Physiology of the Respiratory system Lecture 2

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Elastic properties of the lung (surfactant& compliance)

- The elastic properties of the lung is caused by:-
- **A.** elastic fibers in the tissue
- **B.** surface tension of the fluid in alveoli
- These act against inflation of the lung.

- For ventilation to take place, it should overcome these two causes. However, the two causes are affected by many factors. These are as follow:

1.Surfactant

- ✓A phospholipid produced by type II alveolar cells.
- ✓Act to reduce surface tension of water, separating it from air (because the surface tension occurs at the water/ air interface).
- ✓The surface tension is physical property of fluids. It arises because the cohesive forces between water molecules attract each other, tending to contract their surface& eventually cause alveolar collapse.

Reduction of surface tension prevent:-

- ✓ **1.** Alveolar collapse.
- ✓ **2.** Development of the pulmonary edema (due to negative interstitial pressure caused by alveolar collapse).
- ✓ The presence of the surface tension in the lung was first noticed when air& saline were compared during inflation of excised lung.
- ✓ Inflating lung with saline was found to be easier than inflating them with air.

- ✓ This is because there is no surface tension acting against inflation when saline was used.
- ✓ Surfactant effects are mainly exerted on small alveoli; especially during expiration.
- ✓ This is because these small alveoli have higher tendency to collapse.
- ✓ The higher tendency of small alveoli to collapse can be explained by **law of Laplace**.
- ✓ P= 2T/r

- P= pressure inside alveoli (distending pressure) .
- T= Tension
- r= radius of alveoli

- ✓ This indicates that the smaller the radius the higher distending pressure needed to keep it patent.
- ✓ Production of surfactant start late in pregnancy (after the 32 weeks of pregnancy). Therefor it is deficient in preterm babies.
- ✓ These babies develop cyanosis& difficulty in breathing at birth. A serious condition known as infant respiratory distress syndrome (IRDS) or (hyaline membrane disease); characterized by collapse of alveoli& retention of fluid in interstitial& alveoli.

- ✓ Retention of fluid in alveoli occurs because the surfactant is needed for maturation **of *epithelial sodium channels* (ENaC)** responsible for absorption of sodium& water from the alveoli after birth. **Failure of maturation of these channels due to deficiency of surfactant results in fluid retention.**

- ✓ **Treatment** of (IRDS) requires in addition to higher O_2 supply& fluid balance, inhalation of phospholipid or synthetic surfactant.
- ✓ Surfactant production is increased by glucocorticoids (this is why pregnant women who develop premature labor contraction given injection of hydrocortisone; to increase its production).

✓Surfactant production is decreased by:-

All of them cause (damage of type II cells by hypoxia)

1. Occlusion of pulmonary artery.
2. Occlusion of the main bronchus.
3. Chronic inflation of (100%) oxygen. (the lung is the most vulnerable target for oxygen toxicity at high pressure and concentration)
4. Cigarette smoking.

2. Lung compliance:_

- ✓ It's described as dispensability or stretchibility of the lung (i.e. the **capacity of the lung to expand or stretch**).
- ✓ Also compliance can be defined as **change in the volume per unit change in the pressure**.

- ✓ Compliance differs from elasticity which is the resistance to that stretch (i.e. compliance= 1/elasticity).
- ✓ Therefore when elasticity is decreased the compliance is increased.
- ✓ It's measured in terms **change in the volume per unit change in the pressure**

(i.e C= ∆V/∆P).

- ✓ **Normal values of compliance:-**

A. Compliance of the lung (CL) = 0.2L/cmH_2O.

B. Compliance of the chest (CC) =0.2L/cmH_2O.

C. Compliance of both (CI& CC) =0.1L/cmH_2O.

- ✓ **Factors that increase lung compliance:-**are Emphysema& old age (due to loss of elastic fibers in the lung).
- ✓ **Factors that decrease lung compliance:-** are lung fibrosis, pulmonary edema, high surface tension of fluid in alveoli (surfactant deficiency)& small lung size (in children).

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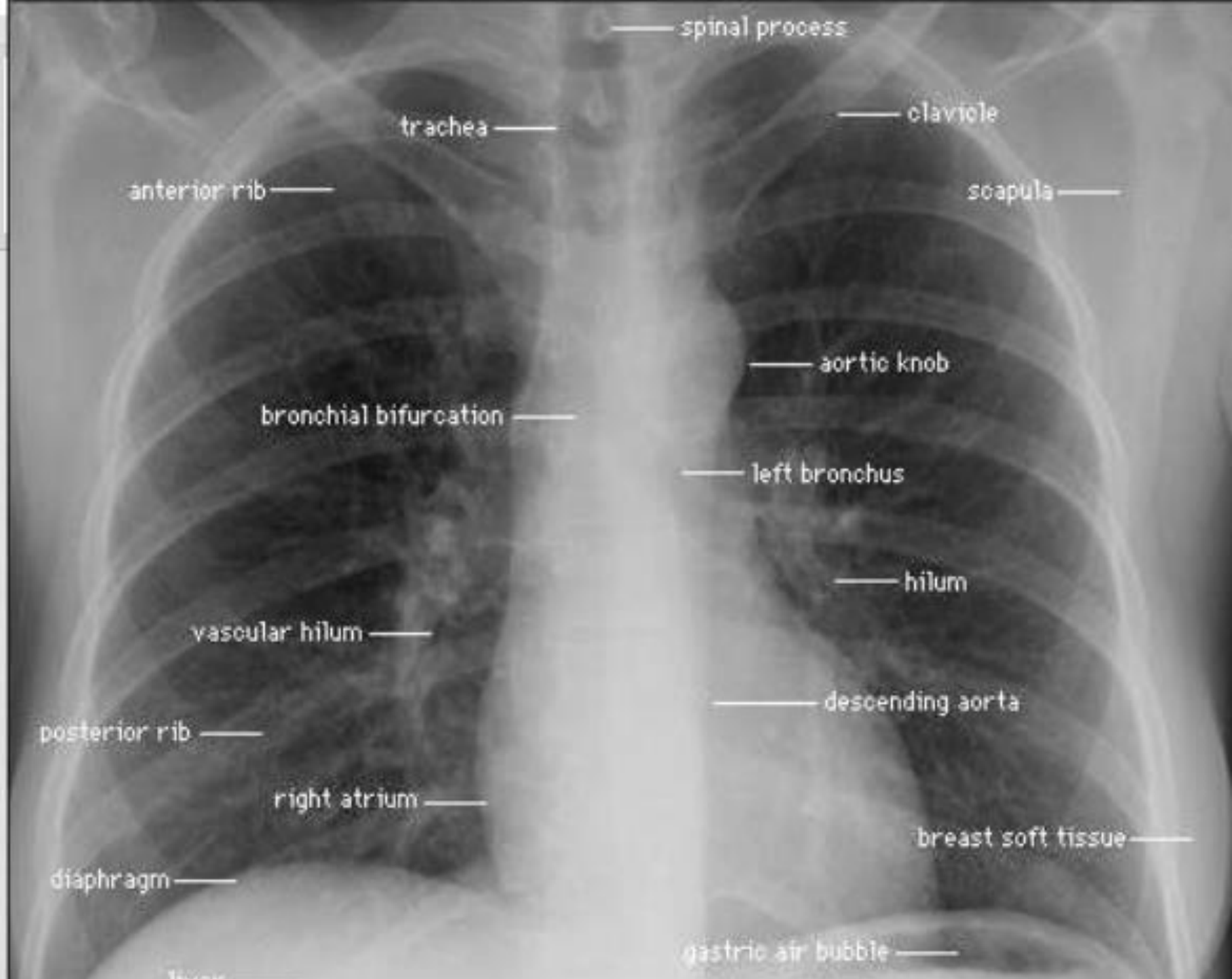
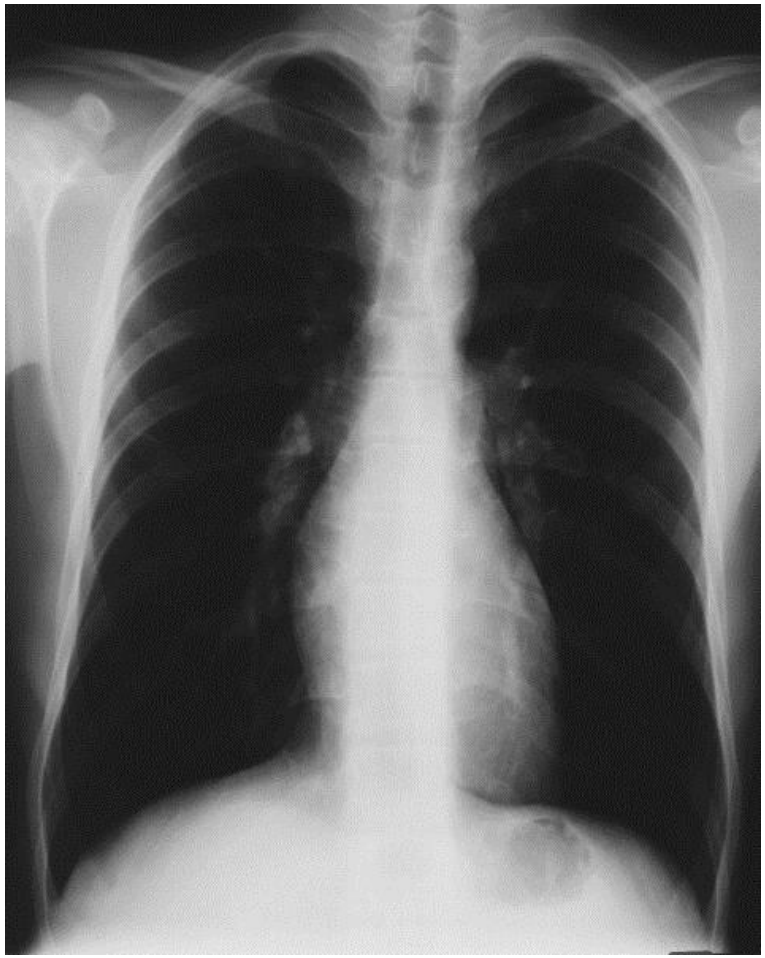

spinal process
trachea
clavicle
anterior rib
scapula
aortic knob
bronchial bifurcation
left bronchus
hilum
vascular hilum
descending aorta
posterior rib
right atrium
breast soft tissue
diaphragm
gastric air bubble

Emphysema



Lung fibrosis

Pulmonary oedema

- ✓ Remember that high lung compliance decrease work of breathing whereas low lung compliance increase work of breathing.
- ✓ Remember that surfactant decrease work of breathing.

Bronchial tone

- The smooth muscles in the bronchial wall are controlled by the autonomic nervous system.
- The sympathetic dilate it (e.g during inspiration) & the parasympathetic constrict it (during expiration).
- However there are multiple irritant, chemical, & hormone that may affect the normal tone of the bronchial tree.

These include:-

1. Factors that cause bronchial constriction:-

I. Irritants& chemicals(e.g. sulfur dioxide).

II. Cool air.

III. Exercise (possibly by the cool air during hyper-ventilation).

IV. Substance P (It is a neuropeptide, acting as a neurotransmitter and as a neuromodulator)

V. Adenosine (Drug)

VI. Many inflammatory modulator& cytokines involved in pathogenesis of asthma (e.g. leukotriene); that is why anti leukotriene's are added for treatment of asthma.

- **Cytokines** are a broad category of small proteins that are important in cell signaling.
- Their release has an effect on the behavior of cells around them.
- It can be said that cytokines are involved in autocrine signalling, paracrine signalling and endocrine signalling as immunomodulating agents.

Factors that cause bronchodilatation:-

I. Catecholamine (Adrenaline and noradrenaline)

II.VIP (vasoactive intestinal peptide).

There is circadian rhythm in bronchial tree tone throughout the day; with maximum constriction early in the morning. That is why asthmatic patients usually suffer from symptoms of air way obstruction early in the morning.

Gas exchange in the lungs

- **There is two sites of gas exchange in the body:-**

1) Between alveoli& pulmonary capillaries (in the lungs).
2) Between tissue cells& systemic capillaries (in the tissues).

- **Gas exchange in the lungs depends on the following factors:-**

1) Pressure gradient of the gas.
2) Surface area of the respiratory membrane.
3) Thickness of the respiratory membrane.
4) Physical properties of the gas.

1) Pressure gradient

- Gasses moved passively from the area of high pressure to area of low pressure.
- The pressure of single gas in a container containing mixture of gasses is called its **partial pressure.**

- The partial pressure of gas is calculated by multiplying its fractional concentration times the total pressure of all gasses.

- The following explain calculation of partial pressures of gasses in atmosphere (dry air).

gas	Percentage	Partial pressure
Nitrogen	78.06	78.06×760=593.3mmHg
Oxygen	20.98	20.98×760=159.4mmHg
Carbone dioxide	0.04	0.04×760=0.3mmHg
Inert gas	0.92	0.92×760=7mmHg
Total	100	760mmHg

➢ Partial pressure of oxygen (PO_2)

- In dry air 159mmHg
- In inspired air (after humidification in the air ways) =149mmHg
- 20.98 %×(760_47); PH2O =47mmHG at the body temperature.
- In alveolar air= 100mmHg (due to rapid diffusion of O_2 into pulmonary capillaries& diffusion of CO_2 into alveoli).
- In venous blood coming to pulmonary capillaries = 40mmHg (Prior to gas exchange).
- Po_2 in arterial blood (leaving pulmonary capillaries) =100mmHg (after the gas exchange; however this value is decreased by the physiological shunt).

➢ Partial pressure of CO_2

- In dry air 0.3mmHg
- In inspired air 0.29mmHg {0.04×(760_47)}. In alveolar air 40mmHg (due to rapid diffusion of CO_2 from pulmonary capillaries to alveoli).
- In venous blood (coming to pulmonary capillaries) 45mmHg (prior to exchange)
- Pco_2 in arterial blood (leaving the pulmonary capillaries) 40mmHg (after gas exchange).

In summary: - the reasons for differences in partial pressure of O_2 & CO_2 between atmosphere and alveolar air are:-

1. The alveolar air is only partially replaced by atmospheric air with each breath.
2. O2 is being constantly absorbed into the pulmonary capillaries from the alveolar air
3. CO2 is constantly diffusing from the pulmonary blood into the alveoli
4. Dry atmospheric air that enters the respiratory passages is humidified even before it reach the alveoli

2) Thickness

- The respiratory membrane consist of the following layers:-

A. Fluid in the alveoli.

B. Alveolar wall (basement membrane+ epithelium).

C. Interstitial tissue.

D. Capillary wall (basement membrane+ endothelium).

- Normal thickness 0.5 micrometer.

2) Gas exchange is inversely proportion to thickness of respiratory membrane.

- For example when the thickness is decreased (as occurs during exercise), the gas exchange increased.

- It is impaired when the thickness is increased (e.g. in lung fibrosis& pulmonary edema). This cause hypoxia (low oxygen in blood); however, thickness of the respiratory membrane is less common cause of hypoxemia than (ventilation perfusion mismatching).

3) Surface area

- The available area for gas exchange is called the effective surface area. It indicates well ventilated alveoli in contact with well perfused capillaries.
- Gas exchange is directly proportional to effective surface area.
- For example when surface area is increased (as occur during exercise), gas exchange is increased.
- **The effective surface area is increased during exercise because:-**

➢ More alveoli are ventilated (due to ↑ ventilation).

➢ More capillaries are perfused (due to ↑perfusion).

- Total surface area equal about 70 m^2 (normal range: 50_100m^2).

4) Diffusion coefficient

- It is defined as the amount of gas that diffuse across the respiratory membrane per unit pressure deference per unit surface area per unit of time.

It depends on:-

- Solubility of the gas (direct relation).
- molecular weight of the gas (inverse relation).

Although molecular weight of CO_2 is larger than O_2. Its diffusion coefficient is higher than O_2. This is due to high solubility of CO_2.

Diffusion capacity of the respiratory membrane:

- The volume of the gas that crosses the respiratory membrane per unit partial pressure difference per unit time. It is affected by:

1. Thickness of membrane (inverse relationship)
2. Surface area of the membrane (direct relationship).

- It is measured by using carbon monoxide (CO) which is highly soluble in blood

The ventilation perfusion ratio (V/Q ratio)

- The ratio of alveolar ventilation to pulmonary blood flow (perfusion)
- Alveolar ventilation is about 4 L/min whereas pulmonary blood flow is about 5 L/min
- Therefore V\Q ratio =0.8 (about 1)

- **V\Q ratio is affected by gravity and lung diseases:-**

1. Effect of gravity on the V/Q ratio

- In the upright position V/Q ratio differs in different parts of the lung due to effect of the gravity

a) <u>At the apex of the lung:-</u>

- Blood flow (Q) is decreased and ventilation (V) is also decrease but to a lesser extent, therefor the ratio increase
- When perfusion is decreased to zero the ratio is increased to infinity (V/Q)=V/Q=infinity
- Since ventilation > perfusion ,the extra air =wasted ventilation (or dead space ventilation)

At the base of the lung

- Blood flow (Q) is increased and ventilation (V) is also increased but to a lesser extent ,therefore the ratio is decreased
- Since ventilation < perfusion ,the extra blood equal to wasted perfusion (or shunt flow)

- **2) Effect of lung diseases on V/Q ratio**

➢ Many lung diseases are characterized by V/Q inequality

➢ These may result in either wasted ventilation e.g. pulmonary embolism or wasted perfusion e.g. lung collapse

➢ The ratio is changed accordingly

- **Remember that V/Q inequality is the most common cause of hypoxemia**

- **Effect of V/Q mismatching on PO_2 and PCO_2 of alveolar air**

- *if ventilation to alveolus is reduced relative to it's perfusion (i.e. less O_2 supply from environment and less CO2 removal)

–PO2 in alveoli (PAO_2)decreases

–PCO2 in alveoli ($PACO_2$)increases

- This normally occur in some alveoli at the base of the lung

- *if perfusion to alveolus is reduced relative to it's ventilation (i.e. less CO2 reach the alveoli from blood).
- *–PO2 in alveoli (PAO_2) increases*
- *–PCO2 in alveoli ($PACO_2$) decreases*

- this normally occurs in some alveoli at the apex of the lung
- **The lung apex is the most favorable site of infection for tubercle bacilli (because of high PAO_2)**

To be continued.....