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.

- IV adenosine (especially in diagnosis of wide-complex tachycardia that could be VTach or if WPW or preexcitation is suspected) or CCBs (diltiazem or verapamil) are the drugs of choice but beta-blockers may also be used. Adenosine may provoke bronchospasm in pts with reactive airway disease, with excessive (prolonged) bradycardia in patients taking carbamazepine, or in denervated heart transplant pts. Higher doses of adenosine may be needed in pts taking methylxanthines (i.e., theophylline). Adenosine may initiate AF in 1–15% of pts; it is usually transient.
- The goal of second-line therapy is to achieve ventricular rate control and possible conversion when PAT does not respond or rapidly recurs after adenosine. IV digoxin is not effective unless CHF is present.
- When AV nodal block is unsuccessful, electrical cardioversion is considered. If infeasible or unsuccessful, antiarrhythmic agents may also be used. When LV function is preserved, IV options include procainamide and amiodarone. The proarrhythmic potential of these agents makes them less desirable than AV nodal blockade. In patients with poor LV function, IV amiodarone is preferred.
- Pts with accessory pathway reentrant rhythms who develop AF are at risk for VFIB; this scenario is exacerbated by agents that reduce the accessory bundle refractory period (digoxin, CCBs, beta-blockers, and adenosine). Hence WPW pts who experience AF should not receive AV nodal blockers. IV procainamide and amiodarone are preferred agents to slow the rate and achieve conversion.
- Multifocal and unifocal PAT: Correct underlying hypoxia and lyte imbalance. Therapy: Electrical cardioversion and procainamide are not effective. Effective IV agents available for use include AV nodal blockers (CCBs, beta-blockers) and amiodarone. Although digoxin slows the ventricular rate, toxicity may provoke automatic atrial tachycardia.

Assessment Points

System	Effect	Assessment by Hx	PE	Test
CV	WPW AV nodal reentry Symptomatic unifocal atrial tachycardia	Palpitations, diaphoresis Hypotension, chest pain	Prominent jugular venous pulsations	ECG (150–250 bpm, abnormal P waves preceding QRS, rarely discernible) Electrophysiologic studies, ECHO
NEURO	Rapid arrhythmia	Fatigue, presyncope or syncope		
RESP	Rapid arrhythmia	Dyspnea	Rales, wheezes	CXR
RENAL	Atrial dilation	Polyuria		BNP, BUN/Cr

Key References: Page RL, Joglar JA, Caldwell MA, et al.: 2015 ACC/AHA/HRS guideline for the management of adult patients with supraventricular tachycardia: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Rhythm Society, *J Am Coll Cardiol* 67:e27–115, 2016; Amar D. Perioperative atrial tachyarrhythmias, Anesthesiology 97:1618–1623, 2002.

Perioperative Implications

Preoperative Preparation

- If possible, continue CCBs and beta-blockers periop to avoid withdrawal-associated arrhythmias.
- Correct hypoxemia and lyte imbalance.
- Consider guideline-driven prophylactic regimens for high-risk pts undergoing cardiac or thoracic surgery.
- Pts with recurrent arrhythmias may be taking drugs such as flecainide, propafenone, amiodarone, or dofetilide for prevention.
- Pts with refractory arrhythmias have usually had electrophysiologic studies and catheter ablation procedures.

Monitoring

- Continuous intraop and postop ECG monitoring in high-risk pts

Induction/Maintenance/Extubation

- Aim for effective postop analgesia.
- Consider beta-blockers in hyperadrenergic postop patients whose cardiac output is adequate.

Anticipated Problems/Concerns

- Transient side effects with adenosine include flushing, dyspnea, and chest pain. Adenosine may provoke hypotension, especially in patients with borderline hemodynamic status.
- Wide-complex rhythms: Adenosine may be used if the rhythm is confirmed by other means to be supraventricular in origin. The use of adenosine to discriminate VT from SVT is now discouraged owing to vasodilatory side effects (worsened hypotension) in pts with VT.
- Diltiazem is highly effective but may be associated with transient hypotension; this can be minimized with slow titration of the drug, α-agonists, and correction of hypovolemia.

Patent Ductus Arteriosus

Aris Sophocles | Mark Twite | Jeffrey D. Roizen

Risk

- Full-term infants: 1:2000
- Preterm infants: 8:1000
- Highest in preterm and low–birth-weight infants
- Female-to-male ratio: 2:1
- Associated with congenital rubella infection and genetic defects, including trisomy 21, CHARGE, and a familial recurrence rate of 3%

Perioperative Risks

- Surgery: Hemorrhage; hemodynamic instability, especially in premature and low–birth-weight neonates; single-lung ventilation resulting in hypoxia, atelectasis, and pneumothorax; injury to the recurrent laryngeal nerve; chylothorax; ligation of the incorrect vessel (aorta or pulm artery); thoracic scoliosis over the long term
- Closure by an occluding device via cardiac cath: Obstruction of the pulm artery and/or aorta from the occluding device, arrhythmias, incomplete closure, and embolization of the device

Worry About

- Premature infant: Lung disease and high mechanical ventilator settings, hemodynamic instability after duct closure due to poor cardiac reserve
- Term infant and young child: Preop dehydration, ability to tolerate single-lung ventilation, postextubation stridor due to injury to the recurrent laryngeal nerve, postop analgesia
- Older child and adult: Pulm Htn

Overview

- Preterm and low–birth-weight infants: PDA may cause CHF and worsening of chronic lung disease, which makes weaning from mechanical ventilation difficult.
- Term and older infants: PDA may be asymptomatic or associated with failure to thrive, recurrent resp infections, and CHF.
- A "silent duct" is a small PDA detected on echocardiography, with no murmur heard.
- PDA leads to an increased risk of endocarditis.

Etiology

- Normal: The arterial duct is the connection between the pulm artery and the aorta; it shunts blood away from the lungs during fetal development in utero. The duct normally constricts shortly after birth owing to the postnatal drop in circulating prostaglandin levels as well as the rise in systemic O_2 tension. Constriction is followed by permanent duct closure due to the hypertrophy of endothelial and smooth muscle cells and eventual formation of the ductal ligament.
- PDA: In preterm infants the ductal muscle layer is thin and poorly contractile; it has a poor constrictor response to changes in arterial oxygen tension.

Usual Treatment

- Medical management: Neonates often receive a trial of ibuprofen or indomethacin. These act by inhibiting prostaglandin-forming COX enzymes. Adverse drug effects include renal dysfunction and NEC.
- Surgical management:
  - Bedside left lateral thoracotomy: Reserved for critically ill ventilated pts who have failed medical therapy.
  - Operated left-lateral muscle-sparing thoracotomy or video-assisted thoracoscopic surgery: For a stable child, technique is surgeon's preference, with most children receiving a thoracotomy. Candidates are usually not suitable for device closure (less than 8 kg) or unusual duct anatomy.
  - Cardiac cath lab: Reserved for children weighing more than 8 kg owing to the size of the femoral sheaths through which the occluding device is introduced. Large PDAs are occluded with an Amplatzer device and small PDAs are occluded with coils.