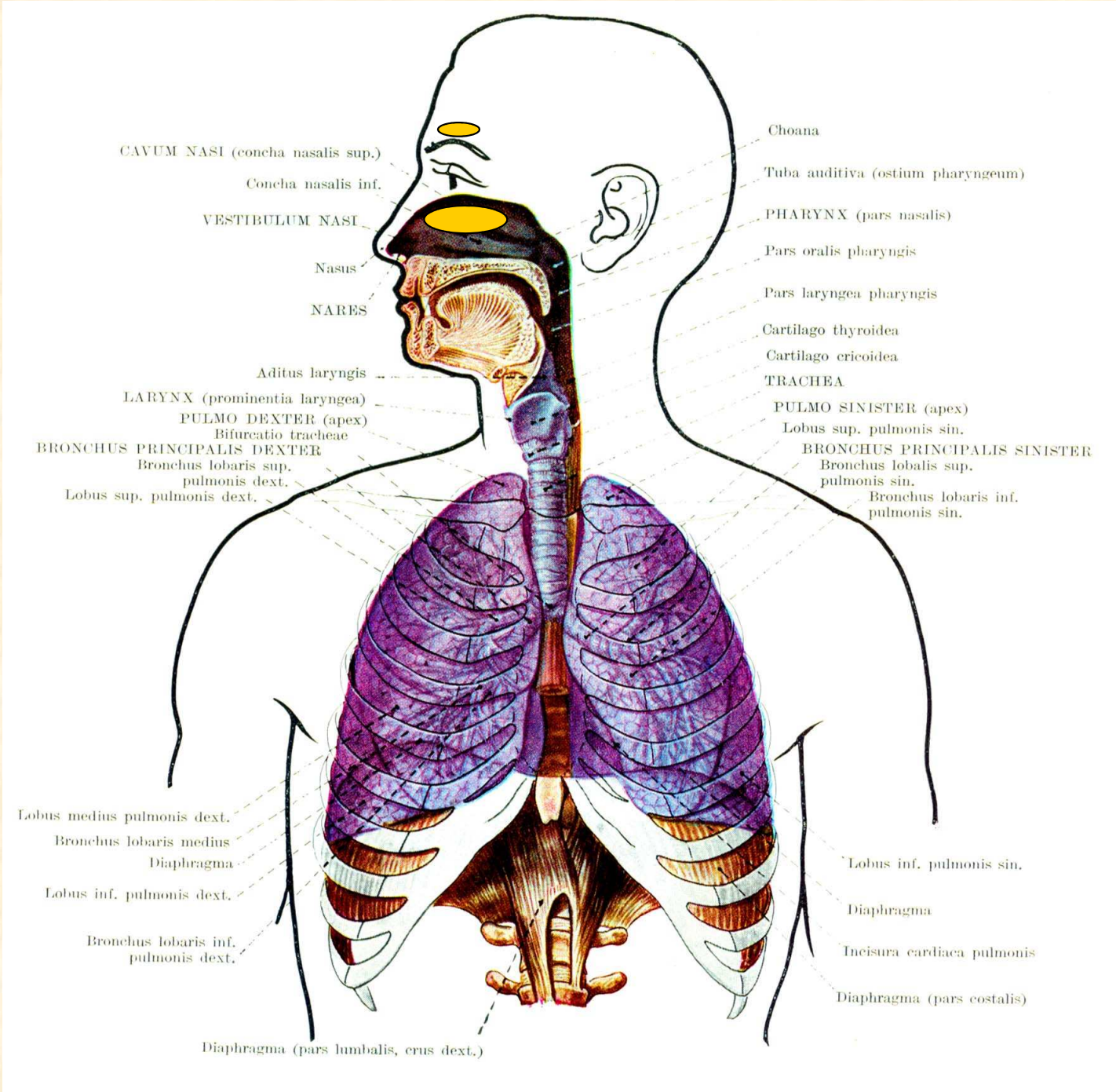
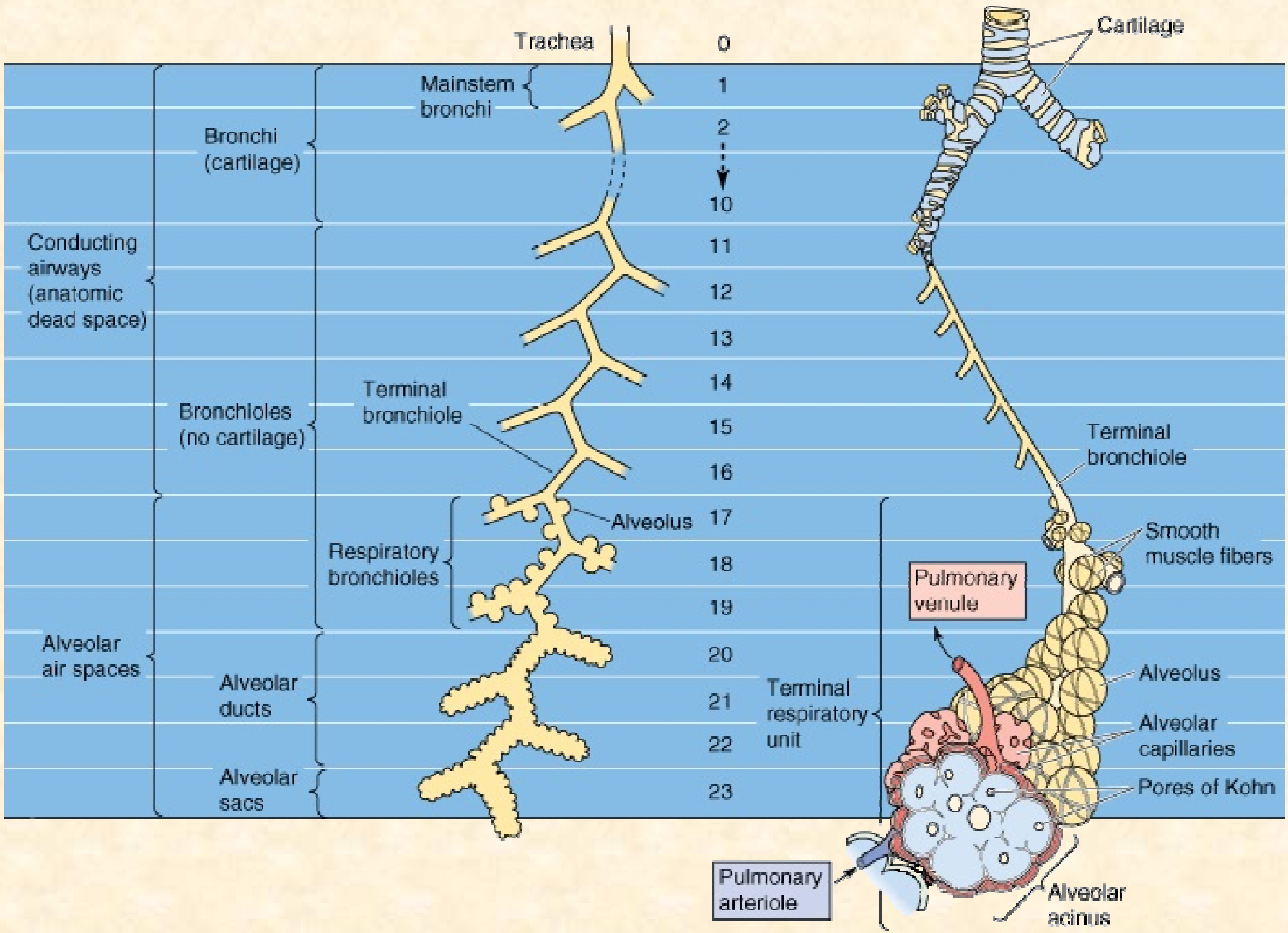
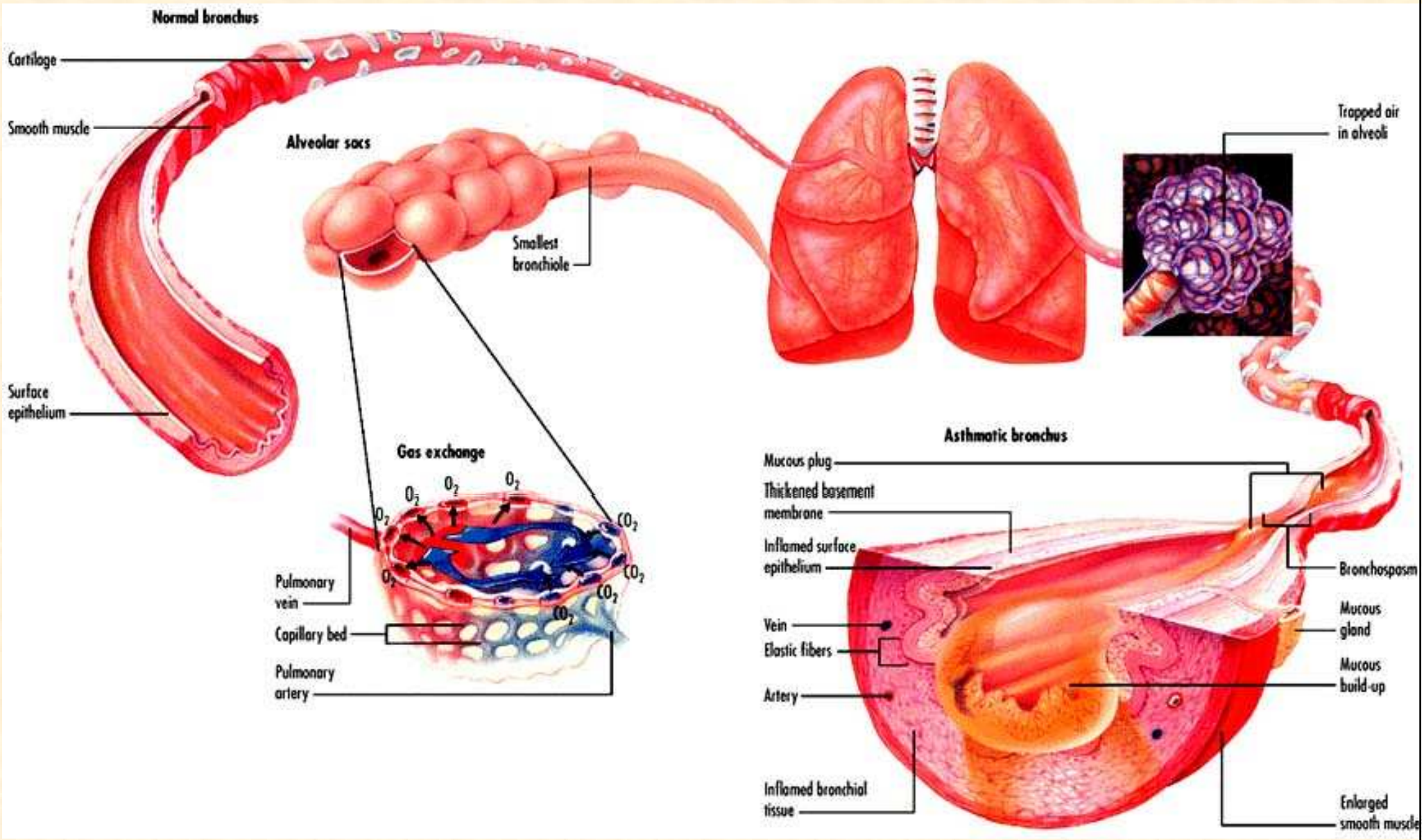
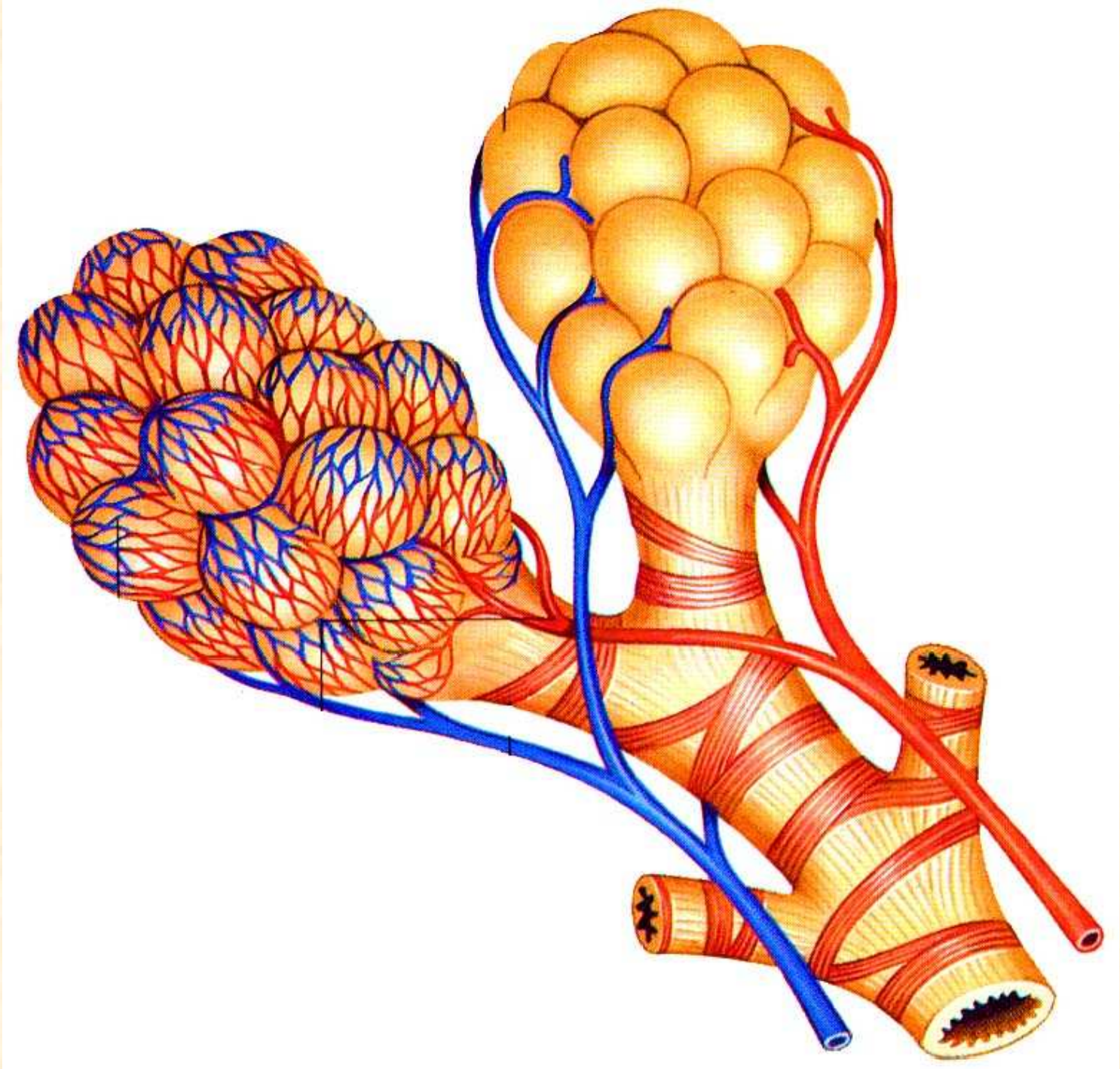
# RESPIRATORY SYSTEM

## RESPIRATORY FUNCTIONS
## MECHANICS OF RESPIRATORY SYSTEM
## GAS TRANSPORT


CAVUM NASI (concha nasalis sup.)
Concha nasalis inf.
VESTIBULUM NASI
Nasus
NARES
Aditus laryngis
LARYNX (prominentia laryngea)
PULMO DEXTER (apex)
Bifurcatio tracheae
BRONCHUS PRINCIPALIS DEXTER
Bronchus lobaris sup. pulmonis dext.
Lobus sup. pulmonis dext.
Lobus medius pulmonis dext.
Bronchus lobaris medius
Diaphragma
Lobus inf. pulmonis dext.
Bronchus lobaris inf. pulmonis dext.
Diaphragma (pars lumbalis, crus dext.)
Choana
Tuba auditiva (ostium pharyngeum)
PHARYNX (pars nasalis)
Pars oralis pharyngis
Pars laryngea pharyngis
Cartilago thyroidea
Cartilago cricoidea
TRACHEA
PULMO SINISTER (apex)
Lobus sup. pulmonis sin.
BRONCHUS PRINCIPALIS SINISTER
Bronchus lobalis sup. pulmonis sin.
Bronchus lobaris inf. pulmonis sin.
Lobus inf. pulmonis sin.
Diaphragma
Incisura cardiaca pulmonis
Diaphragma (pars costalis)


Trachea
0
Mainstem bronchi
1
Bronchi (cartilage)
2
10
Conducting airways (anatomic dead space)
11
12
13
Terminal bronchiole
14
Bronchioles (no cartilage)
15
16
Alveolus
17
Respiratory bronchioles
18
19
Alveolar air spaces
20
Alveolar ducts
21
22
Alveolar sacs
23
Cartilage
Terminal bronchiole
Smooth muscle fibers
Pulmonary venule
Terminal respiratory unit
Alveolus
Alveolar capillaries
Pores of Kohn
Pulmonary arteriole
Alveolar acinus

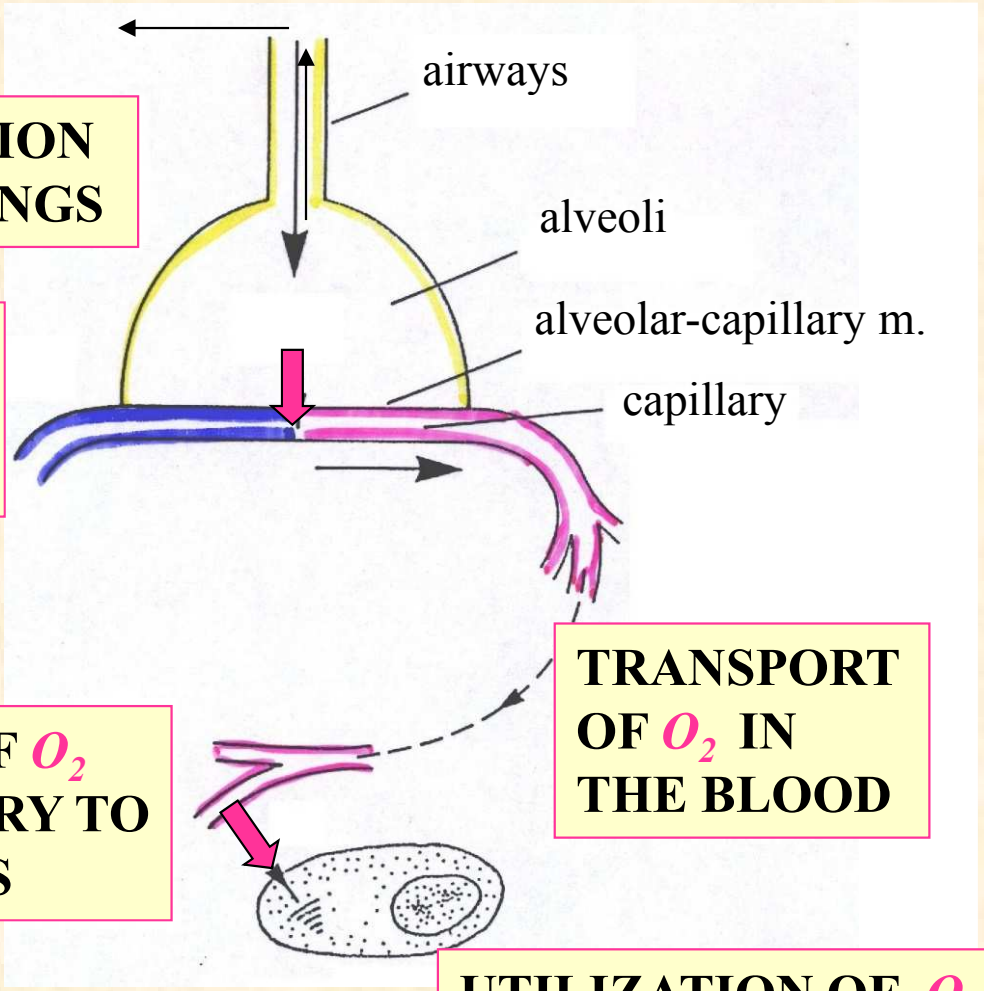

Normal bronchus
Cartilage
Smooth muscle
Surface epithelium
Alveolar sacs
Smallest bronchiole
Gas exchange
$O_2$
$CO_2$
Pulmonary vein
Capillary bed
Pulmonary artery
Trapped air in alveoli
Asthmatic bronchus
Mucous plug
Thickened basement membrane
Inflamed surface epithelium
Bronchospasm
Vein
Elastic fibers
Artery
Mucous gland
Mucous build-up
Inflamed bronchial tissue
Enlarged smooth muscle



# STEPS IN THE DELIVERY OF $O_2$ TO THE CELLS

**VENTILATION OF THE LUNGS**

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




**TRANSPORT OF $O_2$ IN THE BLOOD**

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

**UTILIZATION OF $O_2$ BY MITOCHONDRIA**

AT REST

$O_2$ UPTAKE ~300 ml / min

$CO_2$ OUTPUT ~250 ml / min

INTERNAL RESPIRATION

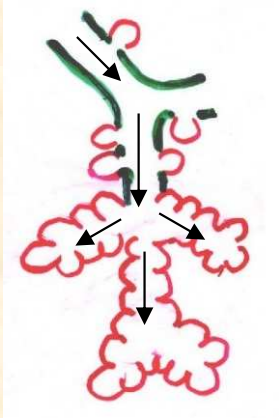
# AIR PASSAGES

## ANATOMICAL DEAD SPACE –CONDUCTING ZONE

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

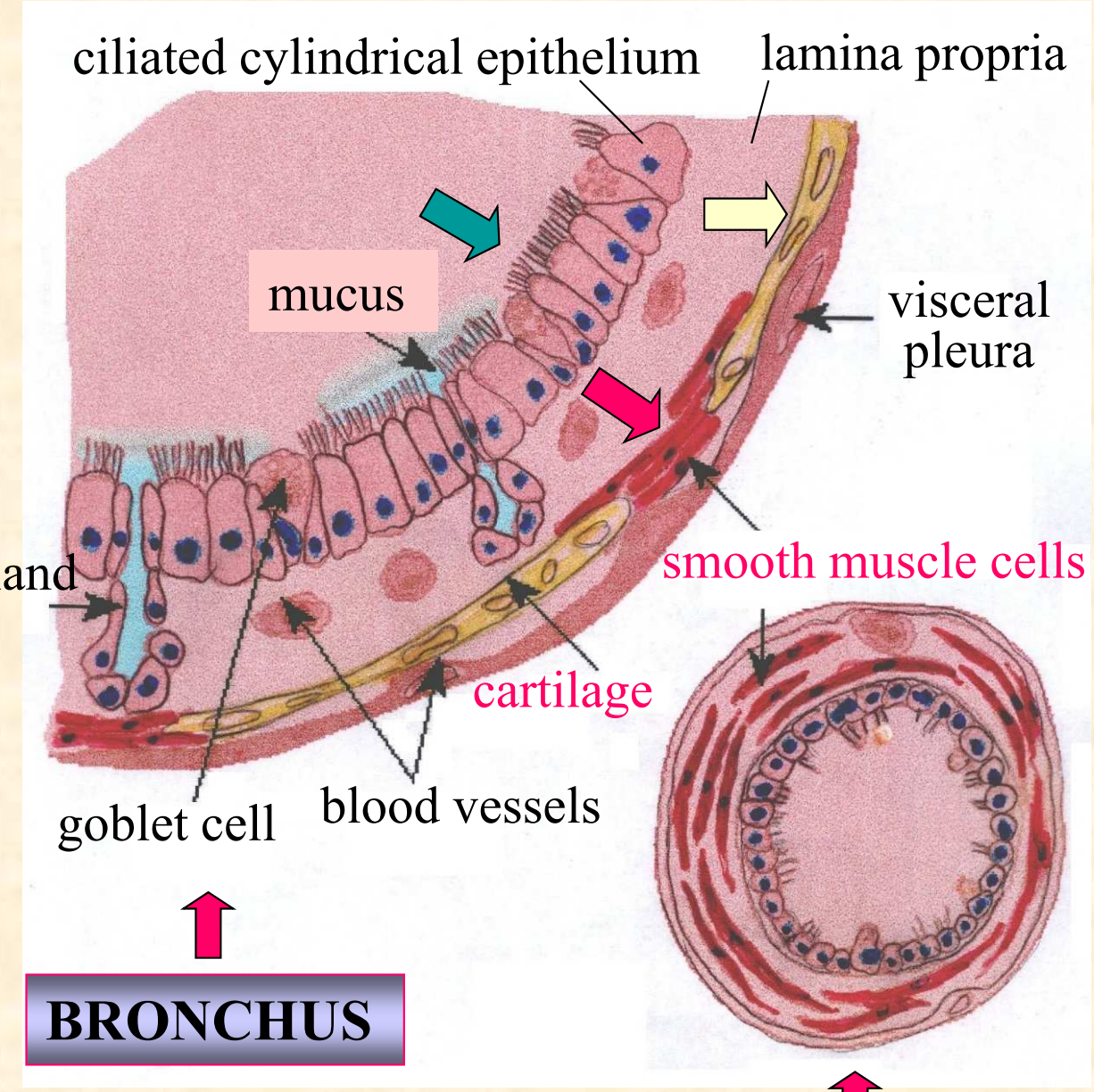
Other physiological functions:

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



## RESPIRATORY ZONE
**(GAS EXCHANGE)**

**Total alveolar area ~100 $m^2$**




**BRONCHUS**

**TERMINAL BRONCHIOLE**

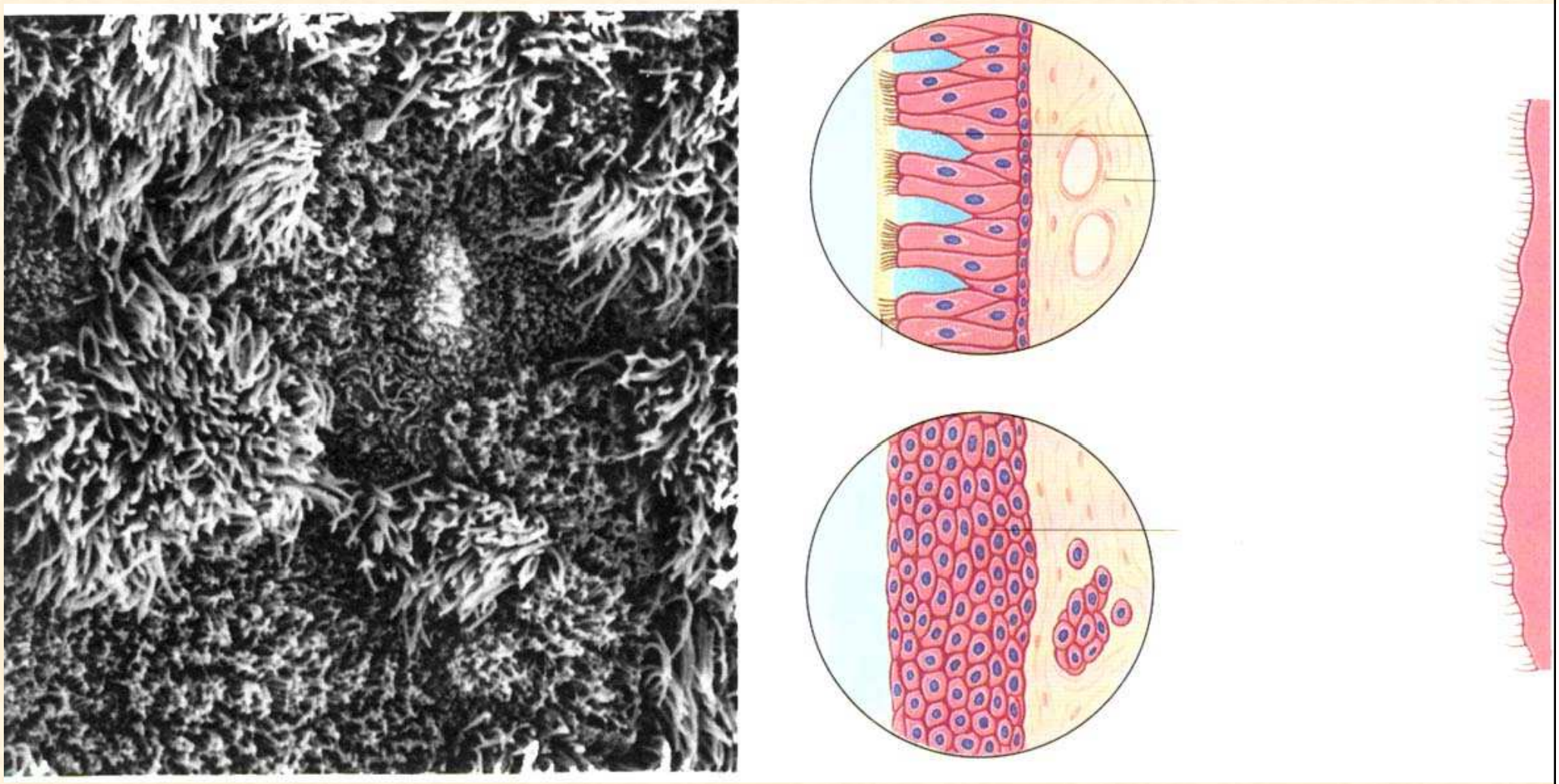
∅ < 1 mm

## 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**

**$\beta_2$-adrenergic receptors: Noradrenaline** activates bronchodilatation



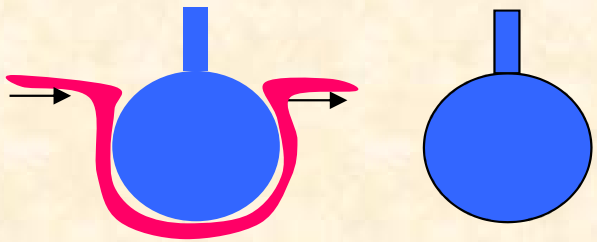
Cylindrical epithelium with cilia

# 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/min

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

**PULMONARY MINUTE VENTILATION**

6 l/min

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

**ALVEOLAR VENTILATION**

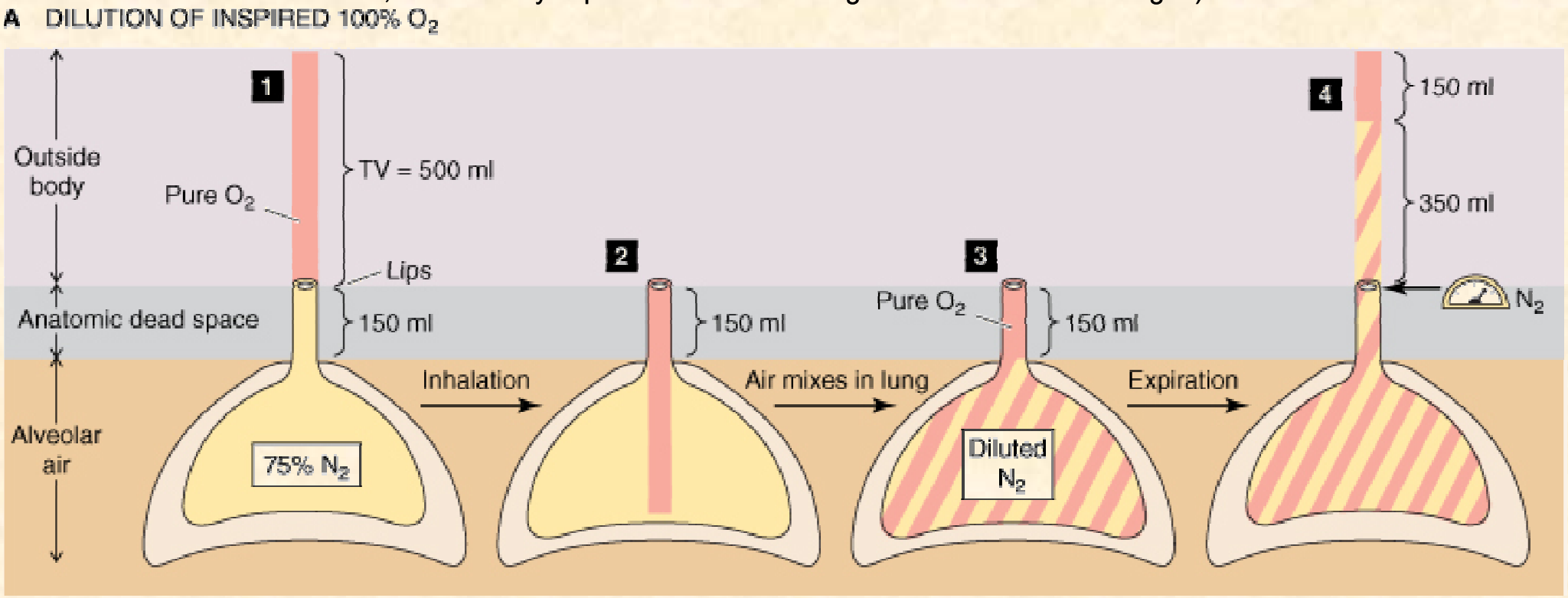
4.2 l/min

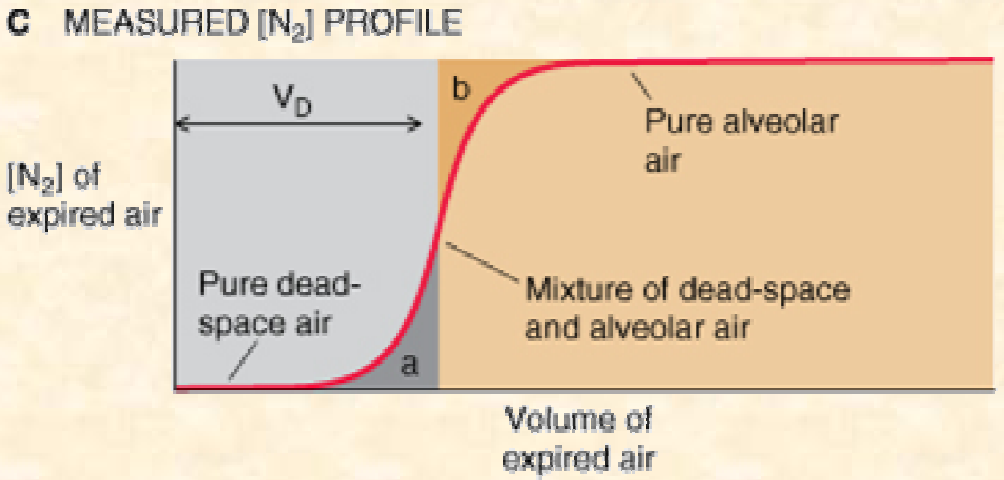
$\dot{V}_D = V_D \times f$

**DEAD SPACE VENTILATION**

1.8 l/min

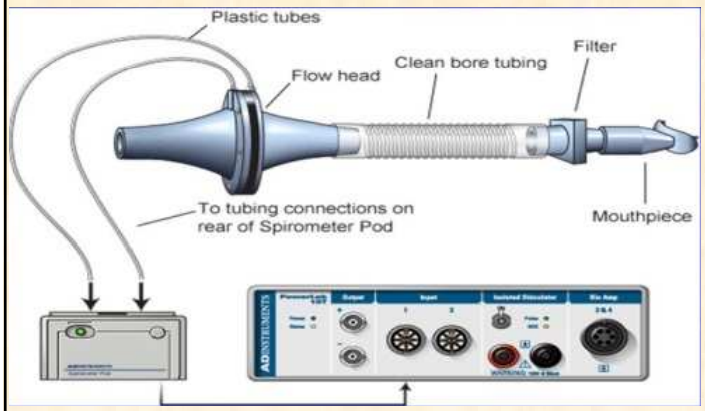
# DEAD SPACE – nitrogen test (force inspiration of pure O2, follow slowly expiration with monitoring of concentration of nitrogen)

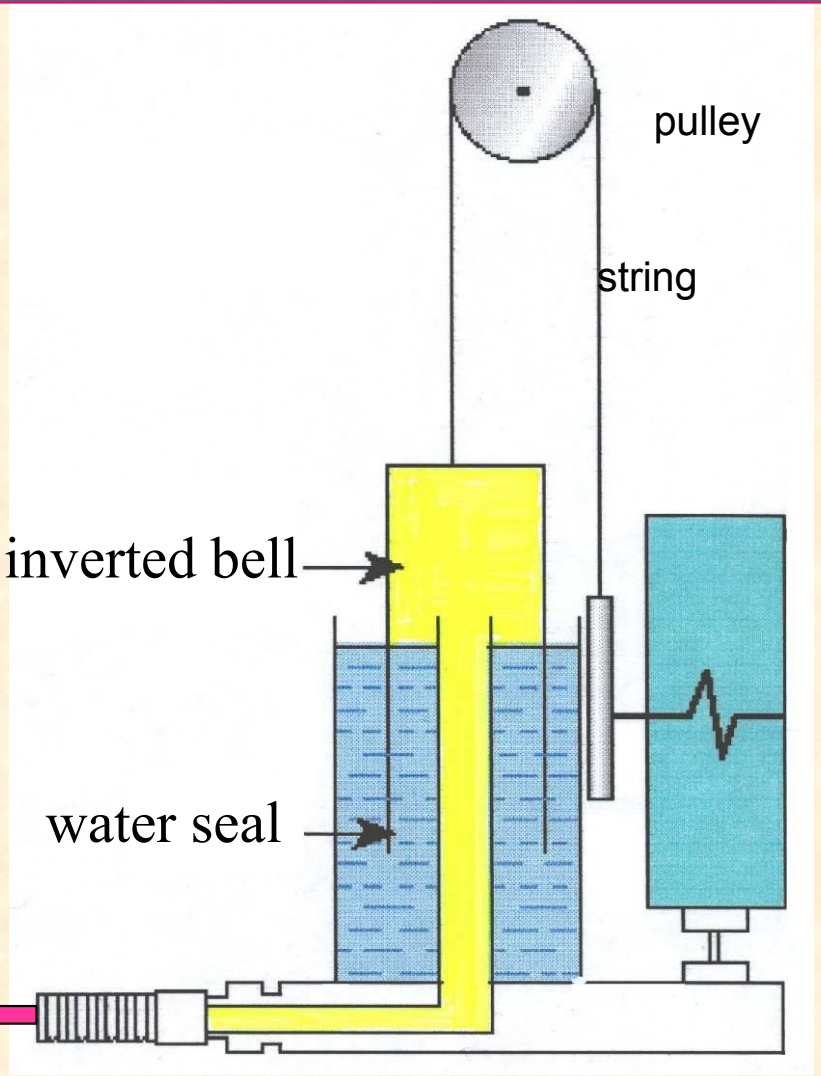


# SPIROMETRY

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




inspiration

expiration

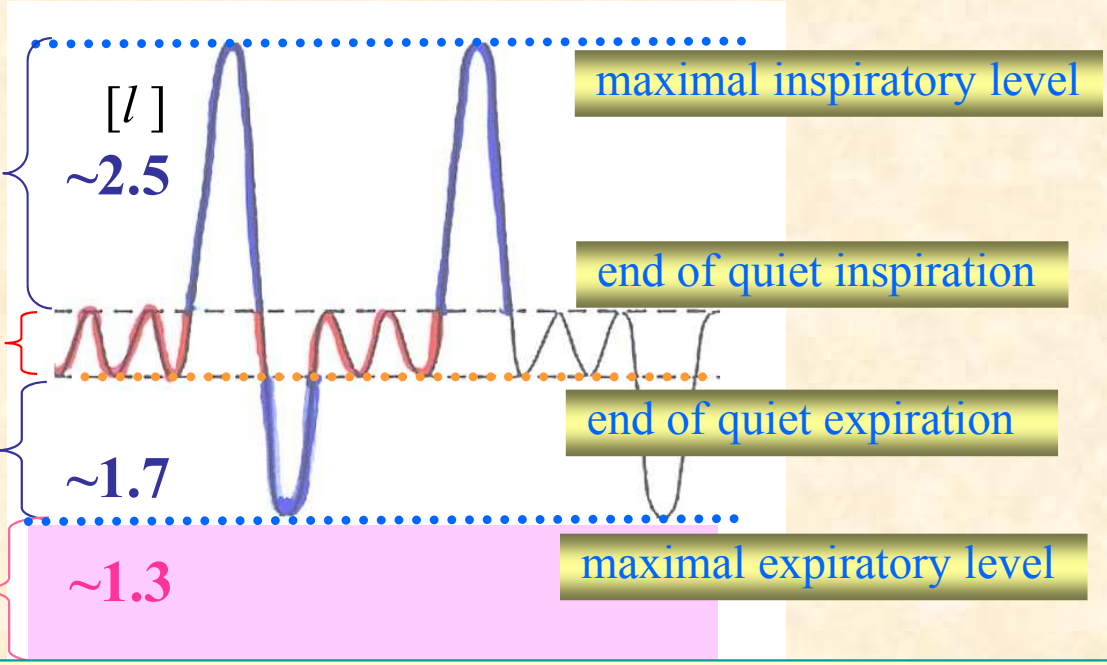
# LUNG VOLUMES

**INSPIRATORY RESERVE VOLUME *IRV***

**TIDAL VOLUME $V_T$**

**EXPIRATORY RESERVE VOLUME *ERV***

**RESIDUAL VOLUME *RV***

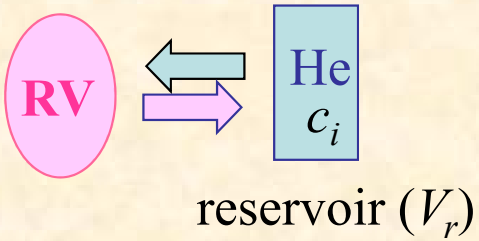



**DILUTION METHOD *He***

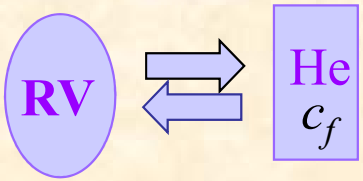
*Principle of method*: **1** Maximal expiration, **2** Repeated inspiration from and expiration into a reservoir (known **volume** $V_r$) with inert gas ***He*** (known concentration $c_i$)

⇒ 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 ($c_i$ , $c_f$).

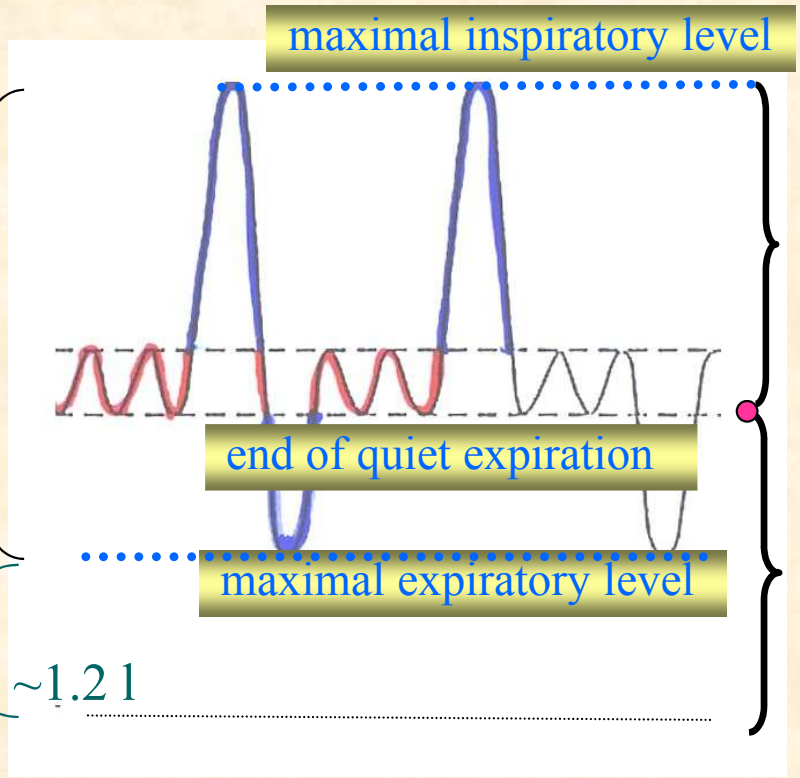



reservoir ($V_r$)




reservoir (V)

$$RV = V_r \frac{c_{i\,He} - c_{f\,He}}{c_{f\,He}}$$




**TLC**

**VC**

**RV**

**INSPIRATORY CAPACITY**

>3.0 l

**FUNCTIONAL RESIDUAL CAPACITY**

<3.0 l

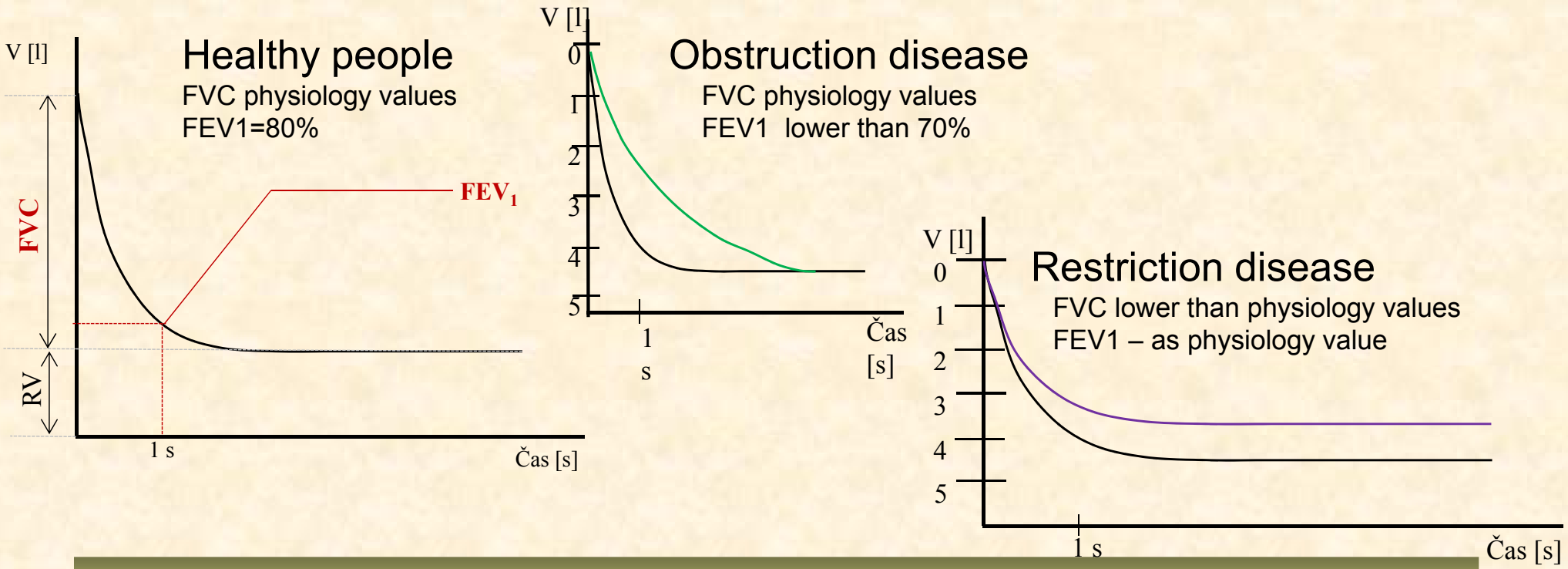
**VC** **VITAL CAPACITY = $V_T$ + IRV + ERV** ~ 4.7 l

**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)**

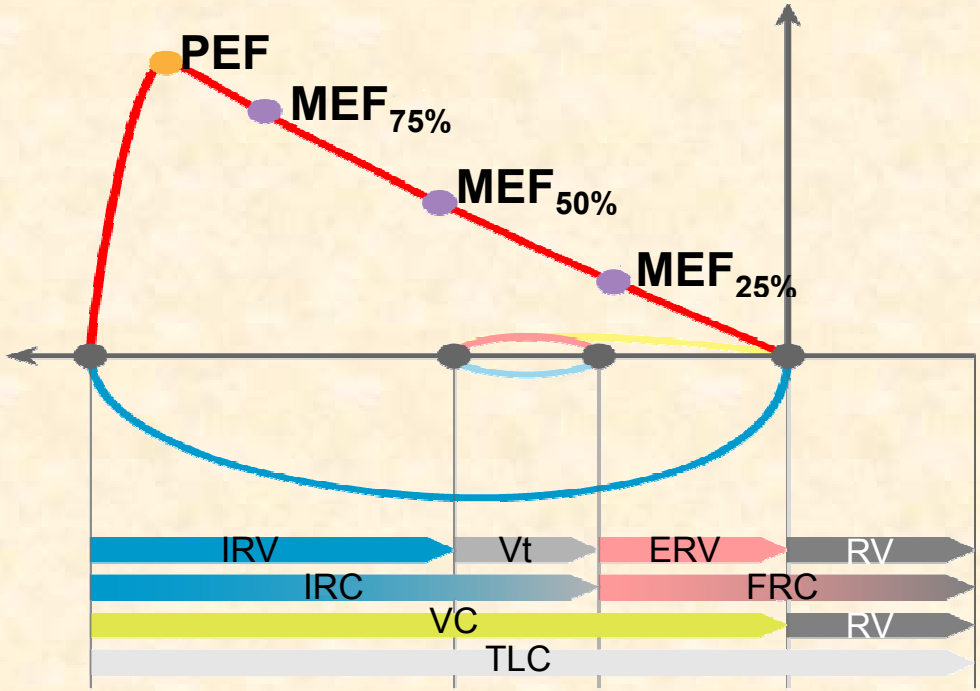



- **PULMONARY MINUTE VENTILATION *RMV* (respiratory minute volume) at rest** (0.5 l x 12 breathes/min = 6 l/min)
- **MAXIMAL VOLUNTARY VENTILATION (*MVV*)** (125-170 l/min)
- **PEAK EXPIRATORY FLOW RATE (*PEFR*)** (~10 l/s)

Tady jsem skončila

# Flow – volume curve

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



# PNEUMOGRAPHY

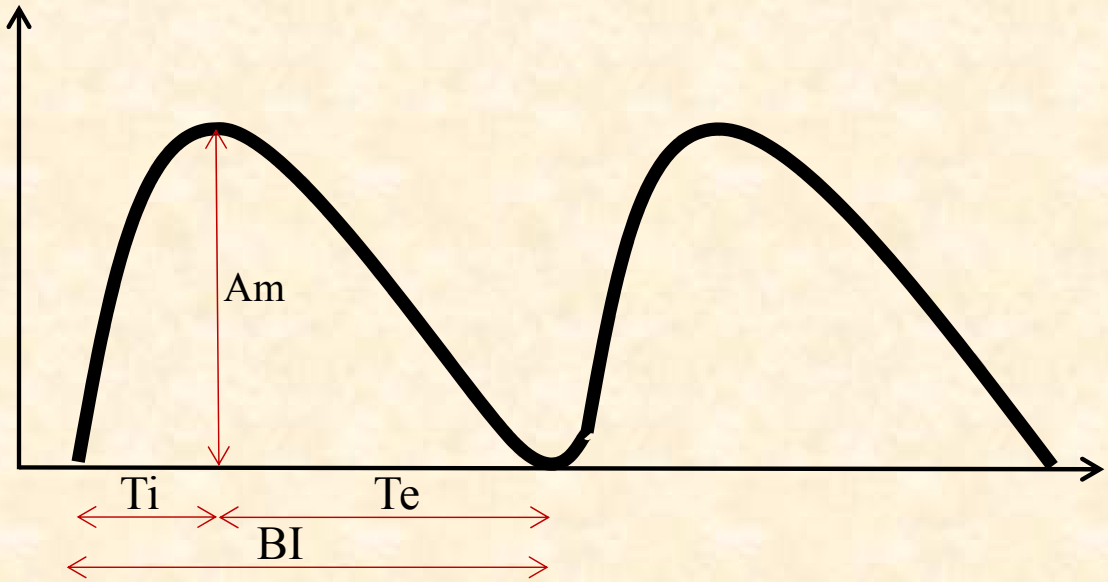
***Principle***

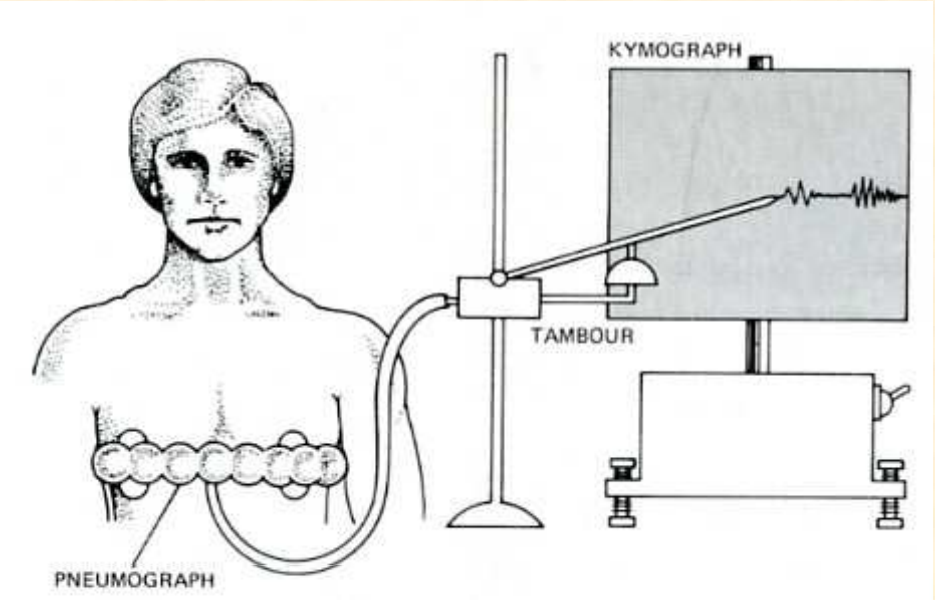
Pneumography – measurement of respiratory movements (via chest or abdomen)

- respiratory belt (piesoelectrical 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 –Ti, Te, BI a Am

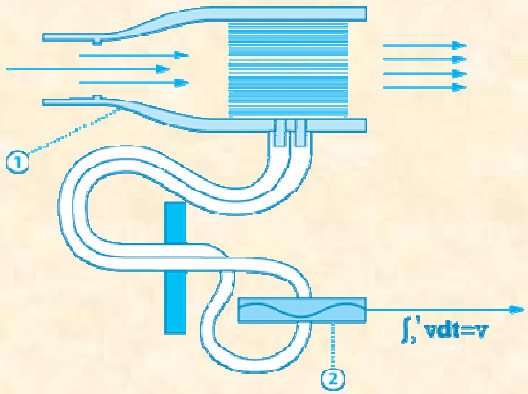


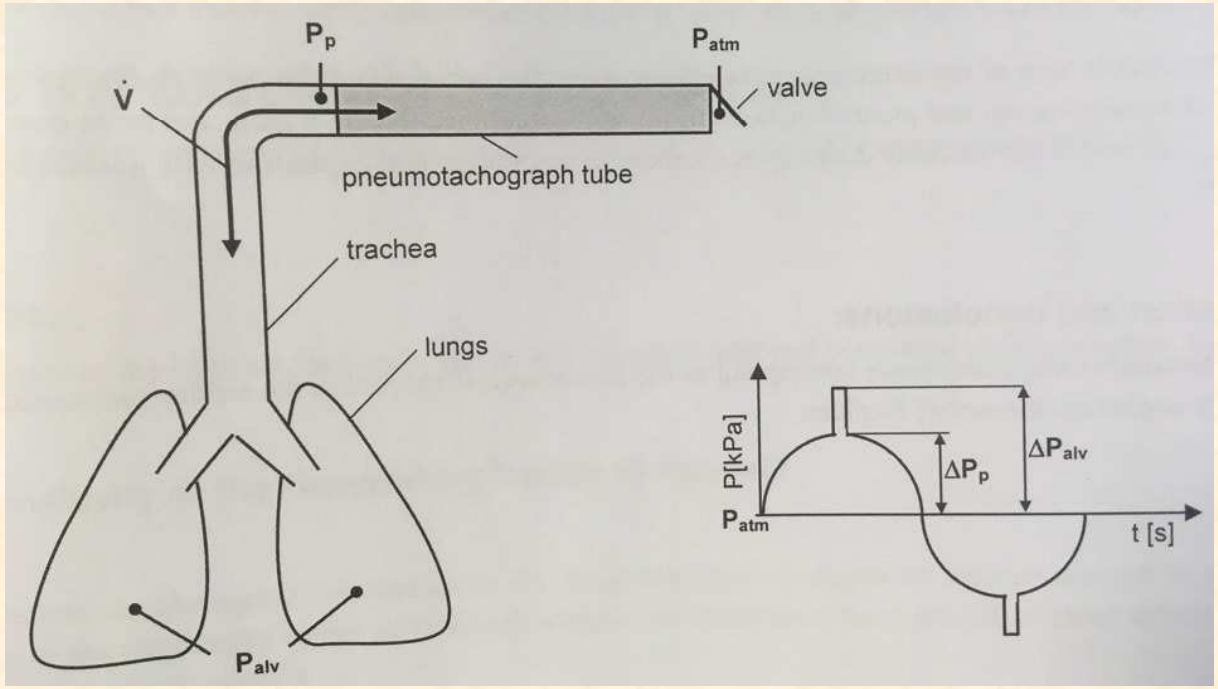



# PNEUMOTACHOGRAPHY

## *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.**




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

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

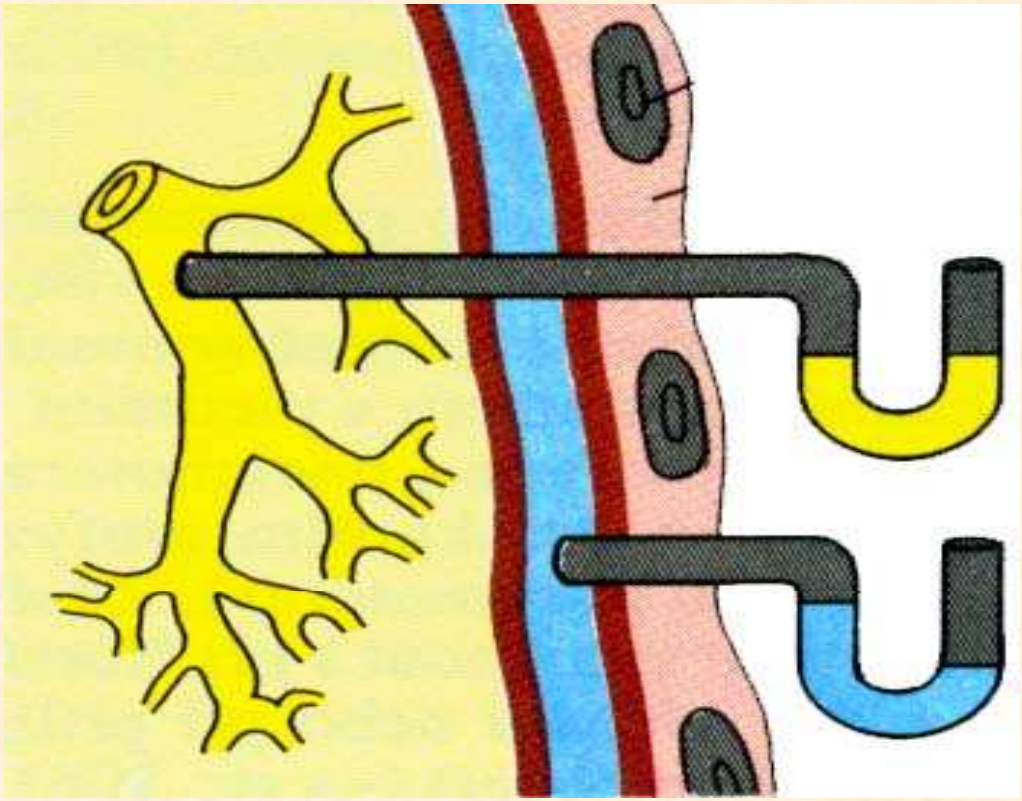
$$\frac{P_p - P_{atm}}{R_p} = \dot{V} = \frac{P_{alv} - P_p}{R_d}$$

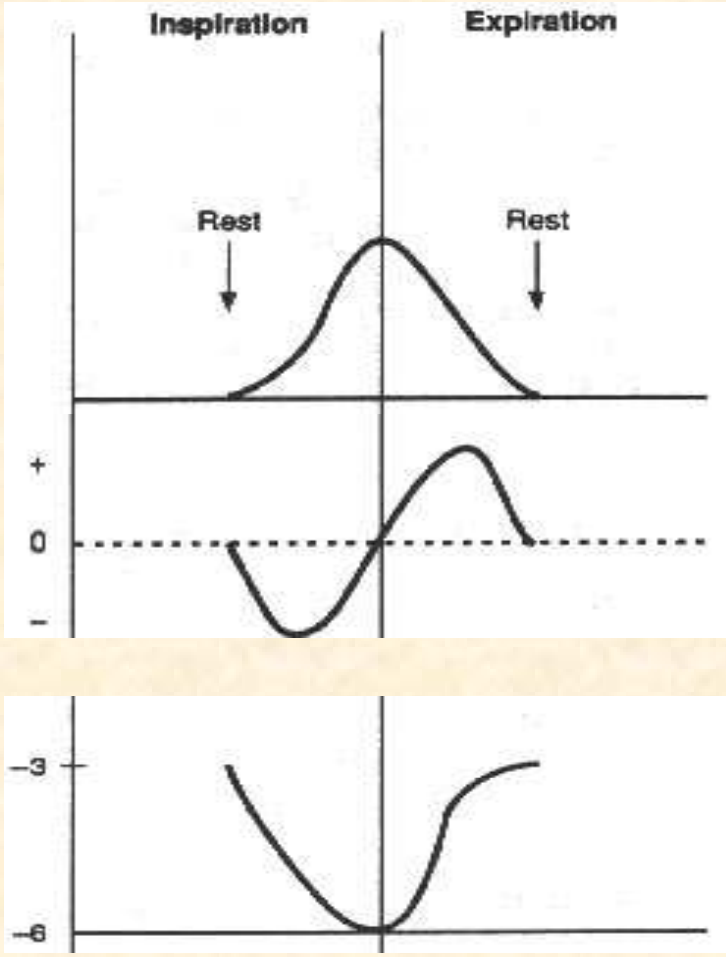
$$R_d = R_p \cdot \left( \frac{\Delta P_{alv}}{\Delta P_p} - 1 \right)$$

- Mechanics of breathing

PLEURA

pulmonalis parietalis





# FORCES PARTICIPATING IN RESPIRATION

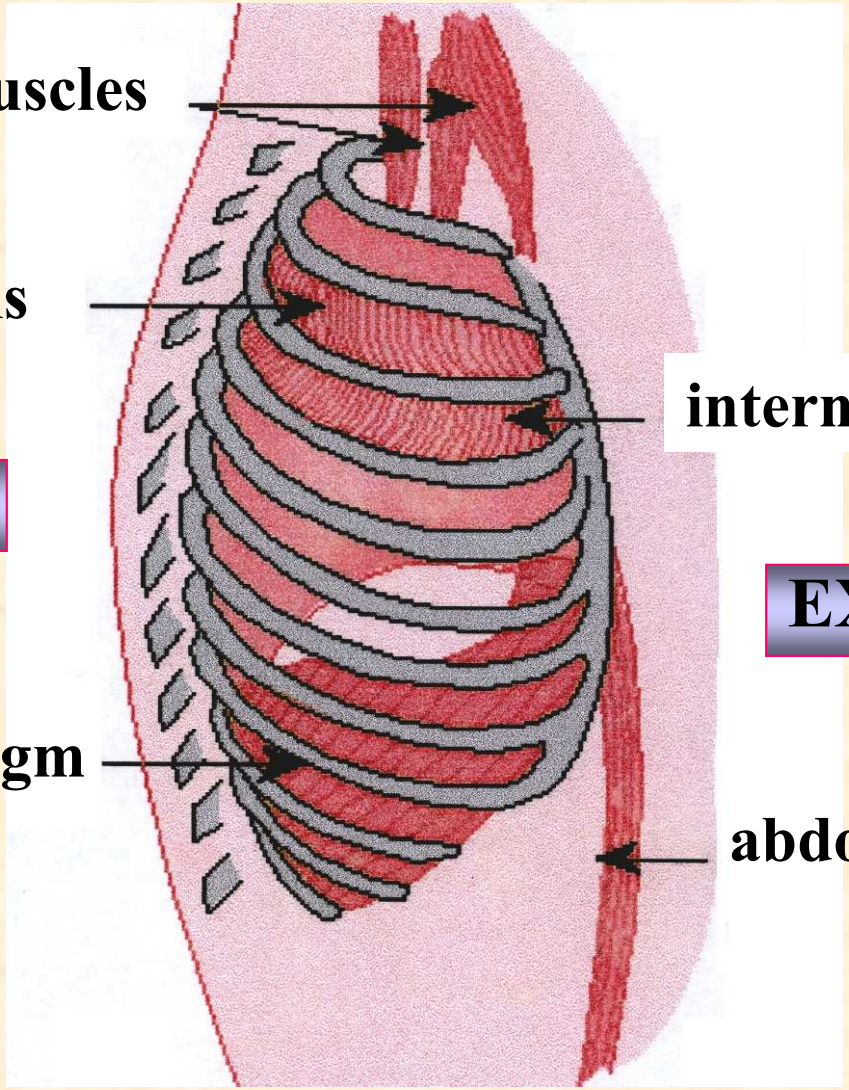
- **ACTIVE FORCES** performed by respiratory muscles
- **PASSIVE FORCES represented by:**
  - **lungs elasticity**
  - **chest elasticity**

## 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

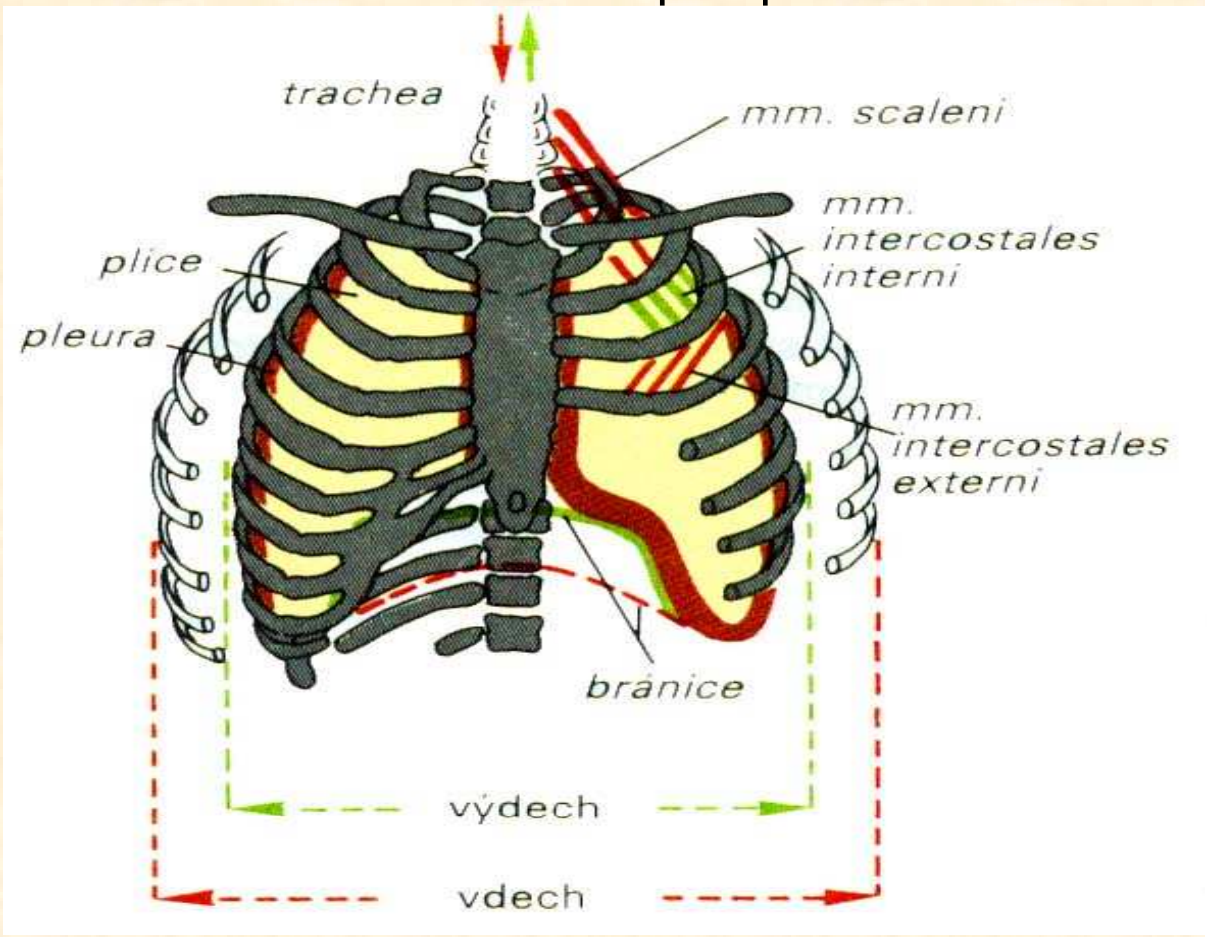
- ***accessory*** **inspiratory muscles** (mm. scalene)

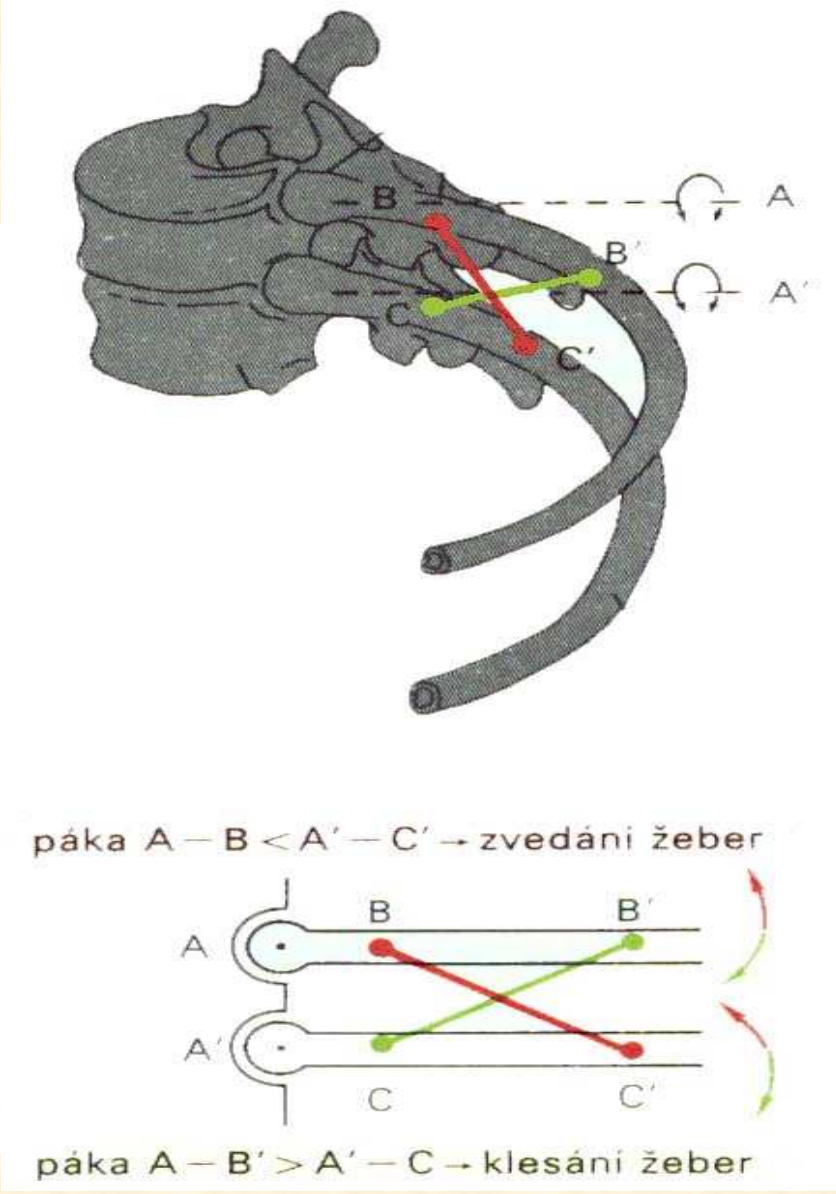
## EXPIRATORY muscles

### Only at FORCED breathing

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

Bucket-handle and water-pump handle effects



## COMPOSITION OF DRY ATMOSPHERIC AIR

| | | | |
|---|---|---|---|
| $O_2$ | 20.98 % | $F_{O2}$ | $\cong 0.21$ |
| $N_2$ | 78.06 % | $F_{N2}$ | $\cong 0.78$ |
| $CO_2$ | 0.04 % | $F_{CO2}$ | $= 0.0004$ |

Other constituents

## BAROMETRIC (ATMOSPHERIC) PRESSURE AT SEA LEVEL

1 atmosphere = 760 mm Hg

## PARTIAL PRESSURES OF GASSES IN DRY AIR AT SEA LEVEL

$P_{O2}$ = 760 x 0.21 = ~160 mm Hg

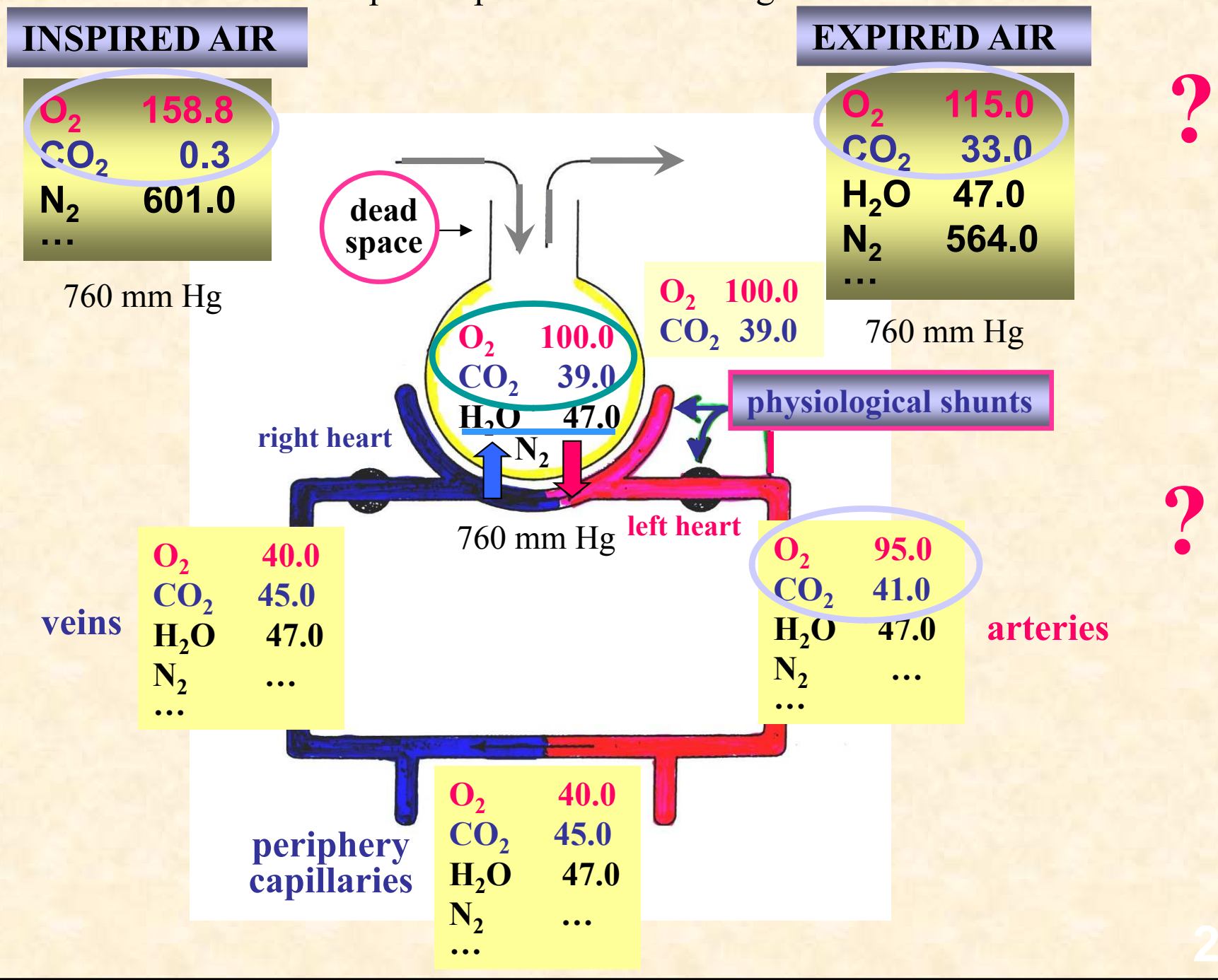
$P_{N2}$ = 760 x 0.78 = ~593 mm Hg

$P_{CO2}$ = 760 x 0.0004 = ~0.3 mm Hg

1 *kPa* = 7.5 *mm Hg (torr)*

# COMPOSITION OF ALVEOLAR AIR

partial pressures in mm Hg

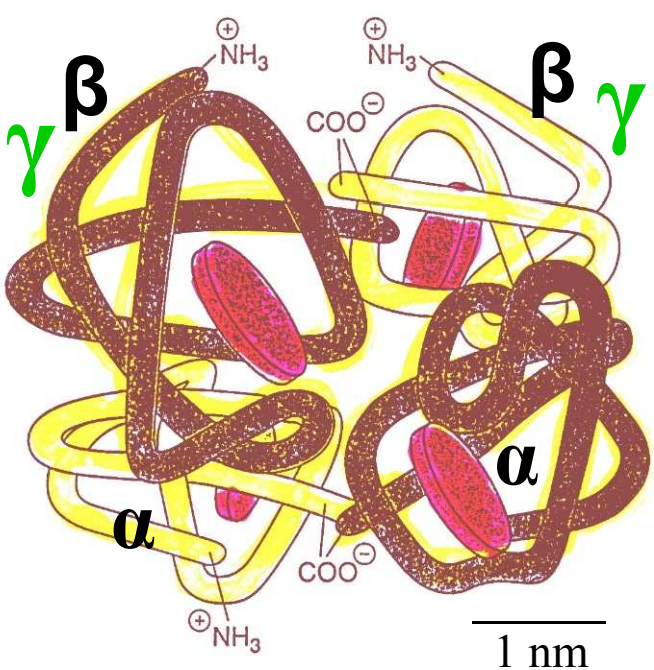


# HAEMOGLOBIN

$Hb_4 + 4\ O_2 \leftrightarrow Hb_4O_8$

**oxygenation**

tetramer

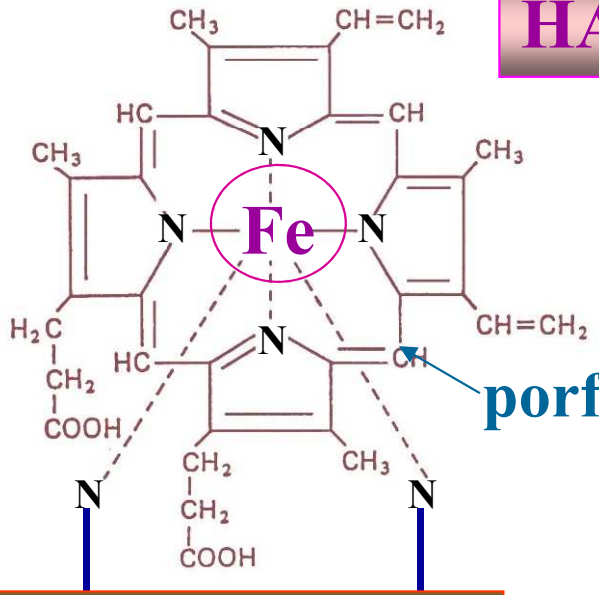


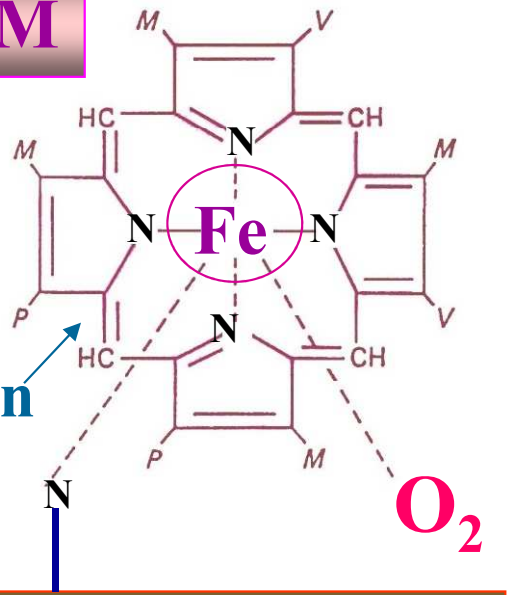

$Fe^{2+}$

**HAEM**

**DEOXY**

**OXY**




**porfyrin**

$O_2$

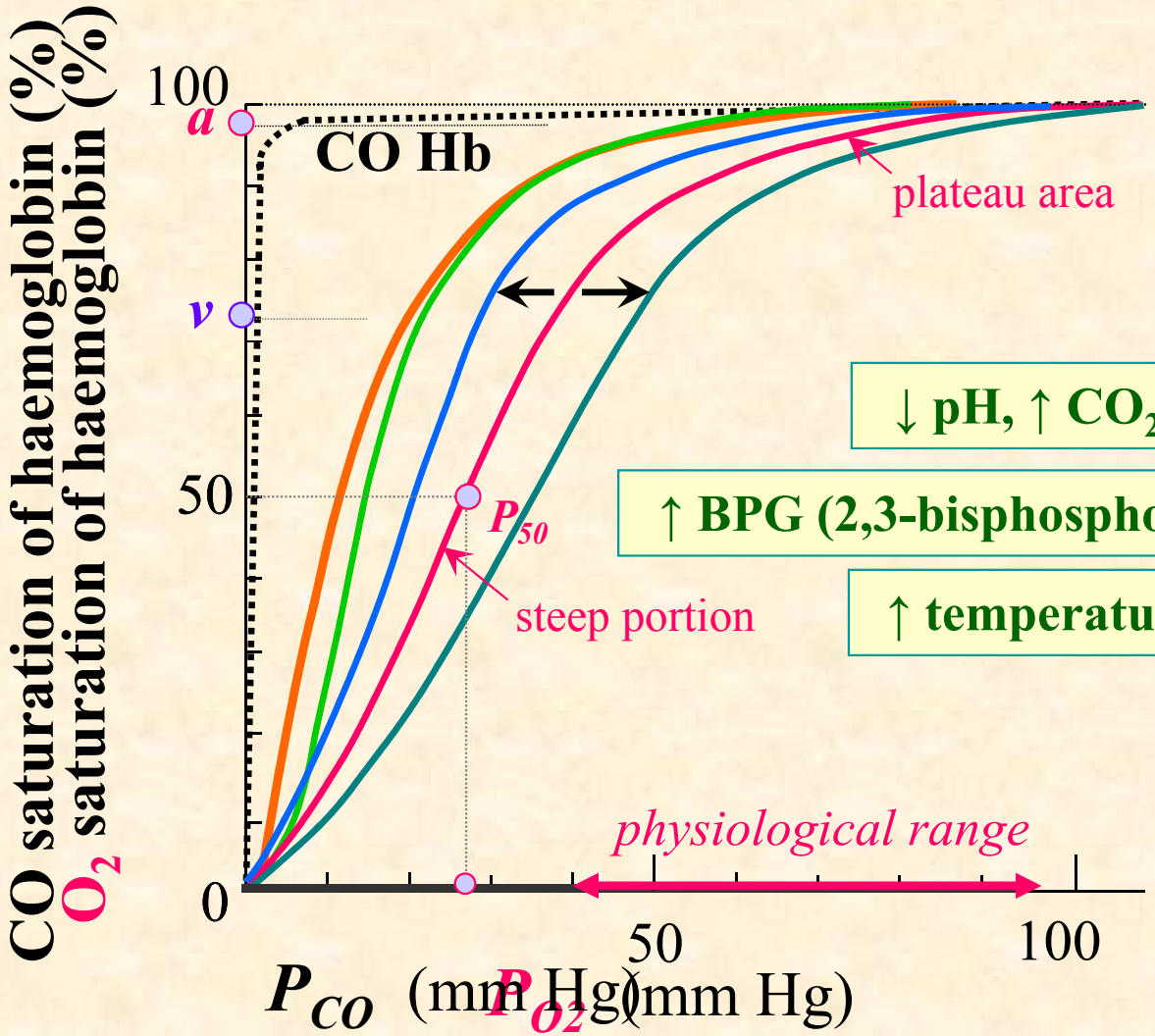
**polypeptide chain**

**polypeptide chain**

**fetal Hb**

**$Fe^{3+}$ (methaemoglobin) oxidation**

# $O_2$–HAEMOGLOBIN DISSOCIATION CURVE




**BOHR´S EFFECT**
**(↓ pH, ↑$CO_2$)**

**↓ pH, ↑ $CO_2$**

**↑ BPG (2,3-bisphosphoglycerate)**

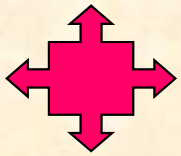
**↑ temperature**

**fetal Hb**

**myoglobin**

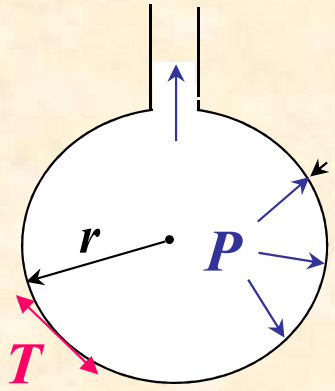
methaemoglobin

**physically dissolved $O_2$ (1.4%)**



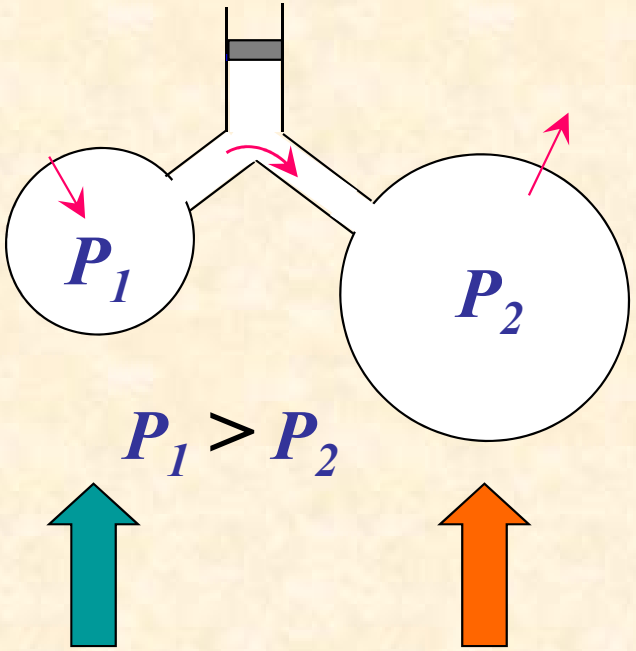
# LAW OF LAPLACE

spherical structures




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

?




*P* pressure

*r* radius

*T* surface tension

**PATHOLOGY**

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

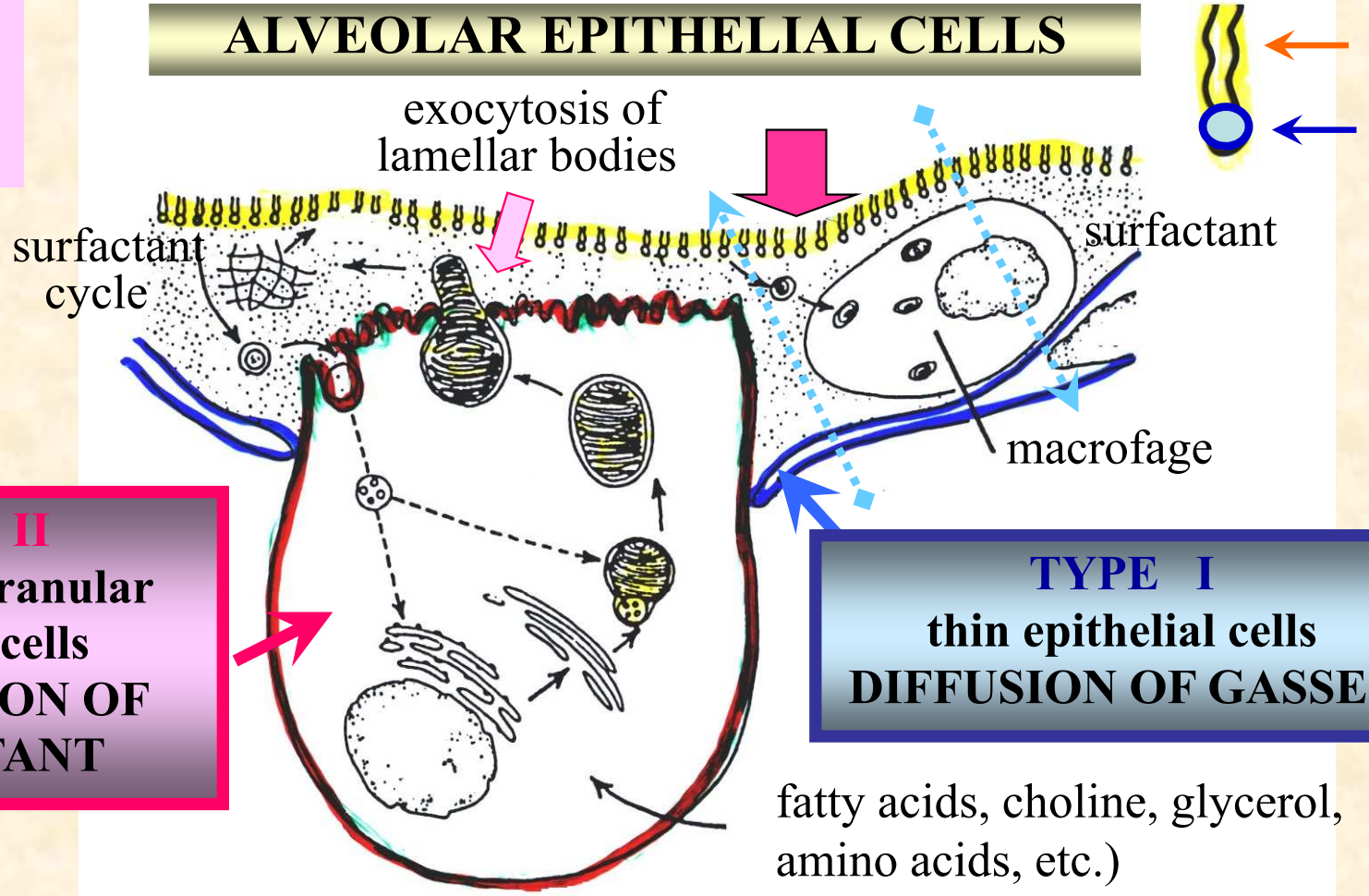
# SURFACTANT

## SURFACE TENSION LOWERING AGENT

## EFFECT MAINLY IN THE EXPIRED POSITION

**PHOSPHOLIPID**
dipalmitoyl
fosfatidyl cholin

## ALVEOLAR EPITHELIAL CELLS



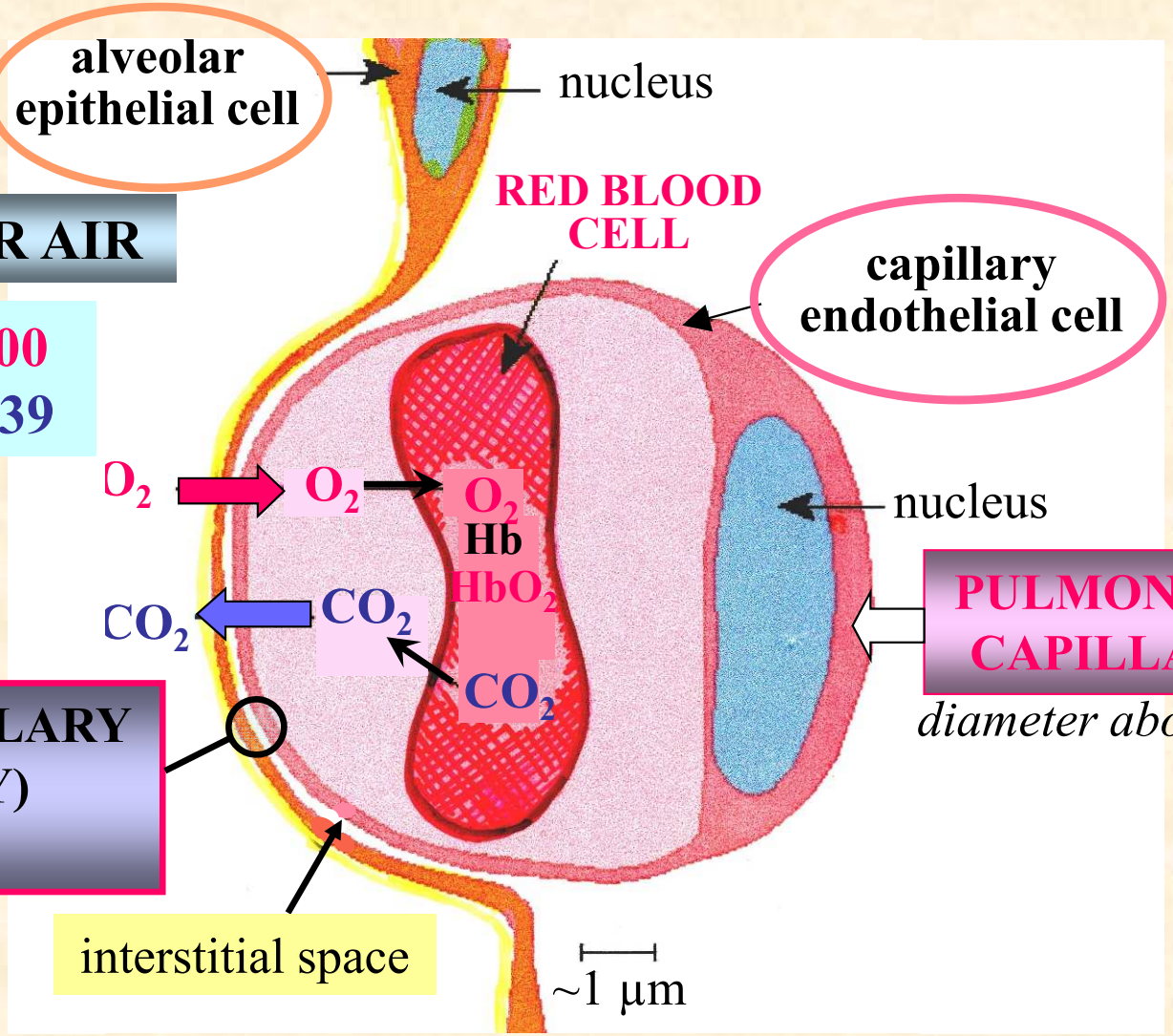

**TYPE II**
**specialized granular epithelial cells**
**PRODUCTION OF SURFACTANT**

**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

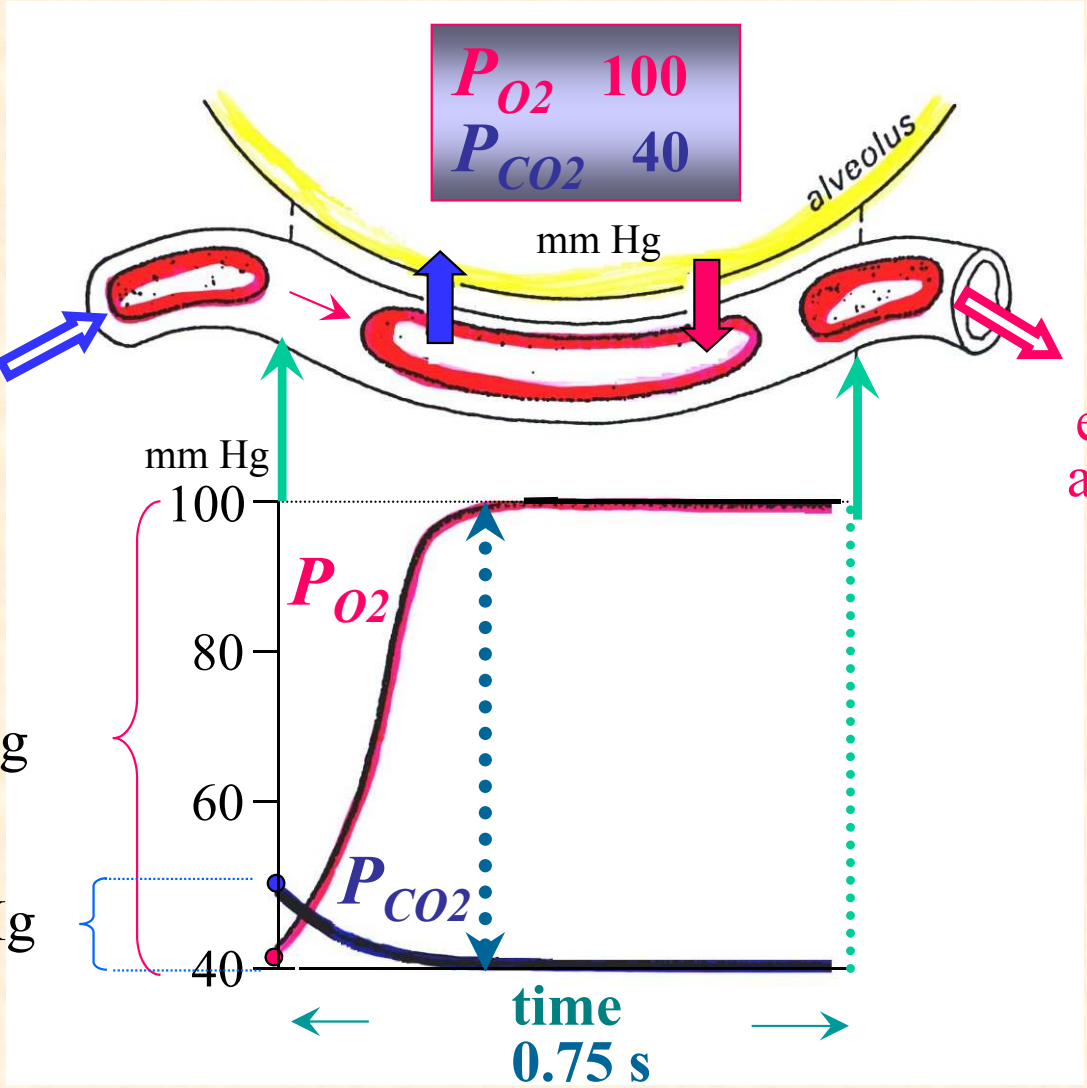
*time interval of erythrocyte contact with respiratory membrane at rest*

# TIME COURSE OF CAPILLARY $P_{O2}$ AND $P_{CO2}$ DURING GRADUAL EQUILIBRATION WITH ALVEOLAR AIR

**venous blood**

$P_{O2}$ 40
$P_{CO2}$ 46

mm Hg




$P_{O2}$ 100
$P_{CO2}$ 40

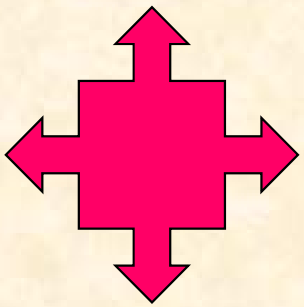
mm Hg

equalization with alveolar pressures

$\Delta P_{O2} \cong 60$ mm Hg

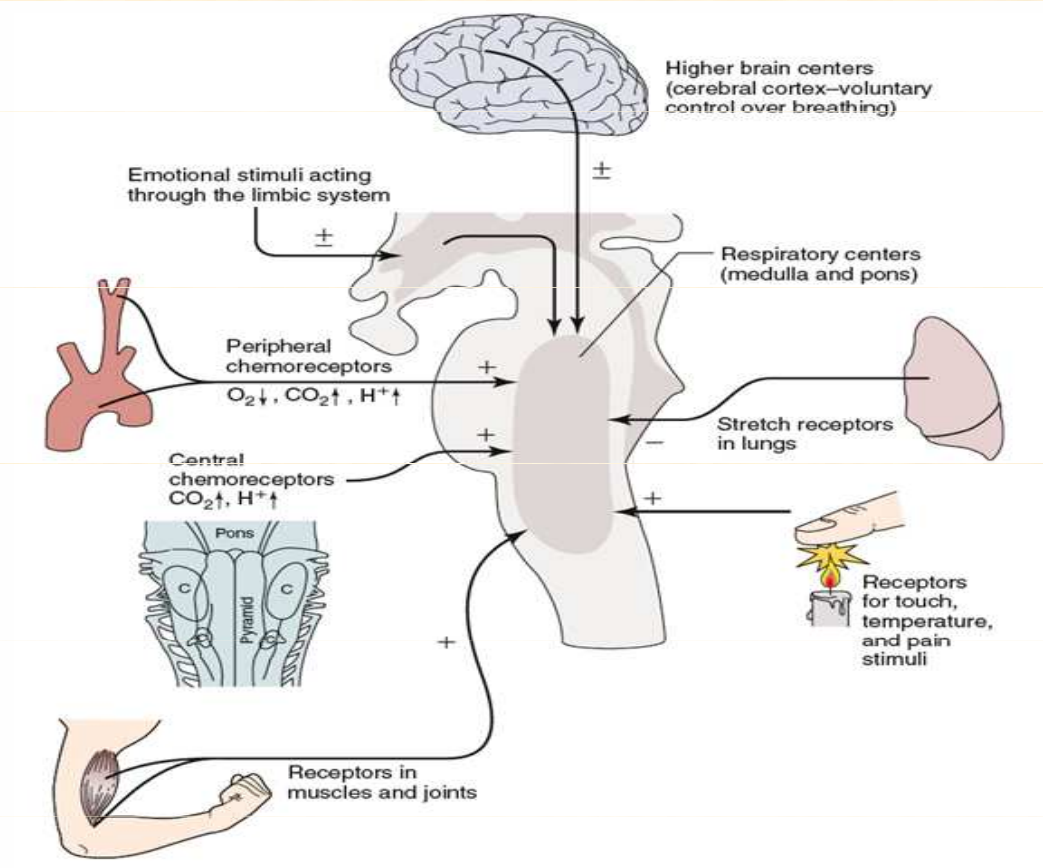
$\Delta P_{CO2} \cong 6$ mm Hg

**time interval of contact of erythrocyte with respiratory membrane at rest**

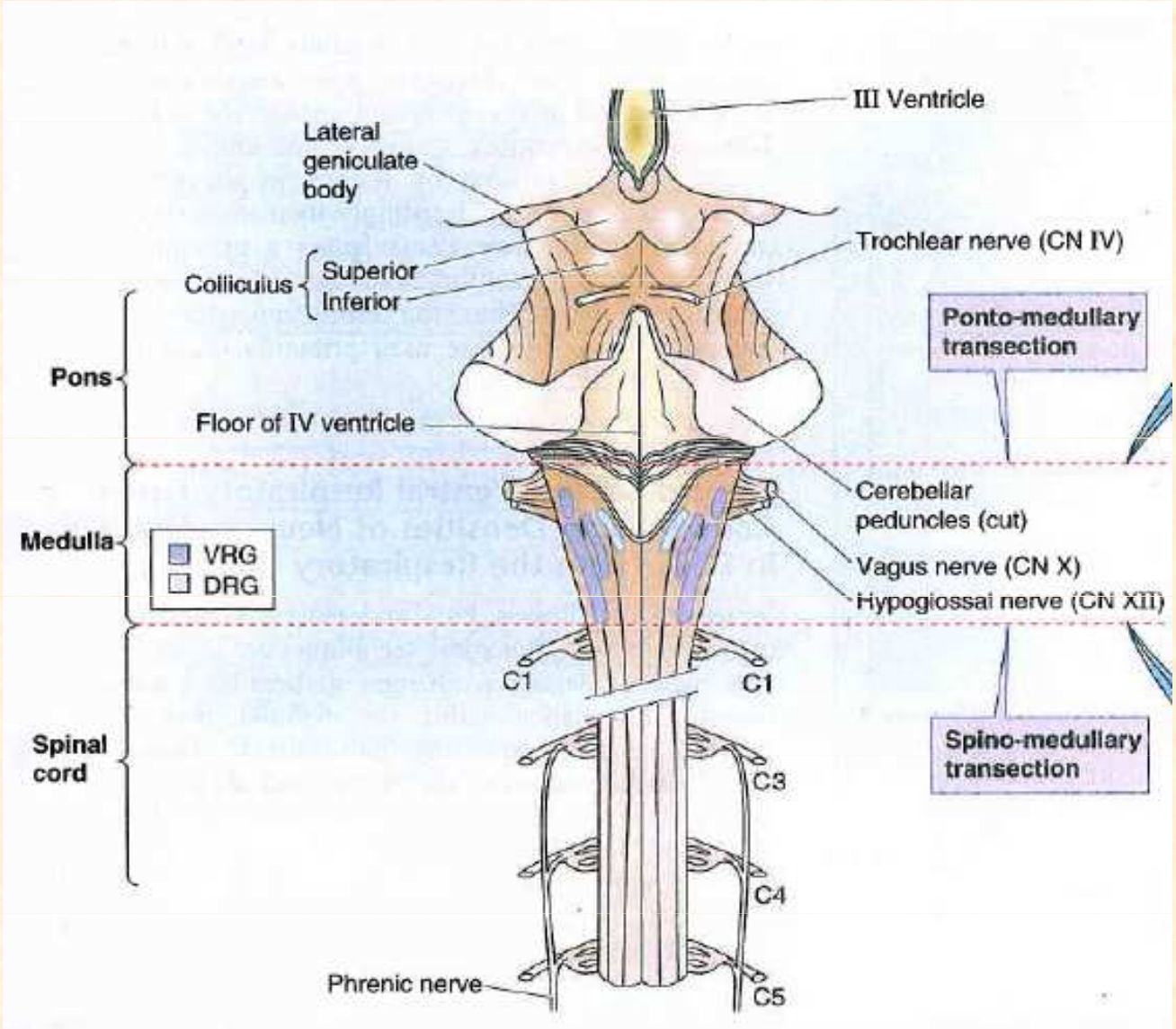


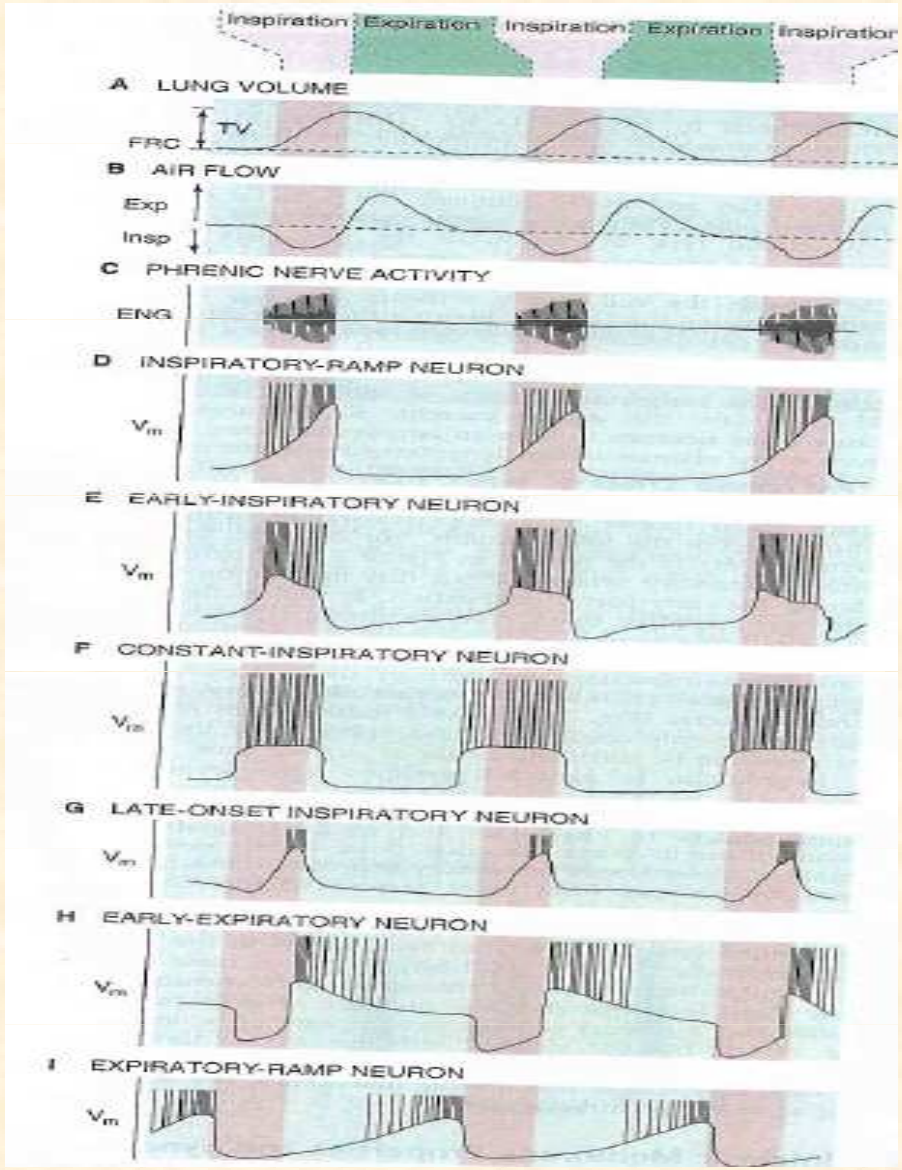
END

# Control of ventilation




https://sleep.sharepoint.com/siteimages/Chapter%203.png


III Ventricle
Lateral geniculate body
Trochlear nerve (CN IV)
Colliculus
Superior
Inferior
Pons
Ponto-medullary transection
Floor of IV ventricle
Cerebellar peduncles (cut)
Medulla
VRG
DRG
Vagus nerve (CN X)
Hypoglossal nerve (CN XII)
C1
C1
Spinal cord
Spino-medullary transection
C3
C4
Phrenic nerve
C5


Inspiration
Expiration
Inspiration
Expiration
Inspiration
A LUNG VOLUME
TV
FRC
B AIR FLOW
Exp
Insp
C PHRENIC NERVE ACTIVITY
ENG
D INSPIRATORY-RAMP NEURON
$V_m$
E EARLY-INSPIRATORY NEURON
$V_m$
F CONSTANT-INSPIRATORY NEURON
$V_m$
G LATE-ONSET INSPIRATORY NEURON
$V_m$
H EARLY-EXPIRATORY NEURON
$V_m$
I EXPIRATORY-RAMP NEURON
$V_m$

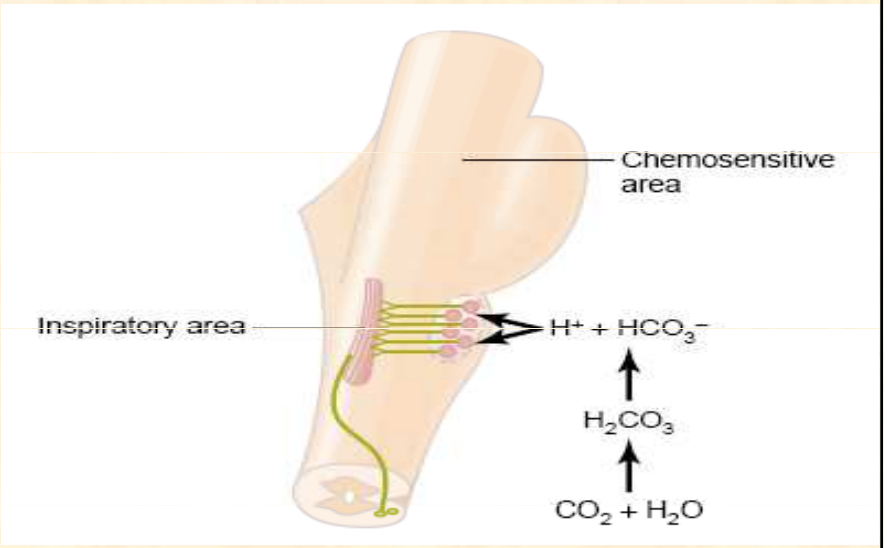
- **Breathing is an automatic process that takes place unconsciously. Automaticity of breathing comes from regular (rhythmic) activity of groups of neurons anatomically localized in the medulla and its vicinity.**

- 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.

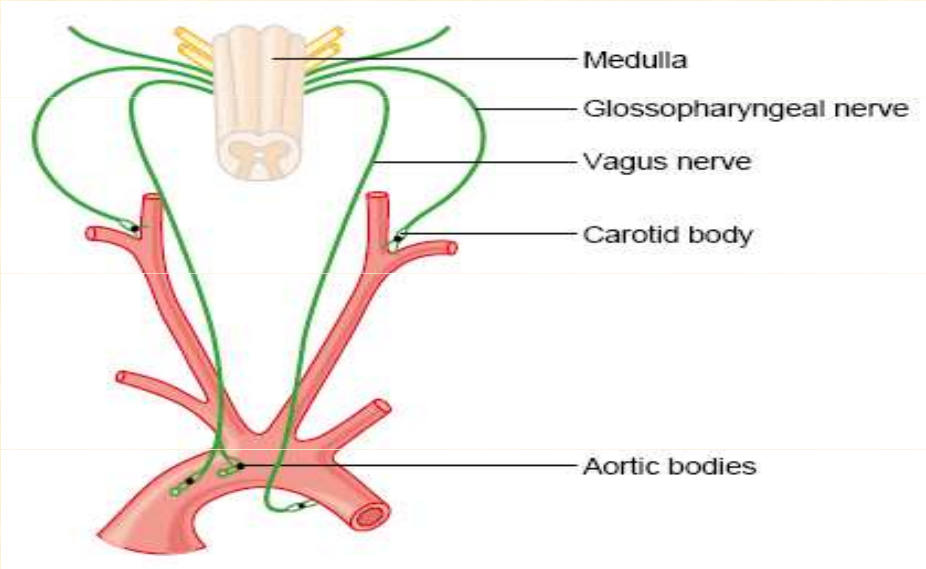
# Chemical factors affecting the respiratory center:

Central chemoreceptors

- on the front side of the medulla
- sensitive only to increase of arterial $pCO_2$ (by increasing $H^+$ )




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

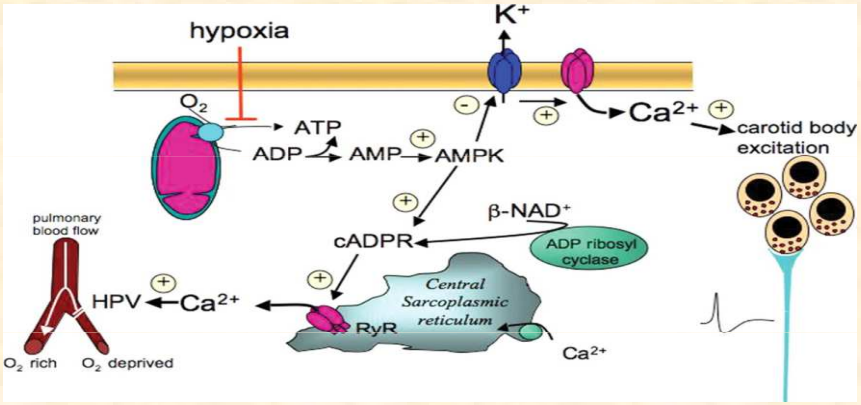



## Peripheral chemoreceptors

– located in the aortic and carotid bodies

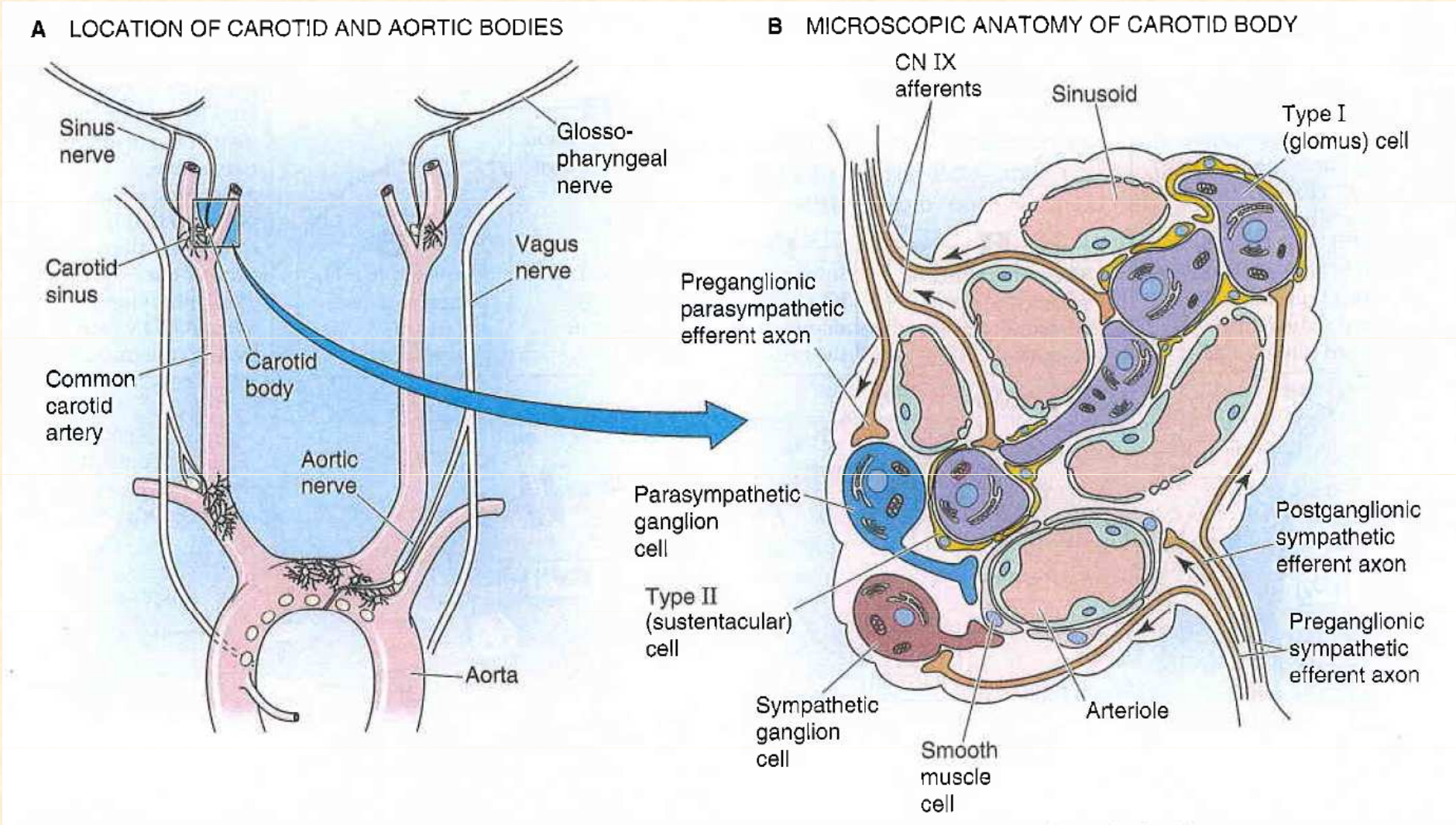
-primarily sensitive to decrease in arterial $pO_2$, particularly to decrease of $O_2$ under 10-13 kPa in the arterial blood.

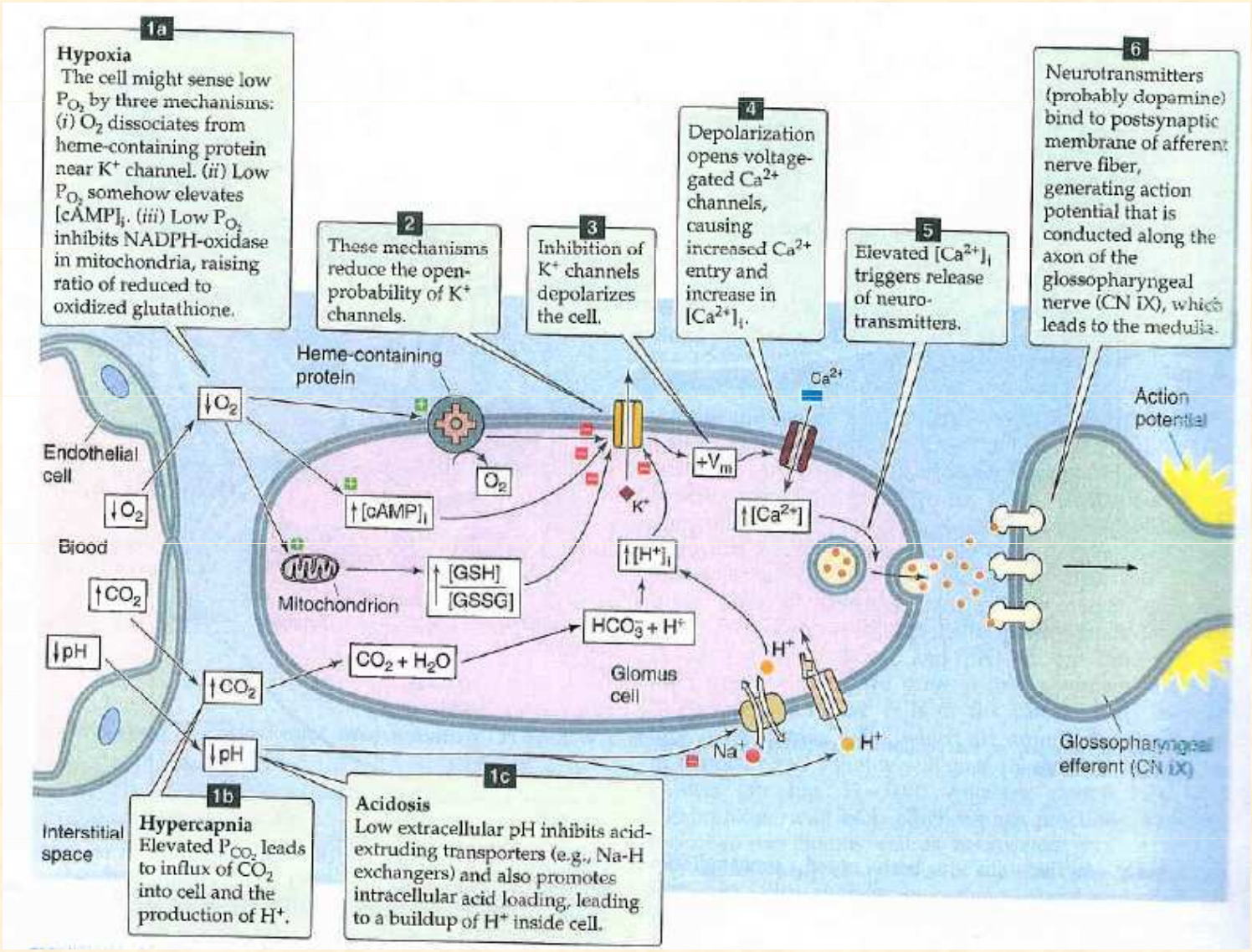
They convey their sensory information to the medulla via the vagus nerve and glossopharyngeal nerve.




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

http://www.medicine.mcgill.ca/physio/resp-web/sect8.htm,


A LOCATION OF CAROTID AND AORTIC BODIES
Sinus nerve
Glosso-pharyngeal nerve
Vagus nerve
Carotid sinus
Common carotid artery
Carotid body
Aortic nerve
Aorta
B MICROSCOPIC ANATOMY OF CAROTID BODY
CN IX afferents
Sinusoid
Type I (glomus) cell
Preganglionic parasympathetic efferent axon
Parasympathetic ganglion cell
Type II (sustentacular) cell
Postganglionic sympathetic efferent axon
Preganglionic sympathetic efferent axon
Sympathetic ganglion cell
Smooth muscle cell
Arteriole


1a
Hypoxia
The cell might sense low $P_{O_2}$ by three mechanisms: (*i*) $O_2$ dissociates from heme-containing protein near $K^+$ channel. (*ii*) Low $P_{O_2}$ somehow elevates $[cAMP]_i$. (*iii*) Low $P_{O_2}$ inhibits NADPH-oxidase in mitochondria, raising ratio of reduced to oxidized glutathione.
2
These mechanisms reduce the open-probability of $K^+$ channels.
3
Inhibition of $K^+$ channels depolarizes the cell.
4
Depolarization opens voltage-gated $Ca^{2+}$ channels, causing increased $Ca^{2+}$ entry and increase in $[Ca^{2+}]_i$.
5
Elevated $[Ca^{2+}]_i$ triggers release of neuro-transmitters.
6
Neurotransmitters (probably dopamine) bind to postsynaptic membrane of afferent nerve fiber, generating action potential that is conducted along the axon of the glossopharyngeal nerve (CN IX), which leads to the medulla.
Heme-containing protein
$\downarrow O_2$
$O_2$
Endothelial cell
$\downarrow O_2$
Blood
$\uparrow CO_2$
$\downarrow pH$
$\uparrow CO_2$
$\downarrow pH$
$\uparrow [cAMP]_i$
Mitochondrion
$\uparrow \frac{[GSH]}{[GSSG]}$
$CO_2 + H_2O$
$HCO_3^- + H^+$
$\uparrow [H^+]_i$
$K^+$
$+V_m$
$Ca^{2+}$
$\uparrow [Ca^{2+}]$
$H^+$
$Na^+$
$H^+$
Glomus cell
Action potential
Glossopharyngeal efferent (CN IX)
Interstitial space
1b
Hypercapnia
Elevated $P_{CO_2}$ leads to influx of $CO_2$ into cell and the production of $H^+$.
1c
Acidosis
Low extracellular pH inhibits acid-extruding transporters (e.g., Na-H exchangers) and also promotes intracellular acid loading, leading to a buildup of $H^+$ inside cell.

# Modulation of respiratory output

Major parameters for feedback control – classical gases:pO2, pCO2, pH

In additin 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 agentsreceptors in respiratory system

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

**Irritants receptors** on mucose of respiratory system – rapidly adapting
Stimulus: agens - chemical substances (histamin, serotonin, prostaglandins, ammonia, cigarette smoke).
Respons: increase mucus secretion, constriction of larynx and brochus

**C-fibre receptors** (juxtacapillary=J receptors)– free nerve ending of n.vagus (unmyelinated axon) in intersticium of bronchus and alveolus;
Stimulus: Mechanical irritans (pulmonary hypertension, pulmonary oedema)+chemical
Response: hypopnoe, rapid shallow breathing, bronchoconstriction, cough

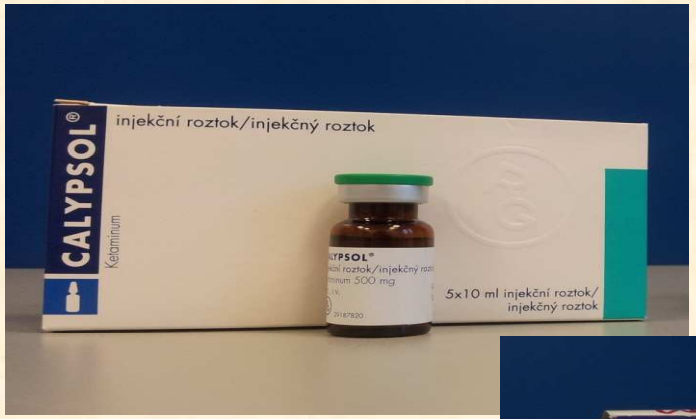
**Stretch receptors** slowly adapting (mechanoreceptors in tracheobronchial tree that detect the changes in lung volume by sensing the stretch receptors of the airway wall), inform to brain about the lung volume to optimize respiratory; its irritants triggered decrese activity of respiratory centre –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

**Baroreceptors** – suppresses activity of  respiratory centre

Irritants of **proprioreceptors of muscles, tendons** during active and pasive 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**

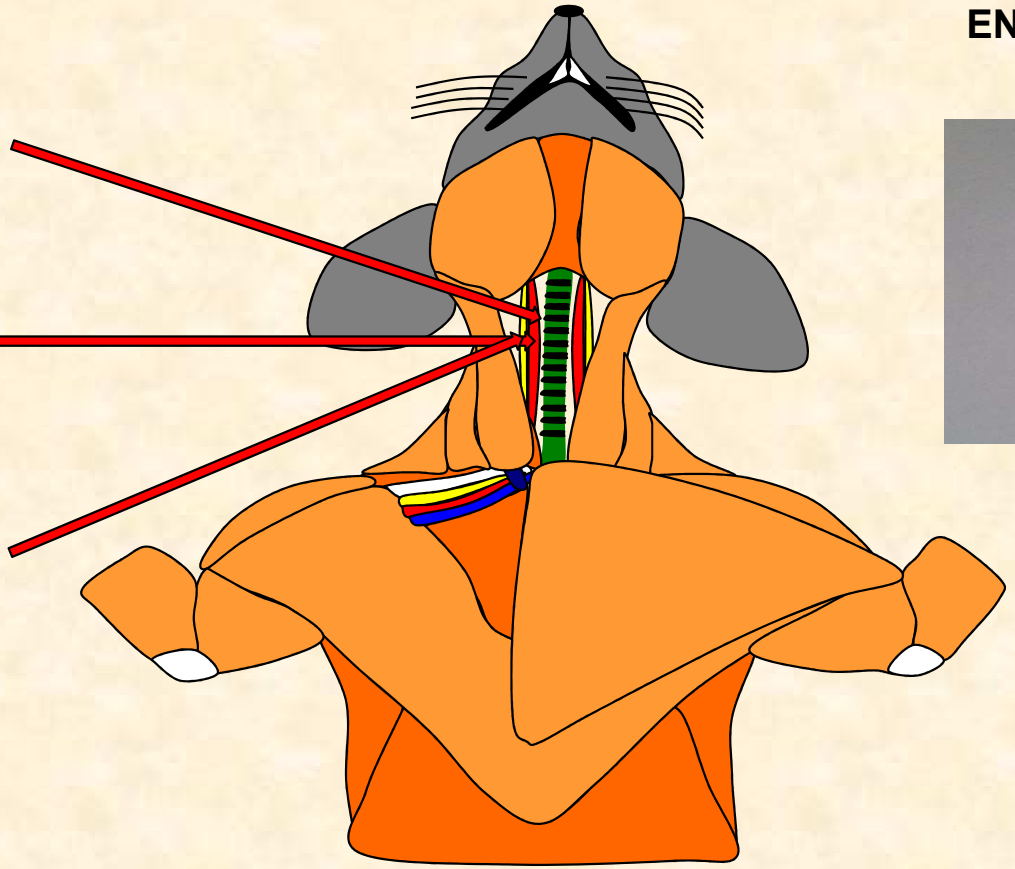

CALYPSOL®
Ketaminum
injekční roztok/injekčný roztok
5x10 ml injekční roztok/
injekčný roztok


Rometar 2%
JEN PRO ZVÍŘATA!
Analgetikum
50 ml
Intramuskulárně, intravenózně
a subkutánně
SPOFA
Rometar 2%
JEN PRO ZVÍŘATA!
SPOFA

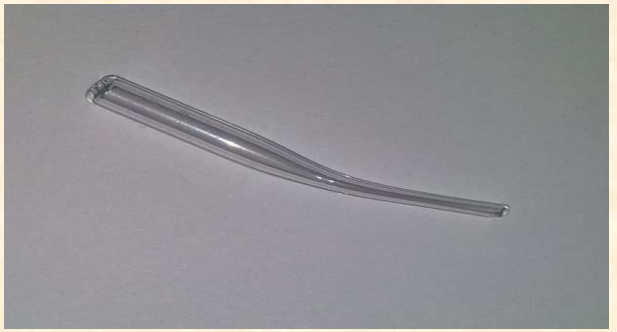
TRACHEA

A. CAROTIS

N. VAGUS

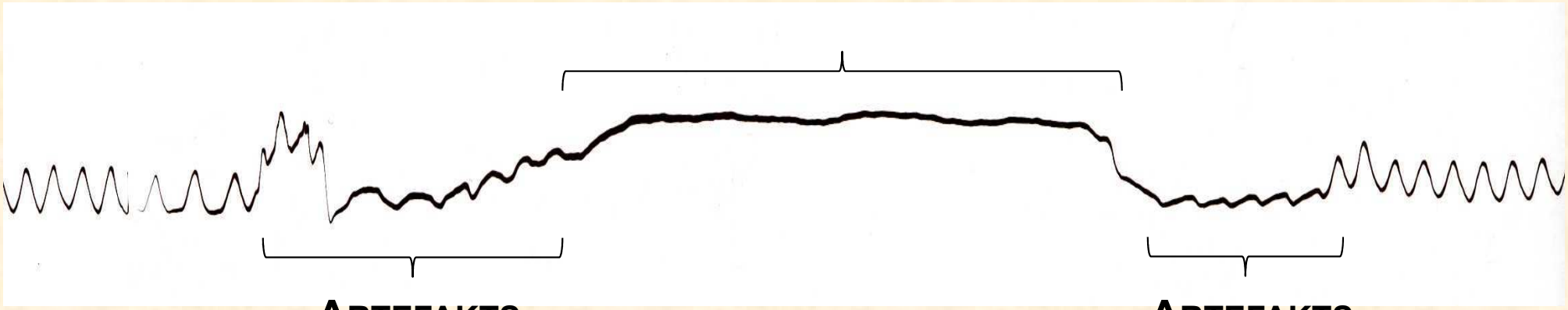


ENDOTRACHEAL CANNULA



# HERING-BREUER REFLEX

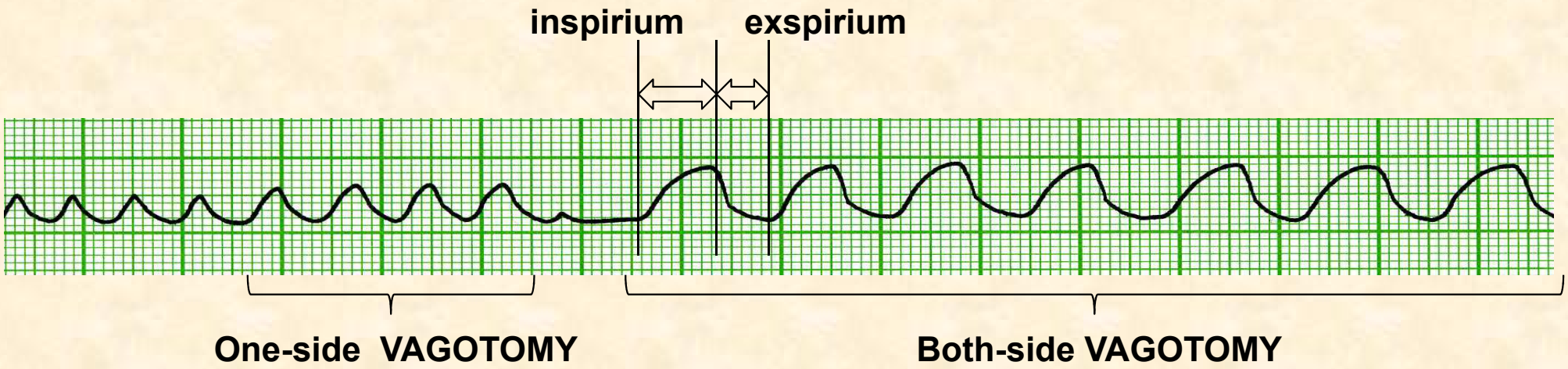
## REFLEX STOP BREATHING



ARTEFAKTS

ARTEFAKTS

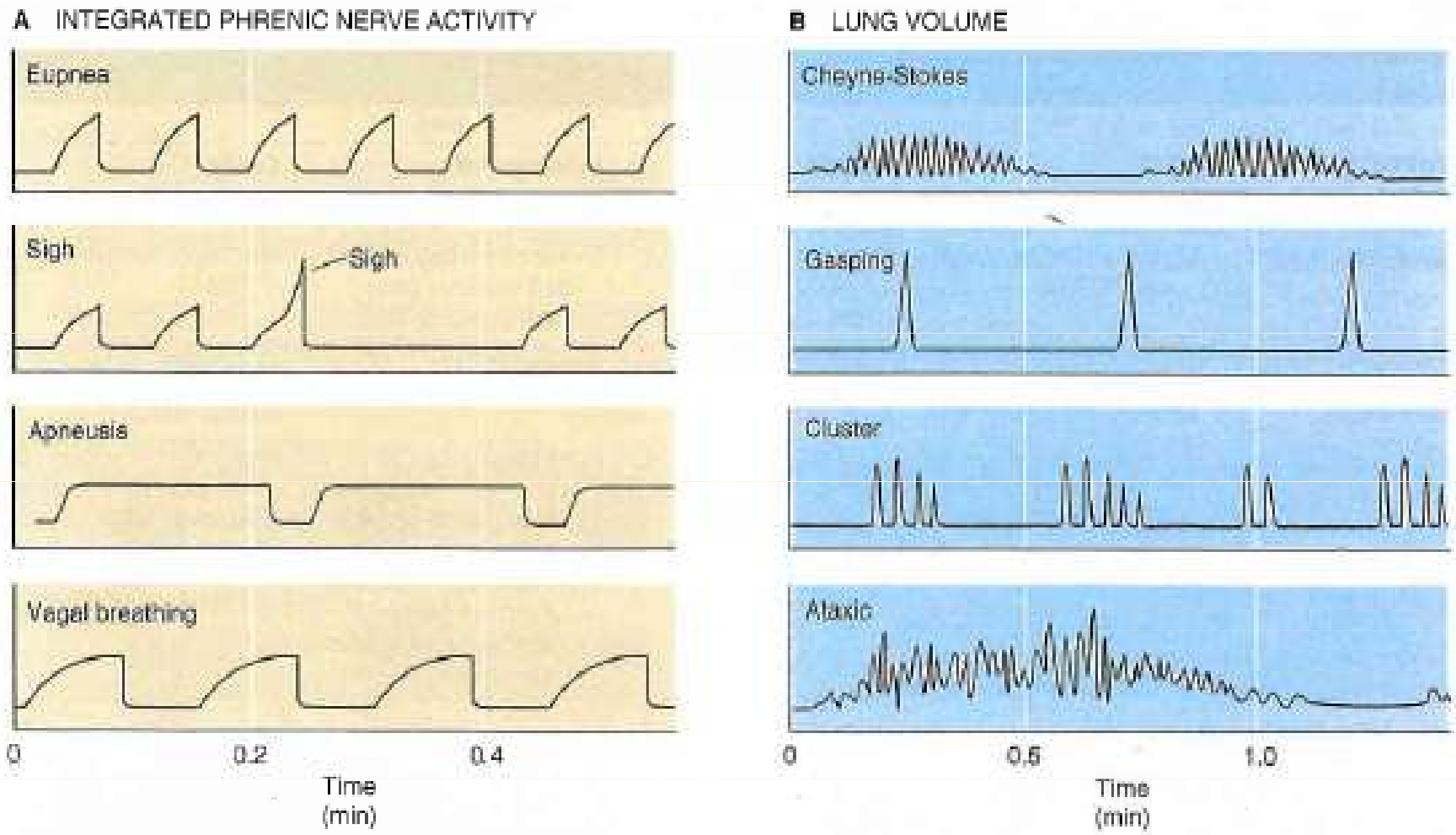
# VAGOTOMY

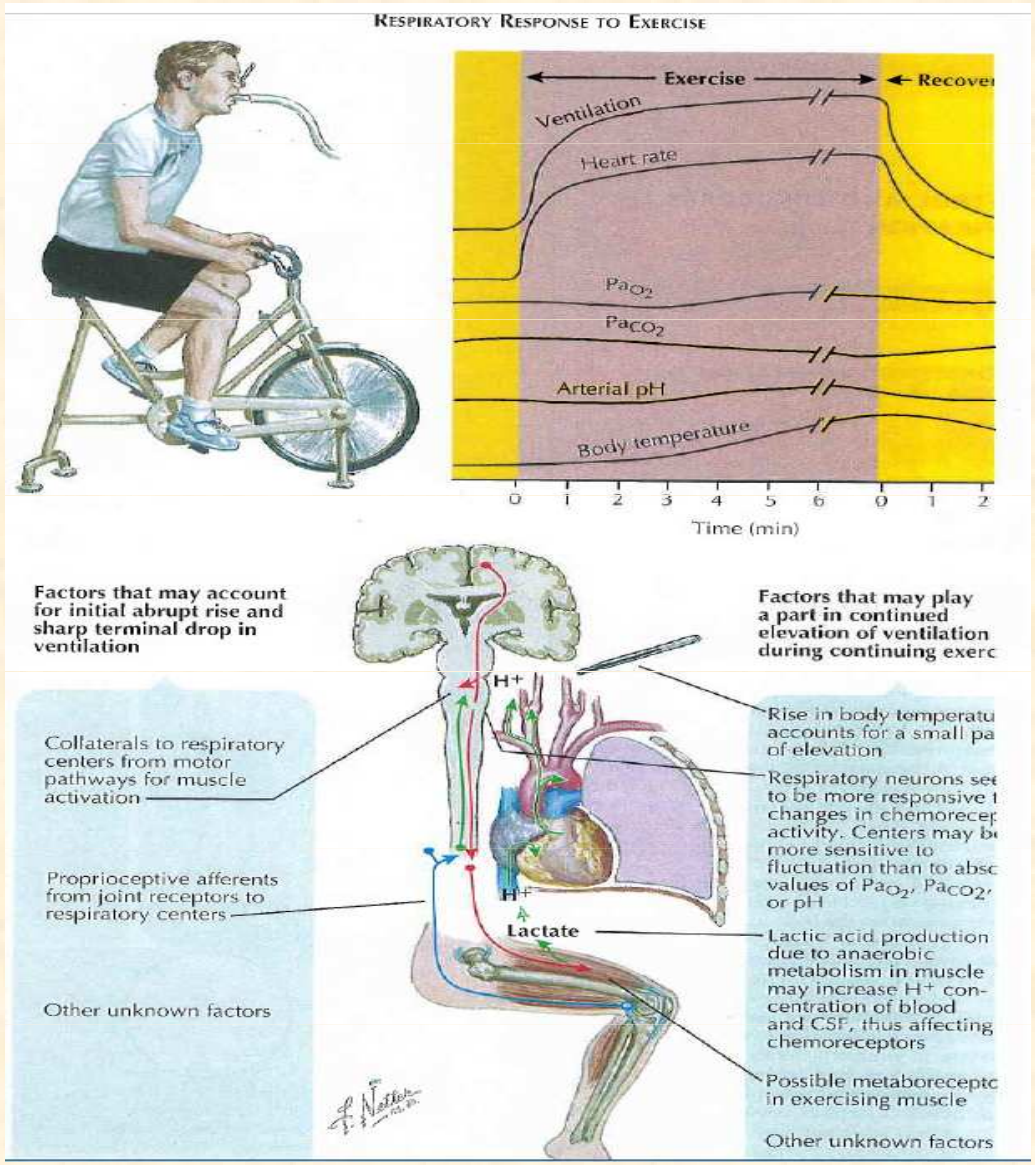


# Periodic breathing

- It is not regular, rhythmic, but respiration occurs in periods ("a moment to breathe, take a moment to not breathe„)

- **CHEYNE-STOKES**
- **BIOT‘S**
- „gasping“
- KUSSMAUL


A INTEGRATED PHRENIC NERVE ACTIVITY
Eupnea
Sigh
Sigh
Apneusis
Vagal breathing
0
0.2
0.4
Time
(min)
B LUNG VOLUME
Cheyne-Stokes
Gasping
Cluster
Ataxic
0
0.5
1.0
Time
(min)


RESPIRATORY RESPONSE TO EXERCISE
Exercise
Recover
Ventilation
Heart rate
$Pa_{O_2}$
$Pa_{CO_2}$
Arterial pH
Body temperature
0 1 2 3 4 5 6 0 1 2
Time (min)
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 exerc
Rise in body temperatu accounts for a small pa of elevation
Respiratory neurons see to be more responsive t changes in chemorecep activity. Centers may b more sensitive to fluctuation than to absc values of $Pa_{O_2}$, $Pa_{CO_2}$, or pH
Lactic acid production due to anaerobic metabolism in muscle may increase $H^+$ con-centration of blood and CSF, thus affecting chemoreceptors
Possible metaborecepto in exercising muscle
Other unknown factors
$H^+$
$H^+$
Lactate

# 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.

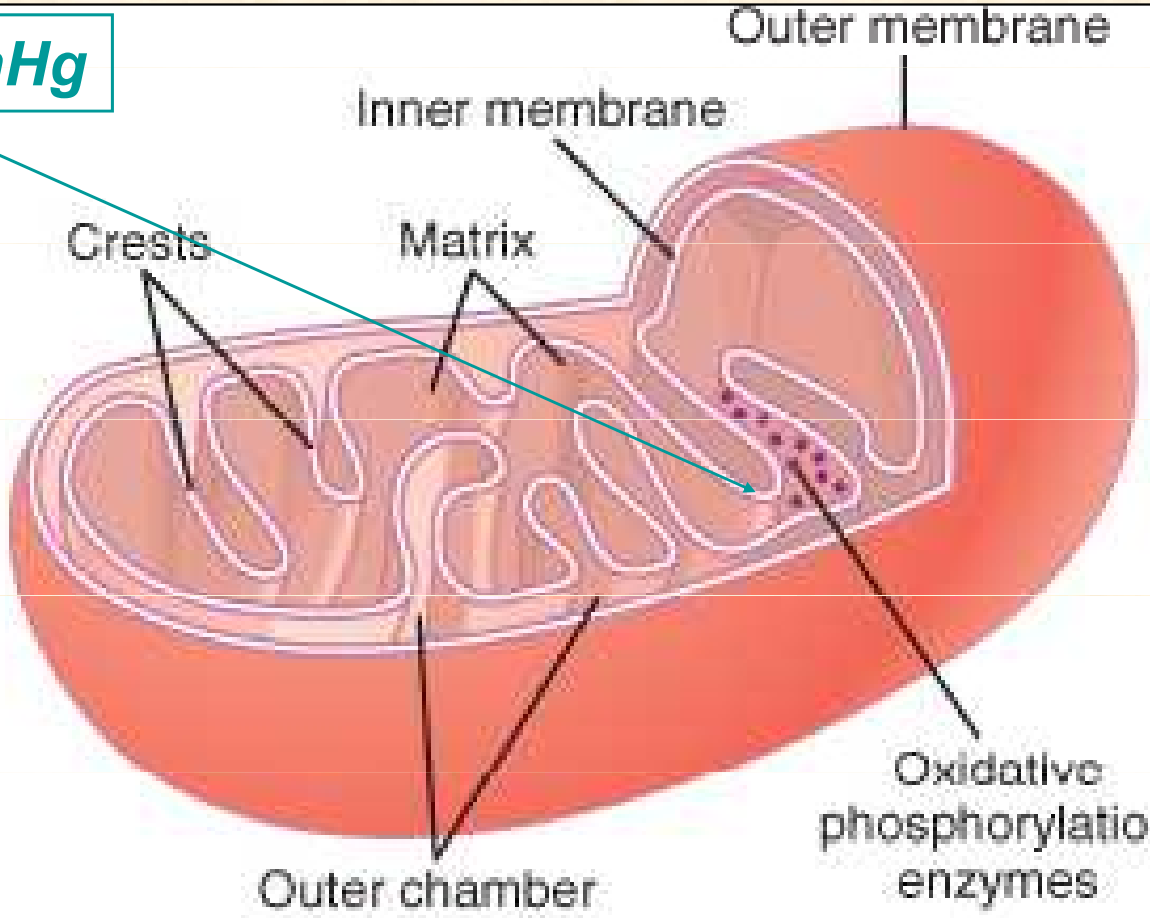
The most common types of hypoxia:

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* |
|---|---|
| *dry atmospheric air* | *159* |
| *humid atmospheric air* | *149* |
| *ideal alveolar gass* | *105* |
| *end-expiration air* | *105* |
| *arterial blood* | *77* |
| *cytoplasma – mitochondria* | *3-10* |
| *mixed venous blood* | *40* |
| *venous blood* | *20* |

$pO_2$ = 1 mmHg



# Hypercapnia

- Hypercapnia - increase of concentration of carbon dioxide in the blood or in tissues that is caused by retention of $CO_2$ 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% $CO_2$)
- hypercapnia around 10 kPa - $CO_2$ 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 aicraft is pressure as on 2000 m above see level)

***<u>High risk for patients with diseases:</u>***

- ***concentration of hemoglobin  lower than 60 %***
- ***severe step of atherosclerosis***
- ***cardial insuficiency***
- ***respiratory insuficiency***
- ***non-treated hypertension (BP ower 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***

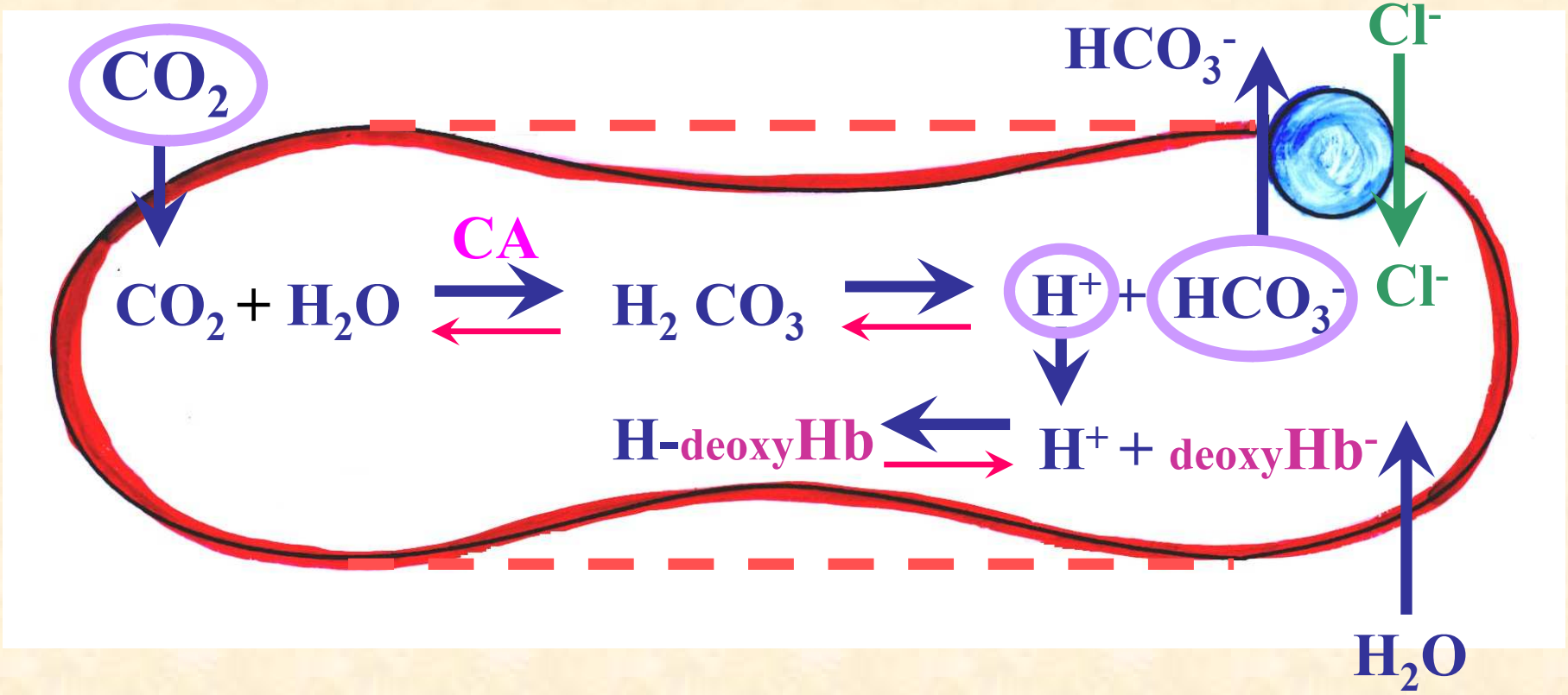
***Recommendation:***

***100 % - give discontinuosly***

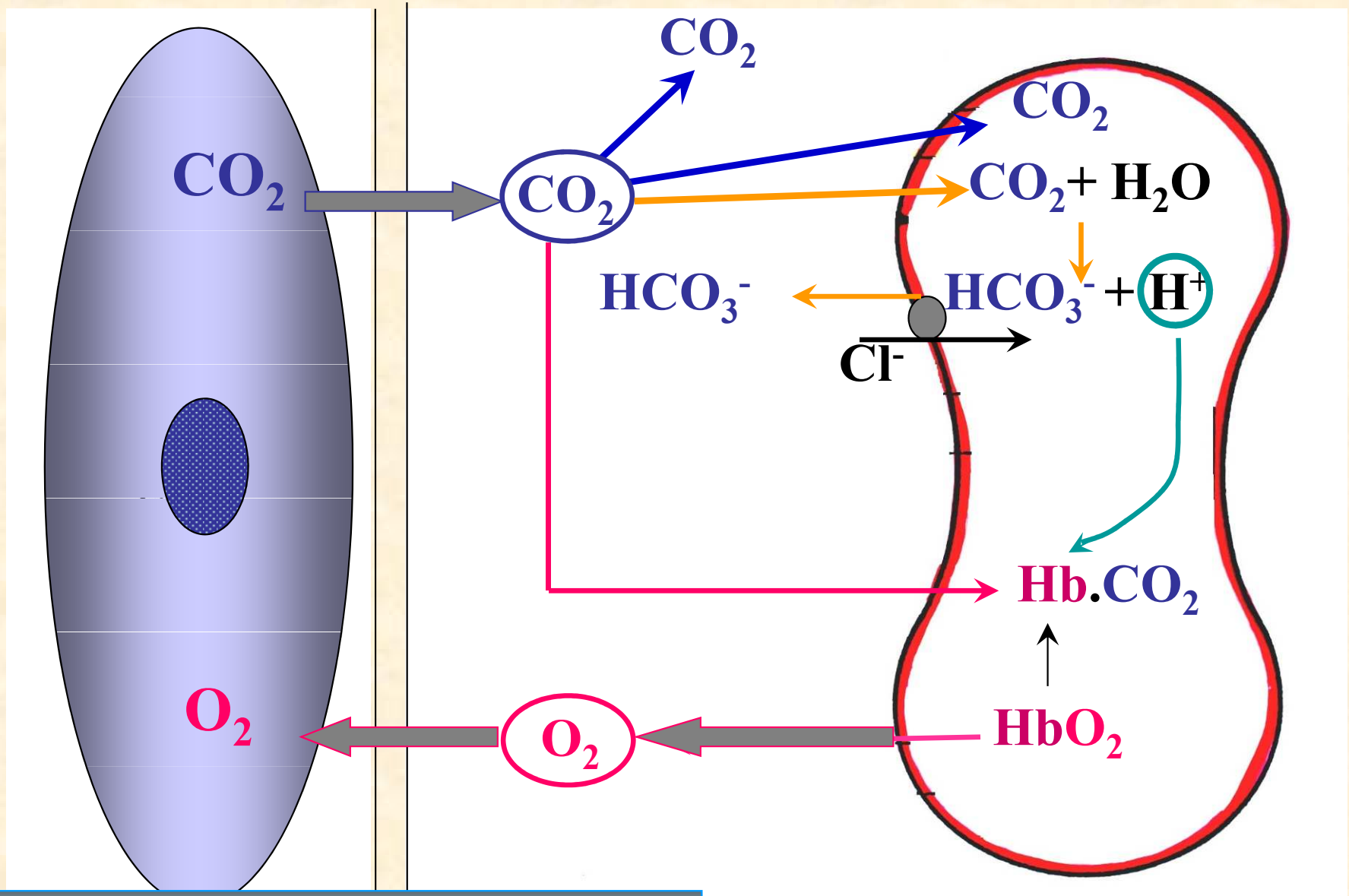
# THANK YOU FOR YOUR ATTENTION

# TRANSPORT OF $CO_2$

**HAMBURGER CHLORIDE SHIFT**



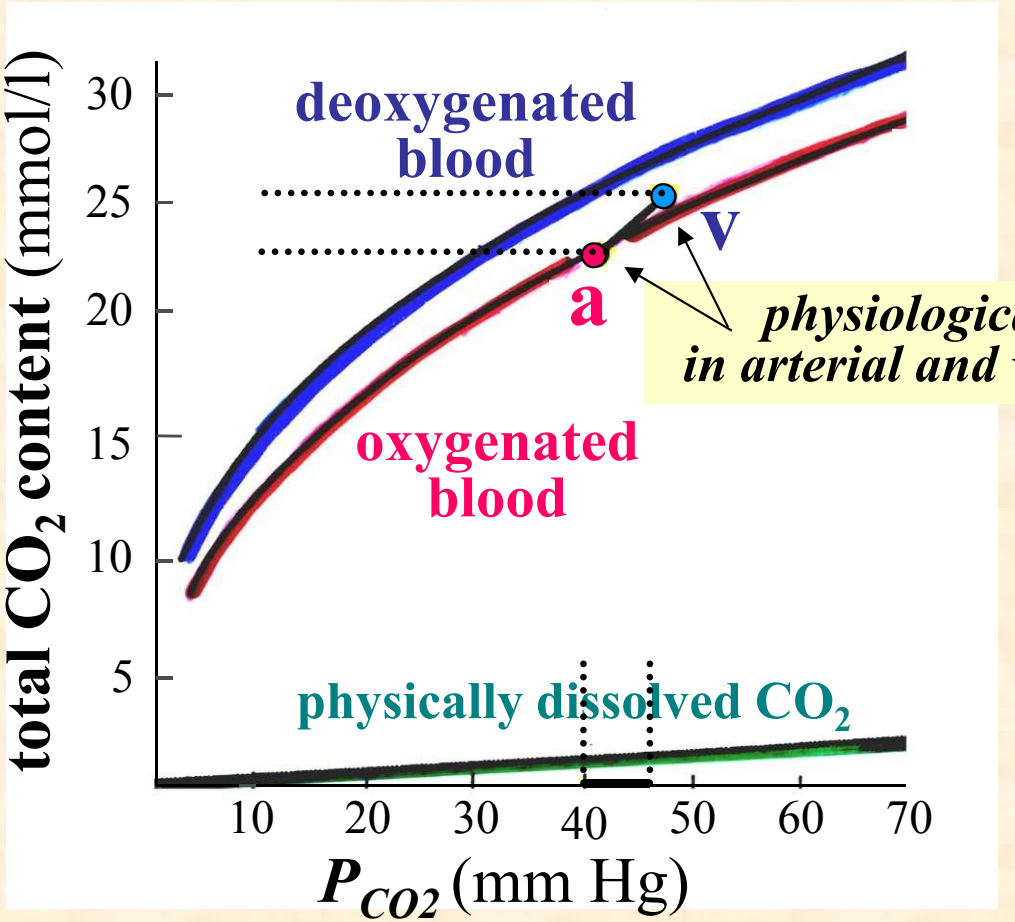

**CA – carbonic anhydrase**




- $CO_2$ physically dissolved (~5.3%)
- $CO_2 + Hb\text{-}NH_2 \rightleftharpoons Hb.NH\text{-}COO^-$ (carbamino-Hb) (~5.3 %)
- $CO_2 + H_2O \rightleftharpoons HCO_3^- + H^+$ (~89%)

60% in plasma, 29% in red blood cell

# $CO_2$ DISSOCIATION CURVE




*physiological values in arterial and venous blood*

## HALDANE EFFECT

?

**DEOXY-Hb**

1 $Hb.NH.COO^-$

2 $Hb^- + H^+ \rightleftarrows HHb$

→ deoxygenated blood in peripheral tissues

← oxygenated blood in the lungs

$$CO_2 + H_2O \rightleftarrows H_2CO_3 \rightleftarrows H^+ + HCO_3^-$$

$$Hb^- + H^+ \rightleftarrows HHb$$

**TISSUES**: DEOXY-Hb binds $H^+$ more readily (weaker acid) ⇒ ↑ amount of chemically bound $CO_2$

**LUNGS**: $H^+$ is released from OXY-Hb ⇒ ↓ amount of chemically bound $CO_2$

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**

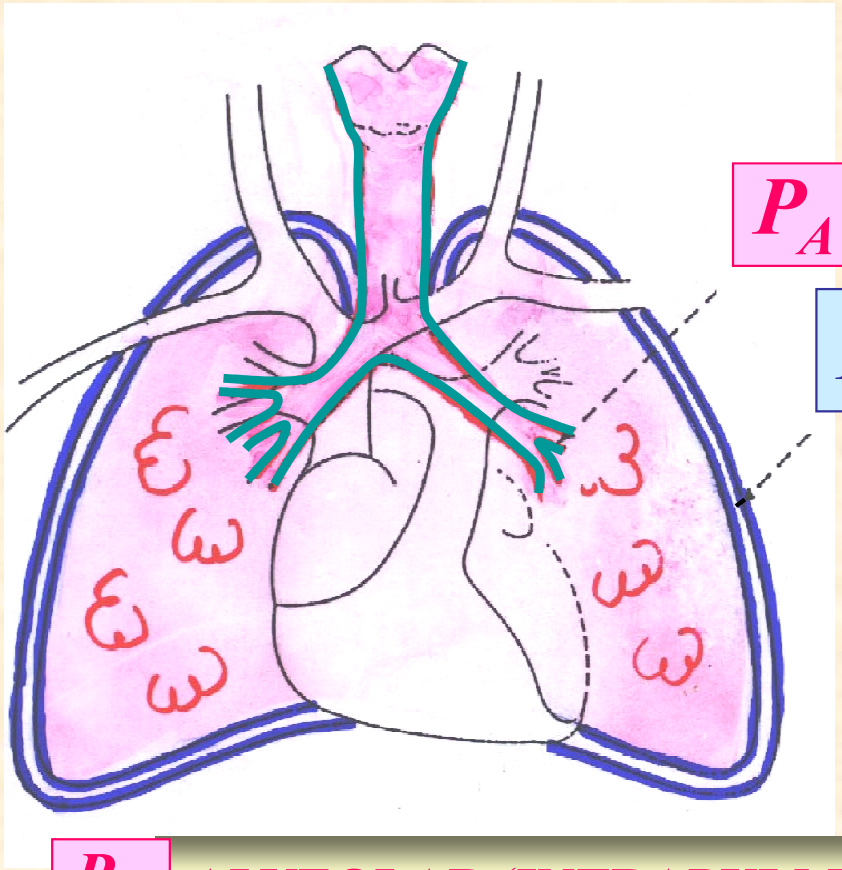
V **ALVEOLAR-CAPILLARY MEMBRANE**

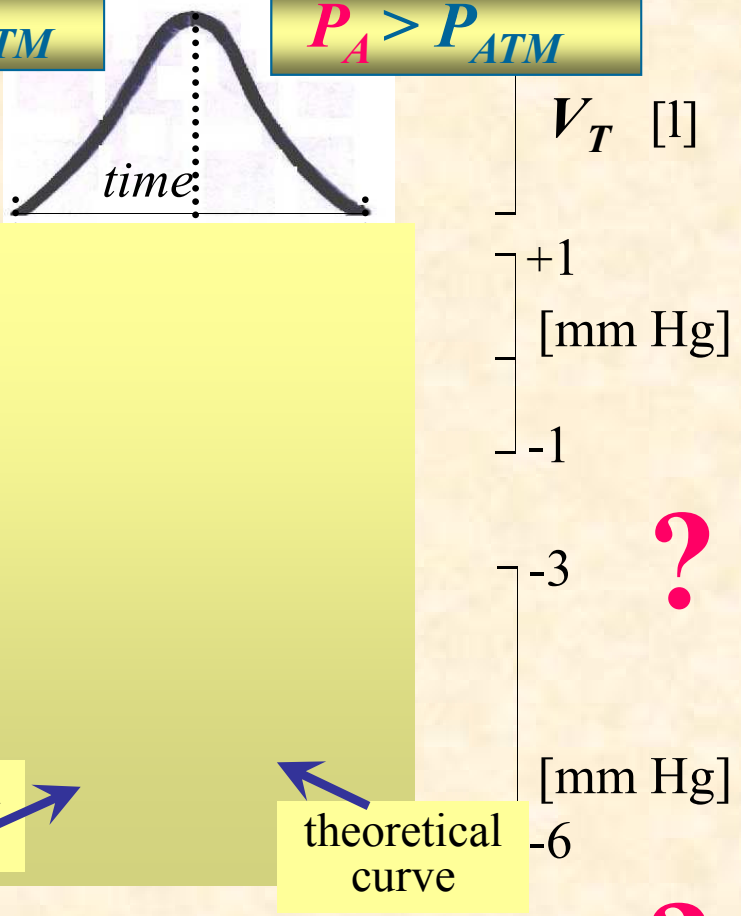
VI **TRANSPORT OF GASSES ($O_2$ and $CO_2$)**

$P.V = const$ ⇒ $P = \frac{const}{V}$

# TIME COURSE OF PRESSURES AT QUIET RESPIRATION

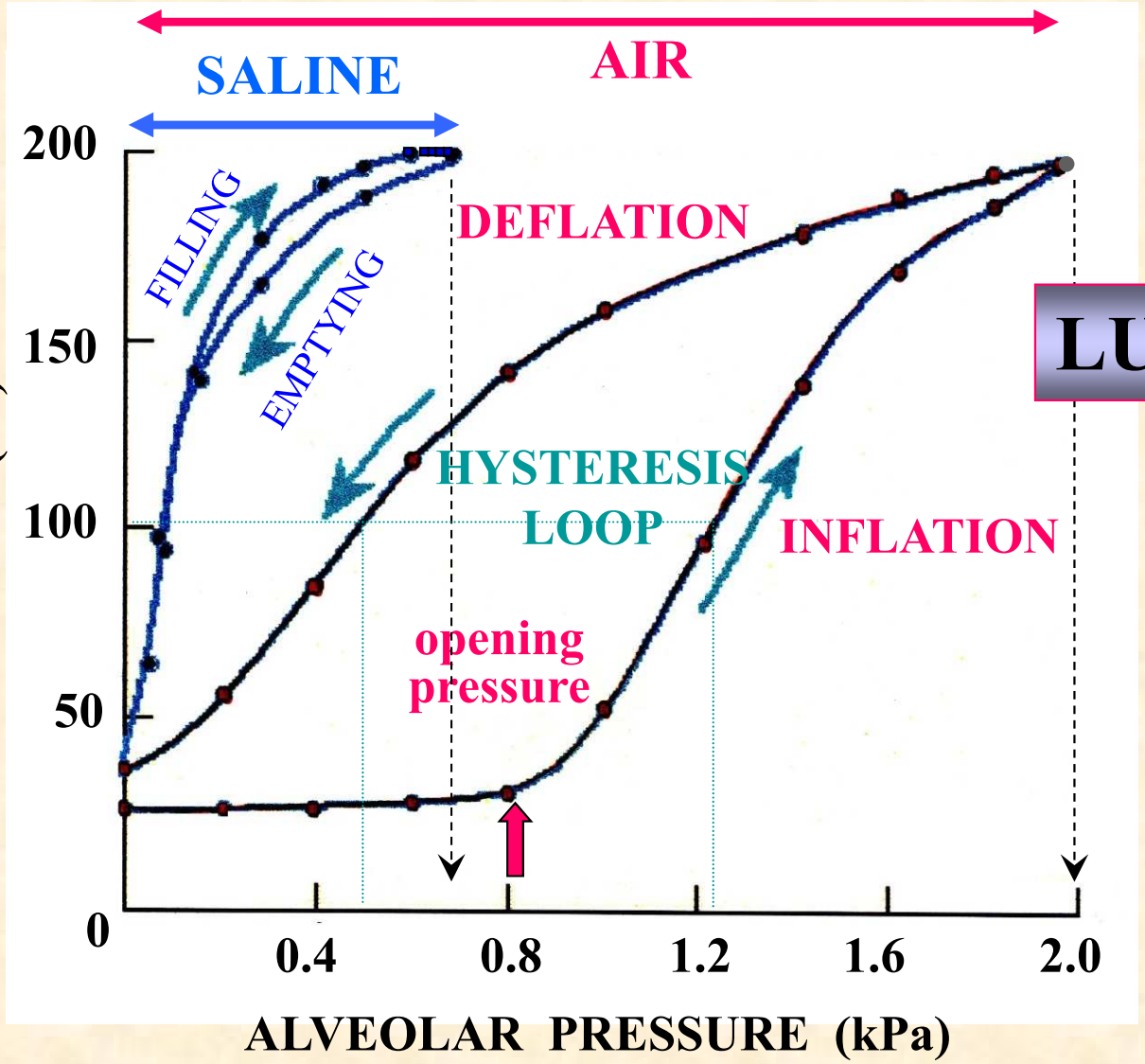
**INSPIRATION** $P_A < P_{ATM}$ | **EXPIRATION** $P_A > P_{ATM}$






$P_A$ **ALVEOLAR (INTRAPULMONARY, LUNG)**

$P_{PL}$ **INTRAPLEURAL (INTRATHORACIC)**

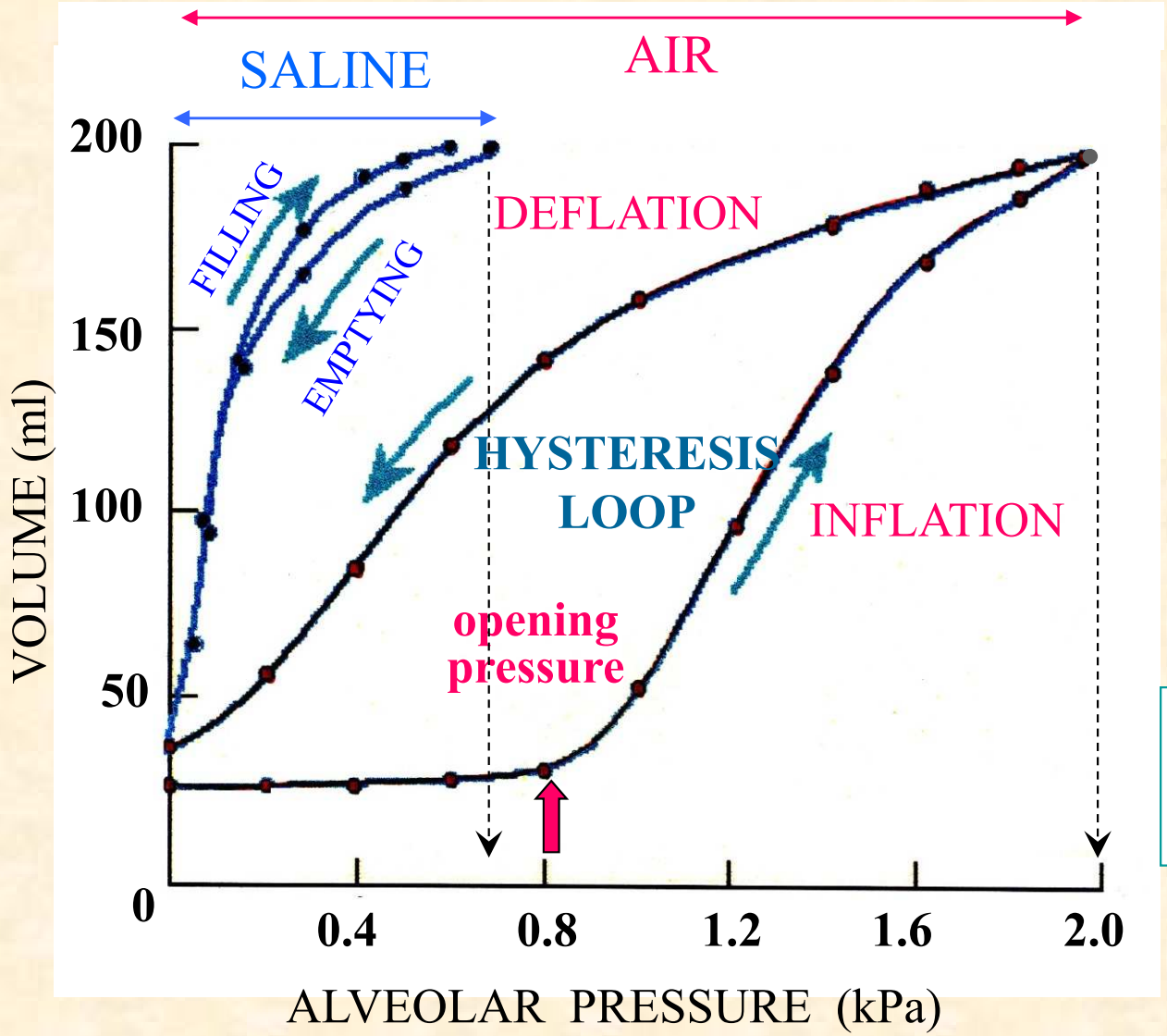



# LUNGS ELASTICITY

1 *kPa* = 7.5 *mm Hg*

## LUNGS ELASTICITY

- **INHERENT TISSUE ELASTICITY**
  **(elastin and collagen fibres)**
- **SURFACE TENSION FORCES**
  **air-liquid interface in alveoli**

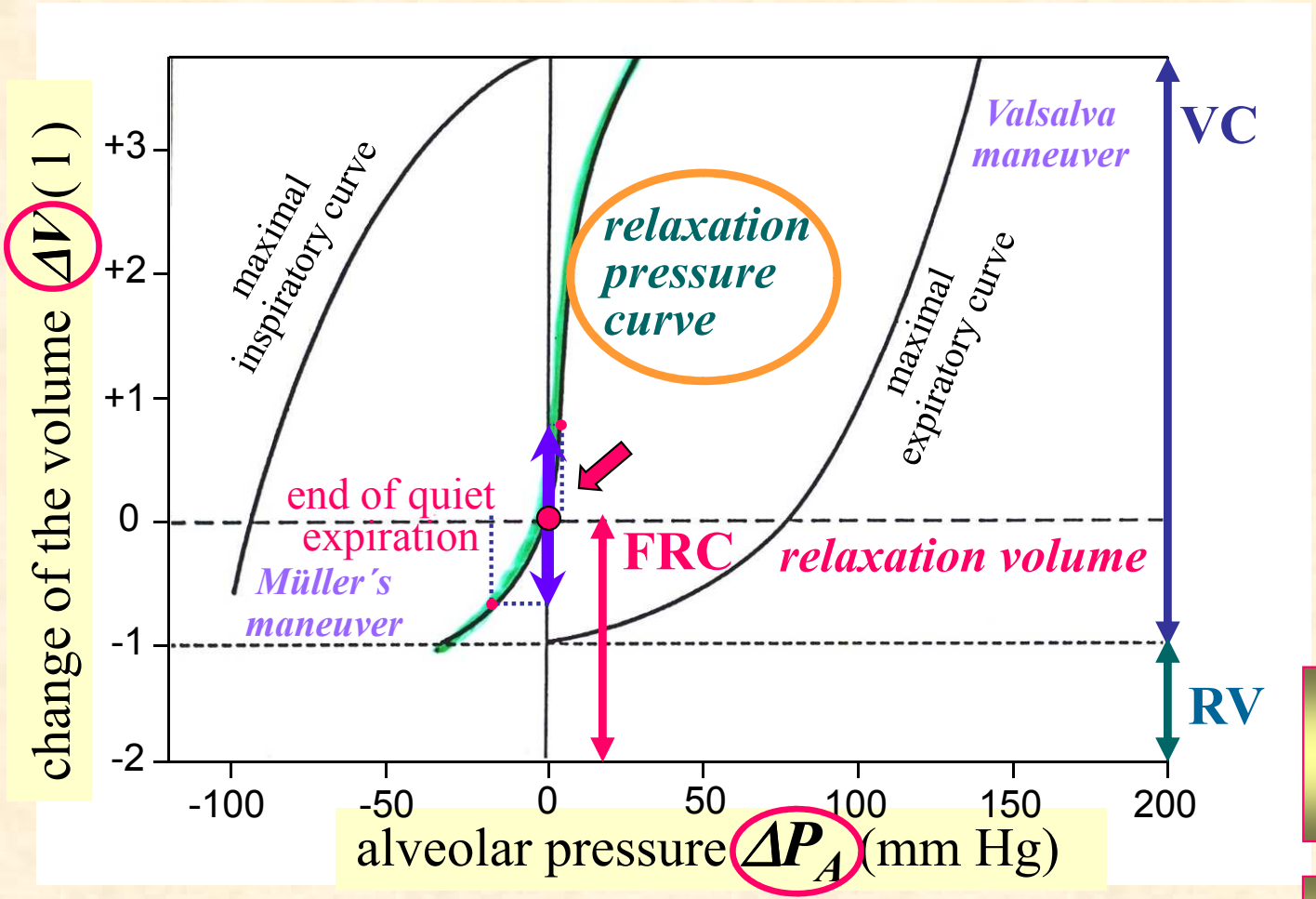



**Factors involved in HYSTERESIS LOOP**

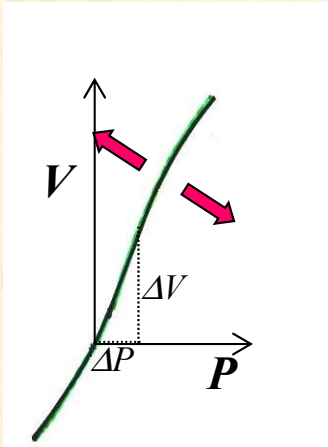
- **LAPLACE LAW (responsible for high opening pressure of alveoli)**
- **Dynamic changes** in the **DENSITY of surfactant molecules** during **INSPIRATION** and **EXPIRATION**

# COMPLIANCE (VOLUME STRETCHABILITY)

## STATIC MEASUREMENT IN CLOSED SYSTEM




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




***compliance is decreased***
↑ ***stiffness of the tissue***

***compliance is increased***
↓ ***stiffness of the tissue***

**TOTAL RESPIRATORY SYSTEM**
**(lungs and chest)**

# 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
- LUNG VOLUMES
- FUNCTIONAL INVESTIGATION
- CHARACTERISTIC PRESSURES

III **ACTIVE AND PASSIVE FORCES**

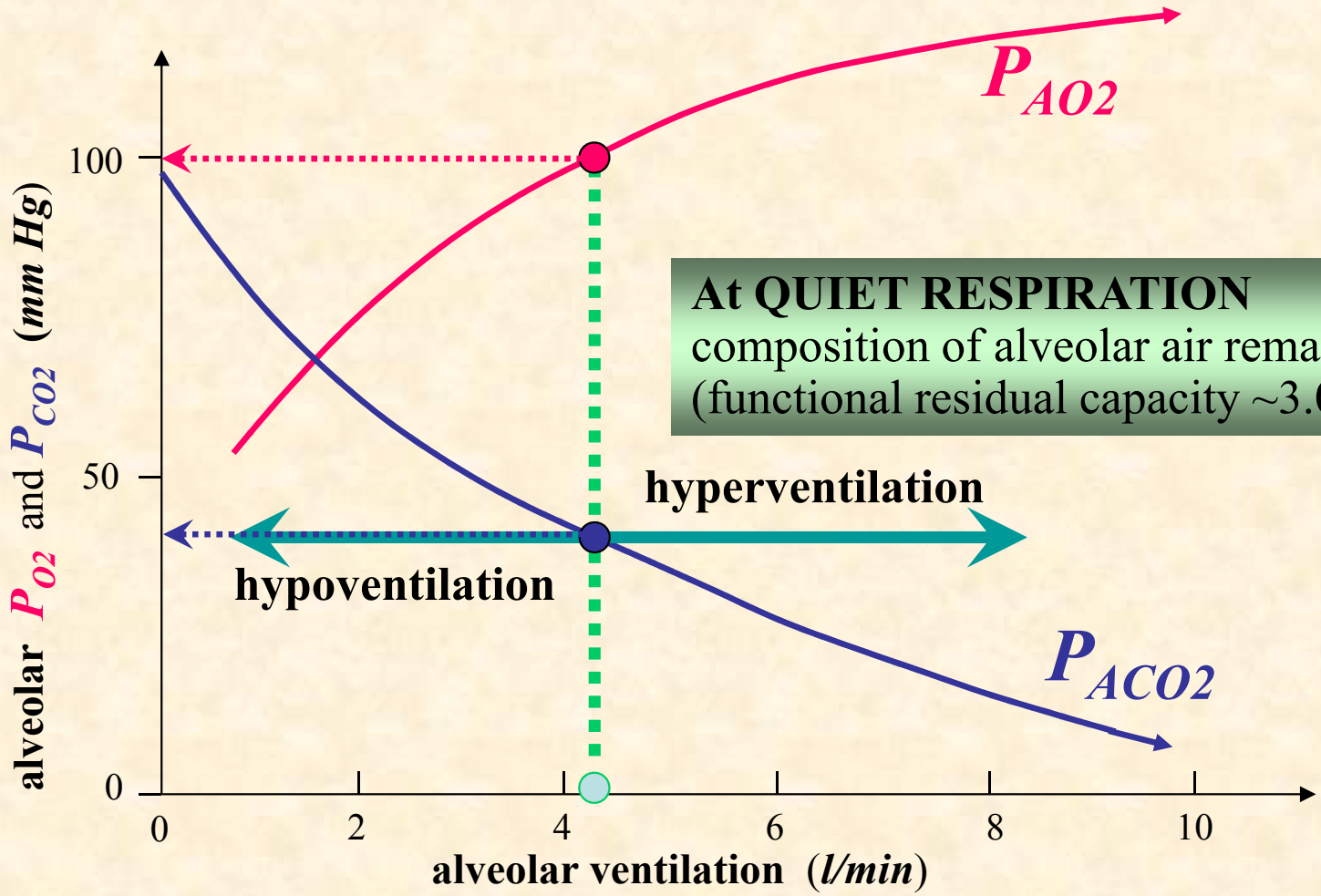
- RESPIRATORY MUSCLES
- LUNGS ELASTICITY
- COMPLIANCE
- WORK OF BREATHING

➡ IV **COMPOSITION OF ALVEOLAR AIR**

V **ALVEOLAR-CAPILLARY MEMBRANE**

VI **TRANSPORT OF GASSES ($O_2$ and $CO_2$)**

# Alveolar $P_{O2}$ and $P_{CO2}$ at voluntary hypo- and hyperventilation




**At QUIET RESPIRATION**
composition of alveolar air remains constant
(functional residual capacity ~3.0 *l*)

**hyperventilation → hypocapnia → respiratory alkalosis**

**hypoventilation → hypercapnia → respiratory acidosis**