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Pacemakers

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Contents

LARGE NUMBERS OF PACEMAKERS of increasing complexity are being implanted each year, making it likely that the anesthesiologist will care for the pacemaker patient scheduled for general surgery. This review will discuss areas of preoperative and intraoperative care that are of concern to the anesthesiologist.

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Definitions

The following definitions are commonly used in the pacing literature and will be used throughout this review[1–3]:

1) *Pulse Generator.* This part of the pacemaker contains the energy source (battery) and electrical circuits for pacing and R- and/or P-wave sensing. It can be implanted or external.
2) *Lead.* This is the insulated wire connecting the electrode and the generator.
3) *Electrode.* The electrode is the exposed metal at the tip of the lead that is in contact with the myocardium.
4) *Unipolar Pacing.* All electrical circuits include positive and negative electrodes. A pacing circuit is no different. Rather than implying a circuit of one pole, unipolar pacing instead implies electrode location. It is accomplished by placing the negative (stimulating) electrode in the atrium or ventricle and the positive (ground) electrode distant from the heart. The ground is typically the metallic portion of the implanted pulse generator, or it can be a wire or metal plate sutured to skin or subcutaneous tissue.
5) *Bipolar Pacing.* In this system, both the positive and negative electrodes are located in the chamber that is being paced. Temporary ventricular pacing generally is bipolar (fig. 1). A temporary bipolar system can be easily converted into a unipolar system by connecting one of the electrodes on the bipolar catheter to the negative terminal of the pacemaker generator and connecting the positive terminal of the generator to an 18-gauge subcutaneous needle.
6) *Endocardial Pacing.* The atrium and ventricle can be stimulated electrically from the epicardium or endocardium. Endocardial pacing, also called transvenous, implies that the lead/electrode system has been passed through a vein to the right atrium or right ventricle. It can be unipolar or bipolar.

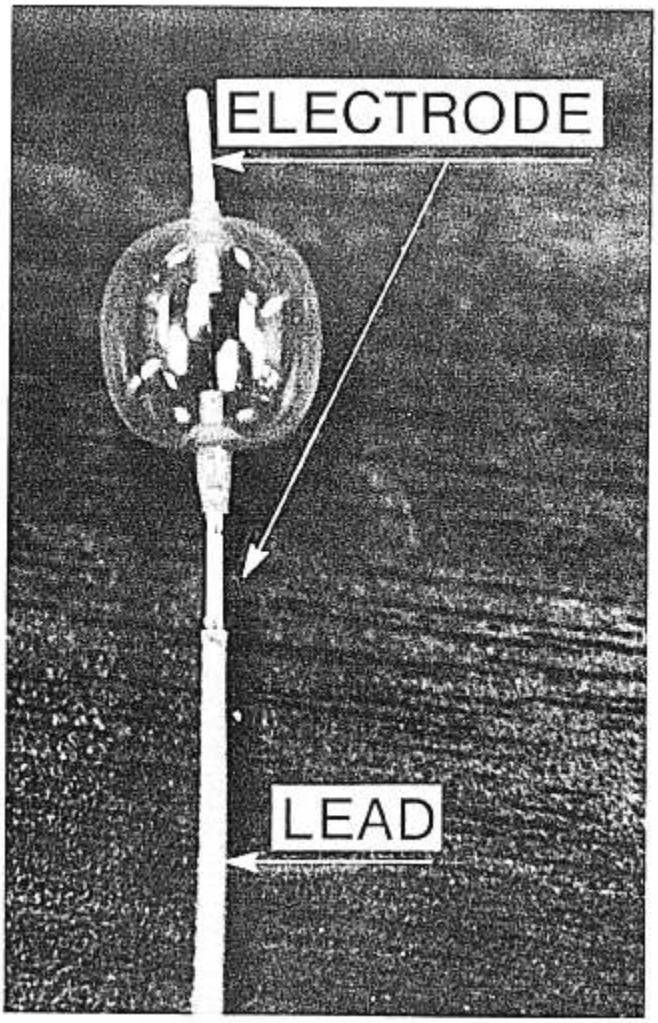

FIG. 1. This figure shows a bipolar transvenous (temporary) electrode catheter. Each one of these electrodes may be used individually to establish unipolar pacing. The inflated balloon is used to float the catheter into the right ventricle. (Reprinted from Zaidan JR: Pacemakers, Thoracic Anesthesia. Edited by Kaplan JA. New York, Churchill Livingstone, 1983, pp 575–595, with permission of author and publisher.)

7) *Epicardial Pacing.* This type of pacing is accomplished by inserting the electrode through the epicardium and into the myocardium, and actually should be called myocardial pacing. Epicardial pacing can be unipolar or bipolar and requires direct access to the heart through a subxiphoid or thoracotomy incision.
8) *Stimulation Threshold.* This is the minimal amount of current or voltage that will stimulate and therefore cause contraction of the chamber that is being paced. The estimates of current threshold (milliamps) and voltage threshold (volts) are made by a pacing system analyzer.
9) *Resistance.* After determining current and voltage thresholds, Ohm's law will provide a measure of the combined resistances of the electrode-myocardial tissue interface, electrode, and lead. The range of this total resistance should be 350 to 1,000 ohms, depending on the lead system.
10) *R-wave Sensitivity.* Intrinsic electrical impulses (R-waves) spread not only throughout the myocardium, but also in retrograde fashion into the electrode and lead back to the pacemaker generator. Once the generator senses an R-wave of sufficient voltage to activate a sensing circuit, the generator will turn off or, in some cases, actually trigger the pacing circuit. The voltage that is just sufficient to activate the sensing circuit to inhibit or trigger the pacing circuit is called the R-wave sensitivity of the generator. It is not a measure of the electrode's ability to transfer the R-wave to the generator, but rather is a measure of the number of millivolts required to activate the generator's sensing circuit. Permanent nonprogrammable pacemakers generally have an R-wave sensitivity of about 2 millivolts. Many programmable pacing systems allow changes to be made in R-wave sensitivity. External generators have a control that allows a choice of R-wave sensitivities. An R-wave sensitivity of approximately 3 millivolts on an external generator will maintain ventricular-inhibited pacing without interference from shivering, atrial depolarization, and ventricular repolarization.
11) *Hysteresis.* Hysteresis is built into many of the older generators and at least one of the new programmable pacemakers, and it occurs when there is a difference between the automatic and escape intervals of the pacemaker. The automatic interval is the number of milliseconds between continuously occurring pacing impulses. An automatic interval of 833 milliseconds corresponds to a pacing rate of 72 beats/minute. The escape interval is the number of milliseconds between a spontaneously occurring R-wave and the first pacing impulse after that R-wave. An escape interval of 1,000 milliseconds indicates that if more than 1,000 milliseconds occurs between the two R-waves, then a pacing impulse will be emitted from the generator. The difference of 167 milliseconds between the automatic and escape intervals of the generator means that the patient's intrinsic heart rate will decrease to 60 beats/minute (60,000/1,000 = 60 beats/minute) before the pacemaker starts pacing. However, once initiated, pacing continues at a rate of 72/minute. This is 12 beat

hysteresis and is normal function in some generators.

Types of Pacemaker Generators

Several different types of pacemaker generators are currently available.

Asynchronous

The circuit in this generator is responsible only for formation of electrical impulses.[4,5] It is the simplest form of generator and can be used safely in patients who have essentially no ventricular activity. Two problems are associated with the asynchronous pacemaker. First, it can compete with the patient's intrinsic conduction system for control of ventricular activation. If the myocardium surrounding a pacing electrode is repolarizing just when an impulse from the generator arrives, ventricular tachycardia could occur. This competition is especially dangerous in the ischemic heart which is being stimulated by the anode.[6,7] The second problem concerns energy conservation. The asynchronous pacemaker wastes energy because it continues to emit impulses even when the patient has a physiologic heart rate.[8,9] The permanent VOO generator is rarely, if ever, used.

Synchronous

A synchronous generator contains two circuits. One circuit is responsible for impulse formation, while the second acts as a sensing circuit. When activated by an intrinsic depolarization (R-wave), the sensing circuit either turns on (triggers) or turns off (inhibits) the pacing circuit. A generator that is inhibited by an R-wave is called an inhibited pacemaker, while a generator that is activated by an R-wave is a triggered pacemaker.

Triggered. This type of synchronous generator is used only in special circumstances. It eliminates the problem of competition, however, because the generator emits an impulse into the unresponsive refractory period of every R-wave; it wastes energy. For instance, if the patient's heart rate is 120/minute, the ventricular-triggered generator emits 120 impulses per minute.

Inhibited. This synchronous generator is turned off by R-waves and therefore eliminates competition while being energy sparing. It is the most common type of permanent pacemaker. Ventricular-inhibited pacing should be used in favor of asynchronous pacing.

Sequential

Sequential pacing is a special type of pacing that is used to preserve the atrioventricular contraction sequence. In critically ill patients who require pacing, sequential pacing will preserve the atrial contribution to preload and, compared with ventricular pacing, will cause a higher cardiac output for any given heart rate (see Cardiovascular Effects of Pacing).

Table 1. Definitions for 3-Letter Code

Letter 1	Chamber Paced
A	Atrium
V	Ventricle
D	Dual (both A and V)
Letter 2	Chamber Sensed
A	Atrium
V	Ventricle
D	Dual (both A and V)
O	Asynchronous, or does not apply
Letter 3	Response to Sensed Signal
I	Inhibition
T	Triggering
O	Asynchronous, or does not apply

Programmable

Pacemakers can be programmable as well as fixed function. While rate, output, and R-wave sensitivity are the most common programmable functions, generators can have programmed refractory periods, PR intervals, synchronous and asynchronous functions, and hysteresis.[9] Continuous and pulsed magnetic fields and radio frequency wave, are common methods of programming. Once this type of generator is programmed, it acts very much like a fixed function generator.

Nomenclature

Several systems of naming pacemaker generators have been developed. Each of these systems makes it possible to precisely describe generator function with minimal confusion. One system uses a three-letter code in which the first letter indicates the chamber that is paced, the second letter denotes the chamber in which sensing of intrinsic depolarizations takes place, and the third letter describes the mode of action of the generator.[10] The letters that are used in this code are listed in table 1. The generators listed below can be implanted or used in external form.

1) *VOO.* Pacing takes place in the ventricle. Since it does not have a sensing circuit, it is an asynchronous generator.

2) *AOO.* This system is basically the same as VOO pacing except that pacing occurs in the atrium.

3) *VVI.* Pacing and sensing activities occur in the ventricle. The generator will be inhibited each time the patient has a ventricular escape beat or a conducted sinus beat. This is the ventricular-inhibited generator described

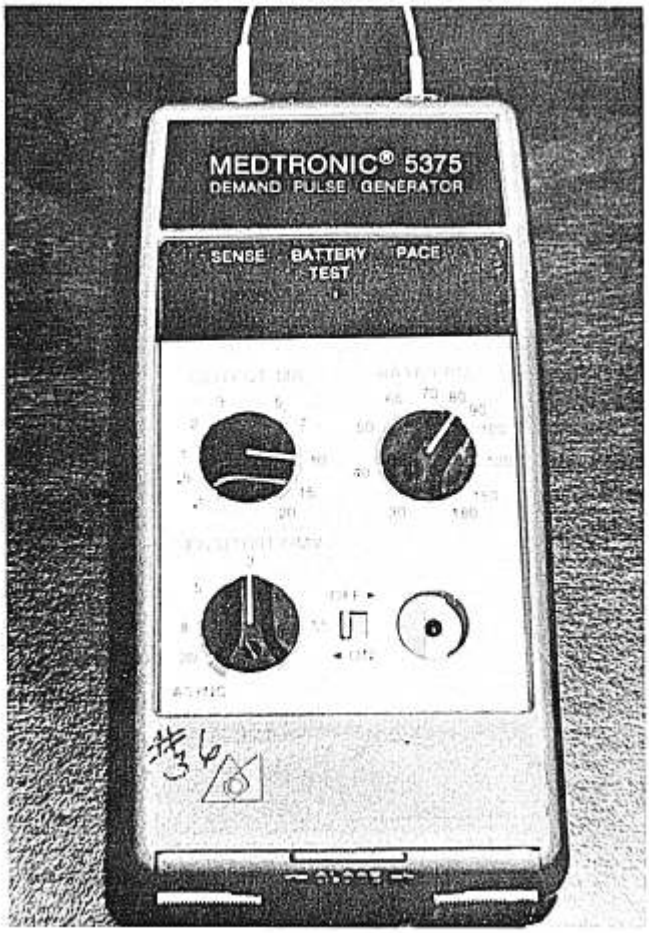

FIG. 2. A typical external pacemaker generator is shown above. The R-wave sensitivity dial controls the ease with which the generator is inhibited by intrinsic depolarizations. This generator therefore is either VOO or VVI depending on the R-wave sensitivity setting. As shown, it is a VVI with an R-wave sensitivity of 3 millivolts. (Reprinted from Zaidan JR: Pacemakers, Thoracic Anesthesia. Edited by Kaplan JA. New York, Churchill Livingstone, 1983, pp 575–595, with permission of author and publisher.)

in the section, Types of Pacemakers. On an external generator, changing the position of the R-wave sensitivity control from asynchronous to 3–5 millivolts converts the generator from a VOO to a VVI (fig. 2).

4) *DVI.* This pacemaker is used for A-V sequential pacing and implies that both the atrium and ventricle are paced. The DVI generator is designed to have both the atrial and ventricular outputs inhibited by R-waves (fig. 3). It is possible for this type of generator to atrially or A-V sequentially pace, depending upon the patient's heart rate and PR interval.

5) *DDD.* This implantable sequential pacemaker is fully automatic and therefore can sense P-waves and R-waves and pace the atrium and ventricle. If P-wave sensing takes place, the generator waits to sense an R-wave before emitting the ventricular pacing impulse. The DDD generator is not available in external form.

The three-letter code was extended to five letters to name the programmable generators. The third position of the five-letter code indicates the response of the generator to sensed R-wave and P-waves. Letter four describes the programmable functions of the generator while the final letter indicates tachyarrhythmia functions. Table 2 defines the letters.[11]

While other codes are used occasionally,[12] the three-letter and five-letter codes are the ones most commonly seen in the literature.

Patient Evaluation

As expected, the history, including allergies and medications, is an important aspect of the evaluation of the patient. Since 50% of these patients have coronary artery disease, 20% are hypertensive, and 10% are diabetic,[13] the list of medications could very possibly include cardiac glycosides, diuretics, β-blockers, antiarrhythmics, and antihypertensives. These drugs, in general, should be continued up to the time of surgery.

The patient should be questioned about the initial indications for pacemakers implantation and also about the return of pre-pacemaker symptoms including lightheadedness, dizziness, or fainting. If the patient states that these symptoms have recurred, then a cardiology consult is advisable.[14]

If the patient cannot answer questions concerning the pacemaker, then the identification card should reveal the name of the pacemaker manufacturer and possibly the type of generator. An X-ray of the generator also may reveal the manufacturer and model.

Myopotentials from skeletal muscles near a VVI generator could cause pacemaker inhibition.[15–17] The patient could possibly describe pre-pacemaker symptoms each time these muscles are exercised. Questioning the patient about this situation before surgery could help to avoid intraoperative problems (see Intraoperative Failure).

A general physical examination is strongly advised. Listen for bruits and evaluate for signs of congestive failure. Noting the location of the generator will help to determine the location of the electrodes. Generally, epicardial electrodes have the generator in the abdominal wall, while endocardial electrodes have the generator over one of the pectoralis muscles.

The laboratory examination is also an important aspect of the evaluation of the patient. A chest X-ray is important as a means of evaluating lead continuity (fig. 4). Knowledge of the course of the lead will allow the surgeon to avoid it during surgery.

Acute potassium imbalance can be associated with loss of pacing or with ventricular tachycardia and will be discussed in detail in the section, Intraoperative Failure. Preoperatively, a stable serum K^+ of 3.5 to 5.0 mEq/l is

adequate. Proceeding with totally elective surgery in the presence of a low serum potassium could lead to serious intraoperative complications. If the patient is hypokalemic and requires emergency surgery, one should proceed with extreme caution.

The usual guidelines for hematocrit should be followed. The presence of a pacemaker is not an indication for a transfusion. For instance, if a pacemaker patient with chronic renal failure has a stable Hct of 25, a transfusion is not necessary.

The laboratory studies listed above are the minimal requirements, and other determinations should be performed as indicated on an individual basis to complete the work-up.

Pacemaker Evaluation

Evaluation of the pacemaker, although important, is secondary to preoperative patient evaluation. One can begin the pacemaker evaluation during the interview simply by noting the rate and regularity of a peripheral pulse. A regular pulse at 70–72 beats per minute indicates that a VOO or uninhibited VVI generator is pacing the heart. A regular rate greater than 70–72 could mean that the patient has a functioning but inhibited VVI pacemaker, but it could imply a nonfunctioning VVI or VOO pacemaker. An irregular pulse is secondary to atrial fibrillation, atrial flutter with varying block, premature ventricular or atrial contractions, a competing VOO generator, or a VVI generator that is not appropriately sensing the R-waves. A programmable pacemaker will act as though it is a VVI generator when being evaluated, unless it is programmed to be a VOO. This would be a very rare situation.

The electrocardiogram must be evaluated in the pacemaker patient to determine one-to-one capture and to help discover the type of generator. One-to-one capture of the pacemaker is tested by monitoring the electrocardiogram while simultaneously palpating a peripheral pulse. Each paced beat should correspond to a pulse. Problems with this evaluation occur when the patient has a VVI generator and a heart rate greater than the pacemaker rate. In this situation, pacemaker impulses will not appear on the ECG. Carotid massage used to slow the heart rate and turn on a VVI generator could result in an arteriosclerotic plaque embolizing to the cerebral circulation. A valsalva maneuver should be used in place of carotid massage. Although it is not contraindicated, application of the magnet to the generator should be avoided if the patient is experiencing angina. It is difficult to circumvent the problem of evaluating one-to-one capture in the presence of an inhibited VVI generator. As a general rule, sensing capabilities are lost before pacing, so that if the generator is less than two years old, the chest X-ray shows no breaks in the lead, and no pacing impulses appear on the electrocardiogram, then the generator probably is working correctly.

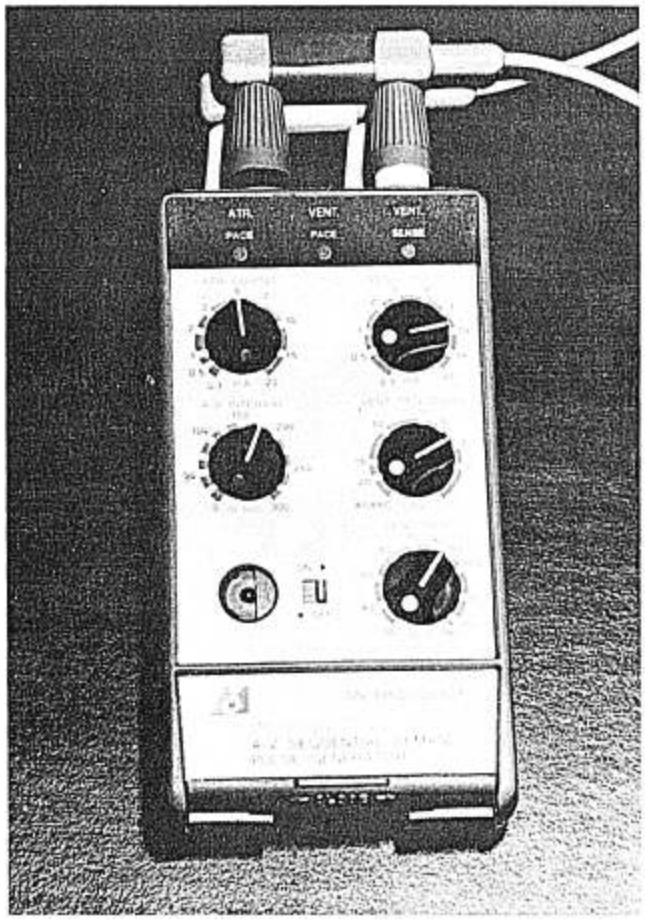

FIG. 3. This DVI generator is used for AV sequential pacing. A second output sends current to the atrium. The PR or AV interval also has a separate control. A PR interval of 175 ms is a reasonable starting point. (Reprinted from Zaidan JR: Pacemakers, Thoracic Anesthesia. Edited by Kaplan JA. New York, Churchill Livingstone, 1983, pp 575–595, with permission of author and publisher.)

TABLE 2. Definitions for 5-Letter Code

Letter 1	See table 1
Letter 2	See table 1
Letter 3	Responses to Sensed Signal
T	Triggered
I	Inhibited
O	Asynchronous
D	Dual (Triggering and Inhibition)
R	Reverse functions (Activation of the generator by rapid heart rates rather than slow heart rates)
Letter 4	Programming
O	No programming
P	Programming only for output and/or rate
M	Multiprogrammable
Letter 5	Tachyarrhythmia Functions

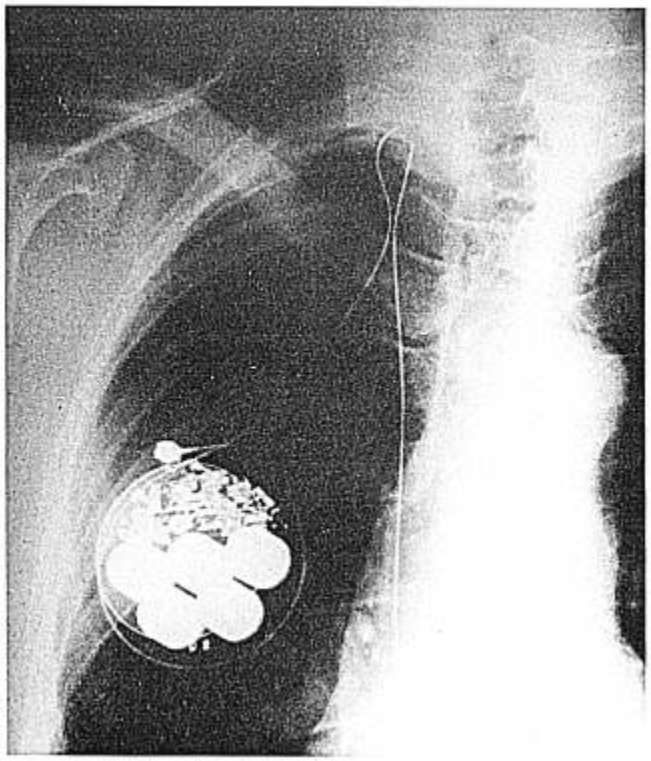

FIG. 4. This X-ray reveals a broken lead located at the acute turn above the clavicle. It is easy to miss a lead fracture such as this one when the patient has a heart rate greater than the pacemaker rate and has a VVI generator. (Reprinted from Mansour MK: Complications of cardiac pacemakers. Am Surg 4:132, 1977, with permission of author and publisher.)

Specific questions that help to evaluate pacemaker function are outlined in table 3.

Intraoperative Management

MONITORS

The selection of monitoring should be based on the patient's underlying disease. The presence of a pacemaker is not an indication for sophisticated monitoring such as a pulmonary artery catheter. If, in the medical judgement of the anesthesiologist, the patient will benefit from the knowledge gained by using a pulmonary artery catheter, then its use should not be avoided. Passing a pulmonary artery catheter through the ventricle will not disturb epicardial electrodes. Although it has not been investigated formally, it is assumed that a pulmonary artery catheter could easily dislodge a freshly placed transvenous (endocardial) electrode. Dislodgement, in our experience, has not occurred when the endocardial electrode has been in place for over four weeks. If the decision is made to insert a pulmonary artery catheter in the presence of a new endocardial electrode, then consider using a Multipurpose (pacing) Swan Ganz® catheter. The use of a central venous pressure monitor should not present a major problem.

ANESTHETIC TECHNIQUES

As with monitoring, the anesthetic technique should be based on the patient's underlying disease. Regional anesthesia represents a reasonable choice of techniques. General anesthesia also is not contraindicated and both narcotic-relaxant and inhalational techniques have been used successfully. Pacing thresholds of acutely placed electrodes recently were investigated during halothane, enflurane, and isoflurane anesthesia.[18] These anesthetic agents were found not to significantly alter the current and voltage thresholds in acutely placed electrodes.

Intraoperative Loss of Pacing

The pacemaker that is functioning normally preoperatively should continue to function intraoperatively without incident. However, several intraoperative situations can occur that are known to alter thresholds or inhibit pacemaker generators.

ELECTROCAUTERY

A VOO or a bipolar VVI generator should not be inhibited by conducted electromagnetic interference (EMI). Older VVI generators, especially if they are unipolar, are inhibited by the electrocautery.[19]

Electromagnetic interference used in the presence of a VVI generator can cause inhibition, reversion into asynchronous pacing, or a power-up sequence. The electrical artifact created by the electrocautery constantly changes amplitude and tends to appear to the pacemaker generator like a muscle myopotential. If the cautery artifact that is picked up by the lead exceeds the R-wave sensitivity of the generator, then the generator will be inhibited. Most VVI pacemakers can be inhibited for one impulse during the process of reversion to VOO pacing; therefore, if the surgeon cauterizes multiple bleeding sites within a short period of time, the generator could be inhibited enough to decrease the heart rate, lower cardiac output, and decrease cerebral and myocardial blood flow. If the electrocautery artifact surpasses the R-wave sensitivity of the generator for a longer period of time, the generator reverts into asynchronous pacing as long as the cautery is being used. In these two situations, when the surgeon stops using the electrocautery, the pulse generator should return to normal function.

A power-up sequence can occur in some generators in response to an EMI. Excessive current from the EMI enters the pacemaker battery and, because of the internal

TABLE 3. Considerations for Pacemaker Evaluation

Question	Comment
1. When was the generator implanted?	The life expectancy of a lithium-powered pacing generator is at least 5 years.
2. What was the factory present rate?	As the battery loses energy over time, the rate drops in stepwise fashion in some generators. A pacemaker rte of 10% below the preset rate is an indication for generator replacement.
3. What type of generator was implanted?	A VVI generator should have no pacing spikes on the ECG in the presence of a physiologic heart rate. Competition commonly occurs with a VOO.
4. What type of electrodes were implanted?	The generator is commonly implanted in the abdominal wall for epicardial electrodes and in the pectoral area for endocardial electrodes. Impliications concern placement of a pulmonary artery catheter.
5. Does the patient have symptoms of decreased cerebral perfusion when exercising muscles around the generator?	Defasciculation or avoidance of succinylcholine is suggested.

resistance of the battery, actually causes the theoretical maximal voltage output of the battery to decrease. When this reduced voltage reaches the pacing circuit, the response is to decrease the rate of impulse formation. Once the excessive current is terminated, voltage output generally increases to normal and the pacemaker rate increases to the factory set rate. A surge of current in an older battery that is operating at reduced voltage can reduce the voltage output to extremely low levels. If battery output decreases to approximately 1.8 volts in some Medtronic pacemakers, then removal of the EMI is not always associated with a return to normal rate of impulse formation, even though voltage output returns to its pre-EMI state. The clinical result is that the pacing rate will falsely reveal an end-of-life pattern in the presence of normal voltage output after the EMI is removed.[20] If this situation occurs, it can be treated by applying the magnet over the generator. After removing the magnet, the generator should resume normal pacing rate in the VVI mode. Ventricular-inhibited generators by Intermedics also show inhibition or reversion to the VOO mode of activity in the presence of EMI. When EMI is removed, they should return to VVI activity without application of the magnet.[21]

Applying a magnet to a VVI converts it to a VOO, almost eliminating the possibility of inhibition by the electrocautery. Since the electrocautery distorts the electrocardiogram, it is impossible to determine if pacemaker inhibition has occurred unless the anesthesiologist is palpating a peripheral pulse or observing a radial artery tracing.

Programmable pacemakers react to conducted electromagnetic interference in several ways. Most probably, the pacemaker will maintain its program. However, since an electrocautery causes surges in current and decreases in battery voltage, the generator could respond by decreasing the pacing rate. It is extremely unlikely that this situation will develop, and it should not present a clinical problem. Once programming has been performed, programmable pacemakers act very much like the more simple VVI or VOO generators. Unlike a standard VVI generator, however, application of the magnet to some programmable pacemakers should be avoided during electrocautery use because the chance for reprogramming increases.[22] If alterations in the program caused by the electrocautery occur, the new program will be unpredictable. These generators can be reprogrammed easily.

For all types of generators, Simon has recommended that: 1) the electrocautery be used in short bursts; 2) the electrocautery ground plate be as remote as possible from the generator to minimize detection of the current by the generator; and 3) the electrocautery current be as low as possible.[23] It also is suggested that the electrocautery ground not be located between the active electrode and the pacing generator.

Electrocautery has been shown to affect more than the generator itself. An investigation by Geddes *et al.*[24] has shown that disconnecting the indifferent electrode from the cautery will cause an endocardial pacemaker electrode to become an active electrode in the cautery circuit and create myocardial burns and ventricular fibrillation. Electrocautery has been reported to cause ventricular fibrillation even in the absence of a pacemaker electrode.[25]

POTASSIUM BALANCE

Potassium equilibrium across the cell membrane determines the resting membrane potential (RMP). It is

TABLE 4. Causes of Acute Changes in K_O

Acute Change in K_O	Cause	Consequence
1. Increase	1. Rapid replacement therapy in a chronically hypokalemic patient 2. Myocardial ischemia 3. Depolarizing muscle relaxants in patients with neuromuscular disease, burns, or major trauma.	Possibility of ventricular tachycardia with VOO pacing or with nonsensing VVI pacing.
2. Decrease	1. Hyperventilation 2. Intraoperative diuretic therapy with excessive urine output. Neurosurgical patients are at a greater risk than other patients because they undergo hyperventilation and diuretic therapy.	Possible loss of pacing

described by the Nernst equation.[26] In very simplified form, the Nernst equation is:

$$RMP = -62 \log \frac{[K]_I}{[K]_O}$$

where $[K]_I$ indicates potassium concentration inside the cell and $[K]_O$ the potassium concentration outside the cell. Potassium concentration inside the resting cell is 150 mEq/l, while $[K]_O$ = 5 mEq/l, so that the RMP becomes $-62 \log 30 = -90$ mv. A pacing impulse depolarizes the membrane, and creates an action potential that eventually causes myocardial contraction. Clinical situations causing the RMP to become less negative and approach the membrane's threshold potential will require less current density at the electrode-tissue interface to initiate an action potential, making capture by the pacemaker easier. If the RMP becomes more negative, an increased current density would be required to raise the RMP to the membrane's threshold potential, therefore making it more difficult for the pacemaker to initiate myocardial contraction.

An acute increase in $[K]_O$ causes a less negative RMP. In this situation, the pacemaker continues to pace. However, the occurrence of ventricular tachycardia is a real possibility if the pacing impulse is emitted into repolarizing myocardial tissue. An acute decrease in $[K]_O$ leads to loss of pacing. Acute changes in potassium concentration therefore are extremely important. Table 4 outlines the causes of acute changes in potassium concentration and their clinical consequences.

Myocardial Infarction

Scar tissue is unresponsive to electrical stimulation; therefore, myocardial infarction causes loss of pacemaker capture if the electrode is centered in the infarcted area. This response is unlike an electrode being located in an ischemic area.

Myopotential Inhibition

Occasionally, a generator can be inhibited by myopotentials. These myopotentials have a variety of causes including exercise, shivering, and muscle fasciculations secondary to succinylcholine. It is suggested that a defasciculating dose of a nondepolarizing relaxant be used or succinylcholine be avoided. Also, measures taken to avoid intraoperative hypothermia could eliminate postoperative shivering-related pacemaker inhibition.

Indications for Pacing

Many times the decision to insert a permanent pacemaker is based on medical judgment; however, guidelines to follow when determining which patient requires pacing has been outlined recently by the Department of Health and Human Services Health Care Financing Administration.[27]

Indications for Permanent Pacing

Sick sinus syndrome is one indication for permanent pacing. It is characterized by recurrent supraventricular tachycardia alternating with sinoatrial arrest and sinus bradycardia. Pacing is indicated if the patient is symptomatic to the bradycardic episodes, or if β-blockers used to control the tachycardia result in symptomatic sinus bradycardia. Electrophysiologic studies showing a corrected sinus node recovery time greater than 525 milliseconds or an insufficient response to intravenous atropine will help in the decision to implant a permanent pacemaker.[28] Other indications for permanent pacing are second degree Mobitz type II heart block and extreme sinus bradycardia in symptomatic patients.[29,30]

Pacing for bifascicular block is controversial. The question in this situation concerns the number of lives that could be saved each year if a pacemaker was im-

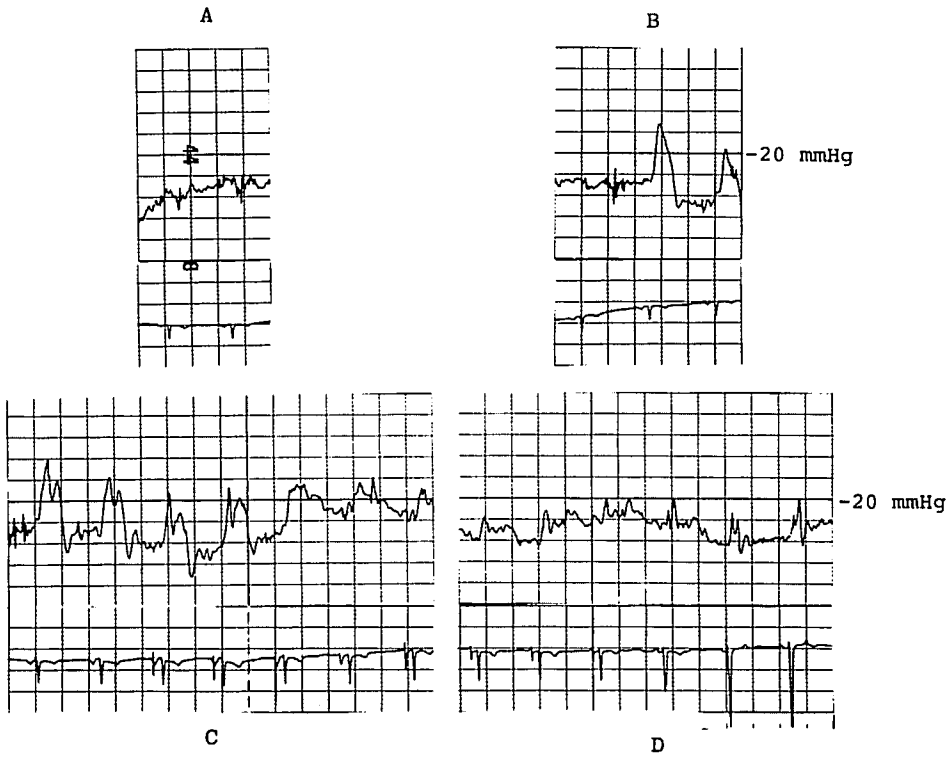

FIG. 5. This figure demonstrates the advancement of the distal ventricular electrode through the heart while monitoring pressures at the distal port on the pulmonary artery catheter. The electrocardiogram is intracavitary and is recorded through the distal ventricular electrode. (*A*) Catheter tip in right atrium and distal electrode in the superior vena cava. (*B*) Catheter tip enters right ventricle. The electrode has not yet entered the atrium. (*C*) Pressure monitoring reveals entrance of the catheter into the pulmonary artery. The distal electrode enters the atrium at the third complex. (*D*) The wedge position is obtained by the third beat. Slight advancement of the catheter pushes the distal electrode past the tricuspid valve (4th beat) and into the right ventricle (5th beat).

planted in every asymptomatic patient who has bifascicular block. Multiple retrospective and prospective studies have shown that a number of deaths will occur in patients with bifascicular block.[31–35] The cause of death could have been related to the development either of ventricular arrhythmias or of third-degree block. Unfortunately, since these studies could not always document the cause of death, the mortality rate associated with bifascicular block remains unknown. Denes *et al.*,[36] McAnulty *et al.*,[37] and Kulbertus *et al.*,[38] have suggested that HV intervals measured by electrophysiologic testing do not help to predict which patient with bifascicular block will develop third-degree heart block. Other investigators, however, have recommended permanent pacing in asymptomatic patients with prolonged HV intervals.[34,39,40] Even though the problem remains unsettled, cardiologists generally do not recommend permanently pacing an asymptomatic patient with bifascicular block.[37]

Acquired complete heart block is another indication for permanent pacing, especially if the patient is symptomatic.[41–44] The block could occur in the AV node, the bundle of His, or in the bundle branches, and generally is secondary to coronary artery disease, calcification,[45] or degeneration[46] of the conduction system.

Ventricular[47–50] or atrial pacing[51,52] can be used to suppress the development of ventricular arrhythmias. Less commonly, atrial and ventricular permanent pacing is used to suppress atrial tachyarrhythmias that are initiated by bradycardia.[53–56]

Permanent pacing is effective in terminating re-entrant atrial tachyarrhythmias.[57–60] One appropriately timed atrial impulse will block the re-entrant pathway and break the dysrhythmia. Since it is impossible to predict the timing of this single stimulus, rapid atrial pacing at rates 120% of the intrinsic atrial rate should be used. Once capture has occurred, the pacing generator may be slowed gradually or stopped abruptly.[61] Ventricular pacing at rates sufficient to provide ventricular capture can be used to terminate ventricular tachycardia.[62–66]

Recently, recurrent ventricular fibrillation has been treated successfully with a permanently implanted automatic defibrillator.[67–70] This implantable "pacemaker" delivers 25 joules per defibrillation with enough stored energy for 100 defibrillations. The anesthetic management for defibrillator implantation has not been described.

INDICATIONS FOR TEMPORARY PACING

Patients scheduled for permanent epicardial electrode placement who have a physiologic ventricular rate at the time of induction of anesthesia do not need temporary

pacing. If this patient were in third-degree block, then a temporary electrode should be positioned before surgery.

Inadequate medical control of supraventricular dysrhythmias in patients who are scheduled for emergency surgery is another indication for temporary pacing. This rare situation requires the use of an external rapid atrial pacing generator and an atrial electrode.

Bradycardia associated with drug therapy or drug toxicity is an indication for temporary pacing. For example, consider placing pacing electrodes in a patient with a heart rate ≤ 35 taking a β-blocking drug. A trained athlete with the same heart rate, however, does not require pacing.

It is reported that 18% to 29% of patients with acute myocardial infarctions develop ventricular conduction blocks.[71,72] Generally, a second-degree Mobitz type I block associated with an inferior myocardial infarction does not require temporary pacing.[73] An anterior myocardial infarction, however, is known to cause second-degree Mobitz type II block that can develop rapidly into complete block. This patient requires temporary pacing.[73] Unfortunately, pacing for heart block developing after an acute myocardial infarction does not decrease the reported 60–70% in-hospital mortality rate.[74–76] If a patient with an acute myocardial infarction and second- or third-degree AV block is scheduled for emergency surgery, then pacing is indicated. Although a cardiologist most likely would not establish pacing for a second-degree Mobitz I block, an anesthesiologist should at least consider temporary pacing to avoid the severe hemodynamic effects of third-degree block should they develop during surgery.

Bifascicular block without first-degree block in an asymptomatic patient is not sufficient reason to insert a prophylactic temporary pacemaker. However, if the patient has a history of dizziness or fainting spells either with or without first-degree block, then a cardiology consult is indicated.

Berg and Kotler studied the occurrence of complete heart block in surgical patients with preoperative bifascicular block.[77] Of the 36 procedures recorded in the study, 18 were preformed with general, seven with spinal, and 11 with local anesthesia. The authors suggested that patients with fascicular blocks do not require prophylactic temporary pacing since no intra- or postoperative complete heart blocks occurred.

Investigations by Atlee[78,79] have shown that enflurane and halothane prolong AH intervals and halothane prolongs HV intervals. Since it is unknown if these agents further prolong conduction in patients with bifascicular blocks, they should be used with caution in these patients.

Consider establishing preoperative temporary pacing in the rare situation in which a comatose patient has a bifascicular block. A transient, third-degree block could have decreased cerebral blood flow enough to result in a head injury and coma.

Cardiovascular Effects of Pacing

One determinant of cardiac output is heart rate. Any means of increasing the heart rate from bradycardic levels will have a positive effect on cardiac output in the presence of normal contractility. Even though ventricular pacing is the most common method of establishing a physiologic rate, it does not always result in the highest possible cardiac output for a given pacing rate.

Atrial systole increases the preload, therefore increasing the cardiac output above that obtained with equal rates of ventricular pacing. The hemodynamic responses to atrial pacing, however, are dependent upon the adequacy of the coronary circulation. Patients with normal coronary arteries are reported to have declines in ventricular volumes with no significant alteration in ejection fraction.[80] Tzivoni *et al.*[81] also found no significant changes in the ejection fraction with atrial pacing. In neither of the above studies did atrial pacing cause significant alterations in cardiac output, systemic vascular resistance, or arterial blood pressure.

Patients with coronary artery disease can have different responses to atrial pacing. End diastolic pressure has been reported to increase in association with decreased ejection fraction.[82–84] Regional wall motion abnormalities appeared with the development of ischemia.[85] Ricci *et al.* found that these wall motion abnormalities persisted even into the post-pacing period.[86] In a study by Arbogast,[87] atrial pacing rates of 140 caused decreases in cardiac output in patients with coronary artery disease, indicating that cardiac output determinations must be performed to establish the most effective pacing rate.

It is well-documented that atrial contraction significantly increases preload[88–91]; However, the influence of atrial contraction of preload diminishes with increased left ventricular end diastolic pressures (LVEDP). Greenberg *et al.*[92] studied the effect of changing LVEDP on the atrial contribution to preload by measuring stroke volume index while changing intravascular volume. Patients with LVEDPs less than 20 mmHg had significantly greater atrial contributions to stroke volume index than those patients with LVEDPs greater than 20 mmHg.

The mere presence of atrial contraction is not the only factor affecting cardiac output during atrial pacing. Another aspect of atrial pacing that is important in determining cardiac output is the temporal relationship between atrial and ventricular contractions. Samet *et al.*[93] found that in normal hearts, sequential pacing was he-

modynamically superior to ventricular pacing but not different from atrial pacing. A postoperative study of cardiac surgical patients by Hartzler *et al.*[94] revealed that atrial pacing caused significant increases in cardiac output compared with ventricular pacing. In Hartzler's study, cardiac output increased even further in several of the patients when they were A-V sequentially paced. Similar cardiovascular responses were found in response to sequential pacing in patients with third-degree heart block both with[95] and without acute myocardial infarctions.[96]

Sequential pacing provided augmentation of cardiac output above that found during ventricular and atrial pacing alone in a study by Curtis *et al.*[97] In this study, sequential pacing resulted in a 35% increase in cardiac output compared with ventricular pacing, and in one patient the cardiac output increased 87%. Sequential pacing created an increase in cardiac output of only 5% over that found with atrial pacing. Shemin *et al.*[98] noted that compared to atrial pacing, ventricular pacing after corrective surgery for idiopathic hypertrophic subaortic stenosis was associated with significant decreases in cardiac output and mean arterial blood pressure and an increase in pulmonary capillary wedge pressure.

Ventricular stimulation can be associated with a low cardiac output for reasons other than the loss of an appropriately timed atrial contraction. Hilton[99] studied the metabolic effects of atrial and ventricular pacing after coronary artery bypass grafting in 19 patients. Coronary sinus blood samples indicated a change from lactate extraction to lactate production when changing from a sinus mechanism to ventricular pacing at a rate of 120. A similar rend was found with atrial pacing, but these changes were not statistically significant.

Several investigators have suggested that the abnormal myocardial activation sequence associated with ventricular pacing is of minor importance in the determination of cardiac output.[93,100] However, other investigators disagree. Daggett[101] found that both myocardial activation sequence and PR interval were important determinants. Badke *et al.*,[102] in a study of regional left ventricular performance in anesthetized dogs, showed that the left ventricle contracted asynchronously during ventricular pacing. Intraventricular volume, therefore, was shifted toward non-contracting areas of the myocardium, resulting in less volume being ejected through the left ventricular outflow tract for a given LVEDP. Wiggers[103] studied ventricular performance by measuring intraventricular pressures during ventricular pacing and found that the isovolumic phase of ejection was prolonged and ventricular ejection time was decreased when compared with normal ventricular activation. Gilmore[104] also studied left ventricular function during ventricular pacing and concluded that abnormal ventricular depolarization resulted in decreased left ventricular function. In an elaborate epicardial mapping study in dogs, Lister *et al.*concluded that different sites of left ventricular and right ventricular activation resulted in significant differences in cardiac output.[105] Ventricular pacing initiated at sites toward the base of the heart, especially at the lateral aspects of each ventricle, resulted in the lowest cardiac outputs.

It is clear, therefore, that ventricular pacing is not the best method of pacing in the acute situation in which control of cardiac output is crucial. In these situations, ventricular pacing is a life-saving maneuver which is adequate until atrial or sequential pacing can be established. It was suggested recently that atrial pacing should be used as the first choice; however, if atrial pacing is inappropriate, such as in the presence of third-degree heart block, A-V sequential pacing should be used.[106]

The Multipurpose Swan Ganz catheter can be used if simultaneous pressure monitoring and pacing capabilities are required.[107] This catheter recently was shown to withstand the manipulations of cardiac surgery and continue to function in the post-cardiopulmonary bypass period.[108] Positioning of this catheter did not require fluoroscopy and the pacing thresholds were well within the limits of an external generator. Figure 5 shows an intracavitary electrogram created by the distal ventricular electrode as it passed through the atrium and into the ventricle. Even though this catheter performs reasonably well in anesthetized patients, awake patients present different problems. Spontaneous ventilation with sighing and coughing, and movement of the head and neck could possibly move the catheter enough to lose pacing.

Electrocardiograms Associated with Pacing

This section shows several typical electrocardiograms associated with pacemakers.

Normal unipolar atrial pacing is demonstrated in figure 6. After each atrial pacing impulse is an atrial wave (P-wave) that assures electrical depolarization but not necessarily atrial contraction. The third beat is a nodal escape beat that has pacemaker impulse in the T-wave. This impulse is not likely to cause ventricular fibrillation since it is occurring in the atrium. However, if it is conducted to the ventricle, it could cause ventricular fibrillation by stimulating in the repolarization phase.

Bipolar ventricular pacing is seen in figure 7. The pacing impulses are very small and contrast with the impulses in figure 6, which are unipolar pacing.

Figure 8 reveals unipolar atrial pacing with a very prolonged PR interval. The only method available to assure capture is to observe the radial arterial and electrocar-

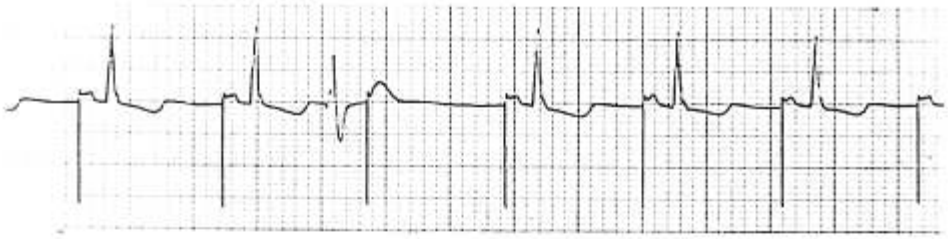

FIG. 6. See text for explanation.

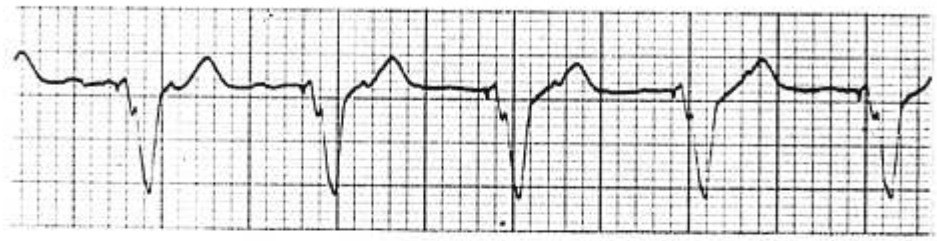

FIG. 7. See text for explanation.

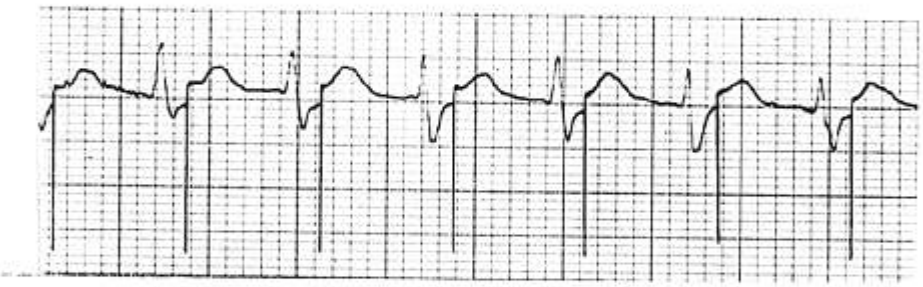

FIG. 8. See text for explanation.

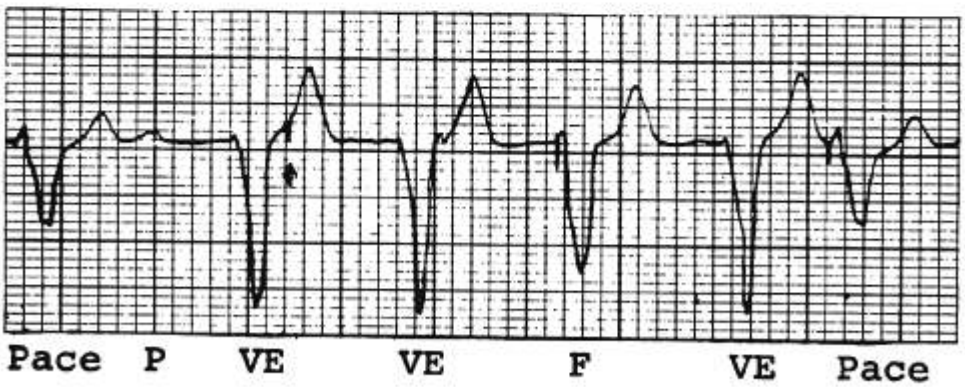

FIG. 9. See text for explanation.

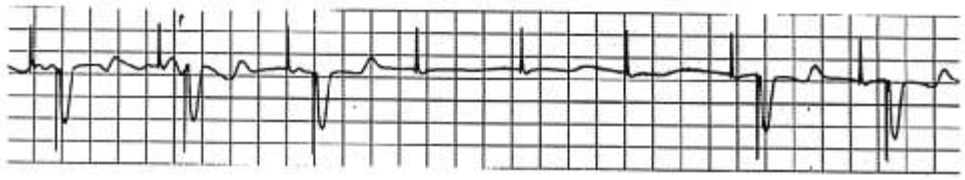

FIG. 10. See text for explanation.

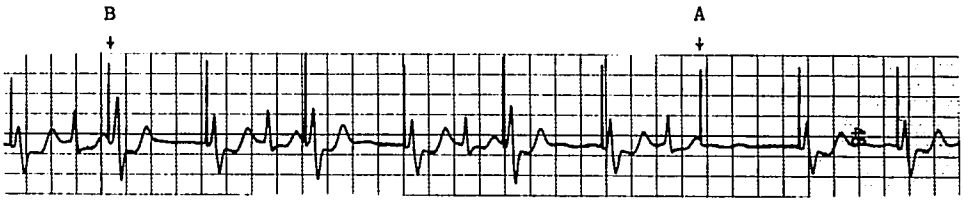

FIG. 11. See text for explanation.

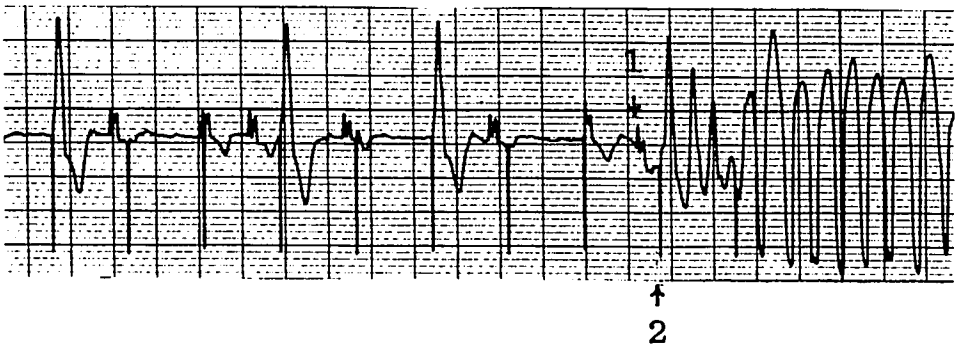

FIG. 12. See text for explanation.

diographic tracings for a one-to-one relationship that continues when the rate control on the generator is changed.

The ECG tracing in figure 9 is from a patient in third-degree AV block and reveals paced beats (pace), P-waves (P), ventricular escape beats (VE), fusion beats (F), and pacing impulses (I). A fusion beat occurs when the pacing impulse and the ectopic focus act together to stimulate the heart and does not indicate abnormal generator function. The abnormal finding in this ECG is the presence of the pacing impulse in the T-wave of the first ventricular escape beat indicating improper R-wave sensing. The hazards of a ventricular pacing impulse occurring on a T wave are shown in figure 12.

A-V sequential pacing with a DVI external generator is demonstrated in figure 10. After the third sequentially paced beat, the R-wave sensitivity of the external generator was set at the most sensitive level causing inhibition of the ventricular pacing circuit by the atrial pacing spike. The last two complexes show restoration of A-V sequential pacing after adjusting the generator to a less sensitive setting. This problem with the external A-V sequential pacer is especially important in patients who are pacemaker-dependent, because the atrial generator output can potentially stop ventricular contraction.

Abnormal sensing of an external VVI generator is shown in figure 11. Several of the pacing impulses fall directly on the T-wave (arrow A) with some of these impulses causing ventricular depolarization (arrow B). In an ischemic myocardium, ventricular tachycardia could occur.

The tracing in figure 12 shows atrial fibrillation and also unipolar ventricular pacing with a VOO generator. It could also be VVI pacing with loss of sensing. Arrow 1 is an intrinsic QRS complex and arrow 2 is a pacing impulse occurring on a T-wave followed by a conducted ventricularly paced beat that initiates ventricular tachycardia. It should be noted that the R-on-T phenomenon occurs with the second, fifth, and seventh pacemaker impulses without causing ventricular tachycardia.

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