Intraoperative Pacemaker Complications

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Intraoperative pacemaker complications associated with electrosurgery-induced interference were first reported in 1965 (during thoracotomy),¹ then again in 1968 (durng open heart surgery),² and in 1969 (during transurethral surgery).³ Because these early problems were caused by electromagnetic interference, pacemakers now have complex circuitry that tries to eliminate problems from external electrical interference. However, the existence of such circuitry can itself result in new intraoperative complications. In addition, newer pacemakers have sensing circuits that can detect a problem with the primary program and activate default mechanisms (that is, backup pacing systems); these sensing circuits and default mechanisms also can play a role in unusual intraoperative complications. We describe two perioperative complications that were related to pacemakers but not owing to their malfunctioning. Each complication was unique to the individual model of pacemaker. Such idiosyncrasies can be dangerous if not understood.

REPORTS OF TWO CASES

Case 1. A 73-year-old woman (65 kg) was admitted for elective right carotid endarterectomy. Evaluation of a syncopal episode 4 months before surgery revealed spontaneous sinus pauses, recorded on Holter monitor tape, lasting as long as 2.5 s. Consequently, a Cordis Bipolar Multicor® II (Model 402A) pacemaker generator was inserted just inferior to the left clavicle and attached to a Cordis tine-tipped endocaridal transvenous ventricular pacing lead (Model 327-563). This pacemaker was programmed to the demand mode at 70 beats/min and was functioning appropriately at the time of insertion. Medical history revealed mild hypertension of several years duration, for which the patient took hydrochlorothiazide. On preoperative physical and laboratory examination, arterial blood pressure

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was 130/70 mmHg and sinus rhythm was 80 beats/min, potassium was 4.3 mEq/l. No medication other than hydrochlorothiazide was given before insertion or application of the monitors, *i.e.*, an 18gauge radial intraarterial catheter, six-lead electrocardiogram (ECG), and a dual channel Holter monitor (Instruments for Cardiac Research, Inc., Liverpool, New York). Anesthesia was induced with thiopental iv and with inhalation of increasing concentrations of isoflurane and oxygen. Intravenous administration of 3 mg *d*-tubocurarine and 100 mg succinylcholine facilitated endotracheal intubation. Induction and maintenance of anesthesia proceeded without complications. Since the pacemaker generator was located near the surgical field, the surgeons were asked to use a bipolar electrosurgery generator (Radionics, Inc., Burlington, Massachusetts; cautery forceps, Codman and Shurtleff, Randolf, Massachusetts).

Twenty minutes after skin incision, the surgeons, unaccustomed to using a bipolar electrosurgery unit, suggested that the usual unipolar electrosurgery generator (electrosurgical unit, Ritter Company, Rochester, New York; electrosurgery pencil, Cooper Medical Devices Corp., San Leandro, California) would provide better hemostasis and requested its use. The electrosurgery ground plate was placed on the patient's right thigh. Before initiation of the unipolar electrosurgery unit, the Holter monitor tape revealed sinus rhythm of 75–108 beats/min, with occasional premature atrial contractions, and paced rhythm when the sinus rate fell below 70 beats/min. Upon initial use of the unipolar electrosurgery unit, the ECG showed that pacemaker spikes, and pacemaker-induced contractions competed with the intrinsic sinus rhythm of the heart (fig. 1).

Further examination of the ECG indicated that the pacemaker converted to a fixed-rate mode of 53 beats/min. Depending on the timing of contractions, competition between the intrinsic heart rhythm and the pacemaker-generated beats caused episodic decreases in arterial blood pressure.

The cardiologist who was consulted immediately after the pacemaker converted to the fixed-rate mode was unable to reprogram the pacemaker with the Cordis[®] programmer. Surgery proceeded for approximately 2 more hours without complications. The trachea was extubated in the operating room, and recovery was uneventful. After the patient arrived in the recovery room and after telephone consultation with the supervising engineer for this model of Cordis[®] pacemaker, the pacemaker was successfully reprogrammed to its original mode.

Case 2. A 62-year-old man was scheduled for elective right thoracotomy to remove a coin lesion in the middle lobe of the right lung. A Siemens Elema[®] pacemaker (Model 678) had been inserted 13 months earlier because of sick sinus syndrome. The pacemaker generator was connected to the heart by a Diag-Medcor[®] atrial lead wire (Model ESI-110) and was programmed to the demand mode at 70 beats/min. He had had no problems related to his heart or to the pacemaker since insertion, and regular clinic follow-up had been unremarkable. Physical examination revealed arterial blood pressure of 100/60 mmHg and weight of 85 kg; ECG recorded an atrial paced rhythm of 70 beats/min. Morphine sulfate 10 mg im and diazepam 10 mg po were given before arrival in the operating room. Monitors included a 20-gauge radial intraarterial catheter and a sixlead ECG. In patients having pacemakers programmed in the demand

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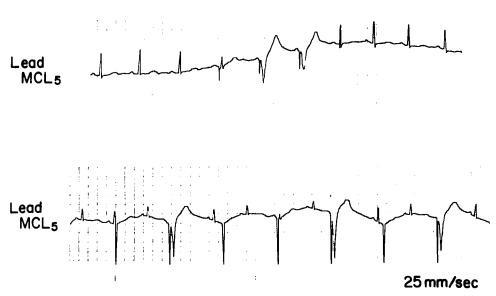
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FIG. 1 (Case 1). Top panel: A sample from the Holter tape (paper speed, 25 mm/s) taken prior to activation of backup permanent pacing demonstrates sinus rhythm that begins at 71.5 beats/min. As the sinus rate spontaneously falls below 70 beats/min, the pacemaker activates for three beats, as programmed in the demand mode. As the sinus rate increases above 70 beats/min, the pacemaker again is inhibited. Bottom panel: Two independent rhythms are now observed. Sinus rhythm, varying slightly at approximately 86 beats/ min, is seen competing with a paced rhythm at 52.5 beats/min. Paced beats 1, 3, and 6 do not initiate a cardiac contraction, falling within the right ventricular effective re-

fractory period. Paced beats 2, 4, 5, and 7 do initiate a contraction. The timing of the contractions seen in the bottom panel resulted in wide variation in blood pressure (see text, Case 1).

mode, we normally perform a magnet test before induction of anesthesia. This test consists of applying a strong magnet over the generator, which normally converts the pacemaker to the fixed-rate mode. Then, when the magnet is removed, the pacemaker returns to its demand mode. However, when the magnet was placed over this pacemaker, the following unexpected sequence of events was recorded (fig. 2). Application of the magnet resulted in the paced rate increasing to approximately 100 beats/min for 16 beats, and then to 122 beats/min for 16 beats, followed by an inability of the pacemaker to elicit cardiac contractions (*i.e.*, to "capture" the heart) for 3.1 s, during which time blood pressure fell to below 60 mmHg. This sequence would repeat until the magnet was removed, at which time the pacemaker automatically returned to the previously programmed demand mode of 70 beats/min. The anesthetist discussed this unexpected occurrence with the surgeons, and all agreed to proceed with the scheduled operation. Surgery and recovery were uneventful.

DISCUSSION

Pacemaker function can be influenced by environmental factors, both electric and nonelectric.^{4,5} Problems unique to the operating room, particularly those related to electrical interference, also have been discussed.^{6,7} The threshold for pacemaker "capture" can be influenced by cellular factors such as acid–base status,⁸

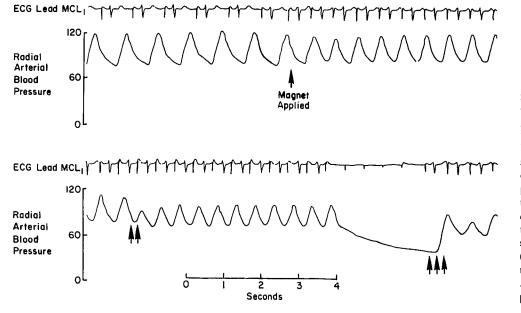


FIG. 2 (Case 2). The top tracing begins with an atrial paced rate of 70 beats/min (paper speed is 25 mm/s; blood pressure is measured in mmHg). The arrow indicates placement of the magnet over the generator, resulting in a paced rate of 100 beats/min. This is the beginning of the "Vario" threshold testing, which consists first of the battery test phase. The lower trace shows the 16-beat "Vario" threshold test phase. Note that the blood pressure falls at the beginning of the threshold test phase (double arrow). Note also that once the threshold test phase is finished, the sequence begins to repeat itself (triple arrow) and will continue to repeat until the magnet is removed. All 16 beats of the battery test phase have not been shown.

320

Anesthesiology V 63, No 3, Sep 1985

arterial oxygen partial pressure,⁹ serum potassium concentration,^{10,11} and use of antiarrhythmic agents.¹² Because normal pacemaker functioning has been reported to change inadvertently, resulting in major complications^{1,2} and even death,¹³ generators for newer pacemakers have back-up (default) pacing modes that prevent pacemakers from being inactivated for long periods of time.

The internal systems monitoring pacemaker function and providing backup pacing are becoming quite sophisticated. The Cordis model 402A® generator has two such systems.** One backup pacing system evaluates incoming signals to determine whether these signals represent electrical noise (interference) or intrinsic cardiac electrical activity. Another back-up pacing system is designed to detect battery depletion or malfunctioning of some of the pacemaker's components associated with the timing of impulse generation. Our first case is unusual because the electrosurgery unit initiated activation of the back-up pacing circuit, which is normally activated by low battery voltage or an alteration in the timing of impulse generation. The pacemaker functioned entirely as programmed by the manufacturer. It was, however, a backup pacing mode with which none of the authors, or the newly designated manufacturer representative, was familiar.

In the first case, electrosurgery interfered with the quartz crystal clock necessary for accurate timing of impulse generation. Because such defects may signify impending battery depletion, a slow (52.5 beats/min), fixed-rate back-up pacing mode was activated, the purpose of which is to preserve battery life. Because this back-up pacing mode is a permanent one, the rate did not revert to the previously programmed rate after the electrosurgery unit was discontinued.

This conversion from a demand mode to a slow fixedrate mode would not have been prevented by prior application of a magnet over the generator. The magnet would certainly have temporarily converted the demand mode to a fixed-rate mode at 70 beats/min before the use of the electrosurgery unit. The unipolar electrosurgical current still would have interfered with the timing of the pacemaker's impulse generation. This interference would have activated the slow (52.5 beats/ min) back-up pacing mode. Since this is a permanent fixed rate default mode, the pacemaker would have continued to fire at 52.5 beats/min, even after removal of the magnet and would have continued at this rate until reprogrammed externally.

This slow (52.5 beats/min), permanently fixed-rate default mode must be distinguished from the back-up pacing mode that is activated when interference from electrical noise is detected. If continuous electrical noise is detected, the pacemaker will temporarily generate fixed-rate pacing at the rate previously programmed for the demand mode. This fixed-rate pacing mode will continue until the electrical interference is no longer present, at which time the pacemaker will automatically revert to the demand mode. In this patient, continuous electrical interference affecting the sensing system would result in a *fixed-rate* pacing of 70 beats/min. As soon as the electrical interference is terminated, the pacemaker would automatically revert back to the demand mode rate of 70 beats/min. If a single electrical signal mimicking cardiac electrical activity is detected during the noise sampling period, the pacemaker will not fire and a new sampling period will begin again. In patients who depend on a pacemaker set to the demand mode, intermittent electrical interference mimicking normal cardiac activity may lead to long periods of asystole. This type of interference (*i.e.*, intermittent electrosurgery) can be prevented by converting the pacemaker to fixedrate pacing by placing a magnet over the pacemaker generator.

This pacemaker conversion occurred during unipolar, but not bipolar, electrosurgical interference. We have not explored the possible electrical interactions between electrosurgery, either unipolar or bipolar, and this or any other pacemaker. The coagulation and cutting circuitry of the electrosurgery units also may affect pacemakers differently.

When the pacemaker generated an impulse fixed at 52.5 beats/min, we became concerned about two potential problems. First, since both the pacemaker rate and the patient's own heart rate differed, the potential existed for ventricular tachycardia induced by an "Ron-T phenomenon."¹⁴ This event consists of the superimposition of the ventricular depolarization ("R wave") of an ectopic beat on the T wave of the preceding beat. Because generator output of a pacemaker is normally low (6 mA in this case), ventricular tachycardia did not occur. We did, however, bring a defibrillator into the operating room. Second, depending on the timing of contractions, systolic blood pressure varied widely. This, too, did not prove to be a problem.

In the second case, human error (forgetfulness) was responsible. Again, as in the first case, the pacemaker functioned entirely as programmed by the manufacturer. Shortly after original insertion of the pacemaker, it was realized that the threshold for capture had changed. (Threshold is defined as the minimum pacemaker-generated voltage required to cause depolarization of the

^{**} Model 402A Multicor[®] II Unipolar/Bipolar, Multiprogrammable Cardiac Pacer with Telemetry. Instructions for Use. April 1983. Cordis Corporation, Miami, Florida, 1983, pp 17, 18, 21, 23, 25.

heart.^{††}) This is not unusual, as scar tissue forms at the site in the heart where the pacemaker lead wire makes contact. The manufacturer has programmed into the generator a special mode for noninvasive threshold testing, and this mode was programmed into the pacemaker at the followup visit to the pacemaker clinic. This threshold function mode, termed the "Vario" mode by the manufacturer, works as follows and accounts for the observed electrocardiographic findings: The "Vario" sequence consists first of 16 fixed-rate pulses at approximately 100 beats/min. These are followed by 16 pulses at appoximately 120 beats/min, the output of which is successively reduced to 0 volts. Since the first 16 beats in the "Vario" mode test the battery (like the normal magnet test), both threshold and battery function can be assessed in the "Vario" mode.⁺⁺ After stable threshold values were obtained, the "Vario" mode simply had not been turned off.

The main problem with leaving the pacemaker in the "Vario" mode is that application of a magnet over the generator will not convert the pacemaker from the demand to the fixed-rate mode. Therefore, electrosurgical interference, which may alter normal pacemakersensing operation, cannot be prevented by applying a magnet over the generator. Additionally, as noted in figure 2, unless the magnet is removed, the pacemaker will continue in the "Vario" mode. Thus, the patient had an asymptomatic decrease in blood pressure during the 3.1-s period that the pacemaker was not able to elicit cardiac contractions. Had the threshold for capture been high, asystole may have lasted longer and signs and symptoms of cerebral hypoperfusion may have occurred. Only removing the magnet from the generator would cause the generator to revert to the previously programmed mode. Ideally, this operation should have been postponed until such was determined.

Both cases illustrate the sophistication of current pacemakers. To date, at least 10 companies manufacture no fewer than 100 types of pacemakers.

It is impossible to know how every pacemaker functions. To acquaint ourselves with pacemaker characteristics, it is important to have access to hospital records pertaining to pacemaker function. As patients move from hospital to hospital and even from state to state, a registry of pacemaker patients and information about their pacemakers may become necessary and would certainly be helpful. Also, industry-wide standardization of pacemaker analyzers, programming modes, and programmers would facilitate pacemaker testing and reprogramming no matter which company manufactured them. Currently, brochures are the only source about specifications and normal operating function. Although company representatives are now available 24 h a day to answer questions about pacemakers, in our first case, this designated representative was not able to correct the defect. Only the senior engineer responsible for the pacemaker was able to tell us what had happened and how to reprogram the pacemaker. We believe that the increasing complexity of newer pacemakers manufactured with noninterchangeable components makes it imperative to obtain information about reprogramming the individual pacemaker and to test such reprogramming strategies before induction of anesthesia.

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References

- 1. Lichter I, Borrie J, Miller WM: Radio-frequency hazards with cardiac pacemakers. Br Med J 1:1513-1518, 1965
- Titel JH, El Etr AA: Fibrillation resulting from pacemaker electrodes and electrocautery during surgery. ANESTHESIOL-OGY 29:845–846, 1968
- Wajszczuk WJ, Mowry FM, Dugan NL: Deactivation of a demand pacemaker by transurethral electrocautery. N Engl J Med 280:34–35, 1969
- Sowton E: Environmental hazards for pacemaker patients. J R Coll Physicians Lond 16:159–164, 1982
- Smyth NPD, Parsonnet V, Escher DJW, Furman S: The pacemaker patient and the electromagnetic environment. JAMA 227:1412, 1974
- Fein RL: Transurethral electrocautery procedures in patients with cardiac pacemakers. JAMA 202:101–103, 1967
- 7. Simon AB: Perioperative management of the pacemaker patient. ANESTHESIOLOGY 46:127–131, 1977
- Hughes HC Jr, Tyers GFO, Torman HA: Effects of acid-base imbalance on myocardial pacing thresholds. J Thorac Cardiovasc Surg 69:743-746, 1975
- Doenecke P, Flöthner R, Rettig G, Bette L: Studies of shortand long-term threshold changes, Advances in Pacemaker Technology. Edited by Schaldach M, Furman S. New York, Springer-Verlag, 1975, pp 283–296
- O-Reilly MV, Murnaghan DP, Williams MB. Transvenous pacemaker failure induced by hyperkalemia. JAMA 228:336-337, 1974
- Al-Abdulla HM, Lulu DJ: Hypokalemia and pacemaker failure. Am Surg 40:234–236, 1974
- Gay RJ, Brown DF: Pacemaker failure due to procainamide toxicity. Am J Cardiol 34:728–732, 1974
- Fleming WH, Toler JC: Degradation of pacemaker function by electromagnetic interference. Circulation 50(suppl 111):111– 226, 1974
- Engel TR, Meister SG, Frankl WS: The "R-on-T" phenomenon. An update and critical review. Ann Intern Med 88:221–225, 1978

^{††} Siemens-Elema Programmable Pulse Generator 678[®]. Instructions for Use. October 1980. Siemens-Elema AB Medicinsk Teknik, Solna, Sweden, 1980, pp 21, 29, 31, 32.