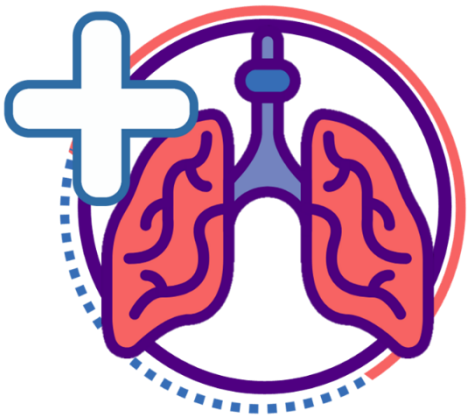

RESPIRATORY SYSTEM

Physiology

Sheet Slide

Number:	- 6
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بسم الله الرحمن الرحيم

This sheet was made from record 6 of section 1 and from the sixth lecture's video of section 2 –the links of both of them you can find them in the last page .

Infant respiratory distress syndrome (IRDS)

A pre mature baby with no sufficient surfactant and a **huge collapsing force of his lungs** so the **inspiration will require too much effort** which result in **multiple abnormalities** :

1)**Hypoxia (decreased Po2 in ABG)**-> a major stimulant for vasoconstriction in the central(pulmonary) arteries ->increased pulmonary pressure -> pulmonary hypertension ->right heart failure. (don't forget that hypoxia is a local vasodilator in systemic circulation)

2)**PATENT DUCTUS ARTERIOSUS:** because the pressure in the right side increases ,the ductus arteriosus (a connection between the aorta and the pulmonary artery which is normally closed ,this was opened in embryo) **reopen again** so the blood goes from right to left heart bypassing the lung (mixing of venous and arterial blood)->**venous pollution (mixture)**

3)**Reopening of foramen ovale** : so reconnecting both atria together resulting in right to left shunt -> increasing hypoxia in addition to the deficient ability to inflate the lung due to the huge collapsing force

4)**Acidosis**

5)**Hypoglycaemia**

So this baby dies from multiple reasons, so in treatment of IRDS we should **fix all the abnormalities** (fixing O2, glucose ,acid-base balance) as well as C-PAP(continuous positive airway pressure :no incubation just keep the airway open)and PEEP(positive end expiratory pressure: require incubation)

Adult/Acute respiratory distress syndrome(ARDS)

-we prefer to use ACUTE because it affects both adults and children

-bad prognosis due to multi organ failure (heart failure ,kidney failure ,liver failure)

-HOW to diagnose?

1)**X-ray**: you see infiltration of the lung

2)**pulmonary capillary wedge pressure** : the pressure measured by wedging a pulmonary catheter with an inflated balloon into a small pulmonary arterial branch .it should be less than 18 mmHg , if it was higher then it is left ventricular(heart)failure

This test is not done by all centres but how is it done ?

We enter with a catheter from subclavian vein to right ventricle to pulmonary arteries ->pulmonary capillaries until it is closed , this pressure is 2-3 mmHg higher than left atrial pressure (so by this we can say it is not caused by the heart, it is caused locally by the lung).

If left ventricular failure-> pressure is more than 18 mmHg leading to pulmonary edema .

3)Po2 = PO2 arterial/fraction of inspired O2

normally it is Po2=100/0.21=476.2

0.21 =21% : the fraction of O2 in the inspired air

We can give pure oxygen (100%) or we can give double of oxygen (0.42) this is what is normally given .

If this Po2 is less than 200 then this is ARDS

If it is between 200-300 then this is Acute lung injury which is a precursor of ARDS

If it is more than 300 then NO ARDS

ARDS is also known as shocked lung/toxic lung/wet lung.

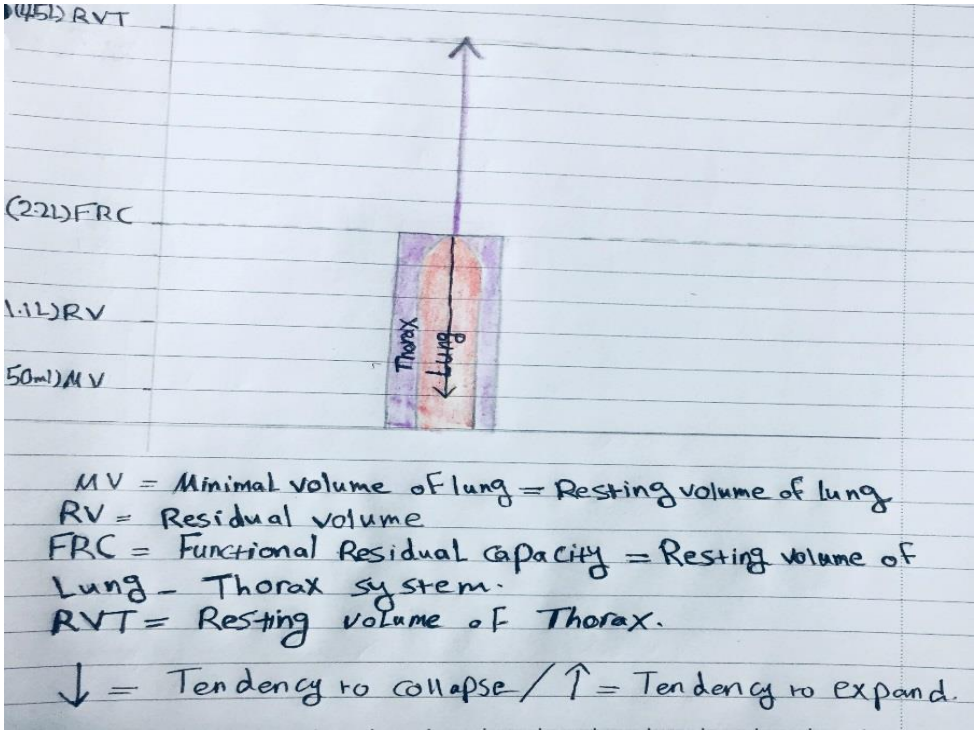
Note : having PO2 of 100 when you give pure oxygen (100%=1) isn't normal , it should be higher .

Lung -thorax system

the lung thorax system is composed of two balloons :

External which is the thorax tending to expand

Internal which is the lung tending to collapse



If you know that Resting state of any subject is the state where its neither collapsed nor stretched

So The resting state (volume)of the lung is the volume when the lung loses its collapsing force , the state where the lung is not tending to collapse any more . **But from where does this collapsing force come ? from the elastic properties of the lung** ,don't forget that the lung is an elastic tissue that have recoil force return it to its resting state .

Lets take an example :

In pneumothorax (air inside the pleura) : the intraplueral pressure will increase till reaching zero(instead of the normal -4) so **we lose the force that opposes collapsing force** so **the lung will automatically collapse to its resting state** which is equal to the minimal volume (150 ml)so when the lung reaches its resting state there is no more tendency to collapse .

Remember even at the FRC, the lung is still tending to collapse but this is balanced (equal and opposite)by the expanding force of the thorax .

Clinical application of the minimal volume

If the baby takes one breath he will have a minimal volume in his lung while there will be **no minimal volume (no air) in the lung in case of still birth** so this is used in forensic medicine(medico-legal purpose) how ?

We take a piece of lung tissue and put it in water if it :

1)**floats** -> there is **air** in the lung -> death happened **after** the baby start breathing (no still birth)

2)**sinks**-> **No air** in the lung ->**still** birth

AGAIN what is the resting volume of the lung ?

The volume when the lung loses its collapsing tendency = the lung doesn't tend to collapse any more

(compliance of the lung in vitro(200ml/cm water) >compliance of lung in vivo (100ml/cm water)why ? simply because in vivo you are filling two balloons not one as in vitro ,also The lung in vitro isn't surrounded by the negative intraplueral pressure but in vivo it located inside the chest cavity .

A note from the video not from the record:

The intrapleurl pressure is needed to overcome the collapsing force of the lung (normally) and it is increased in some cases in order to overcome abnormal expanding force of the chest wall .

the chest wall isn't subjected to abnormalities in most time except if there is congenital defects like kyphosis ,scoliosis.

the thoracic cavity also has a resting volume what is that ?

The volume at which the thoracic cavity loses its tendency to expand. this volume is equal to 75% of TLC =4.5 L out of 6 L.

So let's repeat :

Thorax tend to expand so if we remove the force it will expand to the resting volume

The lung tend to collapse so if we remove the force it will collapse to the resting volume

what about the lung thorax system ?

when the collapsing tendency of the lung is balanced (equal and opposite) by the expanding tendency of the thorax then the system is at rest .

IMPORTANT : what do we call the resting volume of the system ?

We call it FRC (functional residual capacity), yes it is what we Saied previously it is equal to RV+ERV also it is the volume at which the collapsing force of the lung is balanced (equal and opposite) by the expanding force of the thorax .

Remember when you want to stretch a rubber band you need to apply force to change it from its resting state while to return it to its resting state you only release the force. The same applies to any elastic tissue including our lung :

To move any elastic structure from its resting state (deformation) you need a driving force-> ACTIVE process

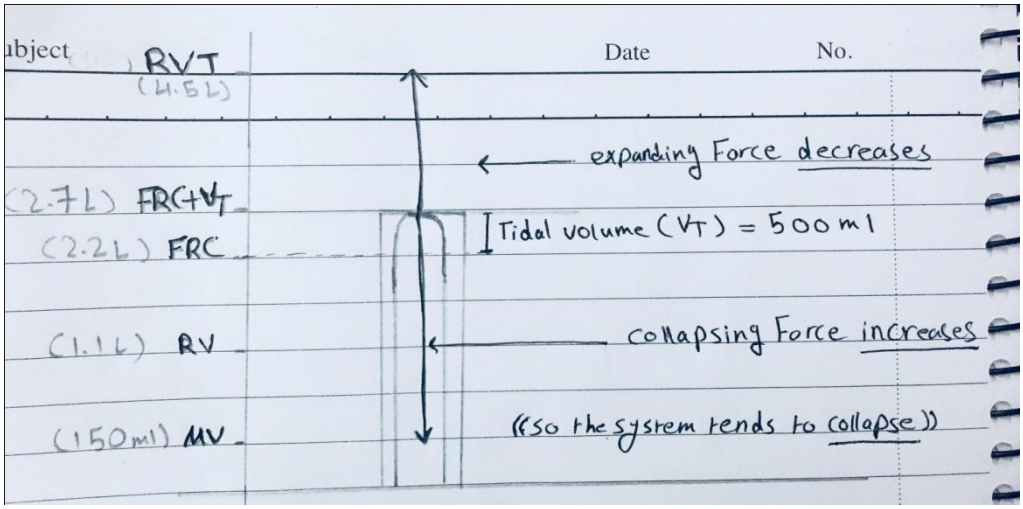
To bring this elasitc tissue back to its resting state its passive because of the recoil tendency

Imagine the resting volume as HOME where you are not tending to collapse or expand you simply feel safe . but when you are away from HOME this tendency increases . (look at the note below)

Note : the closer we are to the resting volume the smaller the collapsing or expanding tendency the more far we are from the resting volume the higher collapsing or expanding tendency

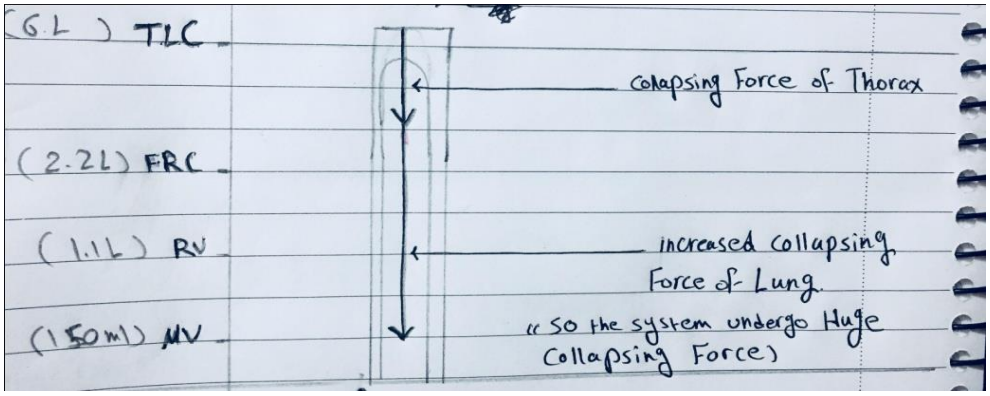
Lets take some examples –look at the graphs of each example while you are reading- :

First :When we take a **tidal volume (normal inspiration)** -> the lung volume **increases** by 500 ml so the lung's **tendency to collapse increase** (since we are more far from its resting) while the **thorax tendency to expand decreases** (because we are closer to its resting) so the **system is tending to collapse** and once you relax your muscles **the lung automatically will collapse again returning to FRC (not the resting) -> in this case the inspiration is active and expiration is passive .(look at the graph in next page)**

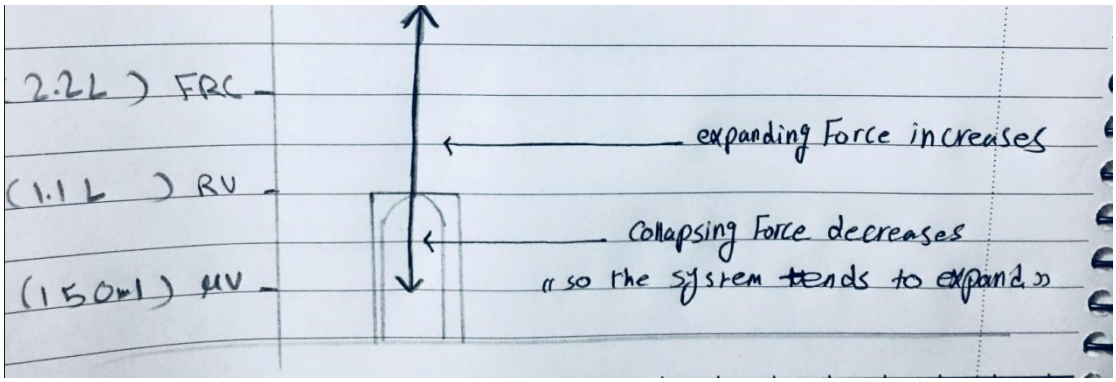


Second :When you **fill your lung to the most** (**reaching TLC**) there is a **huge tendency to collapse of the lung** at this volume and **even the thorax is tending to collapse at TLC** you can

TRY THIS by Filling you lungs to the most, close your mouth and nose ,relax your muscles .. it's very hard ,isn't it ?



Third :So what about if **we are below FRC** ? **the expiration (moving downward in the curve) becomes ACTIVE** by using expiratory muscles(internal intercostals muscles and abdominal muscles) , still the **lung is tending to collapse but it's not that much** (it is decreased since we are coming closer to its resting) while **the tendency of the thorax to expand is high** (since we are far from its resting)so **the system is tending to expand so the inspiration is passive .**



So what should we know by now ?

The expiration is usually but not always passive

The inspiration is usually but not always is active

if the system is tending to collapse then the expiration is passive while if the system is tending to expand the inspiration is passive .

the tendency of the system it is the most important .**in other words, if you want to oppose the system you should pay the price by using your muscles (ACTIVE).**

Let's repeat:

Equilibrium of lung-thorax system

Tendency of the lung to collapse = tendency of the thorax to expand

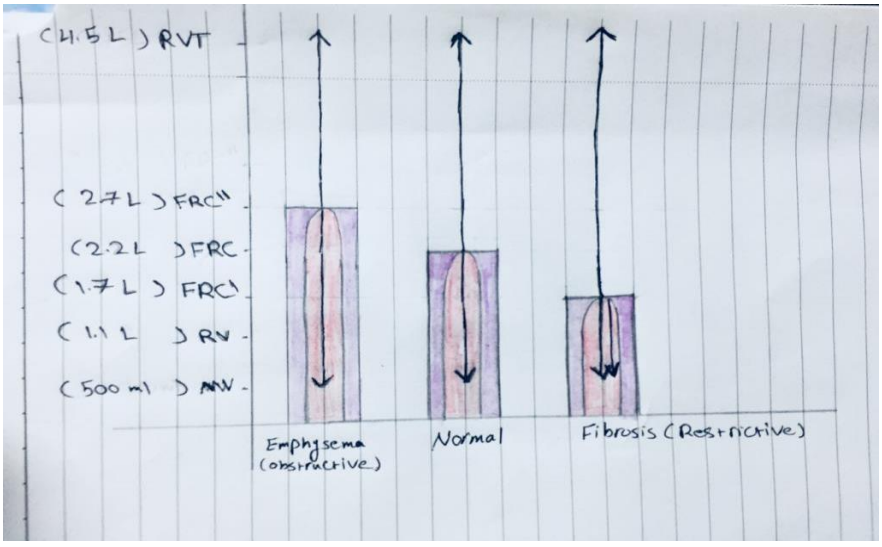
What happens to the system in obstructive and restrictive lung diseases? –see the graph blow while you are reading-

1) In **fibrosis**: lung **compliance is decreased** and the **tendency of the lung to collapse is increased**. therefore **at the original FRC(2.2 L) the tendency of the lung to collapse is greater than the tendency of the thorax to expand so the lung-thorax system will seek NEW LOWER FRC** (1.7L)so that the two opposing forces can be balanced again .

Remember lower FRC means lower tendency to collapse .

so when lung has a high tendency to collapse it will have FRC less than normal -> this applies to restrictive lung diseases

2) **In obstructive like emphysema** : lung **compliance is increased due to the destruction of elastic fibres so the tendency to collapse is decreased** .therefore at the original FRC the tendency of the lung to collapse is less than the tendency of the thorax to expand . the lung-thorax system will seek a NEW HIGHER FRC so that the two opposing forces are balanced again .so increased FRC indicates high compliance .



Always remember that "too much compliance is as bad as too little compliance"

So in case of a stab wound or gunshot **the thorax will equilibrate with the atmospheric pressure becoming zero** so the lung will collapse to its resting and the thorax will expand to its resting .the same happen during thoracic surgeries like CABG but we put the patient on an artificial breathing.
in conclusion here is the differences between obstructive and restrictive lung disease from physiological point :

Pulmonary function test (PFT)	Obstructive	Restrictive
FRC	Higher	Lower
TLC	Higher	Lower
FEV1	Lower	Lower
FVC	Normal or low	Lower
FEV1/FVC	>80%	>= 80%

Cardiopulmonary system

Our goal is to have :

adequate perfusion(Q) : cardiac output from the right ventricle which equals 5 L/min

adequate ventilation(V) : alveolar minute ventilation (350 ml/breath*12breath/min=4.2 L/min)

so normal V/Q (ventilation perfusion ratio)= 4.2/5=0.84 what does this implicate ?

if the V/Q is high then we have ventilation more than perfusion

if the V/Q is low then we have perfusion more than ventilation

we should maintain normal V/Q why? because We don't want an area of the lung to be ventilated and not perfused (wasted ventilation), and we also don't want areas to be perfused but not ventilated (wasted perfusion).

Question: if we have an area that have ventilation but isn't perfused what is V/Q?

Perfusion = zero so V /zero=infinity so at this area the PO2 is 150 mmHg and PCO2 is zero

(no gas exchange has occurred)-look at the PO2-PCO2 curve in the next page-

Another question: why does the PO2 become 150 mmHg(after it has been 160mmHg) when entering the upper respiratory tract ?

Because of the addition of water vapour ,the air becomes **fully humidified before reaching the larynx** which decreases the pressure to 713 after it has been 760 and if we calculate the fraction of PO2 it will be 0.21*713= 150 mmHg but if we want to increase the PO2 in the alveoli what can we do ?

We can do that by **Increasing the percentage of O2 from atmospheric pressure**

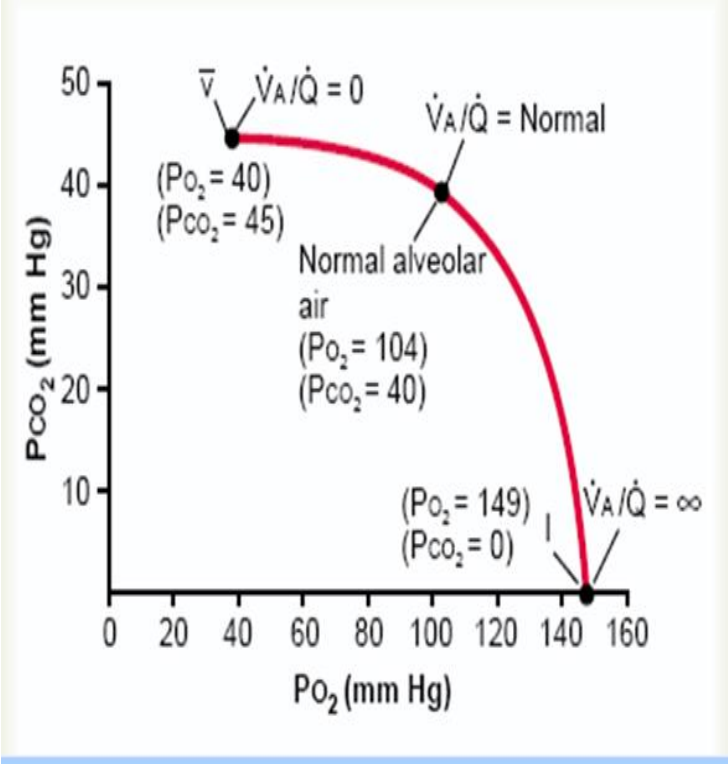
so for example if we double it (making it 42% instead of 21%) then the PO2inspired will be 713*0.42= 300

so the **alveolar PO2= PO2 inspired-(PCO2/R)** : R=0.8 so if we apply this equation we will get

alveolar PO2=300-(40/0.8)=250 mmHg ....**the question NOW is what is R ? this unique R isn't resistance it is what is called respiratory exchange ratio which equals**

CO2 production per minute/O2 consumption per minute =200ml/min/250ml/min=0.8

Note : in normal condition arterial PO2 = Po2inspired(150)-(pco2/R)=150-(40/0.8)=100 mmHg

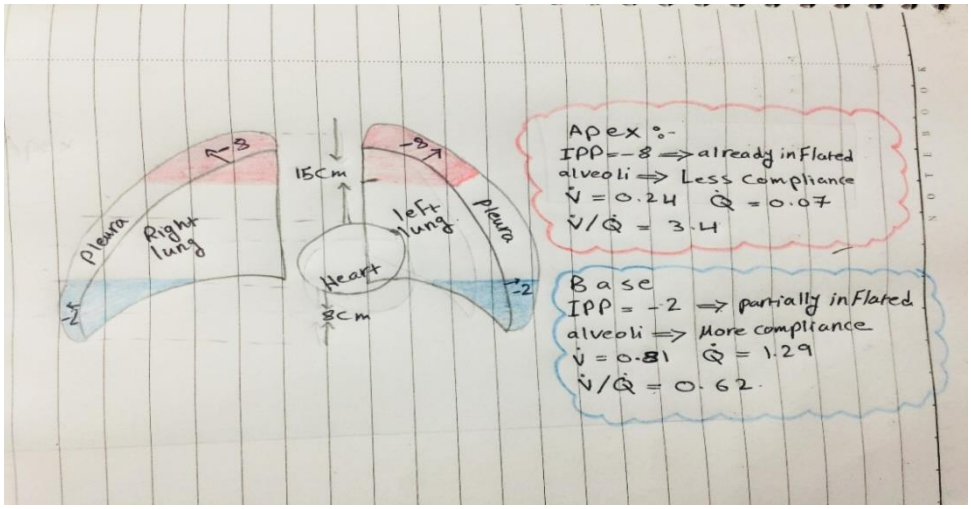


PO2-PCO2 curve :

- When the ventilation (V) is zero, but there is adequate perfusion (Q) of the alveolus, the V/Q is **zero**.
- when there is adequate ventilation, but zero perfusion, the ratio V/Q is **infinity**.
- At a ratio of either zero or infinity, there is no exchange of gases through the respiratory membrane of the affected alveoli

Regional variations in V/Q :

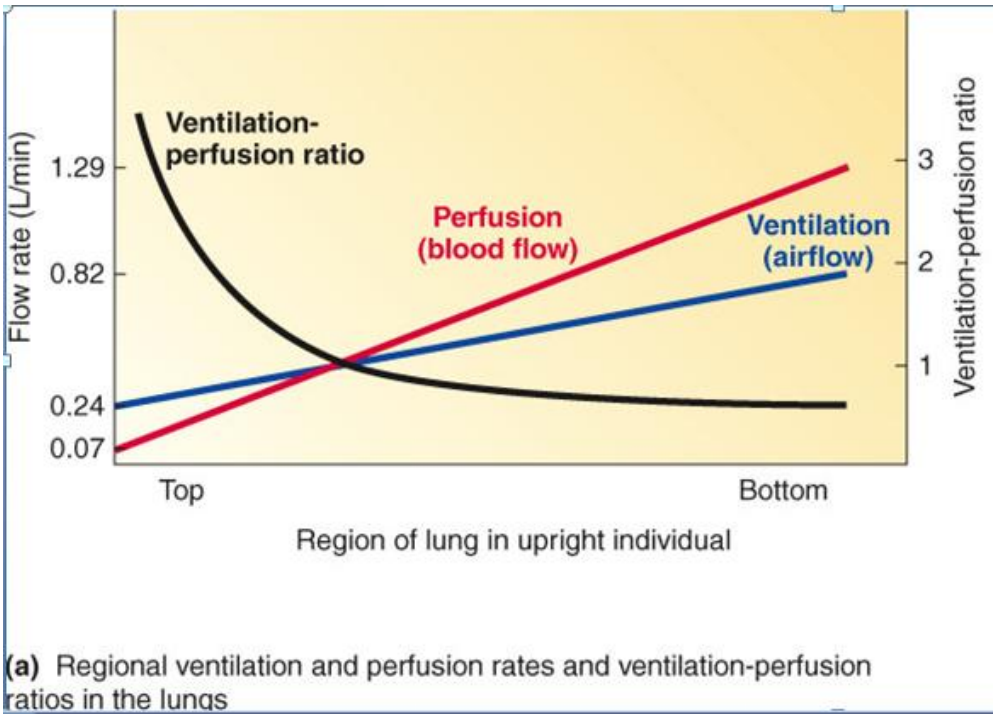
Intraplueral pressure (IPP) isn't the same in different regions of the lungs **only in the erect-standing position (**in the supine position it is the same in all regions) and according to this we divide it to apex and base :



1)APEX: IPP=-8 which makes alveoli already inflated (phase 3 of compliance curve) and you can't inflate the already inflated lung ; we have low compliance so we will have **less ventilation** and because the apex is 15 cm higher than the heart it is more difficult for the blood to flow up than to flow down (due to gravity) so it will have **less perfusion** .

2)BASE: IPP=-2 so the alveoli are partially inflated (phase 2 of compliance curve)so we will have high compliance which means **more ventilation** and since the base is lower than the level of the heart by 8 cm it is easier for the blood to flow down than to flow up(due to gravity) so we will have **more perfusion**

(since blood is more dense than air; it is affected more by gravity and that's why the regional differences in perfusion are greater than ventilation)



(a) Regional ventilation and perfusion rates and ventilation-perfusion ratios in the lungs

The point is perfusion and ventilation are higher in the base than in the apex while V/Q is higher in the apex than in the base , how ?

Imagine you have 1000jd and your friend has 3000jd the ratio of your friend's money to yours is 3 (this what happens at the base)

While if you have 5jd and your friend has 25jd the ratio of your friend's money to yours is 5 (this what happens at the apex)

What is the effect of this difference?

the apex has more ventilation than perfusion(V/Q more than 1) so the PO2 is >100

while

the base has more perfusion than ventilation(V/Q less than 1) so the PO2 <100.

This is clinically beneficial ,HOW ?

The too much oxygen in the apex attracts the aerobic organisms such as TB bacillus to build their nests there. So if you see a shadow in the apical region on the X-ray you should first exclude TB. while if this shadow was in the basal region you should first exclude malignancy (cancer).

Area of lung	Blood flow	ventilation	V/Q	Regional arterial PO2	Regional arterialPCO2
Apex	lowest	lower	higher	highest	lower
Base	highest	higher	lower	lowest	higher

Note : during exercise ventilation ,perfusion,O2consmuption all are increased

Physiological dead space(PDS)

By definition **it is the volume of lung that doesn't participate in gas exchange equal to** :

in abnormal cases : PDS= anatomical dead space (ADS) +alveloar wasted ventilation =150 ml +50 ml = 200 ml

while

in normal people it is the same as ADS

so it can't be less than ADS (either equal or more)

-what is alveolar wasted volume ? the volume of the alveoli that doesn't participate in gas exchange .

normal people don't have alveolar wasted ventilation so it is zero normally while in abnormal condition like **emphysema or in bleeding some apical parts are ventilated but not perfused so they are considered alveolar wasted ventilation.**

Reparatory membrane Respiratory membrane

(mentioned in the video not in the record)

Important prosperities of the membrane : 

Surface area (A) = 50-100 m^2 (directly proportional with diffusion)

Thickness (dx)=0.2-0.6 micrometer (inversely proportional with diffusion)

Only **the first third of the membrane is used** for Gas exchange while the rest 2/3 is a reserve

Clinical application

Example of increased thickness of membrane :

Workers in the phosphate mines are exposed to **phosphate dust which increases the thickness of the membrane so decreasing diffusibilty of gases** , for diagnosis we should know how much they have of the respiratory membrane before stating work and how much is it now ? how can we do this ?

By **DLO2**(diffusion capacity of the lung): a measurement of how much oxygen diffuse through the whole membrane and it is equal to oxygen consumption (VO2) = 250 ml/min normally it is increased during exercise.

Diffusion is a flow and according to Ohm's Law F=Driving force/R

DLO2 or VO2 = difference in PO2 /R =difference in PO2 *K

K=VO2/Difference in PO2

K: permeability =1/R

R: RESISTANCE

**At the end you will find plenty of questions from BRS book -the answers are at the end-:

1. An infant born prematurely in gestational week 25 has neonatal respiratory distress syndrome .which of the following would be expected in this infant ?

a)arterial PO2 of 100 mmHg

b)collapse of small alveoli

b)increased lung compliance

c)normal breathing rate

2. In which vascular bed does hypoxia cause vasoconstriction ?

a)coronary

b)pulmonary

c)cerebral

d)muscle

3. when a person is STANDING ,blood flow in the lungs is

a)equal at the apex and the base

b)highest at the apex owing to Effects of gravity on arterial pressure.

c)highest at the base because that is where the difference between arterial and venous pressure is greatest

d)lowest at the base because that alveolar pressure is greater than arterial pressure .

4. if an area of the lung is not ventilated because of bronchial obstruction , the pulmonary capillary blood serving that area will have a PO2 that is :

a)equal to atmospheric PO2

b)equal to mixed venous PO2

c)equal to normal systemic arterial PO2

d)higher than inspired PO2

5. a person with V//Q defect has hypoxemia and is treated with supplement O2 will be most helpful if this person's predominant V/Q defect is ?

a)dead space

b)shunt

c)high V/Q

d)low V/Q

Link of record 6 :

https://onedrive.live.com/?authkey=%21AKCY6safAjUHHWs&id=7726C7850E9A1950%21109&cid=7726C7850E9A1950

link of sixth lecture video :

https://www.youtube.com/watch?v=V-6248GpKCg&t=0s&list=PLSXxGpUZvfz9TZA-D6OOLoCkvjxYHWHVi&index=7

Answers of the above questions

1	2	3	4	5
B	B	C	B	D

Beauty is power , a smile is its sword .