Respiratory system

What is the function for the respiratory system?!

1. Gas exchange
2. vocalization and resonation
3. Smelling
4. Maintainance for pH
5. Maintain the temprature
6. Metabolism
7. elimination of heat (+ ATP).
8. Olfaction

Respiratory system picture ⇒

Often when we get the, it infected the upper respiratory tract.

The smell and vocalisation happen in the upper respiratory tract, also the resonation occur on the upper respiratory tract which is when you close your nasal cavity, your will not be able to pronounce letters propably.

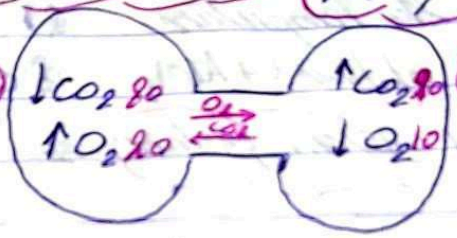
resonation → هو صدى الصوت الي بطلع الكلمات بشكلها الطبيعي.

The total pressure of a mixture of the gases equal the sum of the partial pressure of each gas.

The total pressure for the air is 760 which is the sum 160 O_2 . 716 N_2 ---etc

if we have 2 containers and we connect them to each other and each one have a different mixture of gases ⇒ the gases will move according to their own partial pressure.

Before. (total pressure = 100) (total pressure = 100)

after (total pressure = 100) 

(total pressure = 100)

it will move from the higher pressure to the lower pressure according to its type.

The functions of the nasal cavity ⇒
① Resonation ② Olfaction
③ Trapping for dust particles (Nasal hair) ⇒ Filtration ④ humidification ⇒

For the humidification, we notice after a period of time the mucus will be thick, why ?!
because after a period of time the air takes the water vapor from the mucus when it enter

Why we need to do humidification ?!
because if the air that entered is dry, it will take a water vapor from the cells inside, so the cell inside

will dry cause it to be permeable to infection to break down and damaged lungs so in order to prevent this from occur we need to enter the air with 100% humidity.

When someone sleep with an open mouth or snores ⇒ So it will breath from its' mouth so when he wake up his throat will be dry because the dry air take the water vapor from cells so it will dried and die. or weak or easy to be infected.

So the air that enter the nasal cavity is a mixture of gases with an P_{Bo} pressure but when it enter we add a new gas to the mixture of gases which is the water vapor but the total pressure stayed the same (P_{Bo}) thats mean that all the gases will be lowered in order to not cause a damage to the lungs so we lowered the amount taken of the O_2. The partial pressure of O_2 was 160 in the air when it goes to the lungs it was 150 ⇒ ... thats mean there is no mercury from the O_2 only where replaced by water vapor H_2O.

The air enter the nasal cavity → pharynx → Larynx → trachea → inside bronchus → bronchioles → terminal bronchioles
all this area we called dead space → it means that there's no use for the air to stay in these areas → because we are not doing an gas exchange.

The only part that I do an gas exchange in it is called the alveoli which is the inner part of the last part of the lungs

For normal breathing → 0.5L enter and 0.5L goes out

From the 0.5L there's 150 ml stays in the Nasal cavity, trachea and bronchus and when we take out the air, the 150ml is the first part that goes out.
So there's 150ml is the last one enter and the first one go out and we don't take advantage from it because it came to the dead space.

We have also a physiological dead space.
What is the physiological dead space?!
When someone smoke, there's a material called tar (القطران) enter your lungs and accumulate on the alveoli (المادة التي يستخدمونها في الشوارع الرفلتة) so the alveoli that have to make an gas exchange, can't do an gas exchange now because the tar accumulate on the surface of the alveoli So part of the alveoli will have alot of tar on it (كأنه عملنا سفلت عليها) so it will not do its JOB الجسم يقدر يعوض بمعدل 5 بالمئة بعد ما يتوقف عن التدخين

physiological dead space → dead not because its nature is to be dead it die due to sth prevent it from doing its JOB like the blockage of bronchioles that lead to the alveoli

Also the infection in alveoli will stop it from doing the gas exchange (damaged).

Also the pulmonary edema cause a physiological dead space which means the accumulation of excess fluid in the lungs inside the alveoli which can happen due to the weak heart work on the taking back the blood and fluids which cause it to accumulate the

The fluid in the lungs in the alveoli

The difference between the anatomic dead space and physiological dead space?!

anatomic dead space
normal
areas don't do an gas exchange

physiological dead space
abnormal
areas that was doing an gas exchange but due to any external effects it stopped.

هما الهوا إلي راح ناحية ال alveoli الي إعتبرناها physiological dead space من رح يصير له gas exchange فصار مجموع الغاز الي ما إستفنا عنه 200ml بدل 150ml.

Nasal cavity ⟹

Trachea do the vocalisation while Nasal cavity do the resonation

We also have an air that enter the oral cavity we have the Epiglottis that if solid or watery fluid is coming from the oral cavity once it touched it will close up the trachea (the respiratory system.

Whenever we swallow we actually inhibit the respiration due to the close of the respiratory tube by Epiglottis.

Also the Esophagus extends and push the Trachea.

Choking (الشرقة) ⇒ When we eat the grape and crush it, it will enter quickly before the Epiglottis close which cause it to cough due to the entering a particles that are not gases. (the one that enter is the liquid.).

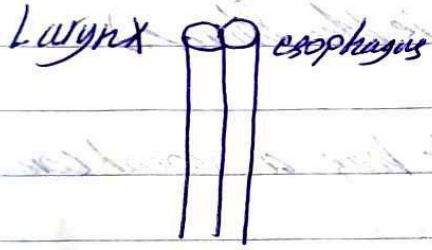
Trachea is the vocal box, it has a vocal cords

The pharynx is 3 parts → Naso pharynx
= or a. pharynx
Laryngo pharynx

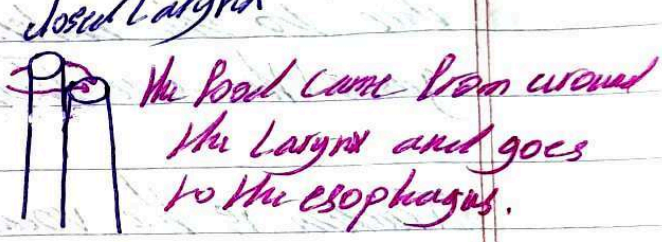
Nasal pharynx ⇒ nose
oral pharynx ⇒ mouth
Laryngeal ⇒ goes to the Esophagus or Larynx
Thats why we called the pharynx a common pathway.

We have the soft palate which has an uvula in its end that located in the end of the nasopharynx which close when we are eating in order to prevent the food from going to the Nasopharynx.

ليش لما بلع تفاحة أدم بنغلع ويسّد ؟!
لأنه لما بلع بترفع Larynx وبسكرها عشان الأكل يمشي من حولها لما يمشي منه فوقها ويجي ع الesophagus



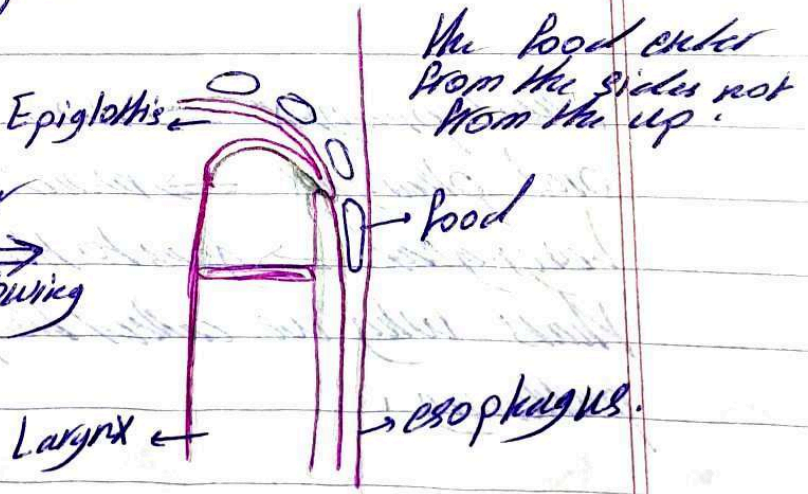
Before swallow



after swallow

Swallowing action picture

after swallowing



تفاحة أدم بتزيد خشونة الصوت .

Trachea →

Why the adventitia is a C-letter not a complete circle ?!

because posterior to it, we will find the esophagus and posterior to the esophagus we have the vertebral column. So in away if the esophagus is all the time is collapsed and we have the food that is going inside, So it means that the esophagus will expand so if we have a cartilage complete between Mousa and esophagus and we have the vertebral column posterior to it that's mean that there is no way for the esophagus to expand so instead of cartilage we have a smooth muscle which has elasticity and it can get expanded inside the trachea and allow esophagus to expand inside the trachea that's why the respiration inhibited because of the expandtion of the esophagus inside it that's why sometimes the choking occur because the food get stuck in the trachea.

Conducting Zone →
if we are talking in our quite breathing 500ml of air and the length of trachea, nasal cavity, bronchus and bronchioles are about 150 mL so we will take a benifit from 350mL
150ml the last thing get in and first one goes out, that's why it's an anatomic dead space.

Alveolar Structure →

Surface tension → alveoli they are too tough to expand, it means they have a high surface tension (أجزاءها ملتصقة ببعض بقوة) because of that, we need a very high energy to make it expand and take the air. So the type 2 synthesis the surfactant that reduces the surface tension. (طبقة عبارة عما يتبقى على الخلايا بتقلل ال surface tension)

So if someone has a problem in Type II cell he will have a difficulty in breathing

When we expand the lungs, we will increase the volume so the pressure will decrease, so the pressure of the air

will be higher than the pressure inside the lungs making the air goes from outside to inside the body. And when the volume decrease the pressure will increase and the air will goes from inside the lungs to outside.

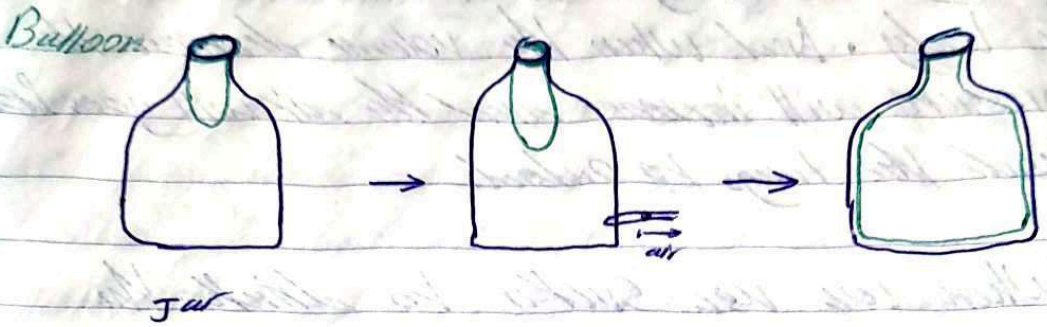
When we use sucker to drink the cola what happen?!
We increase the volume of the oral cavity which decrease the pressure (758) while in the sucker is 760 so the air will enter from the sucker to the oral cavity making the fluid enter the sucker.

(h) Exchange surface of alveoli →

Inorder for the gas to go outside the alveoli and enter the capillary, it have to ⇒
① first dissolve in surfactant ⇒ very thin layer so it didn't increase the space (75m).

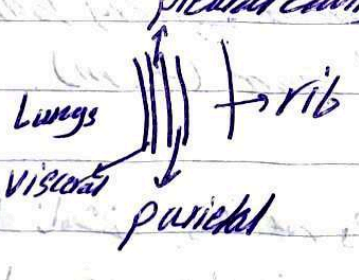
لما يفوت هوا ناشف بوخد منه رطوبة الـ surfactant فطوالي الـ type II بتفرز مكانه- إعشانه هيك بنحس إنه من قادريه نوخد نفس.
ممكن عند بعض الناس ما يكون في surfactant فرح يكون في صعوبة بالتنفس لانه بالأساس هو بسهل التنفس.

pleurae →



If we have a ballon and we put it in the jar as we see in the first pic and we want to blow it in the jar → so we have to take all the air inside the jar. so the balloon will expand and takes the jar shape.

We have the parietal pleurae membrane is stuck on the thoracic cavity from inside and the visceral pleural membrane stuck on the lungs from outside.



يعني ال visceral ملزقة على الرئتين من برا
أما ال parietal ملزقة على القفص الصدري من جوا

So the pressure in the pleural cavity is negative (-3mm Hg) عشان يكون أقل من الضغط الجوي وتقل الأنشية منها.

Inspiration ⇒

① Inspiratory muscles contract (diaphragm descends, ribcage rises) (diaphragm goes down).

② Thoracic cavity volume increases

③ Lungs stretched; Intrapulmonary volume increases

④ Intrapulmonary pressure drops (0-1 mmHg)

⑤ Air (gases) flows into Lungs down its pressure gradient until intrapulmonary pressure is 0 (equal to atmospheric pressure).

Changes in anterior-posterior and superior-inferior dimensions ⇒

* Ribs elevated (مرتفع) and Sternum flares as external intercostals contract.

* Diaphragm moves inferiorly during contraction.

يدخل 0,5L عن طريق تقليل الضغط ويطلع 0.5 L عن طريق زيادة الضغط

Inspiration means that I activate 2 types of the muscles:- ① Diaphragm ② external intercostals cuz Once we activate them that means I increased the volume of the lungs → the pressure is dropped → so the air will come in.

If we want to do the expiration all I need is the relaxation of these muscles.

Once the diaphragm is relaxed it will goes up again and the external intercostals will bring down the ribs and that's mean we are reducing the volume and increasing the pressure so the air will goes out

So we say its an active inspiration and passive expiration, why?!
inspiration need an energy while the expiration we don't need an energy

Why we die if we breath CO?!
not because there's no O_2 but the CO will bind to the Hb instead of O_2 making no O_2 reach the cells

Venous Blood → Dissussion of the picture
The CO_2 is transported in 3 means →
① Dissolved CO_2 ⇒ the most important and similar to O_2 but its percentage is 7% not like the O_2 which was 2%

② About 23% of the CO_2 is transported inside the red

blood cell bind to the Hb (like O_2, but the ~~CO_2~~ binding occur in a different site than the O_2).

③ We have 70% of CO_2 is transported as bicarbonate ⇒ How this happen?!
① CO_2 reacts with H_2O produces the carbonic acid
② Carbonic acid is a very weak acid and and does dissociate into bicarbonate and H^+ ions

③ the H^+ ion will also bind to another binding site on the Hb and is transported as bound to Hb

④ bicarbonate will move out of the red blood cells and will be replaced by the Cl^- and this process we call it chloride shift chloride shift (allowing HCO_3^- to go out and replace it with the negatively charged Cl^- to balance the cell charge).

Why the CO_2 don't react with the H_2O outside the cell but it does react with the ~~CO_2~~ H_2O inside the red blood cell?!
due to the presence of the enzyme CA inside the

red blood cell which called carbonic ~~anhydrase~~ anhydrases

$$CO_2 + H_2O \overset{CA}{\rightleftharpoons} H_2CO_3^- + H^+$$

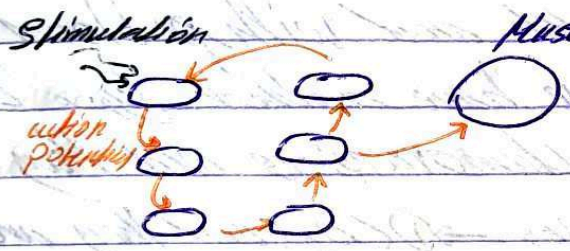
In order to continue producing the HCO_3^- and H^+ we have to decrease the conc of the H^+ and HCO_3^- so if it accumulate on the cell the direction of the equilibrium will go toward CO_2 and H_2O so they will accumulate ~~conc of HCO_3^- and H^+ will HCO_3^- and H^+ will~~

(5) then the red blood cell will move until it reaches the alveoli and due to the low conc of CO_2 in it the dissolved CO_2 will enter and due to the decrease of conc of dissolved CO_2 the CO_2 inside the cell will unbind from the Hb and goes out the cell. ~~and the~~

(6) the HCO_3^- will enter from the plasma to the cell and the Cl^- will goes out and the H^+ will unbind from the Hb and react with the HCO_3^- giving H_2CO_3 that will ~~the~~ dissociate into the CO_2 and H_2O in the presence of CO_2 and the CO_2 will go out.

What the significants of the bicarbonate presents in the blood?!
Buffer => its one of the strongest buffer system that we have in ablood => in order to ~~monitor~~ maintain the pH. Whenever the pH increases or decreases, the bicarbonate workon it.

Figure 18-16 Dissection =>



-> those muscles are the ~~Diag~~ Diaphragm and external intercostals
So they will contract leading to the inspiration

While the action potential do not reach the muscle it will be in relaxation leading to the expiration.

Central pattern generator → is a pattern that has been developed from the moment you are born.

When you are born, the first stimulus will be taken and you don't need anther

في اللحظة اللي بتولد فيها وبتوخد فيها أول نفس هاد الـ stimulus
اللي أعطاها الخلية رقم واحد (بوضحها وخلص) ما بده stimulus
جديد ما بتغير الدورة هي لحالها مستمرة وكل مرة بتعمل
activation للعضلة

carotid → بيضخي الدماغ

Carotid and aortic arteries have 2 bumps which contain receptors, the receptors there are from one type which is the pressoreceptors that they have to do with the stretch and with blood pressure. At the same time, we have another type of receptors that are known as chemoreceptors and they are sensetive to 3 gases → ① CO_2 (and they are very sensetive for it)

② O_2

③ H_2

High levels of CO_2 and low levels of O_2 and pH this will activate the aortic and ~~carotid~~ carotid chemoreceptors → sent the afferent sensory neurons to the Medulla oblongata so that they can activate the sympathetic pathway which will enhance the CO and activate it so to increase the ventilation

Expiration →

① Inspiratory muscles relax (diaphragm rises, rib cage descends due to recoil of costal cartilages).

② Thoracic cavity volume decreases

③ Elastic lungs recoil passively; intrapulmonary volume decreases.

يرجع ضغط الـspace لـ 0 - فيعمل ضغط الرئتين +1 فيطلع الهوا

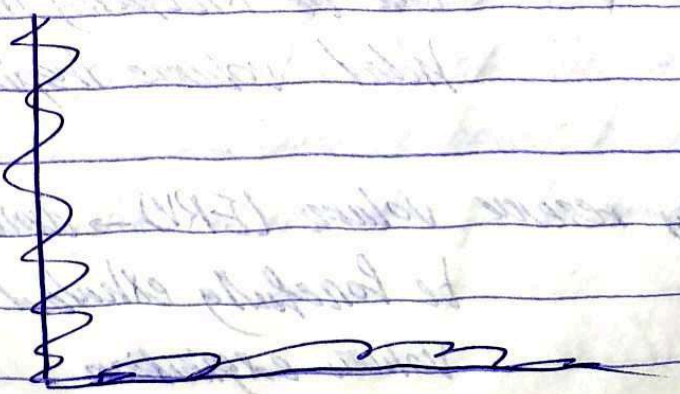
④ Intrapulmonary pressure rises (to +1 mm Hg)

⑤ Air (gases) flows out of lungs down its pressure gradient until intrapulmonary pressure is 0.

Ribs and sternum are depressed as external intercostals relax

Diaphragm moves superiorly as it relaxes.

Figure 18-9 →



At sea level, there's lots of O_2. At a P_{O_2} in the lungs of 100 mm Hg. Hb is 98% saturated

At high altitude, there's less O_2. At a P_{O_2} in the lungs of only 80 mm Hg. Hb is still 95% saturated.

In resting tissue, at a P_{O_2} of 40 mm Hg, Hb is 75% saturated - only 23% of O_2 carried by Hb is released

In metabolically active tissues (eg exercising muscle) the P_{O_2} is even lower. At a P_{O_2} of 20 mm Hg, Hb is only 40% saturated an additional 35% O_2 has been unloaded for tissue use.

Tidal volume → Amount of air inhaled or exhaled with each breath under resting conditions.

Inspiratory reserve volume (IRV) → amount of air that can be forcefully inhaled after a normal tidal volume inspiration

Expiratory reserve volume (ERV) → amount of air that can be forcefully exhaled after a normal tidal volume expiration.

Residual volume → (RV) amount of air remaining in the lungs after a forced expiration

Respiratory capacities → Total lung capacity (TLC) = TV + IRV + ERV + RV

→ Vital capacity (VC) = TV + IRV + ERV

→ Inspiratory capacity (IC) = TV + IRV

→ Functional residual capacity (FRC) = ERV + RV

لو ما بنعمل reclaim وكله بنفقد كميات كبيرة من الماء في حياتنا يومياً بنفقد ١.١ من الماء فقط في التنفس، الا ما برجع جزء منه نفقد ٢ L.

heat → Metabolism اجت من الـ

Normally → Respiration starts from the pulmonary system provide O_2 (incoming air with O_2) then we take it to the arterial blood and distributed to the rest of the body and then we go back again with CO_2 that produce because of the cellular respiration → take it back to the heart and give it to the pulmonary system

pulmonary ventilation →
Its the mechanical process of respiration, for this we allowing air to get in by inspiration and get out by expiration

Sometimes we do more than normal quite breathing as we start walking we will start breathing heavely, we will increase the breath time and increase the volume that we take in air and for this we are going to use other types of muscles.
The third one → scalenes (help to raise up the thoracic cavity and we use sternocleidomastoid. (4th muscle)

- we have 4 muscles that we use them for inspiration quite respiration → diaphragm + external intercostals for active respiration → scalenes + sternocleidomastoids.

For expiration, we don't use muscles (passive process) but for active expiration (coughing) → we will be using another set of muscles → 1- internal intercostals (they contract so they bring the thoracic cavity lower than normal and this will reduce the volume) and also we use abdominal muscles (they contract they cause also the respiration)

عشان ندفع الأعضاء الداخلية أكثر لفوق فنخلوا الهوا يطلع بقوة أكبر

4 muscles for inspiration and 2 for expiration.
- pleurae membrane → it's the membrane that are similar to the heart, each internal organ is surrounded by a double layer membrane).

Similar to the heart

↓

we have pericardium and between them the pericardiumal cavity, within the pericardiumal cavity there's a fluid to reduce the friction of the heart and to allow it to pump in a free friction environment.

Whenever we have a drop in O_2 we call it (below 80) → hypoxic
Whenever we have an increase in the CO_2 → hypercapnia.

- Normally, during rest or normal value we have it within our body CO_2 is about 40mmHg and partial pressure of O_2 will be about 98-100mmHg
- If you do hypoventilation → the partial pressure of O_2 will drop and the partial pressure of CO_2 will increase.

In this case you will feel your body is trying to get to increase O_2 and reduce CO_2, what does it do?

① Increase the respiratory rate ② Increase the value that I take it

* Normal breathing → I take tidal volume → 500ml breath in and 500ml breath out.

* Hypoxic, hypercapnia → increase the amounts مش بس Tidal volume بل أوقد شوي من IRV و أطلع شوي زيادة من ERV هاي العملية هي الزيادة

- زيادة معدل التنفس respiratory rate وزيادة volume that I take يسمى Hyperventilation.

- لما يصير Hypoventilation بدي أعمل Hyperventilation

- Who tells me that I need to take in more gases (more O_2), I need to reduce the partial pressure of CO_2? Receptors مستقبلات كل شي بعمله حسب (sensation receptors → Integration → motor action) لـ

- What are the receptors that tell me that I in hypoxia, hypercapnia? chemoreceptors → تستشعر المواد الكيميائية (CO_2, O_2) our body is more sensitive to CO_2 than O_2

- pain receptors → blood pressure
- Feeling full → الشبع → stretch receptors
صار Stretch في ال stomach.

- عشانه هيك إذا وقفت عا طرف البركة وبدي أنط في المي في كتير ناس بعمل hyper ventilation (بوخدوا كمية O_2 بزيادة) ← we increase the P_{O_2} to the blood and reduce to CO_2
- لما أنزل في المي بصير في إستهلاك أكبر لكمية O_2 وبصير زائد CO_2 رح يبدأ إنتاجه بشكل بطيء، عشانه هيك بوصل لمرحلة O_2 بصير 90 - 85, 80 ولسا CO_2 كمان 18 ← 20 ← 25 ← 30 ← 35 ← 40
بوصل 40 وهو الـ CO_2 بوصل 70 - 75
- مجرد ما نزل تحت 80 بندخل في coma (~~غ~~غيبوبة) إنقص O_2 وهو تحت المي فبموت.

- مالحة الدماغ بتحكي بدي O_2 لإنه ما كان حاسس إنه نسبة CO_2 طلعت و O_2 قلت، فلما حس إنه CO_2 زاد وصار 40 أنا بكون داخل في غيبوبة، لإنه O_2 خلص وأنا مش حاسس.

- our receptors are very sensitive for CO_2 more than O_2
السبب هيك لإنه CO_2 ممكن يلعب في pH، فهو جزء من Buffer system ففي CO_2 بعمل toxicity قاتلة.

- Normally, we have a ratio we have to follow, the ratio we said the perfusion ventilation ratio, they have to be equal, perfusion it means the flow of blood into the blood vessel into the lungs. This flow is how much

blood goes and enter the lungs so it can do gas exchange, لازم يتقابل معها enough ventilation.
↓
منعش الدم circulation سريع وماشي كويس لازم يكون معه تنفس سريع عشان الدم الي بطلع من الرئتين يكون well oxygenated
يعني أي حد بكون increase in heart rate ← بكون معه تنفس سريع.

- في الوقت الي بزيد فيه ال heart rate بصير زيادة في respiratory rate. ليش ؟!

we have to make perfusion + ventilation
أي mismatch تكون في مشكلة، مثلاً واحد بدخن وصار في خلل أو عنده infection خربت المنطقة بلا تشتغل ال alveoli أو مثلاً واحد معه ربو ما بدخل O_2 منيح ← بكون كمية الدم الي واصلة لجزء من ال alveoli غير مفيدة ومباشرة الجسم بحول القناة بعمل vasoconstriction to match ← مع الي جنبها عشان هاي blood vessel الي رايح للمنطقة السيئة بقلل من الدم الي رايحلها عشان to match perfusion ventilation rate متساويات.

واحد غرقه في البحر من إيش بموت ؟! الي ما بتفوت ع الرئة أبداً ؟ الي بصير إنه لما المي تدخل وتوصل الى مباشرة ال epiglottis بسكر فبقطع التنفس ومع استمرار دخول المي التنفس بضل مقطوع فبصير إختناق (suffocation) ولما يموت بفتح ال epiglottis ~~وهو بالأصل~~ وبدخل المي ع الرئتين بس هو بالأصل مات من الإختناق ولما نشغل بطلع المي

ده المعدة مش الرئيسية .

Increase the CO_2 cause an increase in the blood pressure.
pulmonary edema → ما بقيش فيه التهوية → hypertension
pressure.

هو لما الواحد يزيد ضغط الدم عنده بقى عندة تنفخ برجله ده بطلع fluid والجسم ما بيقدرش يرجعها (ضغط الدم عالي الجسم مش قادر يرجعه) والسوائل اللي بتطلع جزء منها برجعها الـ Lymphatic system بحدود 3L

ولكنه هو ده ما بقدر يرجعهم كلهم فبتراكم جزء من السائل وبقى جزء من الرئيسية فيه مي ومش راضي يشتغل

- التضخم يؤدي إلى رفع ضغط الدم .

Transport of O_2 →
O_2 is being transported via hemoglobin not anything else why?!
Because O_2 has a very low solubility coefficient to dissolve in ~~the plasma within~~ water, this means that the max that can't dissolve in the plasma within the blood is about 2% or even less, the rest in order to enter into the RBC where the hemoglobin is present.

- Hemaglobin its a protein that is called a conjugated protein (primary

رابع
Hemoglobin → is a quaternary structure of protein that is connected to a non protein group or groups).

prosthetic group ← قطعة أو قطع صغيرة تسمى (صناعي)
مجموعة المركز تبعها حديد هي الي بترتبط مع O_2 وهي لحالها ما بتشتغل والـ globin لحاله بيشتغل.

- Hemoglobin → عنده active sites for O_2 that does not allow CO_2 to bind to it, but CO compete with O_2 on it, toxicity ← عشان هيك
تعتبر الـ CO كثير خطيرة وعالية لأنه بترتبط مثل الـO_2،
وهو منافس قوي جداً. لو في CO هو الي برتبط مش الـO_2 والمشكلة ما في sensation لـ CO برتبط محل O_2 لـ CO ما الـ receptor وما في إله sensation).

Heme → iron → is an additional group that is non protein group.
its core iron (Heme group → prosthetic group)

- بخار الماء بقلل من O_2 اللي داخل مش برتبط محله، عشان هيك الناس اللي قريبين من البحر بحسوا بإختناق، لأنه كمية الـO_2 هناك قليلة مش إنه إرتبط محل O_2، الأعراض:- بتشوب، نفسه ثقيل.

- الإنسان اللي بكون متوتر جداً بعطوه كيس مشان ينظم التنفس ويصحصح الدماغ ويصير يتنفس من CO_2 لأنه إصلا بحس CO_2

أكثر من O_2، والدماغ يرجع بنظم لدخله ← اللي يكونه متوتر يكون عنده Shalow ventilation ما بياخد Tidal volume بياخد أقل وأنا بدي إياه ياخد كمية O_2 أعلى فبرفعله الـ CO_2 بعديه بصير يوخد نفس عميق.

اصابات نقص حديد ① ← فقر الدم ← Anemic
عدم القدرة على أخذ والحصول على كميات كافية من O_2 ②
والسبب ← ① ما عندي حديد يعمل هيموغلوبين
② ما عنده بروتين يعمل هيموغلوبين

- عدد RBC طبيعي ولكنه عنده أنيميا، RBC مليونه جزيء من الهيموغلوبين ولكنه ما عندها إياه كافي لما في بروتين او حديد او فيتامينات تساعد إنه يرتبط.

في ناس عدد RBC أقل وكمان هادا إنيميا.
لـ الهرمون اللي ~~ترجع~~ بتشجع الـ bone marrow يعمل RBC صح موجود.
- RBC ← erythrocytes
- Hormone ~~Hor~~ ← erythropoietin ← بطلع من الكلى ← لأنه الكلى هي أكثر منطقة فيها مستقبلات لـ RBC ٩٥٪ من الدم عبر من الكلية ففي مستقبلات بتحسب إنه ما عندها RBC كافية وبتكون عندها الخلايا اللي بتفرز الهرمون وبروح مع الدم وبتوصل Bone marrow فبنبح خلايا دم حمراء

Hemoglobin → It has a unique property → you will find it
that it has affinity to O_2 to bind to O_2
but this affinity it has kind of v

whenever the pO_2 is high → increases from capturing O_2, like in alveoli the conc. for pO_2 is high this will increase the saturation of hemoglobin with O_2, but it loses its affinity as soon as the pO_2 that surrounding is low.

بيرخوا O_2 ويطلعوا CO_2 ← resting cell ← عند الخلايا)
عشان هيك بينزل من 98 أو 100 لـ 75 عشان يفلت O_2 هناك
ديرجع يوصل لـ alveoli التركيز العالي فبيمسك O_2 مع الحديد و
بتفضى كثير عند الخلايا .

- What happenes if I'm doing activity (low conc. عند)
يفقد O_2 أكثر لانه الخلايا بدها إياه فيصير shifting وأكثر .
It loses its affinity along with the activity

لو زاد CO_2 في الدم ، هل بتزيد الـ affinity لـ hemoglobin بتظل
ماسك O_2 ولا بفلته ؟ .
لو زادت الحرارة مش بس infection ، بفقده ولا بظل ماسك O_2 ؟
لو الـ pH صار more acidic ؟!
زاد تركيز DPG 2,3-Biphosphoglyceric acid ؟!

- او زاد الـ Metabolism ؟!
- لما بزيد الـ activity عندي بزيد CO_2 ، وبصير الدم more acidic لأنه
CO_2 بذوب ، وحلقة كريبس بتزيد وبطلع DPG أكثر وحرارة الجسم
بتزيد لانه بعمل طاقة ATP + heat وبالتالي راح يفقد O_2 وتقل affinity
تبعت الـ hemoglobin .

CO_2 → solubility coefficient of CO_2 higher than O_2

عشان هيك بذوب 7% منه أقل من ما له والباقي رح يدخل BC و برتبط مع ال Hb و 23% راح يتم نقل في Hb وعند active sites.

مشان HCO_3^- → بطلع في البلازما → Buffer system

يحافظ على pH ثابتة ← 7.4

CFTR → ممكن تسبب الموت

تكون ↳ cystic fibrosis → defective channel

وتبطل تشتغل وتسبب Thick mucus في ال Lungs وتسبب إختناق

وتكون موجودة في الجهاز الهضمي وبتخلي الواحد ما يقدر يهضم الأكل

مع Thick mucus

Cl^- بتجمع حواليه مي والمكان الي لازم يكون فيه مي خارج الخلية بصير

ينشف شوي لأنه بصير فيه Mucus وبياخذ المي من ال Mucus وبصير Thickness.

Cl^- لبرا في ال plasma بصير بسحب مي من ال alveoli و alveolis بتسحب

المي من trachea و bronchioles وبصير هنالك Mucus إلي بحافظ على

رطوبتهم بتصير very thick ← وبسبب إختناق عند الإنسان (مرض

وراثي)!

What is the factors that affect the O_2 content in the blood?!

① O_2 content in arterial blood → أخذ الدم على الرئتين راح

O_2 وبلف فيه بالجسم بحديه مركه و بحديه رجع رجع ياخذ O_2 جديد من الرئتين

فإحنا بنسحب قديش موجود في الشرايين الي في الجسم ← بوصل content 98

إيش إللي بسبب إنه ينزل عنه P98

① عدد الخلايا الدم الحمراء بقل

② هيموغلوبين عدده قليل (Shortenage) المشكلة RBC عندهم

مش بس المشكلة في الـ hemoglobin)

ايش في خطأ في الـ hemoglobin ←

① في Shortenage في الحديد ↓

② نقص في البروتين اللي بتحمله

③ الفيتامينات اللي بتساعد الانزيمات في تصنيع الـ Hemoglobin

We need to regulate our O_2 content

Regulation of O_2 content it means that you have a respiratory control ~~system~~ center that is present within your area where is controlling the vital organs to get enough O_2 all the time (Brain stem → pons, medulla oblongata, mid brain)

Respiratory control center consist of 2 centrals (nucleus)

nucleus in the nerves cells (نفس الخلايا بعملوا نفس الوظيفة)

VRG → control the voluntary expiration أما الزفير الطبيعي ما بيستخدم Muscles

Any action your brain is going to do require input

all the time we do regulate according to the change in the environment

receptors وعشانه أمس بتاع

إذا كنا في مكان ونزل تركيز O_2 فيه معناها بدي مستقبلات تحكي إنه O_2 نزل في البيئة فبدي أزيد الinspiration والـ expiration ، بدل ما أخذ 0.5L بصير أخد 1L .

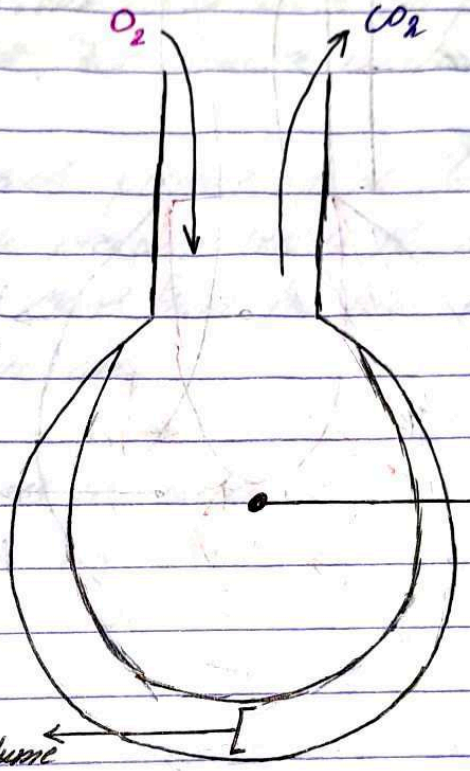
- نوع المستقبلات chemoreceptors موجودين في الـ carotid within الـ aorhi bodies والـ carotid bodies .

Central chemoreceptors → they sense the change of H^+ which is indication of CO_2

any small change in H^+ can cause death.

The Respiratory volumes ⇒

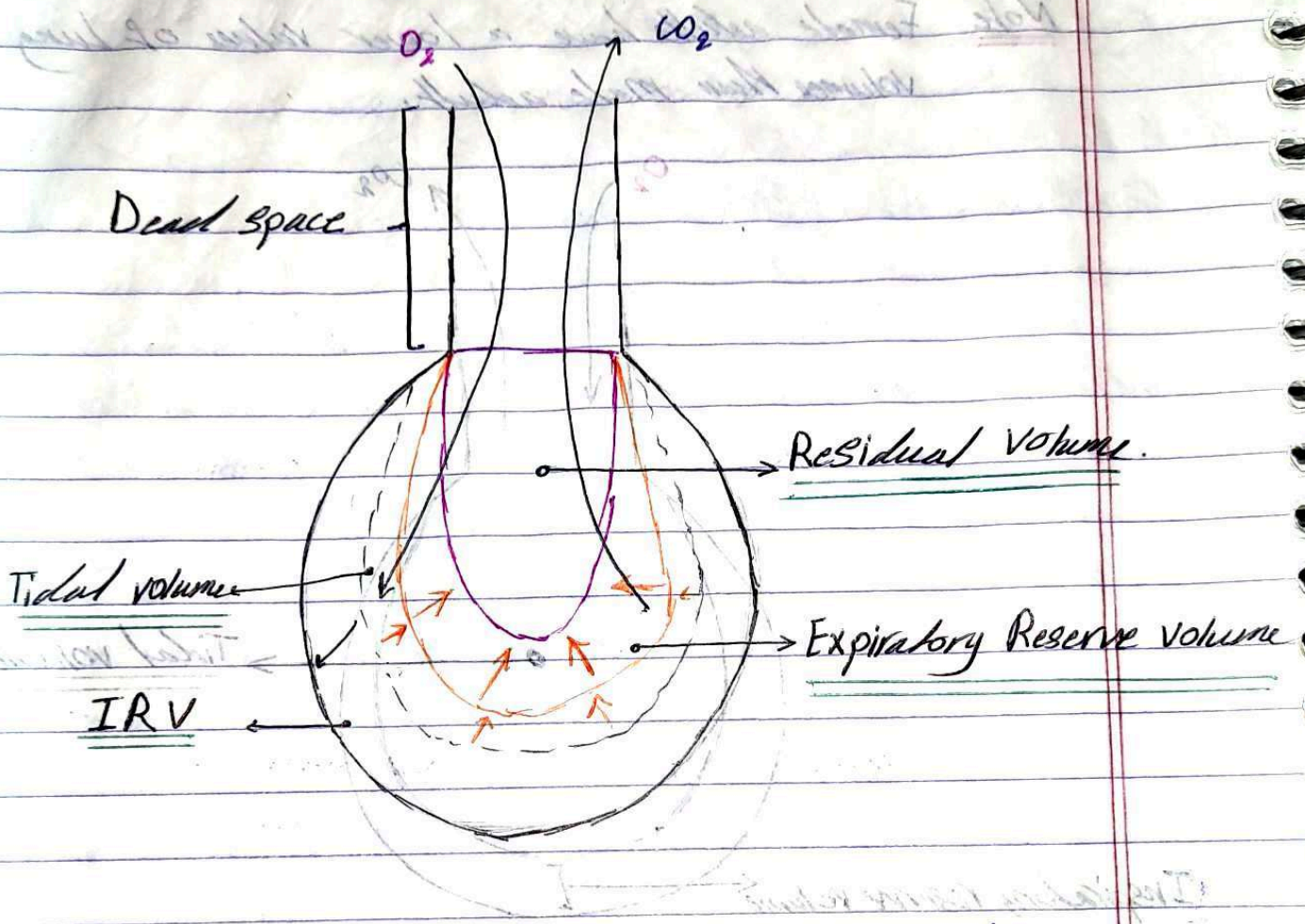
Note Female adults have a lower values of lung volumes than male adult.



Tidal volume ⇒ amount of air we breath in and out in our lungs normally (Normal breathing).

Inspiratory reserve volume ⇒ take a deep breathing in (deep inspiration) i.e their inspired with a maximal

inspiratory effort in excess of the tidal volume.



Expiratory Reserve volume → Exhale more than usual - amount of air we can exhale addition to the TV

Residual volume → At the end of the day, there's always an amount of air or volume remaining in our lungs after a maximal expert of ~~Ef-fort~~ effort