Pulmonary Pathology

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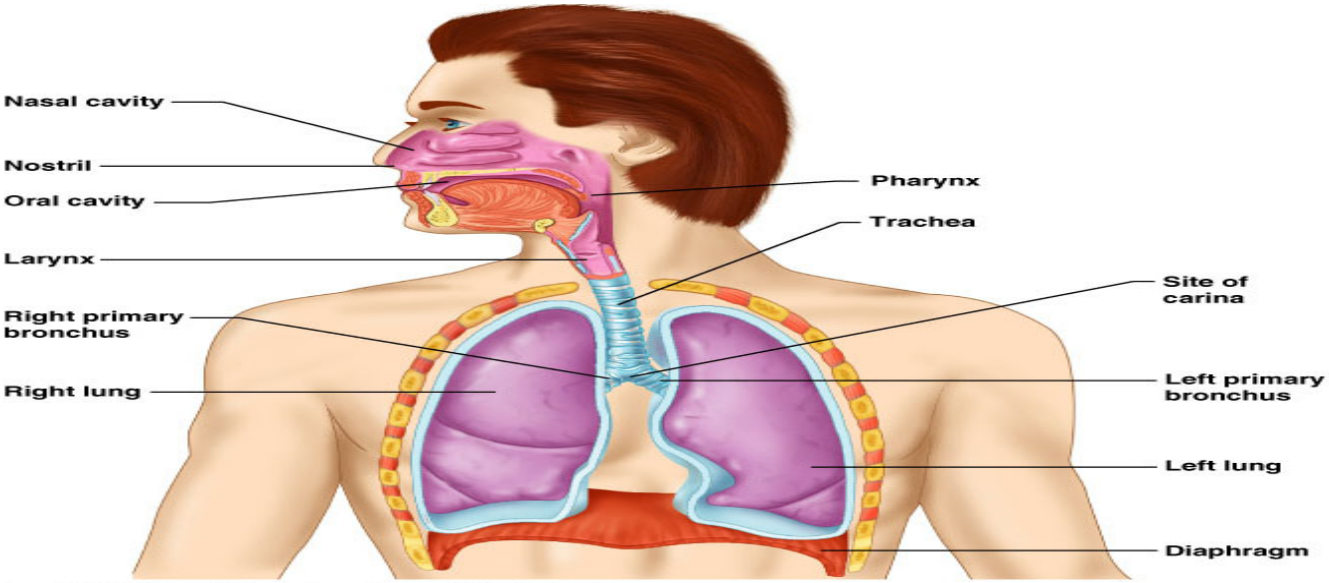
Respiratory Overview

- Respiratory and circulatory systems are closely related structurally and functionally
- External respiration – occurs at the alveoli of the lungs with the capillaries
  - This is where the O2 and CO2 exchange with the lung capillaries
- Internal respiration – takes place between the blood capillaries and the tissue cells.

Major Functions of the Respiratory System

- To supply the body with oxygen and dispose of CO2
- Respiration – four distinct processes must happen
  - Pulmonary ventilation – moving air into and out of the lungs
  - External respiration – gas exchange between the lungs and the blood
  - Transport – transport of oxygen and carbon dioxide between the lungs and tissues
  - Internal respiration – gas exchange between systemic blood vessels and tissues



Respiratory System

- Consists of the respiratory and conducting zones
- Respiratory zone
  - Site of gas exchange
  - Consists of bronchioles, alveolar ducts, and alveoli
- Conducting zone
  - Provides rigid conduits for air to reach the sites of gas exchange
  - Includes all other respiratory structures (e.g., nose, nasal cavity, pharynx, trachea)
- Respiratory muscles – diaphragm and other muscles that promote ventilation

Respiratory Defense Mechanisms

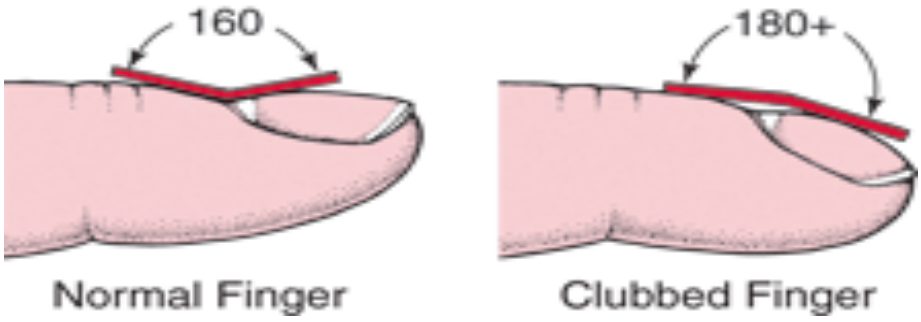
- Warm and humidify incoming air
- Branching of bronchial tree increases its contact with airway mucus
- Cilia prevents particles form reaching distal airways
- Mucus blanket
    - Particle clearance (macrophages)
    - Antibacterial secretions (lysosome)
    - Antiviral secretions (interferons)

Signs and Symptoms of Pulmonary Disease

- **Dyspnea**
    - Subjective sensation of uncomfortable breathing
    - Orthopnea - Dyspnea when a person is lying down
    - Paroxysmal nocturnal dyspnea (PND)
- **Cough**
    - Acute cough
    - Chronic cough
- **Abnormal sputum**
- **Hemoptysis**
- **Abnormal breathing patterns:**
    - Kussmaul respirations (hyperpnea)
    - Cheyne-Stokes respirations
- **Hypoventilation**
    - Hypercapnia - Increased CO_2 due to hypoventilation
- **Hyperventilation**
    - Hypocapnia - Decreased CO_2 due to hyperventilation

Signs and Symptoms of Pulmonary Disease

- **Pain**
- **Cyanosis**
- **Clubbing**
    - Finger clubbing is characterized by enlarged fingertips and a loss of the normal angle at the nail bed.



Conditions Caused by Pulmonary Disease or Injury

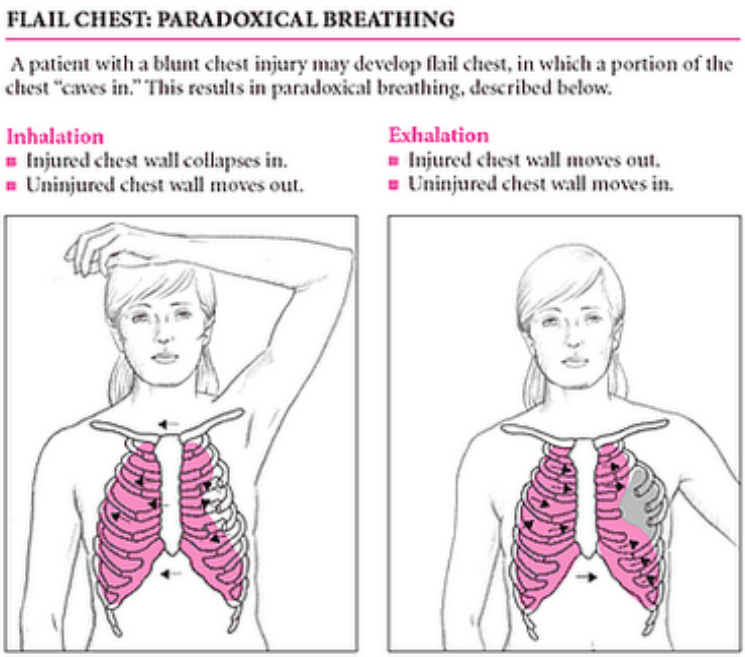
- **Hypercapnia -** Hypoxemia Increased CO2 due to hypoventilation
- **Hypoxemia**
  - Hypoxemia versus hypoxia - The body or a region of the body is deprived of adequate oxygen. Hypoxia may be classified as either *generalized*, affecting the whole body, or *local*.
  - Ventilation-perfusion abnormalities
    - Shunting A pulmonary shunt is a physiological condition which results when the alveoli of the lungs are perfused with blood as normal, but ventilation (the supply of air) fails to supply the perfused region.

Acute Respiratory Failure - Hypoperfusion

- **Hypoperfusion:** inadequate blood flow to pulmonary capillaries
  - **Heart Failure**
    - **Failing left heart**- pulmonary hypertension, reduced blood flow at higher pressure
    - **Failing right heart**- reduced pulmonary blood flow
  - **Thromboembolism** - blockage of vessel
    - Blockage of vessel by embolus
    - Release of vasoconstrictors from activated platelets
  - **Reduced Ventilation**
    - Vasoconstriction response in arteriole

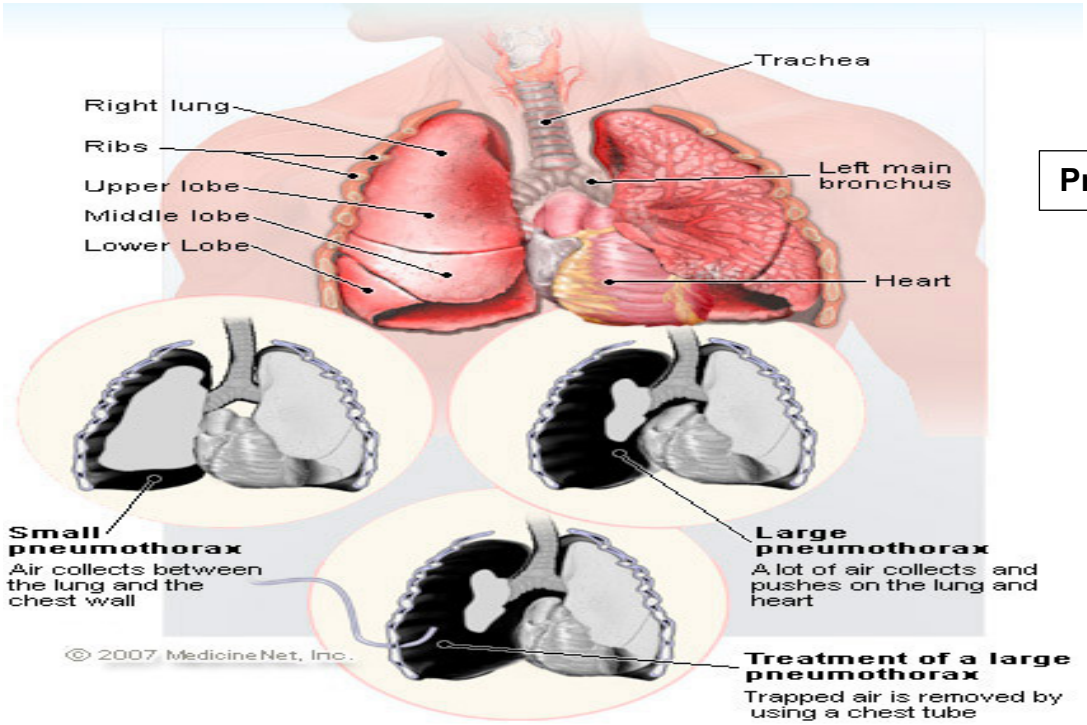
Chest Wall Disorders

- **Chest wall restriction**
  - Compromised chest wall
    - Deformation, immobilization, and/or obesity
- **Flail chest**
  - Instability of a portion of the chest wall

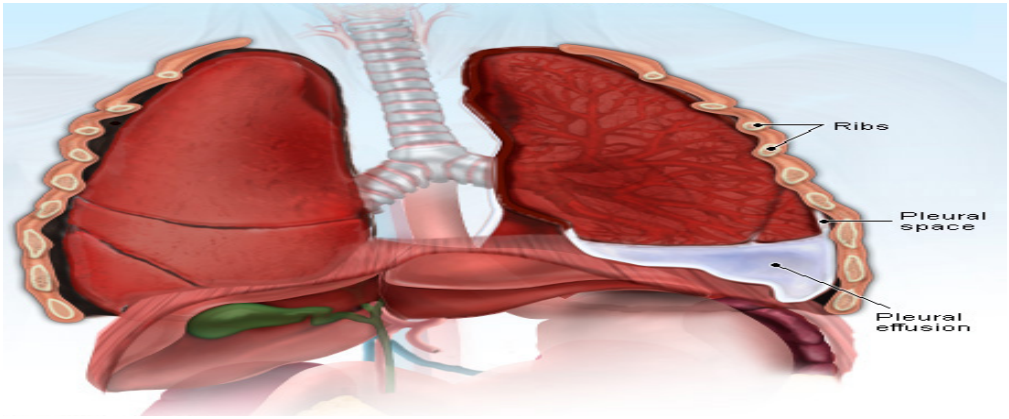


Pleural Abnormalities

- **Pneumothorax**
  - Open pneumothorax
  - Tension pneumothorax
  - Spontaneous pneumothorax
  - Secondary pneumothorax
- **Pleural effusion**
  - Transudative effusion
  - Exudative effusion
  - Hemothorax & Chylothorax
  - Empyema - Infected pleural effusion

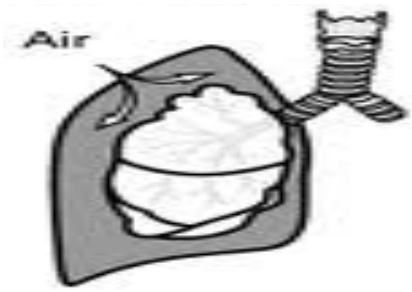


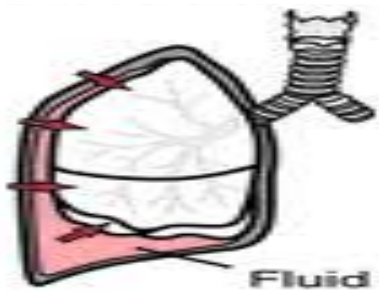
Pneumothorax

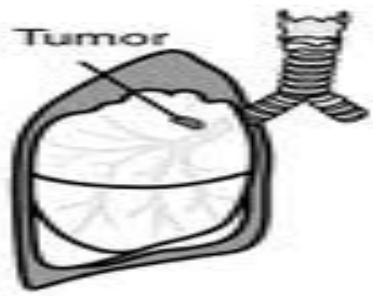


Pleural Effusion

Pneumothorax From Collapse	Hydrothorax From Compression	Pneumothorax From Obstruction







Restrictive Lung Diseases

- Aspiration
  - Passage of fluid and solid particles into the lungs
- Atelectasis
  - Compression atelectasis
  - Absorption atelectasis
- Bronchiectasis
  - Persistent abnormal dilation of the bronchi

Atelectasis

- Atelectasis, also known as collapse, is loss of lung volume caused by inadequate expansion of air spaces.
- It results in shunting of inadequately oxygenated blood from pulmonary arteries into veins, thus giving rise to a ventilation- perfusion imbalance and hypoxia.

Resorption Atelectasis

- Resorption atelectasis occurs when an obstruction prevents air from reaching distal airways. The air already present gradually becomes absorbed, and alveolar collapse follows.
- The most common cause of resorption collapse is obstruction of a bronchus by a mucous or mucopurulent plug.
- This frequently occurs postoperatively but also may complicate bronchial asthma, bronchiectasis, chronic bronchitis, tumor, or foreign body aspiration, particularly in children.

Compression Atelectasis

- Contraction occurs when either local or generalized fibrotic changes in the lung or pleura hamper expansion and increase elastic recoil during expiration.
- This is irreversible
- Atelectasis (except when caused by contraction) is potentially reversible and should be treated promptly to prevent hypoxemia and superimposed infection of the collapsed lung.

Bronchiectasis

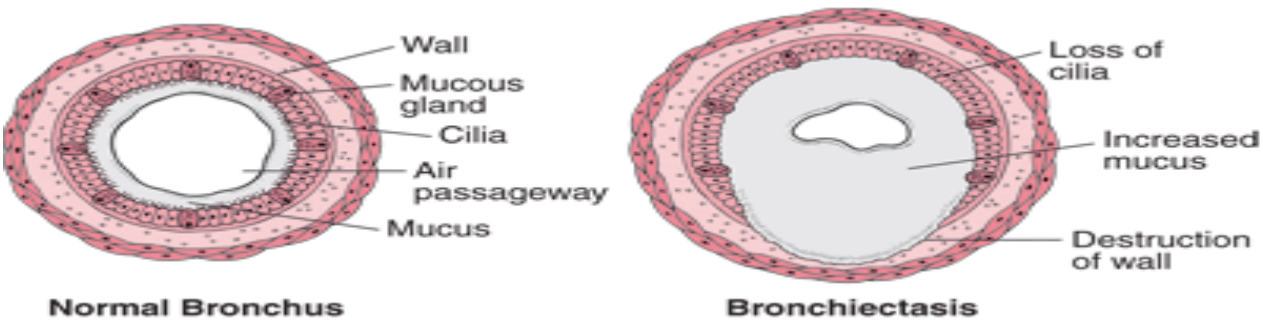
- Bronchiectasis is the permanent dilation of bronchi and bronchioles caused by destruction of the muscle and the supporting elastic tissue, resulting from or associated with chronic necrotizing infections.
- It is not a primary disease but rather secondary to persisting infection or obstruction caused by a variety of conditions.
- Pathophysiology
  - Irreversible dilation of part of the bronchial tree
  - Caused by chronic infection of bronchi & bronchioles
  - Chronic bronchial infection causes a dilatation of the air passages which are trapped with muco-purulent material
  - Caused by slow-growing bacteria and fungi

Bronchiectasis - continued

- S & S
  - Chronic deep hacking cough
  - Copious amounts of foul-spelling pus sputum
  - Frequent attacks of pneumonia
- Diagnosis
  - Localized rales and coarse rhonchi
  - Appears similar to COPD with clubbing
  - Normal blood gases
  - History of chronic infection
  - CT scan confirms the diagnosis
- Treatment
  - Antibiotics – ciprofloxacin
  - Bronchopulmonary drainage
    - Bending over, almost standing on head, to get the mucus up and out
  - Bronchodilators
- Once developed, it gives rise to a characteristic symptom complex dominated by cough and expectoration of copious amounts of purulent sputum.
- Diagnosis depends on an appropriate history along with radiographic demonstration of bronchial dilation.

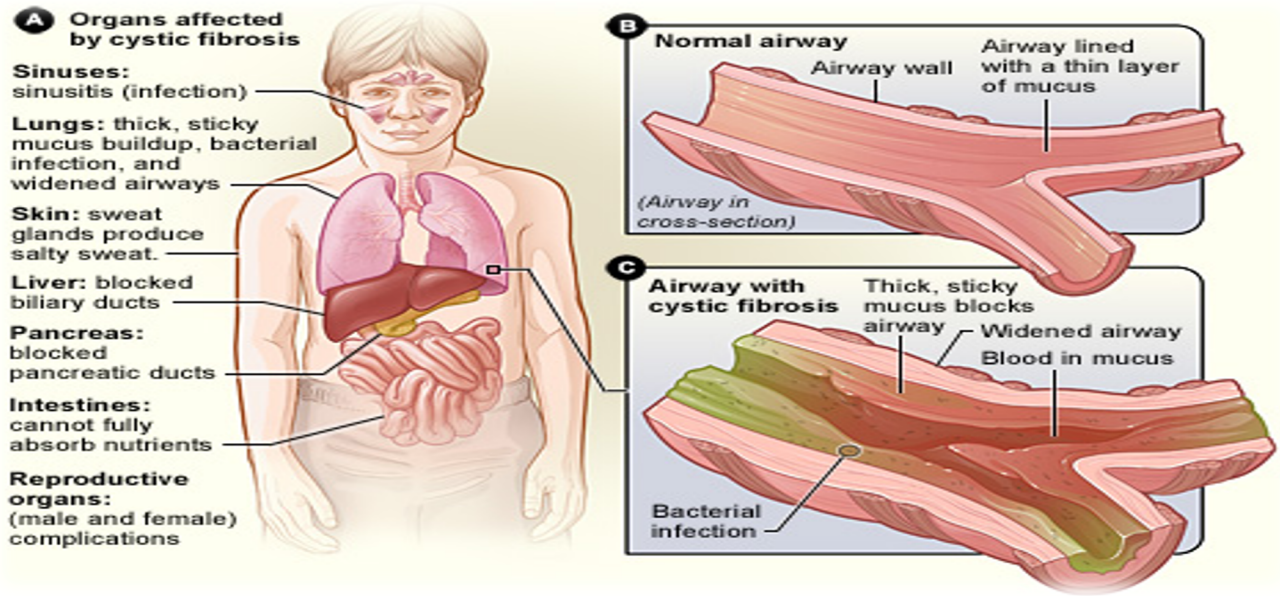
Clinical Features of Bronchiectasis

- The clinical manifestations consist of severe, persistent cough with expectoration of mucopurulent, sometimes fetid, sputum.
  - The sputum may contain flecks of blood; frank hemoptysis can occur.
- Symptoms often are episodic and are precipitated by upper respiratory tract infections or the introduction of new pathogenic agents.
- Clubbing of the fingers may develop.
- The conditions that most commonly predispose to bronchiectasis include:
  - In *cystic fibrosis,* widespread severe bronchiectasis results from obstruction caused by the secretion of abnormally viscid mucus thus predisposing to infections of the bronchial tree.
  - In immunodeficiency states, particularly immunoglobulin deficiencies, localized or diffuse bronchiectasis is likely to develop because of an increased susceptibility to repeated bacterial infections.



Cystic Fibrosis

- Inherited disease that causes thick, sticky mucus to build up in the lungs and digestive tract
- The most common type of chronic lung disease in children and young adults
    - 1 in every 3,300 – most children and teenagers
- May result in early death
- S & S - Pneumonitis, bronchiectasis, lung abscesses, pancreatic insufficiency
- Diagnosis - Established by the sweat electrolyte test



Acute Respiratory Distress Syndrome

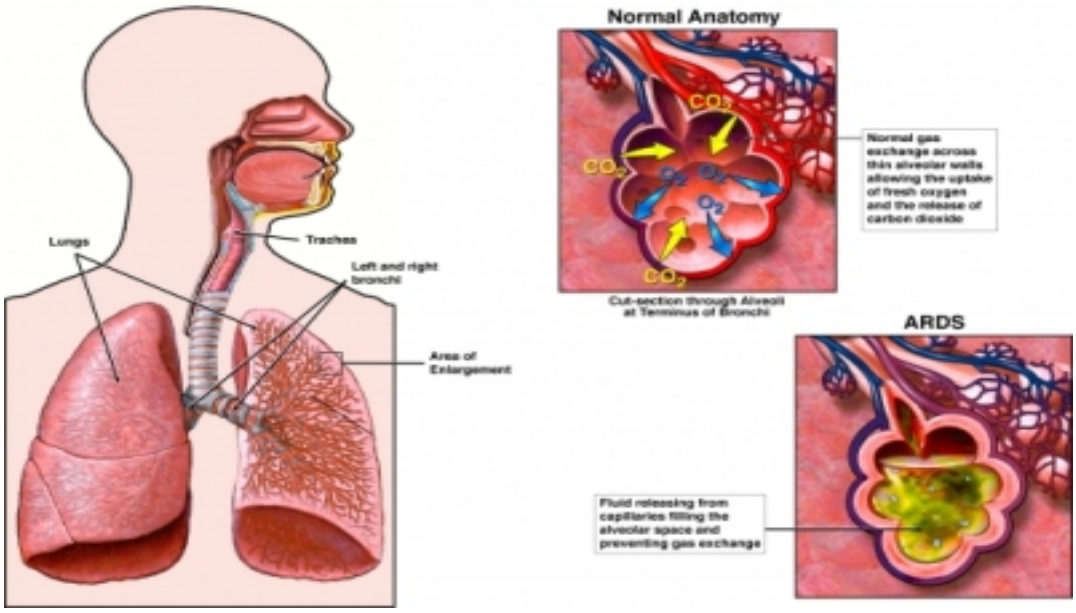
- Acute respiratory distress syndrome (ARDS) is a clinical syndrome caused by diffuse alveolar capillary and epithelial damage.
- Fulminant form of respiratory failure characterized by acute lung inflammation and diffuse alveolocapillary injury
- Injury to the pulmonary capillary endothelium
- Surfactant inactivation
- The usual course is characterized by rapid onset of life-threatening respiratory insufficiency, cyanosis, and severe arterial hypoxemia that is refractory to oxygen therapy and may progress to multisystem organ failure.
- About 85% of patients of patients with an acute lung injury will develop ARDS within 72 hours after the injury.
    - The mortality rate has decreased from 60% to 40% in the last decade.
    - Worse prognosis more common with age, bacteremia, sepsis, and multisystem failure.
- If the patient survives the initial acute phase, diffuse interstitial fibrosis can occur, compromising respiratory function.
- In most patients who survive the acute phase, normal respiratory function returns in 6-12 months.

Acute Respiratory Distress Syndrome - continued

- Manifestations:
  - Hyperventilation
  - Respiratory alkalosis
  - Dyspnea and hypoxemia
  - Metabolic acidosis
  - Hypoventilation
  - Respiratory acidosis
  - Further hypoxemia
  - Hypotension, decreased cardiac output, death
- Evaluation and treatment
  - Physical examination, blood gases, and radiologic examination
  - Supportive therapy with oxygenation and ventilation and prevention of infection
  - Surfactant to improve compliance

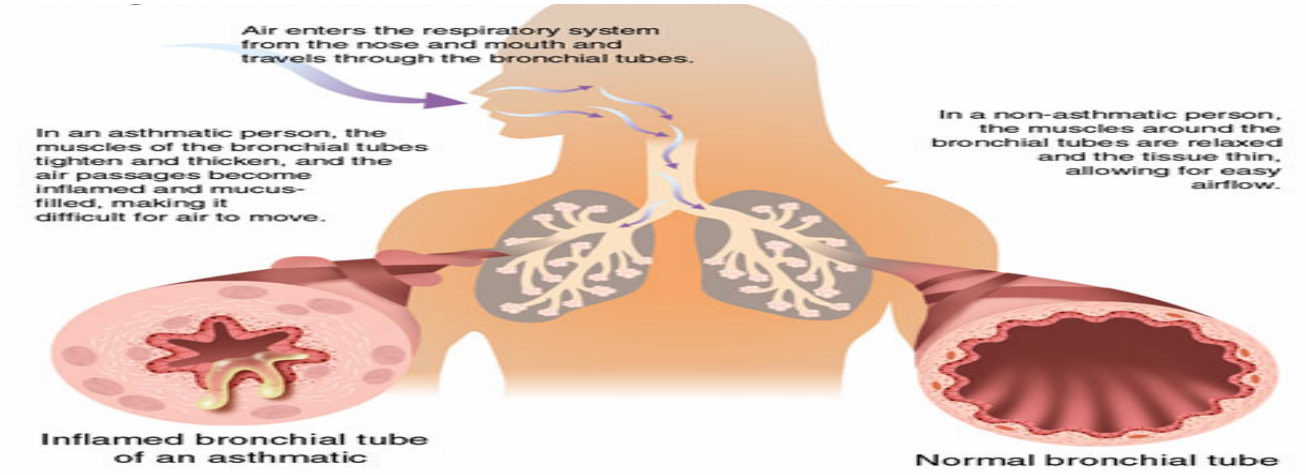
ARDS Summary

- ARDS is a clinical syndrome of progressive respiratory insufficiency caused by diffuse alveolar damage in the setting of sepsis, severe trauma, or diffuse pulmonary infection.
- Neutrophils and their products have a crucial role in the pathogenesis of ARDS by causing endothelial and epithelial injury.
- The characteristic histologic picture is that of alveolar edema, epithelial necrosis, accumulation of neutrophils, and presence of hyaline membranes lining the alveolar ducts.



Obstructive Lung Diseases

- Obstructive airway disease is characterized by limitation of airflow, usually resulting from an increase in resistance by a partial or complete obstruction at any level.
- The major obstructive disorders are asthma, chronic bronchitis, emphysema, and bronchiectasis.
- In patients with these diseases, forced vital capacity (FVC) is either normal or slightly decreased, while the expiratory flow rate, usually measured as the forced expiratory is significantly decreased.
    - Forced vital capacity or FVC, which is the amount of air that an individual can forcibly exhale from his / her lungs after taking the deepest breath they can.
- This is classically observed in asthma, or from loss of elastic recoil, characteristic of emphysema



Asthma

- Asthma is a chronic inflammatory disorder of the airways that causes recurrent episodes of wheezing, breathlessness, chest tightness, and cough, particularly at night and/or early in the morning.
- The hallmarks of the disease are intermittent and reversible airway obstruction, chronic bronchial inflammation with eosinophils, bronchial smooth muscle cell hypertrophy and hyperreactivity, and increased mucus secretion.
- Some of the stimuli that trigger attacks in patients would have little or no effect in persons with normal airways.
    - Many cells play a role in the inflammatory response, in particular eosinophils, mast cells, macrophages, lymphocytes, neutrophils, and epithelial cells.
    - Asthma may be categorized into atopic (evidence of allergen sensitization, often in a patient with a history of allergic rhinitis, eczema) and nonatopic.
        - In either type, episodes of bronchospasm can be triggered by diverse mechanisms, such as respiratory infections (especially viral), environmental exposure to irritants (e.g., smoke, fumes), cold air, stress, and exercise.
- Asthma also may be classified according to the agents or events that trigger bronchoconstriction

Atopic Asthma

- This is the most common type of asthma, usually staring in childhood and is a class Type 1 Hypersensitivity reaction.
- This is sometimes called extrinsic asthma because it is an allergic reaction due to something outside
- A positive family history of atopy and or asthma is common.
- Asthma attacks in children are often preceded by allergic rhinitis, urticaria, or eczema.
- This is triggered by environmental antigens, such as dust, pollens animal dander and foods.
- The antigens are diagnosed with skin tests.

Non Atopic Asthma

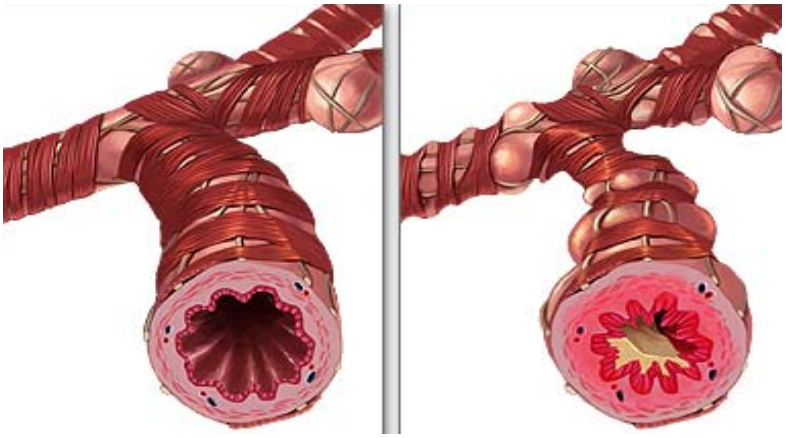
- Sometimes called intrinsic asthma because it is caused by something inside.
- Viral infection, irritants like epithelial damage, mucosal inflammation, emotional upset, parasympathetic input.
- More common in adults
- Patients with nonatopic forms of asthma do not have evidence of allergen sensitization, and skin test results usually are negative. A positive family history of asthma is less common.
- Respiratory infections due to viruses (e.g., rhinovirus, influenza virus) and inhaled air pollutants are common triggers

Drug Induced Asthma

- Several pharmacologic agents provoke asthma, aspirin being the most striking example.
- Patients with aspirin sensitivity present with recurrent rhinitis and nasal polyps, urticaria, and bronchospasm.
- Aspirin induces arachidonic acid metabolism and the increase of bronchoconstrictor leukotrienes.
- It is estimated that 10% of the adult population has this sensitivity.

Occupational Asthma

- This form of asthma is stimulated by fumes (epoxy resins, plastics), organic and chemical dusts (wood, cotton, platinum), gases (toluene), and other chemicals.
- Asthma attacks usually develop after repeated exposure to the inciting antigen(s)
- Sometimes called wood dust asthma.



Asthma Clinical Features

- An attack of asthma is characterized by severe dyspnea with wheezing; the chief difficulty lies in expiration.
- The victim labors to get air into the lungs and then cannot get it out, so that there is progressive hyperinflation of the lungs with air trapped distal to the bronchi, which are constricted and filled with mucus and debris.
- In the usual case, attacks last from 1 to several hours and subside either spontaneously or with therapy, usually bronchodilators and corticosteroids.
- Intervals between attacks are characteristically free from overt respiratory difficulties, but persistent, subtle deficits can be detected by spirometry.

Status Asthmaticus

- Occasionally a severe paroxysm occurs that does not respond to therapy and persists for days and even weeks (*status asthmaticus*).
- The associated hypercapnia, acidosis, and severe hypoxia may be fatal, although in most cases the condition is more disabling than lethal.

Asthma Summary

- Asthma is characterized by reversible bronchoconstriction caused by airway hyperresponsiveness to a variety of stimuli.
- Atopic asthma is caused by a IgE-mediated immunologic reaction to environmental allergens.
- Triggers for nonatopic asthma are less clear but include viral infections and inhaled air pollutants, which can also trigger atopic asthma.
- Eosinophils are key inflammatory cells found in almost all subtypes of asthma; eosinophil products such as major basic protein are responsible for airway damage.
- Airway remodeling (sub-basement membrane thickening and hypertrophy of bronchial glands and smooth muscle) adds an irreversible component to the obstructive disease.

Two Major Types of Asthma – Intrinsic and extrinsic

Intrinsic Asthma	**Extrinsic Asthma**
Caused by a virus	Caused by an allergen
Most common in adults	Most common in children
Non allergic factors, viral infection, irritants like epithelial damage, mucosal inflammation, emotional upset, parasympathetic input	Allergic factors can include dust mites, IgE antibodies, pet dander, and other environmental allergens. Rinovirus (during first 3 years of life) This is why chalkboards are not in schools.
Trigger epithelial cell damage and macrophages	Triggers activate lymphocytes & mast cells.
The affected cells are neutrophils This is sometimes called Non Eosinophilic Asthma	The affected cells are eosinophils (acidophils) This is sometimes called Eosinophilic Asthma
Histamine reactions causes airway inflammation	Histamine reactions causes airway inflammation
Airway hyper responsiveness, irritation, edema, mucous plugging	Airway hyper responsiveness, irritation, edema, mucous plugging

Pulmonary Disorders

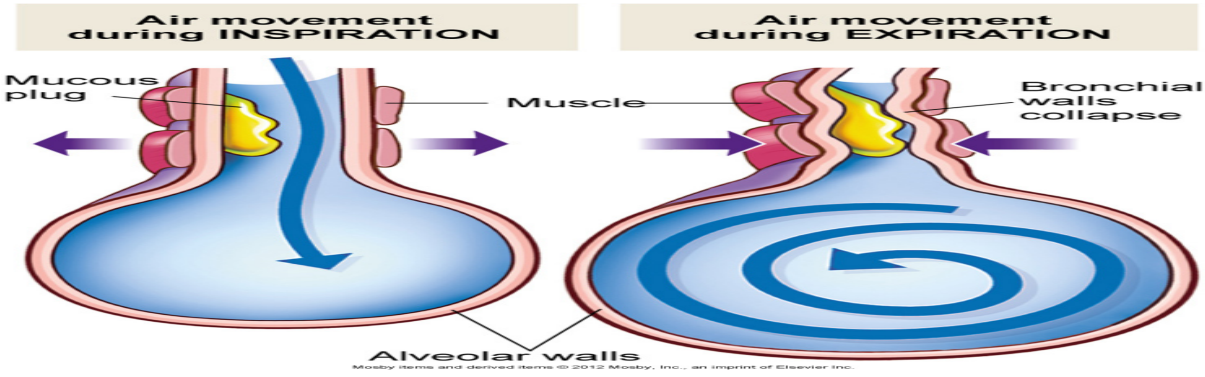
- Obstructive lung diseases: chronic bronchitis
    - Hypersecretion of mucus and chronic productive cough that lasts for at least 3 months of the year and for at least 2 consecutive years
    - Inspired irritants increase mucus production and the size and number of mucous glands
    - The mucus is thicker than normal
    - Bronchodilators, expectorants, and chest physical therapy used to treat
- Obstructive lung diseases: emphysema
    - Abnormal permanent enlargement of the gas-exchange airways accompanied by destruction of alveolar walls without obvious fibrosis
    - Loss of elastic recoil
    - Centriacinar emphysema
        - Most common form usually in proximal respiratory bronchioles in the top parts of the lungs
    - Panacinar emphysema
        - Destroys the entire alveolus and is usually found in the bottom half of the lungs

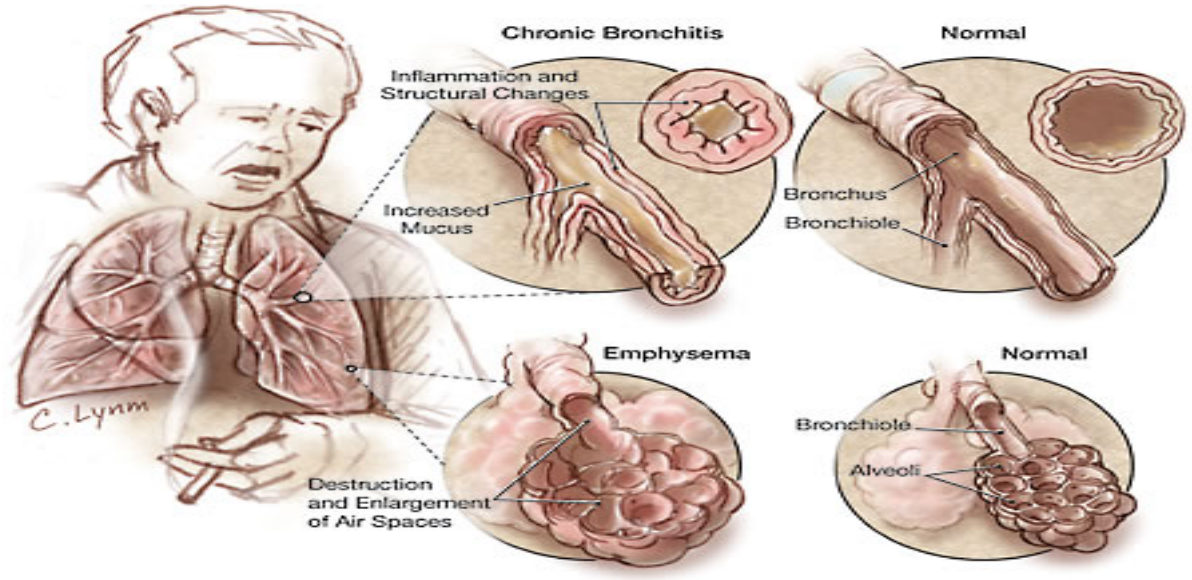
How COPD Develops

- Smoking causes increased mucus production and bronchial inflammation
- Nicotine paralyzes the mucociliary escalator
- Mucociliary escalator traps mucus, bacteria, irritants
- Nicotine blocks protein inhibitors which will eventually dissolve the alveoli
- Pathophysiology - Involves all four parts of the respiratory tract
    - Bronchi
    - Bronchioles
    - Alveoli
    - Parenchyma

Specific Pathology

- Increased resistance to airflow
- Loss of elastic recoil
- Decreased expiratory flow rate
- Alveolar walls frequently break because of the increased resistance of air flows
- The hyper inflated lungs flatten the curvature of the diaphragm and enlarge the rib cage
- The altered configuration of the chest cavity places the respiratory muscles, including the diaphragm, at a mechanical disadvantage and impairs their force-generating capacity
- Consequently, the metabolic work of breathing increases, and dyspnea increases





Interstitial Lung Diseases

- Refers to a group of about 100 chronic lung disorders characterized by inflammation and scarring that make it hard for the lungs to get enough oxygen.
- The scarring is called pulmonary fibrosis.
- The symptoms and course of these diseases may vary from person to person.
- The common link between the many forms of the disease is that they all begin with inflammation.

Inflammatory Signs of Interstitial Lung Diseases

- Bronchiolitis: inflammation of the small airways (bronchioles).
- Alveolitis: inflammation of the air sacs where oxygen and carbon dioxide exchange in the blood takes places (alveoli).
- Vasculitis: inflammation that involves the small blood vessels (capillaries).

Causes of interstitial lung disease

- The cause of interstitial lung disease is not known. Major contributing factors include:
- Smoking
- Certain drugs or medicines
- Exposure to substances at work or in the environment such as organic or inorganic dusts
- Certain connective tissue or collagen diseases and sarcoidosis
- Family history
- Radiation treatment

Symptoms of Interstitial Lung Diseases

- Shortness of breath, especially with activity
- Dry, hacking cough that does not produce phlegm
- Extreme tiredness and weakness
- Loss of appetite
- Unexplained weight loss
- Discomfort in the chest
- Labored breathing, which may be fast and shallow
- Bleeding in the lungs

Pulmonary Fibrosis

- Referred to as interstitial lung diseases
- Causes inflammation and fibrosis of the connective tissue between the alveoli
- Most common causes
  - Environmental causes – inhaled dusts, asbestosis, silicosis, glass makers, construction workers
  - Antigens – hypersensitivity pneumonitis
  - Drugs – Methotrexate
  - Radiation injury
- Other diseases – sarcoidosis, RA
- Mimicking disorders similar presentation but vastly differ
- **Pathology of interstitial lung disease**
  - Inflammation of the alveolar wall and inter-alveolar spaces
  - Fibrous scarring
  - Granuloma formation
  - End stage leads to a mass of scar tissue with contraction and the formation of cystic areas
- Impairment of pulmonary function
  - Decreased lung volume
  - Decreased compliance (stiff lungs)
  - Impairment of diffusion
  - Decreased gas exchange
  - Shunting and spasm of pulmonary arteries
  - Heart failure resulting from pulmonary hypertension

Pulmonary Fibrosis – continued

- **S & S of pulmonary fibrosis**
  - Obvious dyspnea
  - Chronic nonproductive cough
  - Clubbing
  - Mild cyanosis
- **Diagnosis of pulmonary fibrosis**
  - CT scan is confirmatory
- **Specific diseases that can cause pulmonary fibrosis**
  - Silicosis – disease of glass makers, sand blasters, rock miners and stone cutters
    - Takes 20 years to develop
  - Pneumoconiosis – coal miner's disease
    - Severe lung fibrosis with hypoxia
  - Asbestosis – leads to 3 distinct diseases
    - Branchiogenic carcinoma
    - Mesothelioma of lung (cancer of lung pleura)
    - Interstitial fibrosis – takes 20 years to develop
  - Drug-induced pulmonary fibrosis – chemotherapy
- Treatment of pulmonary fibrosis
  - Very little effective care
  - Oxygen 24 / 7
  - Corticosteroids

Pulmonary Diseases of Vascular Origen - Pulmonary Embolism

- Blood clots that occlude the large pulmonary arteries are almost always embolic in origin.
- More than 95% of all pulmonary emboli arise from thrombi within the large deep veins of the lower legs, typically originating in the popliteal vein and larger veins above it**.**
- Thromboembolism causes approximately 50,000 deaths per year in the United States.
- Even when not directly fatal, it can complicate the course of other diseases.
- Some cases of embolism undoubtedly occur outside the hospital in ambulatory patients, in whom the emboli are small and clinically silent.
- Even among hospitalized patients, no more than one third are diagnosed before death.
- Autopsy data on the incidence of pulmonary embolism vary widely, ranging from 1% in the general hospitalized population, to 30% in persons dying after severe burns, trauma, or fractures.

Pulmonary Embolism Risk Factors

- Prolonged bedrest (particularly with immobilization of the legs)
- Surgery, especially orthopedic surgery, of knee and hip
- Severe trauma (including burns or multiple fractures)
- Congestive heart failure
- Women using birth control pills with high estrogen content
- Primary disorders of hypercoagulability
- Consequences of embolic pulmonary arterial occlusion
  - Sudden increase in pulmonary artery pressure
  - Diminished cardiac output
  - Right-sided heart failure or even death
  - Or even death

Clinical Features of Pulmonary Embolism

- Most pulmonary emboli (60% to 80%) are clinically silent because they are small
- In 5% of cases, sudden death, acute right-sided heart failure, or cardiovascular collapse (shock) may occur typically when more than 60% of the total pulmonary vasculature is obstructed
- Massive pulmonary embolism is one of the few causes of literally instantaneous death, even before the person experiences chest pain or dyspnea.

Prophylactic Therapy

- Anticoagulation
- Early ambulation for postoperative and post parturient patients
- Application of elastic stockings
- Intermittent pneumatic calf compression
- Isometric leg exercises for bedridden patients
- Patients with pulmonary embolism are given anticoagulation therapy
- Patients with massive pulmonary embolism are candidates for thrombolytic therapy.

Pulmonary Diseases of Vascular Origen – Pulmonary Hypertension

- Pulmonary hypertension (PH or PHTN) is a condition of increased blood pressure in the arteries of the lungs.
- Symptoms include shortness of breath, fainting, tiredness, chest pain, swelling of the legs, and tachycardia
- Onset is typically gradual.
- Risk Factors
  - Family history, Pulmonary embolism
  - HIV /AIDS, Sickle Cell
  - Cocaine use, COPD
  - Sleep apnea, High altitudes
- Primary pulmonary hypertension (PPH) is a severe and progressive disease
  - Without treatment, the median survival is 2.8 years, with survival rates of 68%, 48%, and 34% at 1, 3, and 5 years.

Pneumonia

- 2-3 million cases in USA yearly causing 45,000 deaths
  - Mortality is 4 times higher over 65
- Predisposing factors
  - Preceded by viral URI causing cilia damage and the production of serous exudates
  - Smoking impairs mucociliary escalation
  - Elderly and compromised immune systems
  - HIV, AIDS, sickle cell disease, diabetes
  - Organ transplant patients
  - Close indoor quarters in the winter
  - Hypostatic pneumonia can occur from constant laying down

Pneumonia diagnosis based upon Acute vs. Chronic

- Acute
  - Symptoms within 1-2 days after exposure
  - Shaking, fever, chills, prostration, dyspnea
  - Common cause of death before antibiotics
- Chronic
  - More slow progressive form
  - Are most viral and fungal pneumonias
  - May last several weeks to months

Pneumonia diagnosis based on symptoms

- Typical pneumonia
  - Rapid onset, productive cough, fever
  - X-ray changes
- Atypical pneumonia
- Common with most viral pneumonias

Pneumonia diagnosis based on part of the lungs affected

- Lobar pneumonia
  - “Classic” pneumonia in which all the alveoli sacs in the lobe are pus filled or fluid filled
- Bronchopneumonia
  - Patchy infiltration throughout the bronchi and bronchioles
- Interstitial pneumonia
  - In the connective tissue between the alveoli with granular infiltration
- Lung abscess
  - Organisms destroy tissue and form pus abscess
- Empyema
  - Purulent infection in the pleural space
- Nodular lung infections
  - TB, coccidiomycosis and histoplasmosis cause nodular infiltrations

Pneumonia diagnosis according to where the pneumonia was acquired

- Community acquired
  - Acquired anywhere in the community, but not in a hospital
- Nosocomial
  - Acquired in a hospitalized setting

Pneumonia diagnosis according to etiologic agent

- Pneumococcal pneumonia
  - Classic bacterial pneumonia
  - AKA streptococcal pneumonia
- Aspiration pneumonia
  - Common in elderly from swallowing gastric or food contents in the trachea
  - Often vomiting with loss on consciousness
- Hemophilus pneumonia
  - Common on smokers with COPD
- Staphylococci pneumonia
  - Virulent infection often after influenza
- Viral pneumonia
  - Most common form

Signs and symptoms of pneumonia

- Cough, sore throat, fever, chills, rapid breathing, wheezing, dyspnea, chest or abdominal pain, exhaustion, vomiting

Diagnosis of pneumonia

- Medical history, physical examination, x-ray

Treatment of pneumonia

- Antibiotics, respiratory therapy with oxygen
- Amoxicillin is first-line therapy
- Steroids for wheezing
- Expectorates and lots of fluids
- Codeine for severe pain

Tuberculosis – TB

- One third of world population have active or latent infection resulting in 3 million deaths per year
- **Pathology and course of TB**
  - A chronic destruction of the lung with scarring
  - Slow progressive lung damage and possible death
  - Systemic symptoms of wasting, fatigue, night sweats, appetite loss – used to be called consumption
- **Causative Agent**
  - *Mycobacterium tuberculosis*
    - Gram-positive cell wall type
    - Slender bacillus
    - Slow growing - Generation time 12 hours or more
    - Resists most prevention methods of control
- **Pathogenesis**
  - Usually contracted by inhalation of airborne organisms
  - Bacteria are taken up by pulmonary macrophages in the lungs
  - Resists destruction within phagocyte
  - Organisms are carried to lymph nodes
    - About 2 weeks post infection intense immune reaction occurs
    - Macrophages fuse together to make large multinucleated cell
    - Macrophages and lymphocytes surround large cell
    - This is an effort to wall off infected tissue
    - Activated macrophages release into infected tissue
    - Causes death of tissue resulting in formation of “cheesy” material
  - **Epidemiology**
    - Estimated 10 million Americans infected
      - Rate highest among non-white, elderly poor people
    - Small infecting dose
      - As little as ten inhaled organisms
    - Factors important in transmission
      - Frequency of coughing, adequacy of ventilation, degree of crowding
    - Tuberculin test used to detect those infected
      - Small amount of tuberculosis antigen is injected under the skin
      - Injection site becomes red and firm if infected
      - Positive test does not indicate active disease
  - **S & S**
    - Chronic productive cough, sputum, hemoptysis.
    - TB spread to organs leads to destruction of organs and organ systems
  - **Prevention**
    - Vaccination for tuberculosis widely used in many parts of the world
    - Vaccine not given in United States because it eliminates use of tuberculin test as diagnostic tool

Tuberculosis – TB

- Treatment
    - Antibiotic treatment is given in cases of active TB
        - Two or more medications are given together to reduce potential antimicrobial resistance
        - Antimicrobials include
            - Rifampin and Isoniazid (INH)
                - Both target actively growing organisms and metabolically inactive intracellular organisms
        - Therapy is pronged
            - Lasting at least 6 months
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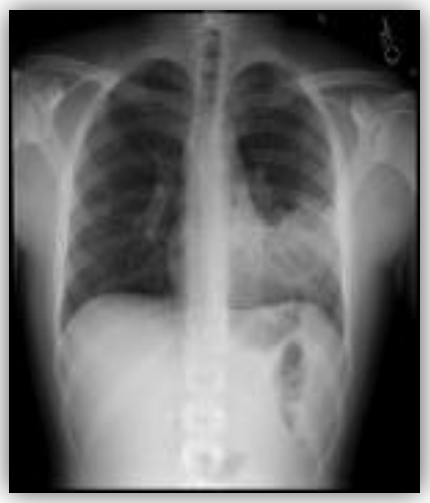
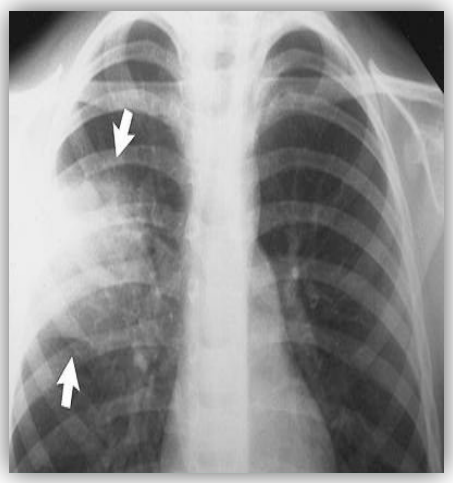




① Airborne *Mycobacterium tuberculosis* bacteria are inhaled and lodge in the lungs.

② The bacteria are phagocytized by lung macrophages and multiply within them, protected by lipid-containing cell walls and other mechanisms.

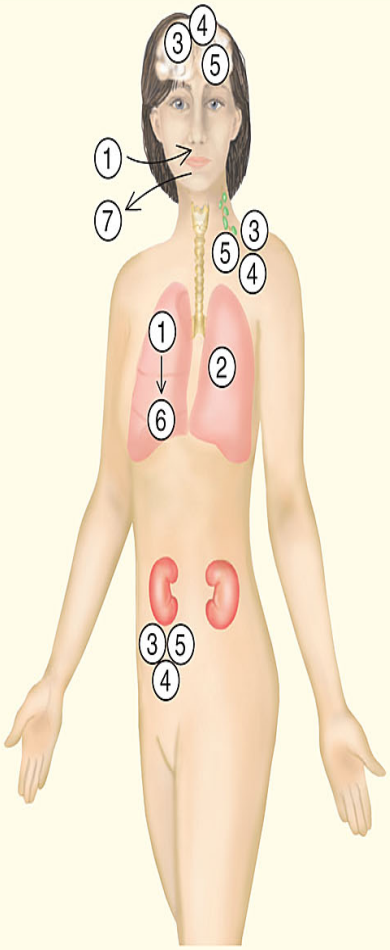
③ Infected macrophages are carried to various parts of the body such as the kidneys, brain, lungs, and lymph nodes; release of *M. tuberculosis* occurs.

④ Delayed hypersensitivity develops; wherever infected *M. tuberculosis* has lodged, an intense inflammatory reaction develops.

⑤ The bacteria are surrounded by macrophages and lymphocytes; growth of the bacteria ceases.

⑥ Intense inflammatory reaction and release of enzymes can cause caseation necrosis and cavity formation.

⑦ With uncontrolled or reactive infection, *M. tuberculosis* exits the body through the mouth with coughing or singing.



Symptoms	Chronic fever, weight loss, cough, sputum production
Incubation period	2 to 10 weeks
Causative agent	*Mycobacterium tuberculosis;* unusual cell wall with high lipid content
Pathogenesis	Colonization of the alveoli incites inflammatory response; ingestion by macrophages follows; organisms survive ingestion and are carried to lymph nodes, lungs, and other body tissues; tubercle bacilli multiply; granulomas form.
Epidemiology	Inhalation of airborne organisms; latent infections can reactivate.
Prevention and treatment	BCG vaccination, not used in the United States; tuberculin (Mantoux) test for detection of infection, allows early therapy of cases; treatment of all high-risk cases including young people with positive tests and individuals whose skin test converts from negative to positive. Treatment: two or more antitubercular medications given simultaneously long term, such as isoniazid (INH) and rifampin; DOTS.