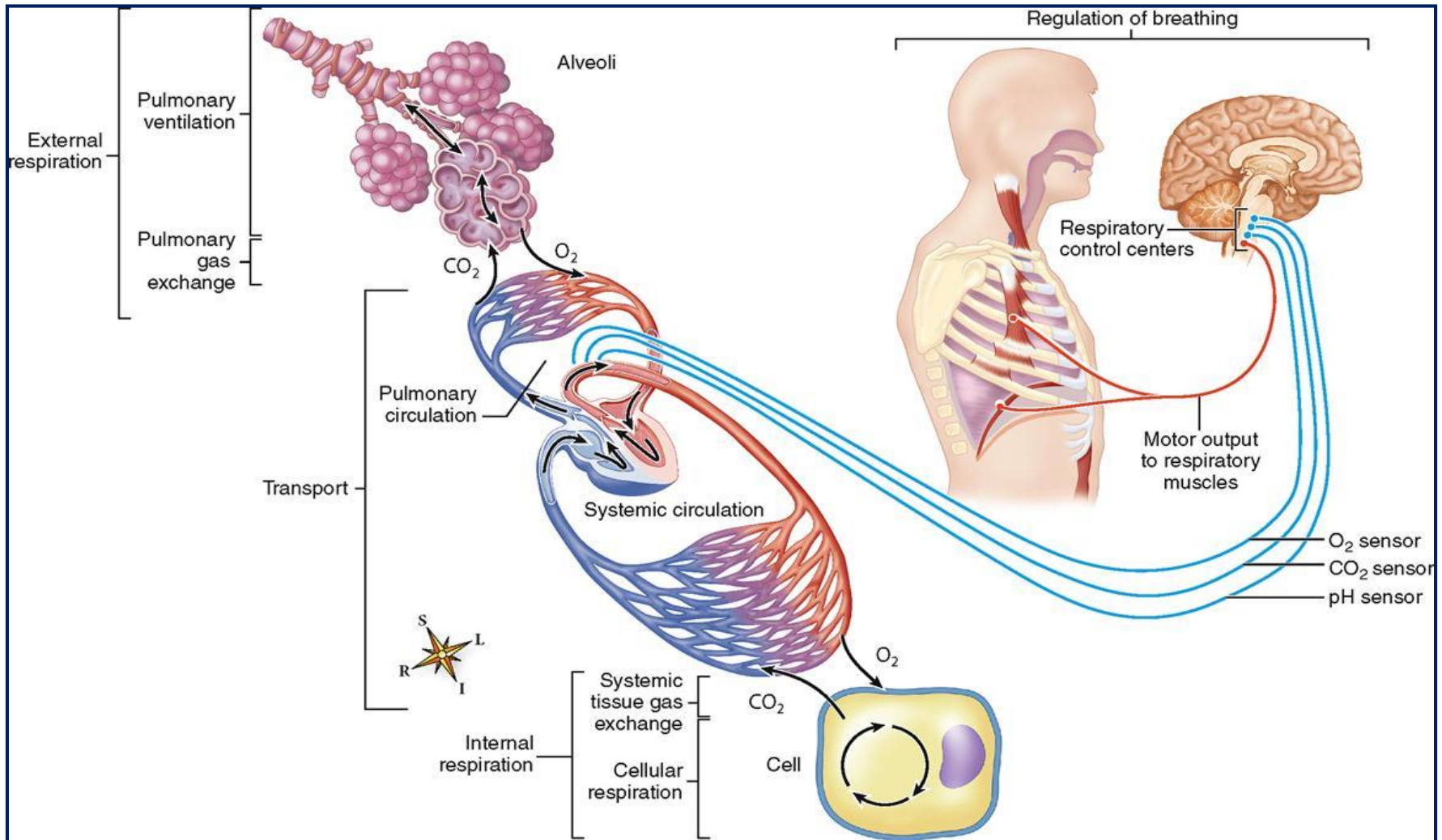


# Control of Breathing

# The overall processes of External Respiration



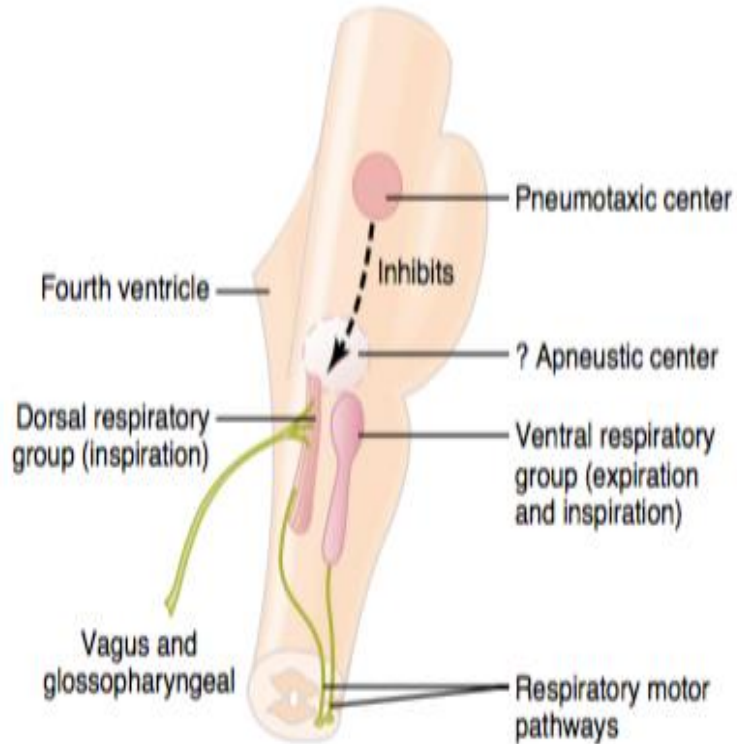
# Objectives

- **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, 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

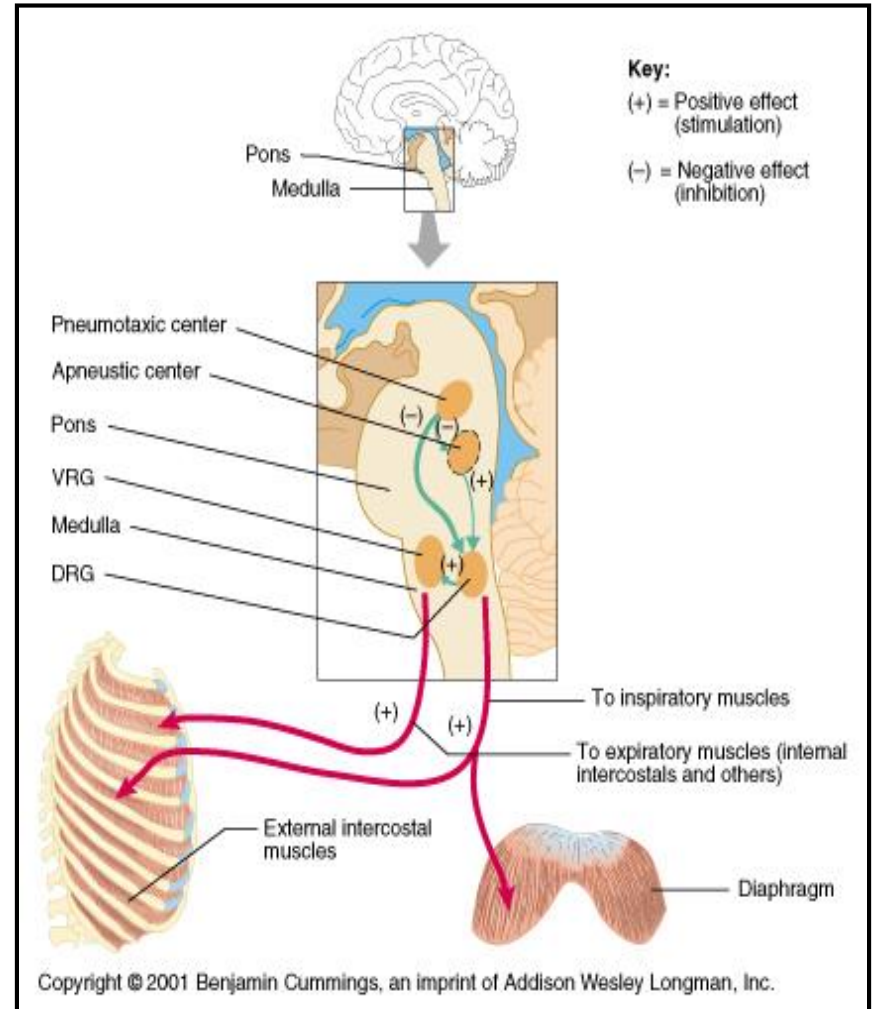
- Arterial PO<sub>2</sub>
  - When PO<sub>2</sub> is VERY low (Hypoxia), ventilation increases.
- Arterial PCO<sub>2</sub>
  - The most important regulator of ventilation is PCO<sub>2</sub>, small increases in PCO<sub>2</sub>, greatly increases ventilation.
- Arterial pH
  - As hydrogen ions increase (acidosis), alveolar ventilation increases.

# Respiratory Centers



**Figure 41-1**

Organization of the respiratory center.



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# 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**
  - Although it contains both inspiratory and expiratory neurons. It is **inactive during normal quiet** breathing.
  - Activated by inspiratory area during **forceful breathing**.
  - Causes contraction of the 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 (Bridge) Respiratory centers

- Transition between inhalation and exhalation is controlled by:

## **Pneumotaxic area**

Inhibits inspiratory area of medulla to stop inhalation.

Therefore, breathing is more rapid when pneumotaxic area is active.

## **Apneustic area**

Stimulates inspiratory area of medulla to **prolong inhalation**. Therefore slow respiration and prolonged respiratory cycles will result if it is stimulated.

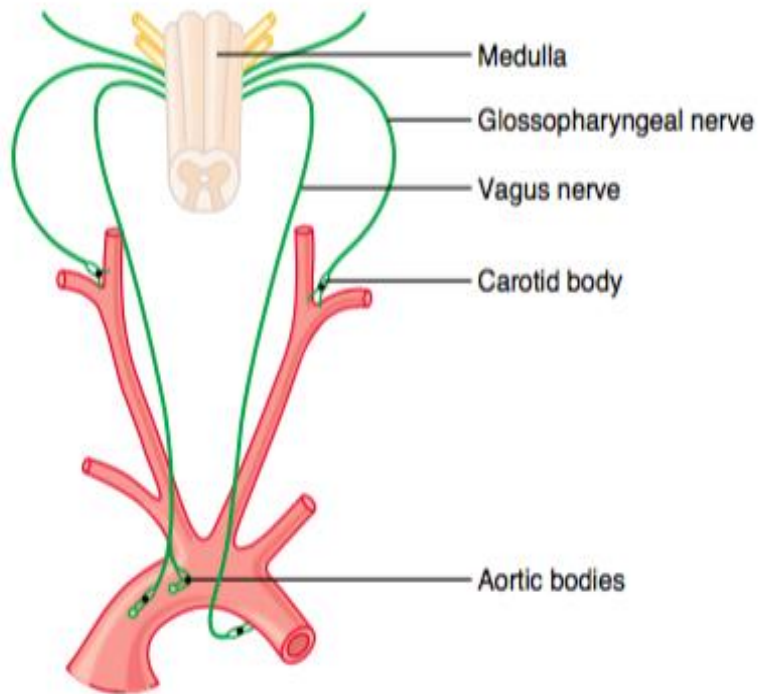
# Hering-Breuer inflation reflex

- When the lung becomes overstretched (tidal volume is about 1.5 L), stretch receptors located in the wall of 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 **increases the rate of respiration** as does the pneumotaxic center.
- This reflex appears to be mainly a **protective mechanism for preventing excess lung inflation**



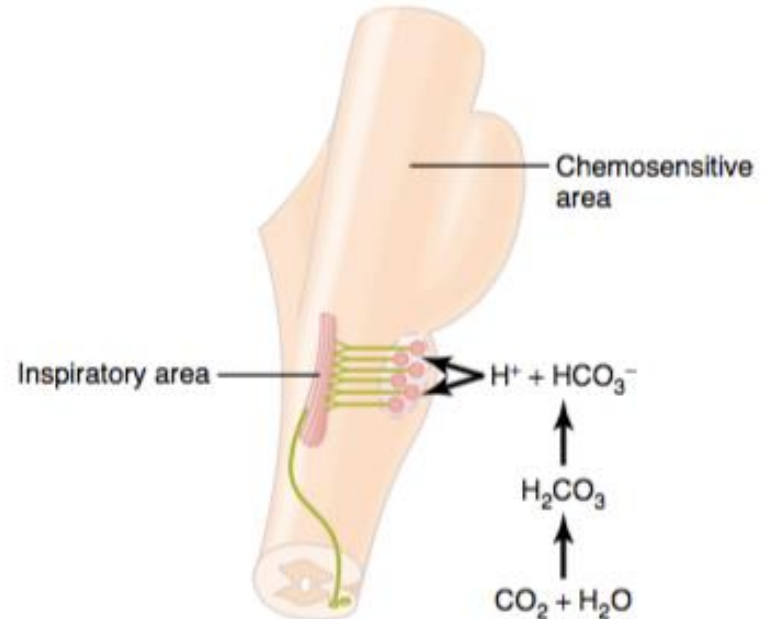
# Chemical Control of Respiration

## *Peripheral and central chemoreceptors*



**Figure 41-4**

Respiratory control by peripheral chemoreceptors in the carotid and aortic bodies.

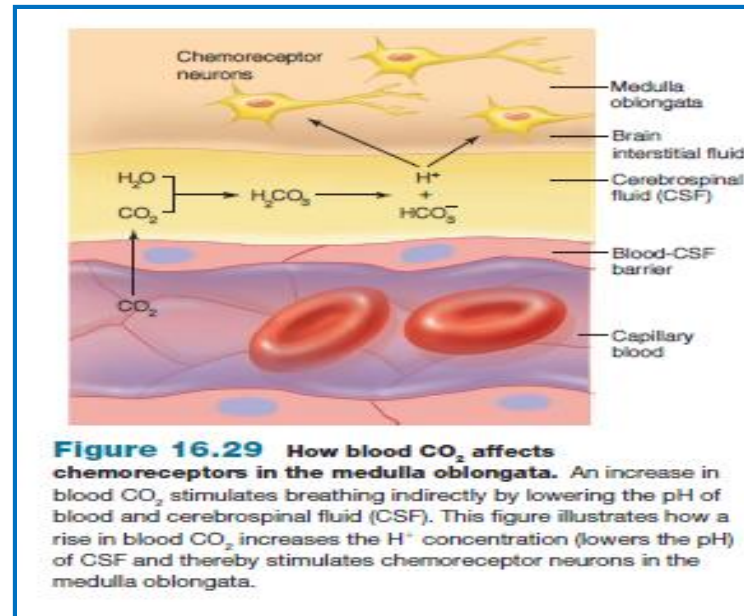
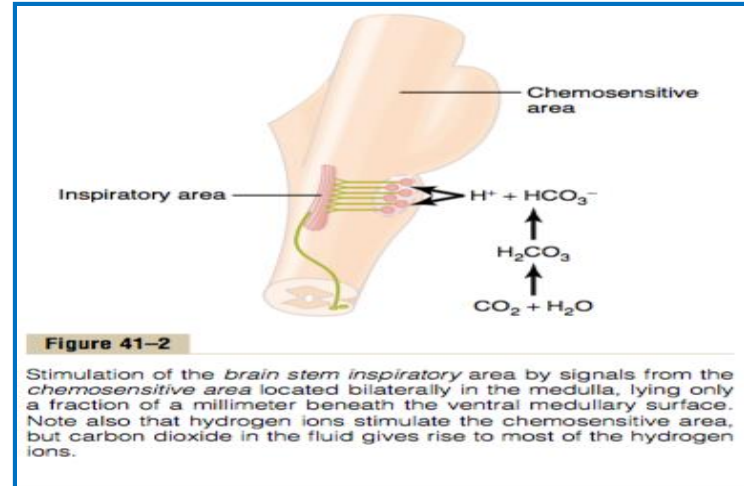


**Figure 41-2**

Stimulation of the *brain stem inspiratory area* by signals from the *chemosensitive area* located bilaterally in the medulla, lying only a fraction of a millimeter beneath the ventral medullary surface. Note also that hydrogen ions stimulate the chemosensitive area, but carbon dioxide in the fluid gives rise to most of the hydrogen ions.

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

Although **carbon dioxide** has little direct effect in stimulating the neurons in the chemosensitive area, **it does have a potent (effective) indirect effect.** It does this by reacting with the water of the tissues to form carbonic acid, which dissociates into hydrogen and bicarbonate ions; **the hydrogen ions then have a potent direct stimulatory effect on respiration.**



## Why does blood carbon dioxide have a more potent effect in stimulating the chemosensitive neurons than do blood hydrogen ions?

The blood- brain barrier (BBB) is nearly impermeable to  $H^+$  ions, but  $CO_2$  passes this barrier very easily. When the blood  $PCO_2$  increases, so does the  $PCO_2$  of both the interstitial fluid of the medulla and the CSF. In these fluids, the  $CO_2$  reacts with the water to form new  $H^+$  ions. Thus, more  $H^+$  ions are released into the respiratory chemosensitive sensory area of the medulla when the blood  $CO_2$  concentration increases than when the blood  $H^+$  ion increases. For this reason, respiratory center activity is increased very strongly by changes in blood  $CO_2$ , a fact that we subsequently discuss quantitatively.

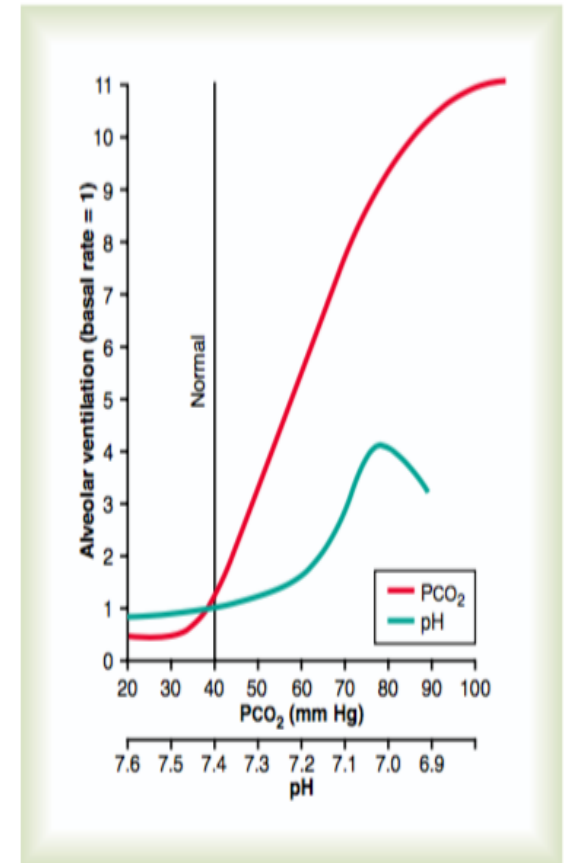


Figure 41-3

Effects of increased arterial blood  $PCO_2$  and decreased arterial pH (increased hydrogen ion concentration) on the rate of alveolar ventilation.

**A change in blood CO<sub>2</sub> concentration has a potent acute effect on controlling respiratory drive but only a weak chronic effect after a few days' adaptation.**

- Excitation of the respiratory center by CO<sub>2</sub> is great after the blood CO<sub>2</sub> first increases, but it gradually **declines over the next 1 to 2 days**.
- Part of this decline results from **renal readjustment** of the H<sup>+</sup> ion concentration in the circulating blood back toward normal after the CO<sub>2</sub> first increases.
- The kidneys increasing the blood HCO<sub>3</sub>, which binds with H<sup>+</sup> ions in the blood and CSF to reduce their concentrations.
- Over a period of hours, the HCO<sub>3</sub> (Bicarbonate) ions slowly diffuse through the BBB (blood- brain barrier) – CSF barriers and combine directly with the H<sup>+</sup> ions adjacent to the respiratory neurons as well, thus reducing the H<sup>+</sup> ions back to near normal.

# Peripheral Chemoreceptor System Activity—Role of Oxygen in Respiratory Control

- Most of the chemoreceptors are in the *carotid bodies*. However, a few are also in the *aortic bodies*.
- When the oxygen concentration in the arterial blood falls below normal, the chemoreceptors **become strongly stimulated**.
- The impulse rate is particularly sensitive to changes in arterial  $PO_2$  in the range of 60 down to 30 mm Hg.
- Under these conditions, low arterial  $PO_2$  obviously drives the ventilatory process quite strongly.

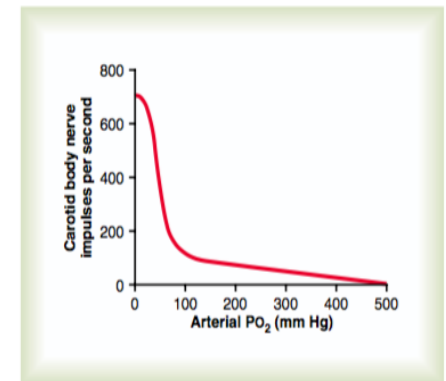


Figure 41-5

Effect of arterial  $PO_2$  on impulse rate from the carotid body of a cat.

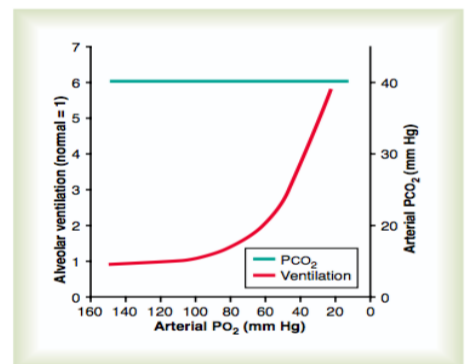
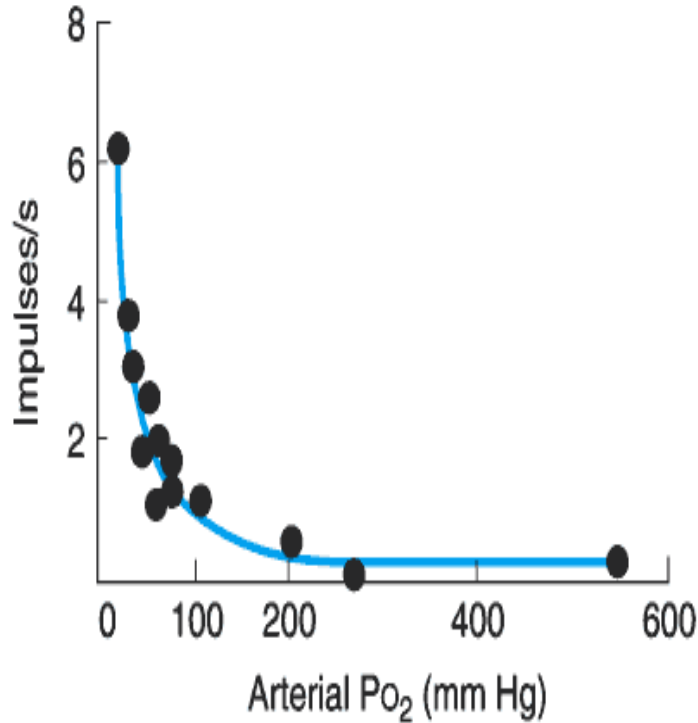


Figure 41-6

The lower curve demonstrates the effect of different levels of arterial  $PO_2$  on alveolar ventilation, showing a sixfold increase in ventilation as the  $PO_2$  decreases from the normal level of 100 mm Hg to 20 mm Hg. The upper line shows that the arterial  $PCO_2$  was kept at a constant level during the measurements of this study; pH also was kept constant.

# Summary of Chemoreceptor Control of Breathing

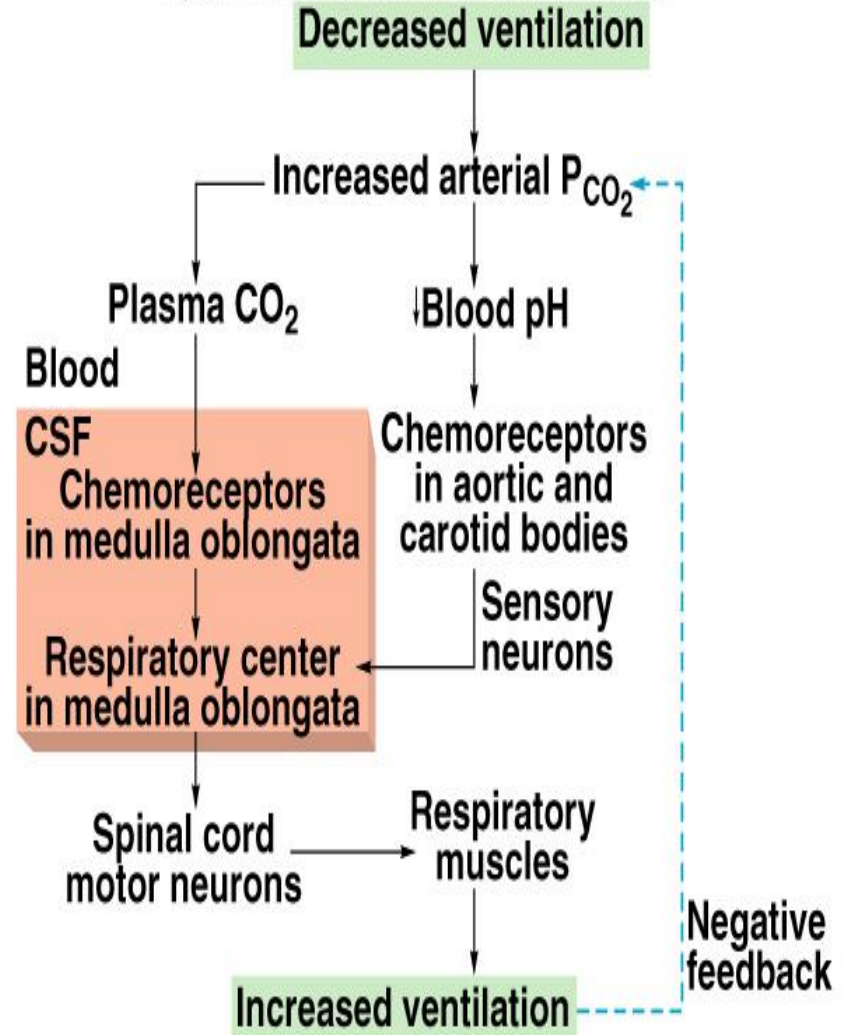
Figure 36-6.



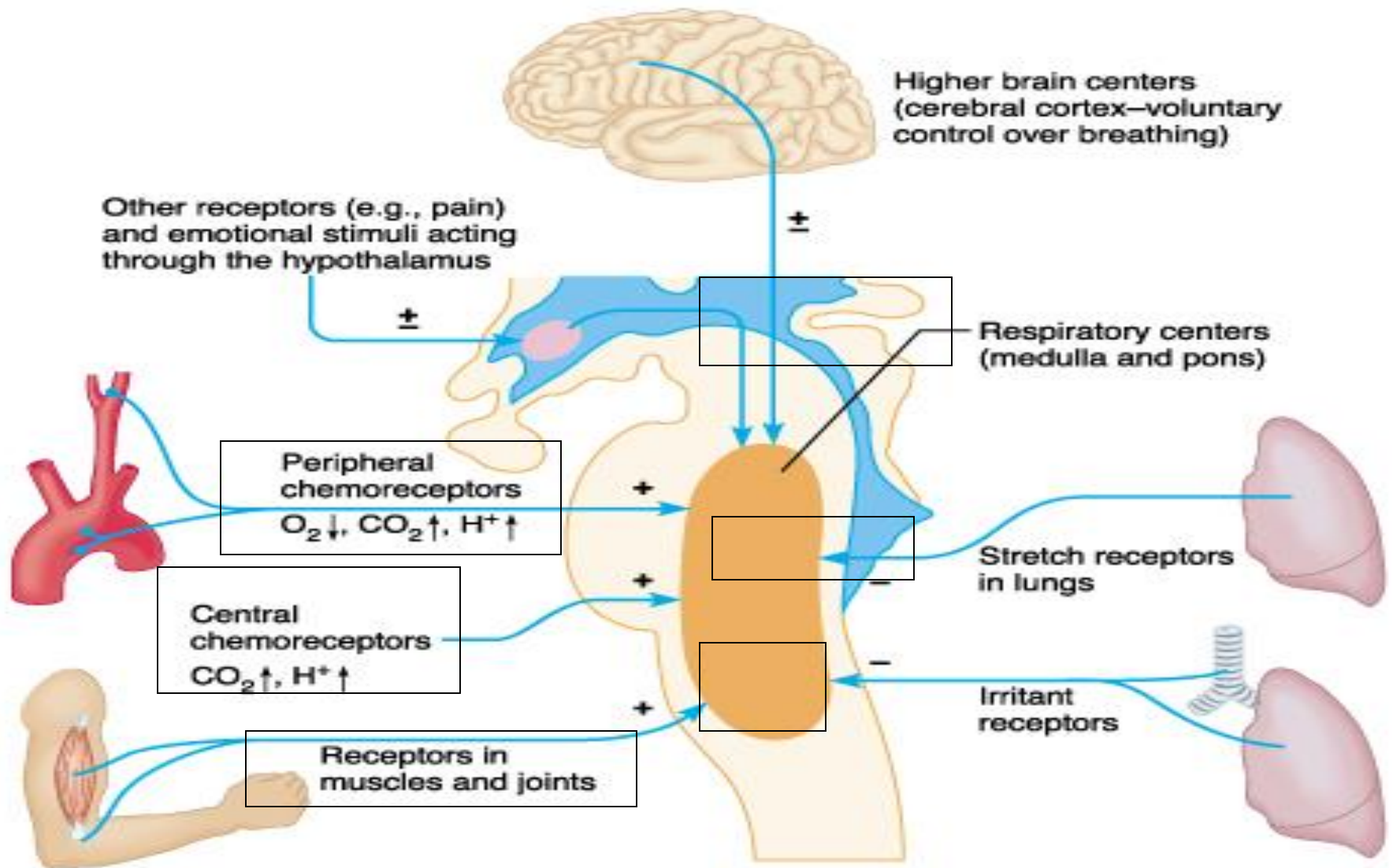
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Change in the rate of discharge of a single afferent fiber from the carotid body when arterial PO<sub>2</sub> is reduced. (Courtesy of S Sampson.)

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# Other Factors Influencing Respiration



## Cont. factor affecting respiratory centers

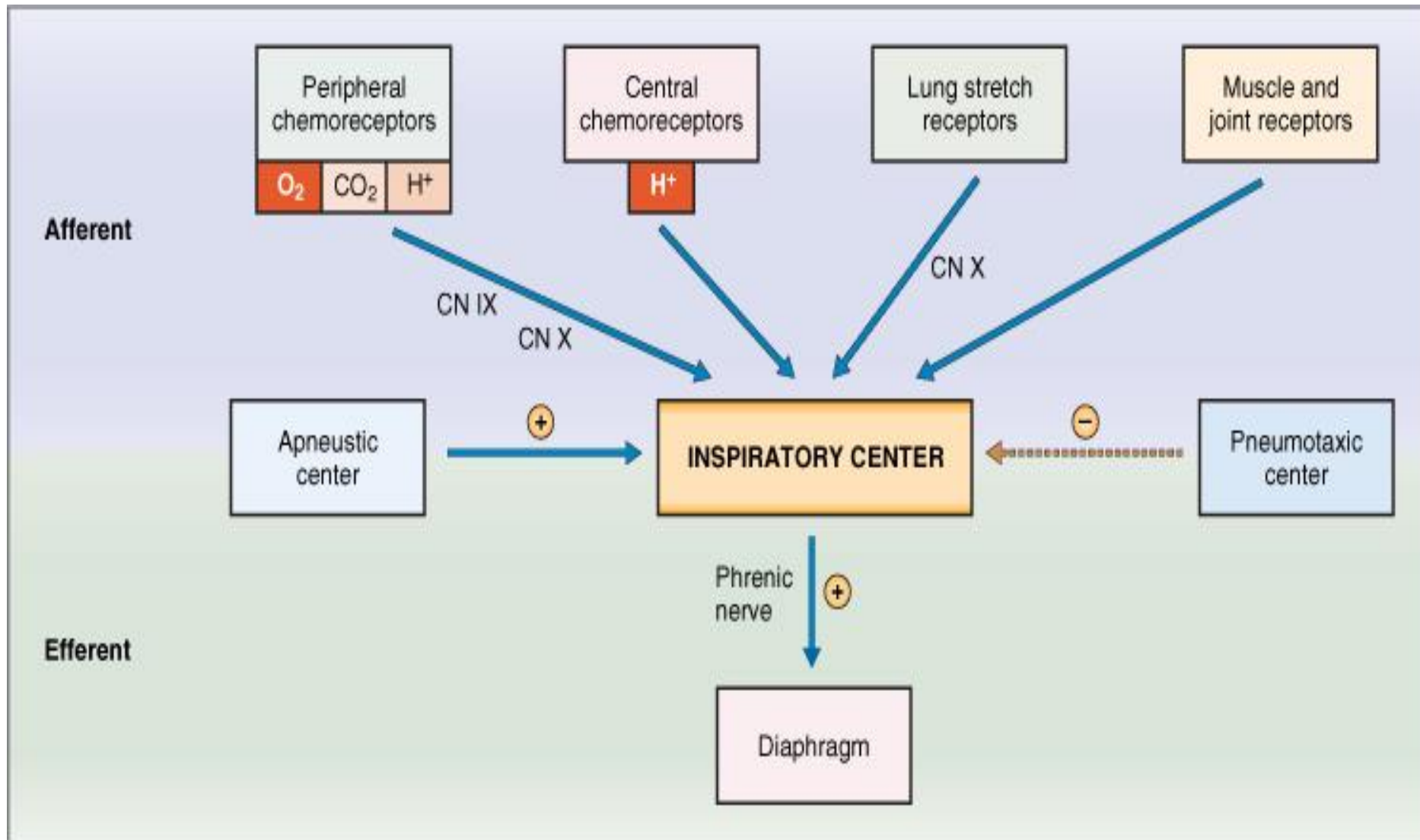
- ⊙ **Effect of Irritant receptors in the airways:** the **epithelium** of trachea, bronchi, and bronchioles is supplied by irritant receptors that are stimulated by irritants that enter the respiratory airways causing coughing, sneezing and bronchoconstriction in bronchial asthma and emphysema.

- ⊙ **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.
  - $\text{P}_{\text{CO}_2}$  increases
  - pH decreases.

## Respiratory Alkalosis

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

## Metabolic Acidosis

- Ingestion (**In**), 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