

## Preconference Form

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Medical Diagnosis/Disease: Chronic obstructive pulmonary disease (COPD) exacerbation

### NCLEX IV (8): Physiological Integrity/Physiological Adaptation

#### Anatomy and Physiology

##### Normal Structures

Main role: Gas exchange, supplying  $O_2$  and removing  $CO_2$ . Upper respiratory: Nose and nasal cavity: air enters through the nose where it is warmed, humidified, and filtered. The nasal cavity contains the mucosa and cilia that trap foreign particles. The pharynx has 3 parts: nasopharynx, oropharynx, & laryngopharynx, it serves as a pathway for food and air. Larynx: contains vocal cords that are responsible for sound production, epiglottis prevents food and liquids from entering the airway when swallowing. Lower: trachea & bronchi: the trachea divides into the right and left mainstream bronchi at the carina, which then divides into smaller bronchi and bronchioles that carry air into the lungs. The bronchioles are divided into alveoli. The alveoli are small sacs within the parenchyma (tissue that supports the lungs) where gas exchange occurs with diffusion across the alveolar capillary membrane and facilitates oxygen entering the blood and  $CO_2$  being expelled. The lungs are split into the right and left. The right has 3 lobes and the left has 2. The lungs are surrounded by the pleura, a double layered membrane that provides lubrication and allows for smooth expansion and contraction. The mechanism of breathing has two parts: Inspiration: the diaphragm (muscle for breathing) contracts and flattens increasing the chest cavity volume, that reduces pressure in the lungs and draws air in. The intercostal muscles also help with this. Expiration: A process where the diaphragm relaxes decreasing chest cavity volume, and forces air out. Breathing is controlled by the medulla and pons in the brainstem that respond to changes in blood  $CO_2$ ,  $O_2$  and PH levels. If carbon dioxide rises the brain tells the body to breathe faster and deeper to get rid of it. Chemoreceptors in the brain and carotid arteries are key in regulating respiratory rate and depth. 2

#### Pathophysiology of Disease

Progressive lung disease is characterized by persistent airflow limitation and a chronic inflammation of the airways, respiratory bronchioles (carry oxygen to alveoli) and alveoli (primary site for gas exchange), and pulmonary blood vessels. The main cause is the loss of elastic recoil (ability of lung tissue to return to shape after being stretched for inhalation) and airflow obstruction, from mucus hypersecretion, mucosal edema (swelling of mucous membranes), and bronchospasm (tightening of the muscles in airway). In severe COPD pulmonary hypertension and systemic manifestations occur. Severely impaired or destroyed areas of lung tissue can exist. The inflammatory process mostly starts with inhaling toxic particles/gases (cigarette smoke), and with repeated exposure, chronic inflammation causes tissue destruction and disrupts the normal defense mechanisms and repair process of the lungs. The inflammatory cells in COPD are neutrophils, macrophages, and lymphocytes (all white blood cells to fight infection). Inflammatory process may be increased by oxidants from cigarettes and other inhaled particles, these affect the lungs by inactivating antiproteases (protect lungs from destruction), stimulate mucus secretion and increase fluid in the lungs. This can result in structural changes. After inhaling toxins proteases increase and antiproteases are inhibited leading to alveolar destruction and loss of elastic recoil. Inability to expire air is the main characteristic of COPD. Main site of airflow limitation is in the smaller airways. As peripheral airways become obstructed air is trapped during expiration, as this happens the chest hyper expands and becomes barrel shaped because the respiratory muscles cannot function effectively. As a result, the patient has to inhale when the lungs are overinflated and becomes dyspneic. Hypoxemia (low levels of Oxygen in blood) occurs usually late in the disease, at first with

### NCLEX IV (7): Reduction of Risk

#### Anticipated Diagnostics

##### Labs:

ABG's (Arterial blood gases) (tell severity of exacerbations by assessing abnormal oxygenation). ( $PCO_2$  35-45) ( $HCO_3$  21-28) (pH 7.35-7.45)  
Sputum (for infection) (normal epithelial cells)  
Serum antitrypsin levels

##### Additional Diagnostics

History and physical assessment  
Chest X ray (flat diaphragm)  
6-min walk test (tell  $O_2\%$  for therapy)  
Spirometry

Circulations: Pulmonary carries deoxygenated blood from the heart to the lungs for gas exchange and returns oxygenated blood back to the heart. Bronchial nourishes the lung tissues but does not participate in gas exchange. The respiratory system has several defenses: nasal hairs, cilia, mucus and cough reflex. These all work to trap and kill foreign particles to fight off infection. Age related: reduced lung elasticity, weaker respiratory muscles, decreased alveolar surface area, which all may impair gas exchange and increase susceptibility to infections.

exercise but then gas exchange problems result in hypoxemia and hypercapnia (increased CO<sub>2</sub>) as the disease gets worse. As air trapping increases, walls of alveoli are destroyed and bullae (large air spaces in parenchyma) and blebs (air spaces next to pleurae (membrane that surrounds the lungs)) can form in and on the lungs. These are not effective in gas exchange because do not contain a capillary that normally surrounds alveolus resulting in hypoxemia, and airway obstruction and CO<sub>2</sub> retention. It can also have pulmonary vascular changes in late COPD, where small pulmonary arteries vasoconstrict due to hypoxia (deficiency of oxygen reaching tissues of the body). The structure of pulmonary arteries change, and smooth vascular muscles thicken causing pulmonary circulation increase. Pulmonary hypertension may progress and lead to hypertrophy of the right ventricle which can dilate and lead to HF. Classification: Based off FEV<sub>1</sub> (how much air a person can exhale in the 1<sup>st</sup> second of breath)

**NCLEX II (3): Health Promotion and Maintenance**

Contributing Risk Factors  
Cigarette smoking, environmental factors like pollutants from job, recurring respiratory infections, physical inactivity, genetics, age, gender (men), history of asthma

Signs and Symptoms  
Chronic cough (productive or not), sputum production, dyspnea (shortness of breath), wheezing, chest tightness, fatigue from work of breathing, weight loss, barrel chest, cyanosis (discoloration of lips or fingers), low o<sub>2</sub> levels

**NCLEX IV (7): Reduction of Risk**

Possible Therapeutic Procedures  
Non-surgical  
Medications, o<sub>2</sub> therapy, respiratory care like breathing exercises, nutrition therapy, smoking cessation support, exercise plan, immunizations, good hydration  
Surgical  
Lung volume reduction surgery to reduce size of lungs by removing diseased parts. Bronchoscopy lung volume reduction  
Bullectomy

Prevention of Complications  
(What are some potential complications associated with this disease process)  
Impaired respiratory function  
Activity intolerance  
Nutritionally compromised  
Ineffective coping  
Sleeping disorders  
Osteoporosis (decreased activity)  
Lung cancer  
Pneumonia  
Pulmonary hypertension

#### NCLEX IV (6): Pharmacological and Parenteral Therapies

##### Anticipated Medication Management

**Bronchodilator drugs** like B2-adrenergic agonists (short or long acting), anticholinergics and methylxanthines.  
Roflumilast (anti-inflammatory drug)  
Triple therapy agents

#### NCLEX IV (5): Basic Care and Comfort

##### Non-Pharmacologic Care Measures

Smoking cessation, breathing techniques, education on COPD and self-managing strategies, addressing anxiety, positioning upright to improve gas exchange, spirometry for deep breathing, explain energy conservation, encourage rest.

#### NCLEX III (4): Psychosocial/Holistic Care Needs

##### What stressors might a patient with this diagnosis be experiencing?

Physical limitations from dyspnea and fatigue, fear of progression, loss of independence, financial concerns, social isolation, anxiety and depression, need to avoid work if working in a toxic pollutant environment, uncertainty, family role

#### Client/Family Education

##### List 3 potential teaching topics/areas

- Healthy nutrition and eating habits**
- Avoid smoking
  
- Importance of breathing exercises and techniques**

#### 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)

Pulmonologist, respiratory therapist, nutritionist, physical therapist, psychologist, social worker, pharmacist, chaplains/spiritual support