

Perioperative Implications**Preoperative Preparation**

- Smoking cessation
- Antibiotics to decrease sputum production
- Resp conditioning

Monitoring

- Consider arterial line to monitor blood gases
- Consider pulm artery catheter for large fluid-shift operations

Airway

- Often, truncal obesity (especially with corticosteroids); may have redundant soft tissue in airway or a short, fat neck

Preinduction/Induction

- Avoid stimulating the airway while pt is in light levels of anesthesia because it may precipitate bronchospasm (although less likely than with asthma).
- Regional anesthesia may be preferable.

Maintenance

- Frequent suctioning of ETT
- Limit narcotic administration (danger of periop CO₂ retention)
- Adjuvant regional anesthesia for postop pain management in procedures that affect resp mechanics (e.g., intercostal nerve blocks, epidural analgesia)

Extubation

- Administer intratracheal bronchodilator in responsive pts before extubation.
- Consider IV lidocaine before extubation.

Anticipated Problems/Concerns

- Postop resp complications (secretions, mucus plugging, atelectasis, pneumonia, and prolonged requirement for mechanical ventilation)

Bronchopulmonary Dysplasia

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Risk

- Incidence in USA: 10,000-15,000 infants annually
- Risk increases with decreasing gestational age and birth weight
- Affects at least one-quarter of infants with birth weights <1500 g
- No race or gender predilection

Perioperative Risks

- Bronchospasm
- Pulm Htn
- Cor pulmonale

Worry About

- Airway obstruction and hyperreactivity
- Pulm Htn and cor pulmonale
- "BPD spells": Acute cyanotic events caused by increases in central airway resistance
- Tracheomalacia and/or bronchomalacia
- Recurrent pulm infections

Overview

- Chronic lung disease associated with premature birth and positive pressure mechanical ventilation, the clinical definition of which has evolved over time
 - "Classic BPD": Associated with characteristic radiographic changes and four stages of lung injury: exudative → necrosis → pulm fibrosis → severe cystic changes, and cor pulmonale
 - "New BPD": Seen after introduction of surfactant therapy, antenatal steroid administration, and improved neonatal ventilator strategies; mild respiratory distress syndrome and continued need for supplemental oxygen; and lung development is uniformly arrested, with simplified alveolar structures and dysmorphic capillaries
- Disease severity (mild, moderate, or severe) determined by the gestational age of the infant, oxygen dependency at 36 wk postconceptional age, total duration of oxygen supplementation, and positive pressure requirements

- Chronic airway obstruction and hyperreactivity present in long-term survivors
- High risk of periop morbidity if pulm Htn present

Etiology

- Multifactorial; arrest of pulm development ± inflammation
- Major risk factors: premature birth, respiratory failure, oxygen supplementation, and mechanical ventilation
- Impaired angiogenesis, which reduces alveolar-capillary gas exchange, leading to hypoxemia and increased PVR

Usual Treatment

- Supplemental oxygen
- Inhaled bronchodilators (e.g., β-agonists)
- Pulm vasodilator therapies (e.g., sildenafil, calcium channel blockers, bosentan) if pulm Htn present

Assessment Points

System	Effect	Assessment by Hx	PE	Test
HEENT	Possible tracheomalacia	SOB Stridor	Retractions Audible stridor	Bronchoscopy for severe cases
CV	PulmHtn Cor pulmonale	Exertional dyspnea Syncope Cyanosis, esp. during feeds Supplemental O ₂ dependence	Hypoxia Cyanosis ± clubbing Rales Peripheral edema Elevated JVP	ECG ECHO Cardiac cath
RESP	Possible bronchomalacia Decreased tidal volumes Increased airway resistance Decreased dynamic lung compliance Hypoxia Hypercapnia	SOB Cyanotic spells Supplemental O ₂ dependence Asthmalike symptoms Recurrent respiratory infections	Tachypnea Retractions Cyanosis ± clubbing Expiratory wheezing Rales	CXR ABG
GI	Failure to thrive	Poor feeding	Low BMI	Generally not needed

Key References: Jensen EA, Schmidt B: Epidemiology of bronchopulmonary dysplasia, *Birth Defects Res A Clin Mol Teratol* 100(3):145–157, 2014; Lauer R, Vadi M, Mason L: Anaesthetic management of the child with co-existing pulmonary disease, *Br J Anaesth* 109(21):i47–i59, 2012.

Perioperative Implications**Preoperative Preparation**

- Determine room-air oxygen saturation and baseline supplemental oxygen requirements.
- Obtain electrolytes (if pt receiving chronic diuretic therapy); ABG (if oxygen requirements recently increased); and ECHO (if clinical markers concerning for pulm Htn).

- Avoid general anesthesia for elective procedures during acute respiratory infection.
- Avoid spinal anesthesia in patients with severe pulm Htn; decreased venous return and bradycardia may precipitate right heart failure.
- Consider preoperative nebulized β₂ agonist and/or steroid administration.
- Administer premedication cautiously in pts with pulm Htn.

Monitoring

- Standard ASA monitors.
 - Monitor pulse oxygen saturation, end-tidal carbon dioxide, and body temperature closely. Abnormalities may worsen pulm Htn.
- Consider arterial cannulation for invasive blood pressure monitoring and central venous line placement for inotrope administration in pts with pulm Htn.

Airway

- No particular association with difficult tracheal intubation
- Risk for subglottic stenosis, airway granulomas, and pseudopolyps if prolonged tracheal intubation occurred during infancy; may require smaller tracheal tubes

Induction

- Induction agents of choice: Sevoflurane (mask induction) and propofol (IV induction). Consider IV ketamine if pt is hemodynamically unstable.
- Ensure deep level of anesthesia before airway manipulation.

- Avoid histamine-releasing neuromuscular blocking agents.

Maintenance

- Treat bronchospasm with inhaled beta-agonists. Use IV terbutaline or epinephrine for refractory wheezing.
- Avoid hypoxia, hypercarbia, and hypothermia (increase PVR).
- Avoid high peak inspiratory pressures; provide longer inspiratory times.
- Consider inhaled nitric oxide for pulm hypertensive crisis.

Postoperative Period

- Continuous pulse oximetry
- May require prolonged postop ventilation
- Pain management strategy may affect postop resp status

Anticipated Problems/Concerns

- Respiratory insufficiency and postop ventilation
- Worsening pulm Htn
- Pain control

Buerger Disease (Thromboangiitis Obliterans)

Jeongae Yoon

Risk

- Current or recent chronic tobacco/nicotine exposure
- Ashkenazi Jewish ethnicity; prevalence much greater in Eastern Europe, Southeast Asia, and India
- Age <45, male gender (M:F ratio: 10–100:1)
- Incidence in USA: Progressively decreasing in association with decreasing smoking prevalence; <8–10/100,000

Perioperative Risks

- Similar to any pt with chronic tobacco exposure
- Risks to already compromised perfusion of distal extremities

Worry About

- Coexisting pulm disease in tobacco smokers
- Abnormal Allen test result in a young (<45 y) male smoker with leg ulcerations (classic clinical scenario for Buerger)
- All extremities because TAO is never confined to a single limb

Overview

- Inflammatory vasculitis of small and medium arteries and veins in extremities.
- Classic distribution is infrapopliteal or distal to the brachial artery.

- Results in extremity ischemia leading to claudication of calf, foot, forearm, or hands.
- Severe ischemia results in ulcerations and gangrene progressing to necrosis and eventual amputation of ischemic extremity.
- Olin (2000) criteria:
 - Age <45 y.
 - Current or recent history of tobacco use.
 - Presence of distal-extremity ischemia indicated by claudication, rest pain, ischemic or gangrenous ulcers, and documentation by noninvasive vascular testing.
 - Exclusion of autoimmune diseases (scleroderma, CREST, sclerodactyly, and telangiectasia), hypercoagulable states (antiphospholipid syndrome or homocysteinemia), or DM.
 - Exclusion of proximal embolic source by ECHO or angiography.
- Diagnosis confirmed with biopsy of active lesion showing a highly cellular thrombus formation with neutrophils, giant cells, and microabscesses but intact internal elastic lamina: differentiates from other vasculitis conditions.
- Antiendothelial antibody titers may allow tracking of disease progression and severity.
- Lesions occasionally occur in coronary, mesenteric, and cerebral vasculature but always present initially in extremities.

Etiology

- Autoimmune reaction against vascular endothelial cells potentiated by nicotine exposure.
- Antiendothelial antibodies trigger immune reaction and microabscesses and thrombosis formation.
- Impaired endothelium-mediated vasodilation in peripheral vasculature, which results in ischemia.
- Angiographic evidence of disease exists before clinical presentation in unaffected limbs.

Usual Treatment

- Complete tobacco and/or nicotine cessation, including nicotine patches/gum and avoidance of passive smoking; all other treatments are palliative.
- Prostaglandins analogue (e.g., IV iloprost), cilostazol, and bosantan, which has shown efficacy in symptom management and disease progression.
- Aspirin and clopidogrel used for secondary prevention.
- Surgical revascularization usually not possible given distal and diffuse nature of vascular lesions.
- Sympathectomy provides palliative short-term pain relief, but no long-term benefit; spinal cord stimulators can provide pain relief.
- Amputation is ultimate treatment option for affected distal digit and/or extremity for nonhealing ulcerations or gangrene.

Assessment Points

System	Effect	Assessment by Hx	PE	Test
CV	Coronary lesions consistent with ischemia	Angina, MI, CHF SOB	Third heart sound, regular rhythm, or no rales	ECG, ECHO, coronary angiography
RESP	Consistent with chronic tobacco exposure, COPD, chronic bronchitis	SOB, cough, increased sputum	Findings consistent with chronic smoker	CXR, PFT results consistent with obstructive pattern
HEME	Carboxyhemoglobin	Smoking Hx		Blood gases with co-oximetry
CNS	Vascular lesions, leading to cerebral ischemia	Syncopal episodes, TIA, CVA	Carotid bruit	Carotid US, CT angiogram
GI	Mesenteric ischemia	"Intestinal angina"	Abdominal bruit	Mesenteric angiography
EXTREMITIES	Distal ischemia, gangrene	Claudication, rest pain, nonhealing ulcers, prior amputations	Cool extremities, poor capillary refill, hair loss, thrombosis, migraines, ulcerations/gangrene	Allen test, Doppler US, angiography with evidence of "corkscrew collateral" revascularization

Key Reference: Olin JW, Shih A: Thromboangiitis obliterans (Buerger's disease). *Curr Opin Rheumatol* 18(1):18–24, 2006.

Perioperative Implications

Preinduction/Induction/Maintenance

- Carefully document locations/extent of distal-extremity ulcerations and thrombosis migraines.
- Optimize preinduction pulmonary status.
- Pay special attention to padding and protection of distal extremities.
- Prevent hypothermia in the entire periop phase by keeping extremities warmed and covered.

Monitoring

- Consider risks versus benefits of distal arterial catheter

- Femoral arterial catheterization would be a viable option for invasive monitoring.
- Pulse oximetry may be more accurate in a proximal location, such as the ear lobe.

General Anesthesia

- Avoid hypothermia (OR ambient temperature and forced-air warmer).
- Maintain intravascular volume and avoid alpha agonists if possible.
- Regional anesthesia can be performed safely.
- Avoid epinephrine in local anesthetic solutions to limit risk of vasospasm.

Postoperative Period

- Keep distal extremities warm; 40% of pts have concurrent Raynaud phenomenon.

Anticipated Problems/Concerns

- Excellent opportunity to reiterate importance of smoking cessation.
- If no critical limb ischemia, smoking cessation will prevent amputation.
- Long-term prognosis for major amputation: 11% at 5 y; 21% at 10 y; and 23% at 20 y.