SEMESTER – IV

ZOO CC409 : Animal Physiology: Life Sustaining Systems, Unit 2

Regulation of Respiration or lung ventilation

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Learning objectives

The students will learn:

- Neural control of respiration
- Chemical control of respiration
- Respiratory centers
- Central chemoreceptors
- Peripheral chemoreceptors

Regulation of respiration or lung ventilation

Respiration is Controlled by two mechanisms

1-Neural control - by respiratory centers

2-Chemical control – by chemoreceptors

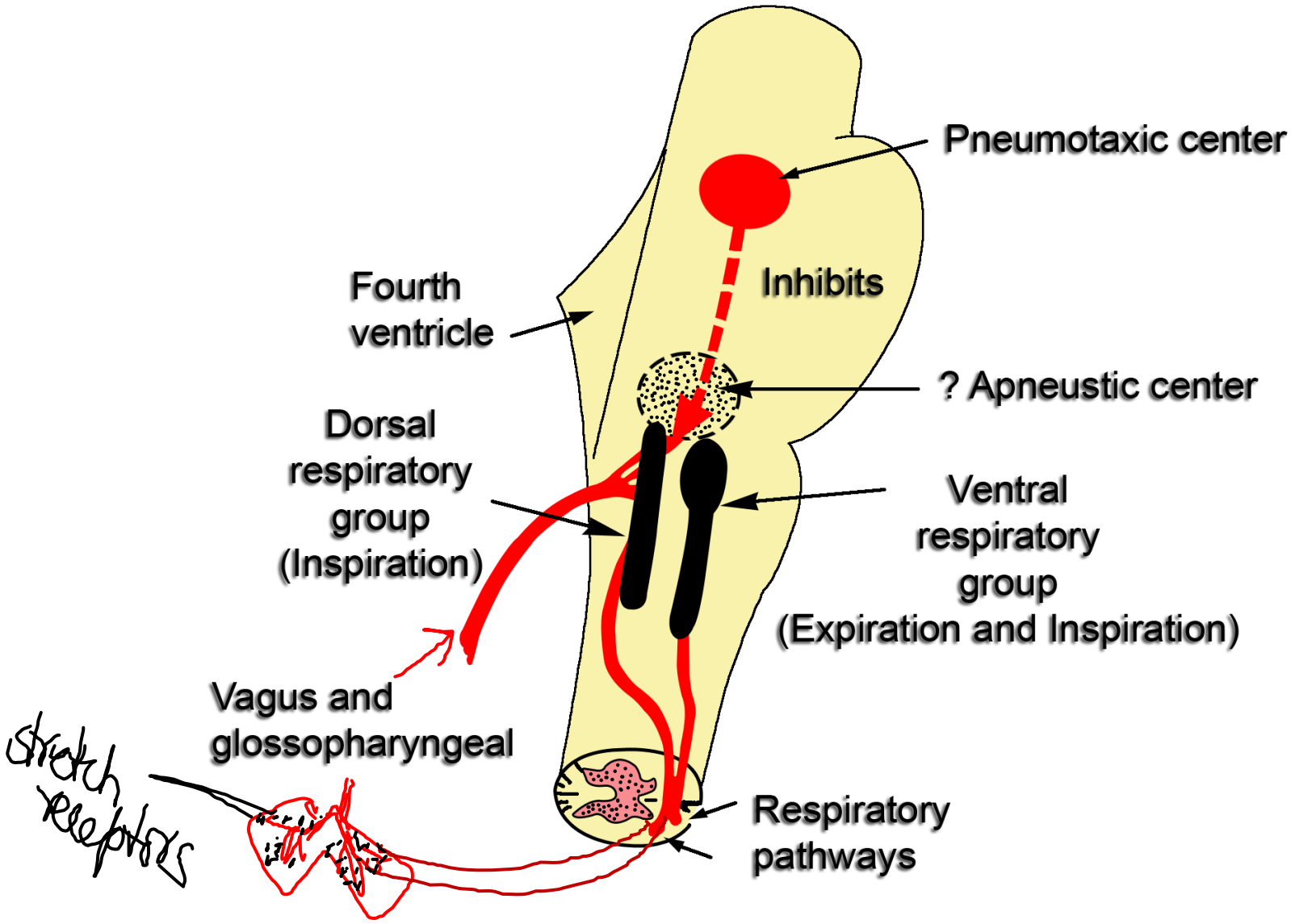
Neural control of respiration

The respiratory center is composed of groups of neurons located bilaterally in the medulla and pons. There are respiratory centers:

1) Dorsal respiratory group in the dorsal portion of the medulla at the posterior end of the 4th ventricle. Causes inspiration. Called inspiratory center.
2) Ventral respiratory group located in the medulla ventral to the floor of 4th ventricle and dorsal to inspiratory center. Contains both expiratory and inspiratory neurons.
3) Pneumotaxic center which is located dorsally in the pons, which helps control both the rate and pattern of breathing.

- Apneustic center (lower pons)
  - The apneustic center of pons sends signals to the dorsal respiratory center to delay the 'switch off' signal of the inspiratory ramp. Duration of inspiration is increased

Organization of Respiratory centers



The dorsal respiratory group

- Plays most fundamental role in the control of respiration.
- Generates the basic rhythm of respiration.
- Controls the normal quiet breathing
- The dorsal neurons send nervous signal to the inspiratory muscles (esp diaphragm) rhythmically .
- The signals begins very weak at first and then increases steadily for 2 seconds in a ramp manner and then it ceases abruptly for 3 seconds which turns off the excitation of the diaphragm and allows elastic recoil of the lungs and chest wall to allow expiration.
- Another inspiratory signal begins for another cycle. The advantage of ramp signal is that it causes a steady ↑ in the volume of the lungs during inspiration rather than inspiratory gasps.

- Located in the muscular portions of the bronchi and bronchioles throughout the lungs are stretch receptors.
- These receptors transmit signals through the vagi to the dorsal respiratory center when the lungs are overstretched.
- This feedback response ‘switches off’ the respiratory ramp signal and stops further inspiration. This action completes a reflex arc called Hering-Breur reflex. It ↑ the rate of respiration.
- In humans, this reflex is not activated until the tidal volume ↑ to more than 3 times normal (>1.5 L per breath). This reflex protects the lungs from overinflation.

The ventral respiratory group

- The ventral respiratory group of neurons functions in both inspiration and expiration. Few neurons cause inspiration while others cause expiration.
- The ventral group of neurons remains almost inactive during normal quiet respiration.
- This group of neurons become active during increase pulmonary ventilation as during exercise.
- Specially important in providing the powerful expiratory signals to the abdominal muscles during heavy expiration.

The pneumotaxic center

The pneumotaxic center limits the duration of inspiration and increases the respiratory rate:

- it sends signals to the inspiratory center and controls the ‘switch off’ point of the inspiratory ramp, thus controlling the duration of the filling phase of the lung cycle.
- a strong signal may cause the inspiration to last for as little as 0.5 second- filling the lungs slightly.
- A weak pneumotaxic signal may cause inspiration to continue for 5 or more seconds, thus filling the lungs with excess of air.

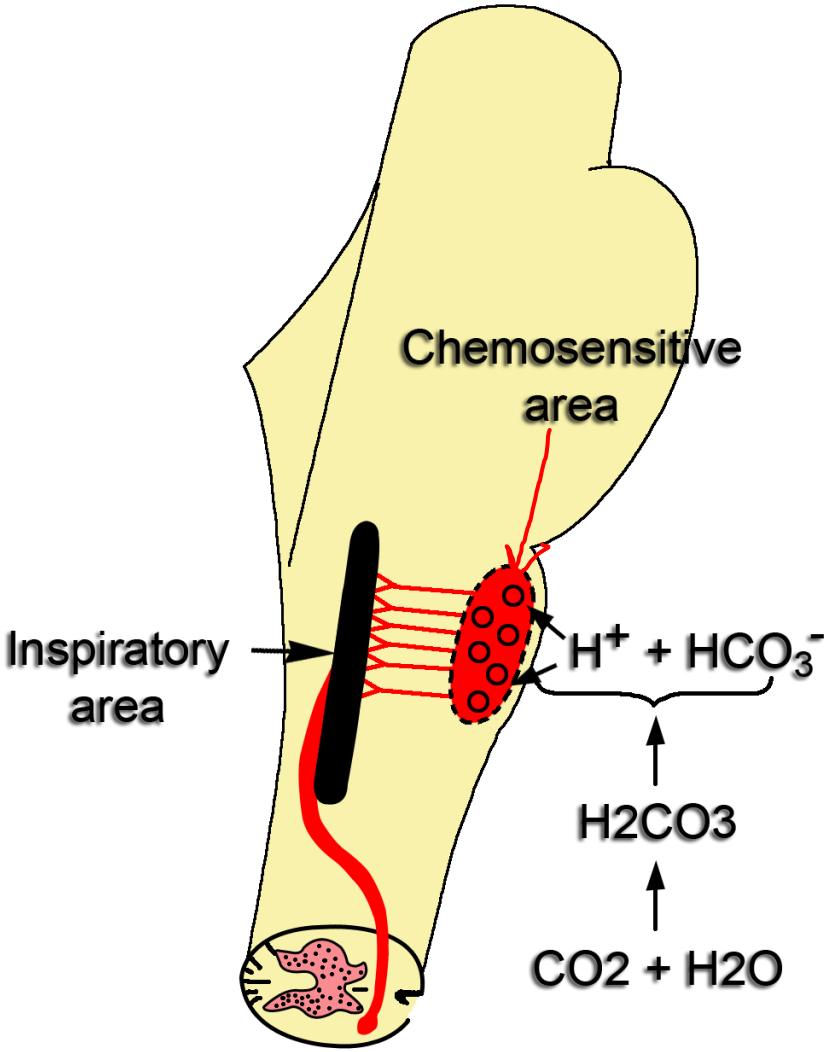
The Hering-Breuer inflation reflex

When the lungs are inflated, this causes stimulation of “stretch receptors” in the walls of bronchi and bronchioles which is transmitted through the vagus nerve to the dorsal respiratory groups to “switch off” inspiration.

Chemical regulation of breathing

- Central Regulation
- Peripheral Regulation

Central chemoreceptors



Central Chemical control of respiration

Excess CO_2 of ↑ H^+ ions mainly stimulate the respiratory center to increase the strength of both inspiratory and expiratory signals to the respiratory muscles.

An additional neuronal area (chemosensitive area) lies 0.2 mm beneath the ventral surface of the medulla.

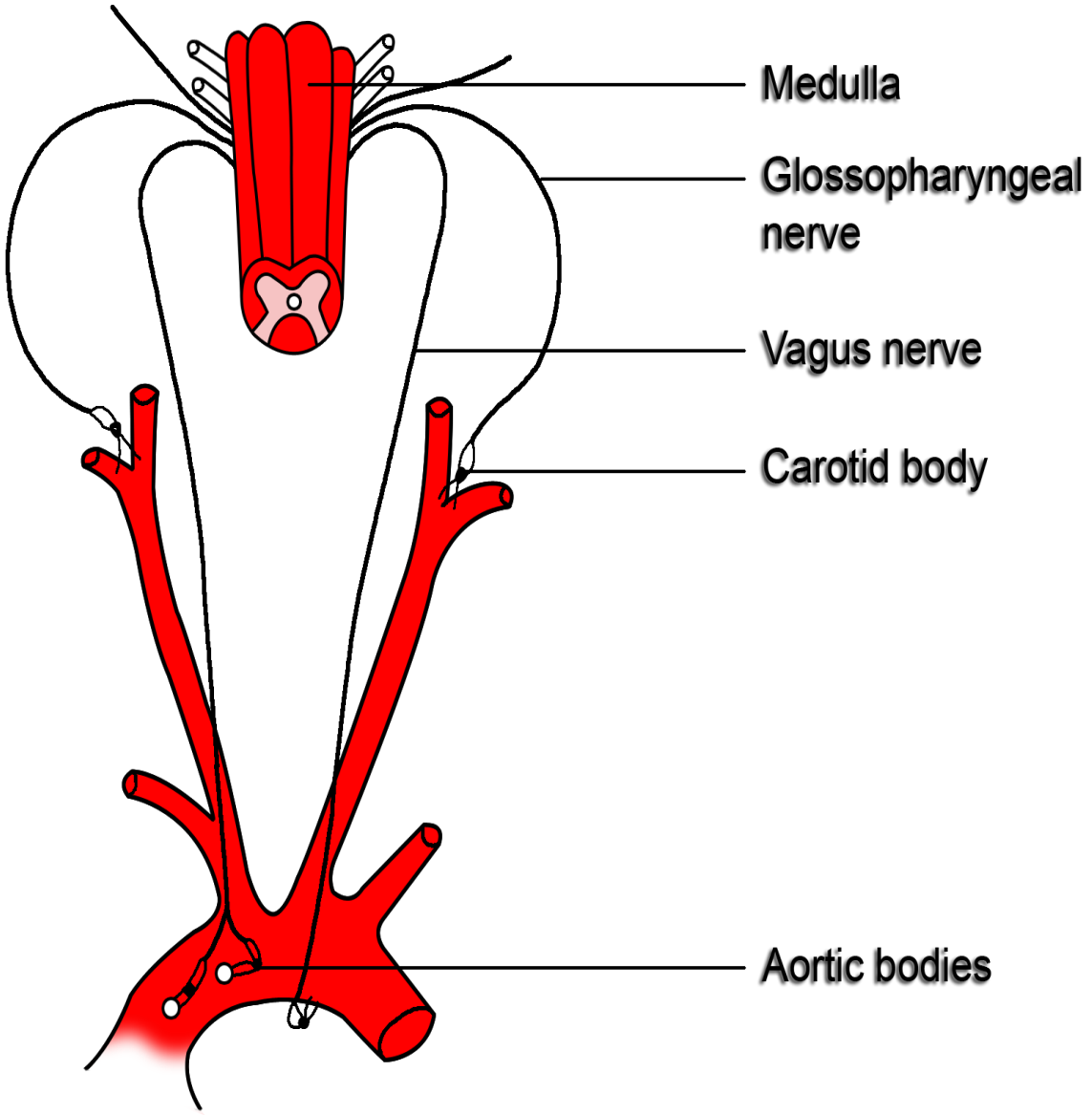
Sensitive to CO_2 or↑ H^+ *, in turn excites the respiratory centre*

Central chemoreceptors

Located on the ventrolateral surfaces of the medulla oblongata (bilaterally). This area is highly sensitive to changes in either blood PCO_2 or H^+ ion concentration. H^+ ions can't cross the blood-brain barrier (BBB). So CO_2 cross the BBB and react with H_2O of CSF to form carbonic acid and then dissociate into H^+ ion and HCO_3^-, then the H^+ ion stimulates the chemosensitive area in the brain.

Oxygen unimportant to control via central chemoreceptors

- This is because oxyhemoglobin buffer system delivers almost exactly normal amounts of O_2 to the tissues even when pulmonary PO_2 changes from a low 60 mm Hg to a high 1000 mm Hg.



Peripheral chemoreceptors

Peripheral chemoreceptors

These chemical receptors are located outside the brain in the carotid bodies and in the aortic bodies. They are highly sensitive to changes in O_2 in the blood. Afferent fibers pass from the carotid bodies via the glossopharyngeal nerves and afferent fibers from the aortic bodies pass via the vagal nerves to the dorsal respiratory area to stimulate respiration. So fall in arterial O_2 concentration below normal or fall in blood PO_2 excites the peripheral chemoreceptors which will cause increase respiration.

This mechanism responds when the blood O_2 falls too low, mainly below 60-70 mm Hg

Peripheral chemoreceptors

- The carotid and aortic bodies receive special blood supply through minute artery from the adjacent arterial trunk.
- Blood flow through these bodies is extreme, 20 times the weight of the bodies per minute.
- These chemoreceptors are always exposed to arterial blood.
- When the O_2 conc in the arterial blood falls below normal, the chemoreceptors become strongly stimulated.

- The body contains about 2 L of stored O_2 that can be used for aerobic metabolism
  - 0.5 L in lungs
  - 0.25 L in body fluids;
  - 1 L combined with hemoglobin
  - 0.3 L in muscle myoglobin
- In heavy exercise stored O_2 is used within 2 mins.

- When the alveolar PCO_2 is above 60-75mmHg, this lead to “air hunger” or called “dyspnea” rapid deep inspiration.
- If CO_2 rises from 80-100mmHg, the person becomes lethargic and semicomatose.
- If PCO_2 rises from 120-150mmHg, this lead to death due to depression of the respiratory center.

Hypoxia

Defined as deficient O_2 supply to the tissue.

Effects of hypoxia on the body:

Severe hypoxia can cause death of the cells, but in less severe cases it results in:

- Depressed mental activity and coma.
- Reduced work capacity of the muscles.

Treatment of hypoxia

By administration of O_2 by:

- Placing the patient head in a “tent” of O_2.
- Allowing the patient to breath either pure or high concentration of O_2 from mask.
- Administration of O_2 through an intranasal tube.

This O_2 therapy is effective in case of atmospheric hypoxia, hypoventilation hypoxia and in hypoxia caused by impaired alveolar membrane diffusion.

- In hypoxia caused by anemia or abnormal hemoglobin, O_2 therapy is less effective because normal O_2 is available in the alveoli but the defect is in transporting O_2 to the tissues.
- Also in hypoxia caused by inadequate tissue use of O_2, O_2 therapy is of no benefit because O_2 is available in the alveoli and no abnormality in O_2 pickup by the lungs or transport to the tissues but tissue enzyme are incapable of utilizing the O_2 that is delivered

Hypercapnea

Means excess CO_2 in the body fluids. It occurs in association with hypoxia which is caused by hypoventilation or circulatory deficiency. Hypoxia caused by too little O_2 in the air, too little Hb, or poisoning of oxidative enzymes, hypercapnea isn't concomitant of these types of hypoxia. If hypoxia caused by poor diffusion through the pulmonary membrane hypercapnea doesn't occur because CO_2 is 20 times more diffusible than O_2 and if it begins to occur it will stimulate pulmonary ventilation to correct the hypercapnea.

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Cyanosis

- Bluish discoloration of the skin and mucus membrane due to more than 5gm/dL of deoxygenated Hb in the blood.
- Anaemic person can’t be cyanotic, he hasn’t enough Hb for 5gm to be deoxygenated in 100mL of blood.
- In polycythemia, excess Hb that can become deoxygenated can cause cyanosis even under normal conditions.

Acclimatization to high altitude

- Atmospheric pressure ↓ with ↑ in altitude
- Atm press at 9000 m on Mount everest =253 mmHg; PO_2=21% of 253=53 mmHg
- Alveolar PO_2 and arterial PO_2 must also ↓
- Causes mountain sickness-breathlessness, headache, nausea, insomnia, fatigue, impaired mental processes- same as responses to hypoxia

Body's compensatory mechanisms

- The peripheral chemoreceptors stimulate ventilation
- Erythropoietin (from kidneys), stimulate RBC synthesis resulting in ↑ RBC and Hb concentration of blood
- DPG ↑ and shifts the Hb dissciation curve to the right, facilitating O_2 unloading in the tissues
- ↑ in capillary density, mitochondria and muscle myoglobin occur, all of which ↑ O_2 transfer

References

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