2023- 2024- RS

SUMMARY

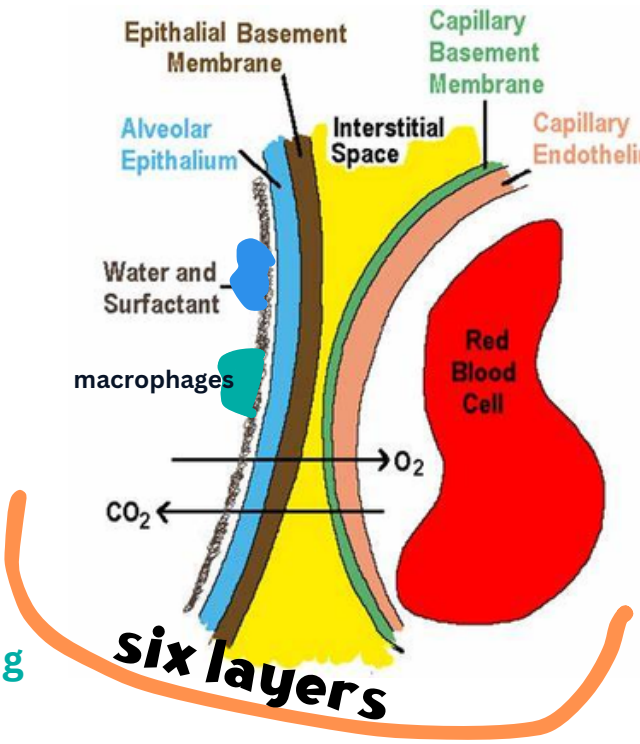
PHYSIOLOGY

ebaa alzubi

left heart failure = pulmonary edema
lung disease = right heart failure

DALTON'S LAW

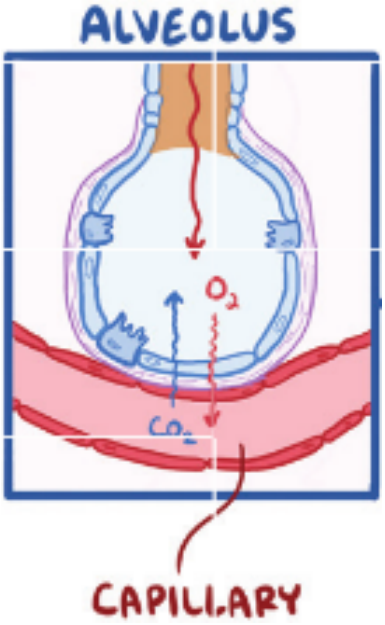
- If the sum of partial pressures in a mixture = total pressure of mixture → barometric pressure (P_B) is sum of the partial pressures of O_2, CO_2, N_2 (nitrogen), and H_2O
- At barometric pressure (760 mmHg) composition of humidified air is O_2, 21%; N_2, 79%; CO_2, 0%
- Within airways, air is humidified thus water vapor pressure is obligatory = to 47mmHg at 37°C/98.6°F



- **at high altitude= low o2 = difficult breathing**
- **respiratory problems (hypoxia): from outside to inside**

1. **abstraction of airways**
2. **restriction of alveolar movement** → **respiratory distress syndrome (RDS) : infants RDS , acute adults RDS .**
3. **diffusion of capillaries blood**

- Emphysema → destruction of alveoli → decreased surface area for gas exchange → decreased DL_{CO}
- Fibrosis/pulmonary edema → increase in membrane thickness (via fluid accumulation in the case of edema) → decreased DL_{CO}
- Anemia → reduced hemoglobin → reduced protein binding in a given time period → decreased DL_{CO}
- Exercise → increased utilization of lung capacity, increased recruitment of pulmonary capillaries → increased DL_{CO}

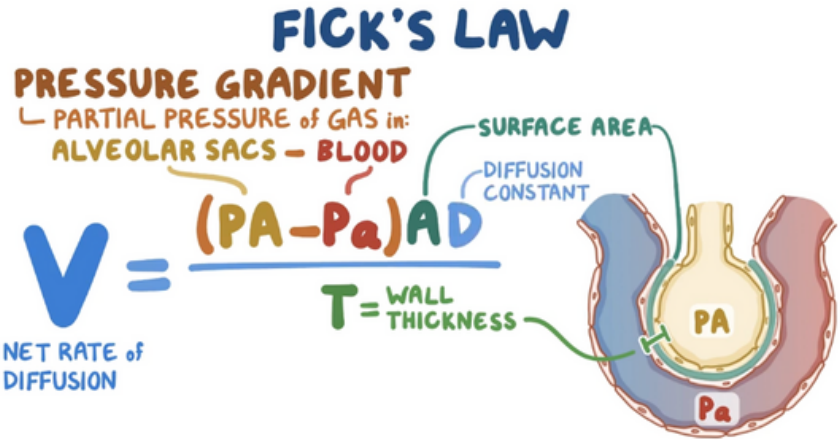


mixed venous blood in pulmonary artery
o2 is the same in all arteries
o2 is differant in veins

blood distribution= 5L

450mL = lungs (190mL pulmonary artery , 190mL pulmonary vein , 70 capillaries)

3L = veins
750mL = arteries
350mL = capillaries
350mL = heart chambers



gas exchange depends on pressure difference

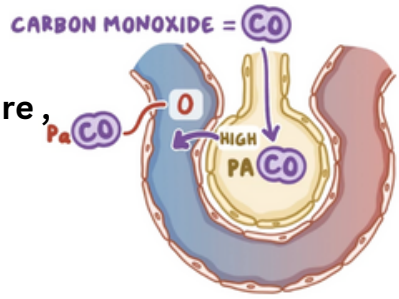
D = gas solubility /√molecular weight
molecular weight is the least important factor

- Diffusion coefficient dramatically affects diffusion rate, e.g. diffusion coefficient for CO_2 is approximately 20x greater than that of O_2 → for a given partial pressure difference CO_2 would diffuse across the same membrane 20x faster than O_2

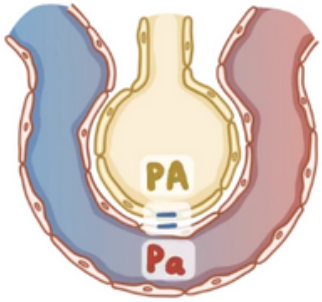
Co2 is strong not affected unless there is a big problem .
so : in respiratory failure type2 , too much damage in lung = high high Co2 in blood

✨diffusion-limited-perfusion-limited-gas-exchange

diffusion -limited :
keep going from high pressure to low pressure , which becomes high , again & again



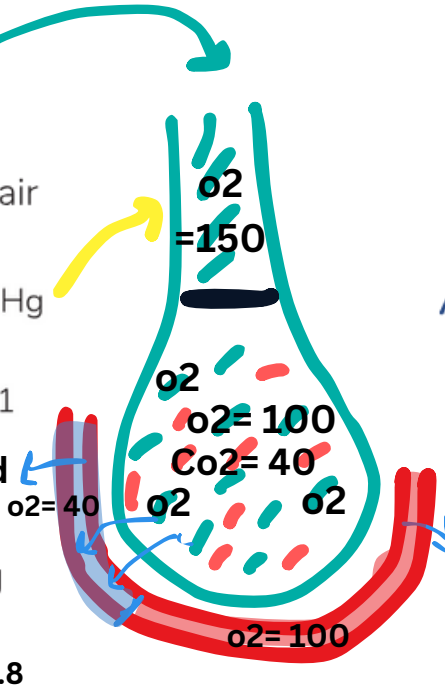
perfusion -limited :
keep going from high to low till achieving equilibrium .



O2 like a ghost , not diffusion- limited , but perfusion limited
the least cause of hypoxia is diffusion

Dry inspired air
- P_{O2} is approximately 160mmHg
  - P_{O2} = 760mmHg x 0.21
  - Assume no CO_2 in dry inspired air

Humidified tracheal air
- P_{O2} of humidified tracheal air is 150mmHg
  - At 37°C/98.6°F, P_{H2O} is 47mmHg
  - P_{O2} = (760mmHg – 47mmHg) x 0.21



Alveolar air
- PA_{O2} = 100mmHg
- PA_{CO2} = 40mmHg

deoxygenated

- If P_{O2} of alveolar air = 100mmHg
- P_{O2} of mixed venous blood entering pulmonary capillary = 40mmHg

oxygenated

blood takes 1 cardiac cycle in capillary 0.8
get oxygenated in 1/3 of capillary cuz O2 is not diffusion limited

so :
high HR = less filling =less diastole = less oxygenated blood = less blood to coronary

oxygen in artery should not be different from alveolar oxygen in normal person

ANATOMIC DEAD SPACE

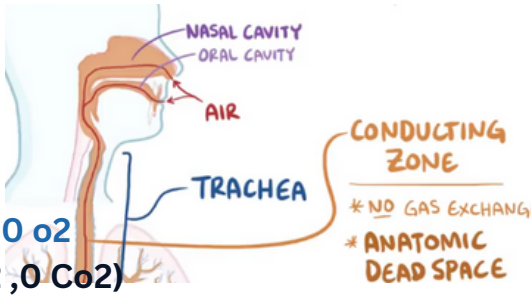
conducting zone - no gas exchange , 1/3 tidal volume = 150ml .
ventaltion -perfusion defect cause physiological dead space "alveolar without exchange"

- Environmental air CO_2 = 0 (actual amount ≅ 0.04%)
- Dead space CO_2 contribution = 0
- All CO_2 in exhaled air comes from functioning alveoli

- $V_D = V_T$ x arterial CO_2 partial pressure ($PaCO_2$) - expired CO_2 partial pressure ($PeCO_2$) ÷ $PaCO_2$

$$V_D = V_T \times \frac{Pa_{CO_2} - Pe_{CO_2}}{Pa_{CO_2}}$$

recap



you inhale 500 mL of air = inspiration = 21% o2 = 160 ml
inspired air gets humified by H2o in trachea = 47 ml H2o then 150 o2
(no gas exchange then it's called anatomical dead space, 150 o2 ,0 Co2)
then in alveoli, o2 get in 1/3 of capillary to reach 100ml equilibrium "perfusion"
and Co2 transport from capillary to alveoli = 40ml "diffusion"
time = 1 cardiac cycle = 0.8
then exhalation : inspired air "fresh air" which stayed in dead zone get out first
then alveolar air (exchanged) so expiration air is mixed air .

so : which air is the highest container of O2 ?

a- alveolar air **c- arterial air**
b-mixed expired air **d- venous air**

VENTILATION

- Air movement between environment, lungs

MINUTE VENTILATION = (TIDAL VOLUME × RESPIRATORY RATE)

but if we want to calculate alveolar ventilation rate , we should first minus dead space volume from the tidal . $V_A = (VT - VD) \times RR$

and as we know Co2 exchange in alveolar so it has a volume in the ventilation , gets affected by its fraction . $V_{CO_2} = V_A \times F_{CO_2}$

$$V_A = \frac{V_{CO_2} \times K}{P_{ACO_2}}$$ → **fraction**

- Law of Laplace: pressure that promotes lungs' collapse is (1) directly proportional to surface tension, (2) inversely proportional to alveoli radius

P = 2T/r

SURFACTANT

- ↓ collapsing pressure in alveoli → ↑ gas exchange, ↑ lung compliance, ↓ work of breathing

(amphipathic nature)

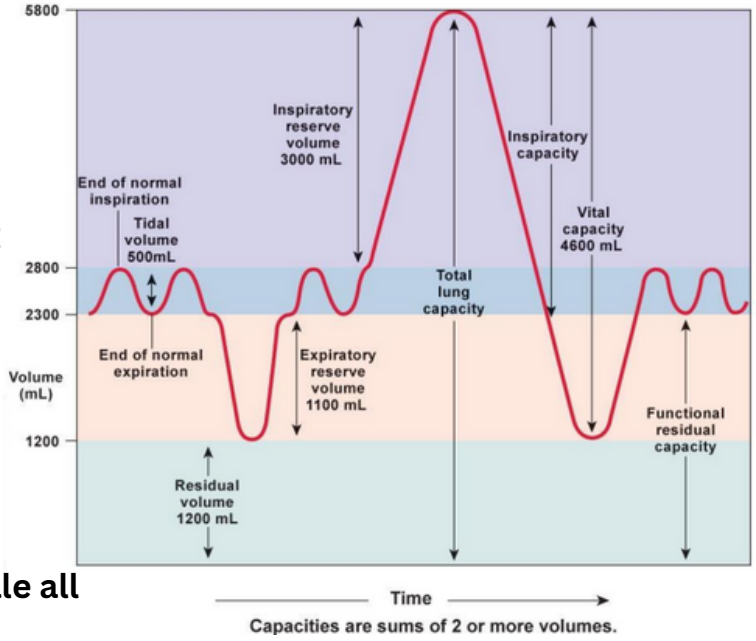
reduce tension & collapsing

LUNG VOLUMES & CAPACITIES

rest / static volume: lung out side the body , without air in it.
capacity is how much air could it contain .

- *Spirometry:* spirometer used to measure air volume moving in, out of lungs
- Tidal volume (V_T)
  - 500mL **normal**
  - Air volume inspired, expired during quiet breathing
- Inspiratory reserve volume
  - Maximum volume inhaled air above V_T = 3L **maximum size of lung**
- Expiratory reserve volume
  - Maximum expired air volume below V_T = 1.2L **maximum use of muscle to exhale all**
- Residual volume (RV)
  - Air remaining in lungs after forced expiration = 1.2L (not measured by spirometry)
- Functional residual capacity (FRC)
  - Expiratory reserve volume (ERV) + RV = 2.4L
- Vital capacity (V_C)
  - V_T + inspiratory reserve volume (IRV) + ERV = 4.7L
- Total lung capacity (TLC)
  - Combination of all lung capacities = 5.9L

A spirometer tracing showing lung volumes and capacities.



Capacities are sums of 2 or more volumes.

resting volume of the system

Helium dilution method

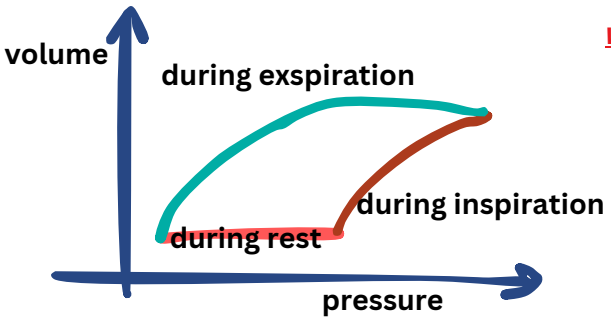
- Helium placed in spirometer → inhaled
- Helium concentration in lungs equalizes with amount of helium placed in spirometer (helium insoluble in blood) after few breaths
- Total helium mass measured in spirometer = FRC

COMPLIANCE OF LUNGS & CHEST WALL

- Compliance measures how changes in pressure → lung volume change
- *Lung, chest wall compliance:* inversely correlated with elastic, "snap back" properties (elastance)
  - Compliance = $\Delta V/\Delta P$
  - Elastance = $\Delta P/\Delta V$
- ↑ compliance → lungs easier to fill with air
  - *Forces promoting open alveoli:* compliance, transmural pressure gradient, surfactant
- ↓ compliance → lungs harder to fill with air
  - *Forces promoting collapse of alveoli:* elastic recoil/elastance, alveolar surface tension

increase elasticity - increase tension - increase pressure = decrease in compliance

calculate from the pic :
IRV : TLC : VT : FRC:
which is one thing that spirometer cant show ?

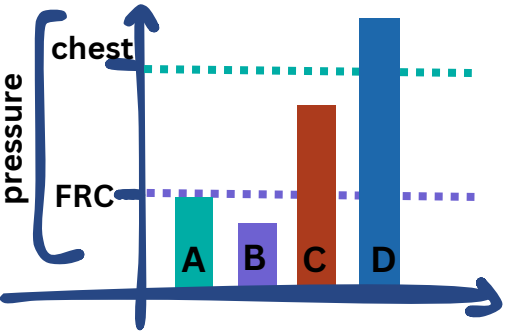


curve = compliance , notice that it was never zero , cuz to *wise to inflate partially inflated lung* , less energy consuming

expiration is not gradual like inspiration cuz you can't hold your muscles+ elastic much longer = passive

notice that expiration it not like inspiration : Hysteresis

active gradual inspiration & passive expiration normally .
opposite in obstruction



ANY pressure less than chest resting pressure cuz chest expansion .
ANY pressure less than FRC resting pressure cuz lung collapse

A- at resting , lungs tend to collapse , chest tends to expand

B- LESS pressure lungs tend to expand, chest tends to expand

C- more pressure lungs tend to collapse , chest tends to expand

D- more pressure lungs tend to collapse , chest tends to collapse

as long as functional residual capacity is systemic resting capacity =FRC

then :

- Volume > FRC — D : lung collapse , chest goes inward
  - Positive transmural pressure
- Volume < FRC (forced expiration) — B : lung expand , chest expand
  - Negative transmural pressure

in emphysema “obstruction”, alveoli enlarged , making new resting pressure , where lung and chest tend to expand = barrel chest = برميل

in restriction diseases , new resting membrane , lung tend to collapse , chest remain in a fixed

RESISTANCE

respiratory resistance = 1% of total resistance

Poiseuille’s law

- Resistance in lungs determined by Poiseuille’s law — asthma + bronchitis + emphysema = more resistance
  - Air flow directly proportional to resistance along airway

$$R = \frac{8nl}{\pi r^4}$$

resistance is uncountable , unmeasurable unit but we can predict it by radius

↑ viscosity (e.g. deep sea diving)

↑ resistance

↓ lung volume

BOYLE’S LAW

- $P_1V_1 = P_2V_2$
- For gas at given temperature, the product of pressure, volume is constant
- Inspiration → diaphragm contraction → ↑ lung volume

this means lung inflation leads to air entry not the opposite

oxygen in artery should not be different from alveolar oxygen in normal person

partial pressure of arterial oxygen (PaO2) / fraction of inspired oxygen (FiO2)

< 200 = ARDS → infant / adults

organ failure