COPD is defined as a disease state characterized by the presence of airflow limitation that is not fully reversible*.

The airflow obstruction is generally progressive, and may be accompanied by airway hyperactivity.

Incidence

4th leading cause of death (smokers)

Estimated that 16 million people in U.S. have some form of COPD

Annual cost around 32.1 billion

Commonly occurs in middle aged adults

Incidence increases with age

The difference between Asthma and COPD

The difference between the two problems are that asthma has reversible inflammation, where as COPD does not.

Patients with COPD may have asthma, and some patients with asthma may go on to develop fixed or irreversible airflow obstruction.

The difference between Chronic Bronchitis and Emphysema

Chronic bronchitis: the presence of chronic productive cough for 3 months in each of 2 successive years in a pt when other causes of chronic cough have been excluded.

Emphysema is defined as destruction of the walls of overdistended alveoli.

There is generally overlap between the two conditions.

Pathophysiology of COPD

Airflow limitation is progressive

Associated with an abnormal inflammatory response to particles or gases

inflammatory response occurs throughout the proximal and peripheral airways, lung parenchyma, and pulmonary vasculature

In the proximal airways, changes include increased numbers of goblet cells and enlarged submucosal glands, both of which lead to hypersecretion of mucus.

Alveolar wall destruction leads to loss of alveolar attachments and a decrease in elastic recoil.

Body attempts to repair itself, narrowing occurs in small peripheral arteries.

Over time scar tissue is formed due to the injury-repair process

Airway narrows due to scar tissue

Finally, the chronic inflammatory process affects the pulmonary vasculature and causes thickening of the lining of the vessel and hypertrophy of smooth muscle, which may lead to pulmonary hypertension

COPD Etiology

Smoking: Affects ciliary tract cleansing system. Causes increased mucous accumulation. CO (carbon monoxide) has a high affinity for hemoglobin and combines with it more readily than O2. The smoker inhales a lower percentage of O2 than normal. Passive smokers are at risk for the same problems.

Infection: Recurring respiratory tract infections are a contributing factor to the aggravation and progression of COPD. This causes impairment of normal defense mechanisms, making the bronchioles and alveoli more susceptible to injury. COPD patients are more susceptible to infection which intensifies their condition

Air Pollution: High levels of urban air pollution are harmful to people with lung disease. There is thought that this is a contributor, because of the increased amount of inhaled particles into the lung.

Heredity: A condition known as alpha 1-Antitrypsin (AAT) is the only known genetic abnormality that leads to COPD.

This is a disorder of alpha protease, which protects the lung parenchyma from injury, deficiency leads to emphysema. Only lung transplant can correct this deformity.

Aging: As people age, a gradual loss of the lung function normally occurs (4% per year after the age of 40). Additionally, there is a loss of elastic recoil of the lung. The lungs become rounder and smaller, as alveoli numbers and thickness decrease. Osteoporosis and calcification of the costal cartilages decrease the thoracic cage. The cage becomes rigid.

All of these changes result in functional residual capacity (loss of???? Increase in????).

COPD Complications

Pulmonary HTN is caused by constriction of the pulmonary vessels in response to alveolar hypoxia.

Cor Pulmonale: hypertrophy of the right side of the heart, w/ or w/out CHF, from pulmonary hypertension (increased afterload)

S&S right ventricle dilation,
systemic venous congestion,
S3 gallop,
JVD,
hepatomegaly and RUQ (right upper quadrant - abdomen) tenderness,
ascites,
peripheral edema and weight gain.

Acute Respiratory Failure
The client generally develops an infection (may be viral) or acute bronchitis and either waits to long for help, or
succumbs to the illness due to other factors: D/C meds or is non-compliant, has been indiscriminately prescribed sedative and or narcotics and develops failure.

S&S fever, ↑cough, dyspnea, and fatigue. This causes hypoventilation and ↑CO2 levels, which eventually suppresses respiration.
And
Chronic elevation of CO2 means that the respiratory center no longer uses ↑CO2 lvls to stimulate breathing. Instead it uses O2 receptors in the arteries and breathing is stimed by ↓ O2 lvls (hypoxic drive).
If too much O2 is administered, the hypoxic drive is abolished and breathing slows or stops.
Low doses of O2, careful screening for surgery, is a must to avoid this complication.
The client must be artificially ventilated.
hypoventilation and ↑ CO2 levels, which eventually suppresses respiration.

Peptic ulcer disease is increased in COPD patients.
The reason is unknown. It is proposed that the medication therapy is the reason as well as the stressful nature of the disease.
Pneumonia This is the most frequent complications of COPD.

Diagnosis and Studies Key factors in determining the dx (as opposed to chronic asthma) are the pts hx and responsiveness to bronchodilators (smelzer).
History and Physical
ABG's are monitored
↓ PaO2, ↑PaCO2, ↓ ph, ↑bicarbonate levels
Pulse oximetry
Chest x-ray/ CT scan
Reduced maximum voluntary ventilation
Reduced vital capacity
Reduced diffusing capacity for CO2
Increased residual volume (air trapping)
Increased total lung capacity
Increased FRC; forced residual capacity

COPD Treatments

Cornerstone of Treatment

Medications same as asthma
Prevention of secondary illnesses
Compliance with medical treatment
Maintenance or improvement of airway

Prevention of Secondary Illnesses

Good hand washing, Immunizations, Stay away from sick people

Oxygen and Respiratory Care Patients with severe disorders may have home O2.

Oxygen: Supplemental O2 will raise the PP of O2 (PO2) in inspired air.
Low doses, such as NC at 3/l min, or Venturi Mask at low dose.
Only low doses should be used, or the drive to breath is suppressed.
Humidification/nebulizers may be used.
Oxygen Toxicity may result from prolonged exposure to a high PaO2.
Development of O2 toxicity is determined by tolerance, exposure, and dose.
It is believed inactivation of pulmonary surfactant leads to ARDS..
S&S: ↑VC, cough, CP, N &V, paresthesia, nasal stuffiness, sore throat, malaise.

Respiratory Care and Teaching

Pursed-lip breathing
Diaphragmatic breathing
Coughing (huff progressing to cascade)
Chest PT: done to ↑clearing of secretions, or for lobar atelectasis (mucus plug).
Family members may be taught to do this at home
Percussion
Vibration
Postural drainage

Nutrition

Needs high protein, high calorie diet in six small meals a day
Nutritional supplements and Vitamins
Icy cold beverages and dairy may exacerbate COPD patients
Foods with low gas
Using supplemental O2 during eating may increase dietary intake
Fluids 3L/day (unless contraindicated)

Spiriva long-acting bronchodilator

First inhaled COPD medication to demonstrate significant and sustained improvements in lung function for 24 hours with once a day dosing
Not for initial treatment of acute episodes of bronchospasm (ie rescue med)
Has less CV side effects than albuterol (ie: less tachycardia, >BP)
Contraindicated w/ hx of hypersensitivity to atropine, or ipratropium.
HandiHaler has: Dust Cap, Mouth Piece, Base, Piercing Button ,Center Chamber
open the dust cap and expose the mouthpiece.
lift the mouthpiece to expose the center chamber
Insert capsule from blister pack into the center chamber
close the mouthpiece firmly until it clicks, Press the piercing button once
Put mouthpiece to mouth & inhale fully,listen for the capsule vibration,
hold breath as long as comfortable. Repeat this step a second time.
Remove and dispose of the capsule
Periodically clean HandiHaler with running water 24 hours prior to next dose.

Activity

Energy conservation is an important component
Upper extremity movements are very difficult because of the use of accessory muscles, and breathing technique.
OT may be necessary.
Walking is by far best physical exercise for the COPD patient using pursed lip breathing and supplemental O2

Sleep

Adequate sleep is difficult for the COPD patient,
often due to medications side effects, congestion, coughing and wheezing.
Sleep meds are not a choice, as they may suppress respiration.
Position in semi-Fowler's for better sleep

Emotional Support

These clients will need to deal with many lifestyle changes (socioeconomic)
Emotions encountered can include: depression, anxiety, isolation, denial, and dependence.
Expressions by the patient can be cooperative to hostile.
Relaxation techniques may be taught to the client. Hypnosis or meditation may be helpful.

Causes
Smoking
pollution
Resp infxns
Heredity
Aging

Teaching
Pursed-lip breathing
Diaphragmatic breathing
Coughing (huff progressing to cascade)
Chest PT
Nutrition
Rest
QUIT SMOKING

COPD

defined as a disease state characterized by the presence of airflow limitation that is not fully reversible*. The airflow obstruction is generally progressive, and may be accompanied by airway hyperactivity and hyperresponsiveness.

Maintenance Tx/Meds
Cornerstone of Treatment
Medications same as asthma
Spiriva
Prevention of secondary illnesses
Compliance with medical treatment
Maintenance or improvement of airway
Prevention of Secondary Illnesses
Oxygen (low dose) and Respiratory Care
Chest PT
Semi fowlers
No sleep meds
Emotional support
Activity- exercise and rest

Empysema (pink puffers)
Hyperinflation of alveoli causes destruction of alveolar walls and capillary walls, no gas exchange. Narrowed airways, loss of lung elasticity. Expiratory obstruction: Air spaces distend beyond terminal bronchioles-same as asthma

Chronic Bronchitis (blue bloaters)
the presence of chronic productive cough for 3 months in each of 2 years in row when other causes of chronic cough have been excluded. Increased mucus secretion and inflammation. Bronchial walls thicken, bronchial lumen narrows, and mucus can plug the airway. Alveoli adjacent to bronchioles become damaged.

Complications
Pulmonary HTN
Cor Pulmonale
Acute Respiratory Failure
Peptic ulcer disease
Pneumonia

Diagnostics
ABG's are monitored
↓ PaO_2, ↑$PaCO_2$, ↓ ph, ↑HCO_3^-
Pulse oximetry
Chest x-ray/ CT scan
↓ MVV, Reduced VC
↓ diffusion of CO_2
↑ RV, TLC, FRC

S&S
Dyspnea, DOE
Minimal coughing is present with small amounts of sputum
Flattened diaphragm with an increased anteroposterior diameter= barrel chest
DOE
Thin extremities and weight loss
Later: finger clubbing, CO_2 elimination is impaired, resulting in hypercapnia - respiratory acidosis

S&S
Productive frequent cough, often exacerbated by respiratory irritants and cold damp air
Bronchospasm
Frequent respiratory infections
Hypoxemia and hypercapnia from hypoventilation
Cyanosis
Normal weight or heavy set

not able to ↑ gas exchange b/c everything coated ☜ mucus

cannot compensate

TREATMENTS - pretty interchangeable across asthma and COPD but some delineation	
Pneumonia	O2, pulse ox QID & PRN, 3L/d fluid, 1500 kcal min, bedrest w/BSC, HOB ↑, Pulmonary Toilet, suction PRN, CBC QD, CXR Q2-3D Acute: pulse ox maintained 93-94%, VS & I/O Q2H, observe for respiratory fatigue/failure
Asthma **Status Asthmaticus**	O2, pulse ox, BP,P,RR, ABGs, cardiac monitor, IV aminophyline & steroids, SQ Epi, Nebs, bedrest w/ bedpan, IVF, I/O Intubated or trached – ICU, nebs in ventilator, IV methylprednisolone (solumedrol), IV Aminophyline, SQ Epi
Emphysema	Cornerstone: meds, prevent 2° infection, compliance w/ treatment, maintain and improve airway. Lowflow O2 , ↑HOB, activity/rest, no se
Chronic Bronchitis	Same as emphysema
Tuberculosis	Aggressive, at least 2 meds; diet,rest, avoid alcohol,
Pneumothorax	Varied but, establish airway, O2, Thoracentesis, needle decompression, chest tubes, pain control, bld transfusions, CXR, IVF
Atelectasis	Incentive Spirometer, Nebs
Pericarditis	Pericardial tap if severe (therapeutic)
Pleurisy	Give NSAIDS
Empyema	Chest tube, sx drain
Pleural effusion	Thoracentesis (therapeutic and treatment)

MEDICATION – meds are pretty interchangeable across asthma, COPD, but sometimes sources seem to delineate.	
Pneumonia	Abs, nebs w/ beta (Albuterol) & antichol (Atrovent), steroids PO prednisone/IV methylprednisilone, antitussive, expect, analgesic, antipyre
Asthma **Status Asthmaticus**	Rescue meds, Maintenance: glucocorticoids, cromolyn, leukitrienes, theophyline, servent, advair, singulair, atrovent, atropine (EIA) See tx section
Emphysema	Meds same as asthma plus spiriva
Chronic Bronchitis	Meds same as asthma plus Abs (bactrim and zpak)
Tuberculosis	Primary:INH- Isoniazide, rifampin, pyrazidnamid, rifabutin, Nydrazid. Secondary for resistance or pt developes tox to primary (but 2nd mor toxic in general). Aminoglycosides can be used as adjuncts, Also Ciprofloxicin. Antiemitics, antitussives, antipyretics, vitamins (esp C),

LUNG FUNC)N

Compliance refers to lungs ability to expand, elasticity and expandability of lungs and chest. Normal when lungs and chest expand easily when pressure is applie

Increased: lungs have lost elasticity and thorax is over distended – emphysema,

Decreased: lungs and thorax are stiff – obesity, Pneumothorax, hemothorax, pleural effusion, pulmonary edema, atelectasis, ARDS, pulmonary fibrosis

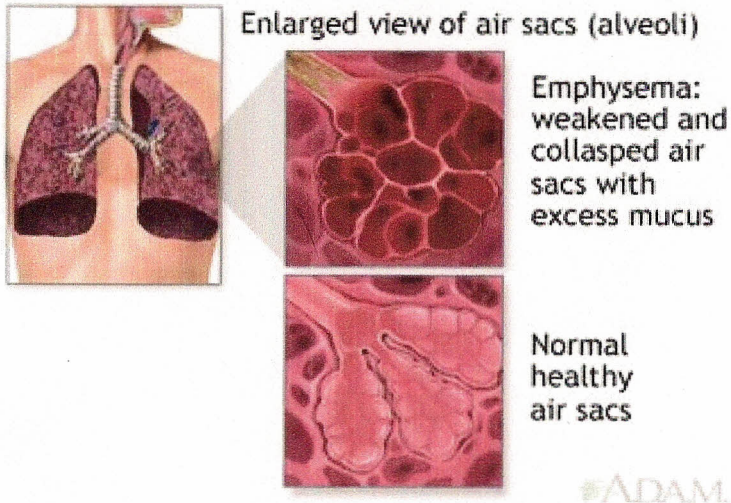
Capacity refers to lung volumes

TLC – Total Lung Capacity	Lung volume after max inspiration	Down in restrictive disease (atelectasis, pneumonia, pneumothor Up in obstructive – trapped air (COPD, Asthma)
VC – Vital Capacity	Max Vol expired after max Vol inspired	Down in atelectasis, pulmonary edema, COPD, pneumonia
FVC – Forced Vital Capacity	VC with max effort	Down in COPD, asthma
FEVt Forced Expiratory Volume w/in specified time FEV1 Forced Expiratory Volume in one second	Volume of expired air with FVC	
So, FEV = maximum air volume expired w/ max force in a specified time frame after a maximum inspiration Or, FEV = FVC w/ in specified time		
FRC - Functional Residual Capacity	Volume in lungs after normal expire	Up in COPD, Asthma

SIGNS & SYMPTOMS –asthma and COPD are all pretty much subject to same S&S depending on stage/exacerbation. Chronic Bronchitis people can't compens for poor ventilation like asthma and emphysema.	
Pneumonia	Sudden fever or gradual, myalgia, fatigue, dyspnea, maybe chills, dry or productive cough, hemoptysis, ↓ compliance, ↓ VC
Asthma **Status Asthmaticus**	WHEEZING, SOB, CHEST TIGHTNESS, COUGH, nasal flaring, ↑BP,P, RR, nocturnal, may be asymptomatic bt attacks w/ norm or abnorm lung fxn Diaphoresis, ↑breath work, cyanosis, **silent chest**, neck vein distention, vent. arrhythmias, pulsus paradox > 40, PEFR< 100, **PCO2> 70**
Emphysema	Dyspnea, usually minimal coughing, barrel chest, thin extremities and wt loss, poss. Bronchospasm, Later: finger clubbing, R. acidosis
Chronic Bronchitis	Productive frequent cough often exacerbated by cold or irritants, freq infections, hypoxemia and hypercapnia, cyanosis, wt: norm-hvy
Tuberculosis	FEVER, HEMOPTYSIS, NIGHT SWEATS, WT LOSS> 10lbs, fatigue, malaise, parenchymal lymph node complex
Pneumothorax	Varied. ARDS, Tension: air hunger, agitation, hypoxemia, cyanosis, hypotension, tachycardia, diaphoresis
ARDS	Interstitial infiltrates, alveolar hemorrhage, atelectasis, ↓ compliance , and refractory (stubborn) hypoxemia
Atelectasis	Insidious. Increasing: dyspnea, cough w/ sputum, diminished breath sounds and crackles, tachypnea, hypoxemia.
ARF	Δ LOC, restlessness, anxiety, confusion. Really low PO2, really high PCO2, and acidotic ph.
Pleurisy	Sharp knife-like pain esp on inspiration

Emphysema

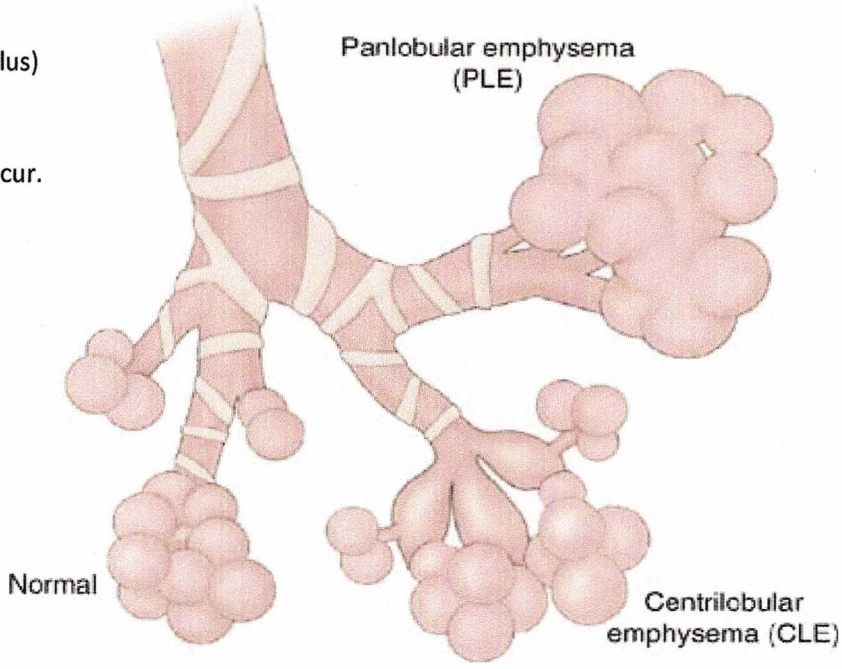
Characterized by destruction of the walls of overdistended alveoli (accelerated by recurrent infection).
Structural changes include:
Hyperinflation of alveoli causes destruction of alveolar walls and destruction of alveolar capillary walls, which causes "dead space" – no gas exchange
Narrowed tortuous small airways
Loss of lung elasticity.
Expiratory obstruction: Air spaces distend beyond the terminal bronchioles.
(same as in asthma)
In later stages of disease, CO2 elimination is impaired, resulting in hypercapnia - respiratory acidosis.
As the alveolar walls continue to break down, the pulmonary capillary bed is reduced in size.
Consequently, resistance to pulmonary blood flow is increased, resulting in cor pulmonale
Congestion, dependent edema, distended neck veins, or pain in the region of the liver suggests the development of cardiac failure.



<u>Two types of Emphysema</u> - A patient can have either type or both types

Panlobular (panacinar)
involves distention & destruction of the whole lobule (respiratory bronchiole, alveolar duct, alveolus)
But there is little inflammatory disease.
There is progressive loss of lung tissue and a decreased alveolar-capillary surface area.
A hyperinflated (hyperexpanded) barrel chest, marked dyspnea on exertion, & wt loss typically occur.
To move air into and out of the lungs, negative pressure is required during inspiration,
and an adequate level of positive pressure must be attained and maintained during expiration.
Expiration becomes active and requires muscular effort.

Centrilobular (centroacinar) This is the more common type of emphysema.
the emphysema primarily involves the central part of the lobule.
Respiratory bronchioles enlarge, and the walls are destroyed.
Frequently, there is a derangement of ventilation–perfusion ratios,
producing chronic hypoxemia, hypercapnia, polycythemia, and episodes of right-sided HF.
This leads to central cyanosis and respiratory failure.
Pt also develops peripheral edema, which is treated with diuretic therapy.
Chronic bronchitis is often associated with this type.



Panlobular bronchioles, alveolar ducts, & alveoli are destroyed, airspaces within the lobule are enlarged.
Centrilobular pathologic changes occur in the lobule, whereas the peripheral portions of the acinus are preserved.

Clinical Manifestations

Dyspnea, DOE
Minimal coughing is present with small amounts of sputum
Flattened diaphragm with an increased anteroposterior diameter= barrel chest
Hypoxemia during activity
Thin extremities and weight loss
Later in the disease finger clubbing

Chronic Bronchitis

Pathophysiology

The presence of chronic productive cough for 3 months in each of 2 successive years in a client in whom other causes of chronic cough have been excluded.
Most times smoke or other environmental factors cause irritation to the airways causing increased mucus secretion and inflammation.
Bronchial walls thicken, bronchial lumen narrows, and mucus can plug the airway.
Alveoli adjacent to bronchioles become damaged
Patient more susceptible to resp infections.
Exacerbations occur most likely during the winter.

Clinical Manifestations

Productive frequent cough,
often exacerbated by respiratory irritants and cold damp air
Bronchospasm
Frequent respiratory infections
Hypoxemia and hypercapnia from hypoventilation
Cyanosis
Normal weight or heavy set