Preconference Form

Student Name: **Yana Quel**

Medical Diagnosis/Disease: **Chronic Obstructive Pulmonary Disease (COPD)**

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

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

Anatomy and Physiology

Normal Structures

Upper respiratory tract:

Nose: Air enters the respiratory tract through the nose. The nasal cavity is made of up pseudostratified ciliated columnar epithelial cells, also called respiratory epithelium, that make up most of the respiratory tract. It is made of bone and cartilage and is divided into 2 nares from the nasal septum. The inside of the nose has 3 turbinates, projections of a passage that increase surface area of nasal mucosa. This mucosa warms and moistens the air as it enters the nose to protect the lower airways and lungs by filtering out small particles. Nose hairs collect any dirt and dust from entering the passages, and cilia which act like hairs to further collect dust to move them outside the body. The internal nose opens directly into the 4 sinuses: frontal, ethmoid, maxillary, and sphenoid. What causes the sense of smell is from the olfactory nerve that is found in the upper mucosa of the nasal cavity.

Mouth: Also called the oral cavity, is where air enters the respiratory tract through to the oropharynx.

Pharynx (3): The pharynx is a tubular passageway that has 3 parts: naso/oro/ laryngo pharynx (superior to inferior) connecting to the epiglottis.

Epiglottis: A small flap behind the tongue that closes when one swallows to prevent anything but air from entering the larynx and further into the lungs.

Larynx: This is where the vocal cords are located and where air travels to after the epiglottis. The opening between the vocal cords is called the glottis, where air then travels though the trachea.

Trachea: This is made of up many U-shaped cartilage rings of connective tissue It is about 5 in. long and 1 in. wide and allows for opening but is flexible enough to allow for esophagus to expand. The trachea further divides into the right/left bronchi at the carina that is located at the *Angle of Louis*, 4-5th thoracic vertebrae. It is highly sensitive and if stimulated, can cause excessive coughing.

Lower respiratory tract:

Bronchi: This consists of the right and left mainstem, that divide further into the lobar, segmental and subsegmental bronchi. The trachea and bronchi transfer gases to and from the alveoli, and this volume is called the anatomic dead space that does not take part in gas exchange. In adults, the air that does take part is called the tidal volume, and the normal amount is 500mL (in a 150 lb man, and about 150mL of this is anatomic dead space; the rest of the 350mL is exchanged in the alveoli).

Bronchioles: The most proximal bronchioles are the non-respiratory while the most distant are the respiratory. They have smooth muscle that constrict and dilate. This is where the term bronchoconstriction and bronchodilation refer to.

Alveolar ducts: They are made up of smooth and connective tissue that connect the bronchioles to the alveoli.

Alveoli: They are small grape-like sacs that are the primary center for gas exchange of CO2 and O2. The

Pathophysiology of Disease

-Progressive and persistent airflow limitation and chronic inflammation of airways, lung parenchyma, and pulmonary blood vessels. There is a loss of elastic recoil and airflow obstruction, from mucous accumulation and hypersecretion, mucosal edema, and bronchospasm. Also from ciliary dysfunction, hyperinflation of lungs, and gas exchange abnormalities. Starts w/ noxious particles and gasses (tobacco, smoke, pollution) that causes chronic inflammation and tissue destruction that disrupt normal defense mechanisms. Oxidants from these 3 causes inactivate natural destruction of the lungs, stimulate mucous secretion, and increase fluid in lungs, all causing structural changes. Oxidants also activate proteases that break down connective tissue and inhibit antiproteases that protect against the breakdown (all subsequently causing elastic recoil). In COPD, inspiring is passive and expiring is active, causing difficulties expelling CO2, causing barrel chest (air is not able to escape through remodeled peripheral airways and dysfunctional respiratory muscles (connections to the alveoli are destroyed causing air trapping). This causes the lungs to overinflate and pt becomes dyspneic and limits exercise tolerance). Severe COPD can cause hypoxemia, that is helped w/ supplemental O2, but as it progresses there is also hypercapnia (increased CO2 retainment), which destroys alveoli. Bullae (large air spaces in the parenchyma) and blebs (air spaces next to pleurae) that causes a V/Q mismatch and hypoxemia and CO2 retention.

-Can be progressed from having both chronic bronchitis and emphysema. Chronic bronchitis is the presence of

Anticipated Diagnostics

Labs:

-ABGs

-Serum alpha 1-antitrypsin levels

-CBC

Additional Diagnostics:

-CXR

-spirometry for FEV1 level to test for severity of mild, moderate, severe, and very severe

-H&P

-6 min. walk test

-COPD assessment test (CAT) or

-Clinical COPD questionnaire (CCQ)

normal adult lung has over 300 million alveoli, each 0.3mm wide. They are interconnected by Pores of Kohn, and deep breathing allows air to move through them to help move mucus out of the bronchioles. Alveoli produce surfactant, a lipoprotein, that lowers surface tension and reduces the amount of pressure needed to inflate them to make them less likely to collapse.
Alveoli-Capillary membrane: This is where gas is exchanged from the alveoli into the pulmonary capillaries of the lungs.
Lungs (2): The right lung is divided into 3 lobes (upper, middle, and lower), and the left lung is divided into 2 lobes (upper and lower). Except for the right and left mainstem bronchi, all other lower respiratory structures are found within the lungs. These, pulmonary vessels, and nerves enter the lungs through the hilus, with the right mainstem bronchi being shorter, wider, and straighter than the left due to the anatomical positioning of the heart. This is also why aspiration is more likely to occur in the right lung.
Defense Mechanism: 1) Air filtration: velocity and air turbulence to remove bacteria and large particles to be removed before they reach the alveoli. **2) Mucociliary Clearance System:** mucociliary escalator moves mucous and debris from distal lung areas. **3) Cough reflex:** high-pressure, high-velocity flow of air acting as a backup for mucociliary clearance. **4) Reflex bronchoconstriction:** inhaling large irritants cause bronchi to constrict to prevent entry. **5) Alveolar Macrophages:** no ciliated cells below respiratory bronchioles, so defense are the macrophages that phagocytize bacteria or other inhaled foreign particles.
2 types of circulation: pulmonary: provides lungs w/ blood, pulmonary artery receives deoxygenated blood rom right ventricle, and gets oxygenated in lungs by gas exchange in pulmonary capillaries, then the pulmonary vein returns oxygenated blood to right atrium/ventricle, then out the aorta to systemically circulate. **Bronchial:** from bronchial arteries that arise from thoracic aorta that does not take part in gas exchange but provides O2 to bronchi and other ling tissues. Deoxygenated blood returns through the azygos vein into the SVC.

cough and sputum for at least 3 months inn each of 2 consecutive years. Can precede or follow air limitation.
Emphysema (destruction of alveoli w/o fibrosis) is a structural abnormality in COPD.
-Predominant inflammatory cells are neutrophils, macrophages, and lymphocytes.
-Men are slightly more at risk and have a worse response from O2 therapy.
-Women are more likely to get it from just cigarette smoking and having smaller lungs and airways.

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

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

Contributing Risk Factors	Signs and Symptoms	Possible Therapeutic Procedures	Prevention of Complications
-Cigarette (tobacco) smoking: increases mucous production, harder to clear them, reduces ciliary activity, abnormal airway remodeling. -Infection: Smoking and HIV develop COPD faster; and TB -Asthma: asthma-COPD overlap syndrome- bronchospasms and air limitations -Air pollution: urban air pollution, coal, other biomass fuels for indoor heating and cooking (even if they never smoked). -Occupational chemical and dusts: dusts, vapors, irritants, or fumes in workplace -Aging: chest wall stiffness, lowered elastic recoil, chest becomes larger to retain volume, decrease in functional alveoli, surface area for gas exchange decreases -Genetics: the "Z" allele of the AAT protein -Alpha-1 Antitrypsin deficiency: it protects lungs and liver from proteolytic enzymes, and w/o enough of the enzymes, they destroy alveoli and can cause lung/ liver disease and can cause early-onset empysema	-chronic, intermittent cough (1st sign usually) -dyspnea -chest heaviness, not being able to take a deep breath, gasping, increased work of breathing **Later stages:** -dyspnea at rest -diaphragm flattens -wheezing and chest tightness -fatigue -weight loss -anorexia -tripoding positioning -barrel chest -hypoxemia and hypercapnia -polycythemia and cyanosis -high hemoglobin and hematocrit	Non-surgical: -O2 therapy -Drug therapy - Respiratory therapy: breathing and coughing -Nutritional therapy Surgical: -**Lung volume reduction surgery (LVRS):** reduce lung size and diseased lung (decreased airway obstruction, increased room for alveoli, allowing diaphragm to return to normal shape) -**Bronchoscopic lung volume reduction (BLVR**): multiple 1-way valves leading to diseased part to allow air to leave during lung exhalation and preventing air from entering during inhalation. -**Bullectomy**: 1+ very large bullae are removed (decreases WOB -**Lung transplant (single or bilateral).**	(What are some potential complications associated with this disease process) -**pulmonary hypertension** (constriction of pulmonary vessels from alveolar collapse). The pressure on the right side of the heart increases to push blood to lungs and right-sided HF can develop -**Cor pulmonale:** results from pulmonary hypertension and a late manifestation of COPD. Dyspnea, maybe crackles, S3 and S4 heart sounds, right HF may develop, distended neck veins, peripheral edema, weight gain, increased pressure on right heart -**Acute exacerbations**: sudden change of Sx of COPD baseline and respiratory infections are usually the cause. Common and increase in frequency as disease progresses **-Acute Respiratory Failure:** when pt's fail to contact HCP about exacerbation and their condition has worsened to the point of most likely needing mechanical ventilation and admission to ICU.

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

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

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

Anticipated Medication Management
-O2 therapy
-antitussive for dry, hacking cough
-expectorant or mucolytic for wet cough
-SABA (short-acting adrenergic agents)- commonly Albuterol
-LABA (long-acting adrenergic agents)- Salmeterol and formoterol
-Bronchodilator (beta-2 adrenergic agonists) or anticholinergics
-Combo of short-acting bronchodilators w/ anticholinergic (improve bronchodilation and can decrease risk of side effects). (albuterol and ipratropium can be nebulized together)
-ICS (inhaled corticosteroid steroid)- can have side effects but lessened if combined w/ LABA like Advair or Symbicort
-Roflumilast (Daliresp)- decrease exacerbations in severe COPD
-vaccinations (flu, COVID-19, pneumonia)

Non-Pharmacologic Care Measures
-elevated HOB or tripoding
-use of IS
-turn, deep breath, and cough
-rest between periods of activity
-smoking cessation
-adequate hydration and nutritional status
-avoiding irritants

What stressors might a patient with this diagnosis be experiencing?
-pain
-inability to breathe properly
-physical body changes (barrel chest)
-inability to exercise w/o periods of rest
-long-term O2
-medical bills, insurance, finances
-quality of life!

Client/Family Education

List 3 potential teaching topics/areas
 smoking cessation programs

 deep breathing and coughing techniques- teach back

 long-term use of supplemental O2

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)
-PT/RT
-surgeon
-radiologist
-pulmonary rehab
-dietician