Maintenance
- Maintain anesthesia with propofol, short-acting opioids, and/or volatile inhalational agents depending on technique used.
- Avoid paralytic agents if possible.
- Pt is usually placed in microlaryngeal suspension for surgical procedures.

Extubation
- Use caution and assess for bleeding or edema.
- Suction thoroughly and extubate awake to prevent aspiration and laryngospasm.
- At the end of procedure, ET tube is usually placed while the pt is given time to wake up from anesthesia.

Adjuvants
- Consider dexamethasone for swelling of airway mucosa from repeated intubations or resections.

Postoperative Period
- Some pts will require humidified oxygen or nebulized racemic epinephrine if stridor occurs in PACU.
- Monitor SpO_2 for desaturation; some pts may require reintubation.

Anticipated Problems/Concerns
- When using CO_2 laser therapy, must use laser-safe ETT to prevent ignition from laser.
- Use of jet ventilation is common for ENT procedures. Concern for dissemination of HPV particles into distal airway and barotrauma from jet ventilator.
- Important to debulk as much pathology as possible while preserving normal tissue to prevent scarring and airway stenosis over time with repeated surgical therapy.

Parkinson Disease (Paralysis Agitans)

Stacie Deiner | Jess Brallier

Risk
- Advanced age
- 1% of population >65 y
- No difference in distribution by gender

Perioperative Risks
- Hemodynamic instability, hypotension, arrhythmias
- Aspiration and upper airway obstruction from poor coordination of upper airway muscles
- Laryngospasm
- Postop confusion and hallucinations

Worry About
- Exacerbation of PD symptoms triggered by dopamine antagonists such as metoclopramide; also phenothiazines, butyrophenones
- Potential drug interactions: MAOIs; meperidine

Overview
- Pathophysiology: Symptoms result from the loss of dopaminergic cells in the pars compacta region of the substantia nigra reticulata. This loss upsets the normal balance between dopaminergic inhibition and cholinergic excitation.
- At least two of the following clinical manifestations are required for the diagnosis of PD: postural instability, bradykinesia, resting tremor, and rigidity. Other common features include depression, anxiety, sensory abnormalities, anosmia, autonomic dysfunction, cognitive impairment, and sleep disturbances.

Etiology
- Etiology unknown; possible genetic predisposition; possible neurotoxin involvement.

Usual Treatment
- Pharmacologic: The goal of current medical therapies is to maintain motor function and quality of life by restoring the dopaminergic/cholinergic balance in the striatum and blocking the effect of Ach.
  - Dopamine precursors
    - l-Dopa (a prodrug converted to dopamine in the brain): Mainstay of therapy, ameliorates all major clinical features of parkinsonism. Often helpful for hyperkinesias. Levodopa treatment is characterized by "on" periods of symptom amelioration and possible dyskinesias followed by "off" periods with decreasing therapeutic levels of dopamine and return of symptoms.
    - Carbidopa: Inhibits dopa decarboxylase, the enzyme responsible for the conversion of levodopa to DA. Limits breakdown of levodopa outside the CNS and increases the effectiveness of levodopa while also minimizing side effects.
    - Sinemet: Combination of carbidopa/levodopa.
  - DAs: Less effective than levodopa in relieving signs/symptoms of PD but less likely to cause dyskinesia and the on-off phenomenon. These drugs include ergot alkaloids (bromocriptine, cabergoline, lisuride), and nonergot alkaloids (pramipexole, ropinirole, rotigotine).
  - Anticholinergics: Trihexyphenidyl benztropine—more helpful for tremor and rigidity; generally less effective than DA drugs.
  - Antivirals: Amantadine—given for mild parkinsonism. Used alone or in combination with anti-Ach. Unclear mechanism of action. Improves all clinical features of PD.
  - MAO-B inhibitors: Selegiline—inhibits breakdown of DA and enhances antiparkinsonian effect of levodopa. May reduce the on-off phenomenon.
  - COMT inhibitors: Entacapone and tolcapone—help sustain plasma levels of levodopa. Decreases the dose and response fluctuations due to carbidopa/levodopa (Sinemet).

Surgical
- Lesioning: Historically, surgical intervention was primarily limited to lesioning of deep brain structures. The idea was that permanent lesioning would remove stimuli due to abnormal CNS activity (thalamotomy, used to treat tremor; pallidotomy, used to treat levodopa-induced dyskinesia/antiparkinsonian effects).
- Although affording some clinical benefits, such operations were also shown to result in permanent side effects, such as paresis, confusion, quadrantanopsia, gait disturbances, dysarthria, and hypersalivation.
- Such surgeries were associated with high complication rates and no possibility of lowering anti-PD drugs. These procedures are rarely performed today, having been replaced by DBS.
- DBS: In the late 1980s it was discovered intraop that high-frequency electrical stimulation could produce the same functional effect as surgical lesioning. This introduced DBS as a primary treatment modality. DBS has revolutionized the treatment of PD.
- The CNS targets of DBS include the ventralis intermedius nucleus (VIM), the subthalamic nucleus (STN), and the globus pallidus (GPi). However, the effects of VIM DBS on the other symptoms of PD (akinesia, rigidity, bradykinesia, etc.) are short-lived or nonexistent. GPi or STN DBS is used to treat these other symptoms.

Assessment Points

System	Effect and Assessment by Hx and PE	Test
ANS	Difficulty with salivation, micturition, temperature regulation, GI function	
CNS	General muscle rigidity, akinesia, tremor, confusion, depression, hallucination, speech impairment	CT, MRI
RESP	Upper airway dysfunction: Retained secretions, atelectasis, respiratory infections, aspiration pneumonia (most common cause of death) Other complications: Postextubation laryngospasm, postop respiratory failure	CXR, CT lung
GI	Dysphagia, esophageal dysfunction, constipation, weight loss, sialorrhea	
ENDO	Abnormal glucose metabolism	Glucose metabolism

Key References: Deiner S, Hagen J: Parkinson's disease and deep brain stimulator placement, *Anesthesiol Clin* 27(3):391–415, 2009; Osborn IP, Kurtis SD, Alterman RL: Functional neurosurgery: anesthetic considerations, *Int Anesthesiol Clin* 53(1):39–52, 2015.

Perioperative Implications of Deep Brain Simulation Surgery

Preoperative Preparation
- A complete assessment of the extent of the pt's PD and other medical comorbidities should occur.
- A full explanation of what to expect with each step of the procedure is imperative and, when possible, should occur prior to the day(s) of surgery. DBS procedures are most often staged, with lead placement performed on one day and the generators placed on another.
- The pt's ability to cooperate should be assessed, and he or she should be mentally prepared to have part of the procedure performed while awake.
- Hold Parkinson medications on the morning of surgery.
- Avoid or limit medications that can affect the microelectrical recordings (MER) used to guide DBS lead placement or suppress PD tremor (i.e., benzodiazepines).

Monitoring
- ASA standard monitoring modalities.
- An arterial line is indicated when severe tremor precludes accurate use of a noninvasive cuff or in pts with significant medical comorbidities.
- Possible use of Foley catheter.

Intravenous Access
- One peripheral IV line is generally sufficient (usually a 20- or 18-gauge catheter).

Airway
- Access to the airway is limited due to the presence of the stereotactic head frame. In the case of an airway emergency, supraglottic airways and ultimately the head frame key (used to remove the frame) should be immediately available.

Intraoperative Period
- DBS surgery requires the pt to be secured in a stereotactic head frame and awake for a portion of the operation. This allows for superior identification of the brain areas involved in the pathology by maximizing the quality of MER and pt cooperation.
- Many practitioners use an awake-asleep-awake technique with sedation utilized during burr-hole creation, interrupted during lead placement and MER, and restarted during surgical closure. A scalp nerve block is performed at the very beginning of the procedure in order to provide analgesia throughout the operation.
- It is important to select anesthetic agents that minimally affect MER quality, tremor, and pt cooperation.
  - Dexmedetomidine (alpha-2 agonist): Has minimal effect on MER, provides sedation while preserving pt cooperation, minimal resp depression, and does not suppress PD tremor.
  - Propofol: Short duration of action, easily titratable. MER and tremor return to baseline with discontinuation of infusion.
  - Opioids (fentanyl and remifentanil): These have minimal effect on MER and suppress PD tremor. Resp depression precludes their use in high doses.
  - Benzodiazepines: Reduce quality of MER. Can cause respiratory depression, suppress PD tremor, and can impair pt cooperation.
- Optimize pt comfort while positioning.
- Complications of DBS surgery:
  - Intracranial hemorrhage (highlights importance of stringent blood pressure control)
  - Seizure
  - Venous air embolism
  - Infection
  - Pneumocephalus

General Anesthesia
- Reserved for pts who cannot tolerate awake procedure (pediatric pts, uncooperative pts).
- The major concerns are that GA can diminish intraop MER used to ensure proper lead placement. GA also inhibits macrostimulation testing by eliminating tremor and preventing pt cooperation and feedback (another tool used by surgeons to ensure proper lead placement).
- Recent studies suggest that the concerns surrounding GA for lead placement may be overstated. Sizable studies have demonstrated successful lead placement under GA and that MER can still be successful as long as anesthetic agents are carefully titrated.
- Additional randomized controlled trials are needed to objectively evaluate the role of GA for DBS cases.

Perioperative Implications for Non–Deep Brain Simulation Surgery

Preoperative Preparation
- Continue PD medications the morning of surgery
- Administer PD medications via OG/NG tube at regularly scheduled intervals during surgery to prevent exacerbation of parkinsonism.

Monitoring
- ASA standard monitoring modalities

Airway
- Aspiration risk
- Upper airway obstruction

Induction
- Many PD pts are exquisitely sensitive to the cardiovascular and respiratory depressant effects of many anesthetic agents. These pts may require dose adjustments. Titrate carefully. Propofol, etomidate, and ketamine are all appropriate.

Maintenance
- Exaggerated vasodilatation and cardiodepressant effects with volatile anesthetics
- Nondepolarizing NMB drugs well tolerated but mask tremor
- Enhanced opioid-induced muscle rigidity following fentanyl administration
- Increased risk of neostigmine-induced bronchoconstriction

General Anesthesia
- May see transient appearance of otherwise pathologic neurologic reflexes (hyperreactive stretch reflexes, ankle clonus, Babinski reflex, decerebrate posturing) on emergence

Regional Anesthesia
- Advantageous
- Diphenhydramine useful for sedation

Postoperative Period
- Confusion, delirium, hallucinations common
- Shivering common

Anticipated Problems/Concerns
- Be aware of all parkinsonian medications and possible drug interactions, particularly with MAO inhibitors.
- Avoid drugs that exacerbate parkinsonism (phenothiazines, butyrophenones, and metoclopramide).
- Use caution with airway management, especially keeping in mind postextubation laryngospasm and respiratory failure.

Paroxysmal Atrial Tachycardia

David Amar

Risk
- May be seen in ICU pts and is indistinguishable from paroxysmal SVT
- Digitalis toxicity, acute lyte or acid-base imbalance
- Incidence of 2% in the periop period (excluding AF)
- No racial prevalence and all age groups
- May be seen with mitral valve prolapse, especially in females

Perioperative Risks
- Rapid heart rate impairs LV filling and may adversely affect LV function in pts with LV failure, hypertrophic cardiomyopathy, and aortic or mitral stenosis.
- Cerebrovascular disease.

Worry About
- Syncope ~15% on initiation or abrupt termination of rapid SVT.
- Syncope may also indicate AF and rapid conduction over an accessory pathway.
- Hypotension in pts with systolic or diastolic dysfunction.
- Chest pain in pts with CAD.
- ST-T segment changes common with rapid rates and reduced coronary filling even with normal coronaries.
- VF in WPW pts who develop AF.
- Digoxin level, lyte, and acid-base status.

Overview
- PAT is among a larger group of narrow (<120 ms) QRS-complex tachycardias defined by the ACC/AHA/ESC task force to include PSVT, AF/flutter, permanent junctional tachycardia and focal atrial tachycardia, and macro-reentrant tachycardia.
- Rapid atrial arrhythmias, primarily AF, occur after any major surgery in pts >45 y of age (2–4%) but with a greater incidence after cardiac (25–45%) or thoracic (4–27%) surgery. Such arrhythmias peak 2–3 d after surgery. Acute postop events such as pneumonia or ARDS may increase the incidence.
- Causes are multifactorial and include autonomic imbalance (sympathetic and vagal excess), oxidative stress, and atrial myocardial inflammation. Predisposing factors include atrial fibrosis, left atrial enlargement, and diastolic dysfunction.
- Common mechanisms of narrow complex tachycardias in the periop period:
  - Reentrant rhythms: AV nodal reentrant tachycardia, AV reciprocating tachycardia through accessory pathway, AF/flutter (most common; seen in over 90% of pts).
  - Unifocal or ectopic atrial tachycardia.
  - Multifocal atrial tachycardia in pts with chronic pulm disease.
- A wide-complex tachycardia (QRS >120 ms) may represent either VTach or SVT with abnormal conduction. Adenosine is suggested as first-line therapy if the arrhythmia is monomorphic, regular, and hemodynamically tolerated because adenosine may help convert the rhythm to sinus and may help in the diagnosis. When doubt exists, it is safest to assume any wide-complex techycardia is VTach. The failure to correctly identify VTach can be potentially life threatening, particularly if misdiagnosis results in VTach being treated with verapamil or diltiazem.

Etiology
- Reentrant rhythms.
- AV nodal reentry: Reentrant pathway within the AV node. Most common form of PAT; seldom associated with organic heart disease.
- Accessory pathway–mediated: Reentrant rhythm that involves an accessory pathway from atrium to ventricle. In sinus rhythm, the bypass tract may cause a preexcitation pattern on ECG (WPW syndrome: short P-R interval and delta wave on ECG) or may not be apparent.
- Unifocal atrial tachycardia arising from a single atrial muscle site other than SA node; associated with catecholamine excess states (uncontrolled pain, light anesthesia) or digitalis toxicity (triggered activity with variable AV block).
- Multifocal atrial tachycardia arising from multiple atrial sites, usually seen in pts with pulm disease or CHF.

Usual Treatment
- Initial therapy: Vagal maneuvers (i.e., Valsalva, carotid massage [avoid in known carotid disease or with presence of bruit] or applying ice-cold wet towel to the face) should be initiated to terminate the arrhythmia.