**Pulmonary Disorders**

- ARDS
- Postoperative Respiratory Failure
- Obstructive Pulmonary Disease
- Respiratory Tract Infections
- Pulmonary Vascular Disease
- Respiratory Neoplasms

**ARDS (Acute Respiratory Distress Syndrome)**

- Fulminant respiratory failure
  - Acute lung inflammation
  - Diffuse alveocapillary injury
  - 30% of all ICU admissions
  - Current mortality < 40%
- Etiology:
  - Sepsis & Multiple trauma (esp w/transfusions)
  - Pneumonia, burns, aspiration, CABG, pancreatitis, drug overdose, smoke, O2, DIC

**ARDS Pathophysiology**

- Starts with alveolocapillary membrane damage and pulmonary edema
  - Direct damage
  - Indirectly (immune mediators)
- Final: Massive inflammatory response
  - Neutrophils, Macrophages, complement, endotoxin, interleukin-1, TNF-α

**Sequence**

- Alveolocapillary membrane damage
- Platelet aggregation & thrombus
  - Attracts Neutrophils
  - Neutrophils release inflammatory mediators
  - Causes further damage, and increases capillary membrane permeability
  - Pulmonary edema & hemorrhage
  - Vasoconstriction → Pulmonary hypertension
    - Uneven → V/Q mismatching

**Meanwhile, back at the ranch...**

- Surfactant production is interrupted
- Compliance is impaired
- Ventilation is impaired
- Results in
  - Right to left shunting
  - Increased work of breathing
  - 24 – 48 hours: hyaline membrane forms
  - 7 days: progressive fibrosis destroys lung

**Associated Problems**

- SIRS
  - Systemic Inflammatory Response Syndrome
- MODS
  - Multi-organ Dysfunction Syndrome
- Death results from combination of Resp Failure and MODS
ARDS Manifestations

• Classic
  – Rapid, shallow, breathing
  – Resp alkalosis
  – Marked dyspnea
  – Hypoxemia
  – Diffuse alveolar infiltrates (x-ray)

• As progresses
  – Diffuse crackles, metabolic acidosis, hypotension, decreased CO, death

ARDS Eval & Tx

• DX: exam, blood gas, x-ray
  – Criteria
    • Hypoxemia, bilat x-ray infiltrates, exclusion of cardiogenic pulmonary edema
  – TX: must catch early
    – Supportive therapy
    – Prevention of complications
    – You’ll learn a lot more about this is Critical Care

Post-Operative Respiratory Failure

• Risk:
  – Any surgery involving chest or thorax, or general anesthesia
  – Smokers or other lung disease
  – Chronic Renal Failure, ↓ cardiac reserve

• Common
  – Atelectasis, pneumonia, pulmonary edema, pulmonary embolism

• Prevention, Prevention, Prevention
  – TCDB, early ambulation, Incentive, O2

Obstructive Pulmonary Diseases

• Diseases that impair airflow
  – Upper or lower tract
  – Increase the work of breathing
  – Typically expiration is harder than inspiration
    • Results in hyperinflated lungs
    • Symptom: dyspnea
    • Sign: wheezing

• Asthma
• Emphysema
• Chronic Bronchitis

Asthma

• Acute, intermittent, or chronic
• Can occur at any age
  – Most common in children (50% of onset)
  – Mortality declining, but incidence rising
  – Familial disease, multiple gene involvement
    • Interleukins 4 & 5, IgE, eosinophils, mast cells, beta adrenergic receptors, bronchial hyperrespons
  – Risk factors: allergen exposure, urban, air pollution, cigarette smoke, hygiene,

Asthma Classification

• Older schema, based on underlying pathophysiology
• Newer classification based on symptoms and severity
  – Mild Intermittent
  – Mild Persistent
  – Moderate Persistent
  – Severe Persistent
Mild Intermittent Asthma

- **Rule of 2's**
  - Symptoms of cough, wheeze, chest tightness or difficulty breathing < twice a week
  - Nighttime symptoms < twice a month
  - Refill albuterol < twice per year
- Flare-ups-brief, but intensity may vary
- Lung function test FEV1 equal to or above 80 percent of normal values
- Peak flow less than 20 percent variability AM-to-AM or AM-to-PM, day-to-day.

Asthma Pathophysiology

- Inflammation → bronchial hyperresponsive
  - IgE & irritants → mast cell degranulation
  - Release of inflammatory mediators
    - Histamine, Leukotrienes, Prostaglandins
  - Release of chemokines
    - Infiltration by neutrophils, eosinophils, lymphocytes

Asthma Pathophysiology

- Inflammatory response
  - Bronchospasm
  - ↑vascular permeability → airway edema
  - Increased mucous production (thick)
  - Impaired mucociliary function
  - Thickening of airway walls
  - Muscarinic receptor stim → increased acetylcholine activity → increased contraction
  - Epithelial destruction by eosinophils (collateral damage)

Asthma Pathophysiology

- End result is airway obstruction
  - Bronchial hyperresponsiveness
  - Inflammatory thickening of airway
- Impaired airflow
  - Hyperinflation distal to obstruction
  - Hyperventilation
  - Decreased perfusion to hyperinflated areas
    - Uneven V/Q relationships
  - Hypoxemia without hypercapnia
Asthma Pathophysiology

- If uncorrected
  - Hyperinflation of resp units results in hyperexpansion of lungs
  - Resp muscles disadvantaged
  - Hypercapnia, resp acidosis
    - Sign of resp failure

Asthma Clinical Manifestations

- Full remission: asymptomatic and PFTs normal
- Partial remission: asymptomatic but PFTs abnormal → sign of impending flare?
- Asthma Attack
  - Slow onset acute asthma: days
    - Often after URI
  - Hyperacute asthma: minutes to hours
    - Often triggered by stress or exercise or allergens

Asthma Attack S/S

- Dyspnea & Wheezing
- Breath sounds decreased
- Peak flow early in attack
- If O2 sat < 90 → ABGs
- Early: nonproductive cough, tachycardia, tachypnea, accessory muscle use
- Resolving: thick stringy mucus

Asthma: Eval & Tx

- Spirometry
  - Decreased FEV1 and FVC
  - Increased FRC & TLC
- Daily Peak flow (RECORD & GRAPH)
- Treatment
  - Avoid triggers (foods, airborne particles, etc.)
  - Get rid of carpets, vacuum regularly
  - Pharmacological Treatment

Asthma Treatment

- Acute treatment:
  - O2, bronchodilation, steroids, hospitalization?
- Chronic treatment:
  - Inflammatory reduction
  - Bronchodilation
  - Mucus reduction
- Status asthmaticus
  - Failure of conventional therapy to relieve attack
  - Life threatening
Chronic Obstructive Pulmonary Disease

- Disease state characterized by airflow limitation that is not fully reversible.
  - Progressive
  - Abnormal inflammatory response
- Mixture of
  - Chronic Bronchitis
  - Emphysema
- Etiology
  - Smoking
  - Occupational exposure, air pollution, genetics

Chronic Bronchitis

- Hypersecretion of mucus and chronic productive cough > 3 month/year for at least 2 consecutive years
- More prevalent during winter
- 20x more incidence in smokers
- More common in elderly
- Associated with repeat infections

Chronic Bronchitis Patho

- Irritants normally cause ↑ mucus secretion
- In CB, irritants also cause
  - Hyperplasia and hypertrophy of goblet cells
  - Thicker, stickier mucus
    - Bacteria love this stuff and colonize it
    - Cilia function impaired, reducing clearance
- End result increased likelihood of infection
- Bronchial walls become inflamed leading to bronchospasm
- Narrowed airway, difficulty expiring

CB Clinical Manifestations

- Decreased exercise tolerance
- Wheezing
- Dyspnea
- Productive cough: Mucus plugs
- Progression
  - Hypercapnia, Hypoxemia
    - Polycythemia and Cyanosis
    - Later, pulmonary hypertension → cor pulmonale
    - Disability and Death

Eval & Tx

- H&P, X-ray, PFT, ABG
- Best treatment? Prevention!!!!
  - Not reversible
  - Stopping smoking can prevent progression
- Tx
  - Bronchodilators, expectorants, anticholinergic
  - Chest PT
  - Antibiotics
  - Low O2
  - Steroids

Emphysema

- Permanent enlargement of acini
- Destruction of alveolar walls w/o fibrosis
- Major limitation to airflow is loss of elasticity due to lung tissue destruction
- Mild is normal with aging (slow decline)
- Earlier and more severe almost always associated with smoking (2° emphysema)
- 1° emphysema (1-2%) genetic disorder
Emphysema Etiology

- Inability to inhibit lung proteolytic enzymes
  - Structural proteins are destroyed
- Primary Emphysema
  - α₁-antitrypsin deficiency (plasma protein responsible for inhibiting proteolytic enzymes)
- Secondary
  - Inhaled toxins inhibit antiproteases
  - Smoking, air pollution, etc.

Emphysema Patho

- Inhaled toxins
  - Epithelial inflammation and infiltration by leukocytes
  - Inflammatory cytokines inhibit endogenous antiproteases (including α₁-antitrypsin)
- Destruction of alveoli - Elastin proteolysis in alveoli septa
  - Decrease surface area → lowered perfusion
  - Capillary destruction → pulmonary HTN
  - Decreased elasticity → difficulty expiring
  - Increased air in acinus → hyperinflation

Emphysema Patho

- Air pocket formation
  - In lung: bullae
  - Adjacent to pleura: blebs
- Location Location Location
  - Centriacinar: mostly in upper lobes
    - More common with chronic bronchitis
  - Panacinar: diffuse, throughout lungs
    - More common in primary emphysema

Clinical Manifestations

- DOE → dyspnea at rest
- Little coughing or sputum unless combined with CB
- Usually thin, tachypneic, prolonged expiration, accessory muscle use
- Barrel chested
- Hyperresonant percussion
Emphysema Eval & Tx

- PFT (TLC can be 2x normal)
- CXR
- ABGs
- Acute Tx
  - CXR, WBCs, O2, Oral Steroids, ABX
- Chronic
  - Stop smoking, bronchodilators, anticholinergic
  - O2 low doses

Respiratory Tract Infections

- Rhinitis
- Sinusitis
- Pharyngitis
- Laryngitis
- Bronchitis
- Pneumonia

Pneumonia

- 6th leading cause of death in U.S.
- Risk factors: age, immunocompromised, lung disease, alcoholism, smoking, intubation, malnutrition, immobilization
- Causative organism: bacteria, fungus, protozoa, parasites
- Source
  - CAP (community acquired pneumonia)
  - Nosocomial

Common Causative agents

<table>
<thead>
<tr>
<th>CAP</th>
<th>Nosocomial</th>
<th>Immunocomp</th>
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<tbody>
<tr>
<td>Strep pneumoniae</td>
<td>Pseudomonas</td>
<td>Pneumocystis carinii</td>
</tr>
<tr>
<td>Haemophilus influenza</td>
<td>Staph aureus</td>
<td>(gorovic)</td>
</tr>
<tr>
<td>Influenza Virus</td>
<td>Klebsiella pneumonia</td>
<td>Mycobacterium</td>
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<tr>
<td>Legionella</td>
<td>E. Coli</td>
<td>tuberculosi</td>
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<td>Chlamydia pneumoniae</td>
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<td>Atypical mycobacteria</td>
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<td>Moraxella catarrhalis</td>
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<td>Fungus</td>
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<td>Uncommon:</td>
<td></td>
<td>Respiratory viruses</td>
</tr>
<tr>
<td>Pneumonic plague</td>
<td></td>
<td>Protozoa</td>
</tr>
</tbody>
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Pneumonia

- Aspiration of oropharyngeal contents or inhalation of infectious particles, or bacteremia
  - Must overcome mucociliary escalator, cough reflex, alveolar macrophage
  - In small numbers, macrophage can eliminate invader without causing inflammation
  - In larger numbers, inflammatory response is set off as organisms colonize lung
  - Localized filling of acini with exudate cellular debris: consolidation
### Pneumonia Manifestations
- Usually preceded by URI or flu
- Cough (productive or unproductive)
- Dyspnea, fever
- Other: malaise, fatigue, chills, pleuritic pain
- Inspiratory crackles, localized decreased breath sounds, increased tactile fremitus

### Eval & Treatment
- CXR (infiltrates: patchy, lobar, diffuse)
- WBC, shift to right or left
- Sputum gram stain and c/s
- Tx
  - Oxygenation & bronchodilation prn
  - Hydration and hygiene
  - Chest therapy
  - Antibiotics as appropriate
    - Gatifloxacin or levofloxacin, ciprofloxacin
    - Ceftriaxone + Azithro or clarithromycin

### Pulmonary Vascular Disease
- Pulmonary Embolism
  - DVT, sudden dyspnea, hypotension, shock
  - Risk factor recognition and prevention
  - O2, rapid anti-coagulation, thrombolytic
- Pulmonary hypertension
- Cor pulmonale
  - Right ventricle enlargement

### Respiratory Neoplasms
- Oral Cancer
- Lung cancer (13% of all U.S. cancer but 25 – 31% of cancer mortality)
  - Heavy smokers 20x risk
  - Second hand smoke 1.3x risk
- Types of Lung Cancer
  - Non-Small Cell Lung Cancer
    - Squamous Cell (30%), Adenocarcinoma (35-40%)
  - Large Cell Carcinoma (10 – 15%)
  - Small Cell Carcinoma (14%)