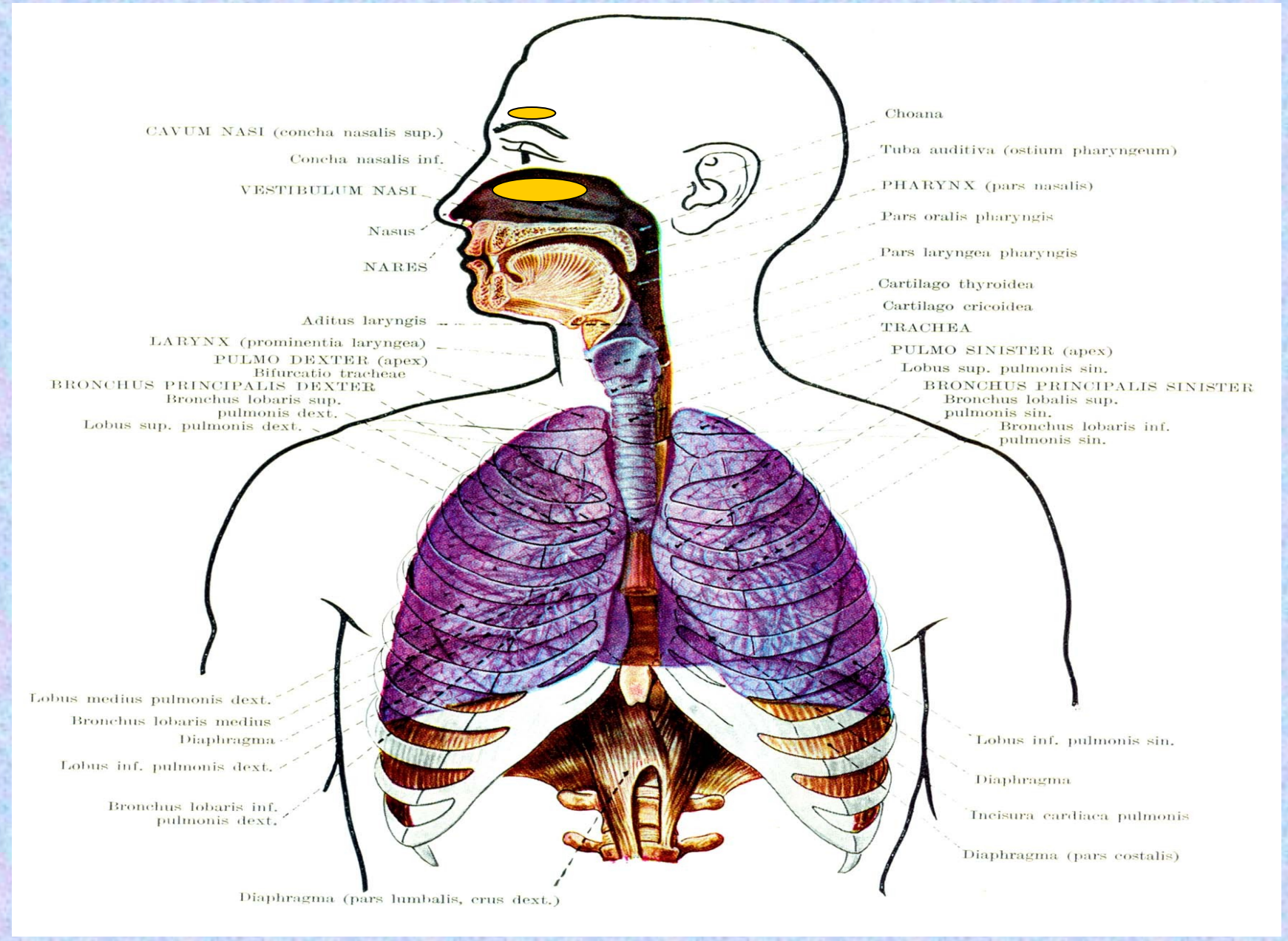
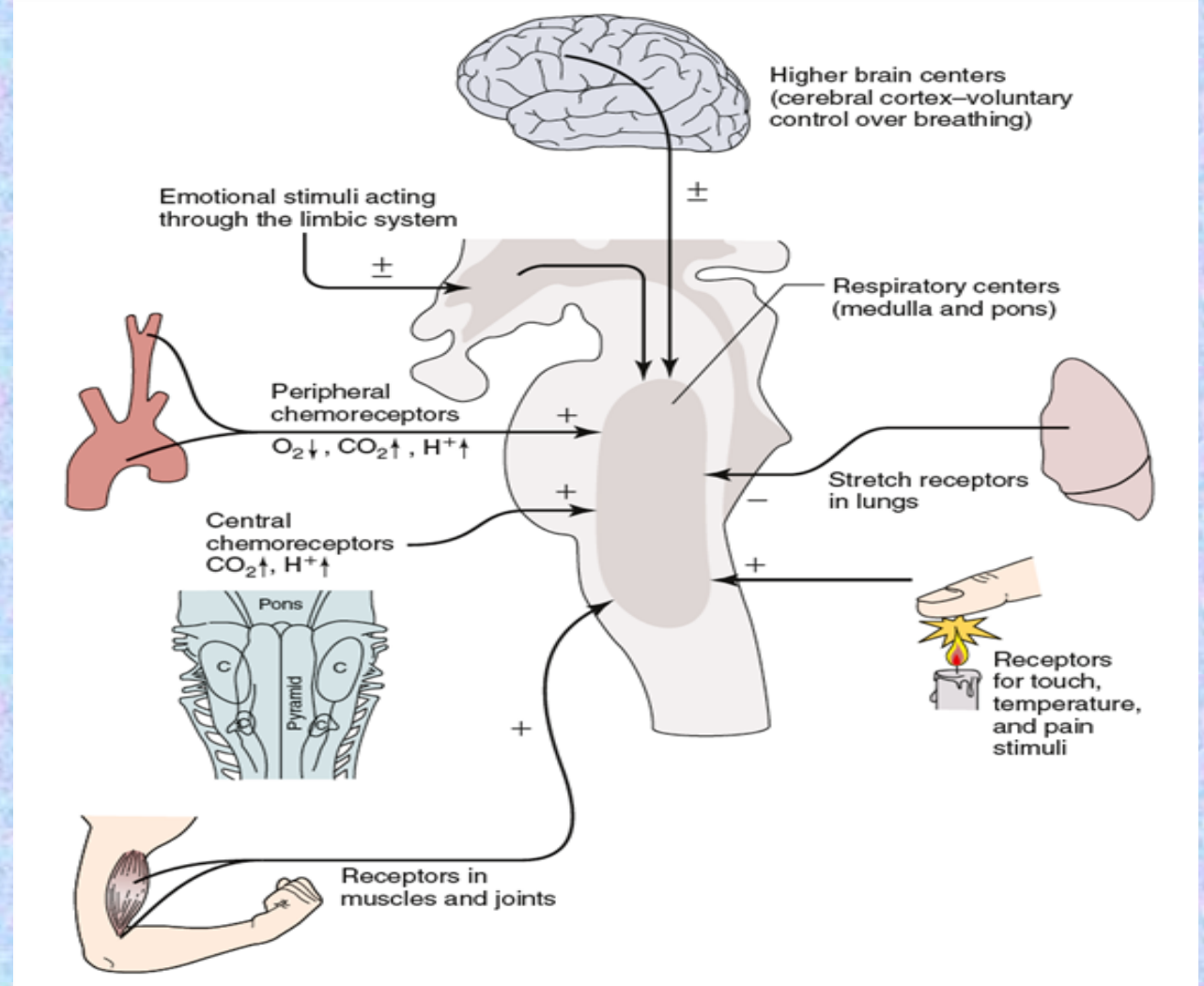
Respiratory system


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)

Regulation of breathing

Control of ventilation



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

- **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.

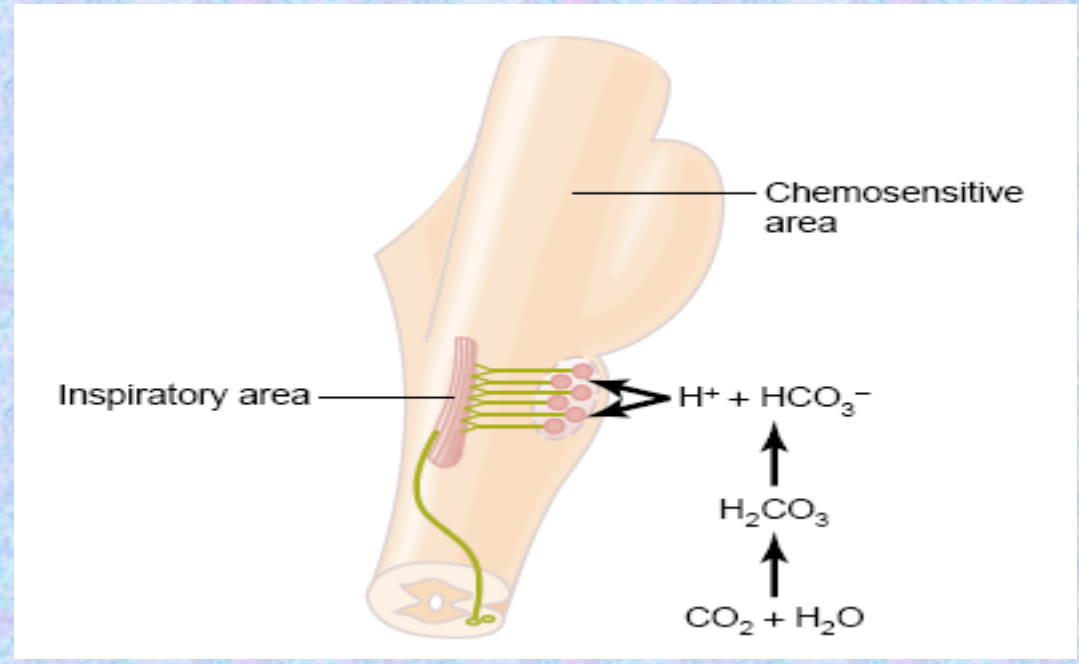
  - *Pontine respiratory group* - *pneumotaxic center* - dorsally placed on top of the pont, contributes to the frequency and depth of breathing; affects the activity of respiratory neurons in the medulla oblongata.

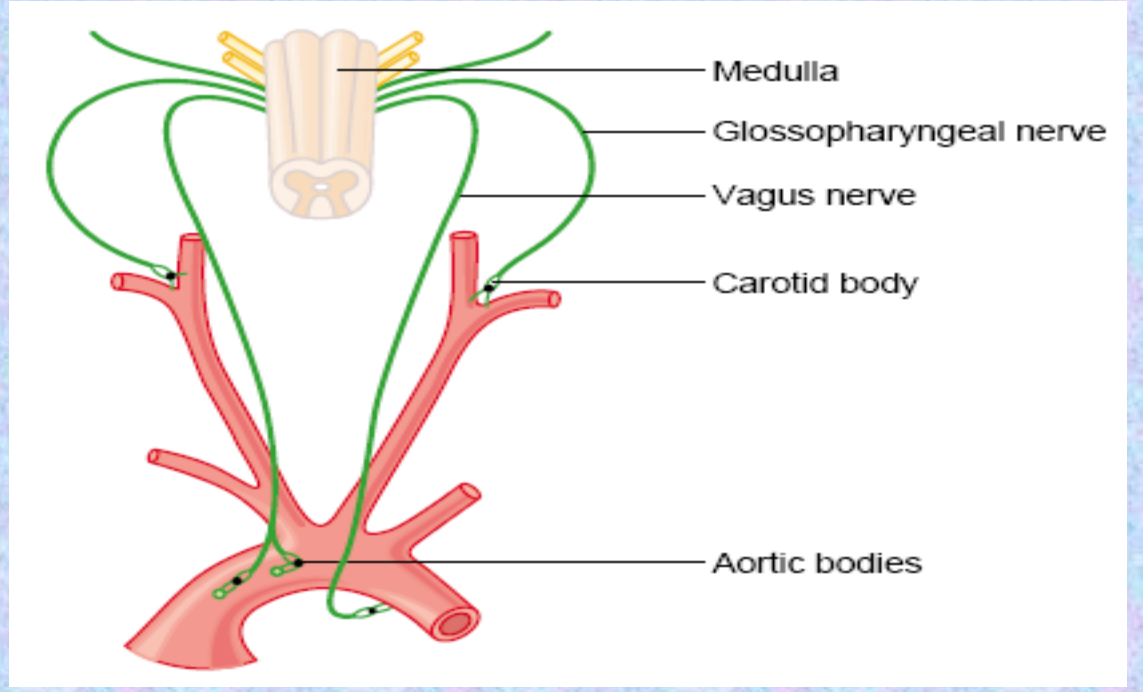
Chemical factors affecting the respiratory center:

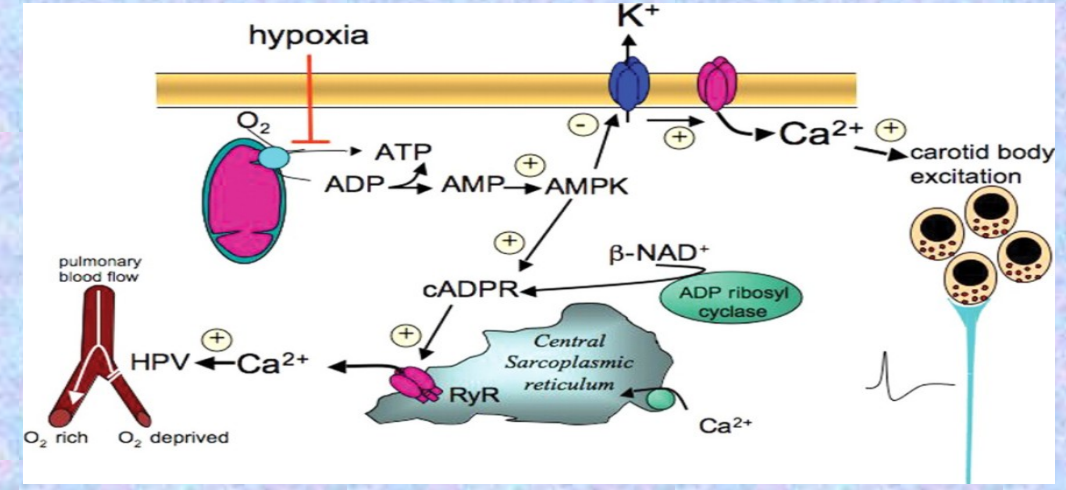
Central chemoreceptors

- 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

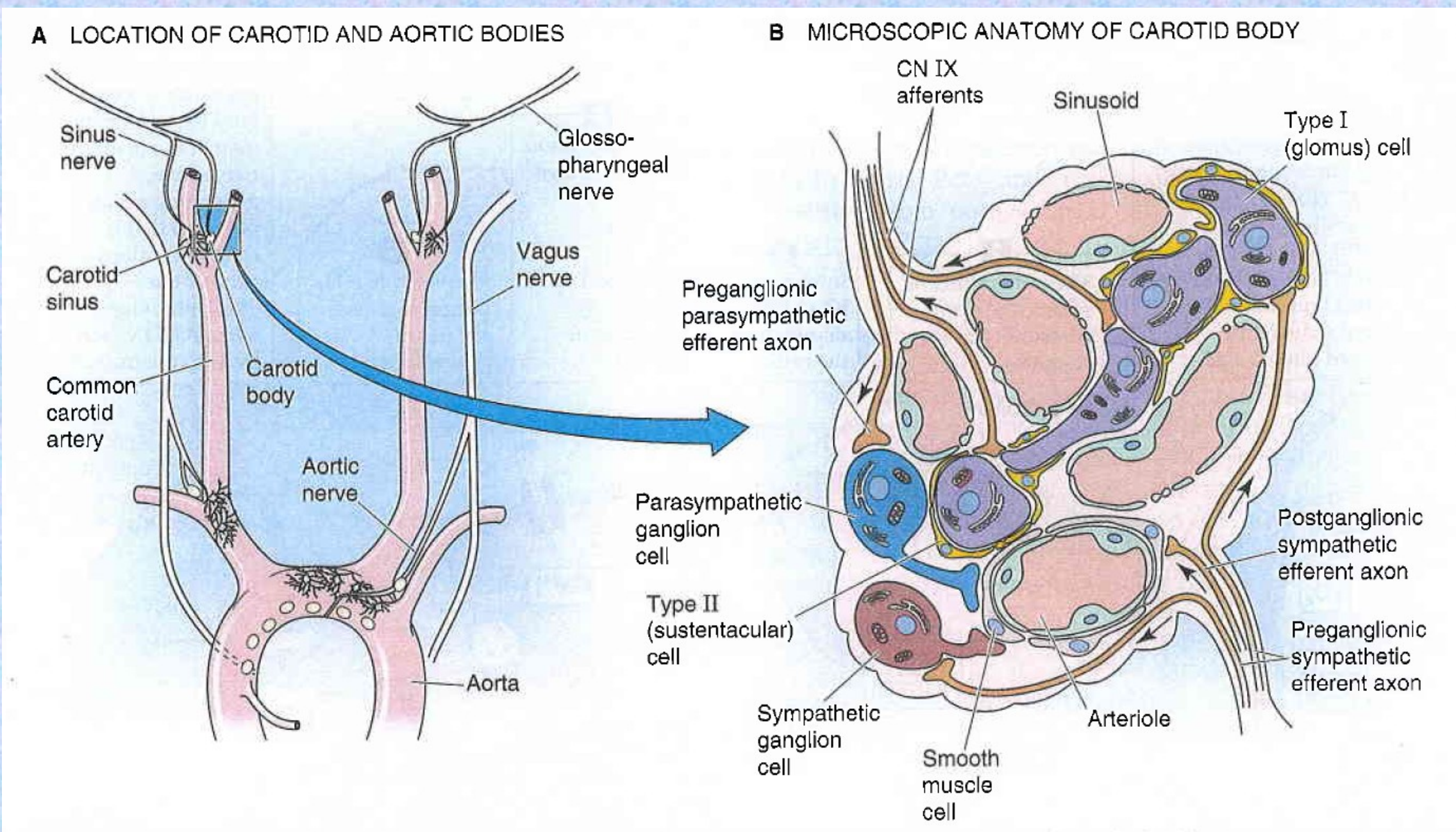
– located in the aortic and carotid bodies

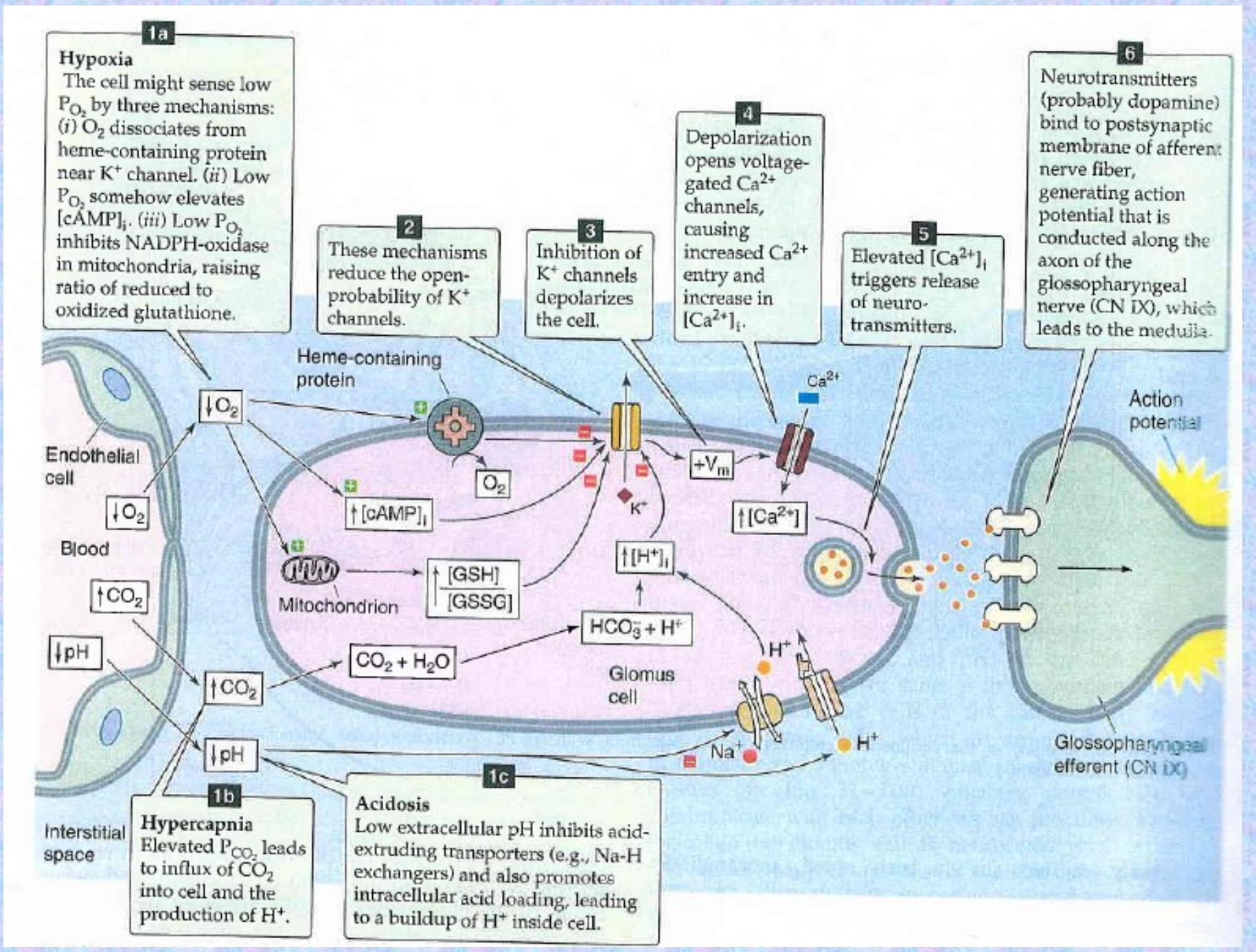
-primarily sensitive to decrease in arterial pO_2, particularly to decrease of O_2 under 10-13 kPa in the arterial blood.

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
$\uparrow [cAMP]_i$
K^+
$+V_m$
Ca^{2+}
$\uparrow [Ca^{2+}]$
Action potential
Endothelial cell
$\downarrow O_2$
Blood
$\uparrow CO_2$
$\downarrow pH$
Mitochondrion
$\uparrow \frac{[GSH]}{[GSSG]}$
$\uparrow [H^+]_i$
$HCO_3^- + H^+$
$CO_2 + H_2O$
$\uparrow CO_2$
$\downarrow pH$
Glomus cell
H^+
Na^+
H^+
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

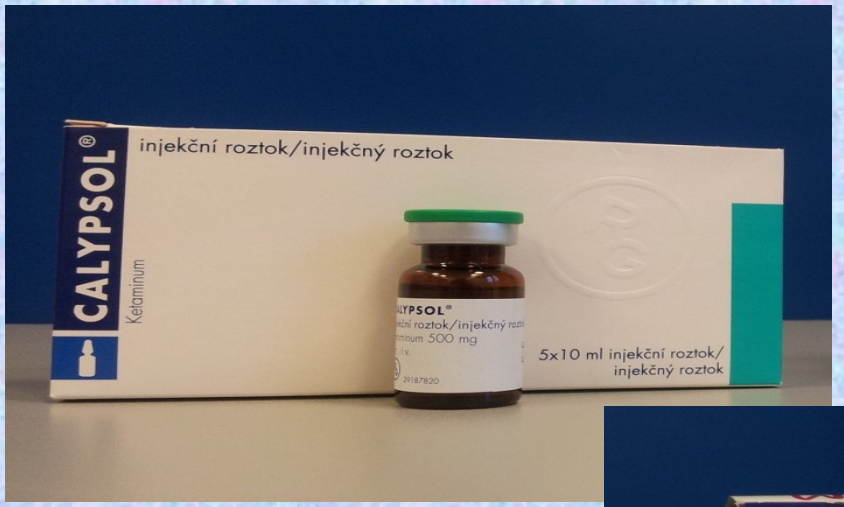
Irritants of **proprioreceptors of muscles, tendons** during active and pasive movements of limbs
Influenced activity of respiratory neurons (increase minute ventilation during work load)

Limbic system, hypothalamus – strong pain, emotion
Tractus corticospinalis =cortex – activated RC during work load

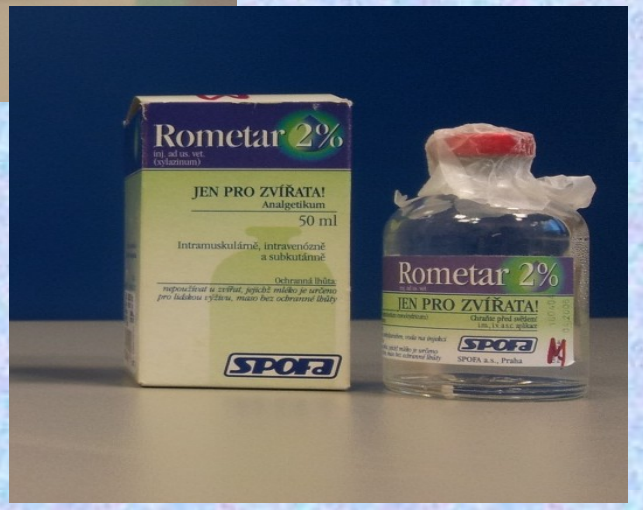
temperature

- Types of anesthaesia in animal experiments

inhalation



Intra-muscular application
= i.m.

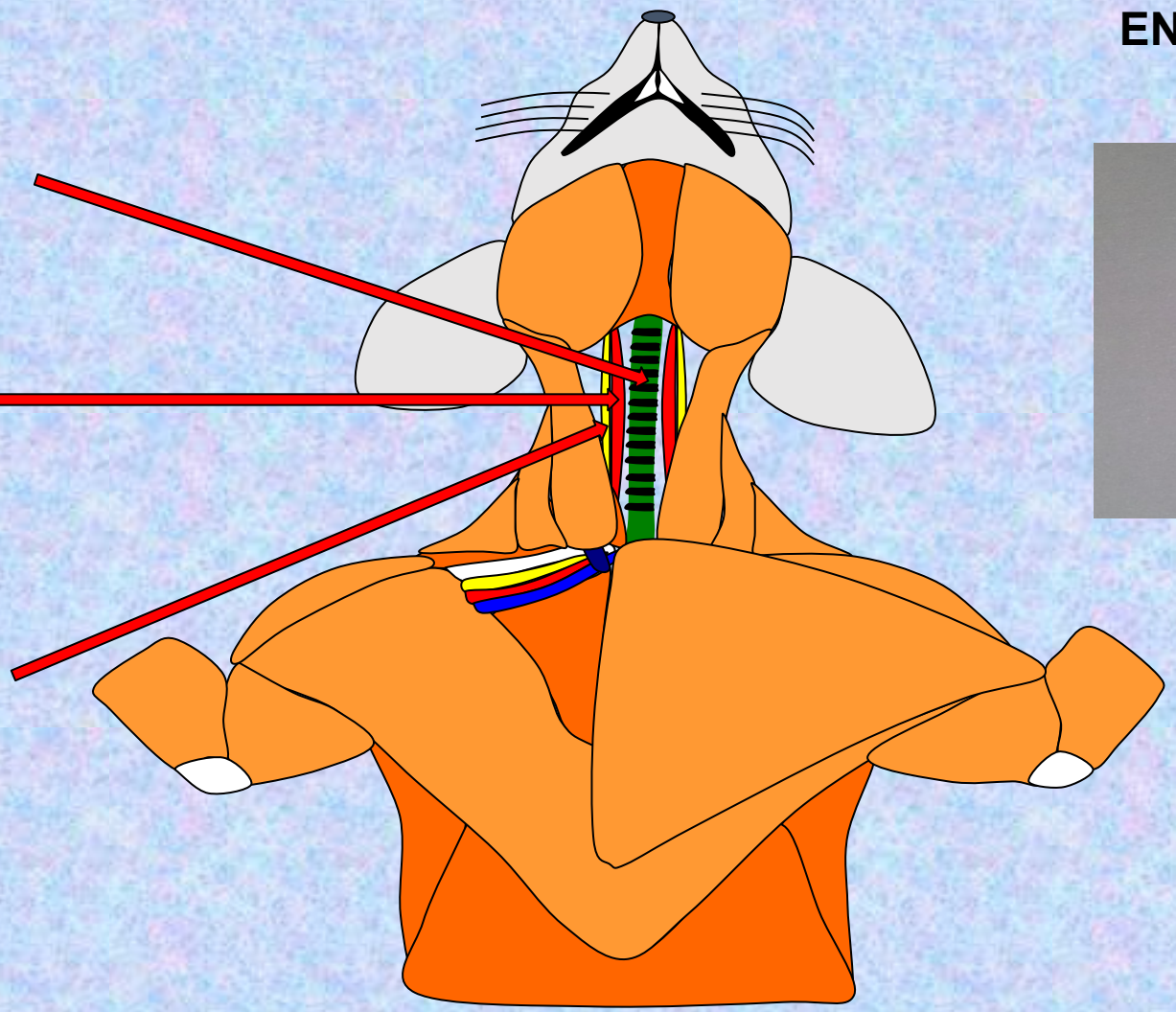


- Anatomy of neck in laboratory rat

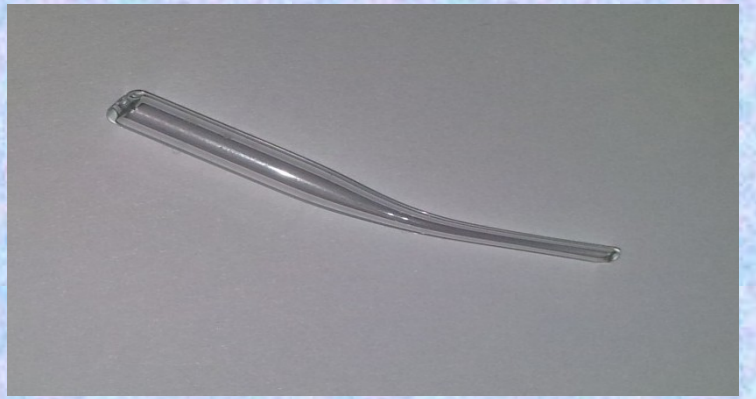
TRACHEA

A. CAROTIS

N. VAGUS

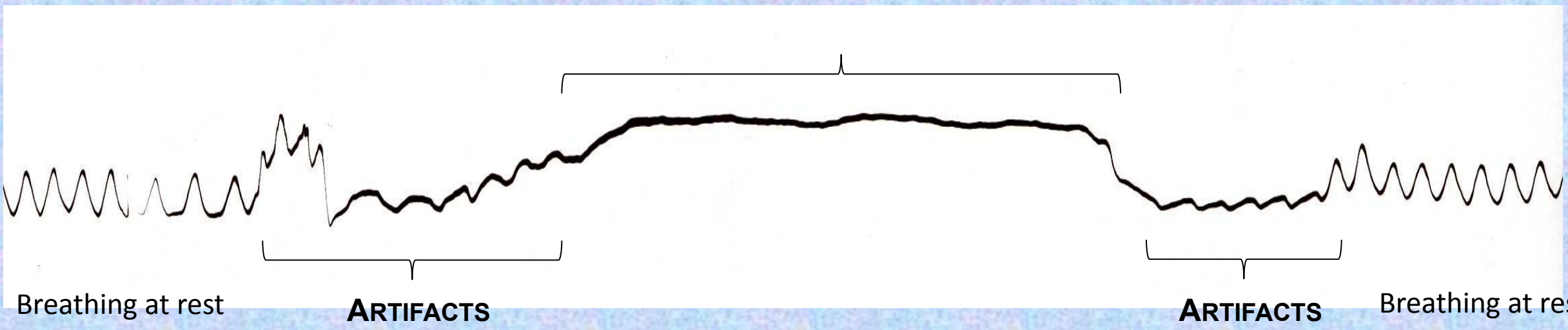


ENDOTRACHEAL CANNULA

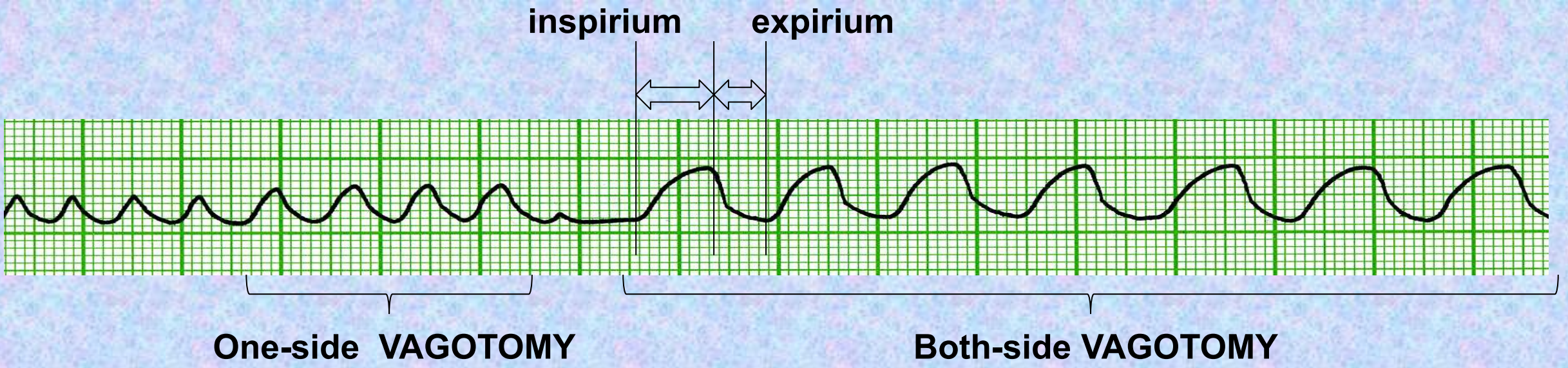


HERING-BREUER „inflation“ REFLEX

REFLEX STOP BREATHING



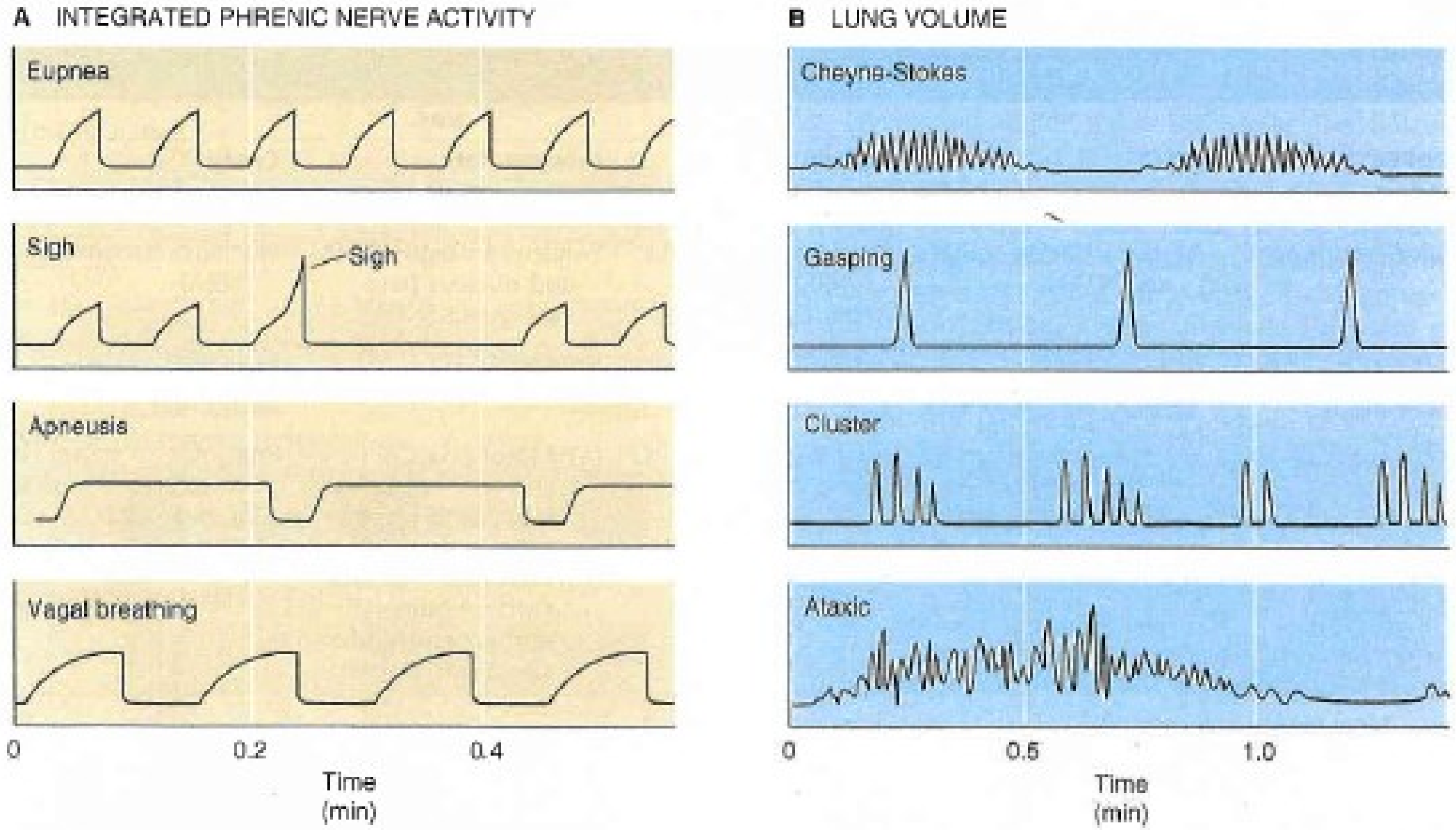
Changes of breathing after VAGOTOMY



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)

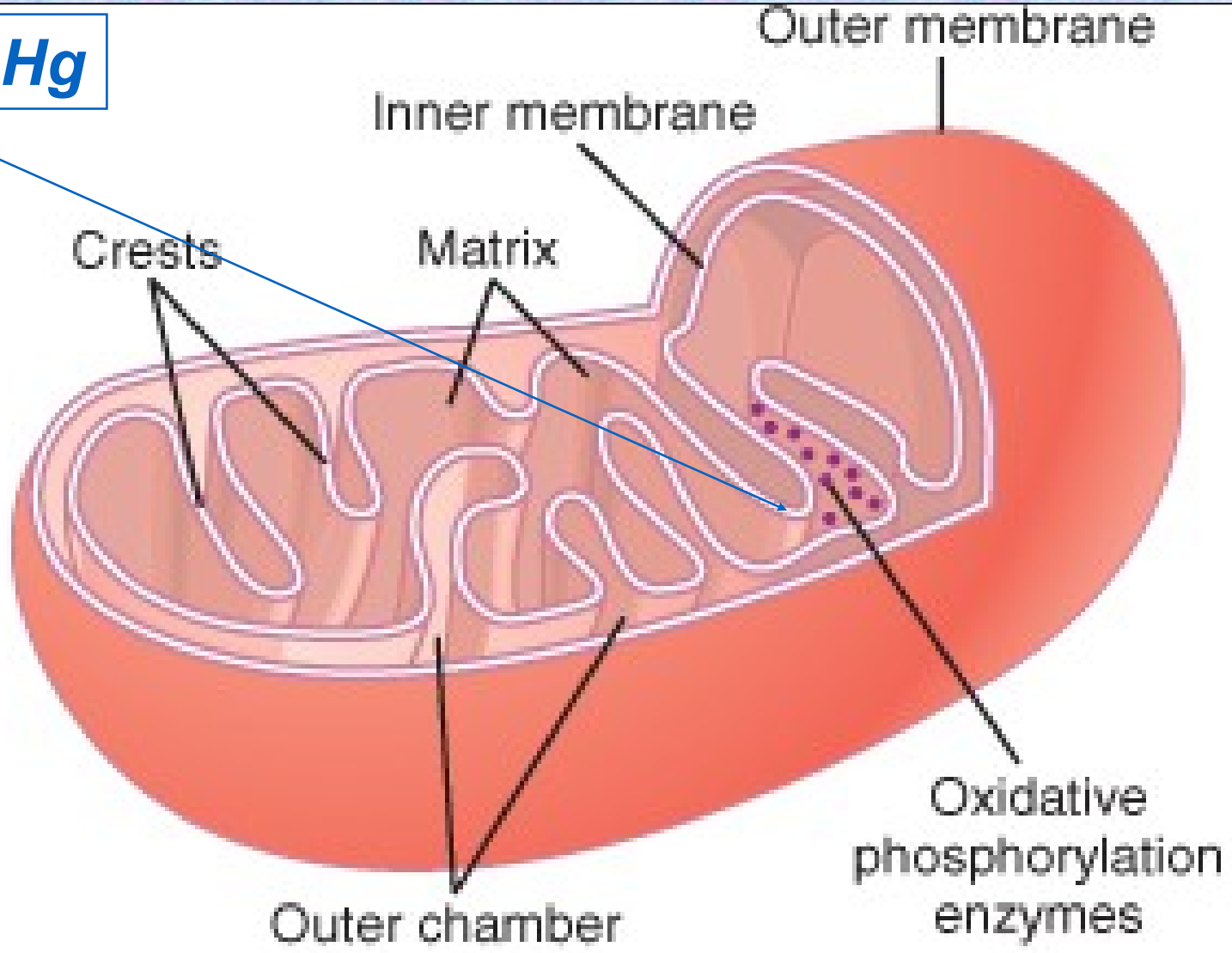
Hypoxia, hypoxemia

- 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)

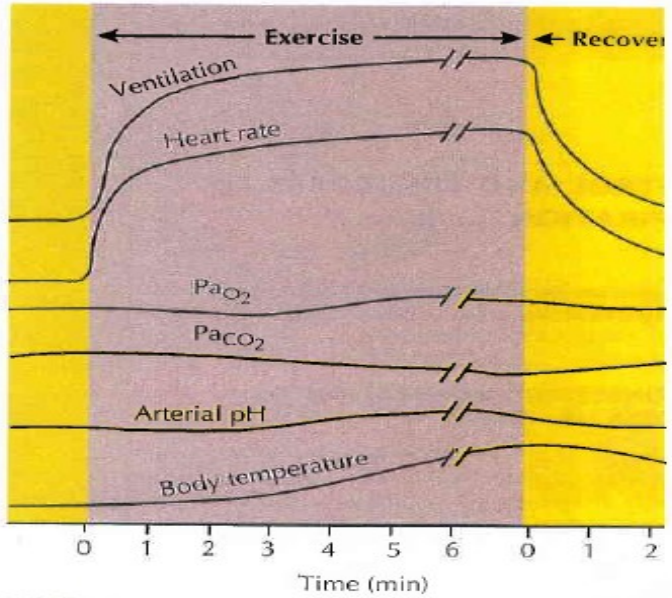
pO_2 = 1 mmHg



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.

Respiratory Response to Exercise

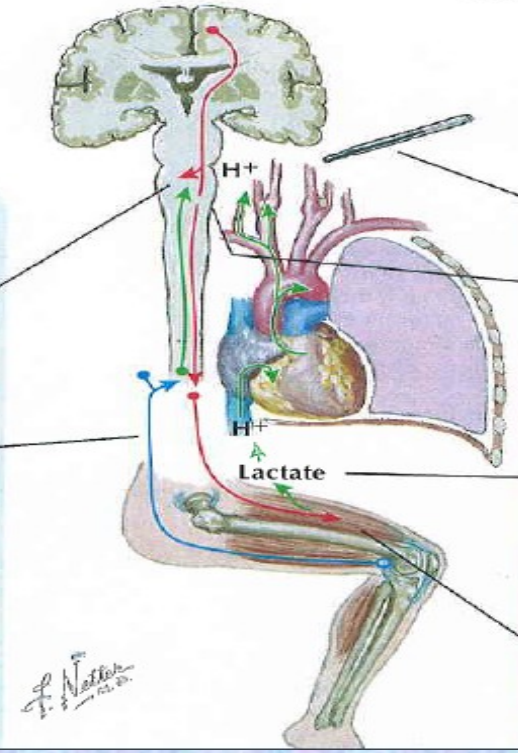


Factors that may account for initial abrupt rise and sharp terminal drop in ventilation

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 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^+ concentration of blood and CSF, thus affecting chemoreceptors

Possible metaboreceptc in exercising muscle

Other unknown factors

Travelling by aircraft

(On board aicraft is pressure as on 2000 m above sea 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