Student Name: Ryan Clagett

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

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

Anatomy and Physiology
Normal Structures

See attached

Pathophysiology of Disease

See attached

NCLEX IV *(7)*: Reduction of Risk

Anticipated Diagnostics

Labs

-CBC
-ABGs (severity of exacerbations)
-Serum alpha1-antitrypsin levels

Additional Diagnostics

-H&P
-CXR (flat diaphragm from hyperinflated lungs
-Spirometry
-6 min walk test (exercise-induced hypoxemia
-COPD Assessment Test
-Clinical COPD Questionnaire
-ECG (right HF, heart function)
-Sputum C&S

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

Contributing Risk Factors

-Cigarette smoking
-Infection (severe recurrent respiratory tract infections in childhood, smoking w/ HIV, TB)
-Asthma (asthma-COPD overlap syndrome)
-Air pollution
-Occupational chemicals and dusts
-Aging
-Genetics
-Alpha-1 Antitrypsin Deficiency (an autosomal recessive disorder that may eventually affect the lungs and liver; main function of AAT is to protect the lung tissue from attack by proteases during inflammation r/t smoking and infections)

Signs and Symptoms

-Chronic intermittent cough
-Dyspnea
-Chest heaviness
-Flattened diaphragm
-Chest breather
-Wheezing
-Fatigue
-Weight loss/anorexia
-Expiratory phase is prolonged
-Decreased lung sounds and/or wheezes in all lung fields
-Anteroposterior diameter increases
-Breathing louder
-Tripod position
-Pursed-lip breathing
-O2 saturation <88% RA
-Hgb concentrations >200 g/L
-Low Hgb/Hct b/c chronic anemia

NCLEX IV *(7)*: Reduction of Risk

Possible Therapeutic Procedures

Non-surgical

-O2 therapy
-Drug therapy
-Respiratory care (breathing techniques
-Nutrition therapy (pts. underweight)

Surgical

-Lung volume reduction
-Bronchoscopic lung volume reduction
-Bullectomy (1 or more very large bullae are removed)
-Lung transplant

Prevention of Complications

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

-Pulmonary HTN
-Cor pulmonale (continuous, long-term, low-flow O2 therapy)
-Acute exacerbations (avoid what causes them, receive immunizations)
-ARF (continue taking meds)

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

Anticipated Medication Management

-Bronchodilator therapy
-Beta-2-adrenergic agonists (smooth muscle relaxation)
-Anticholinergics (block the action of acetylcholine on the muscarinic receptors in the smooth muscles of the bronchotracheal tree)
-Roflumilast (decrease exacerbations in pts. w/ severe COPD
-Mucolytics
-Diuretics

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

Non-Pharmacologic Care Measures

-HOB >30 degrees
-Daily bath/linen change/oral hygiene
-Encouraging all meals/oral fluids
-Cough and deep breathing
-Rest periods
-Assist w/ ambulation and exercise

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

What stressors might a patient with this diagnosis be experiencing?

-Guilt for smoking
-Role as caregiver/provider for family
-Will never live a normal life again
-Progressive decline w/ COPD

Client/Family Education

List 3 potential teaching topics/areas

- Smoking cessation
- Breathing exercises and retraining
- Immunizations: Influenza, pneumococcal, COVID-19

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)

RN, hospitalist, surgeon, respiratory therapy, dietician, pharmacist, chaplain, case management, PT/OT

Anatomy and Physiology/Normal Structures

ANATOMY

- The primary purpose of the respiratory system is gas exchange; this involves the transfer of oxygen and carbon dioxide between the atmosphere and the blood; adequate perfusion depends on adequate oxygenation, requiring a healthy, functioning respiratory system.
- The respiratory system is divided into 2 parts: upper and lower tracts
- Upper Respiratory Tract
    - Includes nose, mouth, pharynx, epiglottis, larynx, and trachea
    - Air enters through the nose; the nose is made of bone and cartilage, divided into 2 nares by the nasal septum; 3 passages in the nose, shaped by projections called turbinates which increase the surface area of the nasal mucosa that warm and moisten the air as it enters the nose; the internal nose opens directly to the sinuses and connects w/ the pharynx: nasopharynx, oropharynx, and laryngopharynx; nose protects the lower airway by warming and humidifying air and filtering small particles before air enters the lungs; the olfactory nerve, found w/n mucosa of the upper nasal cavity, is responsible for the sense of smell.
    - After traveling from the oropharynx to the laryngopharynx, air travels through the epiglottis to the larynx before moving into the trachea; the epiglottis is a small flap behind the tongue that closes over the larynx during swallowing (prevents solids and liquids from entering the lungs; the vocal cords are in the larynx; air passes through the glottis (the opening b/t the vocal cords) and into the trachea.
    - The trachea is a cylindrical tube about 10-12 cm long and 1.5-2.5 cm in diameter; U-shaped cartilages keep the trachea open but allow the adjacent esophagus to expand for swallowing; the trachea divides into right and left mainstem bronchi at the carina; the carina is located at the angle of Louis which is at the level of the 4^{th} and 5^{th} thoracic vertebrae; the carina is highly sensitive and stimulation in that area during suctioning causes vigorous coughing.
- Lower Respiratory Tract
    - Consists of the bronchi, bronchioles, alveolar ducts, and alveoli; except for the right and left mainstem bronchi, all lower airway structures are found w/n the lungs; the right lung is divided into 3 lobes (upper, middle, lower) and the left lung into 2 lobes (upper, lower).
    - The mainstem bronchi, pulmonary vessels, and nerves enter the lungs through a slit called the hilus; the right mainstem bronchus is shorter, wider, and straighter than the left mainstem bronchus (aspiration is more likely to occur in the right lung than the left lung); the mainstem bronchi subdivide several times to form the lobar, segmental (8 branchings), and subsegmental bronchi (15 branchings); further divisions form the bronchioles; the most distant bronchioles are the respiratory bronchioles (24 branchings); the bronchioles are encircled by smooth muscles that constrict and dilate in response to various stimuli; bronchoconstriction and bronchodilation refer to a decrease or increase in the diameter of the airways caused by contraction or relaxation of these muscles; beyond the bronchioles are the alveolar ducts and alveoli.
    - The trachea and the bronchi act as a pathway to conduct gases to and from the alveoli; the volume of air in the trachea and bronchi is called the anatomic dead space (the air doesn't take part in gas exchange); in adults, the normal tidal volume (air exchanged w/ each breath) is about 500 mL; of each 500 mL inhaled, about 150 mL is anatomic dead space.
    - The alveoli (28 branchings from alveolar ducts) are the final part of the respiratory tract; they are small sacs in the lungs that are the primary site of gas exchange of oxygen and carbon dioxide; the adult lung has over 300 million alveoli, each 0.3 mm in diameter; the alveoli are interconnected by pores of Kohn (allow movement of air from alveolus to alveolus; deep breathing promotes air movement through these pores and helps move mucus out of the respiratory bronchioles; bacteria can also move through these pores, spreading infection to previously uninfected areas; alveoli have a total volume of about 2500 mL, w/ a surface area for gas exchange that is about the size of a tennis court; gases are exchanged across the alveolar-capillary membrane where the alveoli come in contact w/ pulmonary capillaries; in conditions such as pulmonary edema, excess fluid fills the interstitial space and alveoli, reducing gas exchange.
- Surfactant
    - Because alveoli are unstable, they have a natural tendency to collapse; alveolar cells secrete surfactant; surfactant is a lipoprotein that lowers the surface tension in the alveoli, reducing the amount of pressure needed to inflate the alveoli and makes them less likely to collapse; normally, each person takes a slightly larger breath, termed a sigh, after every 5 or 6 breaths which stretches the alveoli and promotes surfactant secretion.

- When there isn't enough surfactant, the alveoli collapse (termed atelectasis for collapsed, airless alveoli).

- Blood Supply
  - The lungs have 2 different types of circulation: pulmonary and bronchial
    - Pulmonary circulation provides the lungs w/ blood that takes part in gas exchange; the pulmonary artery receives deoxygenated blood from the right ventricle of the heart and delivers it to pulmonary capillaries that lie directly alongside the alveoli; oxygen-carbon dioxide exchange occurs at this point; the pulmonary veins return oxygenated blood to the left atrium which then delivers it to the left ventricle and into systemic circulation; venous blood is collected from capillary networks of the body and returned to the right atrium via the superior and inferior vena cava.
    - Bronchial circulation starts w/ the bronchial arteries, which arise from the thoracic aorta; bronchial circulation doesn't take part in gas exchange but provides oxygen to the bronchi and other lung tissues; deoxygenated blood returns from the bronchial circulation through the azygos vein into the superior vena cava.
- Chest Wall
  - The chest wall is shaped, supported, and protected by 24 ribs (12 on each side); the thoracic cage (consists of ribs and sternum) protects the lungs and the heart from injury; the mediastinum is the space in the middle of the thoracic cavity; it contains the major organs of the chest, including the heart, aorta, and esophagus; the mediastinum physically separates the right and left lungs into 2 separate components; the chest cavity is lined w/ a membrane called the parietal pleura; the lungs are lined w/ a membrane called the visceral pleura; the parietal and visceral pleurae join to form one continuous membrane; the visceral pleura doesn't have any sensory (pain) fibers or nerve endings; the parietal pleura has pain fivers (irritation and inflammation can cause pain w/ each breath: pleuritic pain).
  - The intrapleural space is the space b/t the pleural layers; normally, it contains 10-20 mL of fluid; this fluid serves 2 purposes: (1) it provides lubrication, allowing the pleural layers to slide over each other during breathing, and (2) it increases unity b/t the pleural layers; this promotes the expansion of the pleurae and lungs during inspiration.
  - Fluid drains from the pleural space via lymphatic circulation; several conditions may cause a pleural effusion (excess fluid in the pleural space); pleural fluid may accumulate b/c of blocked lymphatic drainage (from cancer) or an imbalance b/t intravascular and oncotic fluid pressures; this happens w/ heart failure; purulent pleural fluid w/ bacterial infection is called empyema.
  - The diaphragm is the major muscle of respiration; during inspiration, the diaphragm contracts, moves downward, and increases intrathoracic volume; at the same time, the internal intercostals relax, and the external intercostal muscles contract; this increases the lateral and anteroposterior dimension of the chest; the scalene muscles also contract on inspiration, raising the first and second ribs; this causes the size of the thoracic cavity to increase and intrathoracic pressure to decrease, pulling air into the lungs; the diaphragm is made up of 2 hemidiaphragms, each innervated by the right and left phrenic nerves; the phrenic nerves arise from the spinal cord b/t C3 and C5; injury to the phrenic nerve results in hemidiaphragm paralysis and dependence on a mechanical ventilator

PHYSIOLOGY

- Oxygenation
  - Refers to the process of obtaining oxygen from the atmospheric air and making it available to the organs and tissues of the body; the lungs' ability to oxygenate arterial blood adequately is evaluated by the partial pressure of O2 in arterial blood (PaO2), arterial O2 saturation (SaO2), and patient assessment.
  - O2 is carried in the blood in 2 forms: dissolved and Hgb-bound; the PaO2 represents the amount of O2 dissolved in the plasma; the SaO2 is the amount of O2 bound to Hgb in comparison w/ the amount of O2 the Hgb can carry; e.g., if SaO2 is 90%, then 90% of Hgb attachments for O2 have O2 bound to them.
  - O2 and CO2 move back and forth across the alveolar-capillary membrane by diffusion; the overall direction of movement is from the area of higher concentration to the area of lower concentration; thus, O2 moves from the alveolar gas (atmospheric air) into the arterial blood and CO2 from the arterial blood into the alveolar gas; diffusion continues until equilibrium is reached.
- Ventilation
  - Involved inspiration (movement of air into the lungs) and expiration (movement of air out of the lungs); air moves in and out of the lungs b/c intrathoracic pressure changes in relation to pressure at the airway opening; contraction of the diaphragm and external intercostal and scalene muscles increases chest dimensions; this decreases intrathoracic pressure; gas flows from an area of higher pressure (atmospheric) to one of lower pressure (intrathoracic); when dyspnea (SOB) occurs, neck and shoulder muscles, and other accessory muscles of respiration, can aid the effort.

  - Expiration is passive; elastic recoil is the tendency for the lungs to return to their original size after being stretched or expanded; the elasticity of lung tissue is due to the elastin fibers found in the alveolar walls and surrounding the bronchioles and capillaries; the elastic recoil of the chest wall and lungs allows the chest to passively decrease in size; when intrathoracic pressure rises, air moves out of the lungs.
  - Exacerbations of asthma or COPD cause expiration to become an active, labored process; intercostal, scalene, and accessory muscles (ABD, trapezius) help expel air during labored breathing.
- Compliance and Resistance
  - Changes in compliance and resistance can affect both oxygenation and ventilation; compliance refers to the ability of the lungs to expand; this is a result of the elasticity of the lungs and elastic recoil of the chest wall; w/ decreased compliance, it's harder for the lungs to inflate; this occurs w/ conditions that increase fluid in the lungs (pulmonary edema, acute respiratory distress syndrome, PNA), make lung tissue less elastic or distensible (pulmonary fibrosis, sarcoidosis), or restrict lung movement (pleural effusion); compliance increases when there is destruction of alveolar walls and loss of tissue elasticity, as in COPD.
  - Resistance refers to any obstacle to airflow during inspiration and/or expiration; the main factor affecting airway resistance is changes in the diameter of the airways (acute asthma attacks narrow airways, increasing resistance); giving bronchodilators decreases resistance by increasing the diameter of the bronchi, promoting air entry; the presence of secretions in the bronchi also increases resistance; secretions can partially occlude airways, making airways narrower; this makes it harder for pts. To get air into their lungs; moving secretions from the airway w/ mucolytics or expectorants help decrease resistance.
- Control of Respiration
  - Located in the brainstem, the respiratory center (the medulla) responds to chemical and mechanical signals; the medulla sends impulses to the respiratory muscles through the spinal cord and phrenic nerves
  - A chemoreceptor is a receptor that responds to a change in the chemical composition (PaCO2 and pH) of the fluid around it; an increase in the H+ concentration (acidosis) causes the medulla to increase the respiratory rate and tidal volume; a decrease in H+ concentration (alkalosis) has the opposite effect; changes in PaCO2 regulate ventilation primarily by their effect on the pH of cerebrospinal fluid; when the PaCO2 level is increased, more CO2 is available to combine w/ H2O and form carbonic acid (H2CO3); this lowers the cerebrospinal fluid pH and stimulates an increase in respiratory rate; the opposite process occurs w/ a decrease in PaCO2 level.
  - Peripheral chemoreceptors are found in the carotid bodies and the aortic bodies.
  - In a healthy person, an increase in PaCO2 or a decrease in pH causes an immediate increase in the respiratory rate; the PaCO2 doesn't vary more than about 3 mmHg if lung function is normal; conditions such as COPD change lung function and may result in chronically elevated PaCO2 levels; in these instances, the pt. is not sensitive to further increases in PaCO2 as a stimulus to breathe; they may be maintaining ventilation largely b/c of a hypoxic drive form the peripheral chemoreceptors
  - Mechanical receptors are found in the conducting upper airways, chest wall, diaphragm, and capillaries of the alveoli; they are stimulated by a variety of physiologic factors, such as irritants, muscle stretching, and alveolar wall distortion; 3 major types are irritant, stretch, and juxtacapillary (J) receptors.
    - Irritant receptors are found in the conducting airways; these receptors are sensitive to inhaled particles and aerosols; when stimulated, they initiate the cough reflex; signals from stretch receptors, in the smooth muscles of the airways, aid in the control of respiration.
    - As the lungs inflate, stretch receptors activate the inspiratory center to inhibit further lung expansion; this is the Hering-Breuer reflex, which prevents overdistention of the lungs.
    - Stimulation of the J receptors, found in the capillaries of the alveoli, occurs w/ increased pulmonary capillary pressure; this causes rapid, shallow respiration (tachypnea) seen in pulmonary edema.
- Respiratory Defense Mechanisms
  - Efficient in protecting the lungs from inhaled particles, microorganisms, and toxic gases; the defense mechanisms include air filtration, mucociliary clearance system, cough reflex, reflex bronchoconstriction, and alveolar macrophages.
  - Nasal hairs filter inspired air; in addition, the abrupt changes in the direction of airflow that occur as air moves through the nasopharynx and larynx increase air turbulence, causing particles and bacteria to contact the mucosa lining these structures; the velocity of airflow slows greatly after it passes the larynx, facilitating the deposition of smaller particles, called sedimentation.
  - Below the larynx, the mucociliary clearance system (mucociliary escalator) is responsible for the movement of mucus; goblet cells and submucosal glands continually secrete mucus at a rate of about 100 mL/day; this mucus

forms a blanket that contains the impacted particles and debris from distal lung areas; secretory IgA in the mucus helps protect against bacteria and viruses; cilia cover the airways from the level of the trachea to the respiratory tract; each ciliated cell has about 200 cilia; they beat rhythmically about 1000 times/min in the large airways, moving mucus toward the mouth; the ciliary beat is slower further down the tracheobronchial tree; dehydration, smoking, inhaling high O2 concentrations, infection, and drugs, such as atropine, anesthetics, alcohol, or cocaine, impair ciliary action; pts. w/ COPD and cystic fibrosis often have repeated lower respiratory tract infections; these conditions are associated w/ destroyed cilia, resulting in impaired secretion clearance.

- The cough is a protective reflex that clears the airway by a high-pressure, high-velocity flow of air; it's a backup for mucociliary clearance, especially when this clearance mechanism is overwhelmed or ineffective; coughing is effective in removing secretions only about the subsegmental level; secretions below this level must be moved upward by the mucociliary mechanism before we can remove them by coughing.
- When we inhale large amounts of irritating substances, the bronchi constrict to prevent entry of the irritants; a person w/ hyperreactive airways (asthma), may have bronchoconstriction after inhalation of triggers, such as cold air, perfume, or other strong odors.
- Because there are no ciliated cells below the level of the respiratory bronchioles, the primary defense mechanism at the alveolar level is alveolar macrophages; they rapidly phagocytize inhaled foreign particles, such as bacteria; the debris is moved to the level of the bronchioles for removal by the cilia or removed from the lungs by the lymphatic system; particles (coal dust, silica) that cannot be adequately phagocytized tend to remain in the lungs for indefinite periods and can stimulate inflammatory responses; b/c cigarette smoke impairs alveolar macrophage activity, the smoker who works in an occupation w/ heavy dust exposure (mining, foundries) has an especially high risk for lung disease.

- Gerontologic Considerations
  - Structural changes include calcification of the costal cartilages, which can interfere w/ chest expansion; the outward curvature of the spin is marked, especially w/ osteoporosis, and the lumbar curve flattens; therefore, the chest may appear barrel-shaped, and the person may need to use accessory muscles to breathe; respiratory muscle strength progressively declines after age 50; overall, the lungs in the older adult are harder to inflate.
  - Within the lung, the number of functional alveoli decreases, and they become less elastic; small airways in the lung bases close earlier in expiration; therefore, more inspired air is distributed to the lung apices, and ventilation is less well matched to perfusion, lowering the PaCO2; as a result, older adults have less tolerance for exertion and dyspnea can occur if their activity exceeds their normal exercise.
  - Respiratory defense mechanisms are less effective b/c of a decline in immune function and the ability to produce antibodies.
  - The aging process alters respiratory control, resulting in a more gradual response to changes in blood O2 or CO2; the PaO2 may drop to a slightly lower than normal level and the PaCO2 may rise to a level slightly higher than normal before the respiratory rate changes.
  - The extent of changes varies greatly; e.g., the older adult who has a significant smoking Hx, is obese, and has a chronic illness is at greater risk for adverse outcomes.

Pathophysiology of Disease

- COPD is a progressive lung disease characterized by persistent airflow limitation; it's associated w/ an enhanced chronic inflammatory response in the airways and lungs; the main causes are cigarette smoking and other noxious particles and gases; exacerbations and other coexisting problems may contribute to the severity of the disease; previous definitions of COPD have included such terms as chronic bronchitis and emphysema; each condition has features of COPD, but neither by itself is COPD.
    - Chronic bronchitis is the presence of cough and sputum production for >3 months in each of 2 consecutive years; it's an independent disease that may precede or follow the development of airflow limitation.
    - Emphysema is the destruction of alveoli w/o fibrosis; it describes one of several structural abnormalities in COPD.
- Around 16 million adults in the US have COPD; the number is underestimated b/c the disease is usually not diagnosed until it's moderately advanced; COPD is the 3rd leading cause of death in the US, causing more than 140,000 deaths each year.
- Men
    - COPD is slightly higher in men.
    - Have a poorer response to O2 therapy.
- Women
    - Prevalence of COPD is increasing.
    - The increase I likely due to more women smoking and increased susceptibility (smaller lungs, airways).
    - Women who smoke are 50% more likely to develop COPD compared to men who smoke.
    - Women w/ COPD have a lower quality of life, more exacerbations, less phlegm, and increased dyspnea.
- COPD is characterized by chronic inflammation of the airways, lung parenchyma (respiratory bronchioles and alveoli; sites of gas exchange), and pulmonary blood vessels; the defining feature is airflow limitation not fully reversible during forced exhalation; the main cause is the loss of elastic recoil and airflow obstruction, from mucus hypersecretion, mucosal edema, and bronchospasm.
- As the disease progresses, abnormalities in airflow limitation, air trapping, and gas exchange worsen; in severe COPD, pulmonary hypertension and systemic manifestations occur; COPD has an uneven distribution of pathologic changes; severely impaired and/or destroyed areas of lung tissue exist alongside areas of relatively normal lung.
- The inflammatory process most often starts w/ inhaling noxious particles and gases (cigarette smoke); w/ repeated exposure, chronic inflammation causes tissue destruction and disrupts the normal defense mechanisms and repair process of the lung; may be genetically determined in some people.
- The predominant inflammatory cells in COPD are neutrophils, macrophages, and lymphocytes; inflammatory cells attract other inflammatory mediators (leukotrienes) and proinflammatory cytokines (tumor necrosis factor).
- The inflammatory process may be increased by the oxidants made by cigarette smoke and other inhaled particles and released from the inflammatory cells; oxidants adversely affect the lungs as they inactivate antiproteases (which prevent the natural destruction of the lungs), stimulate mucus secretion, and increase fluid in the lungs; the result is structural changes in the lungs.
- After inhaling oxidants in tobacco or air pollution, the activity of proteases (which break down the connective tissue of the lungs) increases, and antiproteases are inhibited; the natural balance of proteases/antiproteases is tipped in favor of alveolar destruction and loss of the lungs' elastic recoil.
- Inability to expire air is a main characteristic of COPD (airflow limitation from smaller airways); when peripheral airways are obstructed, air is progressively trapped during expiration; as air is trapped in the lungs, the chest hyper-expands and becomes barrel-shaped b/c the respiratory muscles can't function effectively; so, functional residual capacity (FRC) is increased; the residual air, combined w/ the loss of elastic recoil, makes passive expiration of air difficult; the pt. becomes dyspneic w/ limited exercise capacity.
- Typically, the pt. doesn't have problems w/ hypoxemia at rest until late in the disease; at first, hypoxemia may develop during exercise, and the pt. may benefit from supplemental O2; gas exchange problems result in hypoxemia and hypercapnia (increased CO2) as the disease worsens; as the air trapping increases, walls of alveoli are destroyed; bullae (large air spaces in the parenchyma) and blebs (air spaces next to pleurae) can form in and on the lungs; bullae and blebs are not effective in gas exchange b/c they don't contain a capillary that normally surrounds each alveolus; therefore a significant V/Q (ventilation/perfusion) mismatch and hypoxemia result; peripheral airway obstruction also results in V/Q imbalance and combined w/ respiratory muscle impairment leads to CO2 retention, particularly in severe forms of the disease.
- Excess mucus production, resulting in a chronic productive cough, is a feature of persons w/ predominant chronic bronchitis; however, not all COPD pts. Have sputum production; when present, excess mucus production results from an increased

number of mucus-secreting goblet cells, enlarged submucosal glands, cilia dysfunction, and stimulation from inflammatory mediators.

- Pulmonary vascular changes causing mild to moderate pulmonary hypertension may occur late in the course of COPD; the small pulmonary arteries vasoconstrict due to hypoxia; as the disease advances, the structure of the pulmonary arteries changes, resulting in thickening of the vascular smooth muscle; b/c of the loss of alveolar walls and the capillaries surrounding them, pressure in the pulmonary circulation increases; pulmonary HTN may progress and lead to hypertrophy of the right ventricle; the right ventricle dilates and may lead to right HF.
- Classification of COPD
    - A FEC1/FVC ratio (a spirometric parameter that represents the proportion of a pt's vital capacity that they are able to expire in the first second of forced expiration; calculated by dividing forced expiratory volume in one second by the forced vital capacity of the lungs; normal value is >75-85%); <70% establishes the diagnosis of COPD; the severity of obstruction determines the stage of COPD; management of COPD is based on the pt's symptoms, classification, and exacerbation Hx.
    - Gold 1 (Mild; FEV1 >80% predicted)
    - Gold 2 (Moderate; FEV1 50-80% predicted)
    - Gold 3 (Severe; FEV1 30-50% predicted)
    - Gold 4 (Very severe; FEV1 <30% predicted)
- Complications
    - In COPD, the main cause of pulmonary hypertension is constriction of the pulmonary vessels from alveolar hypoxia; chronic hypoxia stimulates RBC production which causes polycythemia; this results in increased viscosity of the blood; these pts. have increased pulmonary vascular resistance and develop pulmonary HTN.
    - Normally, the right ventricle and pulmonary circulatory system are low-pressure systems compared w/ the left ventricle and systemic circulation; w/ pulmonary HTN (pressure w/n the lung increases), the pressures on the right side of the heart must increase to push blood into the lungs; eventually, the pressure is so high, that right HR develops.
    - Cor pulmonale (the enlargement and failure of the right ventricle of the heart) results from pulmonary HTN; it's a late manifestation of COPD; once developed, the prognosis worsens; not all pts. w/ COPD develop cor pulmonale.
    - Dyspnea is the most common symptom of chronic cor pulmonale; lung sounds are normal or crackles at the bases; heart sounds may include S3, S4, and systolic murmurs; other manifestations of right HF may develop: distended neck veins, hepatomegaly w/ RUQ tenderness, peripheral edema, and weight gain.
    - Continuous, long-term, low-flow O2 therapy is often part of care; diuretics may be given if left HF or pulmonary edema are present but must be used w/ caution; decrease in fluid volume from diuresis can worsen heart function; long-term anticoagulation therapy is started to help decrease the risk for venous thromboembolism (VTE).
    - An exacerbation of COPD is an acute event characterized by a worsening of the pt's respiratory symptoms; exacerbations are signaled by a sudden change in the pt's usual dyspnea, cough, and/or sputum; respiratory infections are a common cause; exacerbations are common and increase in frequency (on average 1 or 2 per year) as the disease progresses; as the severity of COPD increases, exacerbations are associated with poorer outcomes.
    - Pts. w/ severe COPD w/ severe exacerbations are at risk for acute respiratory failure (ARF); suddenly stopping a med may cause ARF; pts. often wait to be seen, meaning their condition worsens to the point of needing mechanical ventilation and ICU admission.