CASE REPORTS


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Apparent Failure of a Precordial Magnet and Pacemaker Programmer to Convert a DDD Pacemaker to VOO Mode during the Use of the Electrosurgical Unit


CARDIAC pacemaker failure in patients who are pacemaker dependent results in asystole, and can be catastrophic. A major concern is how electromagnetic interference (EMI) from the electrosurgical unit (ESU) will alter pacemaker function. The effects of EMI on pacemaker function are multiple. Electromagnetic interference, by itself, can reprogram some multiprogrammable pacemakers.3–5 In addition, some pacemakers may automatically convert to the VOO (see table 1 for explanation of pacemaker code) or DOO mode.3–5 Others may be totally inhibited.3–5 If total inhibition occurs in a pacemaker-dependent patient, the result will be asystole. Pacemaker inhibition induced by EMI from the ESU generally responds to placement of a precordial magnet on the skin overlying the pacemaker generator. This effectually activates a magnetic reed switch within the pacemaker that converts the pacemaker to a VOO or DOO mode as long as the magnet overlies the generator unit. Atlee4 recommends having the pacemaker reprogrammed to the VOO mode using a pacemaker programmer. In the VOO mode, the pacemaker is not expected to sense the EMI and, therefore, will continue to pace the heart asynchronously. We report a case of a pacemaker-dependent patient where EMI from the ESU produced total pacemaker inhibition, despite proper placement of a precordial magnet or use of a pacemaker programmer.

Case Report

The patient was a 70-year-old man scheduled for resection of a carcinoma of the left neck. He had a DDDR pacemaker (Model 1254, Teletronics, Englewood, CO) placed for the treatment of complete heart block 3 yr before his current admission. He was currently pacemaker dependent. The pacemaker generator was placed in the left infraclavicular region and attached to endocardial ventricular (Cordis Core, Miami, FL, model 330-201) and atrial (Teletronics, model 330-801) leads. He had no subsequent problems related to his heart or pacemaker since its insertion. Regular clinic visits showed that his pacemaker was working appropriately and the intended programmed settings, in the DDD mode, were operational.

Physical examination revealed an arterial pressure of 150/70 mmHg and a paced heart rate of 70 beats/min.

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Table 1. Understanding Pacemaker Codes

<table>
<thead>
<tr>
<th>Chamber Paced (first position)</th>
<th>Chamber Sensed (second position)</th>
<th>Response to Sensing (third position)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A = Ventricle</td>
<td>V = Ventricle</td>
<td>T = Triggers pacing</td>
</tr>
<tr>
<td>A = Atrium</td>
<td>A = Atrium</td>
<td>I = Inhibits pacing</td>
</tr>
<tr>
<td>D = Dual (A and V)</td>
<td>D = Dual (A and V)</td>
<td>D = Dual (A and V)</td>
</tr>
<tr>
<td>O = None</td>
<td>O = None</td>
<td>O = None</td>
</tr>
</tbody>
</table>

An “R” in the fourth position signifies rate responsiveness.

After appropriate monitors were placed, including a cannula in his right radial artery for continuous blood pressure monitoring, anesthesia was induced with thiopental and, after intravenous succinylcholine, the trachea was intubated. The return plate to the unipolar ESU (System 5000, Power Plus, C. R. Bard, Englewood, CO) was placed under the patient’s left buttock. Desflurane and nitrous oxide were used to maintain anesthesia. The operative course was unremarkable until neck dissection was begun. Activation of the ESU by the surgeon initiated asystole. After placement of a precordial magnet on the skin over the pacemaker generator by the anesthesiologist, the surgeon proceeded. When the ESU was activated again, the pacemaker was totally inhibited, asystole ensued for almost 6 s, and the arterial pressure trace decreased precipitously. A cardiologist then interrogated and reprogrammed the pacemaker to the VOO mode (Teletronics Pacemaker Programmer model 9600). The surgeons proceeded once again with the surgery. Activation of the ESU continued to result in asystole, duplicating the previous findings. The operation was completed using a bipolar ESU. The pacemaker functioned as expected in the programmed VOO mode for the remainder of the surgery, which proceeded uneventfully.

Postoperatively, the pacemaker was interrogated, and found functioning normally. It was then reprogrammed to its previous DDD settings.

Discussion

Use of a precordial magnet to convert a pacemaker to an asynchronous mode (VOO or DOO) should be restricted to instances when EMI can cause life-threatening inhibition of the pacemaker. Routine prophylactic use of the precordial magnet is not recommended because of possible pacemaker reprogramming in the presence of EMI. This is particularly true for those pacemakers that use a magnetic reed switch to enable the pacemaker to receive programming instructions. Radiofrequency signals are used for programming instructions after the magnetic reed switch has been enabled. High frequency EMI from the ESU could be misinterpreted as a programming instruction. The new reprogrammed parameters would then become manifest after the magnet is taken off the skin overlying the pacemaker generator. How the pacemaker would be reprogrammed depends on the pacemaker’s characteristics and is often unpredictable.

With the activation of the ESU, the patient’s arterial pressure decreased precipitously. Because the patient was pacemaker dependent, we elected to change the mode of the pacemaker from DDD to VOO, using the precordial magnet. However, this failed to prevent a recurrence of the events when the ESU was once again activated. Failure of a precordial magnet to change a VVI pacemaker to VOO mode in the surgical setting was described previously. However, in that case, failure of the magnet occurred because the pacemaker had inadvertently been left in a noninvasive threshold testing mode, designated as “vario” by the manufacturer, when the patient was seen in the pacemaker clinic before his surgery. In the “vario” mode, application of a precordial magnet will not convert the pacemaker from VVI to VOO. The Teletronics Model 1524 pacemaker present in our patient does not have a “vario” mode. In addition, our patient’s pacemaker clinic records clearly documented that the pacemaker was in the appropriate DDD mode, and the problem persisted despite appropriate reprogramming of the pacemaker to the VOO mode by the cardiologist.

The events described were not due to technical pacemaker failure, because the pacemaker was subsequently found to be operating within company specifications. This pacemaker, similar to all dual-chambered Teletronics pacemakers, is equipped with a unique, high-voltage protection circuit (fig. 1). This circuit monitors

![Fig 1. Schematic diagram depicting the high-voltage protective circuit. When the protective circuit detects high voltage, switches open (arrow A), which prevents conduction of retrograde currents into the pacemaker. This prevents irreparable damage to the pacemaker's output circuitry. However, this also prevents electrical capture of the heart. When high voltages are no longer detected, the switches close, which allows current to leave the generator and once again capture the heart.](http://anesthesiology.pubs.asahq.org/pdfaccess.ashx?url=/data/journals/jasa/931824/ on 04/03/2017)
the voltages continuously in the electrodes and the case of the generator itself. High voltages monitored include those induced by the ESU or by defibrillator shocks. Once a high voltage, exceeding manufacturer’s threshold, is detected, electronic switches open, effectively disconnecting all electrodes (and the case) from the pacemaker output circuitry. This protects the components of the pacemaker circuitry from these high voltages, which prevents permanent pacemaker damage but also prevents effective output from the pacemaker. In the presence of the EMI from the ESU, the impulses emanating from the pacemaker could not exit and conduct down the pacemaker lead. This led to an apparent and effective pacing failure in the VOO mode even though the pacemaker’s output circuitry was in the VOO mode. This can lead to temporary pacemaker failure, as demonstrated by our case.

All pacemaker manufacturers use a strategy to prevent or limit retrograde currents from entering the pacemaker. The specific circuitry involved varies from company to company. Most companies use a series of zener diodes to prevent retrograde currents."""" This arrangement will theoretically allow an operational VOO mode even in the presence of EMI. However, it also makes possible, in the presence of very high induced voltages, retrograde currents, and, therefore, possible permanent pacemaker damage. The circuit design depicted in figure 1 can lead to temporary operational failure of the VOO mode in the presence of powerful EMI, as it did in our case, but will prevent possible permanent pacemaker damage. Because of the absence of clinical outcome data, each manufacturer determines which tradeoffs to make and, therefore, which circuit design to incorporate into their devices.

A few lessons can be learned from this case. First, in the presence of EMI from a unipolar ESU, a precordial magnet will not effectively convert all pacemakers to a functional VOO mode even when the pacemaker’s output circuitry is functioning properly. Second, even reprogramming this type of pacemaker to the VOO mode will not guarantee continuous pacing in the presence of powerful EMI. We recommend that, if the pacemaker output is inhibited by the ESU, and inhibition results in asystole, a precordial magnet should still be tried, because, without knowledge of pacemaker brand and model, it cannot be known a priori whether the precordial magnet will work. If the use of the precordial magnet still leads to pacemaker inhibition with ESU activation, the anesthesiologist should consider whether brief periods of asystole might be tolerated, provided there was sufficient time for recovery between periods when the surgeon was not using the ESU. If this approach is not feasible, the position of the return plate should be changed. This, conceivably, could change the voltage vector, leading to a smaller amount of current being conducted retrograde by the pacemaker lead. Another strategy that could be tried is to decrease the power of the ESU. If these maneuvers fail to reverse the inhibition of output of the pacemaker, the anesthesiologist and surgeon should consider completion of the operation by using a bipolar ESU. Afterwards, the pacemaker should be interrogated electronically to ensure proper function and programmed settings.

There are two heat-generating surgical scalpels that likely will not interfere with pacemaker function. These devices are now not commonly used. However, anesthesiologists should be aware of them, because they offer possible alternatives to dealing with the problem described in our case. One device is the Shaw Hemostatic Scalpel (Hemostatix Medical Devices, Cherry Hill, NJ). It works by true cautery but is electrically insulated from the patient by a Teflon (E.I. DuPont de Nemours Co., Wilmington, DE) coating. The other is the Ultracision Harmonic Scalpel (Ultracision, Smithfield, RI). This device generates heat for cutting and coagulation by vibrating at a high frequency. The harmonic scalpel, therefore, relies on mechanical, not electrical, energy to cut and coagulate and has been used successfully in a pacemaker-dependent patient.

More than 10 yr ago, Shapiro et al., in concluding his article on intraoperative pacemaker failure wrote that “it is impossible to know how every pacemaker functions.” Because of their growing complexity at the time, he called for industry-wide standardization of all aspects of pacemaker technology. In the ensuing 10 yr, pacemaker technology has grown even more complex, and Shapiro’s sentiments are as true now as they were then.

References


Asystole and Severe Bradycardia during Epidural Anesthesia in Orthopedic Patients

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IN a number of studies1-3 and case reports,4,5 researchers have described bradycardia and asystole during spinal anesthesia. Proposed physiologic mechanisms have been discussed, and clinical factors that contribute to these events, including hypoxemia6,7 and lack of vigilance,8-9 have been described. Factors associated with the development of bradycardia during spinal anesthesia have been studied by Carpenter et al.2 and Liu and colleagues.10 These include low resting heart rate, use of beta blockers, American Society of Anesthesiologists physical status 1, peak block height, and first degree atrio-ventricular block.

In contrast, there has been little reported on the occurrence of bradycardia and asystole during epidural blockade.11,12 This is a report of 7 cases of severe bradycardia and 5 cases of asystole that occurred during orthopedic surgery under epidural anesthesia during the past 9 yr at our institution. These include one case of asystole and one case of severe bradycardia that occurred in the post anesthesia care unit (PACU). Although this report does not provide data on the incidence of bradycardia, these individual cases provide the greatest experience on patterns of onset of acute bradycardia and asystole during epidural anesthesia yet published, and may provide insight into potential etiologic mechanisms.

Methods

Institution

The Hospital for Special Surgery is an elective orthopedic surgical hospital that currently performs approximately 9,000 operations per year. Since 1987, between 80% and 85% have been performed using regional anesthesia, with 4,000-6,000 operations per year being performed using either spinal or epidural anesthesia. Total knee replacements and total hip replacements (THRs), as well as a variety of arthroscopically assisted knee procedures, are operations commonly performed during epidural anesthesia. Whereas tourniquets are often used for procedures that involve the knee, controlled hypotension is routinely used for THRs. The patients are given an epidural block that extends to the upper thoracic dermatomes, whereas cardiac output and mean arterial pressure (MAP) are maintained using an inotropic agent or vasopressor, usually epinephrine.13,14 If MAP does not decrease to an appropriate level to allow for a dry operative field, a vasodilator such as sodium nitroprusside (SNP) may be added, to augment the hypotension.

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