Main function of respiratory system is to supply oxygen, and respiration is divided into two types:
1-external: between lungs and environment, how we breathe in and out
2-internal: between capillary and the tissue, oxygen and CO2 exchange (this is wrong but the doctor said that, the true definition Internal respiration encompasses the intracellular metabolic reactions involving the use of O2 to derive energy (ATP) from food, producing CO2 as a by-product.) *we already explained this in CVS, so we will be discussing External respiration

**RS have other function rather that providing oxygen and expelling CO2, we call them non-respiratory function which are: slide 2
1-water loss and heat elimination, also keeps alveoli wet
2-inhances venous return
3-acid-base balance (pH) → If acid increases → we expel carbon dioxide out
If we want more acid we keep the carbon dioxide inside our body
4-enables speech
5-defends against foreign inhaled matters → stimulates mucous production and coughing
6-removes, modifies, & activate or inactivate materials “prostglandins”
*activating enzymes → like angiotensin converting enzymes
7-smelling
8-shape of the chest
9-protects heart & vessels
10-aireate the blood between respiratory phases

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Min 5

5: Physiology of respiration
As you told in anatomy Structure of RS divides into:

→ Air conducting channels \ respiratory passageway \ conducting zone:
anything between the mouth & the nose until alveoli, doesn’t have a role in exchanging function (no gas exchange), just a passageway for air

→ Respiratory spaces:
1- Alveolar ducts, Alveolar Sacs and alveoli inside Lungs, exchange occurs here

→ Lungs float in the thoracic cavity + pleura
Mechanic of respiration occurs through these steps:

1. **pulmonary ventilation**: air flow between the atmosphere and the lungs.
   *Inspiration*: From Atmosphere **Into the lungs (INter)**
   *Expiration*: From lungs into Atmosphere **(EXit)**

2. diffusion of gasses between the alveoli and the blood.
3. transport. Transport of oxygen and CO2 in blood
4. regulation of respiration

Mechanic of ventilation:

Inflow & outflow (inspiration and expiration) have the same principle as Poistues law same as blood flow, air flow depends on pressure gradient, air enters the lungs when atmospheric pressure is greater the pressure in lungs, when the pressure is the same there is no flow and it’s called equilibrium.
Let’s talk about inspiration, expiration, their mechanism and pressure changes.

**At normal condition** Inspiration is an active process (needs ATP) and expiration is passive (Doesn’t need), why?

Because during inspiration Contraction of the main inspiratory muscles: Diaphragm and External intercostal occur which uses ATP, unlike expiration which is caused by relaxation of those muscles

But what about Pathological cases (like COPD) & Exercise?
Both the inspiration & expiration are ACTIVE, because Expiratory muscles will be used which are 1-Abdominal muscles → mainly 2- Internal intercostal
Also accessory inspiratory muscles (Sternocleidomastoids, Sternum←Scalini (2 ribs) & Anterior serrati) will be used to assist inspiration.

NOW how that helps us in breathing and pressure gradient?
First, inspiration:
before the inspiration begins, Alveolar pressure equals to atmospheric (760 mm Hg) or ZERO cm H2O, so there is no air flow and this is equilibrium
**Diaphragm** is a dome shaped muscle and when it contracts (shortens) it flattens and increases Volume of thorax **longitudinally** (upside-down) and **external intercostal** (By elevation and depression of the ribs **anteroposterior** ribs and sternum moves away from the spine (20% more)) so pressure in thorax decreases (be careful thorax not abdomen, because pressure in abdomen increases), and alveolar pressure becomes 759 mm Hg \(-1\) cm H2O which is less than atmospheric pressure → now we have pressure gradient and air flows inside

Pressure in alveoli will return to 760 because of recoil forces and now we need to do the expiration, Diaphragm relaxes and volume in thorax deceases → pressure in alveoli becomes 761 and air flow will go from lungs to atmosphere until alveolar pressure gets back to 760 and the cycle continues

*regulation of respiration mainly occurs in respiratory center, but we can control it for short period of time. Like in hyperventilation which occurs in hysteria usually in females, washing out of CO2 and Hydrogen happens leading to alkalosis → breathing stops
In hypoventilation like when you stop breathing voluntary, after some period of time breathing starts, عشان هيك بتقدرش تنتحر لما تحبس نفسك:

Min 27.30\[
*As you studied in anatomy (I hope so) lungs are not isolated, they are elastic structure present in thoracic cage and surrounded by pleura a double layered membrane with space between the 2 layers, the external layer → parietal \(\triangledown\) internal\(\triangledown\) visceral \(\triangledown\) and the space between them is filled with fluid with negative pressure(sub atmospheric) called **pleural cavity** which has at REST pressure of \(-4\) cm H2O or 756 mm Hg

*The mechanism that keeps the lungs inflated mainly is sub atmospheric pressure in the pleural cavity(intrapleural pressure) and substance called surfactants we will talk about it very soon

**Negative pressure means less than 760 mm hg or less than 0 cm H2O**
*as we said pleural cavity is not dry, it has fluid and some kind of Suction that helps the lungs to open at rest, there is continuous fluid formation in the sac\cavity and its sucked\absorbed by lymphatic system, pleural pressure is usually between -4 to -7.5 cm H2O during normal inspiration
*At rest or equilibrium the pleural pressure is -4 cm H2O

When we take inspiration there is squeeze and reduction of pleural cavity and pressure will become more negative→ -7.5 cm H2O, during exercise it becomes More negative

Min 30.30 slide 15-21

Alveolar pressure:
In between the breathing, mouth, nose, trachea and glottis are open so alveolar pressure is equal to atmospheric pressure (760 mmHg) no air flow

Inspiration → pressure is less than atmospheric (759 mmHg) -1 cm H2O \2s
Expiration→ more (761 mmHg) +1 H2O \2-3 s

3- Transpulmonary pressure
→ Pressure difference between the alveolar pr. and pleural pr. [pr.differ. b/w alveoli and outer surfaces of the lungs] left transmural pr. Across Lung wall & it is usually 4 →760 – 756 =4
*mainly regulates inflation, during inspiration this transmural pressure increases and increase significantly during exercise
During expiration it will decrease
**What are the two factors that stops continuous inflation?
1-Intrapleural pressure 2-Thoracic wall and cage

→ it measures elastic forces that tend to collapse the lungs each point of expansion [recoil pressure].
**Transmural pressure gradient across thoracic wall =atmospheric pressure -intrapleural pressure
*see slide 20
more volume → more negative interplural pr. → increased transmural/transpulmonary pressure → more air
العكس صحيح

Min 42:

Pneumothorax (pneumo=air) → so it is basically air in pleural cavity, pressure in pleural cavity will be no more negative, it will be equal to atmospheric pressure and the lungs will be collapsed
(traumatic pneumothorax)
if there is puncture in thoracic wall air will inter the pleural cavity → pressure increase → lung collapses
sometimes we have puncture in both sides (both lungs) → patient usually dies, it is an emergency case
but mostly it occurs in one side, so to treat it we put a tube in pleural cavity and connect it to bottle of water (just like shisha) until the air exits the cavity and then we close the puncture
(spontaneous pneumothorax)
in heavy smoker people some alveoli will be ruptured and the air from them will go to pleural cavity → increases pressure in cavity → lung collapses \ there is no injury because it is internally
 treatment just like in traumatic but we puncture the thorax to put the tube

Min 45:

Compliance of the lungs

→ Expandability of the lungs
→ stretch ability of the lungs.
→ The extent to which the lungs will expand for each unit increase in transpulmonary pr.
→ total compliance of both lungs is around (200 ml/cm H2O transpulmonary pressure)
  - this means if transpulmonary pr. Increases 1 cm H2O it will increase the volume by 200 ml of air
Simply, compliance is how much the lungs are distended\inflated

To make it easy imagine a balloon, a child can inflate it, but sometimes he cant so he need more effort\pressure to inflate it so he asks his parents, if we compare the lungs to balloon, the effort needed to inflate the balloon is 100-fold, this means lungs need little effort to be distended

Significantly affected in disease (smokers, COPD, emphysema....) you will need more effort so compliance decreases.
*There is recoil in lungs after distention to return it to its normal size

****now we will start record #2
There are two compliance: one for the lung itself and one for the thoracic cage (lung + thoracic wall)
As we said we distend lungs very easy, but if we put it in thoracic wall it will limit how much it is distended (compliance is decreased) and over inflation will be stopped by thoracic cage and intrapleural pr.
**The compliance of thoracic cage is 50% of lungs compliance**
*anything that limits inflation of lungs will affect compliance like disease, fibrosis, problems in the chest wall and the surface tension of pleural cavity
slide 26 :
compliance of lungs depends on:
1) elastic forces of the lung tissue (elastin and collagen fibres) (1/3 of the total force)
2) Surface tension of the fluid (2/3 of the total force)

→Surface tension is huge when surfactant is absent: H2O molecules on the surface of the alveoli have an extra strong attraction for one another→ attempting to contract and collapse the alveoli →explained below

Surface tension:
In between the alveoli there are molecules of water and surfactants, if water was present alone it will attract each other and the surface tension will be high and the alveoli will collapse, so the surfactants prevent the water molecules to accumulate and reduces the surface tension and keeps the alveoli open.
*in premature babies there are not enough surfactants and the radius of alveoli is small so their alveoli may collapse (6-8 times greater than in adults) → Respiratory distress syndrome of the newborn, we put them in incubator until they mature

Slide 27 → surfactants: surface-active agent in water secreted by type II alveolar epithelial cells (10% of surface area of alveoli).

Phospholipids, dipalmitoylphosphatidylcholine (DPPC), proteins (apoprotein), and ions (calcium) that help in spreading phospholipids

**see slide 28

*So anything that reduces elasticity or increases tension will decrease compliance ,also pleural effusion (fluid accumulation in pleura) will decrease compliance , anything that limits inflation will decrease compliance.

Min 16.40 slide 29

• Pressure generated by Surface Tension = \(2\times \text{Surface Tension}\ \text{Radius}\)

*The bigger the radius the less pressure generated by surface tension
Effect of size of the alveoli on collapsing pr.:
**The Radius is inversely proportional to collapsing pr. So smaller alveoli have greater pr. than the larger ones.

- Slide 30:-Instability of the alveoli (rupture),
- Safety factors:
  1. Interdependence phenomenon (Sharing septal walls)

2. 50 000 functional units w/ fibrous septa

3. Surfactant effect :
   a) reduces S.T from 8 to 3 cm H2O
   b) [surfactant] in smaller alveoli > than in large

slide 31:
- Compliance of thorax and lungs: 110 ml/cm H2O pr.
  we said for the lungs alone it was 200 ml/cm H2O pr. ,but with the thorax it is almost the half
  for 1 cm increased only 110 ml of air enters
- At high volume or compressed to low volumes, the compliance can be as little as one-fifth
  that of lungs alone

min 20\slide 32
- Work of breathing:
  it is divided into 3 parts:

1. work required to expand the lungs against the lung and chest elastic forces (compliance work)

2. work required to overcome the viscosity of the lung and chest wall (tissue resistance work)

3. work required to overcome airway resistance (airway resistance work)

The more the passageway restricted the more we need efforts and less the compliance→less air entered, the most resistance is in large bronchiole ,, in asthma or bronchitis there is constriction so the resistance is increased so need more effort

*anything that increases effort in one of three parts needs more work

*the surface tension in small alveoli is higher
slide 33:

*as we see from the figure the main effort is for the compliance (as the process for inflation of the lung)

*the effort in the tissue resistance work and airway resistance work is for the inspiration process

*the minus sign in the x-axis mean the change in the pressure not the absolute pressure there

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### #the lung volumes and lung capacities

Is how much the lung inflated, how much the air get in and out, how mush air stay in our lung after breathing and so on....

*we do this test to assay the function of the lung and determine the type of disease (obstruction or restriction disease)

*obstruction disease: it’s the obstruct the airway >>difficult to get out

*restriction disease: is the limitation of inflation

>>The volumes

1) Tidal Volume (TV)

The volume of air that we inspire or expire during a resting state with a normal quiet breathing (about 0.5 liter)

2) Inspiratory reserve volume (IRV)

It is the volume of air person can inspire after TV

3) Expiratory reserve volume (ERV)

It is the volume of air person can expire after TV

4) Functional residual volume (FRV)

The volume of the air remain in the lung after normal expiration

5) Residual volume (RV)

The minimum volume of air present in the lung after maximum expiration (as we can’t expire all the air in our lungs) and it’s about 1.2 liter

>> The capacities

1) Vital capacity
It is the maximum amount of air a person can expire it after a maximum inspiration >> its about 5 liter in female and 6 liter in male

Equal (total lung volume – RV)

2) Inspiratory capacity >we can also say excitatory capacity
It is the maximum amount of air a person can inspire

Inspiratory capacity = TV + IRV

3) Total lung volume
It is the amount of air present in the lungs after maximum inspiration

Total lung volume = RV+TV+IRV+ERV

4) Force expiratory volume (FEV1)
* is the volume of air that can forcibly be blown out in one second (first second)
If we take the ratio of how we much expire
*it is very important in diagnosis because .....FEV1 when divided by vital capacity (the result must be more than 70%)
** If less than 70 it’s indicate disease and have grade or level as this
- 50%-70% mild disease
- 40%-50% moderate disease
- less than 30% severe disease

The following slide the doctor don’t read it....

Slide 34

Minute respiratory volume :

Total volume of new air moved into respiratory passages each minute
MRV=TV * freq.
Normal = 500 x 12 = 6L/min
(1.5 L/min fatal). ( high value like 200 L/min is fatal).

- Alveolar ventilation :
  rate at which new air reaches these
  areas (respir. spaces).
  (TV – D.S)* freq. = 4.2L/min
• Respiratory passageway:
  • 1-Main resistance to the airflow present in
  • Large bronchioles and bronchi
  • 2-Sympathetic system dilate bronchioles
  • 3-Parasympathetic system constrict bronchioles
  • 4-Irritation of membrane passageways cause constriction as (smoking, dust, Infection)
  • 5- Histamine and slow reactive substance of anaphylaxis secrete locally by the lungs
  • By mast cells during allergic reaction as in
  • Asthma. These cause bronchiolar constriction
  • 6-Atropine relax respiratory passageway.

Good luck