

## Perioperative Management of the Pacemaker Patient

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TEMPORARY and permanent cardiac pacing have evolved from sophisticated experimental procedures performed in only a few medical centers to the point where they are now performed on a routine basis in many hospitals. It is estimated that 200,000 or more individuals in the United States have permanent, implanted electronic pacemakers; more than half are more than 70 years old.

The ease of insertion of temporary electrodes has made it feasible to manage successfully otherwise fatal arrhythmias until a permanent pacing system can be implanted. The availability of transvenous permanent pacemaker catheter electrodes has made it possible for even the infirm or clinically ill patient to benefit from permanent pacing.

It is the purpose of this report to familiarize the reader with some of the newer concepts in pacing as they relate to preoperative assessment of the patient with conduction disturbances, and the anticipation of potential perioperative problems of the patient who has a permanent pacemaker and their management.

### Areas of Concern

The anesthesiologist's major concerns whenever a patient with a conduction disturbance or bradycardia syndrome comes to the operating room can be divided into three areas: 1) Does the patient who has an atrioventricular or intraventricular conduction disturbance require temporary pacing during the operative procedure or during induction of anesthesia? 2) What special preoperative assessment is necessary for the patient who has a permanent pacemaker? 3) What intraoperative evaluation needs to be made and what special precautions are necessary to avoid the risks of electromagnetic interference with pacemaker function in the operating and recovery rooms?

### Indications for Temporary Pacing in the Operating Room

In recent years the natural history of intraventricular conduction defects has been clarified. Long-term studies have indicated an increased risk of development of complete atrioventricular

block in patients who have bifascicular block.<sup>\*1,2</sup> However, the risk appears to be quite low: complete atrioventricular block occurs in only 5 to 8 per cent of such patients per year. No study is available to demonstrate that progression to complete heart block is enhanced by general anesthesia. Berg *et al.*,<sup>3</sup> Kundstadt *et al.*,<sup>4</sup> and Rooney *et al.*<sup>5</sup> have all demonstrated that patients who have asymptomatic bifascicular block in normal sinus rhythm do not progress to complete atrioventricular block during anesthesia, and therefore prophylactic temporary pacing is not generally indicated for patients in this group. There are situations, however, in management of the patient who has bifascicular block where prophylactic pacing prior to induction of anesthesia may be indicated. Examples include the unconscious or confused emergency patient from whom the history is unobtainable, or a patient undergoing an operation for an accident or traumatic injury that might have been caused by a Stokes-Adams episode. Even with normal intraventricular conduction, temporary pacing may be indicated for patients who have marked sinus bradycardia, or those who have atrial fibrillation with slow ventricular response, who may require a faster rate during or after the operative procedure. In addition, except under unusual circumstances a temporary lead is indicated prior to induction of anesthesia in any patient receiving an epicardial pacemaker electrode system.

Pharmacologic therapy probably has no place whenever temporary pacing is feasible, but may be necessary as an emergency measure until a temporary electrode can be inserted.

Care must be taken during transporting the patient who has a temporary pacemaker to the operating room so that the electrode does not become dislodged or perforate the right ventricle. When the brachial vein has been used for the temporary electrode insertion, this means that the arm must be stabilized during transport and not placed in hyperextension during positioning of the patient on the operating table. If the temporary electrode is inserted in the femoral vein, the trunk should not be flexed or hyperextended. The controls of the external pulse generator must be easily accessible to the anesthesiologist; a lead extender cable may be necessary for this and should be available in the operating room suite. Continuous monitoring

\* Bifascicular block: complete right bundle-branch block with anterior or posterior fascicular block, complete left bundle-branch block.

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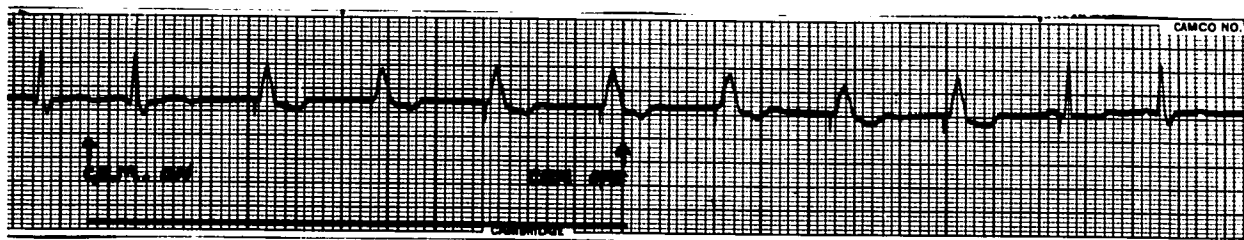


FIG. 1. Normal sinus rhythm. Upon application of carotid sinus massage (CSM ON), the sinus rate slows, and appropriate pacing ensues. The pacemaker stimulus artifact is seen as a small vertical line before each wide QRS complex. Shortly after release of carotid sinus massage (CSM OFF), normal sinus rhythm resumes. Normal demand pacing.

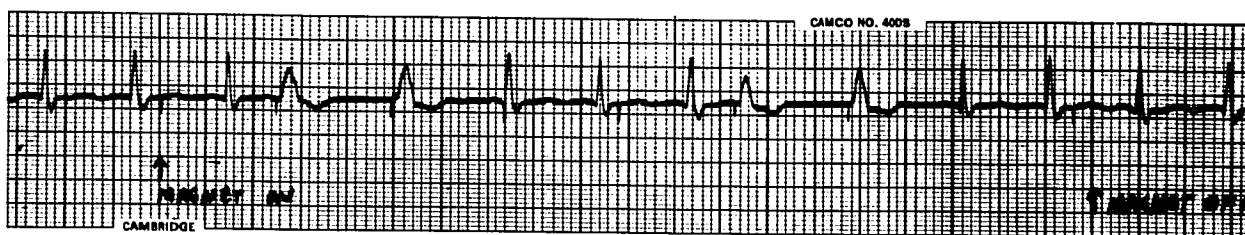


FIG. 2. Normal sinus rhythm. Upon conversion to asynchronous mode (Magnet ON), the pacemaker paces the heart when the stimulus falls outside the refractory period of the ventricle.

is necessary during the operative procedure to detect electrode-induced arrhythmias and to confirm proper temporary pacemaker function.

#### Indications for Permanent Pacing

Initially, permanent pacing was utilized only for the patient with complete atrioventricular block complicated by Stokes-Adams episodes or congestive failure. Subsequently, the indications for permanent pacing were extended to patients who had other arrhythmias as well. For example, patients who have symptomatic sinus bradycardia or periods of sinus node arrest, accompanied by dizziness, syncope or congestive heart failure, currently constitute approximately a quarter of all patients with permanent pacemakers.<sup>8</sup> Patients who have sinus node dysfunction accompanied by paroxysmal supraventricular tachycardia, atrial flutter, or atrial fibrillation (the "bradycardia-tachycardia syndrome") are treated with a combination of antiarrhythmic agents for the supraventricular arrhythmia and a permanent pacemaker for the bradycardia.<sup>8</sup> The hypersensitive carotid sinus syndrome is a less common arrhythmia treated with pacing.<sup>9</sup> This clinical complex includes vasomotor disturbances, pronounced sinus node slowing, or periods of sinus node arrest upon turning of the head to either side, or looking upward, or upon application of light pressure to the carotid sinus. The bradycardia component of this syndrome can be controlled with permanent pacing; associated autonomic dysfunction may necessitate pharmacologic intervention. Permanent pacing has also been utilized to suppress ventricular tachycardia and to convert

supraventricular arrhythmias resistant to antiarrhythmic agents.<sup>10,11</sup>

#### Preoperative Evaluation of the Patient with a Permanent Pacemaker

It needs to be emphasized that the patient who has a permanent pacemaker has significant underlying cardiovascular disease.<sup>12-14</sup> Most frequently ischemic heart disease with previous myocardial infarction has caused the arrhythmia for which the pacing has been instituted. Other causes of complete heart block or sinus node disease are rheumatic heart disease, congenital complete heart block, and cardiomyopathy. Frequently the patient will not have an apparent cause for the conduction system disturbance. These patients may have Lev's disease (fibrosis and calcification of the endocardial skeleton<sup>13</sup>) or "Lenegre's disease" (idiopathic fibrosis of the conduction system<sup>14</sup>).

During stress or sympathetic stimulation, such as during an operative procedure, return to normal sinus rhythm may occur in patients previously in complete heart block; ventricular arrhythmias are also common in patients with pacemakers.<sup>15</sup> It is important that the patient who has a pacemaker be evaluated for any progression of symptoms of the underlying heart disease (*e.g.*, congestive heart failure, new arrhythmias, etc.), as well as for electrolyte disorders and adequacy of digitalization, prior to any planned operative procedure. Likewise, it is important to evaluate the pacemaker itself for its ability to pace the heart properly. The type (fixed-rate or demand), time since implantation, and the pacemaker rate at the time of implantation

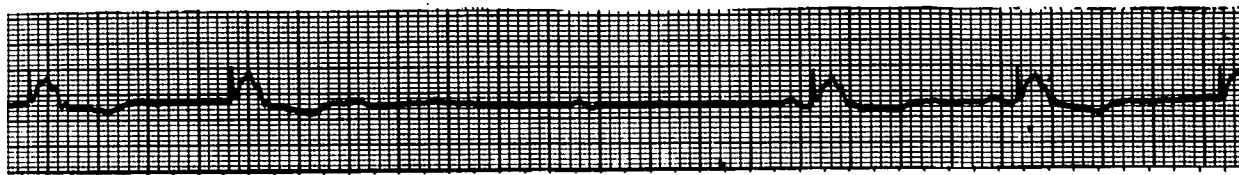


FIG. 3. A short (2.3-sec) period of pacemaker inhibition during manipulation of a mechanized dental chair (field intensity = 1 volt/meter). This is an example of *indirect* electromagnetic interference with pacemaker function.

should be determined. Since all manufacturers give each patient an identification card that carries this information, these data are usually simple to obtain. Assessment of proper pacemaker function also involves careful review of the preoperative electrocardiogram.

Nonadjustable rate units should not differ by more than two beats per minute from the rate at manufacture. A 10 per cent reduction in rate usually indicates power-source depletion, and is one means of identifying pulse-generator failure. If the patient's intrinsic rhythm is faster than the rate of the demand pacemaker, the pacemaker will be suppressed. In such cases it will be necessary to activate the unit. One way to do so is by applying carotid-sinus massage while the patient is being monitored electrocardiographically. This will usually slow the patient's heart rate to less than that of the pacemaker, so that a paced rhythm occurs (fig. 1). This should not be done in patients who have cerebrovascular or carotid-artery disease; alternative methods of increasing vagal tone such as Valsalva maneuver may be more safely utilized in these patients.

If the patient's rate cannot be slowed sufficiently, or if carotid-sinus massage is contraindicated, it will be necessary to activate the pacemaker in the fixed-rate (asynchronous) mode by application of a strong magnet externally. This will cause ventricular activation by the pacemaker when the pacemaker stimulus falls outside the ventricular refractory period (fig. 2). Although pacing stimuli will fall on the T wave of the previously conducted beats, the pacemaker's energy output is sufficiently small that there is no risk of inducing ventricular fibrillation under these conditions. If pacemaker failure is suspected, evaluation of the pacemaker pulse width or waveform may be of additional diagnostic value.

It is also important to establish the etiology of the heart disease responsible for the arrhythmia being treated with the pacemaker, inasmuch as endocarditis prophylaxis will be indicated preoperatively for the patient who has rheumatic or congenital defects, but is not mandatory for patients who have ischemic heart disease or cardiomyopathy. In the past, endocarditis has been reported to occur with transvenous pacing electrodes in place, but this has usually been only following placement of the now-discontinued permanent external pacing system,<sup>6</sup> in cases of a grossly infected implant with bacteremia, or when pacing with temporary

electrodes. Therefore, we do not routinely give endocarditis prophylaxis because of the presence of the permanent pacemaker itself.

Finally, it is important to establish whether electrocautery will be used during the operative procedure. Since 90 per cent or more of currently implanted pacemakers are of the "demand" (synchronous) variety, they are sensitive to direct or indirect electromagnetic interference (EMI).

*Direct* EMI is the most commonly encountered type of EMI in the operating room. In this type the body is part of the current path; EMI from electrocautery devices and the use of dental-pulp vitality testers<sup>17-21</sup> are examples. *Indirect* EMI is interference conducted through the atmosphere, such as with radar, orthopedic saws, or telemetry devices. The latter has received less attention, although rare cases of interference in the hospital setting have been reported<sup>21</sup> (fig. 3). External pacing systems seem especially vulnerable to this type of EMI. While pacemaker inhibition during transurethral prostatectomy has received the most attention in the literature,<sup>17,18</sup> electrocautery during any operation is a potential hazard to the pacemaker-dependent patient. Although most synchronous units will convert to asynchronous mode during *continuous* EMI from any source, intermittent or modulated fields of interference are especially dangerous because reversion to fixed-rate mode will not occur, but repetitive inhibition will. Although there is some variation in opinion as to the risk to the patient who has a pacemaker from electromagnetic interference,<sup>19</sup> deaths have been reported and caution should be exercised.<sup>20</sup>

To decrease the possibility of adverse effects of electrocautery on the pacemaker patient, the following intraoperative measures have been employed at the University of Michigan Medical Center:

- 1) The indifferent plate of the electrocautery unit should be placed as far away from the pulse generator and lead as possible.
- 2) The patient should be electrocardiographically monitored during the operation, looking for significant arrhythmias and proper pacemaker function.
- 3) If the patient is being paced at the time of the procedure, palpation of the pulse or esophageal stethoscopic cardiac monitoring during the electrocauterization is necessary, since the electrical surge will usually render the ECG diagnostically useless. Inhibition of pacemaker function will be detected

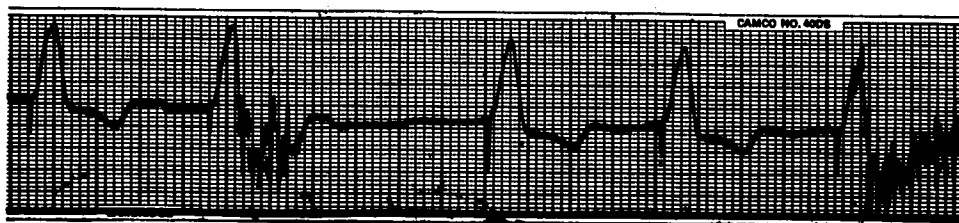


FIG. 4. A short burst of electrocautery causes temporary pacemaker inhibition. Pacing resumes normally when electrocautery ceases. Repetitive surges of electrocautery could significantly slow the rate in the pacemaker-dependent patient. This is an example of the adverse effect of the *direct* form of the electromagnetic interference on pacemaker function.

by a pause in the pulse with electrocautery use (fig. 4). If this occurs, a high-powered magnet should be applied directly over the pacemaker to convert it to "fixed-rate" (asynchronous) mode. In this way inhibition can usually be prevented.

4) When the electromagnetic field intensity is high, *e.g.*, when the electrocautery unit is being used close to the pulse generator or lead, this maneuver may be unsuccessful. In this circumstance the frequency and duration of electrocautery should be limited to one-second bursts every 10 seconds to prevent repetitive asystolic periods.

5) When the pulse generator is located close to the operative field, it may be necessary to bring a sterilized magnet into the field; it can be sutured temporarily to the skin overlying the pulse generator, or it can be held against the skin overlying the pulse generator by an adhesive drape.

Some pacemakers are sensitive to field intensities as low as 1 volt per meter,<sup>21</sup> and since the field intensity from electrocautery may be as high as 60 volts per meter, caution with all sources of electromagnetic interference is indicated. Figures 3 and 4 illustrate pacemaker inhibition during direct and indirect electromagnetic interference.

Since pacemakers may vary in sensitivity to external electromagnetic interference, the maneuvers described above should be utilized with each pacemaker patient for whom electrocautery is used. Although unipolar pulse generators are said to be more sensitive than bipolar units, the anesthesiologist should go through these steps for each patient, regardless of generator type.

Competing rhythms will result when the patient is in normal sinus rhythm during the operative procedure and the unit has been converted to asynchronous mode. Because of the low output of currently available pulse generators, the risk of inducing ventricular fibrillation by induced competing rhythms is negligible so long as hypoxemia is prevented and electrolyte balance is maintained.

#### Additional Perioperative Considerations

Inasmuch as the pacemaker electrode is in direct contact with the endocardium, a direct electrical pathway to the heart is present in the paced

patient. This is most likely to be a hazard in the patient who has a temporary pacemaker, when the electrical contact points between pacemaker lead and external generator are not properly shielded. To protect against the hazard of transmitting electrostatic energy directly to the patient's heart, these contact points should be covered with a rubber glove or the operator should wear rubber gloves when manipulating the leads.

Since patients who have permanent pacemakers have underlying cardiac disease, recognition and treatment of significant arrhythmias in the perioperative period are essential. Hypoxemia should be corrected, and antiarrhythmic agents utilized when necessary. Continuous monitoring is indicated even after the procedure, and until vital signs have stabilized.

Spontaneous skeletal muscle contraction is common in the postoperative period, as is voluntary or involuntary isometric muscle activity. Such activity is capable of generating sufficiently large myopotentials to inhibit unipolar pulse generators.<sup>22,23</sup> This inhibition may result in periods of cardiac standstill. Such events should be looked for by continuous electrocardiographic monitoring. When found, temporary conversion to asynchronous mode by application of an external magnet is indicated. This magnet can be taped to the skin overlying the pulse generator until the patient's condition is stable.

Although permanent pacemakers have protective circuits to guard against externally applied high-voltage discharge, such as with defibrillators, pulse generator malfunction has been reported to occur following external defibrillation or cardioversion. In elective cardioversion the lowest voltage necessary should be utilized. Ventricular fibrillation in the patient who has a permanent pacemaker should be managed with techniques similar to those used for any other patient, with the exception that the defibrillator paddles should not be placed directly over the permanent pulse generator.

#### References

1. Denes P, Dhengra R, Wu D, et al: Sudden cardiac death in patients with chronic bifascicular block. *Circulation* 52:113, 1975 (suppl II)

2. Schuman MM, Brennen M, Maslin G, et al: Cooperative prospective study of bundle-branch block. *Circulation* 52:113, 1975 (suppl II)
3. Berg R, Katler MN: The significance of bilateral bundle-branch block in the preoperative patient. *Chest* 59:62-67, 1972
4. Kunstadt D, Punja M, Cagin N, et al: Bifascicular block: A clinical and electrophysiologic study. *Am Heart J* 46: 173-181, 1973
5. Rooney SM, Goldiner PI, Muss E: Relationship of right bundle-branch block and marked left axis deviation to complete heart block during general anesthesia. *ANESTHESIOLOGY* 44:64-66, 1976
6. Mascarenhas E, Center S: Results of permanent pacemaker therapy. *Cardiac Pacing*. Edited by Samet P. New York, Grune and Stratton, 1973, p 176
7. Rubenstein JJ, Schulman CL, Yurchak PM, et al: Clinical spectrum of the sick sinus syndrome. *Circulation* 46:5-13, 1972
8. Ferrer MI: The sick sinus syndrome. *Circulation* 47:635-641, 1973
9. Thomas JE: Hyperactive carotid sinus reflex and carotid sinus syncope. *Mayo Clin Proc* 44:127-139, 1969
10. Spurrell RAJ, Sowton E: Pacing techniques in the management of paroxysmal supraventricular tachycardia. *J Electrocardiography* 8:287-295, 1975
11. Ticson AR, Whalen RW: Refractory supraventricular tachycardias. *Circulation* 47:642-653, 1973
12. Lev M, Kinare SG, Pick A: The pathogenesis of atrioventricular block in coronary disease. *Circulation* 42: 409-425, 1970
13. Lev M: Anatomic basis for atrioventricular block. *Am J Med* 37:742-747, 1964
14. Lenegre J: Etiology and pathology of bilateral branch block in relation to complete heart block. *Prog Cardiovasc Dis* 6:409-444, 1964
15. Singer E, Gooch AS, Morse D: Exercise-induced arrhythmias in patients with pacemakers. *JAMA* 224:1515-1518, 1973
16. Furman S, Escher DJW: Principles and Techniques of Cardiac Pacing. New York, Harper and Row, 1970, p 170
17. Fein RL: Transurethral electrocautery procedures in patients with cardiac pacemakers. *JAMA* 202:7-9, 1967
18. Wojsycyuk WJ, Mowry FM, Dugan NL: Deactivation of a demand pacemaker by transurethral electrocautery. *N Engl J Med* 280:34, 1969
19. Smyth NPD, Parsonnet V, Escher DJW, et al: The pacemaker patient and the electromagnetic environment. *JAMA* 227:1412, 1974
20. Fleming WH, Toler JC: Degradation of pacemaker function by electromagnetic interference. *Circulation* 50:226, 1974 (suppl III)
21. Simon AB, Linde B, Bonnette GH, et al: The individual with a pacemaker in the dental environment. *JADA* 91:1224-1229, 1975
22. Ohm OI, Bruland H, Pedersen OM, et al: Interference effect of myopotentials on function of unipolar demand pacemakers. *Br Heart J* 36:77-84, 1974
23. McAnulty J, Phillips S, Dobbs J, et al: Demand pacemaker inhibition by isometric skeletal muscle contraction. *Circulation* 50:241, 1974 (suppl III)