Anticipated Problems/Concerns

- Frequently these pts are taking meds that affect platelets or coagulation in addition to having a traumatic injury that increases the risk of a coagulopathy or DIC. Be aggressive to avoid progressive hemorrhagic injury.
- Adverse changes in neuro function may occur. Be alert for posttraumatic hydrocephalus or new bleeding.
- Potential for seizures, SIADH, and DI.
- Neurogenic pulm edema can occur within minutes of the CNS injury or be delayed 12–24 h.
- Concern with neurogenic stunned myocardium.

Bronchiectasis

Matthew Hart | Jeffrey R. Kirsch

Risk

- Incidence in USA <1:10,000 hospital admissions.
  - Cystic fibrosis is the single largest cause of bronchiectasis in industrial nations.
  - A subgroup of Native Americans of Alaskan decent has a four-fold increase in the incidence of bronchiectasis over the general population. Ciliary deformities have been shown in a Polynesian population.
- No gender prevalence.
- Socioeconomic prevalence: Inbreeding and primitive health care, particularly lack of immunization and poor treatment of childhood bronchitides, increase the prevalence.
- Occasionally seen in children:
  - Bronchial cartilage deficiency (Williams-Campbell syndrome)
  - Tracheobronchomegaly (Mounier-Kuhn syndrome)
  - Inherited immunoglobulin deficiencies, impaired phagocytosis, and complement deficiency
  - α_1-Antitrypsin deficiency
- Occasionally seen in adults with acquired γ-globulin deficiency:
  - Cystic fibrosis
  - RA
  - Pulm ciliary dyskinesias (Kartagener syndrome)

Perioperative Risks

- Spillage of infected secretions from bronchiectatic regions to normal lung leads to pneumonitis and retention of secretions
- Risk from bacteremia, after manipulation
- Risk of secondary acute resp failure
- Massive hemoptysis
- Pneumothorax

Worry About

- Exacerbation of asthma
- Amount of sputum produced and its nature
- Fever and hemoptysis: Acute pulm infection
- Right heart function
- Check frequency of cough and daily sputum volume; culture and smear for composition; check body temp and WBC count for acute infection
- Exercise tolerance will indicate associated impairment or disability
- Postop pulmonary decompensation

Overview

- Abnormal widening or dilatation of one or more branches of the bronchial tree, generally caused by permanent damage or destruction to the corresponding segments muscular wall, resulting in a decreased elasticity; widened segments commonly fill with purulent secretions; mucosa is swollen and inflamed and may be ulcerated with granulation tissue exposed; and extensive collateral flow occurs in these chronically inflamed bronchi (3–12% of CO).

Etiology/Pathogenesis

- Exact etiology for acquired form remains unclear but often involves necrotizing infection in tracheobronchial wall. Five mechanisms may predispose pts:
  - Bacterial, viral, or fungal bronchopulmonary infections, including TB, pertussis, and measles
  - Bronchial obstruction
  - Immunodeficiency states, including IgG deficiency, IgA deficiency, and leukocyte dysfunction
  - Hereditary defects in ciliary-mucosal clearance, including Kartagener syndrome, α_1-antitrypsin deficiency, and cystic fibrosis
  - Miscellaneous disorders, including recurrent aspiration, inhaled irritants, Young syndrome, and bronchiolitis obliterans following heart-lung transplantation

Usual Treatment

- Medical therapy: Postural drainage, chest physiotherapy, antibiotics for cultured infection, bronchodilators, and steroids for symptomatic treatment
- Surgical therapy: Resection indicated for uncontrolled hemoptysis; or lobar closely confined disease, age >20 y; bronchopulmonary lavage under GA with divided airway (double-lumen tube)

Assessment Points

System	Effect	Assessment by Hx	PE	Test
HEENT	Sinusitis	Postnasal drip Stuffiness, headache	Translucency	X-ray, US, bright lights
CV	Clubbing, cyanosis CHF (cor pulmonale) Kartagener syndrome	Exercise tolerance Pulm Htn, edema Chronic sinusitis	Situs inversus	ABG Loud P_2 Right heart studies Immotile spermatozoa
RESP	Bronchiectasis	Cough with sputum Hemoptysis Wheezing	Rhonchi CXR: 93% tram lines; 7% normal	Smear, culture, high-resolution CT, bronchogram Bronchoscopy PFTs
HEME	Immunodeficiency Infection	IgG, IgA, WBC Guided antibiotic therapy		
CNS	Brain abscess	CT/MRI		

Key References: O'Brien C, Guest PJ, Hill SL, et al.: Physiological and radiological characterization of patients diagnosed with chronic obstructive pulmonary disease in primary care, *Thorax* 55:635–642, 2000; Nikolaizik WH, Warner JO: Aetiology of chronic suppurative lung disease, *Arch Dis Child* 70(2):141–142, 1994.

Perioperative Implications

Monitoring

- Routine for majority of cases
  - Consider PA catheter for cor pulmonale or CHF.
  - Arterial line for longer/invasive procedures.

Airway

- Careful frequent suctioning and humidification of inspired gases

Induction

- Consider preop pulmonary optimization: Chest physiotherapy and bronchodilator treatment.
- May consider increase steroids if on chronic therapy.
- Consider regional anesthesia when possible.

Maintenance

- Routine

Extubation

- Depends upon degree of pulm and cardiac dysfunction.
- Consider extubation and immediate recovery in sitting position.

Adjuvants
- Routine

Postoperative Period
- Use stir-up regimen and monitor for retained secretions and resp failure.
- Have postop plan for chest physiotherapy.
- Pt may need to continue course of antibiotics.
- Supply supplemental oxygen and monitor SpO_2.
- Check for platypnea-orthodeoxia if right atrial pressures become elevated.

Anticipated Problems/Concerns
- Retained secretions and secondary resp failure
- Right heart decompensation if hypoxemia persists
- Bacteremia from airway manipulations

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Bronchiolitis Obliterans Syndrome

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Risk
- Incidence in USA: 1:40,000
- Racial predilection: None
- Occurs primarily after lung and hematopoietic stem cell transplantation
- Industrial workers exposed to inhalants who have presented with BOS: nylon-flock, battery workers, manufacturer of flavorings (diacetyl butter-like flavoring), and textile workers

Perioperative Risks
- Hypoxemia and severe periop airway obstruction.
- Pulm infection, sepsis, and pulm edema post transplant.
- Injury to tracheal anastomosis due to ETT placement.
- Prolonged intubation (increased sensitivity to medications including muscle relaxants, pulm functions, renal impairment, and pulm edema).
- Complications of immunosuppression (infection, hemorrhage, and renal impairment).
- Preop focus must differentiate between active invasive pulm infection and ongoing chronic rejection with colonization, as well as maximizing medical condition and stratifying risk.

Worry About
- Pulm functions
- Differentiating BOS from untreated invasive pulm infection and other disorders
- Side effects of immunosuppression including infection with invasive techniques, hemorrhage, and renal failure with cyclosporine
- Airway and vascular allograft denervation (physiologic and pharmacologic side effects)
- Other effects of etiologic agents

Overview
- Delayed-onset allograft dysfunction and continual decline in FEV_1 not due to other etiologies of transplant dysfunction; it frequently occurs with signs of airflow obstruction.
- Because BO is difficult to confirm histologically (transbronchial biopsy of larger airways with sporadic involvement often provides insufficient samples and has a high false-negative diagnostic rate), the International Society for Heart and Lung Transplantation proposed a staged clinical definition of BO termed BOS (stages 0 to 3 defined by changes in pulm functions, and based on spirometry, rather than histology).
- BOS clinical staging is important to the clinician because it indicates allograft function.

Etiology
- The mechanism involved in the etiology of BO remains poorly understood.
- Two forms of BOS with inflammation and fibrosis: Rejection-related and non-rejection related.
- After transplant, the syndrome reflects small airway obliterations caused by "chronic rejection."
- Several risk factors, including primary graft dysfunction, lymphocytic bronchiolitis, ischemia-reperfusion injury, acute cellular rejection, mismatches at HLA loci, autoimmunity (collagen V sensitization), persistent neutrophil influx and sequestration (bronchoalveolar lavage neutrophilia), GE reflux with resultant aspiration, loss of cough reflex due to denervation, complication of prematurity (bronchopulmonary dysplasia), toxicant inhalation ("popcorn lung"), and exposure to infectious agents (bacterial, viral, and some atypical organisms including mycoplasma, chlamydia, and fungi) (BO with organizing pneumonia [BOOP]).
- BOS is described after lung, heart-lung, bone marrow, renal, pancreas, and liver and hematopoietic stem cell transplantation; BOS remains the leading cause of death for those who survive beyond 1 y after lung transplantation.

Usual Treatment
- Varies depending on whether or not BOS is rejection related
- Rejection-related BOS: Mainly treated with augmented immunosuppression (systemic corticosteroids, cyclosporine, tacrolimus, and azithromycin) and supportive care, including O_2, bronchodilators, and chest physical therapy
- Non-rejection related BOS is treated with supportive care, anti-infective agents, and medical antireflux therapy, and may respond to steroids (especially toxic fumes and other environmental exposures)
- Newer treatments for rejection-related conditions: Extracorporeal photopheresis, aerosolized cyclosporine, antithymocyte globulin, IV immunoglobulin, statins, bortezomib, interleukin subtype specific antagonists, and montelukast
- Referral to surgeon for potential fundoplication of the GE junction if GE reflux is confirmed
- Severe cases often require lung transplant or even retransplant, with an accompanying increased risk of recurrent BOS and graft dysfunction

Assessment Points

Use previous classification to determine the possible cause of BO, including posttransplantation or environmental exposure(s).

System	Effect	Assessment by Hx	PE	Test
GENERAL	Active infection	Fever and non-rejection related change in status	Increased temp, tachycardia with infection	Increased WBC
RESP	Loss of lung functions (% FEV_1)	Recent change in functional capacity, invasive lung infections, meds, lung colonization (resistant bacteria), risk factors, BOS staging; environmental exposure (e.g., diacetyl production)	Tachypnea, wheezes, cough, fever, cyanosis, pulm edema	CXR, high-resolution computed tomography, PFTs (decreased FEV_1, decreased O_2 saturation hypoxia) bronchoscopy for endobronchial biopsy, culture, bronchoalveolar lavage, lung biopsy (diagnosis)
RENAL	Loss of function due to immunosuppression	Change in status, dialysis	A-V fistula (avoidance for procedures), fluid overload, pulm edema, increased weight	Decreased renal functions, tachycardia, peripheral edema, SOB
HEME	Thrombocytopenia due to medications	Prolonged bleeding	Bruising	CBC with decreased platelets, increased bleeding time

Key References: Meyer KC, Raghu G, Verleden GM, et al.: An international ISHLT/ATS/ERS clinical practice guideline: diagnosis and management of bronchiolitis obliterans syndrome, *Eur Respir J* 44(6):1479–1503, 2014; Feltracco P, Falasco G, Barbieri S, et al.: Anesthetic considerations for nontransplant procedures in lung transplant patients, *J Clin Anesth* 23(6):508–516, 2011.