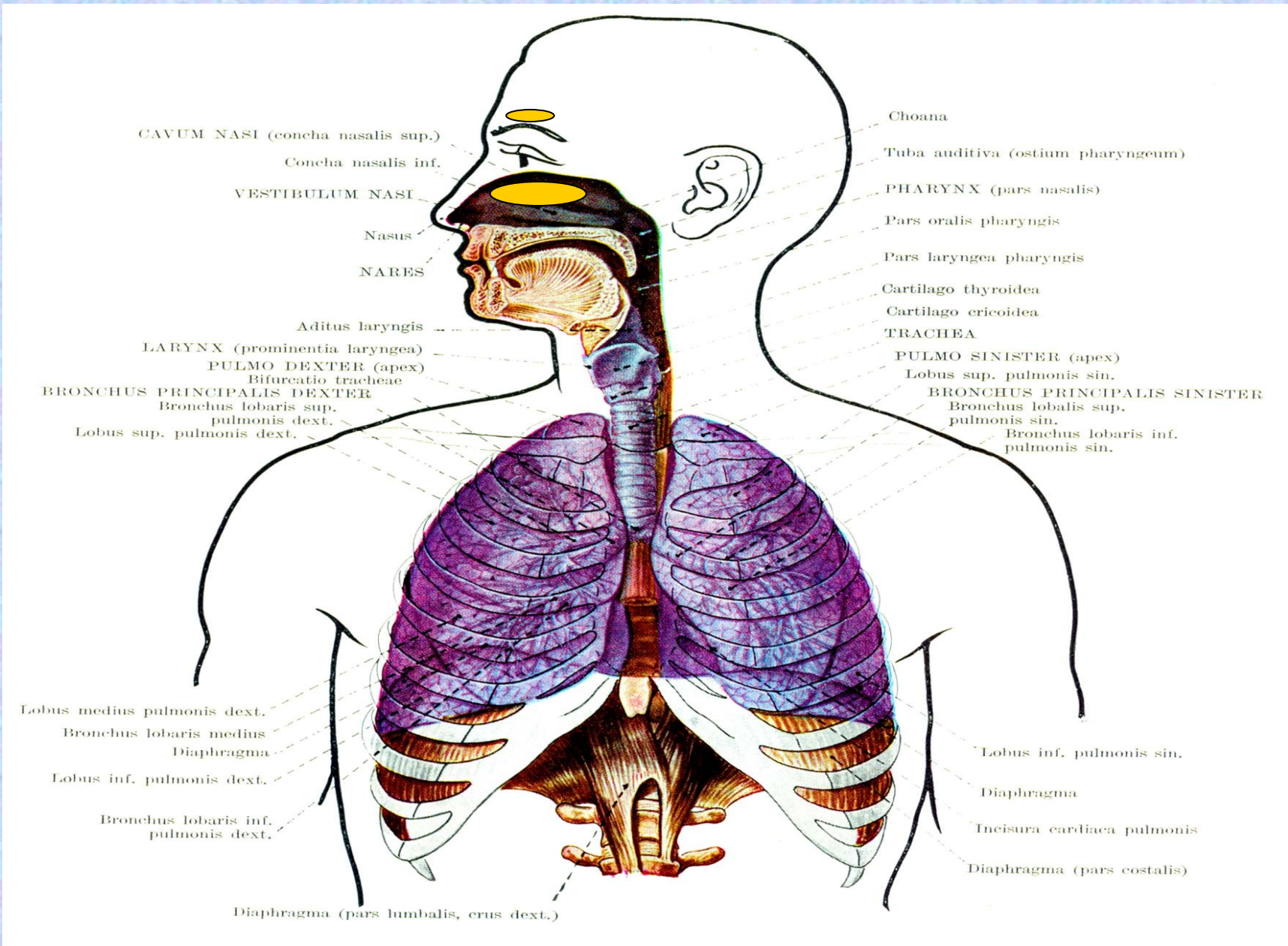
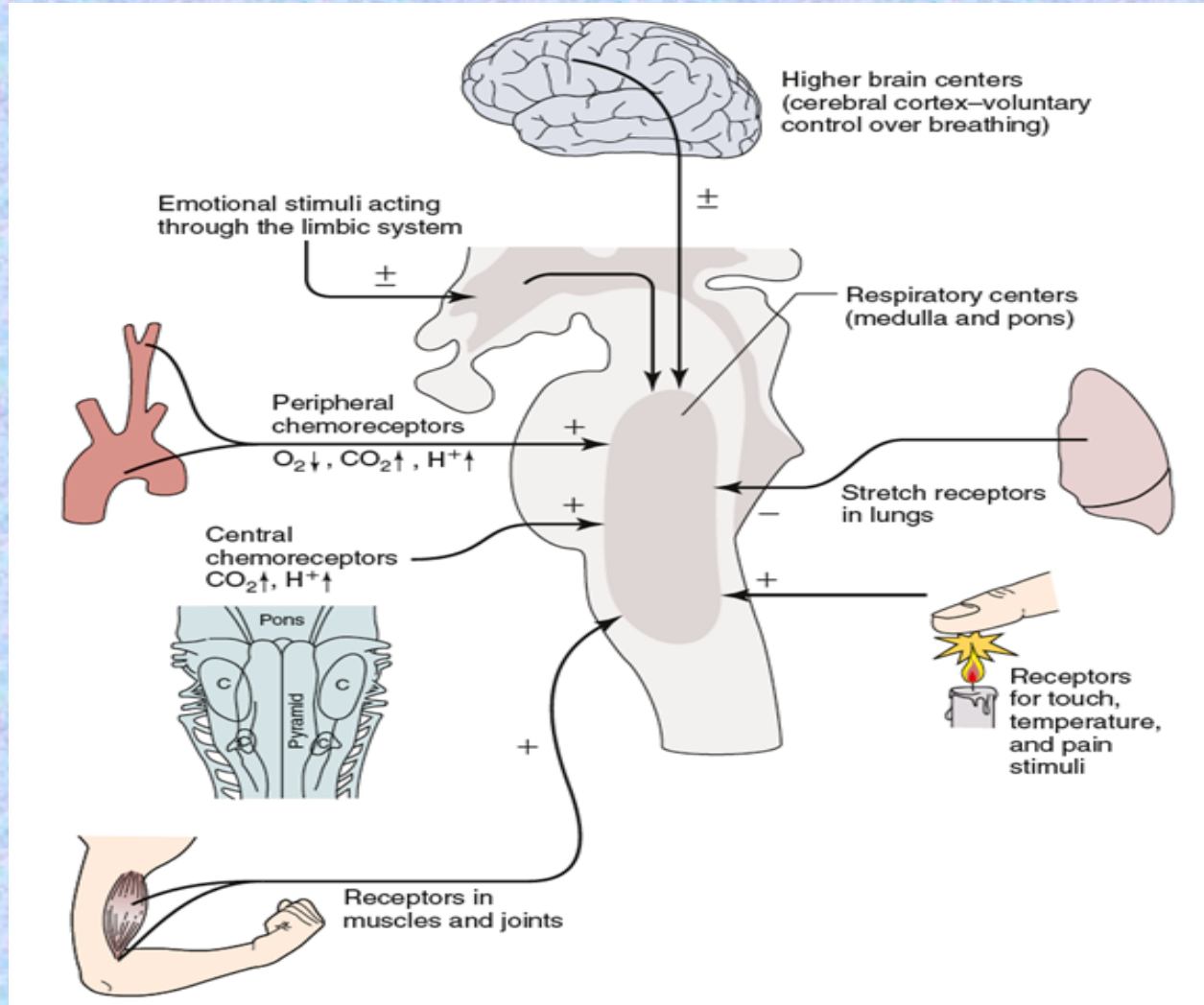


Respiratory system



Regulation of breathing

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.

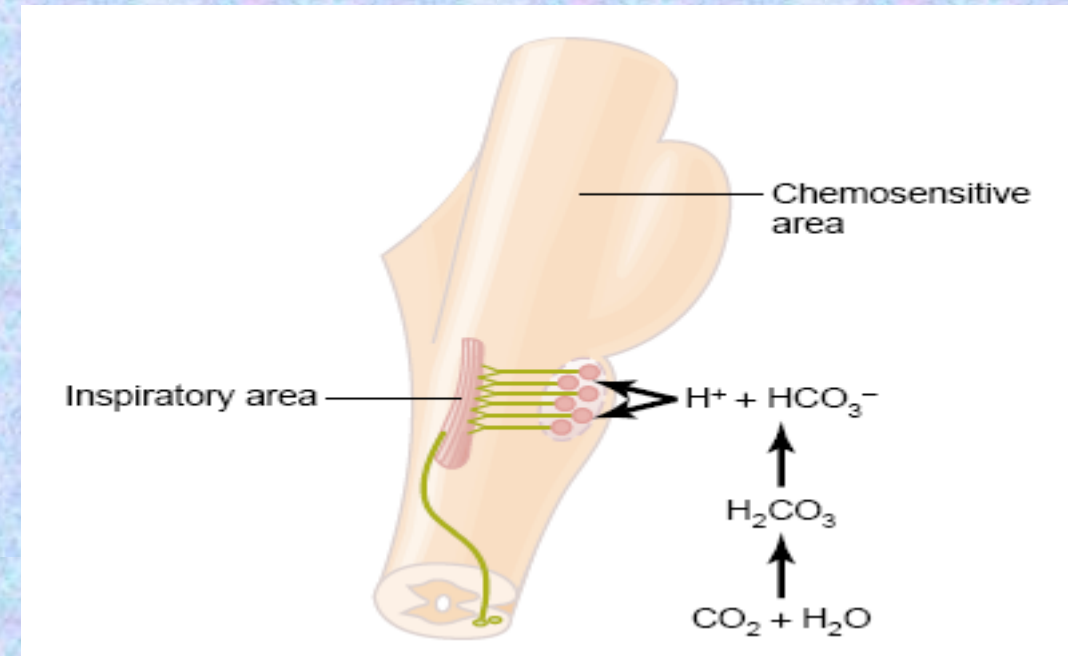
Chemical factors affecting the respiratory center:

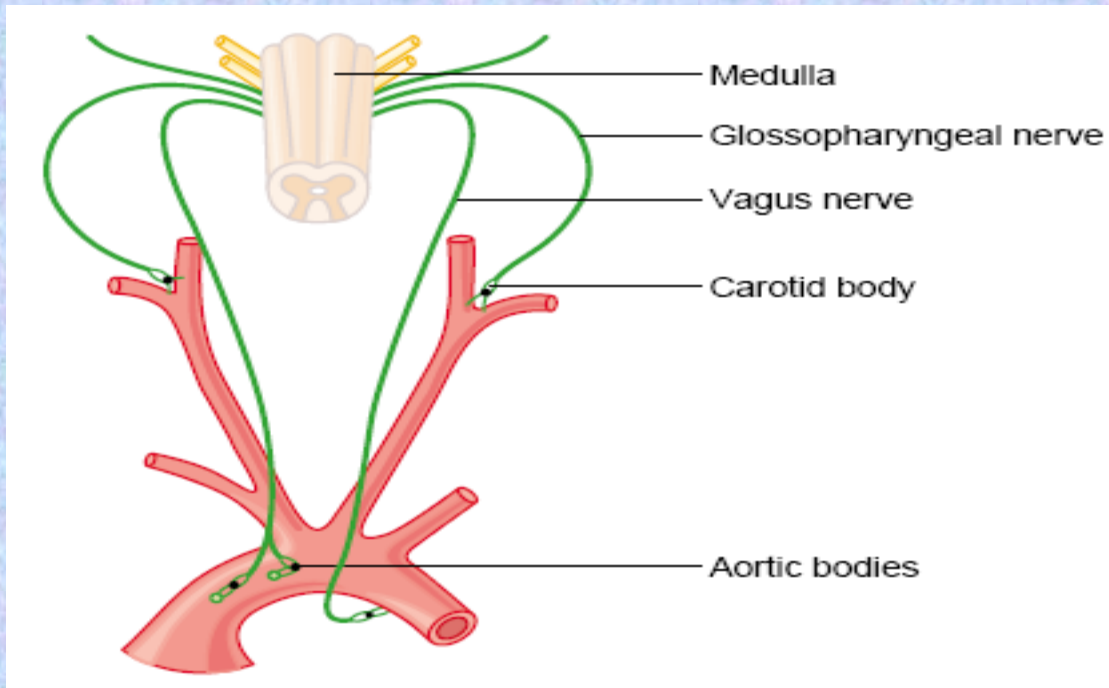
Central chemoreceptors

- 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)



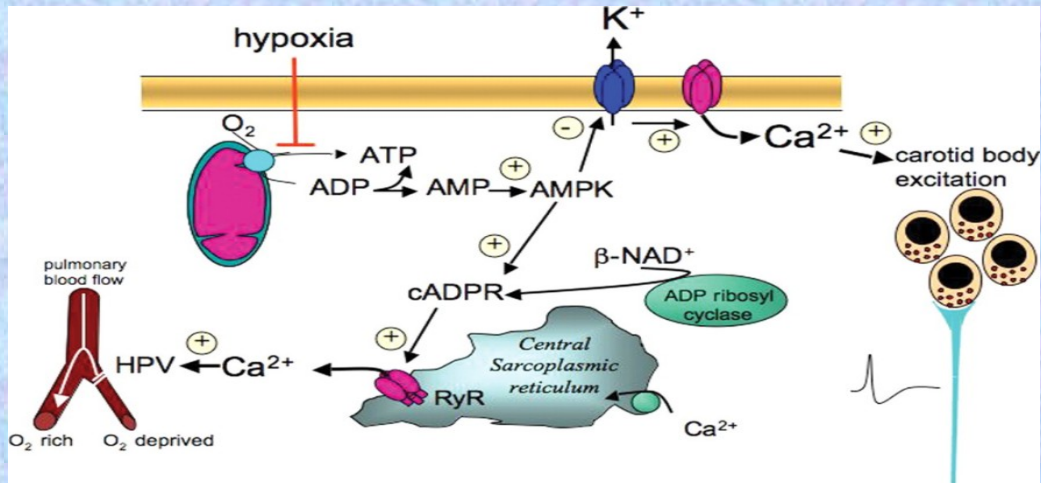


Peripheral chemoreceptors

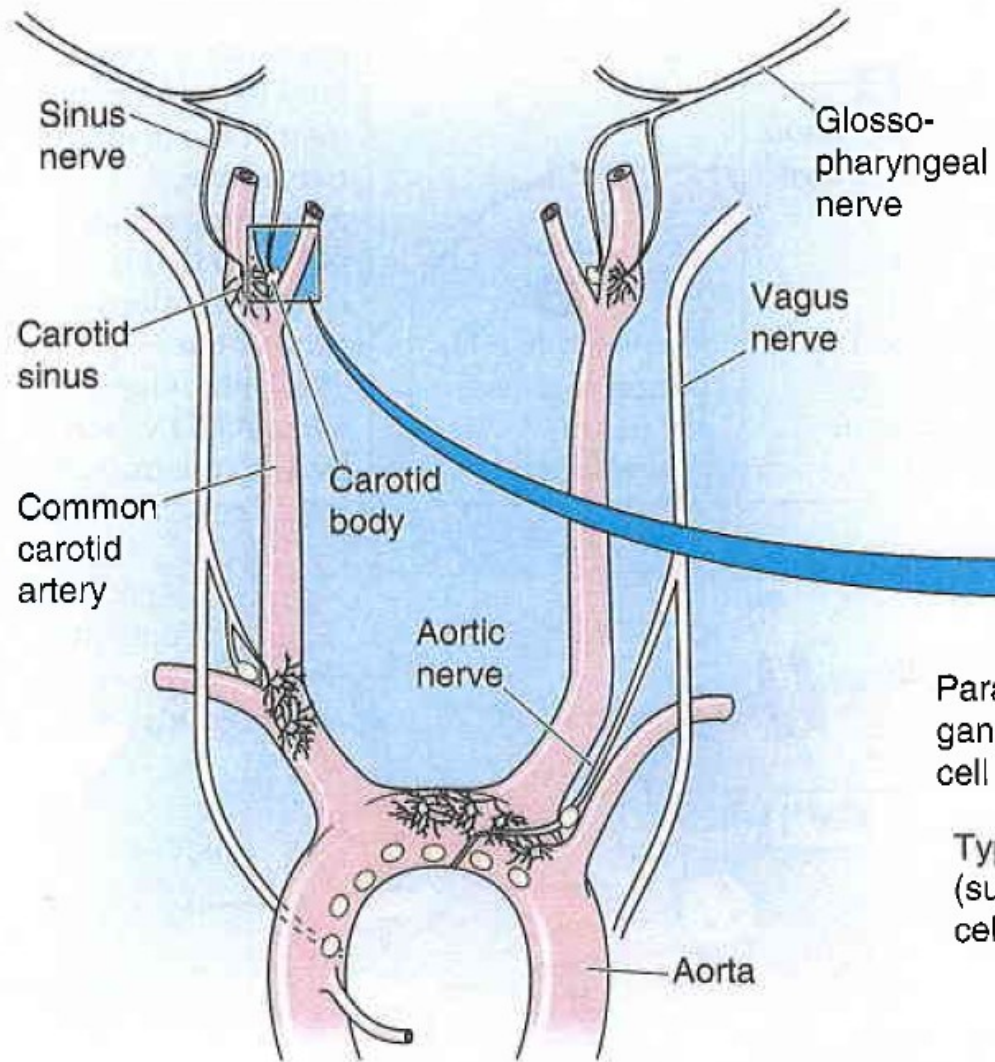
- located in the aortic and carotid bodies
- primarily sensitive to decrease in arterial pO_2 , particularly to decrease of O_2 under 10-13 kPa in the arterial blood.

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

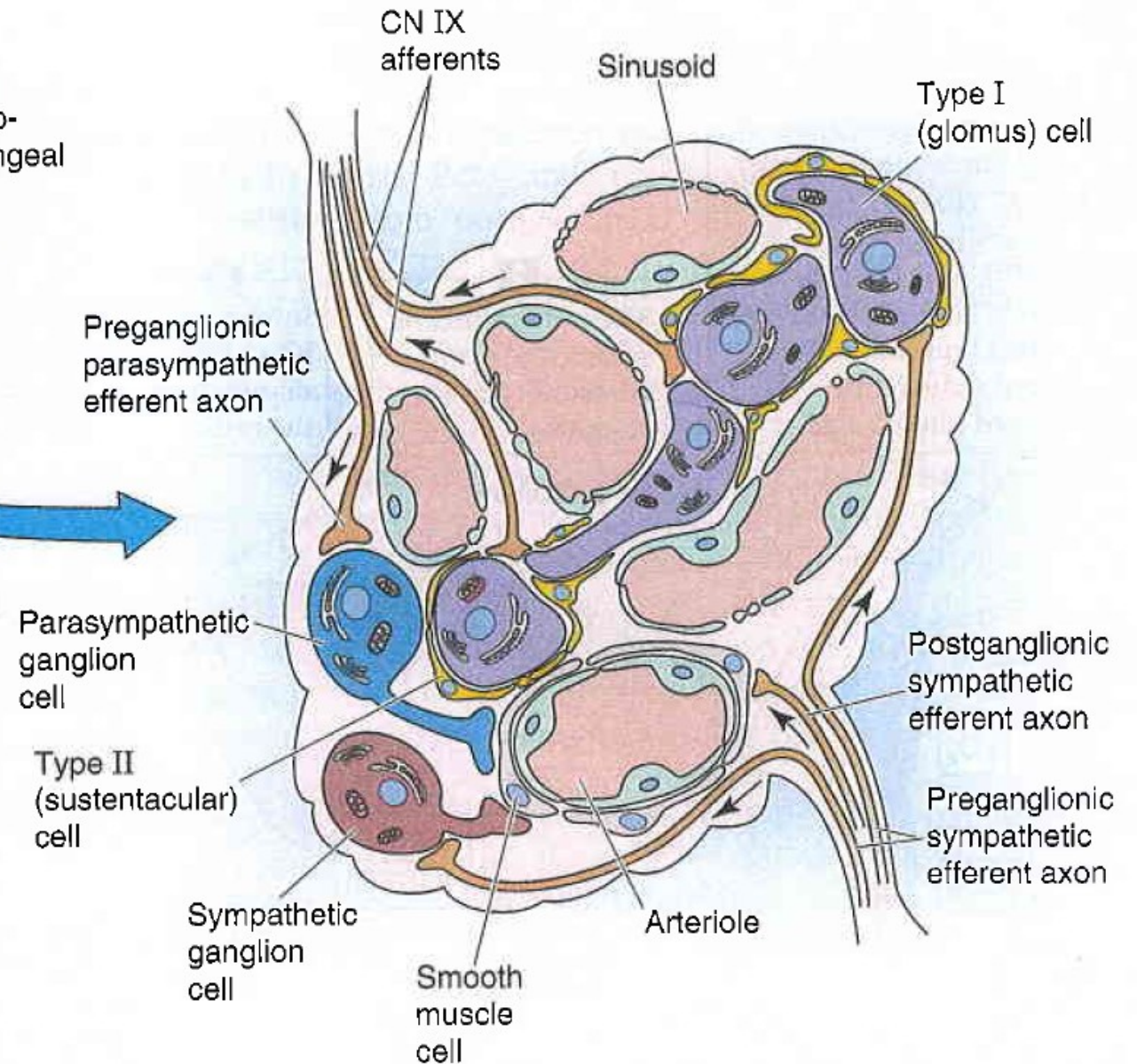
Mechanism of action: Decreased ATP production in mitochondria leads to depolarization of receptors membrane and to excitation of chemoreceptor

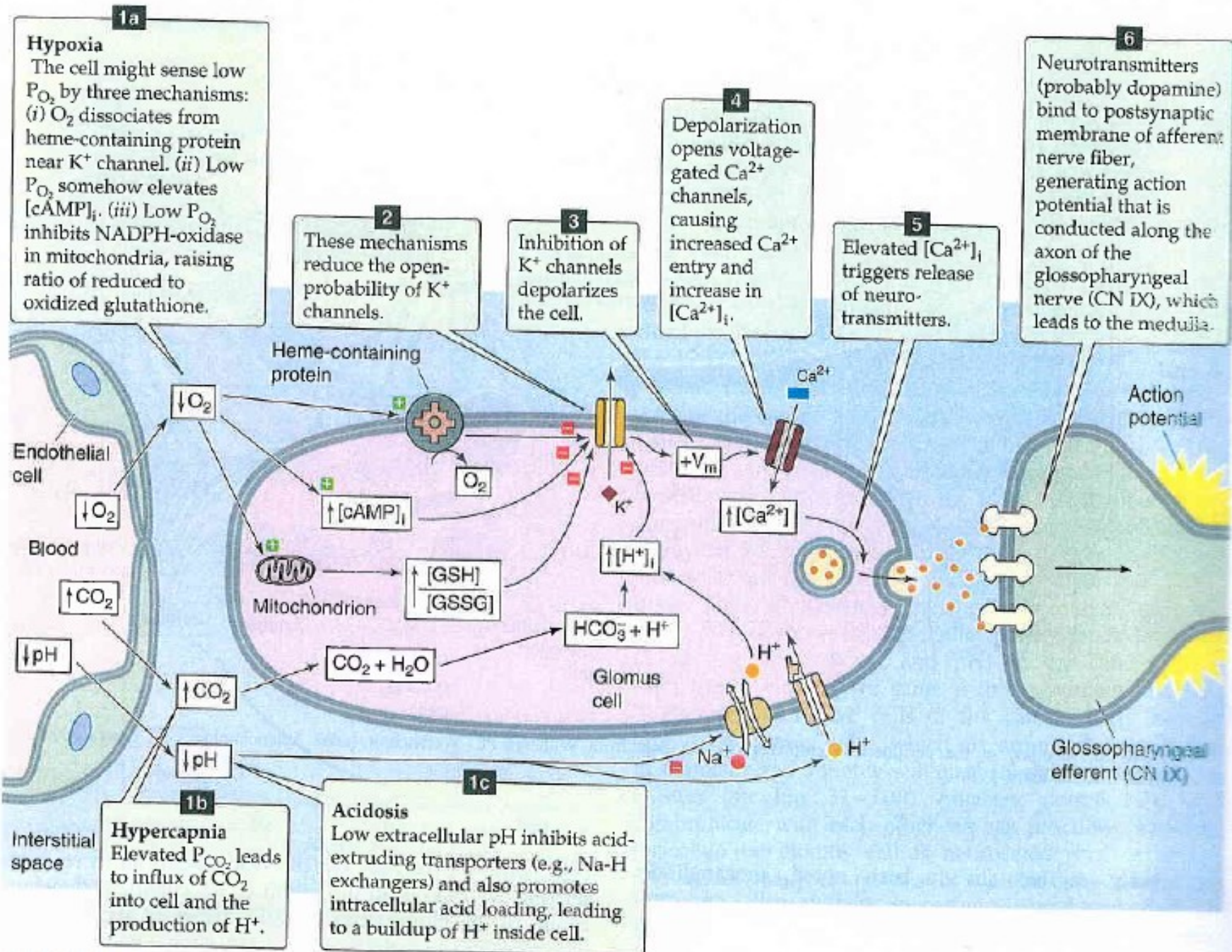


A LOCATION OF CAROTID AND AORTIC BODIES



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; 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

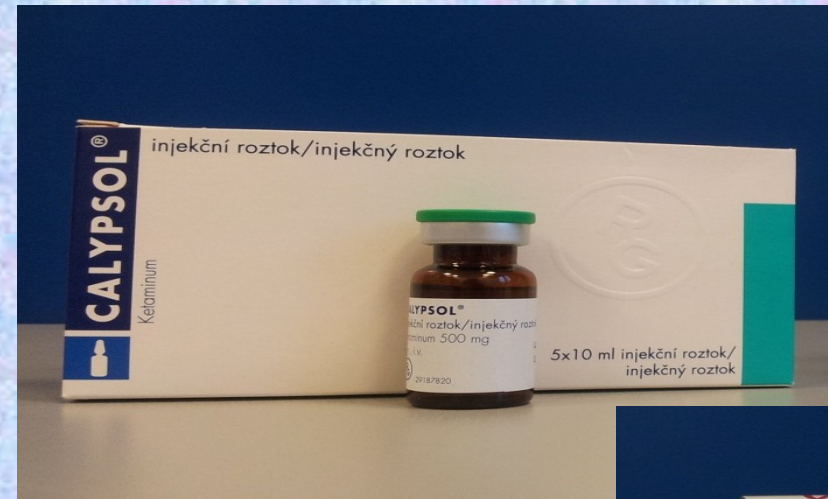
Tractus corticospinalis =cortex – activated RC during work load

temperature

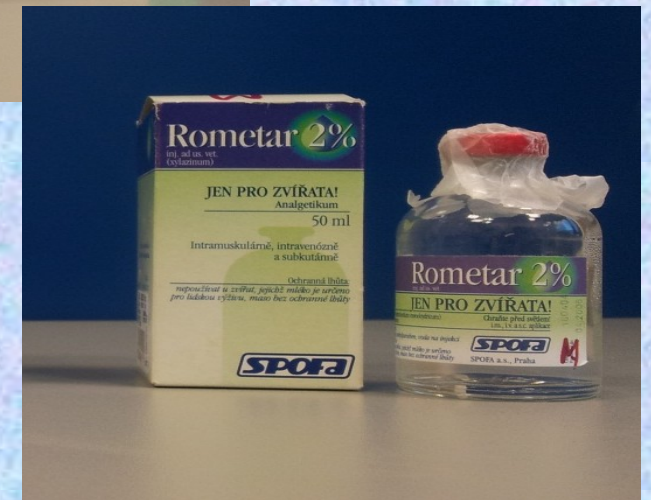
- Types of anaesthesia in animal experiments



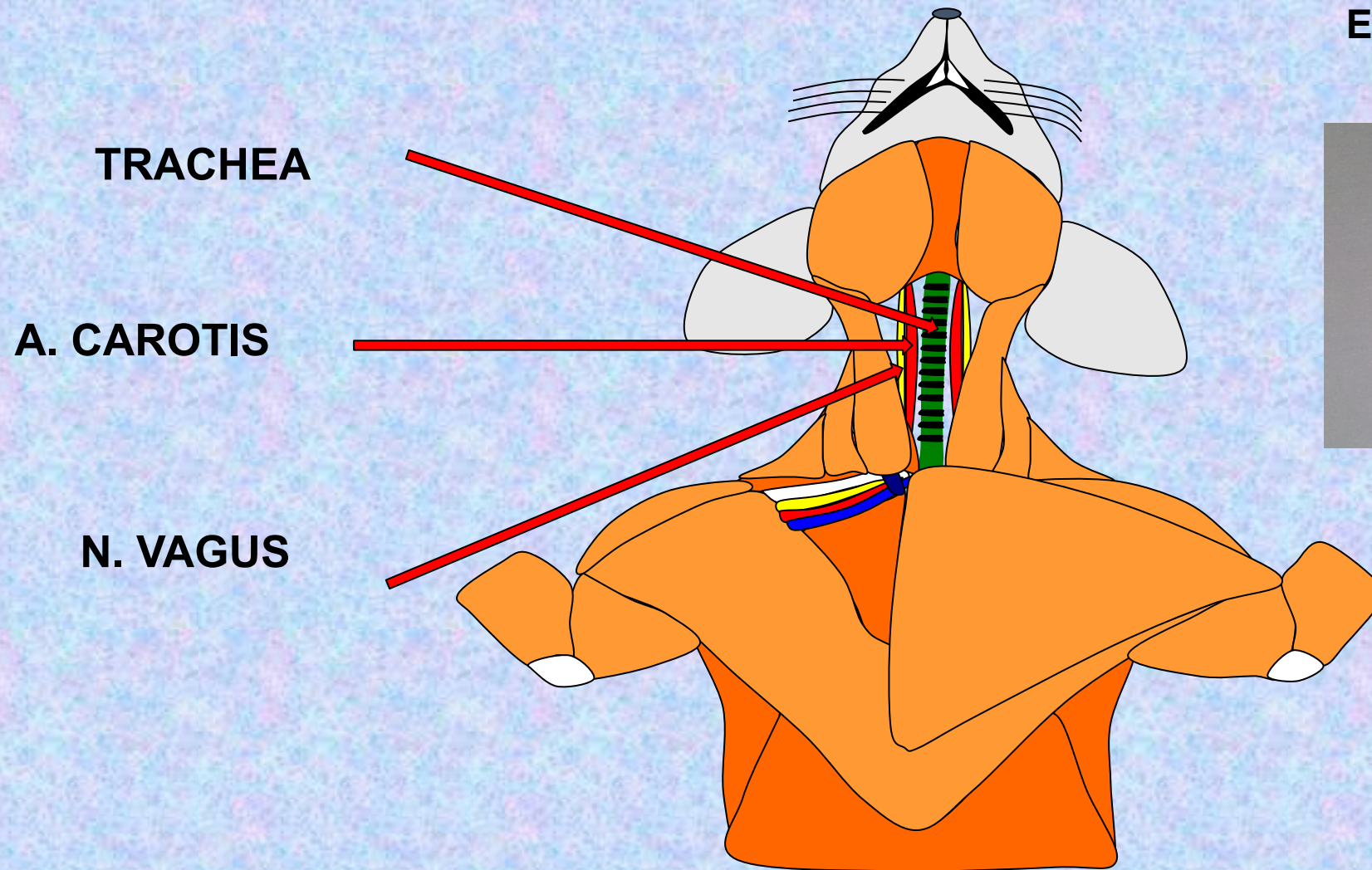
inhalation



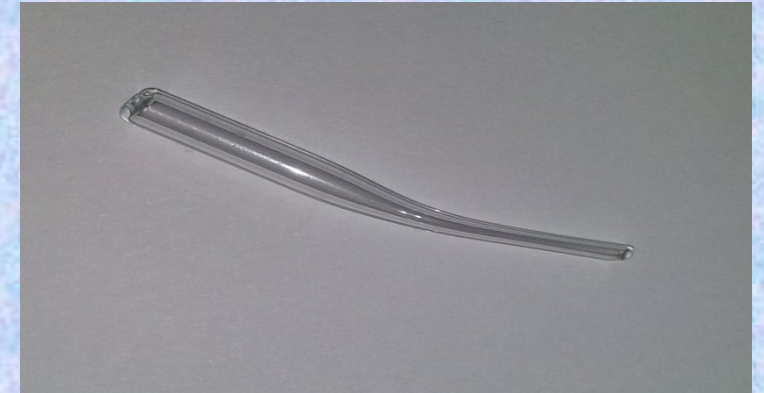
Intra-muscular application
= i.m.



- Anatomy of neck in laboratory rat

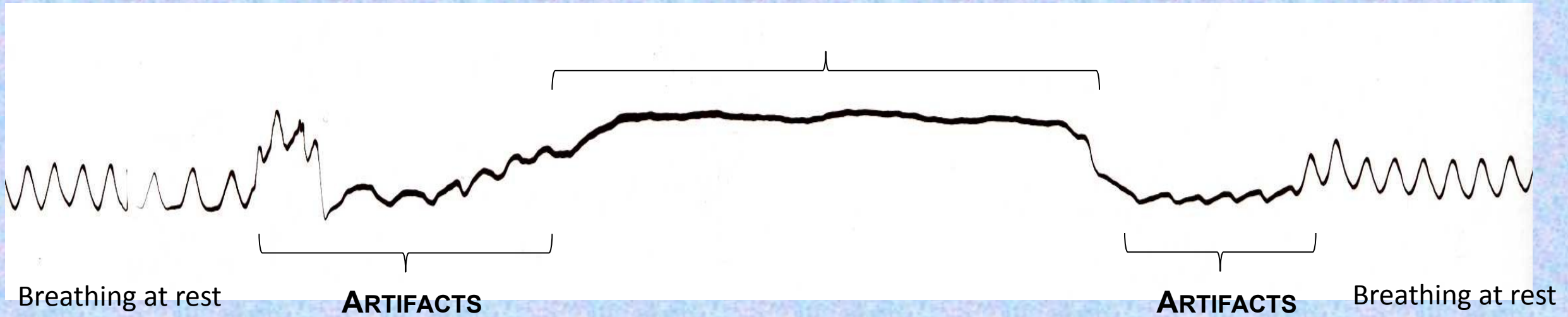


ENDOTRACHEAL CANNULA

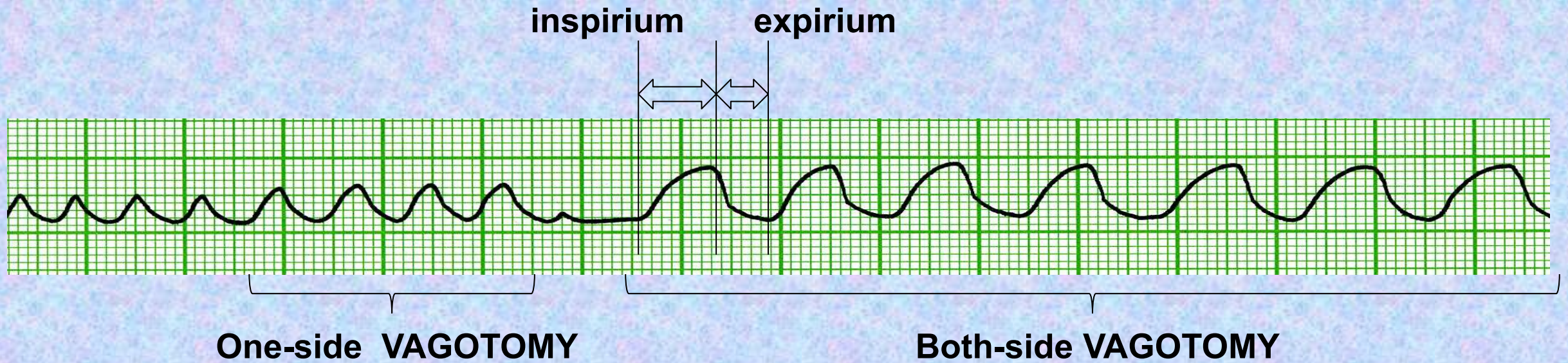


HERING-BREUER „inflation“ REFLEX

REFLEX STOP BREATHING



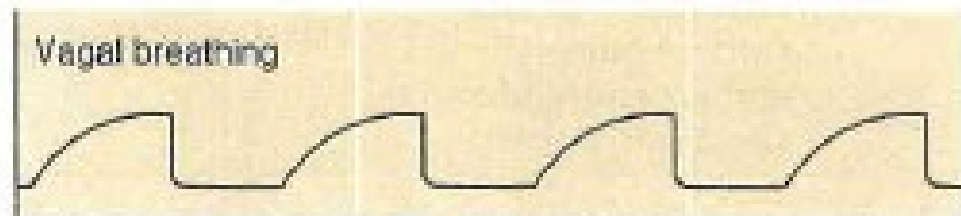
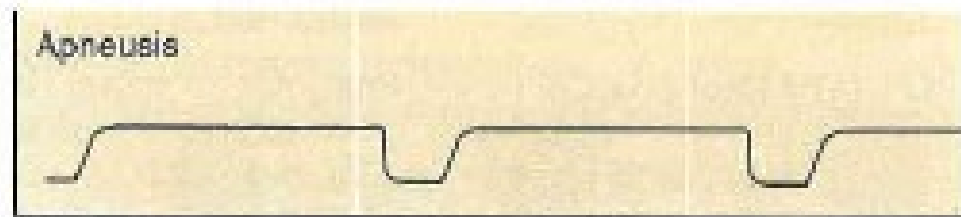
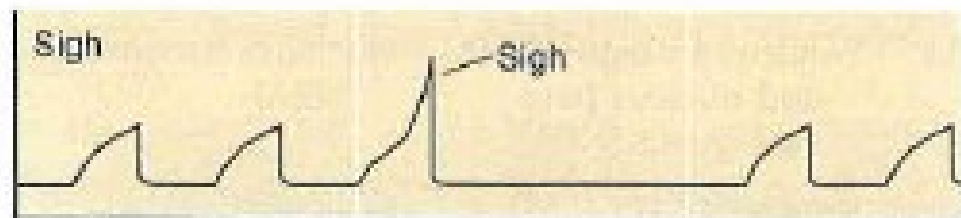
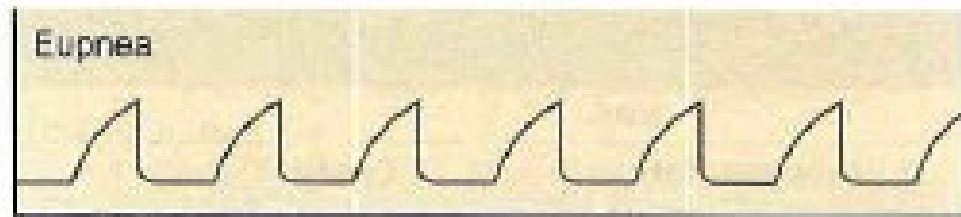
Changes of breathing after VAGOTOMY



Periodic breathing

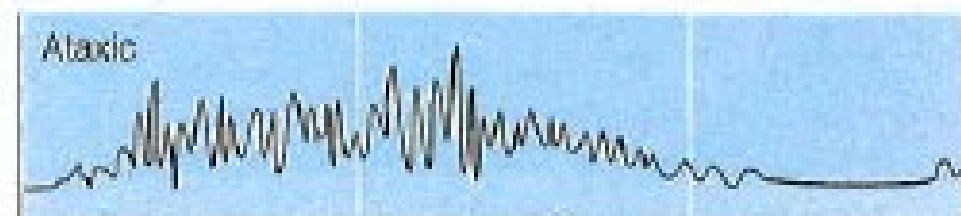
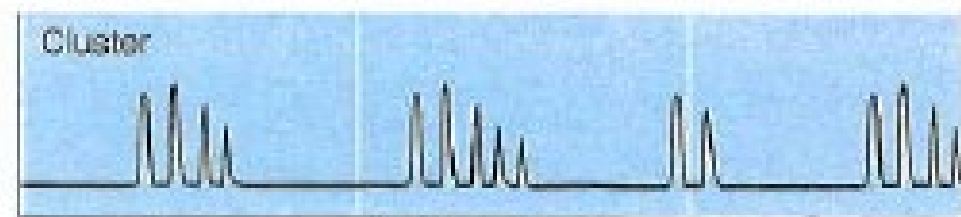
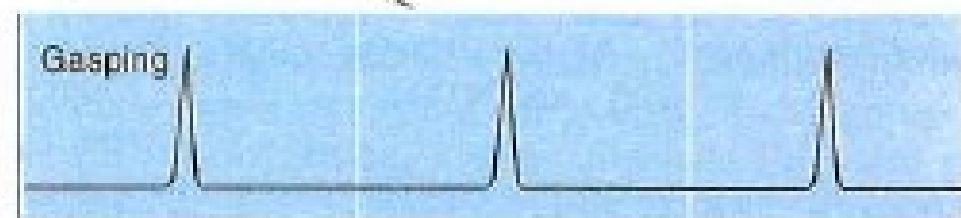
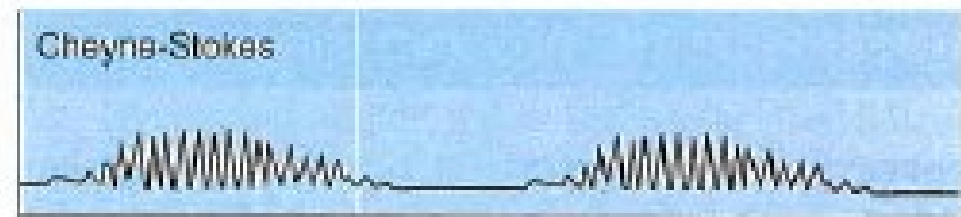
- It is not regular, rhythmic, but respiration occurs in periods ("a moment to breathe, take a moment to not breathe,,")
- **CHEYNE-STOKES**
- **BIOT'S**
- „gaspings“
- **KUSSMAUL**

A INTEGRATED PHRENIC NERVE ACTIVITY



0 0.2 0.4
Time (min)

B LUNG VOLUME



0 0.5 1.0
Time (min)

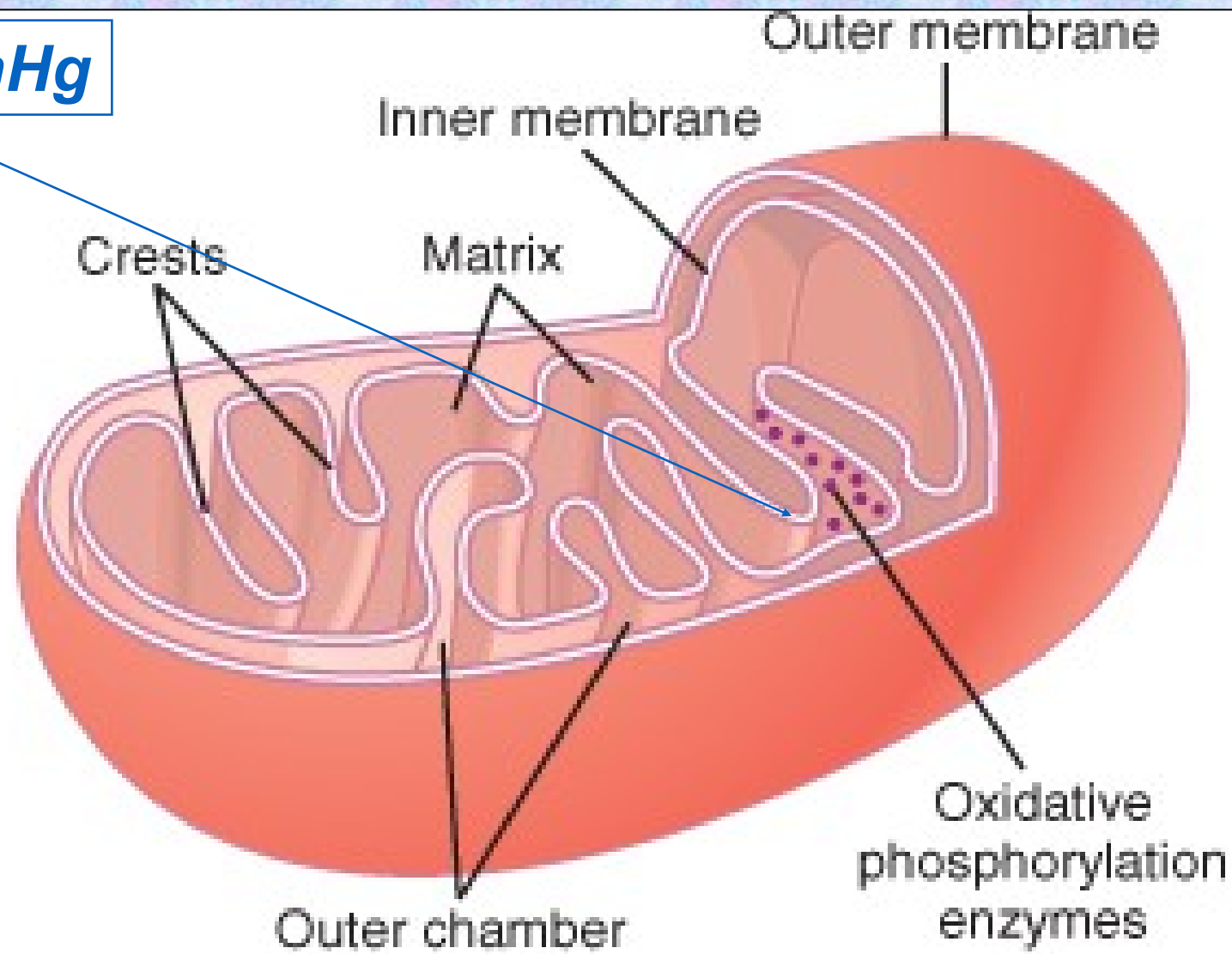
Hypoxia, hypoxemia

- **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)

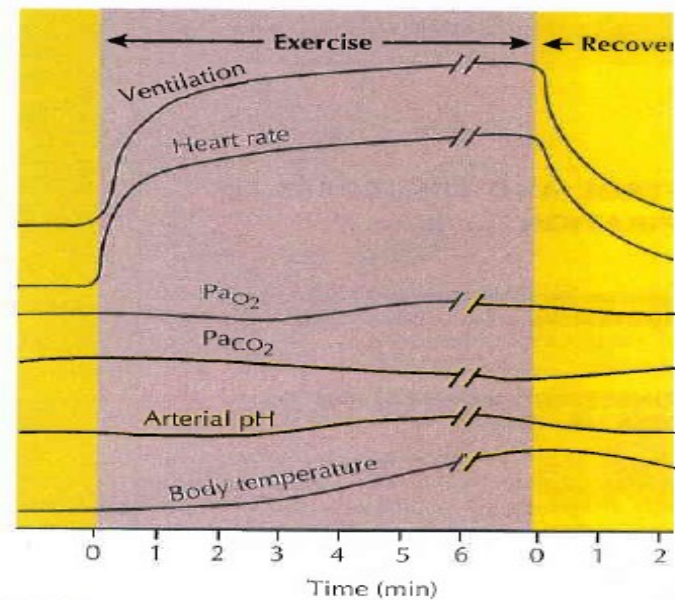
$pO_2 = 1 \text{ mmHg}$



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.

RESPIRATORY RESPONSE TO EXERCISE

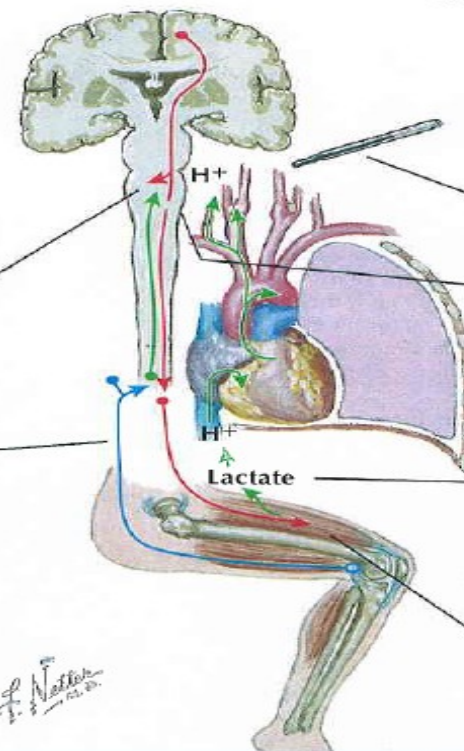


Factors that may account for initial abrupt rise and sharp terminal drop in ventilation

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. N. White
1970

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