Respiratory system physiology

Doctor Yanal Shafagoj's Notes

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• Oxygen transport

The doctor re-emphasized the O2-Hb dissociation curve; it was explained in depth in the last file. We also already established that approximately 98% of oxygen is transported bound to hemoglobin while only 1.5% is transported dissolved in plasma.

- ✓ If the fraction of oxygen in the inspired air (FiO2) was doubled from 21% to 42% what would happen to the amounts of both dissolved and hemoglobin bound oxygen?
  - o Bound oxygen will stay constant, because the population of hemoglobin molecules is almost completely saturated, and no more oxygen can be bound. (remember the sigmoidal dissociation curve)
  - o Dissolved oxygen will increase. Let's prove it using numbers:

    [dissolved O2] = PO2 x solubility of O2

    Solubility is constant and equals 0.003. While increasing the percentage of oxygen will elevate PO2 from 100mmHg to 200mmHg.

    [Dissolved O2] = 200 x 0.003 = **0.6**

    So, it increased from 0.3 at FiO2 = 21% to 0.6 here. It doubled, but it adds only a slight increase to the total amount.
    - *Actually, if you do the calculations, you will notice that PO2 becomes 250 mmHg. However, we're sticking to that mentioned by the doctor "doubled".*

Q: The amount of O2 in the inspired air (FiO2) is doubled, which of the following statements are true and which are false?

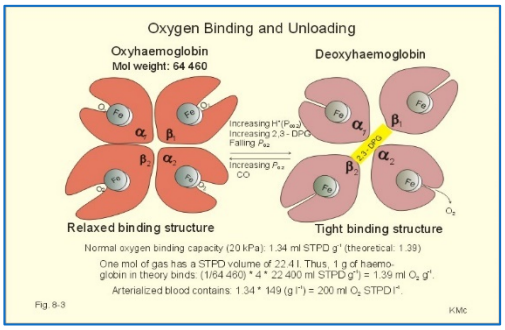
a. The total amount of oxygen will double. (F)
b. The amount of bound oxygen will double. (F)
c. The amount of dissolved oxygen will double. (T)
d. The total amount of oxygen will increase slightly. (T)

Imagine if hemoglobin didn't exist, we would need extraordinary amounts of oxygen in the air to accommodate our oxygen requirements!

➢ Oxygen binding and release

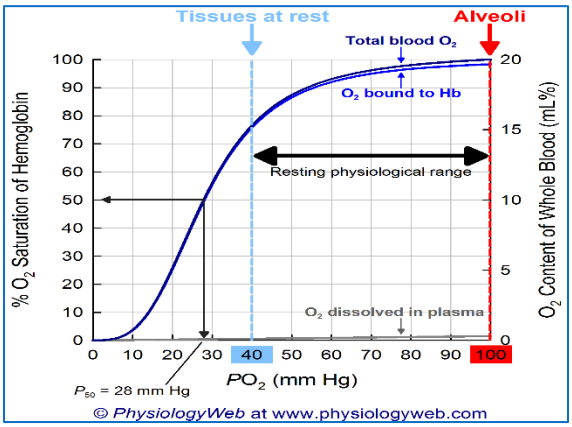
Hemoglobin has a love-hate relationship with oxygen,

- It loves oxygen when the blood is in the **pulmonary capillaries**. Meaning it has high affinity to it, the oxygen carried by hemoglobin is more or the oxygen released is less.
- While it hates oxygen when the blood is in the **systemic capillaries**. Meaning it has low affinity to it, the oxygen carried by hemoglobin is less or the oxygen released is more.
  This love-hate relationship is a simple demonstration of hemoglobin affinity to oxygen at different PO_2 and hemoglobin states.



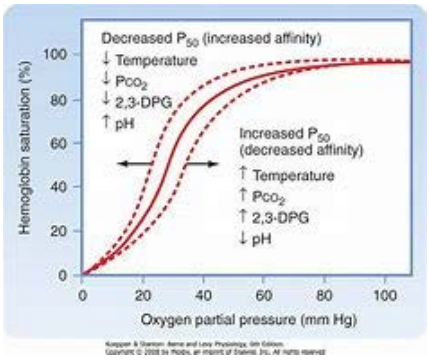
Hemoglobin has two states of structure, the tight binding structure which has low affinity to oxygen and the relaxed binding structure which has high affinity to oxygen. With the equilibrium shifting to one side depending on the surrounding conditions.

What accounts for this change in affinity?



- ✓ The change of PO2, as it is the highest in alveoli at 100mmHg and undergoes a gradual decrease until the tissues are reached where it is almost 40mmHg. Looking at the Oxygen-Hemoglobin dissociation curve you will notice how these changes in PO2 affect the binding to hemoglobin.
- ✓ When the blood is in systemic capillaries near the tissues, the concentration of CO2 is more than that of O2. As a result, CO2 will bind to hemoglobin and O2 will be released, this is called Bohr's effect. As the blood reaches pulmonary capillaries near the alveoli where the concentration of oxygen is very high the reverse Bohr's effect or Haldane's effect takes place as oxygen binds to hemoglobin releasing CO2.

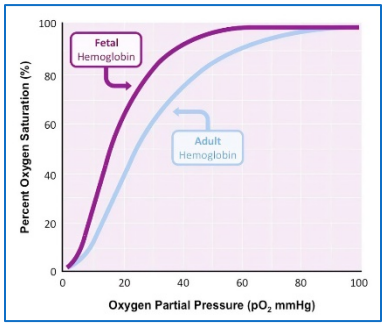
- ✓ Factors that decrease the affinity of hemoglobin to oxygen and **shifts the dissociation curve to the right** are:
    1. Hypercapnia; increase in the concentration of CO2.
    2. Acidosis; increase in concentration of H+.
    3. High temperature.
    4. 2,3-Bisphosphoglycerate, a byproduct of glycolysis, binds to the beta chain of hemoglobin in a 1:1 ratio.



  - o Note that CO2, H+ and temperature are all increased during exercise which leads to more oxygen being released to tissue *(Bohr's effect)*.
- ✓ Decrease in the previously mentioned factors will increase the affinity of hemoglobin to oxygen and will **shift the curve to the left** *(Haldane's effect)*.

Application: fetal hemoglobin

- ✓ Fetal hemoglobin (HbF) is different from adult hemoglobin (HbA) as it has a gamma chain instead of a beta chain $\alpha_2\gamma_2$.
- ✓ What characterizes HbF is that it doesn't bind to 2,3-BPG. Therefore, its dissociation curve would be shifted to the left indicating that it has increased affinity to O2. The oxygen carrying capacity of HbF is 30% more than that of HbA.



- ✓ The significance of this feature lies in the fact that the fetus doesn't have a lung, it acquires its oxygen requirements through the placenta in which the PO2 is 40mmHg. If the fetus had HbA, only 75% of the hemoglobin population would be occupied at this PO2 but a population of HbF -given its characteristics- is able to be almost completely saturated at this PO2.

Application: CO toxicity

- ✓ Hemoglobin has 250x more affinity to CO than to O2. This means that if you have a certain amount of hemoglobin with PO2 of 100mmHg and PCO of 0.4mmHg which is 250 times less than 100, one half of the hemoglobin population would be occupied by oxygen while the other half will be occupied by CO.
- ✓ Breathing CO will have the following effects: it shifts Hb dissociation curve to the left, meaning it increases the affinity to O2, decreases arterial [O2] while PaO2 doesn't change. It will be as if the person has anemia.

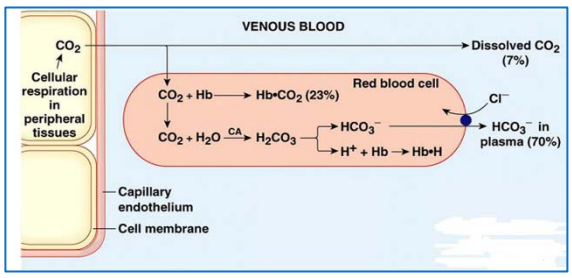
• Carbon dioxide in blood

CO2 coming from the arterial end carries 48ml/dl of CO2, as it passes through the capillary bed, an additional 4ml is added resulting in venous CO2 concentration being 52ml/dl. Since the cardiac output is 50dl/min and 4ml of CO2 are added per dl 50x4=200ml of CO2 are transported per min, this number also resembles CO2 production per min.

How is CO2 transported in the bloodstream?

Percentages differ between sources don't pay attention to them. We're mostly interested in most and least.

1. **7% dissolved in plasma.** Calculated by this equation:
   [dissolved CO2 in arterial blood] = PaCO2 x solubility of CO2
   = 40mmHg x 0.06ml/mmHg = 2.4 ml
   [dissolved CO2 in venous blood] = PvCO2 x solubility of CO2
   = 45mmHg x 0.06ml/mmHg = 2.7 ml
   The difference between arterial and venous dissolved CO2 is 2.7-2.4=**0.3ml**. 0.3ml as a percentage of the total 4ml of CO2 transported in the blood is **7%.**
2. **30% bound to hemoglobin as carbaminohemoglobin.**
3. **63% as bicarbonate.**



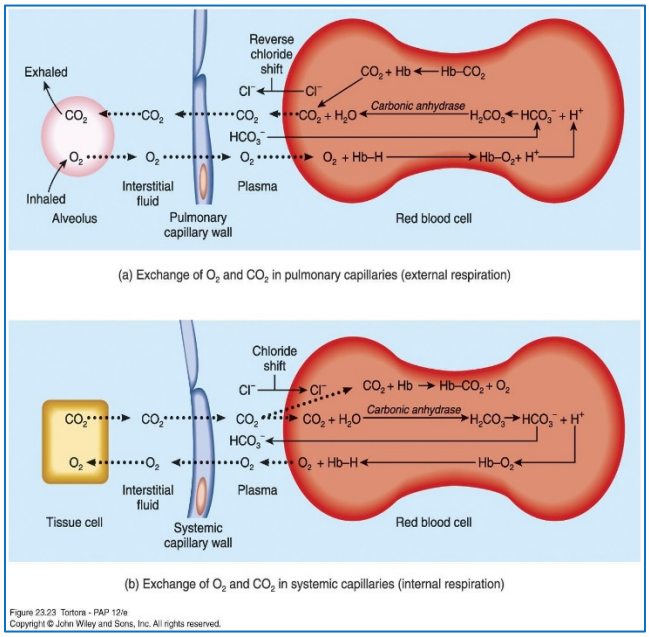
➢ Transport of CO2 as bicarbonate

✓ When blood reaches the **tissues**: $CO_2 + H_2O \rightleftarrows H_2CO_3 \rightleftarrows HCO_3^- + H^+$

- CO2 exits the cells towards the interstitium and enters the blood.
- Inside RBCs, CO_2 binds to water forming carbonic acid which immediately dissociates into HCO3- and H+ in a reversible reaction (the reaction is facilitated by carbonic anhydrase enzyme).
- Converting CO2 to bicarbonate is a step done to maintain a gradient of CO2 between blood and interstitial fluid allowing for more CO2 to flow towards the blood and preventing its accumulation in cells.
- If the enzyme is inhibited by acetazolamide, this will lead to rapidly abolished gradient thus preventing more flow of CO2, which explains the significance of the enzyme.

✓ Bicarbonate is a negatively charged molecule, when it exits the RBC, another negatively charged molecule must enter to compensate for the lost negative charge and maintain the state of **electroneutrality**.

- Cl- enters RBC and this is called **chloride shift**.
- It is only logical to conclude that Cl- concentration is less in venous blood compared arterial blood.



(a) Exchange of O_2 and CO_2 in pulmonary capillaries (external respiration)

(b) Exchange of O_2 and CO_2 in systemic capillaries (internal respiration)

✓ When blood reaches the **lungs**,

- The opposite reaction ensues; bicarbonate enters the RBC and reacts with the readily available H+ to form carbonic acid which immediately dissociates into CO2 and H2O. CO2 leaves the cells towards the alveolus.
- We note here that when bicarbonate enters the RBC, Cl- will exit in its place in a process called reverse **chloride shift**.

Why is hemoglobin present in RBCs and not left to swim freely in plasma? The presence of hemoglobin in RBCs has many advantages, some are:

1. protecting Hb from degradation by plasma proteins.
2. preventing Hb from being filtered in the glomeruli.
3. Reductase that converts Fe+3 to Fe+2.
4. Carbonic anhydrase reaction

• Controller of respiration

✓ The main aim of the control system is to ensure the proper maintenance of normal homeostasis of the arterial blood gases (ABGs). Keeping PaO2=100mmHg PaCO2=40mmHg and Ph=7.4.

✓ One tool that is employed to accomplish this objective is **enhancing ventilation**, doing so it tries to bring the composition of the alveolar air close to that of the outside air, where PCO2 is zero and PO2 is 150 (↑O2 and ↓CO2).

✓ Another tool is **reducing ventilation** (↓O2, ↑CO2).

✓ *hyperventilation* is when alveolar ventilation is more than CO2 production and results in the decrease of PaCO2 while *hypoventilation* when alveolar ventilation is less than CO2 production which increases PaCO2. *PaO2 changes too during hyper and hypoventilation but for the definition, PaCO2 values are considered.*

✓ During normal respiration PaCO2 remains constant as the increase in alveolar ventilation is balanced by the increase in CO2 production.

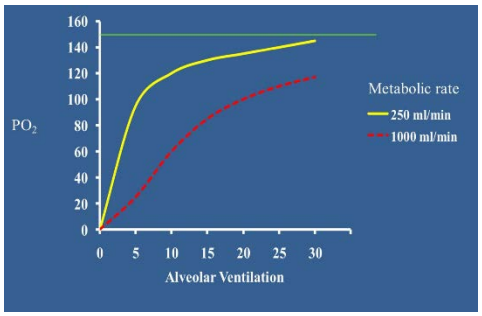
✓ How about O2 levels? The maximum arterial PO2 achievable during normal ventilation is around 150, limited by the oxygen fraction in atmospheric air at sea level (21%). Beyond 150, only increasing the O2 fraction in air can elevate arterial PO2. In exercise, although ventilation does increase, it is faced by a proportionate increase in O2 consumption, keeping PaO2 constant.

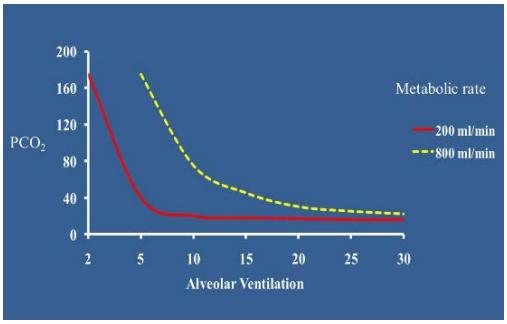
- Increased ventilation without a change in ABG is termed hyperpnea, which is the case in mild to moderate exercise.

What is said here aligns with these equations:

$$PaCO_2 = (VCO_2/VA)*k$$
$$PaO_2 = (VA/VO_2)*k$$

VO2: O2 consumption
VCO2: CO2 production
VA: alveolar ventilation





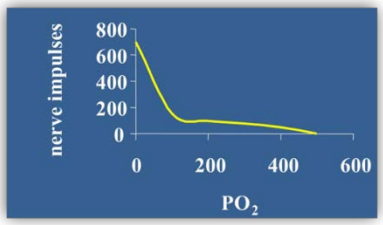
✓ Notice how PaO2 increases with ventilation and PaCo2 decreases with ventilation.

✓ The **feedback regulation** in this system is governed by the levels of ABGs, namely, **PaO2**, **PaCO2**, and **(H+)** sensed by chemoreceptors.

- O2 Level:

  An **increase** in O2 levels **doesn't influence the respiratory center** while a **decrease** in O2 levels below 60mmHg does **activate a response to increase ventilation.**

  Notice how nerve impulses are greatly elevated when PO2 drops below 60 mmHg. To be more accurate, the curve should be horizontal beyond 60.



- CO2 Level:

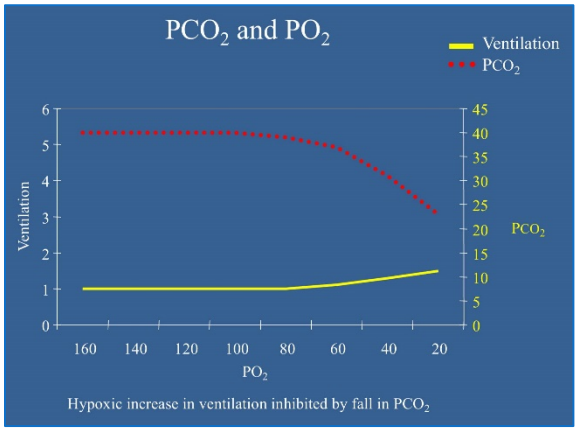
  Elevated CO2 levels activate the center to increase ventilation (to eliminate excess CO2). Low CO2 levels suppress the center (to retain a certain amount of CO2).

  Note: Both an increase and a decrease in PCO2 impact the center because maintaining normal CO2 levels is crucial.

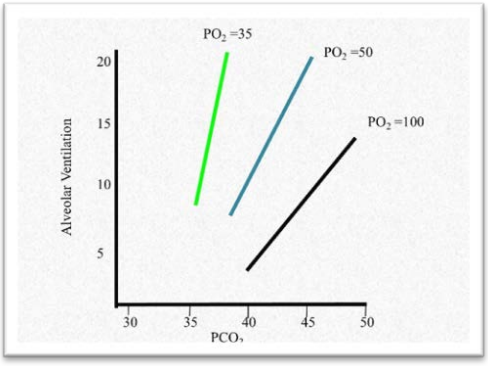
- H+ follows a similar pattern to CO2 where acidosis induces hyperventilation and alkalosis induces hypoventilation.

How do these factors communicate?

Let's look at the relationship between PaCO2 and ventilation under the effect of PaO2.

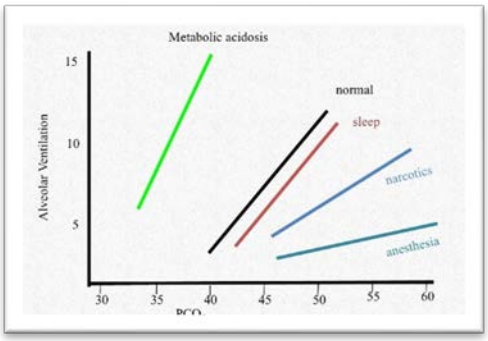


- This graph demonstrates the relationship between PaCO2 (red line) and ventilation (yellow line) and the levels of PaO2 on the x-axis.
- PaCO2 is directly proportional to alveolar ventilation, they have a linear relationship as long as PaO2 is higher than 60mmHg.
- An increase in PaCO2 drives higher ventilation levels, higher ventilation leads to an increase in PaO2 which has no effect on ventilation.
- If $PaCO_2$ decreases, it will lower ventilation levels which in turn will lower PaO_2, if PaO2 drops below 60 mmHg, it will drive more ventilation and **oppose** PCO_2 effect. At this point, PaCO2 and ventilation lose proportionality and will have a nonlinear relationship because hypoventilation driven by a decrease in PaCO2 is opposed by hyperventilation driven by a decrease in PaO2.



- studying the **carbon dioxide response curve** at different O2 levels, we can deduce that hypoxemia potentiates hypercapnia (3+4=11), which means that an increase in PaCO2 will be faced with a disproportionate increase in ventilation **if** it is accompanied by a decrease in PaO2 below 60mmHg, because both of these factors (hypoxemia & hypercapnia) induce hyperventilation. This appears clear in this graph as you notice an increased slope at lower PO2 levels.

- On the other hand, acidosis does not potentiate hypercapnia, it rather has an addition effect (3+4=7). This means that there will be a proportionate increase in ventilation facing the increase in PaCO2 with decreasing ph. In other words, the slope will remain constant, but the line will be shifted to the left.



- The black and green lines in this grahp should be completely parallel (have the same slope). Notice how metabolic acidocis shifted the gragh to the left.

The regulation of respiration involves the collaborative interaction of three primary components:

1. **Sensors, Receptors, and Afferent Pathways:** These elements gather information about key factors such as CO2 levels, O2 levels, and pH.
   - Sensors detect changes, receptors transmit these signals, and the afferent pathways carry the information towards the central controller.
   - The afferent neurons going towards the respiratory centers are the vagus (CN10) and the glossopharyngeal (CN9).
2. **Central Controller:** This component composed of neurons in the brain stem which integrates the signals received from sensors and receptors. It interprets the information and issues commands or orders via efferent pathways, namely the phrenic nerve.
3. **Effectors:** The effectors, in this context represented by **respiratory muscles**, receive the output from the central controller. They then produce a response.

To elaborate more on the central controller,

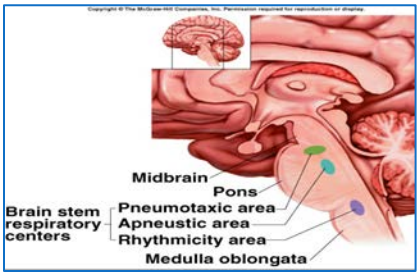
- ✓ The central nervous system (CNS) encompasses the brain, brainstem, and spinal cord. The primary regulator of respiration is the respiratory center,

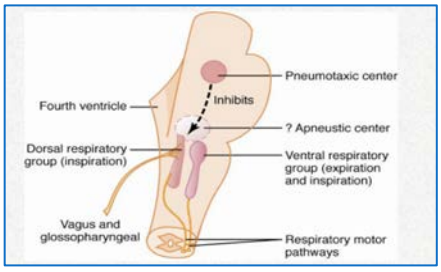
which is a grouping of nuclei found in the brainstem. This center is divided into:

1. **Medullary respiratory center in the medulla oblongata**

- Dorsal respiratory group: INSPIRATORY NEURONS primarily responsible for initiating inspiration, and stimulation of the diaphragm by regulating the activity of the phrenic nerve. They work at rest.
- Ventral respiratory group: INSPIRATORY AND EXPIRATORY NEURONS involved in both inspiration and expirations. They are inactive at rest and active during exercise.
    - During quiet breathing or at rest, expiration is a passive process, and no expiratory muscles are actively engaged. In this state, **the dorsal group takes the lead**, stimulating phrenic neurons located between the third and fifth cervical vertebrae. These phrenic neurons, in turn, activate the diaphragm to initiate the process of inspiration.
    - In situations where breathing requires more effort, such as during forced inspiration or expiration, the **ventral neurons become active**. This shift in neural activity signifies a stronger respiratory effort, involving additional muscles to adjust lung volume.
- Neurons in the reticular formation of the medulla oblongata form the rhythmicity center pacemaker neurons located in the upper part of the ventral respiratory group (VRG)
    - These neurons control automatic breathing as they consist of I neurons that fire during inspiration and E neurons that fire during expiration. They give impulses for 2 seconds and stop firing for 3 seconds, so the cycle is 5 sec. 60/5 = 12 cycle/min which is the respiratory rate. We can also conclude that expiration is longer than inspiration by one second.

2. **Accessory respiratory centers in the upper and lower thirds of pons**
   - ✓ **Pneumotaxic center** in the upper third: it inhibits dorsal respiratory neurons and **apneustic center** therefore it switches inspiration off.
   - ✓ **Apneustic center** in the lower third: promotes inspiration (dorsal neuron) by stimulating the inspiratory neurons in the medulla.
     - If the pneumotaxic center is removed there would be prolonged inspiration and occasional expiration (apneusis); apneustic center loses inhibitory regulation.





- ✓ Note that orders come from the cortex, pass the respiratory center then reach the spinal phrenic nerve. In some instances, the signal can bypass the respiratory center and go directly from the cortex to the spinal nerve through the **cortico-spinal tract.**
  - o This becomes evident when you *decide* to hold your breath, you do it willingly without the effect of the respiratory center as the cortex is responsible for decision making.

➢ Chemoreceptors

Two groups of chemoreceptors that monitor changes in blood PCO2, PO2, and pH.

1. **Central chemoreceptors:** these are medullary chemoreceptors, neighboring the respiratory centers, connected with the dorsal respiratory group (DRG). They are **most sensitive to changes in arterial PCO2, through changes in H+.** $CO_2 + H_2O \rightleftarrows H_2CO_3 \rightleftarrows HCO_3^- + H^+$

   Remember the carbonic anhydrase reaction, you can easily conclude that an increase in CO2 will lead to an increase in H+ and a decrease in pH.

- ✓ CSF is more susceptible to marked changes in pH, this is attributed to the fact that it doesn't have as many protein buffers as the blood.
- ✓ The blood-brain barrier (BBB) and CSF barrier prevent charged hydrogen ions (H+) from easily crossing into the brain, but there's no barrier for carbon dioxide (CO2), it diffuses easily through biological membranes.
- ✓ increased CO2 yields an increase in H+ and a subsequent decrease in pH. If a person voluntarily holds their breath PaCO2 can increase up to 50mmHg, and central chemoreceptors are activated by the decrease in pH caused by CO2 dissemination to the CSF which triggers a response from the dorsal respiratory group to order respiration.
- ✓ Holding one's breath decreases PaO2 as well, but not to less than 80mmHg which is -as stated previously- not enough to elicit a response from the respiratory center.
  - *Keep in mind that increasing H+ the blood directly will not have the same effect as increasing CO2, because H+ can't cross BBB easily, however, in chronic acidosis, H+ may cross BBB and stimulate central chemoreceptors.*
- ✓ So now the signals to the phrenic nerve coming from the cortex through the cortico-spinal tract inhibits respiration (because a person took the decision to hold their breath) are counteracted by signals commanding respiration coming from the dorsal respiratory neurons (because central chemoreceptors sensed a change in pH).
- ✓ The signals from the respiratory center will take over and the person will breathe involuntarily. This is why we say that no one can kill him/herself by holding their breath!

	CSF	***BLOOD***
$HCO3^-$	24	28
protein	**<45 mg/dl**	**6-8 g/dl**
pH	7.32	7.4

2. **Peripheral chemoreceptors:**
   - ✓ The carotid and aortic bodies.
   - ✓ Sensitive to changes in ABGs CO2, H+ and O2, but **especially to changes in PO2**, once they detect these changes, they send signals to the respiratory center through the vagus (CN X) and glossopharyngeal (CN IX) nerves.

- Peripheral chemoreceptors are mainly affected by PO2 and to a lesser extent by PCO2 and H+. The effect of H+ on the peripheral receptors, although one-seventh of the central response, is five times faster.

✓ These receptors are a collection of sensory neurons, they are cells, and like other cells they are surrounded by interstitial fluid and will sense the changes in O2 that happen in it, therefore the composition of the surrounding interstitium must accurately reflect the composition of the blood. There are two possible explanations to this phenomenon:

- If the cell is metabolically inactive and doesn't consume any O2, consequently, PO2 in the interstitium would be equal to PaO2. But this can't be the reason since carotid body cells are the most active cells in our body.
- If the cells had an exceptionally high blood flow providing a high amount of O2 and allowing a very little proportion of it to be consumed despite the high metabolic activity, as a result, the PO2 will not drop significantly as blood passes through and will remain equal to PaO2. This is the correct explanation.

✓ To put things into perspective, blood flow to carotid bodies reaches 20 ml/g, while they only weigh 27 mg, these cells have a dedicated artery known as the carotid body artery. This exceptional vascularization ensures efficient O2 monitoring.

the kidneys, weighing 250 gm, have the second-highest blood flow at 4 ml/g,

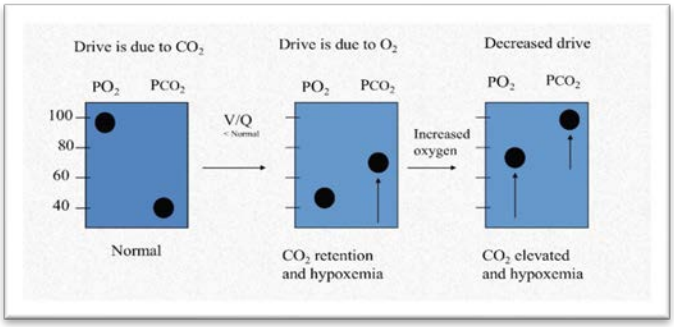
while the heart, with a weight of 300 gm, displays a blood flow of 0.8 ml/g.

Tissue	Blood flow (ml/g/min)	A-V difference (Vol %)	Flow ml/min	O_2 consumption ml/min
Heart	0.8	11	250	27
Brain	0.5	6.2 (25-30% Extraction)	750-900	
Skeletal Muscle	0.03	6	1200	70
Liver	0.6	3.4 Reconditioner organ		
SKIN	0.1			
Kidney	4.2	1.4	1250	18
Carotid bodies	20	0.5	0.6	

The doctor briefly mentioned stretch receptors in alveoli or bronchioles, too much inflation can lead to an order of stopping ventilation by stimulating pneumotaxic and apneustic centers and inhibiting DRG.

Application: treatment of COPD

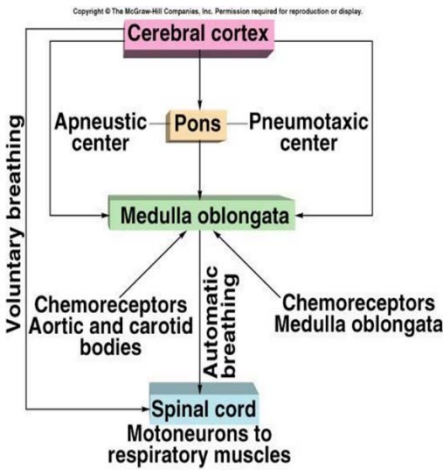
- ✓ COPD patients have hypoxemia, PaO_2 is decreased while $PaCO_2$ is increased. If you treat those patients with pure oxygen, you will most probably kill them.
  - o What drives hyperventilation in these patients is the decreased PaO2 sensed by peripheral receptors sensitive to changes in PaO2. Pure oxygen treatment will elevate PaO_2 above 60mmHg so the driver for hyperventilation is removed, this will ultimately lead to hypoventilation, an increase in PaCO2 which will suppress dorsal respiratory neurons leading to death.
  - o In this case, we treat them with 42% oxygen intermittently.



In COPD, respiratory centers adapt to hypercapnia and become less responsive to it due to chronic exposure. So, they are more sensitive to hypoxia, which is the main driver for hyperventilation.

A wrap up; the Dorsal Respiratory Group receives input from three sources:

1. Accessory neurons in the upper and lower thirds of pons.
2. Peripheral chemosensitive receptors, through the 9th (glossopharyngeal) and 10th (vagus) cranial nerves.
3. Central chemosensitive receptors, located in the medulla, are activated by excessive or insufficient levels of H+.



• Respiration during exercise

This is a thing that we have discussed many times, just a few add ons.

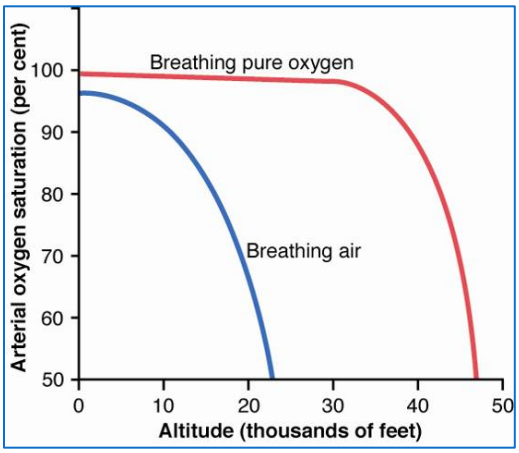
- ✓ ABGs remain unchanged during exercise by the effect of hyperventilation and increased cardiac output, but what drives hyperventilation?
  - o The mechanism of that is not totally understood, however, it is hypothesized that the movement of muscles, tendons and joints stimulates proprioceptors which send impulses to brain respiratory centers inducing hyperventilation.

Remember O_2 is not self-compensatory.

• Ventilation at different altitudes

- At high altitudes:

- ✓ PO_2 at sea level is equal to 21% of Patm (160 mmHg), with ascending, PO_2 decreases because Patm decreases. But the percentage will stay constant. At mount Everest for example, Patm=226mmHg, 21% of that is 45mmHg. If you take the contribution of water vapor in the ADS almost nothing will be left! So, at high altitudes one will suffer from hypoxia.
- ✓ P_AO_2 =100 mmHg, it drops down at high altitudes.
- ✓ If P_aO_2 drops below 60 mmHg, peripheral chemoreceptors will be stimulated driving hyperventilation.
- ✓ PCO_2 is also decreased at high altitudes, and it decreases more with hyperventilation leading to hypocapnia.
  - o Hypocapnia opposes hyperventilation by increasing CSF pH which inhibits central chemoreceptors (suppresses ventilation).
  - o This can explain the much lower instantaneous ventilation than expected after ascending to high altitudes (ventilation at high altitudes instantaneously increases by 70% of ventilation at sea level (30 L/min, 12 L/min at sea level)).



- ✓ However, the body can acclimatize within few days:

- Kidneys perform two functions:
  - They sense the increase in pH and start to eliminate HCO_3- in the urine; eliminating bicarbonate shifts the reaction to the right, hence more H^+ is being produced. Lowering pH in CSF mainly affects central chemoreceptors inducing hyperventilation.

Acclimatization: physiologic adaptation

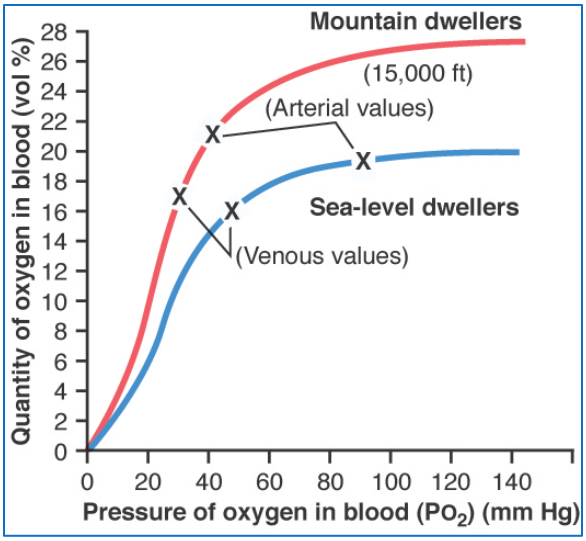
$$H_2CO_3 \rightleftharpoons HCO_3^- + H^+$$

pH = 6.1 + log HCO3-/ (0.03 × PCO2)

  - Hypoxia induces erythropoietin production from the kidneys, which activates bone marrow to produce more RBCs thus increasing hemoglobin concentration and oxygen carrying capacity; those individuals develop physiologic polycythemia.
    →by those two mechanisms, both ventilation and perfusion are increased and matched (normal V/Q ratio); acclimatization is achieved (ventilation reaches up to 400%).
    →kidneys need up to 5 days to sufficiently achieve those two functions.

✓ At this point, sudden return to sea level pressures removes the driver for hyperventilation (hypoxia), so one would expect a normalized ventilation. However, PCO2 increases concurrently leading to acidosis and ultimately hyperventilation; this is explained by kidneys' elimination of HCO3- which is disrupting equilibrium and causing more H+ production at normal PCO2. Again, kidneys take few days to cease bicarbonate elimination.

Polycythemia increases blood viscosity which increases vascular resistance and ultimately leads to hypertension.



- **At low altitudes** (Jordan valley for ex):
  - ✓ PO_2 in the outside air is increased and thus P_aO_2 is also increased, however, ventilation, O_2 saturation and concentration **remain unaffected**.

Questions:

Q1: What is atmospheric PO_2 at 10,000 m (barometric pressure = 508 mmHg)? (Person has normal alveolar ventilation)

a. 95 mmHg
b. 106
c. 149
d. 159

Answer: b, 508*0.21=106

Q2: In which of the following conditions is alveolar PO2 increased and alveolar PCO2 decreased?

a. Breathing air with 19% PO2
b. Increased alveolar ventilation and unchanged metabolism.
c. Decreased alveolar ventilation and unchanged metabolism.
d. Increased metabolism and unchanged alveolar ventilation.

Answer: b, remember PaCO2=(VCO2/VA)*k & PaO2=(VA/VO2)*k

Q3: What is the effect of anemia on ventilation?

a. Decrease ventilation.
b. Increase ventilation.
c. No change in ventilation.

Answer: c, in anemic patients there is no change in PaO2 therefore there is no change in ventilation.

Remember these facts about anemia patients: Hb concentration is low, arterial [O2] is low but PaO2 is normal, O2 extraction ratio is high, mixed venous [O2] is low, PvO2 is low and O2 saturation of Hb in venous blood is decreased.

Q3: breathing CO acutely will ____ respiration:

a. Increase
b. Decrease
c. No change

Answer: c, breathing CO will not change PaO2, so there will be no change in respiration.

Remember these facts about breathing CO: it shifts Hb dissociation curve to the left, meaning it increases the affinity to O2, decreases arterial [O2] while PaO2 doesn't change. It will be as if the person has anemia.

Q4: metabolic rate is doubled but alveolar ventilation is not changed. What happens to PaCO2?

a. Increase
b. Decrease
c. No change

Answer: a, PaCO2=(VCO2/VA)*k

The end of respiratory physiology, GOOD LUCK<3

V2

-two pictures added at the top of page 8

V3