Pulmonary Pathophysiology:

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Acute Respiratory Failure

Failure of the respiratory system to provide for the exchange of oxygen and carbon dioxide between the environment and tissues in quantities sufficient to sustain life.

- **Type I: Hypoxemic Normocapnic**
  - Low PaO2
  - Normal PaCO2
  - Widened A-a gradient

- **Type II: Hypoxemic Hypercapnic**
  - Low PaO2
  - High PaCO2
  - Normal A-a gradient
Acute Respiratory Failure: Causes

- **Type I (oxygenation failure)**
  - **Pathophysiology:** Decreased V/Q ratio (shunting), diffusion defect
    - Pneumonia
    - Pulmonary edema
    - Pleural effusion
    - ARDS
    - **Alveolar / capillary exchange impacted**

- **Type II (acute ventilatory failure)**
  - **Pathophysiology:** Hypoventilation
    - CNS depressant drugs
    - Spinal cord injury
    - Chest trauma
    - Acute exacerbation of COPD

Oxygen Therapy and PCO₂ goals in COPD

COPD

- Disorders of emphysema, chronic bronchitis, and small airway disease.
- Obstructive disease causes resistance to airflow.
- Decreased expiratory airflow is central to COPD.
  - *Residual volume, functional residual capacity, and total lung capacity can increase.*
  - Increased resistance during forced expiration from dynamic compression.
  - FEV₁ (expiratory airflow) / FVC < .80
  - Chronic inflammation of all structures of the lungs.
    - Excessive mucous secretion and ciliary dysfunction.
    - Leads to repeated damage and repair of the airways.
- Vascular changes can lead to pulmonary hypertension and subsequent acute cor pulmonale can develop.
Emphysema and Chronic Bronchitis

- **Emphysema**
  - Destruction of alveolar walls and elastic tissue support of small airways
  - Enlargement of air spaces distal to terminal bronchioles
  - **Air trapping**
  - Airway resistance increased; also loss of pulmonary capillaries
  - Decreased surface area for gas exchange
  - V/Q mismatching

- **Chronic bronchitis**
  - Mucous glands hypertrophy
  - Decreased cilia
  - Increased bronchial wall thickness
  - **Chronic inflammation and excessive secretions block airways**
  - Increased resistance – ventilation impairment

COPD: Clinical Manifestations

**Chronic Bronchitis**
- Chronic cough and sputum production daily for minimum of 3 months/year for at least 2 consecutive years
- Can have chronic hypoxemia / right sided heart failure
- Exacerbations related to infection

**Emphysema**
- Increased responsiveness to hypoxemia
- Dyspnea with adequate oxygenation
- Initial dyspnea on exertion
- Dyspnea at rest
COPD: Clinical Manifestations

- Blended symptoms
- Large lung volumes / diminished breath sounds
- Ventilation / perfusion mismatching
- High PaCO$_2$ / low PaO$_2$
- Increase erythropoietin for increased RBCs
- Right sided heart failure

COPD: Treatment

- Smoking cessation
- Bronchodilators
  - Anticholinergics are the first-line medication in maintenance therapy.
    - ipratropium (Atrovent).
  - Beta-agonists can be added
    - Short acting
      - racemic albuterol (Ventolin, Proventil, Accuneb).
      - levalbuterol (Xopenex).
      - metaproterenol (Alupent).
      - pirbuterol (Exirel, Maxair).
    - Long acting
      - salmeterol (Serevent).
      - formoterol (Foradil, Oxeze).
  - Theophylline is a long acting weak bronchodilator.
COPD: Treatment

- Antibiotics - acute exacerbations can be caused by bacterial infections.
- Corticosteroids: Remains controversial, but they are frequently used in treating exacerbations. Steroids are used as part of chronic treatment in some patients. Corticosteroids can also be combined with other medications.
  - budesonide (Pulmicort)
  - fluticasone and salmeterol (Advair)
- Expectorants/mucolytics.

COPD: Treatment

- Oxygen (Can improve survival in patients who are hypoxemic)
  - Criteria
    - Room air: PaO2 < 55 mm Hg with saturation < 85%.
    - PaO2 56-59 and saturation 86-89%, with a qualifying secondary diagnosis.
    - Goal of oxygen therapy is to obtain PaO2 of 65-80 mm Hg while awake and at rest.
  - Typically delivered at 1-4 L/min, with an increase of 1 L during sleep and exercise.
  - Should be given continuously at least 19 hours of each day.
- Pneumonia and influenza vaccines
Case Example

- Patient history: COPD (CO\textsubscript{2}) retainer
- Initial presentation: Tachypneic with SaO\textsubscript{2} of 78%
- Cause of exacerbation?
- Initial interventions?

Case Example

- ABG
  - 7.29
  - PaCO\textsubscript{2} 60
  - HCO\textsubscript{3} 30
  - PaO\textsubscript{2} 48
- Treatment options?
- Goals for ABG values?
Status Asthmaticus

Exacerbation of acute asthma characterized by severe airflow obstruction that is not relieved after 24 hours of maximal doses of traditional therapy

Characterized by expiratory wheezing

Status Asthmaticus: Etiology

- Extrinsic (specific allergy can be related to attack)
  - Pollen
  - Dust
  - Pets
  - Smoke
  - Food
  - Drugs

- Intrinsic (attack is seemingly unrelated to an allergen)
  - Infection
  - Stress
  - Exercise
  - Aspiration
Status Asthmaticus: Pathophysiology

- Trigger (extrinsic or intrinsic)
- Intrinsic trigger causes imbalance of sympathetic and parasympathetic nervous systems
- Extrinsic: IgE released ➤ histamine and slow-reacting substance of anaphylaxis (SRS-A)
- Histamine ➤ swelling and inflammation of smooth muscle of large bronchi (and mucous membrane swelling)
- Swelling of smooth muscle of small bronchi and release of prostaglandins (enhance histamine)

Status Asthmaticus: Pathophysiology

- Histamine causes excessive secretion of mucous ➤ narrows the airway lumen
- Tachypnea increases insensible water loss ➤ thicker secretions
- Mucous in small airways
- Increased work of breathing (impaired ventilation) (Note: ▲ PaCO2 is late sign)
Status Asthmaticus: Treatment

- Eliminate or treat cause
- Steroids
- Need to ventilate when $\text{PaCO}_2$ becomes elevated
- Additional similar treatment as pneumonia

Pulmonary Embolism

- Obstruction of blood flow to one or more arteries of the lung by a thrombus (other emboli – fat, air, amniotic fluid) lodged in a pulmonary vessel
- Lower lobes frequently affected due to increased perfusion
Risk Factors for PE

- **Stasis of blood**
  - Prolonged immobilization after surgical procedures
  - Plaster casts
  - Venous obstruction
  - Heart failure / Shock / Hypovolemia
  - Varicose veins
  - Obesity

- **Hypercoagulability**
  - Polycythemia vera
  - Sickle cell disease
  - Malignancy
  - Pregnancy
  - Recent trauma
  - Oral contraceptives

- **Injury to the vascular endothelium**
  - Central venous and arterial catheters
  - Phlebitis

Pulmonary Embolism: Pathophysiology

- > 90% of thrombus develop in deep veins of iliofemoral system
  - Can also originate in the right side of the heart, pelvic veins, and axillary or subclavian veins.
  - Another source is around indwelling catheters.

- **Thrombus formation leads to platelet adhesiveness and release of serotonin (vasoconstrictor).**

- **Dislodgement of thrombus**
  - Intravascular pressure changes (standing, massaging legs, fluid challenge, valsalva maneuver).
  - Natural clot dissolution (7-10 days after development).
Pulmonary Embolism: Pathophysiology

- Clot lodges in pulmonary vessels
- Ventilation continues but perfusion decreases
  - Increase in alveolar dead space
  - Alveolar CO₂ decreases (alveolar shrinking). Allows for more inspired air into the perfused alveoli.
- Overperfusion of uninvolved lung results in a decreased V/Q ratio
- Decreased blood flow damages type II pneumocytes, which results in a decrease in surfactant production. (atelectasis)
- Pulmonary edema can develop as secondary complication
- Hypoxemia can occur due to ventilation perfusion mismatching.
- Increased PVR can lead to pulmonary hypertension and potential acute cor pulmonale.
- Cardiogenic shock can occur as the result of right-ventricular failure.

Pulmonary Embolus: Clinical Presentation

- Large to massive when 50% of pulmonary artery bed is occluded
  - Impending doom
  - Hypoxemia
  - Syncope
  - Sign and symptoms of right heart strain or right-ventricular failure
  - Signs of right-ventricular strain on ECG.
  - Sudden shock
  - Pulseless electrical activity

- Medium-sized emboli
  - Dyspnea
  - Substernal chest discomfort/pleuritic chest pain
  - Many non-specific signs
  - Tachypnea
  - Tachycardia
  - Rales
  - Accentuated 2nd heart sound
Pulmonary Infarction

- Pulmonary infarction is infrequent
- More common
  - Large embolus
  - Pre-existing lung disease
- Results in alveoli filling with RBCs and inflammatory cells
- Complicated by infection
  - Abscess

Signs and Symptoms
- Pleuritic chest pain
- Dyspnea
- Hemoptysis
- Cough
- Pleural friction rub
Pulmonary Embolus: Treatment

- Prevent thrombus formation
  - Compression stockings that provide a 30-40 mm Hg or higher gradient
  - Low molecular weight heparin
- Fibrinolytic therapy
  - Indicated in patients with hypotension (even if resolved), hypoxemia, or evidence of right-ventricular strain
  - Troponin levels can also be used to guide decision-making in patients with sub-massive PE
- Pulmonary embolectomy is a surgical option when fibrinolytic therapy is contraindicated
- Treatment for Obstructive Shock!
# Pulmonary Embolus: Treatment

- **Heparin** is the treatment of choice for reducing mortality in PE
  - Initiated prior to a confirmed diagnosis
  - Slows or prevents clot progression and decreases risk of further emboli
- **Oxygen** is indicated, even in the absence of hypoxemia
- Pulmonary vasodilators to help reduce pulmonary vascular resistance
- Treat right-ventricular failure with fluids and inotropes
- **Warfarin**
  - 3 to 6 months if there is identifiable reversible risk factor
  - Minimum of 6 six months if there is no identifiable risk factor
  - Long term with recurrent PE or in patients with ongoing risk factors
- Surgical interruption of inferior vena cava with a filter
  - Patients with contraindication to anticoagulants.
  - Recurrent thromboembolism despite anticoagulant.
  - Survivor of massive PE

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# Special Considerations Fat Emboli

**Risk Factors:**
- Skeletal Trauma: femur and pelvis
- Major orthopedic surgery
- 24 to 72 hours post insult

**Signs and Symptoms:**
- Vague chest pain
- Shortness of breath
- Sudden restlessness - drowsiness
- Fever
- Petechiae (transient - axillary or subconjunctival)

- Release of free fatty acids causes endothelial injury and toxic vasculitis
- Hemorrhage into lungs (decrease H&H and platelets)
- CXR pattern similar to ARDS
- Steroids
Special Considerations Air Emboli

- **Risk Factors**
  - Dialysis
  - Pulmonary artery catheters
  - Surgical procedures
  - CABG

- **Symptoms**
  - Dyspnea, chest pain, agitation, confusion, cough

- **Treatment**
  - Prevent
  - 100% oxygen
  - Hyperbaric oxygen
  - Left lateral / trendelenburg
  - Positive pressure ventilation
  - Aspiration of air

Pulmonary Edema

- **Extra vascular accumulation of fluid in the lungs (cardiac or non cardiac)**
  - Results in impaired diffusion of oxygen due to increase in interstitial space
  - Results in decreased V/Q ratio due to poorly ventilated fluid filled alveoli
  - Fluid in alveoli also impacts compliance of lungs and therefore ventilation

- Capillary endothelium more permeable to water and solute than alveolar endothelium
- Edema accumulates in the interstitium before the alveoli
Pulmonary Edema

- Fluid in pulmonary interstitium is removed by lymphatic drainage of the lung
- Volume of lymph flow from the lung can increase ten fold in pathological conditions
- Only when this large safety factor is taxed does pulmonary edema occur

Pulmonary Edema: Risk Factors and Treatment

- Loss of integrity of alveolar capillary membrane
  - Infection
  - Inhaled toxins
  - Oxygen toxicity
- Increase in pulmonary capillary hydrostatic pressure
  - Left sided heart failure
  - Excessive fluid administration
  - Occlusion of pulmonary vein
  - Other: Blockage of lymphatic system

- Cardiac pulmonary edema is treated as acute decompensated heart failure.
- Non cardiac pulmonary edema is treated like ARDS.
Pneumonia

- Acute infection of the lung parenchyma, including alveolar spaces and interstitial space

Causes:
- Bacteria (Community acquired versus Hospital acquired)
- Virus
- Fungi
- Parasites
- Mycoplasma

Risk Factors for Bacterial Pneumonia

- Previous viral respiratory infection
- Gastro esophageal reflux disease (GERD)
- Chronic alcohol abuse
- Cigarette smoking
- Decreased level of consciousness
- Anesthesia
- Intubation
- Lung disease
- Diabetes mellitus
- Use of corticosteroids
- Elderly
Pneumonia: Pathophysiology

- Causative agent is inhaled or enters pharynx via direct contact
- Alveoli become inflamed
- Alveolar spaces fill with exudate and consolidate
- Diffusion of O2 obstructed
  - Hypoxemia.
- Goblet cells are stimulated to increase mucous
  - Increased airway resistance and work of breathing

Pneumonia: Causative Agents

Common agents in community-acquired pneumonia (younger and healthier population)
- Streptococcus pneumoniae (most common agent in community acquired pneumonia).
- Mycoplasma pneumoniae.
- Chlamydia pneumoniae
- Viral.

_Haemophilus influenza_ common among smokers
_Klebsiella pneumoniae_ in patients with chronic alcoholism

Agents in the older population commonly include gram negative bacilli
- Moraxella catarrhalis (particularly common in patients with chronic bronchitis).
- Staphylococcus aureus (in the setting of post viral influenza).

Methicillin-resistant _Staphylococcus aureus_ (MRSA) also as a cause of community-acquired pneumonia
### Hospital Acquired Pneumonia

#### Causative agents
- **Aerobic gram negative rods**
  - Klebsiella sp.
  - Psuedomonas sp.
  - Enterobacter sp.
  - Escherichia coli.
  - Proteus sp.
- Serratia sp.
- Enterococci.

- **Staphylococcus aureus** (including methicillin-resistant Staphylococcus aureus [MRSA])
- Group B streptococci

#### Nosocomial pneumonia is typically caused by bacterial agents that are more resistant to antibiotic therapy.

#### Sources
- Contamination of pharynx and perhaps stomach with bacteria
- Repeated small aspirations of oral pharyngeal secretions.
- Retrograde contamination from GI tract.

### Pneumonia: Clinical Presentation

- Flu-like symptoms.
- Pleuritic chest pain.
- Confusion in elderly.
- Tachycardia, tachypnea, fever.
- Crackles and wheezes.
- Productive cough.
- Clinical signs of dehydration.

The clinical presentation in the elderly may be more subtle including confusion, dehydration, and fever.
Diagnosis of Pneumonia

- Sputum gram stain
- Sputum culture
- Blood cultures (bacteremia not present in most)
- Leukocytosis / Shift to left of WBCs.
  - Leukocytosis and a left shift is expected in bacterial pneumonia.
  - Failure of the white blood cell count to rise in the presence of a bacterial infection is associated with an increased mortality
- Blood gases/oxygen saturation
- Chest x-ray – produces variable results but infiltrates are frequently seen
  - A chest CT may also be used to aid in the
Complications of Pneumonia

- Abscesses may form and rupture into pleural space leading to pneumothorax and/or empyema
  - Video assisted thoracoscopy with debridement is a treatment option for empyema in the early organizing phase
  - Full thoracotomy with decortication may be necessary in later organizing phases
- Pleural Effusion
- Acute respiratory failure
- ARDS
- Sepsis

Mortality rates for nosocomial or hospital-acquired pneumonia are higher than those for community-acquired pneumonia (particularly in the elderly).

Pneumonia: Treatment

- **Prevent nosocomial infections**
- Timely Antibiotics
- Hydration (Electrolyte Monitoring)
- Deep breathing / incentive spirometry
- Bronchodilators, expectorants, mucolytics
- Avoid: sedatives and antitussives
- Early activity and mobility (DVT Prophylaxis)
Aspiration

- Vomiting or regurgitation
- Large particles – airway obstruction
- pH of liquid determines injury
  - pH<2.5 or large volume
  - Chemical burns destroy type II cells
  - May induce bronchospasm
  - Increase alveolar capillary membrane permeability
    - Decrease compliance
    - Decrease V/Q ratio

Aspiration

- Non acidic aspiration
  - More transient
- Food stuff / small particles
  - Inflammatory reaction
  - Hemorrhagic pneumonia within 6 hours
- Contaminated material with bacteria can be fatal
Aspiration: Possible Prevention Strategies

- Avoiding sedation.
- Resting prior to meal time.
- Eating slowly.
- Flexing the head slightly to the “chin down” position.
- Determining food viscosity best tolerated (thickening liquids will improve swallowing in some patients).

Acute Respiratory Distress Syndrome

A syndrome of acute respiratory failure characterized by non-cardiac pulmonary edema and manifested by refractory hypoxemia. ARDS does not include mild or early acute lung injury, but rather involves severe and diffused lung injury.
Risk Factors in ARDS

- Sepsis (most common)
- Transfusion
- Aspiration
- Trauma
- Massive transfusion
- Pancreatitis

Acute Respiratory Distress Syndrome: Etiology

Direct lung injury
- Chest trauma
- Near drowning
- Smoke inhalation
- Pneumonia
- Pulmonary embolism

Indirect lung injury
- Sepsis
- Shock
- Multi system trauma
- Burns
- CABG
- Head injury

Time from injury of alveolar capillary membrane to onset of symptoms is 12-48 hours.
ARDS: Pathophysiology

- Stimulation of inflammatory and immune systems
- Release of toxic substances, causing micro vascular injury
- Pulmonary capillary membranes are damaged
  - Increase in capillary permeability.
- Cells and fluids leak into interstitium and alveolar spaces
  - Pulmonary Edema
- Impaired production and dysfunction of surfactant
  - Alveolar collapse and massive atelectasis.
- Intrapulmonary shunting
- Hypoxic vasoconstriction
- Decreased the compliance of lung
  - High-peak inspiratory pressures to ventilate the lungs.
- Potential development of pulmonary fibrosis in chronic phase.
  - Endothelium, epithelium, interstitial space expand.
  - Protein exudate inside the alveoli produces a hyaline membrane.

Acute Respiratory Distress Syndrome: Diagnosis

- Predisposing condition
- PaO2/FIO2 ratio < 200
- Chest x-ray: Diffuse bilateral infiltrates
  (Chest CT may also be used)
- Decreased static compliance of lungs
- PAOP < 18 mm Hg or no evidence of increased left-atrial pressure
- No evidence of COPD
- No other explanation for above
ARDS: Treatment

- Optimal ventilation / oxygenation
- Avoid over hydration
- No routine use of steroids
- Pulmonary vasodilators

High Mortality Persists so Prevention Remains Key

Drugs Used to Decrease Right Sided Afterload / Treat Pulmonary Hypertension

- Oxygen
- Pulmonary vasodilators
  - IV
    - NTG
    - Sodium Nitroprusside
    - Prostaglandins (PGE₁, PGI₂)
    - PDE₁ (phosphodiesterase enzyme)
  - Inhaled
    - Any of the above
    - Nitric Oxide
    - Prostacyclin (PGI₂, Epoprostenol, Flolan) or derivative Iloprost
Mechanical Ventilator Management Strategies for ARDS

- Lower tidal volume ventilation
  - Permissive hypercapnia
- Maintain plateau pressure < 30 mmHg

- Uninterrupted PEEP
- Avoidance of auto PEEP

- Airway pressure release ventilation
- High frequency ventilation (Oscillatory)
- Independent lung ventilation
- ECMO

Case Example

- 65 year old female; 85 kg
- Post witnessed cardiac arrest
- Initial \( \text{PaO}_2 / \text{FIO}_2 \) ratio 102

- Initial diagnosis?
Case Example

- Ventilator settings:
  - AC
  - Rate 12
  - TV 700 ml
  - FIO2 80%
  - PEEP 5 cm

- Ventilator adjustment?

- Other treatment considerations?

2nd ABG

- pH = 7.33
- PaCO2 = 40 mmHg
- HCO3 = 14
- PaO2 = 92

Open Lung Strategies: Focus on Mean Airway Pressure

- APRV
  - Similar to CPAP with release
  - Spontaneous breathing allowed throughout cycle
    - Can also be used with no spontaneous effort
  - Release time allows removal of CO2
  - P High (20 - 30 cmH2O) and P low (0) (pressure)
  - T high (4-6 seconds) and T low (0.8 seconds) (time)

- Facilitates oxygenation and CO2 clearance

  - Time triggered
  - Pressure limited
  - Time cycled

Advantages

- Lower peak and plateau pressures for given volume
- Decreased sedation / near elimination of neuromuscular blockade
Open Lung Strategies: Focus on Mean Airway Pressure

- High frequency oscillation
  - Not jet ventilation
  - Constant mean airway pressure
  - TV 1-3ml/kg
  - Delivers and removes gas: 1/3 time delivery in and 2/3 time delivery out
  - Usually set starting at 5 to 6 HZ (60 oscillations / HZ)
  - Chest wiggle
  - JVD: Tamponade effect

Closed (Simple) Pneumothorax

- Air enters the intra pleural space through the lung causing partial or total collapse of the lung
  - Between visceral and parietal pleura

- Possible etiology
  - Primary (no underlying lung disease)
    - Blebs / bullae
    - Smoking
  - Secondary (underlying lung disease)
    - Air enters damaged aveoli
    - COPD

  - Blunt trauma (lung laceration by rib fracture)
  - Positive pressure ventilation (rupture of weak alveoli, bleb or bullous)
  - Iatrogenic – from medical procedure
Closed (Simple) Pneumothorax

- **Pathophysiology**
  - Disruption of normal negative intrapleural pressure
  - Lung collapse
    - Decreased vital capacity
  - Decreased surface area for gas exchange
  - Acute respiratory failure (particularly secondary)

- **Signs and Symptoms**
  - Chest pain, dyspnea, cough, tachycardia
  - Asymmetrical chest excursion, diminished absent breath sounds on affected side, dramatic increases in peak inspiratory pressures on a mechanical ventilator

- **Treatment**
  - Oxygen
  - Analgesics
  - Observation (asymptomatic, small primary)
  - Aspiration (symptomatic small primary)
  - Chest Tube Criteria
    - Secondary

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Tension Pneumothorax

- Accumulation of air into the pleural space without a means of escape causes complete lung collapse and potential mediastinal shift

- **Etiology**
  - Blunt trauma
  - Positive pressure mechanical ventilation
  - Clamped or clotted water seal drainage system
  - Airtight dressing on open pneumothorax

Spontaneous primary pneumothorax may take 12 weeks to resolve.
Tension Pneumothorax

Pathophysiology
- Air rushes in—cannot escape pleural space
- Creates positive pressure in pleural space
- Ipsilateral lung collapse
- Mediastinal shift → contralateral lung compression → potential tearing of thoracic aorta
- Can also compress heart → decrease RV filling → shock

Similar to closed pneumothorax
- If mediastinal shift:
  - Tracheal shift away from affected side
  - JVD
  - Hypotension

Oxygen (100%)
- Emergency decompression
- Chest Tube
- Other as with closed pneumo
Open Pneumothorax

- Air enters the pleural space through the chest wall

**Etiology**
- Penetrating Trauma

**Pathophysiology and Signs and Symptoms**
- Equilibrium between intrathoracic and atmospheric pressures
- Patient condition depends on size of opening compared to trachea
- The affected lung collapses during inspiration
- May cause a tension pneumothorax
- Subcutaneous emphysema usually present

**Treatment**
- Similar to closed pneumothorax
- Closure of open wound with petroleum jelly gauze
- Chest tube and water seal drainage
- End expiration
- Modification for tension pneumothorax
QUESTIONS??

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Our greatest glory is not in never failing, but in rising up every time we fail.

~Ralph Waldo Emerson