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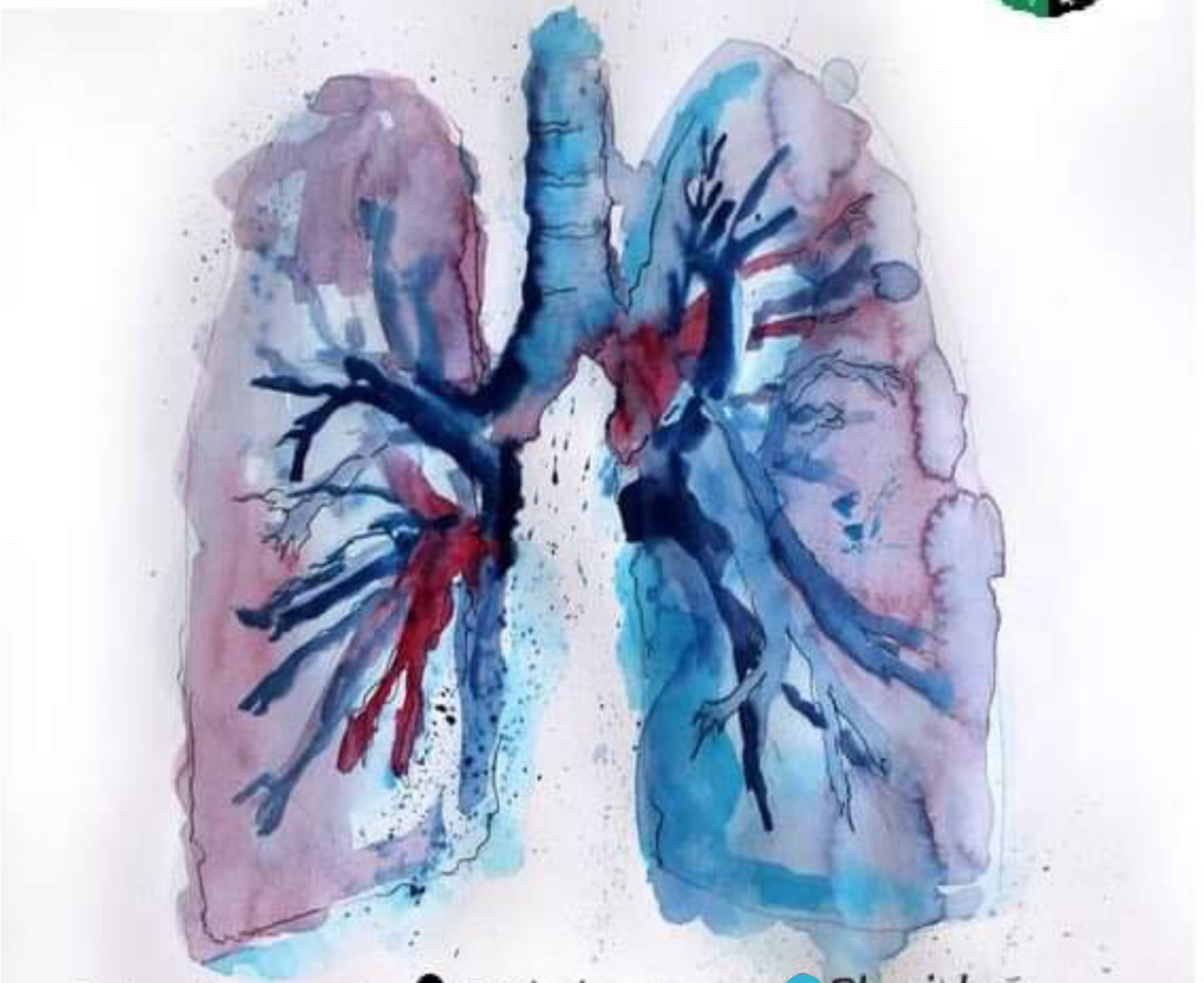

● Anatomy ● Pathology ✓ Physiology

Lecture: Physiology 4 Date: 5/11

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Edited BY : Razan A aburashed

-editor's note : all info from slides were aded "mostly in blue squares",though information might be added in different order to make understanding much easier .

-don't forget to check the sheet correction link: bit.ly/rsphysio

***Review of the previous lecture:**

Imagine that the lungs have 3 parts(physiologically they only have 2).

The blood flow in the lung is affected by both pulmonary arterial pressure and hydrostatic pressure.

In the normal upright position ,the difference between the upper and lower part of the lung in hydrostatic pressure (gravity) = 23mmHg ((hydrostatic pressure from above the heart=15mmHg ,below the heart=8mmHg)).

-The other force is pulmonary arterial pressure (systole,diastole):

During systole: the force that keep the alveolar capillary open is equal to 10mmhg (25-15=10)(**25** is pulmonary arterial systolic pressure ,**15** is hydrostatic pressure form above the heart) so there is blood flow because the systolic pressure is more than the hydrostatic pressure)

During diastole: the pulmonary arterial diastolic pressure =8mmHg so its not sufficient to overcome the 15mmHg hydrostatic pressure so the capillaries stay closed there is no blood flow because the pressure that is present in the pulmonary artery is less than hydrostatic pressure ,so there will be an intermittent blood flow (not zero).

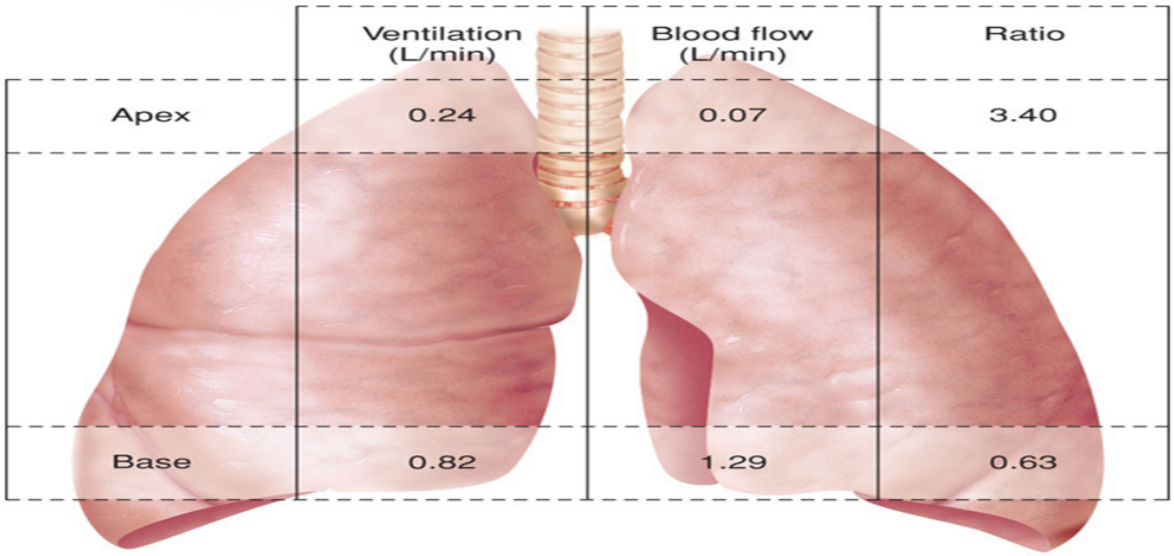
*****As a summary:***

Anything below the heart will have a continuous blood flow and anything above will have intermittent blood flow and the more we go up the more reduction in blood flow.

Any time the pulmonary arterial pressure is more than the hydrostatic pressure there will be blood flow and when pulmonary arterial pressure is less than hydrostatic pressure there will be no flow.

We use the heart as a zero point because during diastole there is no contraction but there is a pulmonary arterial pressure.

In supine position (laying dawn) the hydrostatic effect(pressure) is homogenous in all parts of the lung so all the area of the lung will be perfused with blood (both during systole ,diastole) bcz the pulmonary arterial pressure is higher than hydrostatic pressure)



Pulmonary capillary dynamics

The pulmonary capillaries in the alveolar membrane are side by side to form a sheet in which the blood flow as a sheet of flow rather than individual capillaries

-All the capillaries act as a one sheet so the blood will be distributed all over it.

Mean Pulmonary capillary pressure is 7mmHg which is between mean LF atrial pressure (2) and mean pulmonary arterial pressure (15).

-The mean systemic capillary pressure =17.

In normal cardiac output the blood passes through the pulmonary capillaries in about 0.8 sec but when the cardiac output increases this time can be as short as 0.3 sec.
This shortening develops because opening of capillaries that were normally collapse so can accommodate the increased blood flow.

-The duration of blood which is passing through pulmonary capillaries during rest its 0.8 sec (very short time) but still it's enough to cause full saturation of blood because the area of the capillaries is very large , during exercise(the area significantly increase) so the duration of exposing blood to oxygenation significantly decrease reaching 0.3 sec.

The dynamic of fluid exchange across the lungs capillary membrane are qualitatively the same as for peripheral tissues. But quantitatively are different. The differences are:

1- Pulmonary capillary pressure =7→ in systemic= 17mmHg

2- Interstitial fluid pressure -8→in systemic= -3 mmHg

3- Colloid osmotic pressure of pulmonary interstitial fluid=14→ in systemic =8 mmHg.
4- The alveolar wall is extremely thin. (so, the distance will be short)

-As in systemic circulation we have fluid exchange between capillaries and interstitial space with same forces but different quantities.

	lungs	Systemic cir.
Capillary pressure	7 mmHg	17 mmHg
Interst. Osm. Pr.	14 mmHg	8 mmHg
Interst. Neg. pr.	-8 mmHg	-3 mmHg

Outward Forces

- Pulmonary capillary pressure 7 mmHg
- Interstitial osmotic pressure 14 mmHg
- Negative interstitial pressure 8 mmHg
- Total 29 mmHg

Inward Forces

- Plasma osmotic pressure 28 mmHg

Net filtration pressure **1 mmHg (handled by lymph)**

- **Negative interstitial pressure keeps alveoli dry by sucking any extra fluid appears in the alveoli through small opening between alveolar epithelial cells. This excess fluid then carried out by pulmonary lymphatics**
- Pulmonary capillary dynamics:

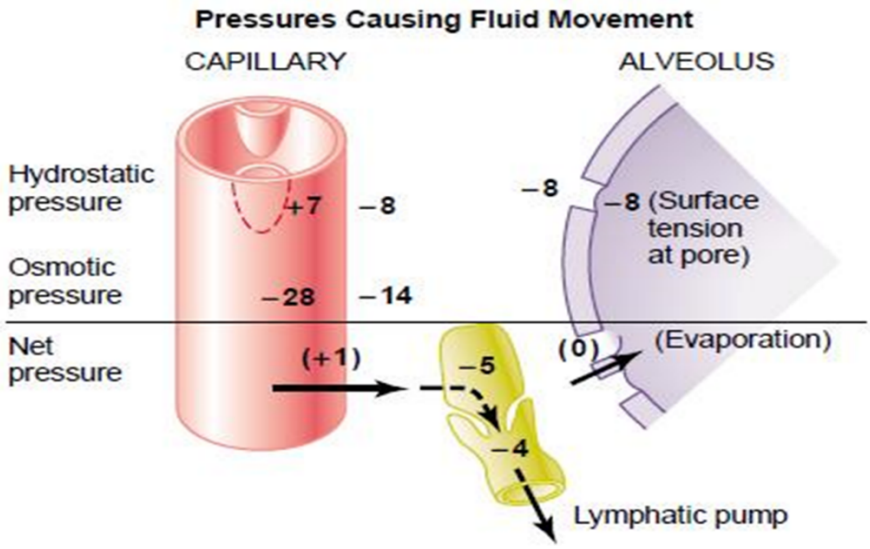
→ 7 mmHg cap. Pressure.

→ blood passes through the capillary in 0.8sec

→ increasing the C.O lowers the time to 0.3sec

-The net filtration =1mmHg so more blood will be coming out from pulmonary capillaries to interstitial and this is handled by lymphatics (in systemic its 3mmHg).

-The negative interstitial pressure is one of the main factors that keep the alveoli dry (it prevents fluid accumulation in the alveoli) in the presence of lymphatics.



Pulmonary edema

As anywhere in the body, Any factor that increase fluid filtration out of pulmonary capillaries or that impedes pulmonary lymphatic function and cause the pulmonary interstitial fluid pressure to become positive(no sucking factor) this will cause filling of pulmonary interstitial space and alveoli with large amount of free fluid.

Alveoli are always dry except for a small amount of fluid secreted by alveolar cells on the alveolar surface.

- **Causes:**

1. **Left-sided heart failure**→ ↑ venous + cap. Pressure

-left ventricle cant pump blood so it accumulates there increasing the pressure in the left ventricle (both end-diastolic volume and end-diastolic pressure increase) so the pressure difference between left ventricle and left atrium will be reduced (blood will not pass form left atrium to left ventricle leading to blood accumulation in left atrium increasing its pressure , now the pulmonary circulation need to build up pressure to be more than the left atrium (pulmonary capillary pressure increase leading to increase in the filtration leading to fluid accumulation in interstitial (interstitial pressure gradually increase till it become positive leading to accumulation of fluid in alveoli (no sucking) ,the lymphatic system will adapt(expand) trying to protect the alveoli until it reach its maximum ability then it will be uncap-able to take more fluid leading to more fluid accumulation.

-People with left heart failure they won't develop edema right away (it develops gradually) the patent will start complaining of dyspnea (difficulty in breathing) because the surface area for exchange will be less.

- GRADING of the disease upon the effort the patient can make (it's done for angina and dyspnea):

- **Stage zero:** When the patient can climb 2 floors either he is normal or have very mild dyspnea/angina
- **Stage one:** if the patient can't 2 floors (he climbs one floor then take rest).
- **Stage two**: if the patient can't climb even one floor (he climbs half of the floor then take a rest) developing chest pain in case of angina and dyspnea in case of heart failure.
- **Stage three(last):** he can't even climb half floor, he merely can do routine activities inside the house (going to bath for example) he only an do very minimal effort it depends on how much fluid there is in dyspnea and how much stenosis in coronaries in angina.

-when the patient develop MI he is either in cariogenic shock or very severe heart failure or sever hypotension (left ventricle cant pump blood leading to sudden decrease in ejection fraction and decrease of left ventricle contractility leading to sudden increase in pulmonary capillary pressure increase in filtration causing *acute pulmonary edema* (its associated with *sever transmural MI*) we mostly lose the patient within 2 min if we don't reach the hospital in the right time :'(.

2-Damage to the pulmonary capillary membrane caused by:

A- infections (pneumonia) B- breathing chlorine gas or sulfur dioxide gas. (affecting the exchange)

-The alveoli in lung increase the surface area of the exchange((if the lung is one area like a balloon the surface are will be small so each alveoli is like an individual balloon with its own surface area increasing total surface area of exchange so anything that effect the alveoli membrane it affect the exchange .

Pulmonary edema safety factors

Pulmonary edema will occur when the pulmonary capillary pressure rises to a value at least equal to colloid osmotic pressure inside the capillaries or more. Keep in mind that any

increase in left atrial pressure, pulmonary capillary pressure must increase at least 1-2mmHg greater to keep the flow going until it reaches to osmotic pressure.

So pulmonary capillary pressure must increase from the normal value of 7 to 28 (21) mmHg the edema will start. This is in acute cases. In chronic cases when the capillary pressure remains elevated for at least 2 weeks, the lungs become more resistance to edema because expand of pulmonary vessels to carry additional fluid accumulated. Thus, in mitral stenosis pulmonary capillary pressure can rise to 40-45 mmHg before pulmonary edema develop

-The forces outward=29 , inward=28 , the pulmonary capillary pressure=7 , colloid osmotic pressure=28 so to reach to the level that the pulmonary capillaries can't absorb fluids it have to be equilibrium to 28 t, (28-7=21)so this how much the pulmonary capillary pressure have to be build up (to reach 28) for edema to develop ,so the safety factor for developing *acute pulmonary edema* in sudden MI is a sudden increase by 21 in pulmonary capillary pressure) the other factors(lymphatics ,interstitial, colloid)are not involved because they gradually build up not *suddenly(acutely)*.**((acute safety factor against pulmonary edema=21mmHg))**

-In *chronic cases* (a patient with cardiac myopathy or mitral stenosis) there is a *gradual* buildup of interstitial pressure and it takes very long time so left arterial pressure can reach 35mmHg before edema can develop. **((chronic safety factor against pulmonary edema=35mmHg))**

-Left arterial pressure can be measured by measuring pulmonary wedge pressure.

-the pulmonary capillary pressure must always be more than left arterial pressure to keep flow until it reaches the level of osmotic pressure.

- Treatment of edema is a very large dose of diuretic ***200-300mg of Lasix*** (if u need 10-20 minute to reach the hospital u'll mostly lose the patient :'(.

-Pulmonary edema experiments are done on animals(dogs) so these figures are for animals and the colloid osmotic pressure in animals=25 mmHg.

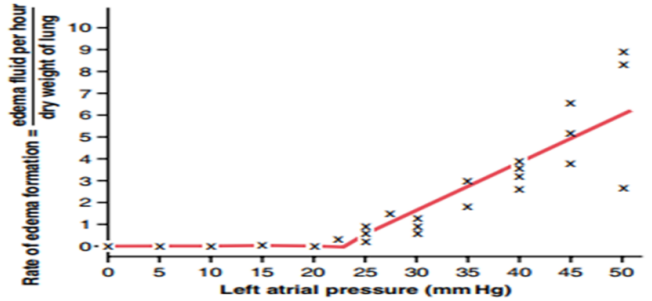


Figure 38-7 Rate of fluid loss into the lung tissues when the left atrial pressure (and pulmonary capillary pressure) is increased. (From Guyton AC, Lindsey AW: Effect of elevated left atrial pressure and decreased plasma protein concentration on the development of pulmonary edema. Circ Res 7:649, 1959.)

Plural effusion

-It’s accumulation of fluids in pleural cavity (edema of the pleural cavity)

-Pleural effusion (edema): Collection of large free fluid in the pleural space it has many causes including:

1- Blockage of lymphatic drainage from pleural space

2- Cardiac failure (increase peripheral and pulmonary cap pressure)

3-Decrease plasma colloid osmotic pressure(it’s a sucking force for fluids from the interstitial , people in poor countries who have malnutrition (they don’t eat enough proteins) the fluid will accumulate in interstitial causing generalized edema (a very good sign of that is accumulation of fluid in the abdomen (ascites) and accumulation of fluid in peritoneal cavity.

4- Infection or inflammation of the surface of pleural cavity (Tb, pneumonia infection leading to blockage in the lymphatics >>no drainage from pleural space>>accumulating fluids >>effusion)

-we have potential spaces that exist in joints, cardiac space, pleural space, peritoneal, mostly there pressure is negative and that’s important (for example if it’s not negative in the knee the tibia and femur will be have direct contact with each other leading to friction thus making it hard to move).

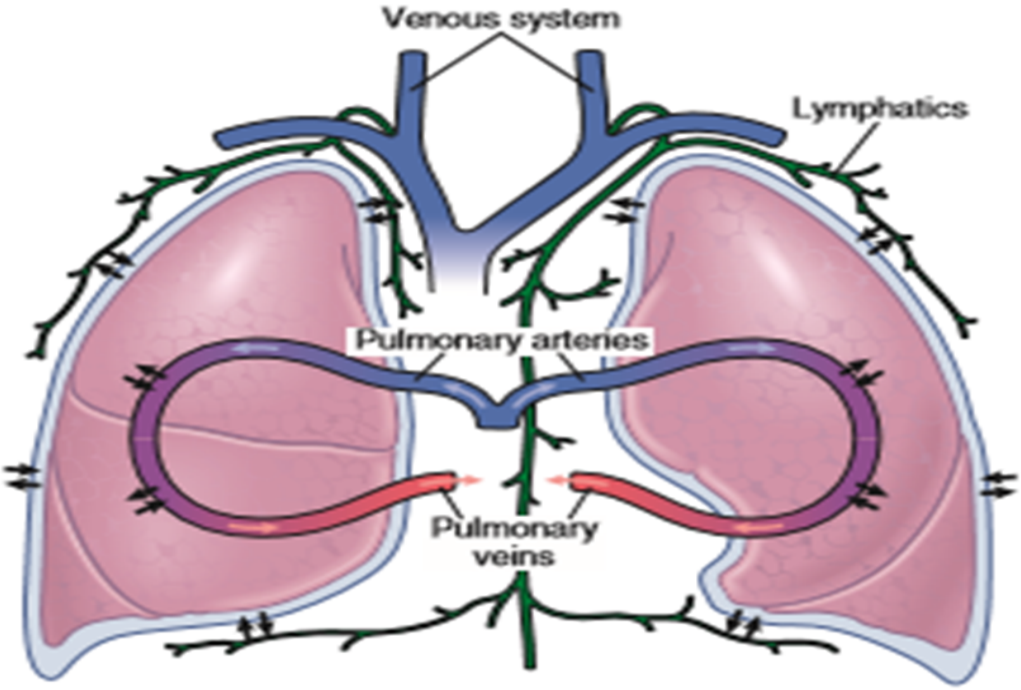


Figure 39-8. Dynamics of fluid exchange in the intrapleural space.