Clinical Essays on The Heart

Volume I

Edited by
J. Willis Hurst

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NOTICE

Medicine is an ever-changing science. As new research and clinical experience broaden our knowledge, changes in treatment and drug therapy are required. The editors and the publisher of this work have made every effort to ensure that the drug dosage schedules herein are accurate and in accord with the standards accepted at the time of publication. Readers are advised, however, to check the product information sheet included in the package of each drug they plan to administer to be certain that changes have not been made in the recommended dose or in the contraindications for administration. This recommendation is of particular importance in regard to new or infrequently used drugs.

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Preface

The Change of the Name

The new name of this series about *The Heart* is *Clinical Essays on the Heart*. The former title, *Update: The Heart*, has been changed for a number of reasons. Some readers believed that the *Updates* simply provided more recent information about the material covered in the last edition of *The Heart*. The new series will maintain that role, as well as accomplish much more, its purpose being to:

- Continue to update the information presented in *The Heart*, which is published every four years. For example, a new procedure, such as nuclear magnetic resonance, may become available after *The Heart* is published; it would be presented to the readership as quickly as possible in the *Essays*.
- Present more detailed information on a subject that is discussed in the last edition of *The Heart*. An old subject such as atrial fibrillation, for example, should be reviewed every few years. The discussion might be one page in *The Heart* but an in-depth discussion of the subject might use thirty pages in an edition of the series.
- Present interesting subjects in the field of cardiology that are seldom discussed.
- Present more illustrations and more complete bibliographies than are possible in the textbook. Space allocations do not permit an author to use an unlimited number of illustrations or listings in a bibliography in a textbook such as *The Heart*. Rather, he or she is forced to bear the pain of omitting a favorite figure or reference in order to keep the book at a manageable size. The new series offers much more freedom.
- Consequently, each edition of the series will be well illustrated and the bibliographies for each article quite complete.
- Permit me, as Editor, to present material not necessarily suited to a textbook or a journal. Accordingly, the series may contain articles on: medical history, controversial subjects not dealt with in a textbook, and many other categories of manuscripts.

Most of the volumes in this series will not have a theme. Volume II, however, does have a central subject, "The Treatment of Atherosclerotic Coronary Heart Disease (Drugs, Coronary Bypass Surgery, and Coronary Angioplasty)."

I would like to thank my colleagues, worldwide, for their warm reception of the *Update* series upon its appearance. It is my hope that *Clinical Essays on the Heart* will enjoy the same enthusiastic response that the *Updates* received.

J. Willis Hurst, M.D.

Four editions of the *Essays* will appear between editions of *The Heart*.

April 1983	*Essays I*
November 1983	*Essays II*
April 1984	*Essays III*
November 1984	*Essays IV*

Contents

Pacemaker Faultfinding

HARRY G. MOND, M.D.

Then of the THEE IN ME who works behind
The Veil of Universe I cried to find
A Lamp to guide me through the darkness; and
Something then said—"An understanding blind"

EDWARD FITZGERALD, TRANS., 1859
The Rubáiyát of Omar Khayyam

The success of an implanted cardiac pacemaker is dependent on the establishment of a harmonious relationship between the artificial pacemaker and the human receiver. Failure of a pacemaker system may result from an electronic or mechanical defect within the pacemaker, a physiologic problem, or from a poor relationship between the normal function of both. In this article, the methods of pacemaker testing and a logical system for investigating patients with suspected pacemaker malfunction will be outlined. Only the ventricular-inhibited pacing system will be discussed.

THE NORMAL VENTRICULAR PACEMAKER ELECTROCARDIOGRAM

The pacemaker QRS (depolarization) consists of a stimulus artifact and the subsequent ventricular response (Fig. 1). The stimulus artifact represents the energy delivered by the pulse generator and is seen as a perpendicular voltage deflection from the ECG baseline. As the energy is dissipated through the myocardium, the stimulus artifact returns to the baseline as a decay curve. It is during this period that actual ventricular depolarization occurs, and thus the QRS is a summation of true depolarization and the decay curve. As seen in Fig. 1, the decay curve can on occasion be as large or larger than the actual QRS. Thus in order to establish true ventricular depolarization, it is important to recognize ventricular repolarization, i.e., the T wave, and a number of ECG leads may be required before satisfactory tracings are obtained.

The major pacing system used today is the ventricular-inhibited (demand or synchronous) system. Here the lead lies at the apex of the right ventricular chamber (endocardial) or is placed on the outer surface of either ventricle (epicardial).

Characteristic ECG patterns are recognized when

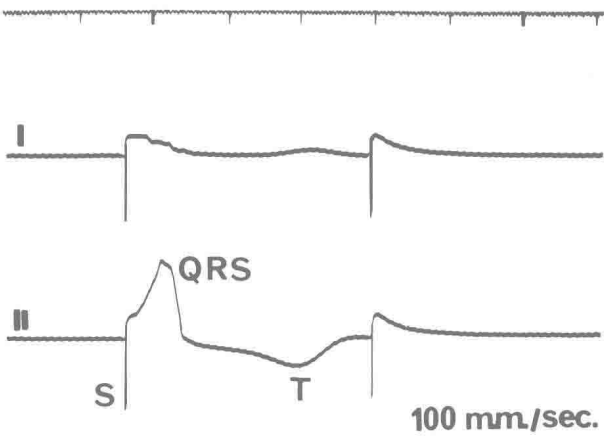
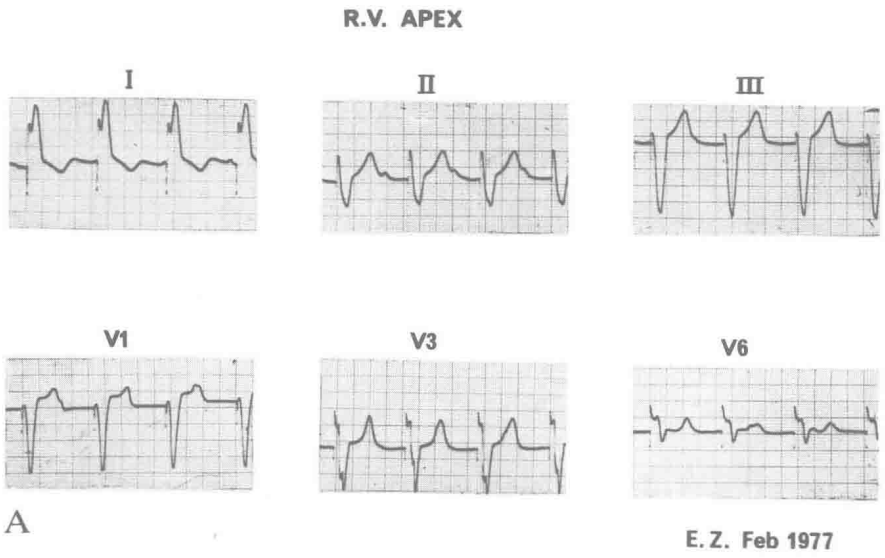


FIGURE 1 Unipolar endocardial lead in right ventricle. The pacemaker ECG consists of a stimulus artifact (S), depolarization (QRS), and repolarization (T). In lead I, first complex, the stimulus artifact is followed by a small QRS and T wave. The second complex is composed of a stimulus artifact and no QRS or T wave, i.e., a failed-paced beat. Note that the stimulus artifact deforms the baseline, and this could be misinterpreted as a QRS. However, there is no T wave. In lead II, the QRS and T wave are prominent, and despite the deformity of the baseline in the second complex, there is no doubt about failed pacing. This illustrates the value of taking more than one lead in interpreting the ECG in patients with pacemakers. The predominant R wave in lead II suggests that the lead has become dislodged and the electrode is in the right ventricular outflow tract.

pacing is established from various parts of the heart. Unipolar endocardial pacing from the apex of the right ventricle results in a left bundle branch block configuration, and the frontal plane vector is to the extreme left (Fig. 2). However, if the electrode is in the right ventricular outflow tract, then the bundle branch block is the same, but the front plane vector is now normal or to the right (Fig. 3). Epicardial left ventricular pacing has a right bundle branch block configuration with an axis depending on the position of the electrode on the left ventricular surface.

With ventricular-inhibited systems, pacing will continue at a regular interval unless inhibited by a premature spontaneous ventricular depolarization (own QRS), such as a ventricular extrasystole or a return to sinus rhythm faster than the pacemaker rate (Fig. 4). Sensing of the spontaneous rhythm occurs via the lead that transmits the electrical information to the sensing circuit of the pulse generator. The other type of pacing is fixed-rate, or asynchronous. Here pacing occurs irrespective of the underlying cardiac rhythm (Fig. 5).

*From the Department of Cardiology, The Royal Melbourne Hospital, Victoria, Australia.



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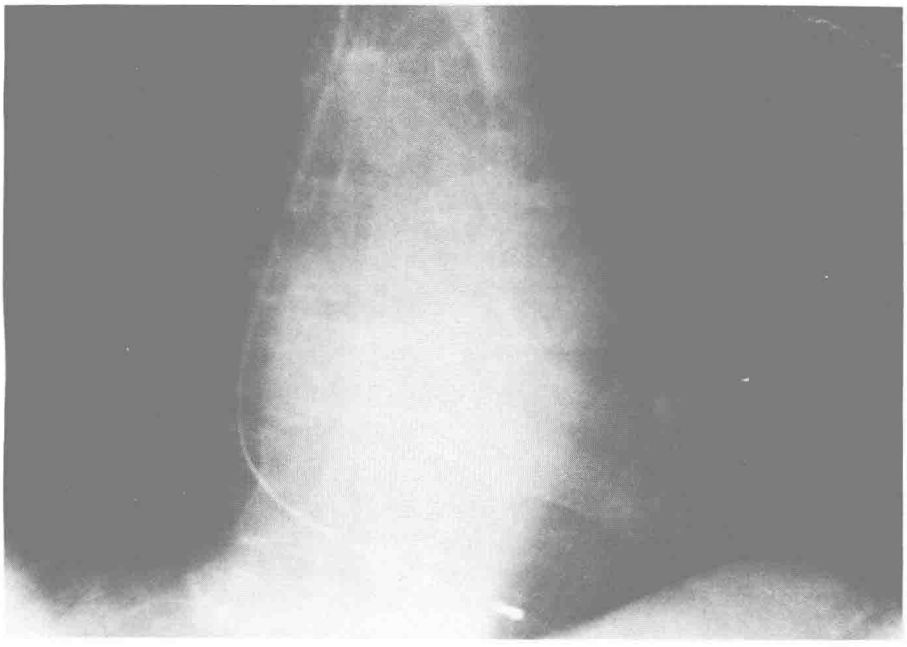

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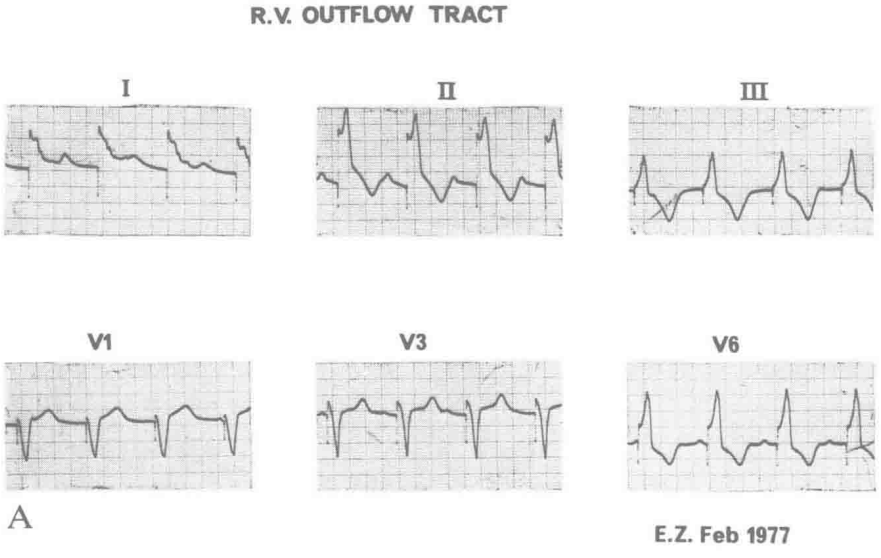
FIGURE 2 (*A*) Typical ECG appearance of a right ventricular endocardial lead with electrode at apex of right ventricle. There is left bundle branch block with a frontal plane axis markedly to the left. (*B*) Chest radiograph to show position of electrode at apex of right ventricle.



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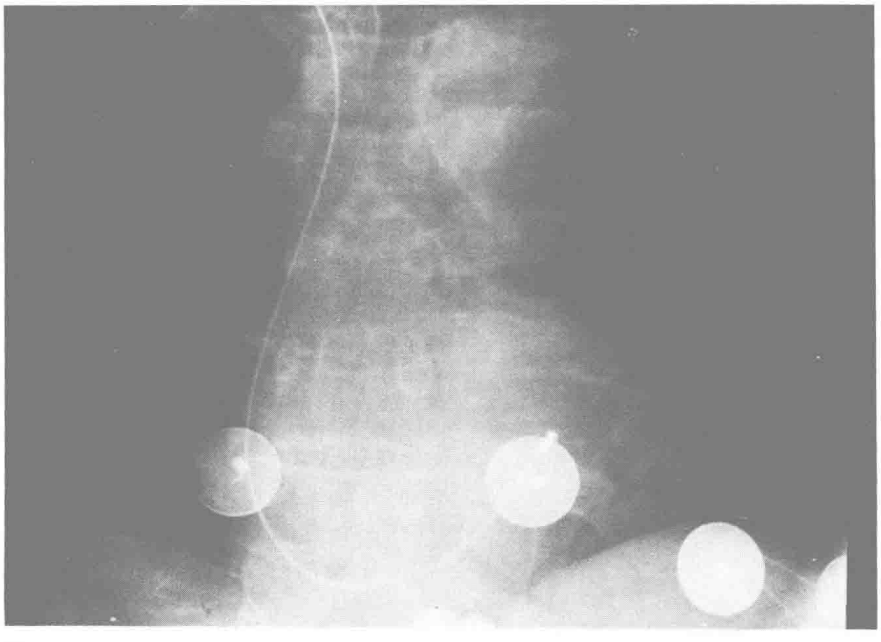

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FIGURE 3 (*A*) ECG appearance of endocardial electrode within the right ventricular outflow tract. There is left bundle branch block and a normal frontal plane axis. (*B*) Chest radiograph to show dislodgement toward the right ventricular outflow tract. Monitoring leads lie on the chest.

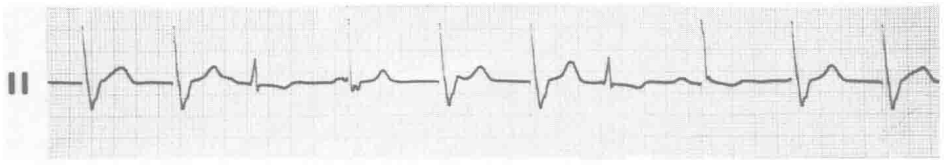

FIGURE 4 ECG, lead II. Ventricular-inhibited pacing with normal sensing. Complexes 1, 2, 5, 6, 9, and 10 are normal-paced beats. Complexes 3 and 7 are spontaneous ventricular complexes that successfully inhibit the pacemaker. Complexes 4 and 8 are fusion beats.

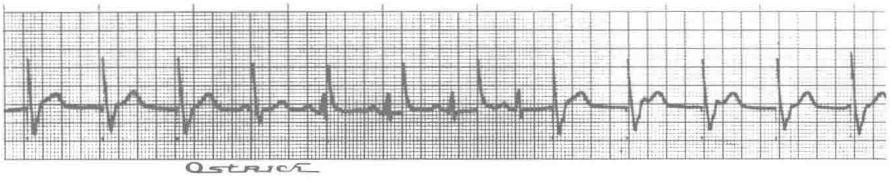

FIGURE 5 ECG, lead II. Asynchronous pacing. The first three and the last five complexes are normal-paced beats. In the middle of the tracing the stimulus artifacts continue at a regular repetition rate irrespective of the sinus beats. Stimulus artifacts that occur within the refractory period of the sinus QRS complexes obviously do not pace the heart. A similar appearance may occur when the sensing mechanism fails (undersensing) or a magnet is placed above the implanted pulse generator.

METHODS USED IN THE EVALUATION OF PACEMAKER FUNCTION

Routine Pacemaker Assessment

Routine pacemaker assessment involves an ECG together with electronic pulse-generator testing. Baseline levels are established and then patients are tested usually at 6 monthly intervals. Oscilloscopic display of the stimulus artifact together with fluoroscopic and radiographic chest examinations should also be carried out shortly after implant to provide baseline data.

ELECTROCARDIOGRAPH

A baseline 12-lead ECG during pacing is necessary postoperatively. At follow-up visits a rhythm strip to confirm pacing is performed. With ventricular-inhibited pacing systems, pacing may be inhibited by the patient's spontaneous rhythm. In this situation testing is performed by positioning a special magnet on the skin over the pulse generator. This activates a reed switch that dislocates the demand function and establishes asynchronous pacing. The stimulus artifact will now occur in competition with the spontaneous rhythm, and pacing will be evident where a QRS and T wave follow the stimulus artifact (Fig. 5).

ELECTRONIC PULSE-GENERATOR TESTING

The two parameters tested are the pulse repetition rate (interval between stimulus artifacts, which is measured in milliseconds or expressed in pulses per minute) and the pulse duration (width or time duration of the stimulus artifact). Both these parameters can now be measured using a variety of inexpensive testing devices. Testing is performed in the synchronous mode and then repeated in the asynchronous mode using a magnet applied over the pulse generator. The results are compared with previous recordings, and usually a fall of 6 to 8 beats per minute of pulse repetition rate and an increase in pulse duration represents impending power-source depletion. However, it must be stressed that each manufacturer has characteristic end-of-life pulse-generator indicators, and these should be known to the physician.

Methods Used in Pacemaker Faultfinding

Initial investigations include routine testing and a clinical examination. Usually, sufficient information is obtained from these simple investigations to diagnose the general cause of malfunction. Further investigations may be necessary. These include the following:

CLINICAL FINDINGS, INCLUDING HISTORY AND EXAMINATION

An unwell patient is examined clinically. Symptomatic patients, with normal routine testing, can often have their symptoms traced to a nonpacemaker cause, such

as vertebrobasilar insufficiency. However, should the patient history indicate a return of original symptoms, such as Stokes-Adams attacks, then the possibility of an intermittent-failed pacemaker system should be entertained.

OSCILLOSCOPIC EXAMINATION

Although still widely used, oscilloscopic examination of the stimulus artifact as a routine procedure in pacemaker clinics diminished with the introduction of simpler methods of measuring pulse duration and repetition rate. Despite this, oscilloscopic examination continues to be a very important tool in the investigation of pacemaker malfunction, especially with pulse-generator electronic faults and lead fracture.[1] Consequently, all personnel responsible for a pacemaker clinic must have a working knowledge of oscilloscopic testing and analysis. It is advisable that all patients undergo such testing soon after implant to obtain baseline documentation of the stimulus artifact so that comparison studies can be made at a later date if indicated (Fig. 6).

ECG MONITORING

In cases of suspected intermittent pacemaker system malfunction, prolonged ECG monitoring may be very valuable. Such systems include 24-h ambulatory monitoring, ECG telemetry, direct bedside monitoring, and telephone transmission of the ECG.

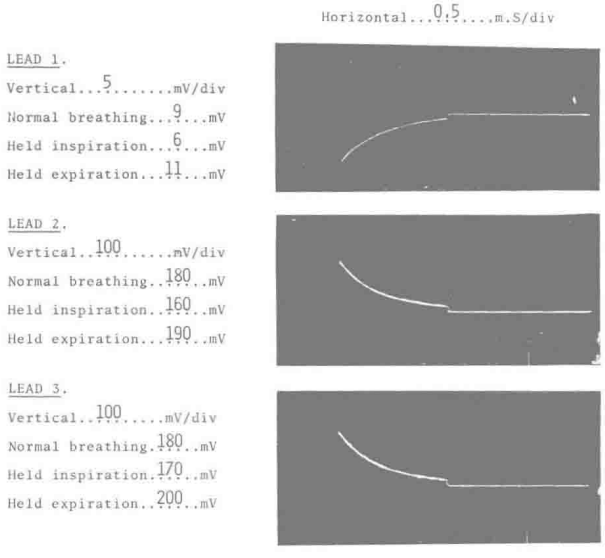

FIGURE 6 Oscilloscopic appearance of the stimulus artifact in leads I, II, and III. The vertical axis is the pulse amplitude and is measured in millivolts. There is a slight fall in voltage on inspiration and a rise on expiration. The horizontal time base is the pulse duration and is, in this case, about 1.75 m/s.

CHEST RADIOGRAPH AND FLUOROSCOPY

These are particularly important noninvasive tools used in the evaluation of suspected pacemaker malfunction. Originally, x-rays were used to judge the depletion of zinc-mercury batteries. Today, however, the chest radiograph and fluoroscopy are essential in determining lead position and possible lead fracture.

PROVOCATIVE MOVEMENTS

Specific movements likely to reproduce symptoms should be performed with ECG monitoring. These include rapid arm movements for skeletal muscle inhibition or unusual postures to demonstrate lead fracture. An intermittent lead fracture or poor connection between setscrew and lead pin can also be demonstrated by abruptly moving the pulse generator beneath the skin. Concomitant fluoroscopy or oscilloscopic analysis are also valuable during provocative movements.

CHEST-WALL STIMULATION

Electric stimuli too weak to affect the heart will inhibit an implanted ventricular-inhibited pulse generator, thus testing its sensing function (Fig. 7). These electric signals can be applied to the chest wall by an external pulse generator.[2,3] As demonstrated in Fig. 7, the technique of inhibiting a pulse generator will allow the physician to examine the patient's underlying, or intrinsic, cardiac rhythm. The technique is also useful in differentiating a pulse generator from a myocardial cause for lack of sensing (undersensing). Successful inhibition of the pulse generator by this technique will exclude a pulse-generator component failure.

SURGICAL EXPLORATION WITH PACING SYSTEM ANALYSIS AND FLUOROSCOPY

On occasion, surgical intervention is necessary as a means of investigating suspected pacemaker malfunction. Many cases of pacemaker malfunction determined by other means will also require surgical intervention. Whether a cause has been established or not, the procedures performed during surgical intervention remain essentially the same. Pulse generator and lead system are mobilized and inspected; both are tested using the pacing system analyzer. The lead system can be observed by fluoroscopy, and where necessary, tension is applied to the lead to demonstrate fractured ends. Any site where nonabsorbable suture material is tied around the lead should be very carefully inspected. Blood inside the lead insulator suggests an insulation break and a possible site for current leakage.

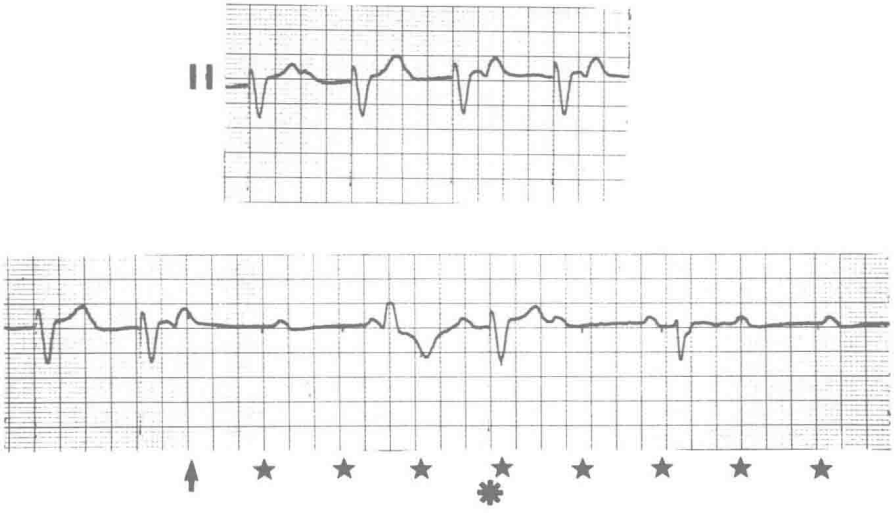

FIGURE 7 Inhibition of an implanted pulse generator by an external pulse generator with the electrodes placed over the anterior chest wall. In the top tracing, normal pacing is recorded prior to turning on the external pulse generator. In the bottom tracing, the external pulse generator is turned on (arrow) and the attenuated stimulus artifact (stars) represents the impulses from the external power source. The implanted pacemaker system assumes that the impulses are coming from the heart and is thus inhibited, revealing the underlying rhythm (complete heart block). However, one paced beat is seen during external inhibition (*). This occurs because a spontaneous QRS complex also inhibits the pulse generator and the next impulse from the external pulse generator occurs in the refractory period of the implanted pulse generator and is not sensed. A normal-paced beat occurs after the appropriate recycling time.

INTRACARDIAC ELECTROGRAPHY

The unipolar intracardiac electrogram is a simple means of recording electric potentials from the heart. Using a battery-operated ECG machine, the V lead is attached to the exposed lead pin of the implanted lead and a recording is taken. With a bipolar lead system, a bipolar recording can be obtained using lead I of the ECG. When the electrode makes contact with the right ventricular endocardium, "a current of injury" pattern characterized by ST elevation and T wave inversion occurs. In contrast, coronary sinus recordings have no such pattern. This current of injury disappears with time, leaving T wave inversion. In about 60 percent of cases, the unipolar ventricular electrogram is biphasic, with the R wave at least 10 percent of the S wave amplitude. In 30 percent of cases, the R wave is less than 10 percent of the S wave (Fig. 8). In the remaining 10 percent there is a dominant R wave without a discrete S wave.[4]

As well as being useful for lead placement, such electrograms are valuable for correct diagnosis of right ventricular perforation. Recordings are performed as the electrode is withdrawn from the epicardial surface.[5]

TESTING OF AN EXPLANTED PULSE GENERATOR

Prior to testing, the explanted pulse generator should be thoroughly washed in a detergent solution and then soaked in 10 percent formaldehyde. This procedure prevents transmission of any infective agent, such as hepatitis B virus.[6] Pulse-generator testing must be performed at body temperature using a water bath or an incubator. The characteristics examined should be compared with the initial test performed and those specified by the manufacturers. These tests are divided into three major categories.

Pulse-generator output This is tested at 37° under a 510-Ω load. Testing is performed with and without the magnet.

Oscilloscope Output pulse voltage shape and pulse duration. The leading- and trailing-edge voltages and pulse duration are recorded.

Rate Counter Pulse repetition rate.

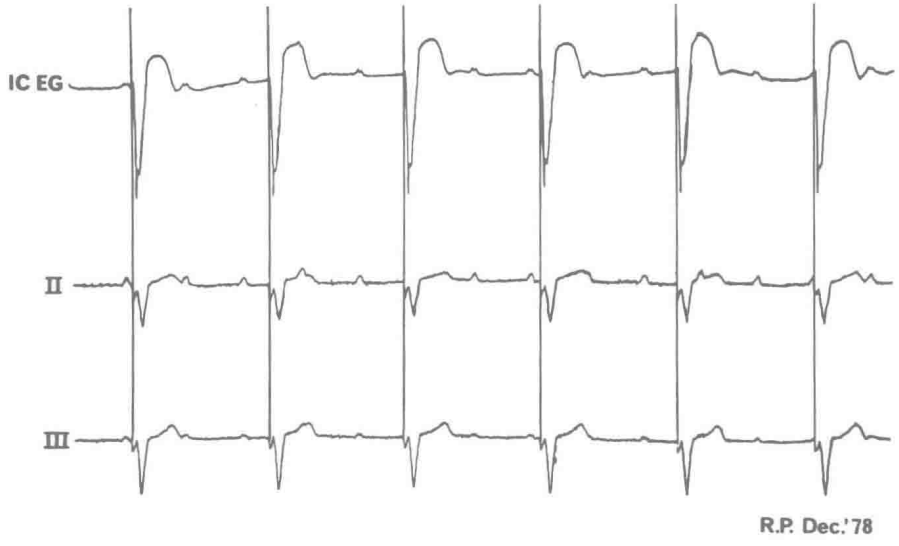

FIGURE 8 Unipolar endocardial electrogram (ICEG) and standard leads II and III of a normal pacing rhythm. The electrogram shows a deep S wave and a current of injury pattern.

Demand function This is performed using a simulated QRS complex having the characteristics of approximately 25 Hz and 4-mV amplitude. Tests include R wave sensitivity, refractory period, and interference frequency.

Missing-pulse detection If a pulse generator passes the preceding tests but there is doubt about performance over a long period, the unit should be tested specifically to identify intermittent failure of operation.[6]

CAUSES OF PACEMAKER MALFUNCTION

In devising a practical classification of pacemaker malfunction, the ECG appearances of pacing, both in the synchronous and asynchronous (magnet or test) modes, are used.

Three principles must be fulfilled to establish normal pacing:

1 A normal stimulus artifact is produced regularly at the preset rate in the asynchronous mode.

2 The stimulus artifact is followed by ventricular depolarization (QRS) and repolarization (T).

3 The pacemaker senses normally.

Using these principles, a flow diagram has been constructed (Fig. 9). The first part investigates the stimulus artifact, and thus the magnet must be applied. The second part investigates the QRS and T wave, and magnet application is not essential. The third part investigates demand function, and the magnet is not used. Within each section of the flow diagram there are a number of end points. One is "normal," indicating that the pacemaker malfunction under investigation is not within this section, since the basic ECG principle being examined has been fulfilled. The other end points represent a pacemaker malfunction group as identified by an ECG abnormality, and specific causes of each malfunction are tabulated (Tables 1 through 6). The tables are subdivided into true malfunctions and pseudomalfunctions. Pseudomalfunctions usually result from misinterpretation or faulty documentation of test data or from a poor understanding of the pacemaker system. Such errors may therefore be human or mechanical.

A second flow diagram (Fig. 10) investigates situations that are not strictly pacemaker malfunction but rather physical or psychologic side effects of pacing (Table 7). This flow diagram also classifies those problems unrelated to the pacemaker system and those where no diagnosis is made but the patient is symptomatic.

Stimulus Artifact

The first principle of normal function stated: "A normal stimulus artifact is produced regularly at the present rate in the asynchronous mode." Figure 9

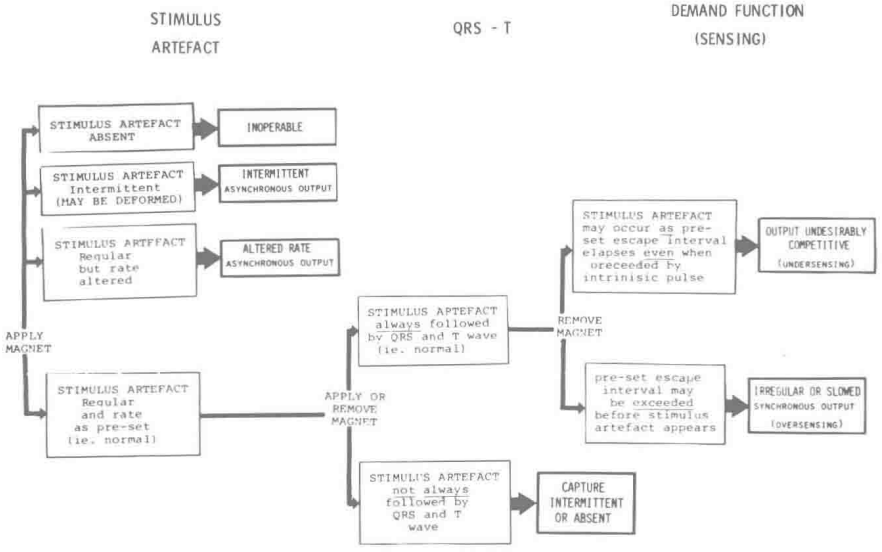

FIGURE 9 Flow diagram to demonstrate steps in faultfinding procedure. The stimulus artifact in the asynchronous mode is inspected, and three abnormalities are recognized: absent, intermittent, or altered rate. The QRS and T wave are then inspected to determine capture. Demand function is investigated in the synchronous mode, and two major abnormalities are recognized: undersensing and oversensing.

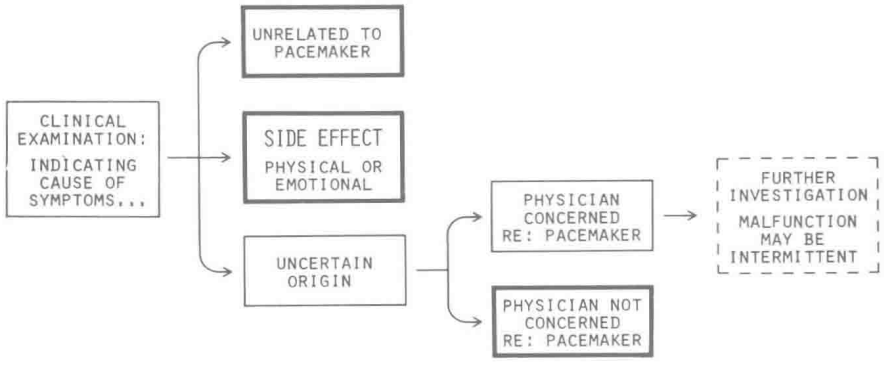

FIGURE 10 The clinical examination suggests that the patient's symptoms are unrelated to the pacemaker system, are due to side effects of the pacing system, or are of uncertain origin. The last group may need further investigation.

investigates the stimulus artifact in the asynchronous mode and identifies three abnormalities.

1 Stimulus artifact absent (inoperable pacemaker system).

2 Stimulus artifact intermittent (may be deformed).

3 Stimulus artifact present but at an altered rate.

STIMULUS ARTIFACT ABSENT—INOPERABLE PACEMAKER SYSTEM (Table 1)

There is no detectable stimulus artifact and thus no pulse-generator output in the asynchronous mode. There are four major true malfunctions: power-source failure, random component failure in the output circuit, a break at some point in the pacemaker-patient electric circuit, and a pacemaker-patient electric short circuit. Clarification of the actual malfunction may be difficult by routine noninvasive testing because the stimulus artifact is absent. Chest radiography and fluoroscopy are usually diagnostic for electrode-lead system fractures with complete discontinuity (Fig. 11). The treatment of all true malfunctions requires surgical intervention. Pseudomalfunctions are extremely important because surgical intervention is not indicated.