- 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.

Assessment Points

System	Effect	Assessment by Hx	PE	Test
CV	CHF	FTT, difficulty feeding	"Machinery" murmur Wide pulse pressure Bounding pulses Pulsus bisferiens	ECHO
	Pulm Htn		Tachycardia Diaphoresis	
RESP	Pulm edema	Recurrent respiratory infections Increased O_2 requirement	Worsening mechanical ventilation parameters Rales	CXR
GI	NEC	Abdominal distention Poor feeding Blood in stool Free air in peritoneum	Distended tense abdomen Edema of abdominal wall Tender abdomen	Abdominal x-ray
RENAL	Oliguria	Decreased UO due to decreased renal blood flow		Serum chemistry
CNS	CNS hemorrhage	Increased fontanel pressure Decreased Hct	Increased fontanel size and tension	Head US

Key Reference: Jacobs JP, Giroud JM: Evolution of strategies for management of the patent arterial duct, *Cardiol Young* 17(Suppl 2):68–74, 2007.

Perioperative Implications

Preoperative Preparation

- Surgery:
  - Unstable neonate, bedside: Cross-matched blood at bedside, adequate IV access with extension tubing. Caretakers must be familiar with ventilator function and settings and must check current running infusions (TPN, vasopressors).
  - Stable child: Cross-matched blood in the OR.
- Cardiac cath lab: Routine setup for general ET anesthesia.

Preinduction/Induction

- Unstable neonate: Induce with fentanyl (10–30 mcg/kg)
- Stable child in cath lab/OR: Premedication and mask induction

Monitoring

- Standard ASA monitors.
- Unstable neonates require an arterial line for continuous BP measurement and blood gas analysis and central venous access for inotrope drug delivery.
- Stable older children do not require invasive monitoring.

Airway

- Critically ill neonates are already intubated and ventilated. Check tube size for leak and confirm position on CXR.
- OR cases: Intubate for single-lung ventilation (right main stem, a single-lumen ETT, bronchial blocker, or double-lumen tube).
- Cath lab cases: Young children often require intubation; older cooperative children may be treated with a natural airway.

Maintenance and Extubation

- Critically ill neonate, bedside: Fentanyl and paralytics; patient should remain intubated at the end of the procedure.
- Stable child, OR: Balanced anesthetic technique with the goal of early extubation and adequate analgesia (consider regional techniques).
- Stable child, cath lab: Balanced anesthetic technique and extubate at the end of the procedure. Analgesic requirements are minimal and related to the femoral vessel puncture sites.

Adjuvants

- Antibiotic prophylaxis for all cases (usually cefazolin 30–50 mg/kg)

Postoperative Period

- Adequate analgesia

Anticipated Problems/Concerns

- Critically ill neonates: Often require a transient increase in BP and respiratory support.
- Stable children: Postop surgical ligation via a thoracotomy; such patients may have atelectasis from single-lung ventilation and also thoracotomy pain.

Patent Foramen Ovale

Ronit Lavi | Daniel Bainbridge

Risk

- Incidence: 25–30% at autopsy.
- Atrial septal aneurysm (a deformity of the septum that results in deviation of the septum more than 15 mm into either atrium) is associated with at least 50% of PFOs and is considered an additional risk factor for stroke.

Perioperative Risk

- Unclear if a PFO increases the risk of stroke or cognitive dysfunction in the periop period.

Worry About

- R-to-L shunting of blood leading to profound hypoxemia
- Paradoxical embolization of air, blood clot, or tissue fragments, potentially resulting in stroke

Overview

- The foramen ovale directs oxygenated blood returning from the placenta and into the right atrium across the intra-atrial septum to the left ventricle.
- As right-sided pressures decrease after birth, the foramen ovale flap is pressed against the septum secundum.
- This results in the fusion of the ovale flap to the septum secundum; irreversible closure of the ovale occurs in 75% of pts.
- Diagnosed by:
  - Right heart cath, with the ability to cross a guide wire across the atrial septum.
  - TEE is considered the "gold standard" imaging technique, using a contrast agent (bubble study) and provocative technique (Valsalva maneuver).
  - TCD is less invasive than TEE with similar sensitivity but reduced specificity.
  - TTE: Sensitivity 50% of TEE, with similar specificity.
- See also Atrial Septal Defect.

Etiology

- Unknown what, if any, risk factors predispose to patent foramen ovale
- A higher incidence of PFO was found in pts who suffer migraine with aura; unclear whether this represents coexistence or a causal relationship between the two entities.

Usual Treatment

- Anticoagulation has not been shown to reduce cryptogenic stroke.
- Percutaneous closure for pts with history of cryptogenic stroke.
- Surgical closure in pts who are not candidates for percutaneous closure.

Assessment Points

System	Effect	Assessment by Hx	PE	Test
CV	Rarely RV overload secondary to chronic L-to-R shunting	Fatigue, abdominal pain	Hepatic enlargement, elevated JVP, peripheral edema	ECG showing right axis deviation ECHO showing large RV PFO
CNS	Assoc with cryptogenic stroke, migraine	Hx of migraine headache, stroke unrelated to carotid Dx, AFIB		

Key References: Sukernik MR, Mets B, Bennett-Guerrero E: Patent foramen ovale and its significance in the perioperative period, *Anesth Analg* 93(5):1137–1146, 2001; Kutty S, Sengupta PP, Khandheria BK: Patent foramen ovale: the known and the to be known, *J Am Coll Cardiol* 59(19):1665–1671, 2012.