Assessment Points

System	Effect	Assessment by Hx	PE	Test
GENERAL	Pt age varies	Childhood vs. adult Dx	Find comorbidities	As indicated
HEENT	Nasal secretions Middle ear fluid/drainage Hypertrophic tonsils and adenoids T&A	Allergy vs. infection Acute vs. chronic OM; ear pain and ear tugging OSA, mouth breathing, snoring	Clear vs. green mucus Fever vs. afebrile, inflamed tympanic membrane (red, opacified, bulging, and immotile) vs. fluid level Inspection	Eosinophil smear Tympanogram
RESP	Cough Laryngo-tracheomalacia Pneumonia	Dry vs. wet OSA/feeding difficulty Fever, cough, dyspnea	Upper vs. lower tract symptoms Retractions and stridor Fever, tachypnea, and crackles	Pulse ox Bronchoscopy Pulse oximetry, CXR, CBC
GI	NPO status and reflux Hx	Clear vs. fatty liquid	Tolerating clears; content	None
CNS	Developmental status Hearing (usually conductive loss) Complications of untreated OM (such as meningitis)	Developmental Hx Delayed speech and cognition Fever, headache, mental status changes, photophobia	Congenital anomalies Fever, Brudzinski and Kernig signs, and meningismus	Genetic testing, Audiometry MRI, lumbar puncture, cultures
DERM	Eczema	Allergy/steroid Hx	Allergic/nonallergic rash	Skin biopsy

Key References: Hoffmann KK, Thompson GK, Burke BL, et al.: Anesthetic complications of tympanostomy tube placement in children. *Arch Otolaryngol Head Neck Surg* 128(9):1040–1043, 2002; Bowatte G, Tham R, Allen KJ, et al.: Breastfeeding and childhood acute otitis media: a systematic review and meta-analysis. *Acta Paediatr* 104(Suppl 467):85–95, 2015.

Perioperative Implications

Preoperative Preparation

- Lower respiratory tract pathology or pneumonia may warrant further evaluation and case rescheduling; runny nose (rhinorrhea) is usually not an indication for case cancellation.
- Children: Avoid oral premed for myringotomy and PETs alone (short surgical time); consider parental presence for induction; allow comfort object in the OR; developmentally appropriate review of procedures; consider preop oral acetaminophen to give the analgesic regimen time to work.
- Adult: IV midazolam or fentanyl before induction; topical local anesthetic drops in ear may be indicated.

Monitoring

- Standard ASA monitors; skin temperature probe
- Precordial stethoscope very helpful

Airway

- Children: Inhalation induction and mask airway maintenance for straightforward cases.
- Adults: IV induction with mask airway or LMA maintenance.
- Oral and/or nasal airways as indicated.
- Preparation for intubation if obstruction is present or as the case direction changes.
- Maintenance.
- Volatile anesthetic in oxygen with NO usually sufficient.
  - 70/30 N_2O/O_2 plus 8% sevoflurane for induction, followed by 50/50 N_2O/O_2 plus 4% sevoflurane for maintenance until first tube in place.
  - Turn off anesthetics at second myringotomy to avoid prolonged anesthesia for short operation.
- Consideration of IV propofol infusion to maintain spontaneous ventilation if laryngoscopy/bronchoscopy is also planned.
- Otherwise as required for additional operative procedures after PETs are placed.

Extubation

- Routine precautions and criteria

Adjuvants

- Determined by the course and complexity of operation(s) to be performed
- PETs are frequently placed before other procedures (cleft lip/palate repair, auditory evoked potentials)

Postoperative Period

- Postop analgesia: Multimodal approach
  - Children: "Belly" analgesia first (bottle, cup, juice, comfort); consideration of nasal fentanyl and/or oral acetaminophen if rectal not given intraop
  - Adults: IV/oral analgesics as needed; antiemetic may be needed more so than in children
- Emergence delirium: Nasal or IV clonidine or dexmedetomidine (an option for children)
- Slow introduction of PO fluids; limited volume if possible
- Plans to reunite child with parent and/or proxy after pt is settled in the PACU

Anticipated Problems/Concerns

- Separation of child and parent and/or proxy: Have a guardian present for induction, oral midazolam if appropriate.
- Separation from child's comfort object: Label the object with pt's name.
- Charting vital signs and maintaining anesthesia record in a short case with much to do: An assistant or electronic medical record is helpful.
- Difficulty maintaining mask airway: Use LMA and ET intubation.
- Laryngospasm: Hold positive pressure, IM/IV succinylcholine and/or atropine, propofol if IV present, possible ET intubation
- Antibiotics: Start PIV if required.
- Ear drops applied by the surgeon: Can sting if the pH is basic.
- Unanticipated pathology includes cerumen impaction, cholesteatoma, other tumors, and ossicular dislocation.
- Excessive bleeding (ear canal trauma): Apply topical epinephrine.
- Small external ear canals: Change type of PE tube used.
- Unable to place PE tube because of prior scarring: Abandon the case.
- PE tube falls into middle ear space: Surgical retrieval is required

Pacemakers

Peter M. Schulman | January Y. Tsai | Marc A. Rozner

Risk

- In USA, over 3 million people have an implantable cardiac PM, and more than 400,000 PMs are implanted annually.
- In addition to a right atrial and right ventricular lead, some PM pts with cardiomyopathy also have left ventricular pacing capability via a transvenous coronary sinus or epicardial lead (this configuration is called CRT-P).
- Because all conventional ICDs provide antibradycardia pacing, that section of this book applies to these pts as well.*
- The incidence of pts with a PM or ICD (collectively called CIEDs) presenting for surgery is substantial.

Perioperative Risks

- Robust data are lacking; however, the presence of a PM might increase periop risk owing to
  - Associated medical problems.
  - Incorrect interpretation of device type (i.e., confusing a PM for an ICD) or events (i.e., pseudomalfunction).
  - Inappropriate periop management, especially for the pacing-dependent pt.
  - Lack of familiarity with new technology, such as LCP.

*MAGNET CAUTION: A magnet will never change the pacing mode or create asynchronous pacing in an ICD. Only ICDs from ELA (Sorin) will change the pacing rate (to 90 bpm if the battery is OK) upon magnet placement. For many ICDs (Boston Scientific and St Jude Medical),† the magnet switch can be programmed "OFF." Only ICDs from Boston Scientific and its previous companies emit ongoing tones that identify correct placement of a magnet (except subcutaneous ICDs, which only emit a tone for 1 min following magnet application). Some older ICDs from Boston Scientific (with the "GDT" or "CPI" x-ray code) can undergo permanent disabling of tachy therapy by magnet placement. Boston Scientific owns the Guidant and CPI brands, and St Jude Medical owns the Pacesetter brand.

Worry About

- Intraop decrease in pacing rate and/or asystole from EMI: induced ventricular oversensing and pacing inhibition in the pacing-dependent pt.
- Intraop increase in ventricular pacing owing to EMI entering a dual chamber PM and causing atrial lead oversensing and ventricular tracking
- Intraop increases in pacing rates resulting from activation of the "exercise sensor," whether because of direct mechanical stimulation (such as preparation of the chest) or pressure on the device (personnel leaning). Cause of this undesirable tachycardia: Possibly mistaken as inadequate anesthetic depth and inappropriately treated with antichronotropic agents
- Failure to capture (i.e., pacing output without myocardial depolarization) because of inappropriate programmed parameters (i.e., inadequate safety margin), or abrupt increase in pacing threshold from myocardial ischemia/infarction, drug administration, or electrolyte shifts. (Note that any or all chambers can undergo failure to capture with possible hemodynamic derangement, even without apparent outright pacing failure.)
- Hemodynamics being degraded by magnet* placement; magnet placement, which typically (but not always) produces asynchronous pacing at 85–100 bpm (depending upon the brand, model, and programming) and shortens the AV interval to 100 ms in some devices; magnet application to a Medtronic Micra leadless PM, which has no effect and is not programmable; magnet application to a St Jude Nanostim leadless PM, which will provide VOO (asynchronous ventricular) pacing at 100/min for 8 cycles, 90/min assuming the battery status normal, and 65/min if the battery status is "elective replacement indicated, assuming that the magnet sensor is programmed 'ON'"
- Thoracic central line placement in a pacing-dependent pt where the guidewire meets the ventricular lead causing over sensing and pacing inhibition; thoracic central line placement that could cause dislodgement of a new CIED lead because the procedure is relatively contraindicated for at least 6 wk following new lead implant. (Note that spontaneous dislodgement of coronary sinus leads occurs in over 10% of pts.)

Overview

- Indications for permanent pacing: Symptomatic failure of impulse formation (sinoatrial disease), symptomatic failure of impulse conduction (AV block), hypertrophic or dilated cardiomyopathy, and long QT syndrome
- Indications for temporary pacing (usually reversible issue): After cardiac surgery, treatment of drug toxicity resulting in dysrhythmias, certain dysrhythmias complicating MI, and bridge to permanent placement
- Nomenclature: Five positions of the North American Society of Pacing and Electrophysiology (NASPE)/British Pacing and Electrophysiology Group (BPEG) generic pacing code, with the first position referring to the chamber(s) paced (A = atrium, V = ventricle, and D = both, O = none); the second position referring to the chamber(s) sensed (A, V, D, and O), the third position identifying the response to sensed events (I = inhibit, D = dual chamber pacing and tracking); the fourth position being "R" if the CIED increases its rate in response to "exercise" or "O" if rate responsiveness is programmed off; and the fifth position identifying a multisite (A = biatrial, V = biventricular, or D = both) CIED

Etiology

- Congenital electrical disease
- Acquired: Mainly idiopathic or resulting from necessary antiarrhythmic drug therapy; neurally mediated syncope (less common indication); other etiologies include AV ablation, CAD, MI, post-cardiac surgery, dilated infiltrative, or hypertrophic cardiomyopathy, inflammatory disease, infection, neoplasm, and radiation

Usual Treatment

- An in-office PM check should occur at least annually, and telephonic checks should occur quarterly. As the PM pulse generator approaches end of life, monthly checks are recommended.

Assessment Points

System	Effect	Assessment by Hx	PE	Test
CV	Dysrhythmia PM	PM indication	ECG/pulse	Preop PM check; CXR unnecessary to evaluate a properly working device except for multisite pacing device
	Palpitations	Exacerbating cause(s), such as arm movement, body position, or exercise	PM pocket manipulation while monitoring PM; arm movement, flexion/extension of shoulder	PM telemetry
	Exercise intolerance	Exercise tolerance, angina, symptoms of CHF	Two-flight walk	Walk test to ensure correct settings of rate response sensor
ENDO	Atrial tachydysrhythmias	Hypothyroidism, hyperthyroidism		TSH, free T_4
CNS	Other causes of syncope	TIA, CVA	Bruits	Carotid Doppler exam
Code	**Indication**	**Function**	**Perioperative Management**	
VVI	Ventricular bradycardia without need for preserved AV conduction	Demand ventricular pacing	Magnet* utilization might be helpful to produce asynchronous (VOO) pacing 85–100 bpm; Magnet* effect can depend upon programming	
VVIR	Ventricular bradycardia without need for preserved AV conduction and chronotropic incompetence	As above, but adjusts the pacing rate to allow somewhat physiologic response to exercise	PM may sense perioperative changes (e.g., mechanical stimulus or respiratory rate) and respond by increasing pacing rate; the etiology of the faster pacing rate might be misinterpreted as pain or device malfunction.	
DDD	Bradycardia when AV synchrony can be preserved	Provides more physiologic response and maintains AV concordance	Magnet* utilization might be helpful to produce asynchronous (DOO) pacing 85–100 bpm; magnet* effect can depend upon programming	
DDDR	Pts requiring AV synchrony and have chronotropic incompetence	Allows somewhat physiologic response to exercise, and maintains AV concordance	PM may sense perioperative changes (e.g., mechanical stimulus or respiratory rate) and respond by increasing pacing rate; the etiology of the faster pacing rate might be misinterpreted as pain or device malfunction	

Key Reference: Rozner MA: Implantable cardiac pulse generators: pacemakers and cardioverter-defibrillators. In Miller RD, Cohen NH, Eriksson LI, et al., editors: *Miller's anesthesia*, ed 8, Philadelphia, 2015, Elsevier, pp 1460–1486; Schulman PM, Rozner MA, Sera V, et al.: Patient with a pacemaker or implantable cardioverter-defibrillator, *Med Clin North Am* 97:1051–1075, 2013.

Perioperative Implications

Preoperative Preparation

- Before an elective procedure, a CIED care team assessment should be obtained. Comprehensive interrogation should be performed within 12 mo before scheduled surgery for a properly working PM system (or perhaps during preop evaluations for surgery expected to be hemodynamically challenging), and within 3 mo before for any CRT device. Remaining battery life, pacing behavior, and magnet response should be documented.
- Many pacing systems (either PM or ICD) have VVI pacing capability; for the pt with intact atria and an AV node, periop care must be directed to prevent the native sinus rate from falling below the VVI pacing rate because ventricular-only pacing could compromise hemodynamics.
- For the pt who is chronotropically incompetent or pacing dependent and undergoing a major procedure, consider increasing the pacing rate.
- For ventricular multisite pacing (called CRT-P), ensure the LV pacing lead is functioning. If placement of a thoracic central venous cannulation is planned in a CRT pt, the position of the LV (coronary sinus) lead on the CXR should be noted because it may be dislodged during central venous cath insertion.
- Alternate pacing modality (e.g., transvenous, transcutaneous) for the pacing-dependent pt should be available. Even though transesophageal pacing might work as backup, its use is contraindicated in CIED pts, as well as atrial fibrillation and AV nodal block.
- IV chronotropes (epinephrine, ephedrine) should be immediately available.
- Discuss monopolar ESU precautions with surgeon and nursing staff. If monopolar ESU will be used

superior to the umbilicus in a pacing-dependent pt, the PM should be programmed to an asynchronous pacing mode. Programming a PM to an asynchronous pacing mode by applying a magnet* is usually possible and sometimes appropriate; however, it is important to ensure that the magnet mode is active and understand that magnet application can occasionally have unintended and untoward consequences.

Monitoring

- Mechanical pulse wave monitoring is required. It can be accomplished with the pulse oximeter plethysmogram, any invasive hemodynamic monitoring modality, or Doppler technique.
- ECG monitoring is an ASA requirement, but EMI perturbs the signal, and monitors frequently report incorrect heart rates (both too high and too low).

Induction

- Succinylcholine or etomidate might lead to muscle fasciculations or myoclonus, resulting in pacing inhibition or increased rates. Succinylcholine-induced potassium fluxes theoretically can change pacing thresholds. No consensus has been reached about the use of these drugs, and appropriately monitored pts should receive appropriate care.

Maintenance

- Monitor ECG/pulse vigilantly.
- Monopolar ESU cautery (i.e., the "Bovie") emits radiofrequency energy, potentially causing EMI, and resulting in transient or permanent changes in PM function. The most common problem is pacing inhibition. Prevention includes the use of bipolar only ESU and pure unblended (CUT MODE) monopolar ESU, and placement of the ESU current return dispersive electrode so that the presumed current path of the ESU does not cross the pulse generator or leads. For all head and neck or contralateral breast surgery, the dispersive electrode can be placed on the shoulder contralateral to the CIED. For ipsilateral breast surgery, the dispersive electrode can be placed on the ipsilateral arm, and the wire prepped into the field if needed.
- Magnet*: Assuming that the magnet is appropriately placed and that the magnet mode is enabled, placement might be useful to convert the PM to an asynchronous pacing mode to prevent asystole from EMI-induced pacing inhibition. However, the asynchronous pacing rate (which depends on manufacturer and battery status) must be greater than the pt's own rate, or competition will result. Atrial competition usually just lowers the blood pressure, but ventricular competition can lead to R-on-T pacing and induce ventricular tachycardia. Some PMs will change the AV delay to 100 ms, which might compromise hemodynamics in some pts.

Postoperative Period

- Monitor the mechanical pulse in the postop care unit.
- Interrogation/reprogramming is required if the PM was reprogrammed before surgery, and it is advisable if monopolar ESU is employed, any problems are noted, or cardioversion/defibrillation has occurred.
- Some pts require programming changes to optimize postop hemodynamics. These changes might include increasing the pacing rate, disabling battery saving features, and adjusting the AV delay.

Anticipated Problems/Concerns

- Intraop failure to pace, most likely related to EMI from monopolar ESU
- Periop pacing and sensing threshold changes
- Risks related to associated medical problems
- Iatrogenic misadventures resulting from misunderstanding of pacing system behavior

Paget Disease

Annie Santi | Lee A. Fleisher

Risk

- Second most common bone disorder after osteoporosis.
- Most common in individuals of Anglo-Saxon descent; men > women
- Prevalence increases with age and can be as high as 9–15% in people >80 y; average age at diagnosis in USA is 58.
- Within USA, prevalence is highest in the Northeast and lowest in the South.

Perioperative Risks

- Bleeding
- Cardiovascular disease
- CNS structures at risk

Worry About

- Excessive blood loss and transfusion requirement
- Potential difficult airway
- High-output heart failure
- Increased ICP

Overview

- Disorder of markedly accelerated bone resorption with excessive formation of bone with abnormal structural integrity, which may predispose to pathologic fracture or cause impingement of surrounding structures.
- Pathophysiology: Early: Exaggerated osteoclastic activity and accelerated bone resorption, resulting in the formation of abnormal bony matrix. Meanwhile bone marrow is replaced by fibrous connective tissue and blood vessels. Late: Pagetic bone becomes large and sclerotic, with reduced tensile strength.
- Typically affected are pelvis, femur, spine, skull, tibia; disease may be limited to one bone (monostotic) or affect many (polyostotic).
- Pain is the most common presenting complaint.
- Radiographic findings include osteolytic and osteosclerotic lesions; "cotton wool" appearance of skull, cortical and trabecular thickening, hyperostosis, bowing of tibia/femur; technetium bone scan is the most sensitive diagnostic tool for determining sites and extent of PD.
- Serum alkaline phosphatase is usually elevated, and the degree of elevation correlates with extent of disease and level of pagetic activity; serum calcium and phosphate are typically normal.
- Risk of progression to neoplasm <0.5%.

Etiology

- The majority of cases are idiopathic; however, a strong genetic component exists in a subset of pts with familial PD; 15–30% of PD pts have a positive family history, which correlates with more severe disease; inheritance is typically in an autosomal dominant pattern with high penetrance.
- Individuals who have first-degree relatives with PD can have up to a 7-fold to 10-fold higher risk.
- Most common mutation is in a gene encoding a scaffold protein, with important effects on osteoclast differentiation and function.
- There is controversial evidence that a viral etiology may play a role in the development of disease in pts already genetically predisposed to PD.

Usual Treatment

- Bisphosphonates are first-line therapy, with remission rates varying from months to years depending on the agent; these drugs inhibit osteoclast activity and bone resorption; therapeutic response is measured by serum alkaline phosphatase levels.
- Calcitonin also inhibits osteoclast activity and has a more rapid onset but is overall less effective, with high rates of recurrence upon drug withdrawal; its use is mostly limited to pts intolerant of bisphosphonates or with renal insufficiency.
- Antipagetic therapy can cause hypocalcemia; adequate calcium intake and vitamin D levels must be ensured.

Assessment Points

System	Effect	Assessment by Hx	PE	Test
HEENT	Mandibular sclerosis	Jaw pain, mobility	Poor mouth opening	X-ray
RESP	Thoracic bony deformity	Chest wall pain		CXR, spirometry
CV	High output heart failure Valvular disease (i.e., aortic stenosis) Conduction abnormalities	Dyspnea Syncope, exertional chest pain	Tachycardia, dyspnea, edema SEM Heart block	TTE ECG
CNS	Increased ICP Spinal stenosis	Headache Radiculopathy	Papilledema	CT, MRI, X-ray
MS	Bony enlargement Reduced tensile strength of bone	Pain, fracture	Enlarged skull Bowing of lower extremities	Technetium bone scan X-ray Serum alkaline phosphatase
METAB	Hypocalcemia (typically on antipagetic drugs) Vitamin D deficiency			Ca^{2+}, Mg^{2+}, phosphate, serum 25-OHD, PTH

Key References: Tucci J: Preoperative management of Paget's disease. In Aaron RK, editor: *Diagnosis and management of hip disease,* Switzerland, 2015, Springer, pp 159–184; Tetzlaff J, Benedetto P: Skin and bone disorders. In Fleisher LA, editor: *Anesthesia and uncommon diseases,* ed 6, Philadelphia, PA, 2012, Saunders, pp 319–349.