Anesthetic implications of implanted pacemakers: A case study

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Pacemakers and the underlying pathophysiology leading to their implantation present challenges to the anesthetist. This case report discusses the anesthetic management of a patient with an implanted pacemaker.

A case is presented of an 80-year-old female who was found unresponsive in a parking lot. She was diagnosed with a right parietal subdural hematoma. She underwent an emergency craniotomy for evacuation of the hematoma. Upon placing the patient on an electrocardiograph, a ventricular paced rhythm was revealed.

Pacemakers are most frequently implanted to initiate electrical activity in a heart unable to maintain its own automaticity. Patients with pacemakers may be unable to react to situations requiring increased cardiac outputs by increasing the heart rate. Depending upon the type of pacemaker present, patients may suffer from diminished cardiac output due to the loss of the "atrial kick." Pacemakers can also be affected by electromagnetic interference from devices used during surgery, particularly the electrosurgical unit. This paper discusses anesthetic management in individuals with implanted permanent pacemakers.

Key words: Electrosurgery, heart block, pacemaker.

Introduction
In 1981 about 118,000 new implantations of permanent cardiac pacemakers were performed. In 1986, there were approximately 350,000 people with pacemakers in the United States. The growing use of pacemakers increases the likelihood that the anesthetist will be required to manage anesthesia for a patient with an implanted pacemaker. In general, the purpose of the pacemaker is to initiate electrical-mechanical activity in a heart that is unable to maintain its own automaticity. There is a wide range of indications for pacemaker insertion. The loss of automaticity may be partial or total, but in any case renders the myocardium incapable of meeting or maintaining the physiologic needs of the patient.

There are several forms of cardiac pacing available. Sites of pacing for modern devices include either the atrium, the ventricle, or both. Pacemakers are also expected to monitor and react to the intrinsic activity of either or both chambers. In addition to the variation in which chamber is paced, it is possible for some pacemakers to sense the needs of the patient's physiological state and to adjust to increasing cardiac output requirements.

The surgical environment presents several opportunities for disturbing pacemaker function and presents a threat to homeostasis of patients under anesthesia. Electrical, mechanical, and chemical considerations may affect pacemaker function. The patient with a permanent cardiac pacemaker may have other pathologic processes which further add to the complexity of their anesthetic management. Fifty percent of permanently paced patients have coronary artery disease, 20% have hypertension, and 10% are diabetic. In order to ensure a positive anesthetic outcome for the patient with a permanent pacemaker, it is essential that the anesthetist be familiar with the anesthetic implications of pacing and be able to monitor and maintain pacemaker function under surgical and anesthetic conditions.

This discussion presents the case of a patient with a permanent pacemaker presenting with severe pathology. Indications for and types of commonly used permanent pacemakers are reviewed.
Preoperative preparation, intraoperative management, and postoperative considerations for the patient with a permanent pacemaker are discussed. The focus is to enable the practice of practical and effective anesthetic management of the patient with a permanent pacemaker.

Case report

An 80-year-old white female weighing approximately 60 kg presented to the emergency department of a level one trauma center after reportedly falling against a curb in a parking lot. She was unresponsive on admission, and endotracheal intubation was performed in the emergency room. Pancuronium was administered after the endotracheal tube was inserted. An emergency computed tomography scan revealed a right parietal subdural hematoma, and the patient was brought immediately to surgery for an emergency craniotomy.

The patient was unresponsive on arrival to the operating room. Placement of electrocardiogram monitors revealed a ventricular paced rhythm with a rate of 70 per minute. Initial blood pressure was 210/76 mmHg. The patient was also noted to have 3+ pitting edema of the lower extremities. Anterior-posterior chest x-ray obtained from the emergency room was remarkable for an enlarged cardiac shadow with a pacemaker generator at the left shoulder with a single lead through the subclavian vein to the right ventricle.

After confirming appropriate endotracheal tube placement, the patient was placed on mechanical ventilation, and the anesthetic was administered with isoflurane 1% and intravenous fentanyl. Pipecuronium was chosen to continue neuromuscular blockade. A left radial arterial catheter and right subclavian central venous access were inserted. Fifty grams of mannitol were administered to reduce intracranial pressure, and slow administration of one gram of phenytoin was begun.

The patient's pacemaker-dominated rhythm, cardiomegaly, and physical appearance suggested the presence of severe cardiovascular compromise. A pulmonary artery catheter was placed via the subclavian access. A pulmonary artery catheter with pacemaker capability was not used. There was concern that a pulmonary artery catheter might entangle and dislodge the pacemaker lead. Initial pulmonary artery pressure was 33/18 mmHg and right atrial pressure was 13 mmHg. Attempts to obtain pulmonary capillary wedge pressure failed. Cardiac output was 3.0 L/min with an estimated cardiac index of 1.8 L/min/m².

An attempt was made to improve cardiac output by stimulating intrinsic rhythm. Placement of a magnet over the pacemaker generator had no effect on rate or rhythm. While considering other options of hemodynamic support, the patient's blood pressure fell abruptly to 80/40 mmHg. Inhalation anesthetic was discontinued. An intravenous bolus of 50 µg epinephrine was administered and a dopamine infusion initiated. Fluid resuscitation consisted of crystalloid, hetastarch, and packed red blood cells. With dopamine infusion at 10 µg/kg/min, the right atrial pressure decreased to 8 mmHg, pulmonary artery pressure was 24/13 mmHg, cardiac output improved to 4.7 L/min (cardiac index 2.8), and systemic blood pressure was held at 95/45 mmHg. The electrocardiogram continued to reveal a ventricular paced rhythm with no evidence of intrinsic activity. The surgical team reported severe bleeding and volume resuscitation was continued with crystalloid and packed red cells. Dobutamine was added to the inotropic support at a rate of 10 µg/kg/min. This resulted in an improvement of cardiac output to 5.4 (cardiac index 3.2) and a systemic blood pressure of 124/55 mmHg. Systemic blood pressure continued to improve and dopamine was discontinued.

Use of bipolar electro surgery had no apparent effect on pacemaker function. Although electrical interference from electrosurgery obscured the electrocardiogram monitor, the arterial line waveform continued to display morphology which indicated rate and pressure. There was discussion among the surgical and anesthesia personnel as to whether using short bursts of electrosurgery would be less likely to affect pacemaker function than continuous use. A right parito-temporal craniotomy was performed, the subdural hematoma evacuated, and the intracranial bleeding was controlled. The patient was transferred to the neurology intensive care unit in critical condition. Fluid resuscitation totaled 4 U of packed red blood cells, 1,000 mL of hetastarch, and 10.5 L of crystalloid.

Significant factors in the management of this case included the lack of opportunity to investigate patient history preoperatively and hesitancy on how to proceed with management of the pacemaker dependent patient. There was concern for the possible dislodging or tangling of the pacemaker wire in situ by placement and subsequent removal of the pulmonary artery catheter. Some personnel were unclear about the effects of electrosurgery on pacemaker function. Familiarity with principles of cardiac pacing and anesthetic management considerations would have been helpful in the decision making process.

Discussion

In the normal human heart, it is the role of
the conduction system to initiate and propagate electrochemical action potentials. The anesthetist should be familiar with the normal anatomy and physiology of the cardiovascular system.

It is important for the anesthetist to understand the underlying pathologies that warrant permanent pacemaker implantation. Indications for permanent pacemakers have been grouped into three classifications. A Class I condition is one for which it is generally accepted that permanent pacemaker implantation is indicated. A Class II condition is one for which permanent pacemakers are frequently used but there is divergence of opinion with respect to their necessity. For patients with a Class III condition, it is generally accepted that pacemaker implantation is not necessary. It is the responsibility of the cardiologist to evaluate the necessity of permanent pacemaker implantation. Common indications for cardiac pacing include atrioventricular block, fascicular block, and sinus node dysfunction. The decision to proceed with pacemaker implantation may be influenced by cardiovascular pathology, drug therapy, or correlation of symptoms to electrocardiographic abnormalities. (Table I).

An uncommon pathology which may merit permanent pacemaker implantation is hypersensitive carotid sinus syndrome. It is defined as syncope resulting from an extreme baroreceptor reflex response to carotid sinus stimulation. Baroreceptors in the carotid sinus are normally stimulated by increases in blood pressure. This stimulation causes a reflex activation of the parasympathetic autonomic system. Vagal discharge results in a decreased chronotropic and inotropic state of the myocardium. The reflex may be manifested by either sinus arrest or block of greater than 3 seconds or a substantial symptomatic decrease in blood pressure.

- **Types of pacemakers.** Functionally, a pacemaker may be thought of as a simple electrical circuit. The components of the circuit are the generator, leads, and heart chamber to be paced. The generator is the power source. The lead is a wire for conducting energy to the heart. The heart chamber to be paced may be thought of as a light bulb. All electrical circuits have positive and negative poles which imply a direction of electron flow. If there are two leads, positive and negative, in contact with the heart chamber, it is called bipolar pacing. Bipolar pacing is a typical circuit used for temporary pacing with an external pacemaker generator. For permanent transvenous pacemakers, unipolar pacing is most common. One lead, which is implanted in the heart chamber, has the function of providing the source of stimulation, or the negative pole. The metal casing of the generator itself serves as the positive, or grounding pole.

Stimulation threshold is the minimal amount of current or voltage that will cause cardiac muscle contraction. Capture is the ability of each pulse delivered to the heart by the pacemaker to initiate a myocardial contraction. Estimates of threshold may be determined by a pacing system analyzer.

### Table I

<table>
<thead>
<tr>
<th>Acquired atrioventricular block</th>
<th>Class I</th>
<th>Class II</th>
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<tbody>
<tr>
<td>Symptomatic complete 3rd degree block</td>
<td>Asymptomatic complete 3rd degree block with pulse ≥40</td>
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<tr>
<td>Symptomatic bradycardia with type II 2nd degree block</td>
<td>Asymptomatic type II 2nd degree block</td>
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<tr>
<td>Atrial dysrhythmia with advanced or complete block</td>
<td>Intra- or infra-Hiss type I 2nd degree block</td>
<td></td>
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<tr>
<td>Atrioventricular block with any bundle branch block associated with myocardial infarction</td>
<td>Bifascicular or trifascicular associated with syncope that is not proved due to heart block but in the absence of other causes of syncope</td>
<td></td>
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<tr>
<th>Fascicular block</th>
<th>Class I</th>
<th>Class II</th>
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<tbody>
<tr>
<td>Symptomatic bifascicular or trifascicular associated with 2nd degree block or complete block</td>
<td>Sinus node dysfunction with symptomatic bradycardia</td>
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<table>
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<tr>
<th>Sinus node dysfunction*</th>
<th>Class I</th>
<th>Class II</th>
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<tbody>
<tr>
<td>Sinus node dysfunction with symptomatic bradycardia</td>
<td>Asymptomatic sinus node dysfunction with pulse ≤40</td>
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*Includes dysfunction induced by drug therapy for which there is no acceptable alternative.
critical point of resistance in the pacemaker system is the point of interface between the electrode of the pacemaker and the myocardium. Resistance will increase if the electrode is attempting to stimulate damaged or infarcted myocardial tissue.⁷

The primary function of early pacemakers was to stimulate the myocardium. There was no ability to sense and accommodate for the presence of a native rhythm. The pacemaker continuously initiated impulses independent of intrinsic activity. This type of pacing is referred to as asynchronous.³

Modern pacemakers have two basic functions: to monitor intrinsic depolarization and to initiate depolarization in its absence. A pacemaker that has the ability to sense and react to intrinsic heart activity is said to be in demand mode. How a pacemaker is intended to accomplish these functions is typically identified by a three letter code.³ With the advent of more complex programmable pacemakers, two letters were added, expanding the code to five letters.⁴ Table II defines and clarifies these codes.

An example of a frequently seen pacemaker is the VVI, or ventricular demand pacemaker.¹ The code indicates that the ventricle is the chamber paced and also the chamber sensed. The third letter indicates that the pacemaker is inhibited in response to sensing intrinsic ventricular activity at a preset rate. A pacemaker with VOO function would have no sensing or programmed response capability. The pacemaker would only deliver stimulation at its preset rate.

Dual chamber pacemakers are designed to preserve atrioventricular sequential contraction.³ Pacing leads are placed in the atrium and the ventricle. The preservation of sequential contraction allows atrial contraction to contribute to cardiac output. Pacemaker activity can be inhibited by either or both chambers. Either chamber can also be stimulated according to need. The dual chamber pacemaker implanted most commonly has a DDD mode.³ The DDD pacemaker is capable of AV sequential pacing, atrial pacing without ventricular pacing, ventricular pacing without atrial pacing, or no pacing.¹⁰

Among the newest trends in programmable pacemakers are pacemakers that can be programmed to respond to the changing physiological needs of the patient.¹¹ These are known as rate responsive pacemakers. Monitoring methods investigated to control pacemaker rate responses have included venous blood pH, QT interval, respiratory rate, venous blood temperature, and muscle activity.²,¹²

Demand pacemakers are provided with a basic program that allows them to revert to an asynchronous pacing mode (VOO). This ensures that the patient will not be left without pacemaker protection if the sensing function is interrupted. The rate for maximal preservation of cardiac index in resting individuals with ventricular pacing is between 70 and 90 beats per minute.¹³ Asynchronous pacing, however, presents risks to patients who have intrinsic cardiac rhythms. Ventricular fibrillation from R on T phenomenon or loss of atrial kick due to pacemaker stimulation are possible, although unlikely.¹²

Anesthetic consideration

- Preoperative considerations. Preoperative evaluation is an essential component in the anesthetic management of the patient with a permanent pacemaker undergoing elective surgery. A proper evaluation anticipates intraoperative problems and provides the basis for administering a safe anesthetic. In addition to pacemaker management considerations, patients in this population have a high incidence of coronary artery disease, hypertension, and diabetes.⁴ Careful history and physical evaluation should focus on optimization of the patient's condition.

The patient should be specifically questioned about the medical history of the cardiac condition which led to the implantation of his or her pacemaker. The original conduction problem and its etiology and presentation should be identified.

<table>
<thead>
<tr>
<th>Table II Pacemaker codes⁸</th>
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<tbody>
<tr>
<td><strong>Letter 1</strong> (pacing)</td>
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<tr>
<td>A Atrium</td>
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<tr>
<td>V Ventricle</td>
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<tr>
<td>D Dual</td>
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<td>O None</td>
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The patient's current cardiac status including any recurrence of symptoms such as dizziness or fainting since implantation requires investigation. The patient should be questioned about the type of pacemaker that he or she has. If unable to answer questions concerning the pacemaker, the patient may be able to produce an identification card. The identification card will identify the manufacturer, type of generator, date of implantation, and may provide helpful information about the indications for implantation.

Systems review should include evaluation of exercise tolerance, signs of congestive heart failure, respiratory disease, and renal dysfunction. The patient's medications may include digitalis, diuretics, beta blockers, antiarrhythmic agents, and antihypertensives. Routine endocarditis prophylaxis is unnecessary for patients with permanent pacemakers but may be considered in the presence of other indications.

Evaluating pacemaker function in the preoperative period is a primary concern. Pacemakers should be checked at least annually. Consultation with the cardiology department will contribute to pacemaker evaluation. If the patient requires only occasional pacemaker function, it is necessary to determine that the pacemaker is functional should the need arise. Demand pacemakers should not show activity on the electrocardiogram if intrinsic rhythm is adequate. The ability to sense generally fails before the ability to pace. The chest x-ray will confirm continuity of the pacemaker leads from the generator to the appropriate heart chambers. It has been suggested that if the chest x-ray shows no lead breaks, and the pacemaker is less than 2 years old, the generator is probably functioning properly.

Knowing the path of the leads will help the surgeon to avoid them during surgery. An x-ray that views the generator may reveal manufacturer and model. Electrocardiogram will confirm the pacemaker's mode of operation if the patient is pacemaker dependent. A pacemaker spike will occur immediately prior to each QRS complex for a ventricular pacemaker. A properly capturing pacemaker should also be confirmed by palpation of the patient's pulse. If the pacemaker is capturing effectively, there will be a pulse felt in association with each pacemaker impulse. The spike will be associated with the P wave for atrial pacemakers. Both P and QRS complexes will be preceded by spikes in atrioventricular sequential pacemakers.

Asynchronous pacing in the presence of a competing intrinsic rhythm can be hazardous. The impulse initiated by the pacemaker produces consequences similar to the early depolarization of a premature ventricular contraction. Early depolarization decreases ventricular filling time and will reduce stroke volume. A premature ventricular contraction (PVC) occurring during the relative refractory period of the ventricle can precipitate ventricular tachycardia or fibrillation. Ventricular tachycardia has been documented resulting from a pacemaker impulse occurring on a T wave. Placement of a magnetic field over the pacemaker causes the generator to revert to asynchronous pacing. The implications of magnetic fields on pacemakers will be discussed in the section on "Intraoperative Considerations."

Blood chemistry values can affect pacemaker function. Severe cases of alkalosis and acidosis will cause increases in the threshold voltage and current requirements of ventricular stimulation. Acute hypokalemia caused by hyperventilation or diuretic therapy causes an increase in threshold and could lead to loss of capture. Acute hyperkalemia increases myocardial irritability and raises the possibility for ventricular dysrhythmias, especially in the ischemic heart.

- Intraoperative considerations. In choosing an anesthetic technique for the patient with a permanent pacemaker, the anesthesiologist should consider requirements for the proposed surgery and the patient's underlying disease. Regional and general anesthetic techniques have been used successfully.

Spinal or epidural anesthesia must be planned carefully. Disadvantages may be encountered due to loss of sympathetic tone. Anesthesia of autonomic nerve fibers arising from T1-T4 dermatomes will result in total preganglionic sympathetic blockade. Vasodilation will cause a decrease in venous return and systemic vascular resistance. Loss of cardioaccelerator fibers results in bradycardia. These events may cause a patient who has an intrinsic rhythm prior to anesthesia to become hypotensive and pacemaker dependent.

All potent inhalational anesthetics cause dose related reductions in cardiac output and mean arterial pressure. Inhalational anesthetics play a part in pacemaker syndrome, which is discussed below. The anesthesiologist should choose intravenous agents that best maintain the patient's cardiovascular stability. Potent opioids can be used as adjuvants to inhaled anesthetics to blunt circulatory responses to laryngoscopy for intubation or sudden changes in the level of surgical stimulation.

Neuromuscular blockade may be instituted as appropriate for the procedure. Loss of pacemaker capture has been reported after the administration of succinylcholine. The myopotentials caused by muscle fasciculations may be sensed by the pacemaker and result in inhibition. If suc-
nylcholine is to be used, the anesthetist should consider preadministration of a nondepolarizing agent to reduce fasciculations.

Although the presence of a permanent pacemaker may not justify invasive monitoring, such monitoring modalities may be indicated due to related considerations such as cardiovascular disease. A permanent pacemaker is not a contraindication for pulmonary artery catheterization if the patient would benefit from pulmonary artery monitoring. Dislodgement of an endocardial electrode due to pulmonary artery catheter manipulation is unlikely if the pacemaker has been in place for more than 4 weeks. The electrocardiogram will be obscured by interference caused by the use of electrosurgery. It is important to remember the value of a pulse oximeter, stethoscope, and palpation of the pulse when evaluating rhythm and pressure. An arterial line may be valuable because it allows immediate evaluation of rate and pressure during electrosurgery use. In the patient with a demand pacemaker and intermittent intrinsic rhythm, an arterial line will reveal mean arterial pressure differences between native rhythm and paced rhythm.

- **Pacemaker syndrome.** Pacemaker syndrome is a phenomenon that primarily occurs in patients with ventricular pacemakers. The awake patient may experience syncope, breathlessness, postural hypotension, and other symptoms associated with a low cardiac output and loss of blood pressure. The syndrome is associated with intact retrograde conductive pathways between the ventricles and atria. This allows asynchronous contraction of the atria following stimulation of the ventricles. Studies have demonstrated that VVI pacing in the presence of intact ventriculoatrial conduction causes significant decreases in mean arterial pressure when compared to sinus rhythm.

Two mechanisms have been proposed to explain the occurrence of pacemaker syndrome. These mechanisms are loss of atrial contribution to preload and diminished peripheral vascular resistance. For the individual with good myocardial compliance, loss of the atrial contribution causes little difficulty. In the heart with poor compliance, atrial contraction contributes a significant volume to ventricular filling and its loss may drastically reduce cardiac output. The second proposed mechanism in creating pacemaker syndrome focuses on retrograde conduction. In the normal individual, a decrease in cardiac output will result in a reflex increase in peripheral vascular resistance. Increased resistance minimizes any loss of perfusion pressure due to decreased flow. This reflex compensatory mechanism is lost in some pacemaker patients. Atrial contraction against a closed atrioventricular valve during ventricular systole increases atrial pressure. The increase in atrial pressure is thought to initiate a paradoxical reflex that prevents the compensatory increase in peripheral vascular resistance. If central venous pressure is monitored, increased atrial pressure can be seen in the appearance of cannon waves in the right atrial tracing during ventricular pacing.

Because of the vasodilating effects of potent inhaled anesthetics, pacemaker syndrome in the anesthetized patient will be more significant than in the awake patient. A change from intrinsic sinus rhythm to ventricular pacing could herald a profound decrease in blood pressure. Measures should be taken to preserve sinus rhythm in the patient who is not completely pacemaker dependent.

Preservation of intrinsic rhythm of the patient with a demand pacemaker is achieved by preventing bradycardia. This can be accomplished through judicious use of anticholinergic drugs such as atropine or glycopyrrolate. A potent beta agonist such as isoproterenol may be considered. Adrenergic stimulation, however, may be a hazardous undertaking. It is important to remember that, in addition to increasing heart rate, beta agonists have potent inotropic and vasodilatory effects. This causes an increase in myocardial oxygen requirements and a decrease in systemic vascular resistance. Ischemia, myocardial infarction, or dysrythmias may result.

- **Electromagnetic interference.** A commonly known intraoperative problem in managing the patient with a permanent pacemaker involves electromagnetic interference (EMI). EMI has been blamed for ventricular fibrillation, myocardial damage, and increases in stimulation threshold leading to loss of capture.

Sources of EMI can be classified as direct and indirect. Indirect EMI refers to electrical interference that is propagated through the air due to induction of current. Examples of indirect EMI sources are orthopaedic saws and telemetry equipment. Most forms of indirect EMI have no effect on the permanent pacemaker. Direct EMI occurs when the patient’s body is part of the electrical current path, such as with the use of electrosurgery.

Two types of electrosurgery are commonly used. In a unipolar system, high frequency electrical current from a single electrode is directed at a point of contact of tissue. After leaving the electrode, the current spreads out and is conducted through the body to a return plate, commonly and incorrectly referred to as the grounding pad. The return plate completes the circuit back to the elec-
trosurgery unit. In bipolar electrosurgery, the current flows between two small electrodes that are positioned close together in the tip of the electrosurgical probe.\(^9\)

A pacemaker may interpret EMI as intrinsic cardiac activity, thus inhibiting the pacemaker from initiating a heart beat.\(^9,14,26\) The best strategy for controlling inhibition of a pacemaker due to EMI is prevention of the event. Bipolar electrosurgery is an effective way to reduce the incidence of pacemaker inhibition due to EMI. Because of the proximity of the electrodes to each other, the electrical field size is minimized, therefore decreasing the possibility of interference with the pacemaker's sensing abilities.\(^24\) Bipolar use may not be suitable because it is low powered and generally used only for small bleeding sites.\(^9\) If monopolar electrosurgery must be used, the lowest effective power setting should be used. The return electrode pad should be placed away from the pacemaker generator and close to the site of electrosurgery use.\(^9,14\)

Attempts at prevention of pacemaker inhibition due to EMI may prove ineffective. In this situation one commonly employed tactic to defeat inhibition of a pacemaker during electrosurgery is positioning a strong magnet over the pacemaker generator.\(^23\) The consistent magnetic field caused by the magnet will induce most demand pacemakers to convert to an asynchronous mode.

There are several considerations that suggest that magnet placement is not an advisable practice to employ in all cases.\(^9\) If the patient is not pacemaker dependent, an asynchronous mode will compete with the intrinsic rhythm and may result in early depolarization, decreased stroke volume, or ventricular tachycardia. Additionally, for some types of pacemakers, application of a magnetic field is a step required to initiate reprogramming of the generator. Phantom or random reprogramming or programmable pacemaker generators when exposed to magnetic fields and electrosurgery has been documented to cause changes in rate and inappropriate modes of operation.\(^28\) As with inhibition, use of bipolar electrosurgery poses less risk of reprogramming than monopolar electrosurgery because bipolar electrosurgery generates a relatively smaller EMI field.\(^25\)

Use of electrosurgery in short bursts of energy is a method of preventing inhibition that has been advocated.\(^9\) This tactic offers no advantage over continuous electrosurgery use unless the short bursts are less than one second in duration and are followed by 5 to 10 second pauses to allow resumption of cardiac rhythm.\(^14,24\) If there is a continued problem, reprogramming the pacemaker to an asynchronous mode should be considered prior to surgery, thus avoiding the possibility of random reprogramming.\(^9,12,21\) There is a risk of inducing a serious tachycardia when using asynchronous mode in a patient with an intrinsic rhythm; however, modern pacemakers use a small amount of current and present less risk of this than older models did.\(^12\)

Strong sources of EMI are capable of inducing electrical current in the pacemaker lead.\(^7,26\) Bipolar electrosurgery minimizes the risk of magnetic inductance. Minimizing monopolar electrosurgery risk is achieved by positioning the return electrode so that the current from the electrosurgical tool passes through the body perpendicular to the path of the pacemaker electrode.\(^9\) This prevents the vector of energy passing through the body from paralleling to the pacemaker lead, decreasing the likelihood of forming an inductive relationship between the EMI source and the lead.\(^7\)

Considerations for a permanent pacemaker during cardiopulmonary resuscitation focus on preserving the pacemaker's ability to function in the postresuscitative period. During defibrillation, a large amount of energy may shunt to the heart through the pacemaker electrode, causing endocardial burns.\(^7\) Acute and chronic elevations in stimulation threshold occur, resulting in loss of capture, thus, the lowest effective voltage should be used in defibrillation or cardioversion.\(^7,14,17\) Defibrillator paddles should be placed as far from the pacemaker generator as possible, and anterior-posterior paddle orientation is recommended if it is available.\(^7,17\)

Patients who have rate-responsive pacemakers may experience changing heart rates during general anesthesia.\(^30\) Anesthetic considerations vary depending on the type of parameters used to determine pacemaker rate. For the patient with a pacemaker sensitive to muscle activity, postoperative shivering may cause an increase in heart rate. A pacemaker that responds to changes in blood temperature is prone to inappropriate pacing rates in response to rapid infusion of warmed intravenous solutions or hypothermia.\(^30\) Pacemaker-induced tachycardia has been reported in a patient with a pacemaker programmed to respond to respiratory patterns; hyperventilation during general anesthesia produced an increase in heart rate. Misinterpretation of the response resulted in inappropriate treatment.\(^31\) In the event of intraoperative problems with pacing rates, rate-responsive pacemakers can be converted to fixed-rate pacing through the application of a magnet over the generator.\(^31\) If difficulty in management is predictable, such as planned hyperventilation, it is sug-

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gested that the pacemaker be reprogrammed preoperatively to exclude the rate-responsive function.30, 31

Postoperative considerations. Management of the permanent pacemaker patient undergoing surgery continues into the postoperative period. Continuous monitoring of the electrocardiogram and vital signs are essential in the anesthesia recovery area. The emphasis of postoperative management should be to ensure that the surgical procedure, especially electrosurgery use, has not resulted in undetected changes in pacemaker functioning. If the patient is not pacemaker dependent, changes in pacemaker function may not be detected until the demand mode is activated. Pacemaker settings should be checked by the cardiology department prior to the patient leaving the monitored area. Arrangements should also be made to check the pacemaker system stimulation threshold. If electrosurgery has caused myocardial damage, changes in threshold may not be apparent for 24 hours or more.2

Conclusion

Several different types of pacemakers are available. The evolution of technology has made multiple modes of operation possible. The increasing sophistication of pacemaker programs has made standardized management strategies inadequate. Commonly used techniques such as application of a magnetic field to counter electrosurgery interference can have variable results. Different types of pacemakers require individual consideration. In some settings, preoperative reprogramming of pacemaker settings may be indicated to facilitate anesthetic management. Proper anesthetic management of the patient with a permanent pacemaker involves evaluation, planning, and monitoring through the postoperative period.

REFERENCES


AUTHORS

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PRECAUTIONS Use of ZEMURON® (rocuronium bromide) injection for the purpose of attenuating some of the side effects of succinylcholine, has not been studied.

Labor and Delivery: The use of ZEMURON® (rocuronium bromide) injection in cesarean section has been studied in a small number of patients in the United States. Because fewer doses of ZEMURON® (rocuronium bromide) injection are required for rapid sequence induction in patients with normal hepatic function than in patients with hepatic dysfunction, the administration of ZEMURON® (rocuronium bromide) injection should only be used in this setting if, in the opinion of the prescribing physician, the specific advantages of the drug outweigh the risk.

Renal Failure: Due to the limited role of the kidney in the excretion of ZEMURON® (rocuronium bromide) injection, adjustment of dosage to the patient’s creatinine clearance is not advised. ZEMURON® 0.6 mg/kg has been evaluated in a limited number of patients (~20) with clinically significant hepatic disease under study. A decrease in ZEMURON® 0.6 mg/kg, from the usual starting dose of 0.5 mg/kg, to 0.1 mg/kg, was administered to patients with a creatinine clearance of 20 to 75 ml/min; 0.1 mg/kg was also given to patients with a creatinine clearance of <20 ml/min. The median duration of 3 mg was also prolonged in patients with creatinine clearance of <20 ml/min.

Hepatic Disease: Since ZEMURON® (rocuronium bromide) injection is primarily excreted by the liver, it should be used with caution in patients with clinical or significant hepatic disease. ZEMURON® 0.6 mg/kg has been shown to be effective in the treatment of patients with hepatic dysfunction. In patients with severe liver disease, the dosage requirement of ZEMURON® 0.6 mg/kg for induction of paralysis was increased to maintain adequate neuromuscular blockade. In patients with cirrhosis, ZEMURON® 0.6 mg/kg for induction of paralysis was increased to maintain adequate neuromuscular blockade. The mean duration of ZEMURON® (rocuronium bromide) injection was 28 minutes (17-51, n=12) without succinylcholine.

Malignant Hyperthermia (MH): In an animal study in MH-susceptible swine, the administration of ZEMURON® (rocuronium bromide) injection did not appear to trigger malignant hyperthermia. ZEMURON® (rocuronium bromide) is contraindicated in patients who have been shown to be clinically susceptible or have been shown to have high concentrations of MH-triggering agents.

Drug Interactions: The use of ZEMURON® (rocuronium bromide) injection before succinylcholine, for the purpose of attenuating some of the side effects of succinylcholine, has not been studied.

Anesthesia: ZEMURON® (rocuronium bromide) injection does not produce irritation or discoloration at the site of injection. A 1 mg/kg dose of rocuronium bromide was administered to patients chronically receiving anticholinergic drugs. The incidence of adverse cardiovascular reactions was no higher than that seen in control patients receiving placebo.

Injection Aesthetics: Use of inhalation anesthesia has been shown to enhance the activity of other muscle relaxants. The use of rocuronium bromide with thiopental sodium, a barbiturate, or with alfentanil or sufentanil, an opioid, has been shown to enhance the action of rocuronium bromide. It has been demonstrated that the use of alfentanil and halothane has been demonstrated. In one study, use of alfentanil in 10 patients resulted in a 20% increase in mean clinical duration of the initial intubating dose, and a 37% increase in the duration of subsequent doses. The use of rocuronium bromide in patients undergoing surgery for orthopedic procedures has been reported. The incidence of adverse cardiovascular reactions was no higher than that seen in control patients receiving placebo.

Antagonists: Antagonism of neuromuscular blockade may be delayed in the presence of debilitation, carcinomatosis, and concomitant use of certain drugs (e.g., calcium channel blockers, 5-hydroxytryptamine receptor antagonists, and corticosteroids). ZEMURON® (rocuronium bromide) injection should not be administered to patients chronically receiving anticholinergic drugs. The incidence of adverse cardiovascular reactions was no higher than that seen in control patients receiving placebo.

ADVERSE REACTIONS ZEMURON® (rocuronium bromide) injection is available in the following forms:

T M 0.6 mg/kg 10 mL multiple dose vials containing 100 mg rocuronium bromide injection (10 mg/mL) as that of prolonged neuromuscular blockade.

ZEMURON® (rocuronium bromide) injection should be stored under refrigeration (2°C to 8°C) and protected from light.

In the European studies, the most commonly reported adverse experiences were transient hypotension (2-3%), tachycardia (3-4%), and injection site pain (0.5-1%). No deaths, life-threatening, or unexpected adverse experiences were reported in the European studies. The incidence of adverse cardiovascular reactions was no higher than that seen in control patients receiving placebo.

OVERDOSE No cases of significant accidental or intentional overdose with ZEMURON® (rocuronium bromide) injection have been reported to date. Overdoses of ZEMURON® (rocuronium bromide) injection have not been studied in the United States. Only a limited number of cases of overdosage have been treated, and the time course of recovery from neuromuscular blockade is not known. The initial clinical signs and symptoms of overdosage are related to potentiation of the central nervous system actions of ZEMURON® (rocuronium bromide) injection. Treatment of overdosage is to support the vital functions as indicated. Supportive and symptomatic therapy is indicated.

HOW SUPPLIED ZEMURON® (rocuronium bromide) injection is available in the following forms:

ZEMURON® 5 mL multiple dose vials containing 50 mg rocuronium bromide injection (10 mg/mL) as that of prolonged neuromuscular blockade.

ZEMURON® 10 mL multiple dose vials containing 100 mg rocuronium bromide injection (10 mg/mL) as that of prolonged neuromuscular blockade.

Antagonists (such as neostigmine) should NOT be administered prior to the demonstration of some spontaneous recovery from neuromuscular blockade. The use of a NERVE STIMULATOR TO DOCUMENT RECOVERY AND ANTAGONISM OF NEUROMUSCULAR BLOCKADE IS INDICATED PRIOR TO ADMINISTRATION OF AN ANTI-MUSCLE RELAXANT AGENT (such as neostigmine, edrophonium chloride) in conjunction with a specific anti-cholinergic agent (such as atropine). The administration of atropine may be necessary to reverse residual effects of ZEMURON® (rocuronium bromide) injection.