Chapter 12

The Respiratory System
Oxygen Delivery: A Cooperative Effort

- Respiratory system oxygenates blood and removes carbon dioxide
- Circulatory system transports gases in the bloodstream
Lung: Structure and Function

• System of tubes conduct air into and out of the lungs
  – Bronchi: largest conducting tube
  – Bronchioles: less than 1 mm
  – Terminal bronchioles: smallest
  – Respiratory bronchioles: distal to terminal bronchiole with alveoli projecting from walls; form alveolar ducts and sacs; transport air and participate in gas exchange

• Alveoli: $O_2$ and $CO_2$ exchange; surrounded by alveolar septum; with cells that produce surfactant

• Lung divided into lobes consisting of smaller units or lobules
Structure Terminal Air Passages
Gas Exchange (1 of 2)

• Two functions of respiration
• Ventilation: movement of air into and out of lungs
  – Inspiration
  – Expiration
• Gas exchange between alveolar air and pulmonary capillaries
  – Atmospheric pressure, sea level = 760 mmHg
  – Partial pressure: part of total atmospheric pressure exerted by a gas
  – Partial pressure of oxygen, \( P_{O_2} \)
  – \( = 0.20 \times 760 \text{ mmHg} = 152 \text{ mmHg} \)
Gas Exchange (2 of 2)

- Gases diffuse between blood, tissues, and pulmonary alveoli due to differences in their partial pressures
  - Alveolar air $\rightarrow$ Blood (Pulm capillaries)
    - $\uparrow$ P0$_2$ 105 mmHg
    - $\downarrow$ PC0$_2$ 35 mmHg
  - P0$_2$ 20 mmHg
  - PC0$_2$ 60 mmHg

- Requirements for efficient gas exchange
  - Large capillary surface area in contact with alveolar membrane
  - Unimpeded diffusion across alveolar membrane
  - Normal pulmonary blood flow
  - Normal pulmonary alveoli
Pulmonary Function Tests

• Evaluate efficiency of pulmonary ventilation and pulmonary gas exchange
• Tested by measuring volume of air that can be moved into and out of lungs under normal conditions
• Vital capacity: maximum volume of air expelled after maximum inspiration
• One-second forced expiratory volume (FEV₁): maximum volume of air expelled in 1 second
• Arterial PO₂ and PCO₂
• Pulse oximeter
The Pleural Cavity

- Pleura: thin membrane covering lungs (visceral pleura) and internal surface of the chest wall (parietal pleura)
- Pleural cavity: potential space between lungs and chest wall
- Intrapleural pressure: pressure within pleural cavity
  - Normally lesser than intrapulmonary pressure
  - Referred as “negative pressure” or subatmospheric because it is lesser than atmospheric pressure
  - Tendency of stretched lung to pull away from chest creates a vacuum
  - Release of vacuum in pleural cavity leads to lung collapse
Pneumothorax (1 of 2)

- Escape of air into pleural space due to lung injury or disease
- Stab wound or penetrating injury to chest wall: atmospheric air enters into pleural space
- Spontaneous pneumothorax – no apparent cause; rupture of small, air-filled subpleural bleb at lung apex

- Manifestations
  - Chest pain
  - Shortness of breath
  - Reduced breath sounds on affected side
  - Chest x-ray: lung collapse + air in pleural cavity
Pneumothorax (2 of 2)

- **Tension pneumothorax**
  - Positive pressure develops in pleural cavity
  - Air flows through perforation into pleural cavity on inspiration but cannot escape on expiration
  - Pressure builds up in pleural cavity displacing heart and mediastinal structures away from affected side

- **Chest tube inserted into pleural cavity; left in place until tear in lung heals**
  - Prevents accumulation of air in pleural cavity
  - Aids re-expansion of lung
Atelectasis (1 of 2)

• Collapse of lung
• Obstructive atelectasis caused by bronchial obstruction from
  – Mucous secretions, tumor, foreign object
  – Part of lung supplied by obstructed bronchus collapses as air absorbed
  – Reduced volume of affected pleural cavity
  – Mediastinal structures shift toward side of atelectasis
  – Diaphragm elevates on affected side
  – May develop as a postoperative complication
Atelectasis (2 of 2)

• Compression atelectasis
  – From external compression of lung by
    • Fluid
    • Air
    • Blood in pleural cavity
  – Reduced lung volume and expansion
Before atelectasis

Atelectasis of entire left lung

Affected lung appears dense with absorption of air; left half of diaphragm elevated; trachea and mediastinal structures shifted to side of collapse
Pneumonia (1 of 3)

• Inflammation of the lung
  – Exudate spreads through lung
  – Exudate fills alveoli
  – Affected lung portion becomes relatively solid (consolidation)
  – Exudate may reach pleural surface causing irritation and inflammation

• Classification
  – By etiology
  – By anatomic distribution of inflammatory process
  – By predisposing factors
Pneumonia (2 of 3)

• Etiology: most important, serves as a guide for treatment
  – Bacteria, viruses, fungi, *Chlamydia*, *Mycoplasma*, *Rickettsia*

• Anatomic distribution of inflammatory process
  – Lobar: infection of entire lung by pathogenic bacteria
  – Legionnaire’s Disease: gram-negative rod
  – Bronchopneumonia: infection of parts of lobes or lobules adjacent to bronchi by pathogenic bacteria
  – Interstitial or primary atypical pneumonia: caused by virus or *Mycoplasma*; involves alveolar septa than alveoli; septa with lymphocytes and plasma cells
Pneumonia (3 of 3)

• Predisposing factors
  – Any condition associated with poor lung ventilation and retention of bronchial secretions
  – Postop pneumonia: accumulation of mucous secretions in bronchi
  – Aspiration pneumonia: foreign body, food, vomit
  – Obstructive pneumonia: distal to bronchial narrowing

• Clinical features of pneumonia
  – Fever, cough, purulent sputum, pain on respiration, shortness of breath
**Pneumocystis Pneumonia**

- **Cause:** *Pneumocystis carinii*, protozoan parasite of low pathogenicity
- **Affects mainly immunocompromised persons**
  - AIDS, receiving immunosuppressive drugs, premature infants
- **Cysts contain sporozoites released from cysts that mature to form trophozoites; sporozoites appear as dark dots at the center of cyst on stained smears**
- **Organisms attack and injure alveolar lining leading to exudation of protein material into alveoli**
- **Cough, dyspnea, pulmonary consolidation**
- **Diagnosis:** lung biopsy by bronchoscopy or from bronchial secretions
Tuberculosis

- Infection from acid-fast bacteria, *Mycobacterium tuberculosis*
- Organism has a capsule composed of waxes and fatty substances; resistant to destruction
- Transmission: airborne droplets
- Granuloma: giant cell with central necrosis, indicates development of cell-mediated immunity
- Multi-nucleated giant cells: bacteria + fused monocytes + periphery of lymphocytes and plasma cells
  - Organisms lodge within pulmonary alveoli
  - Granulomas are formed
  - Spreads into kidneys, bones, uterus, fallopian tubes, others
Granuloma, tuberculosis
Central necrosis

Multinucleated giant cell, tuberculosis
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Tuberculosis-Outcome

• Infection arrested and granulomas heal with scarring
• Infection may be asymptomatic, detected only by chest x-ray and/or Mantoux test
• Infection reactivated: healed granulomas contain viable organisms reactivated with reduced immunity leading to progressive pulmonary TB
• Spread through blood to other organs (extrapulmonary)
  – Secondary focus of infection may progress even if pulmonary infection has healed
• Diagnosis
  – Skin test (Mantoux)
  – Chest x-ray
  – Sputum culture
Reactivated and Miliary Tuberculosis

- Reactivated tuberculosis: active TB in adults from reactivation of an old infection; healed focus of TB flares up with lowered immune resistance

- Miliary tuberculosis
  - Multiple foci (small, white nodules, 1-2 mm in diameter) of disseminated tuberculosis, resembling millet seeds
  - Large numbers of organisms disseminated in body when a mass of tuberculous inflammatory tissue erodes into a large blood vessel
  - Extensive consolidation of one or more lobes of lung
  - At-risk: AIDS and immunocompromised individuals
Drug-Resistant Tuberculosis

- Resistant strains of organisms emerge with failure to complete treatment or premature cessation of treatment
- Multiple drug-resistant tuberculosis, MTB
  - TB caused by organisms resistant to at least two of the anti-TB drugs
  - Course of treatment is prolonged
  - Results less satisfactory
- Extremely drug-resistant tuberculosis, XDR-TB
  - Caused by organisms no longer controlled by many anti-TB drugs
  - Eastern Europe, South Africa, Asia, some cases in the United States
Bronchitis and Bronchiectasis

- Inflammation of the tracheobronchial mucosa
- Acute bronchitis
- Chronic bronchitis: from chronic irritation of respiratory mucosa by smoking or atmospheric pollution
- Bronchiectasis: walls weakened by inflammation become saclike and fusiform
  - Distended bronchi retain secretions
  - Chronic cough; purulent sputum; repeated bouts of pulmonary infection

- Diagnosis: bronchogram
- Only effective treatment: surgical resection of affected segments of lung
Chronic Obstructive Pulmonary Disease (1 of 4)

- Combination of emphysema and chronic bronchitis
- Pulmonary emphysema
  - Destruction of fine alveolar structure of lungs with formation of large cystic spaces
  - Destruction begins in upper lobes eventually affecting all lobes of both lungs
  - Dyspnea, initially on exertion; later, even at rest
- Chronic bronchitis: chronic inflammation of terminal bronchioles; cough and purulent sputum
Chronic Obstructive Pulmonary Disease (2 of 4)

- Three main anatomic derangements in COPD
- Inflammation and narrowing of terminal bronchioles
  - Swelling of bronchial mucosa → reduced caliber of bronchi and bronchioles → increased bronchial secretions → increased resistance to air flow → air enters lungs more readily than it can be expelled → trapping of air at expiration
- Dilatation and coalescence of pulmonary air spaces
  - Diffusion of gases less efficient from large cystic spaces
- Loss of lung elasticity; lungs no longer recoil normally following inspiration
Chronic Obstructive Pulmonary Disease (3 of 4)

- Chronic irritation: smoking and inhalation of injurious agents

- Pathogenesis
  - 1. Inflammatory swelling of mucosa
    - Narrows bronchioles; increased resistance to expiration; causing air to be trapped in lung
  - 2. Leukocytes accumulate in bronchioles and alveoli, releasing proteolytic enzymes that attack elastic fibers of lung’s structural support
  - 3. Coughing and increased intrabronchial pressure convert alveoli into large, cystic air spaces, over-distended lung cannot expel air
  - 4. Retention of secretions predisposes to pulmonary infection
Chronic Obstructive Pulmonary Disease (4 of 4)

• Lungs damaged by emphysema cannot be restored to normal

• Management
  – Promote drainage of bronchial secretions
  – Decrease frequency of superimposed pulmonary infections
  – Surgery does not improve survival, initial benefit is short-term
Bronchial Asthma

- Spasmodic contraction of smooth muscles on walls of bronchi and bronchioles
- Dyspnea and wheezing on expiration
- Greater impact on expiration than on inspiration
- Attacks are precipitated by allergens: inhalation of dust, pollens, animal dander, other allergens

Treatment
- Drugs that dilate bronchial walls: epinephrine or theophylline
- Drugs that block release of mediators from mast cells
Neonatal Respiratory Distress Syndrome

• Progressive respiratory distress soon after birth
• Hyaline membrane disease after red-staining membranes lining alveoli
• Pathogenesis: inadequate surfactant in lungs
  – Alveoli do not expand normally during inspiration
  – Tends to collapse during expiration
• At-risk groups
  – Premature infants
  – Infants delivered by cesarean section
  – Infants born to diabetic mothers
• Treatment
  – Adrenal corticosteroids to mother before delivery
  – Oxygen + surfactant
Neonatal Respiratory Distress Syndrome
Leakage of protein rich in fibrinogen that tends to clot and form adherent eosinophilic hyaline membranes impeding gas exchange.
Adult Respiratory Distress Syndrome

• Shock – major manifestation
• Conditions: fall in blood pressure and reduced blood flow to lungs
  – Severe injury (traumatic shock)
  – Systemic infection (septic shock)
  – Aspiration of acid gastric contents
  – Inhalation of irritant or toxic gases
  – Damage caused by SARS

• Damaged alveolar capillaries leak fluid and protein
• Impaired surfactant production from damaged alveolar lining cells
• Formation of intra-alveolar hyaline membrane
Pulmonary Fibrosis

- Fibrous thickening of alveolar septa from irritant gases, organic, and inorganic particles
  - Makes lungs rigid restricting normal respiratory excursions
  - Diffusion of gases hampered due to increased alveolar thickness
  - Causes progressive respiratory disability similar to emphysema

- Collagen diseases

- Pneumoconiosis: lung injury from inhalation of injurious dust or other particulate material
  - Silicosis (rock dust) and asbestosis (asbestos fibers)
Lung Carcinoma

• Usually smoking-related neoplasm
• Common malignant tumor in both men and women
• Mortality from lung cancer in women exceeds breast cancer
• Arises from mucosa of bronchi and bronchioles
• Rich lymphatic and vascular network in lungs facilitates metastasis
• Often referred as bronchogenic carcinoma because cancer usually arises from bronchial mucosa
• Treatment: surgical resection or radiation and chemotherapy for small cell carcinoma and advanced tumors