

# **Control of Breathing**

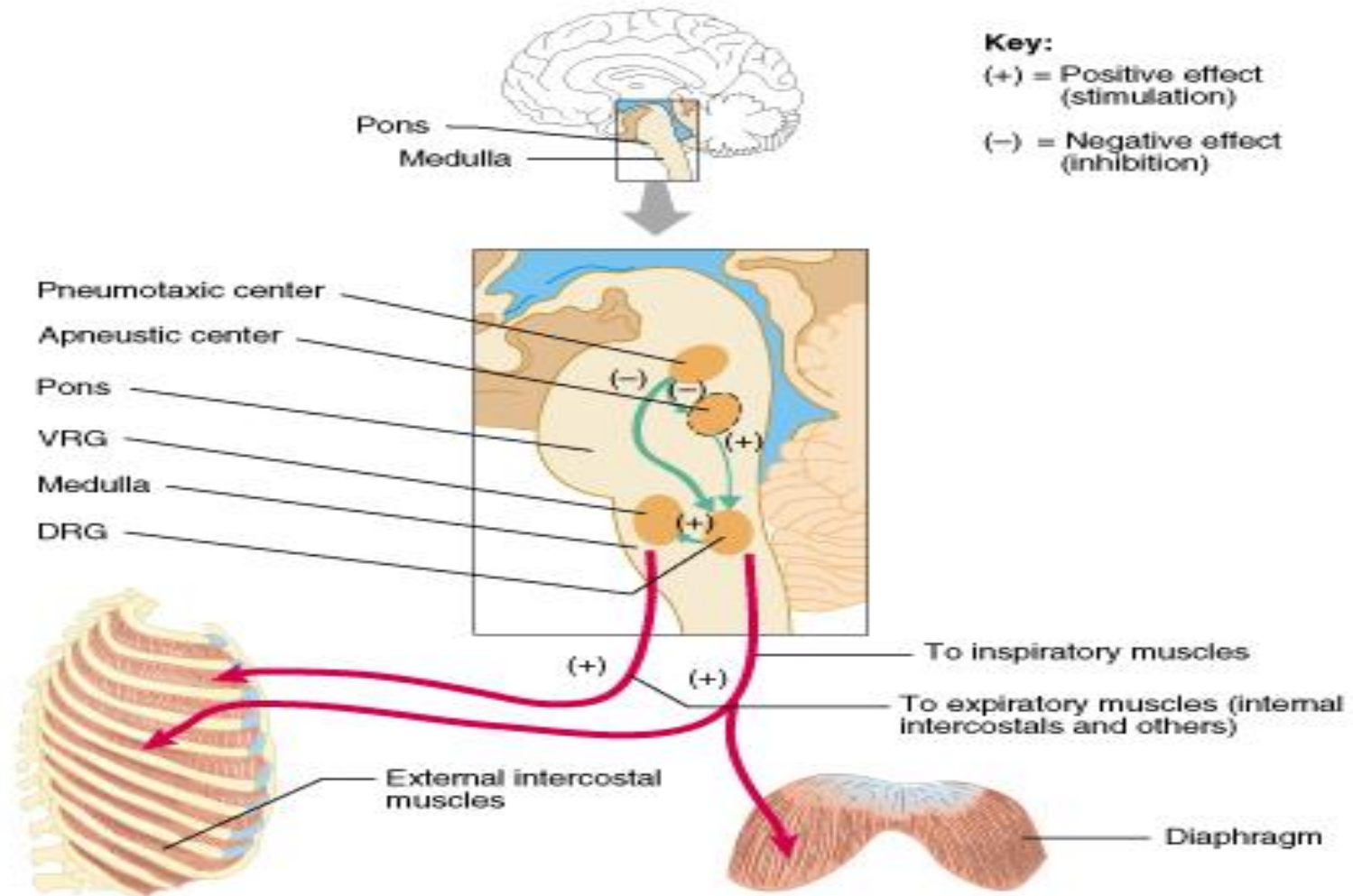
# Objectives

- **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 proprioceptor 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.**
- Compare and contrast **metabolic and respiratory acidosis and metabolic and respiratory alkalosis.**

# Controls of rate and depth of respiration

- Arterial PO<sub>2</sub>
  - When PO<sub>2</sub> is VERY low (Hypoxia), ventilation increases
- Arterial PCO<sub>2</sub>
  - The most important regulator of ventilation is PCO<sub>2</sub>, small increases in PCO<sub>2</sub>, 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 2 seconds and then basic expiration for about 3 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.

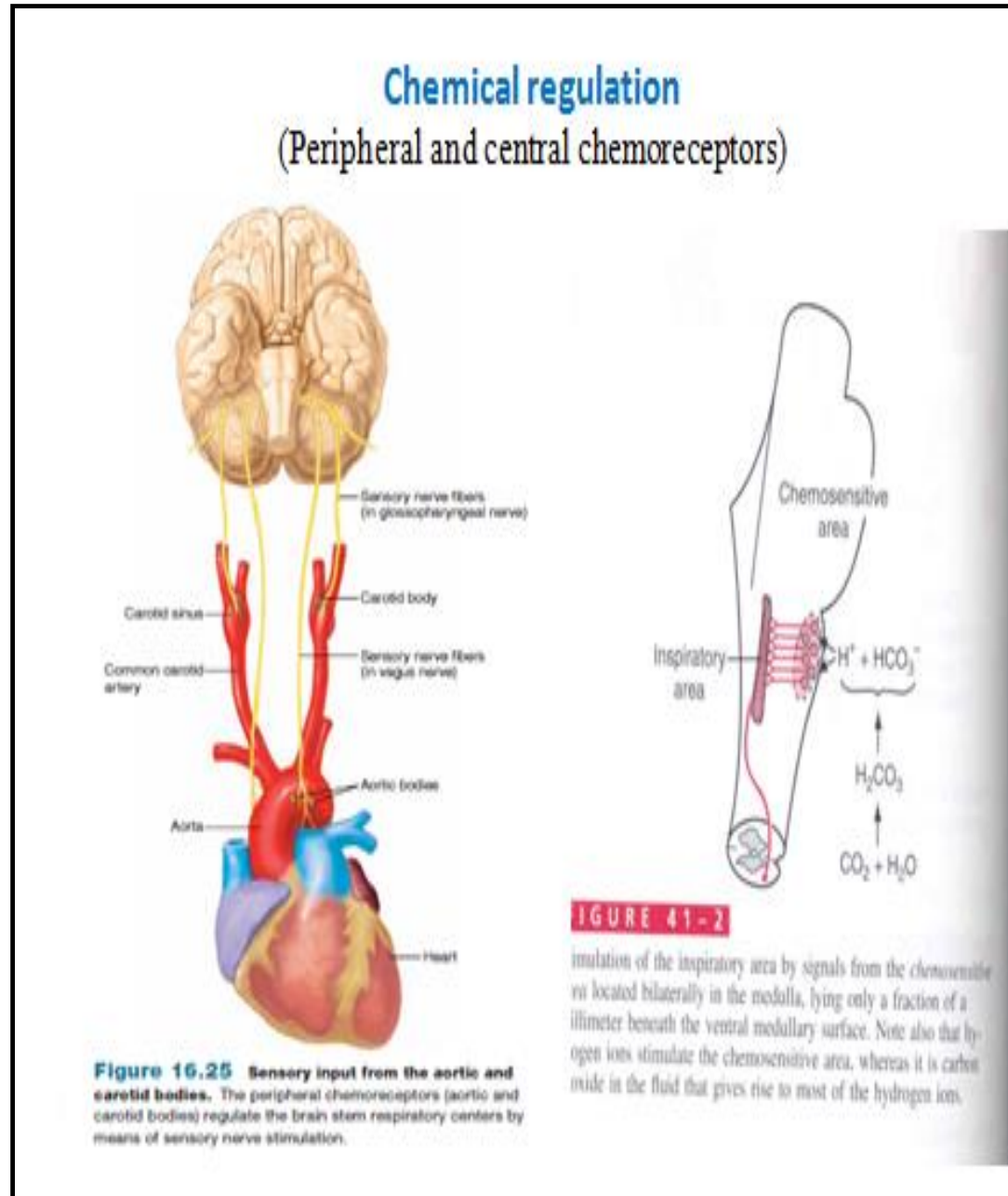
## **Apneustic area**

Stimulates inspiratory area of medulla to prolong inhalation.

Most of the chemoreceptors are in the *carotid bodies*. However, a few are also in the *aortic bodies*, and a very few are located elsewhere in association with other arteries of the thoracic and abdominal regions.

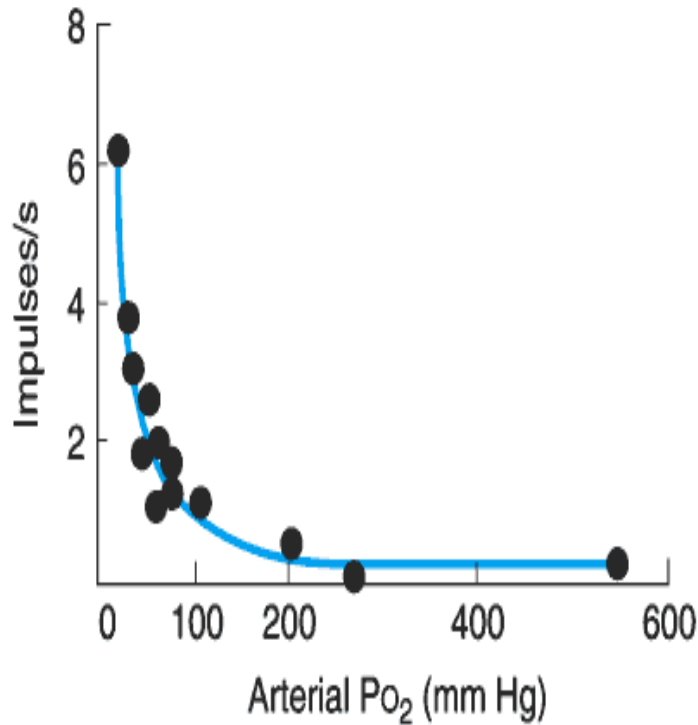
1- Excess  $\text{CO}_2$ ,  $\text{H}^+$  in the blood mainly act directly ---Center

2- other in arteries of the thoracic and abdominal regions peripheral



# Chemoreceptor Control of Breathing

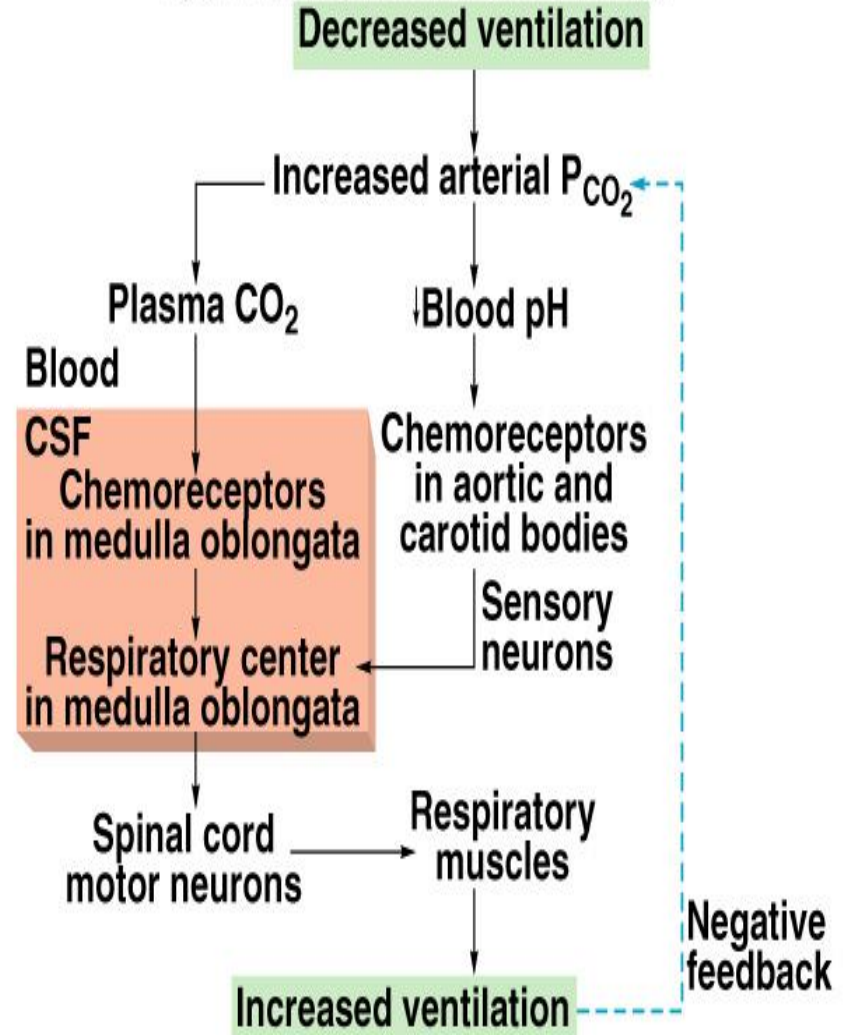
Figure 36-6.



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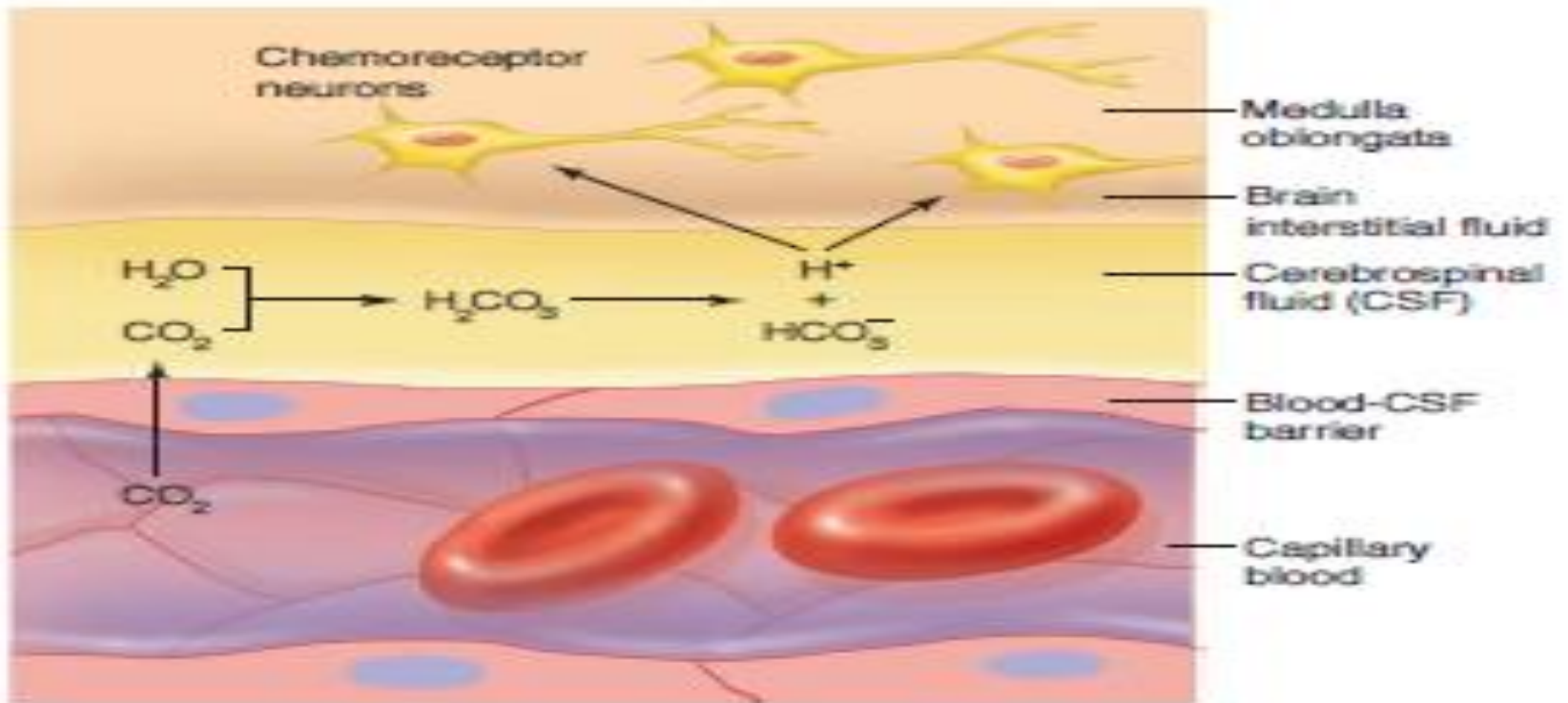
Change in the rate of discharge of a single afferent fiber from the carotid body when arterial PO<sub>2</sub> is reduced. (Courtesy of S Sampson.)

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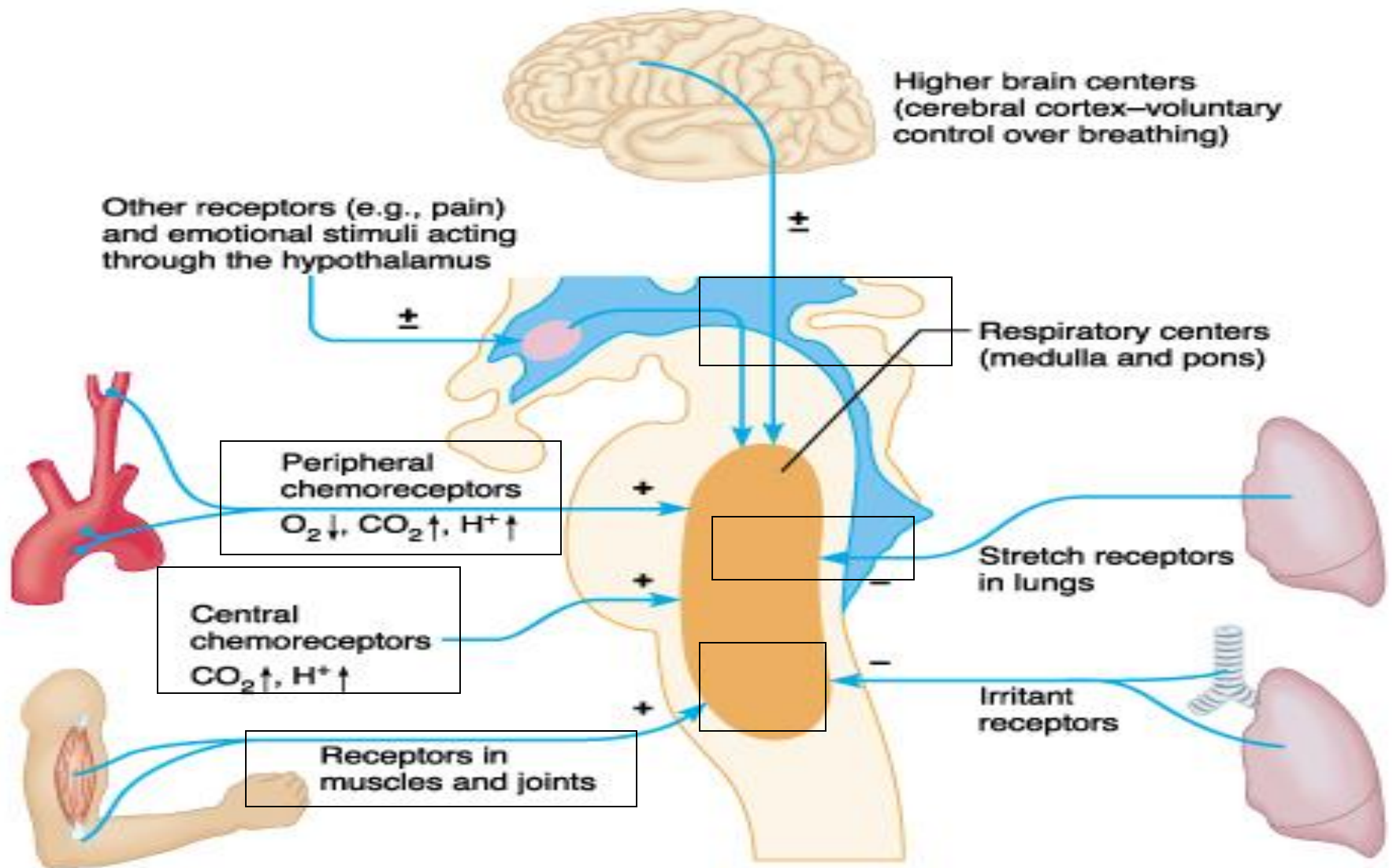


# Effect of blood CO<sub>2</sub> level on central chemoreceptors



**Figure 16.29** How blood CO<sub>2</sub> affects chemoreceptors in the medulla oblongata. An increase in blood CO<sub>2</sub> stimulates breathing indirectly by lowering the pH of blood and cerebrospinal fluid (CSF). This figure illustrates how a rise in blood CO<sub>2</sub> increases the H<sup>+</sup> 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.

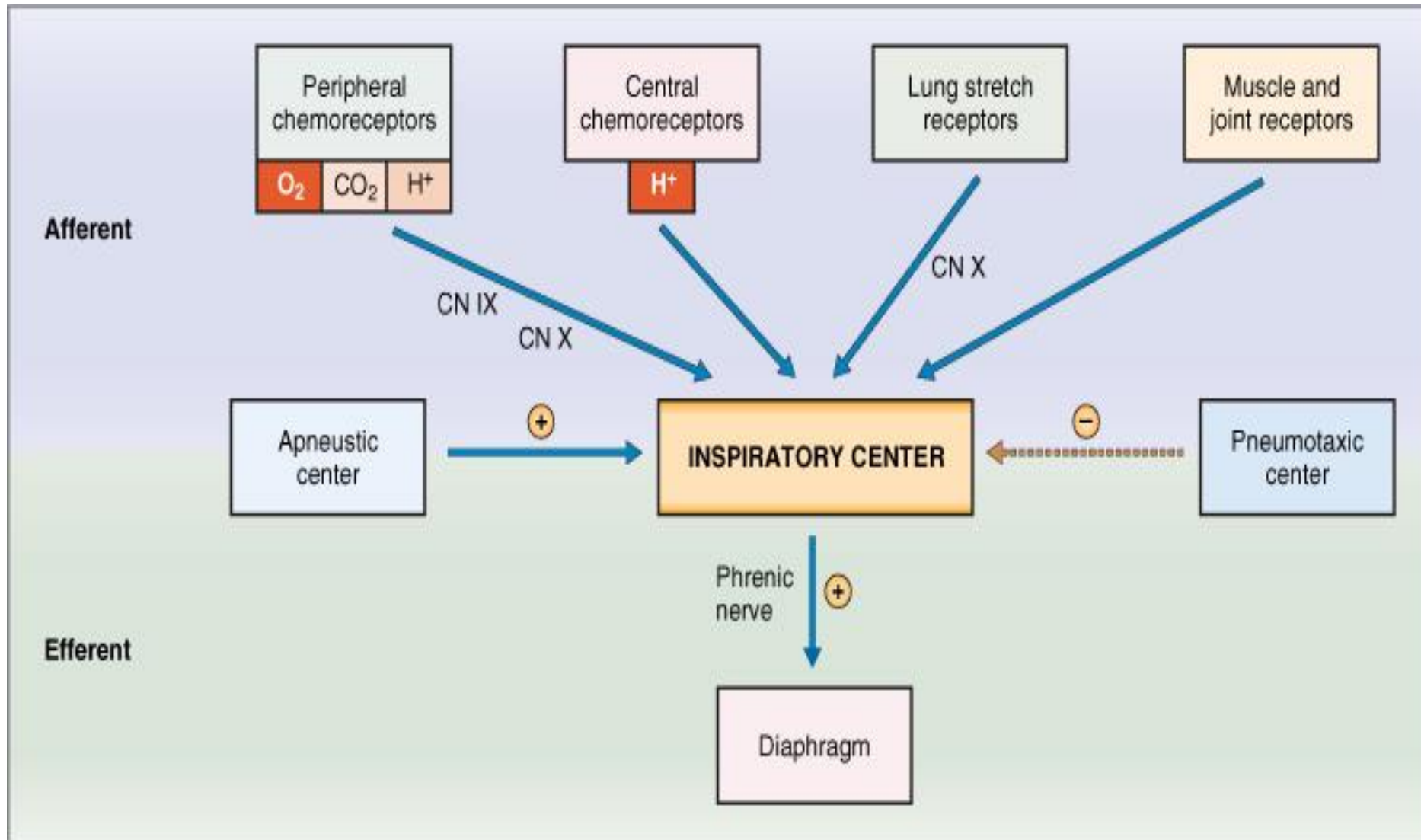
## Cont. factor affecting respiratory centers

- ⊙ **Effect of Irritant receptors in the airways:** the **epithelium** of trachea, bronchi, and bronchioles is supplied by irritant receptors that are stimulated by irritants that enter the respiratory airways causing coughing, sneezing and bronchoconstriction in bronchial asthma and emphysema.

- ⊙ **Function of lung J receptors.**

Few receptors in the wall of the alveoli in juxta position to the pulmonary capillaries. They are stimulated especially when pulmonary capillaries become engorged by blood or when pulmonary edema occur e.g in CHF, their excitation cause the person a feeling of dyspnea.

# Cont..factors affecting respiration



## Respiratory Acidosis

- Hypoventilation.
- Accumulation of  $\text{CO}_2$  in the tissues.
  - $P_{\text{CO}_2}$  increases
  - pH decreases.

## Respiratory Alkalosis

- Hyperventilation.
- Excessive loss of  $\text{CO}_2$ .
  - $P_{\text{CO}_2}$  decreases ( $\downarrow 35$  mmHg).
  - pH increases.

# Effects of low and high gas pressure on the body

# 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 ( manage by) SCUBA (self contained under water breathing apparatus).

# Deep-Sea Diving (High pressure)

- Relationship of Pressure to Sea Depth: a person **33 feet** beneath the ocean surface is exposed to **2 atmospheres pressure**
- At 66 feet the pressure is **3 atmospheres**, and so forth..

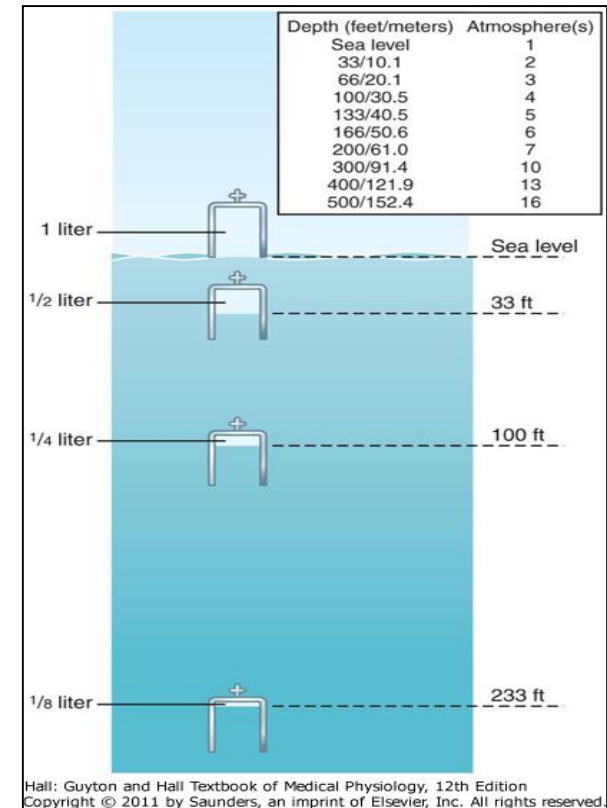


Figure 44-1 Effect of sea depth on pressure (top table) and on gas volume (bottom).

➤ **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 at High Nitrogen Pressures

- About four fifths of the air is nitrogen. At sea-level pressure, the nitrogen has **no significant effect on bodily function** but at high pressures it can cause varying degrees of narcosis
- Nitrogen narcosis has characteristics similar to those of **alcohol intoxication**

## Nitrogen narcosis Cont..

- 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 (happiness )and drowsiness and impaired performance.
- At higher pressure: loss of coordination and finally coma might develop.

# decompression sickness and how it can be avoided.

- Breathes air under high pressure for a long time--**nitrogen dissolved in the body fluids** increases
- And because nitrogen **is not metabolized by the body**, it remains dissolved in all the body tissues until the nitrogen pressure in the lungs is decreased back to some lower level, however, **this removal often takes hours** to occur and **is the source of multiple problems** collectively called **decompression sickness**

# decompression sickness and how it can be avoided. Cont...

- if diver suddenly comes back to the surface of the sea, **nitrogen bubbles can develop** in the body fluids either intracellularly or extracellularly **decompression sickness**.
- Due to decompression gases can escape from the dissolved state and form actual bubbles, in both tissues and blood where they plug many small blood vessels. **The bubbles may not appear for many minutes to hours because sometimes the gases can remain dissolved in the "supersaturated" state for hours before bubbling.**



## 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 N<sub>2</sub> 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** N<sub>2</sub> 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-O<sub>2</sub> mixture** is breathed during the dive..

## Cont.. Treatment of DS

- Helium is more desirable than nitrogen in deep dives because it has:
- $\frac{1}{4}$ - $\frac{1}{5}$  the narcotic effect of nitrogen on CNS.
- $\frac{1}{7}$  the molecular weight of nitrogen.
- low density leading to decreased air way resistance of diver.
- High diffusion through tissues.
- Helium is about  $\frac{1}{2}$  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

# Treatment of Decompression Sickness

- Put the diver into a **pressurized tank** to lower the pressure gradually back to normal atmospheric pressure, by using this time schedule.

10 minutes at 50 feet depth

17 minutes at 40 feet depth

19 minutes at 30 feet depth

50 minutes at 20 feet depth

84 minutes at 10 feet depth

# 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.



# High altitude (Low pressure) Cont ..

Table 43-1. Effects of Acute Exposure to Low Atmospheric Pressures on Alveolar Gas Concentrations and Arterial Oxygen Saturation\*

Altitude (ft/meters)	Barometric Pressure (mm Hg)	Breathing Air				Breathing Pure Oxygen		
		P <sub>O</sub> <sub>2</sub> in Air (mm Hg)	P <sub>CO</sub> <sub>2</sub> in Alveoli (mm Hg)	P <sub>O</sub> <sub>2</sub> in Alveoli (mm Hg)	Arterial Oxygen Saturation (%)	P <sub>CO</sub> <sub>2</sub> in Alveoli (mm Hg)	P <sub>O</sub> <sub>2</sub> in Alveoli (mm Hg)	Arterial Oxygen Saturation (%)
0	760	159	40 (40)	104 (104)	97 (97)	40	673	100
10,000/3048	523	110	36 (23)	67 (77)	90 (92)	40	436	100
20,000/6096	349	73	24 (10)	40 (53)	73 (85)	40	262	100
30,000/9144	226	47	24 (7)	18 (30)	24 (38)	40	139	99
40,000/12,192	141	29				36	58	84
50,000/15,240	87	18				24	16	15

\*Numbers in parentheses are acclimatized values.

## Alveolar PO<sub>2</sub> 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 PO<sub>2</sub>= 159 mmHg.
- At 20,000 feet PO<sub>2</sub>= 40 mmHg.
- At 50,000 feet PO<sub>2</sub>= only 18 mmHg.

## Cont...

- Even at high altitude CO<sub>2</sub> 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 (max and weaker) 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, laziness, mental and muscle fatigue, sometimes headache, occasionally nausea and sometimes euphoria(happiness) .
- 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 PO<sub>2</sub>

- A person remaining at high altitudes for days , weeks or years becomes more and more acclimatized to low PO<sub>2</sub>.
- 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  $PO_2$ .

