Preconference Form

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Medical Diagnosis/Disease: COPD

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

NCLEX IV *(7)*: Reduction of Risk

Anatomy and Physiology

Normal Structures

-The upper respiratory tract contains the nose, mouth, pharynx, epiglottis, larynx, and trachea.
-Air initially enters the upper respiratory tract through the nose which is made of bone/cartilage and is divided into two nares by the nasal septum.
-The nose warms/humidifies the air that we breathe in and filters small particles out before the air reaches the lungs.
-The nasal cavity connects with the pharynx which is divided into three parts which are the nasopharynx, oropharynx, and laryngopharynx.
-Air moves through the oropharynx → laryngopharynx → epiglottis → larynx → trachea.
-Epiglottis: a small flap that covers the larynx to prevent aspiration of food/ liquid into the lungs.
-The trachea divides into the right and left mainstem bronchi at a spot called the carina, which is highly sensitive.
-Once air is through the carina it is now in the lower respiratory tract which consists of bronchi, bronchioles, alveolar ducts, and alveoli.
-Right lung has three lobes (upper- middle-lower)
-Left lung has only two lobes (upper-lower) to accommodate for the heart.
-The mainstem bronchi divides to form the lobar, segmental, and subsegmental bronchi. Which then divide even more to form the bronchioles.
-The respiratory bronchioles are the most distant and they consist of smooth muscles that constrict and dilate as a response to stimuli.
-After the bronchioles there are the alveolar ducts in alveoli.
-The trachea and bronchi are the passageway for gases to go to and from the alveoli. The air in the trachea and bronchi is known as dead

Pathophysiology of Disease

-COPD stands for chronic obstructive pulmonary disease.
-It is a progressive lung disease shown by persistent air flow limitation. It is associated with a chronic inflammatory response in the airways and lungs.
-There are many risk factors for COPD including cigarette smoking which is the leading cause, infection like severe recurring respiratory tract infections, asthma, air pollution, occupational chemicals and dust, aging, and genetics.
-Of the 16 million people in the US have COPD around 38% report that they currently smoke.
- COPD is a high risk for any person who smokes over the age of 40 or has a history of smoking 10 or more pack-years. -The defining feature of COPD is air flow limitation not fully reversible during forced exhalation. The main cause of this is the loss of elastic recoil an air flow obstruction.
-The predominant inflammatory cells in COPD are neutrophils, macrophages, and lymphocytes.
-The inability to expire air is the main characteristic of COPD as the peripheral airways become obstructed, air is trapped during expiration.
-COPD is shown by the chest hyper expanding and becoming barrel shaped due to the respiratory muscle not functioning properly. This increases functional residual capacity.
-Residual air combined with the loss of elastic recoil causes passive expiration to be extremely difficult. This causes the patient to have to inhale when the lungs are already over inflated causing them to become dystonic and have limited exercise capacity.
-Hypoxemia at rest usually isn't until late in the disease but at first hypoxemia can develop during exercise and the patient may benefit from supplemental O2.
-Hypoxemia and hypercapnia may become an issue as the disease worsens.
-As the trapping of air in the lungs increases

Anticipated Diagnostics

Labs

-ABG
-CBC
-BMP

Additional Diagnostics

-Chest X-ray
-ECG
-H & P
-Sputum Test
-6 minute walk test/exercise testing
-PFT's
-Pulse Oximetry
-Genetic testing
-Spirometry
-CT scan

space as this air does not take part in gas exchange.
-Tidal volume: total volume of air exchanged with each breath. In an adult the normal tidal volume is about 500 milliliters with 150 being dead space.
-The alveoli are the final piece of the respiratory tract; they are small sacs in the lungs that are the primary site of gas exchange between O2 and CO2.
-In the alveoli there are pores of Kohn, which connect all the alveoli and allow air flow between alveolus and alveolus.
-Deep breathing increases air movement through these pores and helps move mucus out of the respiratory bronchioles which is where the incentive spirometer comes in.
-Alveoli have a total volume of about 2500 milliliters with a large surface area for gas exchange.
-In the alveolar there is a liquid called surfactant which lowers the surface tension in the alveoli. Surfactant reduces the amount of pressure needed to inflate the alveoli and makes them less likely to collapse. When a person takes a large breath like a sigh this stretches the alveoli and promotes the secretion of surfactant. Without surfactant or when there is not enough surfactant the alveoli can collapse which is termed atelectasis. Many postoperative patients are at risk of this because of anesthesia and decreased mobility which decreases lung expansion and decreases the secretion of surfactant.
-In the lungs there are two types of circulation (pulmonary and bronchial).
-Pulmonary circulation provides the lungs with blood that is used in gas exchange.
-Bronchial circulation does not take part in gas exchange but does provide oxygen to the bronchi and other lung tissues.
-Diaphragm: major muscle of respiration. During inspiration it contracts, moves downwards, and increases intrathoracic volume.
-Oxygenation is the process of attaining oxygen from the atmospheric air and making it available to the organs and tissues.
-Oxygen is carried in the blood as dissolved oxygen and hemoglobin bound oxygen. PAO2

the walls of the alveoli are destroyed means bullae and blebs (air spaces) can form in or on the lungs.
-COPD is classified as either mild, moderate, severe, and very severe.
-The severity of the obstruction is what determines the stage of COPD and this determines how the COPD is managed.
-COPD should be considered an any patient who has a chronic cough, sputum production, dyspnea, and a history of exposure to risk factors like smoking.
-Chronic intermittent cough is usually the first symptom, but this is usually dismissed as patients associate this with the smoking or environmental exposure they are experiencing. There may be reports of chest heaviness, not being able to take a deep breath, gasping, increased effort to breathe, and air hunger. Patients commonly ignore these symptoms and assume that they are a sign of getting older or being out of shape.
-Patients usually seek medical care when the dyspnea becomes so severe that it impairs the ability to complete their activities of daily living.
-Advanced COPD often comes with fatigue, weight loss, and anorexia. You may hear decreased breath sounds and/or wheezes in all lung fields with COPD and the expiratory phase is often prolonged.

represents the amount of oxygen dissolved in the plasma and SAO2 is the amount of oxygen bound to hemoglobin.
-The movement of oxygen and carbon dioxide across the alveolar capillary membrane is called diffusion this direction of movement is from an area of higher concentration to an area of lower concentration.
-Ventilation is the act of inspiration (air in) and expiration (air out).
-Elastic recoil is the tendency for lungs to return their original size after being expanded through breathing.
-Compliance: ability of the lungs to expand, when it is decreased it is harder for the lungs to inflate. Decreased compliance may occur if there is fluid in the lungs (pneumonia). COPD is a common cause of lung compliance increase.
-Resistance: is any obstacle relating to air flow during inspiration/expiration. This is common when there is a change in the diameter of the airway like during an asthma attack where there is a narrowed airway resulting in increased resistance. This resistance is commonly counteracted by the use of bronchodilators. Secretions build up in the airway can also increase resistance by making the airway narrower which can be solved with mucolytics or expectorants.
-Medulla: this is the respiratory center in the brain that responds to chemical and mechanical signals. It sends impulses to respiratory muscles through the spinal cord and phrenic nerves.
-Chemoreceptors are receptors that responds to change in chemical composition like pH or the fluid around it. Central chemoreceptors are found in the medulla and peripheral chemoreceptors are found in the carotid bodies.
-Mechanical receptors are in the conducting upper airways, chest wall, diaphragm, and capillaries of the alveoli. They can be stimulated by a variety of factors such as irritants or stretching of muscles. The three major types of mechanical receptors are irritant, stretch, and juxtacapillary.
-Irritant are in the conducting airways and are sensitive to inhaled particles and aerosols, they initiate the cough reflex.

-Stretch receptors are in the smooth muscle of the airways and aid in control of respiration as lungs inflate.
-Juxtacapillary receptors are found in the capillaries of the alveoli and increase pulmonary capillary pressure.
-There are many respiratory defense mechanisms for example air filtration is when the nasal hairs filter the air we breathe in, there is also the mucociliary clearance system which is responsible for the movement of mucus and this mucus forms a blanket that contains particles/debris to prevent them from reaching the lungs.
-There is also the cough reflex which is a productive reflex that clears the airway by coughing out the air. It is the backup for mucociliary clearance.
-Reflex bronchoconstriction is when the bronchi constrict to prevent entry of irritants.
-Alveolar macrophages rapidly phagocytize inhaled foreign particles such as bacteria.

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

NCLEX IV *(7)*: Reduction of Risk

<u>Contributing Risk Factors</u>

-Smoking
-Age
-Exposure to environmental/occupational factors
-Gender (men are slightly more likely)
-Increased number of respiratory tract infections in childhood
-History of TB
-Asthma
-Alpha 1 antitrypsin deficiency

<u>Signs and Symptoms</u>

-Dyspnea
-Barrel chest
-Tachycardia
-JVD
-Restlessness
-Debilitation
-Muscular atrophy
-Rapid shallow breathing
-Prolonged expiratory phase
-Pursed lip exhaling
-Use of accessory muscles
-Cyanosis/Pallor
-Poor skin turgor
-Peripheral edema

<u>Possible Therapeutic Procedures</u>

<u>Non-surgical</u>

-Noninvasive ventilation
-Endobronchial valve therapy

<u>Surgical</u>

- Lung volume reduction surgery
- Bronchoscope lung volume reduction
- Bullectomy

<u>Prevention of Complications</u>

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

-Cor pulmonale
-Pulmonary hypertension
-Acute respiratory failure
-Acute exacerbations
-Death

	-Easy bruising -Wheezing/crackles -Decreased breath sounds		

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

Anticipated Medication Management
-Supplemental O2
-Bronchodilators
-Nebulizer treatments
-B2 adrenergic agonist (SABA)
-Anticholinergics
-Mucolytics
-Anti inflammatory

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

Non-Pharmacologic Care Measures
-Pulmonary rehabilitation
-Nutrition therapy
-Smoking cessation resources
-Therapy
-Exercise classes

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

What stressors might a patient with this diagnosis be experiencing?
-Fear of death
-Financial stress
-Fear of not being able to breathe properly.

Client/Family Education

List 3 potential teaching topics/areas
 Teaching the patient how to do breathing exercises like pursed-lip breathing and diaphragmatic breathing.

 Teach the patient to perform their activities during the time of day when their breathing is the best like in the morning.

 Teach patient about the effects of smoking with COPD and the benefits of smoking cessation.

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)
-Therapist
-Nutritionist
-Respiratory therapist
-Pharmacist
-Pulmonologist
-Physiotherapist
-Lung function technicians
-Thoracic surgeon
-Pulmonary rehabilitation therapist