PULMONARY CAPILLARY DYNAMICS

Determinants of Net Fluid Movement across Capillaries

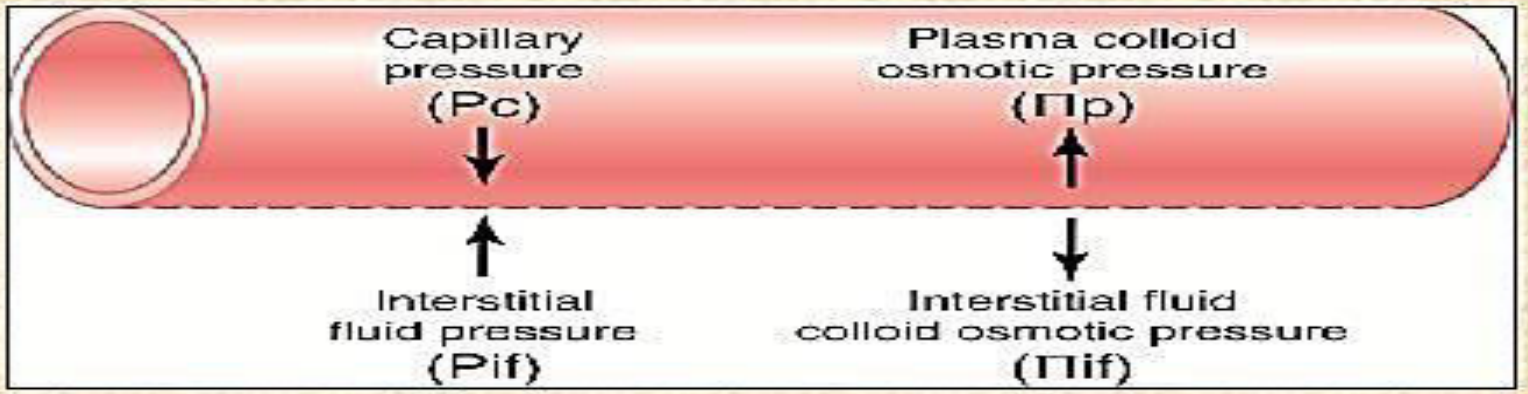


Figure 16-5; Guyton and Hall

- *Plasma colloid osmotic pressure* (π c)- opposes filtration causing osmosis of water inward through the membrane
- *Interstitial fluid colloid pressure* (π if) promotes filtration by causing osmosis of fluid outward through the membrane

$$NP = Pc - \pi c - Pif + \pi if$$

Dynamics of fluid exchange in the pulmonary capillaries

- The formation and drainage of interstitial fluid in the lungs are controlled as in other tissues by **Starling forces** as follows:
  - Forces that favor fluid filtration:
    - These include the following:
    1. The pulmonary capillary hydrostatic pressure (normally about 7 mm Hg).
    2. The pulmonary interstitial fluid colloid osmotic pressure (normally about 14 mm Hg).
    3. Negative interstitial fluid pressure (normally about – 8 mmHg).
  - Forces that favor fluid absorption:

  These include mainly

  The plasma colloid osmotic pressure (normally about 28 mmHg).

Dynamics of fluid exchange in the pulmonary capillaries

❖Forces tending to cause movement of fluid outward from the pulmonary capillaries and into the pulmonary interstitium (filtering forces):

Pulmonary capillary hydrostatic pressure	**7**
Interstitial fluid colloid osmotic pressure	**14**
Negative interstitial fluid pressure	**8**
TOTAL OUTWARD FORCE	**29**

❖Forces tending to cause absorption of fluid into the pulmonary capillaries (absorbing forces):

- Plasma colloid osmotic pressure of pulmonary capillaries = 28 mmHg
- TOTAL INWARD FORCE 28

Dynamics of fluid exchange in the pulmonary capillaries

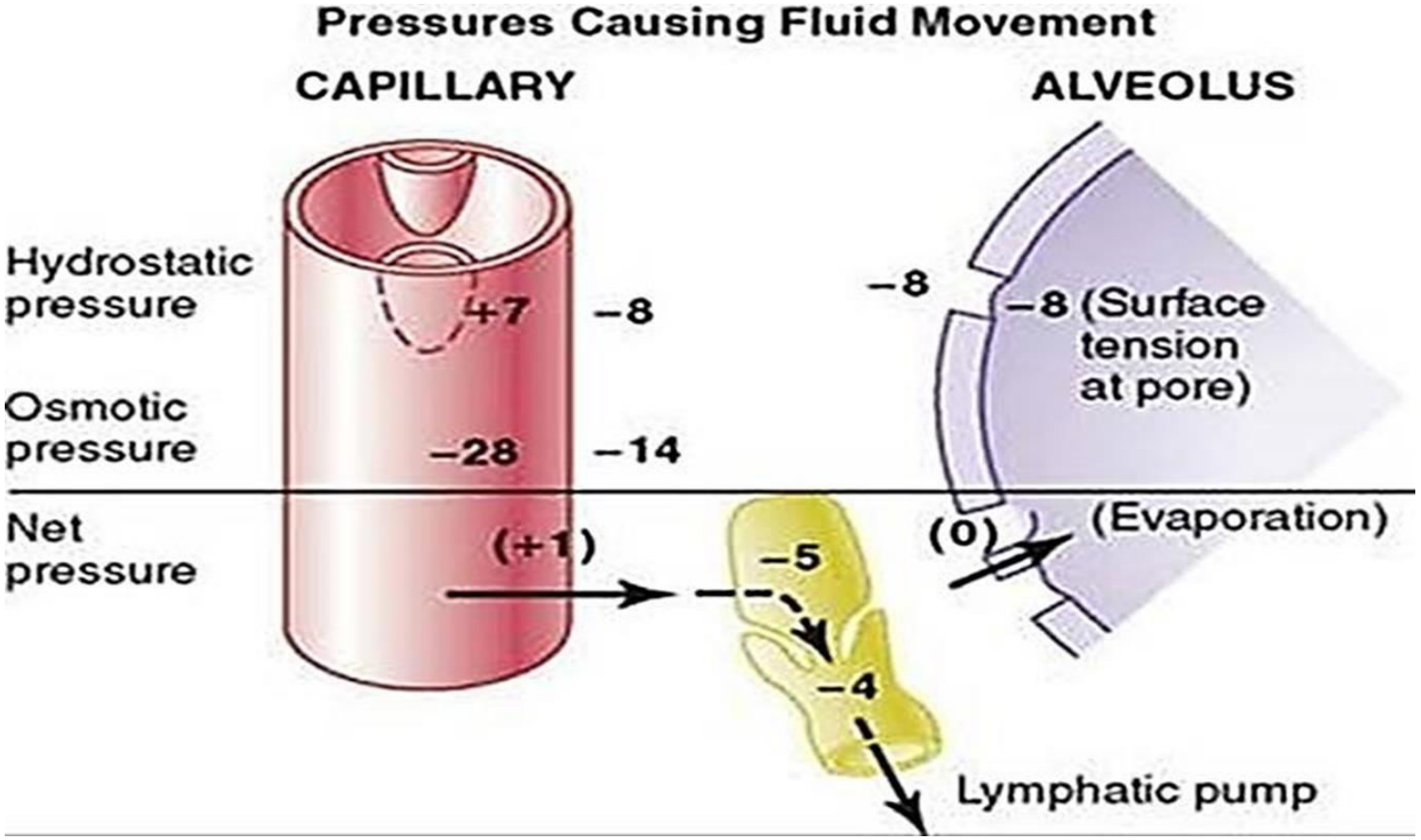
- Thus, the normal outward forces are slightly greater than the inward forces, providing a net filtration pressure at the pulmonary capillary membrane; this can be calculated as follows:

Total outward force	**+29**
Total inward force	**-28**
Net Filtration Pressure	**+1**

- This filtration pressure causes a slight continual flow of fluid from the pulmonary capillaries into the interstitial spaces, and except for a small amount that evaporates in the alveoli, this fluid is pumped back to the circulation through the pulmonary lymphatic system.

Pressures causing fluid movement

❖Figure. Hydrostatic and osmotic forces at the capillary (left) and alveolar membrane (right) of the lungs. Also shown is the tip end of a lymphatic vessel (center) that pumps fluid from the pulmonary interstitial spaces.



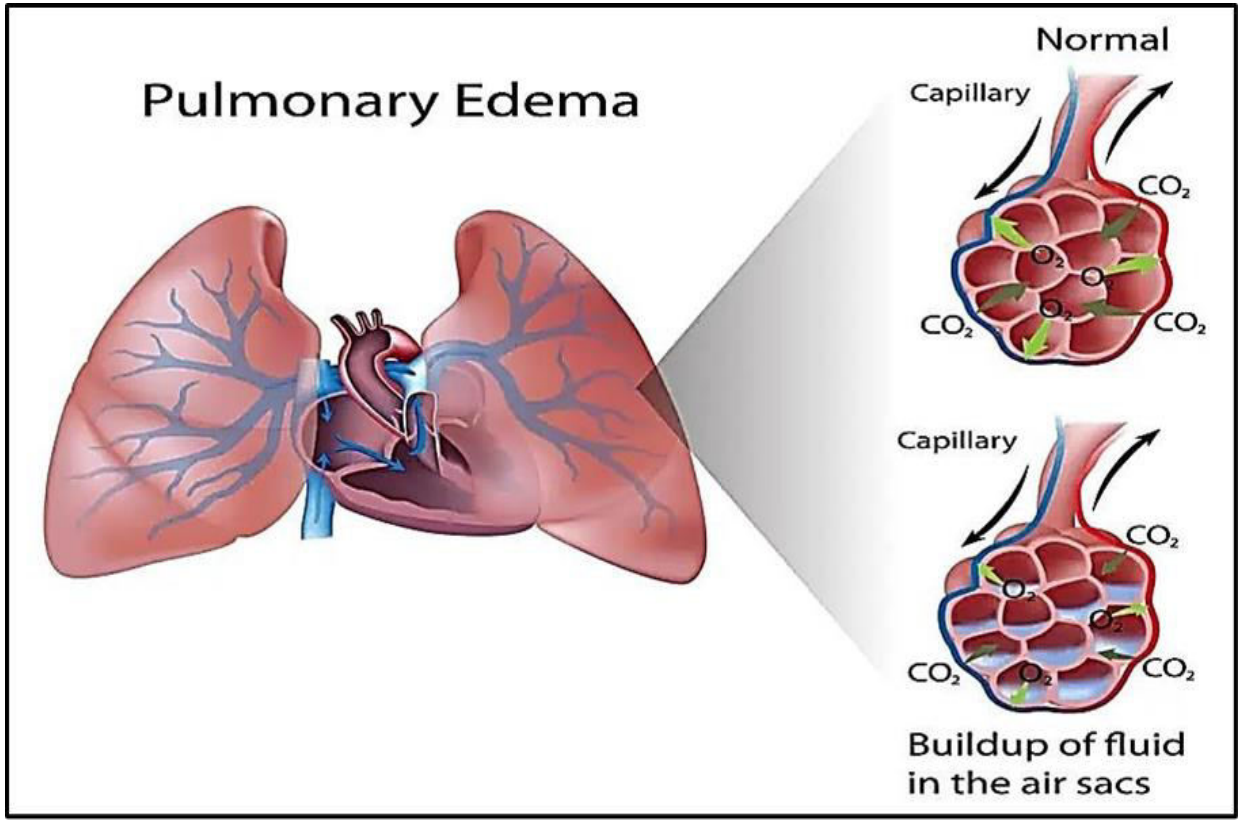
The Mechanism of Keeping the lung alveoli “Dry”

- An adequate gas exchange in the lung alveoli is essential to life, and this requires that they must be kept dry i.e. free of fluids. This is favored by:
  - The low net filtration pressure.
  - The rich lymphatic drainage of the lungs: this removes any excess fluids that may accumulate around the alveoli.
  - The negative pressure in the lung interstitial spaces: this sucks out excess alveolar fluid through the openings between the alveolar epithelial cells.

Pulmonary Edema

❖**Definition**:

- This is accumulation of excess tissue fluid in the lungs.
- It starts in the interstitial spaces, then as the pressure in these spaces rises into the positive range, fluid accumulation suddenly increases resulting in rupture of the alveolar membranes and filling of alveoli with excess fluid.



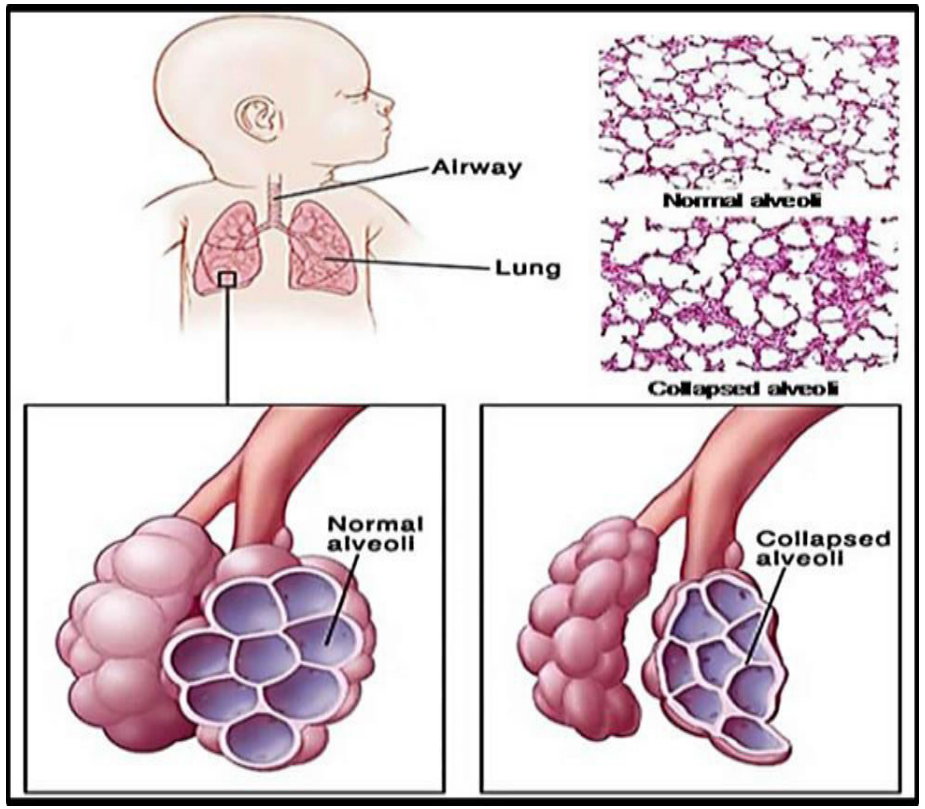
Pulmonary Edema

❖The main causes of pulmonary edema are the following:

1. **Rise of the pulmonary capillary hydrostatic pressure**, commonly due to left ventricular failure or severe mitral stenosis.
2. **Damage of the pulmonary capillary membranes** (due to infections such as pneumonia or inhalation of irritant gases as chlorine gas or sulfur dioxide gas. Each of these causes rapid leakage of both fluid and plasma proteins out of the capillaries (the latter helps fluid filtration by increasing the interstitial fluid colloid osmotic pressure)and into both the lung interstitial spaces and the alveoli.
3. **Obliteration of lymph drainage from the lungs** e.g. by thoracic tumors.
4. **Hypoproteinemia**: this helps occurrence of pulmonary edema by decreasing the plasma colloid osmotic pressure.
5. **The respiratory distress syndrome (RDS).**

Respiratory distress syndrome (RDS)

- A serious lung disease that occurs due to deficient formation of the surfactant.
- Decreased secretion of surfactant → ↑ surface tension of alveolar fluid (i.e. collapsing force) → excessive inspiratory work to distend the collapsed lungs which may fail to distend → respiratory distress syndrome (RDS).
- It may occur in adults but it is more common in newly born infants, particularly:
  - Premature infants: they are born before maturation of the surfactant-forming system. In these infants, lung alveoli are small and the lungs contain areas of atelectasis (alveolar collapse) as well as edema.
  - Hypothyroid infants: thyroid hormones stimulate formation of the surfactant.
  - Infants born by diabetic mothers: because the blood of the infant contains excess insulin which inhibits surfactant formation.



Respiratory distress syndrome (RDS)

❖Clinical Significance of lung surfactant:

❖Prevents pulmonary edema (helps keeping alveoli dry).

- The surface tension of the alveolar fluid can withdraw fluid from the pulmonary capillaries into the alveoli causing → pulmonary edema and respiratory distress in the absence of surfactant.

Pulmonary Edema Safety Factor

- The same factors that prevent edema elsewhere also act in the lungs to prevent pulmonary edema i.e. the following safety factors should be first overcome before appreciable pulmonary edema occurs:
  - The low capillary hydrostatic pressure and high plasma colloid osmotic pressure.
  - The normal negative interstitial fluid pressure.
  - The rich pulmonary lymphatic drainage and the resulting low interstitial fluid colloid osmotic pressure (due to excessive protein washdown)

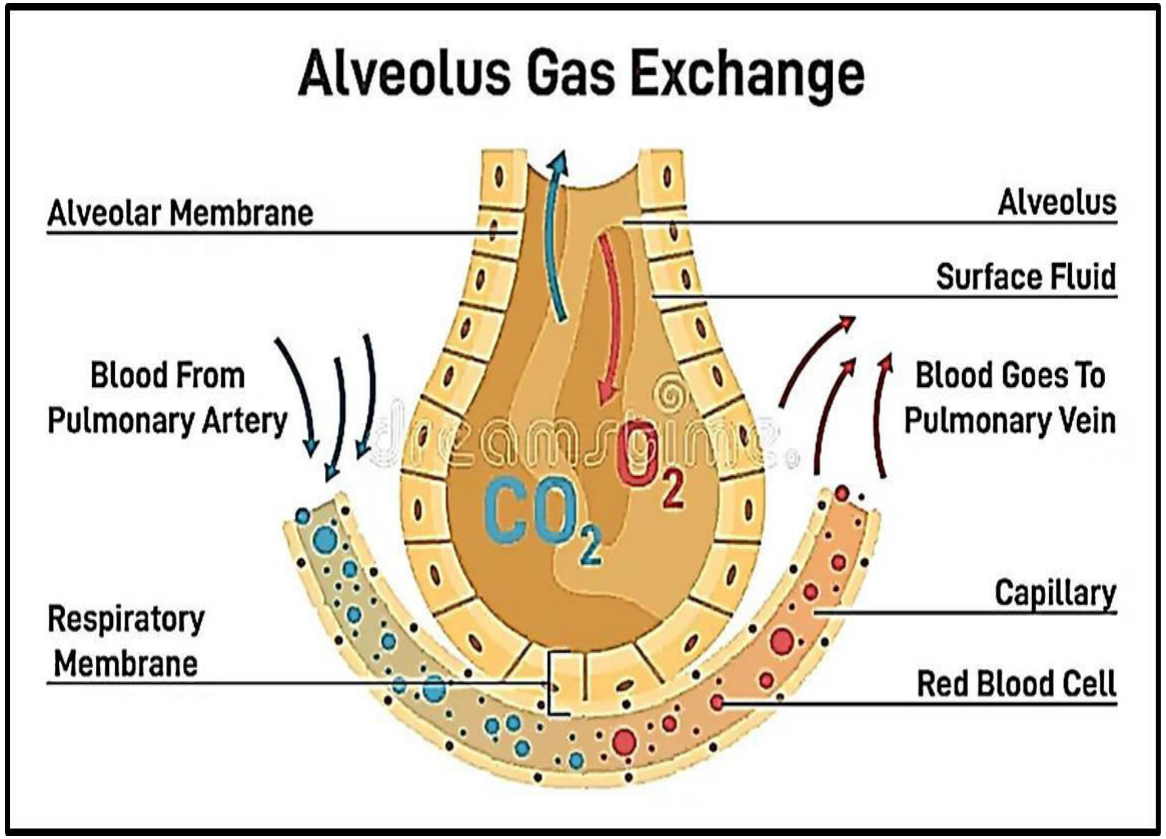
- Low pulmonary capillary pressure
  - in the human being, whose normal plasma colloid osmotic pressure is 28 mm Hg, the pulmonary capillary pressure must rise from the normal level of 7 mm Hg to more than 28 mm Hg to cause pulmonary edema, giving an acute safety factor against pulmonary edema of 21 mm Hg.
- Rapidity of Death in Acute Pulmonary Edema.
  - When the pulmonary capillary pressure rises even slightly above the safety factor level→, lethal pulmonary edema can occur within hours, or even within 20 to 30 minutes if the capillary pressure rises 25 to 30 mm Hg above the safety factor level. Thus, in acute left-sided heart failure, in which the pulmonary capillary pressure occasionally does rise to 50 mm Hg, death frequently occurs afterwards in less than 30 minutes from acute pulmonary edema.

Safety Factor

- Safety Factor in Chronic Conditions.
  - When the pulmonary capillary pressure remains elevated chronically (for at least 2 weeks), the lungs become even more resistant to pulmonary edema because:
    - The lymph vessels expand greatly, increasing their capability of carrying fluid away from the interstitial spaces perhaps as much as 10-fold. Therefore, in patients with chronic mitral stenosis, pulmonary capillary pressures of 40 to 45 mm Hg have been measured without the development of lethal pulmonary edema.

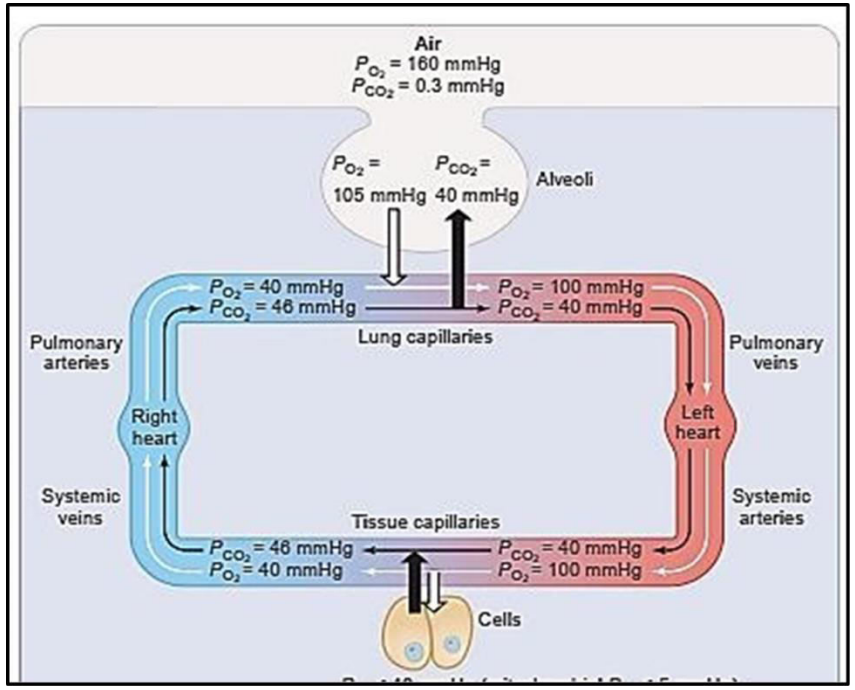
Characters of Pulmonary (i.e. Respiratory) Membrane

- It is also alveolocapillary membrane.
- It is a blood-air barrier because it separates the air in alveoli from the blood in pulmonary capillaries.
- Its average total surface area is about 70 m2 .
- It is very thin; its thickness is only 0.2-0.6 micron (although it consists of several layers) allowing for efficient gas exchange.
- Gas exchange across the respiratory membrane
- This occurs by simple diffusion.
- The alveolocapillary membrane favors O2 and CO2 exchange because:
  - Its surface area is large.
  - It is very thin.
  - It is freely permeable to these gases (these gases are lipid-soluble, so they dissolve easily in the membrane cells which facilitates their transport)



Arterialization of venous blood in the lungs

- This process occurs as follows:
- The average Po2 in the venous blood flowing to the lungs (through pulmonary arteries) is 40 mmHg, while it is about 100 mmHg in the alveolar air. Therefore, O2 diffuses from the alveolar air to the venous blood.
- On the other hand, the average PCO2 in the venous blood is 46 mmHg, while it is about 40 mmHg in the alveolar air. Therefore, CO2 diffuses from the venous blood to the alveolar air .
- After equilibration, the (P) of these gases in the pulmonary veins (which carry arterial blood) become nearly equal to their corresponding values in the alveolar air (the PO2 about 97 mmHg and the PCO2 about 40 mmHg).



Equilibration of the respiratory gases in the lungs

- The molecular weight of O_2 is smaller than that of CO_2 (32 and 44 respectively), thus the diffusion rate of O_2 would be more rapid.
- However, since the solubility coefficient of CO_2 is normally much higher than that of O_2, the diffusion rate of CO_2 is normally about 20 times more than that of O_2.
- In the diseases associated with thickening of the respiratory membrane i.e. cause alveolo-capillary block (e.g. diffuse interstitial pulmonary fibrosis), O_2 diffusion is reduced much earlier than CO_2 diffusion.
- This is because the lung diffusion capacity for O_2 is much smaller than that for CO_2. in these diseases, severe hypoxia may occur without significant CO_2 retention in the body.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Q1 By comparing the dynamic pulmonary fluid exchange with peripheral one?

a. Interstitial lung fluid pressure is slightly negative than that in peripheral tissues

b. Pulmonary capillary pressure is equal with capillary pressure in the peripheral tissues that is about 17mmHg

c. The alveolar walls are extremely thin, which allows dumping of fluid from the interstitial spaces into the alveoli

d. Pulmonary capillaries are more permeable to protein so colloidal osmotic pressure is about 14mmHg with less than half this value in peripheral tissue

e. The excess fluid is carried away through the lymphatics and absorbed by capillaries in pulmonary circulation

Answer : a

Q2 Wrong about blood gas barrier?

a. (NO OTHER CHOICE)

b. Thick

Answer : b

All of the following are edema safety factors except:

a. High osmotic pressure of the plasma proteins
b. Negative ISF
c. High pulmonary capillary hydrostatic pressure
d. Presence of the surfactant

Answer : c