Introduction

Patients with cardiac disease presenting for noncardiac surgery pose a considerable challenge to the anesthesiologists. With the availability of better medical facility and sophisticated diagnostic methods, many patients especially of the elderly age group, are detected to have electrophysiological disorders. Pacemakers are being used with greater frequency for both conduction and arrhythmia problems in such patients. Currently more than 5,00,000 patients in the United States have pacemakers and nearly 1,15,000 new devices are implanted each year. Although, no definite figures are available the number is also increasing in India. These patients may require one or more surgical procedures after receiving the pacemaker. Care of the pacemaker during surgery as well as understanding its anesthetic implications is crucial in the management of these patients. The perioperative management of patients with permanent pacemaker undergoing noncardiac surgery is discussed.

Cardiac pacing is one of the most reliable documented treatment for various cardiac arrhythmias, especially bradyarrhythmias since 1950. The initial pacing system consisted of a single lead asynchronous pacemaker, which paced the heart at a fixed rate. Over the years, the technological advances have revolutionised the pacemakers and currently more sophisticated multiprogrammable devices are available. In addition, automated implantable cardioverter defibrillators (AICD) have been designed to treat fatal tachyarrhythmias. With the availability of pacing devices to suit many conditions, potential indications for pacing are expanding. The American College of Cardiology /American Heart Association (ACC/AHA) established indications for permanent pacemaker or antitachycardia devices in 2002, which are depicted in table 1.

Table 1. Indications of permanent pacemaker implantation.

<table>
<thead>
<tr>
<th>1) Acquired AV block:</th>
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<tr>
<td>A) Third degree AV block</td>
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<tr>
<td>Bradycardia with symptoms</td>
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<tr>
<td>After drug treatment that cause symptomatic bradycardia</td>
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<tr>
<td>Postoperative AV block not expected to resolve</td>
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<tr>
<td>Neuromuscular disease with AV block</td>
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<tr>
<td>Escape rhythm &lt;40 bpm or asystole &gt; 3s</td>
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<tr>
<td>B) Second degree AV block</td>
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<tr>
<td>Permanent or intermittent symptomatic bradycardia</td>
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<tr>
<th>2) After Myocardial infarction:</th>
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<tbody>
<tr>
<td>Persistent second degree or third degree block</td>
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<tr>
<td>Infranodal AV block with LBBB</td>
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<tr>
<td>Symptomatic second or third degree block</td>
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<tr>
<th>3) Bifascicular or Trifascicular block:</th>
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<tbody>
<tr>
<td>Intermittent complete heart block with symptoms</td>
</tr>
<tr>
<td>Type II second degree AV block</td>
</tr>
<tr>
<td>Alternating bundle branch block</td>
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</tbody>
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<th>4) Sinus node dysfunction:</th>
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<tbody>
<tr>
<td>Sinus node dysfunction with symptoms as a result of long term drug therapy</td>
</tr>
<tr>
<td>Symptomatic chronotropic incompetence</td>
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<table>
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<tr>
<th>5) Hypertensive carotid sinus and neurocardiac syndromes:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Recurrent syncope associated with carotid sinus stimulation</td>
</tr>
<tr>
<td>Asystole of &gt;3s duration in absence of any medication</td>
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</tbody>
</table>

AV: atrioventricular, LBBB: Left bundle branch block,
Mercury–Zinc batteries that were used in the early days had a short useful life (2-3 yrs). Currently Lithium-iodine batteries are being used which have longer shelf life (5-10 yrs) and high energy density.

**Leads**

These are insulated wires connecting the pulse generator.

**Electrode**

It is an exposed metal end of the lead in contact with the endocardium or epicardium.

**Unipolar Pacing**

There is one electrode, the cathode (negative pole) or active lead. Current flows from the cathode, stimulates the heart and returns to anode (positive pole) on the casing of pulse generator via the myocardium and adjacent tissue to complete the circuit. Unipolar sensing is more likely to pick up extracardiac signals and myopotentials.

**Bipolar Leads**

They consist of two separate electrodes, anode (positive pole) and cathode (negative pole), both located within the chamber that is being paced. As the electrodes are very close, the possibility of extraneous noise disturbance is less and the signals are sharp.

**Endocardial Pacing**

It is also called as transvenous pacing which implies that the leads/electrodes system has been passed through a vein to the right atrium or right ventricle. It can be unipolar or bipolar.

**Epicardial Pacing**

This type of pacing is accomplished by inserting the electrode through the epicardium into the myocardium. This can also be unipolar or bipolar.

**Pacing Threshold**

This is the minimum amount of energy required.
to consistently cause depolarization and therefore contraction of the heart. Pacing threshold is measured in terms of both amplitude and duration for which it is applied to the myocardium. The amplitude is programmed in volts (V) or in milliampers in some devices, and the duration is measured in milliseconds. Factors affecting the myocardial pacing threshold are listed in table 3. However, only those factors important from the anaesthesia point of view will be discussed.

Table 3. Factors affecting pacing thresholds.

<table>
<thead>
<tr>
<th>Increase</th>
<th>Decrease</th>
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<tbody>
<tr>
<td>1-4 weeks after implantation</td>
<td>Increased catecholamines</td>
</tr>
<tr>
<td>Myocardial ischaemia/infaction</td>
<td>Stress, anxiety</td>
</tr>
<tr>
<td>Hypothermia, hypothyroidism</td>
<td>Sympathomimetic drugs</td>
</tr>
<tr>
<td>Hyperkalaemia, acidosis/alkalosis</td>
<td>Antiarrhythmics (class Ic, 3)</td>
</tr>
<tr>
<td>Antiarrhythmics (class IA/B, 2)*</td>
<td>Glucocorticoids</td>
</tr>
<tr>
<td>Severe hypoxia/hypoglycaemia</td>
<td>Hyperthyroidism</td>
</tr>
<tr>
<td>Inhalation-local anaesthetics**</td>
<td>Hypermetabolic status</td>
</tr>
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*possibly increase thresholds  
**conflicting evidence, probably dose-related

R Wave Sensitivity

It is the measure of minimal voltage of intrinsic R wave, necessary to activate the sensing circuit of the pulse generator and thus inhibit or trigger the pacing circuit. The R wave sensitivity of about 3 mV on an external pulse generator will maintain ventricle inhibited pacing.

Resistance

It can be defined as impedance to the flow of current. In the pacemaker system it amounts to a combination of resistance in lead, resistance through the patient’s tissue and polarization that takes place when voltage and current are delivered into the tissues. Abrupt changes in the impedance may indicate problem with the lead system. Very high resistance can indicate a conductor fracture or poor connection to the pacemaker. A very low resistance indicates an insulation failure.

Hysteresis

It is the difference between intrinsic heart rate at which pacing begins (about 60 beats/min) and pacing rate (e.g., 72 beats/min). It is particularly useful in patients with sick sinus syndrome.

Runaway Pacemaker

It is the acceleration in paced rates due to aging of the pacemaker or damage produced by leakage of the tissue fluids into the pulse generator. Treatment with antiarrhythmic drugs or cardioversion may be ineffective in such cases. It is necessary to change the pacemaker to an asynchronous mode, or reprogram it to lower outputs. If the patient is haemodynamically unstable temporary pacing should be done followed by changing of pulse generator.

Types of Pacing Modes

Asynchronous: (AOO, VOO, and DOO)

It is the simple form of fixed rate pacemaker which discharges at a preset rate irrespective of the inherent heart rate. It can be used safely in cases with no ventricular activity. However, the problems associated with asynchronous pacemaker are that it competes with the patient’s intrinsic rhythm and results in induction of tachyarrhythmias. Continuous pacing wastes energy and also decreases the half-life of the battery.

Single Chamber Atrial Pacing (AAI, AAT)

In this system atrium is paced and the impulse passes down the conducting pathways, thus maintaining atrioventricular synchrony. A single pacing lead with electrode is positioned in the right atrial appendage, which senses the intrinsic P wave and causes inhibition or triggering of the pacemaker. This is useful in patients with sinus arrest and sinus bradycardia provided atrioventricular conduction is adequate. It is inappropriate for chronic atrial fibrillation and long ventricular pauses.

Single Chamber Ventricular Pacing (VVI, VVT)

VVI is the most widely used form of pacing in which ventricle is sensed and paced. It senses the intrinsic R wave and thus inhibits the pacemaker function. This type of pacemaker is indicated in a
patient with complete heart block with chronic atrial flutter, atrial fibrillation and long ventricular pauses. Single chamber ventricular pacing is not recommended for patients with sinus node disease, as these patients are more likely to develop the pacemaker syndrome.

*Dual Chamber AV Sequential Pacing (DDD, DVI, DDI, and VDD)*

Two leads that can be unipolar or bipolar are used, one for the right atrial appendage and the other for right ventricular apex. The atrium is stimulated first to contract, then after an adjustable PR interval ventricle is stimulated to contract. These pacemakers preserve the normal atrioventricular contraction sequence, and are indicated in patients with AV block, carotid sinus syncope, and sinus node disease. In DDD system, both the atrium and ventricle can be sensed and paced. The advantages of dual chamber pacemaker are that they are similar to sinus rhythm and are beneficial in patients, where atrial contraction is important for ventricular filling (e.g. aortic stenosis). The disadvantage of dual chamber pacing is the development of a pacemaker-mediated tachycardia (PMT) due to ventriculoatrial (VA) conduction in which ventricular conduction is conducted back to the atrium and sensed by the atrial circuit, which triggers a ventricular depolarization leading to PMT. This problem can be overcome by careful programming of the pacemaker.

*Programmable Pacemaker*

This is being used since 1980. It provides flexibility to correct abnormal device behavior and adapt the device to patient’s specific and changing needs. The various factors, which can be programmed are pacing rate, pulse duration, voltage output, R wave sensitivity, refractory periods, PR interval, mode of pacing, hysteresis, and atrial tracking rate.

In patients with normal cardiac contractility, the stroke volume increases to its maximal point when only 40% of maximal activity is performed. Thus an increase in heart rate is important during exercise to achieve the peak cardiac output. Patients with fixed stroke volume such as those with dilated cardiomyopathy are not able to effectively increase cardiac output by increase in contractility. They depend entirely on their heart rate. Similarly, patients on pacemaker need to change the paced rate in proportion to the metabolic demand so as to normalize the haemodynamic status. Patients with “chronotropic incompetence” (atrial fibrillation, complete heart block) are unable to change the heart rate according to their metabolic demands. DDD, VVI, and AAI modes also cannot increase heart rate according to the metabolic demands in these patients. In such cases, rate responsive pacemakers (i.e. pacemakers, which not only sense the atrial or ventricular activity but also sense various other stimuli and thus, increase the pacemaker rate) are helpful. Various types of sensors have been designed which respond to the parameters such as vibration, acceleration, minute ventilation, respiratory rate, central venous pressure, central venous pH, QT interval, pre-ejection period, right ventricular stroke volume, mixed venous oxygen saturation, and right atrial pressure. Out of these, sensors capable of detecting body movements (vibrations), changes in ventricular repolarisation, central venous temperature, central venous oxygen saturation, respiratory rate and depth, and right ventricular contractility are commonly used in clinical practice.

*Pacemaker Syndrome*

Most individuals can compensate for the reduction in cardiac output due to loss of atrial systole by activation of baroreceptor reflexes that increase peripheral resistance and maintain systemic blood pressure. Some individuals, particularly those with intact retrograde VA conduction, may not tolerate ventricular pacing and may develop a variety of clinical signs and symptoms resulting from deleterious haemodynamics induced by ventricular pacing termed as pacemaker syndrome. These include hypotension, syncope, vertigo, light-headedness, fatigue, exercise intolerance, malaise, weakness, lethargy, dyspnoea, and induction of congestive heart failure. Cough, awareness of beat-to-beat variation of cardiac response from spontaneous to paced beats, neck pulsation or pressure sensation in the chest, neck, or head, headache, and chest pain are the other symptoms. Symptoms may vary
from mild to severe, and onset may be acute to chronic. The pathophysiology of pacemaker syndrome results from a complex interaction of haemodynamic, neurohumoral and vascular changes induced by the loss of AV synchrony. Patients with retrograde VA conduction are in a state of constant AV dys-synchrony. Retrograde VA conduction is present in about 15% of patients with complete antegrade AV block and in about 67% of patients with intact antegrade AV conduction paced for sinus node disease.

**Pacemaker Failure**

It may be due to generator failure, lead failure, or failure to capture. Failure to capture owing to a defect at the level of myocardium (i.e. the generator continues to fire but no myocardial depolarization takes place) remains the most difficult problem to treat.10

**Haemodynamic Changes During Pacing**

In single chamber pacemaker, atrial pacing increases the cardiac output by about 26% in comparison to ventricular pacing, as atrial contraction contributes 15 to 25% of preload to ventricles. Also atrial systole increases the coronary blood flow and decreases the coronary resistance.

The new AV sequential pacing results in 35 % increase in cardiac output in comparison to the single chamber pacing. This is achieved by the atrial systolic boost (atrial kick) to ventricular filling. While matching pacemaker to a patient, several factors need to be taken into consideration such as patient’s age, symptoms, cardiac rhythm, presence of underlying heart disease, ventricular function, and response of sinus node to activity (chronotropic response). BPEG have issued guidelines on the recommended pacing modes for all types of bradyarrhythmias requiring pacing (Table 4).11 In patients with atrioventricular block, the ventricle must be paced. Ventricular pacing should follow atrial pacing or sensing to maintain atrioventricular synchrony and cardiac output. If the patient is physically active and sinus node is chronotropically incompetent, a rate responsive system is advisable.

Table 4. British pacing and electrophysiology group recommended pacemaker modes.11

<table>
<thead>
<tr>
<th>Sinus node disease</th>
<th>Optimal</th>
<th>Alternative</th>
<th>Inappropriate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Optimal</td>
<td>AAIR</td>
<td>AAI</td>
<td>VVI, VDD</td>
</tr>
<tr>
<td>Alternative</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Inappropriate</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Chronic atrial fibrillation with atrioventricular block**

<table>
<thead>
<tr>
<th>Optimal</th>
<th>VVIR</th>
<th>DD, VVI (with hysteresis)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alternative</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Inappropriate</td>
<td>AAI, VVI, VDD</td>
<td></td>
</tr>
</tbody>
</table>

**Factors Important from Anaesthesia Point of View**

**Physiological**

During the first two weeks, there is an initial sharp increase in the pacing threshold i.e. up to ten times the acute level because of the tissue reaction around the electrode tip. Then it decreases to two to three times the acute level because of the scar formation. In chronic state, it reaches the initial level in 80% of patients. But this has become far less of a problem with the introduction of steroid-eluting leads and other refinements in the lead technology.1

**Potassium**

Its equilibrium across the cell membrane determines the resting membrane potential (RMP). In certain clinical situations, the RMP becomes less negative and approaches the membrane’s threshold potential so that less current density at the electrode tissue interface is required to initiate an action potential, making capture by the pacemaker easier. If the RMP becomes more negative, an increased
current density would be required to raise the RMP to the membrane threshold potential, making it more difficult for the pacemaker to initiate myocardial contraction. An acute increase in extracellular potassium concentration as in patients with myocardial ischaemia, rapid potassium replacement in chronic hypokaemic patients or use of depolarising muscle relaxants in patients with burns, trauma or neuromuscular disease may increase the RMP to less negative value, thus making the capture easier. Similarly, decrease in extracellular potassium (in patients on diuretic therapy or those undergoing hyperventilation such as neurosurgical patients) leads to more negative RMP making the capture difficult.7,9,12

**Myocardial Infarction**

Its scar tissue is unresponsive to electrical stimulation and may cause loss of pacemaker capture.7

**Antiarrhythmic Drug Therapy**

Class Ia (quinidine, procainamide), Ib (lidocaine, diphenylhydrantoine), and Ic (flecainide, encainide, propafenone) drugs have been found to increase the pacing threshold.13,14

**Acid Base Status**

Alkalosis and acidosis both cause increase in pacing threshold.14

**Hypoxia**

It causes increase in pacing threshold.12

**Anaesthetic Drugs**

These drugs are not likely to change the pacing threshold. It is notable that addition of equipotent halothane, enflurane, or isoflurane to opiate based anaesthesia after cardiopulmonary bypass did not increase pacing threshold.14

**Preoperative Evaluation**

Preoperative evaluation is an important aspect of the anaesthetic management of a patient with permanent pacemaker undergoing noncardiac surgery. It includes evaluation of the patient and the pacemaker. It should include not only detailed evaluation of the underlying cardiovascular disease responsible for the insertion of pacemaker, but also other associated medical problems. Since substantial number of these patients suffers from coronary artery disease (50%), hypertension (20%) and diabetis (10%),7 one should know the severity of the cardiac disease, the current functional status, and medication of the patient. The patient should also be questioned about the initial indication for the pacemaker and preimplantation symptoms such as lightheadedness, dizziness or fainting. If these symptoms occur even after the pacemaker insertion, cardiology consultation should be obtained.9 The general physical examination should be done to rule out the presence of any bruits, and signs of congestive heart failure. The location of the pulse generator should be noted. Generally, generator for the epicardial electrodes is kept in the abdomen and over one of the pectoris muscles for the endocardial electrodes.7 Routine biochemical and haematological investigations should be performed as indicated on an individual basis. A 12 lead electrocardiogram, X-ray chest (for visualization of continuity of leads) and measurement of serum electrolytes (especially K+) should be performed.

**Pacemaker Evaluation**

It is equally important to evaluate the function of pacemaker in the preoperative period. Assistance from the cardiologist and the manufacturer’s representative may be obtained for the purpose. Most of the information about the pacemaker, such as type of pacemaker (fixed rate or demand rate), time since implanted, pacemaker rate at the time of implantation, and half-life of the pacemaker battery can be taken from the manufacture’s book kept with the patient.

Ten percent decrease in the rate from the time of implantation indicates power source depletion. In patients with VVI generator, if intrinsic heart rate is greater than pacemaker set rate, evaluation of pacemaker function can be done by slowing down the heart rate by carotid sinus massage, while the patient’s ECG is continuously monitored.15
Carotid sinus massage should be done cautiously in patients with a history suggestive of cerebrovascular disease or carotid artery disease, because the atherosclerotic plaque may embolise to the cerebral circulation. Other methods to slow the heart rate are Valsalva manoeuvre and use of edrophonium (tensilone 5-10 mg).

Reprogramming the pacemaker is generally indicated to disable rate responsiveness. The AICD also needs to be disabled before anaesthesia. ACC/AHA guidelines advise that all antitachycardia therapy should be disabled before anaesthesia. If the risk of electromagnetic interference (EMI) is high, such as, when the electricity is in close proximity to the generator, alternative temporary cardiac pacing device should be available. The use of magnet may also be necessary.

**Effect of the Magnet Application on Pacemaker Function**

Magnet application is an extremely important function. The magnet is placed over the pulse generator to trigger the reed switch present in the pulse generator resulting in a non-sensing asynchronous mode with a fixed pacing rate (magnet rate). Magnet operated reed switches were originally incorporated to produce pacemaker behaviour that would demonstrate remaining battery life and sometimes pacing thresholds. Activation of the reed switch shuts down the demand function so that the pacemaker stimulates asynchronous pacing. Thus, magnets can be used to protect the pacemaker dependent patient during EMI, such as diathermy or electrocautery. However, not all pacemakers switch to asynchronous mode on the application of magnet. The response varies with the model and the manufacturer and may be in the form of no apparent change in rate or rhythm, brief asynchronous pacing, continuous or transient loss of pacing, or asynchronous pacing without rate response. It is advisable to consult the manufacturer to know the magnet response before use. The patient must be connected to an electrocardiograph recorder before the magnet is applied and, remain connected, until after the magnet is removed. The first few paced complexes after magnet application provide information regarding the integrity of the pulse generator and its lead system. A 10% decrease in magnet rate from the time of implantation indicates power source depletion and is an indicator of end of life requiring elective replacement of battery.

If no information is available from the patient about the pacemaker, magnet may identify the particular model with the help of magnet rate, which varies among different manufacturers and thus provide clue for its identification.

Despite the previous recommendations to have a magnet available in the operating room, routine use of magnet during surgery is not without risk and at times may be unjustified. Switching to asynchronous pacing may trigger ventricular asynchrony in patients with myocardial ischaemia, hypoxia, and electrolyte imbalance. The new generation pacemakers are relatively immune to magnet application and may not convert pacemaker to asynchronous mode. Constant magnet application over the pacemaker may alter the programming leading to either inhibited or triggered pacing, or may cause continuous or transient loss of pacing. It has also been seen that if a magnet is placed over a programmable pacemaker, in the presence of EMI, the pulse generator may become inadvertently and unpredictably reprogrammed. This new 'surprise' programme may not be evident until after the magnet is removed. A further problem with magnetic application is the variability of response between devices, as there is no universal standard. Thus, the use of magnet may be safe in non-programmable pacemaker, however, the most current devices should be considered programmable unless known otherwise.

**Intraoperative Management**

Intraoperative monitoring should be based on the patient’s underlying disease and the type of surgery. Continuous ECG monitoring is however, essential to monitor pacemaker functioning. In addition, both electrical and mechanical evidence of the heart function should be monitored by manual palpation of the pulse, pulse oximetry, precordial stethoscope and arterial line, if indicated. Presence of pacemaker is not an indication for insertion of pulmonary artery (PA) or central venous catheter. If these are indicated,
care should be taken during insertion of the guide wire or central venous catheter as they are potentially arrhythmogenic. In a patient in whom the pacemaker or AICD has been recently implanted, while passing the PA catheter, care should be taken as it can easily dislodge the freshly placed transvenous endocardial electrode. It is best to avoid the insertion of PA catheter or use alternative site of insertion in such patients. Multipurpose PA catheter with pacing facilities can also be used.

The anaesthetic technique should be used according to the need of the patient. Both narcotic and inhalational techniques can be used successfully. These anaesthetic agents do not alter current and voltage thresholds of the pacemaker. Skeletal myopotentials, electroconvulsive therapy, succinylcholine fasciculation, myoclonic movements, or direct muscle stimulation can inappropriately inhibit or trigger stimulation, depending on the programmed pacing modes. The muscle fasciculation induced by succinylcholine can be avoided by using nondepolarizing muscle relaxant or defasciculating with nondepolarizing muscle relaxant before giving succinylcholine. Etomidate and ketamine should be avoided as these cause myoclonic movements. Pacemaker function should be verified, before and after initiating mechanical ventilation, as there may be dislodgement of pacemaker leads by positive pressure ventilation, or nitrous oxide entrapment in the pacemaker pocket. In patients with rate responsive pacemakers, rate responsive mode should be deactivated before surgery. If this is not possible for some reason, the mode of rate response must be known so that conditions causing changes in paced heart rate can be avoided. For example, shivering and fasciculations should be avoided if the pacemaker is ‘activity’ rate responsive, ventilation (respiratory rate and tidal volume) should be kept controlled and constant in case of ‘minute ventilation’ rate responsive, and temperature must be kept constant in ‘temperature’ rate responsive pacemakers.

Electromagnetic Interference

Most pacemakers are sensitive to direct or indirect EMI. Strong ionizing beams of radiation, nuclear magnetic resonance imaging, surgical electrocautery or dental pulp vitality tester are the most common direct sources of EMI that could interfere with pacemaker. The indirect sources of EMI include radar, orthopaedic saw, telemetric devices, mechanical ventilators, lithotriptors, cellular telephones, and whole body vibrations are the potential sources of mechanical interferences that could affect pacemaker. Diagnostic radiology and computed tomographic (CT) scans do not affect the function of the pacemaker. Amongst these, electrocautery is the most important source of EMI. It involves radiofrequency current of 300-500 KHz. Fatal arrhythmias and deaths have been reported with the use of electrocautery leading to failure of pacemaker. Between 1984-1997, the US-FDA was notified of 456 adverse events with pulse generators, 255 from electrocautery and significant number of device failures.

One should apply the following measures to decrease the possibility of adverse effects due to electrocautery.

1) Bipolar cautery should be used as much as possible as it has less EMI.
2) If unipolar cautery is to be used during operation, the grounding plate should be placed close to the operative site and as far away as possible from the site of pacemaker, usually on the thigh and should have good skin contact.
3) Electrocautery should not be used within 15 cm of pacemaker. Frequency of electrocautery should be limited to 1-second bursts in every 10 seconds to prevent repeated asystolic periods. Short bursts with long pauses of cautery are preferred.
4) Pacemaker may be programmed to asynchronous mode by a magnet or by a programmer. Before using cautery, the programmer must be available in the operation theatre (OT). During the use of cautery, magnet should not be placed on pulse generator as it may cause pacemaker malfunction.
5) Provision of alternative temporary pacing (transvenous, noninvasive transcutaneous) should be ready in the OT.
6) Drugs such as isoproterenol and atropine
should be available.

7) If defibrillation is required in a patient with pacemaker, paddles should be positioned as far away as possible from the pacemaker generator. If possible, anterior to posterior positioning of paddles should be used. Although permanent pacemakers have protective circuits to guard against externally applied high voltage, pulse generator malfunction has been reported. In elective cardioversion, the lowest voltage necessary should be utilized. However, even with these precautions, defibrillation may result in acute increase in the stimulation threshold, with resultant loss of capture. If this occurs, immediate reprogramming or temporary pacing should be done with increased generator output.

8) Careful monitoring of pulse, pulse oximetry and arterial pressure is necessary during electrocautery, as ECG monitoring can also be affected by interference.

9) The device should always be rechecked after operation.

Specific Perioperative Considerations

Some procedures pose a greater risk of pacemaker malfunction.

Transurethral Resection of Prostate (TURP) and Uterine Hysteroscopy

Coagulation current used during TURP procedure has no effect, but the cutting current at high frequencies (up to 2500 kc/sec) can suppress the output of a bipolar demand ventricular pacemaker. Dresner et al reported a case in which electrocautery, as ECG monitoring can also be affected by interference.

8) Careful monitoring of pulse, pulse oximetry and arterial pressure is necessary during electrocautery, as ECG monitoring can also be affected by interference.

9) The device should always be rechecked after operation.

Electroconvulsive Therapy

ECT appears safe for patients with pacemakers, since little current flows within the heart because of the high impedance of body tissue, but the seizure may generate myopotentials which may inhibit the pacemaker. Thus ECG monitoring is essential and pacemakers should be changed to nonsensing asynchronous mode (fixed mode).

Radiation

Cases where radiation therapy is planned for deep seated tumors, therapeutic radiation can damage the complementary metal oxide semiconductors (CMOS) that are the parts of most modern pacemakers. Generally, doses in excess of 5000 rads are required to cause pacemaker malfunction but as little as 1000 rads may induce pacemaker failure or cause runaway pacemaker. If pacemaker cannot be shielded from the field of radiation, consideration should be given to reimplanting the pacemaker at a distant site. Temporary damage to pacemaker may recover after reprogramming but there may be permanent damage to the pacemaker as well. This effect could be attributed to charge accumulation inside CMOS after radiation therapy leading to failure of various components in the circuitry and therefore, cause pacemaker failure.

Nerve Stimulator Testing or Transcutaneous Electronic Nerve Stimulator Unit (TENS)

TENS is now a widely used method for pain relief. TENS unit consists of several electrodes placed on the skin and connected to a pulse generator that applies 20 µsec rectangular pulses of 1 to 200 V and 0 to 60 mA at a frequency of 20 to 110 Hz. This repeated frequency is similar to the normal range of heart rates, so it can create a far field potential that may inhibit a cardiac pacemaker. Adverse interaction between these devices has been frequently reported, so these patients should be monitored during initial application of TENS. Pacemaker mediated tachycardia has been induced by intraoperative somatosensory evoked potential stimulation.

Lithotripsy

Anaesthesia may be required in patients undergoing extracorporeal shock wave lithotripsy
(ESWL) for immobilisation and to avoid pain in flank at entry site of waves. There may be electrical interference from hydraulic shock waves and can cause mechanical damage. High-energy vibrations produced by lithotripsy machine can cause closure of reed switch causing asynchronous pacing. ‘Activity’ rate responsive pacemaker can be affected due to the damage caused to the piezoelectric crystals by ESWL. The shock waves can produce ventricular extrasystoles, if not synchronized with R wave. Thus, pacemaker malfunction can occur in patients undergoing ESWL, requiring adequate preparation prior to procedure. One should have cardiologist’s opinion, perioperative ECG monitoring, device programmer and a standby cardiologist to deal with any device malfunction. Rate responsive pacemaker should have their activity mode deactivated. Focal point of the lithotriptor should be kept at least six inches (15 cm) away from the pacemaker. Patient with abdominally placed pacemaker generators should not be treated with ESWL. Low shock waves (<16 kilovolts) should be used initially followed by a gradual increase in the level of energy. Dual chamber demand pacemaker is especially sensitive to shock waves and should be reprogrammed to a simpler mode (VOO, VVI) preoperatively.

Magnetic Resonance Imaging (MRI)

MRI is an important diagnostic tool. But its use in patients with pacemaker is contraindicated due to lethal consequences and mortality. Three types of powerful forces exist in the MRI suite. Static Magnetic Field: An intense static field is always present even if the scanner is not imaging. Most of the pacemakers contain ferromagnetic material, which gets attracted to the static magnetic field in the MRI and may exert a torque effect leading to discomfort at the pacemaker pocket. The reed switches used in the pacemaker are known to close at very low magnetic field of 0.5-2 mT, thus reed switch activation by high static field of 0.5-1.5 T can result in switching of pacemaker to a non-sensing asynchronous pacing.

Radiofrequency Field (RF): This field is switched on and off during magnetic resonance imaging and has a frequency of 21-64 MHz depending on the strength of magnetic field. The radiofrequency signals can cause interference with pacemaker output circuits resulting in rapid pacing at multiple of frequency between 60-300 bpm causing rapid pacing rate. It may cause pacemaker reprogramming and destruction of electronic components. It may also cause heating at the electrode-tissue boundary, which may cause thermal injury to endocardium and myocardium.

Gradient Magnetic Field: used for spatial localization changes its strength along different orientations and operates at frequencies in order of 1 kHz. Gradient magnetic field may also interact with reed - switch in pacemaker. Inappropriate sensing and triggering because of the induced voltages can occur. It may induce negligible heating effect.

The results of various studies done to evaluate the safety and feasibility suggest that in the absence of other alternative for getting diagnostic information, MRI can be done in the presence of a cardiologist. However, appropriate patient selection should be done and equipment for resuscitation and temporary pacing should be available. Also patients should be closely monitored with ECG and oxygen saturation. Further studies are necessary to refine the appropriate strategies for performing MRI safely in a patient with implanted pacemaker. The risk-benefit ratio must be individually evaluated in every patient with a pacemaker. Patients, who require head MRI scanning without alternative diagnostic possibilities, may be best served in a carefully monitored setting. Thus patients with pacemakers should not routinely undergo MRI scanning.

Conclusion

Patients with implanted pacemakers can be managed safely for surgery and other non-surgical procedures. It requires thorough understanding about indication of pacemaker insertion, various modes of pacing, and programming of pacemaker. A cardiologist should also be consulted for device evaluation regarding its proper function and life of the batteries. Anaesthetic management should be
planned preoperatively according to patient’s medical status. Careful monitoring of ECG, pulse oximetry and arterial blood pressure should be done. While using electocautery, precaution for minimal EMI should be taken. Magnet should not be placed over pacemaker in the OT in presence of electocautery. Rate responsive pacemakers should have rate responsive mode disabled before surgery. Provision of temporary pacing should be available in the OT to deal with emergency situation of pacemaker malfunction. Pacemaker should be rechecked after the procedure.

References

8. Bourke ME. The patients with a pacemaker or related device. *Can J Anaesth* 1996; 48: R24-R41
10. Rozner MA. Intrathoracic gadgets: Update on pacemakers and implantable cardioverter-defibrillators, ASA refreshers course 1999, pp 212
30. Asroff SW, Kingston TE, Stein BS. Extracorporeal shock...
33. Vahlhaus C, Sommer T, Lewalter T, et al. Interference with cardiac pacemakers by magnetic resonance imaging: are there irreversible changes at 0.5 Tesla. *PACE* 2001; 24: 489-495