Control of Breathing

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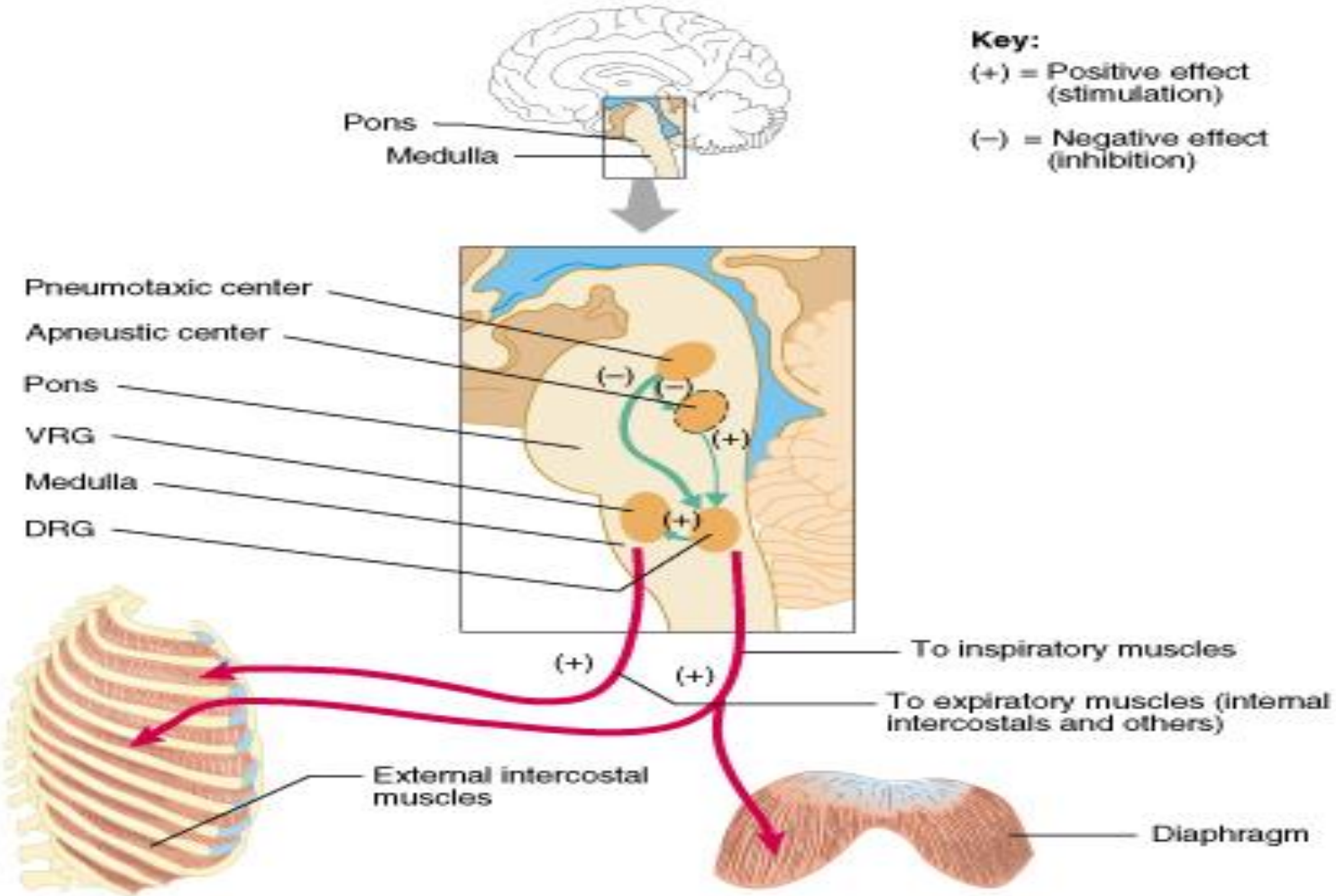
Objectives

- By the end of this lecture you should be able to: -
- Understand the role of the medulla oblongata in determining the basic pattern of respiratory activity.
- List some factors that can modify the basic breathing pattern like e.g.
- a- The Hering-Breuer reflexes, b- The proprioreceptor reflexes, and c- The protective reflexes, like the irritant, and the J-receptors.
- Understand the respiratory consequences of changing PO_2, PCO_2, and pH.
- Describe the locations and roles of the peripheral and central chemoreceptors.

Controls of rate and depth of respiration

- Arterial PO2
  - When PO2 is VERY low(Hypoxia), ventilation increases
- Arterial PCO2
  - The most important regulator of ventilation is PCO2, small increases in PCO2, greatly increases ventilation
- Arterial pH
  - As hydrogen ions increase (acidosis), alveolar ventilation increases.

Respiratory Centers



Medullary Respiratory centers

- Inspiratory area (Dorsal Respiratory Group) DRG
  -Determines basic rhythm of breathing
  -Causes contraction of diaphragm and external intercostals
- Expiratory area (Ventral Respiratory Group) VRG
  -Inactive during normal quiet breathing
  -Activated by inspiratory area during forceful breathing
  -Causes contraction of internal intercostals and abdominal muscles
- The medullary respiratory center stimulates basic inspiration for about 3 seconds and then basic expiration for about 2 seconds (5sec/breath= 12breaths/min).

Pontine Respiratory centers

- Transition between inhalation and exhalation is controlled by:

Pneumotaxic area

Inhibits inspiratory area of medulla to stop inhalation

Breathing is more rapid when pneumotaxic area is active

Apneustic area

stimulates inspiratory area of medulla to prolong inhalation

Chemical regulation

(Peripheral and central chemoreceptors)

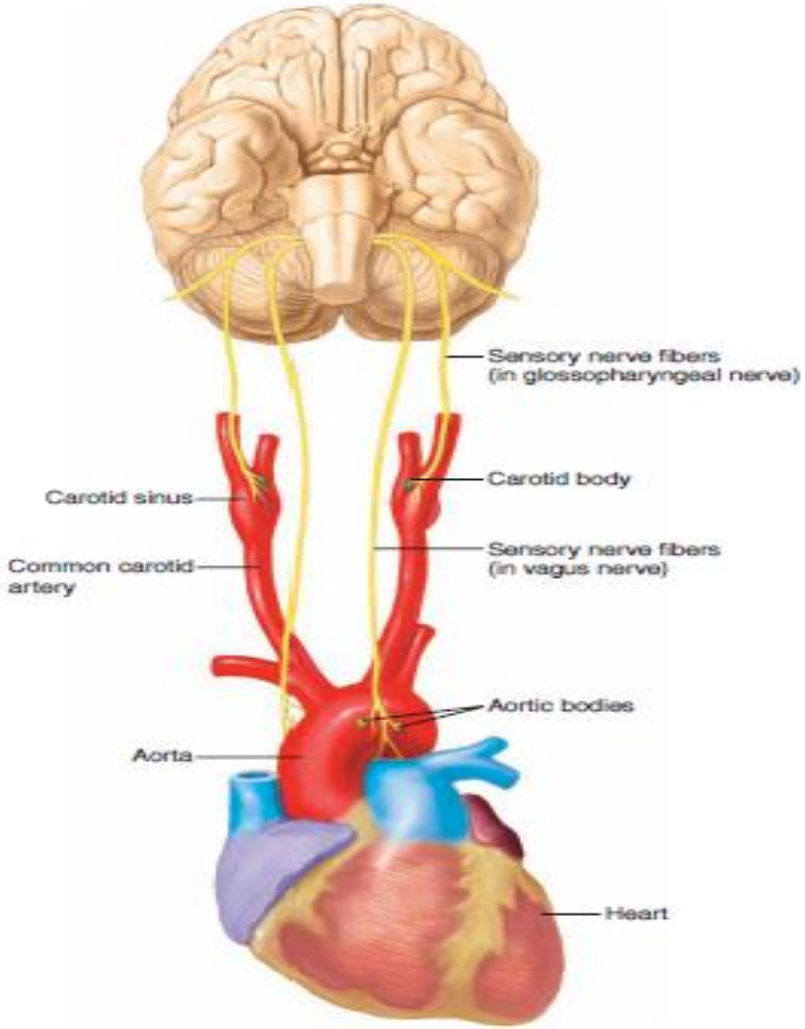
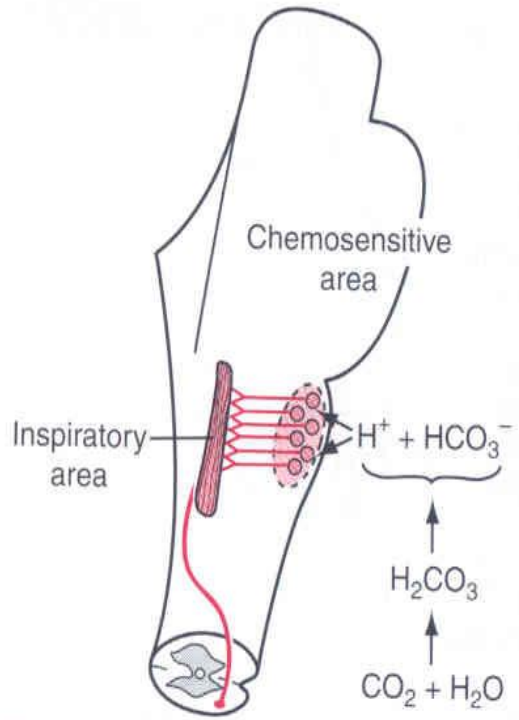


Figure 16.25 **Sensory input from the aortic and carotid bodies.** The peripheral chemoreceptors (aortic and carotid bodies) regulate the brain stem respiratory centers by means of sensory nerve stimulation.

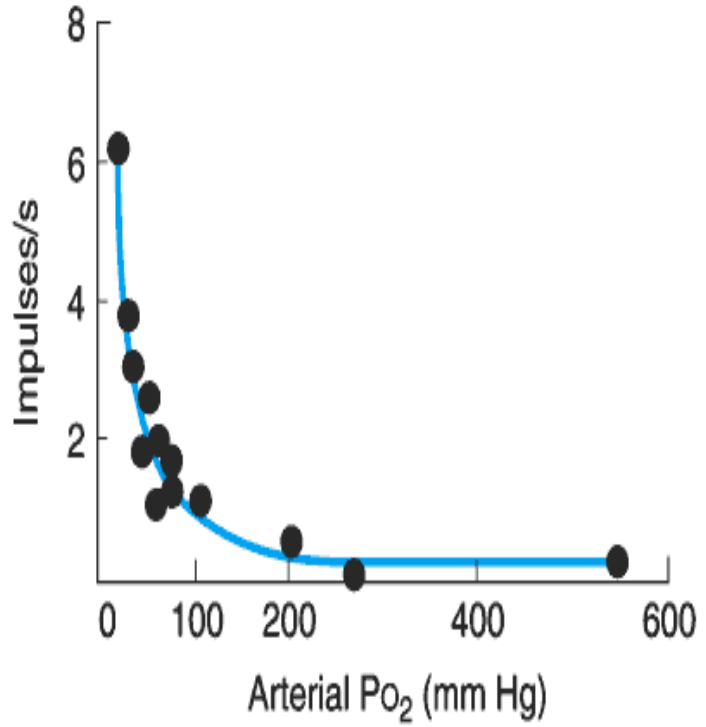


IGURE 41–2

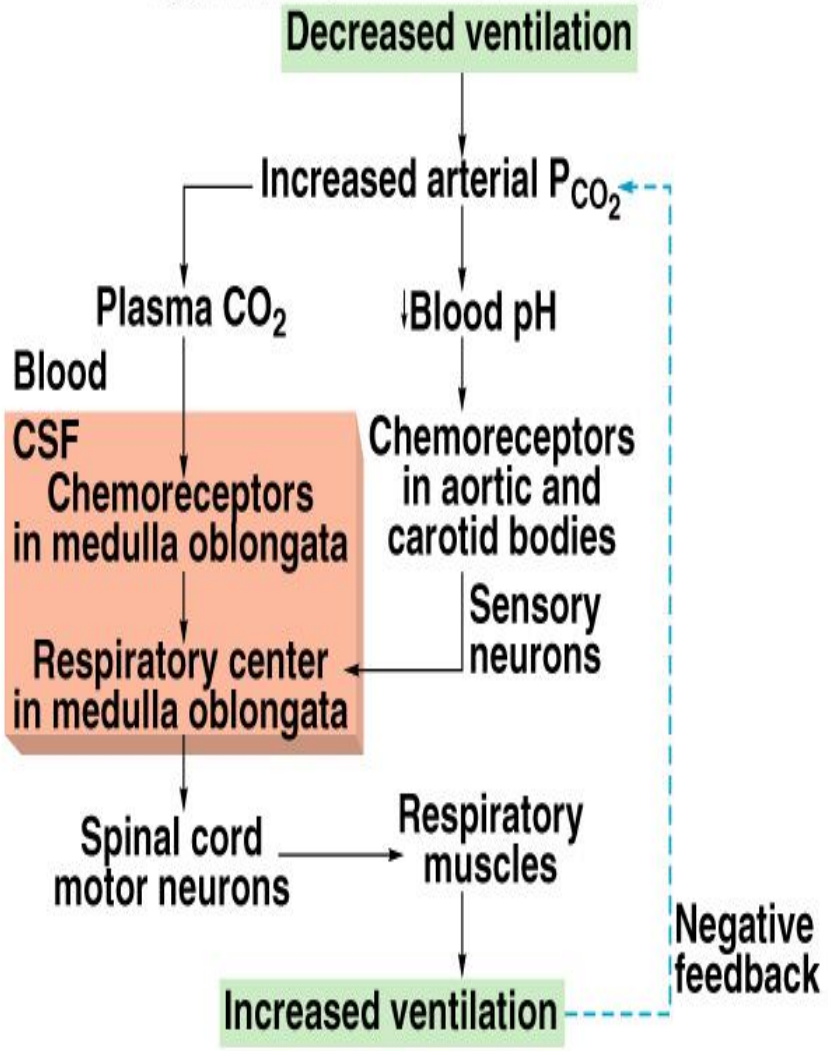
imulation of the inspiratory area by signals from the *chemosensitive ea* located bilaterally in the medulla, lying only a fraction of a illimeter beneath the ventral medullary surface. Note also that hy- ogen ions stimulate the chemosensitive area, whereas it is carbon oxide in the fluid that gives rise to most of the hydrogen ions.

Chemoreceptor Control of Breathing

Figure 36–6.



Change in the rate of discharge of a single afferent fiber from the carotid body when arterial PO_2 is reduced. (Courtesy of S Sampson.)



Effect of blood CO2 level on central chemoreceptors

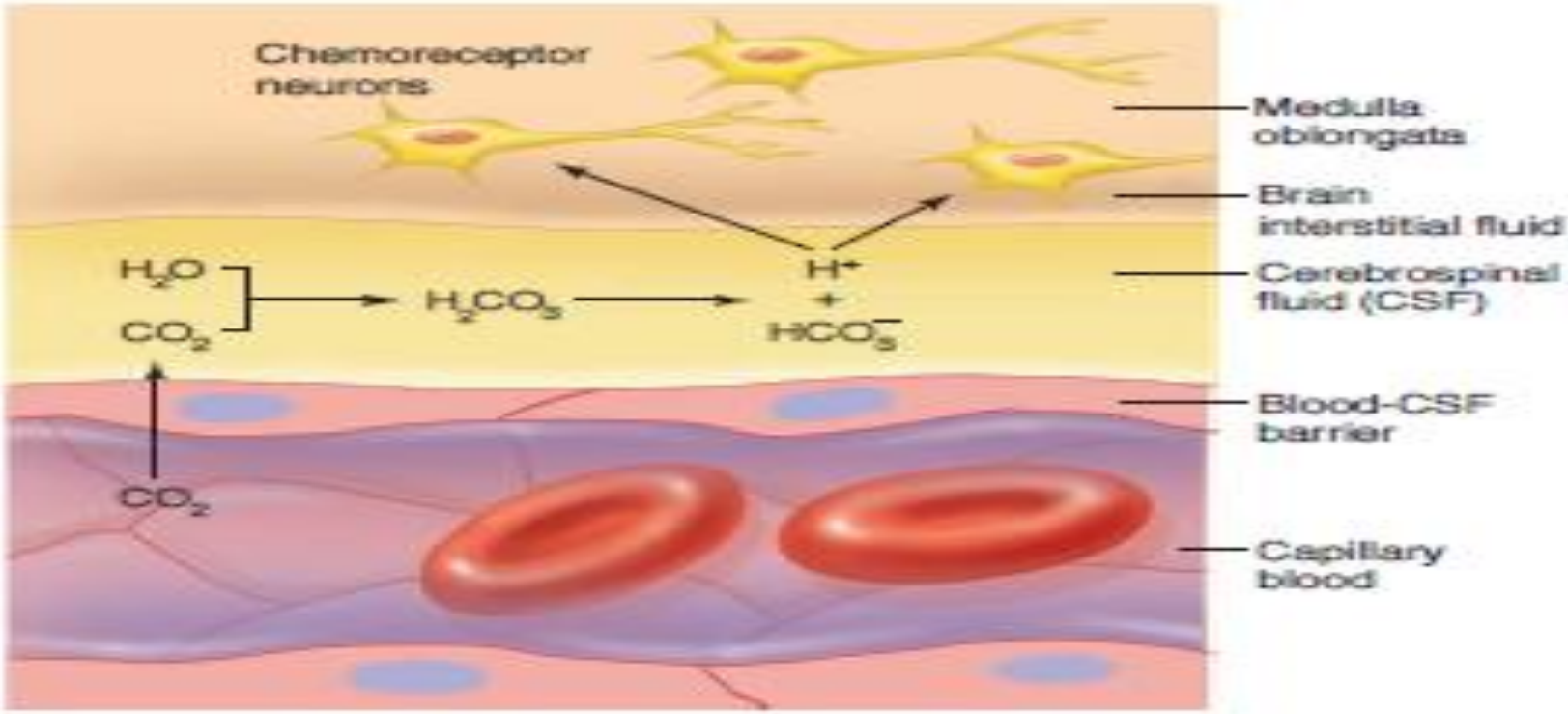
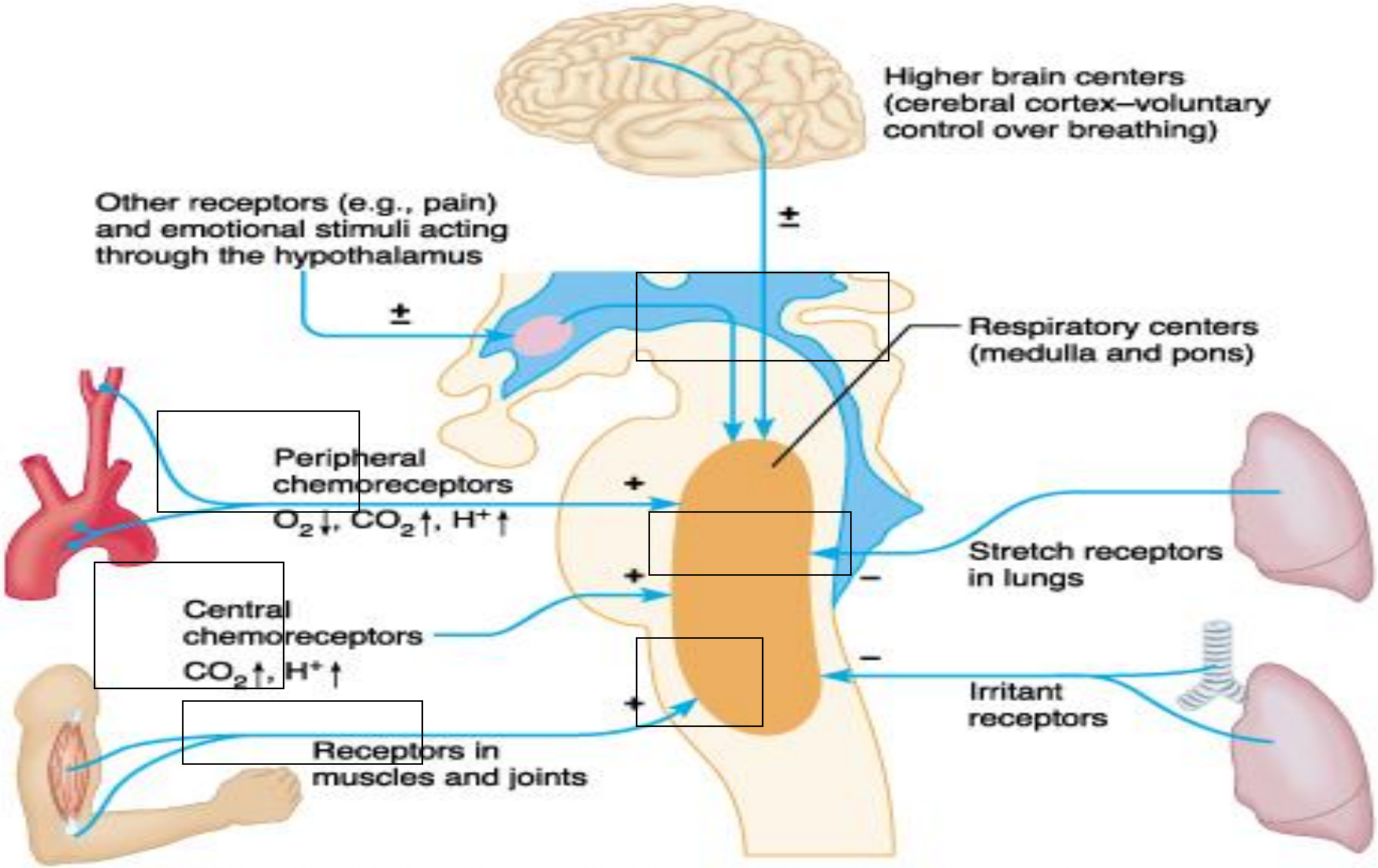


Figure 16.29 **How blood CO_2 affects chemoreceptors in the medulla oblongata.** An increase in blood CO_2 stimulates breathing indirectly by lowering the pH of blood and cerebrospinal fluid (CSF). This figure illustrates how a rise in blood CO_2 increases the H^+ concentration (lowers the pH) of CSF and thereby stimulates chemoreceptor neurons in the medulla oblongata.

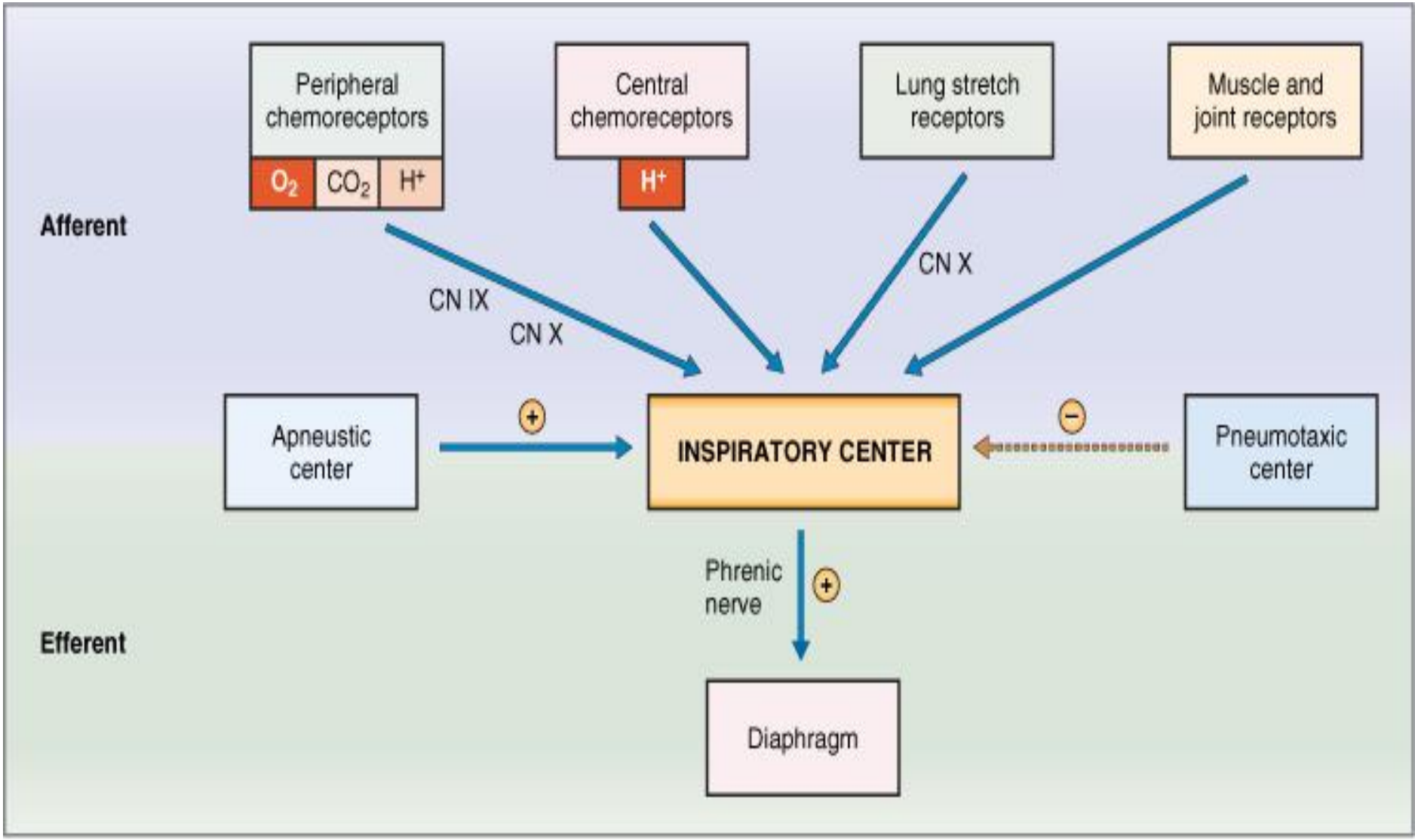
Factors Influencing Respiration



Hering-Breuer inflation reflex

- When the lung becomes overstretched (tidal volume is 1 L or more), stretch receptors located in the wall bronchi and bronchioles transmit signals through vagus nerve to DRG producing effect similar to pneumotaxic center stimulation
- Switches off inspiratory signals and thus stops further inspiration .
- This reflex also increase the rate of respiration as does the pneumotaxic center.

Cont..factors affecting respiration



Respiratory Acidosis

- Hypoventilation.
- Accumulation of CO_2 in the tissues.
  - P_{CO2} increases
  - PH decreases.

Respiratory Alkalosis

- Hyperventilation.
- Excessive loss of CO_2.
  - P_{c02} decreases (↓35 mmHg).
  - pH increases.

Metabolic Acidosis

- Ingestion, infusion, or production of a fixed acid.
- decreased renal excretion of hydrogen ions.
- loss of bicarbonate or other bases from the extracellular compartment

Metabolic Alkalosis

- excessive loss of fixed acids from the body
- ingestion, infusion, or excessive renal reabsorption of bases such as bicarbonate
- pH increases.

The respiratory system can compensate for metabolic acidosis or alkalosis by altering alveolar ventilation

Effects of low and high gas pressure on the body

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Objectives

- **By the end of this lecture you should be able to:**
- 1-Describe the effects of exposure to low and high barometric pressures on the body.
- 2- Describe the body acclimatization to low barometric pressure.
- 3-Define decompression sickness and explain how it can be avoided.
- 4-Understand the effects of high nitrogen pressure, and nitrogen narcosis.

Effect of increased barometric pressure

(Deep sea diving).

- When human descend below the sea, the pressure around them increased.
- To prevent the lungs from collapse , air must be supplied also under high pressure.
- This exposes the blood in the lungs to extremely high alveolar gas pressure(hyperbarism).
- Under certain limits these high pressures cause tremendous alterations in the physiology of the body.

Cont..

- The surrounding pressure increases by 1 atmosphere for every 10 meter (33 feet) of depth in sea water.
- Therefore at a depth of 31 meter (100 feet) in the ocean the diver is exposed to a pressure of 4 atmospheres.
- These problems confront SCUBA (self contained under water breathing apparatus.

- **Effect of depth on the volume of the gases**:

  is compression of gases to smaller and smaller volumes. 1L (sea level)→1/2 L at 33 feet and so on

- **Effect of depth on density of gases**

  increase in density of gas and hence increased work of breathing.

- **Nitrogen effect at high nitrogen pressure**

  has 2 principle effects:

  * Nitrogen narcosis (anesthetic effect)
  * Decompression thickness.

Nitrogen narcosis

- Nitrogen like most other anesthetic gases, dissolve freely in the fats of the body including the membranes and other lipid structures of the neurons.
- This leads to alteration of the electrical conductance of the membranes, reduces their excitability and subsequent narcosis develops.
- At 120 feet: the diver lose many of his cares.
- At 150 feet: there is a feeling of euphoria and drowsiness and impaired performance.
- At higher pressure: loss of coordination and finally coma might develop.

Decompression sickness (Cassion's disease)

- it is a syndrome caused by a decrease in the ambient pressure which occur in animal and men when the tissues of the body contain an excess of physically inert gas.
- During descent , the high partial pressure of nitrogen (encountered when breathing compressed air at depth) forces this poorly soluble gas into solution in body tissue particularly in fat (it has a high N2 solubility).
- On ascending, this inert gas comes out of physical solution forming a gaseous phase (bubbles), leading to symptoms and signs.

Cont.. Decompression sickness

- **During slow ascent** N2 is slowly removed from the tissues since the partial pressure there is higher than that in the arterial blood and alveolar gas.
- **If decompression is rapid** bubbles of gaseous nitrogen are released, in tissues and blood, causing the symptoms of decompression sickness (the bends or caisson disease)

Symptoms & signs of decompression sickness(DS)

- The mildest form of DS is fatigue or drowsiness after decompression.
- Locally there is skin itch
- Other sever symptoms may occur e.g.:

bubbles in the tissues cause sever pains particularly around the joints.

Neurological symptoms include paresthesia, itching, paralysis, and inner ear disturbances.

Cont.. DS

- Thoracic pains: dyspnea, substernal pain, cyanosis, and cough.
- Bubbles in the coronary arteries may cause myocardial damage.
- Decompression sickness shock, capillaries become permeable to plasma and hypovolemia rapidly develop.
- Edema may be prominent and shock is also usually complicated by pulmonary edema

Treatment of decompression symptoms

- Rapid recompression in a pressure chamber followed by slower decompression. This reduces the volume of the bubbles and forces them back into solution.
- In a very deep dives, the risk of decompression sickness can be reduced if a helium-O2 mixture is breathed during the dive..

Cont.. Treatment of DS

- Helium is more desirable than nitrogen in deep dives because it has:
- ¼-1/5 the narcotic effect of nitrogen on CNS.
- 1/7 the molecular weight of nitrogen.
- low density leading to decreased air way resistance of diver.
- High diffusion through tissues.
- Helium is about 1/2 as soluble as nitrogen in body fluids. This reduces the quantity of bubbles that can form in tissues when the diver is decompressed after diving

Effects of low oxygen pressure on the body
(Aviation-ascend to high altitude)

- At the sea level the barometric pressure is 760 mmHg.
- At 10,000 feet is 523 mmHg
- At 50,000 feet 87 mmHg.
- This decrease in barometric pressure is the basic cause of all the hypoxia problems in high altitude in physiology.

Alveolar PO2 at different altitudes

- As the barometric pressure decreases, the oxygen partial pressure decreases proportionally, remaining less than 21 % of the total barometric pressure.
- at sea level PO2= 159 mmHg.
- At 20,000 feet PO2= 40 mmHg.
- At 50,000 feet PO2= only 18 mmHg.

Cont…

- Even at high altitude CO_2 is continuously excreted from the pulmonary blood into the alveoli. Also, water vaporizes into the inspired air from the respiratory surfaces.
- Therefore, these two gases dilute the oxygen in the alveoli, thus reducing the oxygen concentration and therefore hypoxia develops.

Effects of acute hypoxia

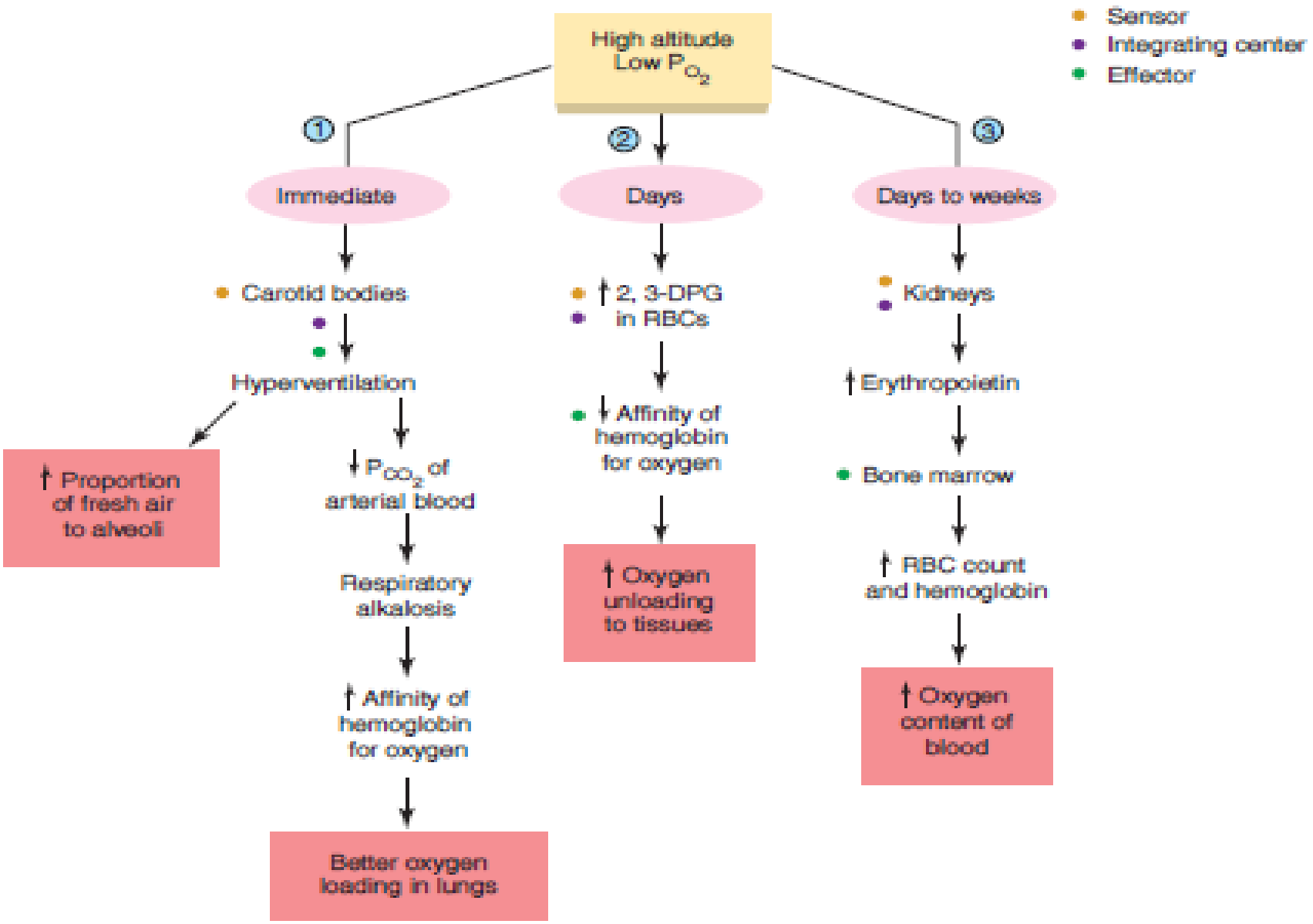
- Some of the important acute effects of hypoxia beginning at an altitude of approximately 12,000 feet, are:
- Drowsiness, lassitude, mental and muscle fatigue, sometimes headache, occasionally nausea and sometimes euphoria.
- All these progress to a stage of twitching or convulsions above 18,000.
- Above 23,000 feet the un acclimatized person can enter into coma.

Acclimatization to low PO2

- A person remaining at high altitudes for days , weeks or years becomes more and more acclimatized to low PO2,
- So that it causes fewer deleterious effects on the body and it becomes possible for the person to work harder without hypoxic effects or to ascend to still higher altitude.

Principle means of acclimatization

- 1- increase in pulmonary ventilation.
- 2- increased red blood cells.
- 3-increased diffusing capacity of the lungs.
- 4- increased vascularity of the tissues.
- 5-Increased ability of the cells to utilize oxygen despite the low PO2.


Sensor
Integrating center
Effector
High altitude
Low P_{O_2}
1
2
3
Immediate
Days
Days to weeks
Carotid bodies
Hyperventilation
↑ Proportion of fresh air to alveoli
↓ P_{CO_2} of arterial blood
Respiratory alkalosis
↑ Affinity of hemoglobin for oxygen
Better oxygen loading in lungs
↑ 2, 3-DPG in RBCs
↓ Affinity of hemoglobin for oxygen
↑ Oxygen unloading to tissues
Kidneys
↑ Erythropoietin
Bone marrow
↑ RBC count and hemoglobin
↑ Oxygen content of blood