

COPD

The sheet is divided into :

- 1- Some types of COPD
- 2- Tests for diagnosis of COPD

Information is arranged in a way to make it easy to understand .

First part: COPDs

The term means **C**hronic **O**bststructive **P**ulmonary **D**isease and they include:

Emphysema, chronic bronchitis.

Asthma can be considered as COPD, if it was chronic which mean that there are permanent changes in the airways.

95% of cases in childhood are cured by themselves (self limited) which means the changes are reversible .

1)Emphysema :

Pathogenesis :

- proteases, such as trypsin, are set free attacking the lung tissue they can't be stopped except with anti-proteases such as antitrypsin .

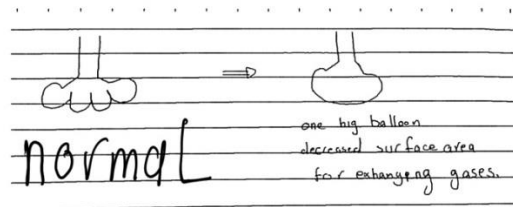
what really happens is that smoking inhibits the anti-proteases from doing there job!

so there will be irreversible destruction(changes) in the lung tissue.

What are these changes?

1- Decreased surface area:

Normally the alveoli's surface area is huge about 50 to 100 m² and will be highly reduced as the alveolar walls are destroyed.

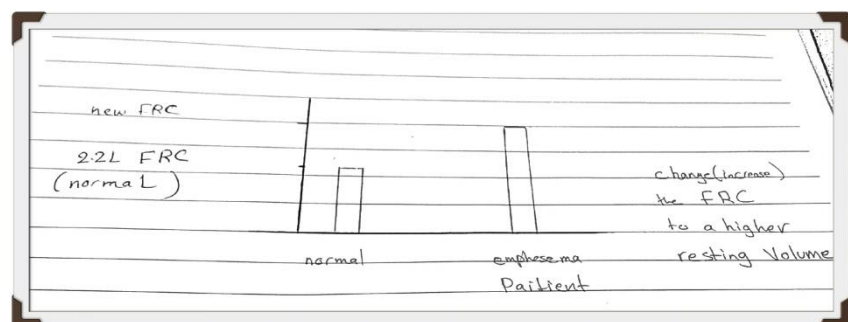


The surface area will be reduced >>The gases exchange will decrease
>>O₂ availability will be reduced and this will result in **hypoxia**.

2- Elastic fibers will be destroyed:

This will increase the compliance of the lung thus the expiration can obstruct the airways.

Emphysema is considered as COPD because it will reduce the recoil tendency, thus the collapsing forces decrease so the system try to change its resting volume into a higher FRC to become close to expanding tendency of the thorax.



this will cause too much air in the lungs:

FRC= 3.5L

TLC= 7L

he can't exhale !!

With aging the muscles become weaker , so the patient can't exhale 1.1 liter to reach RV from FRC so :

ERV will decrease and the RV will increase.

So as a final result, emphysema patient has :

- 1- increased FRC
- 2- increased TLC
- 3- decreased ERV
- 4- increased RV

3- Increase in the pulmonary vascular resistance:

*The cause :

Due to destruction of the capillaries, how they are destructed ?

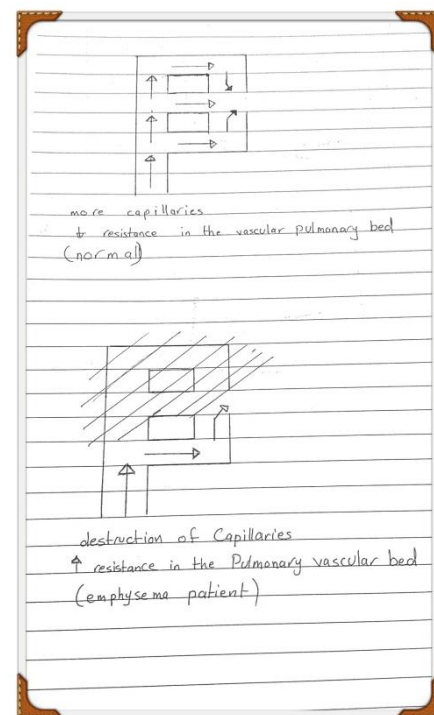
These huge networks of capillaries are part of the alveolar wall, and they are destructed with the wall by the proteases.

Capillaries were used as a route for heart to eject blood toward the lungs.

so when we lose capillaries the resistance in the pulmonary vascular bed will increase, how?

Resistance is inversely proportional to the (cross sectional area) ² .

Losing capillaries will decrease the cross sectional area where blood flows, increasing the resistance. (Check the picture to the left)



*The consequences:

1- Increase in the afterload that faces the right ventricle, which is normally 14(mmHg) now it will be 24 or 34, this will result in right ventricular hypertrophy then dilatation and failure, we call it **congestive heart failure**, and it causes systemic edema (liver, upper/lower limb, hepatomegaly).

The whole condition where we face lung disease and RT.V hypertrophy and enlargement (that may be accompanied by RT.V failure either now or later –it is a matter of time only) is called **Corpolmonale**.

We can see this condition in other cases:

as In high altitudes :

PO₂ is decreased >> hypoxia >> vasoconstriction in the pulmonary vascular bed >> increased afterload >> RT.V hypertrophy >> RT.V failure ... this is called Corpolmonale .

Hypoxia by itself causes vasoconstriction in pulmonary vascular bed >>>>> in the end it will cause Corpolmonale .

Emphysema patient has also heart problem .

Now to sum up :

Cigarettes (which are the most common cause of emphysema) will cause :

- 1- inhibition of anti-trypsin**
- 2- destruction of the alveolar wall and capillaries**
- 3- increased airway resistance**
- 4- decreased surface area for diffusion of gases**
- 5- increased compliance (due to destruction of elastic fibers)**
- 6- increased resistance of the pulmonary vascular bed (results in congestive heart failure).**

It is really serious ! why?

Because the changes are **irreversible (permanent changes)** .. and when the patient get old there will be the aging problems !!

P.S : irreversible changes doesn't mean there is no cells that regenerate elastin but the whole structure is destructed, the alveolar wall and the capillaries .

let's go into the story of aging ..

When an emphysema patient ages, he won't be able to finish his meal or even to walk for short distances.

Aging may increase the TLC a little bit. In normal aging, destruction happens, so:

- 1- TLC increases
- 2- RFC increases
- 3- ERV decreases
- 4-RV increases

and that's why norms in the lung's capacities and volumes differs between patients according to their age, because there is normal degeneration due to aging affecting many tissues including the lungs .

in a person who is 20 years old

the normal consumption of O_2 is $VO_2 = 250\text{ml/ min}$

the max consumption is $VO_2 \text{ max} = 5\text{L/min}$

this is not expected from a 60-old man .

Q: Not all smokers have emphysema, why?

It depends on many factors such as the production of antitrypsin, and somehow there is a genetic factor, as the hypertension patients; some of them is Na^+ sensitive and others are Na^+ insensitive .

But the smoker who survived emphysema, definitely won't survive other complications as lung cancer, bladder cancer, duodenal ulcer .

2)Chronic bronchitis:

caused by smoking, how ?

1- inhibit cilia movement:

so the mucous will accumulate and it is a good media for bacteria growth.

2- causing hyperplasia to goblet cells stimulating it to produce more and more mucous.

and this is another way to obstruct small airways.

3)Asthma :

narrowing of the airways especially those with no cartilage that maintains the opening of the airway, mainly the conducting bronchioles

***causes:**

- 1- From inside such as in exercise (without viruses and bacteria).
- 2- From outside which is most common as in flu infection or with some penetrating substances (we call them allergens).

Allergens or these substances cause our mast cells to produce certain chemicals called slow reactive substances for anaphylaxis such as leukotrienes and histamines and they cause the following :

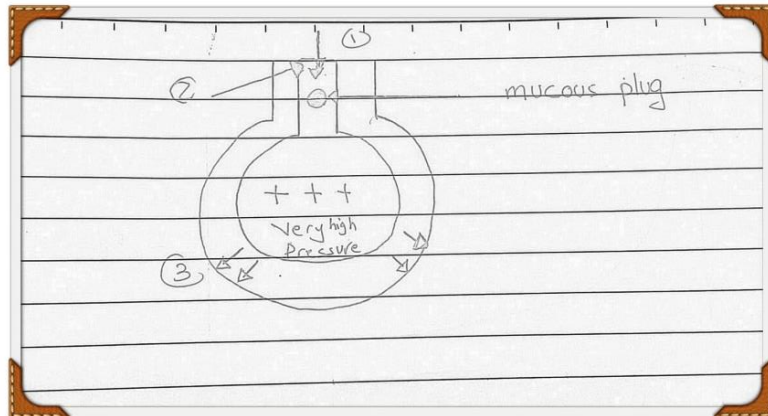
- 1- bronchoconstriction
- 2- inflammation that will lead to edematous layers of the airway >> swelling >>more narrowing
- 3-contraction of smooth muscle >> narrowing
- 4- excessive secretion of mucous

mucous is mostly water and proteins, so water will be absorbed and the protein will remain as a protein plug, that will cause coughing until we get rid of this plug .

First line therapy is bronchodilator.

Dilatation of the bronchi and with some cough we may get rid of the plug.

The principle of the cough; or, what is cough ?



- 1-deep inspiration
- 2- obstruct the outlet of the airways
- 3-contract all the respiratory muscle increase the negative pressure
- 4-pressure inside is very high it may reach 100 mmHg
- 5- sudden open of the outlet and sudden release of the pressure we may push the particle out .

Treatment of asthma :

- 1- First line therapy is bronchodilators :
they are sympathomimic drugs
(beta 2 receptor specific agonists) such as **albuterol** and **salbutamol**
and they are given as inhalers .
beta 2 receptors are found on smooth muscles in the lung.

2- Subcutaneous adrenaline is given in acute stages, such as a 6 years old child fighting for his breath in the ER, he will almost get back to normal in 1min !

3- Glucocorticoids are given as anti-inflammatory inhalers
giving systemic and oral glucocorticoids is dangerous .
Some prophylactic agents are given to prevent the attack
and mainly we should prevent the patient from reaching the causative agent .

Second part : Tests to detect COPD

These tests are used to know if there is an increased obstruction in the airways, and to know the severity and staging of the disease .

Notice that :

- * Tests differs In sensitivity (more sensitive tests will diagnose the obstruction at early stage).
- * Some of them are really the same in normal and in the case of disease!
- * The tests are done by the same procedure but they differ in the values that are measured and the calculations .

So a patient come to us ... asking if he has increased airways resistance ..
what we will do ?

we will keep doing tests (4-5 times) until we get a good diagnosis, and
we will go from a test to a more sensitive test to know the state and
stage of the patient .

1- Forced vital capacity test (FVC) :

* **The goal :**

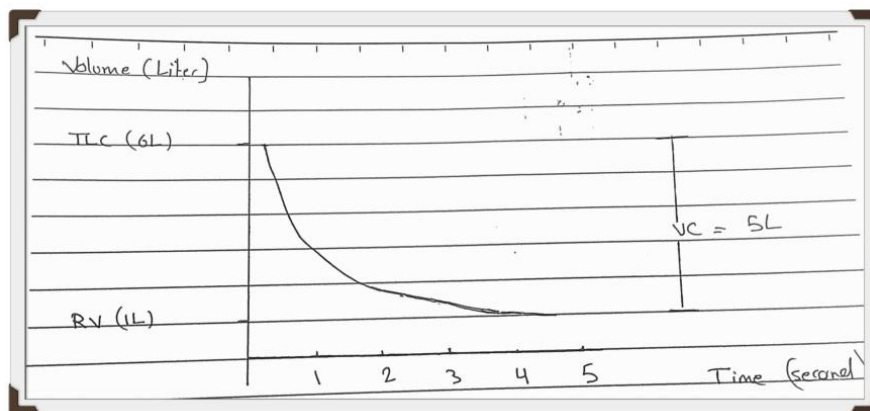
We use this test to measure the time needed by the patient to expire the vital capacity as fast as he can.

*** The procedure :**

We ask him to fill his lungs to the max, then we ask him to empty it as much as he can, as fast as he can down to the residual volume.

normal time needed is 4-5 seconds

FVC = 5L (check the graph below)it is the volume that can forcibly be blown out after full inspiration.



Emptying the lung slowly down to 1L is the same as emptying it fastly, because some airways at the end might close due to increased pressure in fast emptying.

Patients with increased airway resistance might be able to exhale the VC in 4-5 seconds, with no difference than normal people so this test didn't tell us much about the patient's state .

we do more sensitive test !

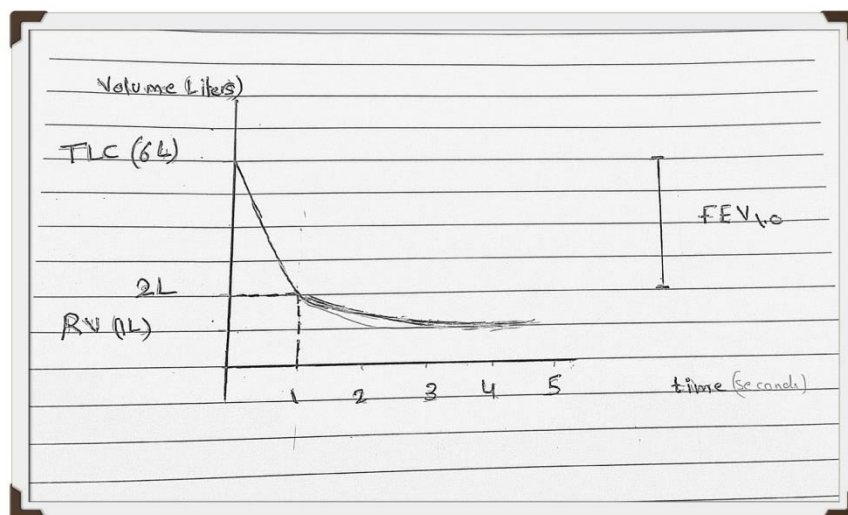
2- Forced expiratory volume in the first one second ($FEV_{1.0}$):

*The procedure :

the same as the previous one.

*The goal:

we measure the volume that was exhaled during the first second and normally it will be 4L (if we considered the FVC = 5L) .



note that he will exhale VC in 5 second in normal situations.

In detecting COPD, what really matters isn't the volume itself
what really matters is **the proportion of ($FEV_{1.0} / FVC$) * 100%**
normally it should equals 80%.

so if the $FEV_{1.0} = 3L$ what does it indicates?

nothing, until we see the FVC, so if it equal 3.8L the proportion will be normal, thus there is no increase in the air way resistance.

***Uses :**

A) We use this test in staging the COPD

1- 60%-79% → mild COPD (or COPD Type I)

2- 40%-59% → moderate COPD

3- <40% → severe COPD

this test can detect changes at 50%-60% obstruction of the airways .

B) Reversibility of asthma, or will the patient benefit from bronchodilators ?

how?

1- we do the previous test

2- give him the bronchodilator

3- repeat the test

If the proportion $(FEV_{1.0}/FVC) * 100\%$ increases by 12% or the $FEV_{1.0}$ increased by 200ml then the patient will benefit and his asthma is reversible .

All in all, these uses mean that this test can be used for staging , prognosis and following up with patients, and it can help in diagnosis.

Note: if the ratio is $\geq 80\%$ we can exclude Obstructive airway diseases.

3- Force expiratory flow rate_{25%-75%} ($FEF_{25\%-75\%}$):

***The procedure :**

is the same as the previous tests.

***The goal:**

to cancel the dilutional effects of the expiration of the first 25% and the last 25% , why ?

Because in normal or obstructed lungs, it is easy to empty the first 25% of the volume of the lung at the beginning of expiration and it is difficult to empty the last 25% of the lung volume at the end of the forceful expiration.

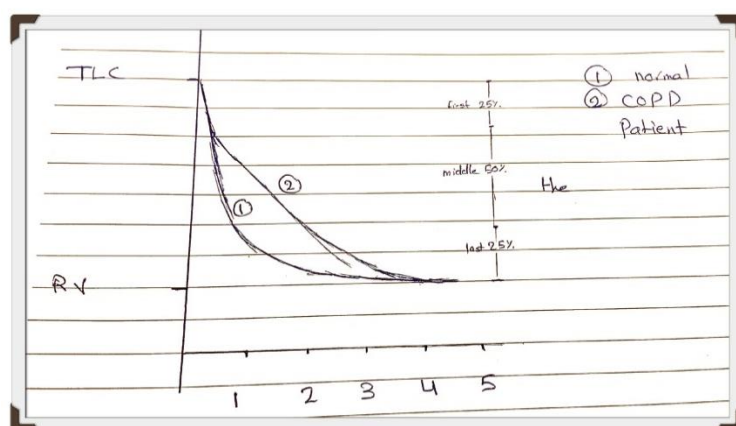
Which means that there is no real difference between the normal person or the patient.

So we take the middle 50% of the lung volume and measures the expiratory rate of it.

And that why it is called,
force expiratory flow
rate25%-75% or
Maximum Mid-expiratory
flow rate(MMEF), which
 means how much time the
 patient needs to remove
 the middle 50% of the lung
 volume during expiration.
 which normally equals
 $FEF_{25\%-75\%} = 3.5L/s$.

How to calculate it ??

$$MMEF = \frac{1}{2} * FVC / \Delta t_{MMEF}$$



To sum up

a patient asks if he has increased airway resistance

1- FVC: he exhaled the VC during five seconds which is normal .

More sensitive test

2-FEV_{1.0}: and it equaled 3.5L

the $[(FEV_{1.0}/FVC)*100\%] = 70\%$

less than normal which is 80% by 10% "not a big deal ".

More sensitive test

3- FEF_{25%-75%} : it equaled for him 1.75L/s

which is decreased to 50% of normal value that equals 3.5L/s

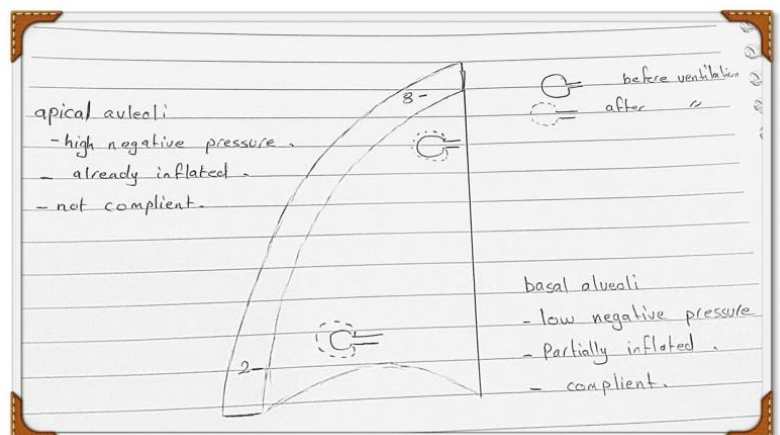
large difference due to increased sensitivity).

4) The closing test :

It is from a different category, and it is the most sensitive one, it can detect any minute obstruction in the airways, all test can be normal but this is not .

- According to the compliance curve of the lung it is difficult to inflate an already inflated alveoli (apical) and it is more easy to inflate less inflated alveoli (basal).
(Check the drawing).

- Air in the apical and basal alveoli contains : O₂, CO₂, N₂, H₂O.



***The procedure:**

We ask the patient to take single breathe of pure O_2 >> tidal volume of O_2 (500ML), most of it will go to the base because the pressure surrounding it is much less than the apex which lead to increased ventilation of the base.

Most of O_2 will go to the base >> more dilution of N_2 in the basal alveoli than the apex :

volume of O_2 in the base is more, volume of N_2 in apex is higher than the base .

During expiration :

- in normal person from the beginning of the expiration until reaching the residual volume the mixed air should come from apical and basal alveoli nothing close until we reach just below the residual volume .

-If we have slight narrowing of the apical or the basal airways which one will have the tendency to close first or is more likely to close at the end of expiration?

The basal alveoli, because they are surrounded by less negative pressure.

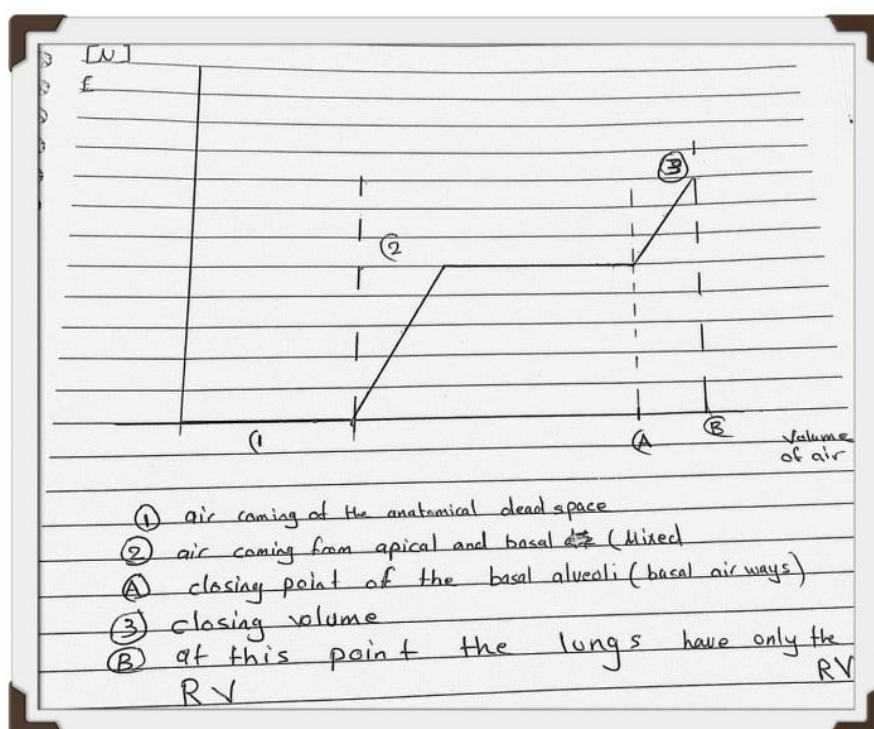
Now during expiration the air is coming out of the apical and basal(mixing of N_2) so it will have a particular volume of N_2 , Then suddenly the N_2 volume will increase, and this marks the closure of the basal airways.

The volume exhaled after this is called the closing volume.

(check the graph in the next page)

Closing volume:

is the volume that is exhaled after the closure of the basal airways up to the residual volume, and normally equals zero.



" إن القيمة الكبرى في ميزان الله هي قيمة العقيدة
 و إن السلعة الرائجة في سوق الله هي سلعة الإيمان
 و إن النصر في أرفع صورته هو انتصار الروح على المادة
 و انتصار العقيدة على الألم
 و انتصار الإيمان على الفتنة "
 سيد قطب
 كتبها : طارق النمل