

## Respiratory system

What is the function for the respiratory system?!

- ① Gas exchange
- ② Vocalization and Resonance
- ③ Smelling
- ④ Maintenance for pH
- ⑤ Maintain the temperature
- ⑥ Metabolism
- ⑦ Elimination of heat (+ ATP).
- ⑧ Olfaction

Respiratory system picture  $\Rightarrow$

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

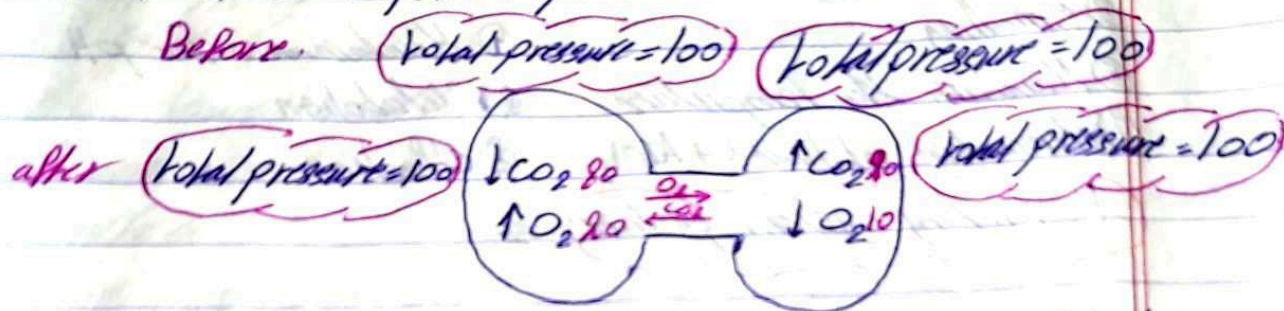
The smell and vocalisation happen in the upper respiratory tract, also the resonance occur on the upper respiratory tract which is when you close your nasal cavity, you will not be able to pronounce letters properly.

Resonance  $\rightarrow$  هو صدى الصوت الي يطلع الكلام سببها 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  $O_2$ , 715  $N_2$  --- etc

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



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

The functions of the nasal cavity  $\Rightarrow$

- ① Resonance
- ② Olfaction
- ③ Trapping for dust particles (mucous hair)  $\Rightarrow$  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 enters.

Why we need to do humidification?

because if the air that enters 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  $\Rightarrow$  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 air 760 pressure  
but when it enter we add a new gases  
the mixture of gases which is the water vapor  
but the total pressure stayed the same (760)  
that's mean that all the gases will be lowered  
in order to not cause a change 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 ... that's  
mean that's 10 mm mercury from the  $O_2$   
only where replaced by water vapor  $H_2O$ .

The air enters 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 enters and 0.5L  
goes out

From the 0.5L there's 150 ml stays in the Nasal  
cavity, Trachea <sup>and</sup> 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 smokes, there's a material called tar (القطران) enter your lungs and accumulate on the alveoli (المادة التي تتجمع فيها في الشوابع الرئوية) so the alveoli that has to make 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 a lot of tar on it (كانت عملها سيئاً جداً) so it will not do its job

الجسم يقترب جداً بعد 5 ساعات بعد يومه غير نراهم  
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 pushing 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 an  
external effects it  
stopped.

هنا الهواء، إلى راح ناحية الـ alveoli إلى اعتبرناها physiological dead space  
من راح يسهل له gas exchange فبعض مجرى الفاز  
إلى ما يستندنا عنه 200ml بدل 150ml.

Nasal cavity →

Trachea do the vocalisation while Nasal cavity  
do the resonance

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)  $\Rightarrow$  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 Pharynx).

Trachea is the vocal box, it has a vocal cords

The pharynx is 3 parts  $\rightarrow$  Naso pharynx  
Oral pharynx  
Laryngo pharynx

Naso pharynx  $\Rightarrow$  nose

oral pharynx  $\Rightarrow$  mouth

Laryngeal  $\Rightarrow$  goes to the Esophagus or Larynx

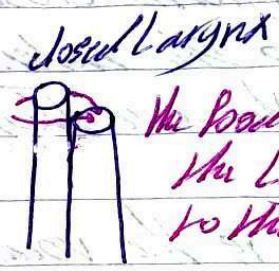
That's why we call the pharynx a common pathway.

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~~ nasopharynx.

ليس لما نبلع تفاعلة أدم بطلع ويستدل! لأنه لما نبلع يرتفع Larynx ويسكر ما عنانه الأكل يعني من حولها لما يعني من فوقها ربي على الـ esophagus



Before swallow



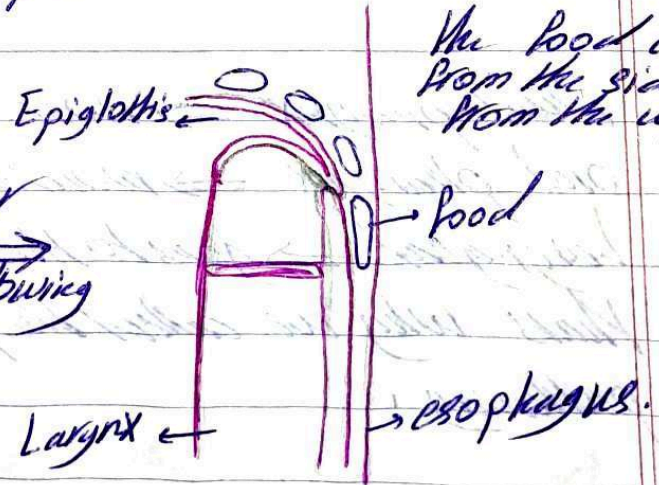
after swallow

The food come from around the Larynx and goes to the esophagus.

Swallowing action picture



after  
swallowing



The food enter from the sides not from the up.



تفاهة 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 anyway 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 trachea 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 expansion 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 quiet breathing 500ml of air and the length of trachea, nasal cavity, bronchi and bronchioles are about 150ml so we will have a residue from 350ml.

150ml the last that get in and first one goes out, that's why it's an anatomic dead space.

### Alveolar Structure →

Surface tension → ~~the~~ alveoli they are so too tough to expand, it means they have a high surface tension.   
 (إبراهيم هادي محمد جواد صبيح تقديس)  
because of that, we need very high energy to make it expand and take the air. 3) The type 2 synthesis the surfactant that reduces the surface tension.   
 (طبقة عمود ما يتجلى)  
الخلايا تنقل 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 ( $P_{58}$ ) while in the sucker is  $P_{70}$  so the air will enter from the sucker to the oral cavity making the fluid enter the sucker.

### (h) Exchange surface of alveoli →

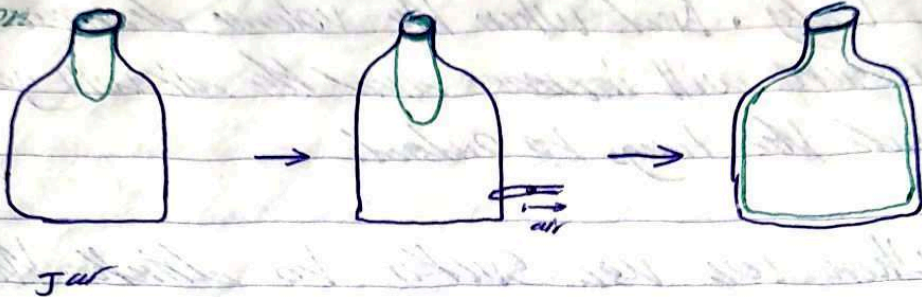
In order for the gas to go outside the alveoli and enter the capillary, it have to ⇒

① First dissolve in surfactant → very thin layer so it didn't increase the space ( $75\text{m}$ ).

لما يفوت هوا ناستف بونف من رطوبة ال surfactant فطوال ال type II بتفرز مكانه - اعطاءه هيك بنحس انه من قانريه نوخذ نفس .  
ممكن عند بعض الناس ما يكون في surfactant فرج يكون في صعوبة بالتنفس لانه بالامس من هو سهل التنفس.

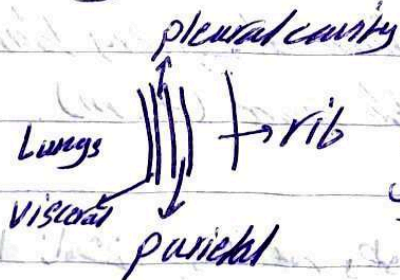
pleurae →

Balloon



If we have a balloon 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~~ lungs from outside.



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

من جوار

so the pressure in the pleural cavity is negative (-3mm Hg)   
 يعني يكون أقل من الضغط الجوي ونقل الأغشية معا

## Inspiration $\Rightarrow$

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  $\Rightarrow$

\* Ribs elevated and sternum flares as external intercostals contract.

\* Diaphragm moves inferiorly during contraction.

بذل 0,5L عن طريقه تقلل الكتل وبتحريك 0,5L عن طريقه زيادة الكتل

Inspiration means that I activate 2 types of the muscles: - ① Diaphragm ② external intercostals

Once we activate them that means I increased the volume of the lungs  $\rightarrow$  the pressure is dropped  $\rightarrow$  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 go up again and the external intercostals will bring down the ribs and that means we are reducing the volume and increasing the pressure so the air will go out.

So we say it's an active inspiration and passive expiration, why?!

Inspiration needs an energy while the expiration we don't need an energy.

Why we die if we breathe CO?!

Not because there's no O<sub>2</sub> but the CO will bind to the Hb instead of O<sub>2</sub> making no O<sub>2</sub> reach the cells.

Venous Blood → Discussion of the picture.

The CO<sub>2</sub> is transported in 3 means →

① Dissolved CO<sub>2</sub> ⇒ the most important and similar to O<sub>2</sub> but its percentage is 7% not like the O<sub>2</sub> which was 2%.

② About 23% of the CO<sub>2</sub> is transported inside the red

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

③ We know 70% of  $CO_2$  is transported as bicarbonate  $\Rightarrow$  How this happen?!

①  $CO_2$  reacts with  $H_2O$  produces the carbonic acid

② Carbonic acid is a very weak acid 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 ~~with~~  $H_2O$  inside the red blood cell?!

due to the presence of the enzyme CA inside the

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



In order to continue producing the  $\text{HCO}_3^-$  and  $\text{H}^+$   
we have to decrease the conc of the  $\text{H}^+$  and  $\text{HCO}_3^-$   
so if it accumulate on the cell the direction  
of the equilibrium will go toward  $\text{CO}_2$  and  $\text{H}_2\text{O}$   
so they will accumulate ~~at the cell and  $\text{H}^+$~~   
~~with  $\text{HCO}_3^-$  and  $\text{H}^+$  will~~

⑤ then the red blood cell will move until  
it reaches the alveoli and due to the low  
conc of  $\text{CO}_2$  in it the dissolved  $\text{CO}_2$  will  
enter and due to the decrease of conc of  
dissolved  $\text{CO}_2$  the  $\text{CO}_2$  inside the cell will unbind  
from the Hb and goes out the cell. ~~and the~~

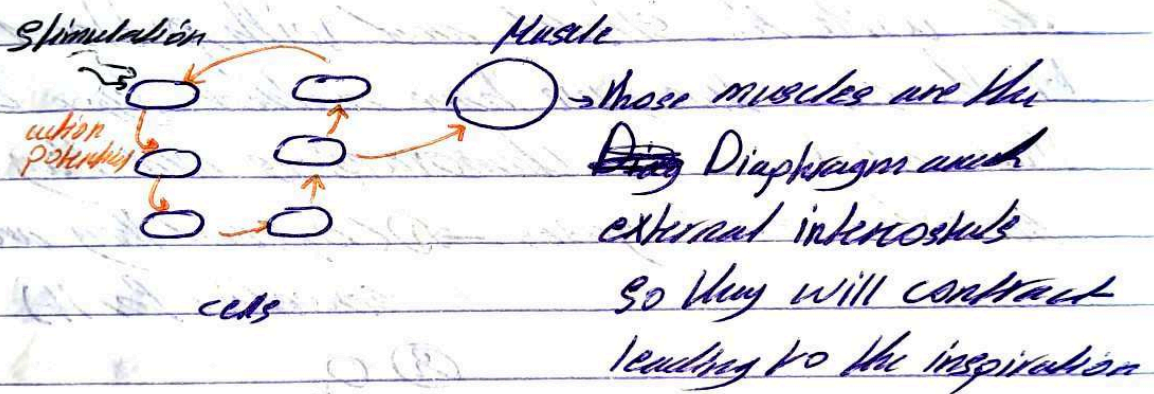
⑥ the  $\text{HCO}_3^-$  will enter from the plasma to the  
cell and the  $\text{Cl}^-$  will goes out and the  $\text{H}^+$  will  
unbind from the Hb and react with the  $\text{HCO}_3^-$   
giving  $\text{H}_2\text{CO}_3$  that will ~~at~~ dissociate into the  
 $\text{CO}_2$  and  $\text{H}_2\text{O}$  in the presence of  $\text{CO}_2$  and the  $\text{CO}_2$   
will go out.



What the significance of the bicarbonate presents in the blood?!

Buffer  $\Rightarrow$  its one of the strongest buffer system that we have in blood  $\Rightarrow$  in order to ~~maintain~~ maintain the pH. Whenever the pH increases or decreases, the bicarbonate work on it.

### Figure 18-16 Dissection $\rightarrow$



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

Central pattern generator  $\rightarrow$  is a <sup>2a</sup> 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 another

في اللفظة الي بتتراد فيها وبتوجد فيها اول نفس هاد ال Stimulus  
الي اعطاهم الخلية رقم واحد ز بتوجد ما وصله ما بدو Stimulus  
جديد لانه بتغير الدورة هي لحاله مستمر وكل مرة بتعمل  
activation للخلية

Carotid side → يعني الدماغ

Carotid and aortic arteries have 2 bumps which contain receptors, the receptors there are from one type which is the pressure receptors 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 sensitive to 3 gases → ①  $CO_2$  (and they are very sensitive for it)

②  $O_2$

③  $H^+$

High levels of  $CO_2$  and low levels of  $O_2$  and pH this will activate the aortic and carotid chemoreceptors → send the afferent sensory neurons to the Medulla oblongata so that they can activate the sympathetic pathway which will enhance the GFR 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 الفراغ إلى space لـ 3 - فيجعل ضغط الرئتين + فيطغى الهواء

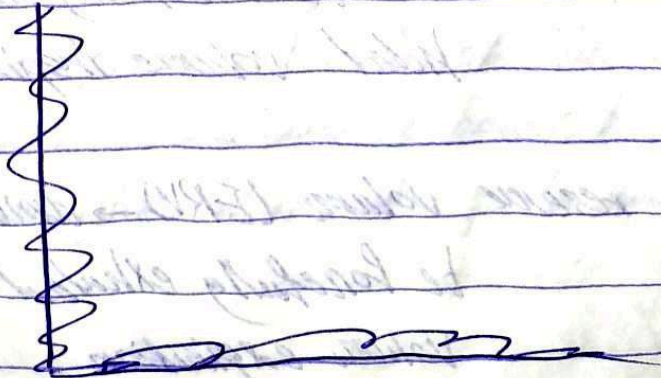
④ Intrapulmonary pressure rises (to +1 mm Hg)

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 mmHg. Hb is 98% saturated

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

In resting tissue, at a  $p_{O_2}$  of 40 mmHg, Hb is 75% saturated - only 23% of  $O_2$  is carried by Hb is released

In metabolically active tissues like exercising muscle the  $p_{O_2}$  is even lower. At a  $p_{O_2}$  of 20 mmHg, Hb is only 40% saturated an additional 35%  $O_2$  has been unloaded for tissue use.

Tidal volume  $\rightarrow$  Amount of air inhaled or exhaled with each breath under resting conditions.

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

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

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

Respiratory capacities  $\rightarrow$  Total lung capacity (TLC) = TV + IRV + ERV + RV

$\rightarrow$  vital capacity (VC) = TV + IRV + ERV

$\rightarrow$  Inspiratory capacity (IC) = TV + IRV

$\rightarrow$  functional residual capacity (FRC) = ERV + RV

لو ما بنعمل reclamation، كانه بيقدر كليات كبيرة من الماء، في حياتنا يومياً بنقدر 1.6 من الماء فقط في التنفس، لو ما برهو جزء ممكن تقدر 2.

heart  $\rightarrow$  Metabolism

Normally  $\rightarrow$  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  $\rightarrow$  take it back to the heart and give it to the pulmonary system

pulmonary ventilation →

It's the mechanical process of respiration, for this we allow 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 heavier, 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 main one → scapulae (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 → scapulae + sternocleidomastoids.

For expiration, we don't use muscles (passive process) but for active expiration (coughing) → we will be using another set of muscles → 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)

عسانه تنفج الاعضاء الداخليه اكثر لفرقة فنتلوا الهوا بطع بقوة المر

- 4 muscles for inspiration and 2 for expiration.
- pleural membrane → its 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 pericardial cavity, within the pericardial 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  $CO_2$  → hypercapnic.

- Normally, during rest or normal value we have it within our body  $CO_2$  is about 40 mmHg and partial pressure of  $O_2$  will be about 98-100 mmHg.
- 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 think 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 volume that I take in

• Normal breathing  $\rightarrow$  I take tidal volume  $\rightarrow$  500ml breath in and 500ml breath out.

• Hypoxic, hypercapnia  $\rightarrow$  increase the amount of IRV  
من قبل كل نفس من tidal volume  
أطلع مستوى زيادة من ERV  
هنا العملية هي الزيادة

زيادة معدل التنفس respiratory rate وزيادة  
Volume that I take  
Hyperventilation

زيادة معدل التنفس Hypoventilation  
يؤدي إلى

- 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  
كل شيء بعد ذلك مستقبلاً  
(sensation receptors  $\rightarrow$  Integration  $\rightarrow$  motor action)

- What are the receptors that tell me that I'm hypoxic, hypercapnic? Chemoreceptors  $\rightarrow$  مستقبلات المواد الكيميائية  
our body is more sensitive to  $CO_2$  than ( $CO_2, O_2$ )  
 $O_2$



- *parareceptors* → blood pressure  
- *feeding full* → الشبع → *stretch receptors*  
صاح *stretch* في ال *Stomach*.

- عشانه هيك اذا رقتك على طرف البركة وبسي أنظ في المي في كثير ناس  
يجلو *hyperventilation* ( بوضوا كمية  $O_2$  بزيادة ) → *we increase*  
*the  $p_{O_2}$  to 120 and reduce to 80*

- لما اتزل في المي بغير في إس هلال أبحر لثمنية  $O_2$  وتسرع وال  
 $CO_2$  مع يتم إنتاجه بشكل بطيء، عشانه هيك بوصول لمرحلة  $O_2$  بغير  
90 - 85, 80 ولسا  $CO_2$  كانه 18 ← 20 ← 25 ← 30 ← 35 ← 40  
بوصول 40 وهو ال  $O_2$  بوصول 75 - 70

- مجرد ما تزل تحت 80 بتدخل في *coma* (عصبية غيبوبة) انخفاض  $O_2$   
وهو تحت المي فقوت .

- مالحة الدماغ يعني بدي  $O_2$  لأنه ما كانه حاسس إنه نسبة  $CO_2$  طلعت  
و  $O_2$  قلت ، فلما حس إنه  $CO_2$  زاد وصار 40 أنا يكونه داخل في غيبوبة  
لأنه  $O_2$  خلص وأنا حاسس حاسس .

- *our receptors are very sensitive for  $CO_2$  more than  $O_2$*   
انقلت هيك لأنه  $CO_2$  ممكن يلعب في *pH* ، فهو جزء من *buffer system*  
ممكنه  $CO_2$  جعل *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, enough ventilation, <sup>معها</sup> كجزء تقابل

↓  
تفعلش الدم circulation سريع وما سيني كويس لازم يكون معه تنفس سريع عشان الدم ابي بطلع منه الرئتين <sup>يكون well oxygenated</sup> يعني ابي حد يكون <sup>increase in heart rate</sup> - يكون معه تنفس سريع

- في الوقت ابي بزيدي فيه ال <sup>heart rate</sup> بغير زيادة في respiratory rate

we have to make perfusion + ventilation أي mismatch تكون في مشكلة، مثلاً واحد يدخل و صار في انفك او عن infection فزيد المنطقه بلك تستقل <sup>alveoli</sup> او مثلاً واحد معه رينو ما يدخل <sup>O<sub>2</sub></sup> فيج - بتكون كمية الدم ابي واصله لجزءه ال <sup>alveoli</sup> غير مفيدة ومباشرة الجسم بحول القنوات بعمل vasoconstriction - <sup>to match</sup> مع ال جنبها عشان هيل <sup>blood vessel</sup> ال رايح للمنطقه اللي بتقال <sup>perfusion</sup> ال رايحها عشان <sup>to match</sup> ال رايح للمنطقه اللي بتقال <sup>ventilation</sup> متاويات <sup>rate</sup>

واحد عنقه في البرصه ايش بمره <sup>IR</sup> ابي ما بتقوت على الرئه ابدأ ابي بغير انه لما ابي تدخل وتوصل الدم مباشرة ال epiglottis بتر فيقطع التنفس ومع استمرار دخول ابي التنفس بخل مقفل وفيه لانتفاخ <sup>Suffocation</sup> ولما يموت بفتح ال epiglottis وهو <sup>الاصلا</sup> يدخل ابي على الرئتين من هو بالاصل ما من ال اختناق ولما بفتح ابي بطلع ابي

في المعدة مثل الرئتين  
Increase the  $CO_2$  cause an increase in the blood pressure.  
pulmonary edema → ما يقرب من التدمية → hyper blood pressure.

عندما الوراثة تزيد ضغط الدم عنده ليس عنده تنفخ برجله من بطلع fluid  
والجسم ما يقدر يرجعها لضغط الدم عالي الجسم مش قادر يرجعها  
والسوائل الي بطلع جزء منها يرجعها ال lymphatic system بحدود 3L

ولكنه هو ما يقدر يرجعها كلها فبتراكم جزء من السائل ويبقى جزء  
من الرئتين فيه مي ومن راضي يستقل

- التنضيم يؤتى الي ارتفاع 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~~ 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.

- Hemoglobin its a protein that is called a conjugated protein (priming) كيناف البروشيا

Hemoglobin <sup>أربع</sup> → is a quaternary structure of protein that is connected to a non protein group or groups).

قطعة أو قطع صغيرة تسمى (صناعي) prosthetic group -  
مجموعة المركز بقومها حديد هي التي ترتبط مع  $O_2$  وهي  
لحاملها ما يشتمل ولا ال globin لحاله مستقل.

- Hemoglobin → active sites for  $O_2$  that does not allow  $CO_2$  to bind to it, but  $CO$  compete with  $O_2$  on it, ~~و~~ toxicity

تعدت ال  $CO$  كثير خطيرة وعالية لأنه يرتبط عند ال  $O_2$  ،  
وهو منافس قوي جداً . لو في  $CO$  هو التي يرتبط مع ال  $O_2$  والمشكلة  
ما في separation ال  $CO$  يرتبط مع ال  $O_2$  (  $CO$  ما ال 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 يقلل من  $O_2$  التي داخل من يرتبط حله وعشانه  
هناك الناس التي قريبيه من البحر يحسوا بإضطرابه لأنه كمية  
ال  $O_2$  هناك قليلة من انه ارتبط حله  $O_2$  ، الأعراض! - بشوابة نفسه  
تقلد .

- الإشبانه يكونه متوتر جداً يكونه كحس مشانه ينظم التنفس  
ويصلح الدماغ ويصل التنفس من  $CO_2$  ، لأنه أيضاً ينفس  $CO_2$

أكثر  $O_2$  هو السامح يرجع بنظم لتدخله التي تكونه متوتر يكونه عنه  
Total ventilation ما يياض  $Tidal volume$  يياض أقل وأنا بدي إياه  
يافد كمية  $O_2$  أعلى فترفعه الـ  $CO_2$  بعديه بغير يوفد نفس عمقه .

أحيانا نقص حديد ① → فقر الدم → Anemic .  
عدم القدرة على أخذ والوصول على كمية كافية من  $O_2$  ②  
والسبب ① ما عندي حديد يعمل هيموغلوسين .  
② ما عنصروسته يعمل هيموغلوسين .

- عدد RBC طبيعي ولكنه عنده أنيميا ، RBC مليونه جزئي منه الهيموغلوبين  
ولكنه ما عندها إياه كافي (ما في بروتينه أو حديد أو فيتامينات) تبعد إياه  
يرتبط .

في ناس عدد RBC أقل وكمان هادا أنيميا .

له الهرمونه التي بترجع الـ  $bone marrow$  يعمل RBC صرع  
صوجود .

- RBC = erythrocytes

- ~~Hormone~~ erythropoietin = هرمون (الكلية) ، لأنه الكلية

هي أكثر منطقة فيها مستقبلات الـ RBC ، لأنه 90% منه الدم عبره  
الكلية ففي مستقبلات بتخبر إنه ما عندها RBC كافية ويكونه عندها  
الخلايا التي بتفرز الهرمونه وبروح مع الدم ويتوصل الـ  $Bone marrow$  فينتج  
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  $\rightarrow$  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.

يؤخذ  $O_2$  ويطرح  $CO_2$   $\rightarrow$  resting cell  $\rightarrow$  عند الخلايا  
 عنده هيك بينزل منه 98 او 100 لـ 75 عنده يطلع  $O_2$  فالك  
 ذيرجع بوصول لـ alveoli التركيز العالي فيبمسك  $O_2$  مع الحديد و  
 يتفلى عااد عند الخلايا.

- What happens if I'm doing activity (low conc.)  
 يفقد  $O_2$  شوي لانه الخلايا بيها ايك فينر Skirting وانحر  
 It loses its affinity along with the activity

لو زاد  $CO_2$  في الدم اهل بتزيد ال affinity, hemoglobin يفقد  
 ما سلك  $O_2$  ولا يفلتة!

لو زاد الحرارة عنده infection, يفقد ولا يفيل مكان  $O_2$ !  
 لـ pH صار more acidic!

زاد تركيز  $Dp$  ال 2,3-Biphosphoglyceric acid!

او زاد ال Metabolism!

- لما يزيد ال activity عندي بتزيد  $CO_2$  وبيد ال more acidic لانه

$CO_2$  بنوب رصلة كحرس بتزيد وبتطلع  $Dp$  انحر وحرارة الجسم  
 بتزيد لانه بفعل طاقة - ATP + heat وبالتالي راع يفقد  $O_2$  ويفيل affinity  
 تبعت ال hemoglobin.

$\text{CO}_2$  → solubility coefficient of  $\text{CO}_2$  higher than  $\text{O}_2$   
عشانه هيل بنوب 7% من اقل من 1% والباقي ربح يدخل  
BC و يرتبط مع ال Hb و 83% راج يتم نقل في Hb و عند  
active sites

مشانه Buffer system → بطاع في البلازما →  $\text{HCO}_3^-$   
يحافظ على pH ثابتة ← 7.4

CFTR → ممكنه تسبب الموت

Cystic Fibrosis → defective channel  
وتبطل تشغل وتسبب *Mucous* في ال Lungs وتسبب إصتانه  
وشها موجودة في الجهاز الهضمي وتدخل الواحدهما يقدر يهضم الأكل  
مع *Mucous*

ال تبصع هو اليه في والمكانه الي لازم يكونه فيه في خارج الخليه بغير  
تتوي مشه لأنه بغير فيه *Mucus* و يياضد اليه من *Mucus* و بغير  
Thickness

- ال لبرا في ال plasma بغير يسحب في من الveoli و الveoli يتسحب  
الي من *Mucus* و *bronchiae* و بغير هناك *Mucus* الي يحافظ على  
رطوبتهم بغير *very thick* ← و بسبب إصتانه عند الانسان اعرض  
ورائها .

What are the factors that affect the  $\text{O}_2$  content  
in the blood?

راغ الدم على الرئيه أفرد →  $\text{O}_2$  content in arterial blood

$\text{O}_2$  وبلغ فيه بالجسم بغيره كركه و بغيره ربح يافد  $\text{O}_2$  جديد من الرئيه  
فأما بسبب قد يس موجود فيه التراسيه الي في الجسم ← بوصول *content* 98

إمراض التي يسبب إنه ينزل عن 98 P

- ① عدد الخلايا الدم الحمراء يقل
- ② هيموغلوبين عوده قليل (shortage) (ممكن RBC عندهم ضيق بسى المشكلة في ال hemoglobin)

أش في ظل في ال hemoglobin ←

- ① في shortage في الحديد
- ② نقل في البروتين التي يتعلمه
- ③ الفيتامينات التي تساعد الأثرية في تصنيع ال 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 centers  
(nucleus)

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

VRC → control the voluntary expiration (أما الزفير الطبيعي ما تستخدم Muscles)

Any action your brain is going to do require input



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

وعشانه أفس بجناح receptors

إذا كنا في مكانه ونزل تركيز  $O_2$  فيه معناها بدى مستقبلات تتكفي  
إنه  $O_2$  نزل في البنية فبدى أزيد الـ *inspiration* والـ *expiration* ،  
بد ما أخذ 0.5L بهر أقر 1L .

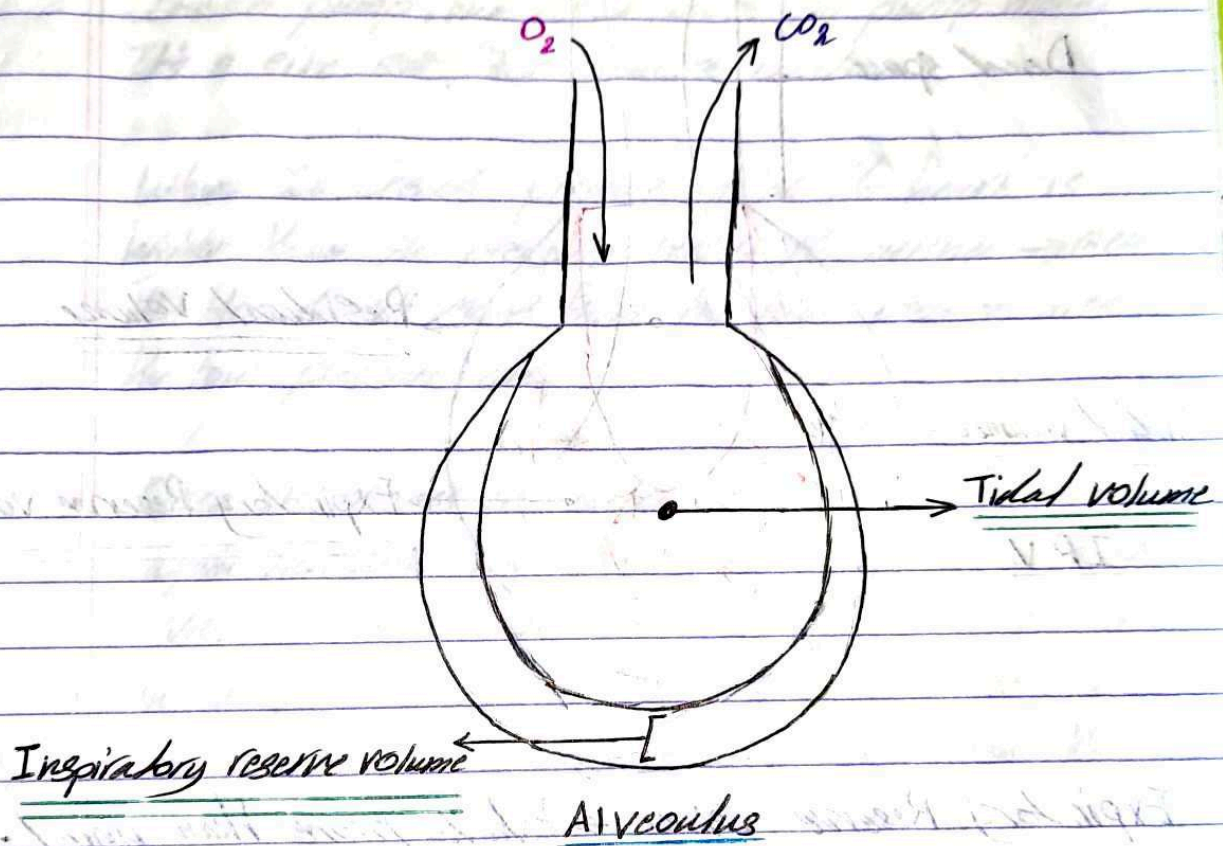
- نوع المستقبلات *chemoreceptors* موجودين في الـ *carotid arteries* في  
الـ *carotid bodies* والـ *aortic bodies* .

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 →

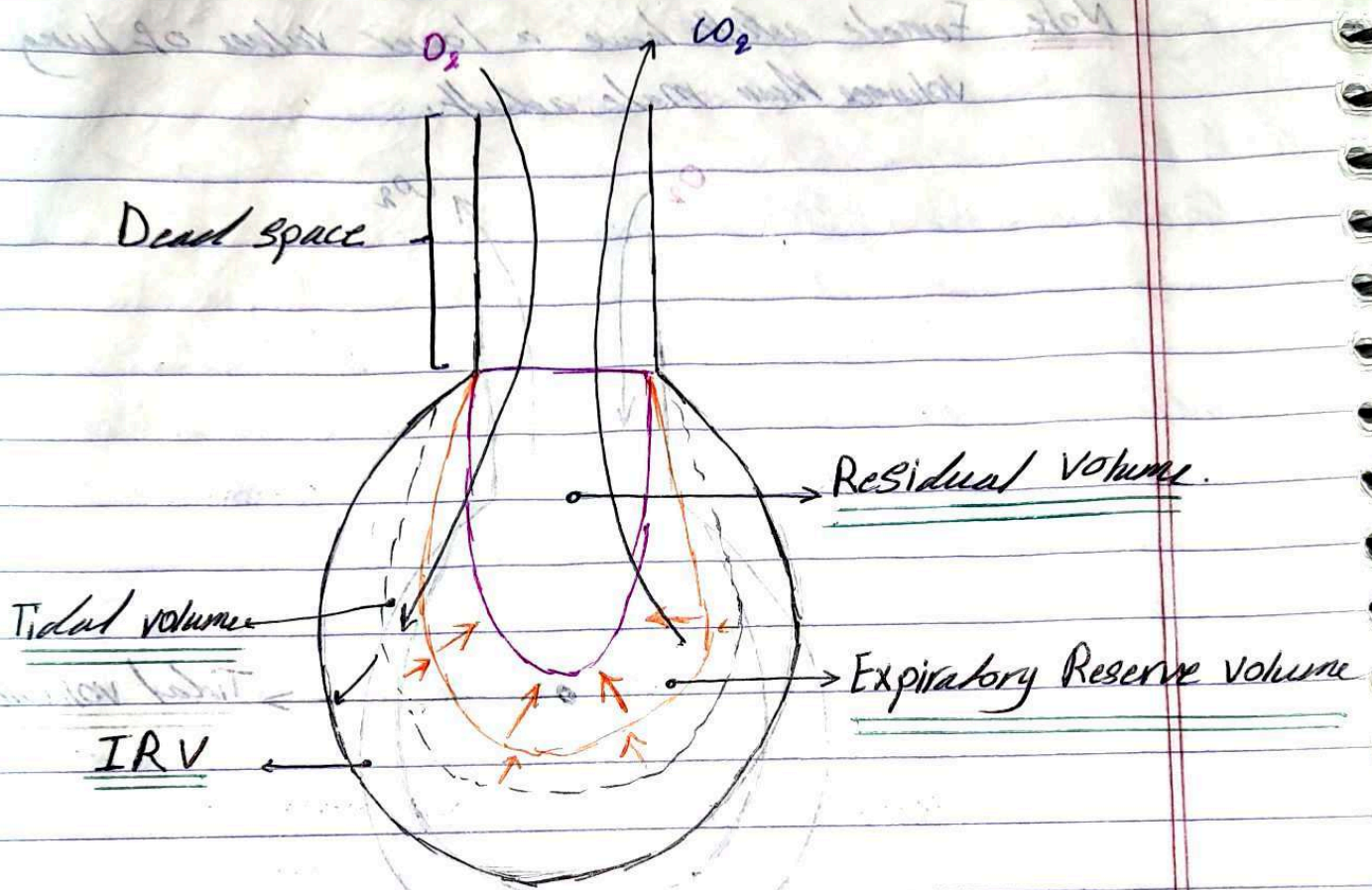
Note Female adults have a lower values of lung volumes than male adult.



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

Inspiratory reserve volume ⇒ take a deep breathing in (deep inspiration) which inspired with a maximal

inspiratory effort in excess of the tidal volume.



Expiratory Reserve volume  $\rightarrow$  Exhale more than usual - amount of air we can exhale in addition to the TV

Residual volume  $\rightarrow$  At the end of the day, there's always an amount of air or volume remaining in our lungs after a maximal effort of ~~Ex~~ expiratory effort