

PHYSIOLOGY:

- Normally we have 300-600 million alveoli
- Respiratory (alveolar) minute ventilation = Respiratory rate * Alveolar air (tidal volume)
- Now why is Alveolar PO₂ equal to Arterial PO₂ ? It's all about the Volume
- if we need more oxygen , making the diffusion easier will not help because it is already easiest possible
- Normal RR is around 12 , and normal TV is 500ml
- PO₂ drop from 160mmHg to 150mmHg is due to additional water vapor in the Airways
- Recoil force : it results from the elastic properties of the lung tissue
- stretch the lung = expanding it = increase the compliance
- Airway resistance is very small and negligible because we need very small driving force to overcome it”.
- Physiologically, Because of the huge cross sectional area of the 15th generation and beyond, the airway resistance in them is almost zero, and most of the resistance resides in the largest airways.
- In pathological condition airway resistance increases, and this increase comes from the small airways because the large airways are supported by cartilage which makes them non collapsible.
- $R = 1/G$, $G=1/R$; where R equals resistance and G equals conduction.
- intrapleural pressure ranges between negative values (stretching forces). But if it becomes positive -due to some reasons- it will act as a collapsing force (aiding the lung recoil force to collapse the alveoli
- too much accumulated air inside the chest (Barrel chest).
- Patients with partially obstructed lungs face more difficulty during expiration.
- emphysema is associated with right heart failure, this is called Cor Pulmonale
- With aging E.R.V decreases & R.V increases.
- Vital capacity (V.C): the total air we can inhale after an active exhalation. $V.C = E.R.V + T.V + I.R.V$
- In order to measure RV, we use the Helium dilution method
- pulmonary vascular resistance increases due to :
 1. Destruction of capillaries.
 2. Hypoxemia causes pulmonary artery vasoconstriction which will increase pressure
- FVC = forced lung capacity
- forced lung capacity (forced expiration) it is the same concept of forced vital capacity, which means how actually you exhale air, while vital lung capacity means how normally you exhale.
- bronchioles have B₂ receptors which induce relaxation by epinephrine and nor epinephrine and induce contraction by acetylcholine, leukotrienes and histamine
- alveolar recruitment; that's mean the opening of one alveolus will help in the opening in the second alveolus (they help each other), in deflation there is no such thing.
- When you fill your lungs with saline you will only have to overcome the elastic fibers because there is no surface tension
- surfactant lowers the surface tension
- Baby to diabetic mother has higher probability to develop IRDS, and if the first baby has IRDS the chance for the second baby to have IRDS is high.
- Dexamethasone will increase the production of surfactants.
- Infant respiratory distress syndrome (IRDS) : A pre mature baby with no sufficient surfactant and a huge collapsing force of his lungs
- pulmonary capillary wedge pressure : the pressure measured by wedging a pulmonary catheter with an inflated balloon into a small pulmonary arterial branch .it should be less than 18

- mmHg , if it was higher then it is left ventricular(heart)failure
- If this P_{O_2} is less than 200 then this is ARDS
 - If it is between 200-300 then this is Acute lung injury which is a precursor of ARDS
 - If it is more than 300 then NO ARDS
 - minimal volume of the lung (150 ml) , after that neither collapsed nor stretched
 - what is the resting volume of the thoracic cavity? The volume at which the thoracic cavity loses its tendency to expand. this volume is equal to 75% of TLC =4.5 L out of 6 L.
 - what do we call the resting volume of the thorax-lung system ? We call it FRC (functional residual capacity)
 - TV = 500 ml = 0.5L
 - if the system is tending to collapse then the expiration is passive while if the system is tending to expand the inspiration is passive .
 - Remember lower FRC means lower tendency to collapse like in restrictive lung disease
 - higher FRC means high compliance
 - normal V/Q (ventilation perfusion ratio)= $4.2/5=0.84$
 - alveolar P_{O_2} = P_{O_2} inspired- (P_{CO_2}/R) where R is the respiratory exchange ration that equals = CO_2 production/ O_2 consumption = 0.8
 - since blood is more dense than air; it is affected more by gravity and that's why the regional differences in perfusion are greater than ventilation)
 - the apex has more ventilation than perfusion(V/Q more than 1) so the P_{O_2} is >100 while the base has more perfusion than ventilation(V/Q less than 1) so the P_{O_2} <100 .
 - DLO₂(diffusion capacity of the lung): a measurement of how much oxygen diffuse through the whole membrane and it is equal to oxygen consumption ($\dot{V}O_2$) = 250 ml/min normally it is increased during exercise.
 - A mnemonic for factors increasing closing capacity is ACLS-S: Age, Chronic bronchitis, LV failure, Smoking, Surgery.
 - lung also participates in acid base balance
 - in each inspiration you actually return the blood to your heart.
 - If you measure the ABG and found that the arterial P_{O_2} is 95 mmHg, then its normal although we said previously that it equals 100 mmHg, the important thing is that the different shouldn't exceed 5 mmHg.
 - P_{O_2} alveolar= $(130+90+90+90)/ 4= 100$
 - in the arteries the hyperventilated blood is unable to correct the hypoventilated blood, unlike the CO_2 which is able to correct itself so the P_{CO_2} in the alveoli = P_{CO_2} in the arteries = 40 mmHg.
 - $\dot{V}O_{2max}$ is the maximum oxygen consumption, and it equals 250 ml O_2 / min in resting state.
 - convert CO diffusing capacity to O_2 diffusing capacity, the value is multiplied by a factor of 1.23 because the diffusion coefficient for O_2 is 1.23* times that for CO. Thus, the average diffusing capacity for CO in healthy young men at rest is 17 ml/min/mm Hg, and the diffusing capacity for O_2 is 1.23 times this, or 21 ml/min/mm Hg.