

Preconference Form

Student Name: Lillian Maslauskas

Medical Diagnosis/Disease: COPD

NCLEX IV (8): Physiological Integrity/Physiological Adaptation

Anatomy and Physiology

Normal Structures

- R lung has 3 lobes (upper, middle, and lower) and L lung has 2 lobes (Upper and lower)

Upper Respiratory: Nose, mouth, pharynx, epiglottis, larynx, trachea.

Nose: Air enters through nose. Nose has 3 passages called turbinate's in which helps increase the surface area of nasal mucosa that warms and moistens air entering the nose. The nasal cavity is connected to your *pharynx* and then split into nasopharynx, oropharynx, and laryngopharynx. Your olfactory nerve, which is found in the mucosa is responsible for sense of smell. Air then moves through your oropharynx to your laryngopharynx. Air travels through epiglottis to *larynx* before moving into the *trachea*. **Epiglottis:** Small flap behind tongue that closes the larynx during swallowing. **Trachea:** Divides into right and left main bronchi at carina point, which is at the 4th and 5th thoracic vertebrae. Stimulation in this area causes vigorous cough.

Lower Respiratory: Bronchi, bronchioles, alveolar ducts and alveoli.

Bronchi: Along with pulmonary vessels and nerves enter the lungs through hilus(slit). There the R main stem bronchus is shorter than the left bronchus, this is why aspiration occurs more on the R lung. *Pathway to conduct gases to and from alveoli, as well as*

Pathophysiology of Disease

Chronic Obstructive Pulmonary Disease.

- Progressive lung disease characterized by persistent airflow limitation and associate with an enhanced, chronic inflammatory response in the airways and lungs.
- Lung parenchyma is a chronic inflammation of the airways in the bronchioles, alveoli and pulmonary blood vessels.
- Cigarette smoking and other noxious particles and gases entering lungs. Smoking speeds up the disease process.
- Classified as mild, moderate, and severe. Can be fatal.
- Wheezing lung sounds.
- It is slightly more common in men due to having poorer response to O₂ therapy. Whereas in women the prevalence of COPD is increasing due to more women starting to smoke and an increased susceptibility due to have smaller lungs and airways. If women get COPD they have a lower quality of life.
- Results in a V/Q mismatch.

*It is a loss of elastic recoil and airflow obstruction from mucus hypersecretion, mucosal edema, and bronchospasm. As the disease progresses airflow limitation, air trapping and gas exchange worsen. In severe COPD pulmonary hypertension and systemic manifestations occur. COPD can severely impair and destroy areas of the lung tissue.

Inflammatory process: Often starts with inhalation of noxious particles and gases,

NCLEX IV (7): Reduction of Risk

Anticipated Diagnostics

Labs

- **ABG**
- Sputum culture
- Blood culture
- CBC
- AAT (Alpha-1 antitrypsin deficiency test)

Additional Diagnostics

- **CXR**
- COPD Assessment Test (CAT)
- Clinical COPD Questionnaire (CCQ)
- 6 minute walk test
- Spirometry

the trachea.

Bronchioles : Smooth muscle that constricts and dilates.

Alveoli: Small sacs in the lungs that are the primary site of gas exchange for O₂ and CO₂.

Alveoli cells secrete surfactant which is a lipoprotein that lowers surface tension in alveoli because they are fragile. It also reduces the pressure needed to inflate the alveoli so they are less likely to collapse.

***Diffusion: Process of alveoli taking CO₂ out.**

Blood Supply: Pulmonary and bronchial circulation.

Pulmonary: Provides lungs with blood that takes part in gas exchange. Pulmonary artery then receives deoxygenated blood from R ventricle and delivers the pulmonary capillaries. O₂-CO₂ occurs. Pulmonary veins then return oxygenated blood to L atrium into L ventricle.

Bronchial: Does not take part in gas exchange. Bronchial arteries arise from thoracic aorta. Provides O₂ to bronchi.

Chest Wall: Lined with parietal pleura and the lungs are lined with visceral pleura. They both work together to form one continuous membrane. The intrapleural space is the space between pleural layers. They slide over each other when breathing, allowing expansion.

**Medulla oblongata controls breathing.*

Oxygenation: Process of obtaining O₂ from atmosphere air and making it available to organs and tissues in the body. O₂ and CO₂ move back and forth across alveolar capillary membrane by diffusion (overall direction of movement is from

such a cigarette smoke. With repeated exposure to this chronic inflammation causes tissue destruction and disrupts the normal defense mechanisms of the lungs. The predominant inflammatory cells in COPD are neutrophils, macrophages, and lymphocytes. The inflammatory process may be increased by the oxidants made by cigarette smoke and other inhaled particles and then released from the inflammatory cells. Oxidants affect the lungs as they inactivate antiproteases (inhibit protease function, which are enzymes that break down protein and prevent the natural destruction of the lungs), stimulate mucus secretion, and increase fluid in the lungs. A result of the inflammatory process is structural change in the lungs.

*Inability to expire air is a main characteristic of COPD. The main site of airflow limitation is in the smaller airways so as the peripheral airways become obstructed, air is progressively trapped during expiration. As air is trapped in the lungs the chest hyper-expands and becomes barrel shaped (very round and large chest) because the respiratory muscles cannot function effectively. This leads to a loss of elastic recoil and makes passive expiration of air very difficult. As a result of this the patient will become dyspneic and will limit exercise capacity, this could lead to hypoxemia later into the disease.

area of high concentration to low). O₂ moves from alveolar gas into arterial blood into the alveolar gas.

NCLEX II (3): Health Promotion and Maintenance

Contributing Risk Factors

- Cigarette smoker
- Chronic smoker
- Chronic bronchitis
- Emphysema
- Asthma
- AATD- Alpha-1 antitrypsin deficiency (Autosomal recessive disorder that effects lungs and liver. Disorder in protection of lung tissue from proteases during inflammation related to smoking and infections).
- Infection in respiratory tract
- Exposure to air pollution
- Aging
- Exposure to chemicals

Signs and Symptoms

- Chronic cough (1st S/S)
- Sputum production
- Dyspnea
- Chest tightness
- Decreased activity
- Fatigue
- SOB
-

NCLEX IV (7): Reduction of Risk

Possible Therapeutic Procedures

Non-surgical

- IS
- O₂
- Breathing exercises
- HOB elevated
- Nutritional supplements

Surgical

- Lung volume reduction
- Bullectomy (removal of air pockets or bulla)
- Lung transplant

Prevention of Complications

(What are some potential complications associated with this disease process)

- Lung infection
- Pulmonary hypertension
- Acute exacerbations (worsen of a disease)
- Bacterial infections
- Collapsed lung



NCLEX IV (6): Pharmacological and Parenteral Therapies

- Anticipated Medication Management
- Anticholinergics
 - Acetaminophen
 - Corticosteroids
 - Methylxanthines
 - Ceftriaxone
 - O2

NCLEX IV (5): Basic Care and Comfort

- Non-Pharmacologic Care Measures
- Cough and deep breathing exercises
 - IS usage
 - Ambulation
 - Exercise plan for walking and upper body strengthen
 - Hydration

NCLEX III (4): Psychosocial/Holistic Care Needs

- What stressors might a patient with this diagnosis be experiencing?
- Fear of not being able to breath
 - Fear of no longer breathing
 - Fear of a collapsed lung
 - Financial expenses from hospital stay
 - Fear of dying

Client/Family Education

- List 3 potential teaching topics/areas
- If a smoker. Teach about the importance of stopping and the risk factors along with smoking.
 - IS usage
 - Cough and deep breathing exercises and the importance of them.

NCLEX I (1): Safe and Effective Care Environment

- Multidisciplinary Team Involvement
(Which other disciplines do you expect to share in the care of this patient)
- Respiratory Therapist
 - Pulmonologist
 - Nurses
 - Chaplin
 - Nutritionist
 - Palliative care team
 - PT
 - Thoracic surgeon