Preinduction/Induction
- No one best technique reported.
- Goal of any technique is to maintain both cardiac output and SVR.
- Combining short-acting IV narcotic (fentanyl), low-dose induction agent (sodium thiopental or ketamine), and inhalational agent (sevoflurane or isoflurane) with muscle relaxant devoid of cardiovascular effects (vecuronium or rocuronium).
- For labor:
  - Provision of effective analgesia prevents increased release of catecholamines, which increases PVR.
  - Coaxial technique: Initial intrathecal dose of narcotic.
- For cesarean section:
  - Regional: Slow induction of epidural anesthesia; counteract sympathectomy with vasopressor and maintenance of preload.
  - General anesthesia: Avoid rapid-sequence with risk of precipitating increase in PVR or inducing myocardial depression; maintain cricoid pressure through induction; avoid increase in PVR, decrease in SVR, hypoxia, hypercarbia, and myocardial depressants.

Maintenance
- GA: Narcotic, low-dose inhalational agent, muscle relaxant.
- Avoid hypotension (decrease SVR), acidosis, hypercarbia and hypoxia (increase PVR).
- For labor:
  - Epidural infusion with low-dose local anesthetic/narcotic solution.
  - Avoid Valsalva maneuver and pushing; delivery with vacuum or forceps.
- For cesarean:
  - High-dose narcotic technique.
  - Amnesia with benzodiazepine.
  - Avoid halogenated agents: Myocardial depression, decrease SVR.
  - Avoid nitrous oxide: Increase PVR, higher FIO_2.

Extubation
- High-dose narcotic technique may necessitate postop ventilation.

Adjuvants
- Avoid N_2O.
- Maintain SVR with dilute solution of phenylephrine.
- Inotrope, vasodilator for treatment of failure.
- Cautious use of oxytocin (systemic vasodilation).
- Avoid prostaglandin F (increase in PVR).
- Resume anticoagulation in postpartum period.

Postoperative Period
- Pain management is critical.
- In pregnant pts, death most often occurs at delivery or postpartum.
- Possible hemodynamic changes:
  - Excessive blood loss: Replace volume.
  - Autotransfusion: Treat with vasodilator, inotrope, judicious use of diuretic.
  - Arrhythmias: Sinus bradycardia, AV block, EMD.
  - Pulm emboli.
  - Postpartum increase in PVR; reason unknown.

Anticipated Problems/Concerns
- Unresponsive, increased PVR or decreased SVR with loss of oxygenation
- CHF

Emphysema

Amy C. Robertson | William R. Furman

Risk
- Incidence in USA: 4.7 million.
- Prevalence, incidence, and mortality increase with age.
- Higher in males than females.
- Higher in whites than nonwhites.

Perioperative Risks
- Intraop bronchospasm
- N_2O expansion of bullae
- Postop respiratory failure
- Postop pulm infection

Worry About
- Worsening of baseline pulm function, caused by:
  - Bronchospasm.
  - Acute bronchitis or pneumonia.
  - Pulm embolism.
- Worsening of baseline cardiac function caused by right heart failure.
- Most common comorbidities include ischemic heart disease, diabetes, skeletal muscle wasting, osteoporosis, and lung cancer.

Overview
- Anatomic: Destruction of interalveolar septa and loss of pulm elastic recoil, leading to formation of bullae and development of irreversible expiratory airflow obstruction.
- Remodeling of the small airway compartment and loss of elastic recoil result in progressive decline of FEV_1 and lead to static and dynamic hyperinflation.
- The "pink puffer" has dyspnea, hyperinflation, distant breath sounds, low diffusing capacity (decreasing D_LCO to <60% predicted).
- The "blue bloater" has chronic bronchitis, leading to hypoxemia, polycythemia, and CO_2 retention.
- Hypoxia, hypercarbia, and cor pulmonale are late developments.
- Mucociliary clearance is often worsened after inhalational anesthetics.
- Diaphragmatic mechanics are impaired by anesthetics, sedatives, NMBs, interscalene blocks, and supine positioning.

Etiology
- According to the elastase-antielastase hypothesis, the lung is normally protected from injury to its elastic tissues by antielastases, including API, which is also called a_1-antitrypsin. According to this theory, emphysema may be acquired or genetic.
- Acquired: Related to inhaled oxidants (cigarette smoke or other occupational exposures), which are believed to inactivate API, thus compromising lung matrix repair after injury.
- Genetic: Absent or abnormal API, also known as a1-antitrypsin deficiency, which accounts for a small fraction of cases.

Usual Treatment
- Smoking cessation (>6–8 wk may lessen anesthetic risk).
- Relief of symptoms by treatment of bronchospasm and infection.
- Most frequent cause of acute exacerbation is viral or bacterial infection. Treatment may consist of increased doses of bronchodilators plus systemic corticosteroids and antibiotics.
- In advanced cases, if hypoxia and cor pulmonale have developed, O_2 therapy may improve quality of life and survival.
- Lung volume reduction surgery may be considered for those with predominantly upper lobe disease and/or low exercise tolerance.

Assessment Points

System	Effect	Assessment by Hx	PE	Test
HEENT	Tumors secondary to smoking	Voice change	Hoarseness, stridor, inspiratory obstruction	Flow-volume loops
CV	Cor pulmonale (late)	Edema, severe dyspnea	Signs of pulm Htn Hepatosplenomegaly Pedal edema, cyanosis, pleural effusions, usually without pulmonary edema	CXR ABG
	Pulm emboli	Episodic SOB Arrhythmias Hard to differentiate from course of underlying illness	May reveal DVT in legs	CXR High-resolution CT V/Q scan Pulm angiogram
RESP	Bronchospasm	Recent increase in dyspnea or decrease in exercise tolerance	Increased respiratory rate Increased expiratory time Increased accessory muscle use	Spirometry pre- and post-bronchodilators
	Pneumonia	Fever, dyspnea, increased sputum	Signs of pulm consolidation	CXR, WBC

Key References: Decramer M, Janssens W, Miravitlles M: Chronic obstructive pulmonary disease, *Lancet* 379(9823):1341–1351, 2012; Hausman MS, Jewell ES Engoren M: Regional versus general anesthesia in surgical patients with chronic obstructive pulmonary disease: does avoiding general anesthesia reduce the risk of postoperative complications? *Anesth Analg* 120(6):1405–1412, 2015.

Perioperative Implications

Preoperative Preparation

- Optimize bronchodilation.
- Eradicate any underlying bacterial infection.
- Encourage smoking cessation if this can occur >6 wk before surgery.
- Consider regional anesthesia where appropriate; associated with lower incidences of pneumonia, prolonged ventilator dependence, and unplanned postop intubation.

Monitoring

- Be cognizant of potential for increased gradient between $PETCO_2$ and $PaCO_2$.

Airway

- None, unless tumor present in airway

Preinduction/Induction

- If pt has airway reactivity, consider issues related to asthma/chronic bronchitis.
- Avoid N_2O when expansion of bullae is a risk.
- May avoid high concentrations of desflurane if airway reactivity is of concern.

Maintenance

- Recumbent positions impair chest wall muscle function, and abdominal muscle function usually needed for spontaneous ventilation.
- Ventilator settings: Avoid dynamic hyperinflation and development of intrinsic PEEP. Long expiratory times may be required; try to avoid high positive pressures (consider pressure controlled ventilation), especially if bullae are present.

Extubation

- Residual anesthetics may blunt the ventilatory response to CO_2, increasing the risk of postop respiratory failure.
- Pre-extubation bronchodilators.
- Unrelieved incisional pain, especially after abdominal or thoracic surgery, will impair breathing; consider postop epidural analgesia.
- Consider regional block and/or NSAIDs to lessen risk of respiratory depression.
- Pts may be semiconscious and combative owing to hypoxia and hypercarbia on emergence.
- Evaluate whether postop ventilation may be the safest approach until the residual anesthetic effects have dissipated. Extubation to NIPPV may be useful in such cases.

Adjuvants

- β-adrenergic agonists and anticholinergic agents for airway reactivity (may consider theophylline)
- Oral or inhaled steroids in selected pts

Anticipated Problems/Concerns

- Postop respiratory failure; consider NIPPV rather than reintubation in selected pts.
- Tension pneumothorax from ventilator-induced barotrauma.
- Airway plugging from secretions.

Encephalitis

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Risk

- Age; animal contact and occupational exposure to animals; ingestion of raw, partially cooked meat, fish, reptiles, or unpasteurized milk; insect contact; laboratory workers; healthcare workers; person-person transmission; recent vaccination and unvaccinated status; season (late summer/early fall, winter); travel and geographic exposure; immunocompromised state; transfusion and transplantation

Perioperative Risks

- Mental status alteration: Delirium, altered level of consciousness, clinical and subclinical seizures, increased ICP, and SIADH
- Unpredictable sedative and amnestic effects of anesthetics and adjunct drugs

Worry About

- Delayed awakening, postop delirium, clinical and subclinical seizures
- Hyperkalemic response to succinylcholine
- Transient myocardial dysfunction
- Paroxysmal sympathetic hyperactivity: Hyperthermia, tachycardia, hypotension, bradycardia
- Electrolyte abnormality secondary to SIADH and CPM with rapid correction of Na^+ abnormality

Overview

- Inflammation of brain parenchyma associated with clinical evidence of neurologic dysfunction.
- Manifestation of disease process or a component of another CNS or systemic illness.
- Organisms enter CNS via bloodstream or peripheral nerves.
- Symptoms: Altered mental status, altered consciousness, with or without focal neurologic abnormality, behavioral and personality changes in the presence of fever, irritability, changes in speech, changes in hearing, headache, photophobia, nuchal rigidity, vomiting, disorientation, lethargy, confusion, hallucinations, memory loss, clinical or subclinical seizures, myoclonus, coma.
- Dx is established by symptoms, epidemiologic Hx (exposure, season, geographic location), culture of blood/sputum/nasopharynx/stool, biopsy skin lesion/lymph node, serologic testing, CSF cells/protein/culture, CSF bacterial and viral antigens, CSF viral PCR, virus specific DNA sequencing, MRI, EEG, and CT scan (if MRI unavailable). Brain biopsy is rarely performed but should be early.

Etiology

- Unknown in most pts; manifestation of illness outside CNS
- Infectious:
  - Viral (most common): Herpes simplex, varicella zoster, CMV, EBV, influenza, RSV, enteroviruses, arboviruses, HIV, JC virus, rabies
  - Nonviral: Bacteria, prion, parasitic, fungal
- Noninfectious:
  - Postinfectious/immune mediated: ADEM immunologic response to antecedent antigenic stimulus
  - Paraneoplastic: Anti-NMDA receptor, which induces glutamatergic transmission impairment

Usual Treatment

- Empiric antibiotics: Acyclovir (important as viral etiology is most common infectious cause), ampicillin, ceftriaxone, vancomycin
- Doxycycline if rickettsial or ehrlichial disease suspected
- ADEM: Steroids, plasma exchange, chemotherapeutic agents
- Human rabies immunoglobulin infiltration of inoculation site immediately after bite
- Specific antimicrobial therapy according to culture and sensitivity
- Supportive care:
  - Intubate, ventilate, if dictated by mental status, airway reflexes
  - Hemodynamic support
  - Nutrition
  - DVT prophylaxis
  - GI prophylaxis
  - Physical therapy
  - Dx and treatment of extracranial infection
- Management of complications: Seizure, increasing ICP, SIADH, resp failure