Respiratory Failure and ARDS

- Respiratory dysfunction refers to the failure of gas exchange with decrease in arterial oxygen tension PaO2 lower than 60 mmHg (hypoxemia)
- It may or may not accompany hypercapnia, a PaCO2 higher than 50 mmHg (decreased CO2 elimination)
- Based on the PaCO2 we divide it into 2 types
    - Type 1 : Hypoxemic Respiratory Failure : PaO2 < 60 mmHg with a normal or low PaCO2
    - Type 2 : Hypercapnic respiratory failure : PaCO2 higher than 50 mmHg and PaO2 lower than 60 mmHg
- Respiratory failure may be further classified as either acute or chronic
- Acute respiratory failure
    - Characterized by life threatening derangements in arterial blood gases and acid base status
    - Acute hypercapnic respiratory failure develops over minutes to hours therefore pH is less than 7.3
    - can be a side effect of a drug like diazepam or morphine which cause respiratory arrest or can be caused by PE
- Chronic respiratory failure
    - Less dramatic and may not be as readily apparent (patients can be asymptomatic but hypoxic)
    - Develops over several days or longer, allowing time for renal compensation and an increase in bicarbonate concentration therefore pH usually is slightly decreased
    - presence of clinical markers of chronic hypoxemia : pulmonary hypertension, polycythemia or corpulmonale
- The distinction between acute and chronic hypoxemic respiratory failure cannot readily be made on the basis of arterial blood gases
- Alveolar oxygen tension PAO2 remains close to 100 mmHg, while alveolar carbon-dioxide tension PACO2 is maintained close to 40 mmHg
- There is a small difference of 5-10 mmHg between Alveolar (A) and arterial (a) oxygen tension because around 2% of the systemic cardiac output bypasses the pulmonary circulation (physiologic shunt) and is not oxygenated .. the Resulting mix of a small amount of deoxygenated blood makes the PaO2 slightly lower than PAO2
- A normal A-a gradient is about < 10 mmHg
- If the A-a gradient is normal, it means there is no defect in the diffusion of gases
- The A-a gradient helps to outline the different causes of respiratory failure
- At a steady state the rate of CO2 production within the body is constant

- The PACO2 depends on and is inversely proportional to the ventilation so the increased ventilation will lead to decreased PACO2 and decreased ventilation will increase PACO2
- PAO2 depends on the concentration of inhaled oxygen FIO2 and PACO2 as in the following equation :
    - PAO2 = FIO2 × (PB – PH2O) – PACO2 / R
        - PAO2 : Alveolar PO2
        - FIO2 : Fractional concentration of O2 in inspired gas : at room air it is 21%
        - PB : Barometric pressure
        - PH2O : water vapor pressure at 37°C = 47
        - PACO2 : Alveolar PCO2
        - R : Respiratory exchange ratio = 0.8
    - any oxygen supply will also be added
- high altitudes reduce O2 concentration in the age
- There are five mechanisms of hypoxemia and respiratory failure
    - Diffusion Impairment : type 1 failure
    - Hypoventilation : type 2 failure
    - High Altitude : type 1 failure
    - Pulmonary Shunt : type 1 failure
    - Ventilation Perfusion V/Q Mismatch : type 1 failure
- Pulmonary shunt (right to left shunt)
    - The venous deoxygenated blood from the body enters the left side of the heart and systemic circulation without getting oxygenated within the alveoli (pulmonary circulation did not get rid of the CO2)
    - So shunt refers to normal perfusion (normal blood supply) but poor ventilation (failure to exchange gases with the incoming deoxygenated blood)
    - so The V/Q ratio is or near to zero
    - The A-a gradient increases as deoxygenated blood enter the arterial systemic circulation & thus decreasing PaO2
    - Increasing the oxygen concentration does not correct the hypoxemia as the blood will bypass the lungs no matter how high the oxygen concentration
    - This failure to increase PaO2 after oxygen administration is a very important point and helps with a differential diagnosis between impaired diffusion and other causes of hypoxemia that resolve with supplemental oxygen.
    - in atelectasis the collapsed lung is not ventilated and the blood within that segment fails to oxygenate
    - In cyanotic heart diseases the blood from right side bypasses (shunts) the lungs and enters the left side causing hypoxemia and cyanosis

    - other causes : pneumonia / ARDS / Congenital problems
- Ventilation Perfusion V/Q Mismatch
    - The V/Q ratio in normal individuals is around 0.8 but it alters if there are significant ventilation or perfusion defects
    - The decreased V/Q ratio < 0.8 may occur either from decreased ventilation (airway or interstitial lung disease) or from over perfusion
        - In these cases the blood is wasted because it fails to properly oxygenate (In extreme conditions it will behave as a pulmonary shunt)
    - The increased V/Q ratio > 0.8 usually occurs when perfusion is decreased (a pulmonary embolism prevents blood flow distal to obstruction) or over ventilation
        - The air is wasted in these cases and is unable to diffuse within the blood (In extreme conditions the alveoli will act as dead space and no diffusion of gases occurs)
- Diffusion Impairment
    - There is a structural problem within the lung
    - There may be decreased surface area as in emphysema or increased thickness of alveolar membranes as in fibrosis and restrictive lung diseases that impairs the diffusion of gases across the alveoli, leading to an increased alveolar arterial gradient
    - the alveolar PO2 will be normal or higher, but arterial PO2 will be lower
    - The greater the structural problem, the greater the alveolar-arterial gradient will be
    - the diffusion of gases is directly proportional to the concentration of gases therefore increasing the concentration of inhaled oxygen will correct PaO2 but the increased A-a gradient will be present as long as the structural problem is present
- High Altitude (Low inspired FiO2)
    - At high altitudes, the barometric pressure PB decreases, which will lead to decreased alveolar PO2 as in the equation
    - The decreased alveolar PAO2 will lead to decreased arterial PaO2 and hypoxemia, but the A-a gradient remains normal since there is no defect within the gas exchange processes
    - Under these conditions, additional oxygen (increasing the FIO2) increases the PAO2 and corrects the hypoxemia
    - When a person suddenly ascends to the high altitude, the body responds to the hypoxemia by hyperventilation, causing respiratory alkalosis and The concentrations of 2,3-diphosphoglycerate (DPG) are increased, shifting the oxygen-hemoglobin dissociation curve to the right
    - Chronically the adjustment takes place and the body responds by increasing the

oxygen carrying capacity of the blood (polycythemia) and The kidneys excrete bicarbonates and maintain the pH within normal limits
    - patients at high risk to develop pulmonary edema
- Hypoventilation (hypercapnic respiratory failure)
    - The minute ventilation depends on the respiratory rate and the tidal volume, which is the amount of inspired air during each normal breath at rest
        - Minute ventilation = Respiratory rate x Tidal volume
        - The normal RR is 12 breathes per minute and the normal tidal volume is 500 mL Therefore the minute respiratory volume normally averages about 6 L/min
    - Occurs when there is a decrease in the respiratory rate or tidal volume so that a lower amount of air is exchanged per minute
    - There will be decreased oxygen entry within the alveoli and the arteries, leading to decreased PaO2
    - The PaCO2 is inversely proportional to the ventilation Hence hypoventilation will lead to increased PaCO2
    - The A-a gradient will be normal and less than 10 mmHg since there is no defect in the diffusion of gases
    - In these cases, increasing the ventilation or increasing O2 concentration will correct the deranged blood gases
- High PaCO2 with also high A-a gradient this means patient has both hypoventilation on top of another mechanism
    - example : asthmatic hypoxic patient - high A-a gradient - got better with O2 - V/Q mismatch - after a while the patient became tachypnic and required more oxygen - high PaCO2 - hypoventilation replaced the V/Q mismatch due to muscle fatigue
    - another example : giving a patient with CO2 retention like COPD that got into a car accident drugs like morphine for pain (from diffusion impairment to hypoventilation since morphine affect respiratory center
- Pathophysiology of Hypercapnia
    - Hypercapnia occurs when PCO2 increases to more than 50 mmHg
    - The PACO2 depends on and is inversely proportional to ventilation, so decreased ventilation will cause increased PACO2 and vice versa
        - $PaCO_2 = VCO_2 \times K/VA$
    - Therefore hypercapnia along with hypoxemia (Type 2) occurs due to conditions that decrease ventilation
- Symptoms of hypercapnia
    - CNS : narcosis / confusion / drowsiness / dimmed sight
    - sweating and muscle tremors with warm hands

    - shortness of breath with tachycardia and high blood pressure
- warm sweaty hand : CO2 retention / cold dry hand : cyanosis
- Treatment of Respiratory Failure
    - The first goal is to correct hypoxemia and by maintaining an PaO2 of 60 mmHg or arterial oxygen saturation SaO2 greater than 90%
    - providing supplemental oxygen and mechanical ventilation which is provided by facial mask or by tracheal intubation
    - Specific respiratory failure treatment depends on the underlying cause
        - steroids and bronchodilators for COPD and asthma
        - antibiotics for pneumonia
        - heparin for pulmonary embolism
- In fixing the O2 levels
    - Type 1 failure : give O2 to reach SaO2 above 95%
    - Type 2 failure : give O2 to reach SaO2 between 88-92% because giving high O2 levels will lead to more CO2 retention

Acute respiratory distress syndrome ARDS

- A rapidly progressive non cardiogenic pulmonary edema that initially manifests as dyspnea, tachypnea, and hypoxemia, then quickly evolves into respiratory failure (right heart catheterization is normal)
- 3 main criteria to diagnose ARDS based on timing of symptom onset (within one week of known clinical insult or new or worsening respiratory symptoms)
    - Bilateral opacities on chest imaging that are not fully explained by effusions, lobar or lung collapse or nodules
    - The likely source of pulmonary edema is respiratory failure not fully explained by cardiac failure of fluid overload (patients might be fluid depleted)
    - Oxygenation : measured by the ratio of PaO2 to fraction of inspired oxygen FiO2 (P-F ratio)
- Severity : based on P-F ratio
    - Mild : P-F ratio between 200-300 mmHg
    - Moderate : P-F ratio between 100-200 mmHg
    - Severe: P-F ratio is less than 100 mmHg
- ARDS often must be differentiated from pneumonia and congestive heart failure which typically has signs of fluid overload
- ARDS is responsible for one in 10 admissions to ICUs and one in four mechanical ventilations

- In hospital mortality for patients with severe ARDS ranges from 46-60%
- Most cases of ARDS in adults are associated with pneumonia with or without sepsis (60%) or with non pulmonary sepsis (16%)
- Chest radiography shows bilateral air space opacification and lack of obvious vascular congestion
- Treatment
    - mechanical ventilation, prophylaxis for stress ulcers and venous thromboembolism, nutritional support, and treatment of the underlying injury
    - Prone positioning is recommended for some moderate and all severe cases