Regulation of respiration

By pharmacist
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Refference: guyton,text book of medical physiology

objectives

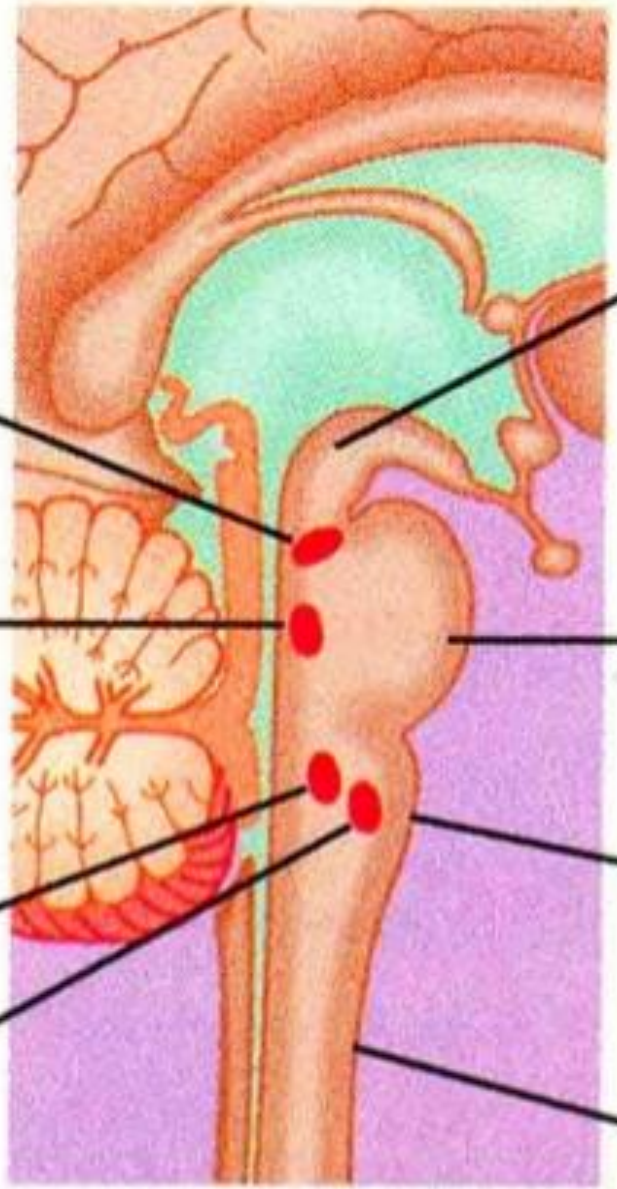
To learn :

1- neural control of respiration.

2- Chemical Control of Respiration

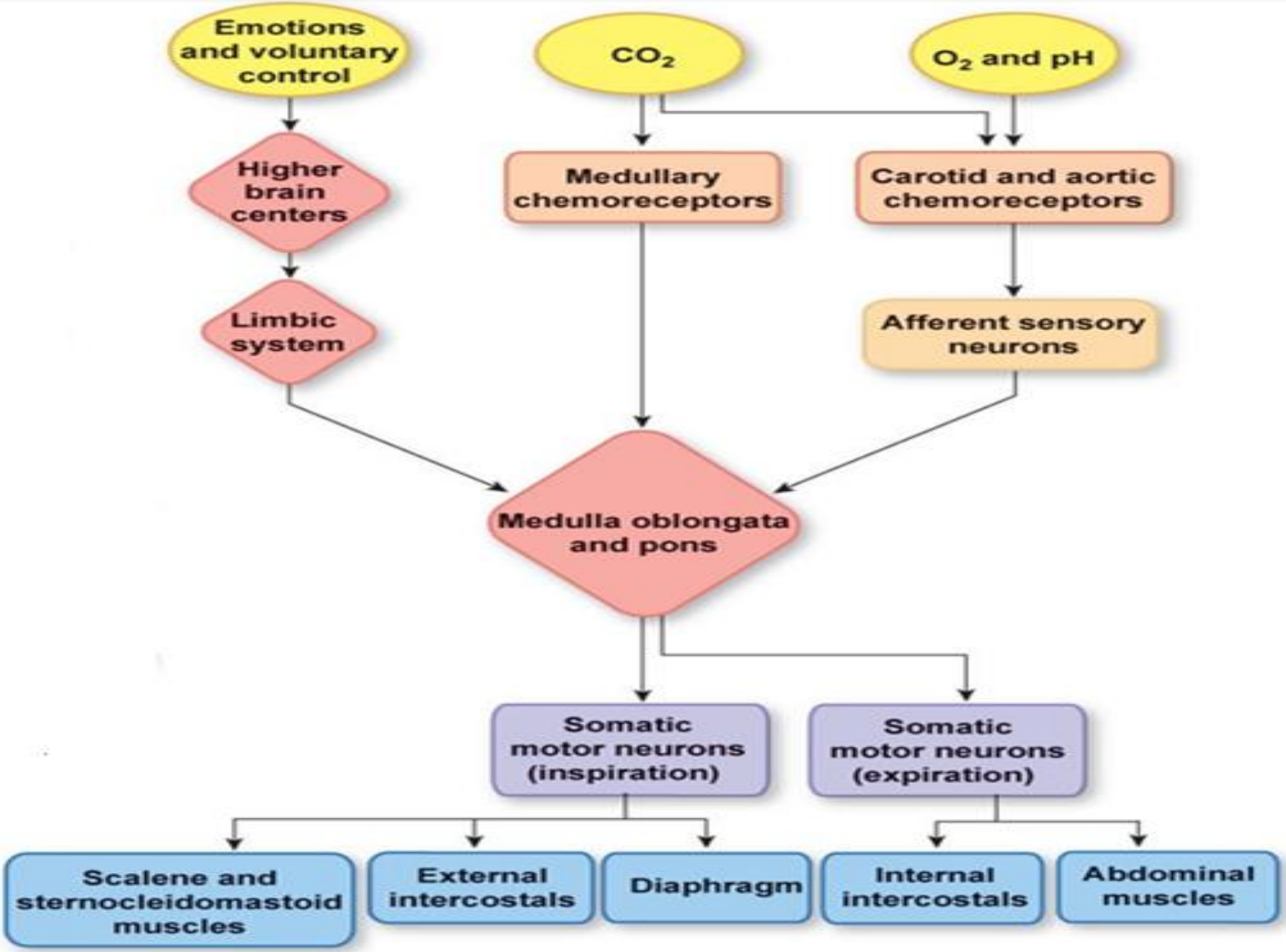
- *respiratory center* located in the *medulla oblongata* & pons of the brain stem,
- (1) a *dorsal respiratory group*,in medulla causes inspiration.
- (2) a *ventral respiratory group in medulla* causes expiration.
- (3) the *pneumotaxic center*,in pons controls rate &depth of breathing.

RESPIRATORY CENTER



Sagittal section of brainstem

- Dorsal Respiratory Group of Neurons located within the *nucleus of the tractus solitarius (NTS)*,which is the sensory termination of both the vagal and the glossopharyngeal nerves, which transmit sensory signals into the respiratory center from
  - (1) peripheral chemoreceptors,
  - (2) baroreceptors,
  - (3) several types of receptors in the lungs.


Emotions and voluntary control
CO_2
O_2 and pH
Higher brain centers
Medullary chemoreceptors
Carotid and aortic chemoreceptors
Limbic system
Afferent sensory neurons
Medulla oblongata and pons
Somatic motor neurons (inspiration)
Somatic motor neurons (expiration)
Scalene and sternocleidomastoid muscles
External intercostals
Diaphragm
Internal intercostals
Abdominal muscles

Inspiratory "Ramp" Signal

- nervous signal that is transmitted to the inspiratory muscles begins weakly & increases steadily in a ramp manner for about 2 seconds in normal respiration.
- Then it ceases abruptly for approximately the next 3 seconds, which turns off the excitation of the diaphragm and allows elastic recoil of the lungs and the chest wall to cause expiration. Next, the inspiratory signal begins again for another cycle; this cycle repeats again and again, with expiration occurring in between.

There are two qualities of the inspiratory ramp that are controlled, as follows:

1- Control of the *rate of increase of the ramp signal* so that during heavy respiration, the ramp increases rapidly and therefore fills the lungs rapidly.

2- Control of the *limiting point at which the ramp suddenly ceases*.

This is the usual method for controlling the rate of respiration; that is, the earlier the ramp ceases, the shorter the duration of inspiration.

This also shortens the duration of expiration.

Thus, the frequency of respiration is ↑.

Ventral Respiratory Group of Neurons-Functions in Both Inspiration and Expiration

- function of this neuronal group differs from that of the dorsal respiratory group in several important ways:
- 1-The neurons of the ventral respiratory group remain *inactive* during normal respiration. Therefore, normal quiet breathing is caused only by repetitive inspiratory signals from the dorsal respiratory group transmitted to the diaphragm, and expiration results from elastic recoil of the lungs and thoracic cage.
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- 2- Electrical stimulation of a few of the neurons in the ventral group causes inspiration, whereas stimulation of others causes expiration.
- Therefore, these neurons contribute to both inspiration and expiration.
- They are especially important in providing the powerful expiratory signals to the abdominal muscles during very heavy expiration.
- Thus, this area operates more or less as an overdrive mechanism when high levels of pulmonary ventilation are required, especially during heavy exercise.

Pneumotaxic Center

- transmits signals to the inspiratory area.
- control the "switch-off" point of the inspiratory ramp& controlling the duration of the filling phase of the lung cycle.
- If pneumotaxic signal is strong, inspiration might last for as little as 0.5 second,
- When pneumotaxic signal is weak, inspiration might continue for 5 seconds, thus filling the lungs with a great excess of air.

- The function of the pneumotaxic center is primarily to limit inspiration

The Hering-Breuer Inflation Reflex

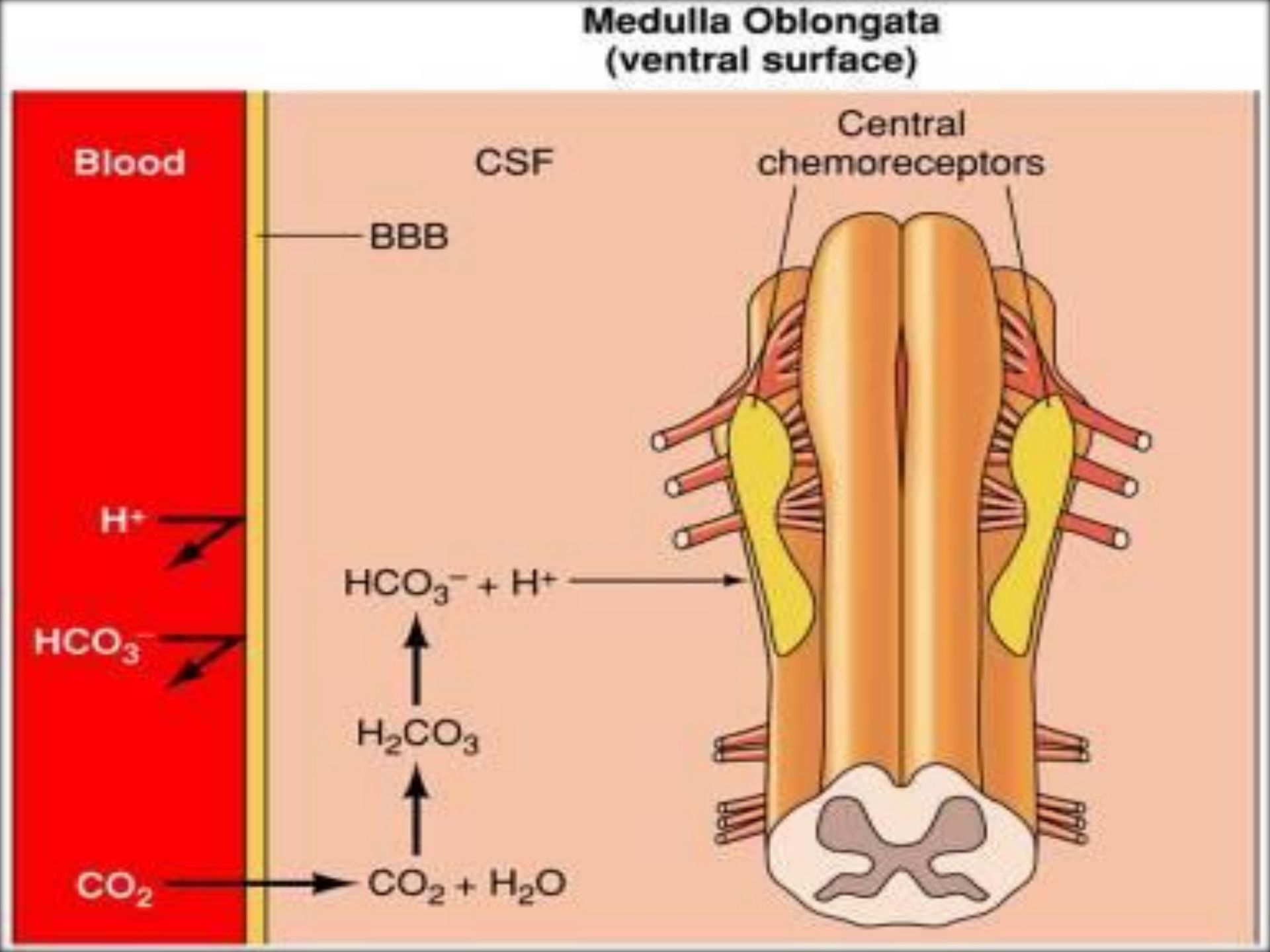
- sensory nerve signals from the lungs also help control respiration.
- *stretch receptors* that transmit signals through the *vagi* into the dorsal respiratory group of neurons when the lungs become overstretched.
- when lungs become overly inflated, stretch receptors activate a feedback response that "switches off" the inspiratory ramp and thus stops further inspiration.
- This is called the *Hering-Breuer inflation reflex*.

- Hering-Breuer reflex is not activated until the tidal volume increases to three times normal (>≈ 1.5 liters per breath).
- Therefore, this reflex appears to be a protective mechanism for preventing excess lung inflation rather than an important ingredient in normal control of ventilation.

Chemical Control of Respiration

- goal of respiration is to maintain proper concentrations of oxygen, carbon dioxide, and hydrogen ions in the tissues.
- Excess carbon dioxide or excess hydrogen ions in the blood mainly act directly on the respiratory center, causing greatly increased strength of both the inspiratory and the expiratory motor signals to the respiratory muscles.

- a *chemosensitive area*, in medulla ,highly sensitive to changes in blood P_{CO_2} or H+ concentration, and it in turn excites the other portions of the respiratory center.
- sensor neurons in chemosensitive area are especially excited by H+.
- H+ do not cross the blood-brain barrier


Medulla Oblongata
(ventral surface)
Blood
CSF
Central
chemoreceptors
BBB
H+
HCO3−
HCO3− + H+
H2CO3
CO2
CO2 + H2O

- so changes in H+ concentration in the blood have less effect in stimulating the chemosensitive neurons than do changes in bloodCO2.
- CO2 have a potent indirect effect.
- by reacting with the water of the tissues to form carbonic acid, which dissociates into hydrogen and bicarbonate ions; the H+ have a potent direct stimulatory effect on respiration.
- respiratory center activity is ↑very strongly by changes in blood CO2.

Excitation of the respiratory center by CO_2 is great the first few hours after the blood CO_2 first increases.

Then gradually declines over the next 1 to 2 days

decline results from renal readjustment of the H^+ concentration in the circulating blood back to normal.

Kidney increasing the bloodHCO_3, which binds with the H^+ in the blood and cerebrospinal fluid to ↓their concentrations.

- After hours, the bicarbonate ions also slowly diffuse through blood-brain and blood-cerebrospinal fluid barriers and combine directly with the H+ adjacent to the respiratory neurons, thus ↓the hydrogen ions back to near normal.
- A change in blood CO2concentration has a potent *acute* effect on controlling respiratory drive but only a weak *chronic* effect after a few days' adaptation

- very marked ↑ in ventilation caused by an ↑in P_{CO_2} *in the normal range* between 35 and 75 mm Hg.

Peripheral Chemoreceptor System for Control of Respiratory Activity-Role of Oxygen in Respiratory Control

- Special nervous chemical receptors, called *peripheral chemoreceptor system* located in the *carotid bodies &aortic bodies*, for detecting changes in O2,CO2 &H+ concentrations in the blood,
- The chemoreceptors transmit nervous signals to the respiratory center in the brain to help regulate respiratory activity.

- Oxygen, not have a significant *direct* effect on the respiratory center of the brain in controlling respiration.
- O2 acts on peripheral *chemoreceptors* located in the *carotid* and *aortic bodies*, and these in turn transmit nervous signals to the respiratory center for control of respiration.

- afferent nerve of *carotid bodies* pass through Hering's nerves to the *glossopharyngeal nerves* and then to the dorsal respiratory area of the medulla.
- afferent nerve fibers of *aortic bodies* pass through the *vagi*, also to the dorsal medullary respiratory area.

- Afferent fiber of carotid bodies •
- Pass through glossopharyngeal nerve •
- Dorsal respiratory area of medulla •

Afferent nerve of oartic bodies

Pass through vegi

To dorsal medullary respiratory area

- *chemoreceptors are exposed at all times to arterial blood*, not venous blood, and their P_{O_2} are arterial P_{O_2}.
- chemoreceptors strongly stimulated if arterial PO2↓, impulse rate is particularly sensitive to changes in arterial P_{O_2} in the range of (60 - 30 mm Hg), ventilation doubles when the arterial P_{O_2} falls to 60 mm Hg and can increase as much as 5fold at very low P_{O_2}.
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- ↑CO_2 or H+ concentration also excites the chemoreceptors,& indirectly increases respiratory activity.
- stimulation of peripheral chemoreceptors by CO_2 occurs as much as 5 times as rapidly as central stimulation,
- peripheral chemoreceptors important in increasing the rapidity of response to CO_2 at the onset of exercise.

Regulation of Respiration During Exercise

- In exercise, O2 consumption & CO2formation ↑as much as 20-fold.
- in the healthy athlete, alveolar ventilation increases almost exactly in step with the increased level of oxygen metabolism.
- The arterial P_{O_2}, P_{CO_2}, and pH remain *almost exactly normal*

What causes intense ventilation during exercise?

- The brain, on transmitting motor impulses to the exercising muscles, transmit at the same time collateral impulses into the brain stem to excite the respiratory center.
- This is analogous to the stimulation of the vasomotor center of the brain stem during exercise that causes a simultaneous increase in arterial pressure.

- when a person begins to exercise, a large share of the total increase in ventilation begins immediately on initiation of the exercise, before any blood chemicals have had time to change.
- It is likely that most of the increase in respiration results from neurogenic signals transmitted directly into the brain stem respiratory center at the same time that signals go to the body muscles to cause muscle contraction.