Pacemakers and implantable cardioverter defibrillators – general and anesthetic considerations

Amy G. Rapsang*, Prithwis Bhattacharyya

Department of Anesthesiology & Intensive Care, North Eastern Indira Gandhi Regional Institute of Health and Medical Sciences, Shillong, Meghalaya, India

Received 21 August 2012; accepted 28 February 2013
Available online 16 October 2013

KEYWORDS
Pacemakers; Implantable cardioverter defibrillators; Anesthesia for device patients

Abstract A pacemaking system consists of an impulse generator and lead or leads to carry the electrical impulse to the patient’s heart. Pacemaker and implantable cardioverter defibrillator codes were made to describe the type of pacemaker or implantable cardioverter defibrillator implanted. Indications for pacing and implantable cardioverter defibrillator implantation were given by the American College of Cardiologists. Certain pacemakers have magnet-operated reed switches incorporated; however, magnet application can have serious adverse effects; hence, devices should be considered programmable unless known otherwise. When a device patient undergoes any procedure (with or without anesthesia), special precautions have to be observed including a focused history/physical examination, interrogation of pacemaker before and after the procedure, emergency drugs/temporary pacing and defibrillation, reprogramming of pacemaker and disabling certain pacemaker functions if required, monitoring of electrolyte and metabolic disturbance and avoiding certain drugs and equipments that can interfere with pacemaker function. If unanticipated device interactions are found, consider discontinuation of the procedure until the source of interference can be eliminated or managed and all corrective measures should be taken to ensure proper pacemaker function should be done. Post procedure, the cardiac rate and rhythm should be monitored continuously and emergency drugs and equipments should be kept ready and consultation with a cardiologist or a pacemaker-implantable cardioverter defibrillator service may be necessary.

© 2013 Sociedade Brasileira de Anestesiologia. Published by Elsevier Editora Ltda.
Este é um artigo Open Access sob a licença de CC BY-NC-ND.

Introduction Battery-operated pacing devices were introduced by C.W. Lillehei and Earl Bakken in 1958. The natural progression of pacemaker (PM) developments led to invention of the implantable cardioverter defibrillator (ICD) around 1980 by Michael Morchower.¹

A pacemaking system consists of an impulse generator and lead or leads to carry the electrical impulse to the patient’s heart. Leads can be unipolar, bipolar or multipolar. Generators with bipolar leads can be programmed to the unipolar mode for pacing, sensing, or both.¹

* Corresponding author.
E-mail: amy_rap@yahoo.com (A.G. Rapsang).

http://dx.doi.org/10.1016/j.bjane.2013.02.005

© 2013 Sociedade Brasileira de Anestesiologia. Published by Elsevier Editora Ltda. Este é um artigo Open Access sob a licença de CC BY-NC-ND.
Pacemaker codes

Table 1 shows the pacemaker codes given by the North American Society of Pacing and Electrophysiology (NASPE)/British Pacing and Electrophysiology Group (BPEG) (2002) Generic Pacemaker Code (NBG).2

<table>
<thead>
<tr>
<th>Position I: pacing chamber(s)</th>
<th>Position II: sensing chamber(s)</th>
<th>Position III: response(s) to sensing</th>
<th>Position IV: programmability</th>
<th>Position V: multisite pacing</th>
</tr>
</thead>
<tbody>
<tr>
<td>O = none</td>
<td>O = none</td>
<td>O = none</td>
<td>O = none</td>
<td>O = none</td>
</tr>
<tr>
<td>A = atrium</td>
<td>A = atrium</td>
<td>I = inhibited</td>
<td>R = rate modulation</td>
<td>A = atrium</td>
</tr>
<tr>
<td>V = ventricle</td>
<td>V = ventricle</td>
<td>T = triggered</td>
<td></td>
<td>V = ventricle</td>
</tr>
<tr>
<td>D = dual (A + V)</td>
<td>D = dual (A + V)</td>
<td>D = dual (T + I)</td>
<td></td>
<td>D = dual (A + V)</td>
</tr>
</tbody>
</table>

Indications for pacemaker implantation3

1. Bradycardia due to Sinus Node Dysfunction (SND) and Atrioventricular Node Dysfunction (AND).
   - SND: Persistent sinus bradycardia and chronotropic incompetence without identifiable causes, symptomatic bradycardia.
   - Acquired atrioventricular (AV) block in adults:
     - Third-degree and advanced second-degree AV block at any anatomic level associated with:
       - Bradycardia with symptoms/ventricular arrhythmias presumed to be due to AV block.
       - Other medical conditions that require drug therapy that results in symptomatic bradycardia.
       - Symptom-free patients in sinus rhythm, with documented periods of asystole greater than or equal to 3 s, an escape rate <40 bpm or an escape rhythm that is below the AV node.
       - Awake, symptom-free patients with AF and bradycardia with ≥1 pauses of at least ≥5 s.
       - After catheter ablation of the AV junction associated with postoperative AV block that is not expected to resolve after cardiac surgery.
       - Associated with neuromuscular diseases with AV block.
   - Second-degree AV block with associated symptomatic bradycardia regardless of type or site of block.
   - Asymptomatic persistent third-degree AV block at any anatomic site with average awake ventricular rates of 40 bpm or faster if cardiomegaly or LV dysfunction is present or if the site of block is below the AV node.
   - Second- or third-degree AV block during exercise in the absence of myocardial ischemia.

2. Chronic bifascicular block: Bifascicular block refers to ECG evidence of impaired conduction below the AV node in the right and left bundles.
   - Advanced second-degree AV block or intermittent third-degree AV block.
   - Type II second-degree AV block.
   - Alternating bundle-branch block.

3. Pacing for atrioventricular block associated with acute myocardial infarction
   - Persistent second-degree AV block after ST-segment elevation MI.
   - Transient advanced second- or third-degree infranodal AV block and associated bundle-branch block.
   - Persistent and symptomatic second- or third-degree AV block.

4. Hypersensitive carotid sinus syndrome and neurocardiogenic syncope.
   - Recurrent syncope caused by spontaneously occurring carotid sinus stimulation and carotid sinus pressure that induces ventricular asystole of more than 3 s.

5. After cardiac transplantation.
   - Persistent inappropriate or symptomatic bradycardia not expected to resolve.

6. Prevention and termination of arrhythmias by pacing.
   - Symptomatic recurrent SVT that is reproducibly terminated by pacing when catheter ablation and/or drugs fail to control the arrhythmia or produce intolerable side effects.
   - Sustained pause-dependent VT, with or without QT prolongation.


   - Advanced second/third-degree AV block associated with symptomatic bradycardia, ventricular dysfunction, or low cardiac output.
   - SND with correlation of symptoms during age-inappropriate bradycardia. (The definition of bradycardia varies with the patient’s age and expected heart rate.)
   - Postoperative advanced second- or third-degree AV block that is not expected to resolve or that persists at least 7 days after cardiac surgery.
   - Congenital third-degree AV block with a wide QRS escape rhythm, complex ventricular ectopy, or ventricular dysfunction.
   - Congenital third-degree AV block in the infant with a ventricular rate less than 55 bpm or with congenital heart disease and a ventricular rate less than 70 bpm.
Indications for implantable cardioverter defibrillators implantation

- Survivors of cardiac arrest due to VF/hemodynamically unstable sustained VT.
- Structural heart disease and spontaneous sustained VT, whether hemodynamically stable or unstable.
- LVEF (Left Ventricular Ejection Fraction) ≤ 35% due to prior MI (NYHA functional Class II/III) or LVEF ≤ 30% due to prior MI (NYHA functional Class I).
- Nonischemic dilated cardiomyopathy having an LVEF ≤ 35% (NYHA functional Class II/III).
- Nonsustained VT due to prior MI, LVEF ≤ 40%.

Indications for implantable cardioverter defibrillators implantation in pediatric patients and patients with congenital heart disease

- Survivor of cardiac arrest.
- Symptomatic sustained VT.

Complications: Complications can be due to the presence of the pacing system as a foreign body (mechanical complications), apparent or real pacing system malfunction, acute complications related to the procedure itself: hemothorax, pneumothorax, subclavian artery puncture and myocardial perforation.

1. Infection: incidence is 0.8–5.7%.
   Staphylococcus aureus (early infections) and S. epidermidis (late infections) are the most common organisms involved.
   - Pacemaker pocket infection: Routine use of anti-staphylococcal antibiotic prophylaxis at the time of implantation or generator change has been shown to have consistent benefit in a meta-analysis of seven randomized controlled trials, in 2023 patients, in decreasing the rates of short-term pocket infection, skin erosion, or septicemia. Therefore routine prophylaxis with anti-staphylococcal antibiotics is recommended.
   - Endocarditis: Complete eradication of the infection usually involves removal of the entire pacing system.

2. Thrombosis: usually subclinical
   - Embolism: Pulmonary (incidence 0.6–3.5%) and systemic embolization. Patients implanted with a VVI(R) pacing system have a higher incidence of paroxysmal and chronic atrial fibrillation, which predisposes to embolic events.

3. Pacing problems
   Failure of capture: The occurrence of the stimulus output, which, when delivered outside the refractory period of atrial or ventricular tissue, is not followed by a P wave or QRS complex. This could be due to elevation of stimulation threshold, which in turn could be due to metabolic disturbances, drugs (mostly anti-arrhythmics), acute myocardial infarction, pacing lead defects (fractures, insulation break), lead maturation, dislodgement or perforation, inappropriately low programmed output, battery end of life.
   - Failure of output: This is manifested on the surface electrocardiogram as an absence of pacing artifacts. This can be due to battery depletion or component failure.

4. Sensing problems:
   - Oversensing of unwanted signals: A pause in paced rhythm (AAI/VVI modes) or, if the oversensed atrial event causes triggered pacing in the ventricle, an earlier than expected paced ventricular event. Under-sensing of the intrinsic intracardiac signal.

5. Rapid paced ventricular rates:
   - Atrial tachyarrhythmias: Sinus tachycardia, atrial tachycardia, atrial flutter and ventricular pacing can resemble an "electronic AV block," and cardiac output can suddenly decrease.
   - Pacemaker-mediated tachycardia (PMT).

Pacemaker magnets

Magnet-operated reed switches within pacemakers were originally incorporated to produce pacemaker behavior that would demonstrate remaining battery life and sometimes pacing thresholds and can be used to protect the pacemaker-dependent patient during diathermy, electrocautery or other sources of pulsed EMI (electro magnetic interference). Magnets can be applied over the pacemaker to avoid inhibition by such pulsed interference. In modern pacemakers, the switch to asynchronous pacing is coupled to the next cardiac event to avoid competition at the output. Placement of a magnet over a generator might produce no change in pacing because not all pacemakers switch to a continuous asynchronous mode when a magnet is applied.

In some devices magnet behavior can be altered by programming, whereas in others, magnet behavior can be completely eliminated by programming. For all generators, calling the manufacturer remains the most reliable method of determining magnet response and using this response to predict remaining battery life. However, the Task Force cautions against the use of the magnet over an ICD.

As battery voltage falls, the magnet response can be used to detect the following:

- IFI (intensified follow-up required) – the device must be checked frequently (approximately every 4 weeks for most models).
- ERI (elective replacement indicator) – the device is nearing the end of its useful life and should be electively replaced.
- EOL (end of life) – the device has insufficient battery power remaining and should be replaced immediately.

Problems with magnet application:

- Switching to asynchronous pacing may trigger ventricular asynchrony in patients with myocardial ischemia, hypoxia, and electrolyte imbalance.
- Constant magnet application over the pacemaker may alter the programming and can also cause continuous or transient loss of pacing.
- Variability of response between devices.
- Occasionally, PMT can ensue on removal of the magnet from a dual-chamber PM.
Thus, in nonprogrammable pacemaker, the use of magnet may be safe. However, the most current devices should be considered programmable unless known otherwise. Table 2 shows the commonly used pacing modes.

**Implantable cardioverter defibrillator (ICD)**

ICD consists of pulse generator, pacing or sensing electrodes, and defibrillation coils. Its function is similar to a pacemaker and hence susceptible to the same complications and emergencies as a pacemaker. In addition, an ICD senses and detects ventricular tachycardia (VT) and ventricular fibrillation (VF) and delivers therapy in the form of overdrive antitachycardia pacing (ATP), low-energy cardioversion, and high-energy defibrillation. Table 3 shows a four-place ICD code, given by the North American Society of Pacing and Electrophysiology (NASPE)/British Pacing and Electrophysiology Group (BPEG) (2002) Generic Defibrillator Code (NBD). The fourth position of the code is the three/five-letter code for the pacemaker capability of the device.

ICD shock can lead to transient post-shock loss of capture and sensing in the pacemaker because of exposure of the myocardium to high current density. In patients with ICDs and separate pacemakers, the pacing stimulation artifact (PSA) can cause over-sensing or under-sensing in the ICD lead resulting in inappropriate ICD therapy. Hence, it is recommended that pacing at the chronic pacing amplitude in sinus rhythm should cause a PSA amplitude <1 mV on the ICD rate sense lead to ensure appropriate ICD sensing during VF,6,10

It is imperative that ICD patients undergo routine evaluation (every three months and after each exposure to EMI).4

**Pacemakers and special circumstances:**

**Magnetic resonance imaging (MRI)**

It is estimated that up to 75% of pacemaker patients will have a medical need for an MRI over the lifetime of their device.11 Scanning device patients is now considered a relative contraindication (vs absolute).12,13
manufacturers have made changes in devices to make them more compatible with MRI (less use of ferromagnetic material in battery construction). MRI affects pacemaker function in various ways:

**Static magnetic field** – can result in actual physical movement of the pulse generator’s internal components. Modulated radio frequency (RF) field – can result in induced voltage across the pacemaker electrodes that may stimulate myocardial tissue, leading to rates equal to the MRI pulsing rates (cycle lengths of 200–1000 ms), heating of cardiac tissue adjacent to lead electrodes causing thermal injury to myocardium and endocardium. Gradient magnetic field – can cause over-sensing or under-sensing, and can induce negligible heating effect.

While doing MRI – magnet response, rate response, noise response, ventricular sense response, premature ventricular contraction response, conducted atrial fibrillation response, and tachyarrhythmia functions – should be disabled. Pacemaker-dependent patients should be programmed to VOO mode and non-pacemaker-dependent patients can be programmed to the VVI or DDI mode.

**Guideline recommendations with regard to performing MRIs in nonpacemaker dependent patients.**

i. Decision should be weighed principally on risk-benefit ratio and the urgency of the clinical indication for MRI.

ii. Obtain a written and verbal informed consent.

iii. Before taking the patient for MRI, pacemaker functions should be pretested.

iv. A cardiologist should decide whether it is necessary to program the pacemaker prior to the MRI.

v. Advanced Cardiac Life Support (ACLS) personnel must be in attendance for the entire MRI examination and a crash cart, and defibrillator must be available onsite.

vi. The patient should be monitored and visual/voice contact with the patient should be maintained continuously during the MRI.

vii. After the MRI examination, a cardiologist/electrophysiologist should interrogate the pacemaker to confirm that the function is consistent with the pre-examination state.

Exposure to MRI has similar effects on an ICD as those described for a cardiac pacemaker, since some of the basic components are comparable. ICDs may falsely detect the Magnetic Resonance Radio Frequency field as VF, charge capacitors, and deliver ATP, cardioversion, or defibrillation therapies. Devices may not be able to deliver ICD therapy in the static magnetic field and the ICD transformer will be magnetically saturated and may not have the voltage necessary to charge the capacitor, and hence can lead to permanent device failure. Magnetic fields may also prevent detection of VT or VF.

**Electrocautery and anesthesia**

Electrocautery is the most common exogenous source of EMI that can interact with pacemakers, resulting in pulse generator inhibition, electrical burns at the myocardial–electrode interface, atrial or ventricular tachycardia and fibrillation, pulse generator component failure, loss of or change in output, reprogramming of rate or mode of function and runaway pacing. The EMI generated by electrocautery that may affect the device is related to the distance and orientation of the current to the patient’s device and leads. A prospective study has shown that unipolar devices are far more susceptible than bipolar devices to electrocautery interference. In bipolar coagulation cautery, the current flow is localized between the two poles of the instrument, and therefore poses minimal problems. However, in unipolar electrocautery devices, the electrical current flow is not restricted to the tissue interposed between two electrodes and spreads throughout the body. The vector of the dipole for the electrocautery device with respect to that of the pacemaker should not intersect with each other. Hence, the EMI generated by electrocautery that may affect the device is related to the distance and orientation of the current to the patient’s device and leads. Special precautions are:

1. Using a bipolar electrocautery system/ultrasonic (harmonic) scalpel, if possible.
2. If only unipolar electrocautery is available, then the indifferent electrode should be placed as far from the pacemaker leads as possible.
3. Assure that the cautery tool and current return pad are positioned so that the current pathway does not pass through or near the CRMD (Cardiac Rhythm Management Devices) pulse generator and leads.
4. Using short, intermittent, and irregular bursts at the lowest feasible energy levels to minimize the hemodynamic effects of pacemaker inhibition.
5. A magnet should not be applied prophylactically, because there is no uniform pulse generator response to this maneuver.
6. Electrocautery can cause electromagnetic noise on the ICD sensing lead that is detected as VF and can lead to inappropriate shock.

---

**Table 3 NASPE/BPG Generic Defibrillator Code (NBG).**

<table>
<thead>
<tr>
<th>Position I shock chambers</th>
<th>Position II antitachycardia pacing chambers</th>
<th>Position III tachycardia detection</th>
<th>Position IV antibradycardia pacing chambers</th>
</tr>
</thead>
<tbody>
<tr>
<td>O = none</td>
<td>O = none</td>
<td>E = electrogram</td>
<td>O = none</td>
</tr>
<tr>
<td>A = atrium</td>
<td>A = atrium</td>
<td>H = hemodynamic monitors</td>
<td>A = atrium</td>
</tr>
<tr>
<td>V = ventricle</td>
<td>V = ventricle</td>
<td></td>
<td>V = ventricle</td>
</tr>
<tr>
<td>D = dual (A + V)</td>
<td>D = dual (A + V)</td>
<td></td>
<td>D = dual (A + V)</td>
</tr>
</tbody>
</table>
7. Therefore the detection and therapy can be programmed to "off" during surgery and turn it "on" postoperatively; external defibrillators are used for intra-operative VT/VF.4
8. The availability of programming equipment and trained personnel is essential.
9. Alternative temporary pacing should be ready in the OT.26
10. Drugs such as isoproterenol and atropine should be available.8
12. The device should always be checked after operation.

Diathermy

Short-wave diathermy should be avoided near the generator site. Potential problems include overheating of the generator circuitry and damage to electronic components.6

Electroconvulsive therapy (ECT)

ECT is relatively safe for patients with pacemakers, because of the localized application of the electrical stimulus to the head, hence a low probability for the occurrence of problems.8 Sometimes the seizure may generate myopotentials which may inhibit the pacemaker, and transient electrocardiographic changes (e.g., increased P-wave amplitude, altered QRS shape, T-wave and ST-T abnormalities) may occur and additional cardiac complications (e.g., arrhythmia or ischemia) may occur in patients with pre-existing cardiac disease. The Task Force believes that ECT may be administered to CRMD patients without significant damage to a disabled CRMD. All CRMDs should undergo a comprehensive interrogation before the procedure(s). ICD functions should be disabled for shock therapy during ECT.6 Therefore, monitoring and standard resuscitation equipment, and a trained programmer, should be available28 and pacemakers can be changed to nonsensing asynchronous mode (fixed mode),29 to avoid myopotential inhibition of the device in pacemaker-dependent patients.6 CRMD-dependent patients may require a temporary pacing system to preserve cardiac rate and rhythm during shock therapy.

Other electrical stimulating techniques

Electrical stimulating techniques such as transcutaneous electrical nerve stimulation (TENS) consists of several electrodes placed on the skin and connected to a pulse generator that applies 20 μs rectangular pulses of 1–200 V and 0–60 mA at a frequency of 20–110 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.5 TENS can be used safely in patients with pacemakers and defibrillators with close monitoring and use in close proximity to the device is not advised.30

PMT has been reported to be induced by intraoperative somatosensory evoked potential stimulation.11

Radiofrequency ablation (RFA)

The radiofrequency current path (electrode tip to current return pad) should be kept as far away from the pulse generator and lead system as possible and to avoid direct contact between the ablation catheter and the CRMD (Cardiac Rhythm Management Devices).6

Radiation therapy

The high-energy ionizing radiation used in radiation therapy can cause significant damage to the semiconductors of pacemakers, even at very small doses.31 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.8 Pulse generator recovery may occur long after the end of the radiation treatment, but it is mostly incomplete, and the pacemaker cannot be used reliably thereafter.12,31 Thus, in pulse generators exposed to radiation, transient loss of function should be regarded as a precursor of permanent damage. Hence it is essential to follow guidelines for ensuring the lowest possible radiation dose to the pacemaker24 and careful follow-up should be performed during and after completion of the radiation therapy. The Task Force believes that radiation therapy can be safely performed for CRMD patients. The device must be outside the field of radiation.6 Therefore, some pulse generators will require surgical relocation before commencing radiation.

The low-energy X-rays used for diagnostic radiology have not been reported to have any adverse effect in pacemakers.4

Other minor surgical procedures (lithotripsy, endoscopic electrocautery)

Extracorporeal shock wave lithotripsy (ESWL) is associated with electromagnetic and mechanical forces that may influence pacing system function.35 Pulse generators employing a piezoelectric crystal sensor seem to be the most susceptible to failure,36 and therefore the sensor mode should be programmed "off" during lithotripsy as this will prevent an unwanted increase in paced heart rate and shatter injury to the piezoelectric element. Therefore, focal point of the lithotriptor should be kept at least six inches (15 cm) away from the pacemaker,37 and disabling atrial pacing if the lithotripsy system triggers on the R wave.7 Low shock waves (<16 kV) should be used initially followed by a gradual increase in the level of energy.38 Endoscopic electrocautery is generally safe in patients with pacemakers, although complications have been reported.39

Cardiopulmonary resuscitation

The standard resuscitation protocols should be followed in patients with permanent pacemakers, and normal pacemaker function should be established after the resuscitation procedure is completed.4 Myocardial stimulation threshold is markedly increased during cardiopulmonary resuscitation4 and even though most sensing and pacing problems are
transient, a thorough evaluation of the pacing system, including interrogation and programming functions, should be made after the resuscitation procedure.\(^7\)

**Direct current cardioversion and defibrillation**

The cardioverter-defibrillator can cause capacitive coupling with the endocardial lead, causing direct discharge at the electrode--endocardium interface,\(^4,40\) thus leading to transient/permanent failure to sense and capture even in the absence of apparent damage to the pulse generator itself.\(^41\) Damaged circuitry,\(^42,43\) changes in programmed mode of function, complete pacemaker failure and microdislodgement of the lead can also occur.\(^4,44\) Defibrillation with internal cardiac paddles requires less energy but may also interfere with pacemaker function.\(^35,46\) Modern pacemakers are equipped with protection mechanisms against damage from DC shock, most common of which is the Zener diode,\(^47\) which directs a surge in current toward the electrode, protecting the pacemaker circuitry but delivering this energy to the endocardium. The general precautions for DC cardioversion and defibrillation in patients with pacemakers attempt to minimize the current delivered to the pacemaker system by using the minimal effective energy setting\(^4,46\) and placing the defibrillator paddles at least 10 cm away from the pulse generator, ensuring that the paddles are placed perpendicular to the dipole of the pacing system. A thorough evaluation of the pacing system should be performed\(^4\) and if loss of capture occurs, and then immediate reprogramming or temporary pacing should be done with increased generator output.\(^48,49\) The Task Force believes that before attempting emergency defibrillation or cardioversion of a patient with an ICD and magnet-disabled therapies, all sources of EMI should be terminated, and the magnet should be removed to re-enable antitachycardia therapies and then consider reenabling therapies through programming.\(^6\)

**Bioelectrical impedance analysis (BIA)**

BIA employs bioimpedence spectroscopy techniques which measures at 50 frequencies over a range from 5 to 1000 kHz to determine the electrical resistances of TBW and ECW to provide an estimate of body cell mass and to describe fluid shifts and fluid balance,\(^50\) but not recommended for participants with a pacemaker.\(^51\)

**Bioimpedance tomography/thoracic electrical bioimpedance (TEB)**

This uses pulsating and polarized electric current at a frequency not higher than 10 Hz, and provides information on the impedance of the extracellular environment.\(^52\) However their use is not recommended for patients with minute ventilation (MV) sensor function pacemakers, since sensors are very sensitive to stray electromagnetic interference, and patients have been inappropriately treated for pacemaker-driven tachycardias as a result. Therefore, rate modulation should be programmed to "OFF" in the peri-operative period to prevent confusion between an intrinsic tachycardia vs a pacemaker-induced.\(^1,14\)

**Metabolic abnormalities**

Electrolyte and metabolic abnormalities (hyperkalemia, hyperglycemia, alkalalemia, acidosis, hypoxemia, hypercapnia and hypothyroidism) increase the pacing threshold, causing failure to capture.\(^53\) Therefore, aggressive correction of the underlying disturbance is essential.

**Systolic heart failure**

The presence of a permanent pacemaker has been reported to be an independent predictor of poor outcome in patients with heart failure,\(^4,45\) and hence evidence of pacemaker malfunction and must be carefully evaluated.

**Pre anesthetic consideration**

Preoperative evaluation of a patient with pacemaker undergoing noncardiac surgery includes general evaluation of the patient and the pacemaker.

The consensus of the Task Force\(^6\) is that a focused preoperative evaluation should include:

a. Establishing whether a patient has a CRMD -- a focused history and physical examination
b. Defining the type of device -- obtaining the manufacturer’s identification card from the patient or other source, chest X-ray (if no other data are available)
c. Determining whether a patient is CRMD dependent for pacemaking function.
d. Determining CRMD function by a comprehensive evaluation of the device, consultation with a cardiologist or a CRMD service, contacting the manufacturer for perioperative recommendations.

ACC guidelines suggest that cardiac testing (stress tests, echocardiograms) be dictated by the patient’s underlying disease, medications, symptomatology, interval from last testing, and planned intervention.\(^55\)

No special laboratory tests or radiographs are needed for patients with a conventional PM. However, a patient with a BiV PM or ICD might need a chest film to document the position of the coronary sinus lead, especially if central line placement is planned. Most current CRMDs have an X-ray code that can be used to identify the manufacturer of the device.\(^6\)

The generator should be identified, and 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.\(^5\) If interrogation is not an option, one can slow the intrinsic heart rate to a rate below that of the pacemaker by carotid massage or a Vasalva maneuver.\(^1,56\) Any device near its elective replacement period should be considered for replacement. Ten percent decrease in the rate from the time of implantation indicates power source depletion.\(^5\) The patient’s underlying rate and rhythm should be determined (which then determines the need for backup/external pacing support). All rate enhancements and minute ventilation rate responsiveness should be programmed to "off".
The lower rate limit can be increase to optimize oxygen delivery to tissues for major cases. A determination as to whether EMI is likely to occur or not. Anesthetic techniques do not influence CRMD function. However, anesthetic-induced physiologic changes (i.e., cardiac rate, rhythm, or ischemia) in the patient may induce unexpected CRMD responses or adversely affect the CRMD–patient interaction.

Appropriate reprogramming is the safest way to avoid intraoperative problems, and is required in any rate responsive device, hypertrophic obstructive/dilated cardiomyopathy, pediatric patients, pacemaker-dependent patients, major procedure in the chest/abdomen, special procedures (lithotripsy, TURP, hysteroscopy, ECT, scoline use, MRI). Temporary pacing and defibrillation equipment should be immediately available before, during, and after a procedure.

Intraoperative considerations

General anesthesia can present a range of problems for the paced patients, although it rarely does so due to contemporary anesthesia techniques. The Task Force agrees that a patient’s electrocardiogram (ECG) should be continuously displayed and continuous peripheral pulse monitoring should be performed for all CRMD patients receiving general or regional anesthesia, sedation, or monitored anesthesia care. Mechanical systole (which depicts paced electrical activity) is best evaluated by pulse oximetry plethysmography, or arterial pressure waveform display. The presence of pacemaker is not an indication for insertion of pulmonary artery (PA) or central venous catheter (CVC). Insertion of the guide wire or CVC is potentially arrhythmogenic and dislodgement of freshly placed transvenous endocardial electrode can occur. Therefore care should be taken and multipurpose PA catheter with pacing facilities can be used. Emergency drugs should be kept ready, including temporary pacing and defibrillation.

Anesthetic induction with succinylcholine can cause significant muscle fasciculations leading to complete inhibition of pacemakers because of oversensing, resulting in cardiac arrest in pacemaker-dependent patients. The muscle fasciculation induced by succinylcholine can be avoided by using non-depolarizing muscle relaxant or defasciculating with non-depolarizing muscle relaxant before giving succinylcholine. This complication can also be prevented by temporary reprogramming of the pacemaker to an asynchronous mode. Narcotics and inhalational techniques do not alter current and voltage thresholds of the pacemaker, and hence can be used safely. Use of N₂O can cause pacemaker malfunction by increasing gas in pre pectoral pacemaker pocket (loss of anodal contact). Eto midate and ketamine should be avoided as these cause myoclonic movements. Positive pressure ventilation can cause dislodgement of pacemaker leads; therefore, pacemaker function should be verified, before and after initiating mechanical ventilation. Many metabolic and electrolyte abnormalities can affect pacemaker function and therefore, monitoring for such events is essential. Shivering should be avoided and temperature must be kept constant in 'temperature' rate responsive pacemakers.

If unanticipated device interactions are found, consider discontinuation of the procedure until the source of interference can be eliminated or managed. If a temporary pacemaker fails intraoperatively, the inspired oxygen concentration should be increased to 100%. All connections and the generator battery should be checked. The generator should be set into the asynchronous mode, and the ven-tricular output should be set on maximum. Pharmacological management (atropine, isoproterenol, or epinephrine) may be useful until the problem is resolved. Failure of a temporary transvenous electrode to capture the ventricle is usually due to displacement of the electrode away from the ventricular endocardium; careful slow advancement of the catheter or wire while pacing often results in capture. If an adequate arterial blood pressure cannot be maintained with adrenergic agonists, cardiopulmonary resuscitation should be instituted until another pacing electrode is placed or a new generator box is obtained. For the patient with an implanted defibrillator, facilities for external defibrillation should be available immediately after the device is disabled. If patient develops VT, surgeons should be advised to terminate all sources of EMI, magnet should be removed to enable anti tachycardia activities, device should be observed and if failed, and then external defibrilation/cardioversion should be initiated.

Electrical interference from surgical electrocautery units, electroconvulsive therapy, lithotripsys has already been discussed.

Post-operative considerations

Monitor cardiac rate and rhythm continuously throughout the immediate postoperative period. Emergency drugs and equipment should be kept ready. The device should be reprogrammed to appropriate settings. For an ICD, all antitachyarrhythmic therapies should be restored. Consultation with a cardiologist or pacemaker–ICD service may be necessary.

Conflicts of interest

The authors declare no conflicts of interest.

References

Pacemakers and ICDs – general and anesthetic consideration


