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.

Perioperative Implications

Preoperative Preparation

- Perform a thorough review of systems to identify coexisting disease in this older pt population and a thorough airway exam, including assessment for atlantoaxial instability; obtain radiographs if necessary.
- Check lytes, serum alkaline phosphatase; CBC for baseline hemoglobin.
- Echocardiography to assess cardiac function and potential sclerotic valvular disease (calcific disease of the aortic valve is most common); ECG to assess to conduction abnormalities; assess for ICP and obtain additional imaging if necessary.
- Ensure blood availability.
- If elective procedure, make sure that antipagetic therapy is instituted preop.

Monitoring

- Invasive BP monitoring may be appropriate.
- Neuromonitoring may be required for procedures involving the spine.

Airway

- Advanced airway equipment may be required for pts with significant cervical spine and/or mandibular disease; fiberoptic intubation may be indicated.

Preinduction/Induction

- If significant cardiovascular or valvular disease is present, aggressive BP management may be required during induction, including the use of cardiac neutral induction agents.
- Neuraxial anesthesia may be difficult if significant spine involvement is present
- Avoid medications contraindicated in pts with increased ICP.

Maintenance

- The majority of pts will be >50 y and may therefore require a lower MAC of anesthesia; there are no specific interactions between anesthetic agents and PD.
- Avoid medications that can increase ICP; can consider hyperventilation if ICP is an issue.
- Maintain normotension and avoid tachycardia in pts with cardiac disease; manage fluids carefully; maintain normothermia to decrease bleeding.

Extubation

- If pt has a difficult airway, it is crucial to avoid the need for emergent reintubation.

Postoperative Period

- Multimodal analgesia will be necessary, as some pts may have chronic pain at baseline due to pagetic activity.
- Mobility may be difficult depending on the extent of disease involvement.
- Lyte monitoring and telemetry may be indicated.
- Postop pulm toilet is important, given an elderly population, especially if there is thoracic involvement.

Anticipated Problems/Concerns

- Risk of injury during positioning.
- Sclerotic bone may be more difficult to manipulate and can prolong surgical time.
- IV access may be challenging if vessels are calcified.

Pancreatitis

Benjamin Rubin | Alexandria Piedmont

Risk

- Incidence of AP varies from 4.9–73.4 cases per 100,000 worldwide.
- Prevalence of CP has recently been estimated at 12–45 cases per 100,000, although its true prevalence is unknown.

Perioperative Risks

- Most mortality occurs with surgery for complications of severe pancreatitis: 10–30%
- Risk of nonpancreatic surgery probably dependent on severity of attack

Worry About

- Severe hypovolemia secondary to sequestration of fluid in the retroperitoneal space
- Lyte abnormalities, including hypocalcemia, hyperglycemia, and acidosis
- Systemic complications such as alcohol withdrawal, ARDS, acute renal failure, DIC, multisystem organ failure, and sepsis

Overview

- AP involves an intense inflammatory response caused by the release of activated pancreatic enzymes, with resultant tissue destruction as well as the loss of fluid and electrolytes.
- AP diagnosis requires at least two of three criteria: amylase and/or lipase >3 times the upper limit of normal, abdominal pain consistent with disease, and/or characteristic abdominal imaging.
- AP is most commonly a mild self-limited disease; it is occasionally severe, with renal, pulm, coagulatory, and septic complications.
- CP results from inflammatory cell infiltration, formation of granulation tissue and fibrosis, and loss of pancreatic parenchyma, leading to exocrine and endocrine insufficiency.

Etiology

- Acute: Most common risk factors are gallstones and excessive alcohol consumption. Others include post-ERCP, drugs, viral infections, metabolic disorders, and abdominal trauma.
- Chronic: In adults, chronic alcohol use accounts for 70% of cases. In children, genetic diseases and anatomic defects are more likely.

Usual Treatment

- In most cases, nonspecific and supportive only.
- Adequate volume replacement and correction of electrolyte abnormalities.
- Intensive care of organ system failures.
- Parenteral opioid analgesia.
- Thromboprophylaxis.
- Early nutritional support; enteral better than parenteral.
- Pts with AP and concurrent acute cholangitis should undergo ERCP within 24 h of admission.
- Rarely, judiciously timed open or endoscopic surgery to drain abscesses or debride necrotic tissue.
- For biliary AP, timing of cholecystectomy dependent on severity.
- CP is primarily managed medically.
- Endoscopic/surgical management of CP is aimed at decreasing pain and treating associated complications, such as strictures (biliary and pancreatic), ductal leaks, intraductal calculi, or pseudocysts.

Assessment Points

System	Effect	Assessment by Hx	PE	Test
CV	Hypovolemia	Orthostatic dizziness Cold	Lying and sitting BP and HR Hypotension Oliguria	BUN/Cr Hct (hemoconcentration)
RESP	ARDS	Dyspnea Tachypnea	Chest exam may be nonspecific	ABG CXR
GI	Ileus GI bleed	N/V Hematemesis		
ENDO	Hyperglycemia			Serum glucose
HEME	DIC		Bleeding	PT/PTT, plts FSP, fibrinogen Hct
RENAL	ARF Hypocalcemia		Tetany	BUN/Cr Serum Ca^{2+}
CNS	Psychosis Encephalopathy		Mental status	

Key References: Inui K, Yoshino J, Miyoshi H, et al.: New developments in diagnosis and non-surgical treatment of chronic pancreatitis. *J Gastroenterol Hepatol* 28(Suppl 4):108–112, 2013; Tenner S, Baillie J, DeWitt J, et al.: American College of Gastroenterology guideline: management of acute pancreatitis. *Am J Gastorenterol* 108(9):1400–1415, 2013.