

Lecture 3

PULMONARY VENTILATION

Objectives

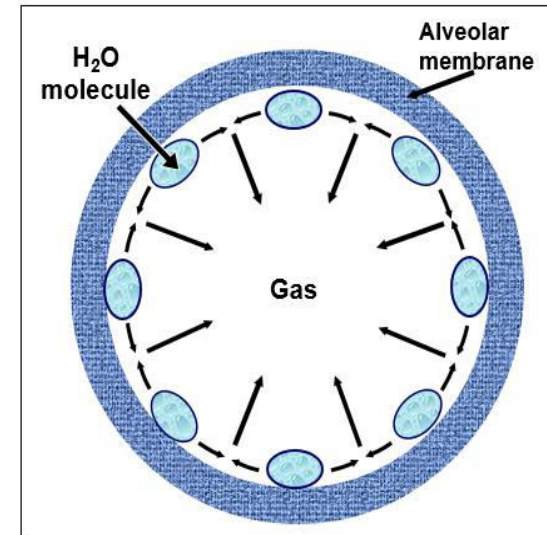
- ✿ The muscles used during ventilation
- ✿ The mechanism of ventilation of the lung
- ✿ The types of respiratory pressures
- ✿ The elastic recoil of the lung (surface tension)
- ✿ The chemical composition, functions and factors affecting surfactant production
- ✿ Types of respiratory dead space
- ✿ Significance of ADS

Recoil tendency of lung

- 1) Tissue elasticity (1/3rd)
- 2) Surface tension (2/3rd)

Surface Tension

- Alveoli lined by thin film of fluid secreted by the epithelial cells (intermolecular attraction) → surface tension → tendency to reduce the size of the alveolus
- Law of Laplace: in spherical structures (alveolus), the distending pressure equals 2 times the tension divided by the radius
 - ☞ Large alveoli (large radii) have low collapsing pressure and are easy to keep open
 - ☞ Small alveoli (small radii) have high collapsing pressures and are more difficult to keep open
- In the absence of surfactant, small alveoli → collapse



$$P = \frac{2T}{r}$$

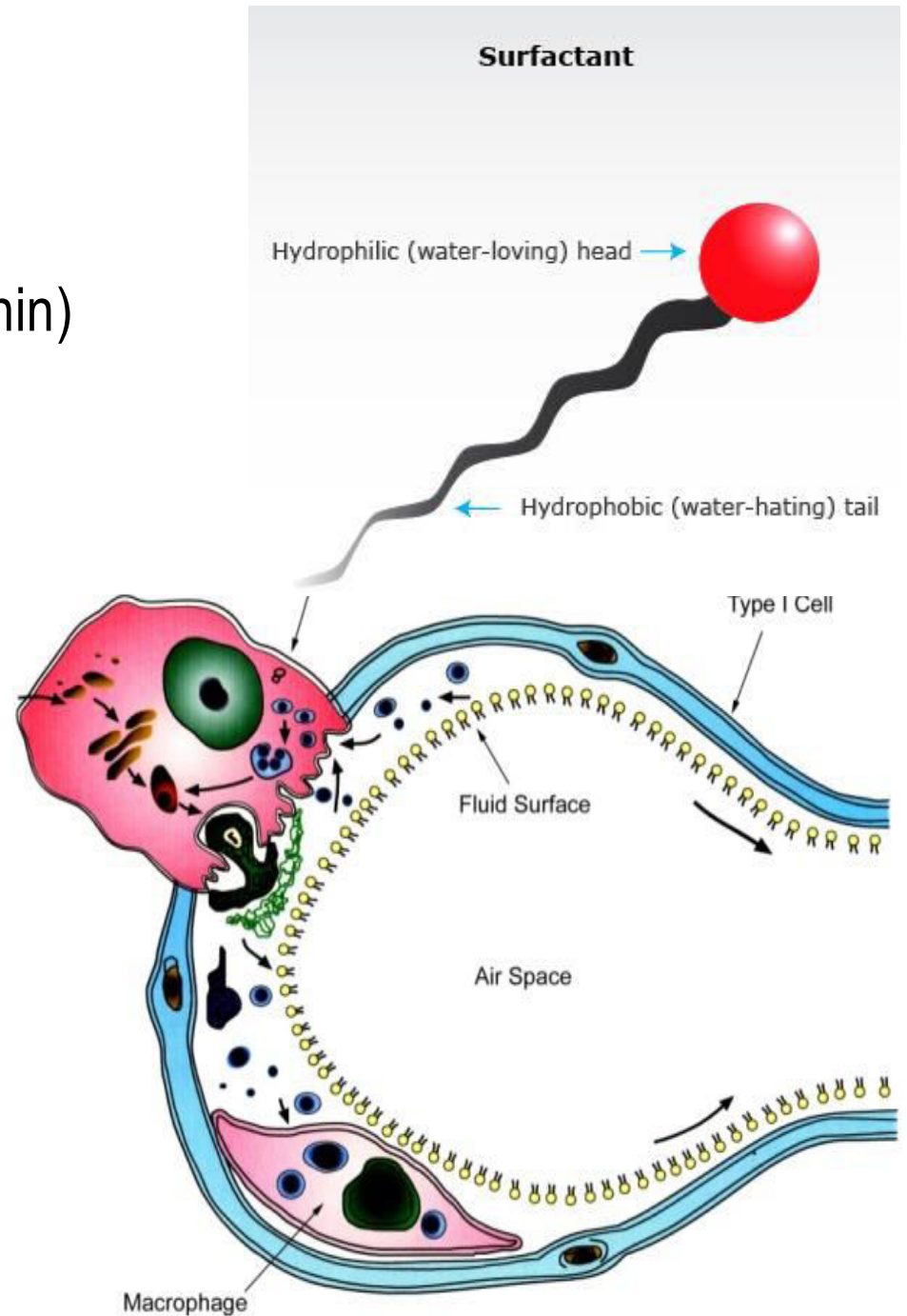
Surfactant

⊙ Chemical nature:

- Surfactant apoprotein
- Phospholipid (Dipalmitoyllecithin)
 - ☞ Hydrophilic head (PO_4)
 - ☞ Hydrophobic tail (lipid)
- Ca^{++}

⊙ Origin:

- Type 2 alveolar cells (granular pneumocytes)

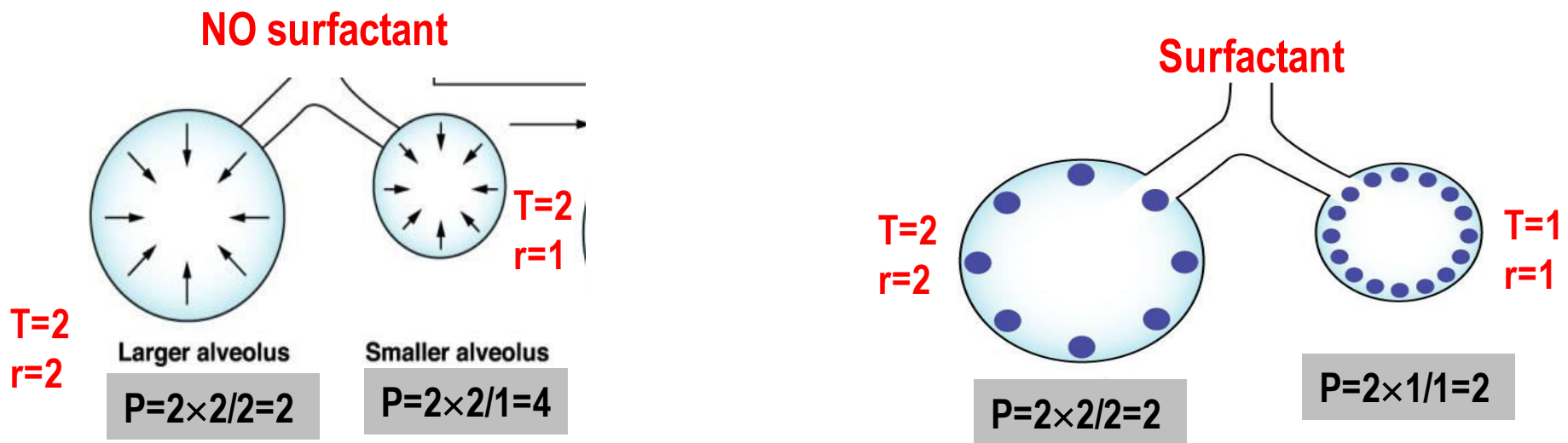


⊙ Functions of Surfactant

1) ↓ Surface tension → ↓ distending pressure from 15 to 3mmHg → ↑ lung compliance → ↓ work of breathing

2) It maintains alveolar stability: Alveolar surface tension $\propto \frac{1}{\text{surfactant conce.}}$

- ↑ Diameter of the alveolus (inspiration) → ↓ surfactant concentration → ↑ surface tension → prevents overdistension
- ↓ Diameter of alveoli (expiration) → ↑ surfactant concentration → ↓ surface tension → prevents collapse



3) Surfactant prevents pul. edema and keeps the alveoli dry.

- If the surfactant was not present, the unopposed surface tension in the alveoli would produce a 20 mm Hg force favoring transudation of fluid from the blood into the alveoli.

Surfactant deficiency

1) Respiratory distress syndrome (RDS)

■ In premature baby

- ☞ The fetus makes respiratory movements in utero, but the lungs remain collapsed until birth. After birth, the infant makes several strong inspiratory movements and the lungs expand. Surfactant keeps them from collapsing again.
- ☞ Surfactant production started by 24 week of pregnancy and completed by 35 week of intrauterine life (cortisol dependent)
- ☞ IRDS: high surface tension → collapse of alveoli + retention of fluid in the lungs → death

2) 100% O₂ therapy for long time

3) Occlusion of pulmonary artery or major bronchus

4) Cigarette smoking

5) Hormones:

- ☞ Hypothyroidism
- ☞ Hypocorticism
- ☞ Hyperinsulinism (occurs in diabetic mothers ⇒ ↑ incidence of IRDS in infants born to diabetic mothers.)

RESPIRATORY DEAD SPACE (RDS)

- Definition: The portion of TV that does not take part in gas exchange
- Types
 1. Anatomical dead space
 2. Alveolar dead space
 3. Total (physiological) dead space

Anatomical dead space (ADS)

Definition

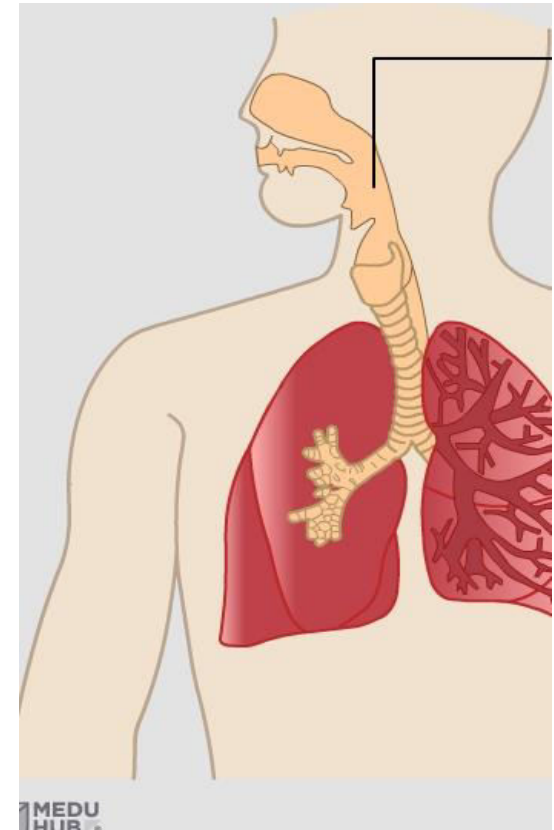
- Volume of air in the respiratory passage from nose to the terminal bronchiole.

Normal value

- Equal in millilitres to your B.WT in pounds. 68 kg male (150 lb) \Rightarrow anatomical dead space \approx 150 ml

Measurement

- Fowler's method (Single-breath N₂ test) and Bohr's method

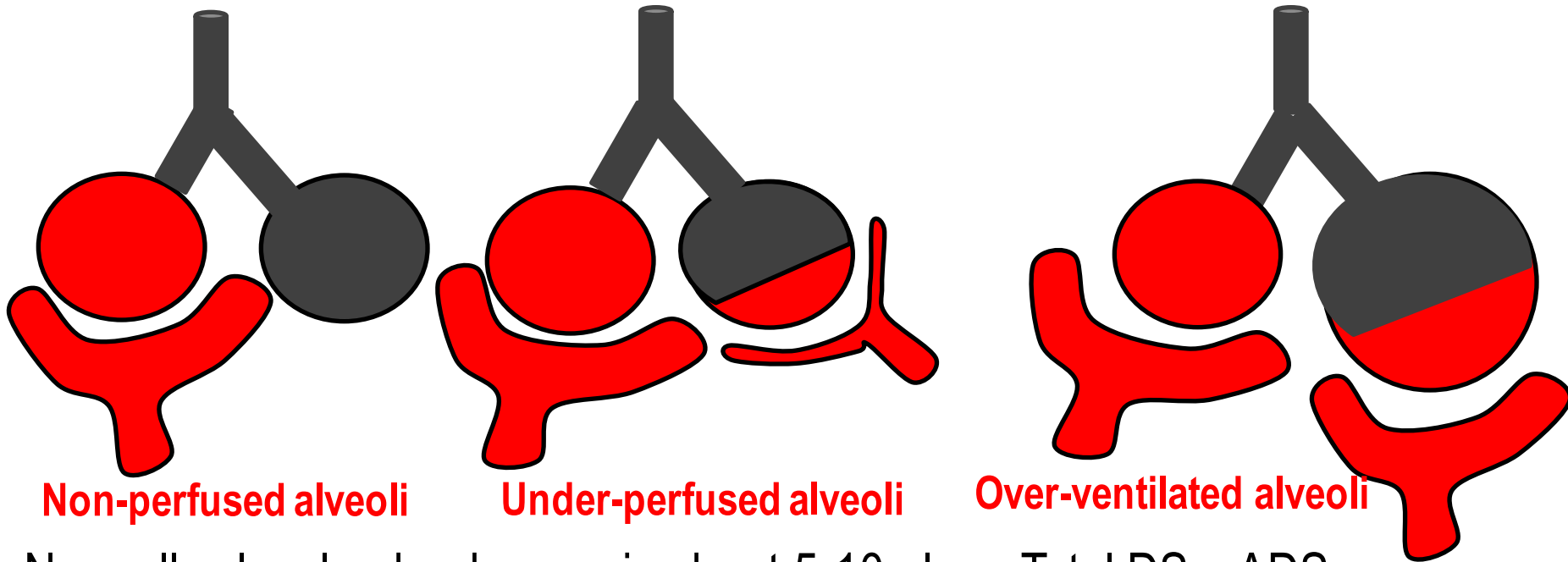


Total (physiologic) dead space

- Total DS = ADS + alveolar DS

Alveolar dead space

- Air in the non functioning alveoli



- Normally alveolar dead space is about 5-10mL → Total DS = ADS
- ↑ Total dead space is seen in pulmonary embolism and COPD.
- Total DS is calculated by Bohr's equation from the PCO_2 of expired air ($PECO_2$), the PCO_2 of arterial blood ($PaCO_2$), and tidal volume (VT)

Bohr's equation (total dead space)

⊙ Amount of CO₂ expired = Amount of CO₂ (alveoli) + Amount of CO₂ (DS)

$$\odot P_{\text{ECO}_2} \times V_T = P_{\text{aCO}_2} (V_T - V_D) + P_{\text{iCO}_2} \times V_D$$

$$\odot P_{\text{ECO}_2} \times V_T = P_{\text{aCO}_2} (V_T - V_D) \quad \begin{array}{l} \blacksquare P_{\text{iCO}_2} = P_{\text{CO}_2} \text{ atmospheric air} = 0.3\text{mmHg} \approx 0 \\ \blacksquare P_{\text{iCO}_2} \times DV = 0 \end{array}$$

$$\odot V_D = V_T - \left(\frac{P_{\text{ECO}_2} \times V_T}{P_{\text{aCO}_2}} \right)$$

⊙ Example: P_{ECO₂} = 28mmHg, P_{aCO₂} = 40mmHg & V_T = 500ml

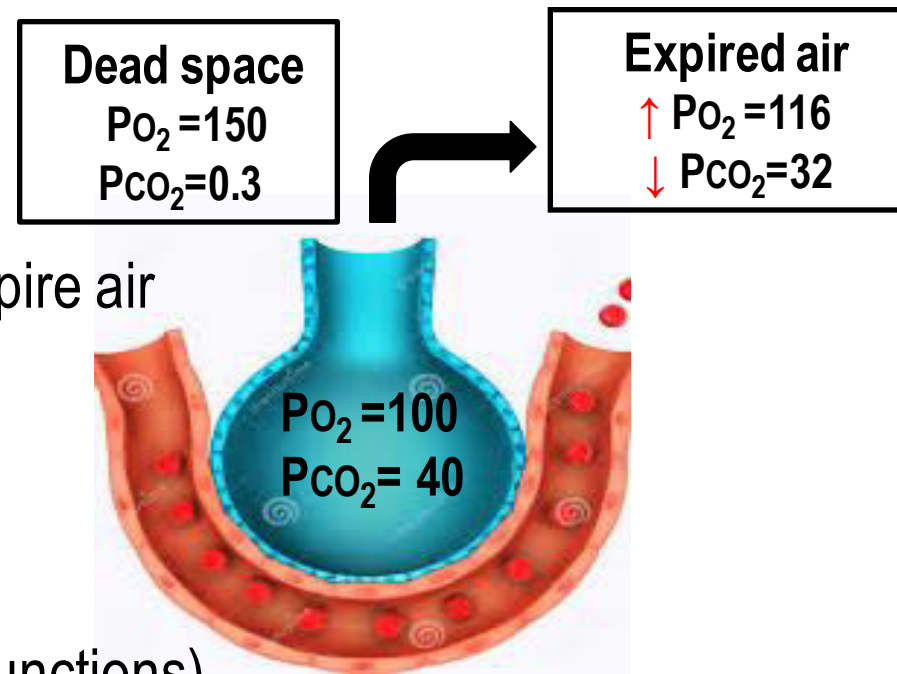
$$V_D = 500 - \left(\frac{28 \times 500}{40} \right) = 500 - 350 = 150\text{ml}$$

⊙ The equation can also be used to measure the ADS if P_{aCO₂} replaced with alveolar P_{co₂} (P_{ACO₂}) ■ P_{ACO₂} = P_{CO₂} of the last 10 ml of expired gas

Significance of ADS

Physiological significance:

1) Differences between alveolar and expire air



2) Protective functions (air conditioning functions)

- Humidification, warming and filtration of inspired air

Particles				
$> 10\mu$	$2-6 \mu$		$2-0.5\mu$	$<0.5\mu$
Nose	Upper airway	Lower airway	Alveoli	
Hair and mucus	Mucus	Mucus	Macrophages	
	Sneezing	coughing		

Clinical significance

3) Differences in alveolar ventilation

	Eupnoea	Shallow rapid	Deep slow
RR	12/min	30/min	6/min
TV	500 ml	200 ml	1000 ml
Pulmonary ventilation	6L	6L	6L
Alveolar ventilation	$12 \times (500 - 150) =$ 4.2L	$30 \times (200 - 150) =$ 1.5L	$6 \times (1000 - 150) =$ 5.1L
		Hypoxia and hypercapnea	Athletes: Early exercise