SURFACTANT

→ Surface tension lowering agent present in alveolus between alveolar fluid and air.

→ Secreted by type II alveolar epithelial cells

→ Composition:- Mixture of lipids (90%), proteins (10%), Ca^{2+} & carbohydrates

- Dipalmitoyl phosphatidyl choline (lecithin)
- Surfactant proteins — Albumin, IgA & apoproteins — SP-A, SP-B, SP-C, SP-D

Functions of Surfactant (8 mk):

1) Decreases surface tension 6 times

- Distending pressure of each alveolus lined by surfactant is only 2 mm Hg. If alveolus was lined by water, the distending pressure may be as high as 15 mm Hg.

2) Maintains alveolar stability

- Prevents overdistension & collapse of alveoli

↓

Helps to keep alveolar size relatively uniform

⇒ Surface tension in alveolus $\propto \frac{1}{\text{No. of surfactant molecules per unit area}}$

During Inspiration

Increase in diameter of alveolus → No. of surfactant molecules per unit area ↓es

↓

Surface tension ↑es → Prevents overdistension of lungs

During Expiration

Decrease in diameter of alveoli → surfactant molecules come closer

↓

No. of surfactant molecules per unit area ↑es

↓

Surface tension ↓es

↓

Prevents collapse of lungs during expiration.

⇓

Surfactant thus maintains alveolar stability.

3) Prevents pulmonary edema & keeps alveoli dry

If surfactant was not present

↓

the unopposed surface tension in the alveoli

↓

Produce a 20 mm Hg force

↓

favors transudation of fluid from interstitium & pulmonary capillary blood into alveoli

↓

pulmonary edema (collection of fluid in alveoli)

⇓

This is prevented by surfactant by ↓ing surface tension

4) Increases compliance of lungs by decreasing surface tension.

5) Reduces work of breathing by decreasing the elastic recoil of lung.

Prevents Hyaline Membrane Disease / (IRDS) Infant Respiratory Distress Syndr

At the time of birth → fetal alveoli is filled with fluid & lungs are completely collapsed

↑

When umbilical cord is cut → fetus develops hypoxia

↓

violent inspiratory efforts

↓

Produce high negativity in mediastinum

↓

Lungs try to expand.

* Surfactant should be there for proper expansion of lungs

* In fetus, development of surfactant is completed only by 31-32 weeks of intrauterine life.

→ If baby birth occurs prematurely, lung maturity is not complete because it lacks surfactant and lungs fail to expand leading to death of baby

⇓

Infant Resp. Distress Syndrome

Treatment: → • Synthetic surfactant / Bovine surfactant
• Steroid therapy (Glucocorticoids)

Q. Glucocorticoid is given to diabetic mothers towards term??

→ IRDS commonly seen in premature babies & babies born to diabetic mothers.

3) Protective function

Surfactant proteins SP-A & SP-D stimulate phagocytosis & chemotaxis & form part of innate immunity.

Q. Application of law of laplace in lung

Laplace's law states that the distending pressure P, in case of a distensible spherical organ $= \frac{2T}{R}$

where, T = Surface Tension & R - Radius of the organ.

P - Pressure required to keep alveoli inflated.

⇒ P will be high when T is high or R is less.

Significance of law of laplace

- If T remains same, small alveoli (with small r) will have high Pressure & vice versa.

↓

Air flows from higher pressure (small alveoli) to low pressure (large alveoli)

↓

Small alveoli becomes smaller & large alveoli becomes larger

↓

Eventually small alveoli collapse & large alveoli overdistends

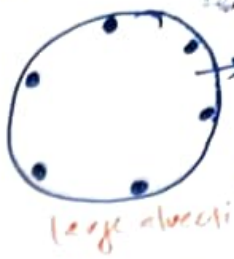
Significance

- To prevent this, we have surfactant molecules
- Small alveoli has higher conc. (no/area) of surfactants

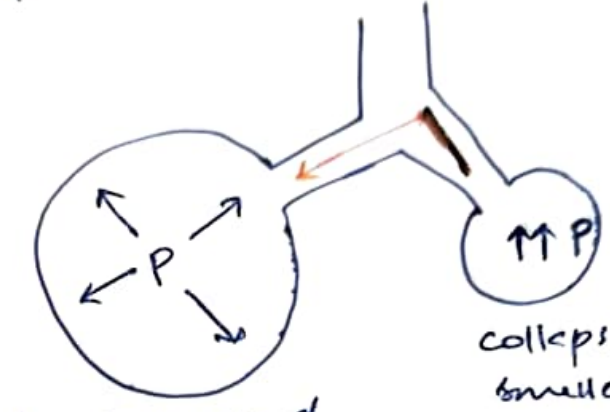
↓

so less surface tension (T)

In large alveoli, surfactant molecules are wide
separated (less concentration)
↓
∴ more surface Tension (T)



⟹ Hence, by changing T, pressure is maintained at a constant level.



Q. Respiratory Dead Space (4 mks)

→ The portion of tidal volume that does not take part in gas exchange - is called dead space (DS)

- Divided into → Anatomical DS
  → Physiological DS

- In normal subjects both are nearly the same.

Anatomical Dead Space

→ Volume of air in the respiratory passage from nose to terminal bronchiole (conducting zone) which does not take part in gas exchange.

→ **Normal Value = 150 ml** ⁂

Physiological Dead Space

Physio. DS = Anatomical dead space + Alveolar Dead Sp

Alveolar Dead Space ⟹ is the air in the non-functio alveoli, ie, in the under-perfused or non-perfused alveoli and over-ventilated alveoli

→ **Normal value = 5-10 ml**

Increase in physiological dead space ⟹ Pulmonary embolism
COPD

Q. Compliance

→ Compliance refers to the ability of lungs and thoracic wall to stretch (distensibility)

→ Expressed as change in lung volume (ΔV) per unit change in transpulmonary pressure (ΔP)

ie, $$\text{Compliance} = \frac{\Delta V}{\Delta P}$$

→ Stiffer the lung, less will be the compliance

→ Total compliance is the compliance of thorax + lungs together

② value = 0.13 L/cm H_2O.

→ when the compliance of thorax & lungs are taken seperately, the value will be more than total compliance.

→ Compliance of thorax alone = 0.22 L/cm of H_2O

" " lungs alone = 0.22 L/cm of H_2O

Hysteresis loop ⇒ For Compliance of Lungs Alone

→ To produce same volume, more pressure is required in inspiration than in expiration.

→ for the same pressure, inspiratory compliance curve lags behind expiratory curve

↓

Hence a hysteresis loop is obtained

Why there is hysteresis? ⇒ Because more pressure (Force) is required to open a closed alveolus than closing an already opened alveolus.

Factors Affecting Compliance

a) Lung Volume ⇒ An individual with only one lung has half the ΔV for a given ΔP ∴ compliance ↓es

b) Phases of respiration

Compliance is slightly greater when measured during deflation than when measured during inflation.

Specific Compliance

→ To eliminate the role of lung volume on compliance, compliance is recorded as a function of FRC (Functional Residual Capacity).

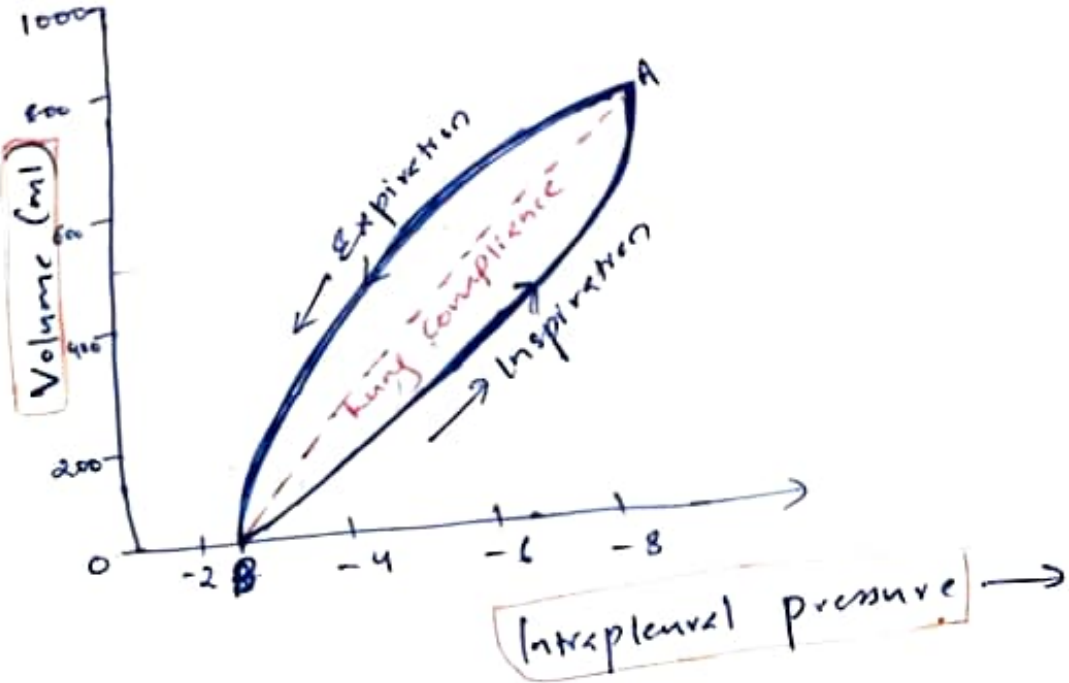
$$\text{Specific Compliance} = \frac{\text{Lung Compliance}}{\text{FRC}}$$

Applied Aspects

1) Decreased Compliance ⇒ Pulmonary fibrosis
Pulmonary Oedema
Pulmonary Congestion

2) Increased Compliance ⇒ Emphysema
Old age.
(Due to loss of elasticity)

Hysteresis loop



Ventilation - Perfusion Ratio (4 mL/8 mL)

→ V-Q ratio is the ratio between alveolar ventilation (V) in 1 minute and pulmonary perfusion (Q) in 1 minute.

→ Resting alveolar ventilation = 4 L/mt
Pulmonary blood flow = cardiac output = 5 L/mt

$V/Q = \frac{4\ L/mt}{5\ L/mt} = 0.8$ at the middle of the lung.

⇓

At this ratio, maximum oxygenation occurs

Effect of gravity on V/Q

→ Because of the effect of gravity

↓

{ Basal alveoli are overperfused
Apical alveoli are underperfused }

Alveolar ventilation also reduced linearly from base to apex ⟹ ∴ Basal alveoli are overventilated
Apical alveoli are underventilated.

⇒ However, gravity affects perfusion more

Hence, the apical alveoli are more underperfused than underventilated.

∴ At apex V/Q ratio $= \frac{\downarrow V}{\downarrow\downarrow Q} =$ ↑ed V/Q ratio $= 3$

Conversely, At base, alveoli are more overperfused than overventilated

∴ $\frac{V}{Q}$ ratio $= \frac{\uparrow V}{\uparrow\uparrow Q} =$ ↓ed V/Q ratio $= 0.6$

Clinical importance of V/Q ratio

4 mks

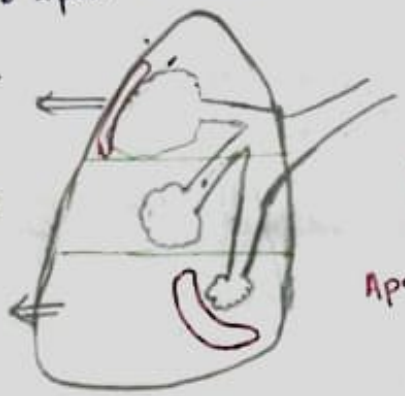
Q. Pulmonary tuberculosis affects apex of lungs first

a) Because of high V/Q ratio, more O_2 is available at the apex of lung (high pO_2).

↓

High alveolar pO_2 provides a favourable environment for the growth of Mycobacterium tuberculosis which are aerobic bacteria.

b) Antibodies in the blood do not reach the apex satisfactorily because of poor perfusion at the apex. So apex is more vulnerable to bacterial attack.



Apex
V ↓
Q ↓↓
V/Q = 3

Base
V ↑
Q ↑↑
V/Q = 0.6

Due to effect of gravity.
Size of alveoli is more at apex & less at base

Apex ⇒ Already enlarged alveolus
↓
less compliant
↓
Ventilation less

Vessels are compressed ⇒ Perfusion also less

Base ⇒ smaller alveolus
↓
More compliant
↓
Ventilation more
Perfusion also more.

Diffusion of Gases through Respiratory Membrane

Q. Respiratory Membrane / Pulmonary Membrane / Alveolo-capillary Membrane

→ Membrane seperating capillary blood from alveolar air.

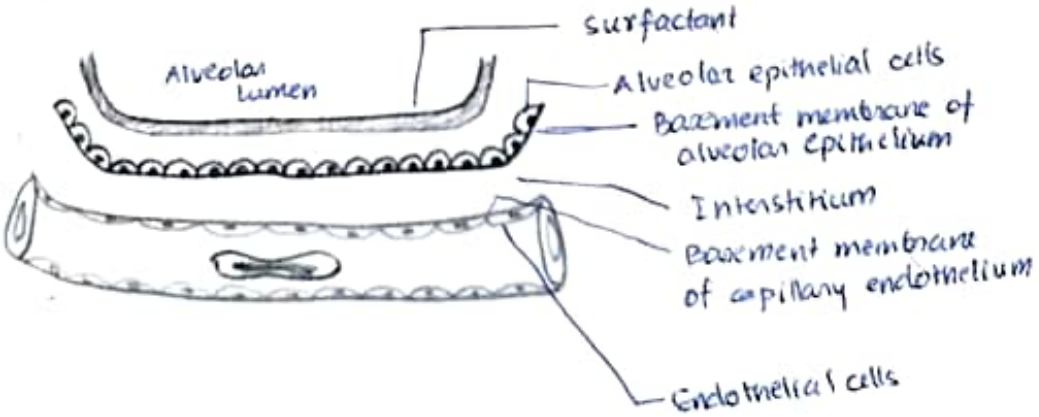
→ Exchange of gases b/w capillary blood & alveolar air requires diffusion through this membrane.

→ **Layers of Respiratory membrane**

I - Layer of alveolar fluid & surfactent
II - Layer of alveolar epithelial cells.
III - Epithelial basement membrane of alveoli
IV - Interstitial space
V - Capillary basement membrane
VI - Layer of capillary endothelial cells.

→ Thickness of Resp membrane = 0.6 Mm

Q. Factors affecting diffusion



JN 8

Factors affecting diffusion across respiratory membrane

1) Thickness of respiratory membrane (d) → inversely proportional

Rate of diffusion $\propto \frac{1}{\text{membrane thickness}}$

2) Surface Area of Respiratory Membrane

Rate of diffusion $\propto$ surface area

3) Diffusion Coefficient (D)

Rate of diffusion $\propto D$

$D = \frac{\text{Solubility of the gas}}{\sqrt{\text{Molecular weight of the gas}}}$

4) Pressure gradient across Respiratory Membrane

Rate of diffusion $\propto$ pressure difference b/w pA & pC $(pC - pA)$ or ΔP

(pA → partial pressure of a gas in alveoli
pC → partial pressure of a gas in pulmo. capillary)

Combining all this ⇒

Rate of diffusion $(V_{gas}) \propto \frac{\Delta P \cdot A \cdot D}{d}$

or $V_{gas} \propto \frac{\Delta P \times A \times S}{d \times \sqrt{MW}}$

This is called fick's law of diffusion.

V_{gas} → Rate of diffusion of gas
ΔP → Pressure gradient
A → Surface Area
S → Solubility of gas
d → Thickness of resp. membrane
MW → Molecular weight of the gas.
D → Diffusion coefficient

118	REEMA
119	RENNA FA
120	RIDHA F
121	RISHEAM