Physiology sheet no. 7

Ventilation/Perfusion ratio (V/Q ratio)

Before you start:

****I advise you to watch Dr. Najeeb's or Kaplan videos because you'll understand the subject really in a better way. You might be confused by some points in the sheet, but this is what the doctor said in the lecture, he's not that organized or clear, I might be wrong, but however, I tried my best to organize it, and I'm sorry for any mistake.**

**Unit of any pressure number in the sheet: mm Hg
**Pa = arterial pressure
**PA = alveolar pressure

The main target always is to maintain normal ABGs (arterial blood gases)

Normal ABGs means: PaO_2= 100 , $PaCO_2$=40

(For hypothetical reasons, we believe that the PaO_2 and PAO_2 are the same).

So the question now is: what makes PAO_2 increases or decreases?

PAO_2 normally fluctuate during respiratory cycle. During inspiration, it might increase to 140 BUT the average is 100.

In general, PAO_2 is a affected by 2 things: input and output!

Input = alveolar ventilation = 4.2 L/min. (350 (mL)*12)
Output = O_2 Consumption = 250 mL/min. (at rest)

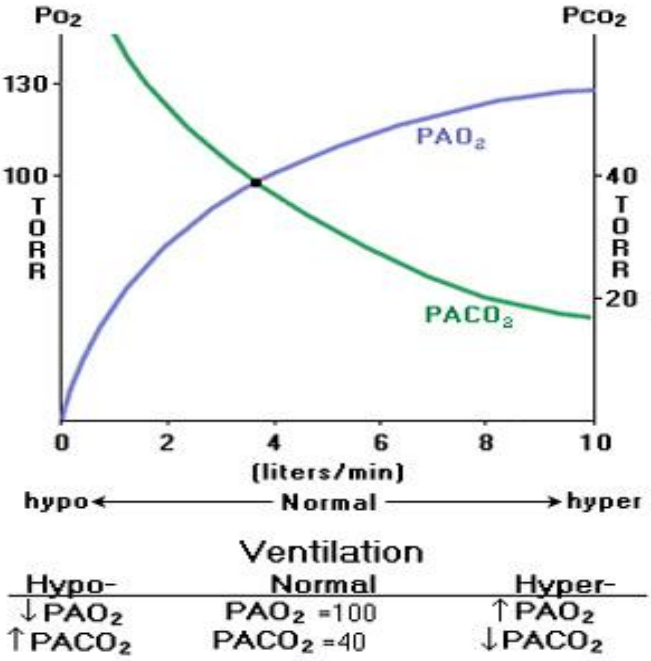
Remember: (O_2 consumption at rest = 250 mL /min. but this increases during exercise.
The maximum O_2 consumption possible = 5 L/min. (20 times the normal rate at rest)) in athletes

And in Normal people it might reach 3.5L\min

A) **If Alveolar ventilation is increased, keeping oxygen consumption constant, then PAO2 increases.**
B) **If oxygen consumption is increased, then PAO2 decreases.**

So, PAO2 is directly proportional to alveolar ventilation, but inversely proportional to O2 consumption! (the take out of O2 from alveoli)

The same thing applies to CO2 but in the opposite direction. The more the ventilation, the less the PACO2 (washing out CO2).



So when you increase the ventilation, the goal is to make the alveolar air composition closer to the outside air composition (which has more oxygen and less CO2).

So hyperventilation by definition is when the input of CO2 is less than the output (The arterial CO2 decreases)

Hyperventilation isn't the same as **Increased Ventilation**! In increased ventilation, PACO2 remains constant, CO2 production is matched with the alveolar washing out (like during exercise, CO2 and O2 in arterial gases remain constant).

So, we can say hyperventilation is when alveolar CO2 decreases below 40! i.e hyperventilation results in hypocapnea

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Some laws:

$PACO_2$ = (CO_2 production (VCO_2)/alveolar ventilation (VA)) *K

PAO_2 = (PO_2 (inspired) – $PACO_2$) / R

(R here is the respiration exchange ratio or the respiratory quotient RQ)

K (gas constant) = 0.863

Respiratory exchange ratio = CO_2 production/O_2 consumption.

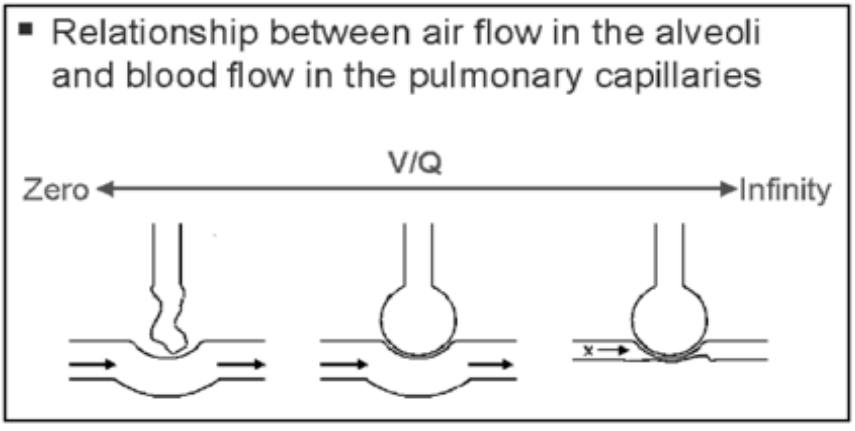
CO_2 production normally isn't 1 molecule for each 1 O_2 molecule consumed, but rather 6 CO_2 molecules for each 1 O_2 molecule.

The respiratory exchange ratio:
for carbohydrates = 1 (completely metabolized)
for fat = 0.7 (not completely metabolized)
for proteins = 0.8 (not completely metabolized)
for mixed food = 0.8

So normally, normal people make 200 mL CO_2 for 250 mL O_2 consumption. (200/250 = 0.8)

So..

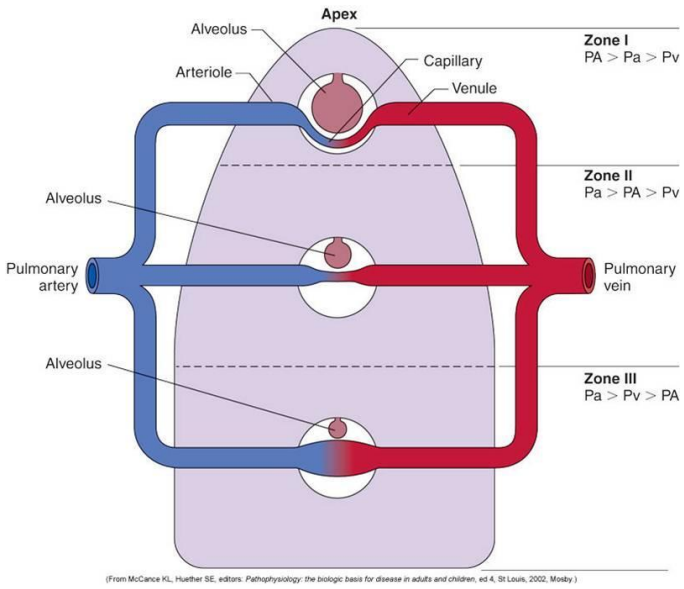
1- If one alveolus is ventilated but not perfused (so V/Q ratio = infinity), then the composition of alveolar air should be the same as the outside air! PAO_2 = 150 (same as the outside air, the only difference is that it's humid because of H_2O mixed with it) and alveolar CO_2 = zero.
2- On the other hand, If one alveolus is obstructed, which means perfused but not ventilated (so V/Q ratio is zero), then the composition of alveolar air should be the same as the venous blood gases composition
PAO_2 = 40, $PACO_2$= 45



Remember that:

- Most of blood flow to tissues occurs mainly during systole (except for the coronary arteries, it happens during diastole).
- Blood flow in systemic capillaries isn't pulsatile (there's damping of pressure pulsation in the capillaries), the flow isn't continuous, it's intermittent! There's pre-capillary sphincter (which controls blood flow) and there's also the vasomotion which affects the flow.
  *in the Pulmonary circulation :
- 2/3 of your capillaries are closed normally! Just 1/3 of them are open! (during exercise, you open all capillaries because of the local vasodilators and this decreases the resistance in the pulmonary circulation so the pulmonary pressure does not rise that much)
- In systemic capillaries, there is more fluctuation in pulse and pressure. While in pulmonary capillaries, because it's near the left ventricle, blood flow is more pulsatile (no damping in blood pressure pulsation)

We divide lungs into 3 zones normally:



Zone 1 (upper zone): No perfusion (neither during systole nor during diastole), but such zone doesn't exist! Might occur during severe bleeding where cardiac output is very weak.

Zone 2: there's perfusion during systole but not during diastole.

Zone 3: there's perfusion during systole and diastole.

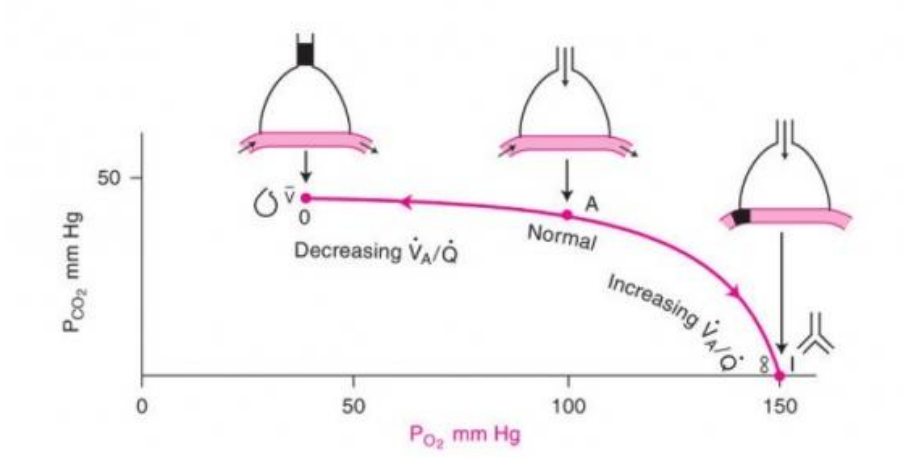
Remember that in lying or exercising individual, all lungs become like zone 3 (pulsation in systole and diastole), but we're talking mainly about standing individual.

**Hypoventilation: making alveolar air composition more CO2 and less O2 (like venous blood).
Increasing CO2 increases hydrogen!

CO2 + H2O → H2CO3 → HCO3- + **H+** (in presence of carbonic anhydrase enzyme), so CO2 is "mask" hydrogen.

So when you have alkalosis, you need to retain hydrogen. The best way is to depress ventilation, maintaining more CO2, so you have more acidity → less pH . That's how lungs help in acid/base balance.

During acidosis: the opposite! Hyperventilation → washing CO2 (washing hydrogen) → going back to the left (in the equation).
PO2-PCO2 diagram:

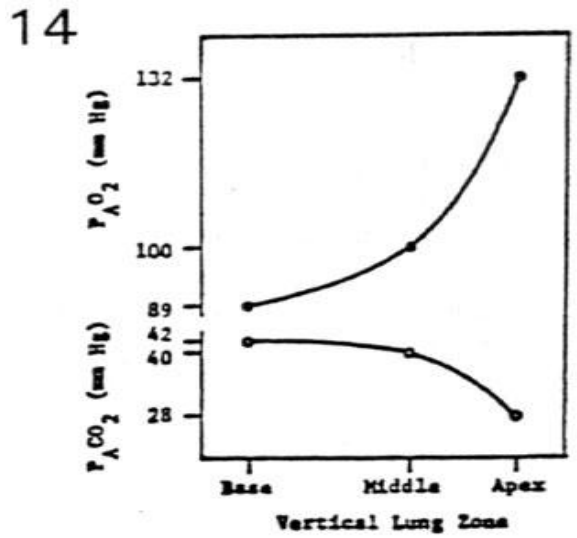


Alveolar wasted volume: when you ventilate but don't perfuse. (like in physiologic dead space). So, it's like you're increasing the dead space.

In Apex: more oxygen, less CO2

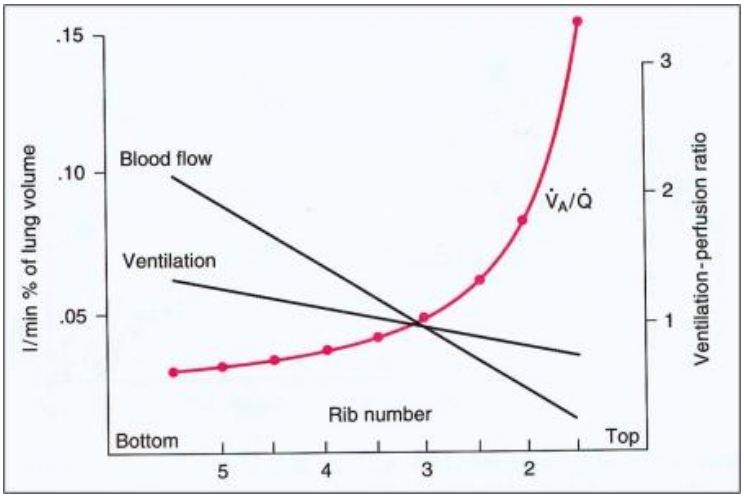
In Base: less O2, more CO2

(Because of the negative pressure (more negative at the apex), and because it's easier for the heart to pump to the base not the apex).



Conclusion:

1- Ventilation in the base more than in the apex
2- Perfusion in base is also more than perfusion in the apex (but here the difference is much more because of the effect of gravity)
3- V/Q ratio of the base is less than the apex
   Ratio in the base is less than 1, because there's much more perfusion.
   Ratio in the apex is more than 1 (almost 3), because there's more ventilation.



Remember that Composition of alveolar air:

At the apex: PAO2 = 130
At the base: PAO2: 90
PAO2 (avg) = 100
PO2 in the blood coming (venous) = 40
PO2 in the blood leaving (arterial) = 100

But PO2 in arterial blood doesn't reach 100 really! It's always between 90 and 95!

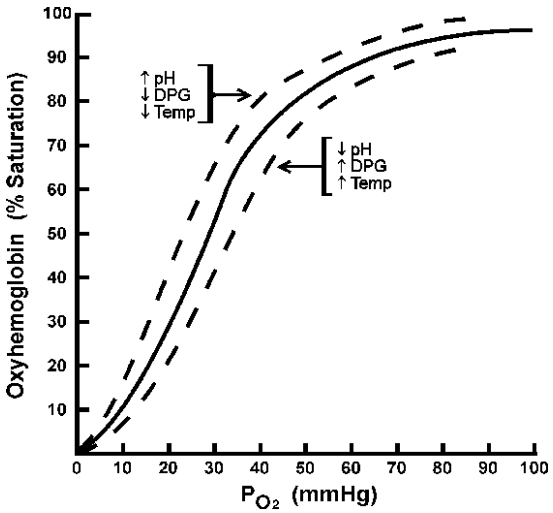
PAO2-PaO2 > zero

Why is that?

There are 2 reasons for it:

1- Venous admixture (pollution): the blood isn't pure arterial, 2% approximately is from venous blood like bronchial veins.
Also, some of the blood bypasses the whole capillary part (shunted blood in the lungs it goes to the lungs but does not play a role in gas exchange)). It goes through what's called arterio-venous anastomosis(physiological shunt)

2- V/Q inequality:
there are different values of PaO2 from different areas of lungs, and there's inability of the hyperventilated blood (that comes from the apex) to correct for the hypoventilated blood (that comes from the base).
WHY?
Blood drained from apex has PO2 = 130, and from the base, PO2 = 90 .. then why the 130 was unable to correct for the 90?
Remember that O2 is either dissolved or bound to Hemoglobin. And the O2-hemoglobin dissociation curve is sigmoidal!



Hemoglobin can bind 4 molecules of O2.
When PO2 is 100, all Hemoglobin binding sites are already saturated with O2. So if we raise the PO2 to 130, it doesn't add any additional O2 to hemoglobin! It's in the state of plateau.
So, PaO2 = 130 doesn't mean increase in O2 content in blood . It's just added to the dissolved form of O2 (after saturating hemoglobin), and the dissolved portion is small and negligible!
So, that's why hyperventilated blood doesn't correct for hypo-ventilated blood! because you're mixing blood which is oxygen deficient (relatively) with blood that has normal oxygen! (not much more oxygen content).

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