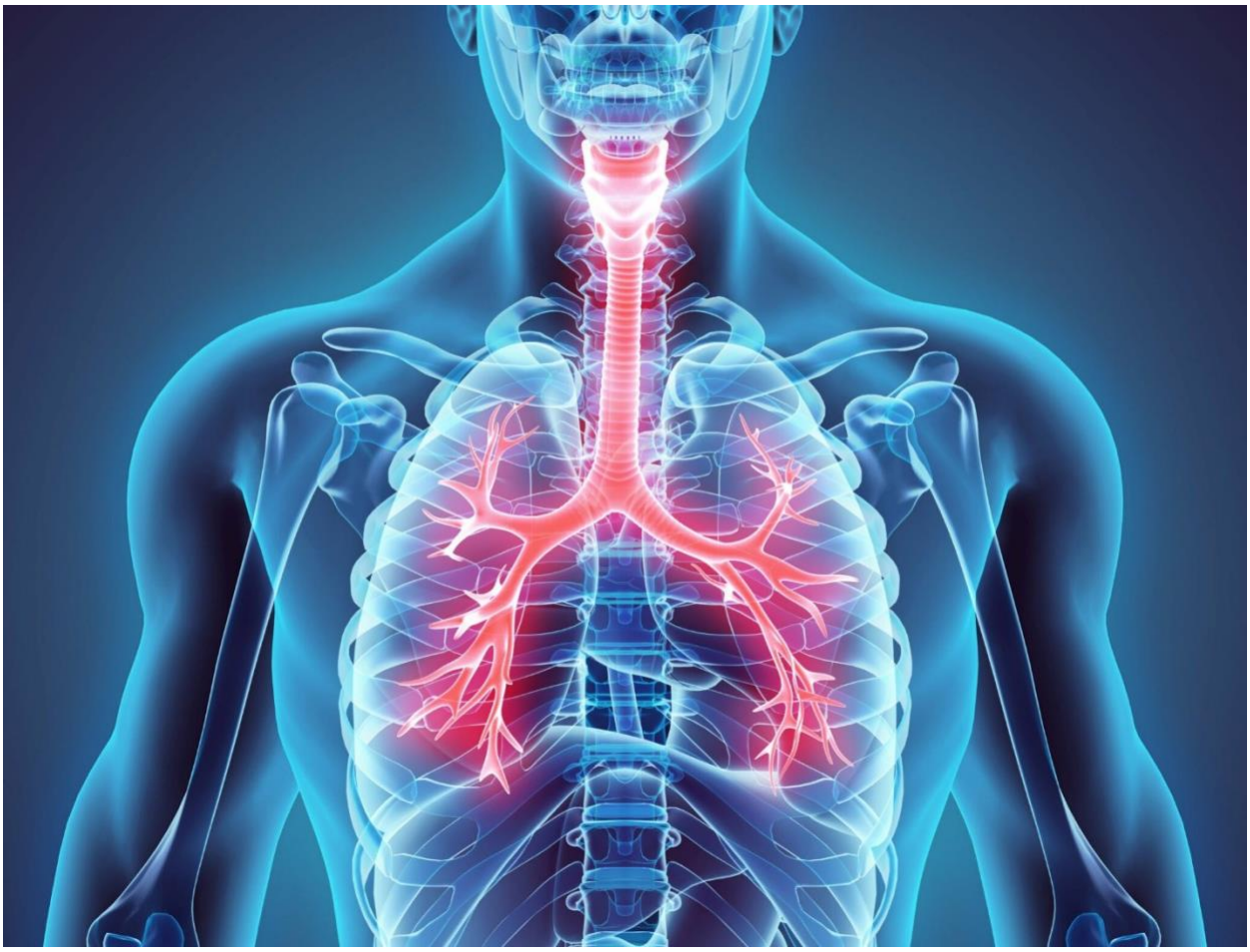


Respiratory system physiology

Doctor yanal shafagoj's notes

Written by Dima Alrafaiah

Corrected by Nermeen Abuhalaweh

- Respiratory system functions mainly to maintain 'homeostasis' of O2, H+, CO2.
  We call those gases the Arterial Blood Gases (ABGs), and it's a test usually performed to assess the efficiency of RS in gas exchange.
  - ✓ Normal ABGs values are: PaO2 = 100
    PaCO2= 40
    pH= 7.4

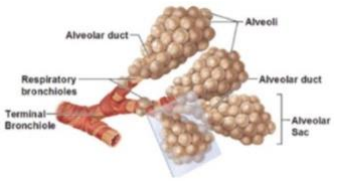
  If ABGs are normal, the lungs are functioning properly **(but it's not necessarily the opposite case if they weren't)**.

- **Functional anatomy**

– Part of the lower respiratory system, which is bronchioles, alveoli and the lungs, is responsible for the exchange of O2 and CO2.
– The trachea branches into two primary bronchi which further divide into smaller airways called bronchioles, at the end of the bronchioles are clusters of air sacs called alveoli.
– We can classify the parts of lower respiratory system by the presence or the absence of cartilage
  A. Bronchi (cartilage)
  B. Bronchioles (no cartilage)

Two important bronchioles are

- ✓ Terminal bronchiole: the final most distal structure in the conducting zone, which deliver air directly into the alveoli.
- ✓ Respiratory bronchiole: a very small alveolar duct and a part of respiratory zone.



- **Alveoli**

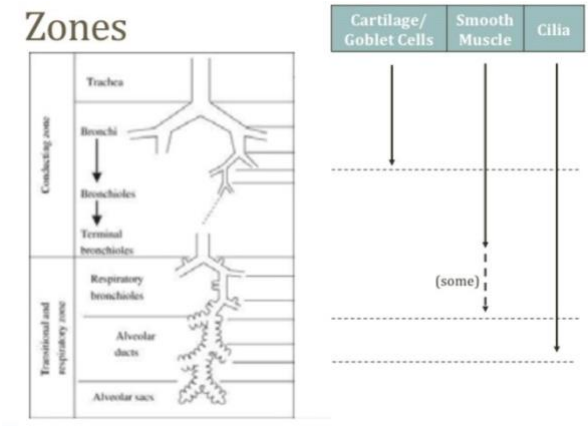
– Small sacs
– Each alveolus is surrounded by a network of capillaries
– Consist of 3 types of epithelial cells
  A. Type 1 (97%), epithelial thin cells for exchange.
  B. Type 2, columnar cells that produce surfactants which are fatty material to reduce surface tension and thus preventing collapse of alveoli when one exhales.
  C. Alveolar macrophages; digest any inhaled particle less than 5 microns.

- **Air way cells**
  A. Goblet cells; secrete mucus which traps and removes inhaled foreign particles like dust, allergen, bacteria and viruses.
  B. Ciliated epithelial cells; move mucus to epiglottis.

- **Respiratory membrane 0.2-0.6 mm (the blood-air barrier)**
  – The network of capillaries surrounding each alveolus makes the respiratory membrane, which is a thin delicate structure that facilitate the exchange of gases between the two. It's composed of 6 layers from alveoli to the lumen of capillary as following:
  ✓ Surfactants
  ✓ Epithelial cells
  ✓ Alveolar basement membrane
  ✓ Interstitium
  ✓ Capillary basement membrane
  ✓ Endothelial cells.

- **Zones**
  - In terms of pulmonary physiology, the respiratory tract is usually divided up in two zones:
    - **A.** Conducting zone
    - **B.** Respiratory zone

A. Conducting zone



- Is also called the Anatomic Dead Space (ADS) because it fills with air but DOES NOT participate in gas exchange and air is simply conducted back and forth.
- It filters, warms, humidifies air or else it will cause physical injury to alveoli.
- Nose, pharynx, trachea, bronchi, bronchioles to the terminal bronchiole.

B. Respiratory zone

- Where gas exchange happens.
- Respiratory bronchiole, alveolar ducts, alveoli.

Respiration

1. Ventilation
2. External respiration (gas exchange at the level of alveoli)
3. Transport
4. Internal respiration (gas exchange at the level of tissues)
5. Cellular respiration (O_2 to ATP)

1. Ventilation

- Is the process of moving air into and out of the lungs (inspiration-inhalation, expiration-exhalation).
- So, it's a flow of air, and like any other flow it depends on two main factors (pressure gradient and resistance).
- To create this pressure gradient between atmospheric pressure and alveolar pressure (P_{atm}-P_A), we either increase P_{atm} OR decrease P_A. We can only modulate alveolar pressure but how can that happen?
- To answer the previous question, you have to learn about intrapleural pressure, the pressure in the pleural space that is normally NEGATIVE to "suck" the alveoli open, and it's generated by the chest wall that tend to expand and spring outward creating this negative pressure.

Going back to our question..

A. Inhalation

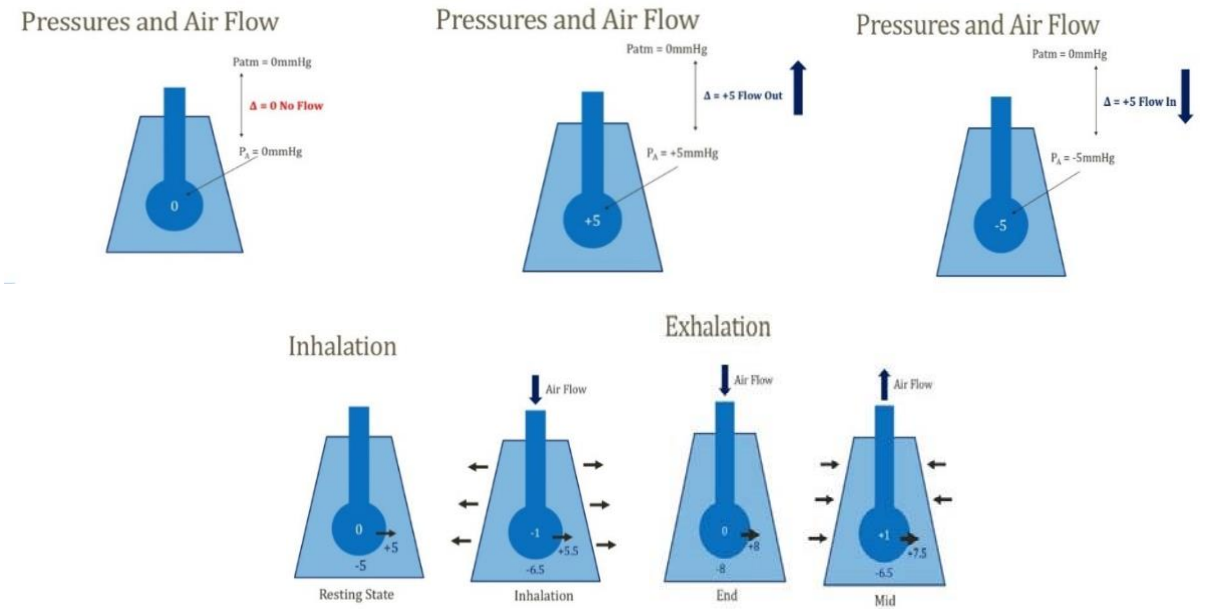
- ✓ Intrapleural pressure becomes MORE negative.
- ✓ Alveolar pressure becomes negative.
- ✓ Air flows INTO the lungs.

B. Exhalation

- ✓ Intrapleural pressure becomes LESS negative.
- ✓ Alveolar pressure becomes positive.
- ✓ Air flows OUT of the lungs.

Atmospheric pressure=760mmHg = 1atm

Alveolar pressure decreased during inhalation, increased during exhalation



- **Lung volumes**
  - ✓ **Tidal volume (TV)**: the volume of air that moves in and out with quiet breathing (the automatic breathing you do when you're not thinking about it, when you're not trying to apply force to inhale or exhale more forcefully) =500mL (150 for ADS, 350 for alveoli).
    Note that there's a fraction of inspired air doesn't perform a physiologic function of gas exchange (remains in ADS).
  - ✓ **Respiratory rate (RR):** how many breaths in a minute =12breath/min.
  - ✓ **Respiratory minute volume (RMV):** total volume a person breath in one minute TV*RR =0.5L*12=6L/min.
    A. Alveolar MV = 0.35L*12= 4.2L/min
    B. Anatomical dead space MV = 0.15L*12= 1.8L/min
  - ➢ During CPR (mouth to mouth ventilation): the rescuer exhales a volume of air that includes both air from alveoli

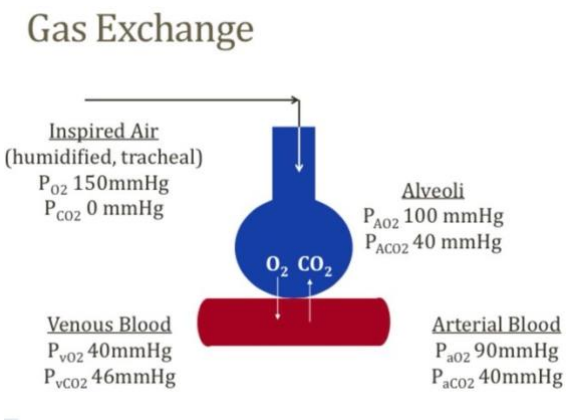
and the air from ADS which is called mixed expiratory air (dead space ventilation+ alveolar ventilation).

- To calculate total O2 delivered in mixed expiratory air:

(Volume of ADS*PO2 of inspired air in airways) + (Alv Vol *PO2 of inspired air in alveoli)/TV
(150*150) +(350*100)/500=116
For CO2 >> (0*150) +(350*40)/500 = 28

- **Important compositions**
  - Outside air at the sea level
    - ✓ Patm= 760mmHg
    - ✓ PO2= 21% * 760 = 160mmHg
    - ✓ PN2= 79% * 760 = 600mmHg
    - ✓ PH20= 0 (dry air)
    - ✓ PCO2= 0
  - Inspired air in airways (humidified, 37c)
    - ✓ PH20= 47 mmHg
    - ✓ PO2= (760-47) * 21% = 150
    - ✓ PCO2= 0

      47 goes for PH2O
  - Inspired air in alveoli after gas exchange
    - ✓ P_AO2= 100mmHg (theoretically, there's some pollution; may drop to 95%)
    - ✓ P_ACO2= 40mmHg
  - Arterial blood in pulmonary veins
    - ✓ P_aO2= 100mmHg
    - ✓ P_aCO2= 40mmHg
  - Mixed venous blood in pulmonary artery



- ✓ P_vO2= 40mmHg
- ✓ PvCO2= 45mmHg

- **Let's go through the journey of CO2:**
  - Mixed venous blood goes from **pulmonary artery** through the pulmonary capillary, and then it leaves into the pulmonary veins, the left atrium, left ventricle, and eventually it makes it to the arteries.
  - Blood from venous system has higher conc of CO2 because it has picked up CO2 from tissue. **PvCO2= 45**
  - Alveolar CO2 conc from inspired air **= 40**
  - Pressure gradient toward alveolus is established.
  - blood flows through the pulmonary capillary, CO2 will leave and enter the alveolus.
  - And by time that blood reaches the exit of pulmonary capillary, EQUILIBRIUM (no further flow) will have been established when the alveolar CO2 is equal to the arterial CO2.
    - ✓ *So why does this equilibrium between artery and alveolus happen with the PACO2 still being the same?* (same thing applies to PAO2)
      - ➢ Normally when gas flows from area 1 with high conc to area 2 with low conc, you expect conc in area 1 to decrease and conc in area 2 to increase until equilibrium is achieved
      - ➢ In our case, CO2 flows from capillaries to alveoli and O2 from alveoli to capillaries until the equilibrium is achieved by the end of pulmonary vein. EXCEPT that the equilibrium here is

achieved when the PaO2 has increased, but the PAO2 is still the same!!

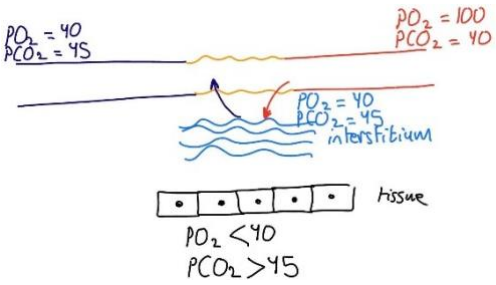
The reason behind this is the much greater volume in lungs in comparison to capillaries

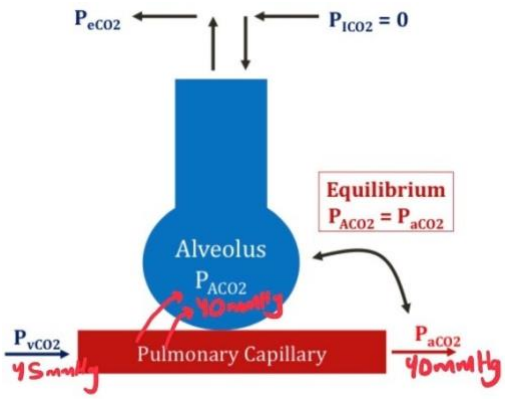
Alv volume (2200ml)>>> pul cap (70ml).

- Same thing regarding gas exchange during internal inspiration at the level of tissues (there's equilibrium between venous end and interstitial fluid composition because

IF volume (11000ml)>>> sys cap (350ml)

P.S: "A" stands for alveoli while "a" for artery.





✓ **This equilibrium is so important to maintain A-a gradient that equals to zero or less than 5 (this 5 is because of pollution), otherwise if it's more than 5 it's an indicator of decreased diffusion. By gradient we mean the difference between Pa and PA, if it was more than five, it means that much of the gas stayed in the alveoli and didn't diffuse to capillaries. Hence, this gradient can be looked at as a measure of diffusion.**

Body fluids 60% of body weight =42L

1/3 (14L) extracellular >>11L

interstitium, 3L plasma2/3 (28L)

intracellular

Blood volume= 7% of body weight =5L

3L sys veins, 750 sys arteries, 350 sys capillaries, 350 in heart chambers, 450 inpulmonary circ (190 pul artery, 190 pul vein, 70 pul capillaries)

- **Diffusion of gases through respiratory membrane**
  - J(diffusion) = ΔP * A/d * S/√MW

    A: surface area

    d: thickness of membrane

    S: solubility of the gas

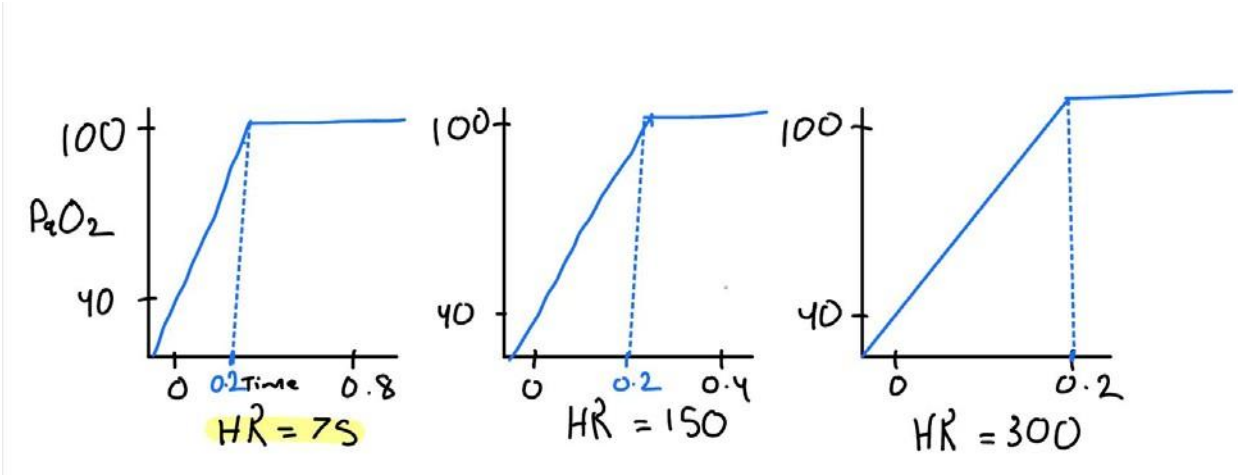
    √MW: square root of MW of the gas (the least contributing factor)
  - S/√MW is the diffusion coefficient, and it's constant for each gas.
  - For a healthy respiratory membrane, the gases differ in their diffusion rate depending on their solubility.
    - ✓ **O2 can cross any membrane as if the membrane doesn't exist, because it's soluble in lipid bilayer.**
    - ✓ **So, it's said to be NOT diffusion limited.**
    - ✓ **However, it's perfusion limited since it depends on the capacity of blood (carried on hemoglobin) to be transported.**
    - ✓ **CO2 is 20 times more soluble and diffusible than O2**

      JCO2 = 20 JO2.
    - ✓ **So, O2 is faster to be affected by diseases affecting diffusion. Example;**

      Respiratory failure TYPE 1, O2 is first affected so you find PO2↓↓ and PCO2 the same or even decreased

      Respiratory failure TYPE 2, PO2↓ and CO2 is just now affected so PCO2↑
  - Blood cells duration in capillaries = cardiac cycle duration.
    - ✓ It only consumes one third of this duration for gas exchange (0.25s)

- ✓ at normal heart rate cardiac cycle =0.8
- ✓ if HR increases blood takes almost the entire duration in gas exchange (HR = 150, CC= 0.4) (HR= 300, CC=0.2 THE WHOLE CYCLE consumed in gas exchange)



- ✓ **so increasing HR will decrease diastolic time (time for coronary perfusion) and time for gas exchange, so the pumped blood is decreased and not fully oxygenated.**

- **Hypoxia (↓O2 utilization by cells) caused by:**

1. Inadequate oxygenation
   - ✓ High altitudes (less Patm)
     - At the sea level Patm= 760mmHg and PO2=21% of Patm=160mmHg
     - Every 5.5km rise in the level will decrease the Patm to the half and consequently will

decrease the PO2; at 5.5km the Patm= 380 and PO2= 80.

- at 11 km the Patm= 190 and PO2= 40.

2. Muscle paralysis

We need diaphragm and external intercostal muscles to breath, those are skeletal muscles innervated by motor neurons coming from respiratory centers in medulla, so any tumor, trauma or bleeding in those centers can cause paralysis. Also, any disease that causes neuropathy like polio.

3. Pulmonary diseases

- ✓ 70% Obstructive (increase the resistance in airway, hard to exhale) such as asthma, emphysema, chronic bronchitis.
- ✓ 20% Restrictive (make intrapleural pressure more positive, hard to inhale, alveoli are less inflatable rigid and non-stretchable) such as fibrosis, infant RDS (respiratory distress syndrome), acute RDS.
- ✓ 10% vascular diseases like pulmonary hypertension.

4. Inadequate transport like anemia, heart failure.
5. Inadequate usage of O2 by cells like septicemia and CN poisoning.

- Questions asked by doctor
  1. **If we took a sample from ADS, what would (PCO2, PO2) be at the end of expiration, at the end of inspiration:**

  a. At the end of expiration ADS composition would be similar to alveolar composition, PCO2 = 40, PO2 =100.

  b. At the end of inspiration, it would be similar to the outside air but after humidification, PCO2=0 PO2=150.

  2. **Where PO2 is highest in following:**

     A. Arterial blood

     B. Mixed venous blood

     C. Interstitium

     D. Expiratory mixed air

     Ans is D (due to dead space ventilation), Moral of story, memorize ALL the numbers.

V2

- Highlighted in gray.

V3

- Highlighted in yellow.