing less palpitation and better cardiac output. Atrial-triggered, ventricular-triggered and inhibited, and atrial-ventricular sequential pulse generators of many varieties have been developed, and some are now being widely used.

Presently, both temporary and long-term pacing are also being applied with considerable enthusiasm in a variety of clinical situations and to a wide variety of difficult arrhythmias. Consequently, the area of applicability of cardiac pacing is growing. Advances in technology and engineering also promise major improvements in new generations of pacemakers so that we can anticipate exciting developments in the whole field of electrical control of cardiac rhythm.
Pacemaker Concepts and Terminology*  
BAROUH V. BERKOVITS, E.E., Ing.
Associate in Surgery, Harvard Medical School, Boston, Mass., Associate in Electrophysiology, Miami University, Miami, Fla., Senior Research Scientist, Cedars-Sinai Medical Research Institute, Los Angeles, Calif., Consultant in Surgery, Peter Bent Brigham Hospital, Boston, Mass., Cardiovascular Research Manager, American Optical Corporation, Framingham, Mass.

Since the introduction of implantable cardiac pacing systems there have been marked advances in concept and design of the equipment available. Whereas intracardiac pacing is usually identified with A-V conduction disturbances, recently gained knowledge of cardiac physiology has shown that electrical stimulation of the heart can be beneficial in the treatment of many arrhythmias. It is important that the physician familiarize himself with the different modalities of pacing. The terminology used to describe the different concepts must be clear and precise so the physician can select the proper pacemaker concept for his patient without ambiguity.

Pacemakers can be divided into two categories: parasystolic and nonparasystolic. While parasystolic-stimulation is independent of intrinsic activity; nonparasystolic-stimulation is controlled by the intrinsic activity. The nonparasystolic pacemakers can be either inhibited, in which intrinsic signals suppress stimulation, or triggered, in which intrinsic signals induce stimulation. According to the site of stimulation the pacemakers can be atrial, ventricular, or A-V sequential. Pacemaker types in present use are:

1. Parasystolic (Continuous)
   a. Fixed or set rate atrial pacer
   b. Fixed or set rate ventricular pacer
   c. Fixed or set rate A-V sequential pacer

2. Triggered (Synchronous)
   a. P-wave triggered ventricular pacer
   b. QRS triggered ventricular pacer

3. Inhibited (Demand)
   a. P-wave inhibited atrial pacer
   b. QRS inhibited ventricular pacer
   c. QRS inhibited A-V sequential (Bifocal)

The parasystolic pacemakers (continuous fixed rate) may be used to stimulate the atria, ventricles, or stimulate both the atria and ventricles with a preset sequential delay. In the presence of natural beats the modality of stimulation is not affected and it competes with the intrinsic activity. The stimuli falling in the absolute refractory period will induce no response, but those falling in the vulnerable interval may induce repetitive response or even fibrillation.

Figure 1 illustrates the differences between the various modalities of nonparasystolic pacing.

Characteristics of P-wave triggered ventricular pacing are:
1. The P waves trigger the pacemaker, which in turn stimulates the ventricles synchronously after a preset delay of 120 milliseconds.
2. This pacemaker usually has a refractory period of 500 milliseconds (measured from the beginning of the P wave) which prevents the pacemaker from following atrial tachycardia or fibrillation. (3) When natural P waves do not appear for a preset interval (1.04 sec.) the P-wave triggered pacemaker escapes. This escape mechanism protects against asystole when there are no P waves present. Thus, the pacemaker works either in synchronous or escape modality.

Characteristics of QRS triggered ventricular pacing are:
1. The QRS complexes trigger the pacemaker, which in turn stimulates the ventricles during its absolute refractory period. In the latest models, this synchronized stimulation is delivered immediately after the detection of the ventricular endocardial signal.
2. The pacemaker has a refractory period of 400 to 500 milliseconds. This built-in refractory period is designed to protect the unit from running at fast rates. A long refractory time may prevent the recognition of premature beats and thus causes escape stimulation competing with these premature beats. Competition with premature beats is even more hazardous than with the normal beats.
3. When natural QRS complexes do not appear for a preset interval (840 milliseconds), pacemaker escapes occur to protect against asystole.

Contrary to the triggered pacemakers, the demand pacemakers work only in escape mode.

1. In the presence of a faster natural rhythm, the demand pacemaker is dormant and no stimuli are delivered to the heart.
2. Stimuli are delivered only if the natural beat fails to occur for a preselected escape interval. Consequently, the heart is stimulated only when needed (on demand), and competitive rhythms or stimulation during the vulnerable phase are thus avoided.

Figure 2 shows how the ventricular demand or...

* Presented at the American College of Cardiology and the Medical College of Virginia Cardiac Pacing Symposium, April 16 and 17, 1971, Williamsburg, Virginia.
Fig 1—Differences between the various modalities of non- parasystolic pacing.

Fig 2—Actual workings of the ventricular demand or inhibited pacemaker.

Fig 3—Schematic illustration of the different modalities of pacing.
Fig 4—Basic construction of the Bifocal demand pacemaker compared to the conventional demand unit.

Fig 5—The Bifocal demand pacemaker facilitating the natural depolarization sequence without competing with spontaneous ventricular activity
PACEMAKER CONCEPTS AND TERMINOLOGY

BIFOCAL DEMAND PACEMAKER
WORKING IN ATRIAL MODE, OCCASIONAL PVC'S & COMPENSATORY PAUSE IN ATRIA

Fig 6—Bifocal pacemaker placed in a patient with sinus bradycardia and premature ventricular contraction.

BIFOCAL DEMAND PACEMAKER
ON-MAGNET-OFF
TESTING RESPONSE OF VENTRICULAR STIMULATION

Fig 8—Testing of a pacemaker with a magnet; unit is converted to a fixed-rate mode.

inhibited pacemaker actually works, imitating the natural escape rhythm of an automatic fiber. The upper part of the tracing demonstrates how the automatic fiber works and how the threshold potential is reached during phase four depolarization so that these fibers can fire by themselves and produce escape beats. The bottom portion of the figure demonstrates how the pacemaker is controlled by a capacitor that restores its charge with each detected beat. In short, each time a depolarization signal is detected the pacemaker is reset and the timing cycle is started again. When the capacitor’s charge reaches the critical level because of the prolonged interval, it permits escape and a stimulus is delivered to the ventricle. The pacemaker actually delivers its stimulation in the same fashion as an automatic fiber.

Figure 3 demonstrates schematically the different modalities of pacing. As shown, Bifocal pacing is similar to A-V sequential pacing, except that it is on demand and it is inhibited and reset by endocardial ventricular depolarizations. The Bifocal demand pacemaker adapts its modality of stimulation to the patient’s need. It combines the advantages of atrial, A-V sequential, and demand stimulation. It may remain dormant, it may stimulate only the atria, or it may stimulate both the atria and the ventricles with a preset sequential A-V interval. The Bifocal demand pacemaker does not compete with the spontaneous ventricular activity and it has no significant refractory time.

Figure 4 illustrates the basic construction of the Bifocal demand pacemaker and compares it to the conventional demand unit. In conventional demand pacing the ventricular signal detected by a QRS detector will control the timing circuit of the ventricular demand stimulator. A magnetic switch is incorporated in these pacers for evaluation of pacemaker function by preventing inhibition and thus converting the unit to a fixed-rate mode. When testing the pacemaker, the rate produced by the magnetic switch is independent of the patient’s physiological condition and should be used to determine the condition of the batteries. It is important that during each outpatient visit the demand pacemaker be checked by applying a magnet and this rate recorded for control. Conceptually, the Bifocal pacemaker is comparable to the conventional demand pacemaker except that atrial stimulation controlled by the same QRS detector has been added.

Two functions of the Bifocal pacemaker are demonstrated in Figs. 5, 6, and 7. Figure 6 shows a Bifocal pacemaker in a patient with sinus bradycardia and premature ventricular contractions. It can be observed that the atrial pacemaker compensates for the premature ventricular beats. Figure 7 shows a Bifocal pacemaker in a patient with first-degree A-V block. The sequential interval of the pacemaker was
similar to the patient's own conducted A-V interval. Therefore, the different degrees of fusion and changes in morphology can be observed.

Figure 8 depicts the testing of a Bifocal pacemaker with a magnet, similar to the ventricular demand pacemaker. The measurement of the interval during a magnet-induced fixed-rate mode should be recorded in order to follow the pacemaker function and determine battery condition. A change greater than 10 percent indicates battery failure.

During the past 18 months we have implanted 60 Bifocal pacers with encouraging results. In the early stages, the basic indication for implantation of Bifocal pacers was to improve cardiac output. Recently, Bifocal pacemakers have also been used for patients with sick sinus syndrome—atrial brady-tachyrythmias. In a number of patients, we have found that 2 to 3 months after implantation, drugs could be progressively discontinued, and that the atrial stimulation not only protected against the bradycardia but also suppressed the episodes of tachycardia.

Pacing therapy has undergone marked evolution during its relatively brief existence. Further developments in concept, clinical applicability and technical areas are forthcoming. The physician has the obligation to understand the various pacing modalities and to select the most suitable concept of pacing for each particular disorder (Table I).

Author's note. "BIFOCAL" is a trademark of the American Optical Corporation for the QRS inhibited A-V sequential pacemaker

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**TABLE I**

THREE DIFFERENT MODALITIES OF A-V PACING

<table>
<thead>
<tr>
<th>P-wave Triggered</th>
<th>A-V Sequential (Continuous)</th>
<th>Bifocal Demand</th>
</tr>
</thead>
<tbody>
<tr>
<td>For Normal Atrial Activity With A-V Block</td>
<td>For Atrial Bradycardia With A-V Block</td>
<td>For Atrial Bradycardia With or Without A-V Block</td>
</tr>
<tr>
<td>Monitors P-waves</td>
<td>No monitoring</td>
<td>Monitors QRS Complexes</td>
</tr>
<tr>
<td>P-waves Control Ventricular Stimulation No Atrial Stimulation Available</td>
<td>Continuous Atrial and Ventricular Stimulation</td>
<td>QRS Complexes Control Both Atrial and Ventricular Stimulation</td>
</tr>
<tr>
<td>Stimulation is Delivered to the Ventricles Continuously</td>
<td>Stimulation is Delivered Both to the Atria and Ventricles Continuously</td>
<td>Stimulation May Be: a Totally Absent b Delivered only to the Atria c Delivered both to the Atria and Ventricles</td>
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Electrocardiographic Recognition of the Various Pacemaker Types and Dysfunction*

LEONARD S. DREIFUS, M.D.

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This discussion deals with electrocardiographic interpretations of the various types of pacemakers as well as with their dysfunction which may be due to pulse generator failure, wire breakage, change in threshold, or polarization of the ventricle. Hemodynamic problems may also arise concerning placement of the pacemaker leads particularly in those patients who lack atrial contribution.

Figure 1 demonstrates competition resulting from a fixed-rate pacemaker. Here are four different types of beats showing the various responses that may occur. It is important to point out that if a T wave is not observed after a pacemaker spike then there is no propagation of the impulse through the ventricle. I prefer not to use this type of pacing because of competition and the possibility of sudden death, but as yet we do not have any cases that verify such a situation. Figure 2 demonstrates the most serious form of competition in which stimulation of the T wave had resulted in ventricular fibrillation. I have observed a number of cases in which the spike did occur on the T wave but repetitive rhythms such as ventricular tachycardia did not occur. However, with those patients who die suddenly one can only surmise that T-wave stimulation and ventricular fibrillation could have been related. I have no proof, but I am worried about this problem as is everyone else. I believe that the sicker the heart the more vulnerable it is and the greater the chance of these complications.

One of the major concerns I have with fixed-rate pacing is the problem of premature ventricular beats other than those associated with ventricular fibrillation. Figure 3, strip b, shows the development of premature ventricular contractions after each paced beat which result in a form of ventricular tachycardia that is hazardous.

Noncompetitive pacemakers such as the R-wave ventricular-triggered pacemaker (Fig. 3, strip c) have a built-in refractory period, but as will be seen in the last portion of the strip, they do not sense all beats. Figure 3, strip a, is an example of coupled rhythm with an R-wave triggered pacemaker that does not produce the form of ventricular tachycardia associated with the fixed-rate unit.

The atrial-triggered pacemakers have given us a host of headaches. There are not many of these around anymore, but when the bifocal type becomes more widely utilized we may run into similar difficulties again. This patient (Fig. 4) developed atrial flutter with an atrial-triggered pacemaker. Digitalization will not work under these circumstances, and there is not much you can do except shock the patient (strip d). I attempted to use quinidine, but the rate went up to 140.

Figure 5 shows a premature ventricular contraction which occurred during use of an atrial-triggered pacemaker. There is no refractory period built into the unit, therefore, the ectopic beat is not sensed. This could create a dangerous situation, and at least the bifocal pacemaker, as Mr. Berkovits pointed out, does not have this problem. In addition to runaway pacemakers, atrial-triggered units include such pitfalls as this instance of a patient in complete A-V block and intermittent function (Fig. 6). Here the "demand," or escape feature of the pacemaker was working, but the atrial threshold increased tremendously, and the atrial electrode was not sensing except in the super-normal period. You can occasionally see atrial-triggered beats coming through and evidence of bigeminy which subsequently developed for some reason. There is nothing wrong with this pacemaker unit. It was just that the atrial circuit was working only intermittently because of build-up of the threshold at the atrial lead. One of the problems with atrial epicardial leads is that after a period of time they may cease to function, and you are again faced with fixed-rate pacing in a patient with high-degree A-V block.

Rate hysteresis is another concern in using demand pacemakers. In order to permit spontaneous sinus activity, the escape rate is set at a slower driving frequency of 60 beats per minute while the driving rate is set at 70 beats per minute. This allows the heart to be paced in the presence of asystole. Following a
Fig 1—Competition resulting from a fixed-rate pacemaker. A, B, C, and D represent four types of beats, showing the various responses that may occur.

Fig 2—The most serious form of competition resulting from a fixed-rate pacemaker. Strip 7 shows stimulation of the T wave which results in ventricular fibrillation (strip 8).

Fig 3—(Strip a) coupled rhythm with an R-wave triggered pacemaker; (strip b) development of premature ventricular contractions after each paced beat with a fixed-rate pacemaker; (strip c) an R-wave ventricular-triggered pacemaker that is not sensing all beats.

Fig 4—Development of atrial flutter with an atrial-triggered pacemaker. Strips a and b represent an EKG recording of atrial flutter with an atrial-triggered pacemaker. Strip c, documents the presence of flutter with a right atrial electrogram. (Strip d) EKG recording after applied shock.
period of sinus rhythm the pacemaker will not return its pacing cycle for at least one second in order to permit slower sinus rates to emerge and maintain optimum cardiac output.

Other problems with demand pacemakers are sometimes encountered. If the tip of the electrode catheter is too close to the atria or the electrodes of the epicardial leads are too close to atrial tissue, P-wave sensing may occur and cause a double reset of the pacemaker. Also, large T waves occasionally can be confused with QRS complexes, and these too can reset pacemakers and produce long diastolic intervals. Sometimes the electrogram does not quite reach two millivolts, and incomplete sensing may occur. If the pacing capacitors are not fully discharged they may recycle at an earlier time and thus, the pacing cycle is shorter than the expected ordinary driving cycle. But in most instances the pacemaker corporations have attempted to correct these problems.

In coronary sinus pacing, the pacemaker may be located above or below the A-V junction. As Dr. Samet pointed out, you have varying delays of propagation to the ventricle from His bundle recordings. However, the coronary sinus is an unreliable place from which to pace. I recently lost a patient completely dependent upon coronary sinus pacing when the catheter flipped out.

The runaway pacemaker (Fig. 7) demonstrates a situation which should no longer occur. For early detection of this problem so that pulse generators can be replaced, it is entirely possible that ten-hour electrocardiographic taping may be of value.

I would now like to deal with some of the interesting aspects of cardiac pacemaking that relate to electrophysiology. In the past we did not know what the supernormal period of the human heart was. We discovered it in the laboratory when we stimulated the heart at half threshold. The subsequent development of pulse generator failure shown in Fig. 8 reveals pacemaker spikes not followed by QRS complexes. However, the supernormal phase is identified at the end of the T wave because of the propagated response. Thus, a defunct pacemaker with less than threshold stimulus remaining is able to propagate an impulse during the supernormal period.

Another example of the supernormal period during pacemaker failure is incomplete A-V dissociation in the presence of 2:1 conduction block (Fig. 9). Why do we get sudden conduction? And sudden conduction occurring only after a paced spike? This is one of the varieties of the so-called supernormal period of conduction. In some way this pacemaker spike is influencing subsequent transmission of facilitation of the next P wave, and this offers an interesting insight into electrocardiographic analysis. The major problem in this situation is to determine whether the failure is due to a defunct battery, broken wire, threshold increase, or
ELECTROCARDIOGRAPHIC RECOGNITION

Fig 8—Development of pulse generator failure. Pacemaker spikes are not followed by QRS complexes, but the supernormal phase is identified at the end of the T wave because of the propagated response.

Fig 9—Electrocardiographic recording of the supernormal period during pacemaker failure in an instance of incomplete A-V dissociation in the presence of 2:1 conduction block.

to actual myocardial contractions.

However, to avoid pacemaker failure, we now rely on an early detection approach of rate counting and recording the arterial pulse. Pulse information can be recorded transtelephonically. Thus, if wire breakage should occur you would see spike, pulse, spike, pulse, spike and no pulse. This means that there was no contraction of the ventricle and no peripheral pulse. The rate is recorded by an interval counter, and if there is a change of 15-20 milliseconds in an R-R interval of 830 milliseconds, you can detect it and recommend a pulse generator change. It is interesting to note that 12 percent of our pacemakers are failing before the indicated battery life span of 18 months. This is a very serious problem, and it is our hope that with the present technique of interval counting we will be able to extend the life of the pulse generator to 23 months.

PANEL DISCUSSION

Dr. Richardson: Regarding the use of pacing to prevent ventricular tachycardia. Does this work by eliminating the VPC's which precipitate ventricular tachycardia or is there another mechanism?

Dr. Dreifus: When you have VPC's falling on the T wave this can set up repetitive phenomena, and it takes only one beat falling at the apex of T wave to produce ventricular fibrillation. Now VPC's may be due to reentry or to increased automaticity. By speeding up the ventricular rate you prevent asynchronous diastolic depolarization and conduction abnormality. When pacing assumes control of the heart it depresses other pacemakers. Therefore, depression of the ectopic pacemaker by the electric one will usually prevent rhythm of development from occurring so that diastolic depolarization does not have a chance to take hold. So there are two basic principles when you use overdrive: you cut down the tendency for inhomogeneous depolarization and reentry, and at the same time you keep the pacemaker depressed.

Questioner: Are you implying parasystolic focus is also depolarized, practically speaking?

Dr. Dreifus: Parasystole implies that already there is undirectional block. The undirectional block is built into the pacemaker. It is true that you may not be able to depolarize the parasystolic pacemaker, but then the chances of its emerging to the rest of the heart may then be inhibited. The most perfect example of parasystole is the electrical parasystole. Obviously it can't get into a fixed-rate pacemaker and so it is going to emerge anyhow, and that is a problem. Fortunately, most ventricular tachycardias with acute infarction are not parasystolic.
Indications for Cardiac Pacing*

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The indications for implantation of a long-term cardiac pacemaker consist of two major categories—the prevention of Stokes-Adams attacks and an inadequate cardiac output caused by a slow ventricular rate.

Stokes-Adams attacks are defined as episodes of cerebral ischemia due to cardiac arrest in patients who have atrioventricular block of any degree, not only during the episode of arrest but at least intermittently at other times as well. One Stokes-Adams episode presents an unquestionable, urgent indication for pacemaker implantation. Questions may arise, however, about the extension of this indication to patients who have high-degree A-V block but have not had an attack. After all, Stokes-Adams attacks are notoriously unpredictable in frequency, severity, and mechanism, and the first attack may be a fatal one. When Stokes-Adams attacks may be considered to have a significant probability, the risk of an initial attack should perhaps be obviated by the implantation of a cardiac pacemaker. A decision of this nature is a matter of legitimate discussion at the present time.

The risks, disadvantages, and complications of the procedure must be balanced against the risk of a Stokes-Adams seizure. Many patients with high-degree A-V block never have a Stokes-Adams seizure. Furthermore, in the presence of transient or reversible factors (such as temporary anoxia, cardiac depressing drugs, anesthetic agents, and acute rheumatic fever or diphtheria) even a documented seizure may not be a sufficient indication for a pacemaker.

The indication may also be considered in patients with multifascicular block, i.e. intermittent bilateral bundle branch block, right bundle branch block plus left anterior hemiblock. These patients may go on to develop complete block and then perhaps suffer Stokes-Adams attacks. The likelihood of this development is not entirely clear, however. Although many patients with high-degree A-V block and Stokes-Adams attacks are known to have had multifascicular block earlier, it is not known how many of all the patients with multifascicular block go through this sequence.

The indication for temporary pacemaker application to prevent Stokes-Adams attacks is even more difficult to assess than for long-term use. It has been proposed in patients with acute myocardial infarction when first-degree A-V block or bundle branch block appears, and even in uncomplicated inferior wall myocardial infarction. Early enthusiasm for pacemaker application in acute myocardial infarction has subsided considerably with the demonstration that this procedure does not improve mortality statistics. The placement of a catheter electrode often constitutes a considerable, undesirable stress for the seriously ill patient with acute myocardial infarction. Furthermore, presence of the electrode in the chamber may provoke dangerous ectopic ventricular activity. Finally, electric stimulation of an ischemic area is especially hazardous since the threshold for repetitive response and ventricular fibrillation is much lower in such an area than in normal myocardium.

At times there may be great difficulty in making a diagnosis of A-V block and even of cardiac syncope in patients with intermittent seizures. A-V block may be incomplete and intermittent, so as to escape detection. Seizures that were thought to be neurological were suffered two or three times a week for 35 years by one patient in our series. His electrocardiograms repeatedly showed normal sinus rhythm with normal intraventricular conduction. Finally, a tracing was obtained from a cardiac monitor equipped with a "memory loop" of recording tape and alarm system that showed transient A-V block and ventricular standstill. His disease was at last cured by the appropriate therapy of implantation of a long-term cardiac pacemaker. His electrocardiograms now almost always show competition of his normally conducted intrinsic ventricular beats with the electrically stimulated beats of his fixed-rate pacemaker, a phenomenon that produces no untoward clinical symptoms. Rarely, A-V block is present and only the pacemaker rhythm is seen.

A final extension of this primary indication for pacemaker application is concerned with the management of cardiac arrhythmias in the absence of A-V block—so that the diagnosis of Stokes-Adams disease is technically improper. The provision of a reliable electrically paced rhythm of appropriate rate will prevent syncopal episodes due to depression of rhythmicity at any site.

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INDICATIONS FOR CARDIAC PACING

regardless of whether A-V block is involved. Furthermore, many tachycardias and even fibrillation may be suppressed by overdriving with electric pacemakers at a sufficiently rapid rate.

The second major indication for pacemaker implantation is an inadequate cardiac output due to a slow ventricular rate. The slow rate may result from a slow sinus rhythm with normal A-V conduction or from varying degrees of A-V block. The inadequate cardiac output may be manifested by congestive heart failure, by diminished tolerance for exercise and consequent limitation of activity, by the presence of angina pectoris, and by reduced renal and cerebral function, i.e. azotemia and confusion or coma. The reduction in renal and cerebral function may at times be insidious and occult, and become apparent only in retrospect after correction by a normal pacemaker rate. In some patients with slow ventricular rates, therefore, a temporary pacemaker at a fast rate should be applied on a trial basis for several days to uncover possible manifestations of inadequate cardiac output. If no significant effect of a slow rate is found, it may be a reasonable clinical judgment not to implant a cardiac pacemaker, but to tolerate a harmless bradycardia. The only regular consequence of a slow ventricular rate is the appearance within a few months of left ventricular hypertrophy.
Techniques and Modes of Insertion of Permanent Pacemakers*

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In this report, I intend to mention most of the methods of implantation of permanent pacemakers while indicating those methods which are currently favored.

The special advantages of epicardial implantation of pacemaking electrodes are the accuracy and reliability of this technique, and the insignificant subsequent rise in pacemaking threshold. However, the indications for epicardial stimulation have contracted in direct proportion to the increased ease and reliability of endocardial catheter pacing, a method which carries a lower morbidity and mortality, particularly in the aged patient.

Permanent Epicardial Pacing. The indications for pacing by direct epicardial stimulation are: (1) for temporary atrial or ventricular pacing in conjunction with cardiac surgery for surgically induced heart block, for the control of sinus or nodal bradycardia, improvement of cardiac output, stabilization of cardiac rhythm, or to overdrive and prevent or correct ventricular arrhythmias, all in conjunction with cardiac surgery. Thus in most patients with acquired heart disease, a pacing wire is implanted on the right atrium at the time of surgery; (2) for control of chronic heart block in children or young adults in whom the risk of thoracotomy is negligible. However, such patients are infrequently seen; (3) if atrial synchronous pacing is being considered and thoracotomy can be tolerated, direct implantation of an atrial electrode is used, since the pervenous techniques are not yet fully reliable; (4) when transvenous endocardial pacing has not been achieved initially or has subsequently failed, epicardial implantation is used. It is in this category that epicardial pacing finds its most common application in chronic heart block.

A variety of surgical procedures can be employed for epicardial pacing. Formerly, the standard procedure was a transpleural, left anterolateral thoracotomy, allowing access to the left ventricle and left atrium. Secondly, the left parasternal, extrapleural approach through the fifth and sixth costal cartilages, which exposes the midanterior right ventricle. Thirdly, the substernal, transdiaphragmatic approach which transgresses neither the peritoneal cavity nor the pleural space, and which exposes the posterior right ventricle. Finally, for the sake of complete description, there is the midsternotomy approach to the anterior right ventricle and right atrium.

Certain technical points deserve emphasis in regard to each of these surgical approaches: The standard anterolateral thoracotomy procedure is usually carried out through the fifth intercostal space. We prefer to position the patient in the full right lateral decubitus since the left ventricle is then more easily exposed. An excellent exposure is obtained after division of the costal cartilages.

If an atrial lead is also to be implanted, the fourth intercostal space is used. Electrode position on the myocardium too close to the phrenic nerve may lead to diaphragmatic stimulation. There is some disagreement regarding the proper position for the electrode. It has been suggested that more effectual contraction of the ventricle can be obtained from stimulation near the apex of the left ventricle. Others deny this. Close to the base of the heart there is greater likelihood of damaging intramyocardial coronary arteries. One of our patients succumbed from ventricular fibrillation on the seventh postoperative day, and at postmortem examination one of the sutures could be seen to encompass a significant intramyocardial coronary artery. As a method of determining optimal electrode position, testing to locate the position on either the left or right heart which yields the most narrow QRS has been suggested. We prefer the commonly employed coil type of electrode originally designed by Chardack, but I am very much intrigued by the electrode presented by Dr. Paul Zoll. Because of the increased susceptibility to ventricular fibrillation, a fixed-rate pacer should no longer be considered for use with epicardial leads in the early postoperative period. The threshold of capture, determined with decreasing current, is recorded for future reference. After a demand or standby

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pacer has been inserted, and if the patient is in sinus rhythm, the pacer must be converted to a fixed-rate mode by magnet to verify the integrity of the system.

Lower thresholds are obtained with electrodes which penetrate into the myocardium. A flat coil epicardial electrode, such as that used for the atrium, can also be used on the ventricle. The late stimulating threshold is somewhat higher, but perfectly acceptable. To avoid the possibility of phrenic stimulation, we have usually placed a thin layer of polyvinyl sponge between the electrode and the pericardium. A wide loop of electrode wire is led into the mediastinum and down into the upper abdomen. Every effort should be made to prevent kinking of the electrode wire or flexion stress. This concept dictates placement of the lead internal to the costal arch and through the diaphragm, if a transpleural approach has been used. If a parasternal approach is used, the lead should traverse the subcostal angle where motion is minimal. We have rarely used axillary placement since the application of the above principles in conjunction with Elgiloy (an alloy of nickel, cobalt, and chromium) wire has completely eliminated lead fracture. We have not favored implanting a generator behind the rectus fascia because of the increased difficulty at the time of subsequent battery replacement. When a unipolar system is used, the ground plate of the generator should be faced anteriorly in the subcutaneous tissue so as to avoid the possibility of muscle stimulation. The subcutaneous pocket should be large enough to prevent tension during closure, but it should not be so large as to allow rotation or turning of the generator unit with resulting stress on the leads. The pocket should not be placed in the lower abdomen, since bending at the waist will lead to increased flexion stress on the leads. The pocket is developed one-third cephalad to the transverse skin incision and two-thirds caudad to it. Absolute hemostasis is essential to minimize the potential for infection in the presence of a foreign body, and we make every effort to avoid catheters or drains. When used within two inches of the generator unit, the coagulation cautery may cause temporary malfunction of the unit. An adequate loop of wire leads should be coiled behind the generator to permit easy and safe extrusion of the unit at reoperation. Antibiotics are instilled into the pocket and used systemically for forty-eight hours postoperatively. Adherence to the above principles, together with a very careful and accurate closure of the subcutaneous tissue with nonabsorbable interrupted sutures, has yielded a record of no wound breakdowns in about 70 cases and only one infection, this beginning in the subcutaneous tissue. A temporary transvenous catheter is employed in all patients with complete heart block who must undergo general anesthesia, such as would be required for a thoracotomy procedure. This type of pacing control facilitates the epicardial implantation of the electrode since sudden discontinuance of rapid pacing achieves a prolonged asystole and quiet heart (Fig. 1). When an atrial surface electrode is employed with atrial synchronous pacing, this is sutured to the epicardium near the base of the auricular appendage.

Alternate methods of direct implantation are important and more commonly employed. As evidenced, the transdiaphragmatic, subcostal incision, an approach which requires general anesthesia, is well tolerated by aged patients, providing endotracheal intubation is used and maintained until full recovery. Through a subcostal incision the rectus sheath is incised and the muscle retracted laterally. Some of the rectus fibers must necessarily be detached from the costal arch. Proprietalon fat is swept away from the undersurface of the diaphragm and an incision made in this structure to gain entry into the pericardial cavity. The undersurface of the right ventricle is exposed where one or two electrodes can always be implanted in an area devoid of epicardial fat. Three maneuvers may facilitate the exposure: the creation of an inverted flap of diaphragm to which traction is applied; the caudad retraction of the diaphragm; and induced paralysis of the diaphragm. The patient with a very narrow costal angle or the very fat individual presents difficulties with this operative approach, and an alternative method may be more suitable. Postoperative endotracheal extubation should not be permitted until full recovery of ventilatory function is assured, and this must be very carefully monitored in the group of elderly patients.

The patient with a very narrow costal angle or the very fat individual presents difficulties with this operative approach, and an alternative method may be more suitable. Postoperative endotracheal extubation should not be permitted until full recovery of ventilatory function is assured, and this must be very carefully monitored in the group of elderly patients. The generator unit is conveniently implanted subcutaneously in the left upper quadrant. There is remarkably little pain associated with this approach and it is well tolerated by the aged.

The left parasternal approach may sometimes be accomplished under intercostal nerve block and local infiltration anesthesia, but one should be prepared to supplement this with general anesthesia when necessary. After resection of the fifth and sixth costal cartilages

![Fig 1—Sudden discontinuance of rapid pacing achieves a prolonged asystole and quiet heart.](image-url)
and division of intercostal bundles, the pleura is retracted laterally, the pericardium incised, and one or two electrodes implanted on the anterior midright ventricle. One surgeon has managed to accomplish this after the resection of only one costal cartilage, but he utilizes a pacemaker catheter sutured into the right ventricular myocardium rather close to the septum.

For the sake of completeness, one should mention the sternotomy approach to the anterior right ventricle. This can be achieved through a complete midsternotomy or by a combined upper midline abdominal and lower midsternotomy incision. Poor healing in the very elderly and debilitated must be considered a drawback to this approach.

**Permanent Transvenous Endocardial Pacing.** We do not electively insert a temporary pacing catheter as a preliminary step for this procedure. However, perhaps 30 percent of our patients have had a temporary catheter in place at the time permanent implantation is undertaken. The temporary catheter should be removed under fluoroscopy before the conclusion of surgery since its withdrawal may dislodge the permanent catheter when withdrawn.

The electrocardiogram should be monitored throughout the procedure and the cardiac rate and rhythm influenced by isoproterenol, lidocaine, or atropine, as needed. All means must be available for possible resuscitation.

The external jugular, the cephalic, and the internal veins are the available routes. The cephalic vein provides the most desirable route on theoretical grounds, but in elderly females the vein is not infrequently atrophic. When this route is elected, the entire surgical procedure is carried out infraclavicular; the initial skin incision being extended medially and inferiorly from the deltopectoral groove for the purposes of developing the subcutaneous pocket. Reports of wire breakage, due to torsion of the leads, strengthen our opinion that a left-sided approach to the cephalic vein provides a better and more gentle curve for the lead. Most often we have used the right external jugular vein. If entry into the superior cava cannot be promptly gained, the transverse incision is extended medially to expose the internal jugular vein between the heads of the sternomastoid muscle. We do not ligate the internal jugular vein, but secure the lead at the point of penetration with several purse string sutures of nonabsorbable suture. After a short loop in the neck, the catheter is carried subcutaneously across the medial end of the clavicle, where there is minimal movement of the catheter with shoulder motion. We have preferred a unipolar catheter. The stylet should be gently curved at its end to facilitate passage of the lead into the right ventricle, but a straight stylet is used for the final positioning in the right ventricular apex. The tip of the catheter should be wedged under the trabeculae at the apex so that cardiac contraction causes a synchronous and slight buckling near the tip, but a gentle curve lies in the right atrium without angulation. The flange on the catheter tip serves admirably to minimize catheter displacement, and this is now an infrequent occurrence. The fibrous sheath developing later tends to minimize dislodgment. Attempted forceful withdrawal at a considerably later date could cause damage to the tricuspid valve where the catheter may be adherent. If there is any doubt about the position of the catheter in the apex of the right ventricle, a lateral X ray should always be obtained before the procedure is terminated. In the correct position the catheter tip lies immediately retrosternal. A position in the coronary sinus may stimulate a correct placement as seen in the anteroposterior view, but a lateral film usually, but not always, discloses the error. In the coronary sinus position, the electrocardiogram reveals current activation from the left ventricle and shows RBBB in Lead V\(_1\) rather than LBBB. Rarely, according to Gulotta, an intracardial electromgram is required to distinguish between an epicardial position anteriorly in the coronary sinus system and the endocardial position at the right ventricular apex. We have not employed this modality. These difficulties, however, can all be obviated if one first sweeps the catheter tip through the right ventricle to identify its position in the RV chamber before seeking a final position at the apex.

Stable pacing at a low threshold should be sought; i.e., under 2 milliamps, and repeated repositioning may be required to achieve this. A position in the coronary sinus system usually, but not always, gives a high threshold. The threshold is measured with an external pacemaker. By decreasing the current after initial capture, the point at which capture is lost determines the threshold. The threshold may be expected to increase several fold before final stabilization.

When a satisfactory position is clearly obtained, the vein should be securely ligated around the catheter with several nonabsorbable sutures of the plastic variety. The emerging loop of catheter is stabilized with a small plastic sleeve which is sutured to surrounding tissue to eliminate kinking. Additional sutures which may be required about the leads should be of absorbable material so that some play in the fibrous sheath which forms will ultimately be permitted. It is essential to close the platysma muscle in the neck to prevent the possibility of late erosion of the catheter through the overlying skin.

The pacer pocket is prepared in the infraclavicular region. We have not favored the axillary position where shoulder motion has been reported to cause wire fracture. In very emaciated patients, we have extended the leads subcutaneously to the upper abdomen where more abundant covering is usually found.

If the original implantation has been made properly, subsequent generator changes are simple. However, before the unit is lifted completely free of the pocket,
TECHNIQUES AND MODES OF INSERTION

The entrapped lead posteriorly should be freed from its sheath so that withdrawal of the unit does not lead to dislodgment of the lead farther distally, indeed, even from the endocardium.

The implantation of pacemakers which stimulate the atrium or synchronize from the atrium necessitates some alteration in the techniques described above.

The indications for this generation of pacemakers are primarily the hemodynamic advantages of the atrial pump and the avoidance of parasystoles. It is the most physiological responsive mode of pacing, and there may be a gradual return to this mode by physicians as reliability of these pacers increases. Theoretically, this mode increases the exercise capability of the healthy individual and provides an advantage for the patient in heart failure. However, the older patient not in heart failure should be provided with a ventricular pacemaker. The atrial pacer is clearly not indicated for the patient with coronary artery disease where a controlled slow rate is to be preferred. When atrial arrhythmias are present or develop subsequently, an atrial synchronous pacer may well increase the difficulties of management.

The most reliable method of atrial pacing is obtained by suturing an epicardial lead directly onto the atrium; to the left atrium, via a left anterolateral thoracotomy, or to the right atrium via a midsternotomy, or if only an atrial lead is to be applied, the right anterolateral approach. The electrode is placed near the base of the atrial appendage. Bruck has described an approach to the right ventricle and right atrium through the subxiphoid route, but this would seem to be both difficult and unreliable.

Currently, the most popular route is transvenous, usually the external jugular, for the atrial lead, with the ventricular lead being inserted through the cephalic vein. The straight catheter has proved unreliable; only the J-shaped catheter against the right lateral wall or in the atrial appendage has yielded an acceptable and reasonably stable atrial pickup. The position is not so critical for atrial stimulation as for atrial pickup. Dodinot has urged that if a P-wave potential throughout the entire atrium of 1.5 mv is not obtained, the procedure should be abandoned. A position in the appendage is to be preferred since potentials of up to 3.0 mv may be obtained in this position. A pacer sensitive to an input signal of .5 mv is used. Carlens and Lagergren have described the insertion of the atrial electrode via mediastinoscopy. The scope is inserted behind the right pulmonary artery slightly to the left of the midline and the lead deposited in a small area behind the left atrium. This approach has been used infrequently or not at all in this country. For the patient with sinoatrial arrest, an atrial pickup electrode used in con-

Fig 2—A tracing obtained by telemetry which reveals one of the problems with P-wave pacing.
junction with a sensitive demand pacemaker is most suitable.

I have had no personal experience with atrial catheter placement except in a few instances of temporary atrial pacing. However, Dr. Nick Smyth of Washington, D.C., kindly supplied me with his own personal statistics relative to his success with a special J-shaped catheter in which placement has been made in the atrial appendage. Of 20 attempts, there were 5 failures either because of low P-wave voltage or high threshold for stimulation. Two of 15 initially successful cases were not stable and subsequently converted to ventricular pacing. The other 13 have remained stable.

Figure 2 is a tracing which was obtained by telemetry and it shows one of the problems with P-wave pacing. The patient was exercised until the rate rose to 120, at which moment the blocking mechanism of the P-wave pacemaker intervened and rather abruptly the rate of the patient dropped from 120 to 50–60. As the patient rested, her intrinsic rate began to fall and the blocking mechanism was cancelled, and her rate suddenly accelerated from 53 up into the 90's. These sudden changes in heart rate are not desirable and represent a drawback of synchronous pacing.

I wish now to present illustrations of pacemaker problems:

A child of eight with congenital heart block had been resuscitated several times by her father. A P-wave synchronous pacemaker was implanted with epicardial leads which crossed the costal arch. Flexion creasing was soon noted at the costal arch in this very active
Fig 4—A direct measurement of arterial pressures before the pacer cut in.

Fig 5—At the moment the pacer cut in, blood pressure decreased abruptly 30-40mm of mercury.
child, and subsequently breakage caused malfunction four months after implantation. Repair was accomplished by splicing the more durable Elgiloy wire to the broken lead, and, in addition, the leads were transposed below the costal arch and through the diaphragm. The P-wave pacer functioned well for another year until it caused a very rapid rate and the child died at school, an example of the so-called runaway pacemaker.

The next case illustrates a suboptimal position of the catheter in the right ventricle. In the lateral roentgenogram the catheter does not reach to the sternum, but pacing continued satisfactorily for three or more years. This position should not usually be accepted. Occasionally the left diaphragm may be stimulated by a catheter which penetrates partially or completely through the right ventricular myocardium. We have also observed stimulation of the right diaphragm by a unique mechanism. In this case, a platinum iridium lead over the right clavicle fractured one to two years after its placement. A new catheter was inserted into the right ventricle and the broken lead was cut off and allowed to retract. From this time on, the patient noted intermittent twitching in the right side of the abdomen and contraction of the right diaphragm was confirmed by fluoroscopy. The tip of the second catheter was seen to lie very close to the tip of the first catheter. We surmised, and I believe correctly, that the phrenic nerve at the superior thoracic inlet was being stimulated retrograde up the original broken lead (Fig. 3). When the generator unit is subsequently changed, we can probably use a low output unit which will suffice for pacing the heart without major leak of current to the other lead.

Another case illustrates the problem of myocardial perforation. Two months after the implantation the catheter appeared to be in good position and was functioning well. Soon thereafter direct diaphragmatic or left phrenic nerve stimulation was noted. One month later a change in catheter position was noted and pacing was now intermittent. One year later stable stimulation of the heart was occurring, but the catheter had again changed position and the oblique roentgenogram revealed that the tip of the catheter was quite far posteriorly, presumably in the pericardium. When pacing ceased soon thereafter, we exposed the generator unit and measured a stimulating threshold of about 12 milliamps. A high output generator was implanted and satisfactory pacing was reestablished.

A Ph.D. engineer sustained a myocardial infarct. A temporary catheter was considered necessary at this time. Six months following discharge from the hospital he developed mild Stokes-Adams attacks due to intermittent heart block. A QRS-triggered demand pacer was inserted. Subsequently, he developed episodes of lightheadedness, faintness, and weakness, and he could detect the moment at which his pacemaker took over functionally. He had fewer symptoms when he was exercising and the intrinsic heart rate exceeded the demand pacer rate of 70. A direct measurement of arterial pressures was made and it was noted that at the moment the pacer cut in, the blood pressure decreased abruptly 30-40mm of mercury (Figs. 4 and 5). When the conducted rhythm returned, the pressure rose to the preëxisting level. With the patient resting the pacer cycled in and out. An explanation for this mechanism may be sought in the paper by Levy and Edelstein.

The above illustrations represent only a few of the many complications of a surgical and electronic origin associated with the art and science of pacemaking. Many of the frustrations of the past have been overcome, but each new advance also brings its problems.

**Author's note:** I was aided in the preparation of this paper by Dr. David H. Sewell who at the time was a Cardiac Surgical Fellow at the Medical College of Virginia.

**PANEL DISCUSSION**

**Dr. Drew:** Do you in any way recommend that an internist and general surgeon team up to implant the permanent transvenous type pacemaker, or do you feel that the internist should concentrate on learning the technique of the temporary pacing catheter and transfer the patient to the nearest medical center?

**Dr. Bosher:** We have different situations at the Medical College of Virginia. Unfortunately, we do not have a cardiologist in the X-ray department when we implant the permanent pacer; they are much too busy with their own duties. However, at hospitals where I have had a cardiologist work very closely with us, I found this to be a very favorable situation. All of the temporary catheters are now implanted by cardiologists in our hospital, and I think this is the way it should be because they see these patients as emergency patients. I think the surgeon can help with the permanent implantation, but I am quite aware of the fact that cardiologists sometimes do the permanent implantation.

**Dr. Drew:** Am I correct in saying that you think permanent pacing would be better reserved for the medical center?

**Dr. Bosher:** No. I think permanent pacing can be carried out in any good hospital where you have an interested team; that is, doctors who have familiarized themselves with these techniques.

**Dr. Drew:** I think this is an interesting point, because we do see people put into ambulances with an isoproterenol drip.

**Dr. Bosher:** This is very bad. Where the patient is in this type of extreme situation, I think it is better to have the surgeon go to the hospital rather than have the patient go to the surgeon.
Emergency Management of Pacemaker Failure*

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The purpose of this report is to describe the more common problems encountered with permanent pacemakers and their management.

One of the serious recurring problems is loss of output from the pacemaker. As a result, no pacemaker impulse whatsoever is seen on the electrocardiogram and the heart rate appears slower than the fixed rate of the pacemaker. In such a situation, if a patient is pacemaker dependent, whereby he will become symptomatic without the pacemaker, a temporary pacemaker electrode must be inserted immediately as an emergency procedure. On the other hand, if the patient can get along for a while without the pacemaker, then the pulse generator can be replaced electively under local anesthesia.

In taking care of any patient, it is extremely important to decide whether or not the patient is pacemaker dependent. This information must be readily available; thus, by providing the patient with some type of identification bracelet or by labeling the chart with a specific tag, the patient will be treated appropriately and without delay.

Another problem that appears frequently is change of pacemaker rate. The most common cause for a change in rate is the drop in output of the battery. This can usually be determined by the exclusion of two other possibilities: competition and change in impedance. If the patient is “competing,” the pulse at the wrist may be considerably slower than the rate seen on the electrocardiogram. An electrocardiogram will demonstrate whether or not the pacemaker is behaving normally, and if the pulse deficit is a result of an inability to palpate early coupled beats. A change in the impedance of the electrode, as seen when the tip of an electrode breaks, can also change the pacemaker rate if that particular model is load sensitive. However, most of the time if there is a real change in the rate, as measured on the electrocardiogram or any suitable interval counter, the usual cause is a drop in battery voltage and pulse generator output, which is treated by the replacement of the unit.

A noncompetitive pacemaker firing at a fixed rate indicates that the sensing circuit has failed. Sensing failure causes difficulties in making a proper diagnosis by external examination, X ray or electrocardiograms alone. Many judgments can only be made by operating on the patient and exposing the pulse generator and electrodes. However, an X ray, both PA and lateral, may reveal malposition of the electrode or perforation of the heart. There is also the possibility that scarring has occurred in the area of the electrode with a drop in the amplitude of the intracardiac signal to which the pacemaker no longer responds. With the pacemaker Surgically exposed, the amplitude of the R wave and the output of the pacemaker should be measured. If pacemaker output has dropped, failure to sense has been caused by battery exhaustion. If a new pacemaker does not behave properly in its sensing mode, something has happened at the electrode myocardial interface; consequently, a new electrode position must be found or a more sensitive pacemaker implanted. When sensing failure has occurred after the 20th month, a safe assumption may be made that battery exhaustion was the cause.

If a pacemaker fails to elicit a ventricular response and the rate is the same as it was when the pacemaker was inserted, there is probably something wrong with the electrode position. Either the electrode is dislodged or the threshold for excitation exceeds the pacemaker output. High threshold occurs most commonly from sepsis along the electrode, scarring of the myocardium, or a break in the wire within the insulating sheet. Short circuits can also occur, shunting some of the current away from the heart; this is usually due to a set screw on the sur-

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face of the pacemaker that is not properly insulated, leaving a path between the anode and the cathode. Short circuits occur at other areas in the same way, but most of these problems are detected on the operating table. If careful analysis reveals a problem that can be easily corrected, such as splicing a broken wire or repairing broken insulation, it is wrong to replace the pacemaker.

If there is failure to capture and the rate of the pacemaker has also changed, a safe assumption would be that the output is down. The possibility that something has happened to the impedance must also become a consideration; however, the former is by far the more common reason. Exposure of the pacemaker and testing of the threshold for stimulation and output of the unit will solve this problem.

Runaway pacemakers, unfortunately, continue to present a problem. In runaway cases, modern pacemakers have been constructed to ensure a simultaneous drop in output to avoid capture of the heart. In other words, when the rate increases, the output will fall below threshold. Removal of a runaway pacemaker is urgent even if the rate is only slightly increased. This will help prevent further increases that may occur within a short period of time. The treatment for a runaway pacemaker is either rapid incision to cut the wire, pacing on that wire, or removal of the pacemaker from its pocket to disconnect it. The latter is preferable if time is available. Pacing can be continued with an external unit on the same electrode, thereby preserving it; a new pulse generator can then be implanted using the original wire.

Pacemaker extrusion is another complication that may be encountered. Causes of extrusion are pressure necrosis of the skin overlying the pacemaker, acute or chronic infection and hematomas. Whatever the mechanism, once the pacemaker erodes through the skin, several therapeutic options can be considered. The first is not to do anything, because once the pacemaker appears through the skin, enlargement of the hole often occurs slowly. There will be a draining sinus that can be covered with a bandage through the full 20 to 24 months of pacemaker life, if the patient is willing to put up with the inconvenience. Whether the unit is changed at once or at the end of the battery life, the surgical technique for replacement is the same; an incision is made on the opposite side, isolating the field from the infected side. By sewing drapes to the skin after the new pacemaker electrode is inserted, the infected unit can then be removed by a separate surgical team. Once the old pacemaker electrode has been withdrawn, the new one is definitely positioned and, if necessary, used for temporary pacing. When the clean operation is finished, the wound on the original side is drained and closed. Another option may be irrigation of the infected wound with antibiotics. On rare occasions, this method has been successful in aborting infection and pacemaker extrusion. Veins are always available for permanent transvenous pacing. If the cephalic vein on one side has been used, other veins will still be acceptable at a later date, including the subclavian vein, or its tributaries, and the external and internal jugular vein.

Another troublesome complication is twitching of the diaphragm. This is uncommon, but when present it is usually a sign of trouble; such as perforation of the heart or malposition of the epicardial electrodes. Occasionally, a transvenous electrode may have pulled back into the atrium where it would stimulate the right phrenic nerve. Once in a while the diaphragm can be stimulated through the intact ventricle, especially if the ventricle is very thin. All of these possibilities must be corrected by repositioning the electrode. If there are two epicardial electrodes near the left phrenic nerve, the problem can be corrected by converting it to a unipolar system. Upon determining which electrode produces the twitch, it should be used as the indifferent electrode, using the other one as the stimulating electrode. In this way, implantation of an entirely new system can be avoided.

There have been instances in which pacemakers have been seen jumping back and forth in their pockets. When these pectoral muscles near the pulse generator twitch, some form of correction should be made. If it is a bipolar pacemaker with both electrodes in the heart, obviously there must be a bare wire or an uninsulated set screw. These defects produce direct stimulation of a somatic muscle. A break in the insulation is a more common problem, but is often difficult to fix. The broken Silastic sheath must be reinsulated and shifted to a unipolar system, or a new system must be installed.

Twitching in unipolar electrodes can be caused by various other factors. There have been cases in which an electrode has been pulled out entirely, wrapping around the pacemaker so that the cathode was in the subclavian vein against the pectoralis major muscle. The pacemaker can also turn over with the anode facing the muscle rather than the skin. Although this does not always cause twitching, a twitch is less likely to occur when the pacer is plate up rather than plate down. Since a twitch can occur in any position, it is important not to use muscle relaxants on those occasions when general anesthesia is required during insertion of the pacemaker. If a pacemaker has flipped over, it can be recognized by palpating the pacemaker, observing if it is loose in the pocket and then flipping it to the proper position. In unipolar pacing systems there are other kinds of leaks around the electrode connector, par-
particularly around the Silastic collar or the set screw, that can produce short circuit, high current density paths to somatic muscles.

There has been some recent discussion about electrical interference. An unusual example of this occurred with a patient who had an external Medtronic pacemaker with a bipolar electrode in the heart and an EKG telemeter attached to his chest. Several times he had experienced complete asystole. Thinking that the cause might be electrode malposition or that the pacemaker was defective, he had been taken to the catheterization laboratory. After trying many different external pacemakers, all of which showed the same problem, the telemeter was removed, revealing that the radio frequency transmission from the telemeter had been blocking the external pacemaker. Such interference with pacemaker function is rare, but is something to be aware of in coronary care units. If a telemeter is used in a patient with a pacemaker, the telemeter should be kept far away from the pacemaker. Better still, a different type of monitoring system should be used.

Another problem encountered is the handling of a patient who complains of dizzy spells, yet the electrocardiogram reveals that the pacemaker is functioning normally. To decide what is the problem, a series of exclusions must be considered. Is the output of the pacemaker dropping? This is likely if the pacemaker is 22 months old and there is also a change in rate. Is there a high threshold so that pacemaker output is just borderline? Is there intermittent malposition of the electrode? This is difficult to diagnose, but often can be suspected by the X-ray configuration of the leads. Is there intermittent contact of a broken wire within its insulation? Does the patient have intermittent bursts of ventricular arrhythmias? This must be suspected if in a long rhythm strip frequent ventricular premature contractions are seen.

"Pacemaker syndrome" is still another area that causes problems. This is characterized by the symptomatic fluctuation of the blood pressure, usually in a patient with a fixed-rate pacemaker, from atrial and ventricular asynchrony. The diagnosis can be confirmed only by ruling out all other causes of weakness and by recording wide blood pressure fluctuations. What about electromagnetic interference? Did the patient do anything unusual preceding the weak spells? When all of these ruminations have been considered and all causes of weakness have been discarded, it must be assumed that the patient is right; something is wrong with his pacemaker system. If you cannot make a definitive diagnosis, the pacemaker should be exposed and its output and excitation threshold should be tested. Nine times out of ten, the patient is right and it is necessary to adjust the pacemaker.
Pacemaker longevity is a serious problem to overcome. However, our immediate concern is to get as much useful life from a pacemaker as possible. On the average, pacemakers fail in about 23 months. The only pacemaker that lasted much longer was a fixed-rate unit, the Medtronic 5860, which is no longer available.

A past suggestion in handling this problem has been to change the pacemaker electively at an arbitrary time, but that time has varied tremendously as the manufacturers once selected 30 months, and more recently 15 months. If a pacemaker is replaced at 15 months, very few of them will actually be near the end of their life span.

For those who like to experiment, there are several kinds of pacemakers available. Presently, we have used 33 different models from eight manufacturers with varying pacing modes. Pacemakers can be fixed rate, atrial synchronous or noncompetitive; bifocal and cyclic; or attached to unipolar or bipolar electrodes. Electrodes have been situated in the myocardium or endocardium, using a variety of testing devices.

Because of the importance of replacing pacemakers as late as possible, the study of the electrical output of the pacemaker has begun in a pacemaker clinic. A pacemaker impulse has certain definable characteristics: its amplitude can be measured at its leading edge, trailing edge, and halfway between; and the electrical impulse has width or pulse duration, measured from the leading edge to the trailing edge, displaying a typical configuration (Fig. 1). A sensitive index of pacemaker function is the repetition rate or pulse interval. Hopefully, if a comparison in pacemaker impulse is made between its appearance at 4 months, which is the best time to establish a base line, and its appearance at 22 months, the following changes may be noted: the amplitude decreases, the pulse width shortens, but the configuration remains about the same (Fig. 2).

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Fig 1—Examples of pulse generator impulses (schematic) as seen on the oscilloscope. Measurements of A, B, C, and D are made and recorded for sequential comparisons. Impulses 1 and 4 seen commonly with Medtronic pacemakers; impulse 3 with Electrodyne; impulses 1, 2, and 5 with Cordis; impulse 4 with American Optical; impulses 2 and 4 with Vitatron; impulse 4 with General Electric.

Fig 2—Typical changes in pacemaker impulse over 18-month period.
Laboratory tests have produced a study of American pacemaker models. Our engineers, working in conjunction with engineers from each company, evaluated the behavior of each pacemaker on the bench as a number of battery cells varied. This study showed that some pacemakers cease to function where one cell fails, but most others cut off after at least two of the five cells have been lost. The drop of pulse generator is directly related to the number of cells and varies from unit to unit, and the output of the pulse generator is directly related to the number of cells and also to the resistive load. The amplitude changes with the load and so does the pulse width. Pulse duration also varies with the number of cells. In some pacemakers, the pulse width will widen as the number of cells change; in others, the pulse width will shorten. Pulse width is also affected by the load.

Pacemaker sensing circuits also have a failure threshold. As the number of cells are reduced, the sensing circuits begin to fail. In most pacemakers, the sensing circuit will fail before there is a loss of output from the pulse generator. The sensing circuit of the American Optical pacemaker, however, will continue to function well into late pacemaker life. This parameter can be measured and tested in the clinic.

In evaluation of the pulse interval, it seems preferable, although there is no general agreement on this, that a pacemaker should slow down as the number of cells decrease. If the pacemaker stops when it is going slowly, idioventricular rhythm will start fairly soon after the last effective beat. If the pacemaker speeds up as it fails and then stops entirely, there will be a rather prolonged period of asystole before spontaneous cardiac action resumes. Although most new pacemakers are designed to slow down as batteries are exhausted, actual performance varies considerably. The number of cells is not the only thing that will affect the rate; change in resistive load also affects the rate with considerable variation from model to model. All things considered, clinical evaluation of this pacemaker problem is rather complex. There is some doubt as to whether the resistive load has changed from minute fractures in a lead wire or electrode corrosion, or whether the batteries are gradually becoming exhausted.

Within the physical set up of our clinic, there are five examination rooms in which various test devices are connected to the patient by an electrocardiographic cable attached to the limbs. Here lead I of the electrocardiogram is taken, also a "P" lead in which one electrode is on the left arm and the other over the pulse generator. Lead I is used to record a rhythm strip. The P lead is used for evaluation of the amplitude of the electrical impulse. The amplitude of the impulse depends upon the relative position of the stimulating and exploring electrodes. Therefore, it is of great importance to eliminate all factors that will affect the positioning. Hence, this examination must be performed with the patient in the same position at each visit, using the same exploring electrodes.

In the testing room there is a computer and peripheral equipment. A monitor is used to observe the electrocardiogram during the test process. The output of an amplifier goes to an interval counter and to an oscilloscope where the wave shape is photographed and the amplitude and pulse width are measured. An electrocardiogram is also taken. All this information is then inserted into a computer which types a report, via the peripheral equipment, displaying the rhythm strip, a picture of the wave form, and the doctor's comments. Within seconds, the entire history of the particular pacemaker under test can be seen.

Patients come to the clinic every four months for the first year; every two months for the next six months; and every month after eighteen months. It is quite important that the patient be emotionally reinforced during his visits to the clinic. Great care is taken during the examination to provide the kind of emotional attention and support that he really needs. As a result, the patient likes the clinical atmosphere and will appear for further visits.

For patients who find it difficult to come to the clinic because of infirmity, age, or distance from the hospital, a telephone system has been devised. This system is used to count only pacemaker pulse rate. The patient holds an electrode in his left hand and puts the other electrode against the pacemaker with his right hand. There is a magnet in the electrode which turns the pacemaker to its fixed-rate mode. At the hospital our technician places the phone in a cradle, and then records the rate to within 1/10th of a millisecond accuracy on a digital counter.

There is an advantage of not only counting the electrical impulse, but also counting the physical pulse at the fingertips. A click is heard for the electrical impulse and a musical note for the physical pulse, enabling the physician or technician to tell whether one follows the other. This method has proven to be quite valuable as adjunct to the clinic. Of 89 cases where a satisfactory analysis could be made, interval change occurred as a sign of failure in 67; interval change alone occurred in only 14 cases. In the great majority of cases, the interval changed simultaneously with another parameter. Therefore, the total analysis gives somewhat more information than measuring the pulse interval alone.

This test may seem to be complicated, but the entire procedure takes exactly three minutes to perform; the patient can actually go through the whole process in about five minutes. In New Jersey there is now a network of these clinics, all working in identical fashion. There are also nine affiliated centers, and five or six others waiting to join. At each center the evaluations are done in the same way, including the telephone communication. Each center communicates by telephone
with the computer at the Beth Israel Medical Center and then receives a typed report immediately at their own hospital.

There are many potential advantages to this system. Within one year there will be 1500 pacemaker histories in the computer in New Jersey alone. A physician will be able to know very quickly the actual behavior of a large sample of many different kinds of pacemakers. If a physician decides to use X pacemaker for the first time and something goes wrong with it, the computer system will provide data on the behavior of 30 other X units inserted by other physicians in the state to which the pacemaker in question can be compared. However, the computer is not a doctor and does not replace good clinical acumen. A careful office examination by a doctor, in which the rate is accurately measured and an evaluation of the rhythm strip made correctly, will provide enough information to determine if things are grossly wrong.

Until recently, these were the considerations in replacing a pacemaker. If in one month there was a certain change of rate within 10 milliseconds (not readily detectable by taking a peripheral pulse), or if there was a change in the pulse duration or amplitude (also not detectable at all by examination), the patient would be reevaluated within a few weeks. If any two of these changes occurred simultaneously or if the sensing circuit failed, the pacemaker would be replaced immediately.

In 1970, 318 patients were studied and 126 pacemakers replaced. Forty of these replacements could not be evaluated in the clinic because replacement was for reasons other than battery exhaustion. For example, 7 pacemakers were changed because they were extruding; several pacemakers were replaced because some patients did not want to wait; others were changed because patients did not come to the clinic often enough. Other instances involved pacemakers with high thresholds, runaway rates, plug corrosion, and broken insulation. Of 87 that were suitable for analysis, 76 (87%) were removed electively with only 13% having errors. These errors occurred for several reasons: nine were actually new pacemakers and not much was known about their behavior; three were cases in which the change seen was underestimated; several patients missed crucial visits; three cases were without clues whatsoever. Some of these cases were due to sudden failures of a component that were not detectable.

The results from the clinic in comparison to the bench tests revealed, pulse duration the one exception, that bench tests corresponded to what was actually observed in the clinic. Because the bench analysis has proven so helpful, in the future whenever a new model pacemaker is introduced, it will be tested to provide some idea of what the performance will be in its failure mode.

In conclusion, the clinical approach to pacemaker evaluation is important because it is at least 90% accurate. Hopefully in the near future, with the accumulation of accurate data to rely on, computers will be used to a greater degree in aiding the diagnosis of pacemaker malfunction: the computer will "remember" how things ought to be and will tell when things are wrong, suggesting when the pacemaker should be changed. However, it is hoped to soon have pacemakers that will operate reliably for ten years. Elective replacement at nine years will then be feasible and frequent clinic tests will no longer be required.

**PANEL DISCUSSION**

**Questioner:** What would be the indication for using a low voltage pacemaker?

**Dr. Parsonnet:** Pacemakers are made with a rather sizeable leeway between the anticipated threshold and its output. Presumably, if you can use or have a lower output, you can get the pacemaker to last longer. An alternative would be to use a fewer number of batteries, thus making a smaller pacemaker. The Cordis people have experimented with this idea, and I am sure other companies will do the same. As a matter of fact, the Medtronic company tried to do the same thing by having a potentiometer on it; you just turned your output down and caused a decrease of a drain on the battery. Because of this substantial decrease, we are planning to go back to this method soon.

**Questioner:** Why haven't they implanted a set of batteries in parallel elsewhere in the body to take over when and if the other batteries fail?

**Mr. Berkovits:** Because most of the problems in batteries involve short life, the extra set may fail as quickly as the first set. There are many battery failures that are unrelated to exhaustion.

**Dr. Drew:** Do any of the companies make any financial adjustments if the pacemakers go out prematurely?

**Mr. Berkovits:** To my knowledge, all manufacturers in the United States are giving some credit for pacemakers returned with premature exhaustion.

**Questioner:** How accurate is the phone monitor?

**Dr. Parsonnet:** It is accurate to a tenth of a millisecond for the interval counter, which just measures pulse interval.

**Questioner:** Have you used the transmission of a rhythm strip to give you the intervals?

**Dr. Parsonnet:** No, we have not. Although it would give you some information, it would not give you quite enough. You would have to count the rate on an electrocardiograph which has a sizable error in paper speed, 2 to 3 percent, I think. It will tell you whether or not the pacemaker is capturing the heart, but there are other ways to find that out.
Dr. Drew: Do you predict that other states will have to follow suit as the number of pacemakers grows, in order to have the type of follow-up that you have in New Jersey?

Dr. Parsonnet: Perhaps a modification of it. I am not quite certain what the phone is going to bring. Right now, at least for my purpose, it is an ancillary test procedure which is helpful but not sufficiently diagnostic. I think every patient with a pacemaker should go to some kind of an evaluation clinic. He should be going either to a doctor who sees him regularly or to a center with a group of patients.

Dr. Tarjan: There is a company in Philadelphia that follows these people with pacemakers. I would imagine that their methods of evaluation are somewhat similar. Should a doctor entrust his patients' care to this sort of an organization?

Dr. Parsonnet: I do not know if there is anything wrong with the organization. Personally, I would rather test the rate myself. I don't trust myself half the time, but I do trust myself more than I would a company located elsewhere. The reason we use the system I showed you today is because we have control over it; we can count the rate with our own technicians, and if something doesn't look right, we can run downstairs and see what the matter is. With the Pacer Tracer company, which is the one you referred to, they count the pulse at the fingertip and the rate, and call you on the phone. It is that middle man that makes me a little uncomfortable. They have a clever system and I don't want to knock them in any way; it may turn out to be a good idea.

Questioner: How do you prepare a patient for surgery with a pacemaker, where cautery is needed?

Dr. Parsonnet: The danger is having the ground plate of the cautery near the pacemaker, and the active end of the cautery directly across from it. If you are going to do a transurethral resection, it is best if your pacemaker is up in the infraclavicular area. Be sure the patient is sitting on the ground plate and that you are monitoring the heart. Because the cautery causes interference, monitor the pulse manually. The only precaution necessary is that you don't have a direct line across the pacemaker. Although other opinions may vary, we do not restrict the surgeon in any other way; we just tell them that we will be around in case they need any help.

Dr. Drew: What restrictions are on the patient if they should decide to go for ultrasound treatment on the back?

Dr. Parsonnet: Tell them to avoid diathermy, ultrasound, and everything else. We have had only two instances of interference in our patients; both in the early days of pacemakers when adequate protection was not provided. Nevertheless, there has to be a delicate balance between warning the patients and scaring them.
His Bundle Recordings*

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When we record atrial activity on the His bundle electrogram we are recording from the area of the atrium which is around the tricuspid valve, that is, the lower part of the right atrium. The normal range of conduction time from the region of the sinus node to the low right atrium is about 25 to 45 milliseconds. When we are at the A-V junction we can record the local atrial activity, the His bundle potentials, and the ventricular activation. Normally, we record this simultaneously with three surface EKG leads. We are using this technique for quantitative measurements, because it enables us to know where the P wave and the QRS complex begins, and depending upon what EKG lead you chose, it could vary the onset of these complexes. The three leads are Lead 1, AVF, and V1, which give us the three plains combination: frontal, sagital, and horizontal. A breakdown of the PR intervals which we measure are: (1) the PA, or the intra-atrial conduction from the sinus node down to the low right atrium; (2) the AH, or the atrial activation to the His bundle and represents conduction through the A-V node (a normal range for this time in our laboratory is 50 to 120 milliseconds); and (3) the HV, which is measured from the beginning of the His bundle to the earliest ventricular activation seen either on the EKG or on the bipolar electrogram. In our laboratory the normal values for this are about 35 to 45 milliseconds. The His bundle deflection per se, is about 15 to 20 milliseconds in duration, indicating how long it takes for the impulse to go through the main His bundle before it bifurcates.

Up to now it has been taught that the left bundle activates the myocardium earlier than the right bundle branch or that the septum gets activated earlier from the left side than the right. The present data which we have collected by recording from both right and left heart in man as well as from original experimental work done by Hoffman’s laboratory and by Rosen in a Chicago laboratory indicates that impulse arrives at the terminals of the left and the right bundle branches at the same time. Also, the anterior and posterior divisions of the left bundle receive the impulse at the same time. This means that if we were to damage one bundle branch, there should be no delay in the impulse arrival to the ventricle. Thus it would follow that if we have a unilateral bundle branch block the HV time should not increase. It is considered now that we have three branches: the main right bundle and two divisions of the left. Therefore, if we were to damage either of the two of these three, we still should have normal HV time because the undamaged branch would take the impulse to the ventricle without any delay. We found clinical evidence to support this new data when we studied about 29 patients who had left axis deviation with a narrow QRS complex yet maintained normal HV time. There were also indications that if we produced rate-dependent right bundle branch block through atrial pacing, the HV time would not change. In one patient atrial pacing was done at a rate of 150 beats per minute. This produced second-degree Wenckebach type of block at the A-V node and right bundle branch block. The QRS complex following the blocked P wave was narrow. The H-V time in this beat with a narrow QRS complex was the same as in the following beat which showed right bundle branch block. In studies of the left side, the same phenomena was observed. We had a patient who had a rate-dependent left bundle branch block and the HV time did not change when the QRS complex changed from narrow to left bundle.

Further, we have studied about 25 to 30 patients with pure right bundle branch block. Up to this time the feeling was that most of the patients with right bundle branch block should have a normal HV time. But 20 percent of the patients with pure right bundle branch block show abnormal HV time. This indicates that these patients have additional disease either in the main stem His bundle or in the left main bundle or its two subdivisions. This would explain why statistically 70 percent of patients with right bundle, left axis deviation show abnormal HV time. They are presumed to have additional disease elsewhere. Since this is not reflected in the EKG, what we see is the dominant one-bundle lesion which is the most diseased one.

For years it has been said that whenever you see a combination of first-degree block and right bundle branch block, it indicates bilateral bundle branch

* Presented at the American College of Cardiology and the Medical College of Virginia Cardiac Pacing Symposium, April 16 and 17, 1971, Williamsburg, Virginia.
block. But this is not always the case. The major delay which produces first-degree block occurs in the A-V node, not in the His-Purkinje system. Patients may have additional disease in the Purkinje system, but the major part comes from the delay in the A-V node. So first-degree right bundle doesn't always mean bilateral bundle branch block. The only way to validate the His bundle potential is by His bundle stimulation. Right bundle deflections look like His bundle, and there is no way to differentiate these except by this means. This is very important when we are making quantitative analysis and diagnosis, because if you have a wrong deflection the diagnosis is worthless.

We studied a patient who for several years had right bundle branch block and a normal PR. On the day of admission, he showed 2:1 A-V block with right bundle branch block. That evening he showed a transient episode of complete heart block, and again the QRS complex showed right bundle branch block. The EKG made us suspect that the block was occurring somewhere in the A-V node, because the complex was identical and the patient had only right bundle without any axis deviation. When we studied him four days afterwards, he was showing 2:1 A-V block, the same as when he was admitted. On the EKG, every P wave was blocked beyond the His bundle deflection, and the conduction time through the A-V node, that is the AH time, was completely normal—60 milliseconds. In the beats which were conducted, the HV time was prolonged at 85 milliseconds. The His bundle deflection was about 35 milliseconds. What was happening was that the lesion which produced the block was in the main stem His bundle. Unfortunately, His bundle has been ignored for a long time as far as lesions are concerned because they do not show anything on the EKG. And for that reason some people have never made a diagnosis of His bundle blocks. But if right and left bundle produce lesions, why should the His bundle be immune to pathology since it is the same tissue. I believe about 40 percent of the patients with bundle branch lesions have disease in the main stem His bundle, and I think that as we collect more data we will be able to stress this.

We have studied about 90 patients with right bundle branch block, left axis deviation. As I suggested earlier, if there is a lesion in either of the two divisions the HV time theoretically should be normal. His bundle records in a patient with right bundle, left axis and normal PR showed a normal HV time of 40 milliseconds. In another patient under the same conditions we observed the HV time at 75 milliseconds. This indicates that these patients are not a homogeneous group and may explain why some patients with right bundle, left axis have gone for ten years without an A-V block and some develop it within a year. It follows that some of them have a significant disease which is not reflected in the EKG's and others have no disease in the third division.

We have observed that when the H-V time is abnormal, the third division is damaged. A patient came to the E.R. with second-degree A-V block. We brought him to the cath lab with 1:1 conduction and at that time the first beat on the surface EKG showed right bundle, left axis deviation. The His recording revealed an abnormal H-V time of 75 milliseconds which indicated that we should expect disease elsewhere in the conduction system. Our expectations were corroborated when the EKG spontaneously showed us right bundle, right axis deviation in the next two beats.

We have studied 25 patients with right bundle branch block, right axis deviation, and all of them except one showed abnormal HV times. Statistically this indicates that in patients with right bundle, right axis deviation the posterior division is damaged and there is additional disease which extensively involves the main His bundle or the anterior division.

**PANEL DISCUSSION**

**Questioner:** Dr. Narula, did I understand you to say that the majority of patients with right bundle, left axis had abnormal HV times, and does this mean that they have disease here more than the right bundle itself?

**Dr. Narula:** Clinically, it is felt from the EKG pattern that right bundle, left axis deviation means right bundle branch block and damage of the anterior division. Right bundle branch block with right axis deviation...
deviation means damage in the right bundle and the posterior division. This is what has been proposed by Rosenbaum, and theoretically it is right. In reality this is not always true, however. It is misleading to assume from the surface EKG pattern that a right bundle, left axis deviation indicates that a third division, i.e. the posterior division, is okay. Seventy percent of these patients have additional disease elsewhere, and probably in the posterior division itself, which we can't see from the EKG. The right bundle, right axis theoretically could be normal, but statistically this is unlikely, and the majority of the patients have additional disease elsewhere involving either the anterior division or the main stem His bundle. Just to reemphasize that point again: there are three divisions. If disease is present in only two of the three divisions, the HV time should be normal. Ninety-five percent of the patients with right bundle, right axis have abnormal HV time; therefore, disease is probably present in the third division as well, despite the fact that the EKG doesn't show it.

Questioner: Are you giving pacemakers to patients who are asymptomatic but the EKG shows right bundle, left axis deviation?

Dr. Narula: As yet, we do not make a decision to implant a pacemaker based on His bundle recordings. We rely instead on the clinical symptoms the patient is having, a history of syncope, and an abnormal HV time. Some day I hope we will have enough data to show that patients with abnormal HV time develop complete heart block in three years. Then we can give pacemakers prophylactically.

Dr. Samet: What if we have a patient with right bundle, left axis, right bundle, right axis and a normal heart rate, and he has a syncopal episode? Can we automatically assume that his syncopal episode is related to his conduction disturbance? We have had at least two patients with right bundle, left axis and one patient with right bundle, right axis who have come in with syncopal episodes and the referring physician was pressing for the insertion of a pacemaker. In these three cases, His bundle study revealed a normal HV time, but in none of them was a pacemaker implanted on this basis. We think this is a correct decision and we are following this approach now.

Dr. Dreifus: I think that what Dr. Samet said is right. The patient should have symptoms before you put in the pacemaker whether it is first-, second-, third-, or fourth-degree A-V block. Lenègre did a good deal of anatomic studies, and he makes it clear that it doesn't make much difference whether block is above or below. It is the importance of the symptoms based on block. There are many patients walking around with high-grade block in which there is no conduction from atria to ventricle who do not have symptoms. The minute symptoms develop and block is present, then I think it is time to put in pacemakers. If you do not prove block is the cause of the symptoms the decision rests on a clinical diagnosis. I think there are other ways to prove whether the symptoms are due to block. You can use exercise stress tests, drugs, or tape recordings, and a number of these patients will show intermittent periods of nonconducted beats. Then the decision whether or not to implant a pacemaker can be made on that basis.

Dr. Narula: I just want to clarify a point. Dr. Dreifus just said that you can stress the conduction system to bring out the block whether by drugs or by exercise. The His-Purkinje system is so immune to some of these stresses that if you were to increase the rate by pacing or by giving atropine, the majority of these patients would still conduct 1:1, despite severely prolonged HV time. For some strange reason it acts in such a capacity that either it conducts or it doesn't, so that neither atrial pacing nor drugs will stress the system. When patients with 1:1 conduction do develop second-degree A-V block at high atrial pacing rates, it almost always occurs in the A-V node. Only a few cases develop block distal to the His bundle with increase in atrial rate and most of these latter cases even spontaneously manifest second-degree block. The degree of block in the patient with His-Purkinje system lesions will increase, but if he has 1:1 conduction it usually doesn't. We have had patients who came to the E.R. with complete heart block. We brought them to the cath lab, put in a temporary pacemaker, and within two hours we were able to pace them 1:1 up to 160, and they had HV times of 75 milliseconds. So I think atrial pacing might be misleading in checking the condition of the conduction system.

Questioner: Dr. Narula, regarding control of the HV interval which you have been placing so much reliance on. Have you studied the same patient a number of times, knowing what the previous HV interval was, so that you could understand what variance you are getting?

Dr. Narula: We had follow-up studies on the patients with normal conduction times and also on the patients with abnormal ones. For some strange reason the HV interval seems to be exactly reproducible, but not the AH. The AH interval varies from sitting to sitting, and from time to time you recatheterize the patient. But the HV is the same whether you recatheterize them a week or a month afterwards. We have studied at least 100 patients in this length of time where the purpose was to observe the progress of disease. The majority of them have not shown even a 5 millisecond change as yet. So the test, as far as HV is concerned is reproducible, provided you validate that the His bundle is what you are recording.
Techniques of Bedside Pacing*

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In temporary pacing the primary approach is transvenous—either percutaneous or via a cutdown in the jugular or brachial vein system. Tripolar and quadripolar pacemaker catheters which have been used for His bundle recordings are now obtainable as well as the bipolar variety. Among the latter is the floating catheter which may also be used as an intracavitary electrocardiogram. Doctor Furman of New York City has been extremely successful with this type in approximately 500 patients.

The problem with the floating catheter is the potential hazard of displacement. Thus, it may be worthwhile to consider using a stiffer or semi-floating catheter. Remember that the catheter may have to remain in place for as long as three or four days, and you want to be sure that you can depend on it to stay in position. One type of semi-floating catheter has a slight bend at the end. It is much more likely to remain securely in the trabeculae of the right ventricle, and it may be passed blindly when image intensification is not available.

The rigid bipolar catheter is too large for the percutaneous route and a cutdown must be performed through an invasion either in the external, internal jugular or brachial vein. But if you want to save yourself a lot of time and trouble in making these temporary pacemakers remain in place you may have to use this kind. There is one type of rigid catheter on which the electrodes are located 15cm from the tip. It is softer than the other rigid catheters, and it is to be inserted into the pulmonary artery so that right ventricular outflow pacing is possible. Figure 1 shows the electrodes in the outflow tract of the right ventricle and the tip in the right pulmonary artery. The importance of this type of catheter in addition to the position of the electrodes is its softness which doesn't irritate the ventricle. We have had a number of cases of ventricular fibrillation during catheter placement in the apex of the right ventricle in patients with an acute myocardial infarction. We have defibrillated them calmly during the

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catheterization, but this is always a risk, and a bolus of lidocaine 100mg is administered intravenously prior to the insertion in patients with this condition. This prophylactic measure results in less ectopic beats and runs of ventricular tachycardia, and I would recommend it in such patients in order to avoid serious ventricular rhythms.

When using percutaneous catheters it is important to select a needle with a good 13-gauge lumen in order to pass the catheter. Percutaneous pacemaker catheters may be inserted at mid clavicle at a 45 degree angle as shown (Fig. 2) or above or below the clavicle. The problem in approaching the subclavian vein is that you really have to fish for it. But any third-year medical student can accomplish this, and fortunately you can get into the vein blindly. The catheter can then be placed down into the superior vena cava, right atrium, and on into the ventricle. In addition to catheter placement in the outflow tract of the right ventricle it is possible to pace from the apex, the right atrium, and the coronary sinus. Usually you can document the position of the catheter by attaching the external lead to the electrocardiogram and observing the electrocardiographic changes. It must be remembered, however, that if the electrode is touched in an ungrounded situation ventricular fibrillation may occur with the intracavitary electrode technique. Therefore, the use of image intensification is preferred.

I invariably use demand pacing particularly in patients with recent myocardial infarction to avoid competition. An exception to demand pacing during an infarction would be the use of overdrive pacing for ventricular tachycardia.

There are several types of pulse generators, and you may make your own if you like. However, to provide effective pacing rates for atrioventricular sequential pacing, rapid atrial stimulation, and paired pacing you have to use a coupled pulse generator (Fig. 3).

Coupled or S,S paired pacing is a bedside technique that I would not recommend in ventricular pacing unless you have a computer that has measured the cycle length ahead and programmed the next S,S interval. Otherwise, ventricular fibrillation will very likely occur. Paired pacing of the atria in cases where atrial tachycardia are uncontrollable by any other means can be used to reduce the rate by one half. The problem with ventricular pacing is that there is only one pulse in the brachial artery for every two electrical stimuli and this may cause a decrease in the stroke volume which could lead to acute pulmonary edema.

Dr. Philip Samet’s approach of rapid atrial stimulation as a means of converting atrial arrhythmias is of value in the digitalized or over-digitalized patient in whom electrical countershock may not be feasible. Additional indications for rapid atrial stimulation are recurrent atrial tachycardia or flutter particularly during an acute myocardial infarction as well as during cardiac catheterization when transient arrhythmias may result in changes in the oxygen determinations. The restoration of normal sinus rhythm would then allow one to obtain more reliable data. The use of rapid atrial stimulation also avoids the need for administration of an anesthetic.

One indication for the use of temporary pacing is premature ventricular beats in the presence of a prolonged QT interval. The use of drugs would further prolong the QT interval and increase the opportunity for ventricular tachycardia. I would also recommend pacing in patients with acute myocardial infarction and in patients with high-degree A-V block associated with ventricular tachycardia. In these instances overdrive pacing as a bedside technique has worked very well.

Another indication for temporary pacing is reentry atrial tachycardia in patients with Wolff-Parkinson-White syndrome. I would recommend, however, that pacing not be performed in patients with WPW as a means of converting atrial flutter or fibrillation or to induce these arrhythmias in patients with this condition. As Dr. Neil Moore has demonstrated so beautifully, you may buzz or overdrive the atria to the point of ventricular fibrillation.
Figure 4 shows an example of a six-week-old child with paroxysmal atrial tachycardia in which overdrive pacing was used. The electrocardiogram shows delta waves during sinus rhythm and is termed the type A form of WPW syndrome. A floating catheter was placed and the rhythm converted by atrial pacing. The child was managed with a radio frequency pacemaker on a permanent basis. The aerial of the transmitter was placed over the heart and sinus rhythm was then re-established each time. The child's mother learned to stop the tachycardia by broadcasting the pacing at home.

Ingenuity is an important requirement in using any of the above bedside techniques because many problems will have to be solved on the spur of the moment. You should be knowledgeable about these techniques and have all the equipment around you so that you will have a system you can depend on.

TECHNIQUES OF BEDSIDE PACING

PANEL DISCUSSION

Questioner: Dr. Dreifus, what are your criteria for a floating catheter versus a rigid catheter?

Dr. Dreifus: I would use a floating catheter in the extreme emergency. I would go to the rigid or semi-rigid ones if I had a little more time to make decisions. Then I would use image intensification to get the catheter into the trabeculae, making sure it is well placed and that the threshold is adequate and low enough so that both sensing and pacing are possible. If you lose the sensing part, you then have a fixed-rate pacemaker, which will not sense premature beats and will give you a new impulse on top of a T wave. Premature ventricular beats with an acute infarction could be dangerous, so it is imperative that you get the best position so that the catheter doesn't slip out. You may need it in there for a week, two weeks, three weeks, four weeks, sometimes. I know Dr. Furman used to put them in for a year back in 1959 or so, and they remained in place and patients stayed alive because of these rigid catheters. But if I had my choice, I would choose a rigid one, and I would spend some time getting it into position with plenty of antiseptics so I wouldn't get infection.

Dr. Parsonnet: May I make one comment about the rigid catheters? We have some interest in them since the one you are talking about was designed by Dr. Zucker at our hospital and is sometimes called a Zucker catheter. We are not cutting down any longer on those. We are putting them in through the subclavian vein with a needle, and we pass a rather large sheath over the needle and then pass the catheter through the sheath. But we also feel quite strongly about the statement you've already made, Leonard, that these electrodes should be fixed and placed with anatomical control using fluoroscopy and not with the free-floating technique unless you are in a terrible hurry and unable to obtain bedside fluoroscopy.

Dr. Zoll: Dr. Dreifus has made a point of great importance and I want to take this opportunity to say that I agree with it. I think it is extremely important to use demand pacemakers in patients with acute myocardial infarction. The risk of repetitive response in fibrillation is a very serious one—indeed, so serious that I try not to use catheters at all in these circumstances. But if you are going to use one, for goodness sakes, make it a demand. In this regard, I should point out that although it seems difficult in Philadelphia, and perhaps in other places, in some areas one finds that emergency control can be accomplished readily. While you are preparing to move the patient or the bedside X-ray machine, you can manage the situation possibly with external electric stimulation and by intravenous drug therapy with epinephrine or isoproterenol. Most of the time it is a matter really of only a minute or so to control rhythm.

Fig 4—Type A form of Wolff-Parkinson-White syndrome complicated by PAT.
with intravenous drug therapy. You can arouse a ventricular pacemaker, you can accelerate it, you can overdrive ectopic activity, and you can gain control very often with drugs, but then you are not in such a desperate hurry to get the catheter in place.

Dr. Baird: How reliable is demand pacing in a temporary situation as far as over-sensing or undersensing either with the temporary floating, semi-floating, or stiff catheter electrode systems?

Mr. Berkovits: For temporary pacing you must have the electrode placed as well as you would for permanent pacing, because detection of the endocardial depolarization depends on proper electrode placement. You must have an endocardial signal to control the demand pacemaker, otherwise you don't have a demand pacemaker. The endocardial signal of the floating electrodes is not acceptable and will not be reliable for controlling demand pacing.

Dr. Samet: I would like to add something here. Anyone who is trained in catheterization can pass at least three quarters of the rigid catheters into the right ventricle blindly just by repeated manipulation. You won't be in an ideal position, but you will be in a reasonable position to capture the ventricle and then move the patient over to a catheterization laboratory with X-ray facilities in order to gain a final position. Therefore, we rarely, if ever, use a semi-floating catheter to capture the ventricle.

Dr. Richardson: Is anybody using the Swan-Ganz balloon technique? Do they make a pacemaker catheter?

Dr. Dreifus: Yes, they do make a pacemaker. (Have you used it?) Yes, we have used it. It is an excellent catheter, incidentally, because it has a little balloon that is inflated, and the stream will then carry it on its way. Its most important use of course is to put it in the pulmonary artery when you want to monitor pulmonary artery pressure or something like that, rather than as a pacemaker. But what Dr. Samet said is true. You can pass a rigid catheter into the ventricle without EKG control. Even though my machines are constantly tested, I still have the fear of leakage in a ground. We had a 3-prong plug ground on one of our new Marquettes, and we had a 65 ma leak in that machine. You never know when you are going to run into this, and I have fibrillated patients. Even with putting rigid catheters in under fluoroscopic control, they may become displaced. So you must have golden fingers.

Dr. Parsonnet: We have been playing with semi-floating catheters for a long time, and again, Dr. Zucker has one with a little parachute on the end of it, like an umbrella, which guides the catheter into the ventricle. Our experience has been much the same as most other people's with these temporary electrodes. Even though you know where the tip is and obviously you can tell that it is in the ventricle from the electrocardiogram, you don't know where the rest of the loop is. The loop of the electrode can be in the pulmonary artery, or up in the atrium, or in the ventricle. This insecure position of the loop can lead to malposition, and then it will pass with a bolus of blood and pull the tip out of position. The free-floating catheters have knotted around themselves and around trabeculae, and we have had to go after them. In our experience, and the same with Tom Killip in New York and others, about 20 percent have pacing failure, even though they have been positioned with an electrocardiogram.

Dr. Baird: Dr. Samet, how do you verify your position in blind insertion of the stiff catheter?

Dr. Samet: It is not important to verify it, because all you want to do is put it into a position where you can gain at least temporary pacing to control the situation and then move the patient.

Dr. Baird: Do you rely on intracavitary electrocardiograms?

Dr. Samet: No.

Dr. Richardson: It seems to me that it is far safer than EKG monitoring hooked up to 110 volts.

Dr. Narula: I just wanted to make a comment that if you do want to use temporary pacing by the bedside in an emergency situation, instead of hooking up the catheter to the EKG for finding out the position, a simple way may be to hook up the catheter to the pacemaker so you don't defibrillate the patient by EKG output. Then turn the pacemaker on a 5 or 10 ma, and when you get into the right atrium, you will have atrial pacing and you just record a surface EKG. When you get in the RV you will find ventricular pacing. This way you may bypass the problem of shocking the patient at the bedside. If you are in the shoulder, you may have some muscle twitching, so that it is much simpler to use this method.

Dr. Samet: I just want to emphasize one point again. The term “bedside” has been used several times this morning. When we use rapid atrial stimulation this is not a bedside technique. It is a technique to be used only under fluoroscopic control—not under EKG control. You don't dare turn on rapid atrial stimulation at 300, 400, 500, or 1000 without having absolute fluoroscopic control of the tip of your catheter or you fibrillate the ventricles. This was done once at our hospital and fortunately the patient was revived. So rapid atrial stimulation is not a bedside technique in the sense of a technique in the ward, or even in the ICU, away from X-ray facilities.
Electric current is both valuable and hazardous in cardiac therapy. Small amounts of electric current are enough to electrocute the patient, especially if the current is delivered through a catheter in the heart. It has been demonstrated that currents as low as 10–15 microamperes are capable of inducing ventricular fibrillation. This minute amount of current is actually a thousand times less than what you can barely feel with your hand; nevertheless, this amount of current, internally, can cause many problems. Therefore, electrical equipment, when used, must be used carefully. We must be careful of the amount of current induced by capacitive coupling, or other sources of leakage.

The problem becomes further complicated in a hospital where electrical equipment is used with inadequate maintenance. The most desirable situation would be to have an electrical technician on the staff to maintain the equipment, making certain that the equipment has been installed correctly. Both the technician and the physician should have the responsibility of insuring proper use of the electrical apparatus. The manufacturer designs the equipment to work in a certain environment; if it is used properly within this environment, it is usually safe.

Presently, there is no perfect solution to the problems of electrical hazards in cardiac therapy. Although electrical hazards are continually occurring due to improper wiring, it would be an enormous expense to have all hospitals suitably rewired. Committees and workshops have been created to study these problems. In 1970, the National Academy of Sciences published an excellent report on Electrical Hazards in Hospitals. However, the complexity of the problem remains with no universal solution.

What can be done and what can the physician do? One recommendation is proper grounding; but for this to be effective, proper grounding would have to correlate directly with the design of the equipment. Some equipment is designed to be used without ground. Should the patient touch ground or any surface at a different potential, a dangerous current may flow through the patient.

Although solutions to individual problems are usually found as each situation arises, there does not appear to be, as yet, a universal solution.

**PANEL DISCUSSION**

**Questioner:** How are the devices for recognition of electrical hazards in coronary units?

**Mr. Berkovits:** If you have a system that is designed to be grounded, a testing circuit may detect when you have a malfunction.

**Questioner:** What about the use of an electric bed?

**Mr. Berkovits:** As long as it functions properly you have no problem. The question is whether or not the convenience of an electric bed justifies the risk of a potential danger. The electrical equipment in a coronary care unit should be kept to a minimum. When functioning properly, the high isolation impedance of this system will improve your reliability and safety. However, capacitive currents and currents induced magnetically in a magnetic field can be hazardous.

**Dr. Zoll:** I think it is important to mention that there is a risk of electrical fibrillation not only in the coronary care unit, but in the operating room at the time of implantation of pacemaker replacement. Several of us have talked about the means to measure threshold at the time of operative manipulation at the end of the catheter electrode. At this time, we attach the electrode to a variable voltage source and a measuring device, usually an oscilloscope which is powered by alternating current. Although some oscilloscopes are now battery powered, every time one is connected, we risk inducing alternating current ventricular fibrillation. However, in experienced hands, like those of Dr. Parsonnet, this is a safe procedure. Before we attach these instruments to the patient's catheter, we routinely test for AC interference. With a quickly breakable
connection we then attach the instruments. In this way, if any ectopic interference activity is produced, we can disconnect the instruments properly. Extreme caution must be exercised when a low resistance pathway to the heart is exposed to the environment.

**Mr. Berkovits:** I would like to point out that in order to avoid confusion in an emergency situation, there should not be wires hanging in all directions. Whenever you work in this type of situation, you should have a clear vision of everything that is happening.

**Questioner:** What is the value of a voltmeter?

**Mr. Berkovits:** If you have a battery-powered voltmeter, you can measure voltage, milliamps, and resistance, but because the leakage currents are minute (10–15 microamps), no conventional voltmeter will detect them.

**Questioner:** Can AC-powered voltmeters be used?

**Mr. Berkovits:** AC-powered voltmeters do not offer appreciable increased sensitivity in resistance measurements, and you run the risk of inducing dangerous leakage currents.

**Questioner:** Concerning the usage of electrical instruments, how can you provide for maximum safety?

**Mr. Berkovits:** Because each hospital has a different wiring system, there is no single solution. A number of recommendations have been made for the formation of committees. The National Academy of Sciences has formed a committee on safety and hazards. Although they have not found a solution that would apply to all electrical hospital equipment, they do agree that the AC current delivered to the heart should be kept below 10 microamps.

**Questioner:** Is the battery-powered oscilloscope safe?

**Mr. Berkovits:** It is safer than a conventionally powered oscilloscope, but there are external conditions that may create a danger regardless of whether an AC- or DC-powered oscilloscope is used. Because electric currents can be inductively or capacitively induced, any wire near the heart can act as an antenna and induce more than 10 microamps.

**Questioner:** What about the hazards of static electricity?

**Mr. Berkovits:** Static electricity gives only a single discharge and will appear as a single stimulus.

**Questioner:** Dr. Zoll, please make additional comments on external shock therapy for cardiac standstill in a straightline EKG with arrest.

**Dr. Zoll:** External electrical stimulation, properly applied, will provide an effective and adequate stimulus to produce an electrical and mechanical response if the heart is capable of responding. If one acts quickly, within a matter of seconds after the onset of arrest, the resuscitation from ventricular standstill by external electric stimulation is an effective emergency measure that should be successful no matter what the background of the arrest may be. It is most commonly done in patients with Stokes-Adams disease with A-V blockage because these patients are most commonly affected with ventricular standstill. But, it is also effective in patients who have reflex vagal stimulation and arrest, in patients who have arrest due to drugs of one sort or another, in patients who have so-called "unexpected arrest" in the operating room while under anesthesia, and in patients with acute myocardial infarction. Because of the availability of this method of resuscitation and the availability of intravenous drug therapy, which can also be used to maintain and restore cardiac rhythm, we are less eager to introduce all the complications, risks, and troubles involved by the use of temporary cardiac pacing in many patients with acute myocardial infarction. I agree that there is an important place for temporary pacing in acute myocardial infarction, but I strongly maintain that if one is careful with patients with acute myocardial infarction, uses drugs properly, and stands by with emergency resuscitation, the need for catheter electrodes and the corresponding risk can be greatly diminished. I say that one must balance the various factors involved with one's clinical judgment and that one doesn't need to use temporary pacing quite as often as might be suggested today.
Case Presentations

CASE I:
A 43-year-old white male construction worker was admitted to the coronary care unit for evaluation of left anterior chest discomfort. How would you manage the following intermittent arrhythmia, which was noted shortly after admission? Vital signs remained stable.

Dr. Baird: Dr. Dreifus, how would you manage this patient’s arrhythmia?

Dr. Dreifus: You have here a premature ventricular systole which begins a run of slow ectopic beating presumably from the ventricle. It is not occurring on top of the T wave. The victim is in ventricular tachycardia at a rate of about 60 or 62, and it appears to be a slow ventricular idioventricular rate without retrograde P waves. In this case I would try speeding up the sinus rate with a little atropine to eliminate the slow rate.

Dr. Baird: Would any other panelists like to comment or disagree?

Dr. Zoll: I would simply watch the patient and do nothing else.

Dr. Parsonnet: In our coronary care unit with continuous monitoring, this is not an infrequent arrhythmia, though Dr. Samet has just whispered in my ear that he sees it rarely in his unit. It is totally benign and has no serious consequences, therefore it is not treated.
CASE II

This 75-year-old black male represents an arrhythmia related to digitalis toxicity. Is temporary pacing indicated?

CASE II

Dr. Baird: This is a frequent problem in city hospitals. There is no adequate history available on this patient, but apparently he has been taking too much digitalis. The question is, if he has atrial fibrillation with high-degree A-V block, how would you manage him during the interim period?

Dr. Samet: First, how symptomatic is this individual?

Dr. Baird: He is confused, and he has been nauseated, but there has been no evidence of Stokes-Adams seizures. He has mild congestive failure.

Dr. Samet: Well, the rate here is in the low 40's. If he is symptomatic with something that would be improved by increased rate, then I would pace him. If he is not symptomatic with this arrhythmia, and if I felt it was due to digitalis, I would not pace, but stop the digitalis and just watch him. One point I want to stress is that when high-degree A-V block is a manifestation of digitalis toxicity, the administration of potassium is inappropriate and may have untoward consequences. If this is just complete heart block, then the potassium may have no further block-producing tendencies.

Dr. Baird: How do the other panelists feel? Dr. Dreifus, are you concerned about pacing a patient who has an arrhythmia due to digitalis?

Dr. Dreifus: I think if he is confused, I would do as Dr. Samet stated. The question is whether or not he is symptomatic. Now there is some controversy over potassium. I would tend to agree that if in the sub-junctional regions you give enough potassium or give it too fast, you will cause further block. However, there is no doubt that potassium will antagonize digitalis in the N region of the A-V node and increase conduction. If the serum potassium were depressed and if I were worrying about causing further depression, I would then pace the patient and still administer potassium.

Dr. Baird: Thank you. What are the hazards with temporary pacing in patients with digitalis-induced arrhythmia? What has been your experience in this situation?

Dr. Dreifus: What you are worried about is the mechanical stimulation of that catheter number 1 and 2. If you induce a propagated response from the pacemaker on top of T wave in an over-digitalized patient, is the ventricle more vulnerable? Yes, the ventricle is more vulnerable, and the risk is increased. But with proper placement and prophylactic use of agents while you are passing the catheter and with great skill and few premature ventricular beats, this is really not a problem.

Questioner: What is your attitude toward treating this patient with diphenylhydantoin?

Dr. Dreifus: I don't think diphenylhydantoin would be of value in this instance. In the concentrations used clinically, we have shown that it increases block in the A-V node. If there were ectopic ventricular beats with block here, I certainly would avoid drugs and go the pacing route.
CASE PRESENTATIONS

CASE III:

This patient with a permanent pacemaker developed substernal pain and hypotension. Would sympathomimetic amines be helpful to restore his blood pressure?

CASE III

Dr. Baird: Dr. Zoll, how do you feel about the concomitant use of sympathomimetic amines in patients with pacemakers?

Dr. Zoll: With the functioning permanent pacemaker in place, one would treat hypotension with drugs as if the patient were in normal sinus rhythm. As long as the pacemaker rate is reasonably rapid, as in this case, the appropriate vasoconstrictor agents (i.e., neosynephrine or isoproterenol) can be used. Most of these drugs do have a tendency to arouse ectopic activity as well as acceleration of dominant rhythm; however, with a well-functioning pacemaker, a positive inotropic action is not immediately seen.

Dr. Samet: My first reaction after seeing this tracing would be to ask for other leads. While this tracing probably represents paced beats followed by a T wave, I am not mathematically certain whether or not these other beats represent premature beats. I would have to see other leads in order to be certain.

Dr. Baird: I have used this case to suggest that in certain patients sympathomimetic amines might lower the threshold for ventricular fibrillation, and there might be instances in which untoward administration of these drugs might precipitate ventricular tachyrhythmias. Anybody have any comments or experience with this situation? Dr. Dreifus?

Dr. Dreifus: Yes. I learned my lesson on the cardiac surgery table where we used a good deal of sympathomimetic amines, particularly isoproterenol. Beyond a flow of 3 micrograms per minute of isoproterenol, you will get increased ectopic beating that could be fatal to the patient. Therefore, it is a matter of degree how you use these agents. I don't think we can say that they are all good or all bad; we should, however, be aware of the dangers and the limitations of administration.

Dr. Zoll: Isoproterenol clearly does arouse and accelerate ventricular pacemakers. There is a definite risk with isoproterenol; but if one gives the drug in a diluted solution, say 1 or 2 micrograms per minute, we can then step it up every several minutes. We usually change the dose every 4 or 5 minutes, watching what is going on all the time. You can keep on going until you get either the desired therapeutic effect of an increased cardiac output or increased blood pressure. If untoward effects appear (i.e., ectopic beats), reduce the dosage or adjust the administration of the dosage until no adverse effects are seen. There is no real limit as to how much you should give a patient. We have given over 30 micrograms per minute of isoproterenol to some patients with complete heart block, in what I consider to be a safe and effective way. On the other hand, some patients might run into trouble with a much smaller dose, in which case you stop and take care of the situation accordingly.

Dr. Samet: The arrhythmic effects of the vasoconstrictors really have two aspects. One is the direct aspect which has been alluded to, and the other is an aspect which follows the development of hypertension. That is, when one gets the desired effect by overshooting the mark, reflex arrhythmias secondary to hypertension can develop. Which leads me to my next point: One of the things we have learned to mistrust is the peripheral arterial pressure under these circumstances. We have, in a number of instances, obtained hypotensive readings with the usual blood pressure determination. Because you can readily overshoot the mark, we prefer monitoring the arterial pressure interarterially almost routinely when we feel we have to administer vasopressors. So I warn you not to trust the routinely determined blood pressures in these hypotensive patients.
CASE IV:

This 70-year-old white male was admitted for hematologic evaluation when he suddenly developed chest pain and acute EKG changes suggestive of diaphragmatic myocardial infarction. Several days later, he was placed on oral procainamide to suppress VPC’s. However, the following arrhythmia developed.

**Dr. Baird:** Dr. Narula, how would you manage the patient in this situation?

**Dr. Narula:** This looks to be a very interesting arrhythmia which terminated spontaneously. Because of the sudden sinus beat, the possibility of fusion beat is less likely. Therefore, I would say it is probably another ectopic beat which suppresses the ventricular tachyrhythmias and gives the sinus rhythm a chance.

**Dr. Baird:** You would give more suppressant drug therapy?

**Dr. Narula:** Because sinus rhythm is fairly stable in this patient, within the normal physiological range, I think this patient would require suppressant therapy.

**Dr. Baird:** Any other comments?

**Dr. Samet:** In this discussion about the treatment of cardiac arrest and ventricular arrhythmia, I would like to stress that Dr. Zoll and I were just talking about a very simple physical mode which does really work; we have seen a blow on the chest administered by a clinched fist stop ventricular fibrillation; we have also seen it restart the heart that was arrested in just a few seconds.

**Dr. Parsonnet:** As far as therapy in the cardiac care unit is concerned, there is a tendency to worry about the electrocardiogram and forget about the other things happening to the patient. For example, a person who has been in the intensive care unit for a few days, hypoventilating and getting all kinds of drugs, may have serious abnormalities in their blood gases and blood electrolytes. Before you rush off and start using a lot of other things, I think you ought to make sure that you have a normal electrolyte balance. Sometimes, by simply directing the blood gases or blood electrolytes, you can stop the arrhythmias.

**Dr. Dreifus:** I agree with Dr. Parsonnet; you have to look for other problems. But remember, these ventricular premature beats are bisecting the T wave. In other words, the onset is very close to the T wave, and vasoconstrictors will respond to drugs like lidocaine and procainamide. This repeated occurrence in the development of fibrillation brings up an interesting hiatus in drug therapy: Do we use a drug like procainamide or lidocaine, which actually decreases fibrillation threshold, or do we switch to an agent like bretylium? We just might end up treating these arrhythmias with bretylium when they are prefibrillatory or fibrillatory. If there are single ventricular premature systoles, you may take the lidocaine approach first just to get rid of the ectopic beating period. However, if they are prefibrillatory, as in this particular record, we may be using bretylium in the future. I think you had better keep a very close eye on the literature to see where we go from here.

**Dr. Baird:** Dr. Samet, do you agree with Dr. Dreifus’s enthusiasm in regard to bretylium?

**Dr. Samet:** If we are talking about bretylium as a drug of initial approach, I do not agree. However, if we are talking about bretylium as a drug to be used when others have failed, then I agree emphatically. There are side effects with bretylium even in the horizontal position which does not make it the drug of initial choice. We still rely upon lidocaine, quinidine, procainamide, etc., but have used bretylium with success on a number of occasions. The role of bretylium has to be
CASE PRESENTATIONS

kept in proper perspective.

**Dr. Richardson:** What about procainamide as an inciting drug?

**Dr. Baird:** Well, that's a point I was trying to emphasize. Dr. Dreifus, when ventricular tachyrhythmias revert spontaneously, doesn't this give you a clue that quinidine or procainamide was induced?

**Dr. Dreifus:** No.

**Dr. Baird:** Do any of the panelists have experience with recurrent ventricular fibrillation due to quinidine? Our experience is, in a limited number of cases, that they do not require defibrillation, but may be controlled with overdrive pacing.

**Dr. Samet:** Would you tell us a little more about the circumstances developed? I suspect that some of those cases do not represent quinidine sensitivity but inappropriate use of quinidine.

**Dr. Baird:** That is an important point in determining whether it is really a toxic effect, a rate-dependent problem, or an effect related to a true sensitivity as originally described; but this a point of argument. The procainamide was stopped and the patient's ventricular irritability decreased. He did not require any overdrive pacing as a technique suppressing drug-induced recurrent ventricular tachyrhythmias.

**Dr. Narula:** Could it be that the chest pains and the ischemic changes disappeared with time and were responsible for the disappearance of the arrhythmia rather than the discontinuance of the procainamide?

**Dr. Baird:** No.

CASE V:

A 2½-year-old child was in excellent health until he developed a viral illness, which was complicated by rapid heart rate. Initial therapy was digitalization to control "PAT." This failed and recurrent bouts of "VT" required 350 countershocks (100-200 W.S.).
Dr. Baird: Here we discuss the advantages and disadvantages of pacing. I have just mentioned the fact that atrial overdrive for ventricular pacing may control a certain arrhythmia. In this case the child, who had been in excellent health, developed a flu-like syndrome associated with recurrent bouts of tachyrhythmia. At first it was thought that these were paroxysmal atrial tachycardia. However, an intra-atrial lead demonstrated clearly dissociation between the atrial activity and the ventricular rhythm disturbance. The diagnosis was made that this was recurrent ventricular tachycardia in a child who possibly had some type of myocarditis or myocardiopathy. Assuming the diagnosis is correct, what would you recommend in the way of pacing or drugs to control this particular problem? The child had been given 350 countershocks by the pediatric service for recurrent bouts of ventricular tachycardia associated with hypotension. Dr. Dreifus, what are your thoughts on this problem?

Dr. Dreifus: Obviously this is a very difficult situation. I have not seen ventricular tachycardia very often in children. The use of digitalis here is contraindicated under the circumstances. I am not at all convinced that this is ventricular tachycardia. It could be junctional with aberrant conduction. Atrial pacing might work in this case if you could get the catheter to stay in place, but that is tough in a kid. Ventricular pacing may also work, but that too is difficult. What I have dealt with in children is the use of propranolol and quinidine. The problem is getting them to take that combination because it tastes terrible. But sometimes that will control the ventricular tachycardia. I have not used bretylium, but I think that if I had my choice after all the electricity that has passed through this kid, I might say this is one of the prefibrillatory or prerepetitive situations and switch to a drug like bretylium rather than push my luck with the other depressant agents.

Dr. Baird: Before we comment on bretylium, do you think there is any clinical trial justification of internal defibrillation?

Dr. Dreifus: I don't know that we could do it in a child though we do it in dogs. It takes a lot of hardware, and I am wondering if it would really be practical in the end. It works, but maybe Mr. Berkovits will have some ideas on how to develop the electronics to improve it. I would try to solve all problems like this pharmacologically rather than on an internal or external defibrillation basis. Once the patients are put on a prophylactic pharmacological treatment, the arrhythmia usually goes away.

Dr. Baird: Any other comments? After bretylium was administered intravenously in this child, acceleration of the ventricular rate occurred. Whether continued administration of bretylium would have been effective as an antiarrhythmia, I don't know. After I had caused rate acceleration with the drug, I was no longer asked to follow the patient. Dr. Dreifus?

Dr. Dreifus: Small doses of bretylium do have a tendency to aggravate the arrhythmia. There is no doubt about it. There are some electrophysiological reasons why this might occur. The trick is to use enough, but using bretylium in a kid for a rather unknown problem is risky. You may have been right to back off. I might have tried a large dose.

Dr. Baird: It has been an enjoyable day and a half. I would like to acknowledge the moderators, Drs. Richardson* and Drew* as well as the panelists for the support and advice required to make this a successful program.

*DONALD W. DREW, M.D., Director of Cardiology and Acting Director of Internal Medicine, Norfolk General Hospital, Norfolk, Virginia.

*DAVID W. RICHARDSON, M.D., Professor of Medicine, The Medical College of Virginia, Richmond, Virginia.
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Current Trends in College Health Medicine
PHILIP B. CHASE, Medford, Massachusetts

The Adolescent and Competitive Athletics
ISAO HIRATA, JR., New Haven, Connecticut

Psychological Aspects of the Management of Adolescents with Malignancy
CHARLENE P. HOLTON, Denver, Colorado

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Cardiac Pacing: Sponsored by The American College of Cardiology and the Medical College of Virginia
CHARLES L. BAIRD, JR., M.D., Guest Editor

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CHARLES L. BAIRD, JR., M.D., Richmond, Virginia

Historical Development of Cardiac Pacing
PAUL M. ZOLL, Boston, Massachusetts

Pacemaker Concepts and Terminology
BAROUH V. BERKOVITS, Miami, Florida

Electrocardiographic Recognition of the Various Pacemaker Types and Dysfunction
LEONARD S. DREIFUS, Philadelphia, Pennsylvania

Indications for Cardiac Pacing
PAUL M. ZOLL, Boston, Massachusetts

Techniques and Modes of Insertion
LEWIS H. BOSHER, JR., Richmond, Virginia

Emergency Management of Pacemaker Failure
VICTOR PARSONNET, Newark, New Jersey

Pacemaker Clinic
VICTOR PARSONNET, Newark, New Jersey

His Bundle Recordings
ONKAR S. NARULA, Miami Beach, Florida

Techniques of Bedside Pacing
LEONARD S. DREIFUS, Philadelphia, Pennsylvania

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BAROUH V. BERKOVITS, Miami, Florida

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Indications for Temporary Pacing


**Diagnosis:**
spasm reactor

**Decision:**
Donnatal

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*Note: Habit forming*

**Brief summary.** Side effects: Blurring of vision, dry mouth, difficult urination, and flushing or dryness of the skin may occur on higher dosage levels, rarely on usual dosage. Administer with caution to patients with incipient glaucoma or urinary bladder neck obstruction as in prostatic hypertrophy. Contraindicated in patients with acute glaucoma, advanced renal or hepatic disease or a hypersensitivity to any of the ingredients.

A·H·ROBINS A. H. Robins Company, Richmond, Virginia 23220
Diagnosis:
spasm reactor

Decision:
Donnatal®

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