Chronic Obstructive Pulmonary Disease (COPD)

- COPD is a combination of emphysema and chronic bronchitis. It is a preventable and treatable condition.
- It is characterized by restricted airflow that gets progressively worse.
- COPD is the most common lung disease in the world and is the fourth leading cause of death globally.

Risk Factors

- Smoking is #1 risk factor
- Occupational dusts and chemicals
- Air pollution
- Lung growth limitations
- Genetic abnormalities with alpha 1-antitrypsin deficiency (this gene inhibits proteolysis in lung tissue)

<table>
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<tr>
<th>PATHOPHYSIOLOGY</th>
<th>SYMPTOMS</th>
<th>MANAGEMENT</th>
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<tbody>
<tr>
<td>Tobacco smoke</td>
<td>Inflammation of the airway epithelium</td>
<td>Inhibition of normal endogenous antiproteases</td>
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<tr>
<td>Air pollution</td>
<td>Infiltration of inflammatory cells and release of cytokines (neutrophils, macrophages, lymphocytes, interleukins)</td>
<td>Increased protease activity with breakdown of elastin in connective tissue of lungs (elastases, cathepsins, etc.)</td>
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<tr>
<td>Systemic effects (muscle weakness, weight loss)</td>
<td>Continuous bronchial irritation and inflammation</td>
<td>Chronic bronchitis (bronchial edema, hypersecretion of mucus, bacterial colonization of airways)</td>
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<tr>
<td>Inhaled irritants and chemicals</td>
<td>Airway obstruction</td>
<td>Emphysema (destruction of alveolar septa and loss of elastic recoil of bronchial walls)</td>
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<tr>
<td>Inhaled irritants and chemicals</td>
<td>Loss of surface area for gas exchange</td>
<td>Dyspnea</td>
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<tr>
<td>Inhaled irritants and chemicals</td>
<td>Frequent exacerbations (infections, bronchoconstriction)</td>
<td>Cough</td>
</tr>
<tr>
<td>Inhaled irritants and chemicals</td>
<td></td>
<td>Hypoxemia</td>
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<tr>
<td>Inhaled irritants and chemicals</td>
<td></td>
<td>Hypercapnia</td>
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<tr>
<td>Inhaled irritants and chemicals</td>
<td></td>
<td>Cor pulmonale</td>
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**Chronic Bronchitis**
- Is hyper secretion of mucus with a chronic productive cough for a minimum of 3 months occurring for at least two years.
- Chronic inflammation of the bronchi leads to bronchial edema from an increase in the number and size of mucous glands and goblet cells in the airway. An increase in the size and number of cells causes the airway to become more narrow. Ciliary functions impaired and tenacious mucus cannot be expelled.
- Chronic bronchitis increases risk for infections and ineffective airway repair.
- Eventually the obstruction of bronchi will lead to ventilation – perfusion mismatch with hypoxemia.
- Hyperinflation is a result from gas being trapped in the distal portions of the lungs from early collapse of the airways during expiration.
- Manifestations: hypoventilation, hypercapnia, and decreased tidal volume

**Emphysema**
- Emphysema occurs from acini (gas-exchange airways) growing in size along with alveolar walls being destroyed. Fibrosis will not be obvious in emphysema.
- Loss of elastic recoil is the main mechanisms of airflow limitation.
- As alveolar are destroyed blebs of air are produced in the lung parenchyma which are not effective in gas exchange.
- Air trapping causes hyper expansion off the chest placing the muscles of respiration at a mechanical disadvantage. Late side effects of air trading are hypoventilation and hypercapnia.
- Pulmonary hypertension and cor pulmonale result from destruction of pulmonary capillaries and alveolar walls.

<table>
<thead>
<tr>
<th>Clinical Manifestations</th>
<th>Bronchitis</th>
<th>Emphysema</th>
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<tbody>
<tr>
<td>Productive cough</td>
<td>Classic</td>
<td>With infection</td>
</tr>
<tr>
<td>Dyspnea</td>
<td>Late in course</td>
<td>Common</td>
</tr>
<tr>
<td>Wheezing</td>
<td>Intermittent</td>
<td>Common</td>
</tr>
<tr>
<td>History of smoking</td>
<td>Common</td>
<td>Common</td>
</tr>
<tr>
<td>Barrel chest</td>
<td>Occasionally</td>
<td>Classic</td>
</tr>
<tr>
<td>Prolonged expiration</td>
<td>Always present</td>
<td>Always present</td>
</tr>
<tr>
<td>Cyanosis</td>
<td>Common</td>
<td>Uncommon</td>
</tr>
<tr>
<td>Chronic hypoventilation</td>
<td>Common</td>
<td>Late in course</td>
</tr>
<tr>
<td>Polycythemia</td>
<td>Common</td>
<td>Late in course</td>
</tr>
<tr>
<td>Cor pulmonale</td>
<td>Common</td>
<td>Late in course</td>
</tr>
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</table>
Nurses Role

Interventions

- **Assessment of respiratory system:** vital sounds, RR sound and pattern, present and past medical history, risk factors

- **Achieve airway clearance:** to decrease O2 demands
  - Administer bronchodilators and corticosteroids
  - Direct / controlled coughing to reduce fatigue

- **Improve breathing pattern:**
  - Inspiratory muscle training
  - Diaphragmatic breathing to reduce RR, increase alveolar ventilation and expel max amount of air during expiration
  - Pursed lip breathing to slow expiration

- **Improve activity intolerance:**
  - Manage daily activities; must be paced throughout the day
  - Exercise training to help strengthen muscles to improve tolerance and endurance
  - Walking aids to improve ambulation

- **Monitor for complications:**
  - Monitor cognitive changes
  - Monitor pulse oximetry levels (if inaccurate always get ABG)
  - Prevent infection!! GET FLU SHOT

Nursing Priorities

- 1: Maintain patent airway
- 2: Assist with measure to facilitate gas exchange
- 3: Enhance nutritional intake
- 4: Prevent complications, slow progression of condition
- 5: Provide information about disease process, prognosis and treatment regimen
**TREATMENT**

**Leukotriene Modifiers**
- zafirlukast, montelukast
  - Antagonizes the effects of leukotrienes
  - Monitor liver function
  - S/E: suicidal thoughts and headache

**Leukotriene Synthesis Inhibitors**
- zileuton
  - Inhibits enzyme 5-lipoxygenase that catalyses formation of leukotrienes
  - Monitor liver function
  - S/E: suicidal thoughts and headache

**Glucocorticoids**
- Suppresses inflammation and the normal immune response
- S/E: hypertension, peptic ulcers, anorexia, nausea, acne, decreased wound healing, ecchymoses, fragility, hirsutism, petaechiae, adrenal suppression, hyperglycemia, muscle wasting, osteoporosis

**Glucocorticoids + Beta2 Agonist**
- fluticasone propionate + (salmetrol or vilanterol)
  - Caution with CPY34A inhibitors
  - S/E: pneumonia, hypokalemia
  - If patient is allergic to milk may be allergic to fluticasone vilanterol

**Mast Cell Stabilizer**
- cromolyn sodium, nedocromil sodium
  - Prevents release of histamine and SRS-A (slow reacting substance of anaphylaxis) from sensitized mast cells
  - S/E: nasal irritation with inhalation, unpleasant taste
  - PO cromolyn: mix in glass of water & administer 30 minutes before meals and at bedtime

**Treatment**
- Bronchodilators to open airways
- Glucocorticoids to decrease inflammation
- Leukotriene modifiers to reduce lung inflammation
- Expectorants to loosen mucus from airways
- Antibiotics to prevent further infections

**Clinical Management**
- Implementing Collaborative Care:
  - Teaching!! >>> Medication compliance and exacerbation prevention
  - Major interventions include smoking cessation, drug therapy, and oxygen

- Nutrition Management: increase calories with focus on protein and fat. Will need more fat than carbs (carbs produce carbon dioxide which could exacerbate illness).
  - Meals should be small and frequent
    - Weight loss is a bad idea because every body percent they lose their mortality is increased by 10%
    - Avoid or limit milk products
    - Fluid intake of at least 2-3 L / day

- Monitoring breathing pattern: very important, as respiratory is the first system that will show symptoms of inadequate perfusion.
  - Breathing retraining (pursed lip and abdominal breathing), effective coughing, chest physiotherapy, home nebulizers

**References:**