

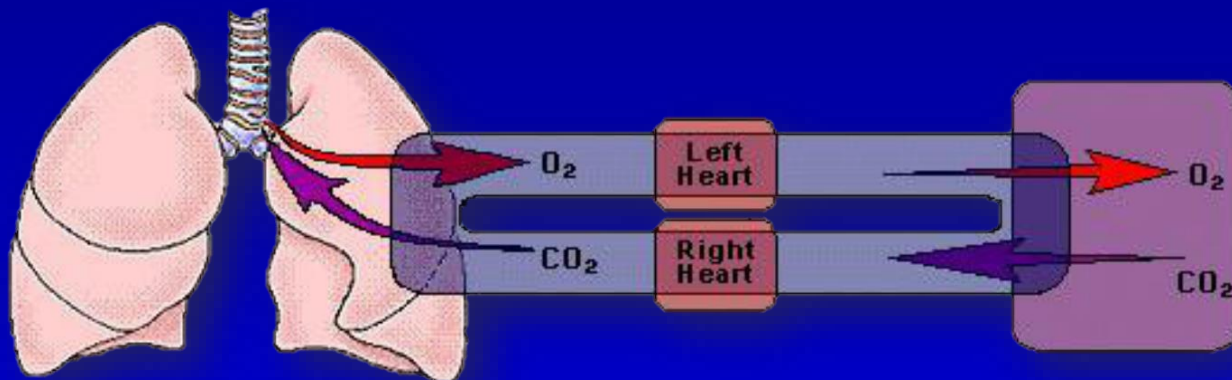
Respiratory Physiology

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為什麼要學呼吸生理學？
不知道這個也活得好好的



slido



何時需刻意地增加呼吸效率？

① Start presenting to display the poll results on this slide.

何時需刻意地增加呼吸效率？

- 正常休息



- ✓ 延腦，不需要特別注意

- 周圍環境缺氣：高山，礦坑



- 疾病：長期阻塞性肺病



- 運動

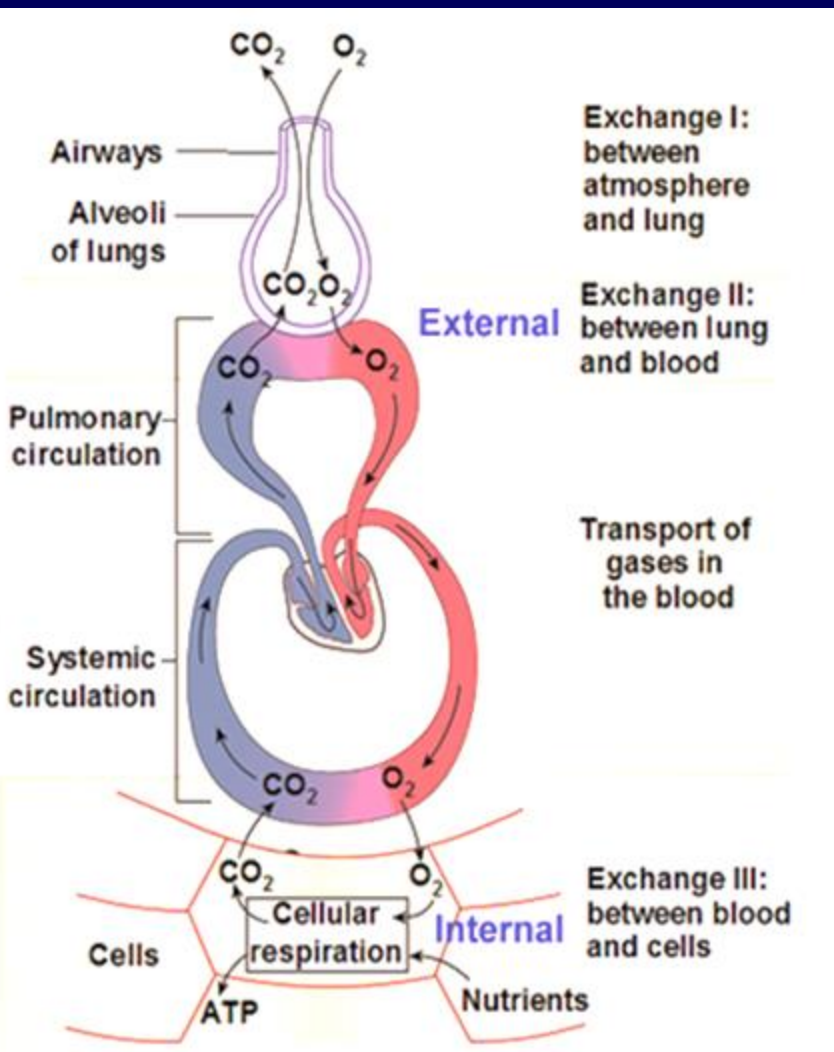


- ✓ 身體代謝增加

Outline

- **Background (背景)**
- Structure and function
- Ventilation
- Perfusion and ventilation/perfusion ratio
- Static/Dynamic respiratory mechanics
- Diffusion and gas transport
- Neural control of respiration
- Chemical control of respiration
- Acid-base balance
- Examples: exercise and high altitude adaptation₅

Background



- **Systemic respiration:** gas exchange between the external environment and the body
- **Cellular respiration:** the utilization of O_2 in metabolic pathways of cells for nutrient breakdown to get ATP
- Primary sites of gas exchange in lungs: **alveoli (肺泡)**
- Primary sites of cellular respiration: **mitochondria (粒線體)**

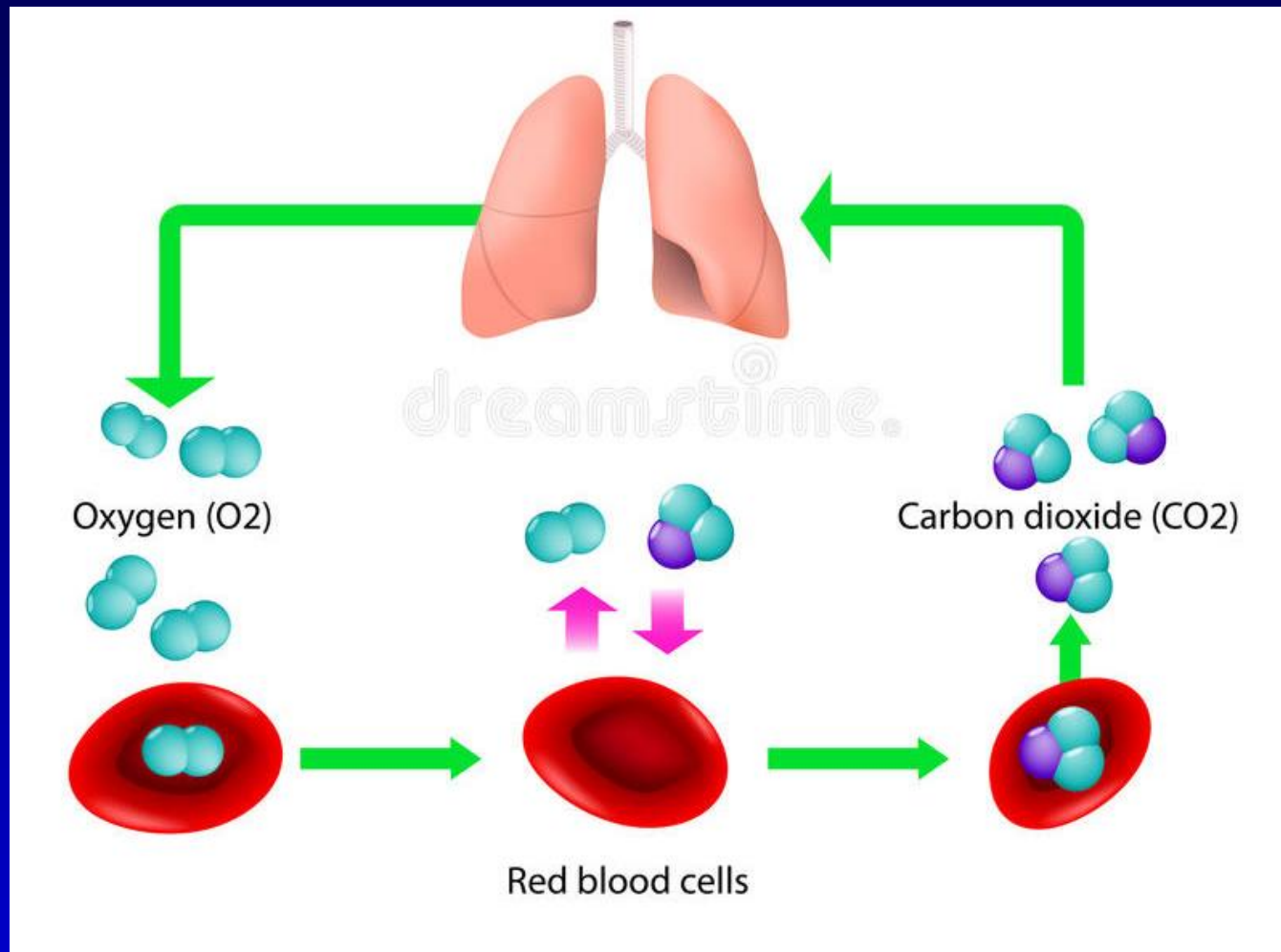
Background

- Symbols and abbreviation
 - ✓ P: pressure
 - ✓ V: volume
 - ✓ \dot{V} : $\frac{dV}{dt}$; gas volume per unit time
 - rate of gas flow
- Conditions for measuring pressure and volume

- STPD**
- Sandard temperature (0 °C)
 - Standard pressure (1 atm; 760 mmHg)
 - Dry air (no humidity)

- BTPS**
- Body temperature (37 °C)
 - Ambient pressure (variable)
 - Air saturated with water vapor at body temp. (47 mmHg)

除了氣體交換(吸氧排二氧化碳)外，
呼吸系統還有那些功能？



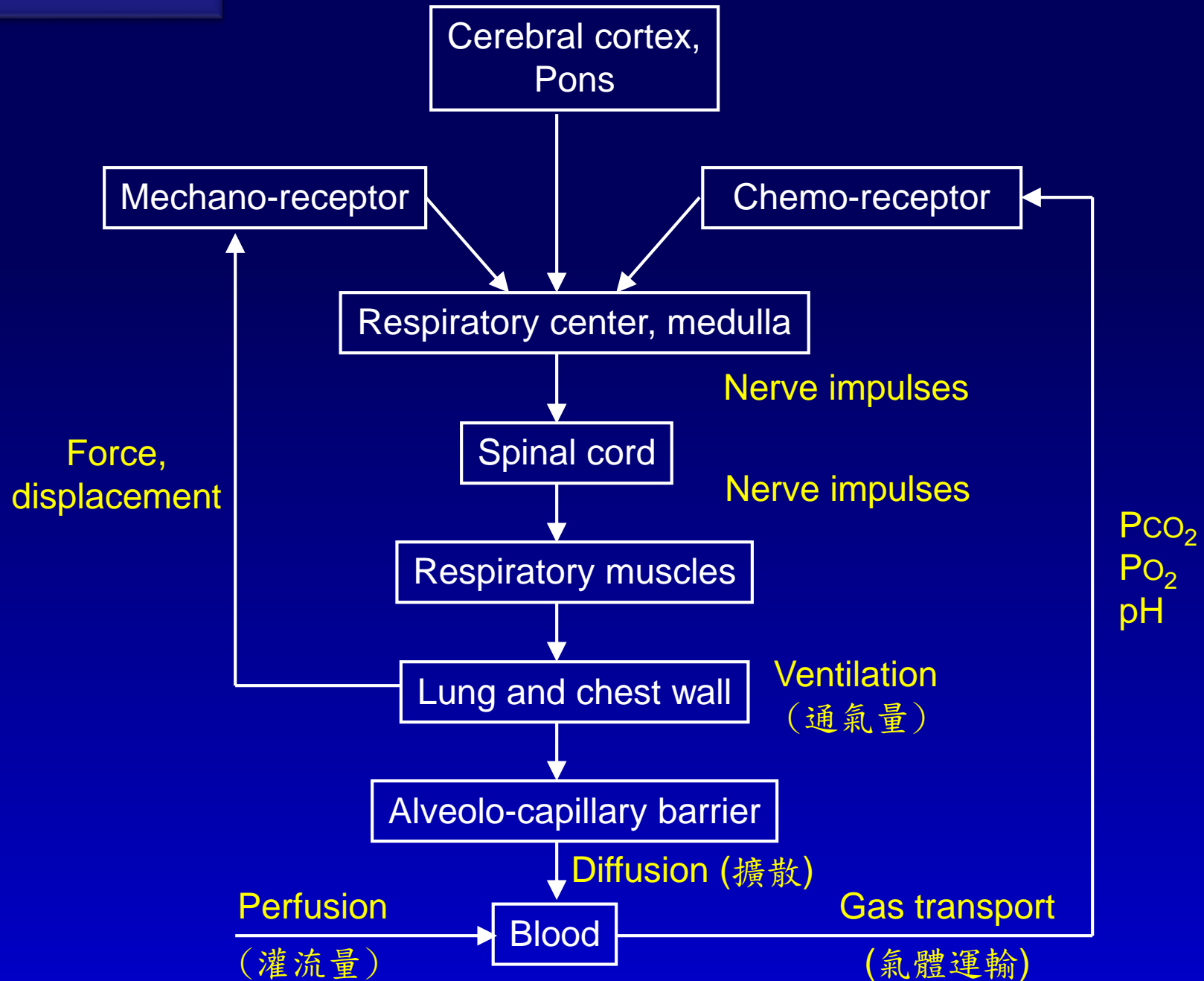


除了氣體交換(吸氧排二氧化碳)外,呼吸系統還有那些功能?

Functions of Respiratory Sys.

1. Supply O_2 to the body for metabolic processes in order to produce energy
2. Remove the byproducts of metabolism (CO_2 & H_2O)
3. Aid in acid/base regulation of blood (acidosis; alkalosis)
4. Temperature regulation
5. Enable vocalizations
6. Stress relief
7. Defend against inhaled foreign matter
8. Enhance venous return — respiratory pump
9. Modify materials passing through the circulatory system
 - ✓ Activates angiotensin II (第二型血管張力素)
 - ✓ Inactivates prostaglandins (前列腺素)

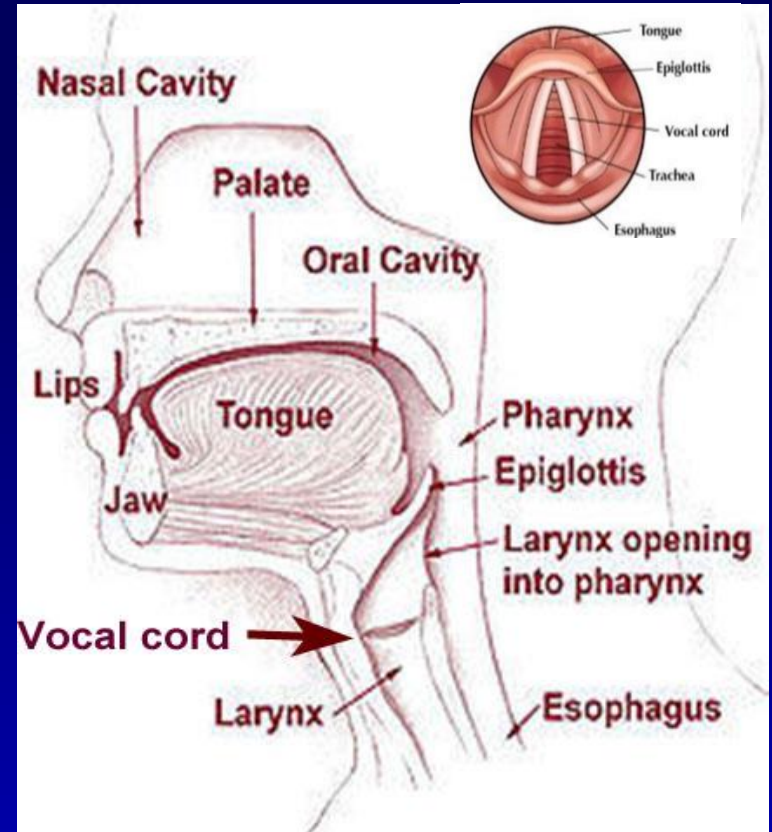
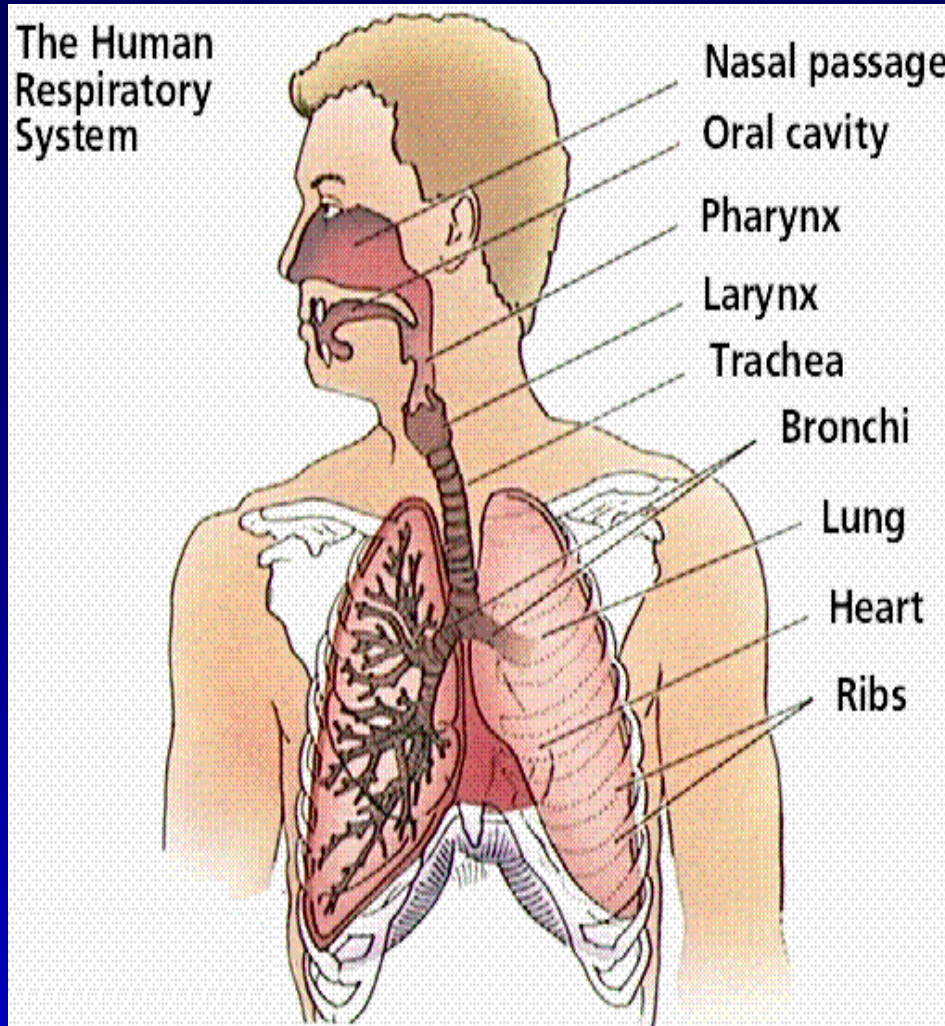
Overview



Outline

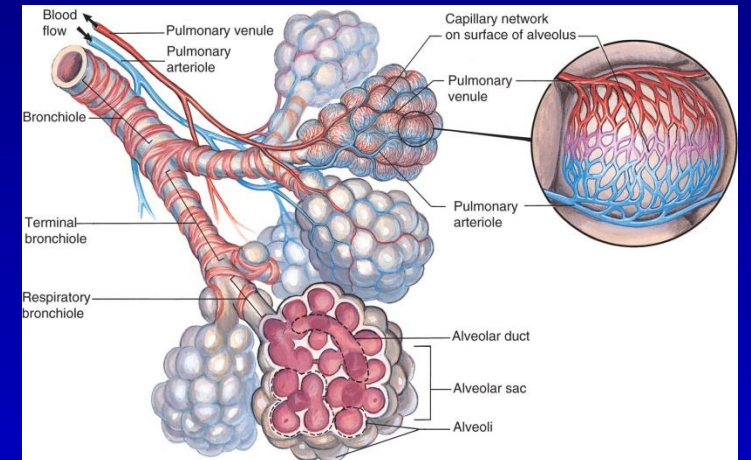
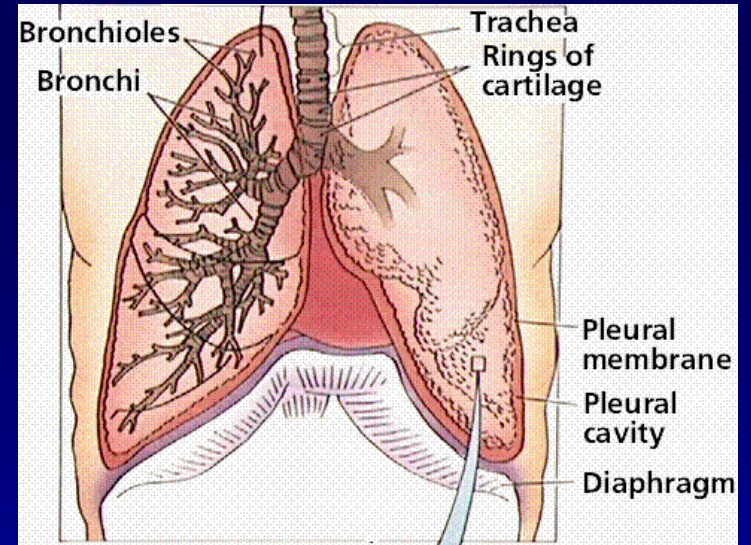
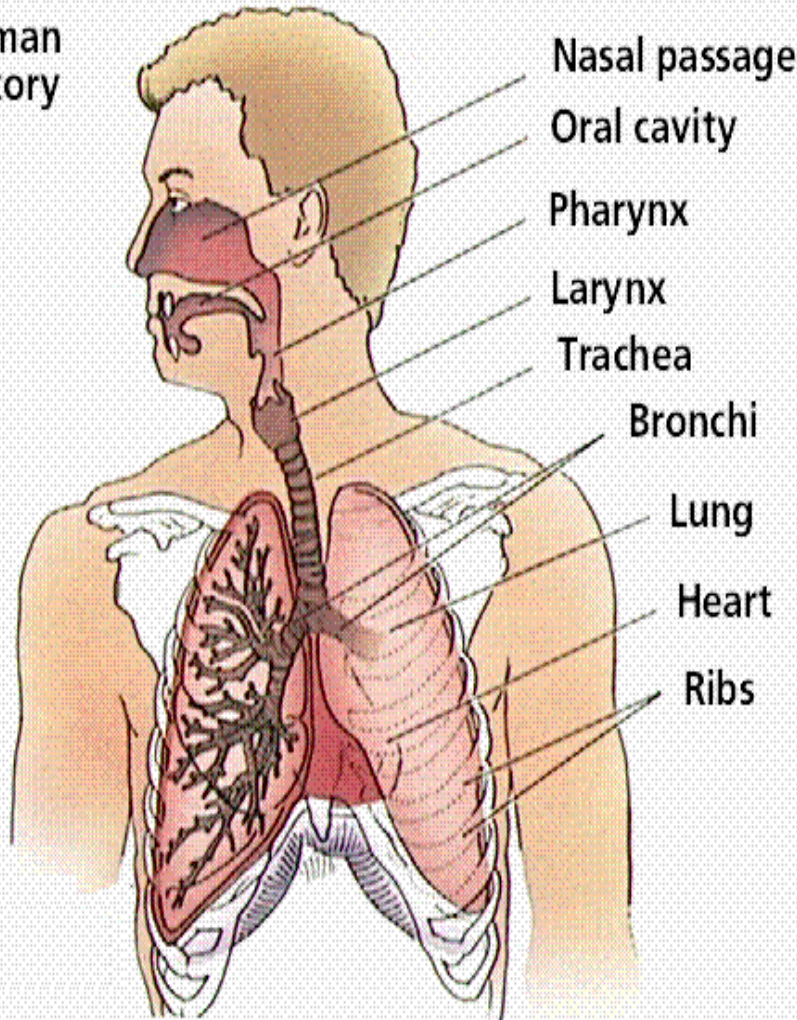
- Background
- **Structure and function (結構與功能)**
- Ventilation
- Perfusion and ventilation/perfusion ratio
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- Chemical control of respiration
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Structure of Respiratory Sys.



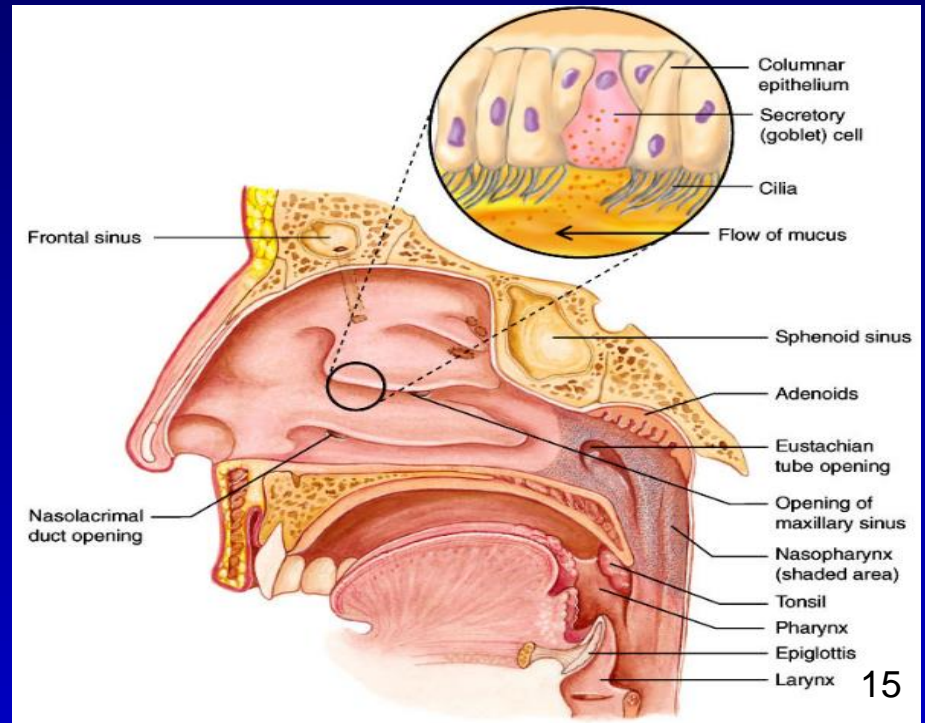
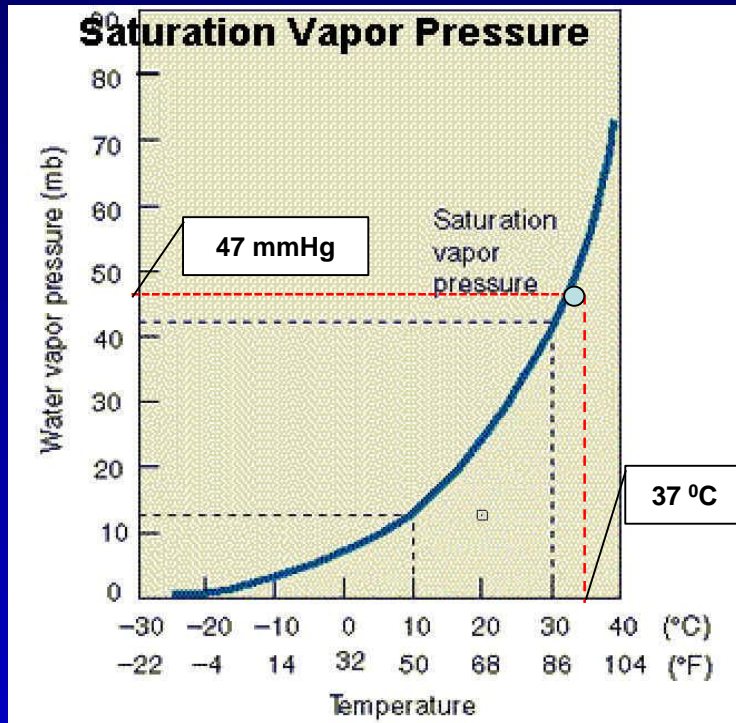
Structure of Respiratory Sys.

The Human Respiratory System



Function of Nasal Passage

- **Clean** the air – mucus and cilia filter airborne particles
- **Warm** the air – become as body temperature (37 °C)
- **Humidify** the air – saturated with H₂O to match vapor pressure (47 mmHg) within the body



Comparison of Airway Structure

Trachea: supported by 15 to 20 **C-shaped** cartilages

- Oriented posteriorly and filled by smooth muscle

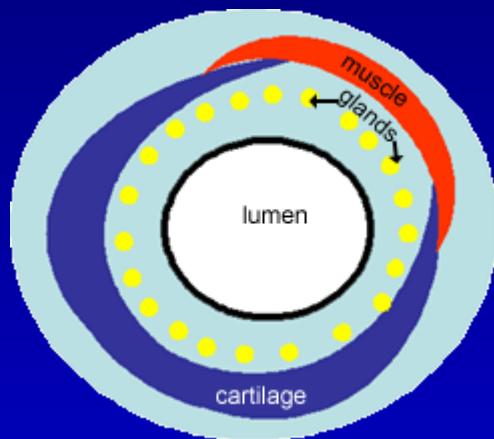
Bronchus: cartilage is in the form of **irregular plates**

- Smooth muscle forms complete rings

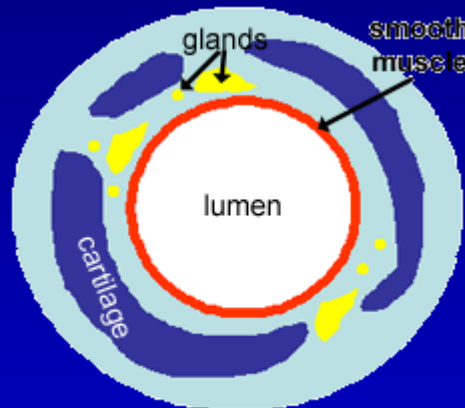
Bronchiole: **no** cartilage

- Smooth muscle layer is relatively thick

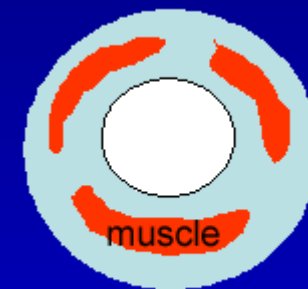
- All three structures are lined by a respiratory mucosa



Trachea

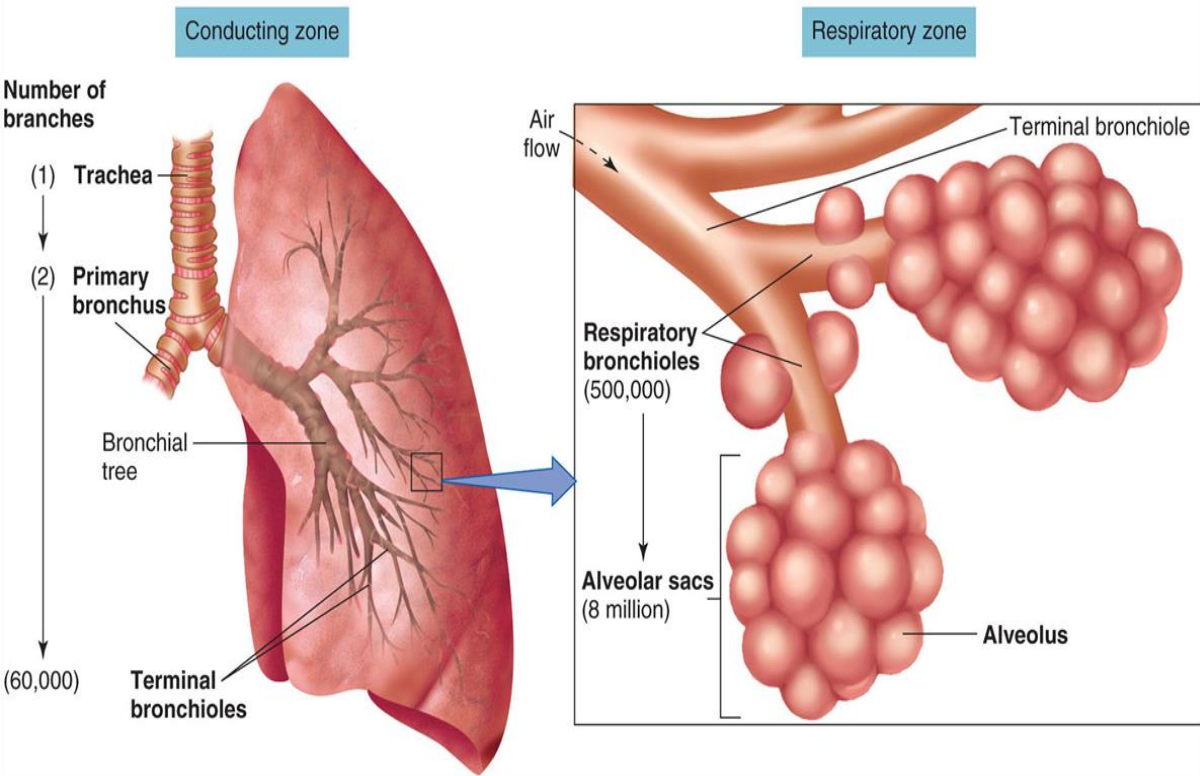


Bronchus



Bronchiole

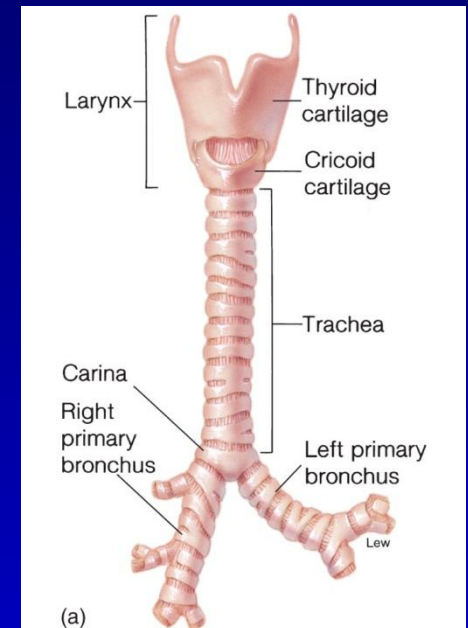
Airways of a Human Lung



	Z	
Conducting zone	Trachea	0
	Bronchi	1
		2
		3
	Bronchioles	4
Transitional and respiratory zones	Terminal bronchioles	5
		↓
		16
	Respiratory bronchioles	17
		18
		19
	Alveolar ducts	20
		21
22		
Alveolar sacs		23

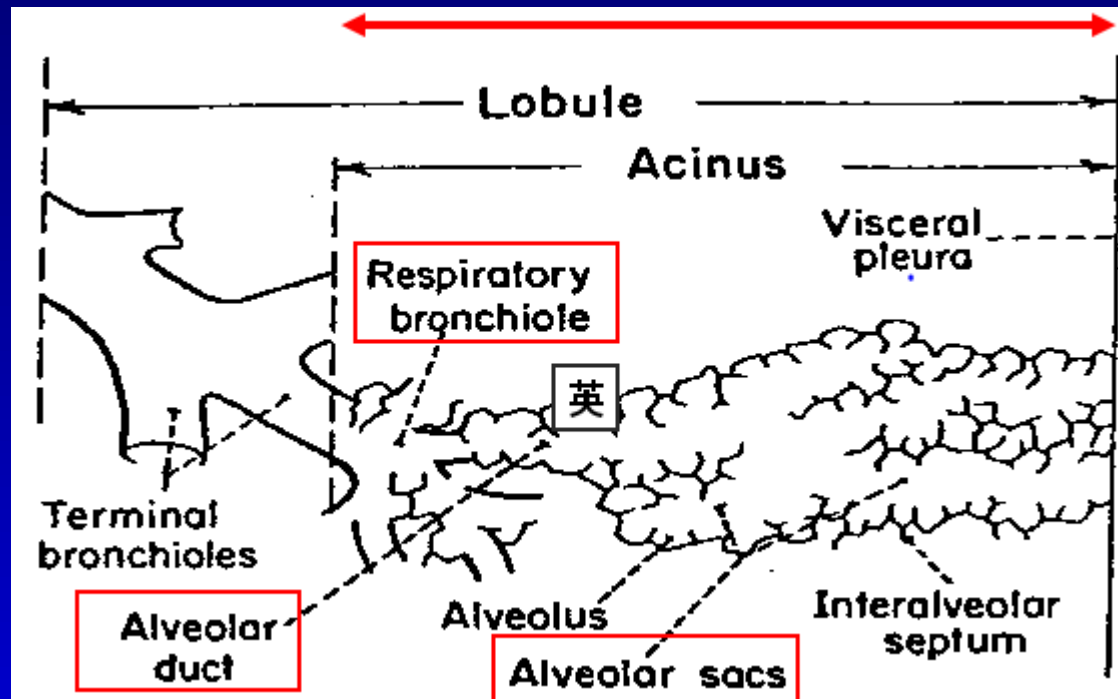
Main Airway Branches & Zones

- Conducting Zone (傳導區) (1-16 generations)
(No gas exchange)
 - ✓ Trachea (1)
 - R + L main bronchi (R't is less sharply angled)
 - lobar bronchi
 - segmental bronchi
 - bronchioles
 - terminal bronchioles (6×10^4)
- The first 16 branches are responsible for
 - ✓ Conducting air movement (by pressure)
 - ✓ Cleansing the air



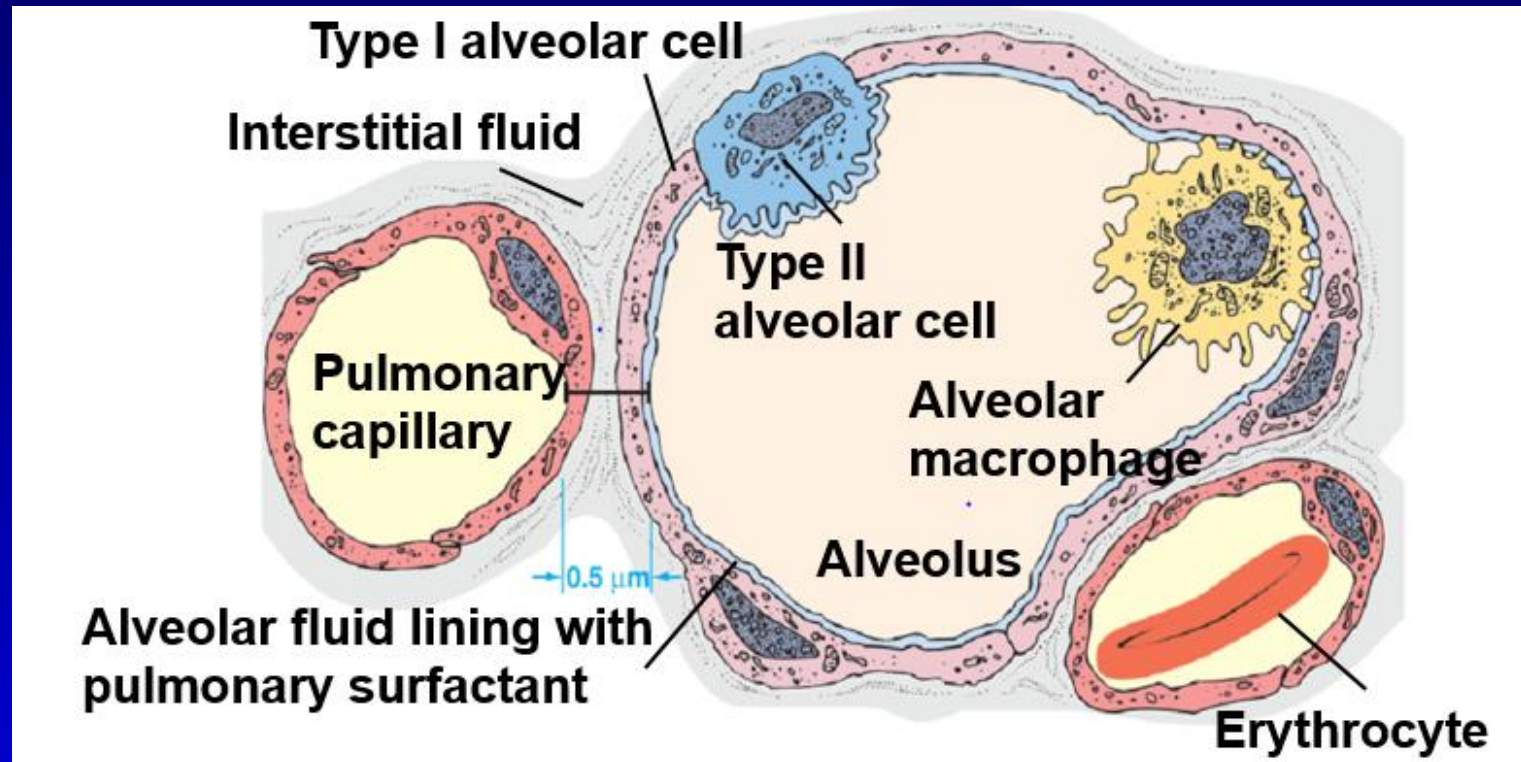
Main Airway Branches & Zones

- Respiratory Zone (呼吸區) (17-23 generations)
 - ✓ Gas movement by **diffusion** (擴散)
 - ✓ Respiratory bronchioles
 - alveolar ducts
 - alveolar sacs (8×10^6)

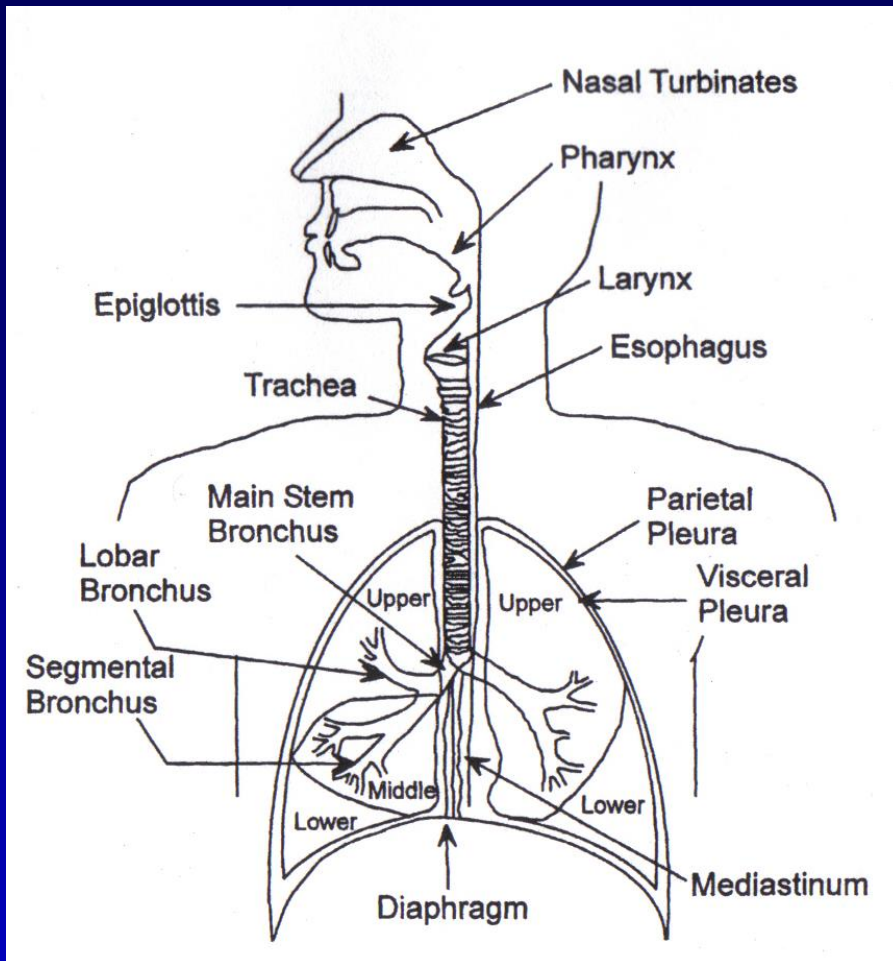


Alveoli

- Thin-walled, inflatable sacs
- Formed by a single layer of flattened Type I alveolar cells
- **Type II alveolar cells** secretes **pulmonary surfactant**
 - This substance facilitates lung expansion
- Encircled by pulmonary capillaries, offering tremendous surface area for gas exchange by **diffusion**

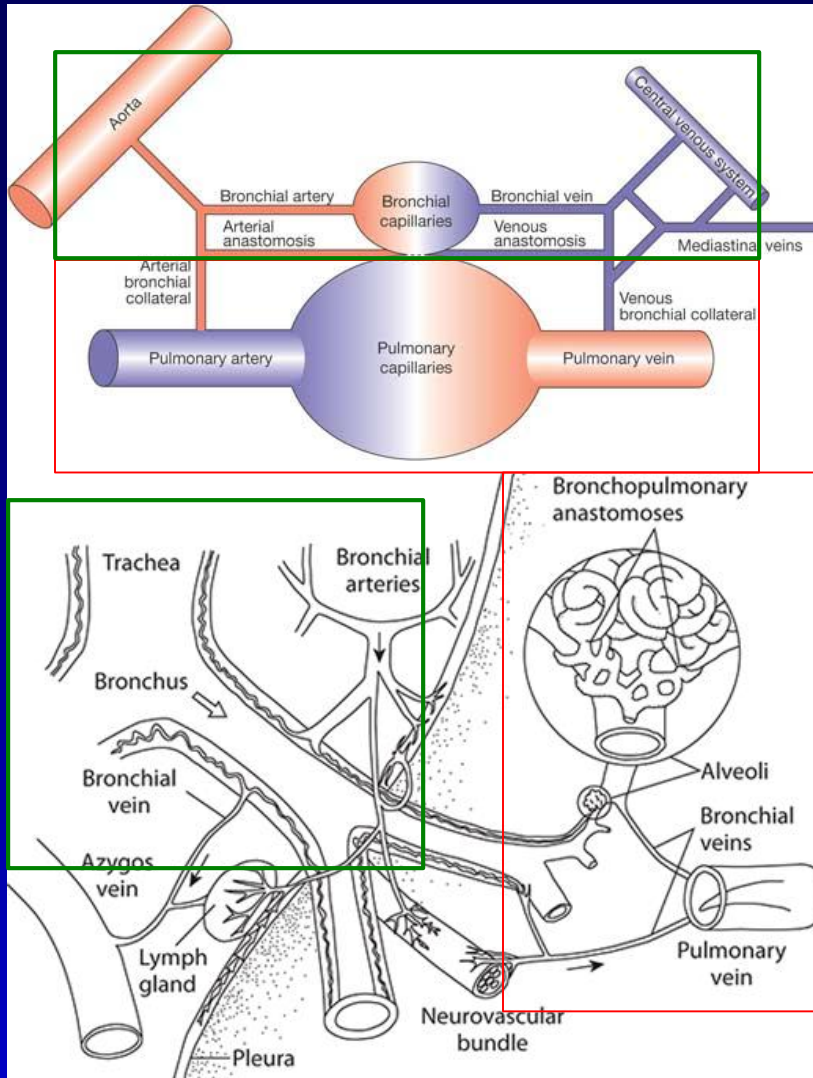


The Human Lung



- Includes **airways** and **parenchyma (基質)**
- **Parenchyma**: connective tissues and other non-airway components
- Parenchyma provides mainly the **elastic recoil force**

Blood Supply of Lungs

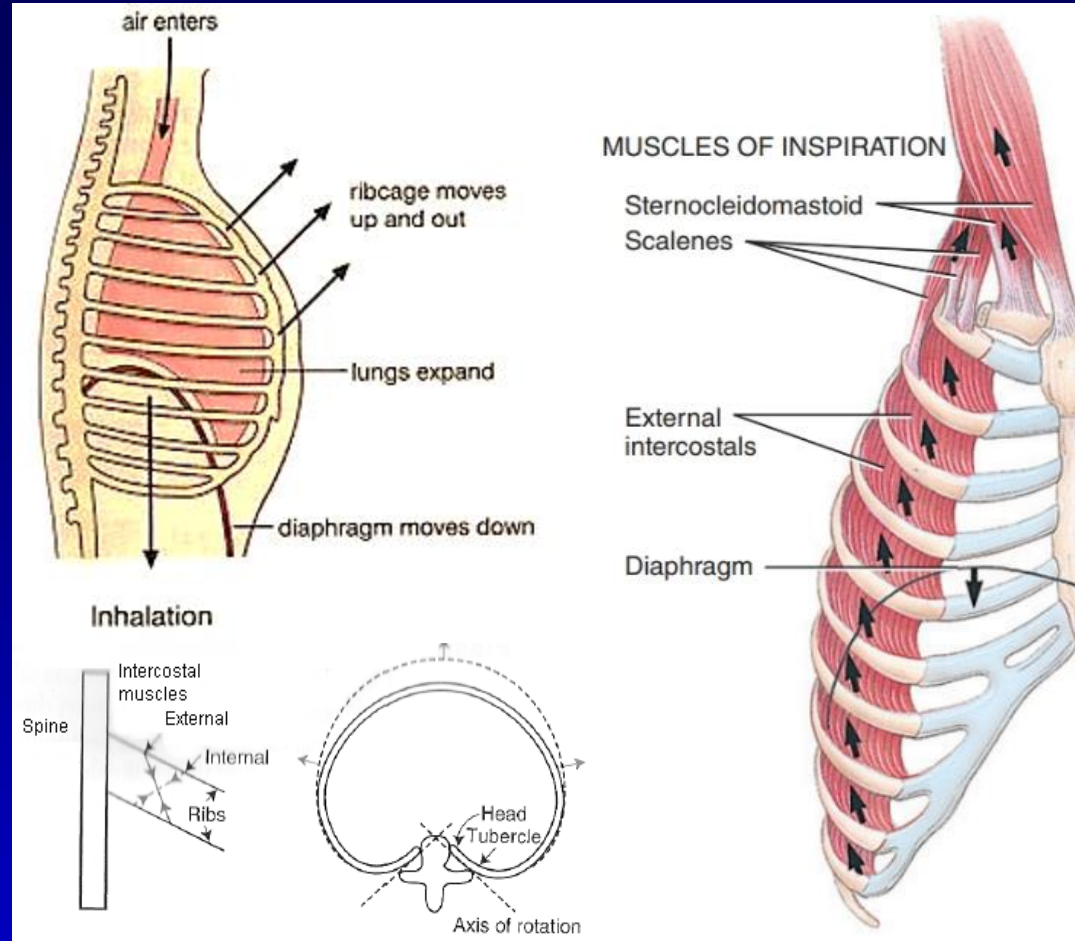


- **Pulmonary circulation:** gas exchange with the **alveoli** in the parenchyma → **respiratory zone**
- **Bronchial circulation:** main nutrient and O₂ supply for the **airways** → **conducting zone**

Respiratory Muscles

Inspiration

- **Diaphragm**: ↑longitudinal dimension of thorax
 - ✓ Major m. for inspiration, innervated by **phrenic n.**
 - hiccup
- **External intercostal m.**: ↑ ant-post. dimension of thorax
- **Accessory m. of respiration**: sternomastoids & scalenes



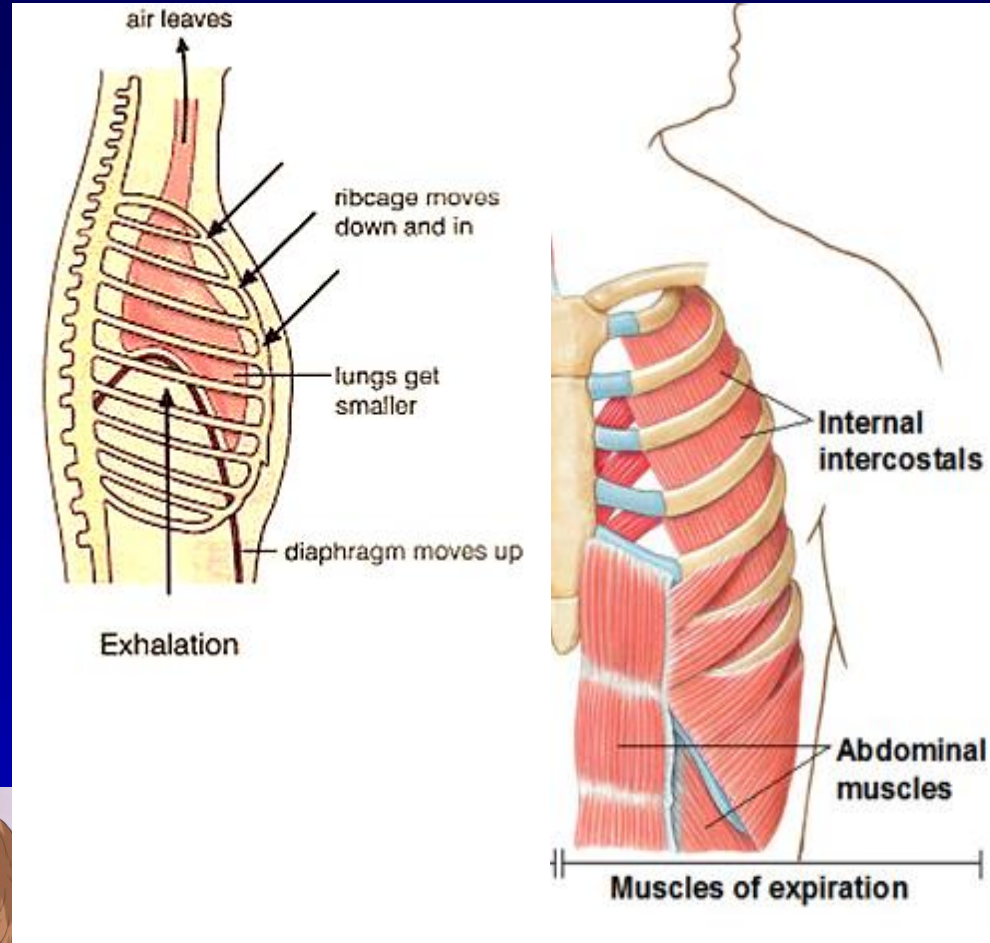
Respiratory Muscles

Expiration:

Under **normal resting** condition, expiration is a **passive** process, relying on the **elastic recoil of the lung and chest wall**

During forced expiration:

- **Internal intercostal m.:** ↓ ant-post. dimension of thorax
- **Abdominal m.**



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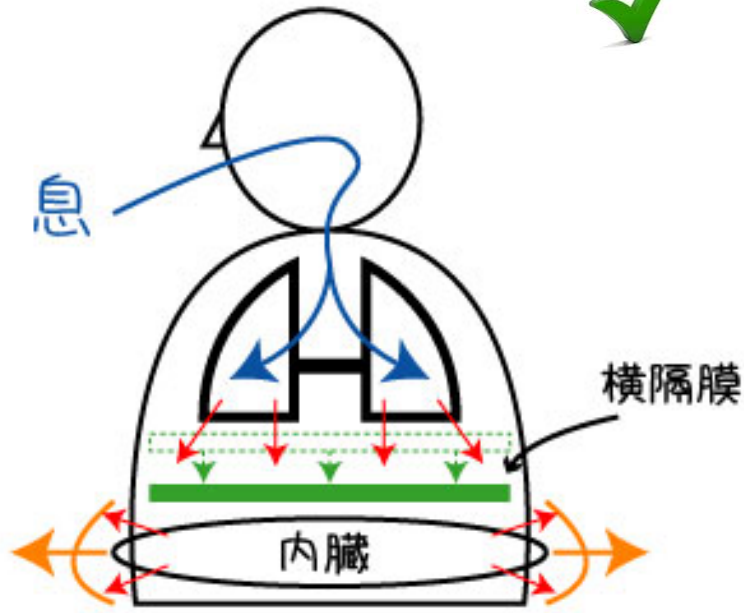


那種呼吸方式比較有效率？

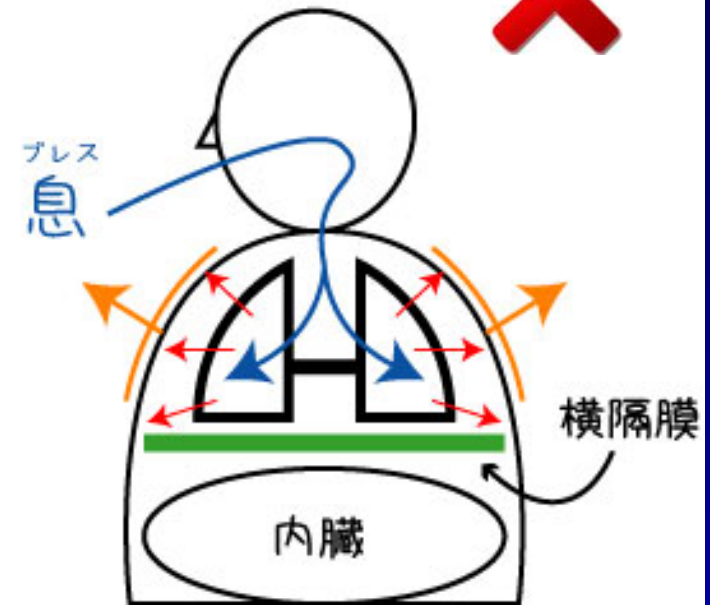
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腹式呼吸

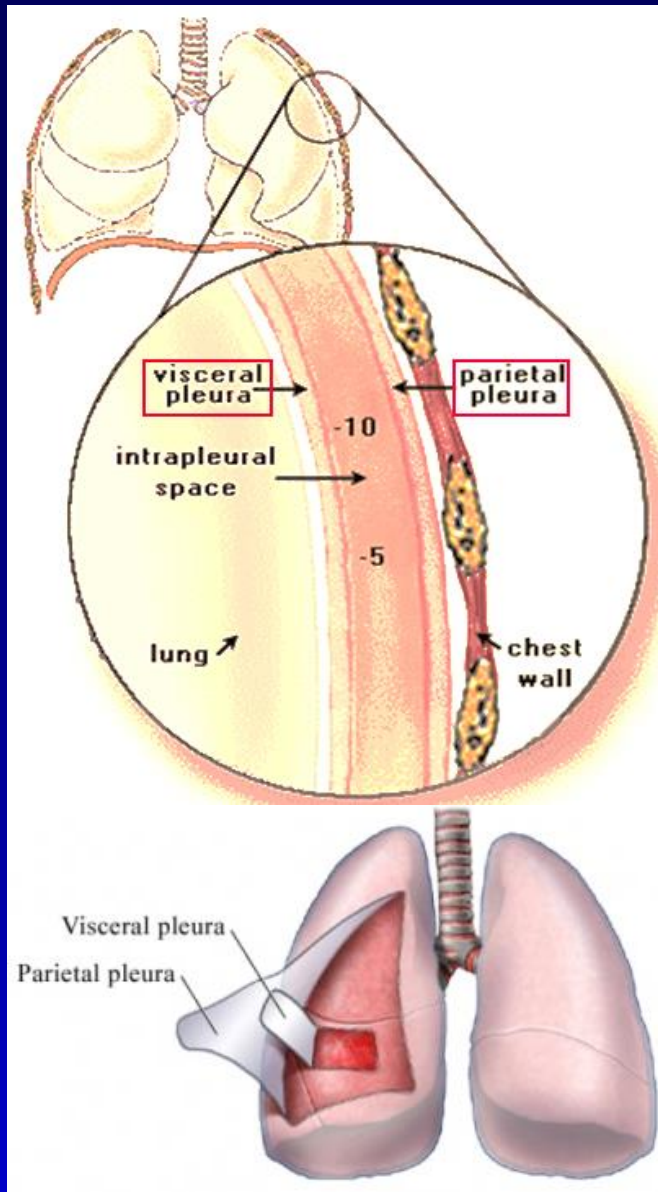


胸式呼吸



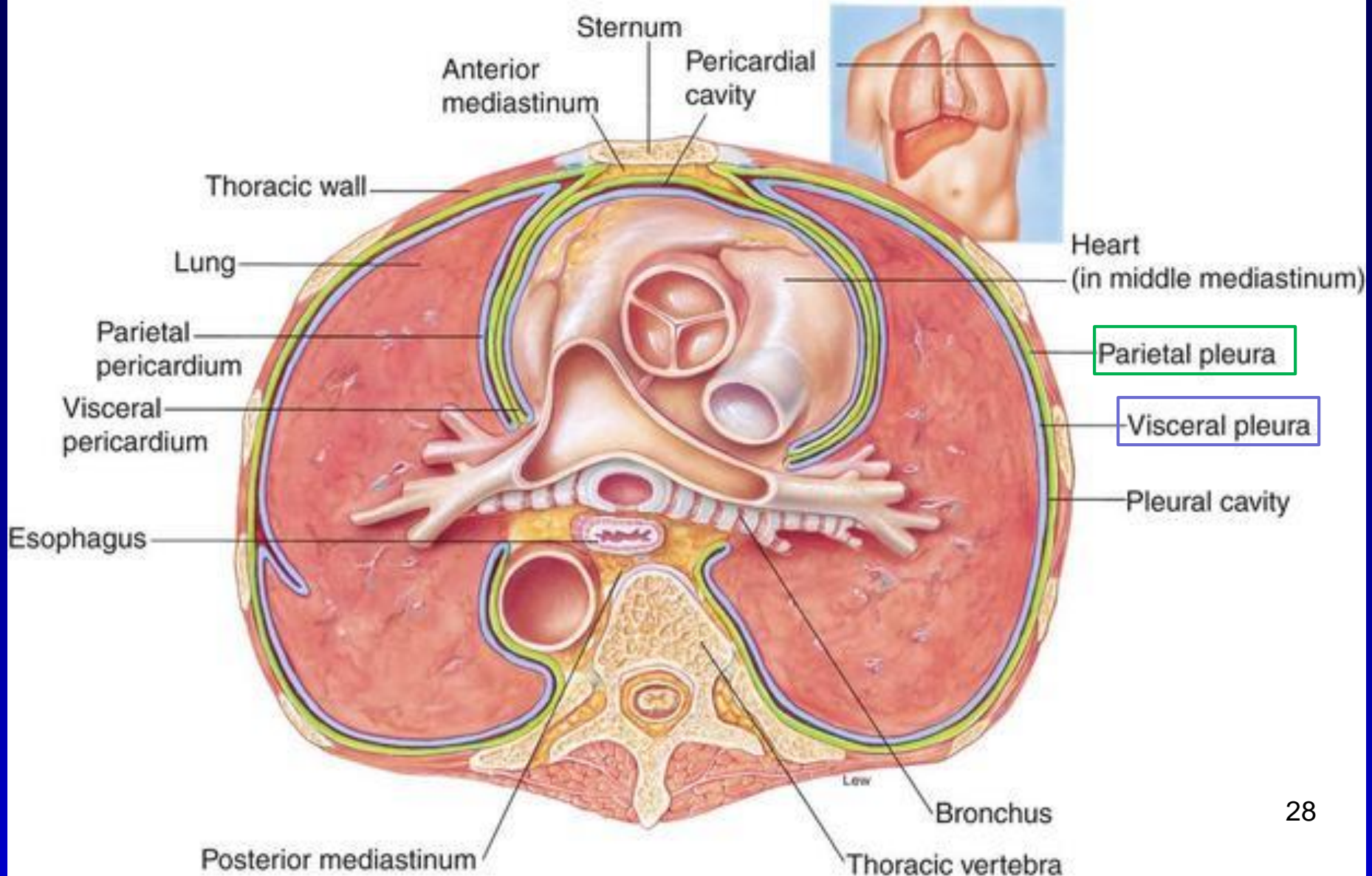
- ✓ 橫膈為最主要吸氣肌
- ✓ 當腹肌收縮，使吐氣吐的完全(較多廢氣排出)，下次吸氣即能吸較多的新鮮空氣

Pleural layers



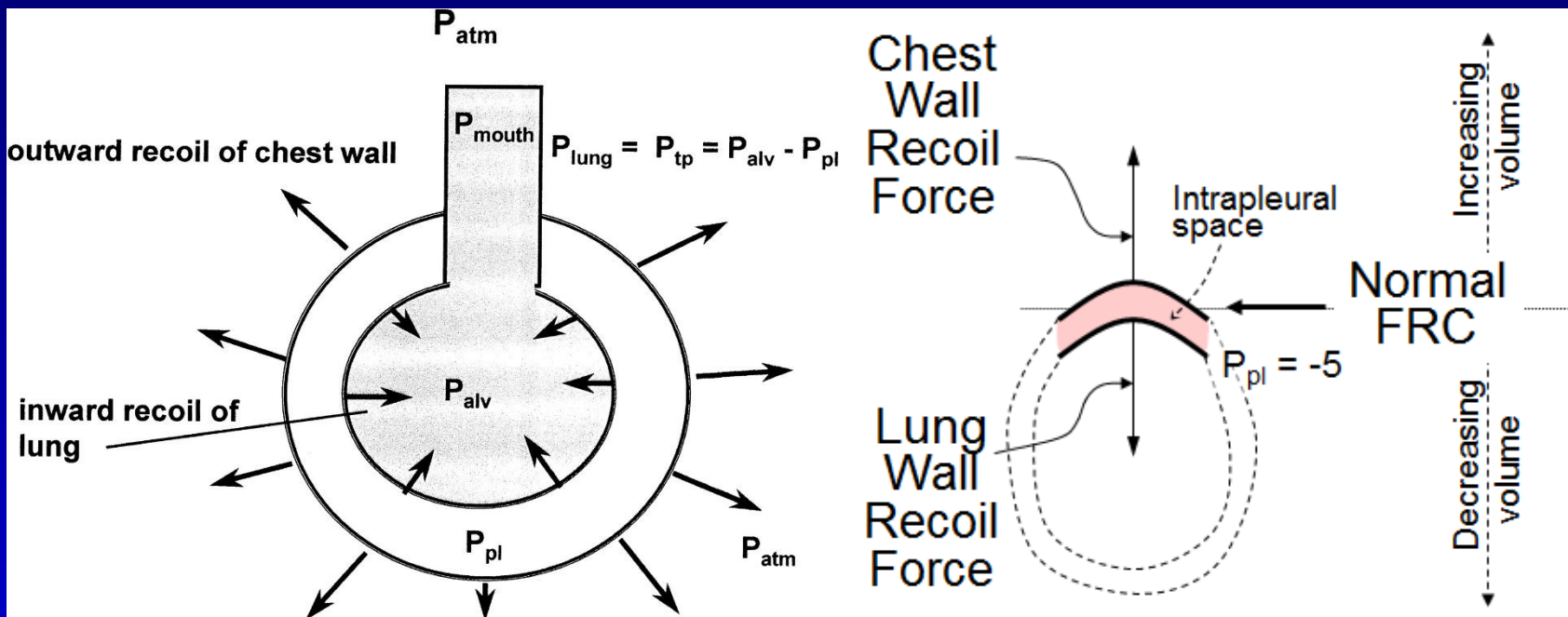
- **Visceral pleura (臟側肋膜)**: covers lungs
- **Parietal pleura (壁側肋膜)**: covers inside of chest wall
- **Intrapleural space (肋膜間腔)**: space between visceral pleura and parietal pleural
- * **Pleural coupling**: lungs move with movement of chest wall

Cross Section of the Thoracic Cavity



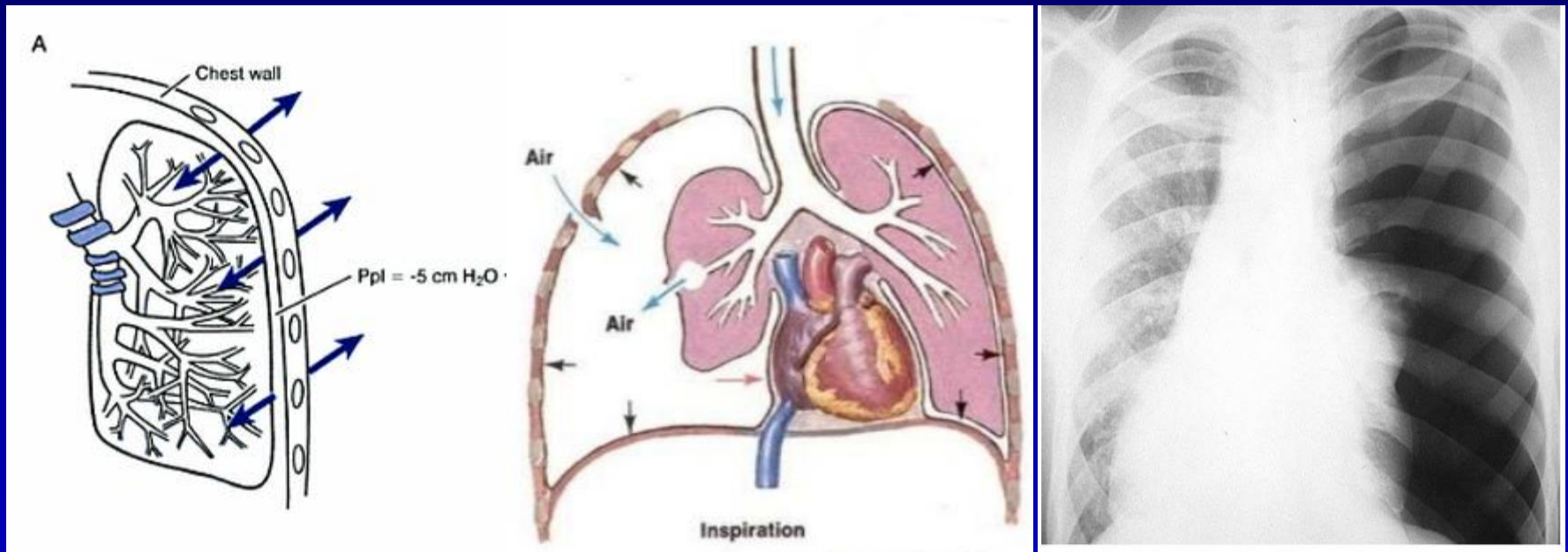
Intrapleural Space

- Intrapleural pressure (P_{pl}): -4~5 cmH₂O at end-expiration (FRC, functional residual capacity)
 - ✓ Lungs have a tendency to collapse
 - ✓ Chest wall has a tendency to expand act in opposite direction
- **Negative** Intrapleural pressure



Pneumothorax

- **Pneumothorax (氣胸)**: air is introduced to the fluid layer between the pleura causing them to come apart ($P_{pl} = 0$)
 - ✓ Loss of **pleural coupling**

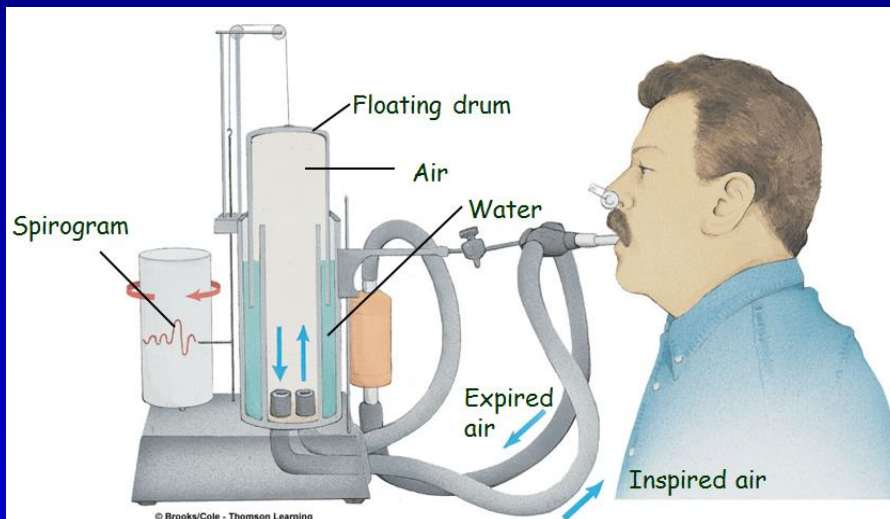


Outline

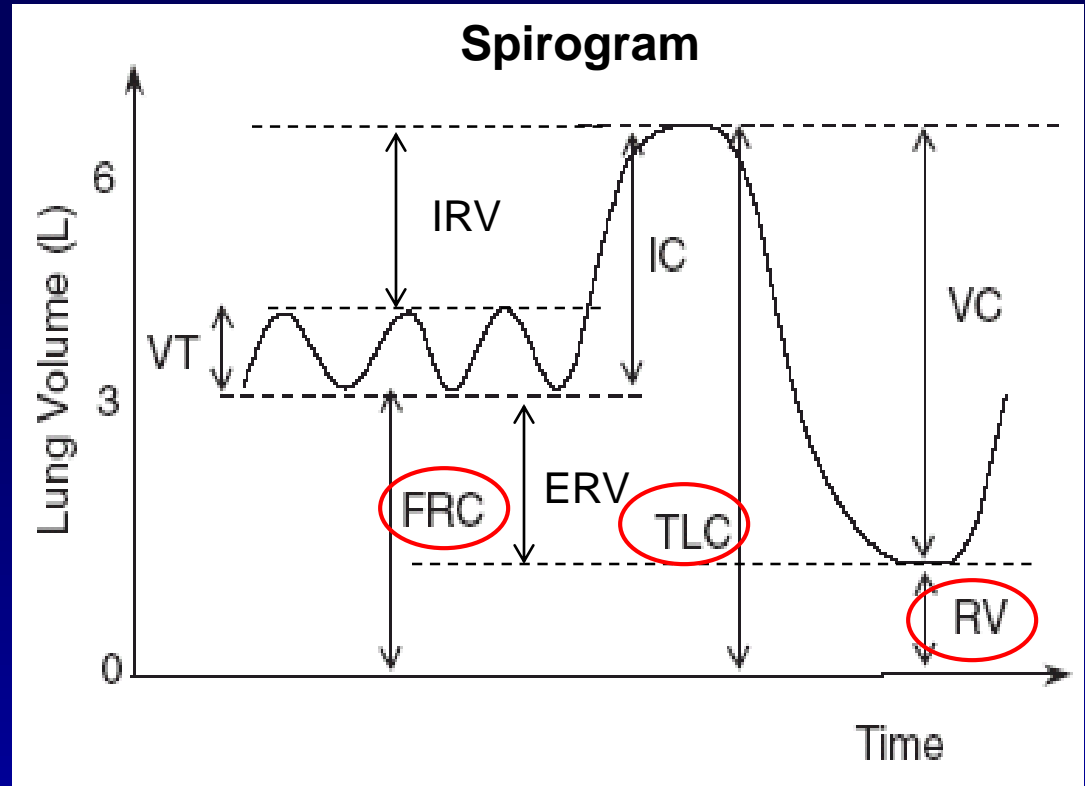
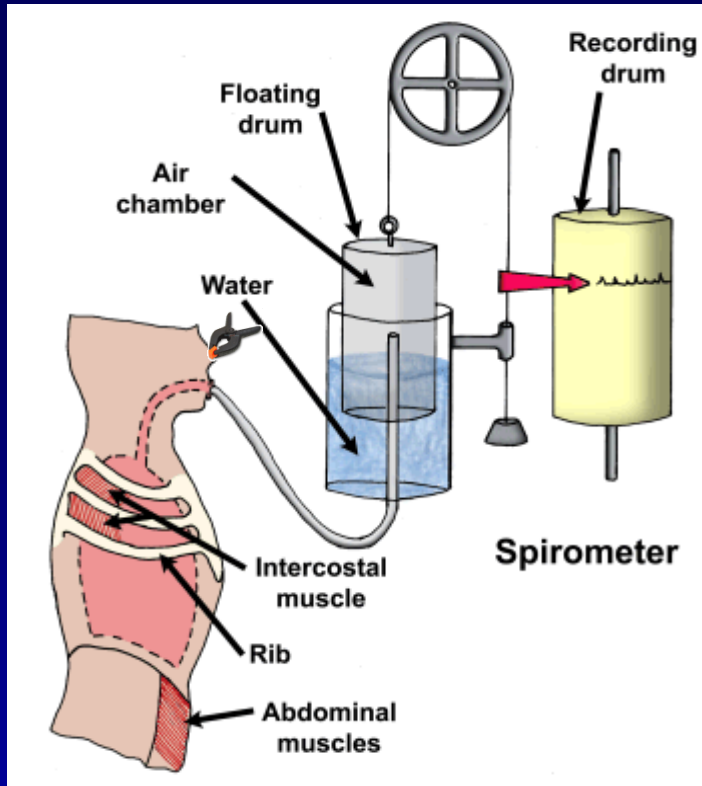
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Instrument for Measuring Lung Vol.

- **Spirometer** (肺活量計): a device for measuring lung volumes (except *functional residual capacity*, *residual volume*, *total lung capacity*)
- **Body plethysmograph** (身體體積描記器): a method of obtaining the absolute volume of air within one's lungs
- **Pneumotachograph** (呼吸速度描記器): a device for measuring airflow velocity (Vol. is calculated by integration of flow)



Spirometer, Lung Volumes and Capacities

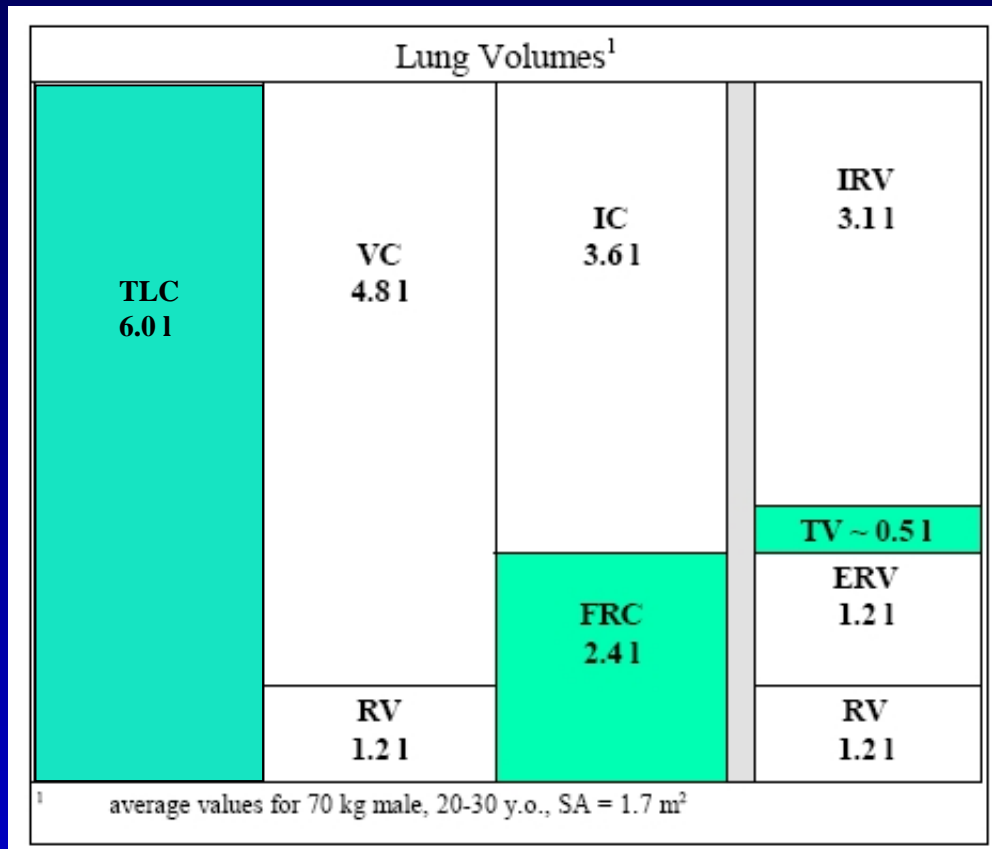


V_T : tidal volume (潮氣容積)
IRV: inspiratory reserve volume
(吸氣儲備容積)
ERV: expiratory reserve volume
(吐氣儲備容積)
IC: inspiratory capacity (吸氣量)

VC: vital capacity (肺活量)
RV: residual volume (殘餘容積)
TLC: total lung capacity (總肺量)
FRC: functional residual capacity
(功能肺餘量)

Lung Volumes and Capacities

- Capacity (量) = the summation of volume (容積)
- Primary lung volume: RV, ERV, V_T , IRV
- Secondary derived capacities: TLC, VC, IC, FRC



- $IC = IRV + V_T$
- $FRC = ERV + RV$
- $VC = IRV + V_T + ERV$
- $TLC = IC + FRC$
 $= IRV + V_T + ERV + RV$

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什麼時候正常吐氣結束？

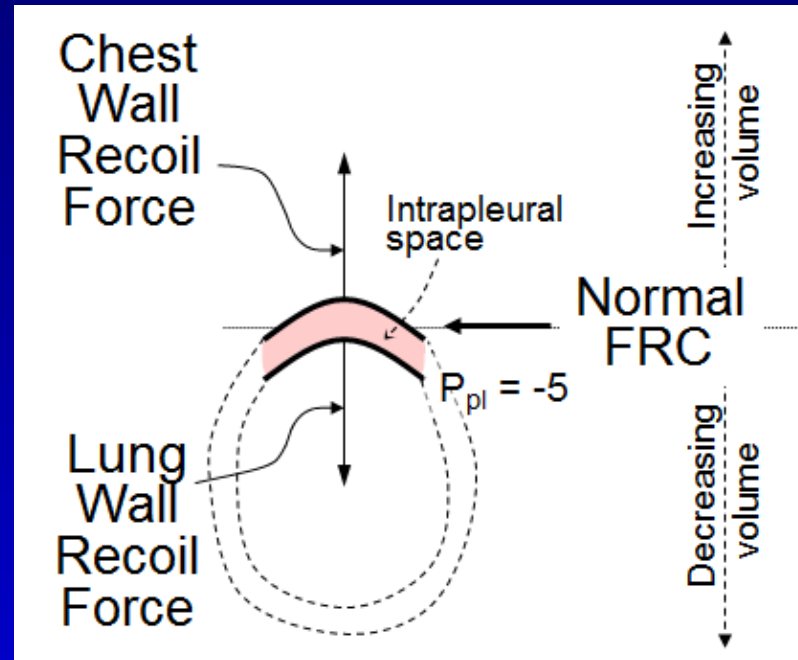
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什麼時候正常吐氣結束？

A) 肺中的氣體完全吐光

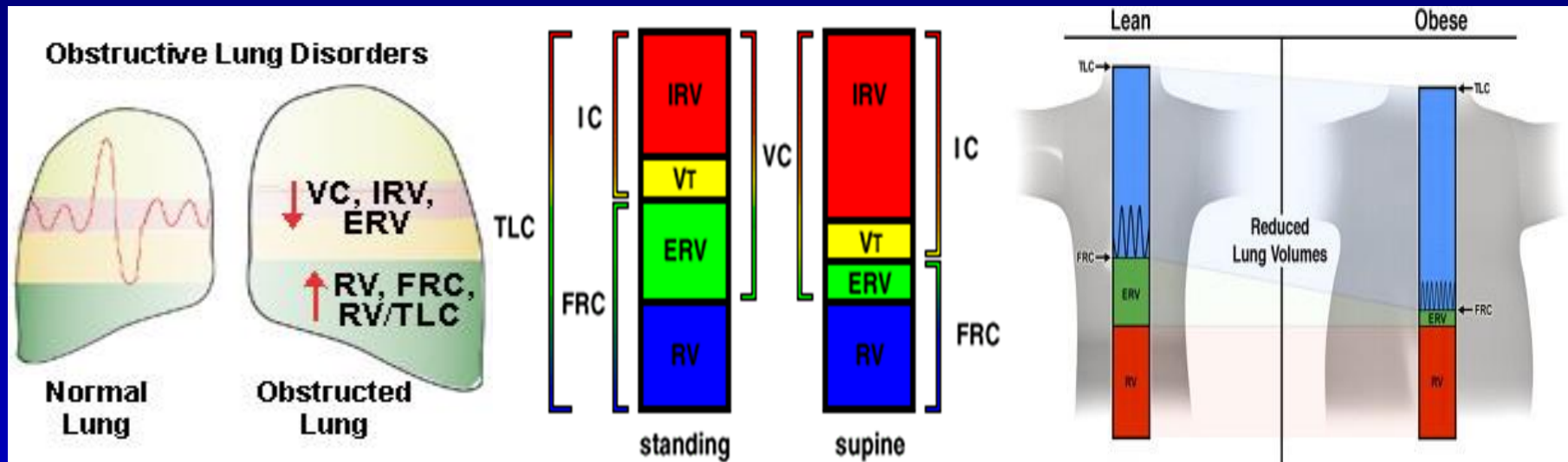


B) 肺向內縮的彈力等於胸腔壁向外擴張的力



Functional Residual Capacity

- The vol. of gas left in the lungs at the end of normal tidal expiration
- Determined by a balance between the inward elastic forces of the lung and the outward forces of the chest wall
- Factors ↓ FRC: supine, obesity, pregnancy, anesthesia
- Factors ↑ FRC: height, obstructive lung disease

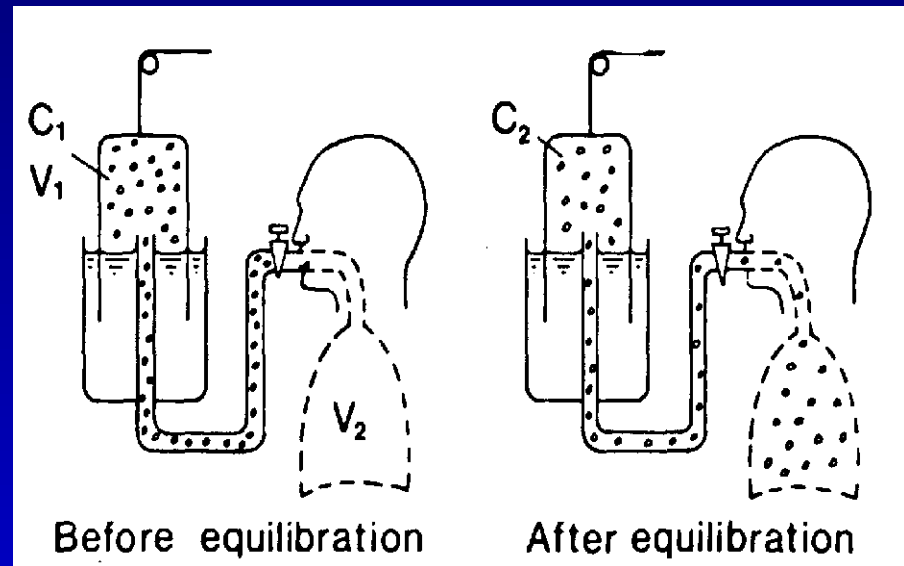
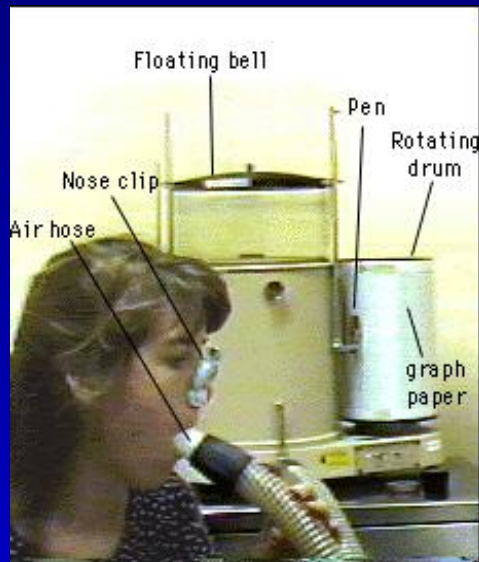


Measurement of FRC

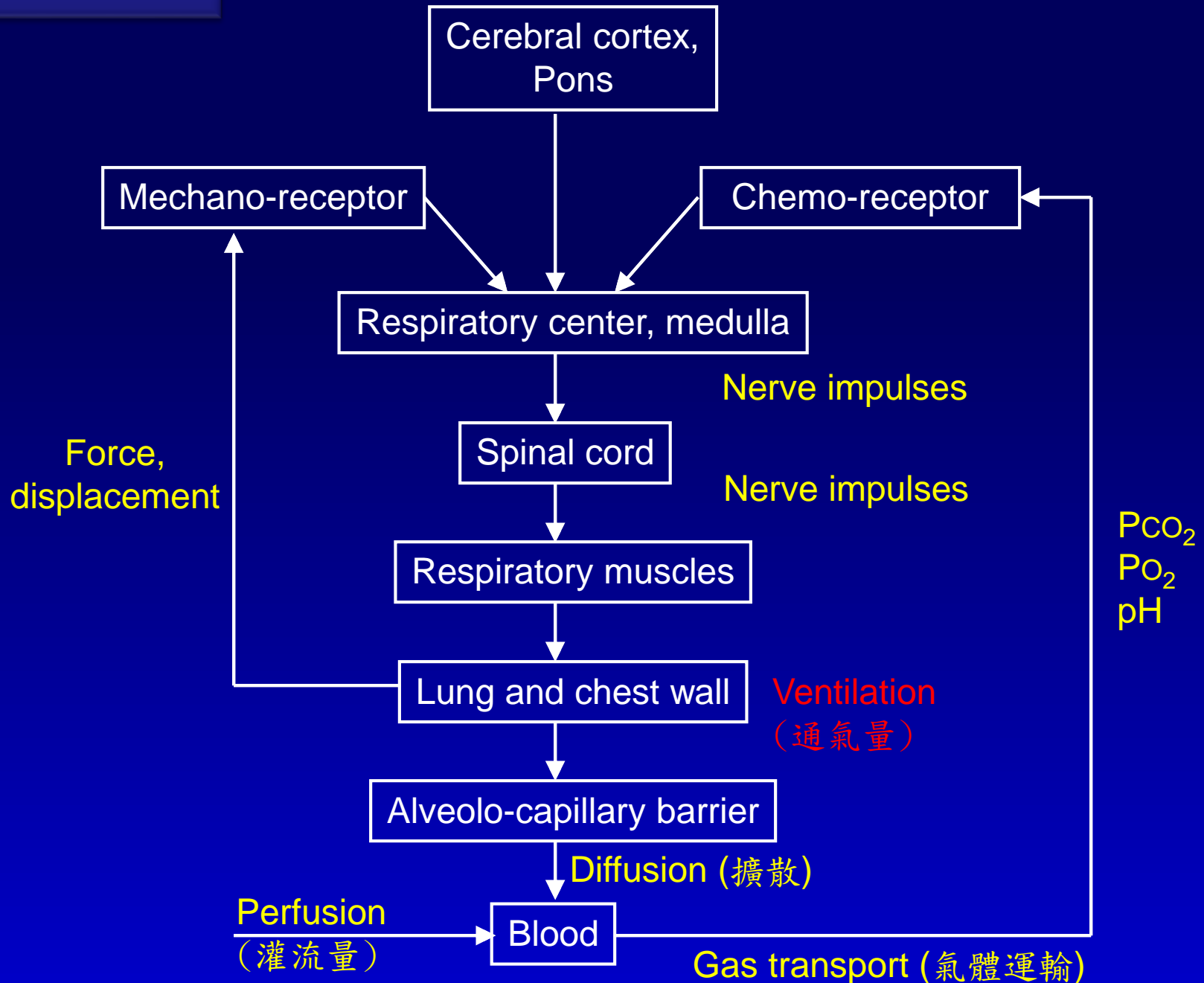
Method 1: Closed circuit helium dilution

- Gas: insoluble inert gases (e.g. helium or neon)
- Principle: law of conservation of mass
→ check concentration change

$$C_1 V_1 = C_2 (FRC + V_1)$$



Overview

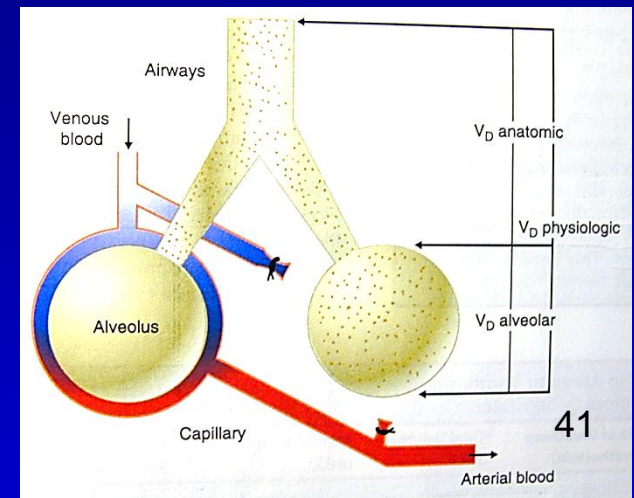


Ventilation (通氣量)

- The movement of air in and out of the resp. system
- **Minute ventilation** (\dot{V}): **volume** of gas leaving (V_E) or entering (V_I) lungs **per min**
- \dot{V} (ml/min) = V_T (ml) x resp. rate (1/min)
E.g., $\dot{V}_E = V_T \times f$
 $= 500 \times 15 = 7500$ ml/min
- Changes in **respiratory rate** cause proportionate changes in **minute ventilation** (\dot{V}_E)
- **NOT ALL** inspired air is gas exchanged
- **Dead space** (死腔; V_D): area where there is **no** gas exchange, e.g. 1-16 generation of airway

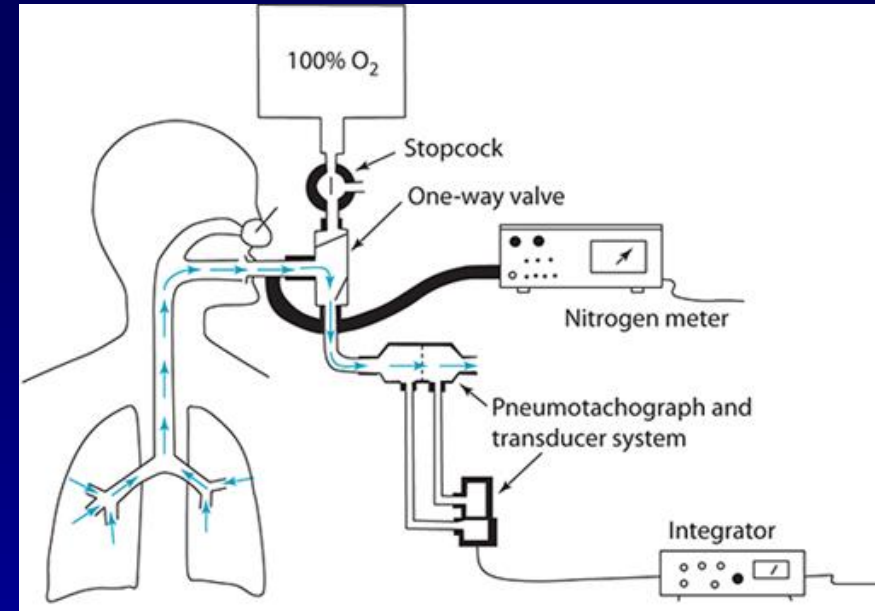
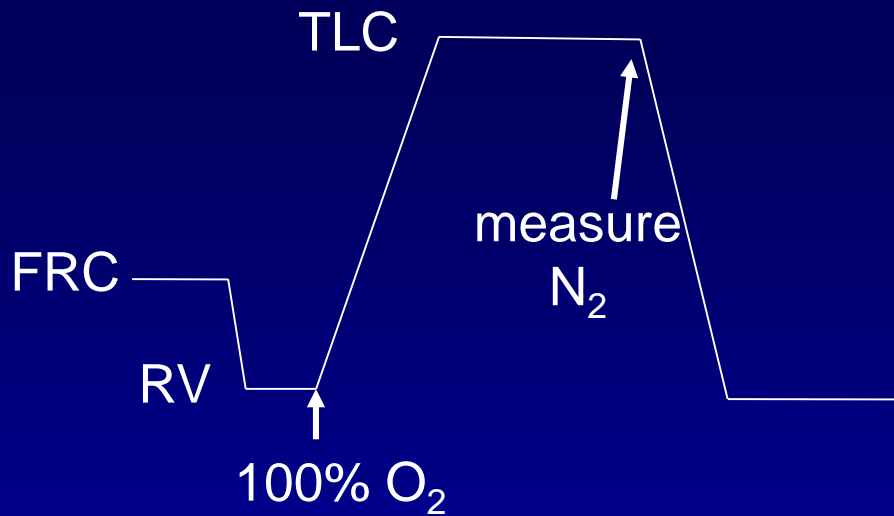
Dead Space (死腔)

- **Anatomic dead space** (V_D^{Anat}): the volume of the conducting airways in which no gas exchange takes place
- **Alveolar dead space** (V_D^{Alv}): inspired gas which enters alveoli (respiratory zone), however is **ineffective in arterializing** mixed venous blood
 - ✓ Alveoli with no perfusion or reduced perfusion
- **Physiologic dead space** (V_D^{Phys}): the volume of gas that does not eliminate CO_2
 - ✓ $V_D^{Phys} = V_D^{Anat} + V_D^{Alv}$
- Methods to measure dead space
 - ✓ Anatomic V_D : Fowler's method
 - ✓ Physiological V_D : Bohr's method

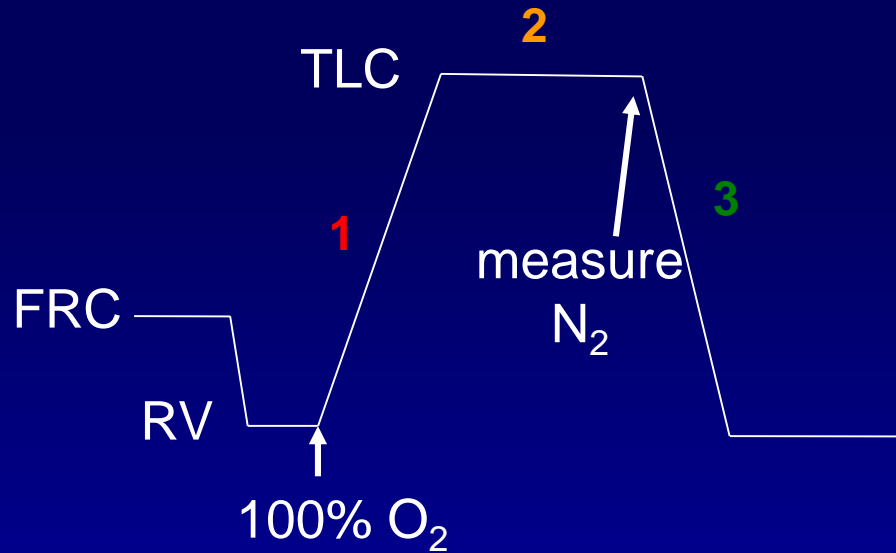


Fowler's Method:

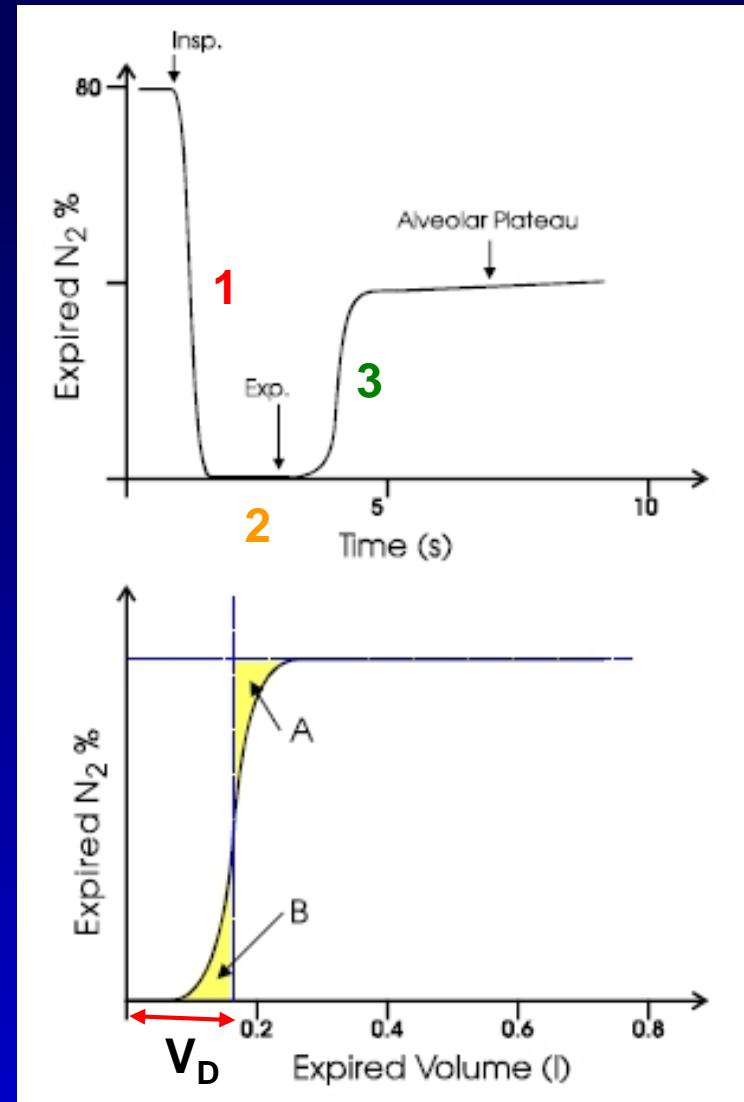
Single-Breath Nitrogen Washout



Fowler's Method: Single-Breath Nitrogen Washout

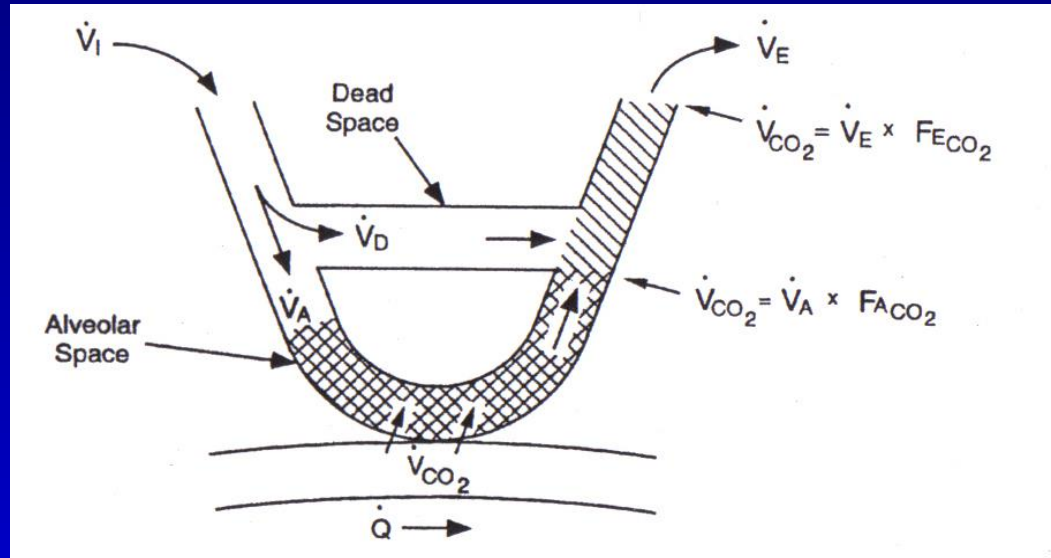


- **Anatomic dead space** is the exhaled volume to the point of **transition** between **dead space** and **alveolar gas**
- $V_D^{Anat} \sim 2.2$ ml/kg of body weight



Bohr's Method: Conservation of Mass

- Principle: V_D does not contribute to expired CO_2
- $\dot{V}_T \times F_{ECO_2} = \dot{V}_A \times F_{ACO_2}$
- $\dot{V}_A = \dot{V}_T - \dot{V}_D$
 $\rightarrow \dot{V}_T \times F_{ECO_2} = (\dot{V}_T - \dot{V}_D) \times F_{ACO_2}$
 $\rightarrow \frac{\dot{V}_D}{\dot{V}_T} = \frac{F_{ACO_2} - F_{ECO_2}}{F_{ACO_2}}$ (Bohr Equation)



Dalton's Law

- Atmosphere contains a mixture of gases
 - ✓ O₂ (20.93%); N₂ (78.09%); CO₂ (0.03%); inert gas
- Dalton's law:

$$P_x = F_x \times P_{\text{total}}$$

✓ In STPD, $P_{\text{O}_2} = F_{\text{O}_2} \times P_{\text{atm}} = 0.2093 \times 760 = 159 \text{ mmHg}$

✓ In BTPS, $P_{\text{O}_2} = F_{\text{O}_2} \times (P_{\text{atm}} - P_{\text{H}_2\text{O}})$
 $= 0.2093 \times (760 - 47) = 150 \text{ mmHg}$

- The sum of gases must equal barometric pressure
- $P_{\text{H}_2\text{O}} = 47 \text{ mmHg}$ at body temp.

Bohr's Method (2)

- $\frac{\dot{V}_D}{\dot{V}_T} = \frac{F_{ACO_2} - F_{ECO_2}}{F_{ACO_2}}$ (Bohr Equation)

Dalton's law:

- $P_x = F_x \times P_{atm}$ [dry] (STPD)
- $P_x = F_x \times (P_{atm} - P_{H_2O})$ [wet] (BTPS)

$$\rightarrow \dot{V}_D = \frac{P_{ACO_2} - P_{ECO_2}}{P_{ACO_2}} \times \dot{V}_T$$

Example:

$$P_{ACO_2} = 40 \text{ mmHg}; P_{ECO_2} = 28 \text{ mmHg}$$

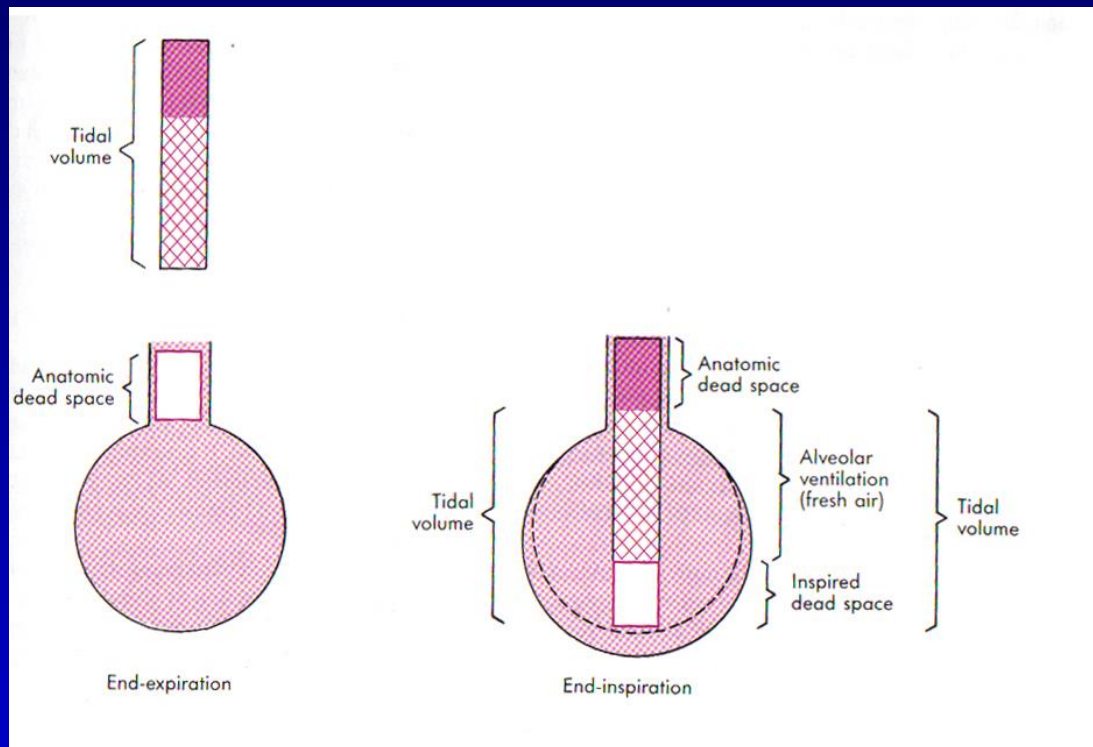
$$V_D = \frac{40 - 28}{40} \times 500 = 150 \text{ ml}$$

Alveolar Ventilation

- **Alveolar vol.:** the volume of fresh gas **entering** the alveoli and effective in **arterializing** mixed venous blood

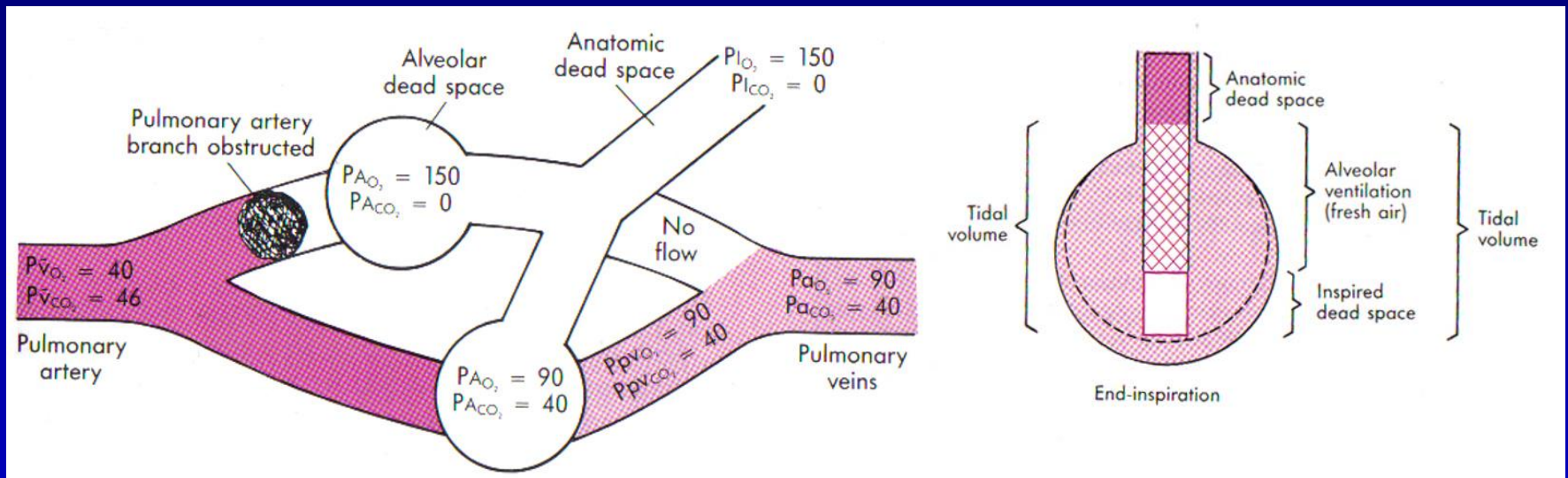
$$V_A = V_T - V_D^{\text{Phys}}$$

V_A : alveolar vol.



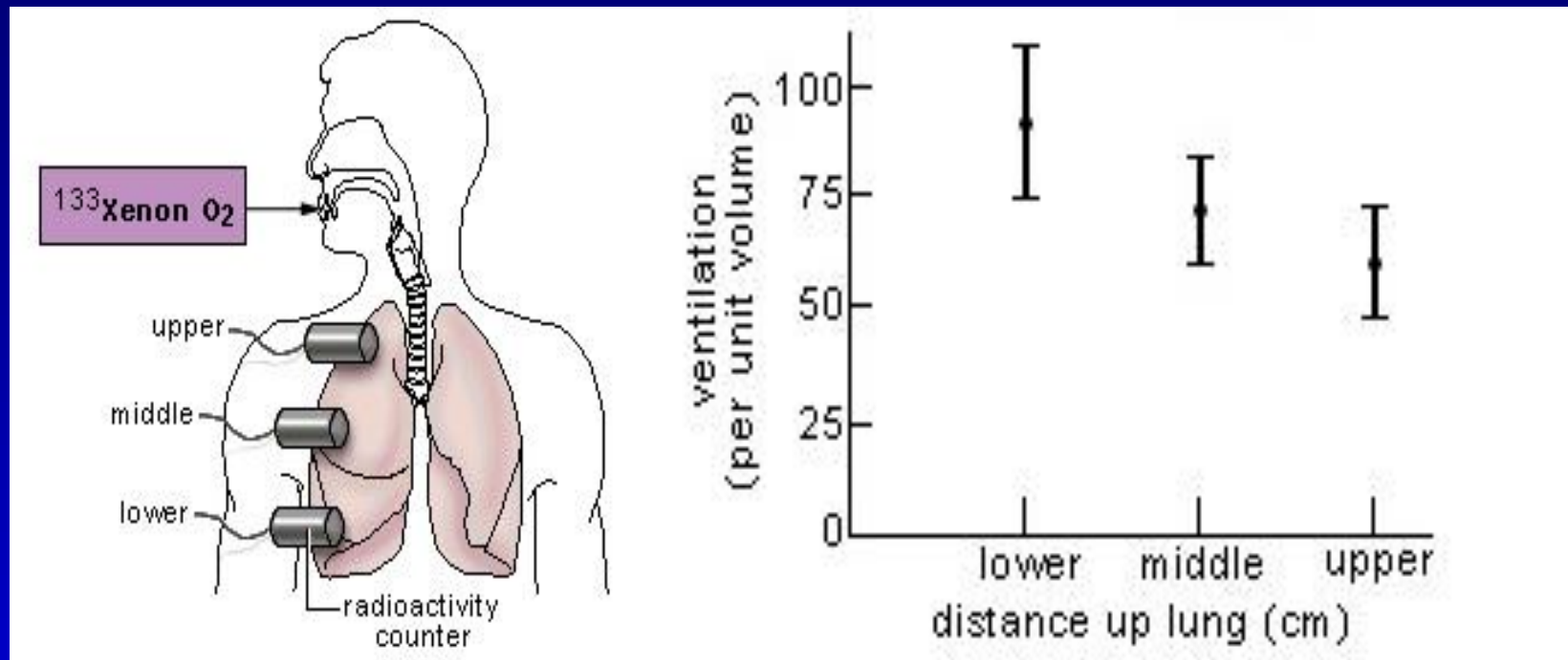
Alveolar Ventilation

- $V_D^{\text{Phys}} = V_D^{\text{Alv}} + V_D^{\text{Anat}}$
- In normal supine man, $V_D^{\text{Alv}} \sim 0 \rightarrow V_D^{\text{Phys}} \approx V_D^{\text{Anat}}$
- $\dot{V}_A = \dot{V}_T - \dot{V}_D^{\text{anat}} = (V_T - V_D) \times f$
- Changes in **respiratory rate** cause proportionate changes in **alveolar ventilation (\dot{V}_A)**



Uneven Ventilation in Upright Position

- Regional differences in airway **resistance** & **compliance** → different alveolar filling time
- In the upright position, ventilation is maximal at the lung bases, decreasing linearly to the apices



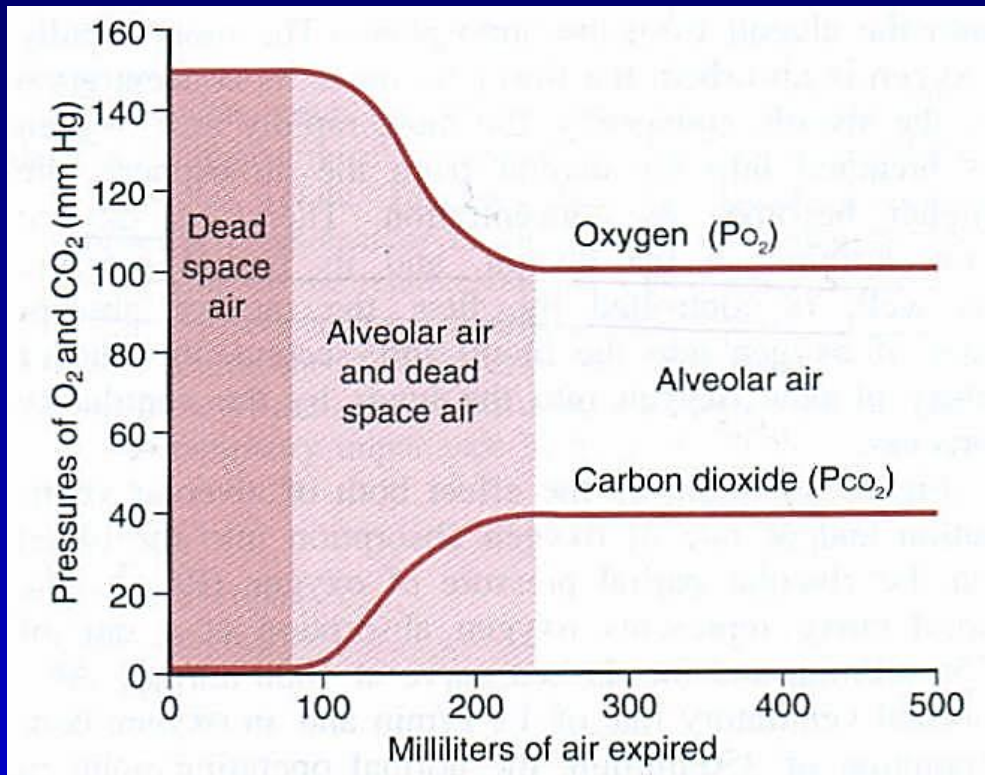
Partial Pressures of Gases in Various Parts

- In the alveoli, the percentage of oxygen decreases and CO₂ increases, changing the partial pressure of each

	Inspired air	Alveolar air
H ₂ O	Variable	47 mmHg
CO ₂	000.3 mmHg	40 mmHg
O ₂	159 mmHg	105 mmHg
N ₂	601 mmHg	568 mmHg
Total pressure	760 mmHg	760 mmHg

O₂ and CO₂ Concentrations in Exhaled Gas

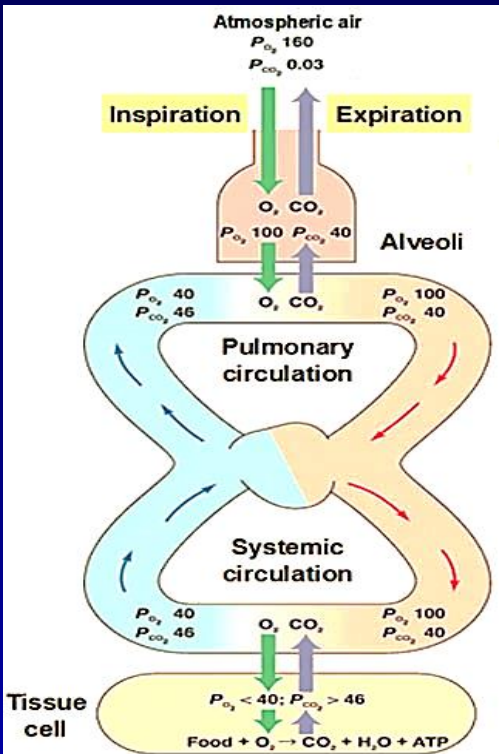
- A good way to evaluate alveolar gas content in normal subjects is to **examine gas coming out late in exhalation** after the gas in the conducting airways has been cleared



先進去的氣體，後出來
後進去的氣體，先出來



Overview of P_{O_2} and P_{CO_2}



Ambient

$$PO_2 = 160 \text{ mmHg} = 760 \times 0.21$$

$$PCO_2 = 0 \text{ mmHg}$$

Inspired

$$PO_2 = 150 \text{ mmHg}$$

$$PCO_2 = 0 \text{ mmHg}$$

$$P_{I}O_2 = F_{I}O_2 \times (P_B - 47)$$

$$= 0.21 \times (760 - 47)$$

Alveolar

$$P_{A}O_2 = 100 \text{ mmHg}$$

$$P_{A}CO_2 = 40 \text{ mmHg}$$

Mixed
venous

$$P_vO_2 = 40 \text{ mmHg}$$

$$P_vCO_2 = 46 \text{ mmHg}$$

gas exchange

End
capillary

$$P_aO_2 = 100 \text{ mmHg}$$

$$P_aCO_2 = 40 \text{ mmHg}$$

Pulmonary
artery

Pulmonary
vein

slido



改變呼吸方式可改變那種氣體的分壓？

① Start presenting to display the poll results on this slide.

Hyper-, Hypo-ventilation & Hyperpnea

- Changes in **alveolar ventilation** (\dot{V}_A) cause **reciprocal changes** in **alveolar P_{CO_2}**
- **Hyperventilation**: an increase in alveolar ventilation (\dot{V}_A) out of proportion to metabolism
→ ↓ P_{aCO_2} (<37 mmHg)
- **Hypoventilation**: an decrease in alveolar ventilation (\dot{V}_A) out of proportion to metabolism
→ ↑ P_{aCO_2} (>43 mmHg)
- **Hyperpnea**: an increase in alveolar ventilation (\dot{V}_A) is proportional to metabolism → ↔ P_{aCO_2} (40 mmHg)
✓ increased breathing (usual ↑ V_T)
- **Tachypnea** – increased frequency of respiration

跑步後很喘, 如何快速回到 正常的呼吸速率?



- A) Hyperpnea
(深呼吸)
- B) Tachypnea
(淺快呼吸)

Why hyperpnea is more efficient?

Case	Tidal vol. (ml)	Freq. (/min)	Min. ventilation (ml/min)	Dead space (ml)	Alveolar ventilation (ml/min)
A	150	40	6000	150	$(150-150) \times 40 = 0$
B	500	12	6000	150	$(500-150) \times 12 = 4200$
C	1000	6	6000	150	$(1000-150) \times 6 = 5100$

A: Tachypnea

B: Normal

C: Hyperpnea

Respiration efficiency: hyperpnea > tachypnea

- **NOT** all inhaled air can be gas exchanged → dead space

Since dead space volume is **fixed**,

→ Increase frequency

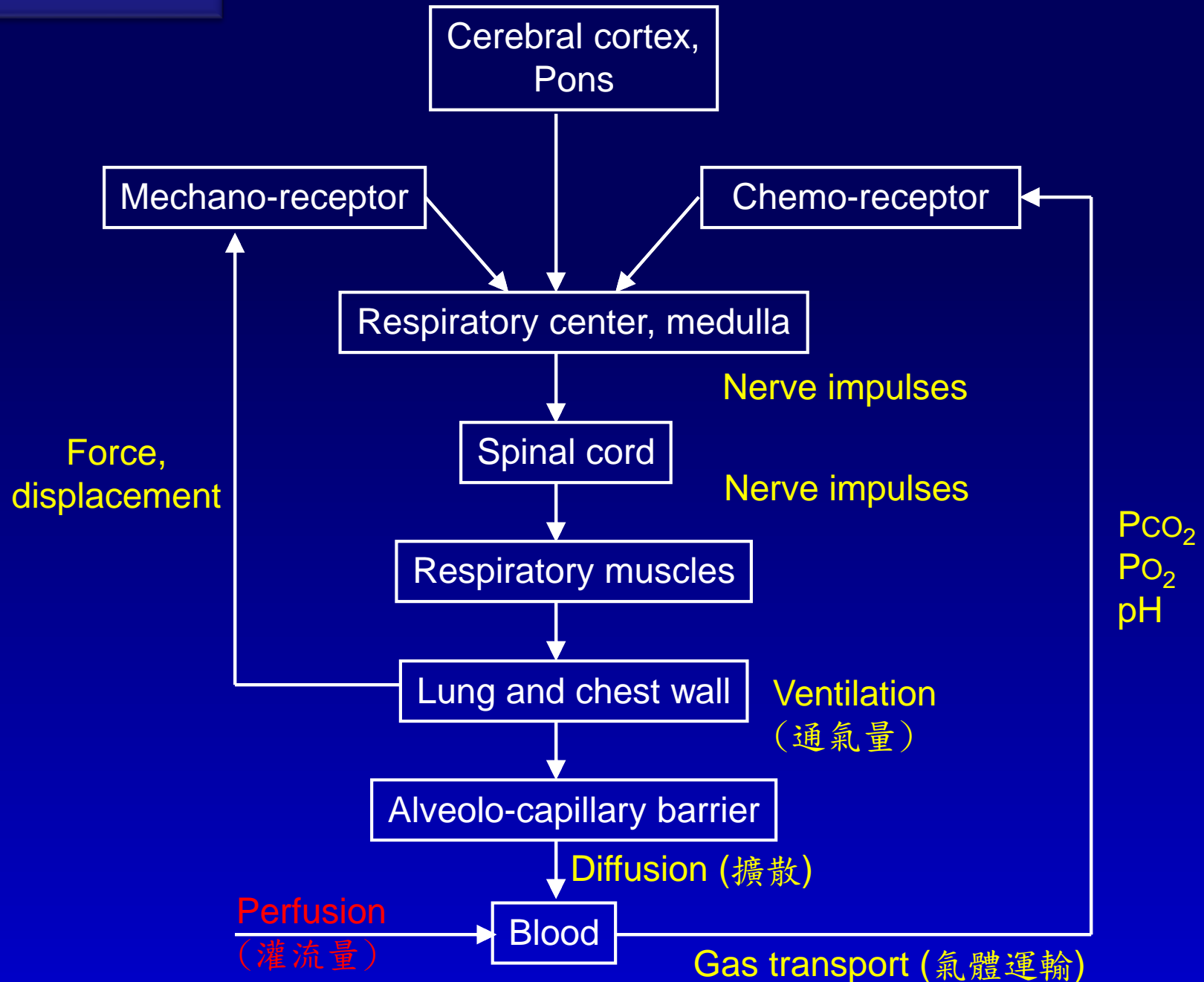
→ Decrease tidal volume

→ Decrease alveolar ventilation

Outline

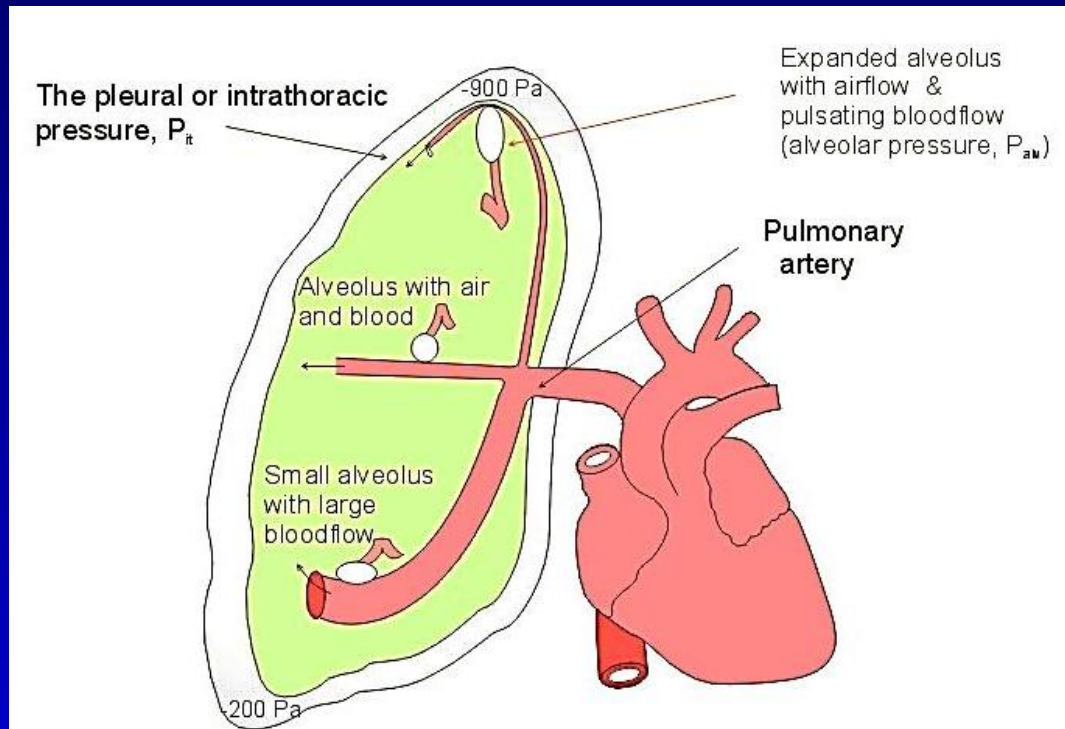
- Background
- Structure and function
- Ventilation
- **Perfusion (灌流量) and ventilation/perfusion ratio**
- Static/Dynamic respiratory mechanics
- Diffusion and gas transport
- Neural control of respiration
- Chemical control of respiration
- Acid-base balance
- Examples: exercise and high altitude adaptation

Overview



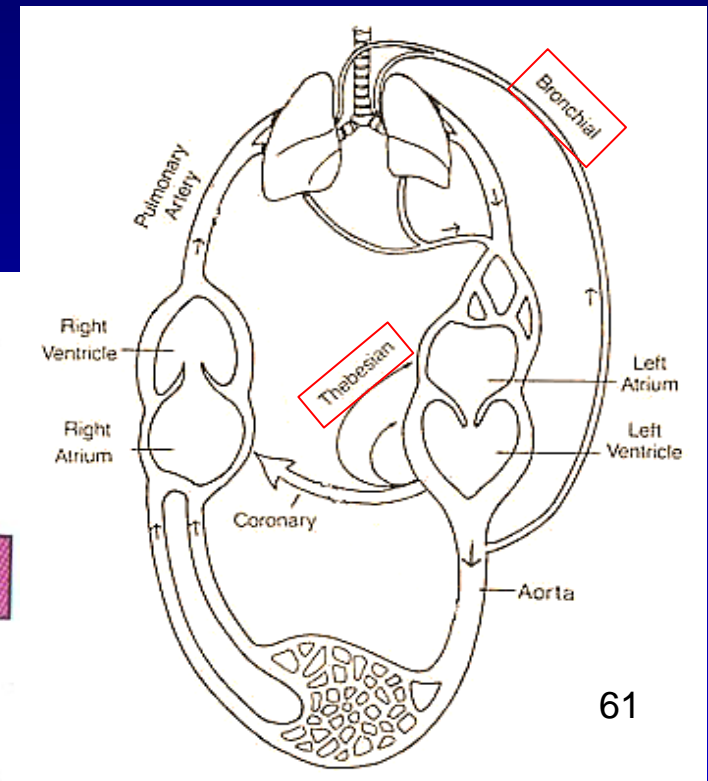
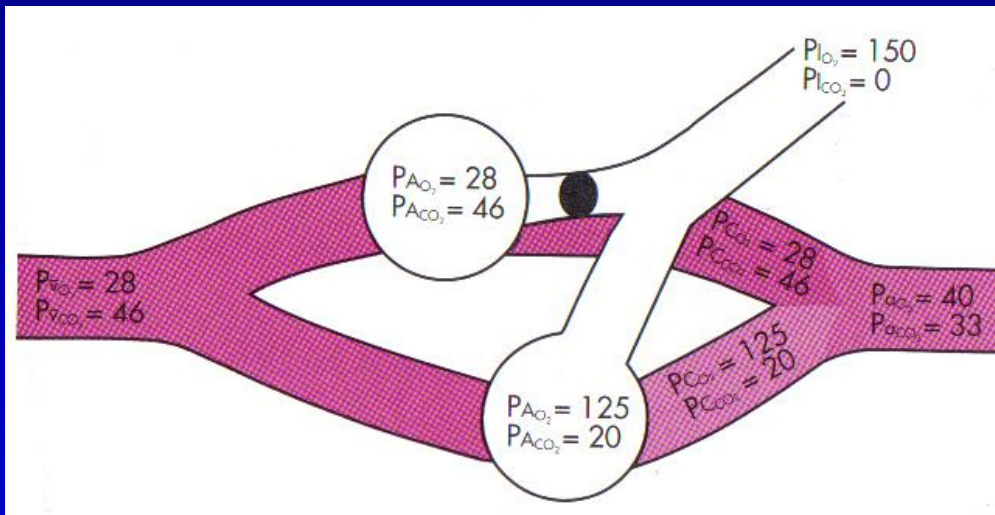
Perfusion (灌流量)

- **Perfusion** (Q): blood flow through the lung
 - ✓ The distribution of blood flow is largely due to the effects of **gravity**
i.e. the effect of hydrostatic pressure



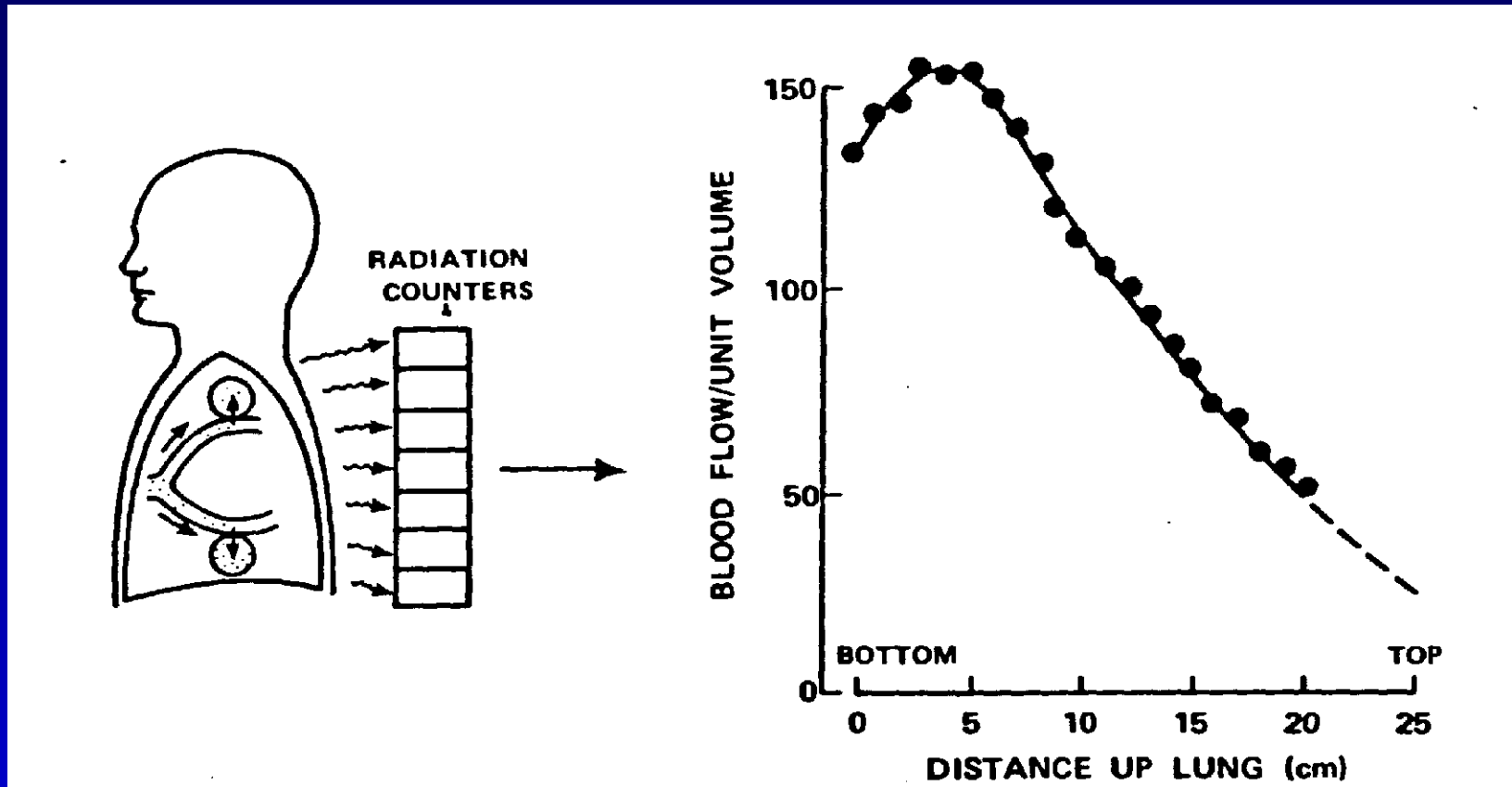
Shunt (分流)

- **Shunt**: blood without gas exchange with alveoli
 - ✓ **Intrapulmonary shunts**: blood perfuses alveoli but is not ventilated
 - ✓ **Anatomical shunts**
 - **Bronchial circulation** enters the pulmonary veins
 - **Coronary circulation** enters LV via thebesian veins

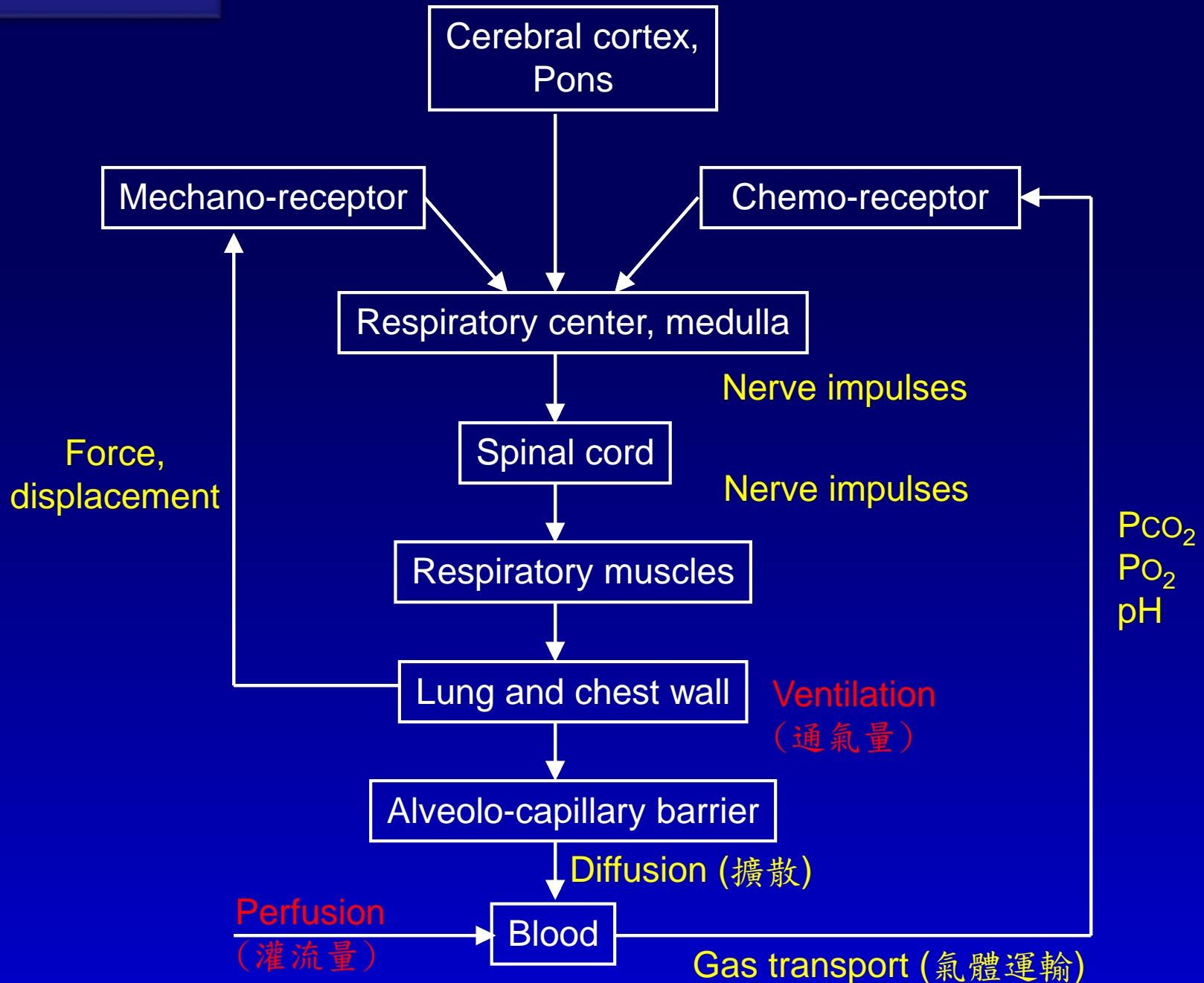


Uneven Perfusion in Upright Position

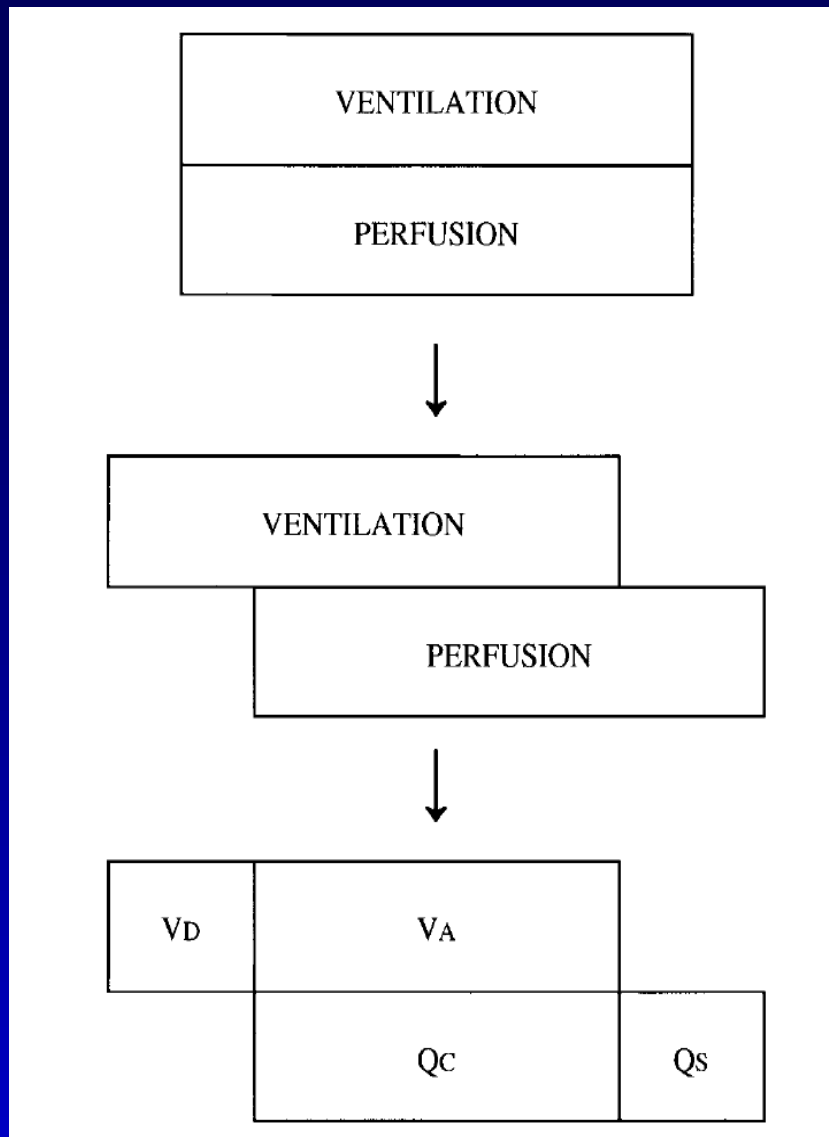
In the upright position, blood flow is maximal at the lung bases, decreasing linearly to the apices



Overview



Matching of Ventilation & Perfusion



Perfect matching

$$\rightarrow \dot{V}/Q=1$$

Mismatching of \dot{V}/Q

$$\dot{V} = \dot{V}_A + \dot{V}_D$$

\dot{V}_A : alveolar ventilation

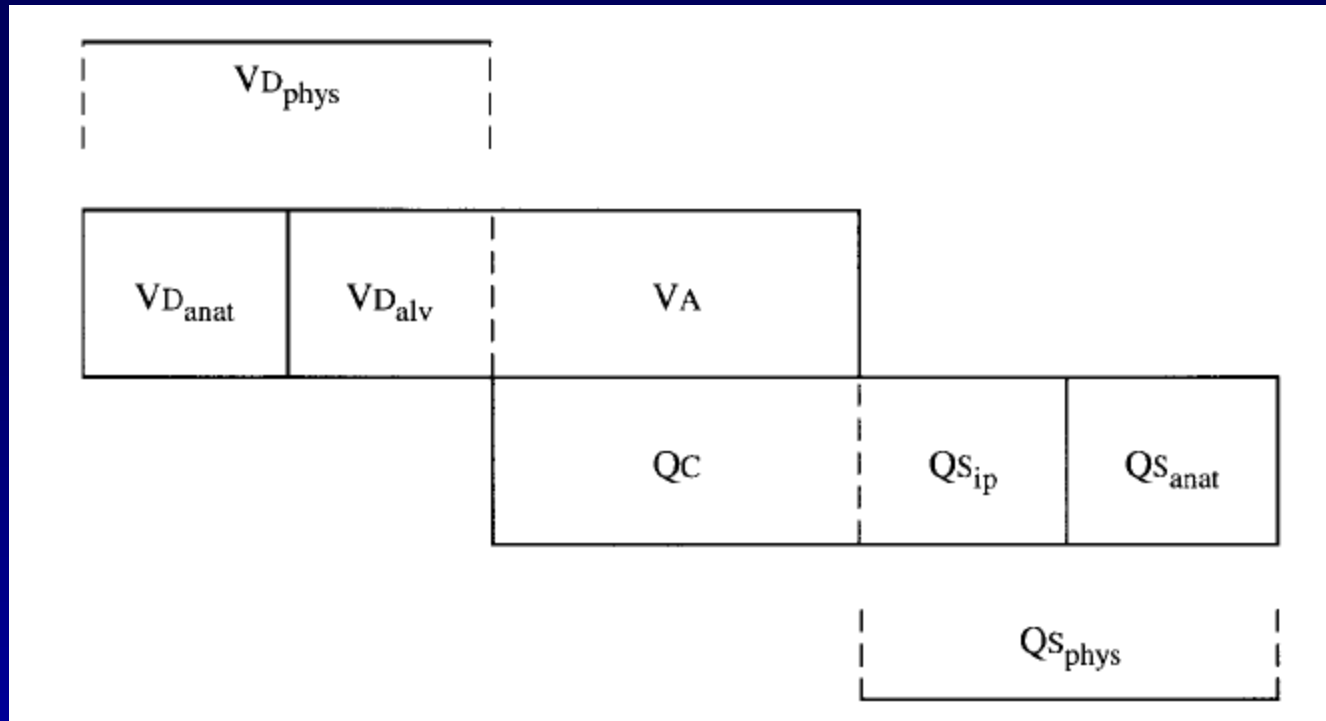
\dot{V}_D : dead-space ventilation

$$Q = Q_C + Q_S$$

Q_C : capillary flow

Q_S : shunt flow

Mis-matching of Ventilation & Perfusion



$$\dot{V}_D^{phys} = \dot{V}_D^{anat} + \dot{V}_D^{alv}$$

\dot{V}_D^{phys} : physiological \dot{V}_D

\dot{V}_D^{anat} : anatomic \dot{V}_D

\dot{V}_D^{alv} : alveolar \dot{V}_D

$$Q_{S_{phys}} = Q_{S_{ip}} + Q_{S_{anat}}$$

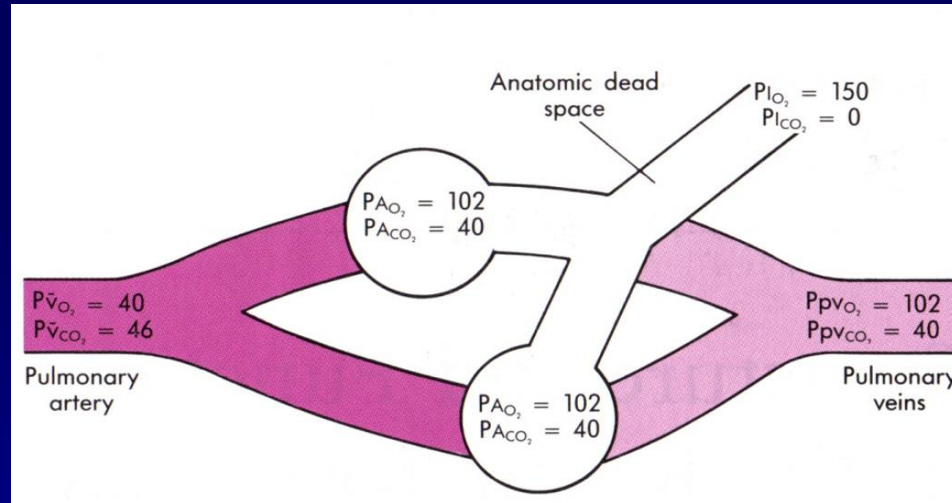
$Q_{S_{phys}}$: physiological shunt

$Q_{S_{ip}}$: intrapulmonary shunt

$Q_{S_{anat}}$: anatomical shunt

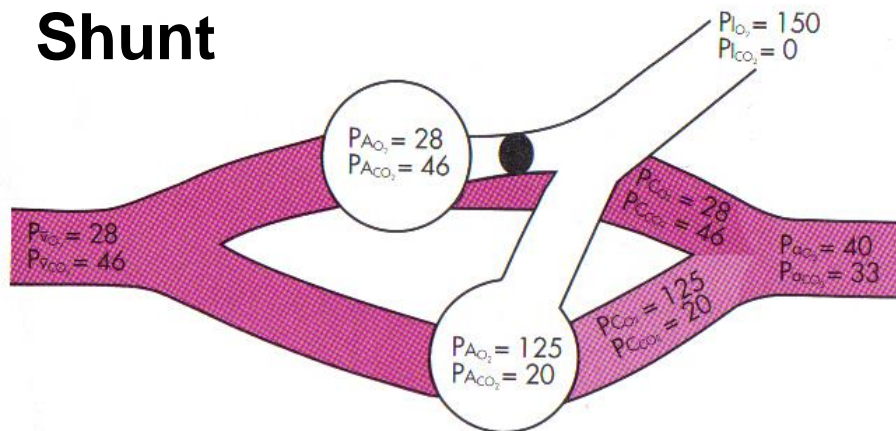
\dot{V}/Q 受什麼影響？

Matching of Ventilation & Perfusion



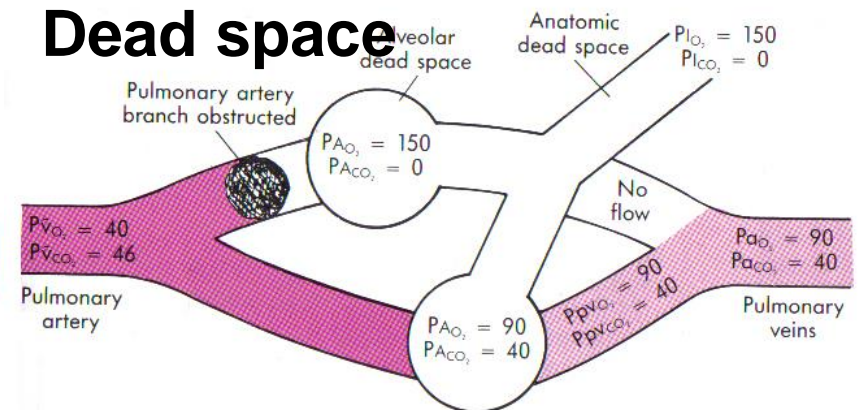
$$\dot{V}/Q \sim 0.8$$

Shunt



$$\dot{V}/Q \downarrow$$

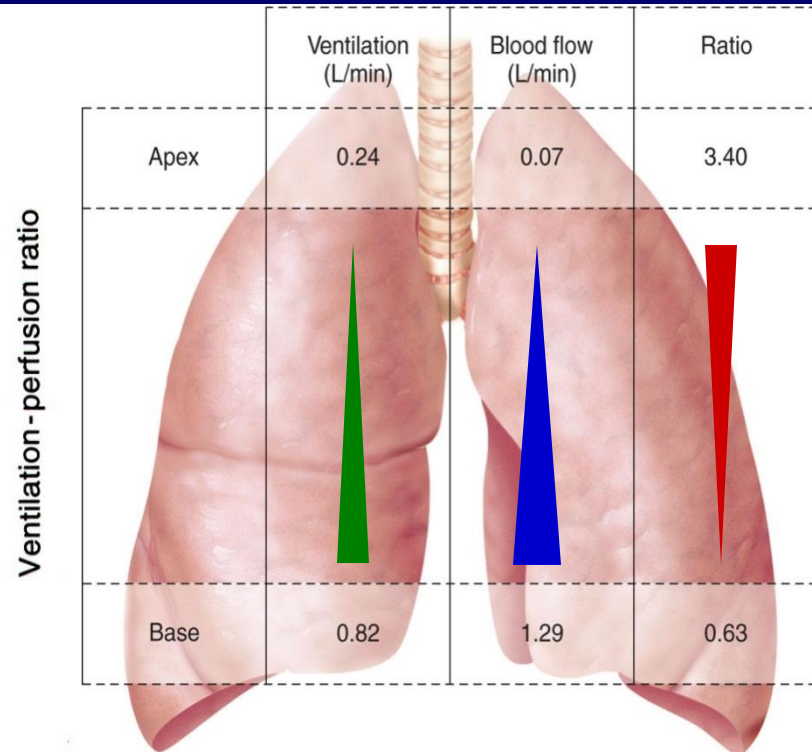
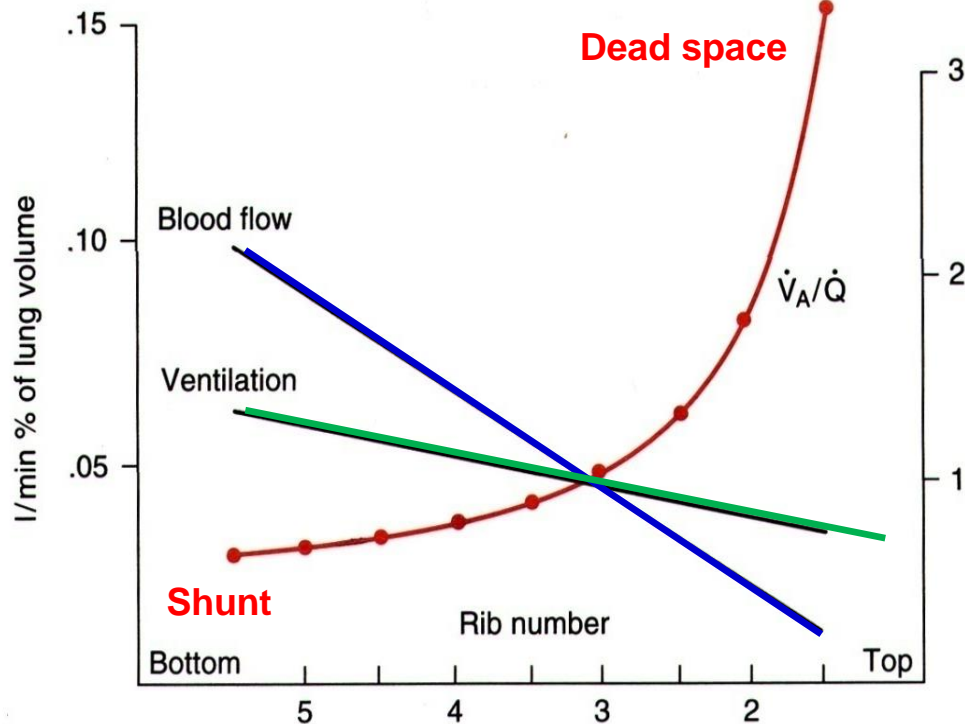
Dead space



$$\dot{V}/Q \uparrow$$

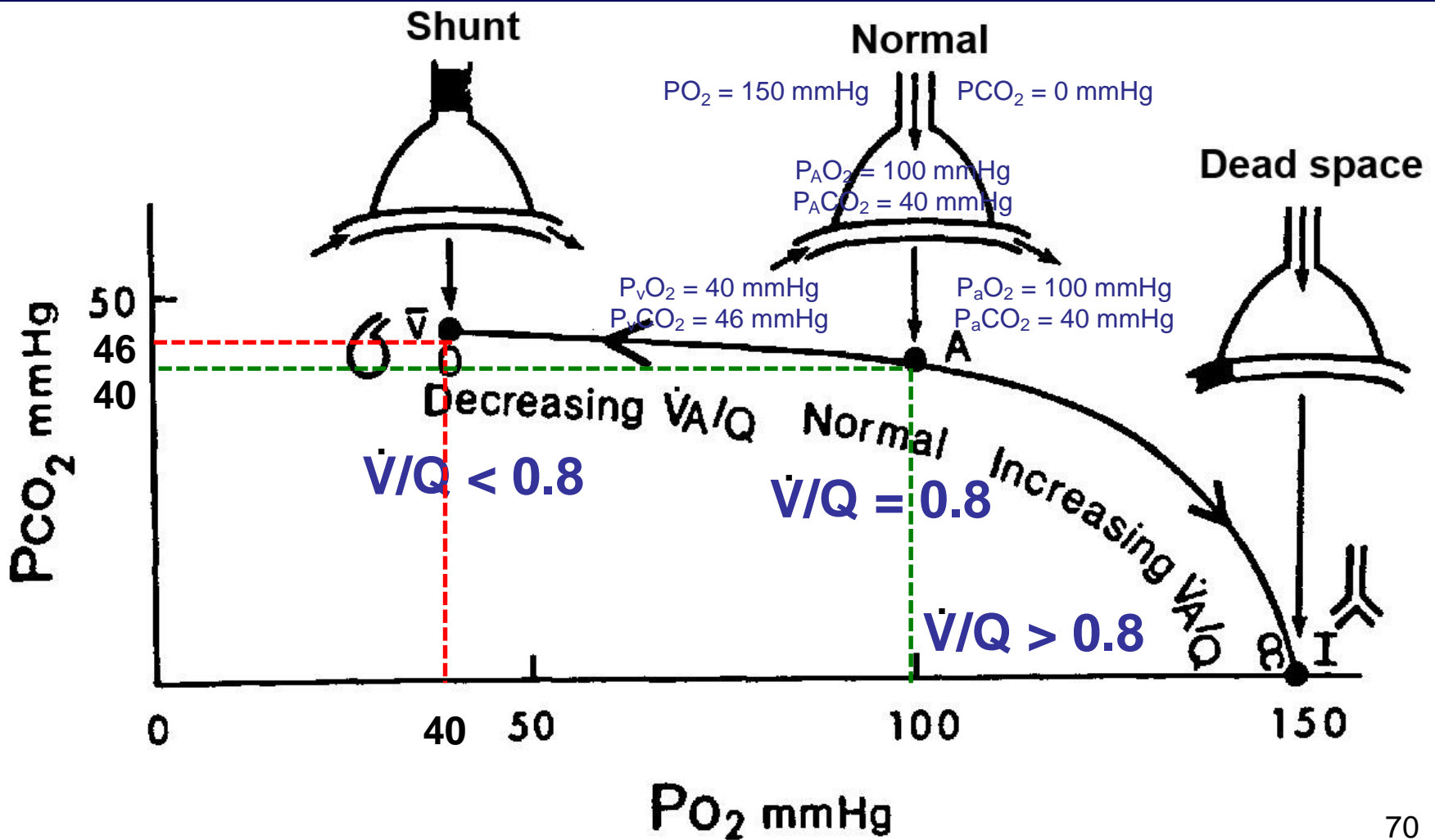
Distribution of \dot{V} and Q Within the Lung in the Upright Position

- \dot{V} ↓ from base to apex of lung
- Q ↓↓ from base to apex of lung
- $\rightarrow \dot{V}/Q \uparrow$ from base to apex of lung



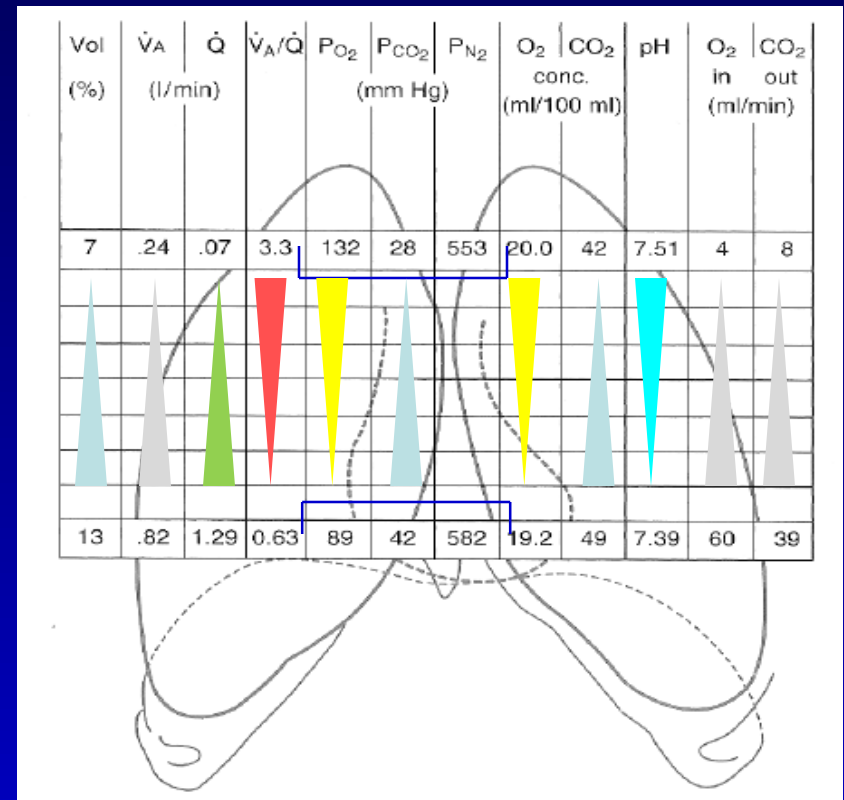
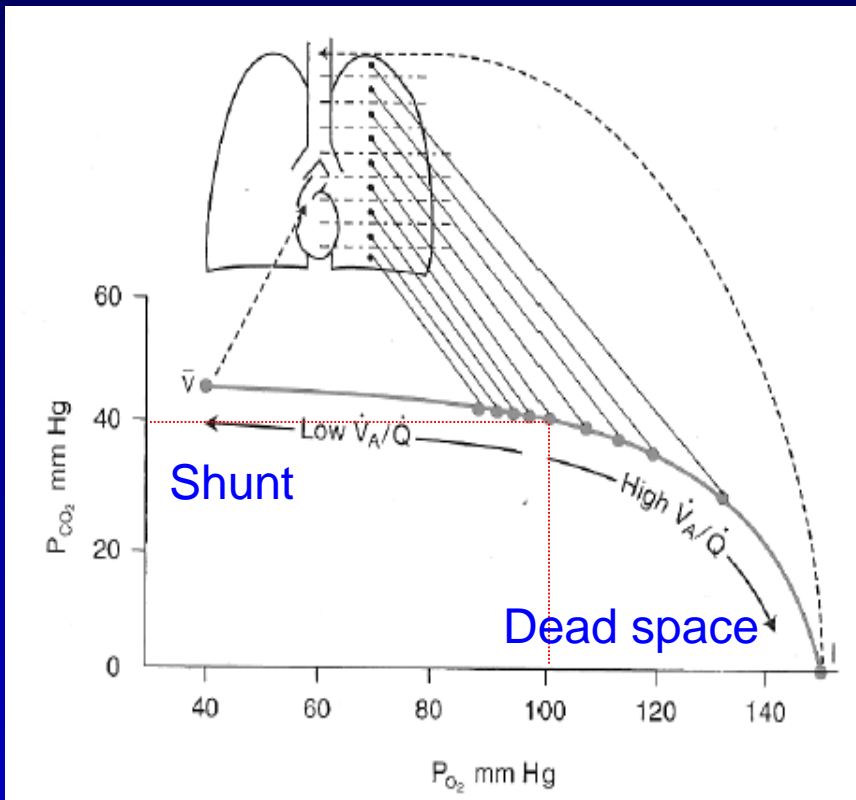
\dot{V}/Q 如何影響氣體的分壓？

\dot{V}/Q v.s. P_{O_2} & P_{CO_2}



\dot{V}/Q Inequality of Normal Lung in the Upright Position

- High \dot{V}/Q ratio at the apex \rightarrow high P_{O_2} and low P_{CO_2}



Outline

- Background
- Structure and function
- Ventilation
- Perfusion and ventilation/perfusion ratio
- **Static/Dynamic respiratory mechanics (呼吸力學)**
- Diffusion and gas transport
- Neural control of respiration
- Chemical control of respiration
- Acid-base balance
- Examples: exercise and high altitude adaptation

Key Points

- General concepts and terminology
- Mechanical properties
 - 1. Compliance
 - 2. Resistance
 - 3. Pressure-volume (P-V) curve of the lungs
 - 4. Lung-chest wall coupling

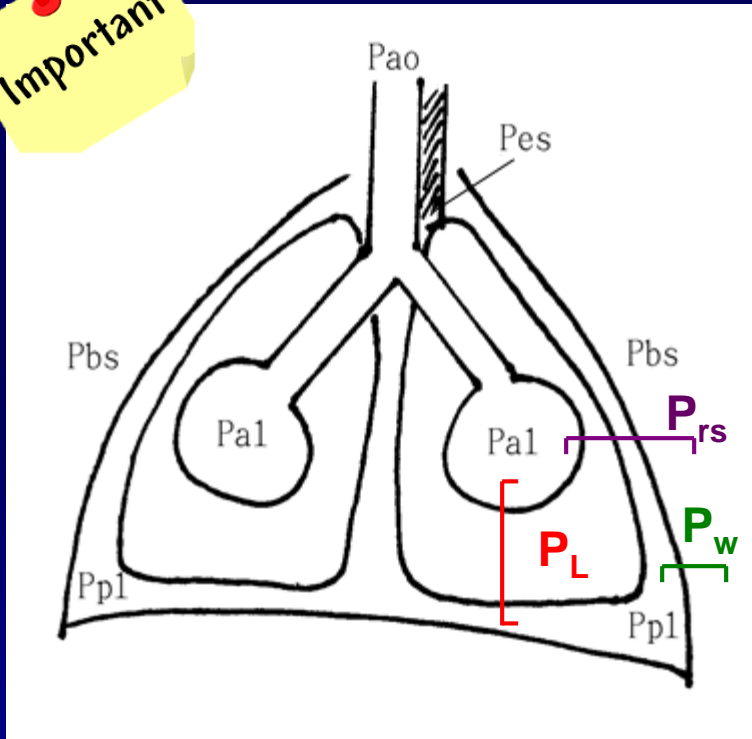
General Concepts and Terminology

- $P_{\text{total}} = \text{resistive } P_r + \text{elastic } P_r = \dot{V}R + \frac{\Delta V}{C}$
 - ✓ In spontaneous breathing, $P_{\text{total}} = P_{\text{muscle}}$
 - ✓ In mechanical ventilation, $P_{\text{muscle}} = 0$, P_{total} is driven by ventilator
- *Active* ($P_{\text{muscle}} > 0$) or *passive* ($P_{\text{muscle}} = 0$)
- *Static* ($\dot{V} = 0$) or *dynamic* ($\dot{V} \neq 0$)
- **Transmural pressure (跨壁壓)**: pressure difference from the **inside** to the **outside**
- Atmospheric pressure is considered = 0,
→ positive pressure meaning the value greater than atmospheric pressure, vice versa

General Concepts and Terminology

- $P_{\text{total}} = \text{resistive } P_r + \text{elastic } P_r = \dot{V}R + \frac{\Delta V}{C}$
- Under static conditions,
transmural pressure = **elastic recoil pressure** of the compartment
- Static properties (when flow=0) mean **lung elastic recoil**
 - ✓ Elastic properties of the lung tissue itself
 - ✓ Surface tension

Important



$$P_L = P_{al} - P_{pl} \quad (1)$$

$$P_w = P_{pl} - P_{bs} \quad (2)$$

$$(1) + (2)$$

$$P_{rs} = P_L + P_w = P_{al} - P_{bs}$$

$$a) P_{bs} = 0 \rightarrow P_w = P_{pl} = P_{es}$$

$$P_{rs} = P_{al}$$

P_L : transpulmonary Pr. (跨肺壓)

P_{al} : alveolar Pr.

P_{pl} : intrapleural Pr.

P_w : trans-chest wall Pr. (跨胸壁壓)

P_{bs} : body surface Pr.

P_{rs} : respiratory sys. Pr.

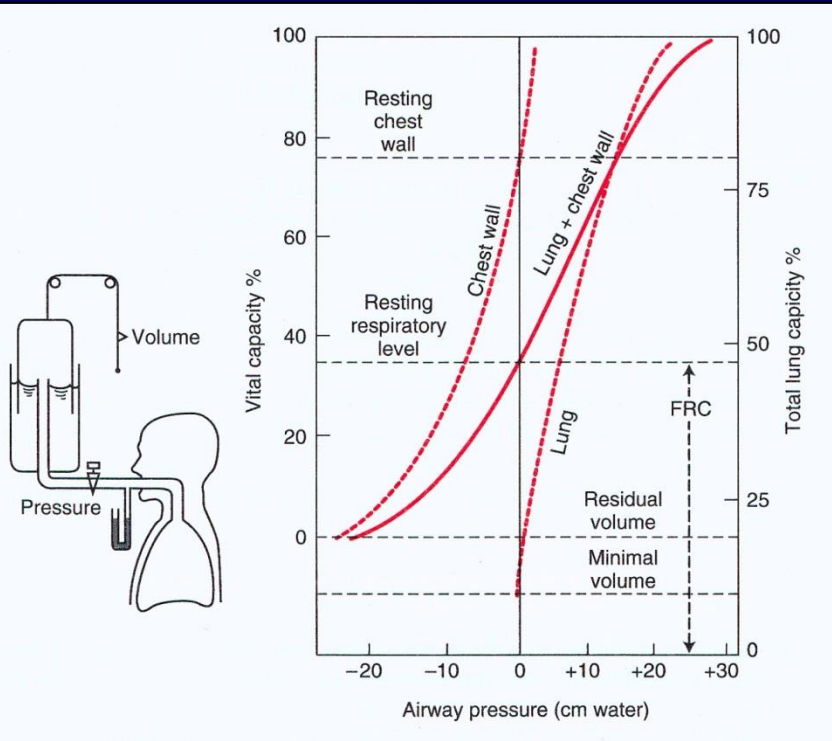
P_{ao} : airway opening Pr.

P_{es} : esophageal Pr.

$$b) P_{ao} = \dot{V}R_{aw} + P_{al}$$

When flow=0, $P_{ao} = P_{al} = P_{rs}$

$$c) P_L = P_{al} - P_{pl} = P_{ao|flow=0} - P_{es}$$



$$P_L = P_{al} - P_{pl} \quad (1)$$

$$P_w = P_{pl} - P_{bs} \quad (2)$$

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Key Points

- General concepts and terminology
- Mechanical properties
 - 1. Compliance (順應性)
 - 2. Resistance
 - 3. Pressure-volume (P-V) curve of the lungs
 - 4. Lung-chest wall coupling

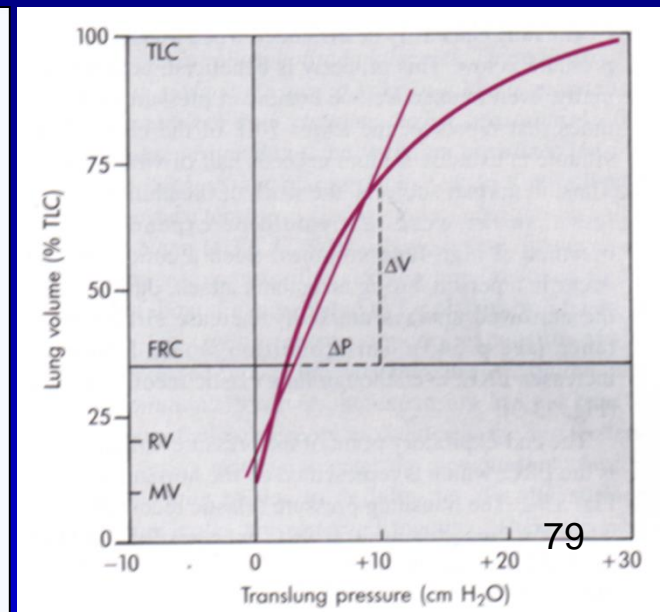
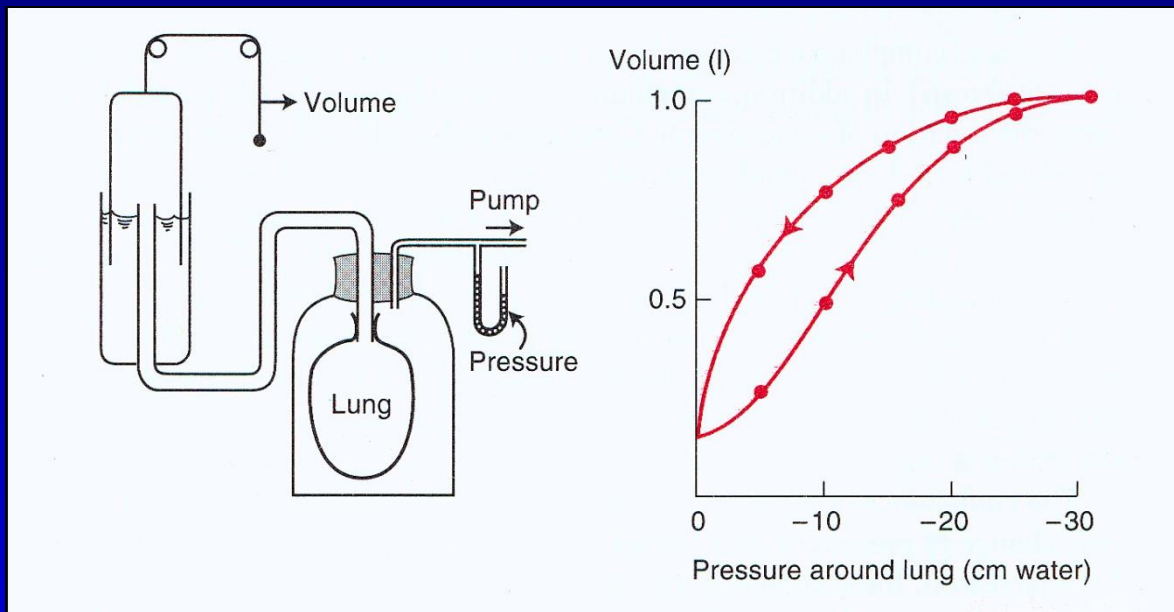
Static Mechanical Properties

- **Compliance (順應性; C)**: the ease with which an object can be deformed

Elastic Recoil of the Lung

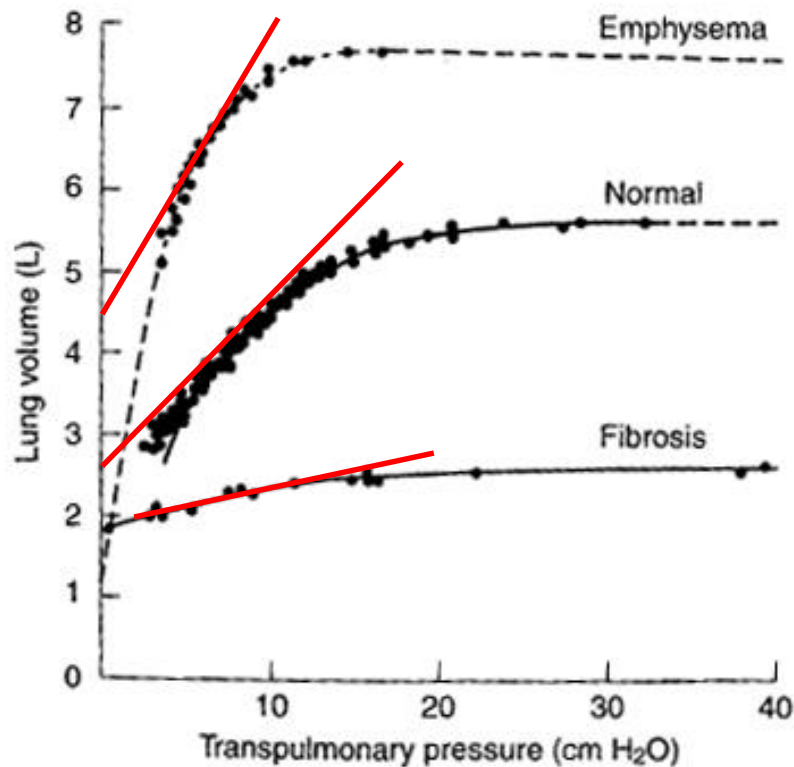
- Lung compliance: the **slope** of the line between any two points on the **deflation limb** of the pressure-volume loop

$$C_L = \frac{\Delta V_L}{\Delta P_L}$$



反吸煙宣傳：
吸煙豬肺示範

Compliance Changes in Different Diseases



In chronic obstructive pulmonary disease (COPD), alveolar walls progressively degenerate
→ C_L increase

In pulmonary fibrosis,
→ C_L decrease

Key Points

- General concepts and terminology
- Mechanical properties
 - 1. Compliance
 - 2. **Resistance (阻力)**
 - 3. Pressure-volume (P-V) curve of the lungs
 - 4. Lung-chest wall coupling

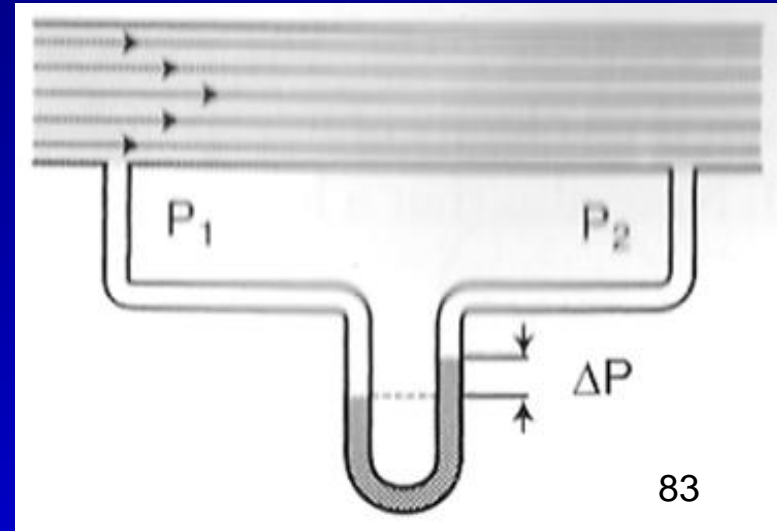
Resistance

- Resp. Resistance:
 - Airway resistance (70~80%)
 - Tissue resistance (20%): movement of lung tissue, chest wall and abdominal contents
- **Airway resistance (氣管阻力)**: the pressure difference between the alveoli and the mouth per unit of airflow

$$P_{ao} = \dot{V}R_{aw} + P_{al}$$

$$\rightarrow R_{aw} = \frac{P_{ao} - P_{al}}{\dot{V}}$$

P_{ao} : airway opening Pr.



The Airway Resistance

- $P_{rs} = \text{resistive Pr} + \text{elastic Pr} = \dot{V}R_{aw} + \frac{\Delta V}{C}$

$$\rightarrow R_{aw} = \frac{P_{rs} - \frac{\Delta V}{C}}{\dot{V}} \quad (1)$$

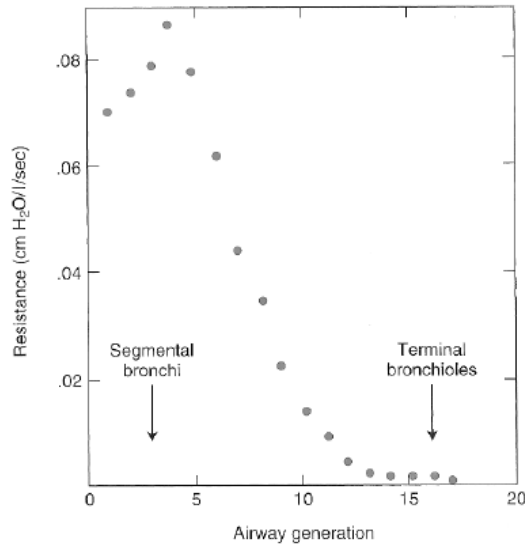
- In laminar flow, flow is proportional to ΔP by Poiseuille's law

$$Q = \frac{\pi r^4 (P_1 - P_2)}{8\eta l} \quad (2)$$

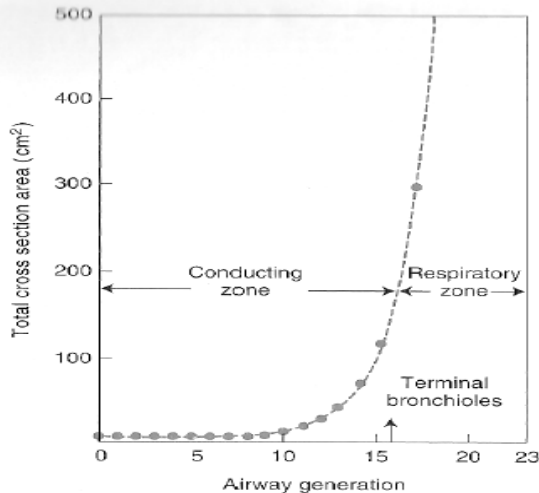
$$\rightarrow R_{aw} = \frac{(P_{rs} - \frac{\Delta V}{C}) \times 8\eta l}{\pi r^4 (P_1 - P_2)}$$

- Resistance is **inversely proportional** to the **fourth power** of the airway radius

The Airway Resistance

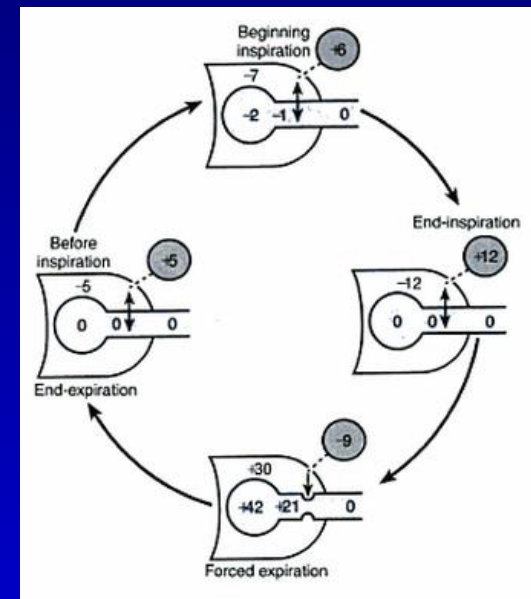
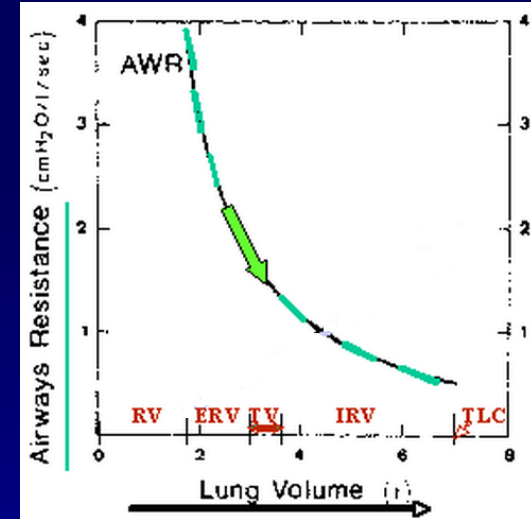


- Poiseuille's law: $R \propto \frac{1}{r^4}$
- Individual resistance:
small airway \gg large airway
- Total resistance:
small airway $<$ large airway
- ✓ the effective cross-sectional area of many bronchioles in parallel increases

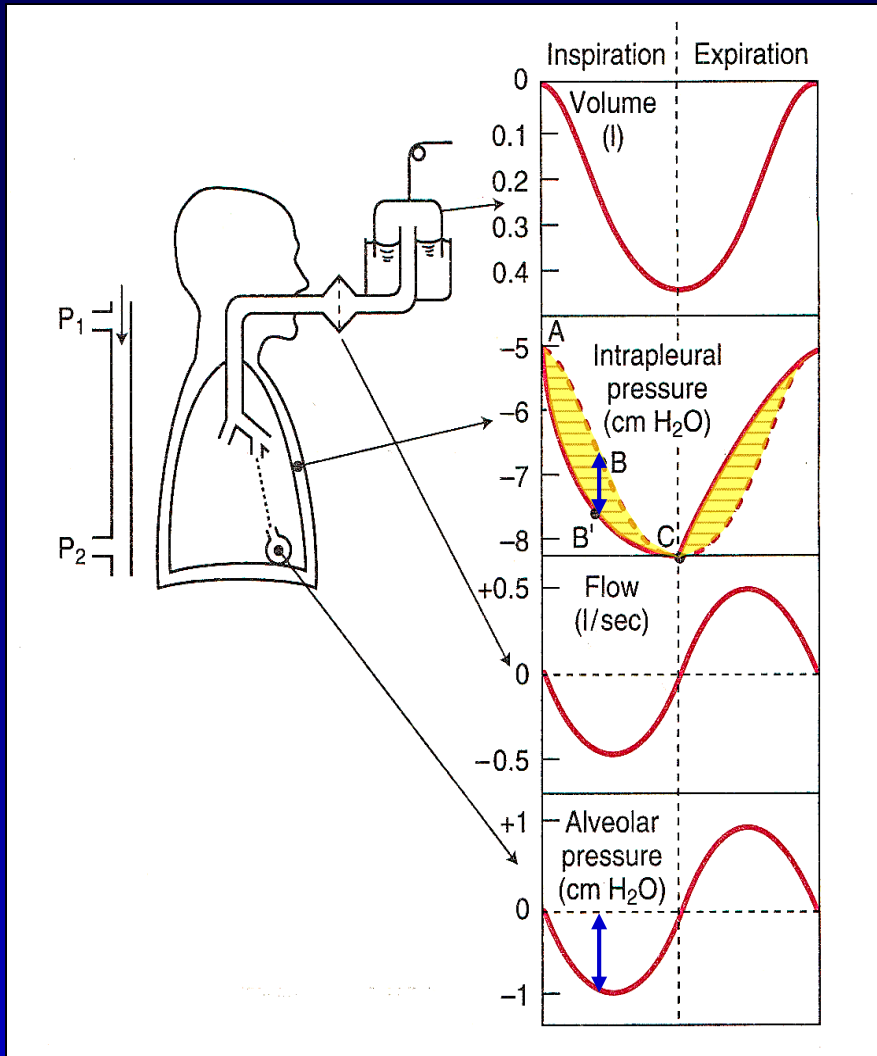


The Airway Resistance

- Airway resistance \downarrow as lung volume \uparrow
→ the airways distend as the lungs inflate
- The airways are narrower during expiration
→ $R_{\text{exp}} > R_{\text{insp}}$
- Factors affecting the radius of bronchioles
 - **Airway constriction:** histamine; parasymp. n.
 - **Airway dilation:** epinephrine; symp. n.

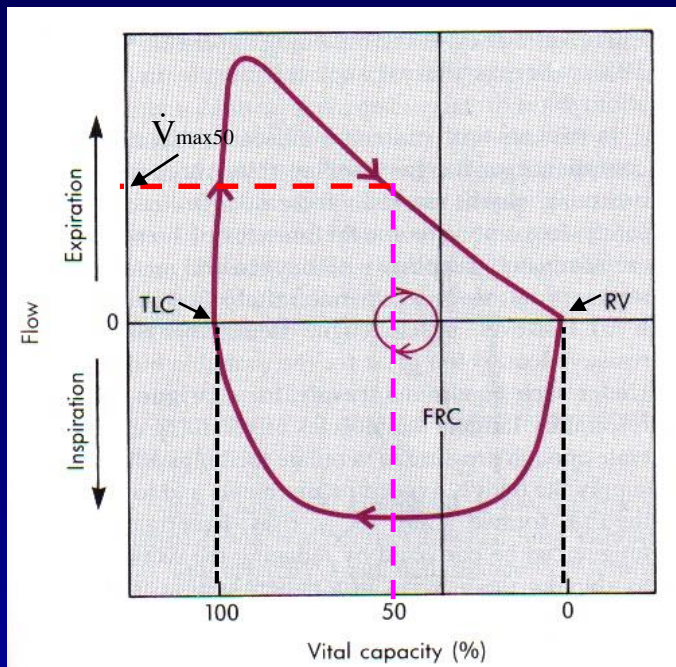


The Airway Resistance



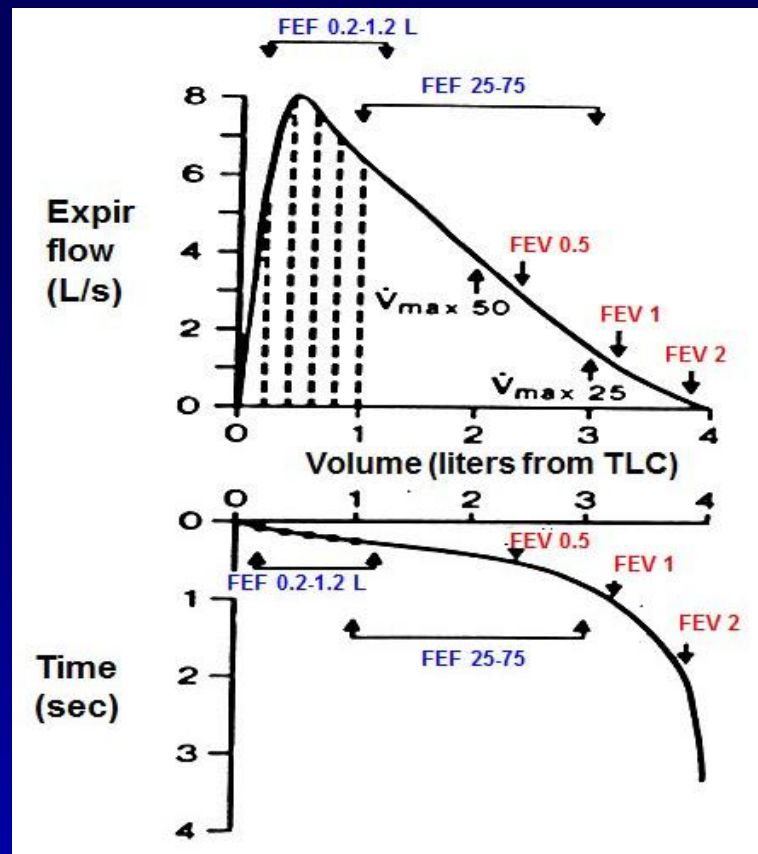
- If no resistance exists, intrapleural pressure should be along the broken line
- The vertical distance between lines ABC and AB'C reflects the alveolar pressure
- Airway resistance contributes the hatched portion of intrapleural pressure

Evaluation of Airway Resistance



Flow-Volume Curve

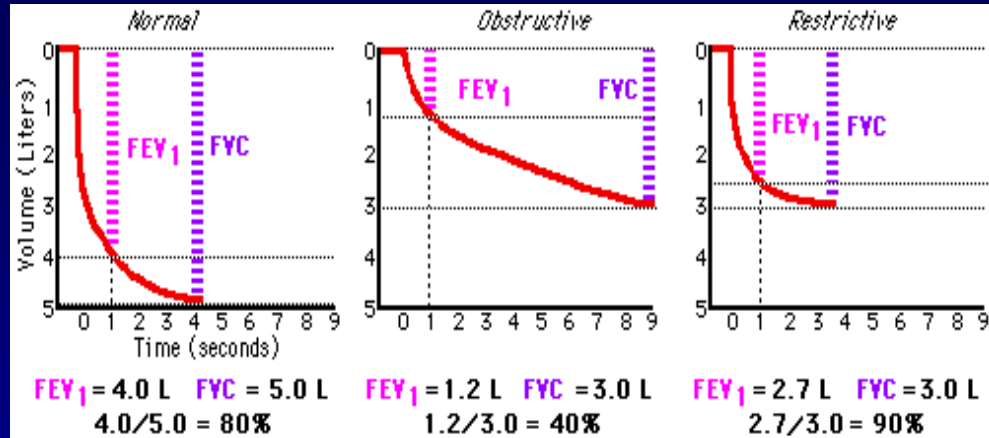
$\dot{V}_{\max 50}$: \dot{V}_{\max} at 50% of VC



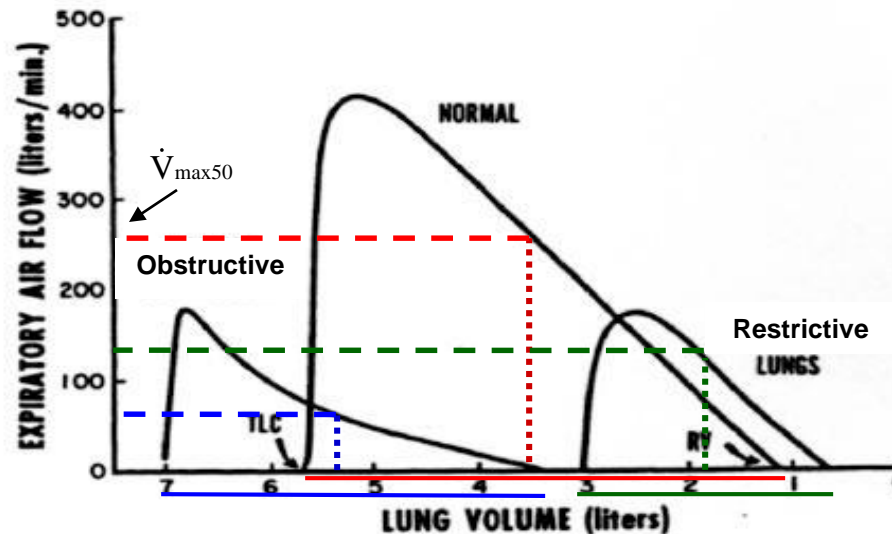
FEF: forced expiratory flow
 FEV₁: forced expiratory vol.
 in one second

Evaluation of Abnormality in Lung Vol.

FEV_1



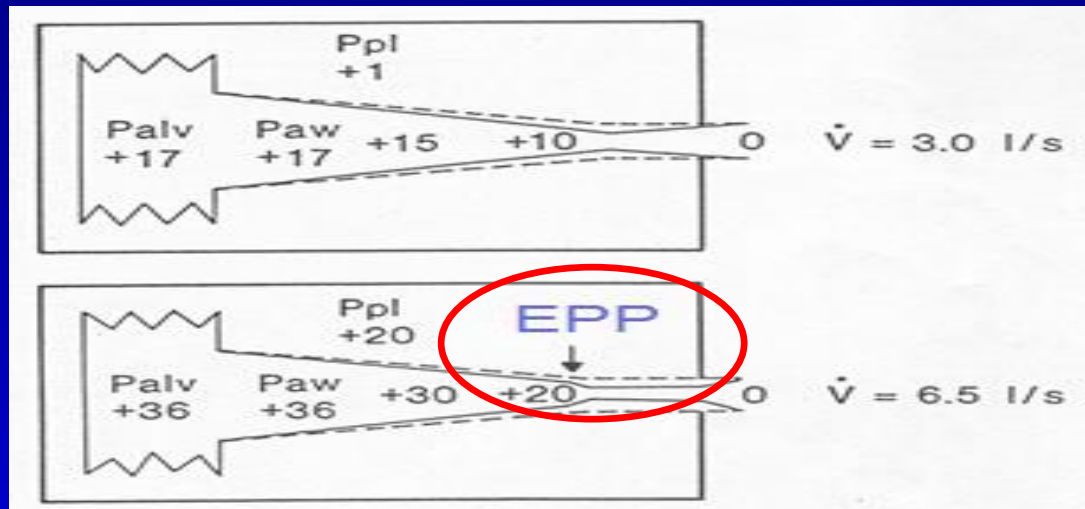
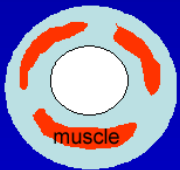
\dot{V}_{max50}



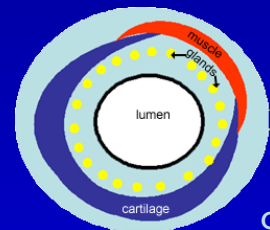
Pursed Lip Breathing

- **Equal pressure point (EPP)**
 - ✓ The point where intrapleural pressure (P_{pl}) equals airway pressure (P_{aw}) during forced expiration
 - ✓ Downstream airway (close to mouth) is more compressed → ↑ airway resistance → hard to expel air
- **Pursed lip breathing (噉嘴吐氣)**
 - ✓ Increase mouth pressure → EPP is moved from smaller collapsible airways toward larger cartilaginous (non-collapsible) airways

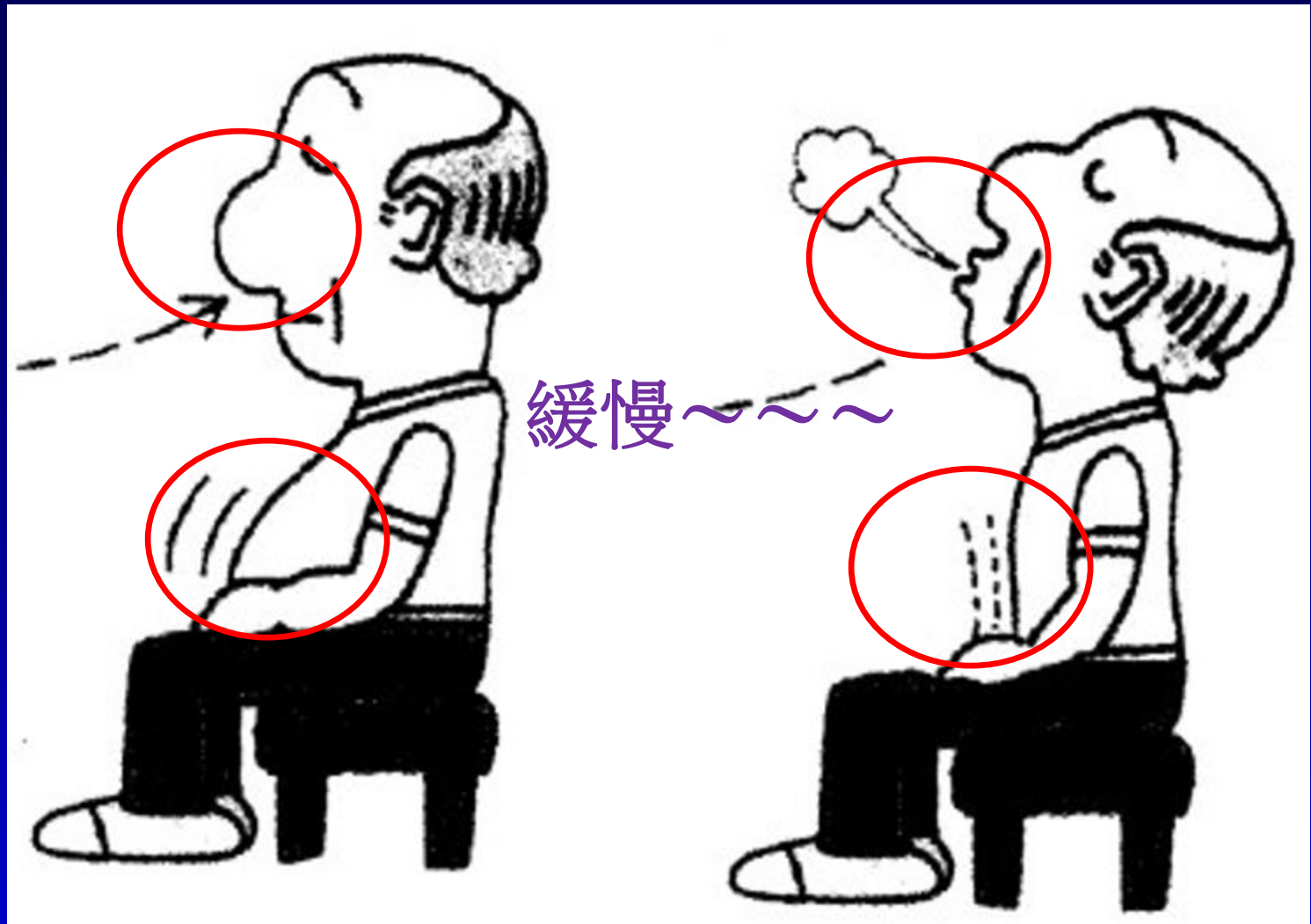
Bronchiole



Trachea



做那些動作使呼吸效率增加? 為什麼?



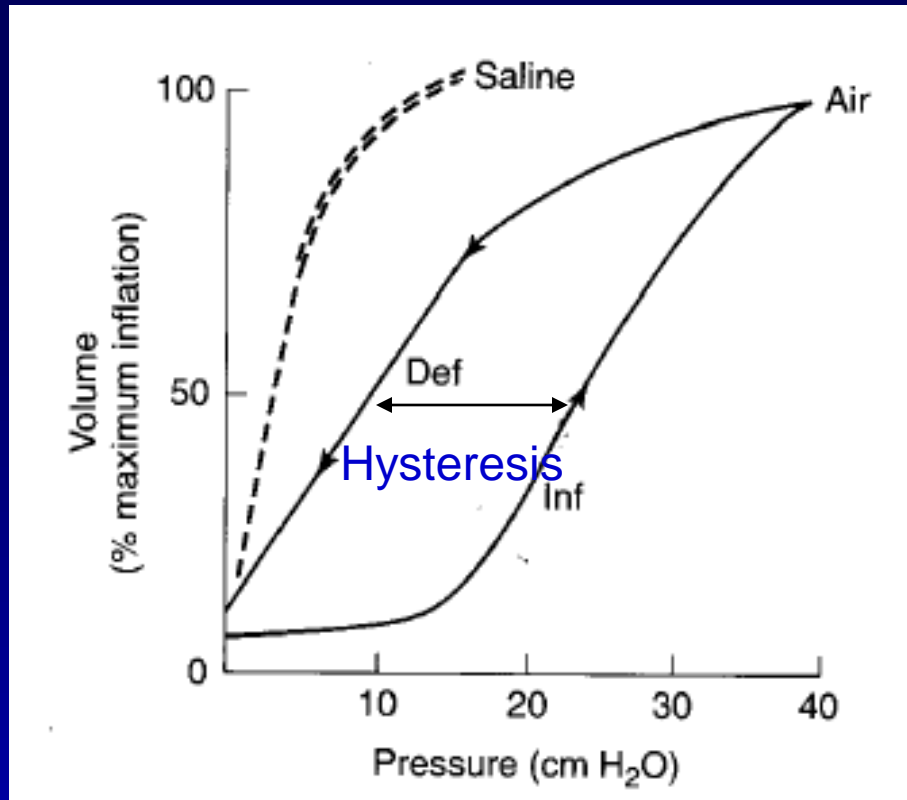
做那些動作使呼吸效率增加？ 為什麼？

- 腹式呼吸：
 - ✓ 橫膈為最主要吸氣肌
 - ✓ 使吐氣吐的完全
- 深緩呼吸：↑ 肺泡通氣量
- 鼻子吸氣，嘴巴噤嘴吐氣
 - ✓ 鼻子有過濾及溫度、濕度調節作用
 - ✓ 嘴巴噤嘴可↓氣管被壓縮程度，↑排氣

Key Points

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P-V Curve of the Lungs



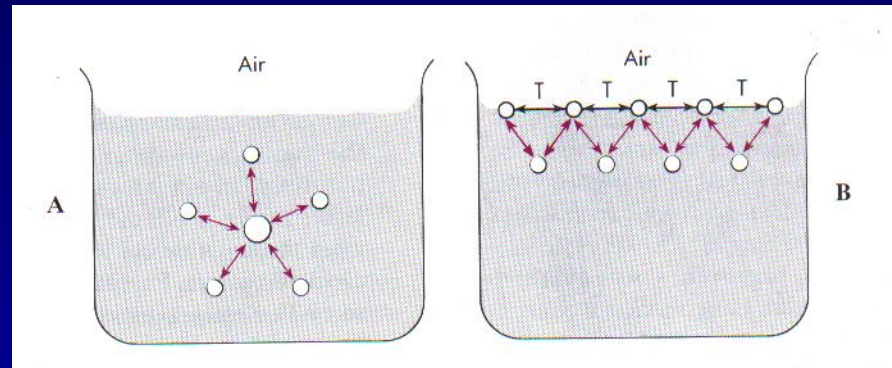
Hysteresis (遲滯; disparity between responses to inflation and deflation) is **NOT** due to tissue elastic recoil forces

BUT

disappearance of alveolar air-liquid interface (i.e. work against **surface tension** during inflation)

Surface Tension

A molecular cohesive force existing in the surface film of all liquids which tends to contract the surface to the smallest possible area



A. Force is relatively uniform on molecules in the interior

B. At the surface the molecules are pulled toward the interior and generate a compression tension (T) in the plane of the surface

Example: a soap bubble on the end of a tube

Law of LaPlace

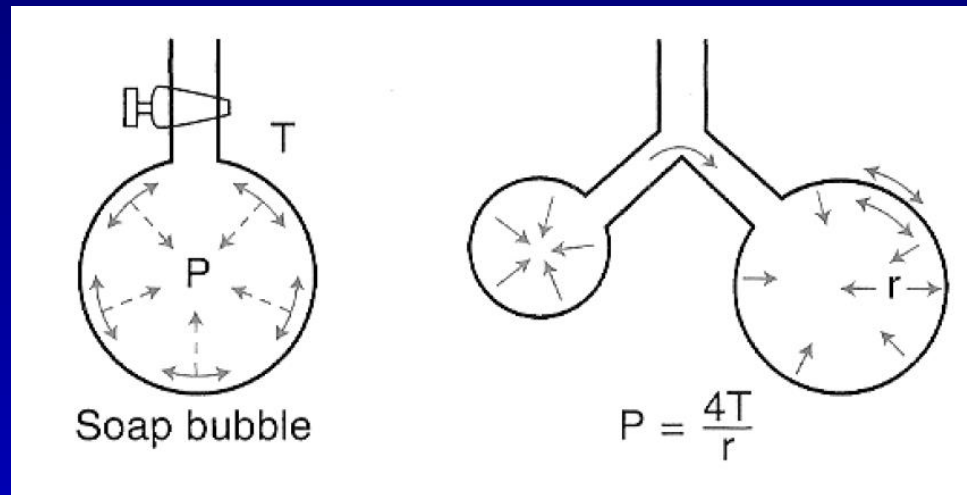
- Surface forces in a soap bubble tend to reduce the area of the surface and generate a pressure within the bubble
- LaPlace's Law:

$$P = \frac{4T}{r}$$

P: trans-mural pressure

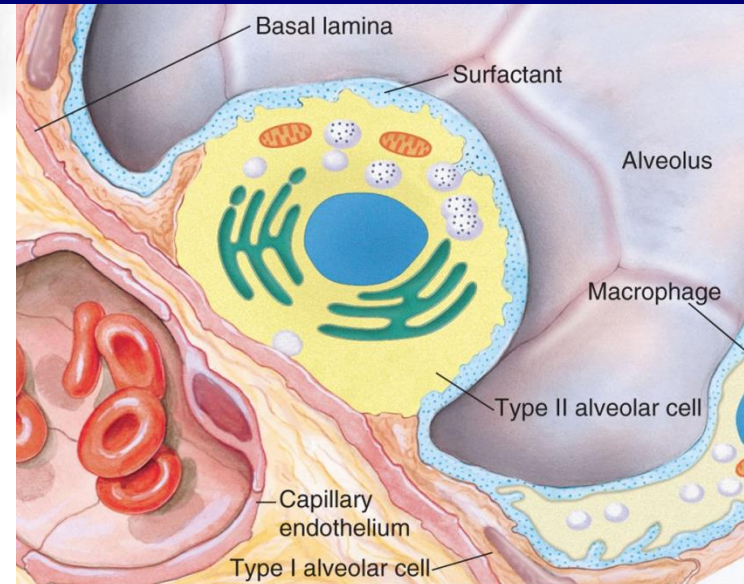
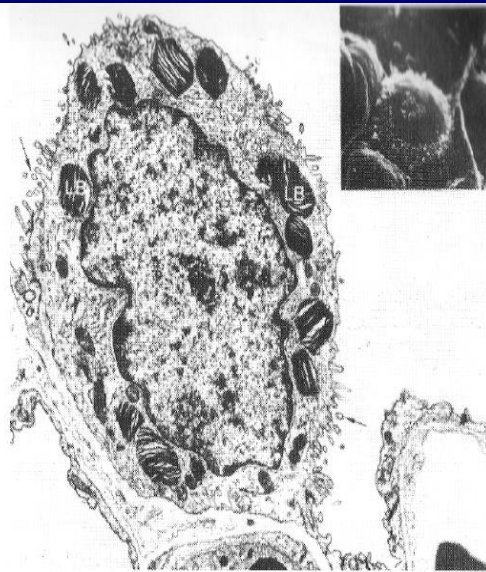
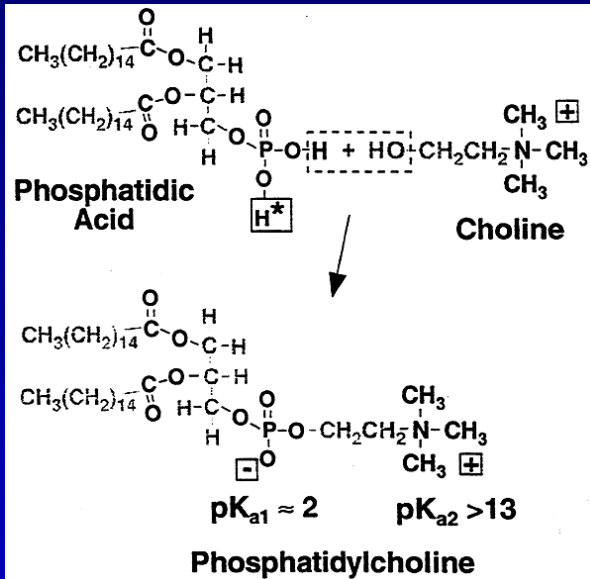
T: surface tension

r: radius

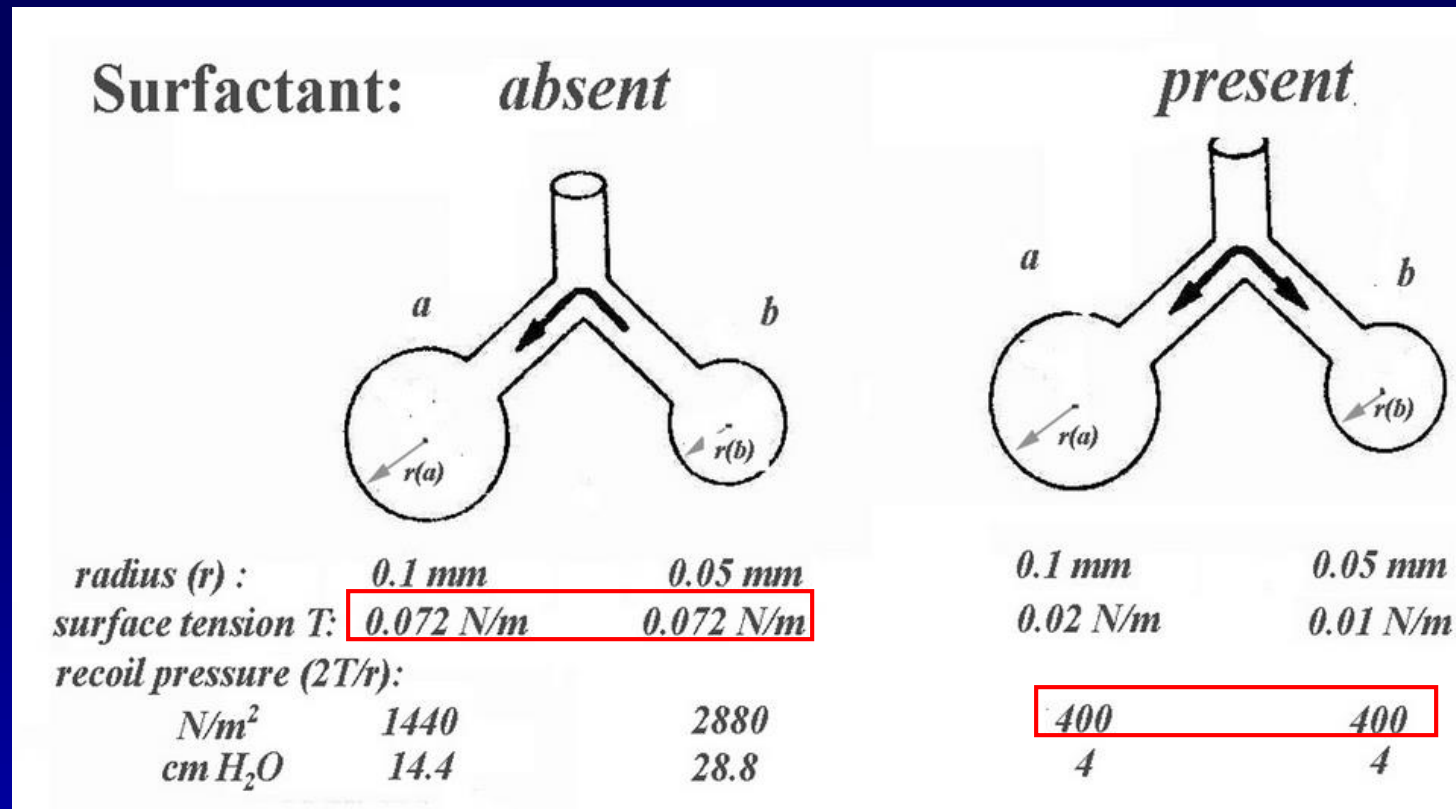


Surfactants

- Surfactants (界面活性劑): (e.g. detergents) **lower** the surface tension of water
- Lung surfactant (**dipalmitoyl phosphatidylcholine**, DPPC; secreted by alveolar epithelial cells type II) **allows the surface tension to vary**

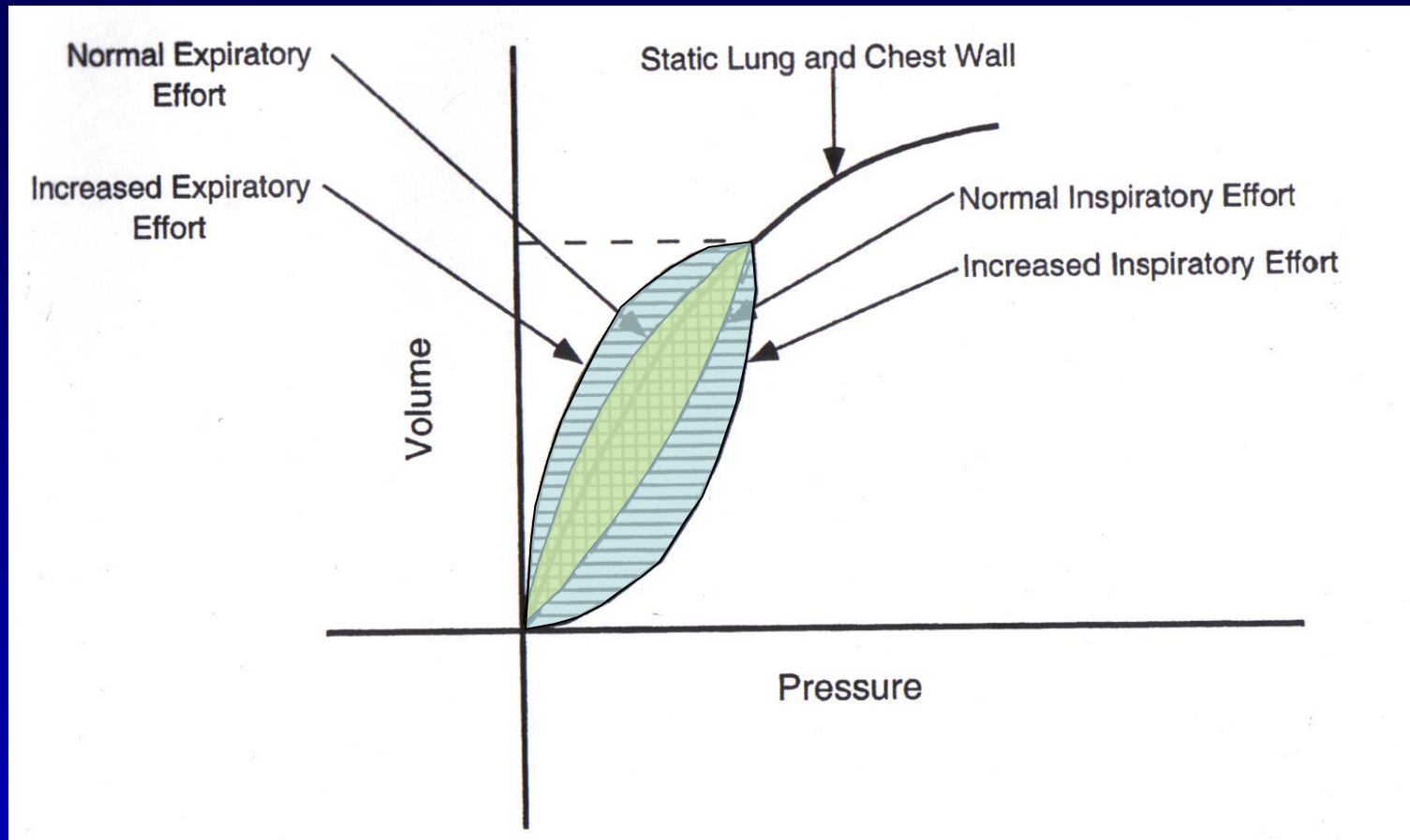


Importance of Lung Surfactant



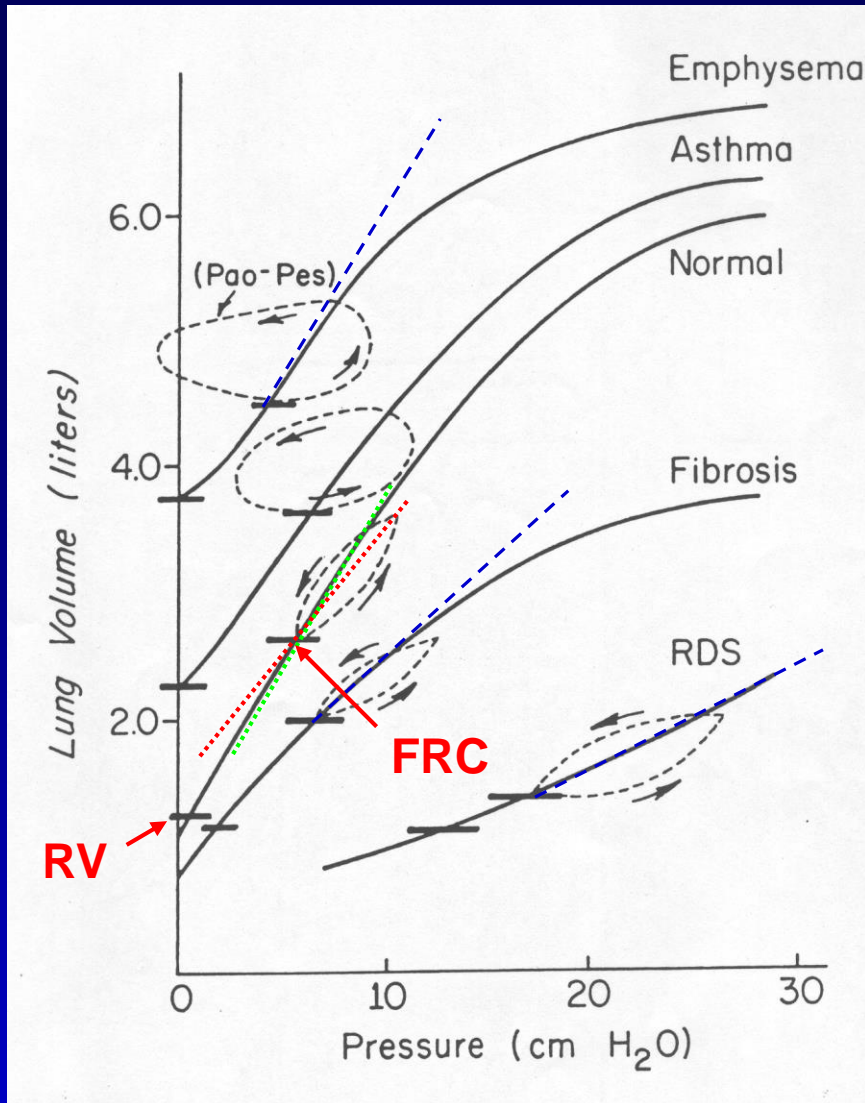
- ↓ surface tension to keep the **same recoil pressure**; ↑ compliance
- ↑ stability of alveoli
- ↓ vascular leakage, ↓ edema

Work of Breathing



- Area of PV loop is the work of breathing

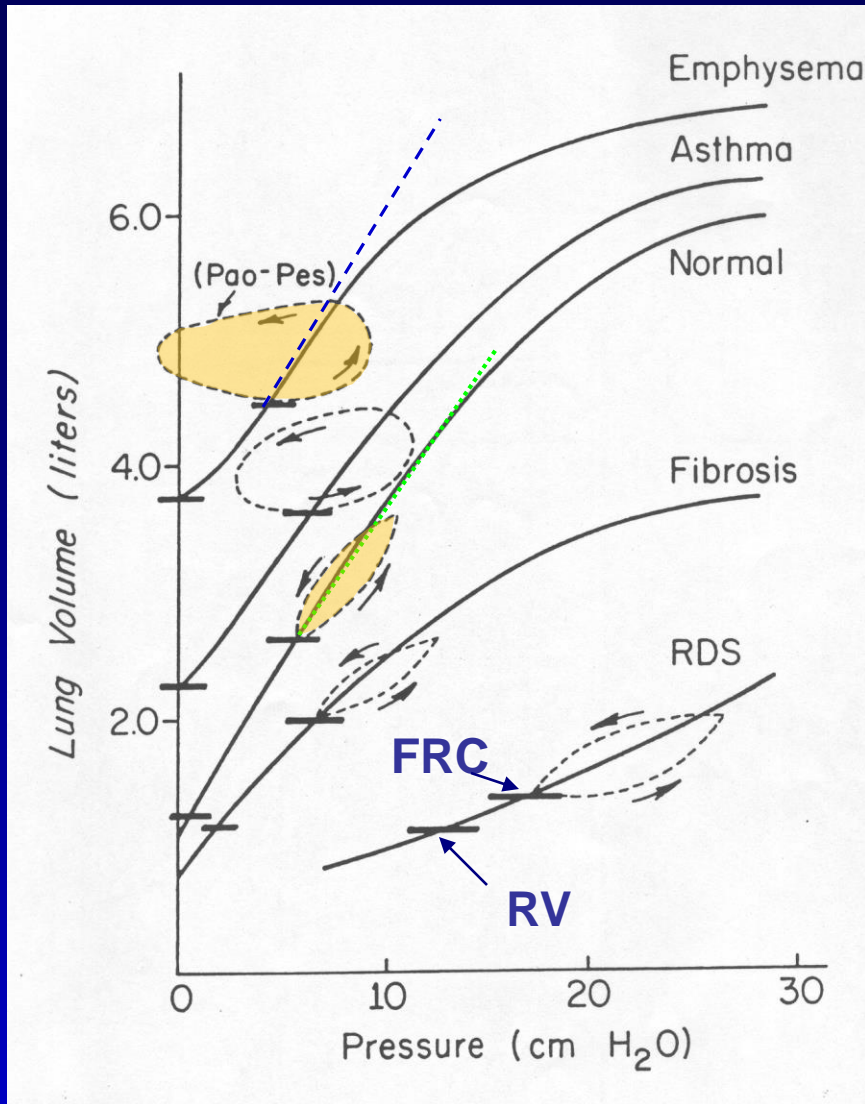
Effects of Diseases on PV Curve



- FRC, RV
- Compliance (Elastic Pr.)
 - **Emphysema (肺氣腫):** ↑ compliance
 - **Fibrosis (肺纖維化):** ↓ compliance
 - **RDS (呼吸性窘迫症候群; Resp. Distress Syndrome):** ↑ surface tension; ↓↓ compliance

為什麼肺氣腫的病人每次的
呼吸都很吃力？

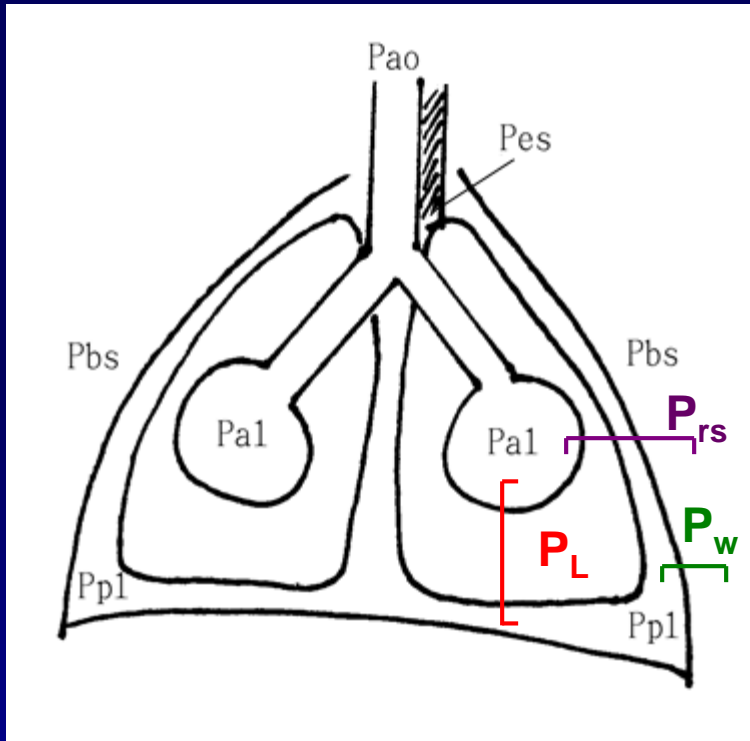
Effects of Diseases on PV Curve



- Work: area of PV loop
 $P_L = \text{resistive } Pr + \text{elastic } Pr$
- Emphysema:
 ✓ elastic $Pr \downarrow$, but resistive Pr
 ↑↑
 → work ↑

Key Points

- General concepts and terminology
- Mechanical properties
 - 1. Compliance
 - 2. Resistance
 - 3. Pressure-volume (P-V) curve of the lungs
 - 4. Lung-chest wall coupling



$$P_L = P_{al} - P_{pl} \quad (1)$$

$$P_w = P_{pl} - P_{bs} \quad (2)$$

$$(1) + (2)$$

$$P_{rs} = P_L + P_w = P_{al} - P_{bs}$$

$$a) P_{bs} = 0 \rightarrow P_w = P_{pl} = P_{es}$$

$$P_{rs} = P_{al}$$

P_L : transpulmonary Pr. (跨肺壓)

P_{al} : alveolar Pr.

P_{pl} : intrapleural Pr.

P_w : trans-chest wall Pr. (跨胸壁壓)

P_{bs} : body surface Pr.

P_{rs} : respiratory sys. Pr.

P_{ao} : airway opening Pr.

P_{es} : esophageal Pr.

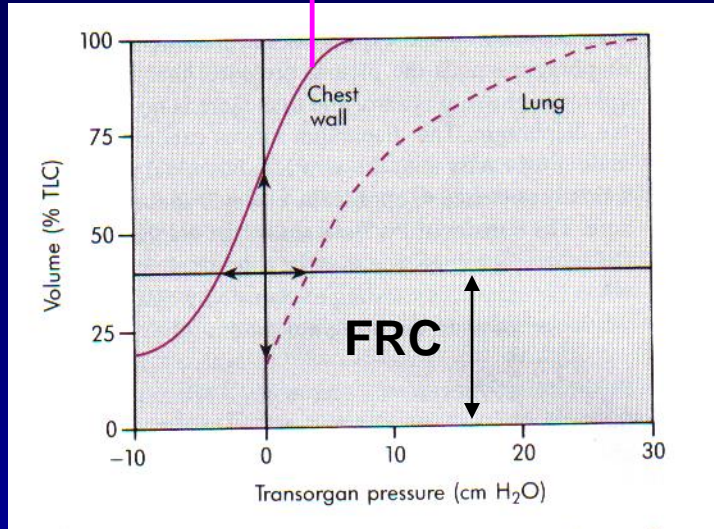
$$b) P_{ao} = \dot{V}R_{aw} + P_{al}$$

When flow=0, $P_{ao} = P_{al} = P_{rs}$

$$c) P_L = P_{al} - P_{pl} = P_{ao|flow=0} - P_{es}$$

Elastic Recoil of the Chest Wall

$$P_w = P_{pl} = P_{es}$$



$P_w < 0$, the chest wall is compressed

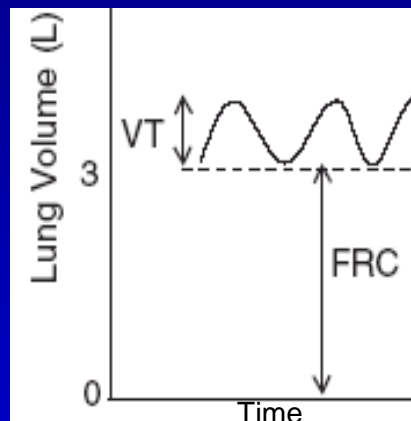
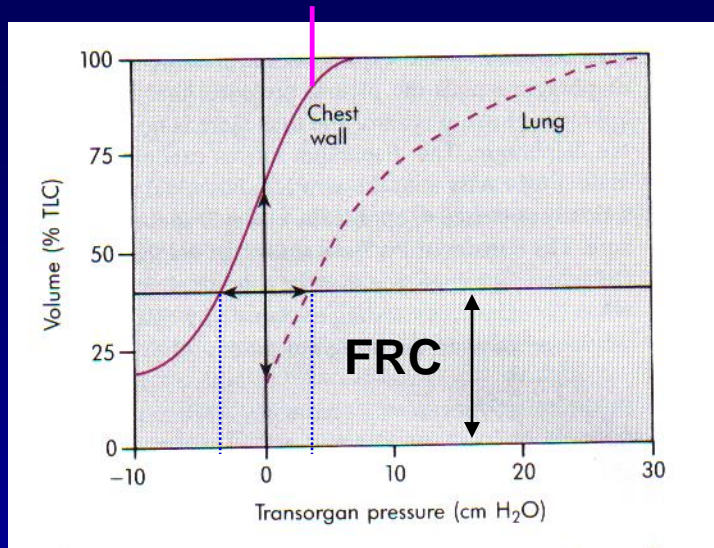
So, in pneumothorax

⇒ thoracic cavity increases

The dynamic compliance of cell wall is not different from its static compliance

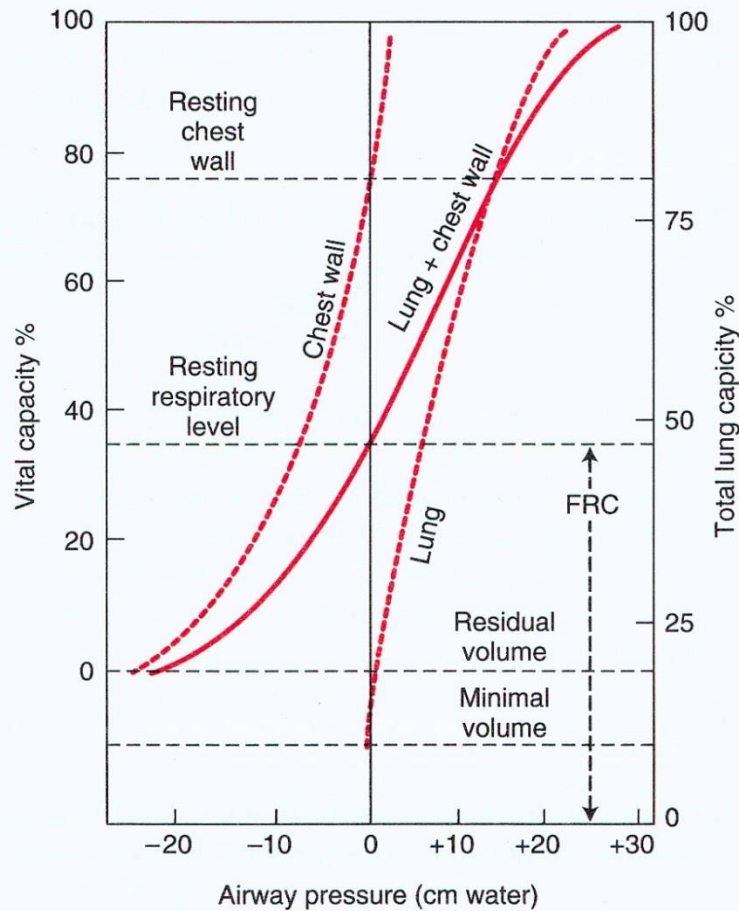
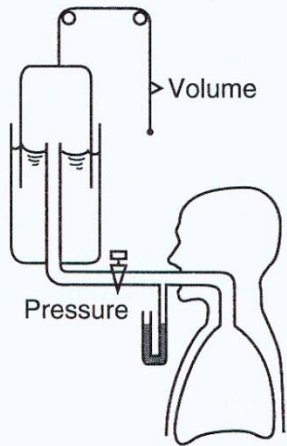
Elastic Recoil of the Chest Wall

$$P_w = P_{pl} = P_{es}$$



- FRC (*functional residual capacity*) is where the recoil forces of chest wall is **equal** but **opposite** to the recoil forces of the lung
- When lung vol. is below FRC, the chest wall becomes progressively stiffer (C_w decreases)
- When lung vol. is above FRC, $\rightarrow P_w$ changes from negative to positive
 $\rightarrow C_w$ increases and constant until the lung vol. is near TLC

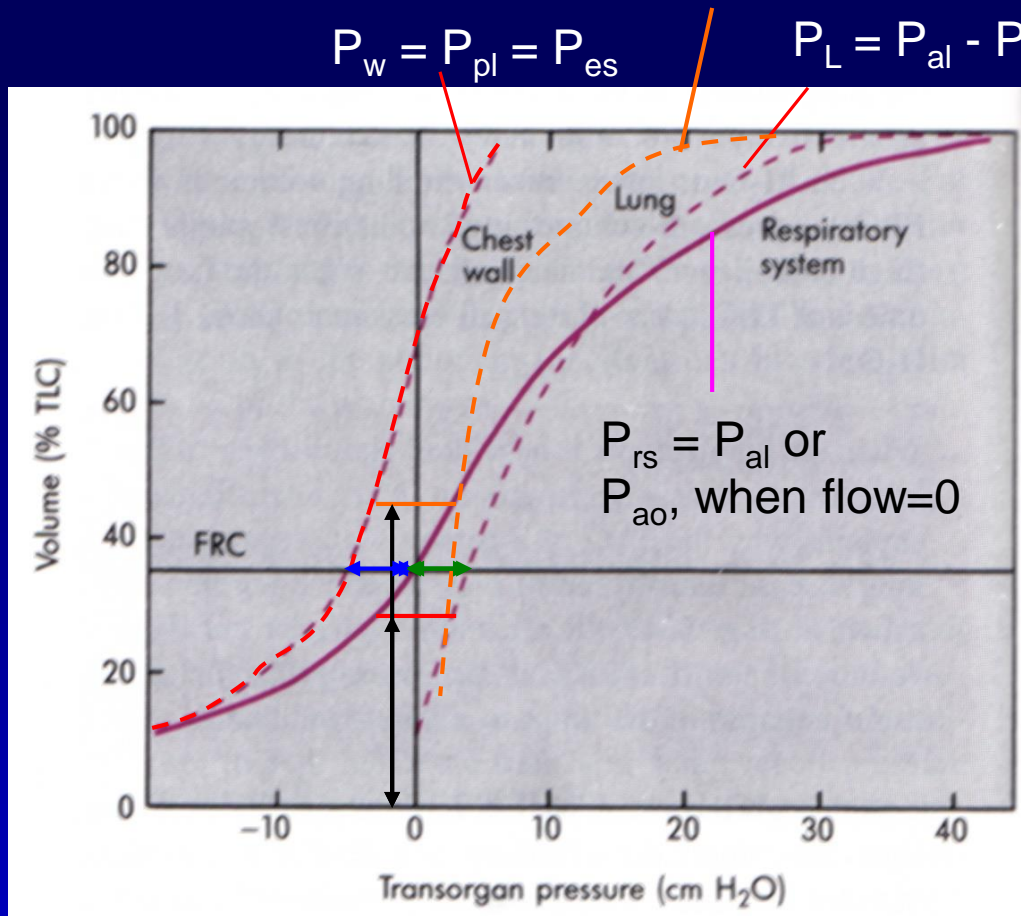
Lung-Chest Wall Coupling in **Static** Status at Different Lung Volume



- $P_w = P_{pl} = P_{es}$
- $P_{rs} = P_{al}$ or P_{ao} , when flow=0
- $P_L = P_{al} - P_{pl}$

Lung-chest Wall Coupling to Determine FRC

Emphysema

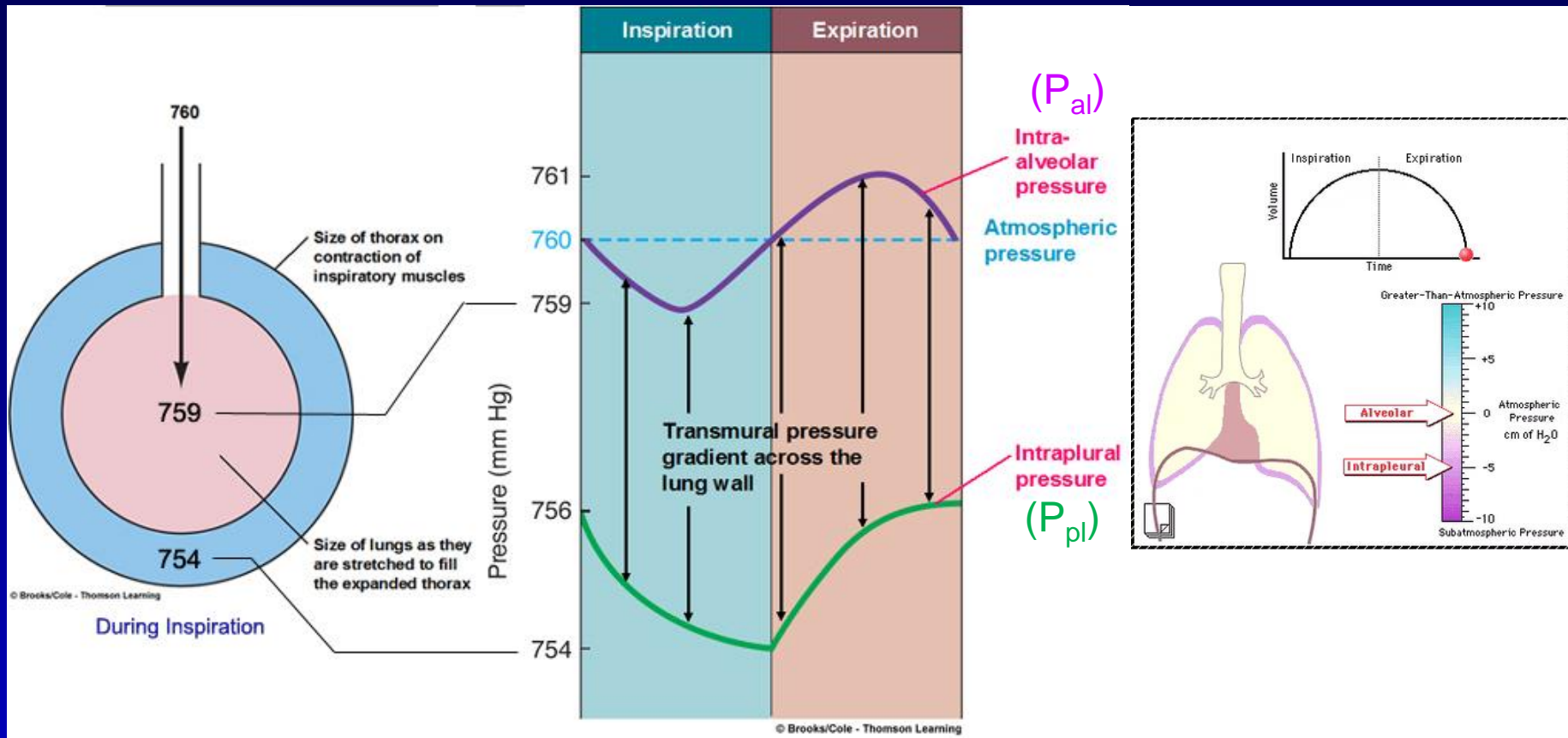


At FRC, $P_{rs} = 0 = P_L + P_W$

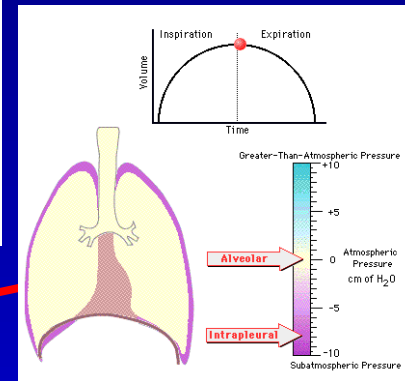
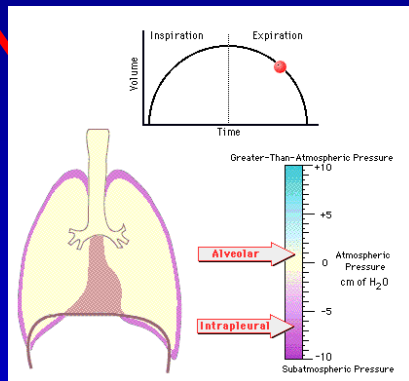
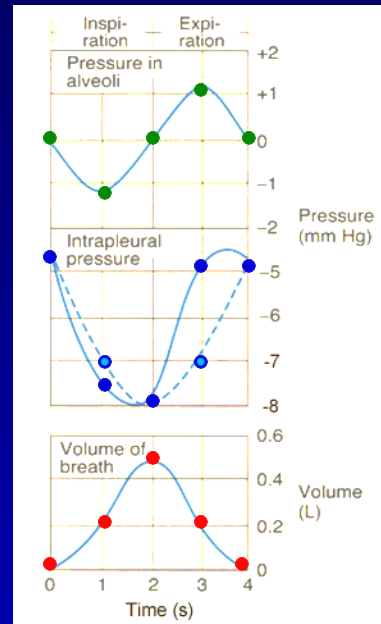
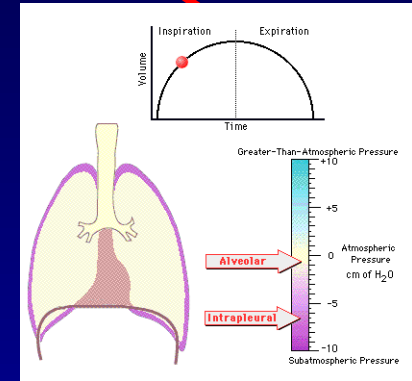
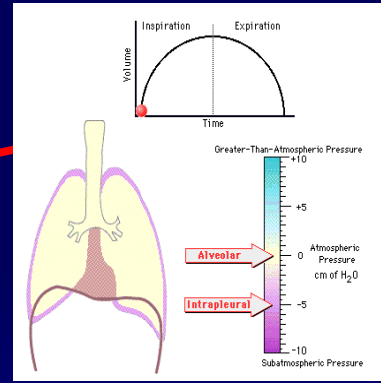
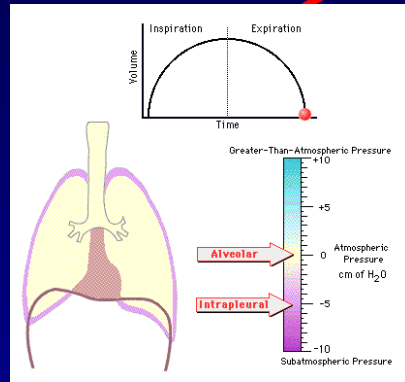
If P_w shifts to right, e.g. kyphoscoliosis (restrictive lung disease)
 → FRC decrease

In emphysema (obstructive lung disease), C_L increases
 → P_L shifts to left
 → FRC increase

Transmural Pressure Across the Lung Wall in **Dynamic** Status

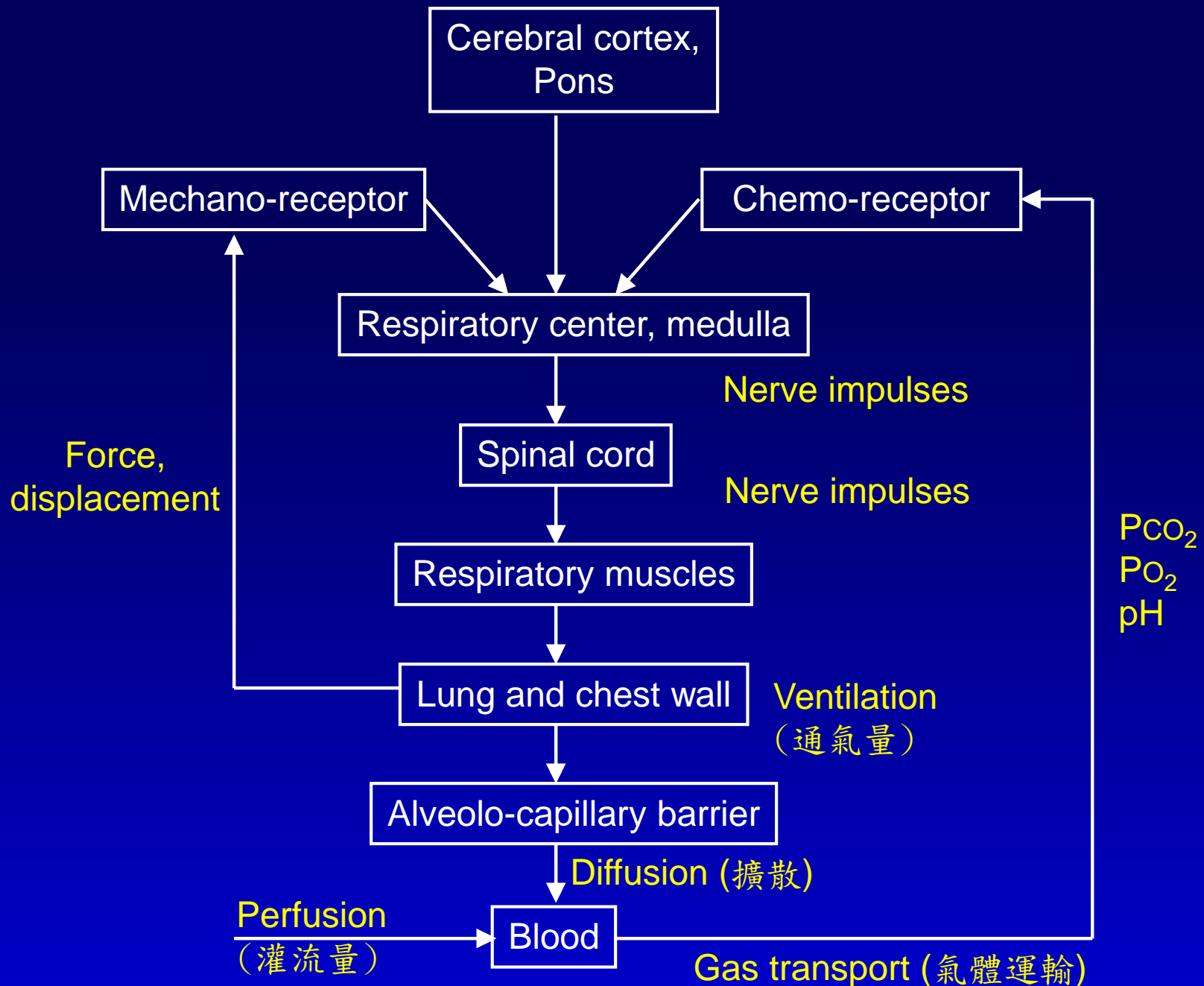


The Mechanics of Quiet Breathing



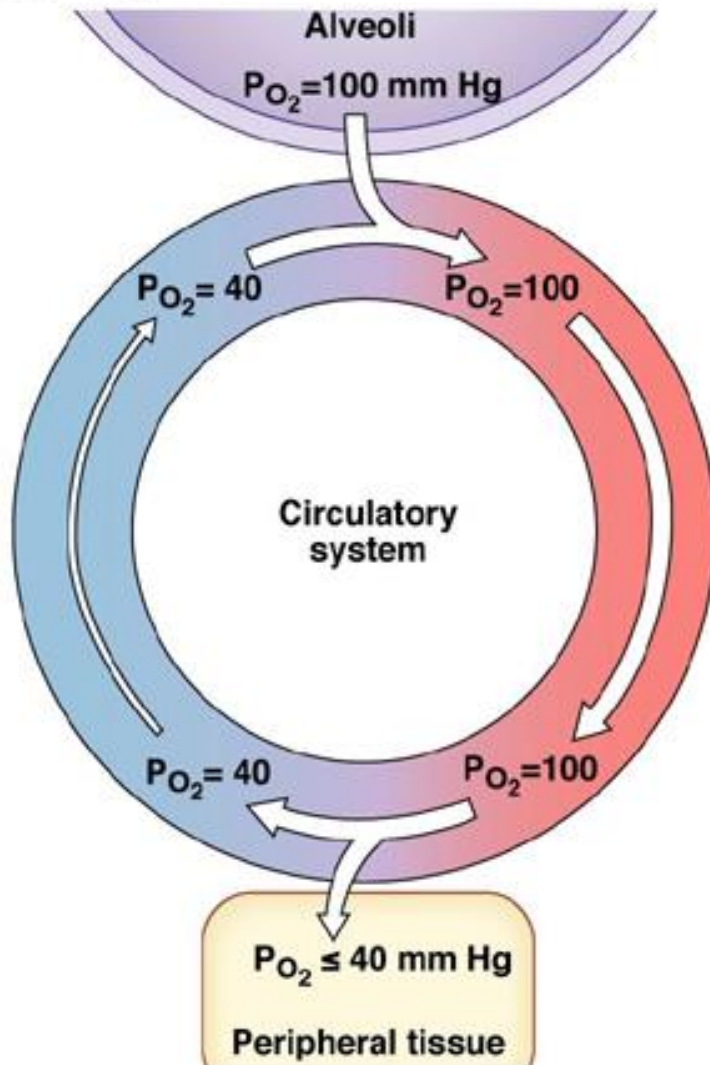
Outline

- Background
- Structure and function
- Ventilation
- Perfusion and ventilation/perfusion ratio
- Static/Dynamic respiratory mechanics
- **Diffusion (擴散) and gas transport (氣體運輸)**
- Neural control of respiration
- Chemical control of respiration
- Acid-base balance

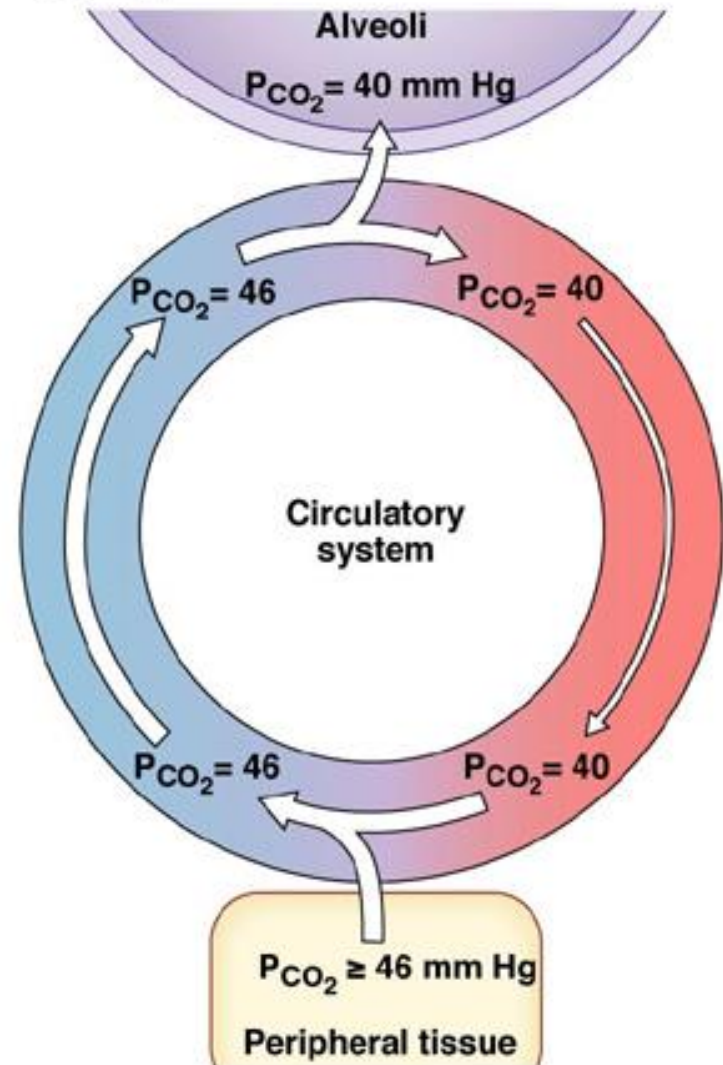


Diffusion and Gas Transport

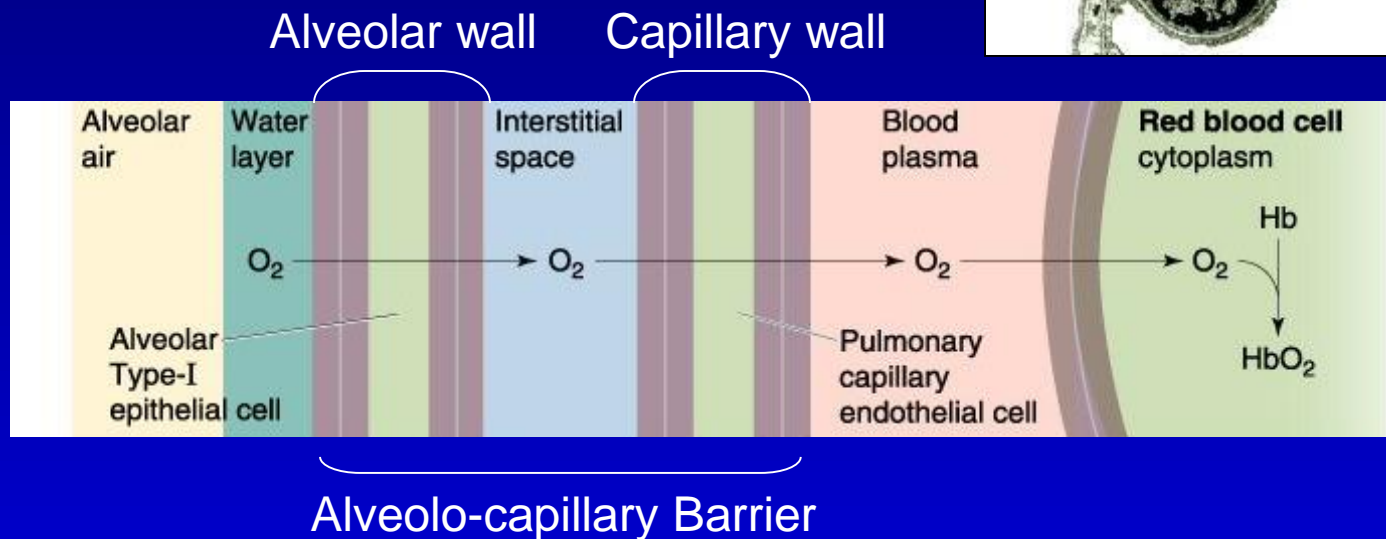
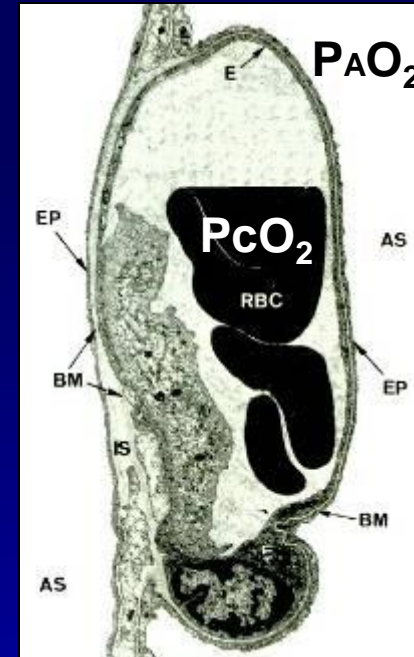
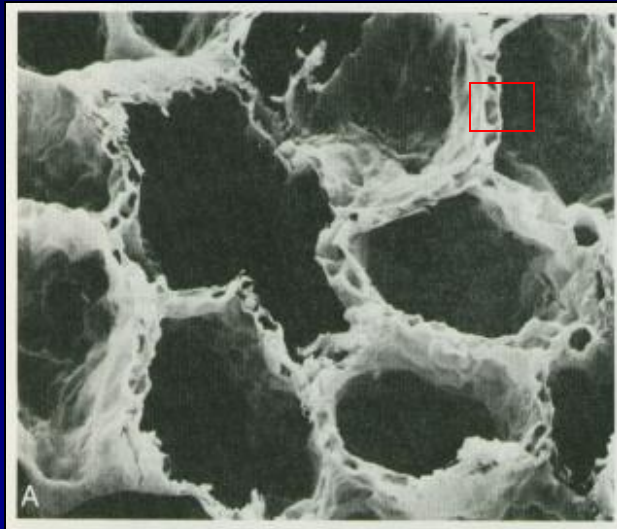
(a) Oxygen diffusion



(b) CO₂ diffusion

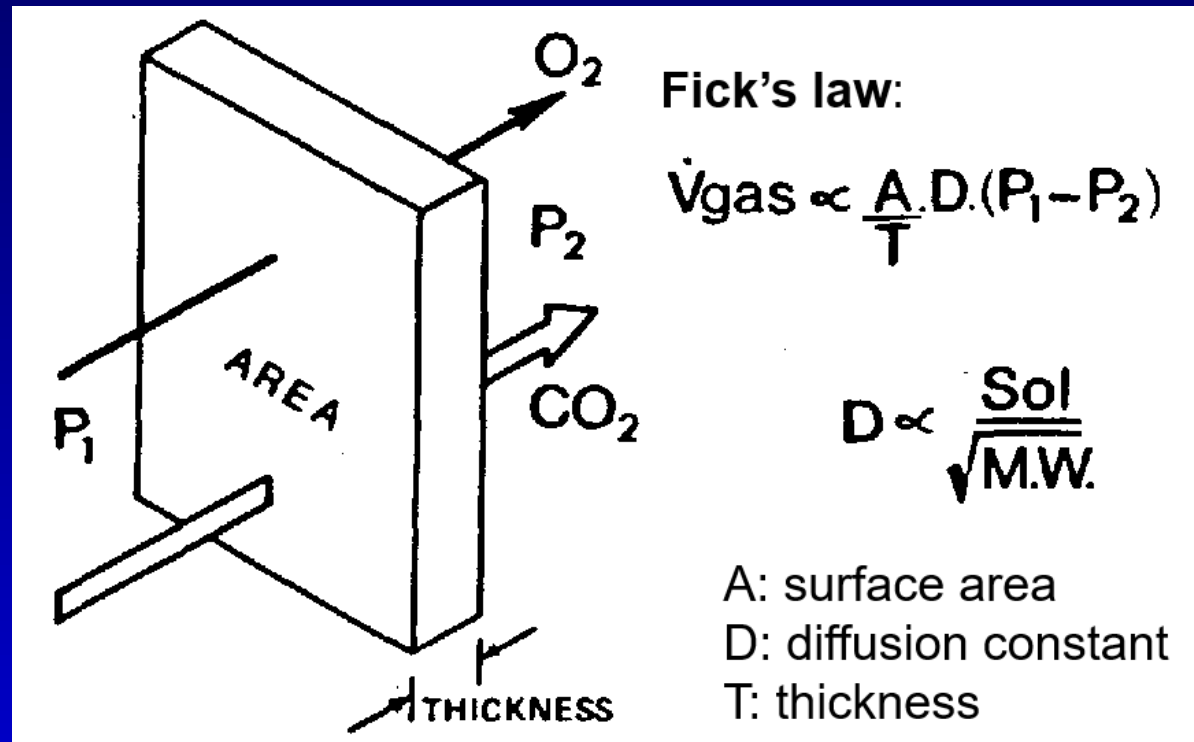


Alveolo-Capillary Barrier



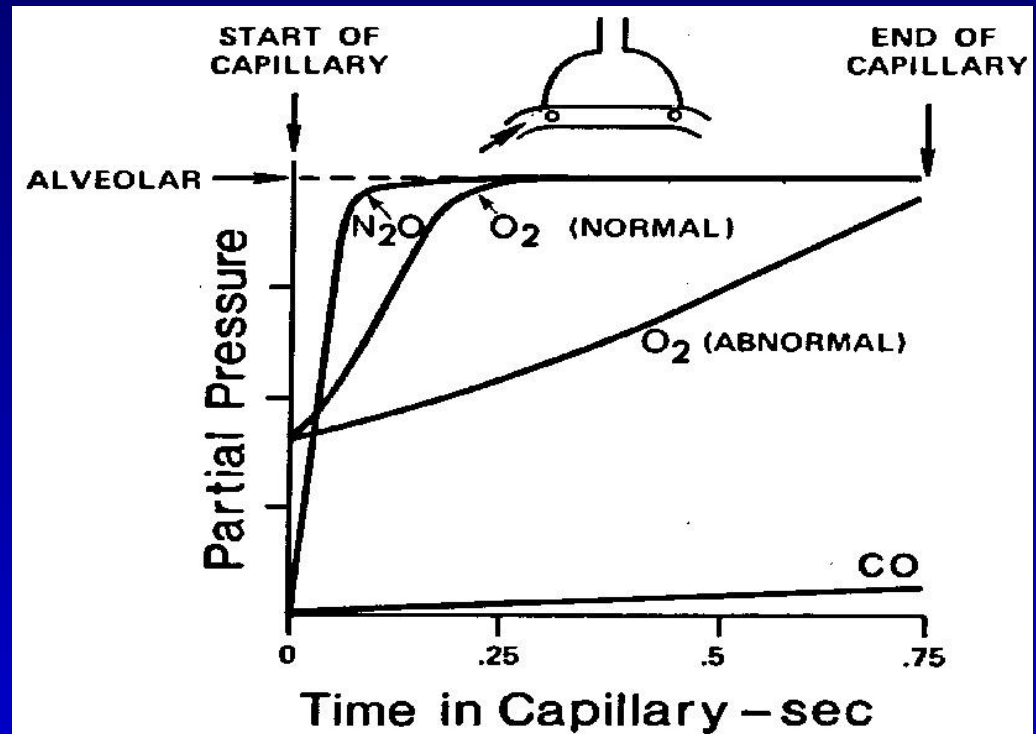
Diffusion

- The movement of molecules from a area in which they are highly concentrated to a area in which they are less concentrated
- Fick's law



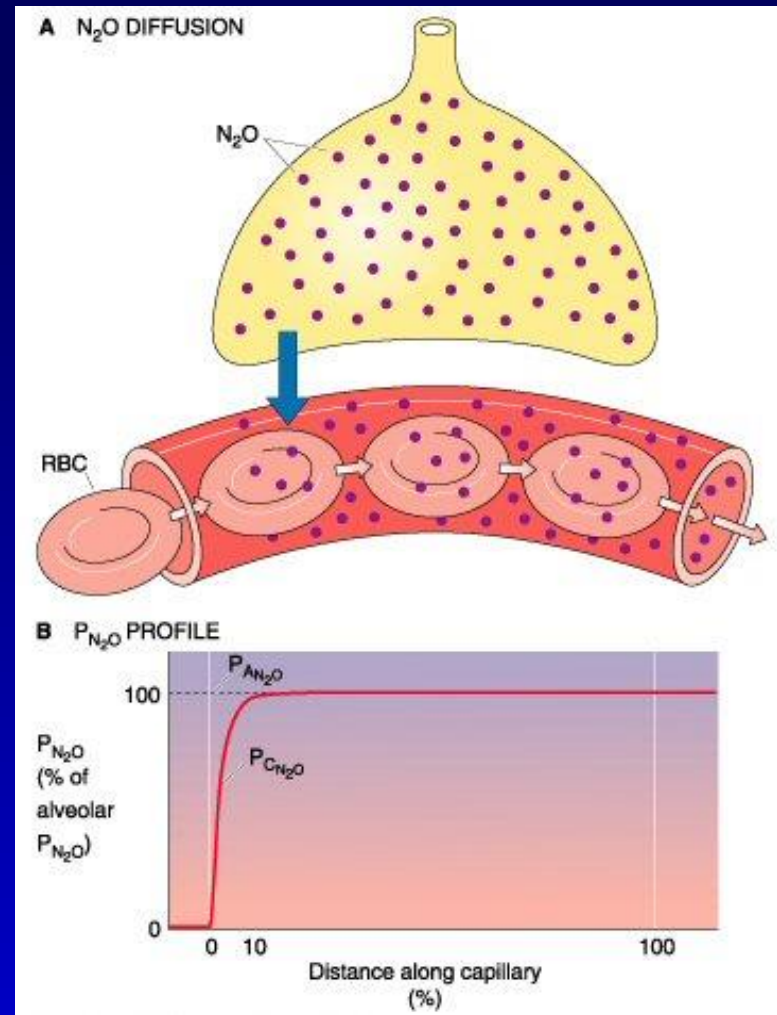
Capillary Transit Time

- Capillary transit time is ~ 0.75 sec
- If diffusion defects, **exercise** results in poor oxygenation of blood
- N_2O : perfusion-limited
- CO : diffusion-limited



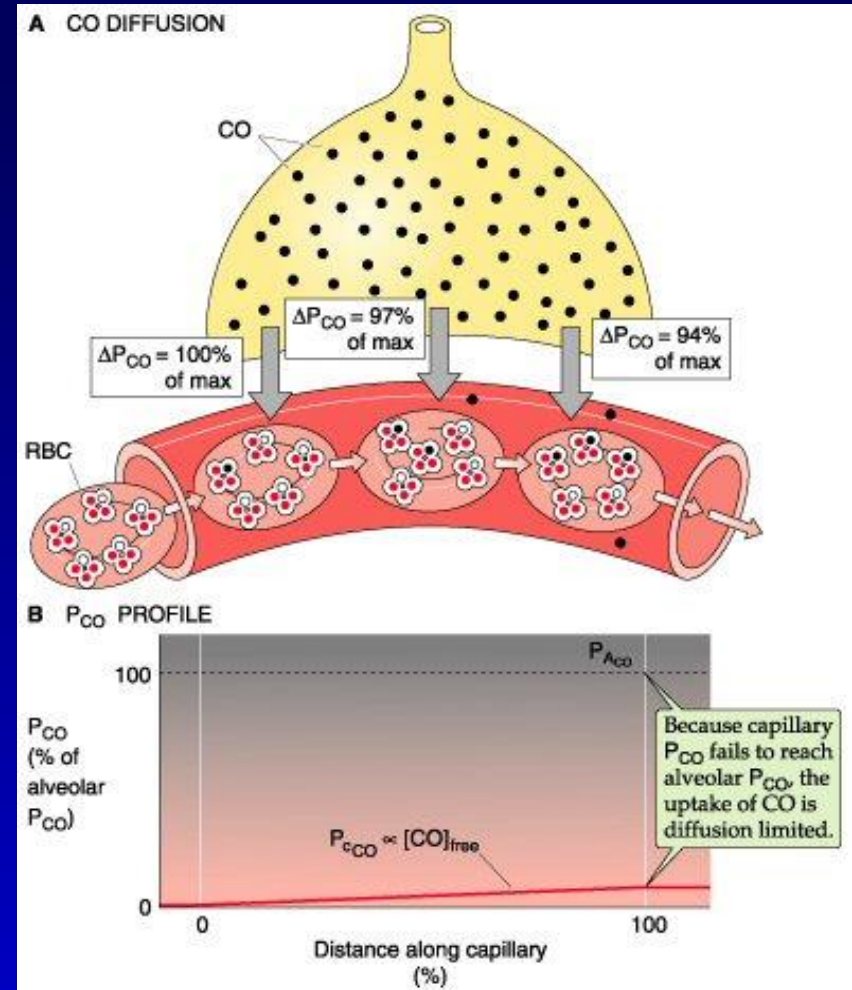
Perfusion-limited Gas

- Uptake of nitrous oxide (N_2O) is perfusion-limited
- Hb does not bind N_2O
- P_{AN_2O} and P_{cN_2O} rapidly equilibrate
- To increase uptake of a perfusion-limited gas, blood flow must increase

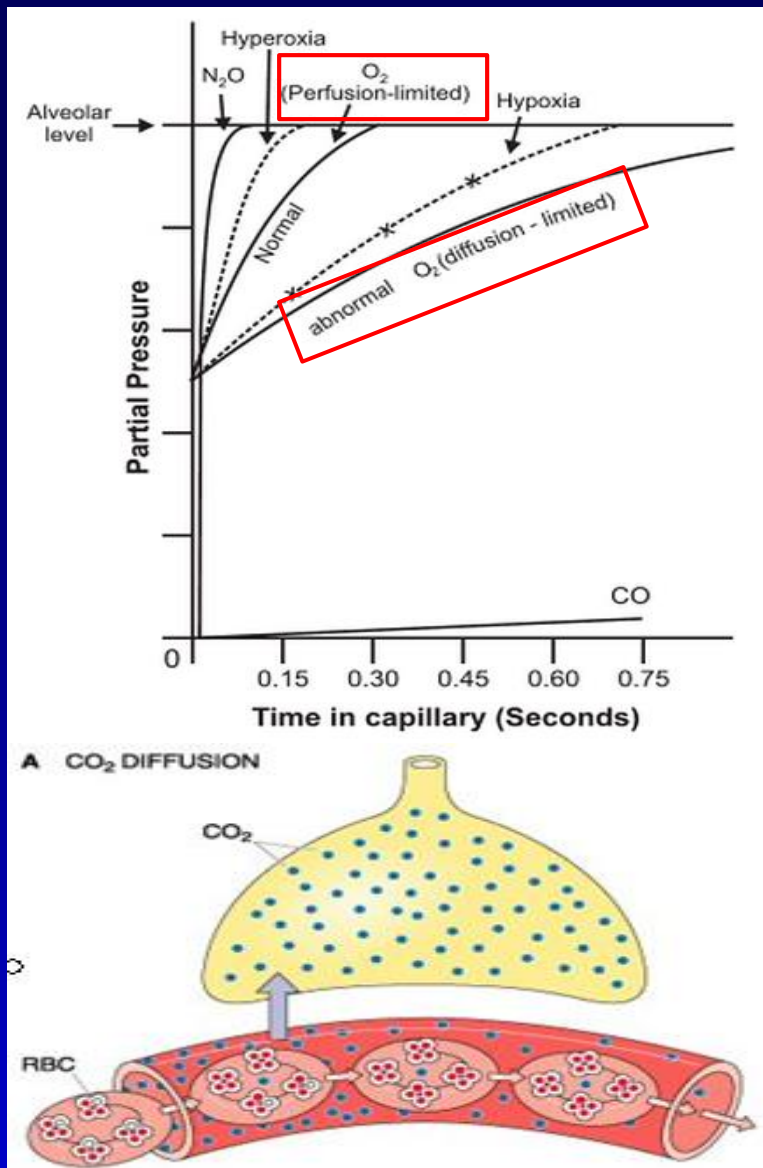


Diffusion-limited Gas

- Uptake of CO is diffusion-limited
- High affinity of Hb for CO
- No equilibration
 $P_c\text{CO} \approx P_v\text{CO} \approx 0$
- To increase uptake of a diffusion-limited gas, ΔP must increase

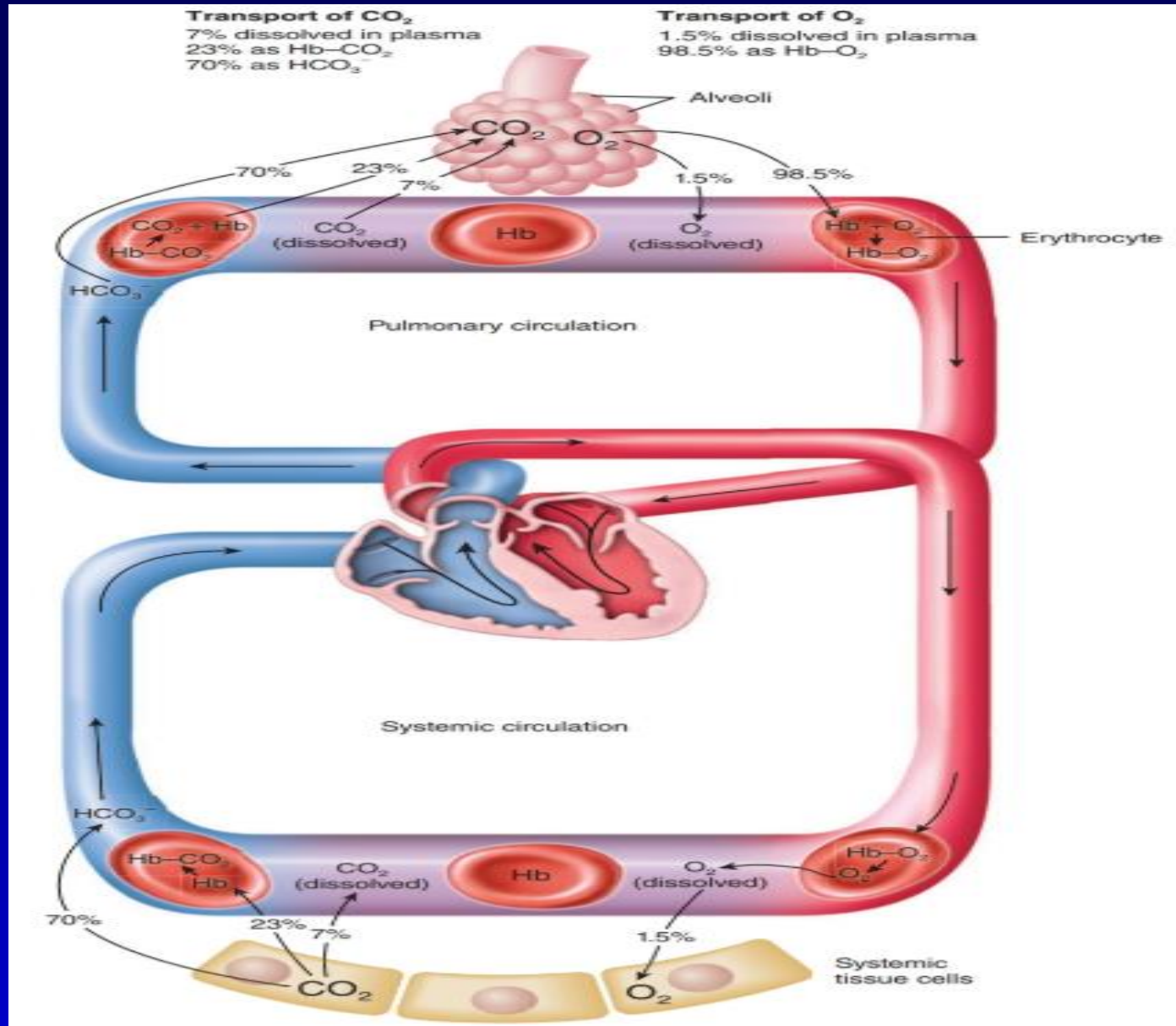


Diffusion and Perfusion Limitations



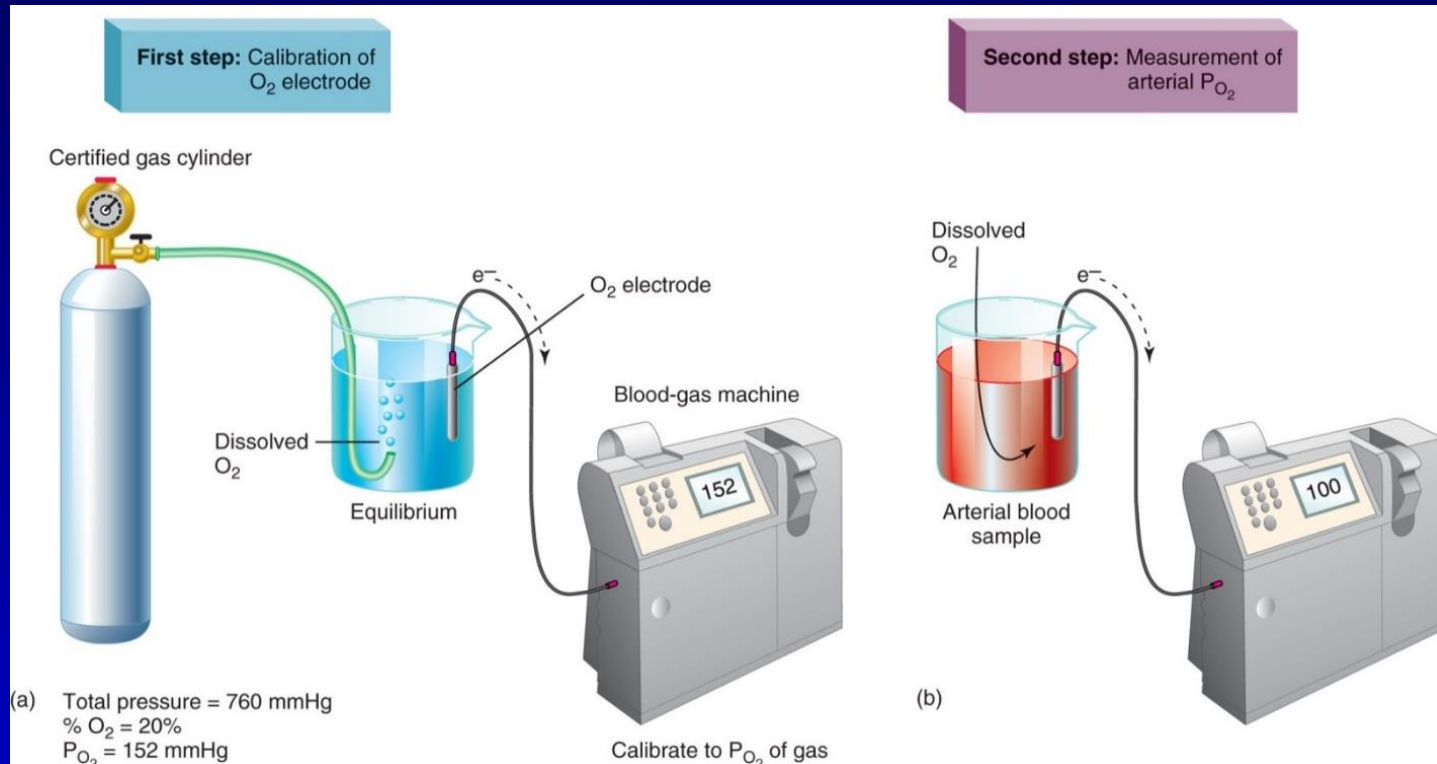
- O₂ is normally **perfusion-limited gas**
 - ✓ E.g., exercise
- If $D_{L}O_2$ is decreased in disease, O₂ becomes more diffusion limited
- CO₂ exchange is much **less affected** when perfusion increases or D_L decreases

Transport of O₂ and CO₂



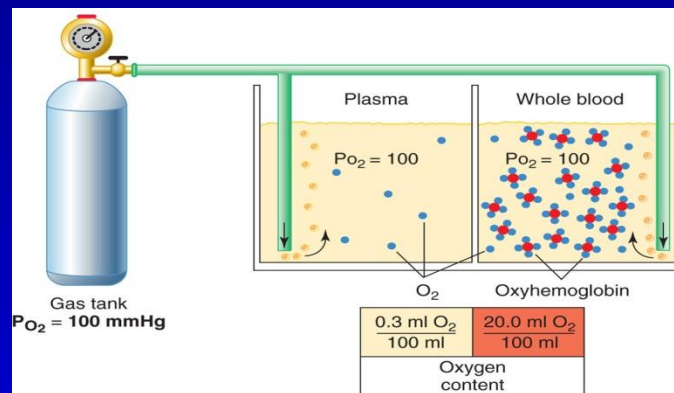
Blood Gas Measurement

- **Arterial blood gas (ABG):** a sample of arterial blood, which provides you with P_{aO_2} , P_{aCO_2} , pH
- Uses an oxygen electrode



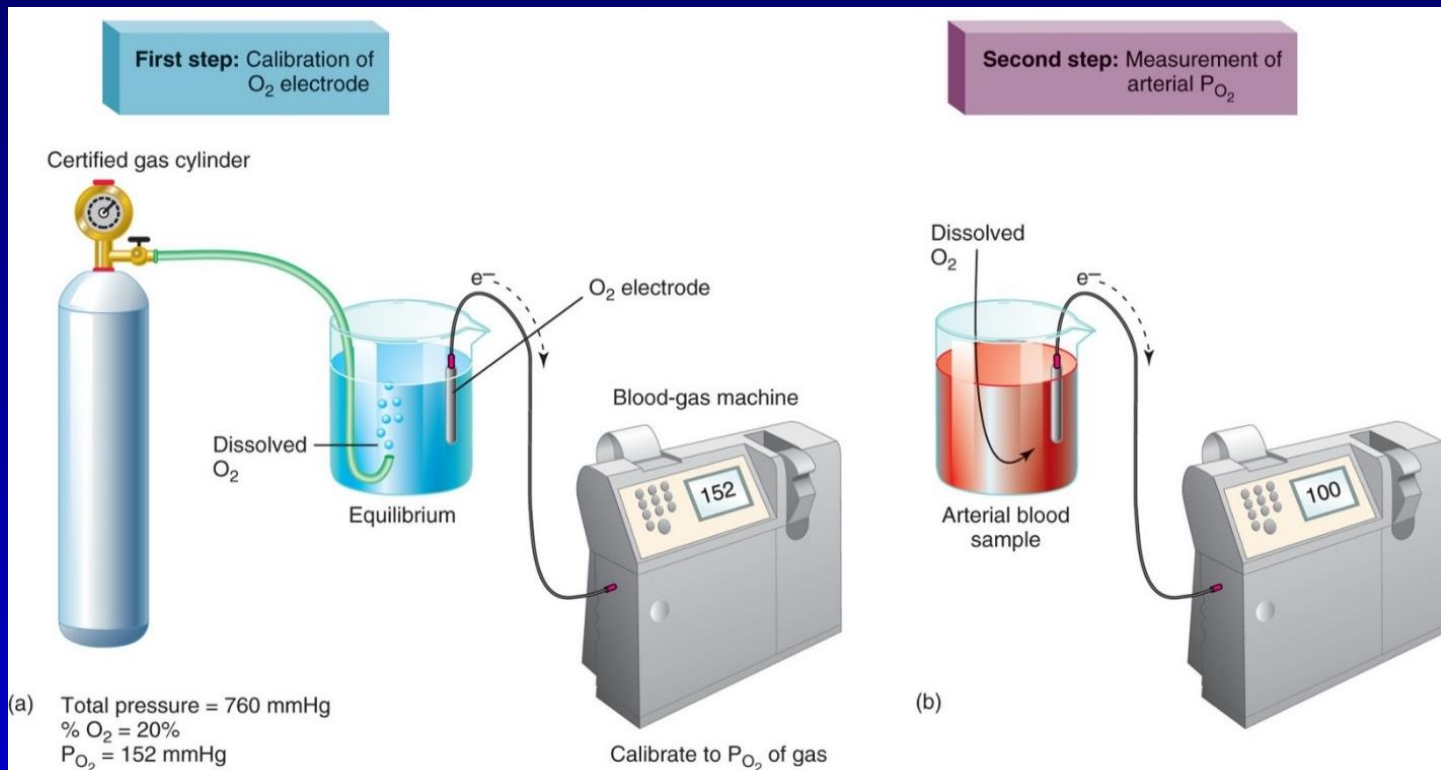
Oxygen Transport

- Two ways of O₂ carried in blood
 - ✓ Dissolved O₂ in plasma (<5%)
 - ✓ Bound to hemoglobin (Hb) (> 95%)
- Dissolved O₂
 - ✓ Normal arterial blood with a P_{O₂} of 100 mmHg contains 0.3 ml dissolved O₂/100 ml of blood
- Bound to hemoglobin (Hb)
 - ✓ Oxygen dissociation curve and factors affecting the curve



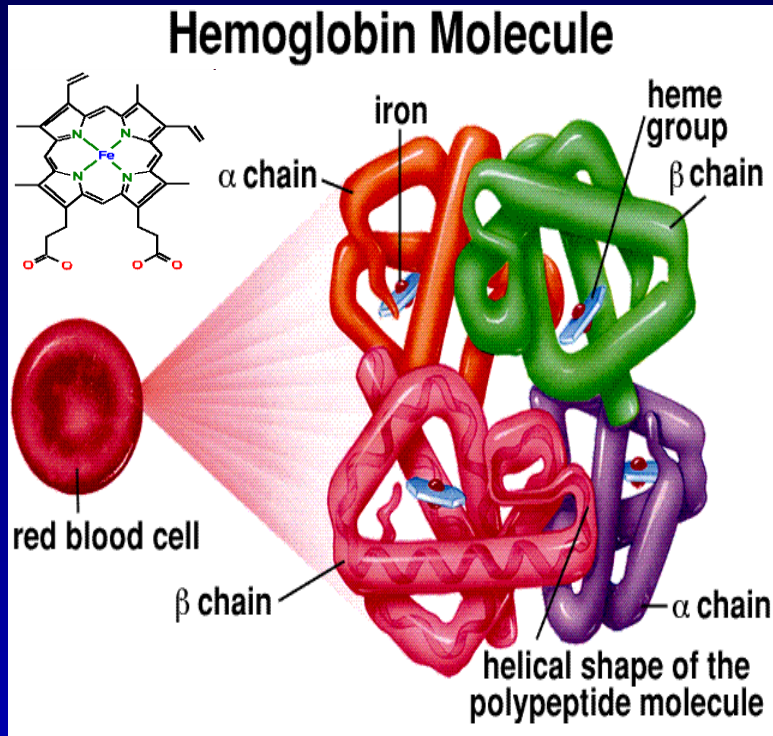
Blood Gas Measurement

- Only measures oxygen dissolved in the blood plasma
 - ✓ Not measure oxygen in red blood cells
 - ✓ Anemia dose not affect P_aO_2 , P_aCO_2 , but decrease P_vO_2
 - anemia → tissue hypoxia → P_{vO_2} , S_{vO_2} decreases



O₂ Bound to Hb

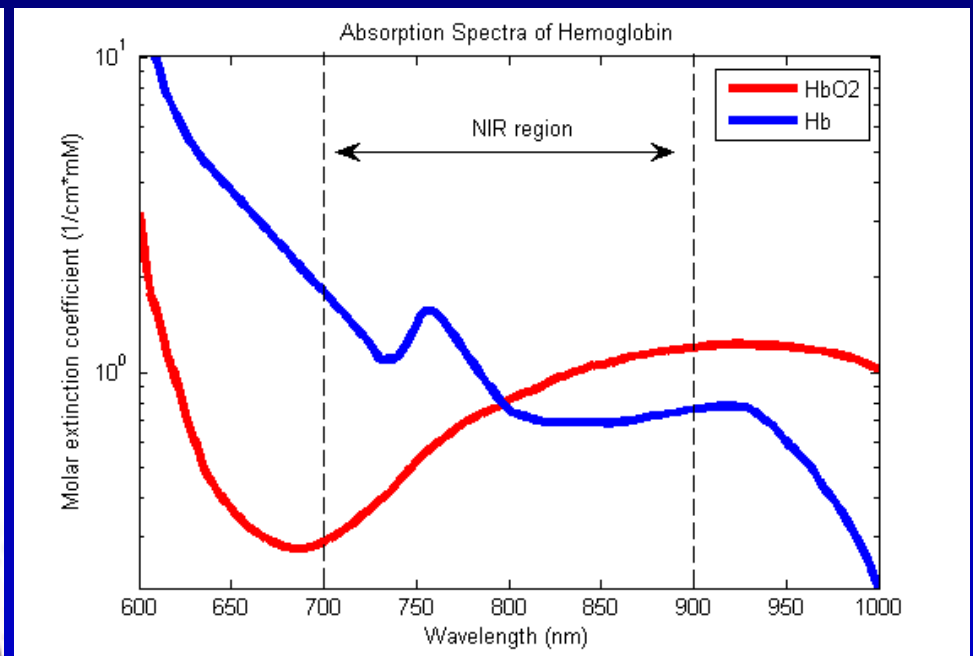
- Hemoglobin (Hb): heme + globin



- ✓ A [$\alpha(2):\beta(2)$] tetrameric hemoprotein that is carried by **erythrocytes**
- ✓ An **iron** atom in heme is responsible for the binding of **oxygen**
- ✓ Each Hb combines with **4 O₂** molecules

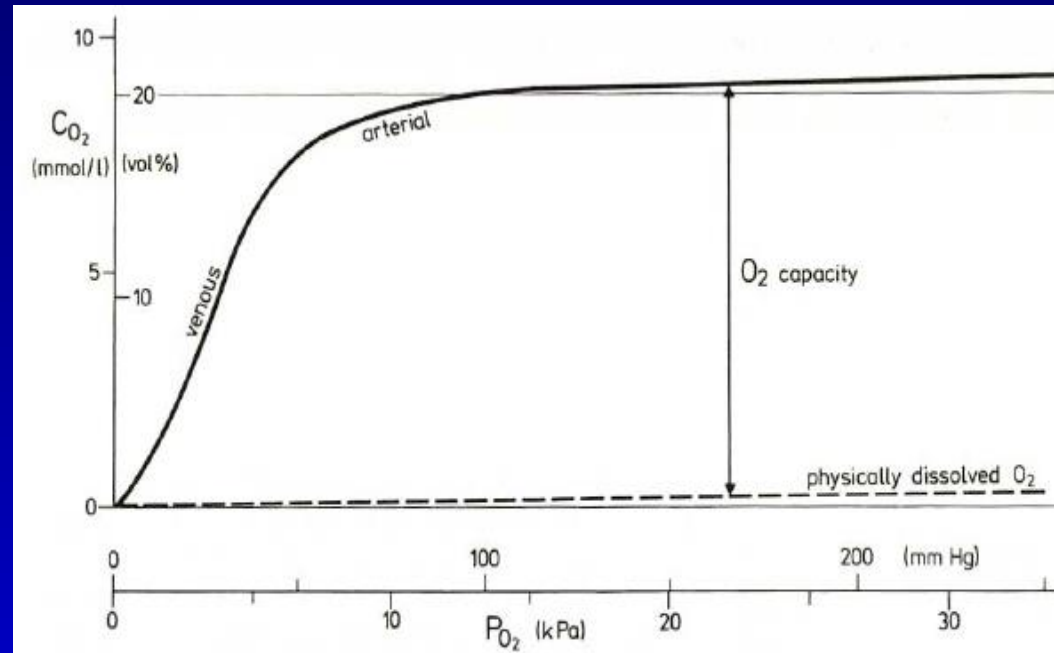
What does pulse oximeter measure?

1. O_2 dissolved in the blood plasma
2. O_2 bound with hemoglobin



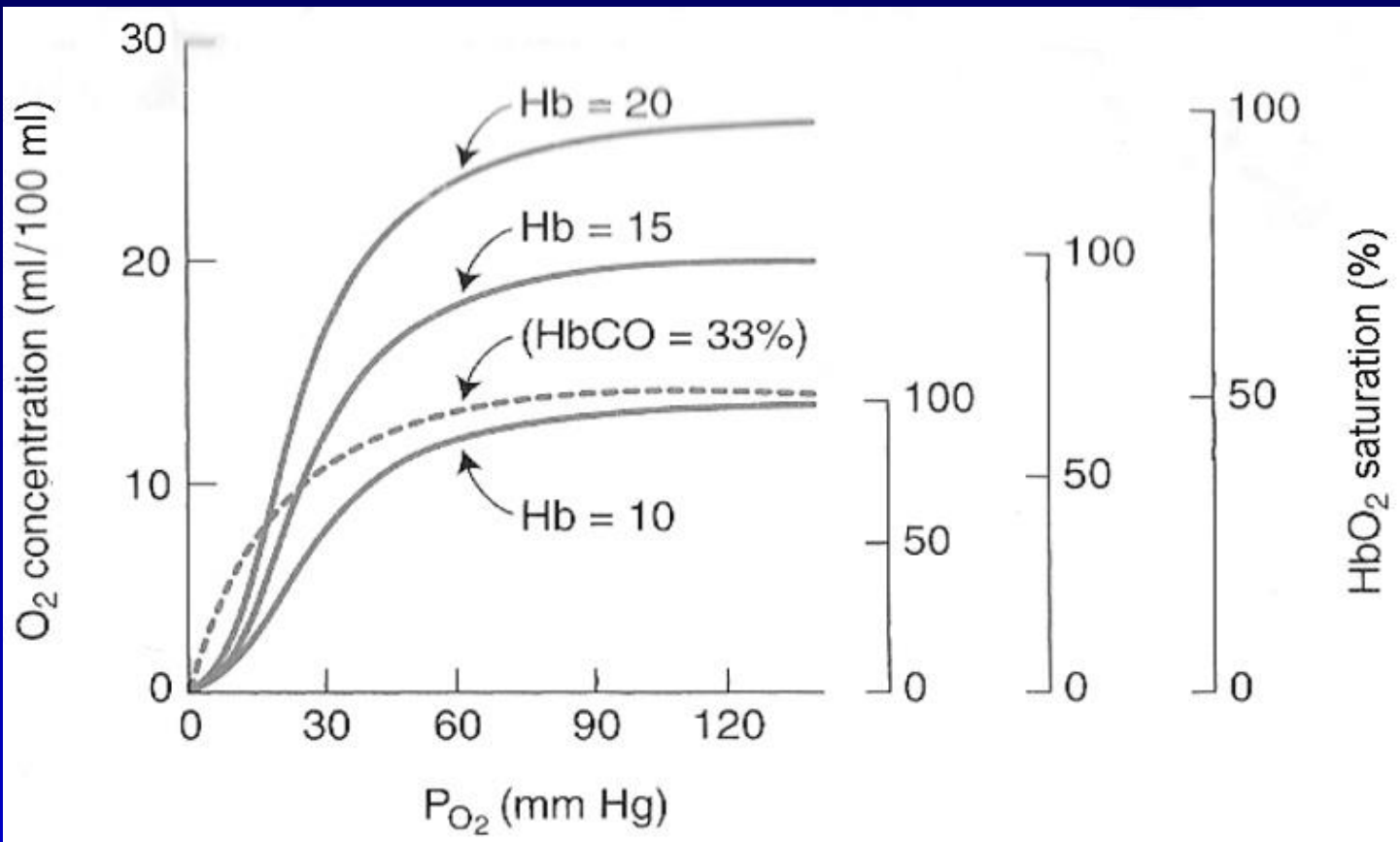
O₂ Bound to Hb

- **O₂ capacity**: max. amount of O₂ that can combine with Hb
= 15 g of Hb/100ml blood x 1.39 ml/g of Hb
= 20.9 ml /100ml blood
- **O₂ capacity varies individually**
- % saturation =
$$\frac{\text{Hb-bound O}_2}{\text{O}_2 \text{ Capacity}} \times 100\%$$
- O₂ dissociation curve
 - * Pulse oximeter:
Measures the absorbance of A940 (HbO₂) and A660 (Hb) to calculate saturation of peripheral O₂ (SpO₂)



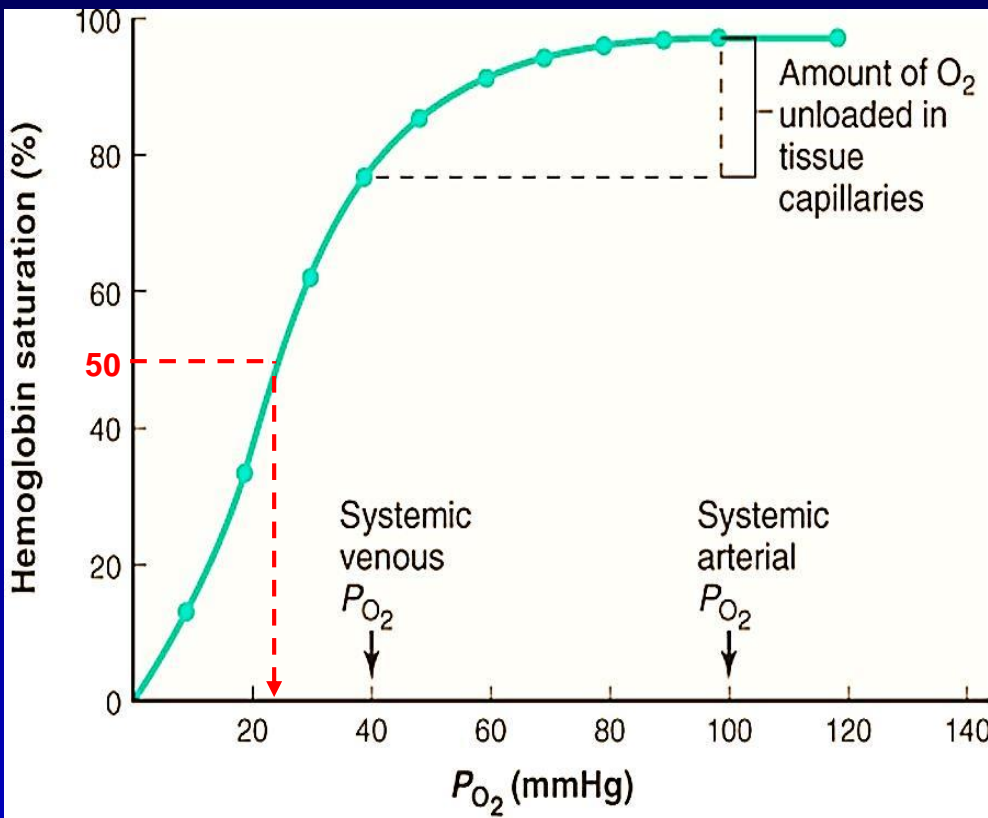
O₂ Concentration & Saturation in Anemia

- **Anemia (貧血)**: low O₂ concentration (low O₂ capacity) **but** normal O₂ saturation



O₂ Bound to Hb

- Characteristics of O₂ dissociation curve

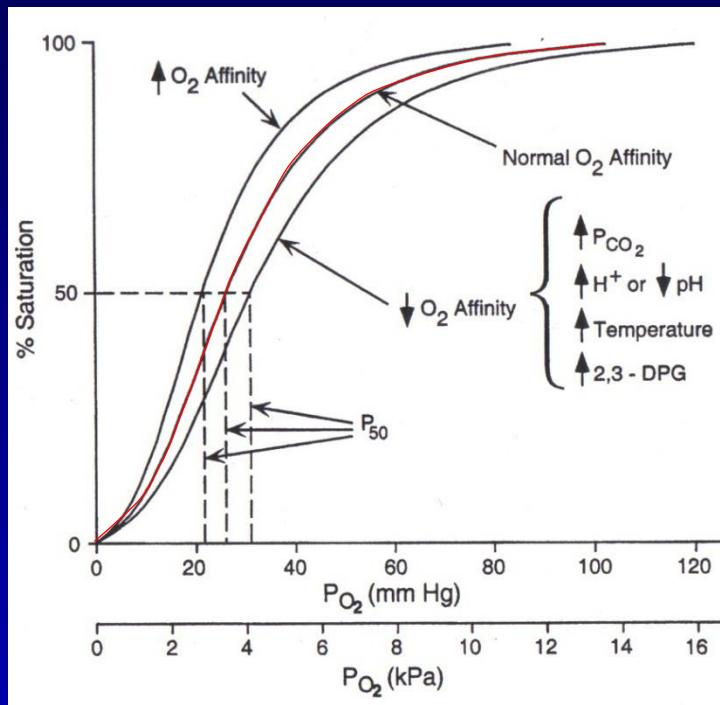


- ✓ P_{O_2} = 100 mmHg (alveolar)
 - near saturated
 - affinity good
- ✓ P_{O_2} ~ 70-100 mmHg
 - little change
 - affinity changed little
- ✓ P_{O_2} ~ 40-50 mmHg (tissue)
 - unload O₂ easily
 - affinity decrease

- P_{50} : P_{O_2} at 50% of saturation
- Higher P_{50} → lower affinity

O₂ Bound to Hb

- Factors affecting O₂ saturation curve



Right shift of curve (O₂ unloading):

→ P₅₀ ↑ (↓ affinity)

✓ ↑ P_{CO₂}: **Bohr effect**

✓ ↑ H⁺ (↓ pH)

✓ ↑ body temp

✓ ↑ 2,3-DPG (**d**iphosphoglycerate):

formed during anaerobic metabolism of RBC

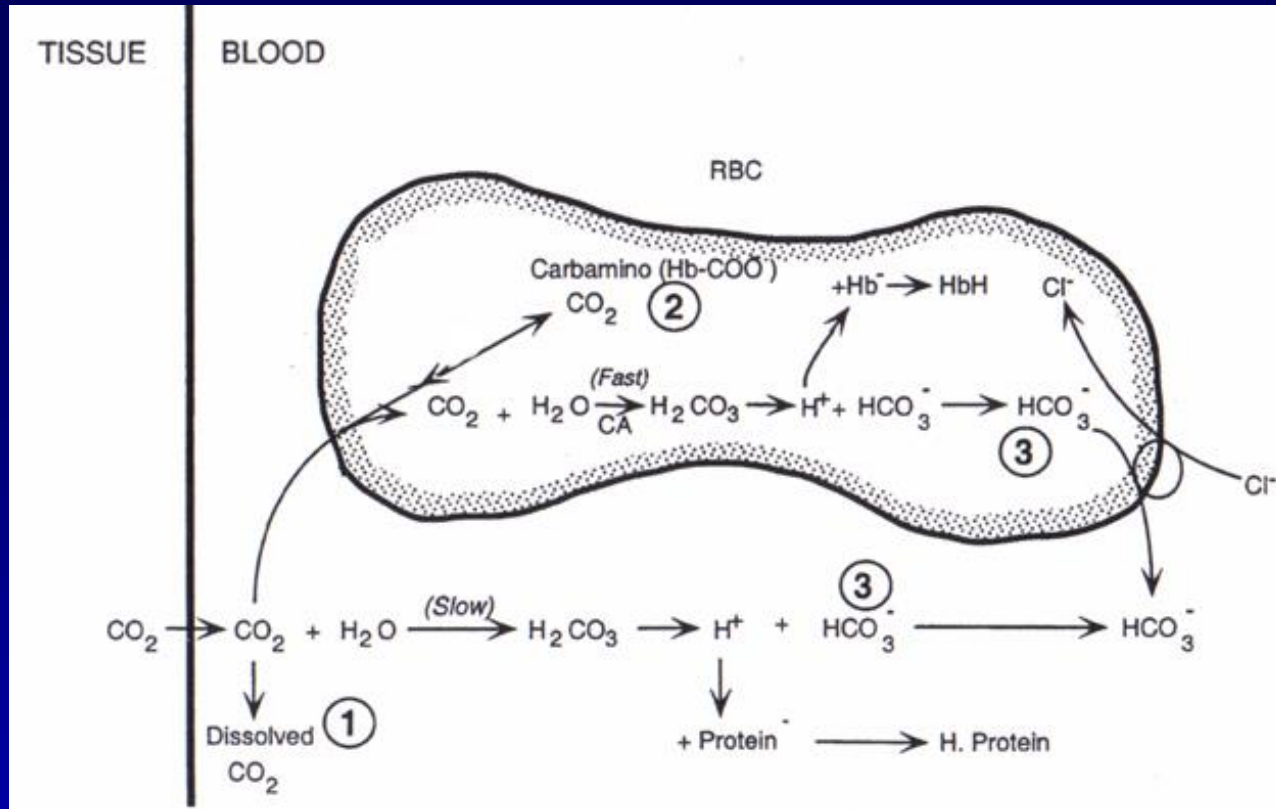
➤ high altitude, hypoxia, chronic lung disease

Example: exercise

CO₂ Transport

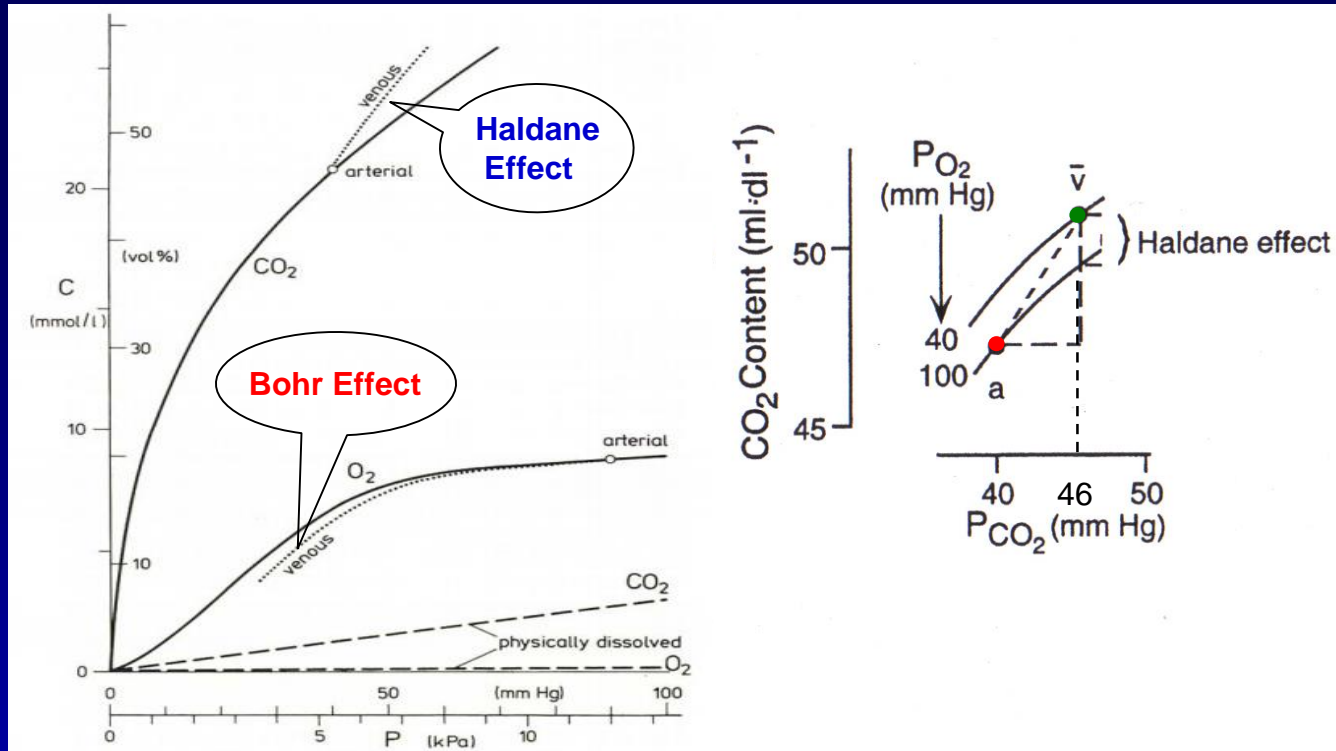
- Three ways of CO₂ carried in blood: transported from the body cells back to the lungs
 - ✓ Dissolved CO₂ in plasma (7-10%)
 - ✓ Carbamino Hb (15-30%): bound to hemoglobin (Hb)
 - ✓ Bicarbonate (HCO₃⁻) (60-70%):
 - most transport in **plasma**
 - most formed in RBC by **carbonic anhydrase**

CO₂ Transport



- H⁺ + Hb: to maintain the blood pH
- CA: carbonic anhydrase
- Chloride shift: exchange with HCO₃⁻ to maintain electrical neutrality

CO₂ Equilibrium Curve



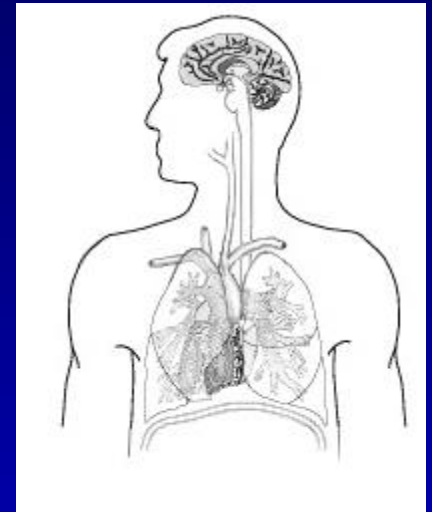
- **Haldane effect:** deoxygenation of Hb increases its affinity for CO₂ (curve left shift)
- * **Bohr effect:** P_{CO₂} decreases the binding affinity of O₂ to hemoglobin (curve right shift)

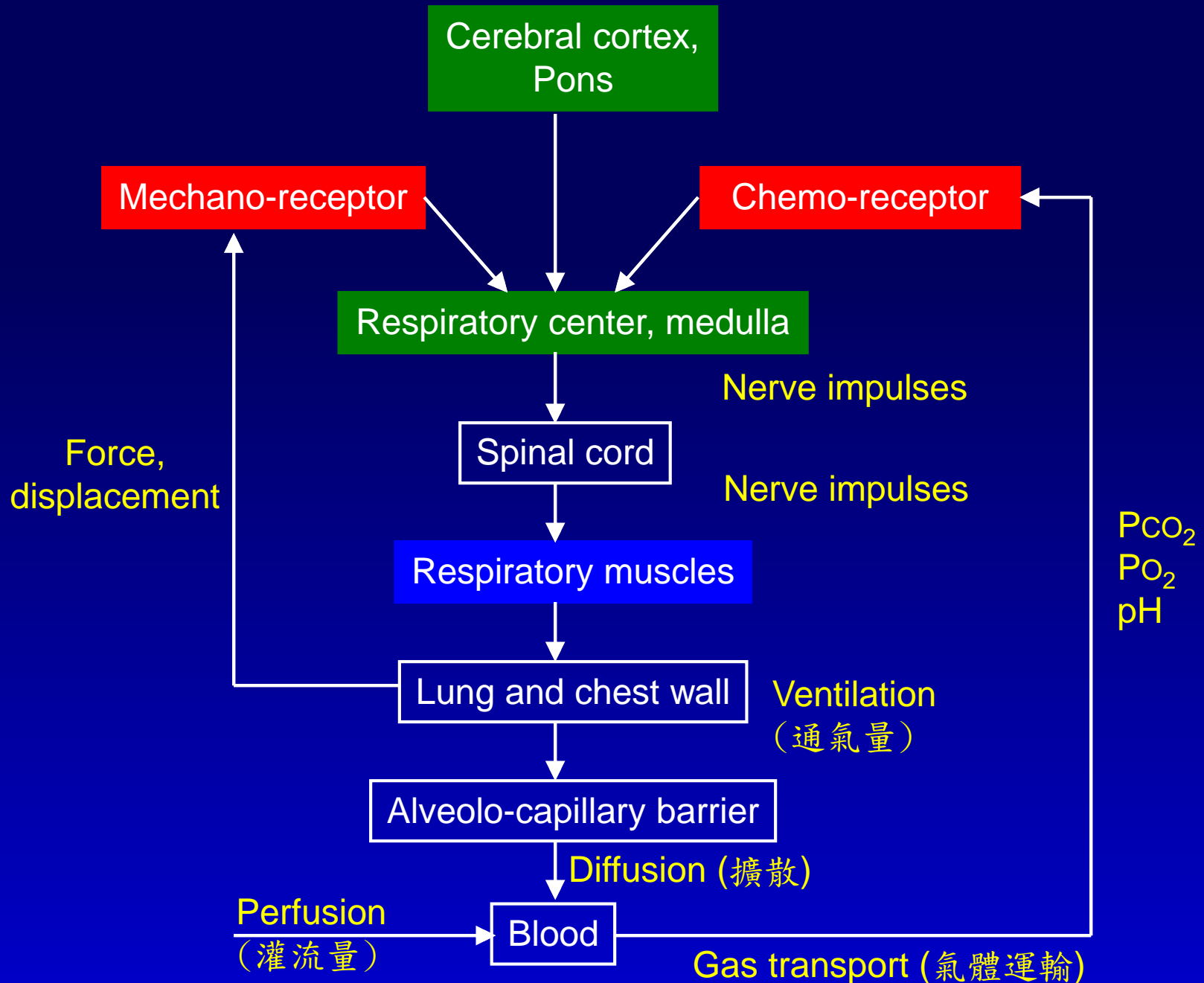
Outline

- Background
- Structure and function
- Ventilation
- Perfusion and ventilation/perfusion ratio
- Static/Dynamic respiratory mechanics
- Diffusion and gas transport
- **Neural control of respiration** (呼吸的神經調控)
- Chemical control of respiration
- Acid-base balance

Control of Respiration

- Three components of resp. control system:
 - ✓ **Sensors (receptors):** e.g. mechanoreceptor
 - ✓ **Central controller:** e.g. medulla
 - ✓ **Effectors:** e.g. resp. muscle
- Central control of breathing
 - ✓ **Origination:** cause of resp. drive in the brain
 - ✓ **Rhythmicity:** how do neurons integrate to give insp./exp.
 - ✓ **Adjustment:** meet different conditions, e.g. exercise



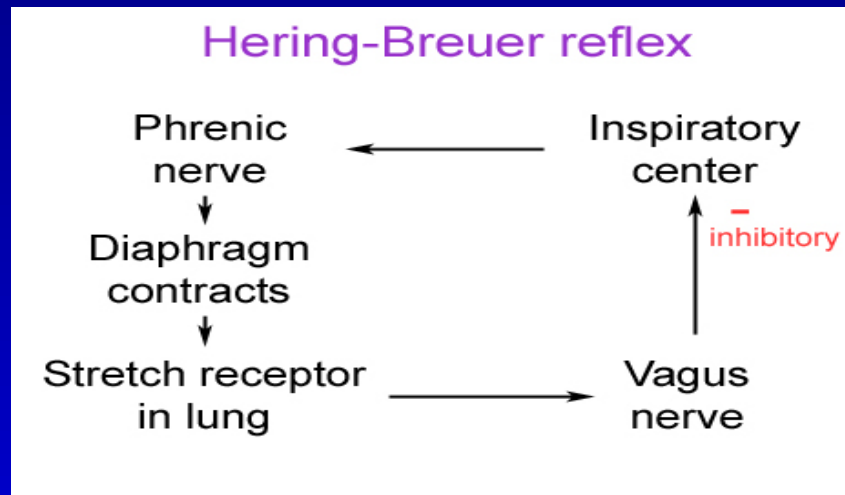


Receptors

- Chemoreceptors
 - ✓ Peripheral: carotid bodies; aortic bodies
 - ✓ Central: medulla
- Lung receptors:
 - ✓ Rapidly adapting receptor (irritant R.)
 - Located between airway epithelial cell
 - Stimulated by noxious gas; cigarette smoke; inhaled dusts; cold air
 - Effect: hyperpnea; bronchoconstriction; coughing; mucous secretion

Receptors

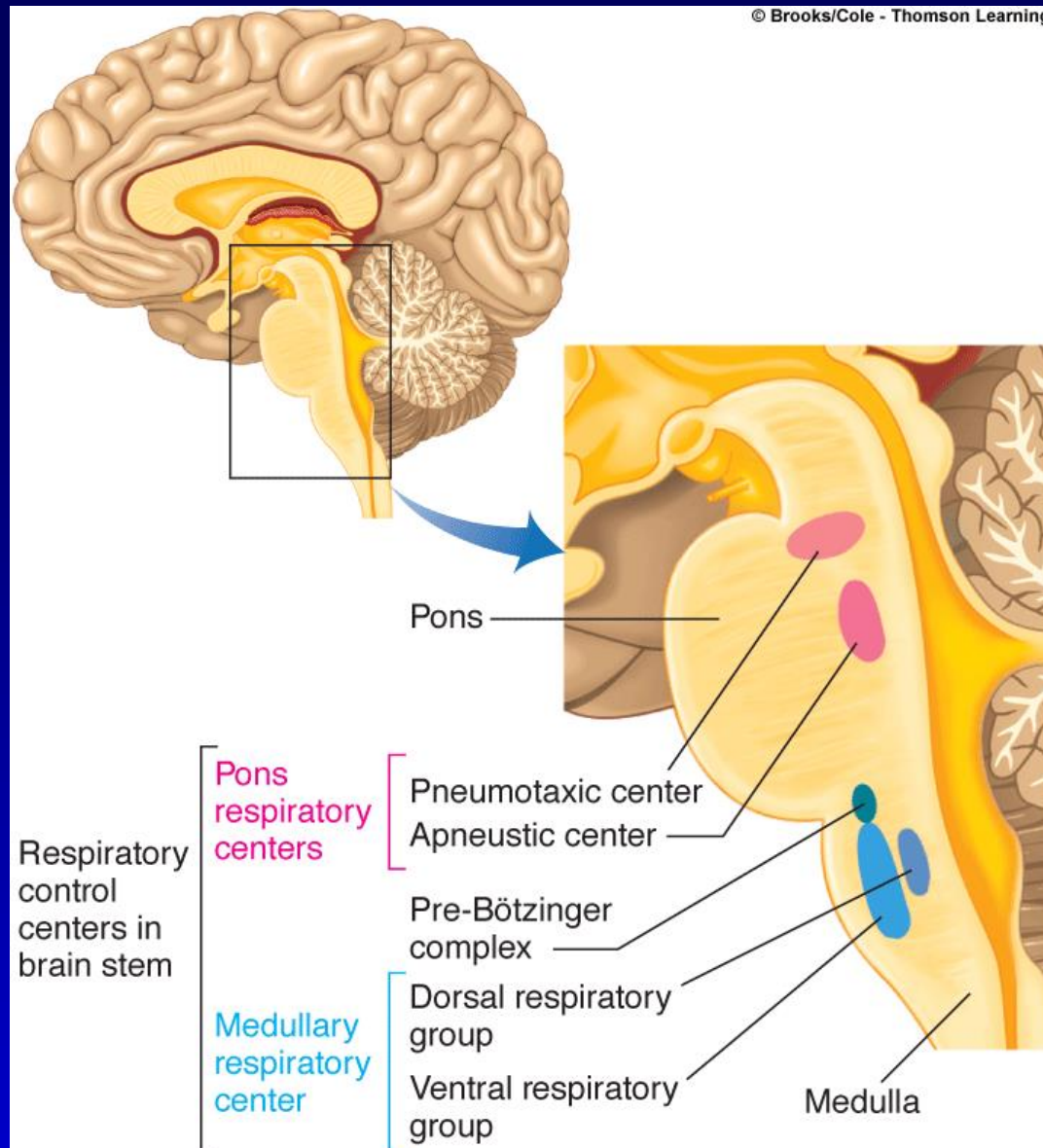
- Lung receptors (continue)
 - ✓ Slowly adapting receptor (pulmonary stretch R.)
 - Located at airway smooth m
 - Stimulated by lung inflation
 - Hering-Breuer inflation Reflex:
 - ↑ lung vol. → ↓ inspiration activity
 - Distention of lung → activate pul. stretch R. → vagus n. → brain → inhibition of insp. activity



Receptors

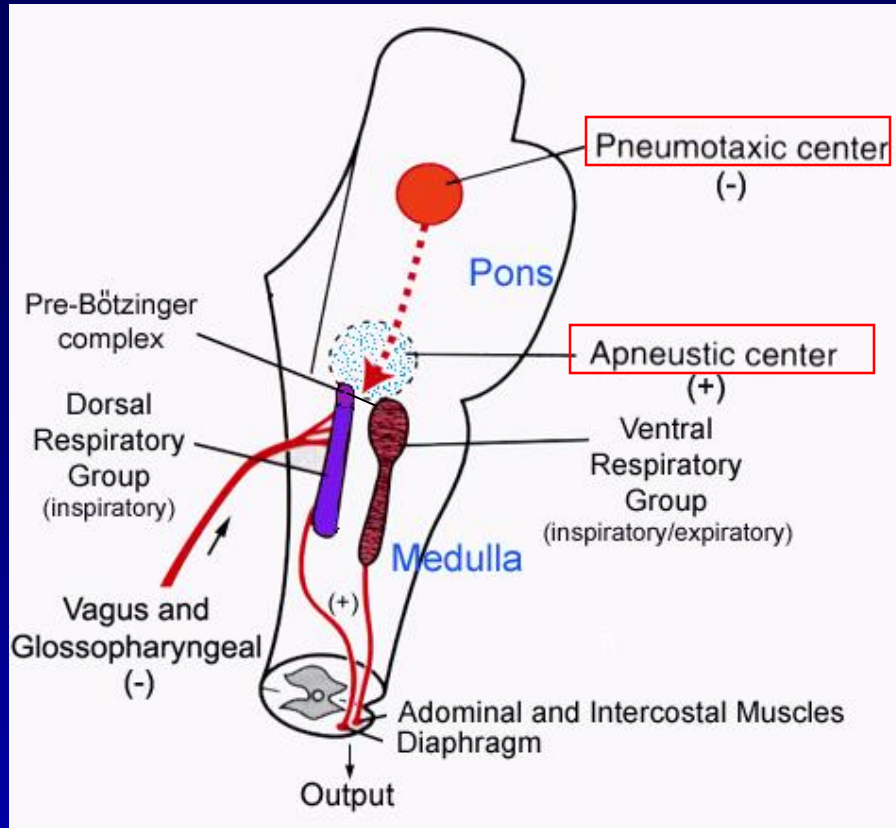
- **Lung receptors** (continue)
 - ✓ **J receptor**
 - Located in the alveolar wall close to capillaries – “juxta-capillary receptor”
 - Innervated by non-myelinated fibers
 - Stimulated by pulmonary edema; congestion
 - Effect: apnea; rapid shallow breathing (tachypnea)
- **Nociceptors (pain)**
 - ✓ Found in every tissue
 - ✓ Effect: ↑ breathing
- **Skeletal m R: thoracic stretch R.**
 - ✓ At intercostals m.
 - ✓ Activated by m. elongation

Central Controller in Brain Stem



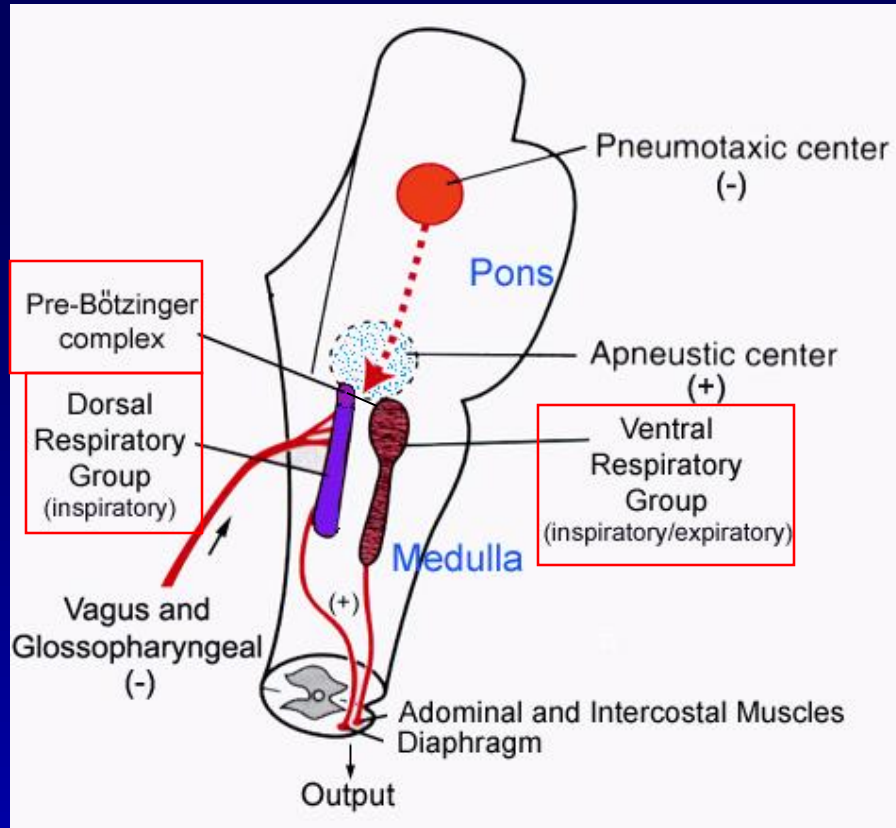
你覺得吸氣比較重要
還是呼氣比較重要？

Central Controller



- **Pons:**
 - ✓ **Pneumotaxic center**
 - fine tune respiratory rate and rhythm
 - switch off of the inspiratory ramp, thus controlling the duration of the filling phase of the lung cycle
 - ✓ **Apneustic center**
 - located in the lower part of the pons
 - stimulates/prolongs inspiration

Central Controller



- **Medulla**

- ✓ **Dorsal medullary resp. group**

- generate basic rhythm of respiration
- causes inspiration

- ✓ **Ventral medullary resp. group**

- cause either expiration or inspiration

- ✓ **Pre-Botzinger complex:**

- ventral side of medulla
- involve in oscillatory (pacemaker) rhythm

正常情況下, 什麼時候呼吸的
型態會改變?

Central Controller

- The resp. sys. is absolutely dependent on an external neural drive
- **Reflex** alters respiratory movements
- For example:
 - **Sneezing**: short inspiration, forced expirations with glottis open
 - **Swallowing**: inhibition of respiration
 - **Coughing**: short inspiration → series of forced expirations with glottis closed (pressure created in airway) → glottis opens suddenly → blast of air carries out irritant material

Central Controller

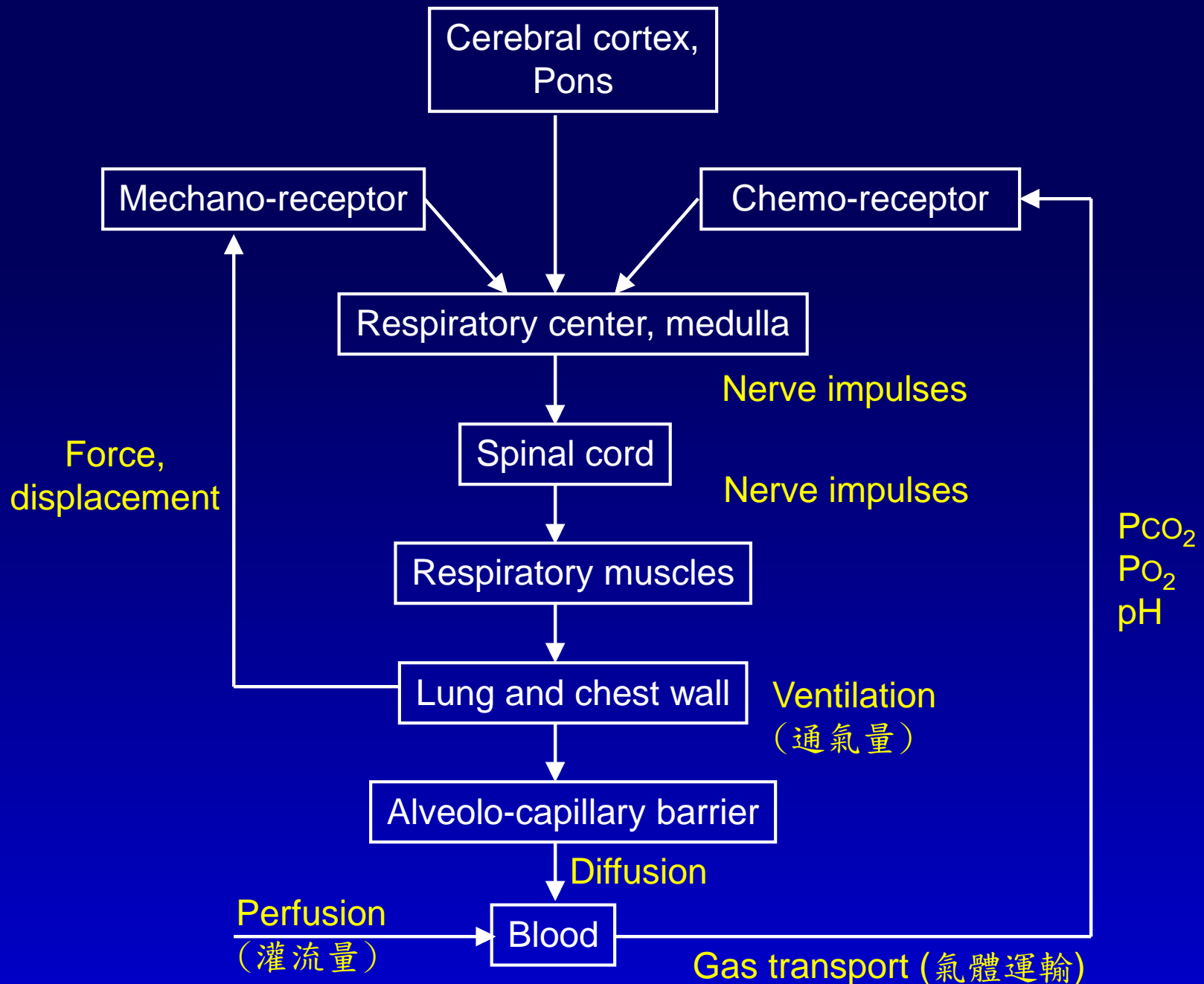
- **Cortical override**: voluntary alterations in breathing on a short term basis
- For example:
 - ✓ **Diving**: hold breath
 - ✓ **Speech & singing**: interruptions of expiration
 - ✓ **Laughter & weeping**: deep inspiration then short spasmodic expiration
 - ✓ **Sighing**: prolong expiration
 - ✓ **Yawning**: deep inspiration with mouth open
 - ✓ **Fear & excitement**: rapid breathing

Effectors

- Dorsal & ventral resp. group **cross the midline** and descend in ventrolateral column of cord
- Inspiratory m: diaphragm, external intercostal m.
- Expiratory m: **passive process**
 - ✓ forced expiration: internal intercostal m., abdominal m.

Outline

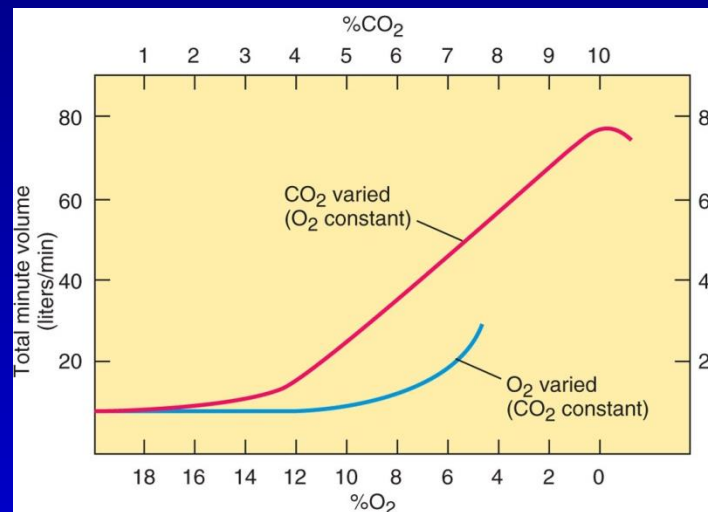
- Background
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- Perfusion and ventilation/perfusion ratio
- Static/Dynamic respiratory mechanics
- Diffusion and gas transport
- Neural control of respiration
- **Chemical control of respiration (呼吸的化學調控)**
- Acid-base balance



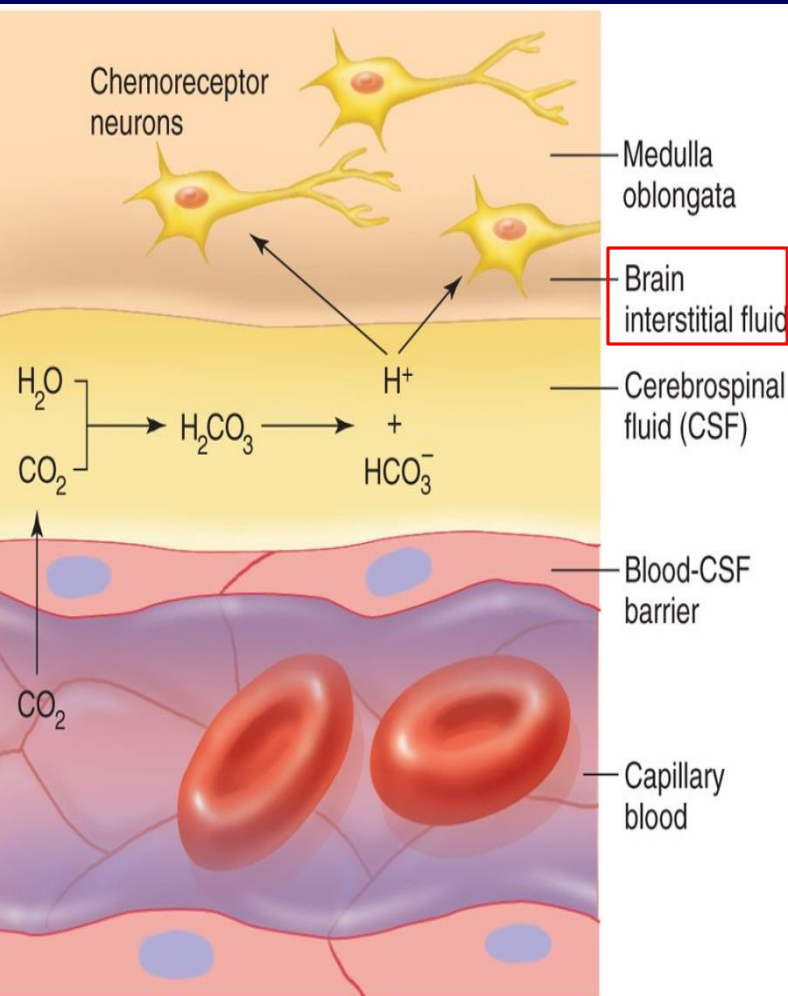
你覺得吸氧氣比較重要
還是排二氧化碳比較重要？

Chemical Control of Resp.

- Two sets of chemoreceptors:
 - ✓ **Central chemoreceptors:** Responsive to arterial P_{CO_2} by way of $[H^+]$ in **extracellular fluid**
 - ✓ **Peripheral chemoreceptors:** Responsive to arterial P_{O_2} , P_{CO_2} , and $[H^+]$
- The most important single driver of ventilation is **P_{CO_2}** acting on the central chemoreceptors by altering **extracellular fluid $[H^+]$**

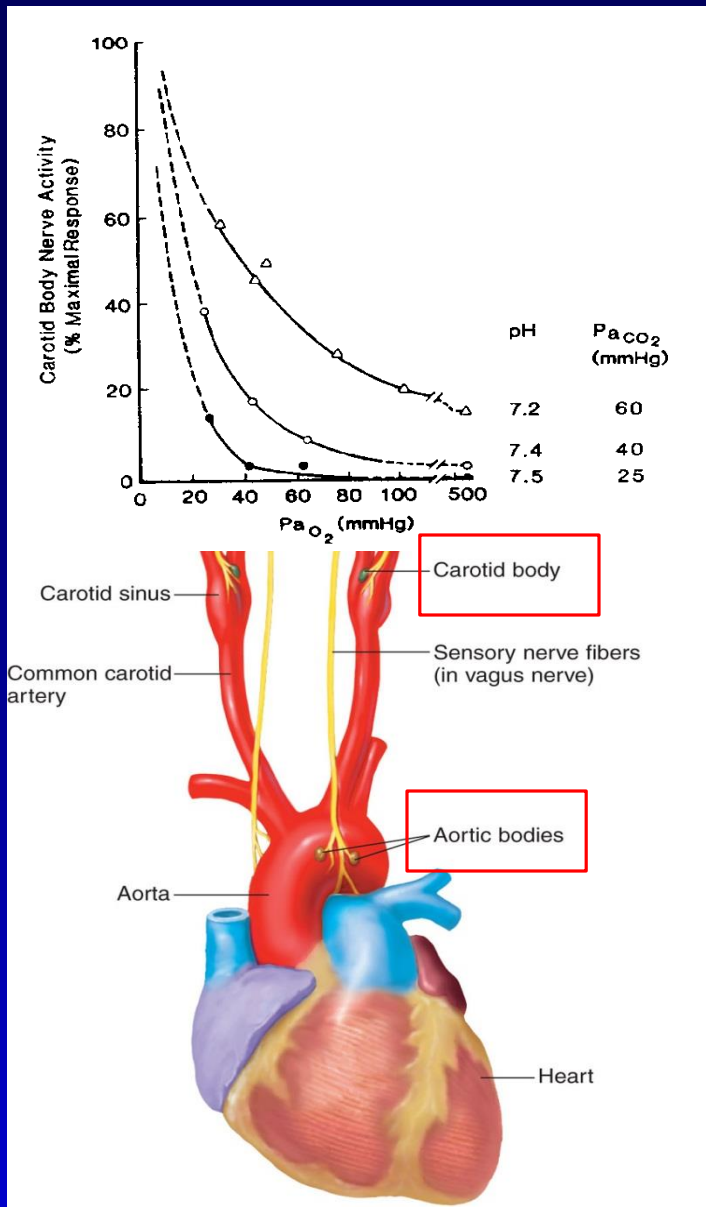


Central Chemoreceptor



- Located in ventrolateral surface of medulla, exposed to **extracellular fluid**
- Respond to $\text{Pa}_{\text{CO}_2} \uparrow$, $\text{pH} \downarrow$ in extracellular fluid (not in blood, due to blood brain barrier) \rightarrow \uparrow ventilation
 - ✓ CO_2 diffuse across BBB easier, H^+ and HCO_3^- do not
- $\text{Pa}_{\text{O}_2} \downarrow \rightarrow$ depresses ventilation by depressing oxidative metabolism in neural tissue
- Insensitive to changes in P_{aO_2} above about 50–60 mm Hg

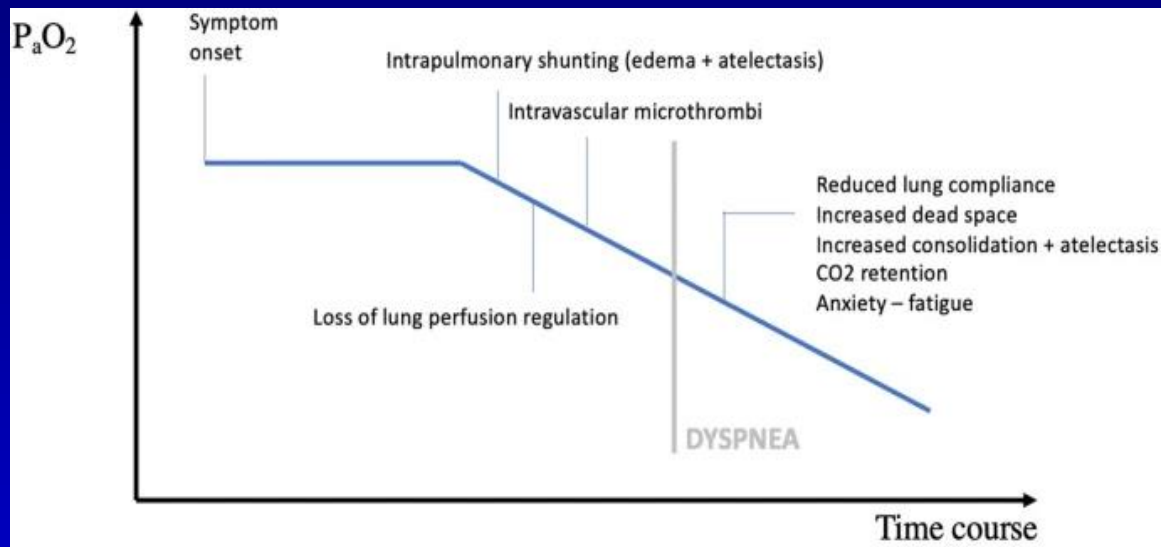
Peripheral Chemoreceptor



- Glomus cells in carotid body & aortic body (Respond to $P_{aO_2} \downarrow$, $P_{aCO_2} \uparrow$, $pH \downarrow$)
 $\rightarrow \uparrow V_T$ & \uparrow freq.
- Neural impulses from the carotid body increase as $P_{aO_2} \downarrow$
 - ✓ potentiated by acidosis and hypercapnia
- Peripheral chemoreceptor is the **ONLY** way to sense low P_{O_2} with stimulatory response

Silent Hypoxia (Happy Hypoxia)

- Hypoxia that does not coincide with shortness of breath
- Causes of hypoxemia in COVID-19
 - ✓ Intrapulmonary shunting
 - ✓ Loss of lung perfusion regulation
 - ✓ Intravascular microthrombi
 - ✓ Impaired diffusion capacity



Outline

- Background
- Structure and function
- Ventilation
- Perfusion and ventilation/perfusion ratio
- Static/Dynamic respiratory mechanics
- Diffusion and gas transport
- Neural control of respiration
- Chemical control of respiration
- **Acid-base balance (酸鹼平衡)**

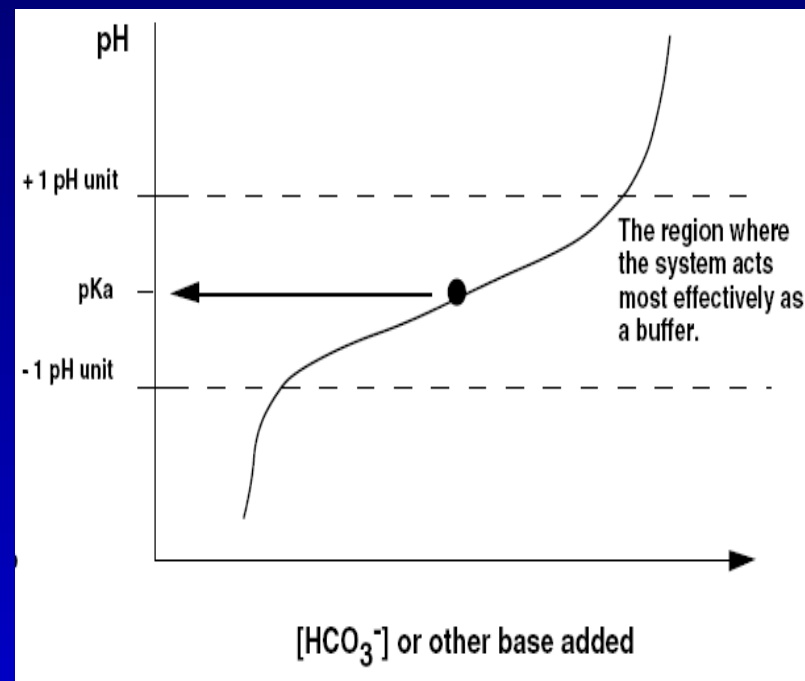
Acid-base Balance

- Blood pH ~ 7.4 +/- 0.05
 - ✓ Acidosis (酸中毒): too much acid in blood, pH ↓
 - ✓ Alkalosis (鹼中毒): too much base in blood, pH ↑
- Categorized by primary cause:
 - ✓ Respiratory: lung; Pco₂ changes
 - ✓ Metabolic: kidney, liver; [HCO₃⁻] changes
- Three ways of controlling blood pH:
 - ✓ Buffer systems: bicarbonate, phosphate and Hb
 - ✓ Release of CO₂ from the lung (fast)
 - ✓ Excretion of acids or bases from the kidney (slow)

Effectiveness of a Buffer System

- pKa
 - ✓ Gives the pH where a buffer is most effective
 - ✓ Phosphate (pKa = 7.2), Hb (imidazole group of histidine, pKa=6.8), bicarbonate (pKa = 6.1)
- Amount (concentration) of the buffer
 - ✓ Bicarbonate & Hb

* **Bicarbonate** is the most important buffer in the body



Bicarbonate



$$\text{pH} = \text{pKa} + \log \frac{[\text{conjugate base}]}{[\text{acid}]}$$

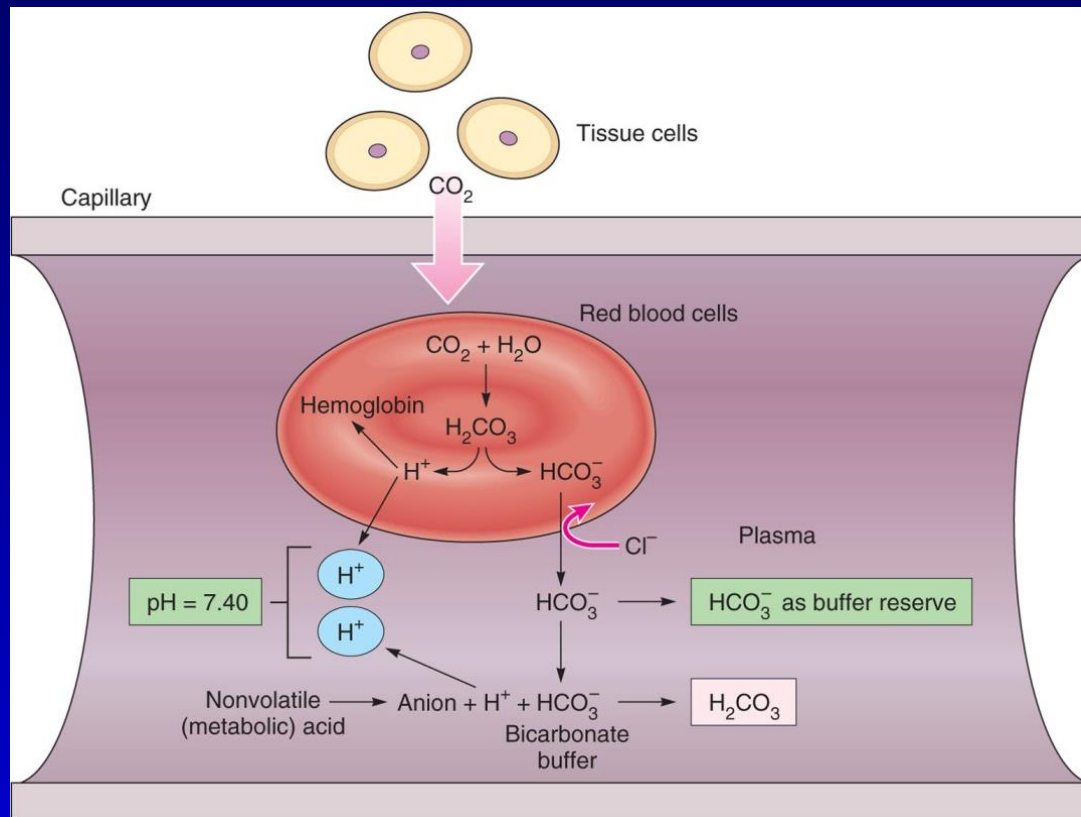
$$\text{pH} = \text{pKa} + \log \frac{[\text{bicarbonate}]}{[\text{acid}]}$$

$$\text{pH} = 6.1 + \log \frac{[\text{HCO}_3^-]}{\alpha_{\text{CO}_2} \times P_{\text{CO}_2}}$$

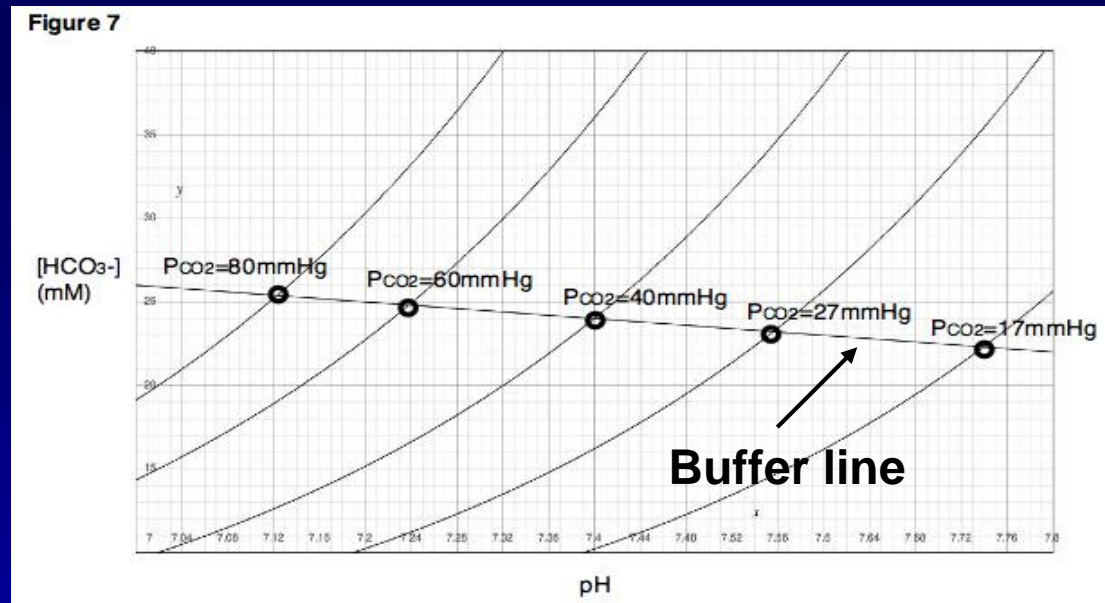
$$[\text{H}_2\text{CO}_3] = 0.03 \times P_{\text{CO}_2} \text{ (Henry's law)}$$

The Effect of Bicarbonate on Blood pH

- Released into the plasma from RBC buffers the H^+ produced by the ionization of metabolic acids (lactic acid, fatty acids, ketone bodies, etc.)



Davenport Diagram



$$pH = 6.1 + \log \frac{[HCO_3^-]}{\alpha_{CO_2} \times P_{CO_2}}$$

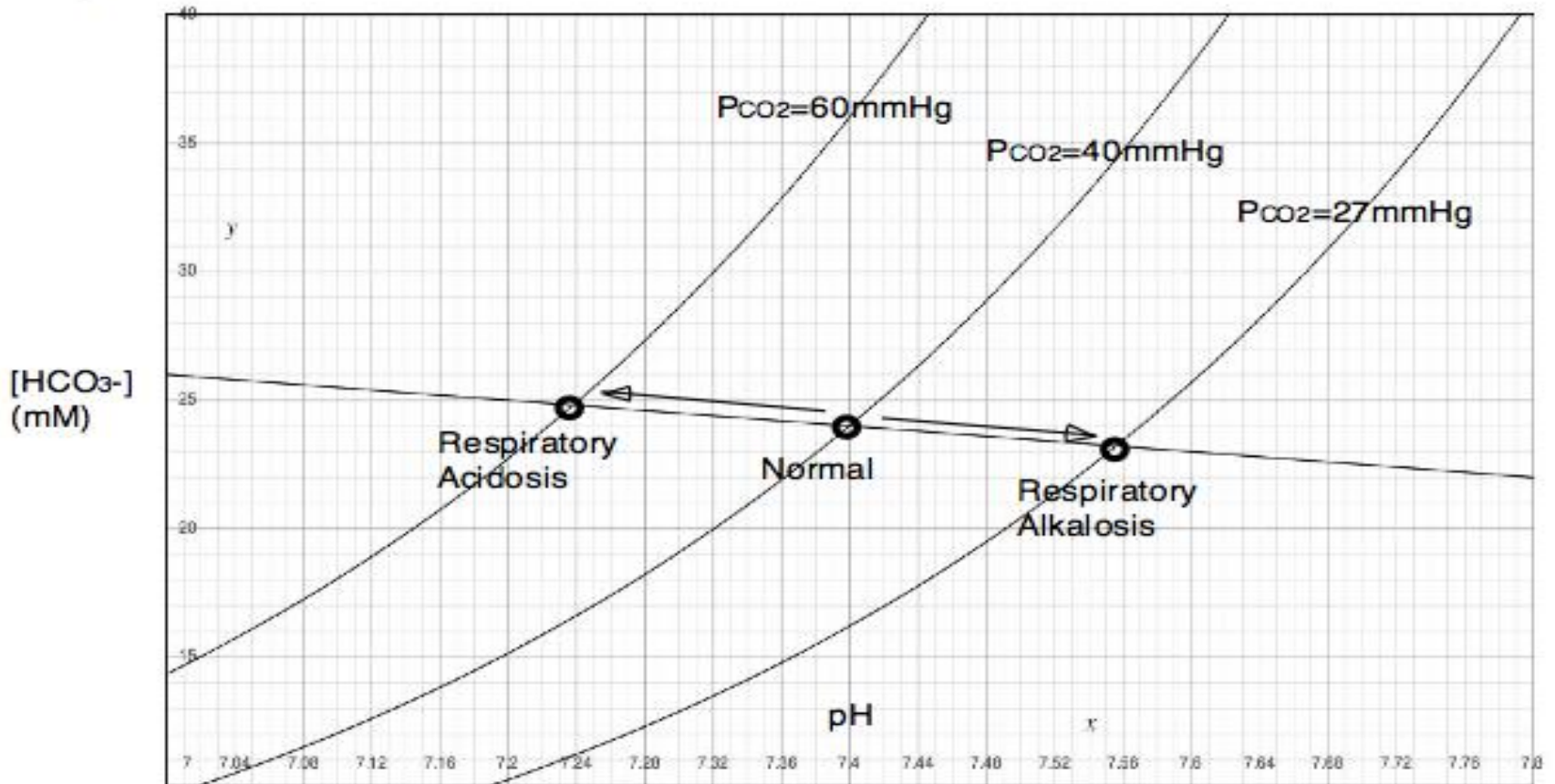
arterial blood: $P_{CO_2} = 40$ mmHg

- pH 7.4, $\alpha_{CO_2} = 0.03$

- $[HCO_3^-] = 24$ mM

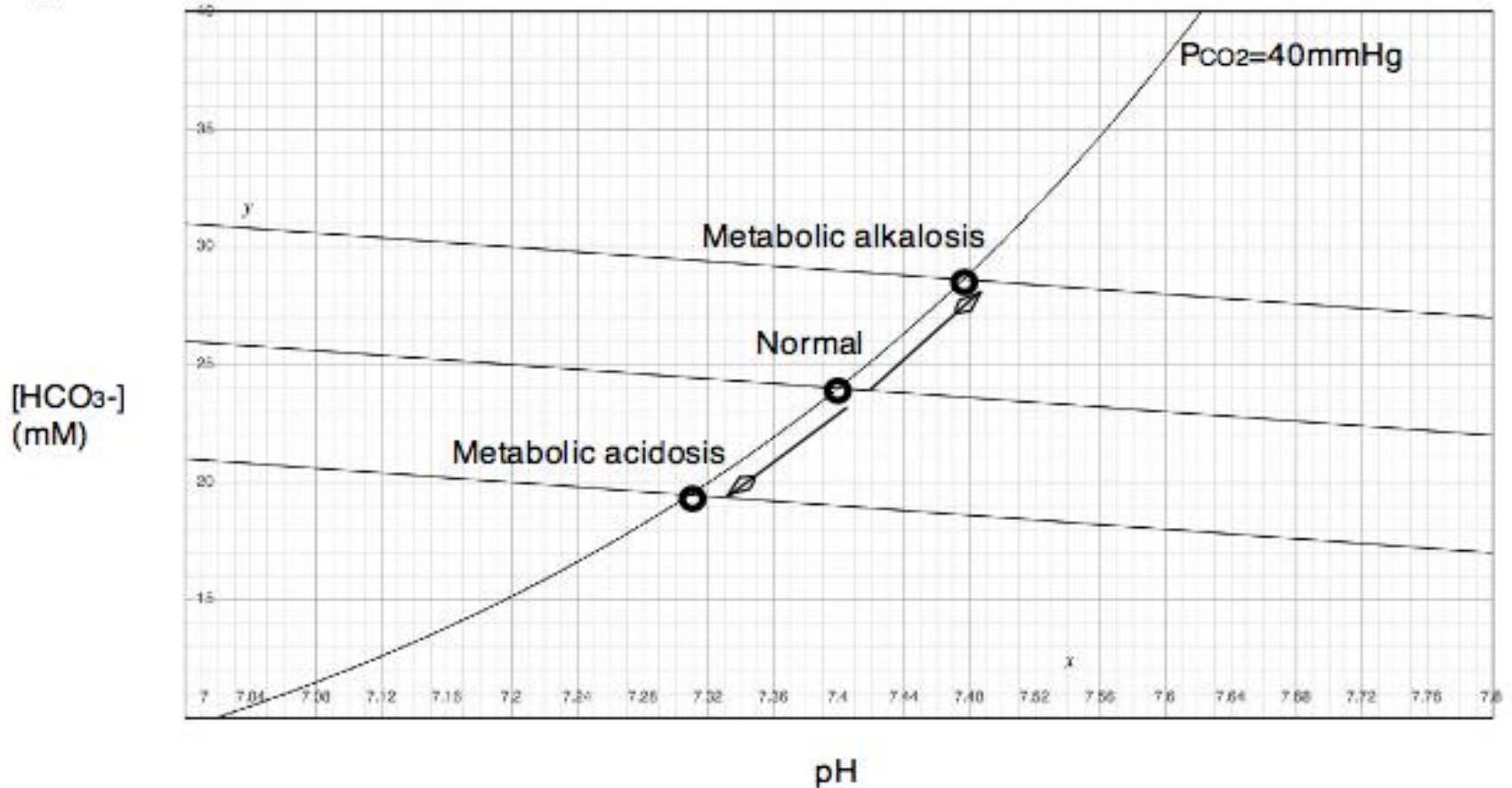
Respiratory Disturbances

Figure 11

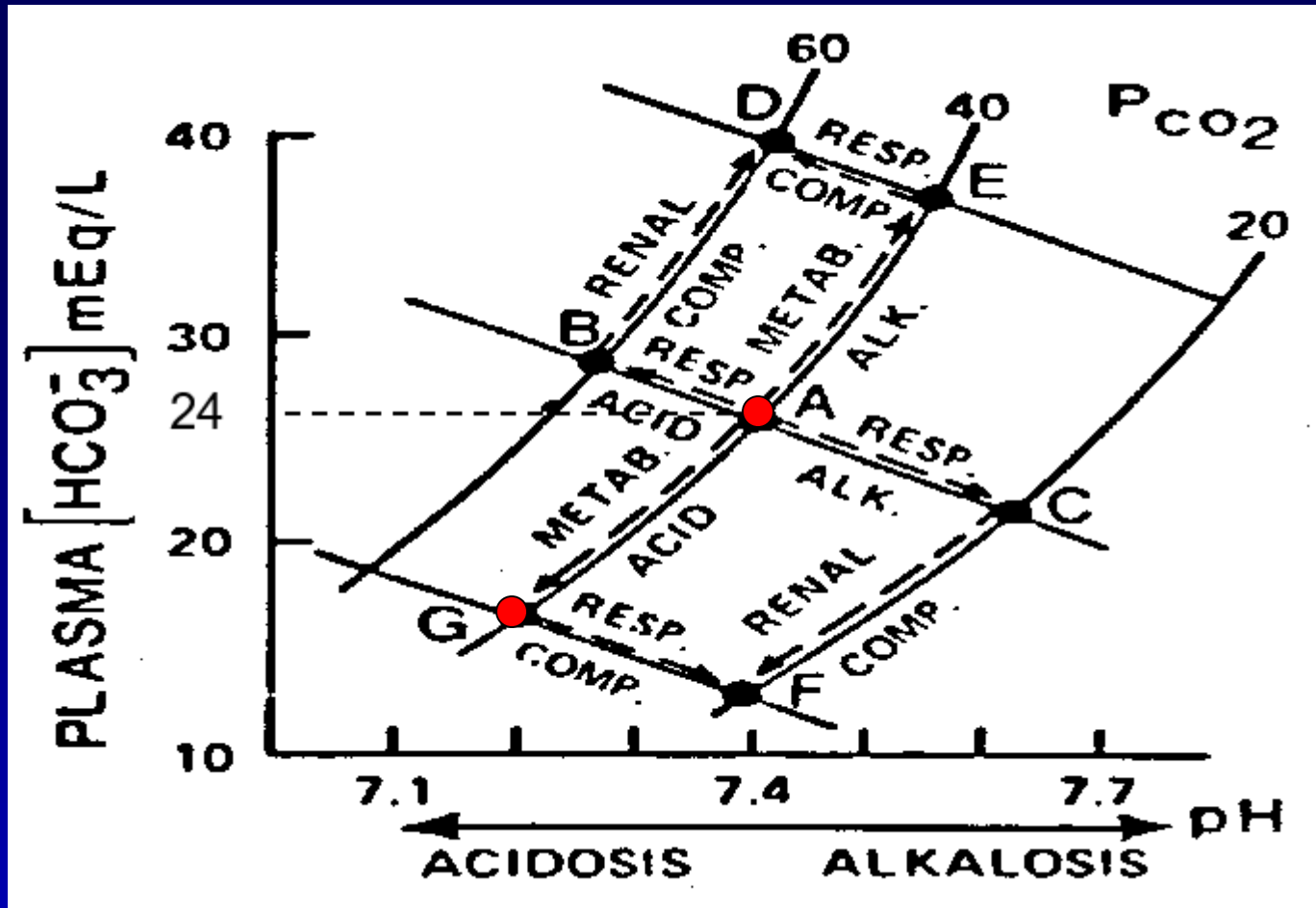


Metabolic Disturbances

Figure 12



Compensatory Responses



E.g., diabetic patient: ketoacidosis, hyperventilation, pH=7.4

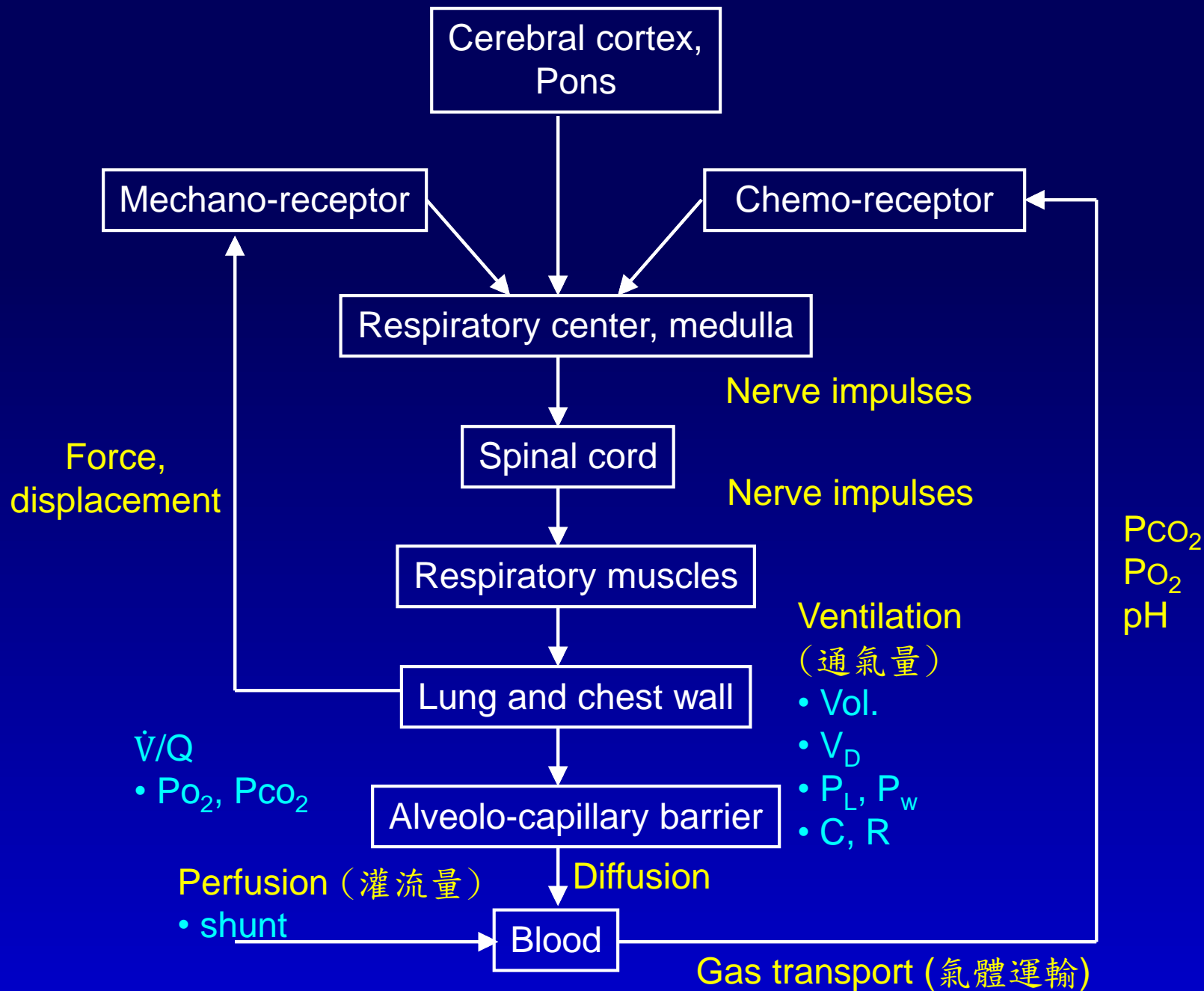
Metabolic acidosis with respiratory alkalosis

How to Increase Resp. Function During Exercise?

- Deeper, faster breathing to match oxygen utilization and CO₂ production → hyperpnea (ventilation ↑; Pco₂ ↔)
 - Expiration muscle contraction
 - Pursed lip breathing
 - Body heat is expelled during exhalation
- Excitation of sympathetic nerve → bronchodilation (resistance ↓)
- Increase blood flow → O₂ diffusion ↑ (perfusion-limited gas), perfusion ↑, transport time ↓
- Decrease O₂ affinity of hemoglobin → unload O₂ to tissue

How to Increase Resp. Function During Exercise?

- **Neurogenic** and **humoral** mechanisms control this
 - **Neurogenic** mechanisms
 - **Cerebral cortex** stimulates respiration via respiratory centers
 - **Sensory n. activity** from exercising m. stimulates respiration via **spinal reflexes** or **brain stem resp. centers**
 - **Humoral** mechanisms (oxygen debt)
 - Rapid and deep breathing continues **after exercise** due to **humoral factors**
 - P_{CO_2} and pH differences at sensors



The text "The End!" is rendered in a large, colorful, 3D-style font. Each letter is filled with a different color: 'T' is pink, 'h' is orange, 'e' is yellow, 'E' is green, 'n' is blue, and 'd!' is purple. The letters have a white outline and a grey shadow underneath, giving them a three-dimensional appearance. The background is a solid dark blue.

References:

- 賴義隆等，呼吸生理學，金名圖書
- KE Barrett et. al., Ganong's Review of Medical Physiology
- SI Fox, Human Physiology
- JB West, Respiratory Physiology: the essentials