

Nsng Mngmnt of Adults with Respiratory Disorders Part I (lecture)

- ▼ The "I"s...watch for these! One or more of these will be at the physiological basis of EVERY disease process!
 - Ischemia
 - Inflammation
 - Immune alteration
- ▼ Respiratory Failure
 - The primary role of the respiratory system is to deliver O₂ and get rid of CO₂. Of special importance is the concept of system interdependence...when respiratory failure occurs it can be because of other systems which all need to work well together: CNS, pulmonary, heart, vascular. Problems with any of these four systems can cause problems with the respiratory system. For example, if the heart isn't pumping out enough blood (or the pt is anemic), then this is going to affect oxygenation. If the medulla is damaged, the respiratory centers of the brain are going to be affected, etc...
- ▼ Acute Respiratory Failure is defined as the inability of the body to meet tissue O₂ needs or CO₂ removal needs.
 - PaO₂ < 50 mm Hg ---OR---
 - PaCO₂ > 50 mm Hg on room air
 - It is diagnosed by ABGs...and please remember, that the clinical symptoms do not necessarily tell you anything about the severity of the problem. For example, a COPD patient would have different symptoms than a healthy individual.
 - NOTE: at first, the problem with oxygenation will show that the CO₂ may be ok while the O₂ is low. This doesn't last though! Eventually the CO₂ will rise!
- ▼ There are two types of ARF: arterial hypoxemia and ventilatory or hypercapnic (high CO₂). You will usually see both types together in clinical cases (see below)!
 - **Arterial hypoxemia** is defined as a lower than normal amount of oxygen dissolved in the plasma. Recall that each RBC can hold 4 oxygen molecules. So, the hypoxemia occurs when the body cannot get O₂ into the bloodstream because of an alveolar or capillary membrane issue. Examples of this would be pulmonary edema, ARDS, drowning, pulmonary embolism, lung tumors and bleeding. RECALL THAT HYPOXEMIA relates to blood (emia = blood)
- ▼ Levels of hypoxemia
 - Mild hypoxemia = PaO₂ 60-80 mm Hg
 - Moderate hypoxemia = PaO₂ 50-60 mm Hg (the SaO₂ would be around 88-90% at this point)
 - Severe hypoxemia = < 50 mm Hg (pt would be on 100% non-rebreather)
 - Life threatening hypoxemia = < 40 mm Hg (pt would be on the 100% NRB and intubated!)
 - NOTE: when you see levels below 50, you need to move quickly!
- ▼ The cause of arterial hypoxemia:
 - Basically, the issue here is alveolar hypoventilation. The alveoli is receiving little or NO oxygen, but has normal perfusion (the blood is flowing right on by as though nothing were wrong!). So, what happens? The blood comes by but it can't pick up any more O₂...it's going to keep on trucking to the left side of the heart and get pumped out into circulation with less and less O₂ each time. This is called "SHUNT" and it's a very bad thing! So why can't the alveoli receive any oxygen? Well, it is either collapsed, blocked (by a mucus plug maybe) or filled with fluid (pulmonary edema).
 - V/Q mismatch is another important concept to understand. V = ventilation, and Q = perfusion. If you have "low VQ", this means the ventilation is the problem and perfusion is ok. If you have "high VQ", this means the ventilation is OK, but the perfusion is a problem.
- ▼ What happens when an alveoli is blocked by a mucus plug?
 - Glad you asked! The Pore of Kohn is this great little "secret passageway" that connects one alveolus to another. When the alveolus is blocked, the pressure in the full-of-air alveolus next door causes the Pore of Kohn to open, bringing oxygen into the collapsed alveoli. We can open these secret passageways by having our pts deep breath, use IS, cough. A big breath on the IS should be 1000-1200 ml. If we needed to remove a mucus plug, we could do this via bronchoscopy or by encouraging coughing. I think I would prefer coughing!
- ▼ The cause of hypoxemia

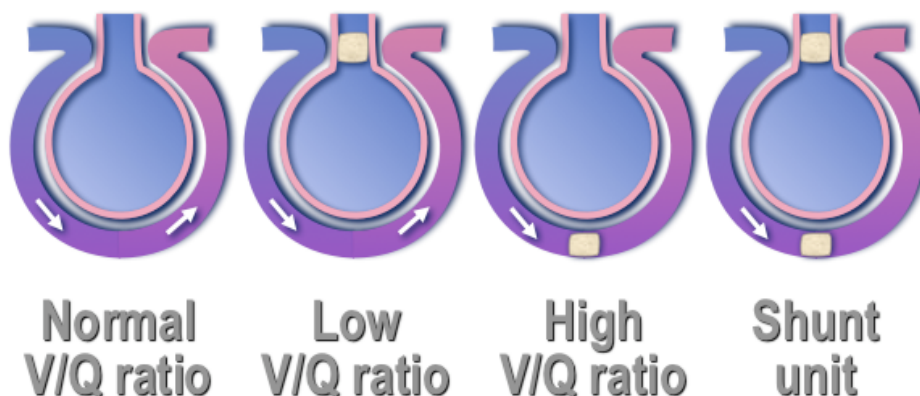
- Alveolar Dead Space means there is nowhere for the O₂ to go. The alveoli are fully ventilated, but blood is blocked in the capillary (so there is no perfusion) and the alveolus is unable to participate in gas exchange...there is no blood flowing through for it to exchange gasses with! Severe "wasted" ventilation is referred to as "dead space". A shunt can occur from this as well...the blood going to the heart for systemic circulation has less and less oxygen in it. This could be caused by a pulmonary embolism and is this a high or low V/Q ratio? It is a HIGH V/Q ratio! Good job!

▼ ***The signs and symptoms of hypoxemia *** these are core pulmonary assessments! muy importante!

- Increased RR
- Increased HR
- Dyspnea
- Agitation
- Increased WOB

▼ Let's review the range of ventilation to perfusion ratios!

Alveolar air flow & Capillary blood flow



- Normal V/Q: blood is flowing and the alveoli are full of yummy air (ventilation and perfusion are good!)
- Low V/Q ratio: ventilation is low, but the blood is flowing (could be a mucus plug in the alveolus)
- High V/Q ratio: ventilation is good, but the blood flow is blocked (pulmonary embolism)
- Shunt unit: alveolus plugged AND blood flow clogged

- Ventilatory or hypercapnic ARF** occurs when the body cannot get CO₂ out of the bloodstream due to a pulmonary structure issue such as COPD or status asthmaticus, or CNS issue such as CNS depression or neurological injury. When CO₂ builds up this is hypercapnia...a bad thing! If you wanted to stop an asthma attack, what would you give? Bronchodilators and mast cell stabilizers to prevent the release of histamine!

▼ The signs and symptoms of hypercapnia:

- lethargy (early sign)
- Decreased LOC
- Decreased RR
- Low tidal volume (shallow breaths)
- NOTE: As the CO₂ builds up in the system, the pt transitions between the agitation stage of hypoxemia to looking like they are just worn out...lethargy is that sign that they are wearing out and hypercapnic!

▼ Signs of respiratory distress...catch it before it becomes respiratory failure or respiratory arrest!

- Increased WOB
- Increased HR
- Increased RR
- Increased use of accessory muscles
- Tripod position
- Nasal flaring (occurs when pt is trying to lengthen exhale)
- Unable to speak in full sentences. If your pt can only get a few words out, this is a bad thing!

▼ **Hypoxemia + Hypercapnia = respiratory distress and failure.**

- These two disease states most commonly occur together in respiratory distress and failure. How does this work? Well...the PaO₂ will decrease first causing the pt to be very agitated (plus cause all the other symptoms of

hypoxemia outlined above). Agitation and whatnot (HR, RR, WOB) increase O₂ demands, the RR increases to try to meet those demands and your pt will be blowing off their CO₂...so initially their PaCO₂ levels will DROP. However, this can't go on forever and the pt will get tired. When they get tired, the RR decreases and the CO₂ levels go up. The result is low PaO₂ and high PaCO₂.

▼ **Tissue Hypoxia = another bad thing that happens with ARF**

▼ Tissue hypoxia is defined as lower-than-normal O₂ delivered to the cells...since all cells need O₂ to live, this leads to organ and system failure. This can be due to a supply problem or a demand problem.

▼ Supply problems can be caused by any of the four systems mentioned previously:

- CNS system: drugs cause CNS depression, and neurological injury would affect respiration as well
- Pulmonary system: hypoxemia...not enough O₂ in the blood d/t pneumonia or asthma (for instance)
- Cardiac system: decrease cardiac output is going to mean less blood (and thus less oxygen) is getting to the tissues. This can occur with hypovolemia or cardiomyopathy.
- Vascular system: abnormal hemoglobin such as in sickle cell disease.

▼ Demand problems occur when O₂ demands go up and they just can't be met:

- Fever increases oxygen requirements...so if your pt has a fever this could put them into even more trouble. Fever levels of concern would be > 102 in an otherwise healthy adult, > 101 in an older adult, > 100 in an immunocompromised pt
- Infection
- ADLs. Bed baths increase O₂ demands the most out of any ADLs. Provide rest for pts with respiratory issues.
- Agitation on mechanical ventilation

▼ Effects of tissue hypoxia are multi-system

- Cardiovascular effects: tachycardia, hypertension, dysrhythmias, polycythemia (HR above 120-ish is hypoxia!)
- Respiratory effects: tachypnea, hypoxemia (get the ABGs!), cyanosis is a late sign...if cyanosis occurs, you missed something earlier on! Bad nurse!
- Renal effects: low urinary output. Recall that the kidneys get 25% of cardiac output...so any decrease in urinary output (below 0.5mg/kg/hr) is going to tell you that the organs are not being perfused adequately.
- Neurological: anxiety and agitation (early signs), confusion, headache, weakness/drowsiness, double vision, impaired judgment, coma

▼ So, what are you going to do about it? TREAT THE CAUSE!

- Low O₂ means the pt gets aggressive O₂ therapy. Aggressive means 100% non re-breather most likely
- If the pt has high Co₂, you need to increase the rate and depth of breathing...get them up and moving!
- Frequently, patients have both problems, and this is trickier. You wouldn't want to ambulate a pt with low O₂, because that would just increase their oxygen demands...use your critical thinker!

● **Pulmonary Embolism = yet another very bad thing**

● A pulmonary embolism is a blockage of the pulmonary artery from a thrombus that has come from the systemic veins. Sadly, PEs account for 100,000 deaths in the U.S. each year. The most common site of origin are the deep veins of the pelvis. Pulmonary embolism is a MAJOR RISK FACTOR for surgical patients, especially those who have had ortho surgery (50% risk of DVT) and other surgeries are at 30%. Make sure you reference the DVT prevention table...pretty sure she is going to ask test questions about this!

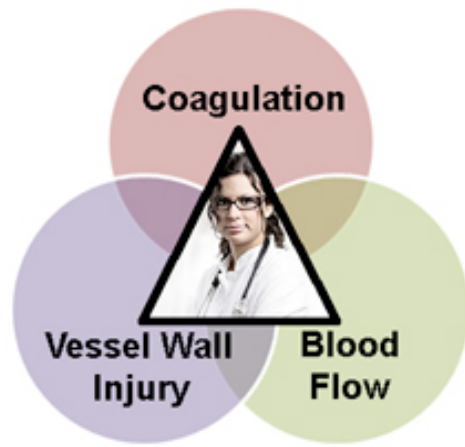
▼ Classifications of PE: acute and massive

- Acute PE is a partial occlusion. this would be a PE that blocks < 2 lobes or < 50% of lung volume.
- A massive PE blocks 2+ lobes or > 50% of lung volume...it is a complete blockage of one or both of the major pulmonary arteries.
- RECALL the route of a PE: IVC to the SVC to the RA (where it could cause dysrhythmias) to the PA where it can block one lung, both lungs or scatter and block lots of smaller vessels.

▼ What are the big risk factors for a PE? Remember Virchow's Triad?

Virchow's Triad

- 1) Blood flow/venous stasis
- 2) Altered coagulability
- 3) Vessel wall damage



Virchow's Triad

- 1) Blood flow/venous stasis
- 2) Altered coagulability
- 3) Vessel wall damage

- Blood flow/venous stasis problems can be caused by: obesity, immobility, lower limb paralysis
- Altered coagulation risk factors are cancer, arterial fibrillation, hx of DVT
- Vessel damage risk factors are trauma, bone fractures, sepsis, atherosclerosis, orthopedic surgery (50%), general surgery (30%).
- ▼ What happens in a pulmonary embolism?
 - Blood flow distal to the embolus is eliminated. Not only does this mean those alveoli do not get perfused...it also means that lung tissue gets necrotic. Lung compliance goes down, the surfactant drops which leads to unequal gas distribution, WOB goes up, and the alveoli stiffen. Pulmonary hypertension occurs (which I believe can lead to dropping BP d/t decreased cardiac output) In general, not a good situation at all.
 - ▼ Clinical findings...a SUDDEN ONSET of:
 - Dyspnea, tachypnea
 - Apprehension, diaphoresis, syncope
 - Tachycardia, chest pain
 - Cough, hemoptysis
 - ▼ Diagnosis of a PE
 - Physical exam with hx...find out if the pt has any risk factors
 - ABG (low O₂, initial low PaCO₂, then increasing PaCO₂)
 - Doppler ultrasound will show presence of DVT
 - Spiral CT is the best method...it can show the blocked area clearly
 - V/Q scan is an older test...not as valid
 - Pulmonary angiogram (dye injected into pulmonary vasculature)
 - ▼ What do you do about it? PREVENT IT!
 - ▼ Nsg interventions would be to screen pts for DVT/PE risk, ambulate pt, educate pt on use of SCDs, and making sure MDs orders are implemented. (see DVT prevention table!)
 - High risk pt (ortho or major trauma) will get LMWH
 - Moderate risk pt (ill, post-op) will get low-dose unfractionated heparin or LMWH
 - Low risk pt that have a high risk of bleeding will get SCDs, TEDs
 - ▼ What do you do if you SUSPECT a PE?
 - Take a thorough respiratory assessment...report onset of symptoms IMMEDIATELY and administer increasing O₂ IMMEDIATELY. The goal is to get O₂ sats > 94%.
 - To treat the PE...you want to make sure the pt has good pain control so they can breath deeply...use narcotics and NSAIDS. You will also make sure the pt is on heparin therapy to prevent further clots (continuous IV infusion). You will adjust this dose according to PTT...goal is to have it at a therapeutic level which is 2 to 2.5x the normal PTT. You could also do thrombolytic therapy to lyse the clot (this is done in Interventional Radiology)...note that you cannot do this on a recent surgery patient...risk for bleeding is too high! You can place filters surgically and also do a surgical embolectomy (removal of the embolus).