Doctor.021

no. 2

RS PHYSIOLOGY

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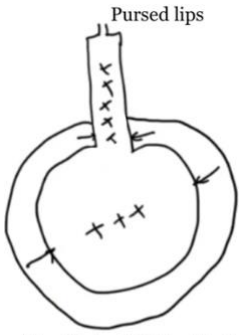
بسم الله الرحمن الرحيم
"اللهم لا سهل إلا ما جعلته سهلًا و أنت تجعل الحزن إذا شئت سهلا ، ربِّ اشرح لي صدري و يسر لي أمري واحلل عقدة من لساني يفقهه قولي، اللهم علمني ما ينفعني و انفعني بما علمتني و زدني علمًا، اللهم أرني الحق حقًا و ارزقني اتباعه و أرني الباطل باطلًا و ارزقني اجتنابه"

This sheet includes the notes in the ppt slides which the doctor said are important.



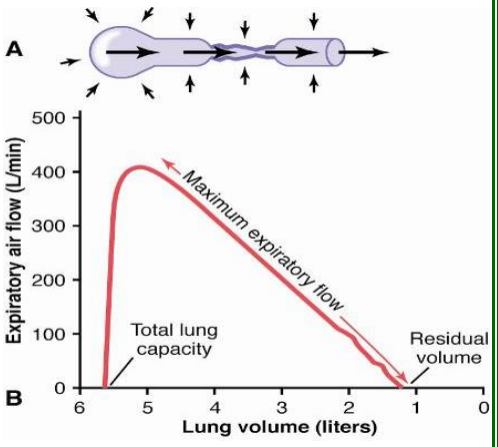
Alveoli of a COPD patient

As we said COPD patients have a problem of high resistance and to overcome that they need a higher driving force and to do that they need to increase the pressure inside the alveoli and that is by making the IPP positive which will help in overcoming the resistance, but it arises another problem, this increase in pressure will happen also at the end of the airways and this will decrease the flow through it, and to overcome that COPD patients will close their lips in a “pursed manner” which will increase the pressure through the airways and help keeping them open after the pressure was increased in them, but for this technique to happen it needs the patient to be awake so when he sleeps he won’t be able to do it and the airways will close and this will case suffocation.

Pursed Lip Breathing in COPD patients: (the doctor didn’t read this)
Collapse of your airways on expiration, as your lungs are getting smaller as you breathe out. This is a particularly serious problem in people with Emphysema, as the elastic supporting lung structure helping to keep the airways open is deficient.
Pursed Lip Breathing simply imposes a slight obstruction to expiration air flow at the mouth, which generates a back pressure throughout the airways, and therefore a stenting effect to help prop open the airways and assist expiration and lung emptying. It must be emphasized, the amount of pressure supplied by you by pursing your lips together must, as usually described, be "minimal," or "gentle."
Another test to know whether the patient has obstruction or not (add it to the other tests we talked about in the previous lecture):

1. We ask the patient to fill his lungs to the TLC (6L in normal people).

2. After forced expiration, we measure the maximum expiratory flow rate (MEFR)≈400L/min in normal.
3. Measure the residual volume (Rv) in normal conditions it is 1L and the maximum flow rate at this point should be zero, because there is no flow. And by that we will get the curve below.



Notice that the first phase of expiration is the fastest, if you filled a balloon to the max and let it loose at first it will deflate fast and forcefully, but if you only filled the balloon with air halfway through and let it deflate, the air will go out fast but not as fast as the first case.

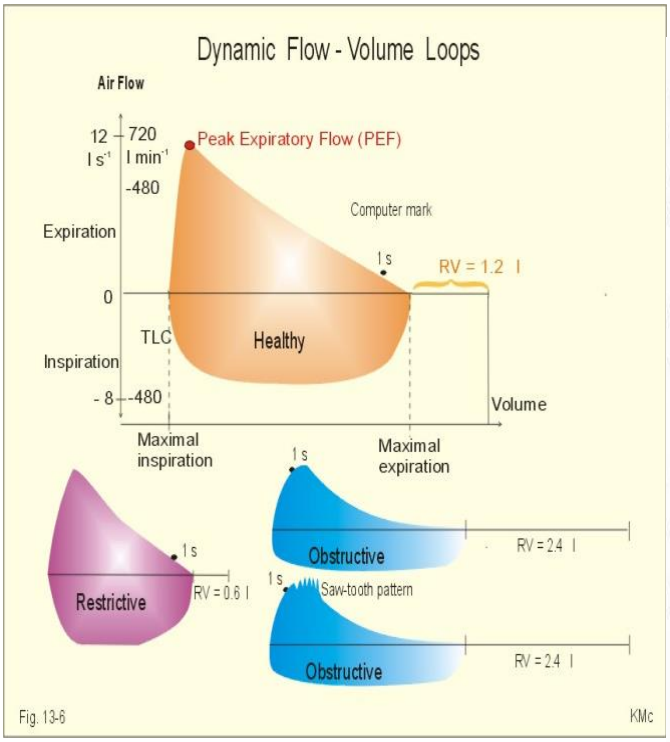
In the figure what is above the lone represents the expiration and below is the inspiration.

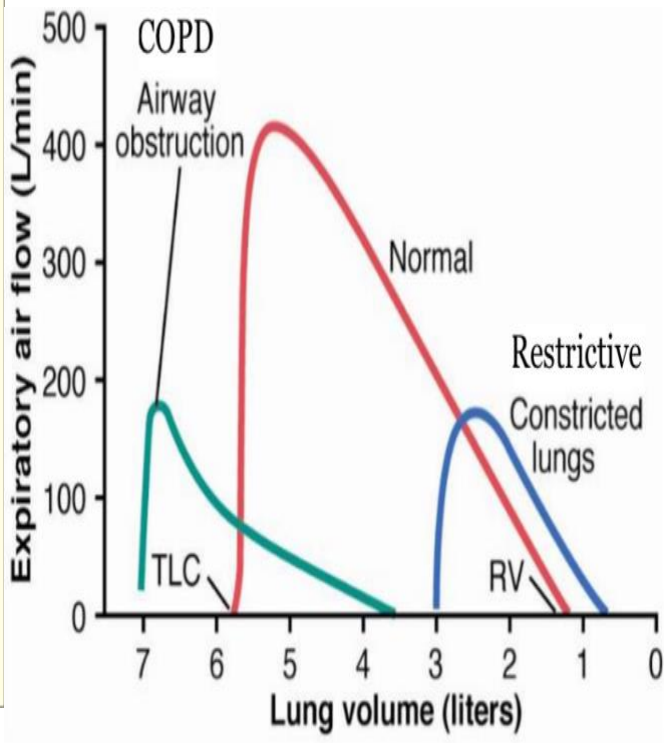
For patients with **COPD** (obstructive lung diseases) the **TLC will be 7L** instead of 6, and because their problem is with expiration, their **maximum expiratory flow rate (MEFR) will be 200L/min** instead of 400, and the deflation will be quite easy at first but then it becomes very difficult so that stage won't be linear as in normal people, but it will be **pushed to the inside and curved** (see the picture), and the **Rv is higher** than the normal and is approximately **3L** instead of 1L (because these patients can't expire as much air remember they are called "bloaters").

At every lung volume there exists a maximal rate of flow which cannot be exceeded. When an individual tries to exceed his maximal flow rate, he forcefully contracts his abdominal muscles to increase his already positive pleural pressure. This increases the driving pressure for air flow from the alveoli to the mouth but also causes the bronchi (whose pressure lies somewhere between that in the alveoli and that at the mouth, but is less than pleural pressure) to collapse. Thus, the airways become occluded and flow

is slowed until the pressure difference across the airways drops a bit, the airways can reopen, and flow can continue.

On the other hand, in patients with **restrictive lung diseases**, the TLC will be less reaching **4L,** and the **maximum expiratory flow rate (MEFR) will be 200L/min** (the bigger the volume you start exhaling from, the faster the maximum expiratory rate)**,** and the shape of the expiration curve will be **like the normal** but smaller (not curved like the figure in COPD), provided that at any volume when we compare the normal and the curve of restrictive patients the expiratory flow rate is always higher than the normal at comparable volumes (see the figure) because the collapsing tendency in restrictive patients is higher and the speed of deflation will be higher thus the rate will also be higher, but when we compare the maximum flow rate of the two we will see that the normal will be higher (400 in normal and 200 in restrictive), but that is just because in restrictive lung patients they can't reach the maximum volume TLC a normal person can reach (6 in normal and 4 in restricted) (refer to the figure), and the **Rv will be less than normal =.5L** instead of 1L in normal.





- In case of obstruction pulmonary disease:

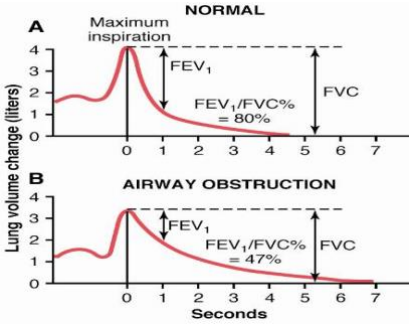
1. The compliance increases, and the lung is easier to inflate ⟶ higher TLC.
2. Due to the obstruction the flow in interrupted during exhalation ⟶ lower maximum expiratory rate.
3. The shape of the curve is different than the normal مقعر.
4. The curve is shifted to the left.

- In case of restriction pulmonary disease:

1. The compliance decreases but the collapsing force increase ⟶ Lower residual volume.
2. Inflation of the lung is difficult ⟶ Lower TLC.
3. The shape of the curve is like the normal one.
4. The curve is shifted to the right, with lower peak.

Restrictive Diseases:

- **Decreased expansion of the lungs.**
- **Lung volumes.**
  - **reduced VC, FRC, RV and normal airway resistance.**
- **Diffuse Interstitial Pulmonary Fibrosis.**
  - **thick collagen deposits.**
- **Pneumothorax.**



As we said in the last lecture, FCV is the forced vital capacity, and FEV1/FVC=80% in normal people, but in airway obstruction as you can see in the figure, FEV1 is lower than the normal and the FEV1/ FVC=47% (less than the normal).

Forced expiration curves are particularly useful because they are so reproducible.

FEV1 (Forced Expiratory Volume in 1 Second) -- This is the volume of air expired in the first second during maximal expiratory effort. The FEV1 is reduced in both obstructive and restrictive lung disease. The FEV1 is reduced in obstructive lung disease because of increased airway resistance. It is reduced in restrictive lung disease because of the low vital capacity.

Now we will start with a new topic which is **the pulmonary circulation.**

Pulmonary Pressures

Pulmonary artery pressure:

- **systolic 25 mmHg**
- **diastolic 8 mmHg**
- **mean 15 mmHg**
- **capillary 7-10 mmHg**

• Left Atrial and Pulmonary Venous Pressures = 2 (1-5) mm Hg (estimated).

• Pulmonary wedge pressure = 5 mm Hg (usually its 2 to 3 mm Hg greater than the left atrial pressure.

Pulmonary circulation is similar to the systemic circulation in some aspects (the cardiac output is 5L for both, the same colloid osmotic pressure π=28 (which is the protein concentration (mainly albumin), so it is the same in all arteries (except for the renal)) and different in other (the capillaries pressure and the hydrostatic pressure).

The pressure in the arterial side of the systemic capillary =40 and on the venous end=20 and the middle =30, while the pulmonary capillary pressure is 7-10 (less).

In the systemic we have 20L filtered and 17L reabsorbed and 3L return back through the lymphatics in 24hrs.

In lungs, the purpose of the blood flow isn't for filtration, but to exchange gases, so the lung must be kept dry, we cannot tolerate pulmonary edema it causes death, but we can tolerate edema in limps or abdomen (ascites), other types of edema that can't be tolerated other than pulmonary: cardiac, laryngeal, brain.

And to prevent edema in lungs, the hydrostatic pressure in the pulmonary capillary is lower than the systemic.

In a pulmonary capillary we can see the following:

- Pulmonary capillary hydrostatic pressure Pc=10 (remember its direction is to the outside of the capillary, favours filtration).

- Pulmonary capillary colloid pressure πc=28 (directed to the inside of the capillary, it opposes the filtration it favours reabsorption).
- Interstitial hydrostatic pressure Pi=5
- Interstitial colloid pressure πi=14

The forces that favour filtration are:

1. Pc
2. Pi
3. πi

10+5+14=29 (for the forces that favour filtration)
And the reabsorption is 28, so the net is +1 which is low, and the lung can deal with it through the lymphatics since it's filled with lymphatics which drains in the right lymphatic duct (with the exception of the left apex which drains in the thoracic duct)
The flow rate in the thoracic duct is 100ml/hr= 2400/day, while the right lymphatic duct rate is 20ml/hr =480ml/day, and the net by the two≈ 3000ml/day which is the 3L that return back through the lymphatics from the systemic blood.
πi = 14 this means that the capillary is leaky to proteins.
Where can you find the highest πi in the body? In the liver, due to the very high permeability of sinusoids.

Protein concentration in liver = 4-6 g/dl

Protein concentration in plasma = 6-8 g/dl

Protein concentration in muscles = 1-2 g/dl

A side note from the doctor: Pi value differs from tissue to other according to function, mostly it's positive which means that it favours absorption, in kidneys it's highly positive, but in lungs as we said it's around -5 meaning that it favours filtration, in skin (subcutaneous tissue) it's -3 or -2 that's why skin is adherent to our body.

Reasons Why Pressures Are Different in Pulmonary and Systemic Circulations:

- **Gravity and Distance:**

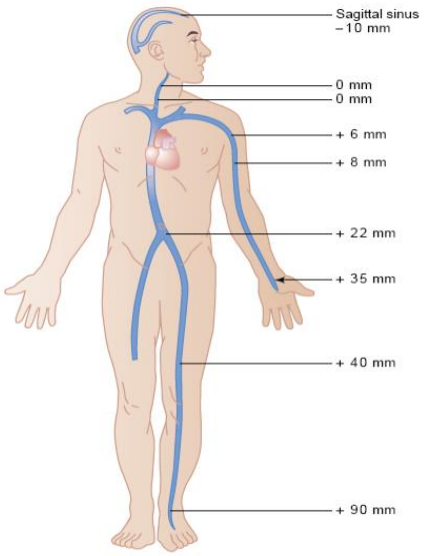
- Distance above or below the heart adds to, or subtracts from, both arterial and venous pressure.
- Distance between Apex and Base affected by gravity.

- **Systemic:**

Aorta	**100mmHg**
Head	**50 mmHg**
Feet	**180mmHg**

- **Pulmonary:**

Mean PA	**15 mmHg**
Apex	**2 mmHg**
Base	**25 mmHg** (Much more than the apex)



Effect of hydrostatic pressure on venous pressure in the standing position

Difference between pulmonary capillary & that of systemic:

	Pulmonary capillary	Systemic capillary
Pc	**10 mm Hg**	**17 mm Hg in certain tissues** **30 mmHg in muscles** **60 in kidneys**
Πc (constant, the same btw pulmonary and systemic)	**28 mmHg**	**28 mmHg**
Pi	**- 5 mmHg**	**Zero (it differs from site to other, but we put it 0 for calculation purposes to be easier)**
Πi	**14 mmHg***	**7 mmHg**

The composition of alveolar air reflects the harmony by which respiratory & cardiovascular systems are working Ventilation: Perfusion Ratio (V/Q).

Now we have alveolar oxygen and carbon dioxide pressure (P_AO_2 &P_ACO_2) So what affect them?

$P_AO_2 \propto \dot{V}_A/\dot{V}_{O2}$ (alveolar ventilation/ O_2 consumption)

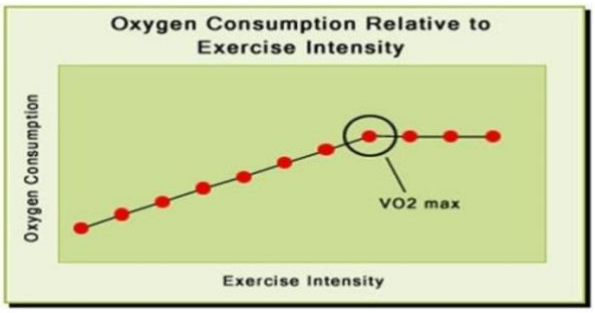
Oxygen consumption at rest is around 250ml/min, as you exercise it will increase until 3 L in normal person (5 L in athletes) and that's what we call maximal oxygen consumption.

V_{O2} max is something you were born with and it's a fixed value for each person, but by exercising it may increase by 10% or less.

$\dot{V}_{O2}$ max is maximal oxygen consumption in maximal exercise.

Actually, high $\dot{V}_{O2}$ max is essential for marathoners with other needs as high amount of red muscular fibers (white fibers is needed for weightlifters), high TLC and diffusion capacity …etc

The alveolar ventilation it can be calculated,in normal person its around 4.2L/min at rest, so this is the normal ventilation.



- Hyperventilation is when you trying to keep composition of air inside alveoli close to outside air (means more oxygen, & less CO_2)

By definition it's when arterial CO_2 pressure become less than 40.

In normal ABGs P_aCO_2=40, in hyperventilation P_aCO_2<40.

- Hypoventilation is when you trying to make composition of air inside alveoli close to mixed venous blood (the composition of air they have is similar to that comes to them by capillaries), if the hall lung is hypoventilated the person will die.

$P_ACO_2 = \dot{V}_{co2}/\dot{V}_A * 0.863$

$\dot{V}_{co2}$ from carbon dioxide production its around 200ml/min

So $P_ACO_2 = \dot{V}_{co2}/\dot{V}_A * 0.863$=200/4.2*0.863= 41

During exercise carbon dioxide production will increase, alveolar ventilation increases too, that keeps P_aCO_2 constant (ABGs values will stay normal).

Another important term you should know is exchange ratio or RQ (respiratory quotient) …. RQ= $\dot{V}_{co2}/\dot{V}_{o2}$

If you eat only carbohydrates, you will make one co2 molecule for each o2 molecule you consump (RQ=1)

If you eat only proteins (RQ=0.9), only fats (RQ=0.7)

But for normal person who eats mixed food (RQ=200/250=0.8)

Pressure in the different areas of the lungs

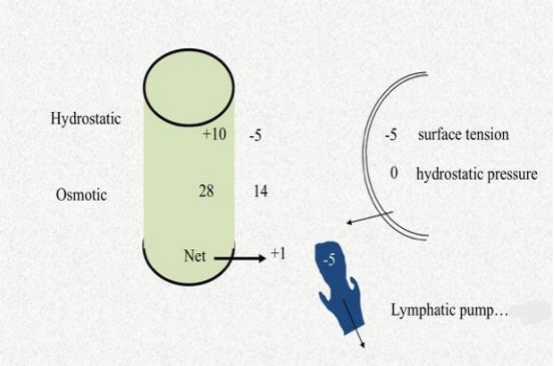
- **At the top, 15 mm Hg less than the pulmonary arterial pressure at the level of the heart.**
- **At the bottom, 8 mm Hg greater than the pulmonary arterial pressure at the level of the heart.**
- **23 mm Hg pressure difference between the top and the bottom of the lung.**
- **These differences have effects on blood flow through the different areas of the lungs.**

PULMONARY RESISTANCE TO FLOW

- **Pressure drop of 12 mmHg (P_m-PRA).**
- **Flow of 5 l/min.**
- **Resistance 1/7 systemic circulation.**

Pulmonary Capillary Dynamics

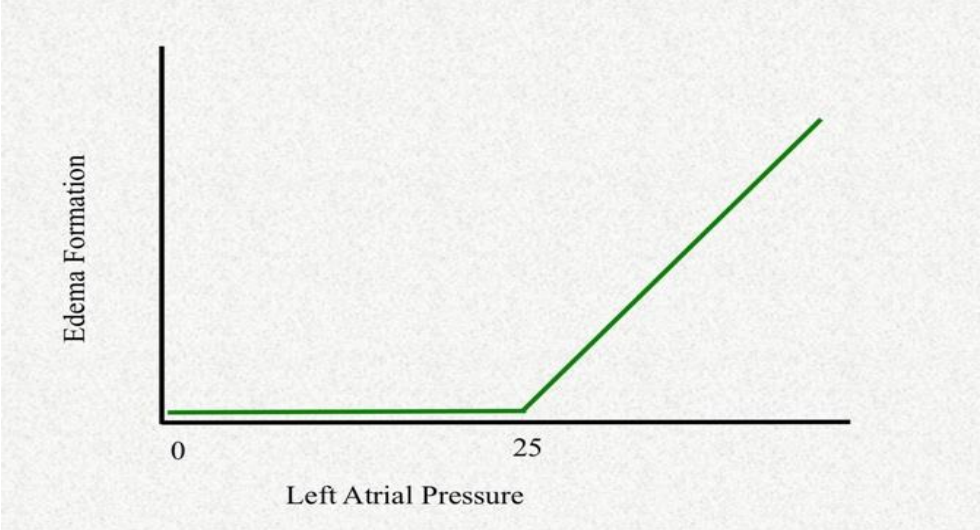
- **Outward Forces**
  - **Pulmonary capillary pressure 10 mmHg (in pathological conditions it may raise to 28mmHg and the lung still handle it and edema doesn't occur, due to very high lymphatic drainage).**



  - Interstitial colloid osmotic pressure: 14 mmHg
  - Negative interstitial pressure: 5 mmHg
  - Total: 29 mmHg
- Inward Forces
  - Plasma osmotic pressure: 28 mmHg
- Net filtration pressure: 1 mmHg
- Lymphatic vessels take care of this extra filtrate.
- There is plenty lymphatics which empty in the right lymphatic duct to prevent the occurrence of pulmonary edema. The left apex empties in the thoracic duct.
- The filtrate will be pumped by the highly effective lymphatic drainage. Actually, if, due to Left heart failure the pulmonary capillary P reaches 28 mmHg (equals blood colloid osmotic pressure P) :(21 mm Hg above normal) pulmonary edema would not develop. (21 mm Hg is a safety factor). That is true in case of acute state, however, in chronic conditions (> 2 WKS) the lung become even more resistant to pulmonary edema and a capillary P of 40-45 develop without significant pulmonary edema.

Pulmonary Edema

- Causes of pulmonary edema!



- **left heart failure.**
- **damage to pulmonary membrane: infection or noxious gas such as, chlorine, sulfur dioxide.**

- **Safety factors:**

- **negative interstitial pressure.**
- **lymphatic pumping.**

Past papers

• Hyperventilation can result from:

a- increase alveolar Pco2.

b- increase alveolar Po2.

c- decrease arterial Pco2 below 30 mmHg.

d- direct stimulation of central chemosensitive receptors due to increase PH.

e- a decline of arterial Po2 from 100 mmHg to 70 mmHg.

• Hypoventilation causes one of the following changes in arterial blood gases:

a. Increase in arterial PO2, increase in arterial PCO2, and decrease pH.

b. Increase in arterial PO2, decrease in arterial PCO2, and increase pH.

c. Decrease in arterial PO2, decrease in arterial PCO2, and increase pH.

d. Increase arterial PO2, no change in arterial PCO2, and increase pH.

e. Decrease in arterial PO2, increase in arterial PCO2, and decrease pH.

• Regarding bronchial asthma, all the following statements are true EXCEPT:

a- Cough suppressants are highly indicated.

b- Airway resistance is increased.

c- During the attack, FEV1.0/FVC is < 80%.

d- Bronchodilators can be given to asthmatic patients.

e- Patients might be allergic to pollens.

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c- decrease arterial Pco2 below 30 mmHg.
d- direct stimulation of central chemosensitive receptors due to increase PH.
e- a decline of arterial Po2 from 100 mmHg to 70 mmHg.

• **One of the followings is expected in idiopathic pulmonary fibrosis:**
a- lower than normal FRC.
b- higher than normal tidal volume.
c- lower than normal pulmonary vascular resistance.
d- higher than normal TLC.
e- higher than normal lung compliance.

• **In diving, divers first hyperventilate before they go into water. This hyperventilation allows one to hold one's breath for a longer period of time, because hyperventilation:**
a. increases the oxygen reserve of systemic arterial blood.
b. decreases the PCO2 of systemic arterial blood.
c. decreases the pH of systemic arterial blood.
d. increases brain blood flow.
e. makes alveolar air full of O2 which divers can use while diving.

• **True regarding a patient with pulmonary fibrosis:**
Decreased peak expiratory flow, decreased FEV1 and increase collapsing forces.

(وَكَأَيِّن مِّن نَّبِيٍّ قَاتَلَ مَعَهُ رِبِّيُّونَ كَثِيرٌ فَمَا وَهَنُوا لِمَا أَصَابَهُمْ فِي سَبِيلِ اللَّهِ وَمَا ضَعُفُوا وَمَا اسْتَكَانُوا ۗ وَاللَّهُ يُحِبُّ الصَّابِرِينَ (146) وَمَا كَانَ قَوْلَهُمْ إِلَّا أَن قَالُوا رَبَّنَا اغْفِرْ لَنَا ذُنُوبَنَا وَإِسْرَافَنَا فِي أَمْرِنَا وَثَبِّتْ أَقْدَامَنَا وَانصُرْنَا عَلَى الْقَوْمِ الْكَافِرِينَ (147) فَآتَاهُمُ اللَّهُ ثَوَابَ الدُّنْيَا وَحُسْنَ ثَوَابِ الْآخِرَةِ ۗ وَاللَّهُ يُحِبُّ الْمُحْسِنِينَ(١٤٨))
دعواتكم لأهلنا في غزة و السودان و للمستضعفين من المؤمنين في كل بقاع الأرض

Answers: A, E, C, A, A, B