RESPIRATORY SYSTEM

- Imaging Protocols
- Proper positioning
- Erect
  - 5 – 10 mm
- Horizontal beam
- Inspiration
- SID
- Exposure time
  - <10 msec
- Kvp
  - Contrast scale

Infectious Diseases
Croup

- Etiology
  - Acute viral infection causing diffuse laryngeal swelling localized to subglottic segment of trachea
- Diagnosis
  - Stridor, AP soft tissue neck x-ray
- Treatment
  - Steam, corticosteroids, intubation if life threatening
- Prognosis
- Radiologic Demonstration
  - Narrowing of subglottic segment of trachea
CROUP

• Viral infection, occurs in young children.
• Caused from inflammation of subglottic area of the trachea.
• APPEARANCE:
  – Soft tissue neck radiograph shows tapered, narrowed subglottic airway.
• TREATMENT:
  – Cool mist.
  – Steam from a hot shower @ 15-20 min intervals.
  – Corticosteroid medications in severe cases.

Pneumonia

• Etiology
  – Inflammation of the lung caused by bacteria or viruses. Produces one basic radiographic pattern.
• Types:
  • Alveolar or Air-Space
    – Focal (limited to alveoli) or Diffuse (alveoli & bronchi, lobar pneumonia)
    – Pneumococcal bacteria produces an exudate replacing air
    – Affects pulmonary segments or entire lobe (lobar) due to communication with alveolar channels
    – Common in debilitated patients

Pneumonia Types

• Bronchopneumonia
  – Staphylococcal bacteria affects segmental bronchi, can spread to alveoli

• Interstitial
  – Viruses or mycoplasma pneumoniae
  – Affects walls & lining of alveoli
  – Often diffuse and bilateral

• Aspiration
  – Inhalation of gastric fluids due to dysphagia, anesthesia, tracheostomy, esophageal obstruction, trauma, or coma
  – Common in debilitated patients
  – Affects the posterior portions of lung lobes
Pneumonia

• **Diagnosis**
  – CXR erect (PA, Lateral)

• **Treatment**
  – Antibiotic (All), Corticosteroid (Aspiration)
  – Bedrest, Hydration, Breathing exercises

• **Prognosis**
  – Dependent upon prompt treatment

Radiologic Demonstration

• **Alveolar Pneumonia**
  – Consolidation of lung with air-bronchogram sign

• **Bronchopneumonia**
  – Scattered lung consolidation
  – Atelectasis when airways are obstructed

• **Interstitial**
  – Linear or reticular appearance

• **Aspiration**
  – Patchy, diffuse densities throughout lungs
  – Posterior segments primarily affected

ALVEOLAR PNEUMONIA

• Also called air space pneumonia.
• Organism causes inflammatory exudate that replaces the air in the alveoli.
• This makes the parts of the lung that used to contain air (radiolucent) appear solid (radiopaque).
• May involve segments of an entire lobe (lobar pneumonia).
• APPEARANCE: Air-bronchogram sign caused by consolidation of the lung parenchyma.

Consolidations in both lobes of the right lung. Arrows point to air bronchograms.
BRONCHOPNEUMONIA

- Inflammation of bronchi or bronchiolar mucosa spreading to the adjacent alveoli.
- Produces small patches of consolidation.
- If the inflammation causes airway obstruction, atelectasis (lung collapse) may occur.
- APPEARANCE: Radiopaque patches of consolidation scattered throughout lungs, with air-containing lung tissue in between. No air bronchograms. If airway obstruction occurs, atelectasis will be evident.

INTERSTITIAL PNEUMONIA

- Inflammation most commonly produced by viral infections.
- Involves the alveoli and the supporting structures of the lung (alveolar septa).
- APPEARANCE: Linear pattern. Small nodular densities produced. The heart border may be obscured, creating the shaggy heart sign.

ASPIRATION PNEUMONIA

- Caused by aspiration of esophageal or gastric contents into the stomach.
  - Food
  - Liquids
  - Vomitus
- APPEARANCE: Multiple alveolar densities, distributed widely and diffusely between both lungs.
Tuberculosis

- Affects 25K persons in US annually
- Primary TB often unrecognized, incidence rate may be higher
- Spread by coughing up droplets
  - RT particulate respirator
  - Patient sputum tested to determine presence of bacillus
  - Patient is not infectious when bacillus is no longer detected in sputum

Mantoux Skin Test

<table>
<thead>
<tr>
<th>Induration</th>
<th>Interpretation</th>
<th>Analysis</th>
</tr>
</thead>
<tbody>
<tr>
<td>None</td>
<td>Negative</td>
<td>Not Infected</td>
</tr>
</tbody>
</table>
| 5-10 mm    | Positive       | Infected; at high risk for developing active TB
  - Prior active pulmonary TB
  - Contact with patient with active TB |
| 10-15 mm   | Positive       | Infected; at increased risk for developing active TB
  - Recent immigrants
  - IV drug abuser
  - TB lab personnel
  - COPD |
| >15 mm     | Positive       | Infected; healthy persons with no known TB risk factors. Unlikely to develop active TB |

TB Etiology

- Chronic bacterial infectious disease caused by Mycobacterium tuberculosis
- Primary infection (initial) results in localized lung inflammation (Gohn complex)
- 95% of cases Gohn heals spontaneously leaving calcification
- Symptoms (non-specific)
  - Low fever, mild pulmonary disease

High-power microscopic study of acid-fast stain of sputum. Cells in the background are from the bronchi and mouth.
Secondary TB

- Reactivation of dormant M.TB or reinfection
- Bacteria spread to apices of lung causing cavities
  - Cavities are source of hemoptysis
- Tissue destruction facilitates spread by:
  - Lymphatics
  - Pulmonary blood vessels
  - Airways
- Symptoms
  - Dry cough, loss of appetite, weight loss, malaise, night sweats, low-fever

The natural history of tuberculosis

Initial (primary) infection rarely progresses to disease because an effective immune response arrests most infections before they spread beyond the initial site (Ghon tubercle), or beyond the lung and a few mediastinal (hilar) lymph nodes. Some initial infections (~5%) spread quickly and widely as primary progressive tuberculosis. Primary progressive tuberculosis produces many small lesions and is called miliary (seed-like) tuberculosis. Most clinical tuberculosis is secondary TB stemming from reactivation of dormant, old infections as microbes escape from their long containment by the immune system. A few cases of secondary tuberculosis arise from a second (new) infection.

Spread of TB
TUBERCULOSIS

• **Treatment**
  – Goal to prevent drug resistance and to monitor function of kidneys, liver, eyes, ears due to drug therapy
  – Isoniazid, rifampin, pyrazinamide (combination drug therapy @ 1 year)
  – Patient usually hospitalized to minimize spread of disease, nutrition education, drug compliance

• **Prognosis**
  – Early diagnosis & treatment essential

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TB Diagnosis

• **Confirmed by CXR**

• **Radiographic Appearance**
  – **Primary TB**
    • Consolidation (Apical)
    • Hilar/lymph enlargement
    • Ghon lesion
    • Pleural effusion
  – **Secondary TB**
    • Apical, posterior segments affected most
    • Fibrotic changes with healing calcifications

---

PRIMARY TB

• **APPEARANCE:**
  – Lobar or segmental air-space consolidation that is homogenous, dense, and well defined.
  – Enlargement of hilar or mediastinal lymph nodes.
  – Focal parenchymal lesion.
  – Pleural effusion.
SECONDARY (RE_ACTIVATION) TB

- "Re-infection TB."
- TB bacterium remains inactive for many years and emerges due to a compromise in the body's immune system.
- Hilum becomes elevated.
- Heals slowly with extensive calcifications and fibrosis.
- APPEARANCE: Hazy infiltrate radiating outwards from the hilum. Hilum is usually retracted upwards. Calcifications indicate "healing."

Notice apical calcifications and upward retraction of the hilum.

RESPIRATORY SYSTEM

- Genetic/Congenital Diseases

Cystic Fibrosis - Mucoviscidosis

- Defect in the transport of chloride across the cell membrane results in a lack of NaCl in the glandular secretions of all exocrine glands
- Disorder of the exocrine glands most importantly the pancreas, intestine, and the bronchi
  - Since NaCl has an osmotic effect, those secretions contain less water and are viscid leading to obstruction of the lumen of these organs
- Abnormalities of the sweat glands are present leads to excessive sweating
Cystic Fibrosis

• **Etiology**
  – Most common autosomal recessive disease
    • Defective gene on chromosome 7
    • Affects 1 in 2500 newborns in the U.S.
    • Primarily afflicts whites, rare in other races

Cystic Fibrosis

• **Diagnosis**
  – Usually made in infancy or early childhood
  • Earliest manifestation is muconium ileus, an obstruction of the SB by viscous stool
  • Chronic cough, frequent foul smelling stool, chronic respiratory infection, weight gain due to inability to digest fat
  • Sweat Test to measure elevation of sodium and chloride
    – Loss of sodium, potassium, chloride (2-3X)

Cystic Fibrosis

• **Treatment**
  – No cure, Palliative
    • Prophylactic antibiotics
      • Deoxyribonuclease (DNase)
        » Inhalation therapy to control infections
        » Enzyme ingests DNA of bacteria and inflammatory cells
    • Chest physiotherapy
    • Bronchodilators
    • Research being conducted
      – Control production/absorption of chloride
      – Gene therapy
  • **Prognosis**
    – Early adulthood age
    – 90% morbidity due to respiratory impairment
Cystic Fibrosis

- **Radiologic Demonstration**
  - **Abdomen**
    - SB Obstruction gas/meconium
    - Bowel perforation leads to fatal peritonitis
  - **Chest**
    - Irregular thickening of linear markings throughout lungs
    - Hyperinflation
  - **CT**
    - Lung damage, disease progression

**Cystic Fibrosis Radiographic Appearance**

- Irregular thickening of markings throughout the lungs.

*Meconium ileus in cystic fibrosis.*

*Notice the small cyst-like lesions within the lungs.*

Hyaline Membrane Disease – Idiopathic RDS (Neonatal RDS)

- **Etiology**
  - Premature birth. C-Section, Diabetic mothers
  - Lungs mature during last 3 months of pregnancy
  - Lungs expand and principle components of alveoli are formed
    - In preparation for respiratory function after birth, the alveolar pneumocytes begin secreting a surfactant rich in lipids, proteins, carbohydrates (> surface tension)
    - During fetal life, the surfactant is released into the amniotic fluid that fills the fetal lungs
    - Surfactant is not required during fetal life since oxygen is delivered by the placenta
    - When fetus is born prematurely, the functionally immature lungs cannot sustain respiration; the alveoli collapse causing atelectasis due to lack of surfactant
    - Plasma proteins covering the alveolar ducts coagulate and form hyaline membranes
HYALINE MEMBRANE DISEASE

• AKA Ideopathic Respiratory Distress Syndrome (IRDS).
• Occurs primarily in premature infants from a lack of surfactant.
• APPEARANCE:
  – Granular (grainy) appearance of lungs.
  – Air bronchogram sign.
• TREATMENT:
  – Artificial surfactant given.
  – Endotracheal tube.

Arrows show the air bronchograms

IRDS

• Diagnosis
  – Respiratory evaluation at birth up to 6 hours evidenced
  – CXR
• Treatment
  – Artificial surfactant to maintain patency of alveoli
  – Positive ventilation, high O₂ delivery
• Prognosis
  – Variable due to level of oxygenation
• Radiologic Demonstration
  – Air bronchograms in atelectatic lungs

RESPIRATORY SYSTEM

• Chronic Obstructive Pulmonary Disease (COPD)
  – Multiple conditions that result in ineffective oxygen exchange
Emphysema

- Enlargement of the airspaces distal to the terminal bronchioles with destruction of the alveolar walls
- Hyperinflation of the lungs due to bronchial narrowing and loss of elasticity of alveolar walls; alveoli may collapse
  - Loss of oxygen in body
- **Etiology**
  - Cigarettes, chronic bronchitis, air pollution, chronic irritant exposure

Emphysema Pathologic Findings

- Diagnosis
  - Physical exam
    - Dyspnea on exertion, tachypnea, tachycardia, cough, barrel chest, anxiety, carbon dioxide retention weakness, anorexia
  - CXR
- **Treatment:** Palliative
  - Oxygen saturation @92%, expectorants, antibiotics
- **Prognosis**
  - Variable due to chronic condition
- **Radiologic Demonstration**
  - Pulmonary hyperinflation
  - Flattened diaphragm
  - Radiolucent retrosternal space
  - Bullae formation (air-filled spaces)
**Emphysema**

Fig 2-27 A & B. Emphysema. Frontal and lateral chest views demonstrate hyperaeration of the lungs, flattening of the bases of the hemidiaphragms, and the “Barrel chest” appearance.

**Emphysema**

Fig 2-28. Emphysematous bulla.

Fig 2-29. Emphysematous bullae (blebs) seen on a CT scan.

**Asthma**

- Common multifactorial disease characterized by constriction of the bronchi due to increased reactivity to various stimuli
- **Extrinsic**
  - Primarily affects children and is associate with allergies (50% recovery)
- **Intrinsic**
  - Non-specific factors < 40 yrs
    - Exercise
    - Stress
    - Irritants, air pollution
    - Bronchial infection
    - Heat/Cold
Asthma

- **Diagnosis**
  - Physical Exam
    - Dyspnea, wheezing when breathing caused by bronchial constriction, coughing, viscous sputum
  - CXR
- **Treatment**
  - Nebulizer, bronchodilators, allergy injections, removal of irritant, bronchomuscular spasm medication, inhaled steroids to reduce infections
- **Prognosis**
- **Radiologic Demonstration**
  - Bronchial narrowing, hyperlucent lungs during attack
  - Normal pulmonary vascular markings different from emphysema

Asthma

Frontal chest radiograph demonstrates the “dirty chest” appearance.

RESPIRATORY SYSTEM: Miscellaneous Lung Disorder

- **Atelectasis**
  - Incomplete expansion of the lung or collapse of the alveoli
  - Prevents the respiratory exchange of carbon dioxide and oxygen
Atelectasis

• **Etiology**
  – **Deficiency of surfactant**
    • Premature infants
  – **Compression of lungs**
    • Fluid in the pleural cavity
      – Transudate formed due to heart failure
      – Exudate produced due to inflammation
  – **Resorption of air distal to bronchial obstruction**
    • Foreign body, mucous (surgery or chronic bronchitis), tumor
  – **Iatrogenic**
    • ET tube placement

• **Diagnosis**
  – Physical Exam
    • Diminished breath sounds, dyspnea, fever, low O₂
  – CXR

• **Treatment**
  – Removal of causative agent, alveoli reinflate
  – Spirometry

• **Prognosis**
  – Reversible, return to normal

• **Radiologic Demonstration**
  – Radiopaque plate-like streaks

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**Fig 2-65. Atelectasis in platelike form. Arrows point to linear streaking caused from the lung collapse.**
RESPIRATORY SYSTEM

• Pleural Diseases
  – Pneumothorax
    • Air within the pleural cavity that results in partial or complete collapse of the lung
  – Etiology
    • Ruptured bulla (emphysema)
    • Spontaneous event
    • Trauma (Stabbing, fx rib, gunshot)
    • Iatrogenic (Chest tube placement)
    • Hyaline membrane disease
      – (Long-term ventilation)

Pneumothorax

• Diagnosis
  – Physical exam
    • Sudden, intense chest pain
    • Dypnea, tachypnea
    • Tachycardia, weak pulse
    • Hypotension,
    • Diaphoresis
    • Fever, Pallor, Anxiety

Pneumothorax

• Treatment
  – Minor:
    • Spontaneous absorption
  – Major:
    • Chest tube to suction air and ensure inflation
    • Oxygen administration, Fowler’s position

• Prognosis
  • Radiologic Demonstration
    – Pulmonary markings absent
    – PA Erect (Inspiration, Expiration)
    – Lateral Decubitus
Pneumothorax

Fig 2-72. Pneumothorax. The right lung is completely collapsed.

Pneumothorax

Fig 2-74. Tension pneumothorax. Notice extreme mediastinal shift to the right and complete radiolucency of the left lung field.

Pleural Effusion

- Collection of fluid in the pleural space
  - Exudate (inflammation)
  - Transudate (CHF)
- Etiology
  - Multiple pathological processes that include:
    - CHF, Pulmonary embolism, Infection (TB), Neoplasm, Surgery, Ascites
- Diagnosis
  - Physical Exam
    - Fever, Chest pain, dyspnea, non-productive cough
  - CXR
- Treatment
  - Thoracocentesis
  - Eradicate causative agent
Pleural Effusion

• Prognosis
• Radiologic Demonstration
  – Fluid level (lateral decubitus: side down)
  – Repeat images as prescribed to determine progress of fluid removal

PLEURAL EFFUSION

Arrows point to blunting of the left costophrenic angle.
Massive left sided pleural effusion causing mediastinal shift to the right.

Neoplasms

• Solitary pulmonary nodule
  – Benign or Malignant
  – Usually asymptomatic, found as incidental finding
  – < 30 yrs <1% risk of malignancy
  – 30-45 yrs @ 15% risk malignancy
  – 50 yrs @ 50% risk malignancy

  ➢ Growth rate comparisons used to determine possible classification
  ➢ Double volume <month or > 18 months benign (unreliable indicator)
  ➢ CT Scan differentiate benign/malignant tumor
  ➢ PET Scan Stage Tumor
Benign vs. malignant

Fig 2-39 and 2-40. Solitary pulmonary nodule, benign vs. malignant. The benign nodule has a sharp, well defined border. The malignant nodule has a fuzzy, blurry appearing border.

Bronchogenic Carcinoma

- Etiology
  - Cigarettes, chemicals, irritants, air pollution
- Non-small cell carcinoma / Large cell (80%)
  - Squamous cell carcinoma
    - Most common, central bronchi origination causes blockage bronchi
    - Anaplasia of respiratory squamous epithelial cells
    - Slow-growing tumor that is late to metastasize but can be associated with metastases to the bone.
  - Adenocarcinoma
    - Peripheral location which can result in pleural and thoracic wall involvement
    - Anaplasia of the ciliated and mucus cells. Also includes a more rare form of lung cancer called bronchoalveolar carcinoma (bronchiolar)
  - Large cell carcinoma (undifferentiated)
    - Poorly differentiated, highly anaplastic and aggressive tumor that grows rapidly and metastasizes early.

Bronchogenic Carcinoma

- Appearance: Can produce a broad spectrum of radiographic abnormalities.
  - Airway obstruction caused by the carcinoma can lead to atelectasis or pneumonia.
  - Unilateral enlargement of the hilum is sometimes apparent, demonstrated on serial radiography of the chest.
  - Cavitation commonly occurs in bronchogenic carcinoma.

Cavitary RUL mass with air-fluid interface.
Small Cell Carcinoma (20%)

- Anaplasia of the neuroendocrine cells
- Enlargement of hilar lymph nodes
- Frequently associated with the production of hormones
- Very aggressive and has a poor prognosis
  - It has already metastasized widely in the body (liver, bone marrow, brain, adrenal glands) in the majority of individuals by the time the diagnosis is made
  - Treatment is usually palliative rather than curative

Pulmonary Metastases

- **Solitary**
  - Solitary may be resected, 25% of cases
  - CT, PET Scan to differentiate low cellular activity (benign) or high cellular activity (malignant)
- **Hematogenous**
  - Lung Mets common due to primary tumor cell floating in blood passes through pulmonary capillaries.
  - Thoracic duct (main lymph vessel) is confluent with SVC, thus tumor cells transported in the lymph will also lodge in the lungs.
  - Vascular tumor spread from kidney, bone sarcoma, thyroid
  - Lesions scattered in lungs well-circumscribed or miliary nodules (thyroid low malignancy)
- **Lymphangitic**
  - Lymphangitic mets
  - Breast, stomach, pancreas, prostate, cervix, larynx
  - Irregular, poorly defined, coarsened

Pulmonary Metastases

![Image: Pulmonary metastases. Multiple nodules scattered throughout both lungs.](Image)
Tumor Grading & Staging

• Grading based on histology results
  – Grade I: well differentiated
  – Grade II: moderate differentiation
  – Grade III: undifferentiated
• Staging based on clinical exam, imaging studies, biopsy, or surgical exploration
  – Size of tumor, lymph mets, distant mets