Chronic Obstructive Airway Diseases

Chronic Bronchitis & Emphysema

RSPT 2310

Obstructive Airways Diseases

- Obstructive lung diseases are characterized by a variety of pathologic conditions
  - bronchial inflammation
  - excessive airway secretions
  - mucous plugging
  - bronchospasm
  - distal airway weakening
- The most common obstructive lung disorders:
  - chronic bronchitis
  - emphysema
  - asthma
- Chronic bronchitis, emphysema, and asthma may appear alone, but often appear in combination
- When chronic bronchitis and emphysema appear together as one disease complex, the patient is said to have chronic obstructive pulmonary disease (COPD)
- Although asthma can be chronic, it is usually a more acute and intermittent respiratory disorder.
- Other obstructive lung disorders include cystic fibrosis and bronchiectasis

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- Excessive airway secretions
- Mucous plugging
- Bronchospasm
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American Thoracic Society Guidelines
American Thoracic Society Guidelines

- Chronic obstructive pulmonary disease is a preventable and treatable disease state characterized by airflow limitation that is not fully reversible
  - The airflow limitation is usually progressive and is associated with an abnormal inflammatory response of the lungs to noxious particles or gases, primarily caused by cigarette smoking
  - Although COPD affects the lungs, it also produces significant systemic consequences

American Thoracic Society Guidelines

- Chronic bronchitis is defined clinically as chronic productive cough for 3 months in each of 2 successive years in a patient in whom other causes of productive chronic cough have been excluded

American Thoracic Society Guidelines

- Emphysema is defined pathologically as the presence of permanent enlargement of the airspaces distal to the terminal bronchioles, accompanied by destruction of their walls and without obvious fibrosis

Chronic Obstructive Diseases

- Chronic bronchitis and emphysema
  - Can each develop alone
  - Often occur together as one disease complex called chronic obstructive pulmonary disease
  - COPD refers to two diseases occurring concurrently
  - Patients with COPD demonstrate a variety of clinical manifestations associated with both disorders
  - The treatment of chronic bronchitis, emphysema, or a combination of both disorders (COPD) is essentially the same in the clinical setting

Anatomic Alterations of the Lungs

- Chronic Bronchitis:
  - Chronic inflammation and swelling of the wall of the peripheral airways
  - Excessive mucous production and accumulation
  - Partial or total mucous plugging of the airways
  - Smooth muscle constriction of bronchial airways (bronchospasm)
  - Air trapping and hyperinflation of alveoli—occasionally in late stages
Anatomic Alterations of the Lungs

- Chronic Bronchitis
  - The conducting airways (particularly the bronchi) are the primary structures that undergo change in chronic bronchitis.
  - As a result of chronic inflammation the bronchial walls are narrowed by vasodilation, congestion, and mucosal edema.
  - This condition is often accompanied by bronchial smooth muscle constriction.

- Emphysema
  - Distal airways, weakened in the process, collapse during expiration in response to increased intrapleural pressure, trapping gas in the alveoli.
  - Two major types of emphysema: panacinar (panlobular) emphysema and centriacinar (centrilobular) emphysema.
Anatomic Alterations of the Lungs

• Emphysema
  – The alveolar-capillary surface area is significantly decreased
  – Panlobular emphysema commonly is found in the lower parts of the lungs and often is associated with a deficiency of alpha_{1}-antitrypsin
  – Panlobular emphysema is the most severe type of emphysema and therefore the most likely to produce significant clinical manifestations

• Emphysema
  – In centriacinar emphysema, or centrilobular emphysema, the pathology involves the respiratory bronchioles in the proximal portion of the acinus
  – The respiratory bronchiolar walls enlarge, become confluent, and are then destroyed

• Emphysema
  – A rim of parenchyma remains relatively unaffected
  – Centriacinar emphysema is the most common form of emphysema and is strongly associated with cigarette smoking and with chronic bronchitis

• Emphysema
  – Major pathologic or structural changes
  • Permanent enlargement and destruction of the air spaces distal to the terminal bronchioles
  • Destruction of pulmonary capillaries
  • Weakening of the distal airways, primarily the respiratory bronchioles
  • Air trapping and hyperinflation

Etiology and Epidemiology

• Incidence of COPD
  – Precise incidence is unknown
    • Estimated that 10 to 15 million people in the United States have chronic bronchitis, emphysema, or a combination of both
    • Most authorities agree that COPD is underdiagnosed
      – If the people who have not been “officially” diagnosed with COPD are considered, the incidence is probably over 20 million people in the United States

• Incidence of COPD
  – Generally accepted that more people have chronic bronchitis than emphysema
    • National Center for Health Statistics estimates that in the United States about 9.5 million people have chronic bronchitis and 4.1 million people have emphysema

  – Annual cost related to COPD in the United States was about $37.2 billion—including $20.9 billion in direct costs, $7.4 billion in morbidity costs, and $8.9 billion in indirect costs
Etiology and Epidemiology

- COPD is the fourth leading cause of death, claiming more than 100,000 Americans each year
  - It is estimated that COPD will become the third leading cause of death by 2020
- Historically, more men than women have died from COPD each year
  - Since the year 2000, however, more women than men have died from COPD each year

Risk Factors

- COPD risk factors are related to the total burden of inhaled particles a person encounters over his or her lifetime
  - This is why we questions patients about
    - Smoking history
      - how long did they smoke/how many packs per day
      - 1 pack year = 1 ppd for 1 year
      - if they have quit smoking, when?
      - current and past employment
      - residence locales

- Risk Factors for COPD
  - Tobacco smoke
    - Includes smoke from cigarette, pipe, cigar, and other types of tobacco smoking
  - Environmental tobacco smoke
    - According to GOLD, cigarette smoking is the most commonly encountered risk factor for COPD worldwide

- Risk Factors for COPD
  - Occupational dusts and chemicals
    - Vapors, irritants, and fumes, when the exposures are sufficiently intense or prolonged
  - Indoor air pollution
    - From biomass fuel used for cooking and heating in poorly vented dwellings, a risk factor that particularly affects women in developing countries

- Risk Factors for COPD
  - Outdoor air pollution
    - Also contributes to the lungs' total burden of inhaled particles and gases (e.g., silicates, sulfur dioxide, the nitrogen oxides, and ozone)
    - Appears to have a relatively small effect in causing COPD
Risk Factors

- Risk Factors for COPD
  - Conditions that affect normal lung growth
    - Any condition that affects lung growth during gestation and childhood (e.g., low birth weight, respiratory infections) has the potential for increasing the risk of developing COPD

Risk Factors

- Risk Factors for COPD
  - Genetic predisposition (alpha1-antitrypsin deficiency)
    - In about 1 out of every 50 cases of emphysema, there is a specific hereditary basis for panlobular emphysema called alpha1 (or α1)-antitrypsin deficiency

Risk Factors

- Risk Factors for COPD
  - Genetic predisposition (α1-antitrypsin deficiency)
    - Major protein in the blood
    - Produced by the liver
    - Protects the lungs by blocking the effects of a powerful enzyme called elastase (carried by the body’s white cells to help kill invading bacteria and to neutralize small particles inhaled into the lung)

Risk Factors

- Risk Factors for COPD
  - Genetic predisposition (α1-antitrypsin deficiency)
    - The normal level of alpha1-antitrypsin is 200 to 400 mg/dL
    - Patients with normal levels of alpha1-antitrypsin are referred to genetically as having an MM phenotype or simply an M phenotype (homozygote)
    - The phenotype associated with severely low serum concentrations is the ZZ phenotype, or simply Z

Risk Factors

- Risk Factors for COPD
  - Genetic predisposition (α1-antitrypsin deficiency)
    - When old white cells are destroyed in the lungs, elastase is released
    - Under normal circumstances, α1-antitrypsin works to inactivate the released elastase
    - When the α1-antitrypsin level is low, the elastase is free to attack and destroy the elastic tissue of the lungs

Risk Factors

- Risk Factors for COPD
  - Genetic predisposition (α1-antitrypsin deficiency)
    - The heterozygous offspring of parents with the M and Z phenotypes have an MZ phenotype
    - This phenotype results in an intermediate deficiency of alpha1-antitrypsin, the precise effect which is unclear
    - It is strongly recommended, however, that individuals with this phenotype not smoke or work in areas having significant environmental air pollution
RSPT 2310
Chronic Obstructive Airway Diseases

Risk Factors
• Risk Factors for COPD
  – One other possible risk factor is the remodeling of airways that occurs in asthma
  – Not yet proven

Diagnosis
• Recommendations
  – The key indicators for considering a COPD diagnosis are as follows:
    • Over 40 years of age with
      – dyspnea
      – chronic cough
      – chronic sputum production
      – history of exposure to risk factors such as tobacco smoke

Diagnosis
• Recommendations
  – Although these indicators are not diagnostic by themselves, the presence of multiple indicators significantly increases the probability of a diagnosis of COPD
  – When multiple key indicators are present, the diagnosis of COPD should be confirmed by a pulmonary function study

Diagnosis
• The Pulmonary Function Study
  – The three main spirometry tests are used
    • Forced vital capacity (FVC)
    • Forced expiratory volume in 1 second (FEV₁)
    • Forced expiratory volume in 1 second/forced vital capacity ratio (FEV₁/FVC ratio)
  – Clinically, the FEV₁/FVC ratio is also commonly called the forced expiratory volume 1 second percentage (FEV₁%)
Diagnosis

- COPD is confirmed when both FEV\textsubscript{1} and FEV\textsubscript{1}/FVC ratio are decreased
  - A post-bronchodilator FEV\textsubscript{1} is recommended for both the diagnosis and assessment of the severity of COPD
  - The degree of spirometric abnormality usually determines the severity of COPD
  - The extent of the symptoms should also be considered when developing individualized management programs

Additional Diagnostic Studies

- In patients who are diagnosed with Stage II-IV COPD
  - Arterial blood gas measurement in patients with FEV\textsubscript{1} <50% predicted or with clinical signs suggestive of ventilatory failure or right-sided heart failure
    - the major clinical sign of ventilatory failure is cyanosis
    - clinical signs of right-sided heart failure include ankle edema and an increase in the jugular venous pressure
    - ventilatory failure is indicated by a PaO\textsubscript{2} <60 mm Hg, with or without a PaCO\textsubscript{2} >50 mm Hg while breathing room air

- Additional Diagnostic Studies
  - In patients who are diagnosed with Stage II-IV COPD
    - Bronchodilator reversibility testing to rule out a diagnosis of asthma, particularly in patients with an atypical history
      - e.g., asthma in childhood and regular nocturnal night waking with cough and wheeze
    - Chest x-ray examination is seldom diagnostic in COPD but valuable to exclude alternative and/or additional diagnoses
      - e.g., pulmonary tuberculosis, and pneumonia, and to identify comorbidities such as cardiac failure

- Alpha\textsubscript{1}-antitrypsin deficiency screening: Perform when COPD develops in patients of Caucasian descent under 45 years of age or with a strong family history of COPD.
Diagnosis

- **Type A – “Pink Puffer”**
  - In addition to the marked dyspnea and ruddy complexion, the pink puffer
    - Tends to be thin
    - Muscle wasting and weight loss due to increased WOB
    - Has a barrel chest
    - Overinflated lungs
    - Uses accessory muscles of inspiration
    - Exhales through pursed lips

- **Type B – “Blue Bloater”**
  - Term comes from the cyanosis seen chronic bronchitis
    - The pulmonary capillaries in the patient with chronic bronchitis are not damaged
    - Patients with chronic bronchitis respond to the increased airway obstruction by decreasing ventilation and increasing cardiac output
    - This decreased V/Q ratio leads to decreased PaO₂, increased PaCO₂, and a compensated (normal) pH
      - Compensated respiratory acidosis
      - Depressed respiratory drive
    - The low V/Q ratio and depressed respiratory drive both lead to a chronically reduced PaO₂ and polycythemia—which, in turn, causes cyanosis

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**Common Key Distinguishing Features Between Emphysema & Chronic Bronchitis**

<table>
<thead>
<tr>
<th>Clinical Manifestations</th>
<th>Emphysema (Type A: Pink Puffer)</th>
<th>Chronic Bronchitis (Type B: Blue Bloater)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inspection</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Body build</td>
<td>Thin</td>
<td>Stocky, overweight</td>
</tr>
<tr>
<td>Barrel Chest</td>
<td>Common—classic sign</td>
<td>Normal</td>
</tr>
<tr>
<td>Respiratory pattern</td>
<td>Hypoventilation &amp; marked dyspnea; often occurs at rest Late stage: diminished respiratory drive &amp; hyperventilation</td>
<td>Diminished respiratory drive Hyperventilation common, with resultant hypoxia and hypercapnia</td>
</tr>
<tr>
<td>Pursed-lip breathing</td>
<td>Common</td>
<td>Uncommon</td>
</tr>
<tr>
<td>Cough</td>
<td>Uncommon</td>
<td>Common—classic sign</td>
</tr>
<tr>
<td>Sputum</td>
<td>Uncommon</td>
<td>Common—classic sign, Copious amounts, purulent</td>
</tr>
</tbody>
</table>
## Common Key Distinguishing Features Between Emphysema & Chronic Bronchitis (Cont’d)

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<tr>
<th>Clinical Manifestations</th>
<th>Emphysema (Type A: Pink Puffer)</th>
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</thead>
<tbody>
<tr>
<td>Cyanosis</td>
<td>Uncommon (reddish skin)</td>
<td>Common</td>
</tr>
<tr>
<td>Peripheral edema</td>
<td>Uncommon</td>
<td>Common Right-heart failure</td>
</tr>
<tr>
<td>Neck vein distention</td>
<td>Uncommon</td>
<td>Common Right-heart failure</td>
</tr>
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### Clinical Manifestations

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<tr>
<td>Auscultation</td>
<td>Decreased breath sounds, decreased heart sounds; prolonged expiration</td>
</tr>
<tr>
<td>Percussion</td>
<td>Hyperresonance</td>
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<tbody>
<tr>
<td>Infecitons</td>
<td>Occasionally</td>
</tr>
<tr>
<td>Polycythemia</td>
<td>Uncommon</td>
</tr>
</tbody>
</table>

### Pulmonary Function Study

<table>
<thead>
<tr>
<th>Emphysema (Type A: Pink Puffer)</th>
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</tr>
</thead>
<tbody>
<tr>
<td>DL(CO) and DL(CO)/VA</td>
<td>Decrease</td>
</tr>
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### Other

<table>
<thead>
<tr>
<th>Emphysema (Type A: Pink Puffer)</th>
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<tbody>
<tr>
<td>Pulmonary hypertension</td>
<td>Uncommon</td>
</tr>
<tr>
<td>Cor pulmonale</td>
<td>Uncommon</td>
</tr>
</tbody>
</table>
Overview of the Cardiopulmonary Clinical Manifestations Associated with Chronic Bronchitis and Emphysema (COPD)

The following clinical manifestations result from the pathophysiologic mechanisms caused (or activated) by

- Excessive Bronchial Secretions
- Bronchospasm
- Distal Airway and Alveolar Weakening

Clinical Data Obtained at the Bedside

<table>
<thead>
<tr>
<th>Vital Signs</th>
<th>Chronic Bronchitis &amp; Emphysema</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate and respiratory rate</td>
<td>Stable patients: normal vital signs Exacerbations: Usually acute increase in heart rate and respiratory rate (Tachypnea) Classic sign of hypoxemia</td>
</tr>
</tbody>
</table>
### Chest Assessment Findings

<table>
<thead>
<tr>
<th>Emphysema</th>
<th>Chronic Bronchitis</th>
</tr>
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<tbody>
<tr>
<td><strong>Inspection</strong></td>
<td></td>
</tr>
<tr>
<td>General body build</td>
<td>Thin, underweight Stocky, overweight</td>
</tr>
<tr>
<td>Altered Sensorium— anxiety, irritability</td>
<td>Common—severe stage Classic sign of hypoxemia Common—during moderate and severe stage Classic sign of hypoxemia</td>
</tr>
<tr>
<td>Barrel Chest</td>
<td>Yes—classic sign Occasionally</td>
</tr>
<tr>
<td>Digital Clubbing</td>
<td>Late-stage Common</td>
</tr>
</tbody>
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### Chest Assessment Findings (Cont'd)

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<td><strong>Inspection</strong></td>
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<tr>
<td>Cyanosis</td>
<td>Uncommon—often reddish skin Common</td>
</tr>
<tr>
<td>Peripheral edema and venous distention</td>
<td>End-stage emphysema Common—Because polycythemia &amp; cor pulmonale are common, the following are often seen: Distended neck veins Pitting edema Enlarged &amp; tender liver</td>
</tr>
</tbody>
</table>

### Chest Assessment Findings (Cont'd)

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<tr>
<td><strong>Inspection</strong></td>
<td></td>
</tr>
<tr>
<td>Use of accessory muscles</td>
<td>Common Especially during exacerbations Uncommon End-stage in some chronic bronchitis</td>
</tr>
<tr>
<td>Hoover’s Sign - The inward movement of the lower lateral chest wall during each inspiration—indicates severe hyperinflation</td>
<td>Common—Severe Stage Uncommon</td>
</tr>
</tbody>
</table>

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<tr>
<td>Pursed-lip breathing</td>
<td>Common Uncommon</td>
</tr>
<tr>
<td>Cough</td>
<td>Uncommon during mild and moderate stage Some coughing during severe-stage with infection Classic sign More severe in the mornings</td>
</tr>
<tr>
<td>Sputum</td>
<td>Uncommon Little, mucoid Common Classic sign; copious amounts, purulent</td>
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<tr>
<td><strong>Inspection</strong></td>
<td></td>
</tr>
<tr>
<td>Palpation of the Chest</td>
<td>Decreased tactile fremitus Decreased chest expansion PMI often shifts to the epigastric area Normal</td>
</tr>
</tbody>
</table>

### Chest Assessment Findings (Cont'd)

<table>
<thead>
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<th>Chronic Bronchitis</th>
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<tr>
<td><strong>Inspection</strong></td>
<td></td>
</tr>
<tr>
<td>Percussion of the Chest</td>
<td>Hyperresonance Decreased diaphragmatic excursion Normal</td>
</tr>
<tr>
<td>Auscultation of the Chest</td>
<td>Diminished breath sounds Prolonged expiration Diminished heart sounds Rhonchi Crackles Wheezes</td>
</tr>
</tbody>
</table>
Clinical Data Obtained from Laboratory Tests and Special Procedures

**Pulmonary Function Test Findings**

Moderate to Severe Chronic Bronchitis & Emphysema (Obstructive Lung Pathophysiology)

<table>
<thead>
<tr>
<th>Forced Expiratory Flow Rate Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>FVC</td>
</tr>
<tr>
<td>↓</td>
</tr>
<tr>
<td>FEF₂₀₀-₁₂₀₀</td>
</tr>
<tr>
<td>↓</td>
</tr>
</tbody>
</table>

**Pulmonary Function Test Findings**

Moderate to Severe Chronic Bronchitis & Emphysema (Obstructive Lung Pathophysiology)

<table>
<thead>
<tr>
<th>Lung Volume &amp; Capacity Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>VT</td>
</tr>
<tr>
<td>N or ↑</td>
</tr>
<tr>
<td>IC</td>
</tr>
<tr>
<td>N or ↓</td>
</tr>
</tbody>
</table>

**Diffusion Capacity (DL₂CO)**

<table>
<thead>
<tr>
<th>Emphysema</th>
<th>Chronic Bronchitis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Decreased</td>
<td>Decreased DL₂CO is a classic diagnostic sign of emphysema</td>
</tr>
<tr>
<td>Normal</td>
<td></td>
</tr>
</tbody>
</table>

**Arterial Blood Gases**

Chronic Bronchitis & Emphysema

Mild to Moderate Stages

Acute Alveolar Hyperventilation with Hypoxemia

<table>
<thead>
<tr>
<th>pH</th>
<th>PaCO₂</th>
<th>HCO₃</th>
<th>PaO₂</th>
</tr>
</thead>
<tbody>
<tr>
<td>↑</td>
<td>↓</td>
<td>↓ (slightly)</td>
<td>↓</td>
</tr>
</tbody>
</table>
**Arterial Blood Gases**

**Chronic Bronchitis & Emphysema**

Chronic Ventilatory Failure with Hypoxemia

<table>
<thead>
<tr>
<th>pH</th>
<th>PaCO₂</th>
<th>HCO₃⁻</th>
<th>PaO₂</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>↓</td>
<td>↓ (significantly)</td>
<td>↓</td>
</tr>
</tbody>
</table>

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**Arterial Blood Gases**

**Chronic Bronchitis & Emphysema**

Acute Ventilatory Changes Superimposed On Chronic Ventilatory Failure

- Because acute ventilatory changes are frequently seen in patients with chronic ventilatory failure, the respiratory care practitioner must be familiar with and alert for the following:
  - Acute alveolar hyperventilation superimposed on chronic ventilatory failure
  - Acute ventilatory failure (acute hypoventilation) superimposed on chronic ventilatory failure.

---

**Oxygenation Indices**

**Chronic Bronchitis and Emphysema**

Moderate to Severe Stages

<table>
<thead>
<tr>
<th>Qₐ/Qₜ</th>
<th>DO₂</th>
<th>VO₂</th>
<th>C(a-v)O₂</th>
<th>O₂ER</th>
<th>SvO₂</th>
</tr>
</thead>
<tbody>
<tr>
<td>↑</td>
<td>↓</td>
<td>N</td>
<td>N</td>
<td>↑</td>
<td>↓</td>
</tr>
</tbody>
</table>

---

**Hemodynamic Indices**

**Chronic Bronchitis and Emphysema**

Moderate to Severe Stages

<table>
<thead>
<tr>
<th>CVP</th>
<th>RAP</th>
<th>PA</th>
<th>PCWP</th>
<th>CO</th>
<th>SV</th>
</tr>
</thead>
<tbody>
<tr>
<td>↑</td>
<td>↑</td>
<td>↑</td>
<td>N</td>
<td>N</td>
<td>N</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>SVI</th>
<th>CI</th>
<th>RVSWI</th>
<th>LVSWI</th>
<th>PVR</th>
<th>SVR</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>N</td>
<td>↑</td>
<td>N</td>
<td>↑</td>
<td>N</td>
</tr>
</tbody>
</table>

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**Laboratory Tests and Procedures**

**Emphysema**

- Hematocrit & Hemoglobin: Normal—mild moderate stage, Elevated—late stage
- Electrolytes (abnormal): Late stage: Hypochloremia (CL⁻)
  - When chronic ventilatory failure is present: Hypernatremia (Na⁺)

**Chronic Bronchitis**

- Polycythemia common during the early and late stage
- Electrolytes (abnormal): Early & Late stages: Hypochloremia (CL⁻)
  - When chronic ventilatory failure is present: Hypernatremia (Na⁺)
Chronic Obstructive Airway Diseases

<table>
<thead>
<tr>
<th>Laboratory Tests and Procedures</th>
<th>Emphysema</th>
<th>Chronic Bronchitis</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Sputum examination (culture)</strong></td>
<td>Normal</td>
<td>Streptococcus pneumoniae Haemophilus influenzae Moraxella catarrhalis</td>
</tr>
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<table>
<thead>
<tr>
<th>Radiology Findings</th>
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<tbody>
<tr>
<td><strong>Test</strong></td>
<td>Emphysema</td>
<td>Chronic Bronchitis</td>
</tr>
<tr>
<td>Chest Radiograph</td>
<td>Common</td>
<td>Lungs may be clear if only large bronchi are affected Occasionally Translucent Depressed or flattened diaphragms Common Cor pulmonale</td>
</tr>
<tr>
<td>Bronchogram</td>
<td>Small spikelike protrusions</td>
<td></td>
</tr>
</tbody>
</table>

Chronic bronchitis. Bronchogram with localized view of left hilum. Rounded collections of contrast are adjacent to bronchial walls and are particularly well demonstrated below the left main stem bronchus (arrow) in this film. They are caused by contrast in dilated mucous gland ducts.

Radiology Findings

<table>
<thead>
<tr>
<th>Test</th>
<th>Emphysema</th>
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</thead>
<tbody>
<tr>
<td>Chest Radiograph</td>
<td>Common Translucent Depressed or flattened diaphragms Long &amp; narrow heart Increased retrosternal air space Occasionally cor pulmonale</td>
</tr>
</tbody>
</table>

Chronic bronchitis. Chest x-ray film from a patient with chronic bronchitis. Note the translucent (dark) lung fields at the bases, depressed diaphragms, and long and narrow heart.

Chronic bronchitis. Chest x-ray film of a patient with emphysema. The heart often appears long and narrow as a result of being drawn downward by the descending diaphragm.
Emphysema. Lateral chest radiograph demonstrates a characteristically large retrosternal radiolucency.

General Management of COPD

• Patient and Family Education
  – Help patient/family understand the disease and its effects on the body
  – Home care therapies and administration of medications
    • Although sympathomimetic, parasympatholytic, and xanthine agents are often prescribed, these drugs are minimally effective except in special cases
    • Excessive bronchial secretions may require expectorants, mucolytics and respiratory care modalities to mobilize secretions

• General Management of COPD

  • Patient and Family Education
    – Home care therapies and administration of medications
      • ICS therapy to reduce inflammation
      • Antibiotics to treat secondary respiratory tract infections
      • Prolastin may help patients with alpha, deficiency although long-term benefits have not been demonstrated
      • Oxygen therapy
        – If hypoxemia is present oxygen is usually required
        – Reevaluate patient on oxygen to avoid eliminating patients hypoxic drive

• General Management of COPD

  • Behavioral Management
    – Avoidance of smoking and inhaled irritants (smoking cessation clinics)
    – Avoidance of infections (immunizations, pneumococcal vaccine)
    – Encourage physician and patient involvement in a pulmonary rehabilitation, if available

• General Management of COPD

  • RC Treatment Protocols
    – Oxygen Therapy Protocol
    – Bronchial Hygiene Protocol
    – Aerosolized Medication Protocol
    – Mechanical Ventilation Protocol

GOLD STANDARDS

<table>
<thead>
<tr>
<th>Global Initiative for Chronic Obstructive Lung Disease</th>
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<tbody>
<tr>
<td>G I lobal Initiative for Chronic</td>
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<td>O bstructive</td>
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<td>L ung</td>
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<td>D isease</td>
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</table>
Components of Care: A COPD Management Program

The goals of COPD management include:
- Relieve symptoms
- Prevent disease progression
- Improve exercise tolerance
- Improve health status
- Prevent and treat complications
- Prevent and treat exacerbations
- Reduce mortality
- Prevent or minimize side effects from treatment

Four Components

- Assess and Monitor Disease
- Reduce Risk Factors
- Manage Stable COPD
- Manage Exacerbations

Component 1: Assess and Monitor

- Exposure to risk factors, including intensity and duration
- Past medical history, including asthma, allergy, sinusitis or nasal polyps, respiratory infections in childhood, and other respiratory diseases
- Family history of COPD or other chronic respiratory disease
- Pattern of symptom development
- History of exacerbations or previous hospitalizations for respiratory disorder.

Component 2: Reduce Risk Factors

- Counseling to quit smoking
- Pharmacotherapy
- Smoking prevention
- Occupational exposures
- Indoor and outdoor air pollution

Component 1: Assess and Monitor

- Presence of comorbidities, such as obesity, heart disease, malignancies, osteoporosis, and musculoskeletal disorders, which may also contribute to restriction of activity.
- Appropriateness of current medical treatments.

Component 1: Assess and Monitor

- Impact of disease on patient’s life, including limitation of activity; missed work and economic impact; effect on family routines; and feelings of depression or anxiety.
- Social and family support available to the patient.
- Possibilities for reducing risk factors, especially smoking cessation.
### Strategy to Help a Patient Quit Smoking

- Ask—Systematically identify all tobacco users at every visit.
- Advise—Strongly urge all tobacco users to quit.
- Assess—Determine willingness to make a quit attempt.
- Assist—Aid the patient in quitting.
- Arrange—Schedule follow-up contact.

### Component 3: Manage Stable COPD

Management of stable COPD should be guided by the following general principles:

- Determine disease severity
- Implement a stepwise treatment plan that reflects this assessment of disease severity
- Choose treatments according to national and cultural preferences, the patient’s skills and preferences, and local availability of medications

#### Component 3: Manage Stable COPD

- **Patient education**

#### Component 3: Manage Stable COPD

- **Pharmacologic treatments**
  - β₂-agonists
    - Short-acting
      - Fenoterol
      - Levalbuterol
      - Salbutamol
      - Terbutaline
  - Long-acting
    - Formoterol
    - Saleterol
    - Anticholinergics
      - Short-acting
        - Ipratropium bromide
        - Oxitropium bromide
      - Long-acting
        - Tiotropium

- **Combination short-acting β₂-agonists plus anticholinergic in one inhaler**
  - Fenoterol/ipratropium
  - Oxitropium bromide
### Component 3: Manage Stable COPD

- Methylxanthines
  - Aminophylline
  - Theophylline

### Component 3: Manage Stable COPD

- Inhaled glucocorticosteroids
  - Beclomethasone
  - Budesonide
  - Fluticasone
  - Triamcinolone

### Component 3: Manage Stable COPD

- Combination long-acting β₂-agonists plus glucocorticosteroids in one inhaler
  - Formoterol/Budesonide
  - Salmeterol/Fluticasone

### Component 3: Manage Stable COPD

- Systemic glucocorticosteroids
  - Prednisone
  - Methylprednisolone

### Component 3: Manage Stable COPD

- Glucocorticosteroids
- Vaccines
- Antibiotics
- Mucolytic
- Antitussives
- Non-pharmacologic treatment
- Rehabilitation
- Oxygen therapy
- Surgical treatment
## Component 4: Manage Exacerbation

- Exacerbation of COPD is defined as an event in the natural course of the disease characterized by a change in the patient’s baseline dyspnea, cough, and/or sputum that is beyond normal-day-to-day variations, is acute in onset, and may warrant a change in regular medication in a patient with underlying COPD.

## Additional Treatment Considerations for Emphysema

- Alpha 1 antitrypsin therapy
- Lung volume reduction surgery
- Lung transplantation