**COPD/ ASSOCIATED RESPIRATORY DISEASES**

**COPD** is a treatable slowly progressive disease. Incidence is increased with age, onset is usually middle age.

- Characterized by limitation of air flow that is NOT fully reservable, chronic inflammation can cause tissue damage
- Scar tissue: results in narrowing airway, decreases elastic recoil (compliance), causes thickened vessel lining, hypertrophy of smooth muscle and pulmonary hypertension
- Can cause airflow obstruction from noxious particles or gases
- Pts will exhibit chronic cough, sputum, dyspnea symptoms must be present 3 months at a time for 2 years and can lead to cor pulmonale

**COPD assessment**

- barrel chest and use of accessory muscles
- prolonged expiration
- orthopnea
- congestion/hyperinflation seen on chest x-ray/flattened diaphragm
- ABG's that will yield respiratory acidosis and hypoxemia
- Pulmonary FX test that shows decreased vital capacity.
- Get a good HX and examination
- Reviewed dx tests

**COPD interventions**

- smoking cessation is the single most effective intervention, set a quit date w/ the patient and possibly start them on anti smoking medications (gum, patch, pill, nasal spray) chantix is a nicotinic receptor agonist not for teens and decrease 10 ciggaretts a day. Follow up 3-5 days
- monitor vitals /weight/ nutrition high calorie high protein / encourage fluids 3000 ml/day to thin secreations
- administer low O2 levels to avoid blowing out the respiratory tract / breathe in low arterial P02 instead of increased pc02. Administering oxygen can lead to the retention of Co2, to much O2 can cause chemo receptors not work on their own and suppress the hypoxic drive.
- monitor pulse ox
- provide chest physiotherapy and respiratory tx /bronchodilators/ corticosteroids/antibiotics and mucolytics as ordered.
- educate the client on diaphragmatic breathing and pursed lip breathing to increase airway.
- suction the client/ lift the bedside table and have the patient their arms on it

**COPD Risk Factors**

- smoking/exposure to smoke
- Increased age
- Exposure to occupational chemicals
- Pollution
- Genetic abnormality / alpha 1 anti-trypsin deficiency (enzyme that counter acts lung damage)
**COPD Pharmacological therapies**
- Bronchodilators relax smooth muscle in the bronchioles, rinse your mouth after use to avoid thrush, administered throughout the day as well as PRN can be used prophylactically (albuterol)
- Beta 2 agonist
- Short acting: start with this one then 30 secs later do your long acting
- Long action: advair/ symbacort
- Corticosteroids

**Exacerbation of COPD**
- Primary cause is tracheobronchial infection and air pollution
- Tx requires identification of the primary cause
- Optimization of bronchodilators (first line therapy)
- Depending on s/sx antibiotic tx, corticosteroids and nebulizer tx
- Hospitalization is dyspnea doesn't respond to treatment, worsening of symptoms, or central cyanosis
- Educate on the importance of pneumonia vaccine

**Emphysema "pink puffer" R/T hypercapnia**
- Abnormal distention of air spaces beyond the bronchioles w/ destruction of the alveoli
- Progresses slowly/many years
- Decreased alveolar surface area increases dead space (no gas exchange)
- Hypoxemia decreased oxygen to the blood/right sided HF may occur due to pressure (cor pulmonale)
- Manifestations: peripheral edema, liver enlargement, mismatched 02/c02, respiratory acidosis, jugular vein distention
- Wheezes will be noted on auscultation

**Two main types of emphysema**

**Panlobular:**
- Destruction of the respiratory bronchioles, alveolar ducts, and alveolus.
- All airspaces are enlarged but little inflammatory disease
- Hyperinflation chest, dyspnea on exertion, weight loss
- Respiration becomes voluntary and use of accessory muscles is noted

**Centrilobular**
- Changes noted mainly in the center of the secondary lobule
- Chronic hypoxemia, hypercapnia, polycythemia, Rt side HF
- Peripheral edema

* you can have a patient that exhibits both types of emphysema*
**Chronic Bronchitis "Blue Bloater"**
- ciliary fx is reduced, bronchial walls thicken, airways narrow, mucous plugs airways
- alveoli become damaged and fibroed, alveolar macrophage fx diminishes
- patient becomes more susceptible to respiratory infection
- A wide range of viruses, bacteria, and mycoplasmial infections can occur
- Exacerbation of chronic bronchitis are most likely to occur during the winter, when viral and bacterial infections are most common

**BRONCHIECTASIS**
- chronic and irreversible dilation of the bronchi and bronchioles
- Caused by: airway obstruction, pulmonary infection, diffuse airway, genetic disorders (cystic fibrosis), idiopathic causes
- Distortion of vessel walls
- Mismatch 02/CO2 causes decreased perfusion

**BRONCHIECTASIS Management**
- turn cough deep breathe
- Pt education on clearing secretions
- can be mistaken as chronic bronchitis dx via difference in cough and sputum amount
- can treat with bronchoscopy to remove mucous plugs
- smoking can cause ciliary dysfunction
- hand hygiene is very important
- Have the pt avoid air pollutions if possible
- Give psychosocial support
- Have the pt make the choice of how much exercise they can do
- Daily weights
**ASTHMA**

- Abnormal airway clearance that is characterized by reversible inflammation either spontaneously or with tx
- Causes hyperresponsiveness, mucosal edema, mucous production
- S/sx: chest tightness, cough, wheezes, and dyspnea
- Most common disease of childhood
- Ethnic/racial disparities relate to morbidity (African and latinos) are most at risk
- Aspirin can trigger an asthma attack, make sure your educating at risk patients

**Allergies:** the strongest predisposing risk factor for asthma

**Common allergies:** can be seasonal (grass, trees) or perennial (mold, dust, roaches, or animal dander).

**Common triggers:** airway irritants, temperature changes, weather changes, smoke, perfume, hormones, medications, respiratory tract infections and GERD. Most asthma patients are sensitive to more than one trigger.