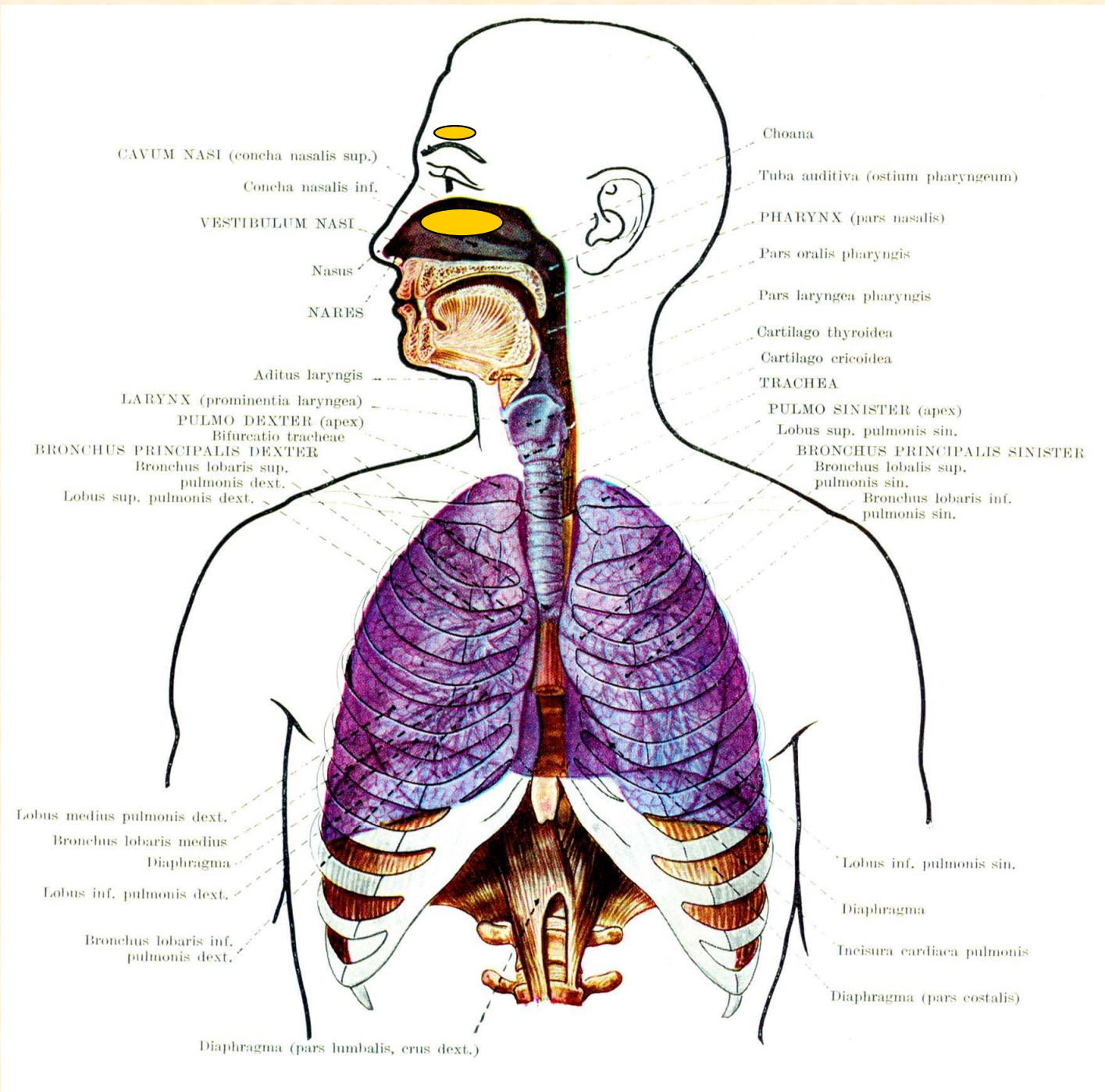
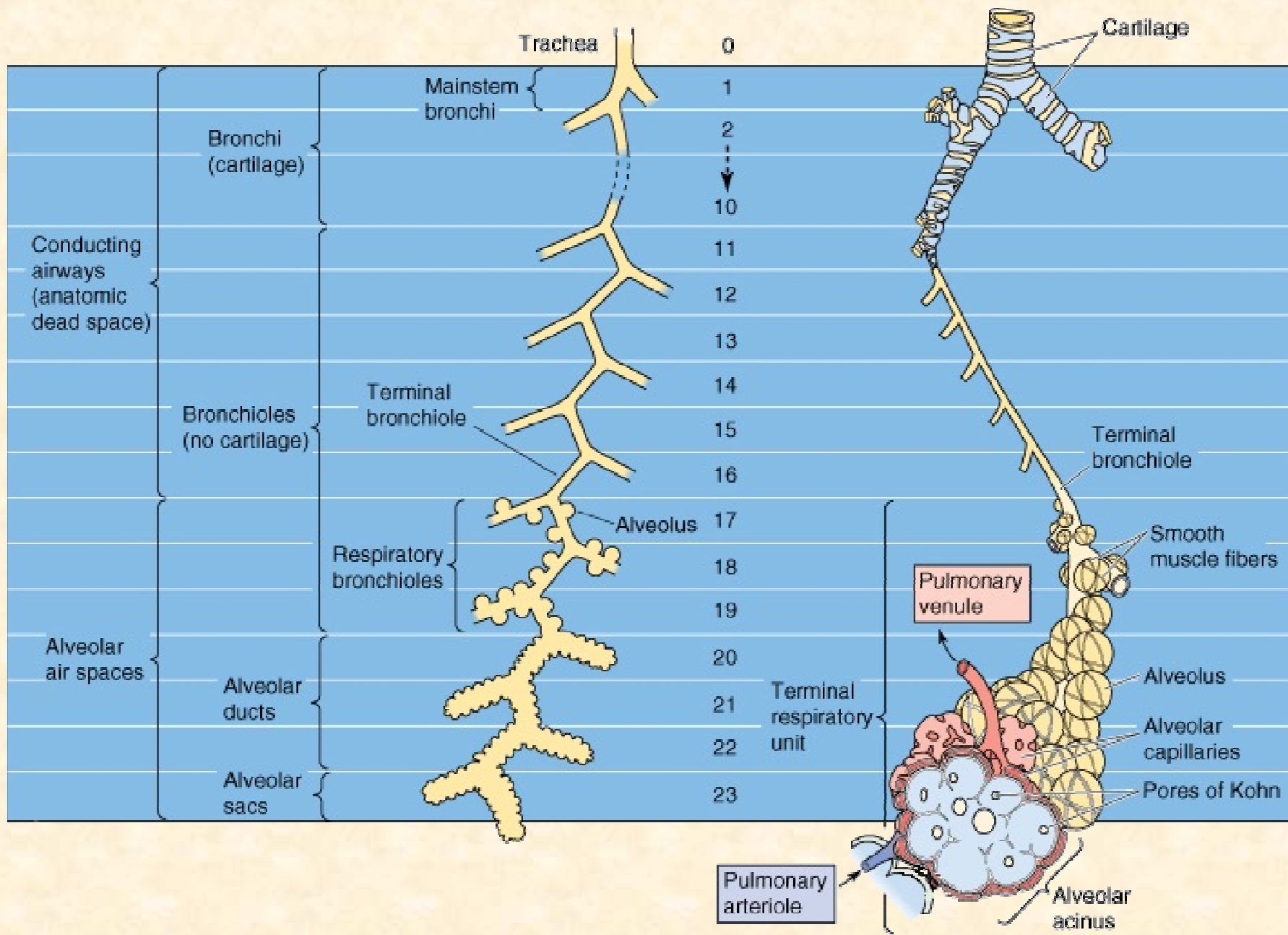
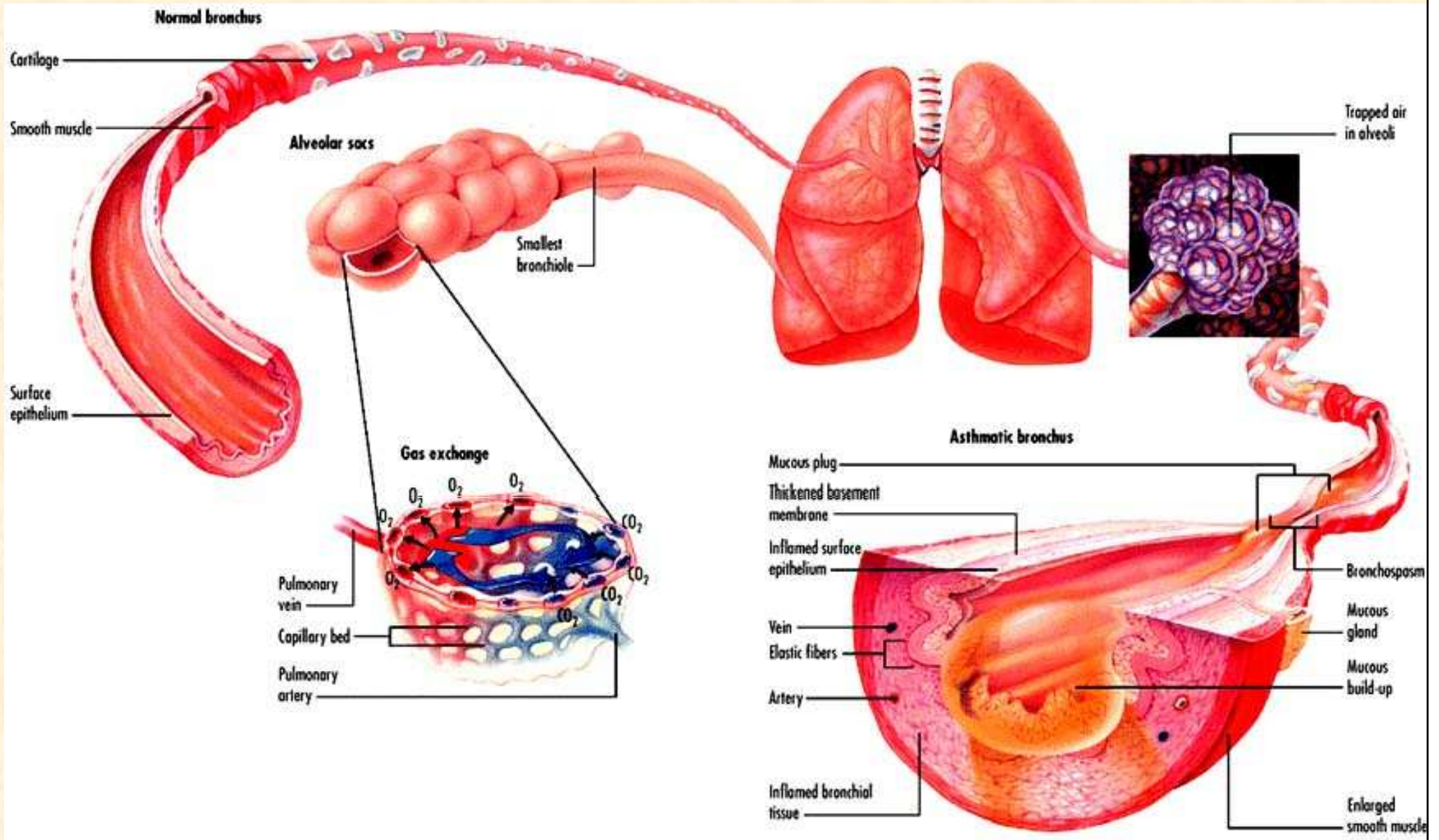


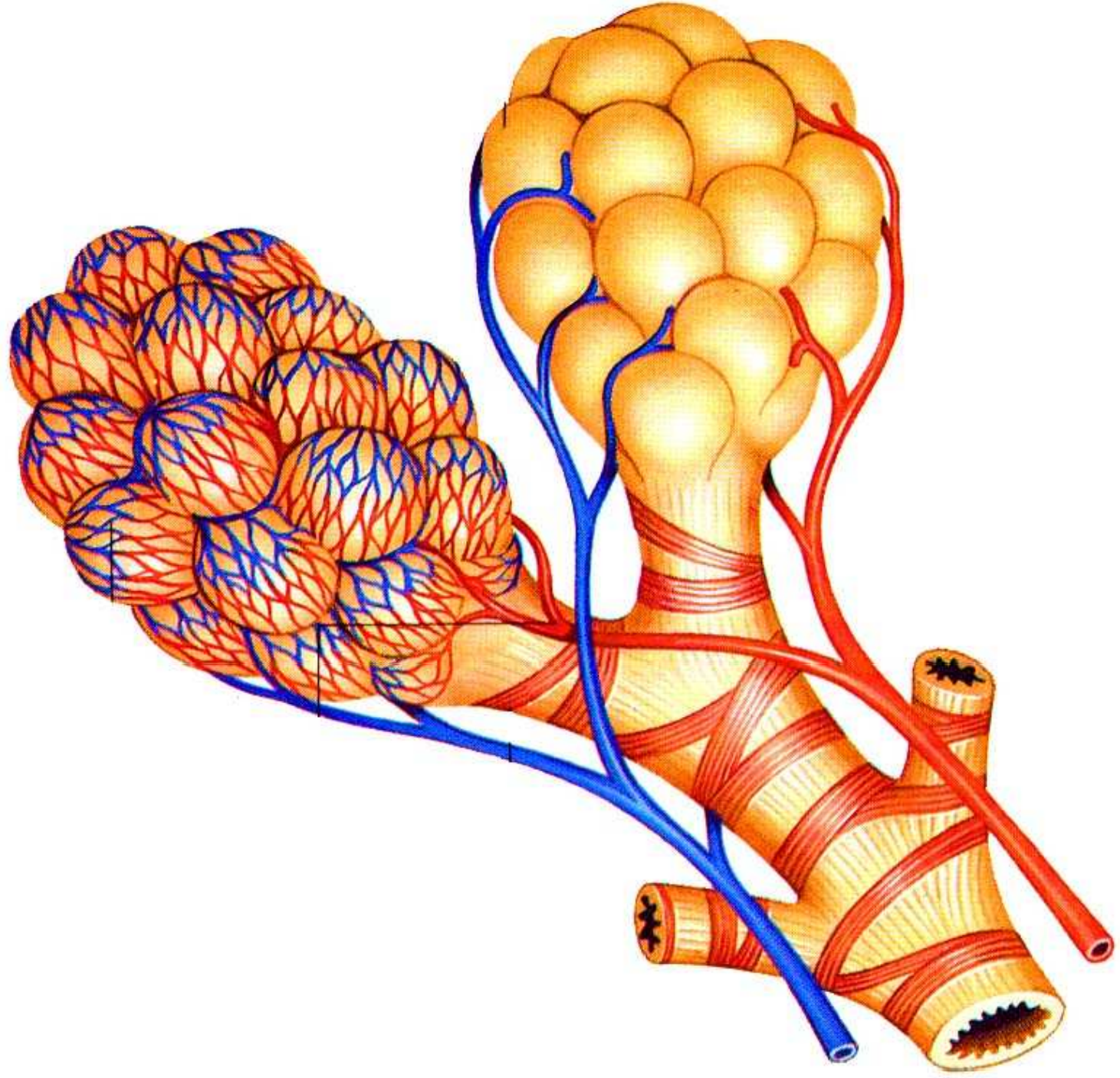
RESPIRATORY SYSTEM

**RESPIRATORY FUNCTIONS
MECHANICS OF RESPIRATORY SYSTEM
GAS TRANSPORT**









STEPS IN THE DELIVERY OF O_2 TO THE CELLS

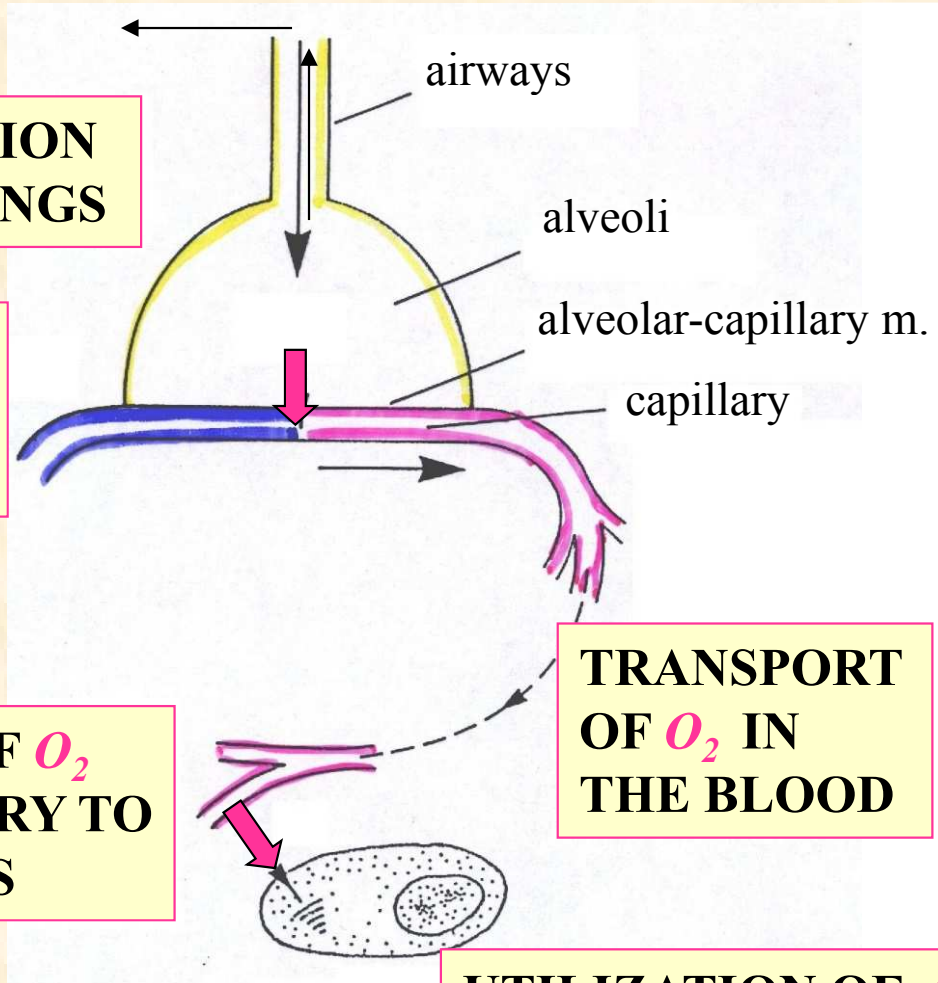
**VENTILATION
OF THE LUNGS**

**DIFFUSION OF O_2 ACROSS
ALVEOLAR-CAPILLARY
MEMBRANE**

**DIFFUSION OF O_2
FROM CAPILLARY TO
THE CELLS**

**TRANSPORT
OF O_2 IN
THE BLOOD**

**UTILIZATION OF O_2
BY MITOCHONDRIA**



AT REST

O_2 UPTAKE ~300 ml / min

CO_2 OUTPUT ~250 ml / min

INTERNAL RESPIRATION

AIR PASSAGES

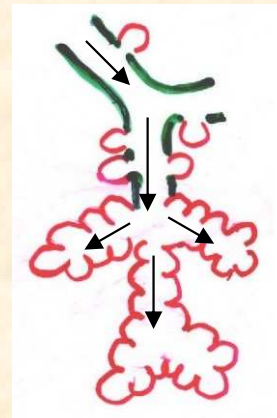
ANATOMICAL DEAD SPACE – **CONDUCTING ZONE**



- **NASAL PASSAGES**
- **PHARYNX**
- **LARYNX**
- **TRACHEA**
- **BRONCHI**
- **BRONCHIOLES**
- **TERMINAL BRONCHIOLES**

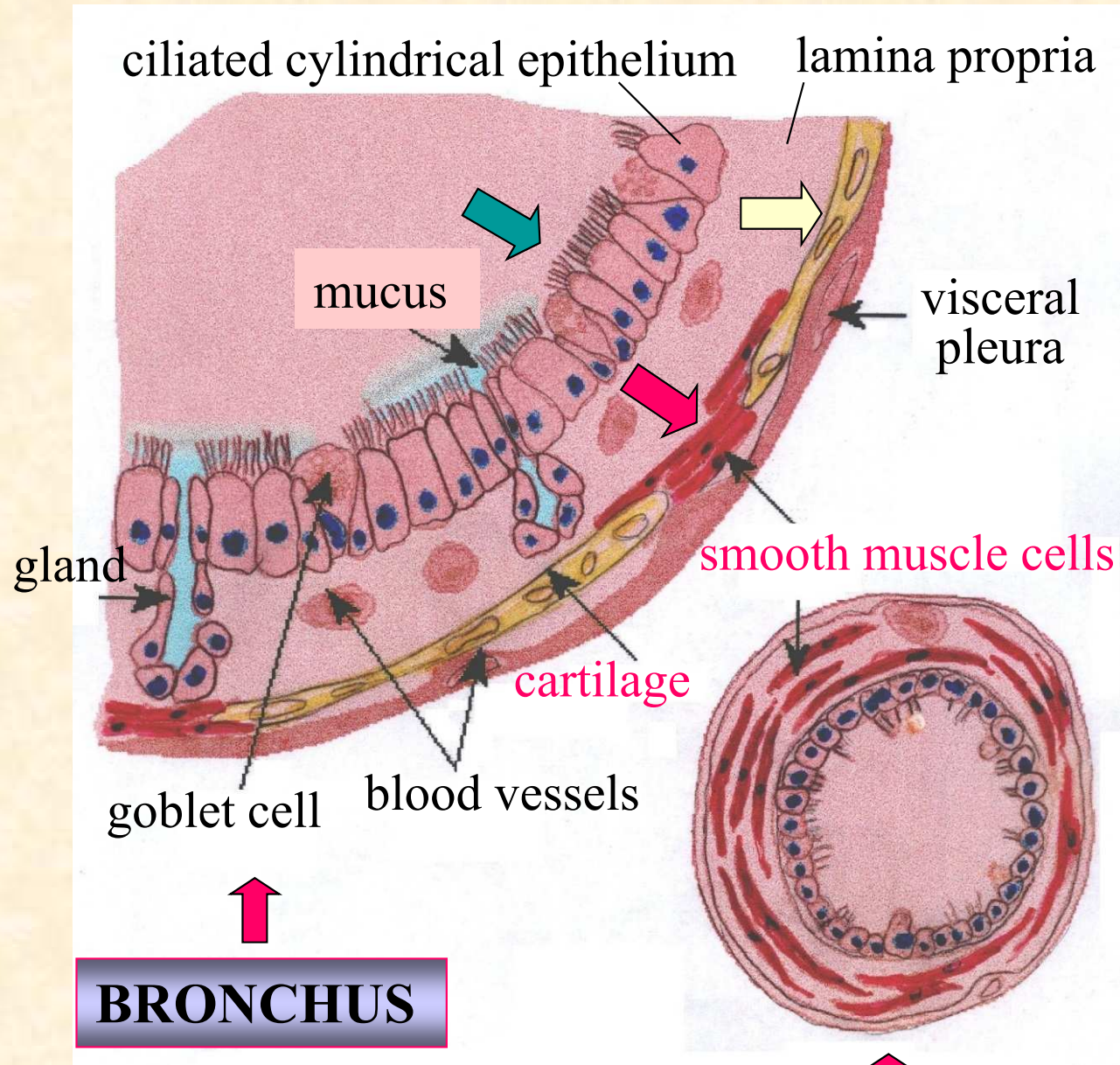
Other physiological functions:

- air is warmed, cleaned and takes up water vapour
- respiratory reflex responses to the irritants
- speech and singing (function of larynx, vocal cords)



RESPIRATORY ZONE (GAS EXCHANGE)

Total alveolar area $\sim 100 \text{ m}^2$



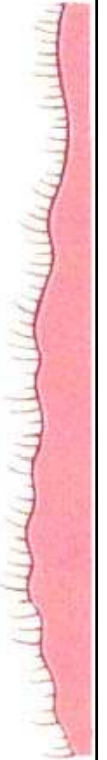
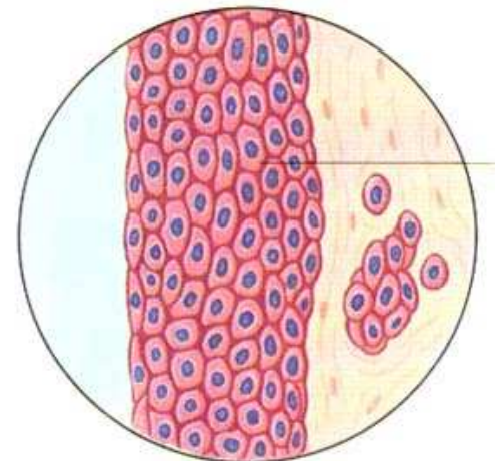
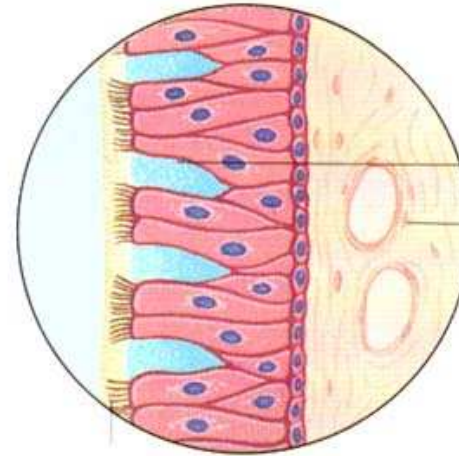
AUTONOMIC INNERVATION of smooth muscle cells

Stimulation via parasympathetic NS - n.vagus due to **Muscarinic** receptors: Acetylcholine activates bronchoconstriction

Stimulation via to sympathetic NS – due to catecholamins in circulation
 β_2 -adrenergic receptors:
Noradrenaline activates bronchodilatation

TERMINAL BRONCHIOLE

$\varnothing < 1 \text{ mm}$



Cylindrical epithelium with cilia

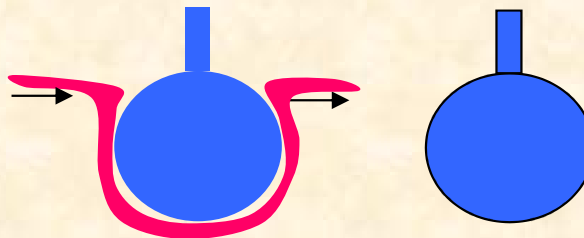
Picture: the idea of a seabed with sea anemones

DEAD SPACE

**TOTAL GAS VOLUME NOT EQUILIBRATED WITH BLOOD
(without exchange of gasses)**

- **ANATOMICAL** dead space - volume of air passages
- **FUNCTIONAL (total)** dead space

ANATOMICAL dead space + total **VOLUME** of **ALVEOLI** without functional capillary bed



IN HEALTHY INDIVIDUALS
both spaces are practically identical

V_T tidal volume ~ 500 ml

$$V_T = V_A + V_D$$

V_A part of tidal volume entering alveoli ~ 350 ml

V_D part of tidal volume remaining in the dead space ~ 150 ml

$$f = 12/\text{min}$$

$$\dot{V} = V_T \times f$$

**PULMONARY
MINUTE
VENTILATION**

6 l/min

$$\dot{V}_A = V_A \times f$$

ALVEOLAR VENTILATION

4.2 l/min

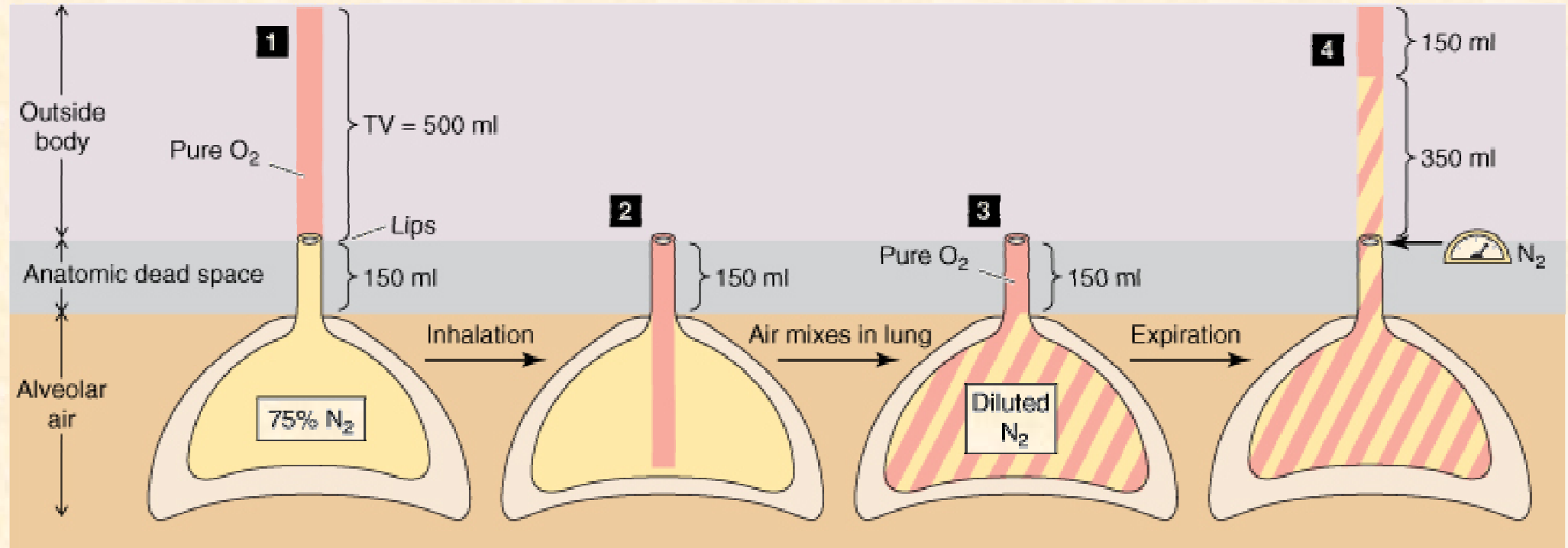
$$\dot{V}_D = V_D \times f$$

DEAD SPACE VENTILATION

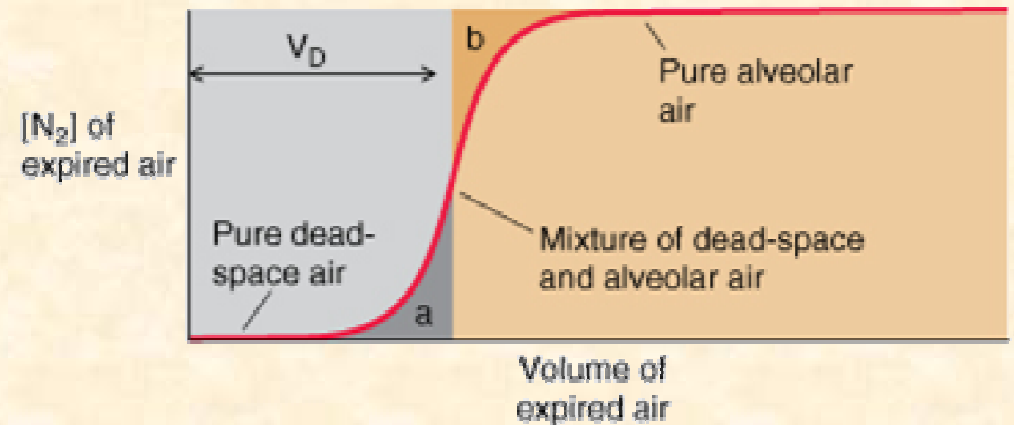
1.8 l/min

DEAD SPACE – nitrogen test (force inspiration of pure O₂, follow slowly expiration with monitoring of concentration of nitrogen)

A DILUTION OF INSPIRED 100% O₂

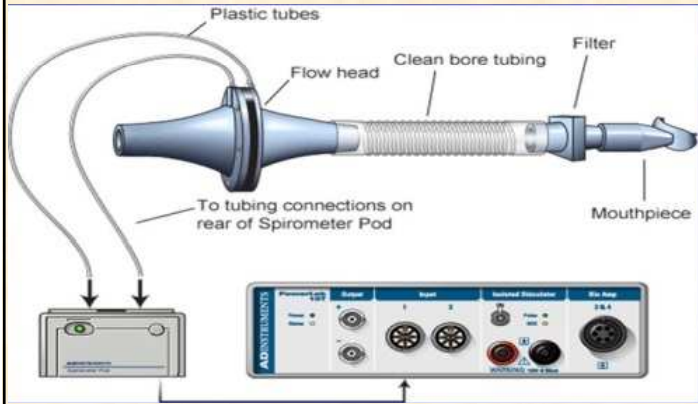


C MEASURED [N₂] PROFILE

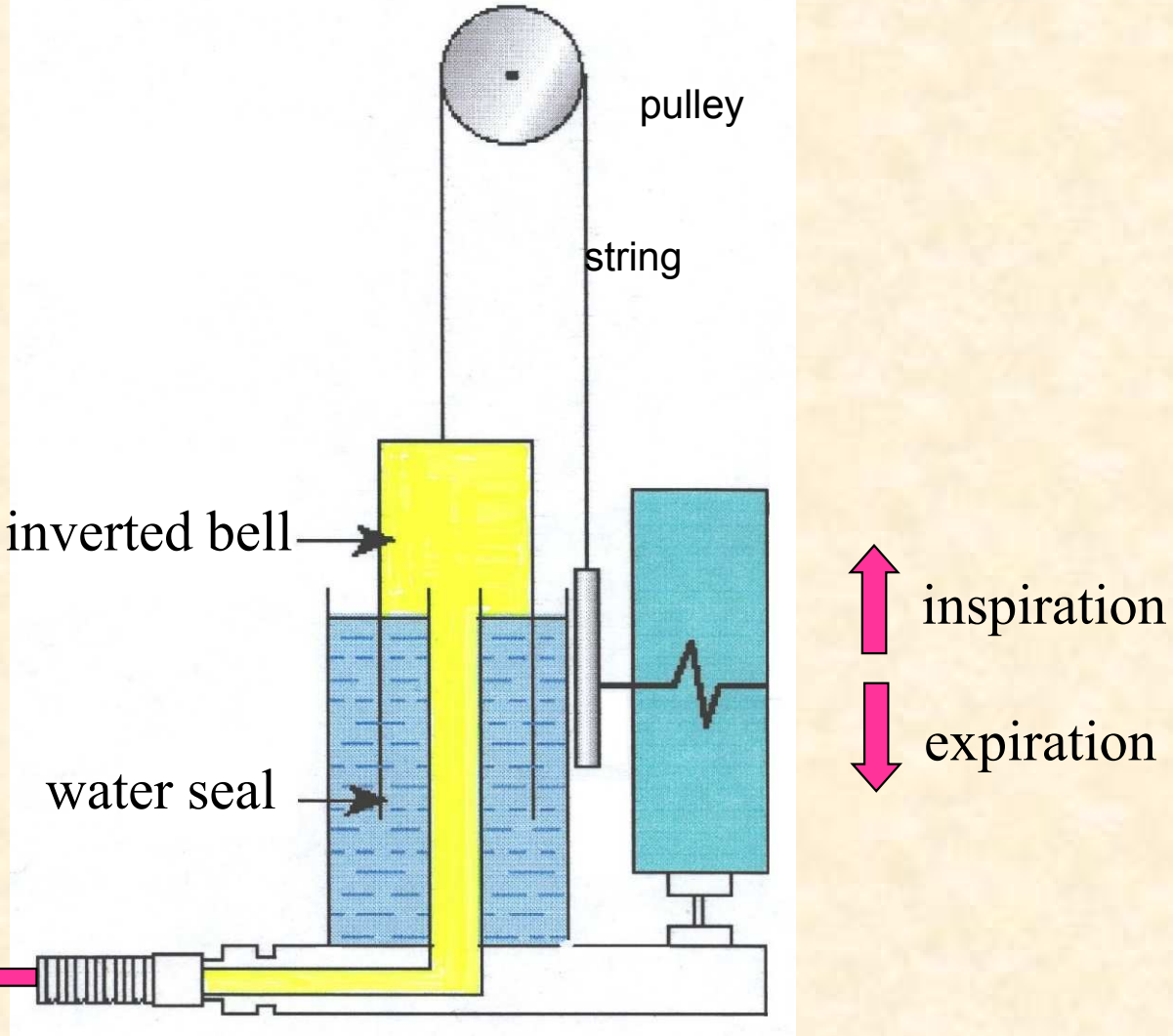


SPIROMETRY

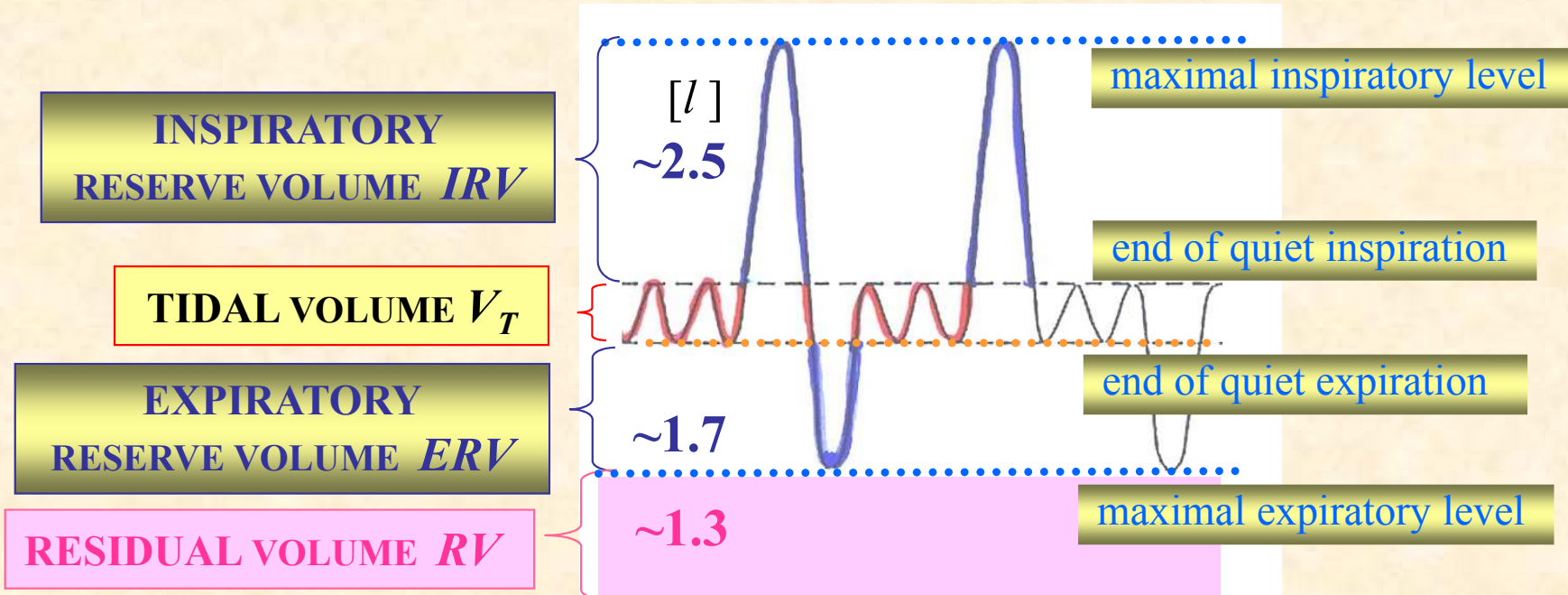
(measurements of lung volumes, capacities, functional investigations, ...)



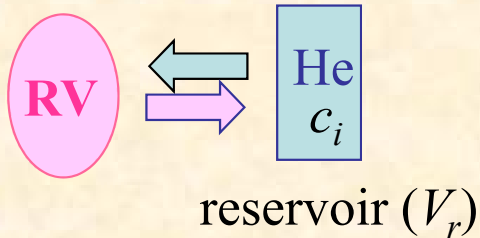
subject



LUNG VOLUMES



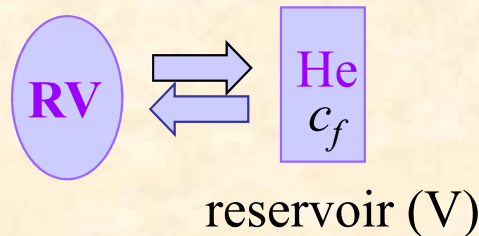
DILUTION METHOD He



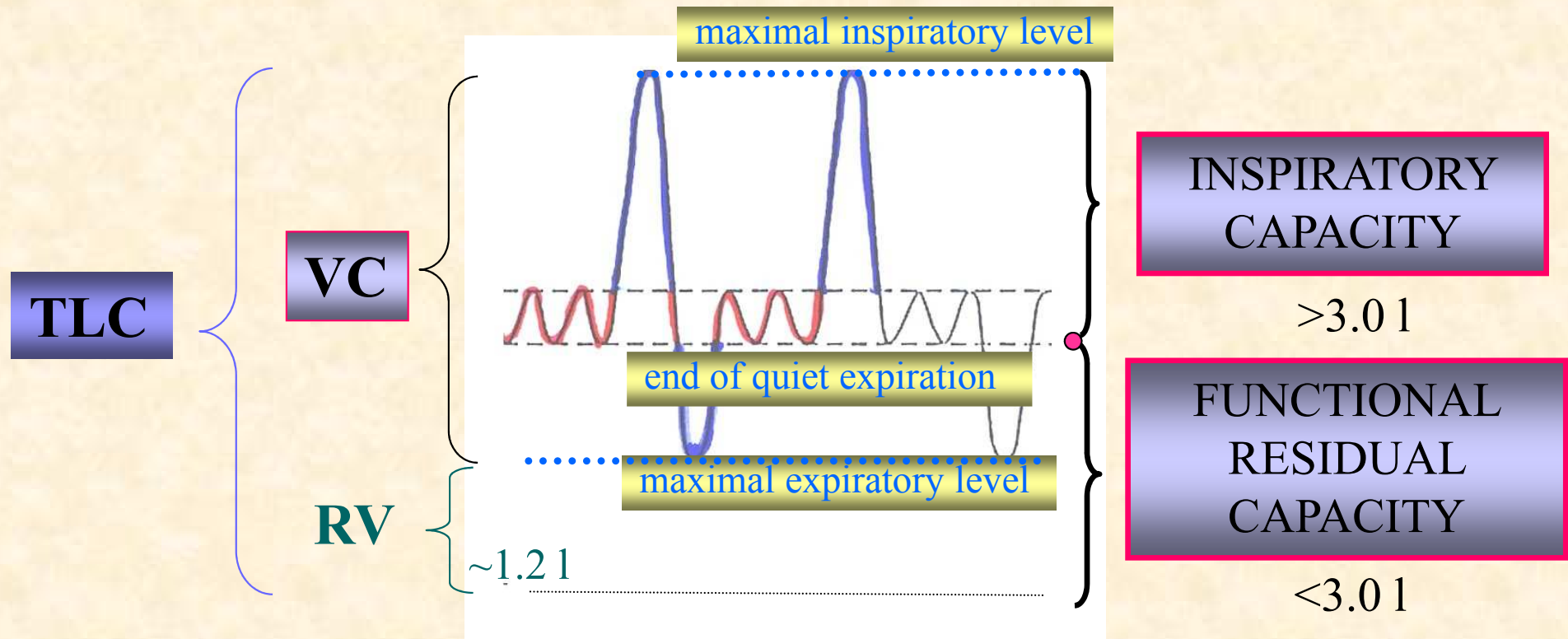
Principle of method: **1** Maximal expiration, **2** Repeated inspiration from and expiration into a reservoir (known volume V_r) with inert gas He (known concentration c_i)

\Rightarrow Equilibration of the air in the residual volume and reservoir

3 Calculation of **residual volume** RV from the initial and final He concentrations in reservoir (c_i, c_f).



$$RV = V_r \frac{c_{iHe} - c_{fHe}}{c_{fHe}}$$



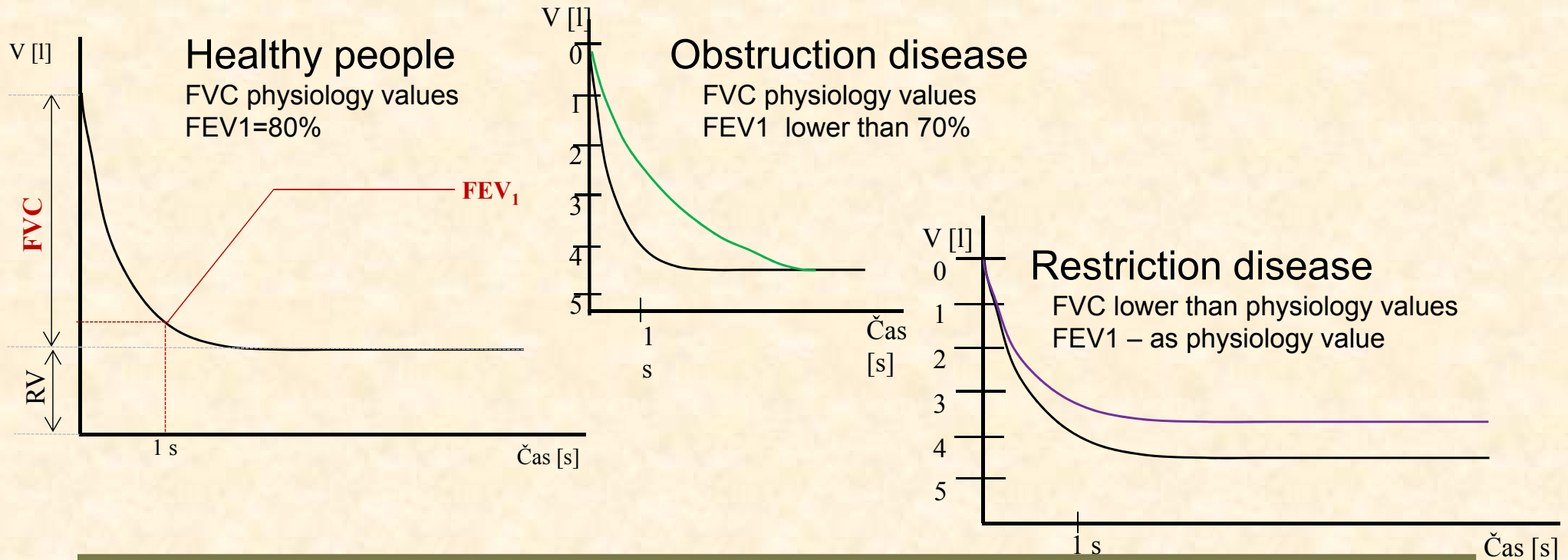
VC **VITAL CAPACITY** = $V_T + IRV + ERV$ ~ 4.7 l

VC - the largest amount of air that can be expired after maximal inspiration

TLC **TOTAL LUNG CAPACITY** = **VC** + **RV** ~ 6.0 l

FUNCTIONAL INVESTIGATION OF THE LUNGS

- **TIMED VITAL CAPACITY (FEV_1 - forced expiratory volume per 1 s)**

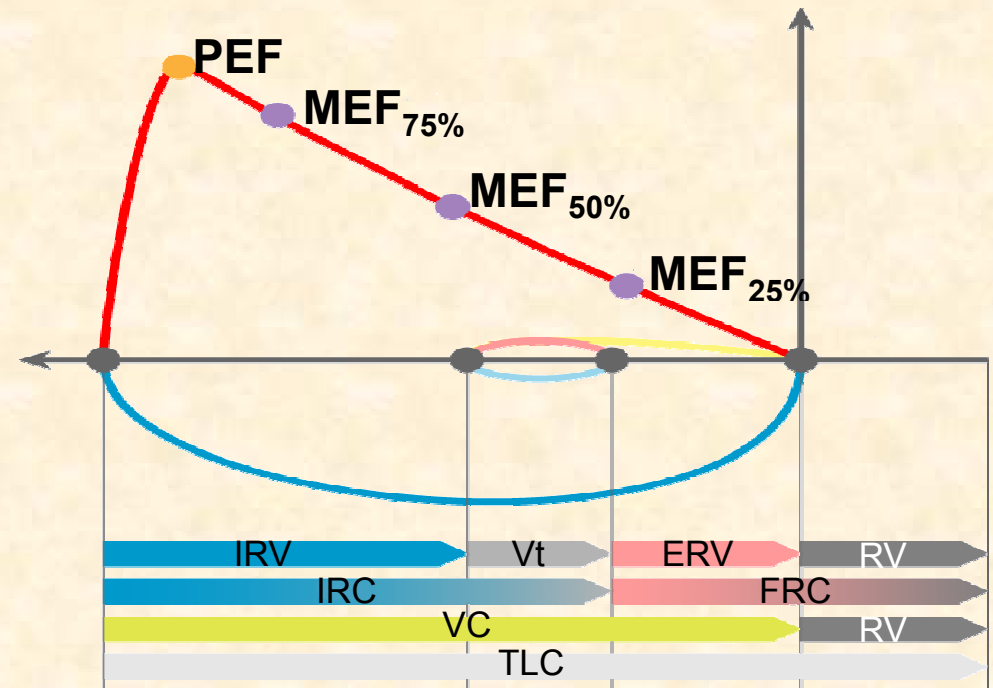


- **PULMONARY MINUTE VENTILATION RMV (respiratory minute volume) at rest** ($0.5 \text{ l} \times 12 \text{ breathes/min} = 6 \text{ l/min}$)
- **MAXIMAL VOLUNTARY VENTILATION (MVV)** (125-170 l/min)
- **PEAK EXPIRATORY FLOW RATE ($PEFR$)** ($\sim 10 \text{ l/s}$)

Flow – volume curve



- **PEF** – peak expiratory flow
- **MEF** – maximální maximal expiratory flow on the differential levels of FVC - 75 %, 50 % a 25 % FVC



Tady jsem skončila

PNEUMOGRAPHY

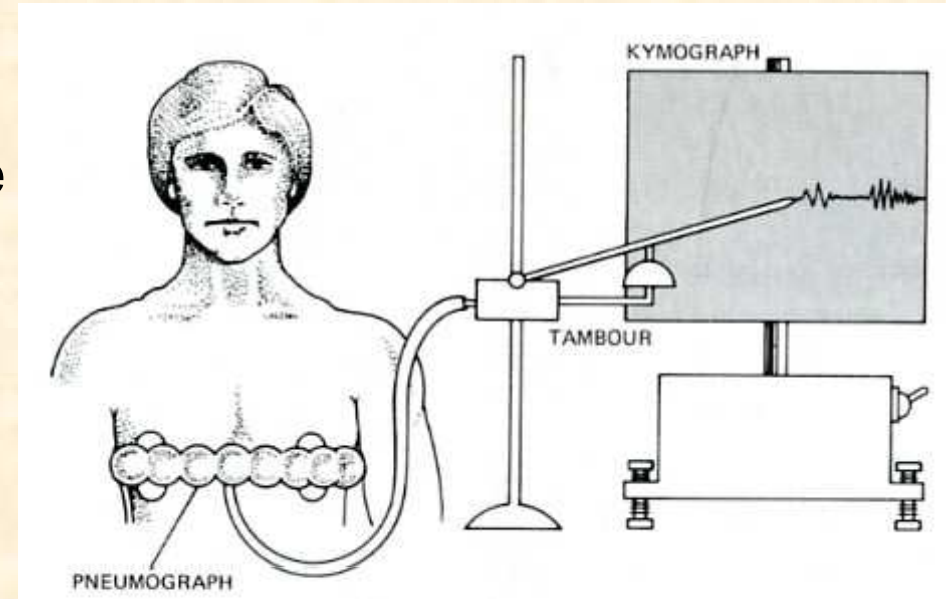
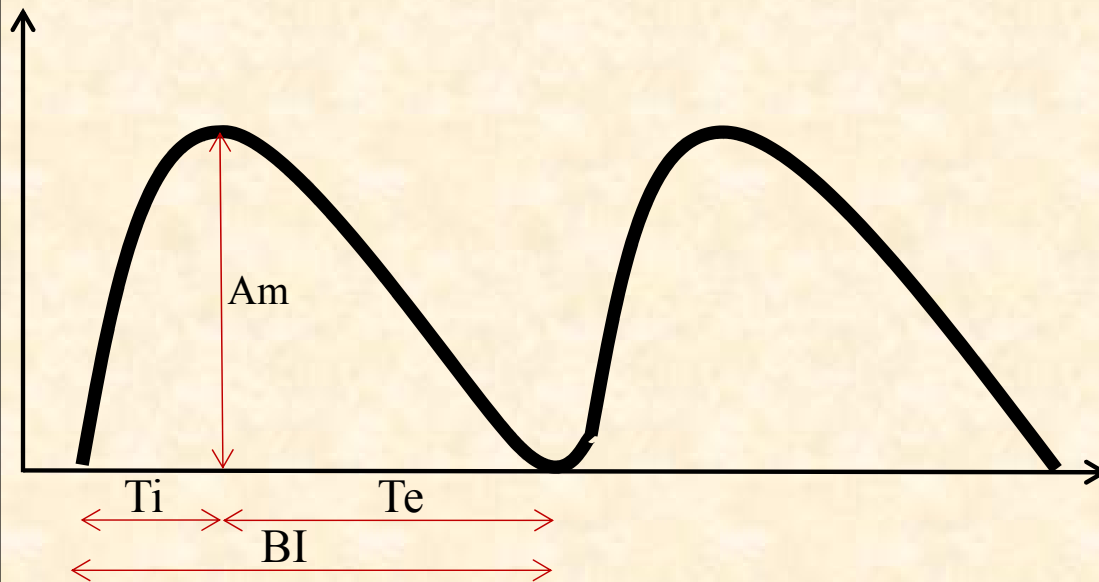
Principle

Pneumography – measurement of respiratory movements (via chest or abdomen)

- respiratory belt (piezoelectrical principle – is the ability of crystal to generate of electrical voltage during its deformation)

Record:

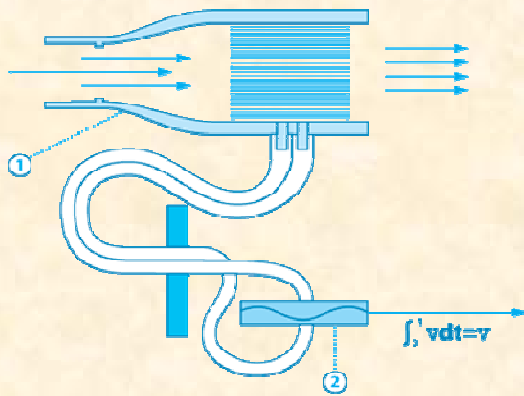
- Resting breathing
- Breathing after mild or intensive exercise
- Evaluation of record – T_i , T_e , BI a A_m



PNEUMOTACHOGRAPHY

Principle

Pneumotachograph - the device consists of tubes of the same diameter arranged in parallel. One of the tubes has branches with tubes near both its ends (oral and external). These are connected to a pressure sensor that allows you to measure the differences in air pressure at the beginning and end of the pneumotachograph in proportion to the speed of the inhaled or exhaled air.

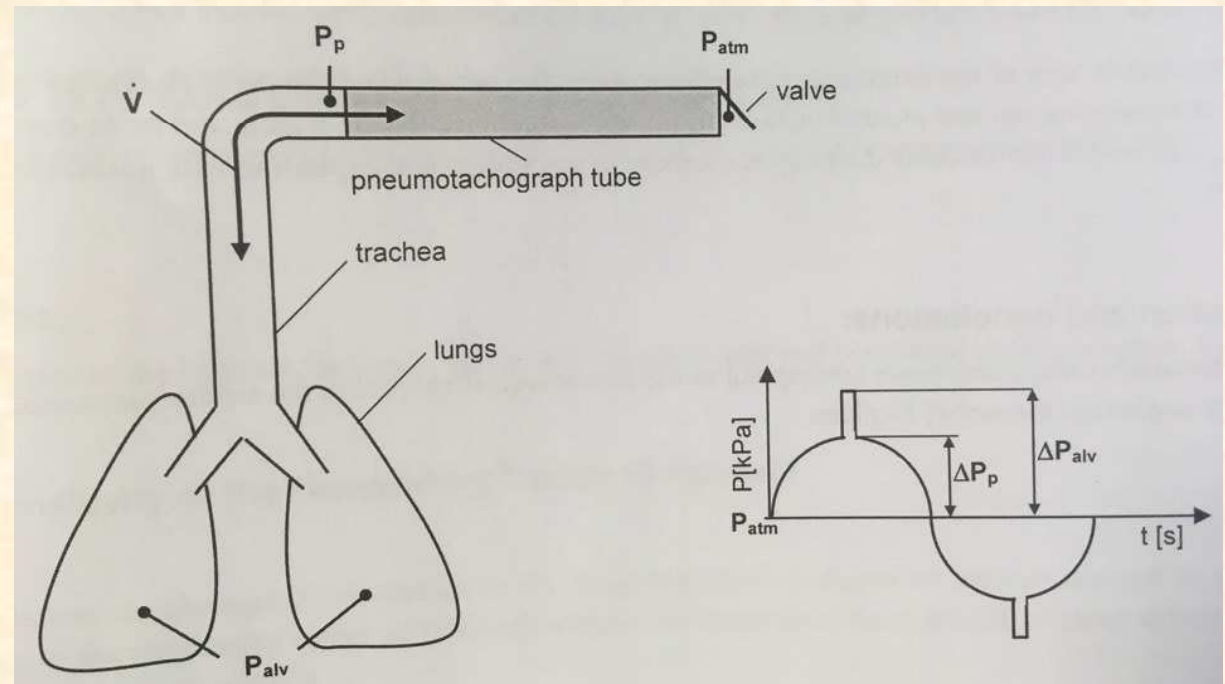


$$\Delta P_p = P_p - P_{atm}$$

$$\Delta P_{alv} = P_{alv} - P_{atm}$$

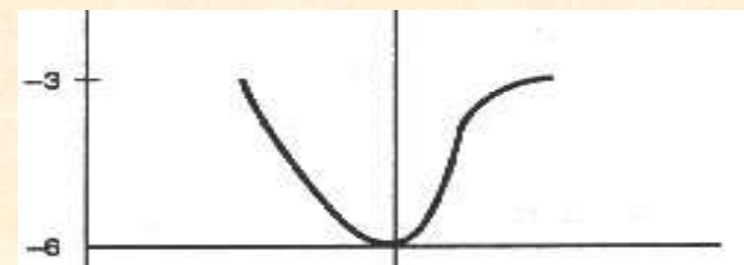
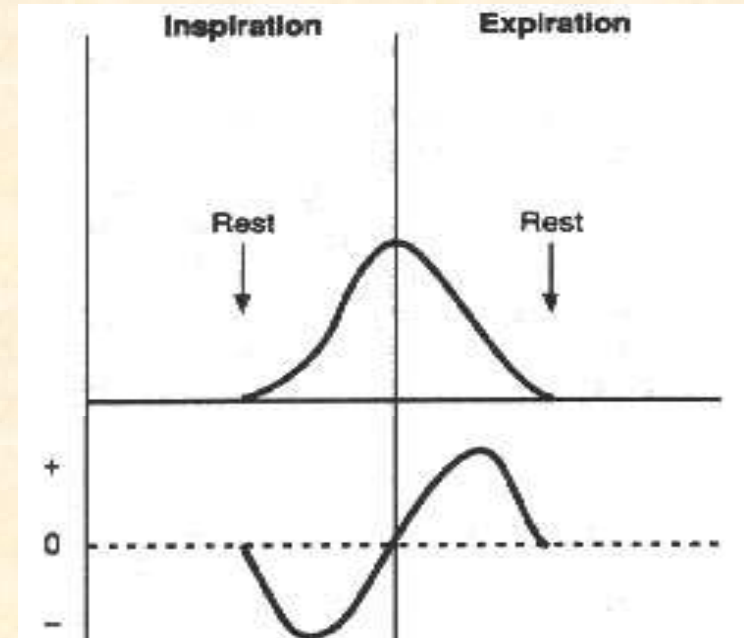
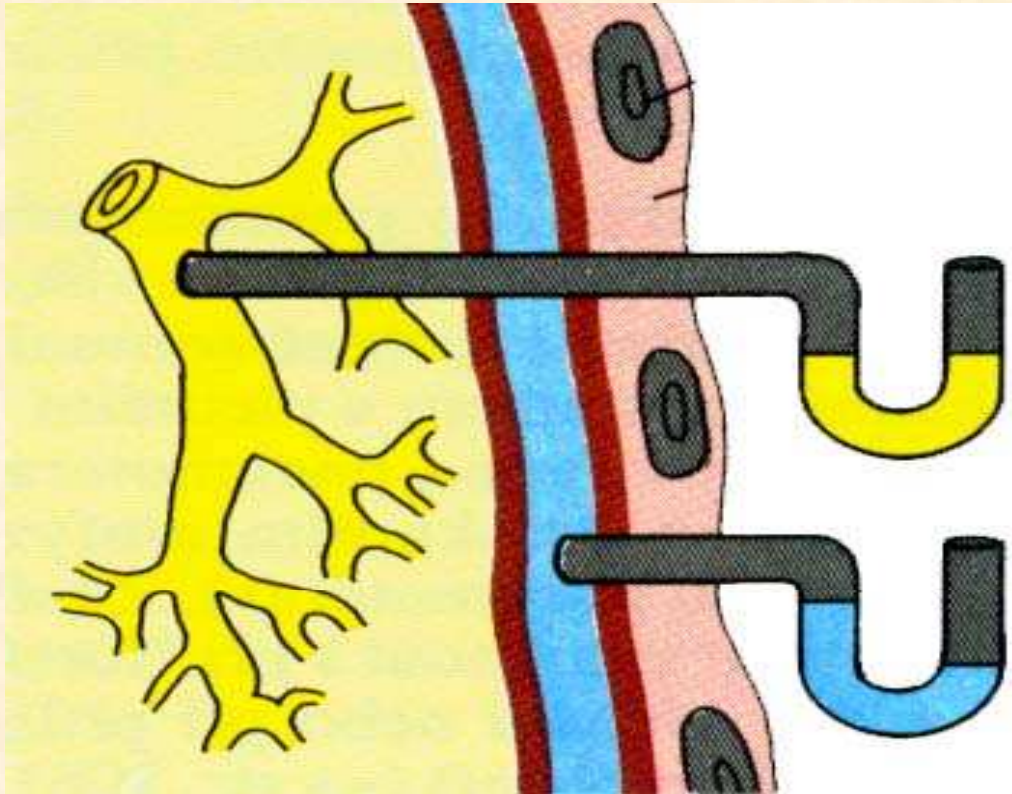
$$\frac{P_p - P_{atm}}{R_p} = \dot{V} = \frac{P_{alv} - P_p}{R_d}$$

$$R_d = R_p \cdot \left(\frac{\Delta P_{alv}}{\Delta P_p} - 1 \right)$$



- **Mechanics of breathing**

PLEURA
pulmonalis parietalis



FORCES PARTICIPATING IN RESPIRATION

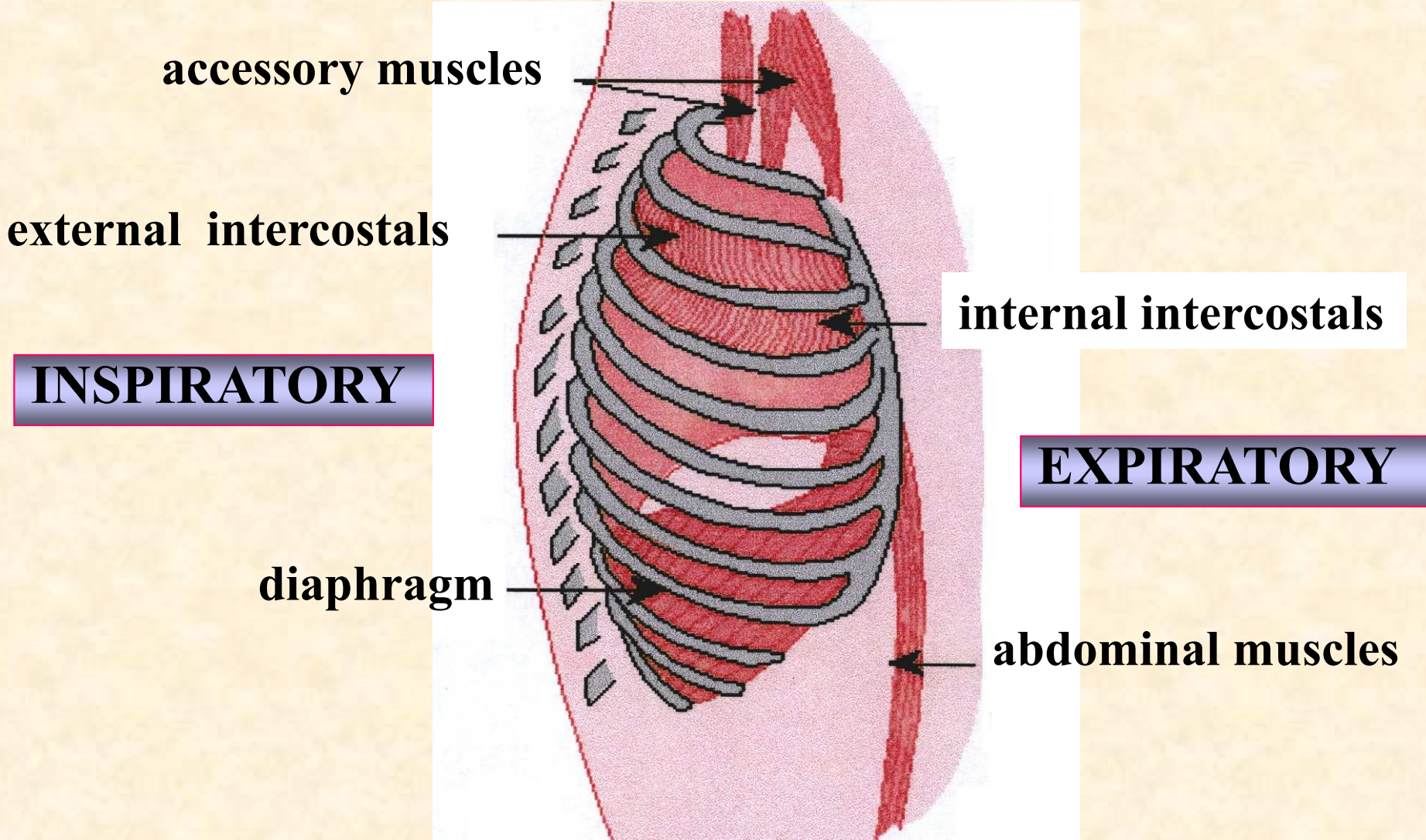
- **ACTIVE FORCES** performed by respiratory muscles
- **PASSIVE FORCES** represented by:
 - lungs elasticity
 - chest elasticity

QUIET RESPIRATION

INSPIRATION - active forces of inspiratory muscles prevail

EXPIRATION - only passive (elastic) forces are in action

RESPIRATORY MUSCLES



INSPIRATORY muscles

QUIET breathing

- *diaphragm* ($> 80\%$)
- *external intercostals* ($< 20\%$)

FORCED breathing in addition

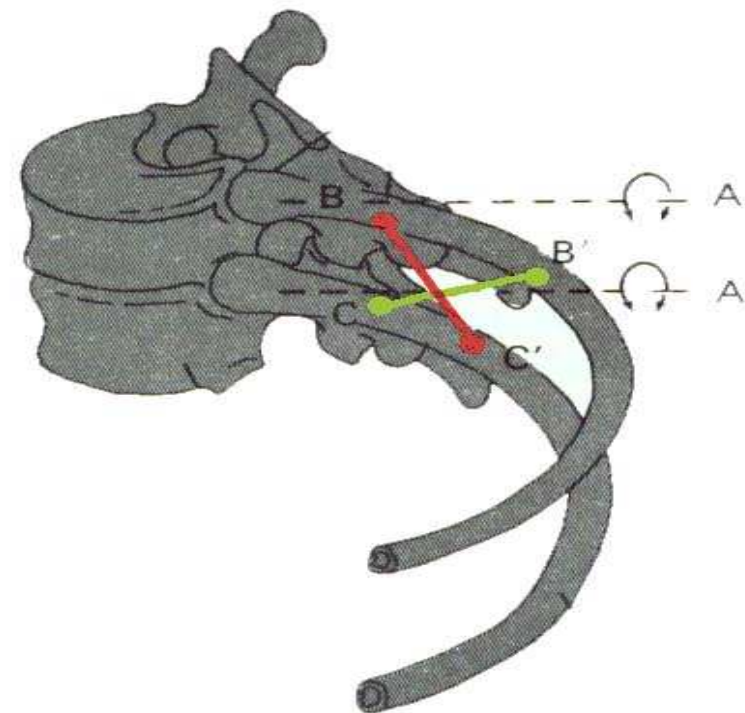
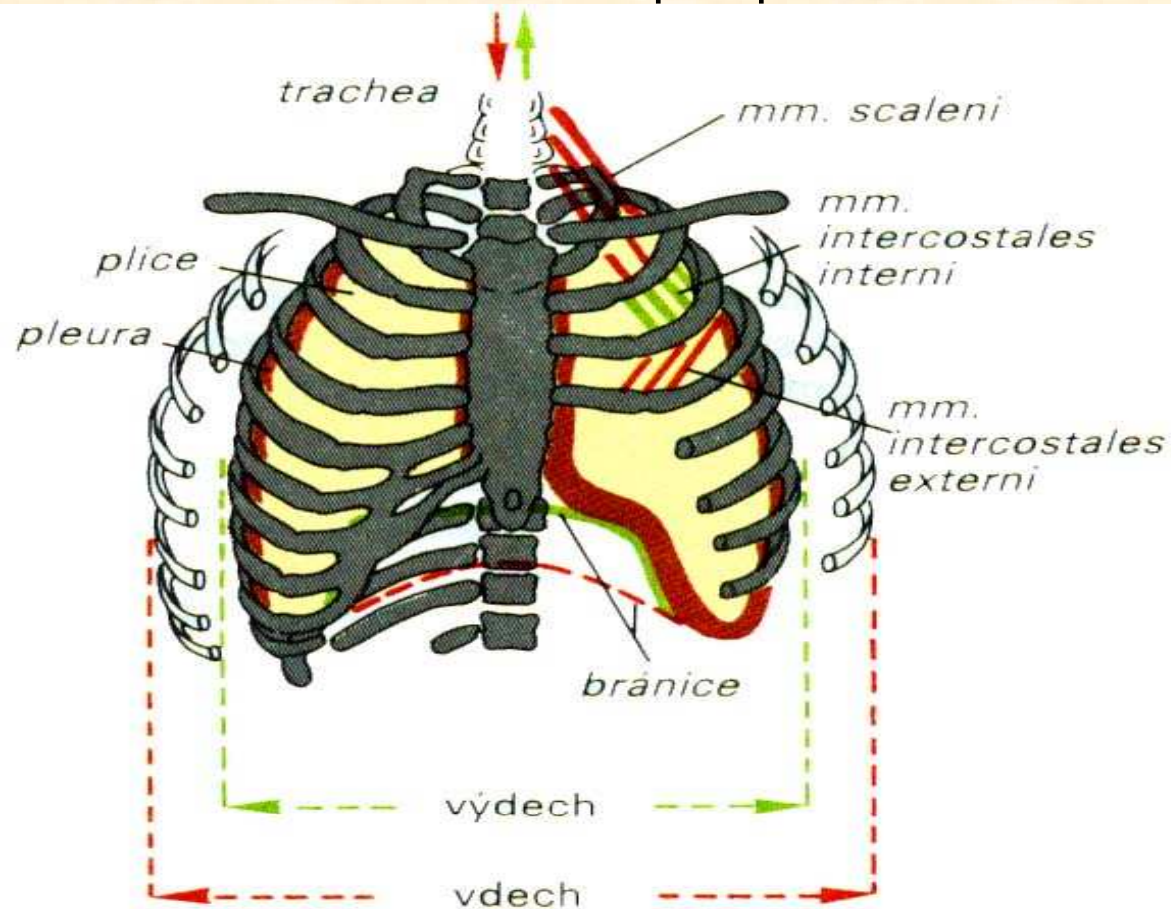
- *accessory inspiratory muscles* (mm. scalene)

EXPIRATORY muscles

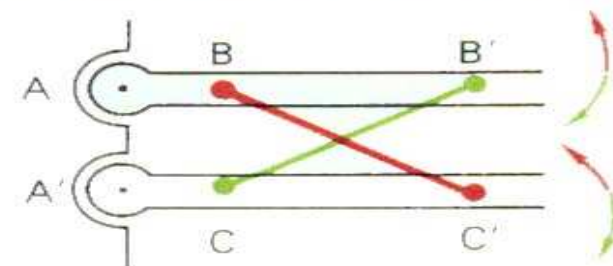
Only at FORCED breathing

- *internal intercostals*
- *muscles of the anterior abdominal wall*
(abdominal recti, ...)

Bucket-handle and water-pump handle effects



páka $A - B < A' - C' \rightarrow$ zvedání žeber



páka $A - B' > A' - C \rightarrow$ klesání žeber

COMPOSITION OF DRY ATMOSPHERIC AIR

O₂ **20.98 %**

N₂ **78.06 %**

CO₂ **0.04 %**

Other constituents

F_{O₂} **≅ 0.21**

F_{N₂} **≅ 0.78**

F_{CO₂} **= 0.0004**

BAROMETRIC (ATMOSPHERIC) PRESSURE AT SEA LEVEL

1 atmosphere = 760 mm Hg

PARTIAL PRESSURES OF GASSES IN DRY AIR AT SEA LEVEL

$$P_{O_2} = 760 \times 0.21 = \sim 160 \text{ mm Hg}$$

$$P_{N_2} = 760 \times 0.78 = \sim 593 \text{ mm Hg}$$

$$P_{CO_2} = 760 \times 0.0004 = \sim 0.3 \text{ mm Hg}$$

$$1 \text{ kPa} = 7.5 \text{ mm Hg (torr)}$$

COMPOSITION OF ALVEOLAR AIR

partial pressures in mm Hg

INSPIRED AIR

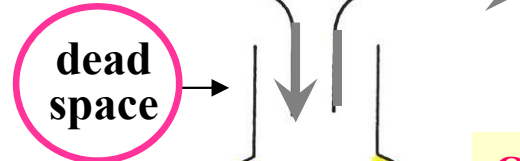
| | |
|-----------------|-------|
| O ₂ | 158.8 |
| CO ₂ | 0.3 |
| N ₂ | 601.0 |
| ... | |

760 mm Hg

EXPIRED AIR

| | |
|------------------|-------|
| O ₂ | 115.0 |
| CO ₂ | 33.0 |
| H ₂ O | 47.0 |
| N ₂ | 564.0 |
| ... | |

760 mm Hg



| | |
|------------------|-------|
| O ₂ | 100.0 |
| CO ₂ | 39.0 |
| H ₂ O | 47.0 |
| N ₂ | ... |

| | |
|-----------------|-------|
| O ₂ | 100.0 |
| CO ₂ | 39.0 |

physiological shunts

right heart

760 mm Hg

left heart

veins

| | |
|------------------|------|
| O ₂ | 40.0 |
| CO ₂ | 45.0 |
| H ₂ O | 47.0 |
| N ₂ | ... |
| ... | |

arteries

| | |
|------------------|------|
| O ₂ | 95.0 |
| CO ₂ | 41.0 |
| H ₂ O | 47.0 |
| N ₂ | ... |
| ... | |

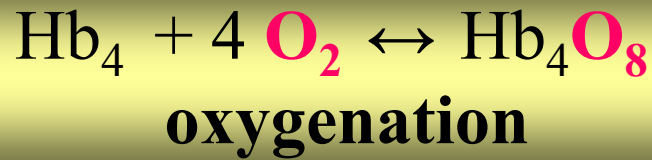
periphery capillaries

| | |
|------------------|------|
| O ₂ | 40.0 |
| CO ₂ | 45.0 |
| H ₂ O | 47.0 |
| N ₂ | ... |
| ... | |

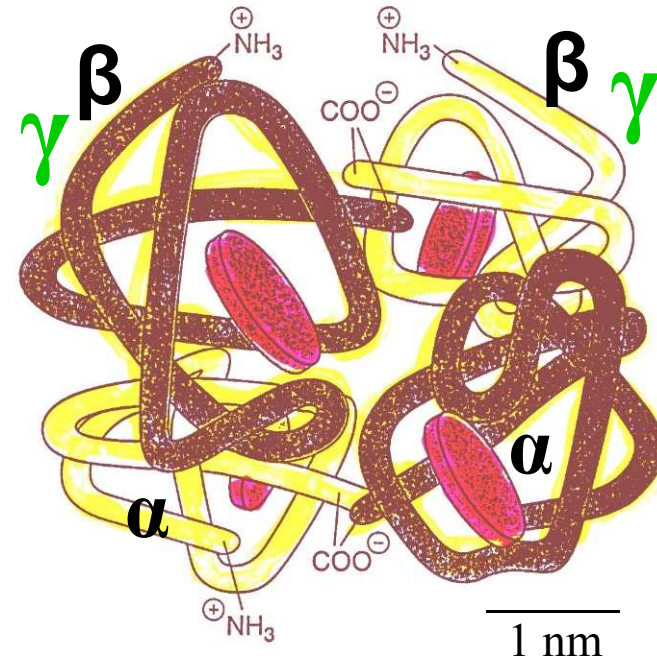
?

?

HAEMOGLOBIN



tetramer

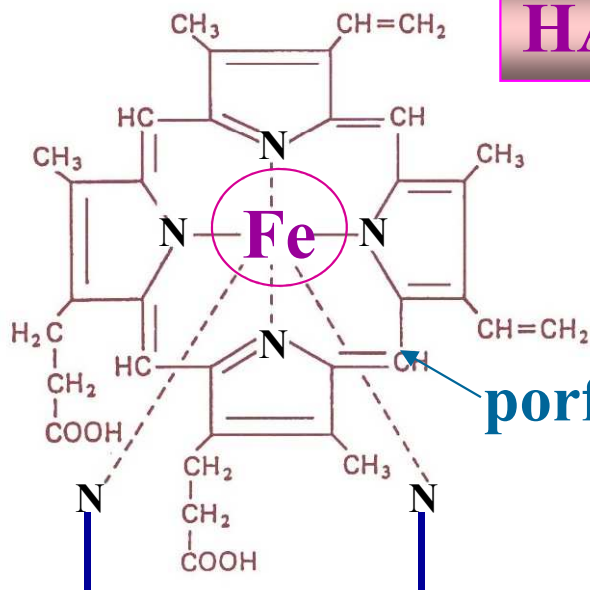


Fe^{2+}

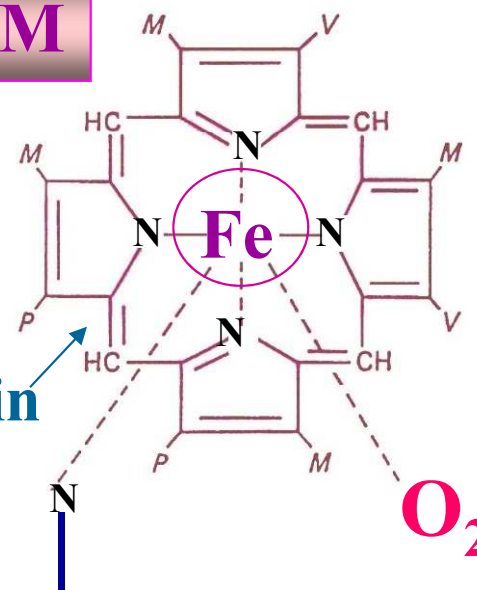
DEOXY

OXY

HAEM



porphyrin



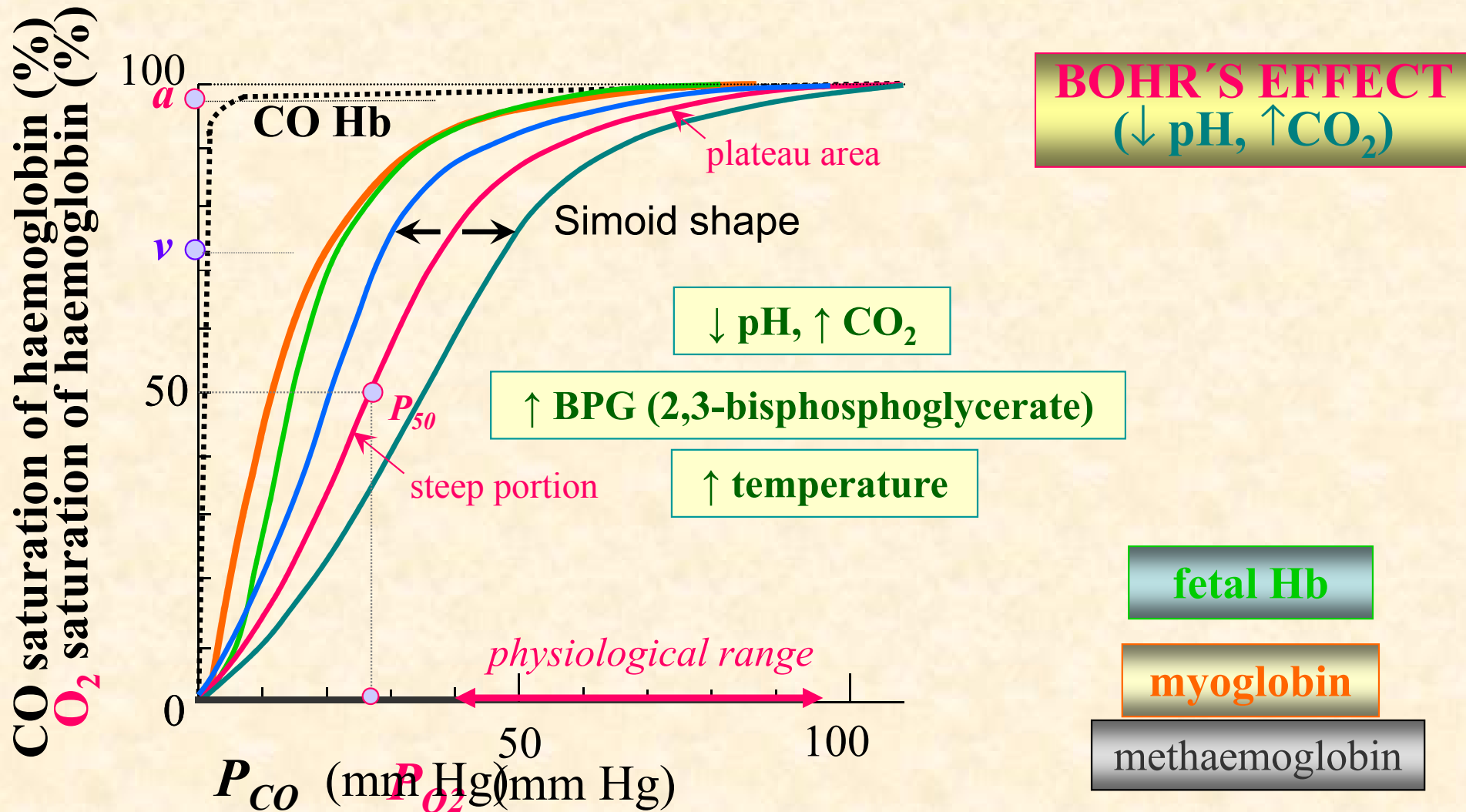
fetal Hb

Fe^{3+} (methaemoglobin)
oxidation

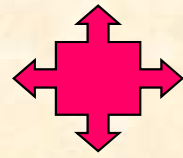
polypeptide chain

polypeptide chain

O_2 -HAEMOGLOBIN DISSOCIATION CURVE

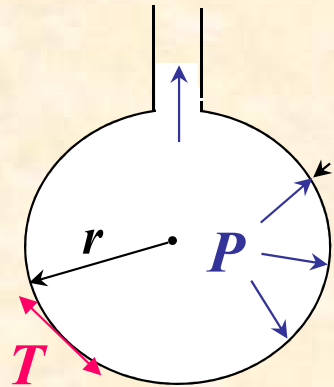


physically dissolved O_2 (1.4%)

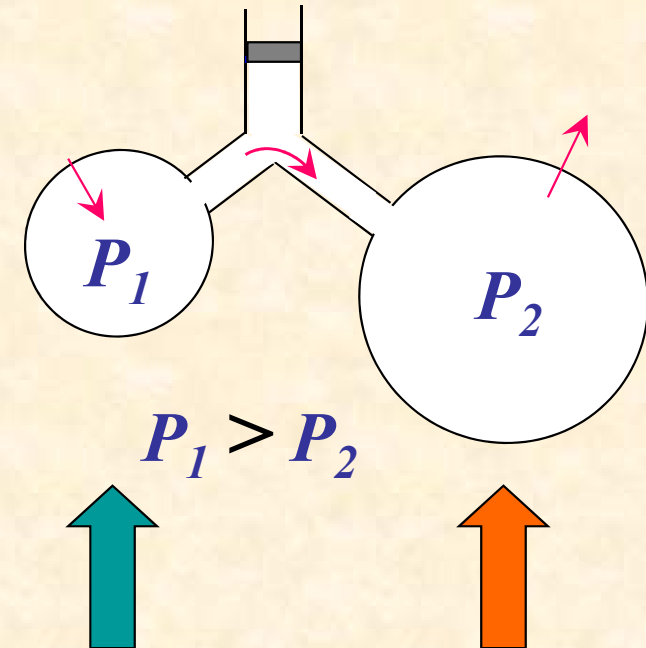


LAW OF LAPLACE

spherical structures



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



P pressure

r radius

T surface tension

PATHOLOGY

- **COLLAPSE OF ALVEOLI - ATELECTASIS**
- **EXPANSION OF ALVEOLI**

SURFACTANT

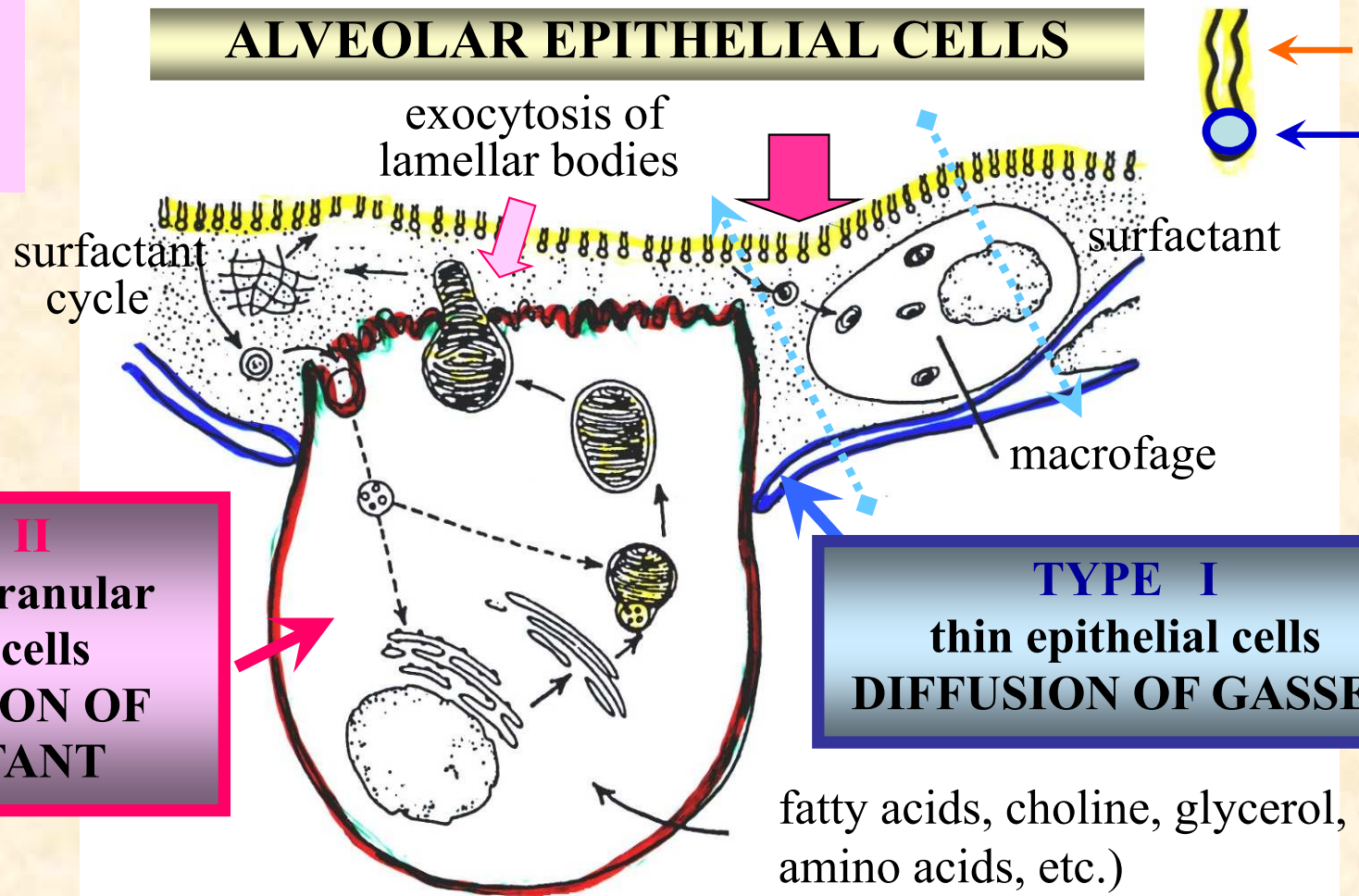
SURFACE TENSION LOWERING AGENT

EFFECT MAINLY IN THE EXPIRED POSITION

PHOSPHOLIPID

dipalmitoyl
fosfatidyl cholin

ALVEOLAR EPITHELIAL CELLS



TYPE II
specialized granular
epithelial cells
**PRODUCTION OF
SURFACTANT**

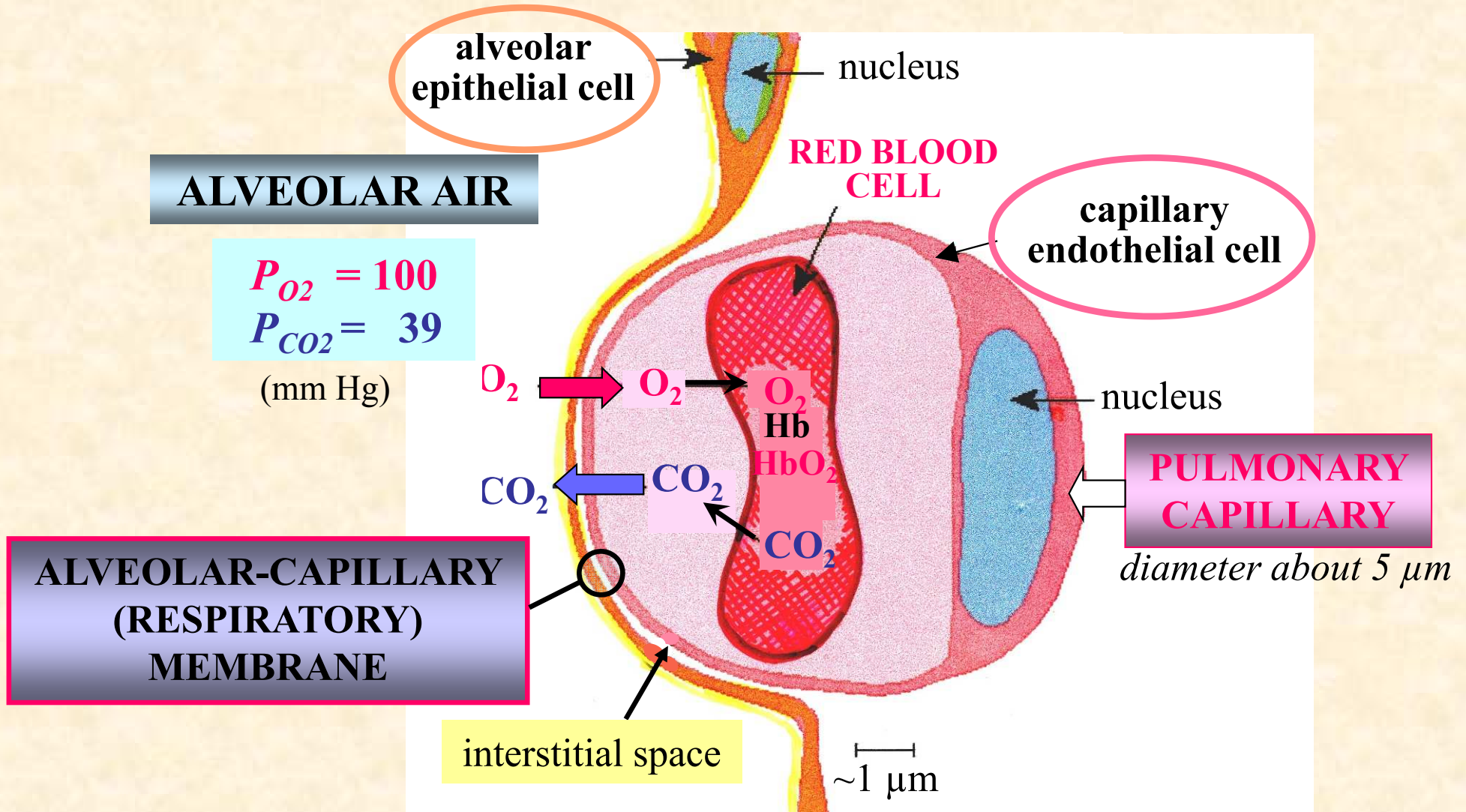
TYPE I
thin epithelial cells
DIFFUSION OF GASSES

fatty acids, choline, glycerol,
amino acids, etc.)



ALVEOLAR-CAPILLARY (RESPIRATORY) MEMBRANE

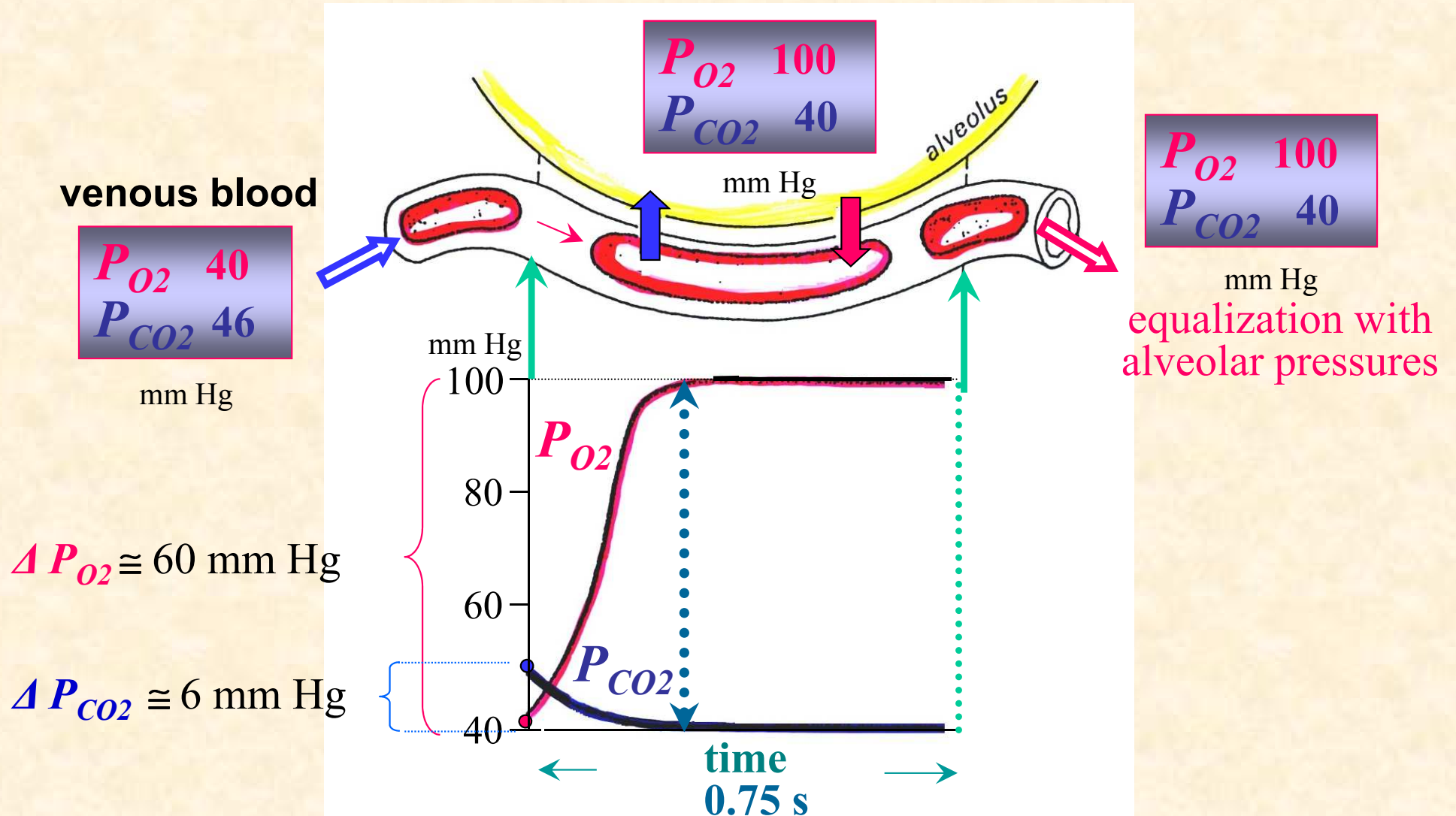
DIFFUSION OF GASES



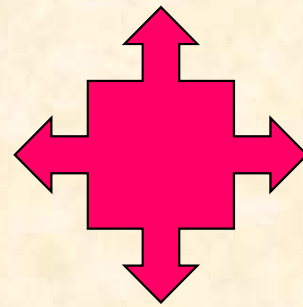
0.75 s

time interval of erythrocyte contact with respiratory membrane at rest

TIME COURSE OF CAPILLARY P_{O_2} AND P_{CO_2} DURING GRADUAL EQUILIBRATION WITH ALVEOLAR AIR



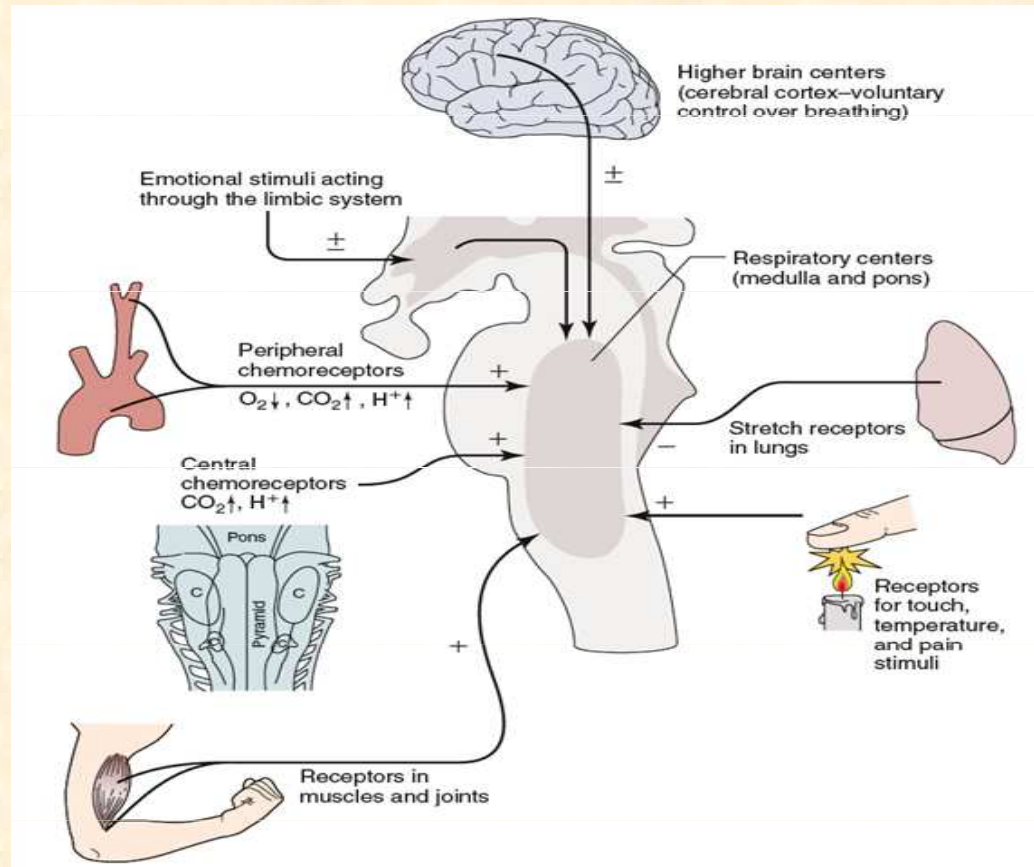
time interval of contact of erythrocyte with respiratory membrane at rest

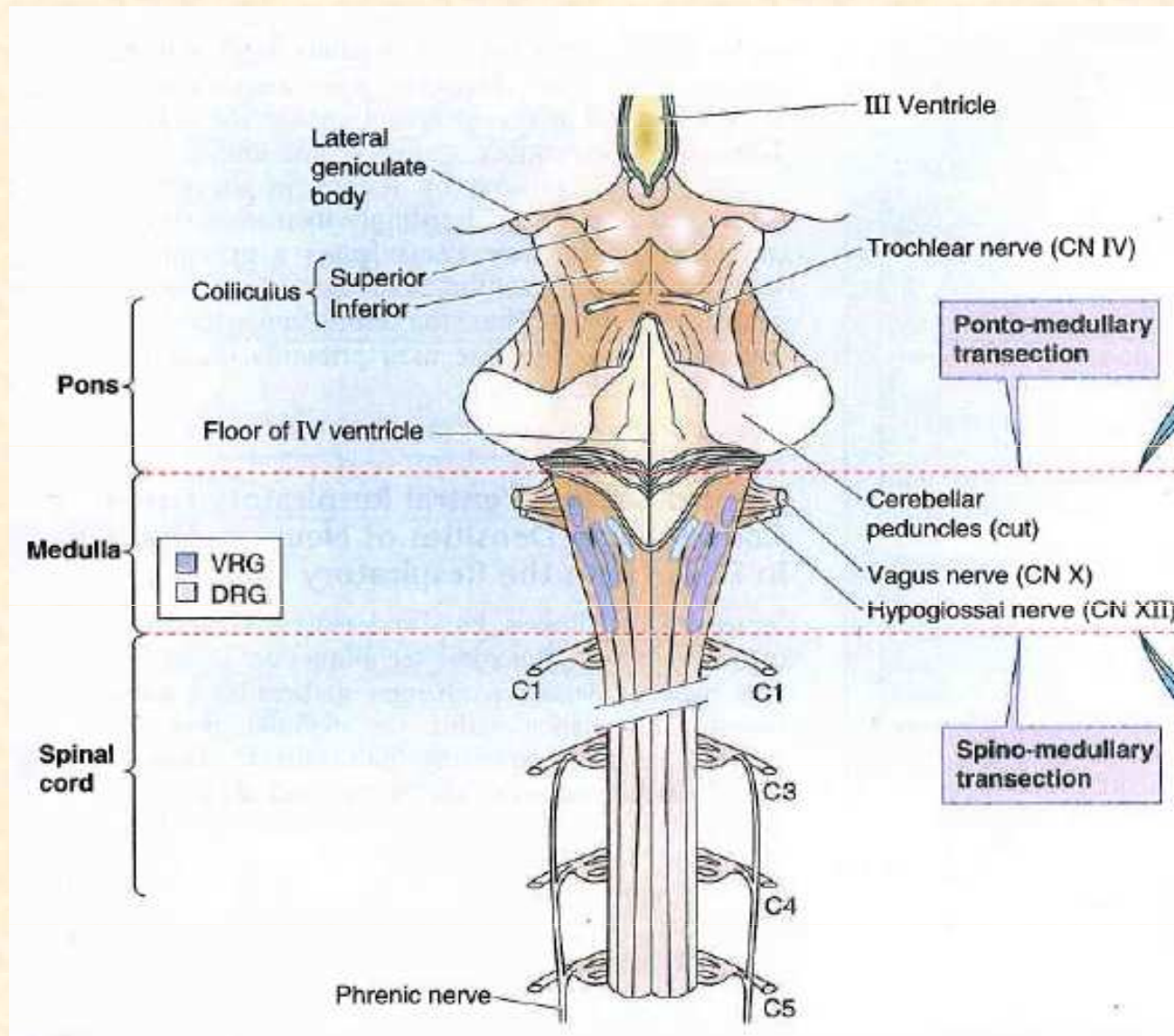


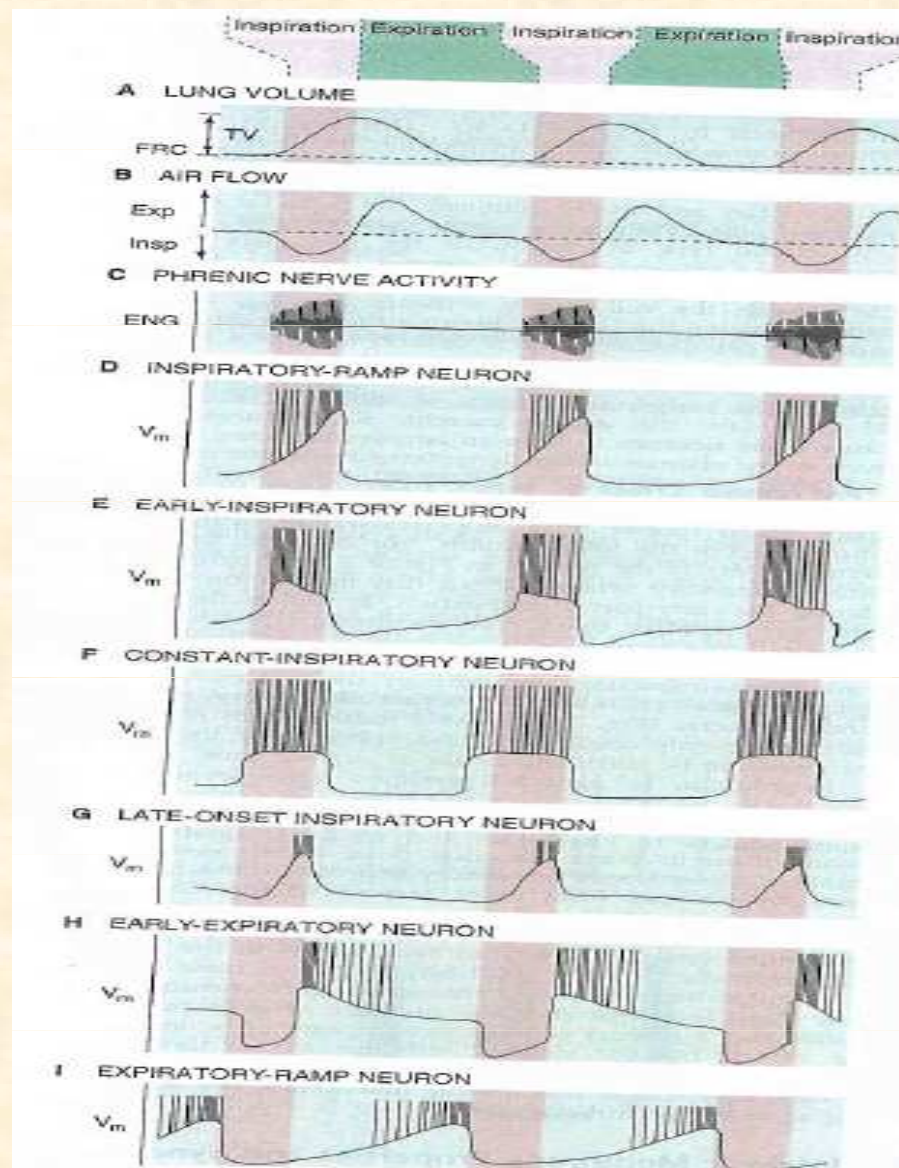
END



Control of ventilation







- **Breathing is an automatic process that takes place unconsciously. Automaticity of breathing comes from regular (rhythmic) activity of groups of neurons anatomically localized in the medulla and its vicinity.**

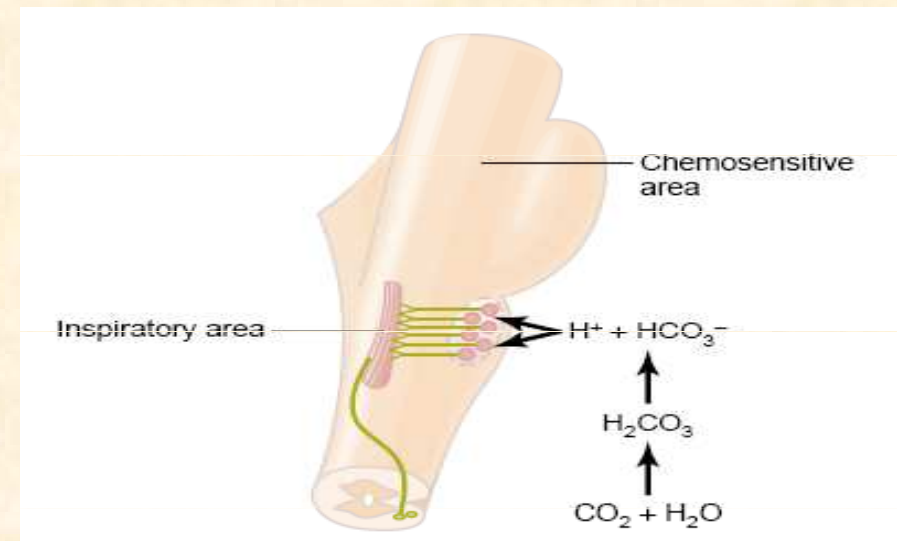
- They can be divided into three **main groups**:
 - *dorsal respiratory group* – placed bilaterally on the dorsal side of the medulla oblongata, only inspiratory neurons, sending axons to motoneurons of inspiratory muscles (diaphragm, external intercostal muscles; their activation=inspiration, their relaxation=expiration; participates on inspiration at rest and forced inspiration
 - *ventral respiratory group* - located on the ventrolateral part of the medulla oblongata, the upper part: neurons whose axons of motor neurons activate the main and auxiliary inspiratory muscles; the lower part: expiratory neurons which innervate expiratory muscles (internal intercostal muscles). Neurons in this group operate only during forced inspiration and forced expiration.
 - *Pontine respiratory group* - *pneumotaxic center* - dorsally placed on top of the pont, contributes to the frequency and depth of breathing; affects the activity of respiratory neurons in the medulla oblongata.

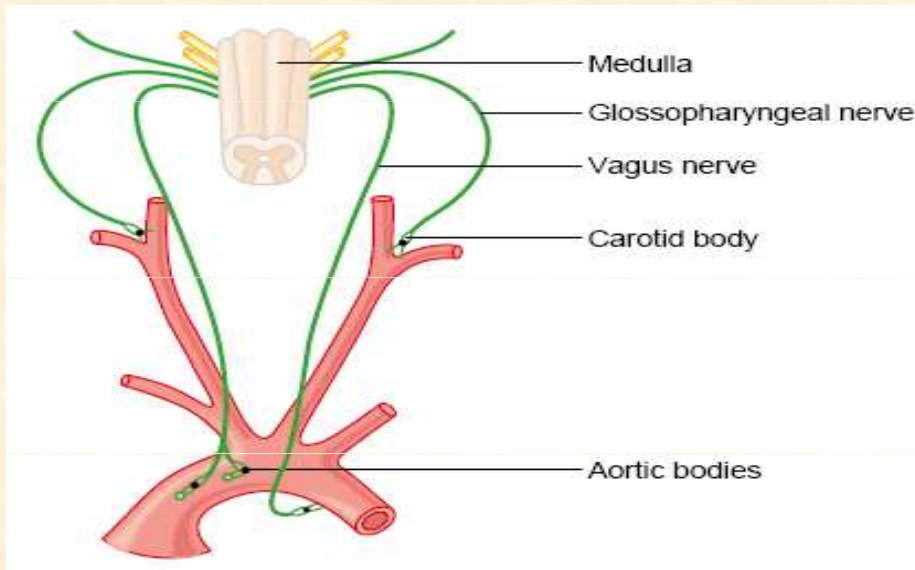
Chemical factors affecting the respiratory center:

Central chemoreceptors

- on the front side of the medulla
- sensitive only to increase of arterial $p\text{CO}_2$ (by increasing H^+)

- Notice:
- central chemoreceptor are stimulated by other types of acidosis (lactate acidosis, ketoacidosis)



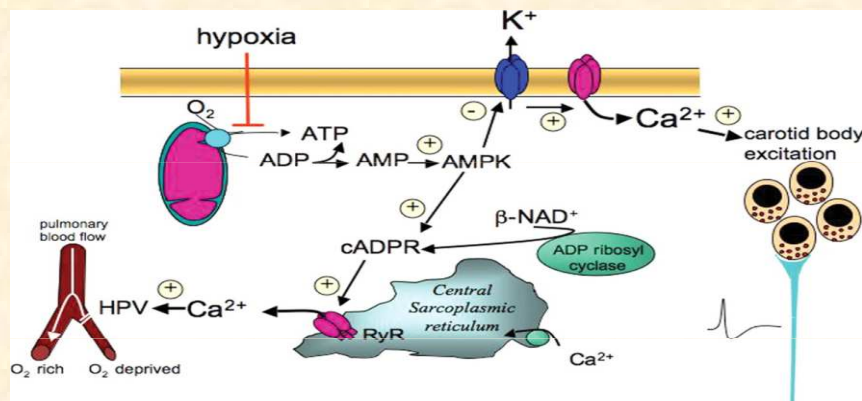


Peripheral chemoreceptors

– located in the aortic and carotid bodies

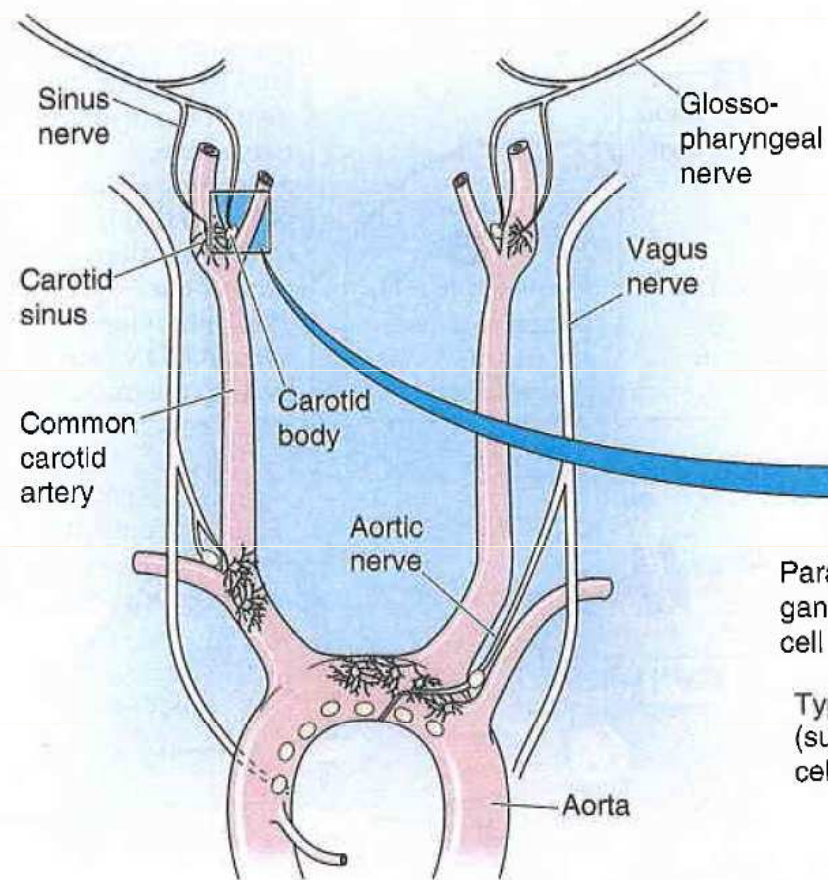
-primarily sensitive to decrease in arterial pO_2 , particularly to decrease of O_2 under 10-13 kPa in the arterial blood.

They convey their sensory information to the medulla via the vagus nerve and glossopharyngeal nerve.

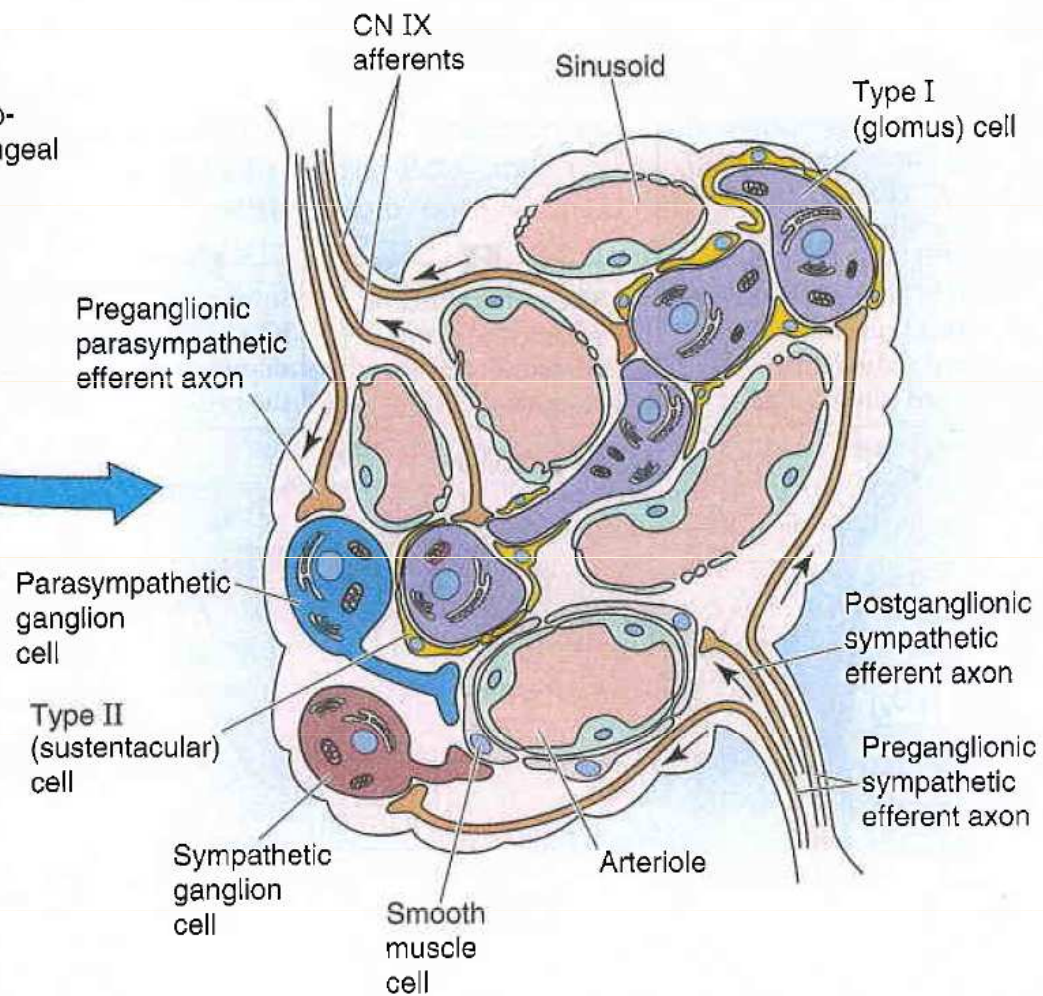


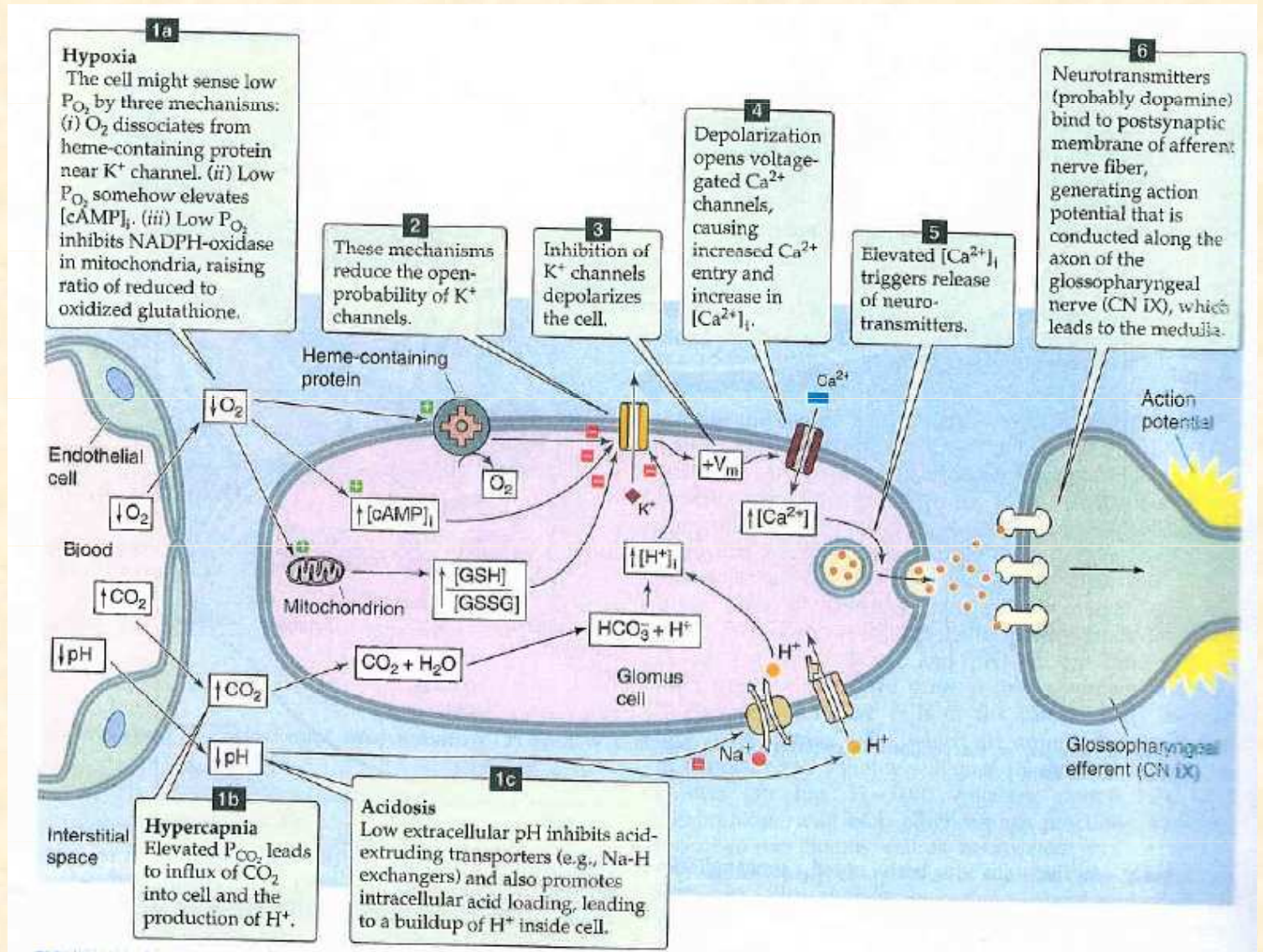
Mechanism of action: Decreased ATP production in mitochondria leads to depolarization of receptors membrane and to excitation of chemoreceptor

A LOCATION OF CAROTID AND AORTIC BODIES



B MICROSCOPIC ANATOMY OF CAROTID BODY





Modulation of respiratory output

Major parameters for feedback control – classical gases: pO_2 , pCO_2 , pH

In addition to these, the respiratory system receives input from two other major sources:

1. **variety of stretch and chemical/irritant receptors** that monitor the size of airways and the presence of noxious agents/receptors in respiratory system

2. **Higher CNS centers** that modulate respiratory activity for the sake of nonrespiratory activities

Irritants receptors on mucosa of respiratory system – rapidly adapting

Stimulus: agents - chemical substances (histamine, serotonin, prostaglandins, ammonia, cigarette smoke).

Response: increase mucus secretion, constriction of larynx and bronchus

C-fibre receptors (juxtacapillary=J receptors) – free nerve ending of n.vagus (unmyelinated axon) in interstitium of bronchus and alveolus;

Stimulus: Mechanical irritants (pulmonary hypertension, pulmonary oedema)+chemical

Response: hypopnoea, rapid shallow breathing, bronchoconstriction, cough

Stretch receptors slowly adapting (mechanoreceptors in tracheobronchial tree that detect the changes in lung volume by sensing the stretch receptors of the airway wall), inform to brain about the lung volume to optimize respiratory; its irritants triggered decrease activity of respiratory centre – **Hering-Breuer's reflexes**. (protecting the lungs from overinflation/deflation)

Baroreceptors – suppresses activity of respiratory centre

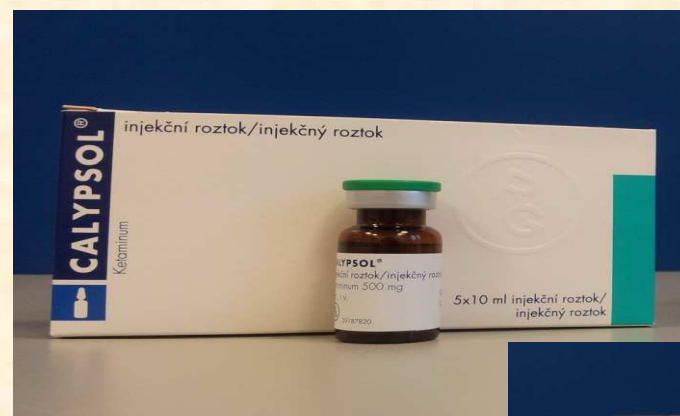
Irritants of **proprioceptors of muscles, tendons** during active and passive movements of limbs

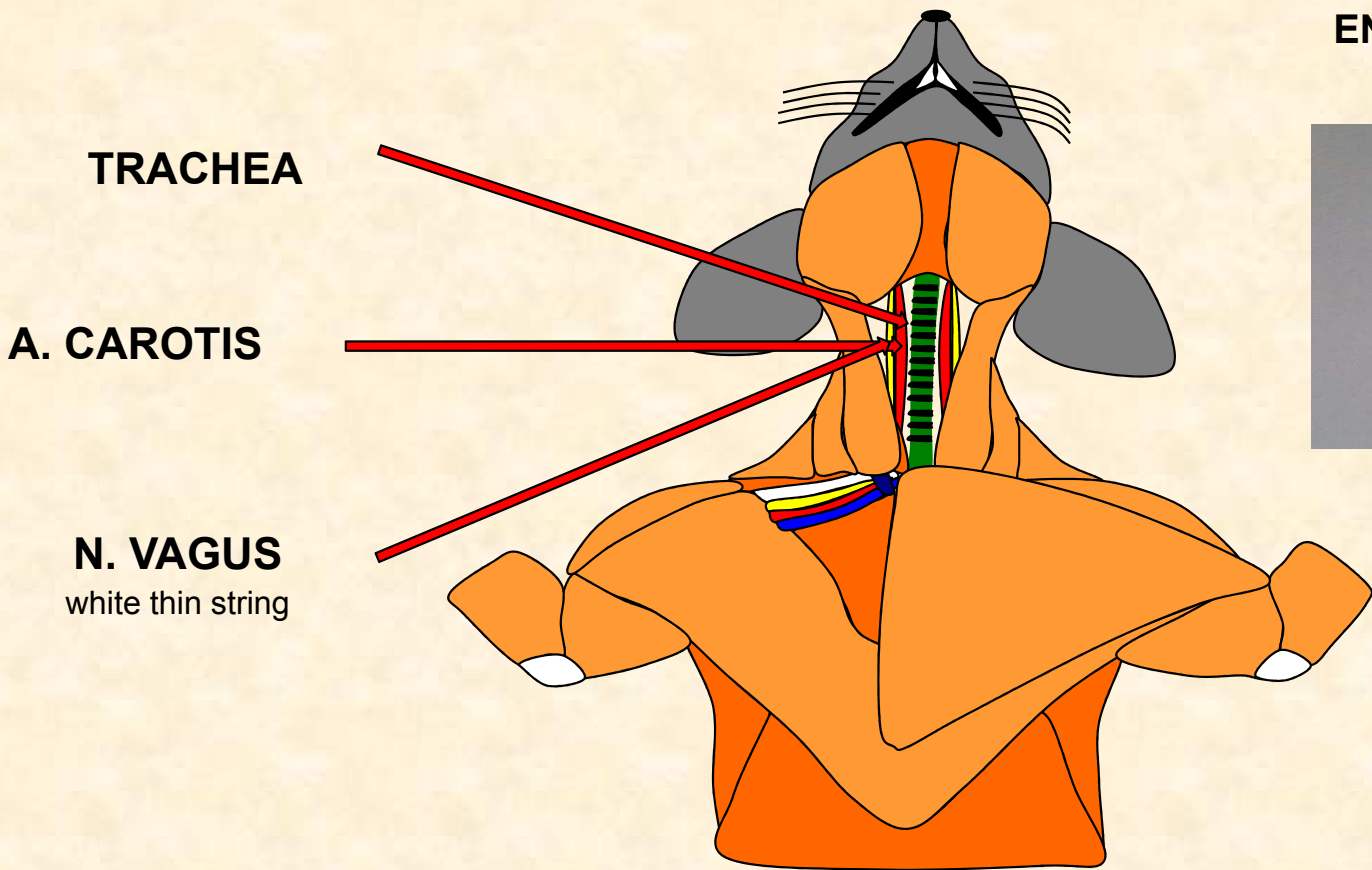
Influenced activity of respiratory neurons (increase minute ventilation during work load)

Limbic system, hypothalamus – strong pain, emotion

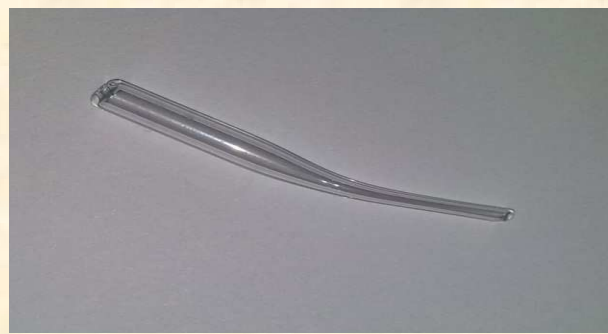
Tractus corticospinalis =cortex – activated RC during work load

temperature



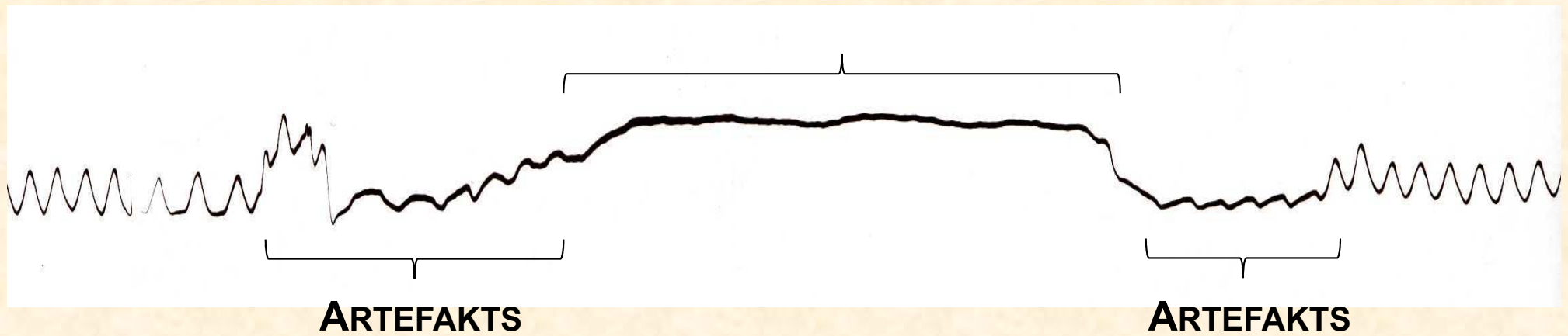


ENDOTRACHEAL CANNULA

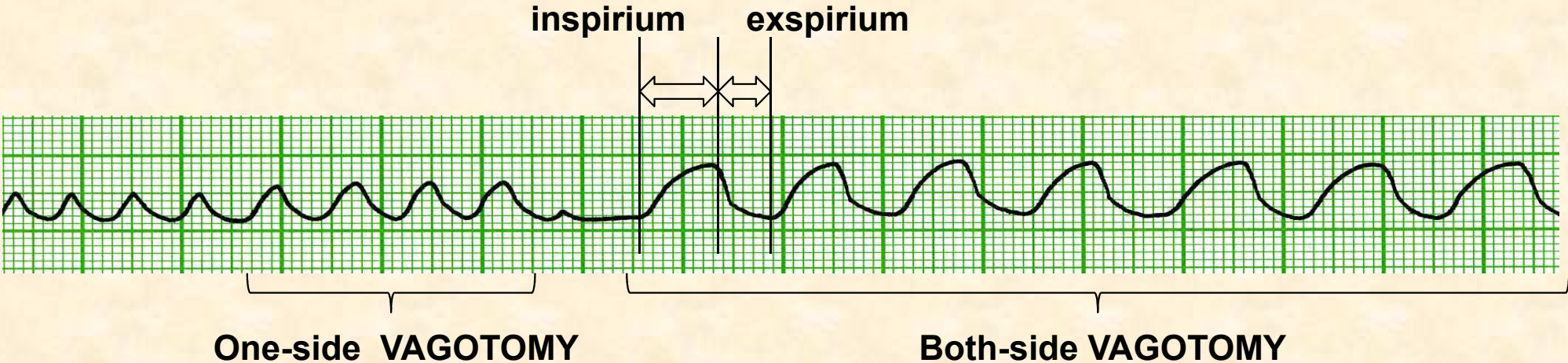


HERING-BREUER REFLEX

REFLEX STOP BREATHING



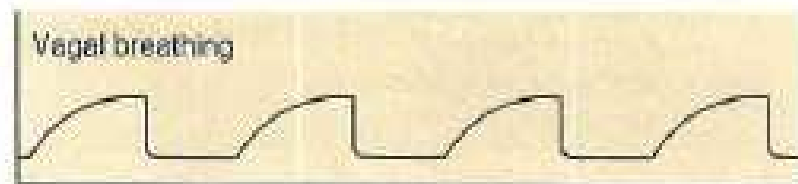
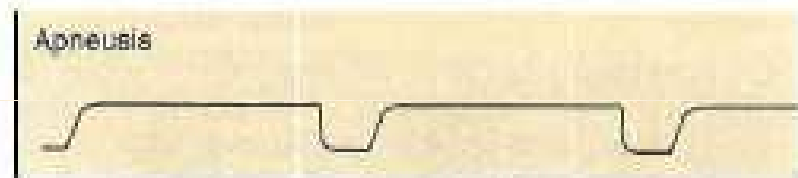
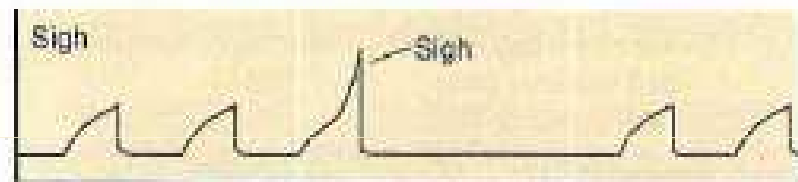
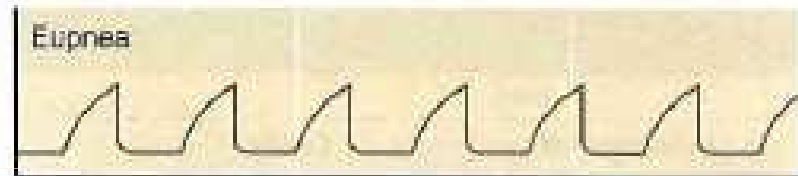
VAGOTOMY



Periodic breathing

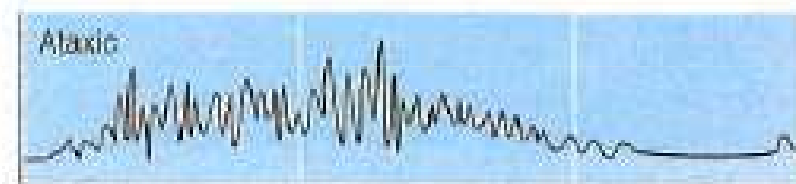
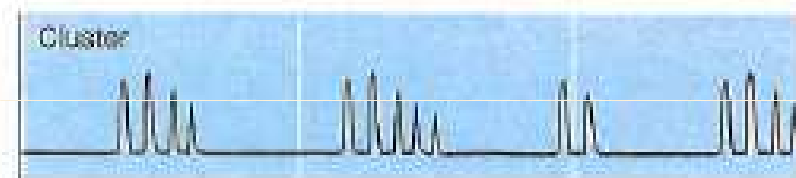
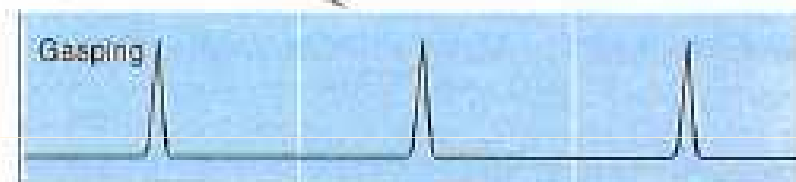
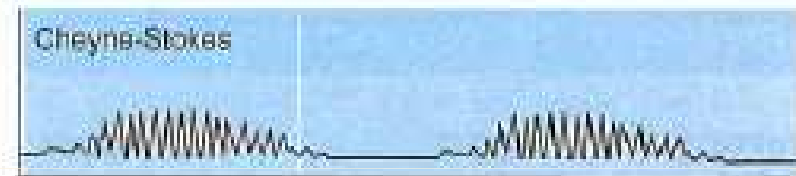
- It is not regular, rhythmic, but respiration occurs in periods ("a moment to breathe, take a moment to not breathe,,")
- **CHEYNE-STOKES**
- **BIOT'S**
- „gaspig“
- **KUSSMAUL**

A INTEGRATED PHRENIC NERVE ACTIVITY



0 0.2 0.4
Time (min)

B LUNG VOLUME



0 0.5 1.0
Time (min)

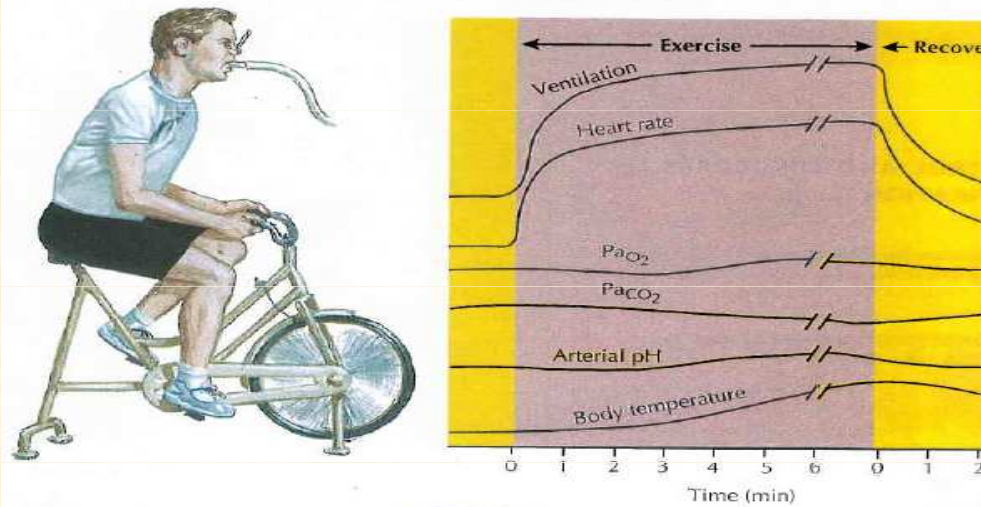


Christmas custom – cutting an apple - asterisk means health and happiness
for the New Year



*Merry Christmas
and Happy New Year 2022*

RESPIRATORY RESPONSE TO EXERCISE

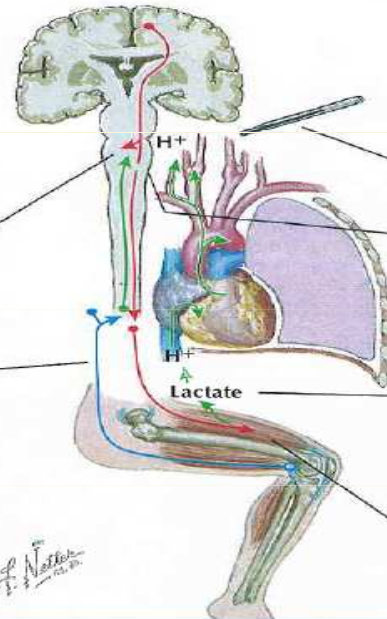


Factors that may account for initial abrupt rise and sharp terminal drop in ventilation

Collaterals to respiratory centers from motor pathways for muscle activation

Proprioceptive afferents from joint receptors to respiratory centers

Other unknown factors



Factors that may play a part in continued elevation of ventilation during continuing exercise

Rise in body temperature accounts for a small part of elevation

Respiratory neurons seem to be more responsive to changes in chemoreceptor activity. Centers may be more sensitive to fluctuation than to absolute values of PaO₂, PaCO₂, or pH

Lactic acid production due to anaerobic metabolism in muscle may increase H⁺ concentration of blood and CSF, thus affecting chemoreceptors

Possible metaboreceptors in exercising muscle

Other unknown factors

F. Netter M.D.

Hypoxia, hypoxemia

- **Hypoxia** is a general name for a lack of oxygen in the body or individual tissues.
- Hypoxemia is lack of oxygen in arterial blood.
- Complete lack of oxygen is known as anoxia.

The most common types of hypoxia:

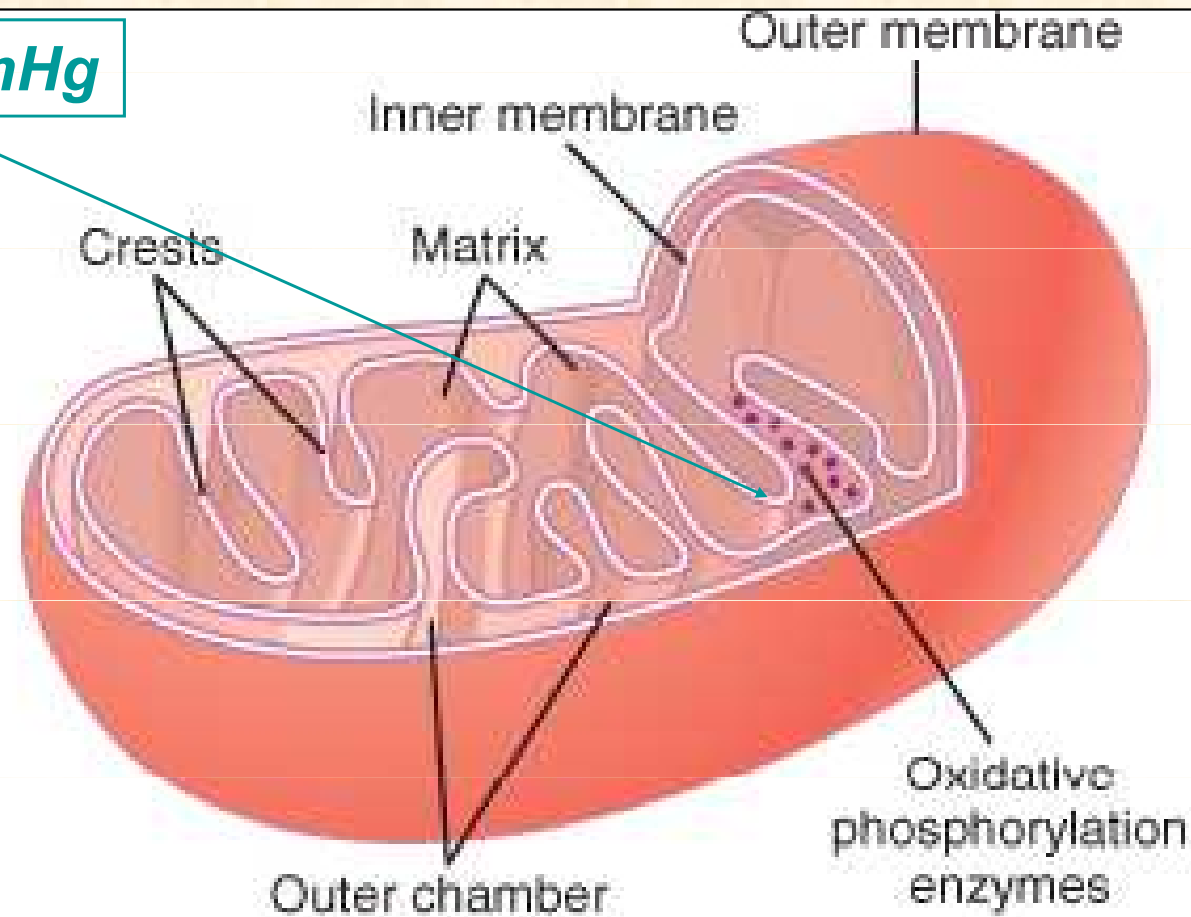
1. Hypoxic - physiological: stay at higher altitudes, pathological: hypoventilation during lung or neuromuscular diseases
2. Transport (anemic) - reduced transport capacity of blood for oxygen (anemia, blood loss, CO poisoning)
3. Ischemic (stagnation) - restricted blood flow to tissue (heart failure, shock states, obstruction of an artery)
4. Histotoxic - cells are unable to utilize oxygen (cyanide poisoning - damage to the respiratory chain)

OXYGEN FALL

mmHg

| | |
|---------------------------------|-------------|
| <i>dry atmospheric air</i> | 159 |
| <i>humid atmospheric air</i> | 149 |
| <i>ideal alveolar gass</i> | 105 |
| <i>end-expiration air</i> | 105 |
| <i>arterial blood</i> | 77 |
| <i>cytoplasm – mitochondria</i> | 3-10 |
| <i>mixed venous blood</i> | 40 |
| <i>venous blood</i> | 20 |

$pO_2 = 1 \text{ mmHg}$



Hypercapnia

- Hypercapnia - increase of concentration of carbon dioxide in the blood or in tissues that is caused by retention of CO₂ in the body
- possible causes: total alveolar hypoventilation (decreased respiration or extension of dead space)
- mild hypercapnia (5 -7 kPa) causes stimulation of the respiratory center (therapeutic use: pneumoxid = mixture of oxygen + 2-5% CO₂)
- hypercapnia around 10 kPa - CO₂ narcosis - respiratory depression (preceded by headache, confusion, disorientation, a feeling of breathlessness)
- hypercapnia over 12 kPa - significant respiratory depression - coma and death.

Travelling by aircraft

(On board aircraft is pressure as on 2000 m above sea level)

High risk for patients with diseases:

- **concentration of hemoglobin lower than 60 %**
- **severe step of atherosclerosis**
- **cardial insufficiency**
- **respiratory insufficiency**
- **non-treated hypertension (BP over 200/100mmHg)**

Toxicity of oxygen

The toxicity seems to be due to the production of the superoxid anion and H_2O_2

Causes:

- lost of possibility binding CO_2 in venous blood*
- in lungs – pulmonary edoema – decrease CO_2 expenditure*

Critical values > 40 kPa (300 mmHg) –dependence on time

Toxicity of oxygen

Exposure – 8 hours:- respiratory passages became irritated

- Substernal distress***
- Nasal congestion***
- Sore throat***
- Cough***

- 24-48 hours:

- damage of lungs – decrease production of surfactant***

Recommendation:

100 % - give discontinuosly

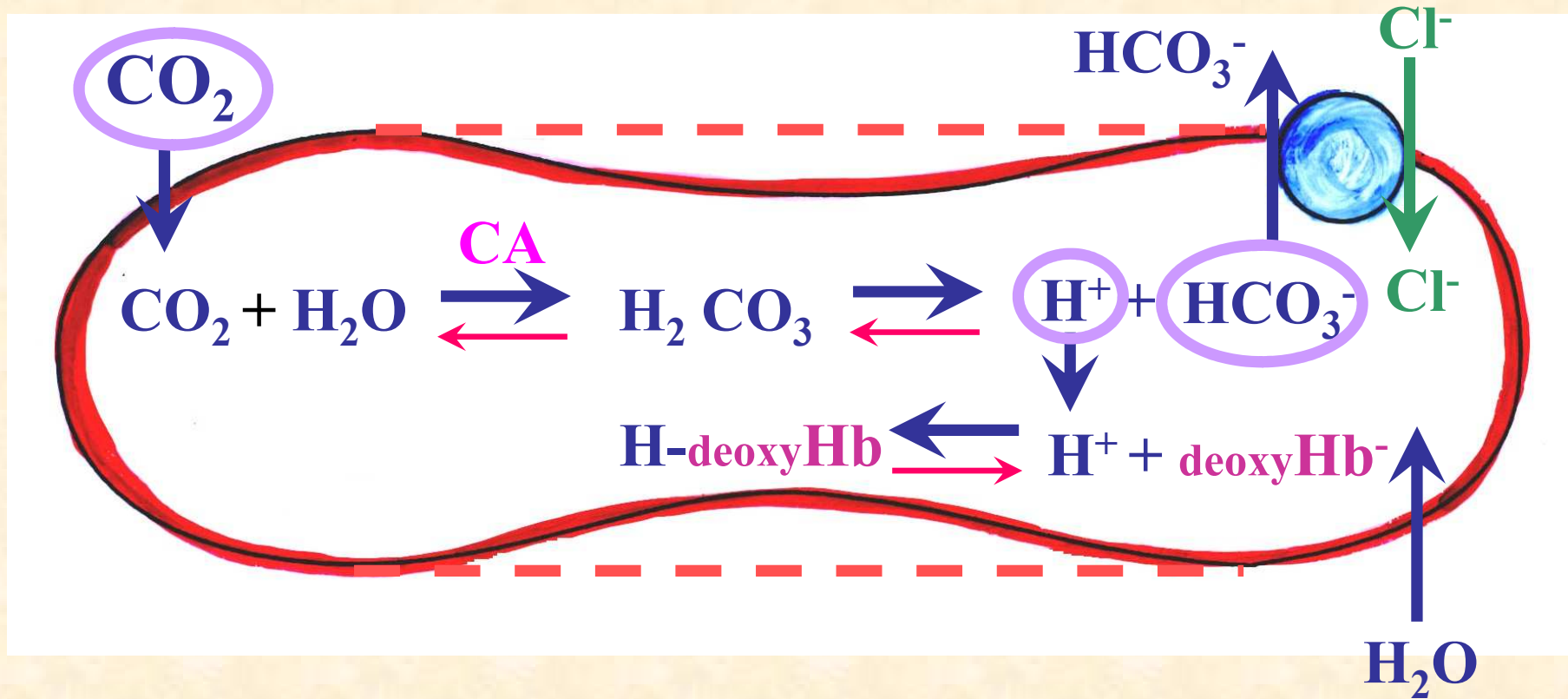
THANK YOU FOR YOUR
ATTENTION



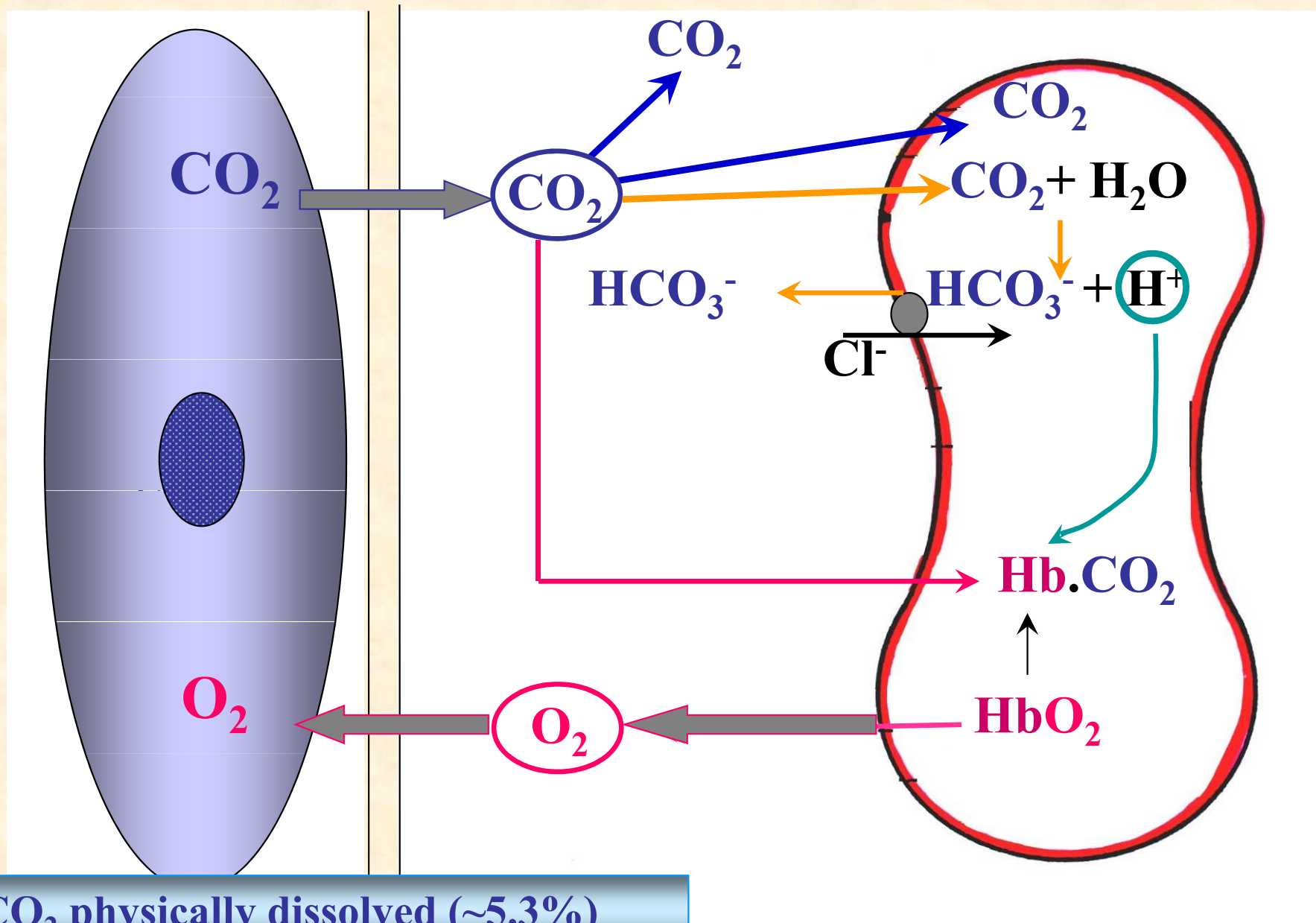


TRANSPORT OF CO₂

HAMBURGER CHLORIDE SHIFT



CA – carbonic anhydrase



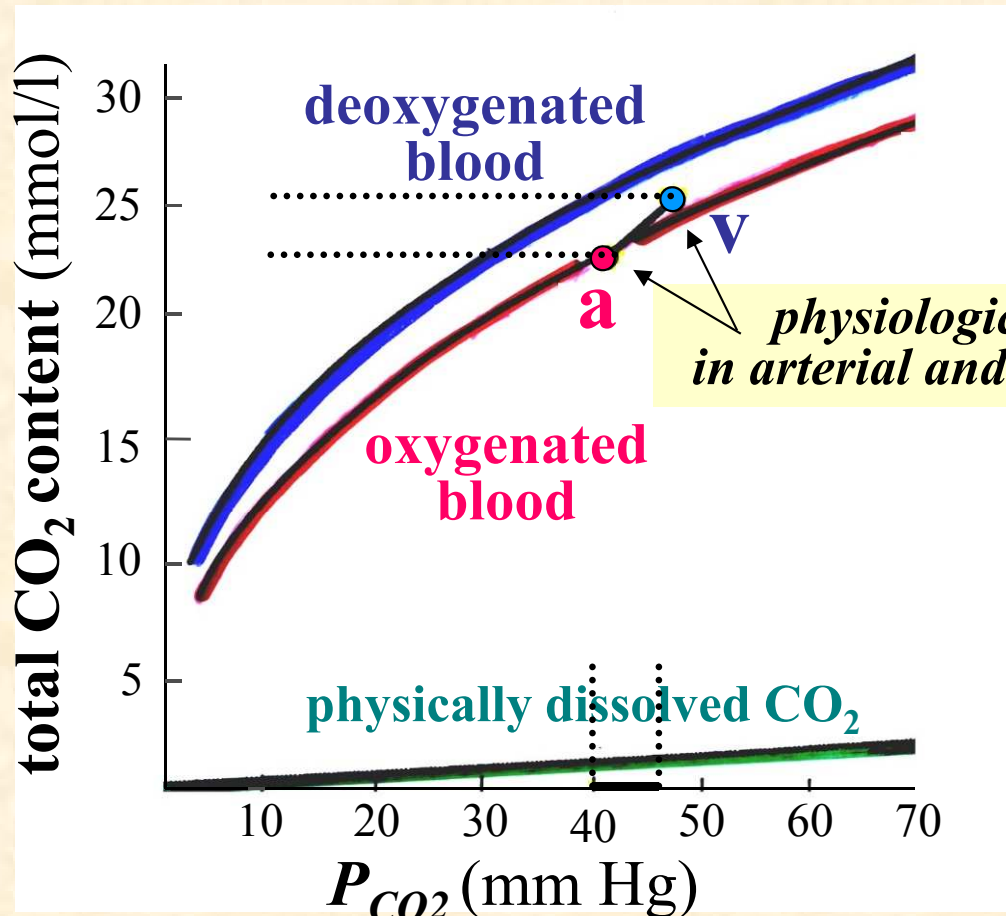
- CO₂ physically dissolved (~5.3%)

- $\text{CO}_2 + \text{Hb-NH}_2 \rightleftharpoons \text{Hb.NH-COO}^-$ (carbamino-Hb) (~5.3 %)

- $\text{CO}_2 + \text{H}_2\text{O} \rightleftharpoons \text{HCO}_3^- + \text{H}^+$ (~89%)

60% in plasma, 29% in red blood cell

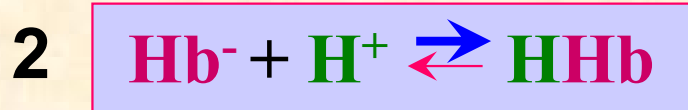
CO₂ DISSOCIATION CURVE



HALDANE EFFECT

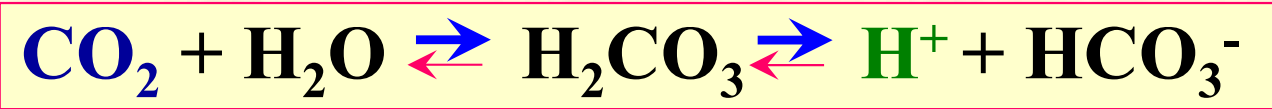
?

DEOXY-Hb

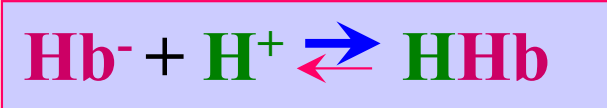


→ deoxygenated blood in peripheral tissues

← oxygenated blood in the lungs




↑↓



TISSUES: DEOXY-Hb binds H⁺ more readily (weaker acid) ⇒ ↑ amount of chemically bound CO₂

LUNGS: H⁺ is released from OXY-Hb ⇒ ↓ amount of chemically bound CO₂

- 
- I PHYSIOLOGY OF AIR PASSAGES**
 - II BASIC MEASURABLE PARAMETERS**
 - III ACTIVE AND PASSIVE FORCES**
 - IV COMPOSITION OF ALVEOLAR AIR**
 - V ALVEOLAR-CAPILLARY MEMBRANE**
 - VI TRANSPORT OF O_2 AND CO_2 IN THE BLOOD**

I AIR PASSAGES

→ II MEASURABLE PARAMETERS

- DEAD SPACE
- LUNG VOLUMES
- FUNCTIONAL INVESTIGATION
- CHARACTERISTIC PRESSURES

III ACTIVE AND PASSIVE FORCES

- RESPIRATORY MUSCLES
- LUNGS ELASTICITY
- COMPLIANCE
- WORK OF BREATHING

IV COMPOSITION OF ALVEOLAR AIR

V ALVEOLAR-CAPILLARY MEMBRANE

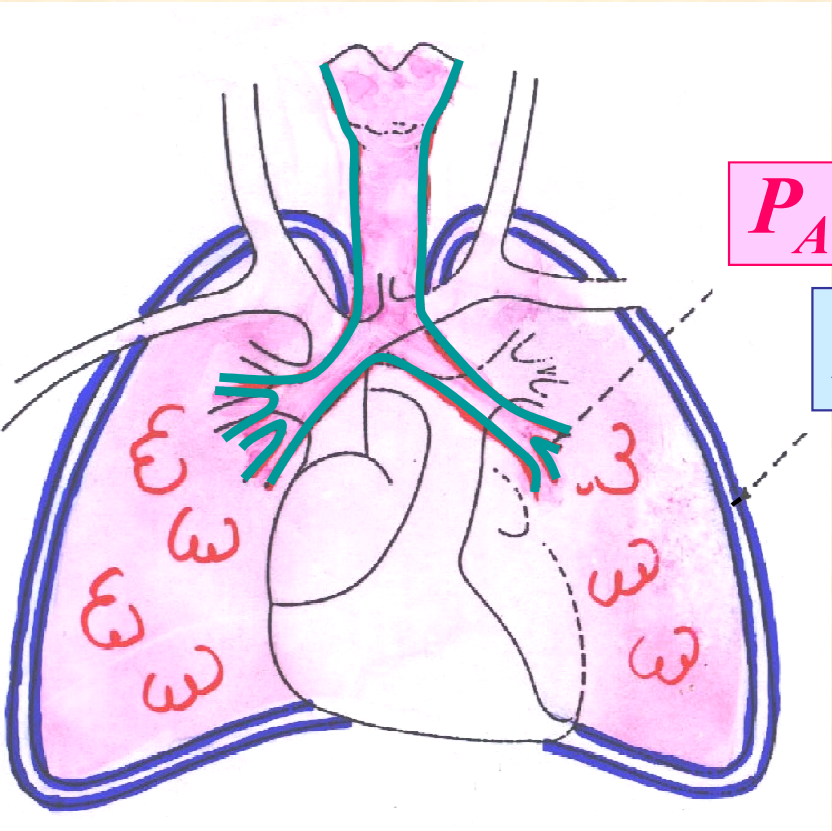
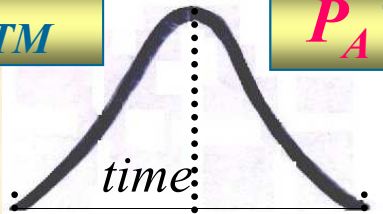
VI TRANSPORT OF GASSES (O_2 and CO_2)

$$P \cdot V = \text{const} \rightarrow P = \frac{\text{const}}{V}$$

TIME COURSE OF PRESSURES AT QUIET RESPIRATION

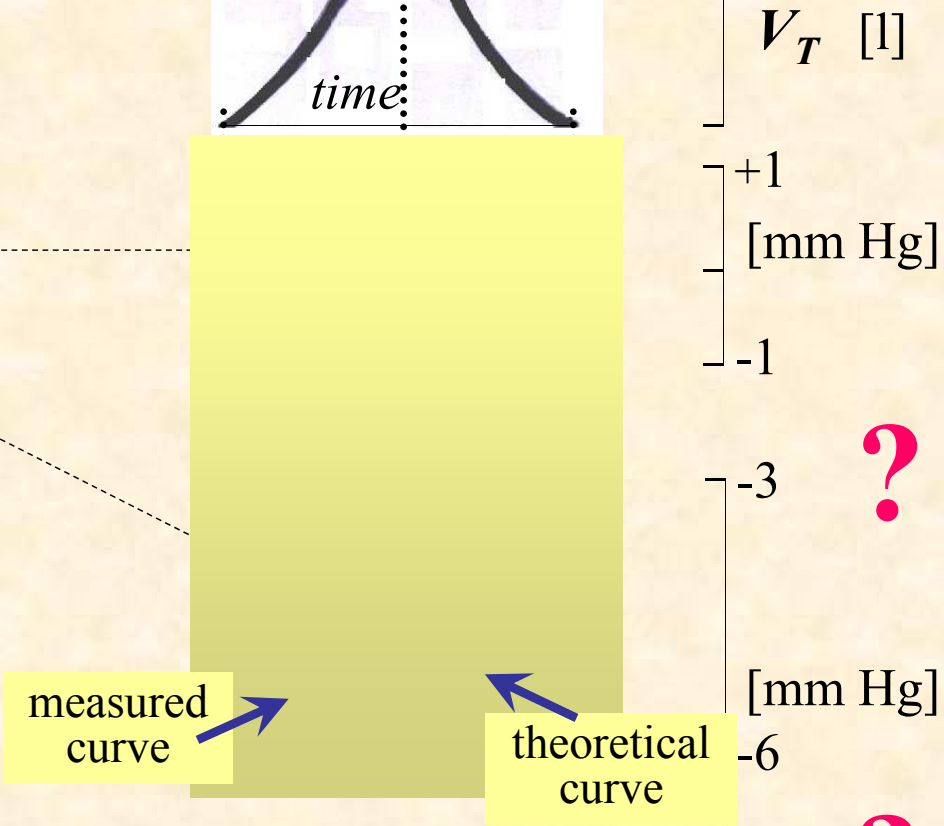
INSPIRATION EXPIRATION

$P_A < P_{ATM}$ $P_A > P_{ATM}$



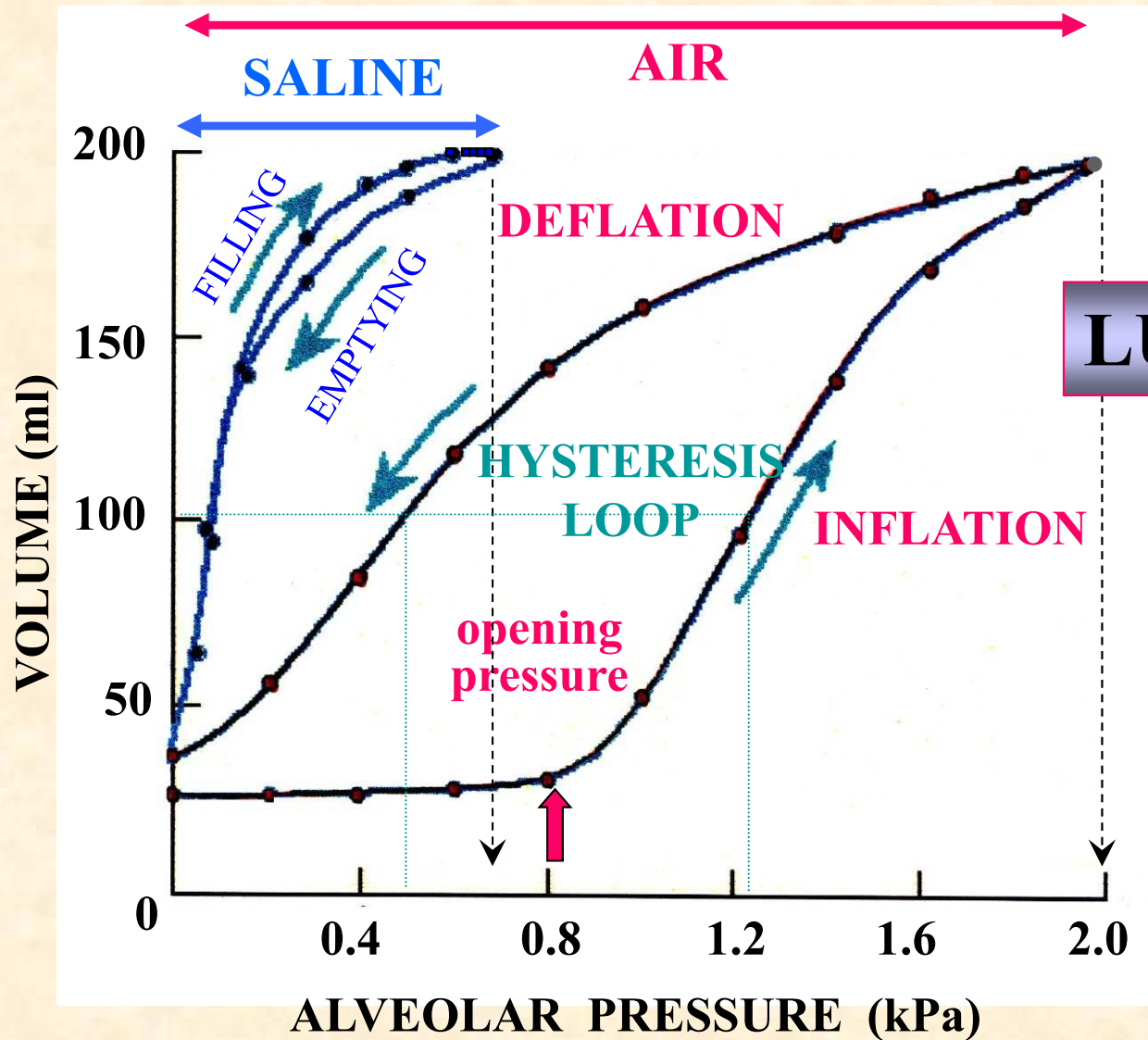
P_A

P_{PL}



P_A ALVEOLAR (INTRAPULMONARY, LUNG)

P_{PL} INTRAPLEURAL (INTRATHORACIC)



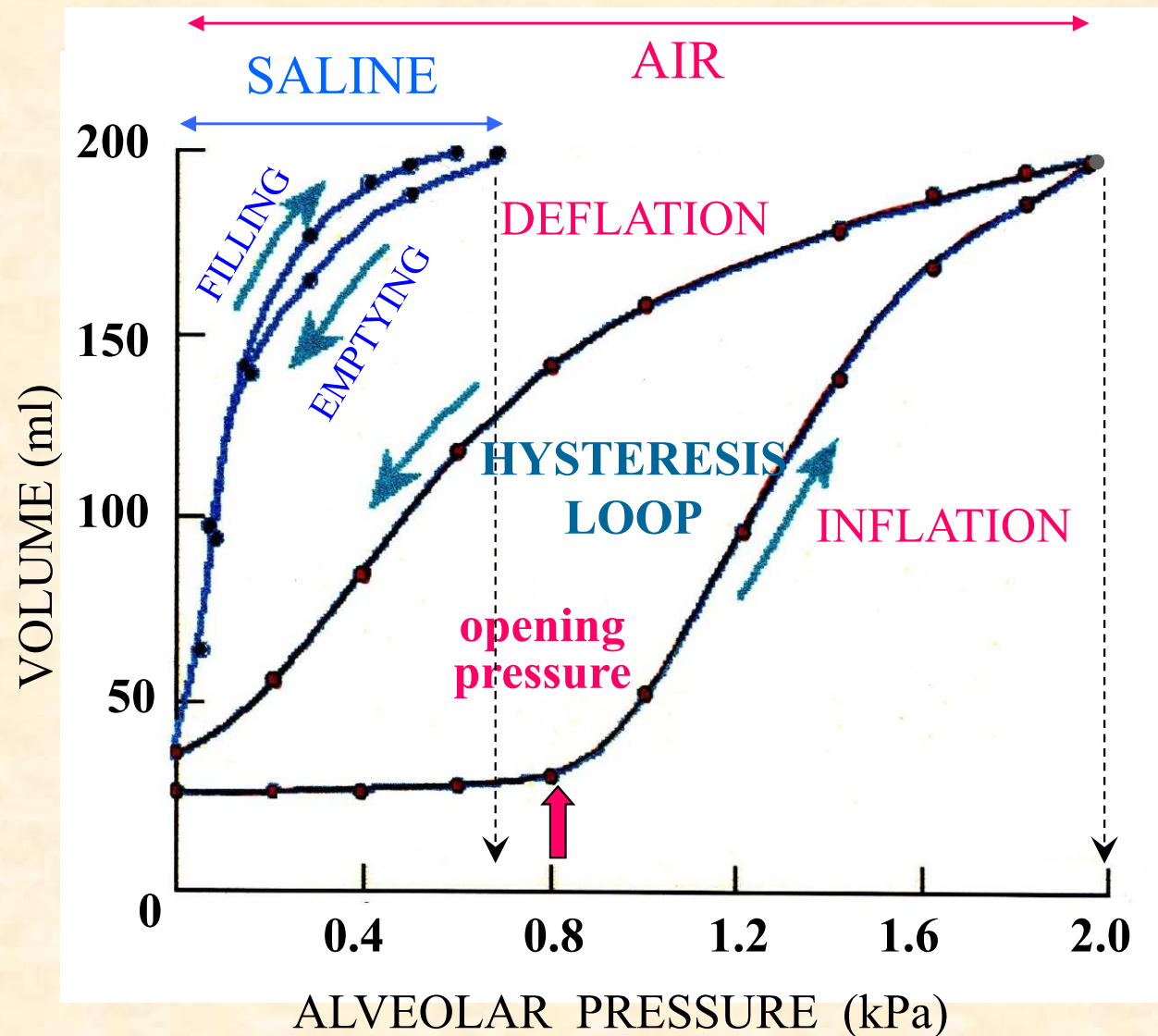
LUNGS ELASTICITY

1 kPa = 7.5 mm Hg

LUNGS ELASTICITY

INHERENT TISSUE ELASTICITY
(elastin and collagen fibres)

SURFACE TENSION FORCES
air-liquid interface in alveoli



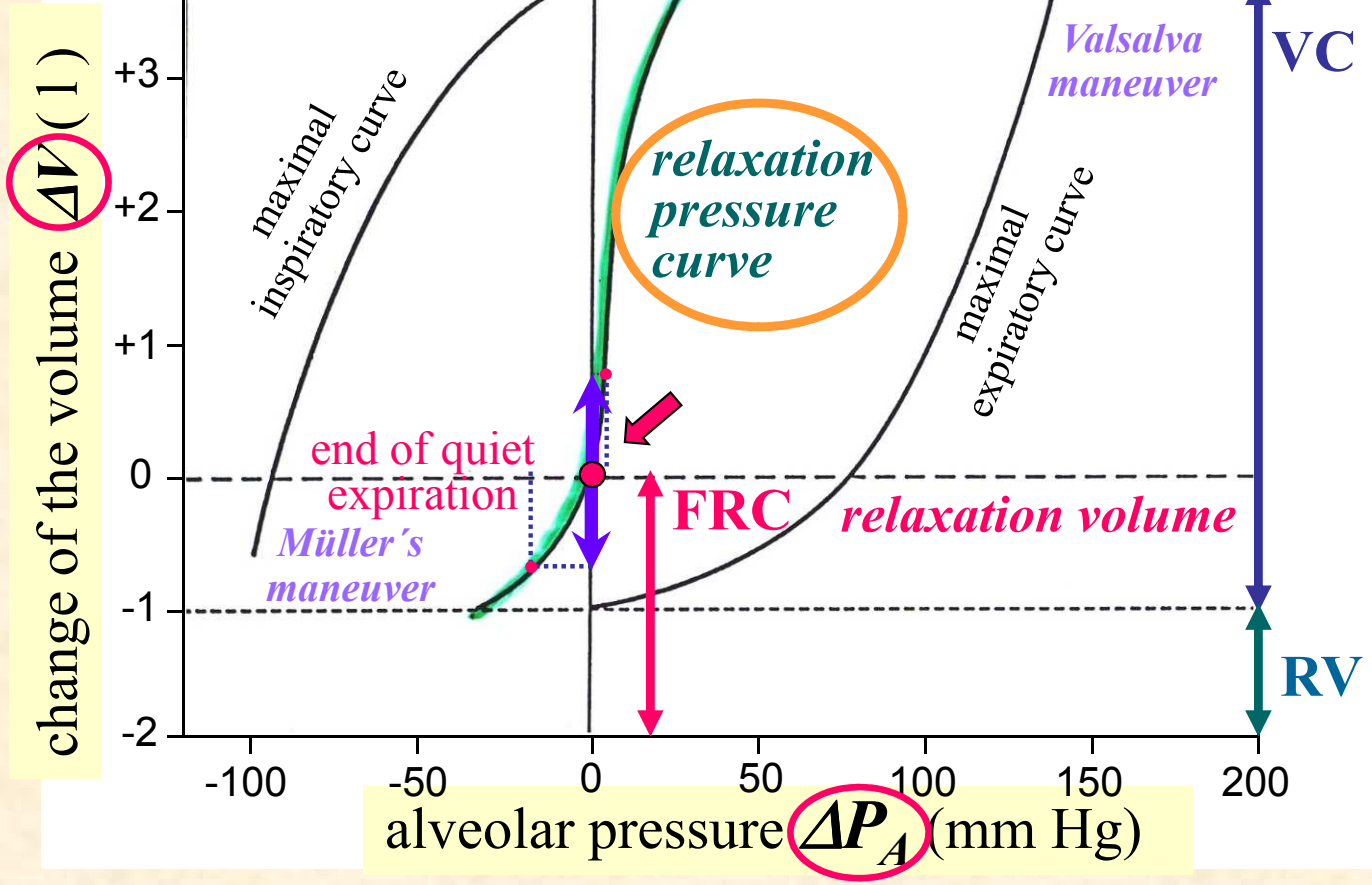
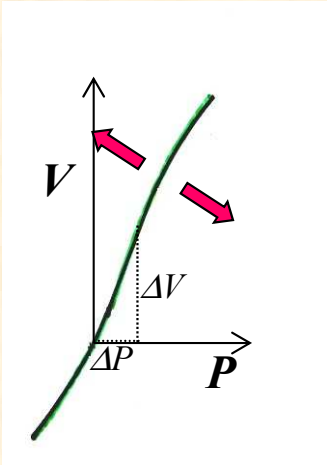
Factors involved in HYSTERSIS LOOP

- **LAPLACE LAW** (responsible for high **opening pressure** of alveoli)
- **Dynamic changes in the DENSITY** of surfactant molecules during **INSPIRATION** and **EXPIRATION**

COMPLIANCE (VOLUME STRETCHABILITY)

STATIC MEASUREMENT IN CLOSED SYSTEM

$$C = \frac{\Delta V}{\Delta P}$$



TOTAL RESPIRATORY SYSTEM
(lungs and chest)

compliance is decreased
↑ *stiffness of the tissue*

compliance is increased
↓ *stiffness of the tissue*

TOTAL WORK OF RESPIRATORY MUSCLES AT QUIET BREATHING

ELASTIC (STATIC) WORK (65%)

to overcome the elastic forces of the chest and lungs

DYNAMIC WORK (35%)

- to overcome the resistance of air passages during the air movement – **AERODYNAMIC RESISTANCE** (~ 28%)
- to overcome the friction during mutual movement of inelastic tissues – **VISCOUS RESISTANCE** (~ 7%)

I AIR PASSAGES

II MEASURABLE PARAMETERS

- DEAD SPACE
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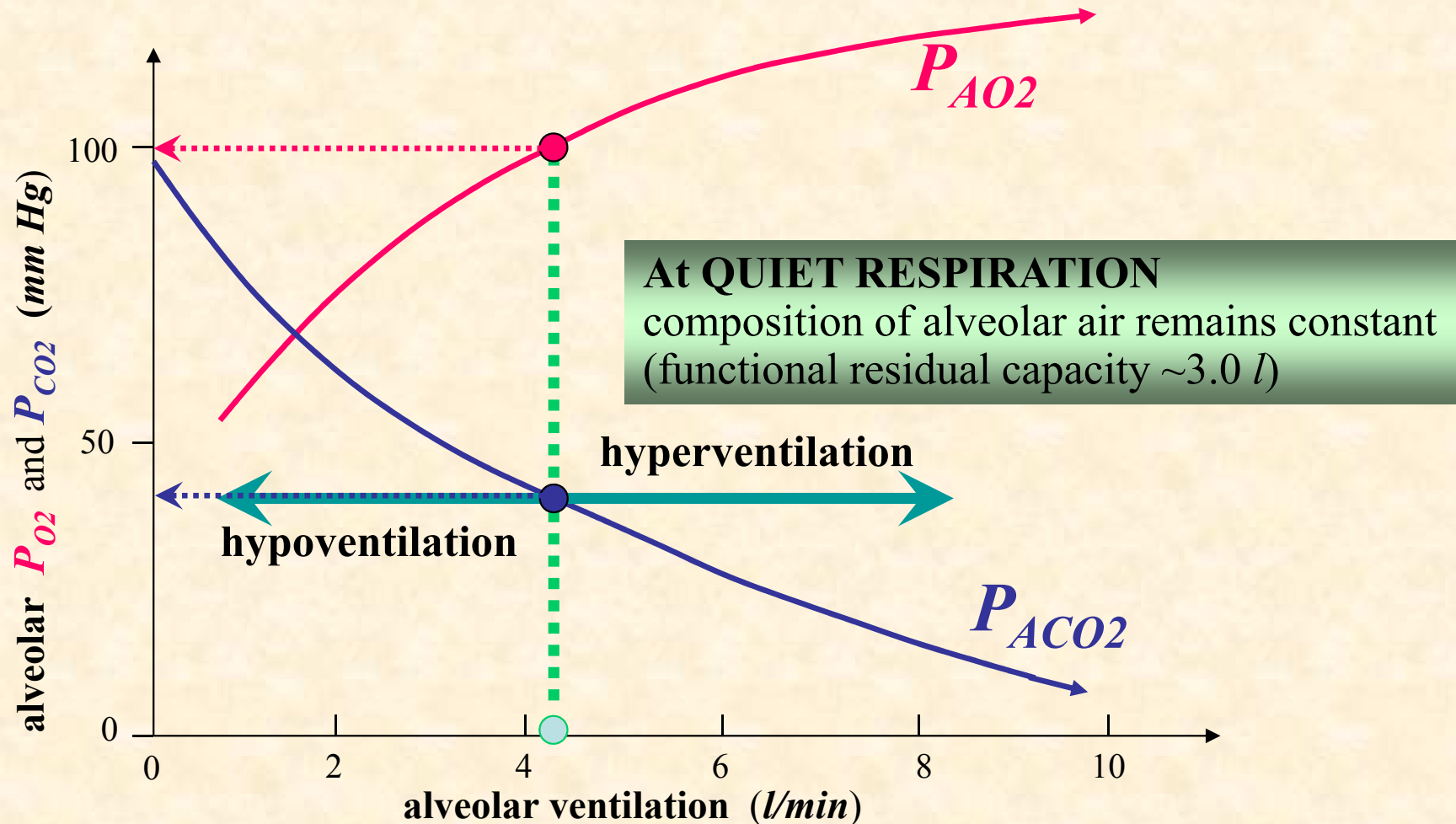
- RESPIRATORY MUSCLES
- LUNGS ELASTICITY
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- WORK OF BREATHING

➔ IV COMPOSITION OF ALVEOLAR AIR

V ALVEOLAR-CAPILLARY MEMBRANE

VI TRANSPORT OF GASSES (O_2 and CO_2)

Alveolar P_{O_2} and P_{CO_2} at voluntary hypo- and hyperventilation



hyperventilation \rightarrow hypocapnia \rightarrow respiratory alkalosis

hypoventilation \rightarrow hypercapnia \rightarrow respiratory acidosis