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Lec: Pulmonary circulation
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**Note:** you will find a lot of information from cardiovascular system; so I will add many details about CVS, to explain many points regarding our lecture.

I will do my best for covering all the information in this big lecture;;

so sorry in any mistake You may find it

**the doctor started with these preview about last lec**

--keep in your mind DSV mainly a constant value 150 ml

so if you breath 1500 ml there is a 150ml DSP (more air reach alveoli)

IF you breath 200ml (like rapid breathing) there is a 150ml also DSP (very less air in alveoli) people with this case must be in ICU

--although expiration mainly its passive process (no need for energy); but still you find some resistance in a process of exit of the air

mainly it comes from bronchi and large broncholes

becuase as we know from histology these structures have a muscles >> these muscles can resist exit of air

unlike small bronchioles which not have a muscles and do not resist expiration.

--sympathetic nervous system --- bronchodilation (cause you need more air in state of work and stress)

patient with asthma because of obstruction of air way you can give him adrenaline >> bronchodilation

--parasympathetic --- bronchoconstriction (no need for more air)

--Histamine -- vasodilation – bronchoconstrction

--Atropine relax respiratory passageway.

**Pulmonary circulation**

bronchial arteries after supplying lung tissue >> their blood becomes deoxygenated and drains in 2 way

some of the blood will go th bronchial veins and then to azygos vein until SVC (This is customary)

And others blood will drain into pulmonary veins---L.atrium ---L.ventricle

this thing will affect Cardiac output in left ventricle (by increasing it 1-2% in more than R.ventricle)
and will affect saturation of O2 in blood of pulmonary veins (cause mixing BW oxygenated and deoxygenated blood)

**the Artery is 5cm thin (1/3 of aorta diameter), while the branches are short and have more diameter so it will have a large compliance (7 ml/mmHg).**

**pulmonary veins are thin & short.**

**bronchial arteries originate from the systemic circulation (carry 1-2% of C.O - oxygenated blood) then empty into pulmonary veins then to the left atrium so the left ventricle pumps 1-2% more than the right ventricle.**

**rich lymphatic drainage to the right thoracic duct to prevent edema**

*Pressures in the Pulmonary Systems*

- **Right Ventricle**: The systolic pressure of the right ventricle about **25 mmHg** whilst the diastolic **about 0 to 1 mmHg**; values that are only one-fifth those of the left ventricle. The low diastolic pressure is actually normal since it is required for blood to flow into the ventricles from the atria.

pulmonary artery: During systole, the pressure in the pulmonary artery is equal to the pressure in the right ventricle (cause they connect with each other). The diastolic pressure is about **8 mmHg** << The mean pressure is equal to **15 mmHg**.

pulmonary capillary: The mean pulmonary capillary pressure is about **7 mmHg** which is less than that of the systemic capillaries that has a systolic pressure of **30 mmHg** and diastolic pressure of **10 mmHg**.

The mean pressure in the left atrium and the major pulmonary veins averages about **2 mmHg**

As you can not remember from cvs ☹️ ;;You able to calculate mean blood pressure by:

\[
(2 \times \text{diastolic.P} + \text{Systolic.P})/3
\]

-So as we see here the pulmonary circulation is a low pressure system compared to the systemic circulation. This is due to a shorter distance and a lower resistance present in pulmonary circulation

Blood volume of the lungs:

450ml of blood present in lungs or pulmonary circulation thats equal to 9% of total blood volume.

from these 450ml >> 75ml found in capillaries
That’s pic explain to you a large difference of pressure bw left ventricle and aorta (systemic pressure) with right ventricle and pulmonary arteries (pulmonary pressure)

From this pic you can find

- Systole and diastole pressure of aorta and l.ventricle 120—80
- Systole and diastole P of pulmonary artery 25—8
- Systol and diastole P of R.ventricle 25—near to zero

-again another pic show you pressure in pulmonary artery;; vein and capillary (left atrium the same of pulmonary veins)
- mean blood pressure of heart =100 mmhg

**Blood volume of the lungs**
the lung containing as we said 450 ml of blood and acting as reservoir of blood .. How??
in a case of hypotension like bleeding the lung will shifted more than half of their own blood (250ml) to support systemic circulation
at the case of left heart failure the volume of blood in lungs will reach 900ml
((Left-sided heart failure occurs when the left ventricle doesn't pump efficiently. This prevents your body from getting enough oxygen-rich blood. The blood backs up into your lungs instead, which causes shortness of breath and a buildup of fluid))

Fick principle

give us cardiac output by measuring oxygen consumption by knowing O2 content in venous side and arterial side and using these equation

\[ Vo2 = Q(Ca_o2 - Cv_o2) \]

where

- \( Vo2 \): oxygen consumption
- \( CaO2 \): conc.of O2 in artery
- \( CvO2 \): conc of O2 in vein
- \( Q \): blood flow

from where you can take a blood

-to have a good mixed venous blood take it from "pulmonary artery"

-for the arterial blood dose not matter from where you take the sample because the blood comes from pulmonary veins and go to l.ventricle (the same o2 content) and go to aorta the same >> to any artery of the body still the same until reach the capillary it will be changing

**Blood flow:**

--when there is hypoxia in systemic circulation >> Must be accompanied with vasodilation (Even to compensate for the lack of oxygen)

but whats happen in pulmonary circulation its opposite  why???

the blood reaching alveoli reduced with O2 ;;so that will lead to vasoconstriction IN PULMONARY CIRCULATION << in order to transport blood from non ventilated alveoli to ventilated one

cause the blood going to alveoli must be undergo exchanging

so if you have hypoxia >> blood going to alveoli reduced that will lead to vasoconstriction for proper ventilation area

(i hope its become clear)

why thats happen

maybe because of vasoconstrictor substances

**rec. min 24) hydrostatic pressure**

: The pressure exerted by a fluid at equilibrium at a given point within the fluid, due to the force of gravity. Hydrostatic pressure increases in proportion to depth measured from the surface because of the increasing weight of fluid exerting downward force from above.

in the R.ventricle systolic pressure 25mmhg go up it will decrease

go down it will increase
thats the effect of hydrostatic pressure.

blood flow during heavy exercise:

- cardiac output increase >> open more capillary in pulmonary circulation << more blood coming here (extra flow)
- these extra flow from where coming ??
  - increase num of open capillary
  - distending all capillary and increasing flow rate
  - increasing pulmonary arterial pressure

- observe that normal cardiac output 5L ;And the mean pulmonary blood pressure 15 mmhg.
- at the case of heavy exercise you will able to see more CO and more mean blood pressure.

(Rec. min32) Regional pulmonary blood flow :

we can divide lung to 3 region
- zone1 (our lung did not have this zone)
- blood pressure in capillary less than alvoeli so that will lead to blockage of flow
- zone2
  - during systole capillary pressure more than alveolar pressure
so there is a flow here; but this flow not continuous cause in diastole >> alveolar pressure will be again more than capillary pressure so there is no flow here and we call it 
(intermittent flow)

-zone 3

always capillary pressure more than alveolar pressure so there is a continuous flow here during exercise what will happen

upper part of lung increasing with blood supply 7-8 fold than rest state; whereas lower part of lung increasing only with 2-3 fold << that because the opening of pulmonary capillary and hydrostatic effect

Regional pulmonary blood flow: (slides)

arterial pressure in pulmonary artery is 25-8 mmHg

at apex systolic 25-15= 10 mmHg
during diastole 8-15 mmHg= -7?

(Hydrostatic pressure difference is 15 mmHg between the level of the heart and the lung apex)

if the person is lying down or exercising only zone 3 can be seen.

(Rec. min38) Pulmonary capillary dynamics:

blood which going from heart to pulmonary capillary its take a time

this time mainly 0.8 sec to reach pulmonary capillary

in the case of exercise this time will be less and reach to 0.3 sec.

** these is comparison of capillary in lung and systemic circulation

but before start let’s remember these terms
- interstitial osmotic pressure: (some protein leakage to interstitial space >> these leakage will lead to interstitial osmotic pressure)

- interstitial negative pressure: fluid (not protein) that leakage or going into interstitial space

**lung**

<table>
<thead>
<tr>
<th>Capillary Pressure</th>
<th>7 mmHg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Interstitial Osmotic Pressure</td>
<td>14 mmHg</td>
</tr>
<tr>
<td>Interstitial Negative Pressure</td>
<td>-8 mmHg</td>
</tr>
</tbody>
</table>

**systemic circulation**

<table>
<thead>
<tr>
<th>Capillary Pressure</th>
<th>17 mmHg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Interstitial Osmotic Pressure</td>
<td>8 mmHg</td>
</tr>
<tr>
<td>Interstitial Negative Pressure</td>
<td>-3 mmHg</td>
</tr>
</tbody>
</table>

-- Pulmonary Capillary Dynamics

**Outward Forces**

- Pulmonary capillary pressure 7 mmHg
- Interstitial osmotic pressure 14 mmHg
- Negative interstitial pressure 8 mmHg
- Total 25 mmHg

**Inward Forces**

- Plasma osmotic pressure 28 mmHg
- Net filtration pressure 1 mmHg (that will lead to little more comes out than in >> it handle by lymphatic system)
- Negative interstitial pressure keeps alveoli dry

**(rec. min44) Pulmonary edema**

- is a condition caused by excess fluid in the lungs. This fluid collects in the numerous air sacs in the lungs, making it difficult to breathe. In most cases, heart problems cause pulmonary edema

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

-When interstitial pressure becomes (+) water will fill the alveoli
so
capillary pressure increase << outward forces increase << interstitial fluid increase that will lead to edema

when pressure gradual increase in L.atrium --L.A can not Receive more blood so the blood will stay in pulmonary circulation -- lead to increase pressure in capillary -- pulmonary edema

so as we see its mainly caused when pressure of L.A more than pressure in colloid pressure that will lead to prevent outflow and inflow bw capillary and interstitial >> pulmonary edema

other causes

-Damage to the pulmonary capillary membrane caused by :
  a- infections
  b- breathing chlorine gas or sulfur dioxide gas.

safety factors

21 mmHg in acute states

35 mmHg in chronic cases

in acute one mainly its come from myocardial infarction that will lead to less ejection fraction << less Cardiac output << more left atrial pressure leading to pulmonary edema as we know

and the saftey factor here is atrial pressure less than 21 mmhg

in chronic state there is gradual increase in saftey factor

that will reach to 35mmhg thses increase coming from lymphatic system(because lymphatic system can remove fluid)
Rate of fluid loss into the lung tissues when the left atrial pressure (and pulmonary capillary pressure) is increased

look at this pic you will see when atrial pressure begin to increase more than 25 mmhg pulmonary edema start to develop

(rec min53) ended

Now rec2 to complete with Pleural effusion (edema)

Pleural effusion or pleural edema
-accumulation of fluid in pleural cavity that will lead to edema
-normally there is no fluid in this cavity "very little amount"
any fluid comes here it will clean by lymphatic system
-pleural effusion takes time to happen so its chronic not acute
there are many reasons for pleural effusion
-Blockage of lymphatic drainage from pleural space
2- Cardiac failure (increase cap pressure)
3- Decrease plasma colloid osmotic pressure
4- Infection or inflammation of the surface of pleural cavity
the most common one comes from tuberculosis infection
our lecture has ended
best of luck for you