Another nearly pointless class brought to you by Mr. Philosophy.

Final review: 1 of several -

Define the following (per Dr. Stewart)

- Systolic bp – from contraction of the left ventricle
- Diastolic bp – pressure in circulatory system present during the relaxation of the left ventricle
- Mean arterial pressure – average blood pressure between systolic and diastolic. CHF patients if untreated, will have a high mean arterial blood pressure.
- Pulse pressure – difference between sys and dia bps. “Narrow” pulse pressure is a small difference between the two sys and dia. Later stages of shock will cause narrow pulse pressures, though starts out as a very wide difference. (sometimes can’t even measure the lower number.

Renal artery stenosis – if artery to kidney is narrow/stenotic generating narrow pulse pressures like 190/110. Aortic stenosis can also cause this - Kidney clamps down, raises blood pressure.

Know terms and concepts and try to explain phenomenon based on these. Some :straightforward easy dumb questions” like anemia from chronic blood loss most likely results in? iron deficiency … hypochromic . . . would be microchromic (very small) b/c lack of iron causes smaller size, paleness because they cannot carry the O2.

Can also have anemia that is a pernicious or Vitamin B12 deficiency. Need Vitamin B12 Intrinsic Factor which is a stomach enzyme that helps us break the vitamin down. Beets, cruciferous veggies (cabbage and derivatives, leafy greens, radishes of both kinds, etc.) and liver/fish/eggs/cheese.
Chapter 31 – Disorders of Ventilation and Gas Exchange

**Pleuritis and Pleurodynia**

Pain of pleuritis. Slide 40 of [Chapter 29 – Control of Respiratory Function](#).

When you exhale the pleural sac (which adheres in some spots and not in others) will circulate fluid around so that the protective funx of the pleura works properly. When there is an absence of this fluid, the pleura will adhere like mad – patient will try to inhale, but cannot because the pleura is restricting it. This is an example of pleuritis.

Pleuritis is thought generally to be caused by infection, but not always. Pleural lining in pleuritis will be thicker and drier with an absence of fluid generating ability. Pleural fluid is produced in excess in pneumonia, TB, pulmonary abcess and lung cancer. An excess in pleural fluid can be treated by removing the fluids with a needle – a Pleural Tap. This too is a type of pleuritis, presenting with pleuritic pain. It is sharp, intense, worse on inspiration, will keep them from breathing deeply. Can still oxygenate just fine. Typically, will have increased CO2 levels, later will pant more and blow off CO2.

Pleurodynia = pain in the pleura

Many patients with arthritic changes in chest wall may use the term pleuritis – this isn’t really pleuresy. This is a musculo-skeletal pain. Ironically, many people who really have pleuritis will assume musculo-skeletal reasons for the pain they feel.

**Pleural Effusion**

Pleural fluid is normally very little – 10-20cc’s -- and is really a thin film over the pleural surface which gives enough lubrication to keep the sac from sticking to the lungs. Two types:

- **Transudate**
  Water that gets into the pleural space from pulmonary edema.

- **Exudate**
  Patho process of lungs causing the production of pleural fluid. Has protein, WBCs, pus contained within the fluid.

Empyema – refers to pus in the pleural cavity
Chylothorax – lymph in the thoracic cavity
Hemothorax – blood in the thoracic cavity. Usually due to trauma or something like lung cancer or TB.
Pneumothorax

Pneumothorax is a rupture of the lung caused by 1) rupture of a “bleb” (blister) on the surface of the lung, 2) chest wall trauma or 3) lung trauma. Air escapes into the pleural or “potential space.”

There are several types:

1. **Spontaneous pneumothorax**
   - Air accumulates in the pleura and the lung collapses until there is no longer a gradient can no long force air in or until the collapse causes the leak to seal. Emphysema is the most common cause of spontaneous pneumothorax.
     a. **Primary**
        - In otherwise healthy people from a bleb rupture. The cause of the bleb is unknown. More common in tall men. Go figure.
     b. **Secondary**
        - More serious – occurring in people w/underlying lung diseases such as emphysema, asthma, CHF, etc. which further exacerbates existing compromised lung function.
     c. **Catamenial**
        - Occurs during menses and is related to endometriosis in the pleura or diaphragm.

2. **Traumatic pneumothorax**
   - Can be penetrating (like a bullet wound or knife wound) or non-penetrating (broken rib). May also present with a hemothorax…duh! The severity of this type depends upon the nature and severity of the injury.

3. **Tension pneumothorax**
   - A “sucking chest wound.” Air accumulates rapidly in the pleura but can’t escape. It continues to accumulate, shifting the mediastinum to the right, compressing the vena cava and impairing the venous return.

4. **Iatrogenic**
   - “Iatrogenic” means an injury caused by a doc or healthcare provider/facility or unfavorable result of a medical procedure. Could come from a thoracentesis, acupuncture, biopsy. Could also be from a difficult intubation or aggressive ventilation resulting in damage to the alveoli, trachea, bronchi, etc.

Manifestations depend upon the degree of the pnthx. Can be:
- Ipsilateral (same side) pain, dyspnea, tachypnea, increased heart rate
- Hypoxia
- Tension pnthx leads to mediastinal shift to opposite side.

When you percuss it, it sounds resonant or hyper-resonant. Auscultation (listening usually w/stethoscope) = can’t hear the breathing or lowered breath sounds over the area.
Asthma and Bronchitis

Both are referred to as obstructive lung diseases. Lung movement/expansion is obstructed.

In asthma there is a precipitating factor – allergen or smoke or toxin or poison which triggers an inflammatory process. Book focuses on mast cells and degranulization which means they are releasing histamines. For whatever reason, once inflammation is triggered = swelling in mucosal airway. Passages are lined by circular smooth muscle – when constricted gets very narrow. Inflammation triggers broncho-constriction…to keep cooties out of the air sacs. The primary job of the airways is to 1) get gases down to the arterioles and air sacs and 2) to keep cooties out of the air sacs.

Asthma is thus a reactive airway disease. There are a variety of triggers. The inflammatory component of this is the major one – broncho constrx is a 2ndary phenomenon and almost never happens without the inflammation response. Antihistamies don’t really help – most are H2 blockers, one type of histamine receptor. Many types of receptors are involved in the response, so not enough is blocked to help with asthma.

Inflammation:
Current asthma therapies are thus gauged toward supressing inflammation. Corticosteriods are the most common in use now. Unfortunately, corticosteroids come with very bad side effects. Most of the problems come from the fact that they are very non-specific and generalized – sort of like carpet bombing. Singulair on the flip side, only gets one facet of the asthma phase – blocking of leukotrienes.

Though asthma is complex and a lot of crap happens therein, basically: there’s an allergen triggering a release of IgE antibodies and then an inflammatory cascade which ends in the restriction of the airways.

Bronchoconstriction:
In asthma, there is a thickening of the mucosal lining by smooth muscle thickening. When this occurs can narrow the linings even more upon inflammation. Eventually even the outside of the airway will be affected and then the condition can be considered to be chronic bronchitis. Chronic bronchitis in children is actually indistinguishable from asthma. If you escape asthma as a child, you have a better prognosis if you get it as an adult because you don’t have that long-term thickening.

Symptoms and Signs of Asthma:
- Mild to severe to fatal
- Wheezing, tightness, dyspnea, fatigue, cough (which is not effective because it cannot clear the secretions)
- Symptoms are worse at night
- Prolonged expiration as compared to inspiration.

Trapped air leads to hyperinflation of the lungs. The decreased effectiveness of the ventilation process results in hypoxia (low oxygen) and hypercapnia (abnormally high CO2 in the blood). And remember that high CO2 levels cause a feeling of needing breath and can really make you panic, dude. Breathe slowly and deeply to breathe off the CO2.

Things causing Asthma:
- Atopic (allergens of various kinds). Can also express along with hay fever, eczema, hives, etc.
Other: exercise, cold, infections, inhaled irritants of all kinds, NSAIDs and aspirin. Also significant is GERD, sulfites, emotional factors, etc.

“Cardiac Asthma” is not asthma at all, but pulmonary edema and is CHF related. Peak Flow Meters allow you to measure of the volume of air that can be moved with a forceful exhale. PEV is the prolonged expiratory volume.

**COPD (Chronic obstructive pulmonary disease)**

Basically chronic bronchitis (acute can be infectious and noninfectious). A bronchitis patient may thusly be on antibiotics or not. Sadly, treating chronic bronchitis with antibiotics actually makes it worse. COPD is a group of disorders characterized by chronic and recurrent obstruction of airflow. COPD is progressive and is accompanied by inflammation. Most cases of COPD are related to smoking.

COPD is an adult disease. Some ppl with it had asthma as a child, however. Fundamentally the same disease process: thickening of airways, reactivity of airways, excessive production of mucus which is a result of the inflammatory process that underlies the disease. Asthma has a stronger autoimmune component and is far more multifaceted. In COPD it is less complex, but worse and more progressive.

COPD has 2 stages of the process: 1) Emphysema, 2) chronic bronchitis. Some ppl are predisposed to losing their lung elasticity quickly and emphysema is the result. There is not necessarily a causal relationship however between asthma and emphysema though they are both characterized by the inflammatory process and both cause reactive airways (so the treating meds are very similar).

COPD (emphysema) is a loss of lung elasticity with abnormal enlargement of the air spaces below the terminal bronchioles and a destruction of the alveolar walls and capillary beds. There is a breakdown of elastin and other alveolar components by enzymes released as an inflammatory reaction to smoking. Actually, the destruction of the alveoli reduces the surface area available for gas exchange, causes loss of elasticity, and airway collapse.

Impaired airflow of expiration traps air, causing a loss of ventilation-perfusion. The result is “puffing”. Again, this patient thinks he or she needs more air and puffs – hence the term “pink puffer” for a COPD patient.

COPD (bronchitis) patients are termed as “blue bloaters.” Again, the airways are obstructed by inflammation of major and small airways. Edema and hyperplasia of the submucosal glands cause excess mucus production which is hard to clear. These folks look blue.

Most COPD patients have elements of both of the above. See slide 72. These people have right sided heart failure.

**Pneumoconiosis**

= occupationally caused lung disease. See slide 74.