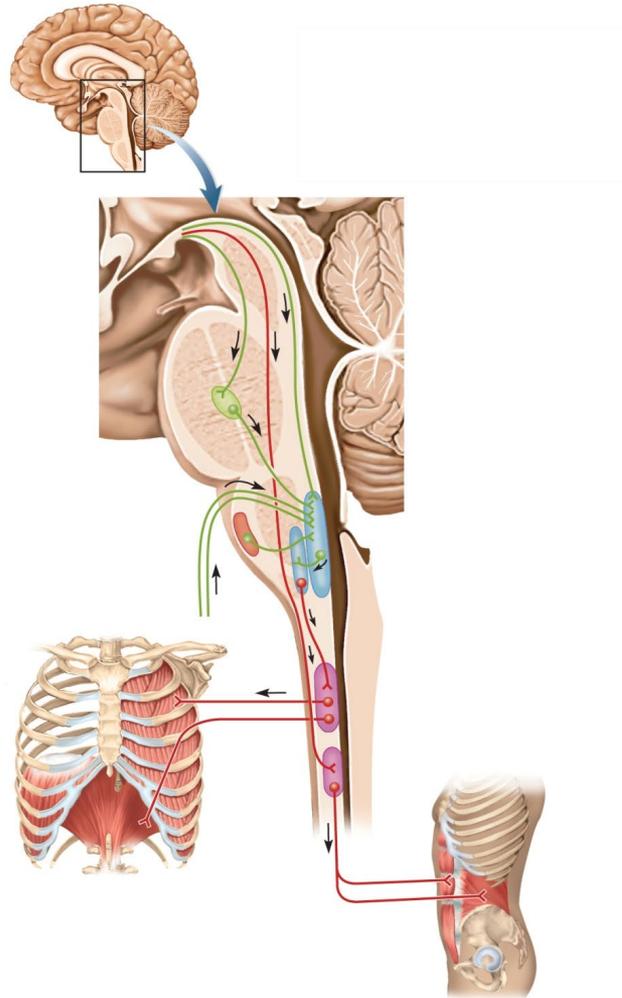


## Chapter 22.3

# Neural Control of Breathing



# Neural Control of Breathing

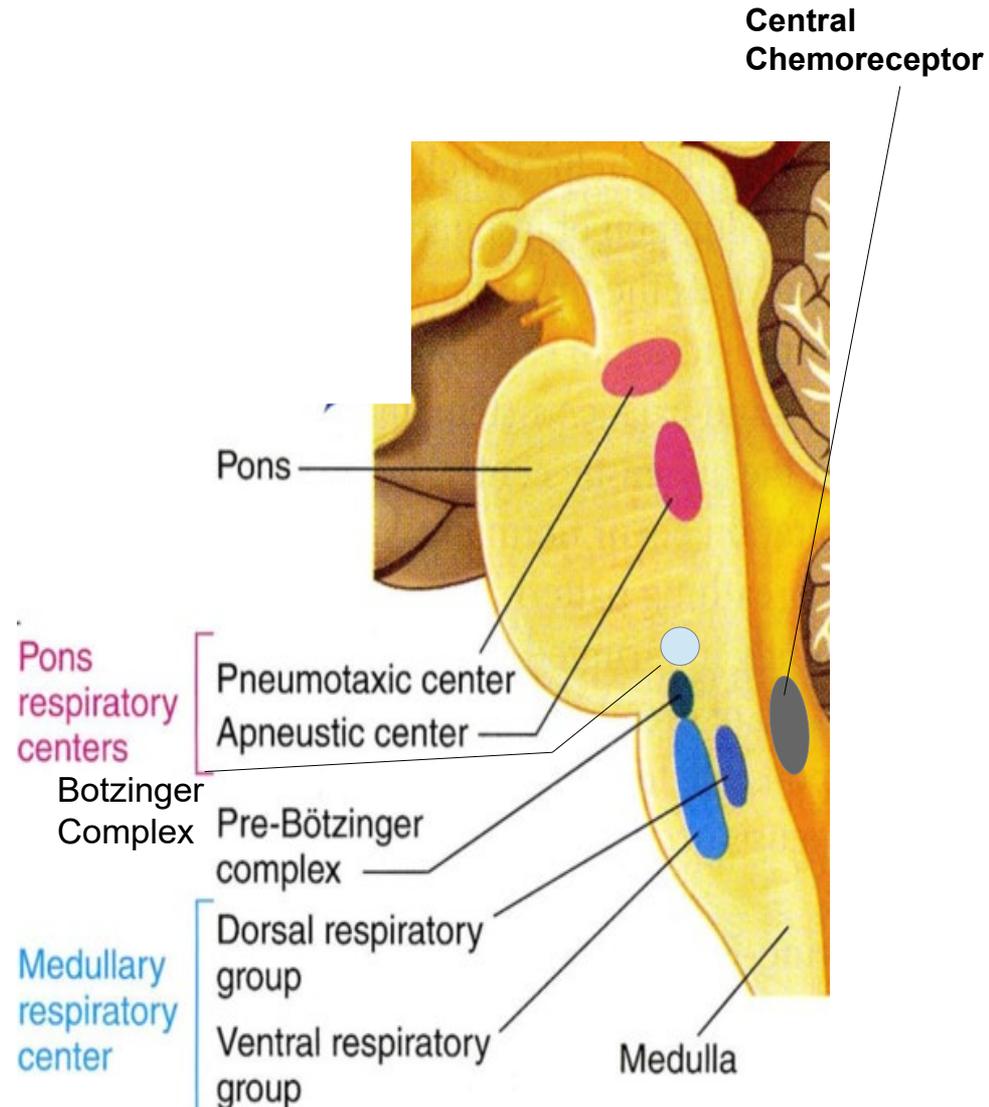
Breathing is the movement of air in and out of the lungs

Voluntary somatic motor pathways may initiate inspiration, however

Nuclei in the brain stem regulate rhythmic breathing // Two primary centers are the **Pons Respiratory Center and Medullary Respiratory Center**

*Breathing is also influenced by higher brain centers, central and peripheral chemo-receptors, stretch receptors, irritant receptors, and proprioceptors.*

No air moves in the transition between inspiration (air in) vs expiration (air out)



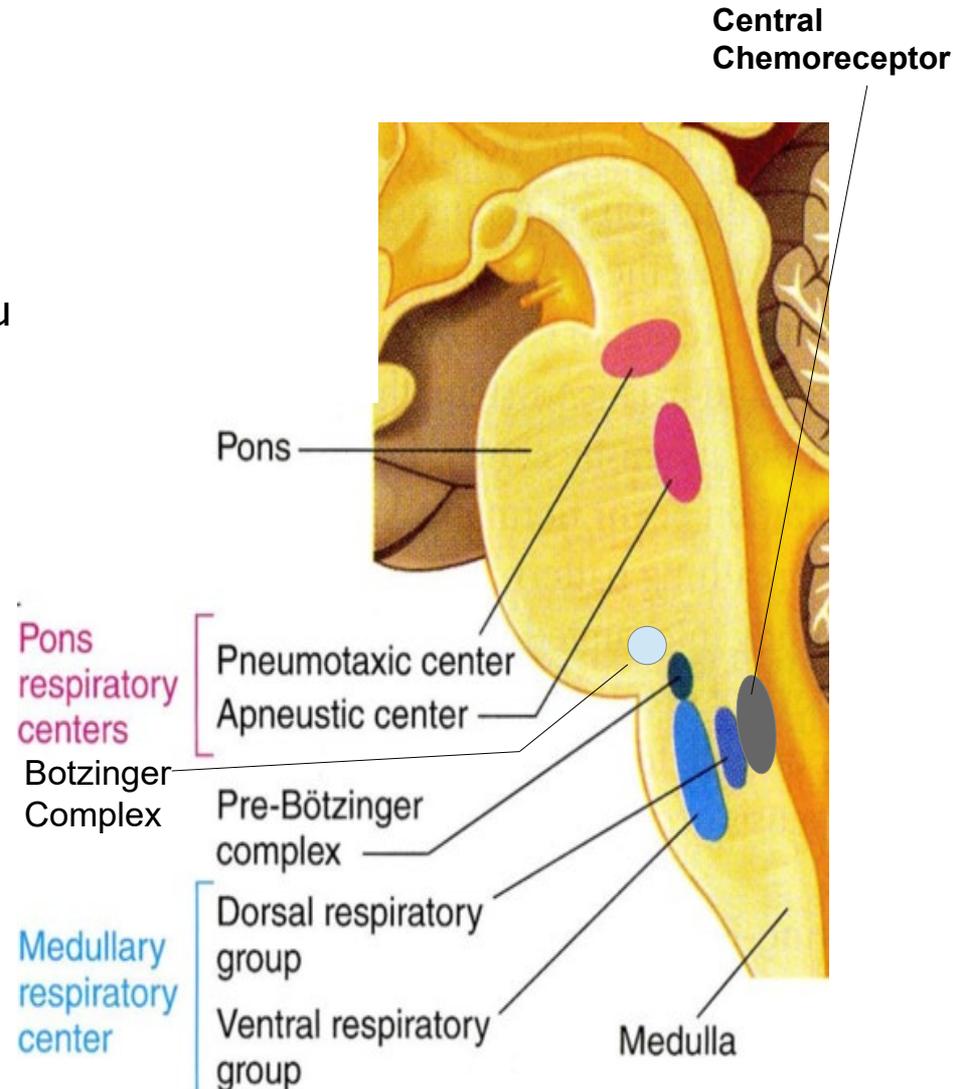
# Neural Control of Breathing

**Pneumotaxic center** fine tunes the transition between inspiration and expiration by inhibiting inspiration.

**Apneustic center** prolongs inspiration // if you destroy the pneumotaxic center then you get prolonged inspiration // called apneustic breathing.

**Central chemoreceptors** monitor CO<sub>2</sub> in cerebral spinal fluid // increase in CO<sub>2</sub> stimulates inspiration // decrease CO<sub>2</sub> inhibits inspiration

**Dorsal respiratory group** receives signals to regulate breathing from lung stretch & irritant receptors, proprioceptors, central and peripheral chemoreceptors // send signals to spinal cord integrating centers and to the ventral respiratory group // Influence both inspiration and forced expiration



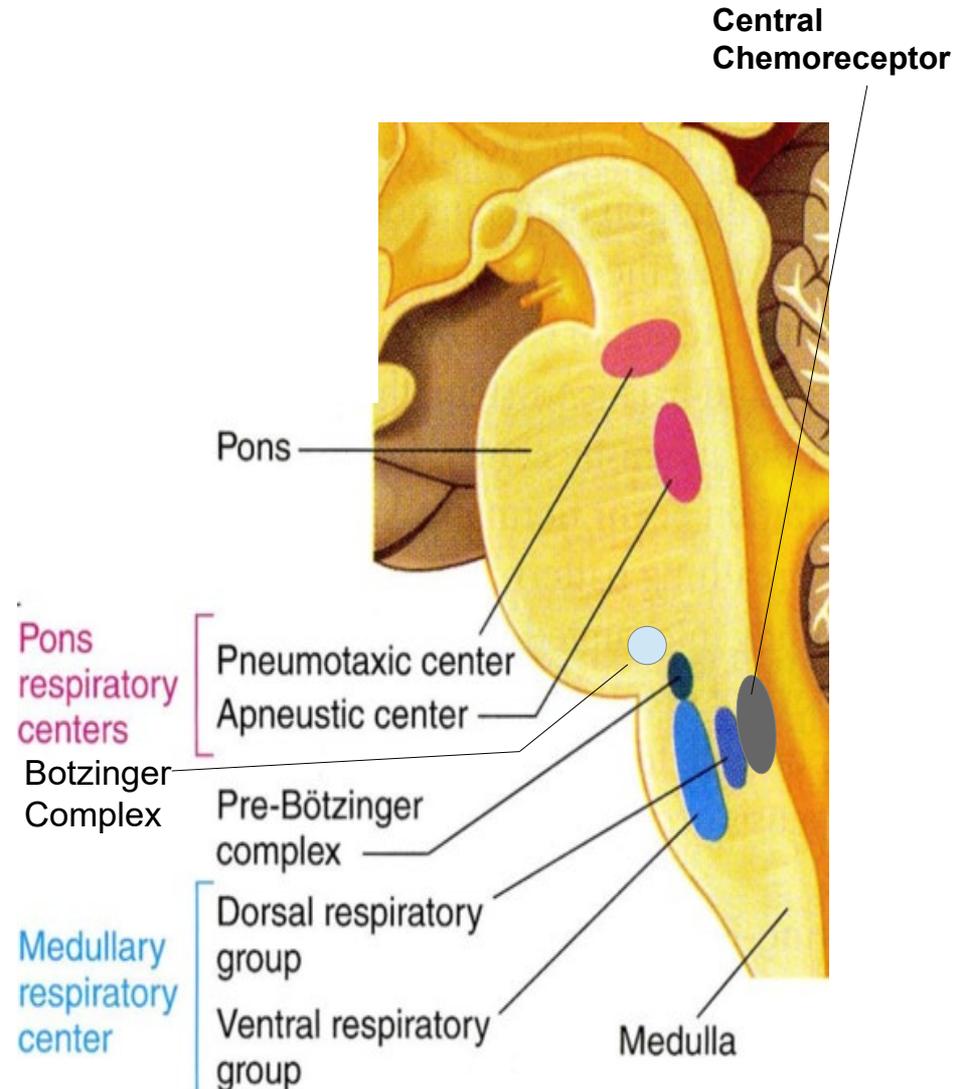
# Neural Control of Breathing

**Ventral respiratory center** receives signals from the dorsal respiratory group // send descending inspiratory and inhibitory action potentials to intercostal muscles and diaphragm

**Pre-Botzinger Complex** is a nuclei unable to maintain a resting membrane potential // leaks cations // this becomes the pacemaker for inspiration by sending action potential to the ventral respiratory group.

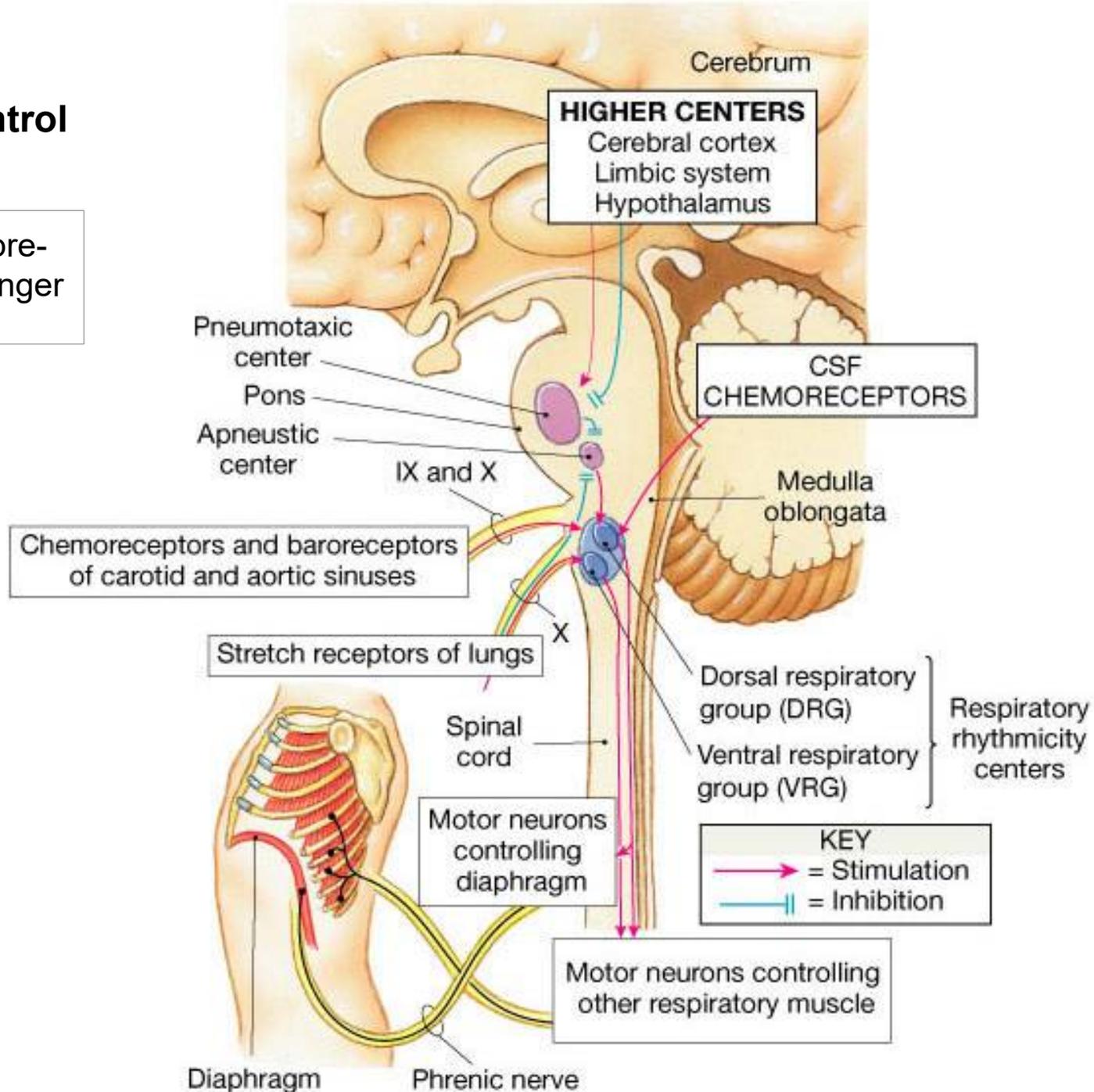
**Botzinger complex** // inhibits the pre-Botzinger complex

**Dorsal and ventral respiratory group** send Inspiratory neurons to spinal cord anterior Horn // C3 to C5 lower motor pathway to phrenic nerve (diaphragm) and T1 to T11 lower motor pathway to intercostal muscles



# Respiratory Control Centers

Not shown are the pre-Botzinger and Botzinger centers.



# Neural Control of Breathing

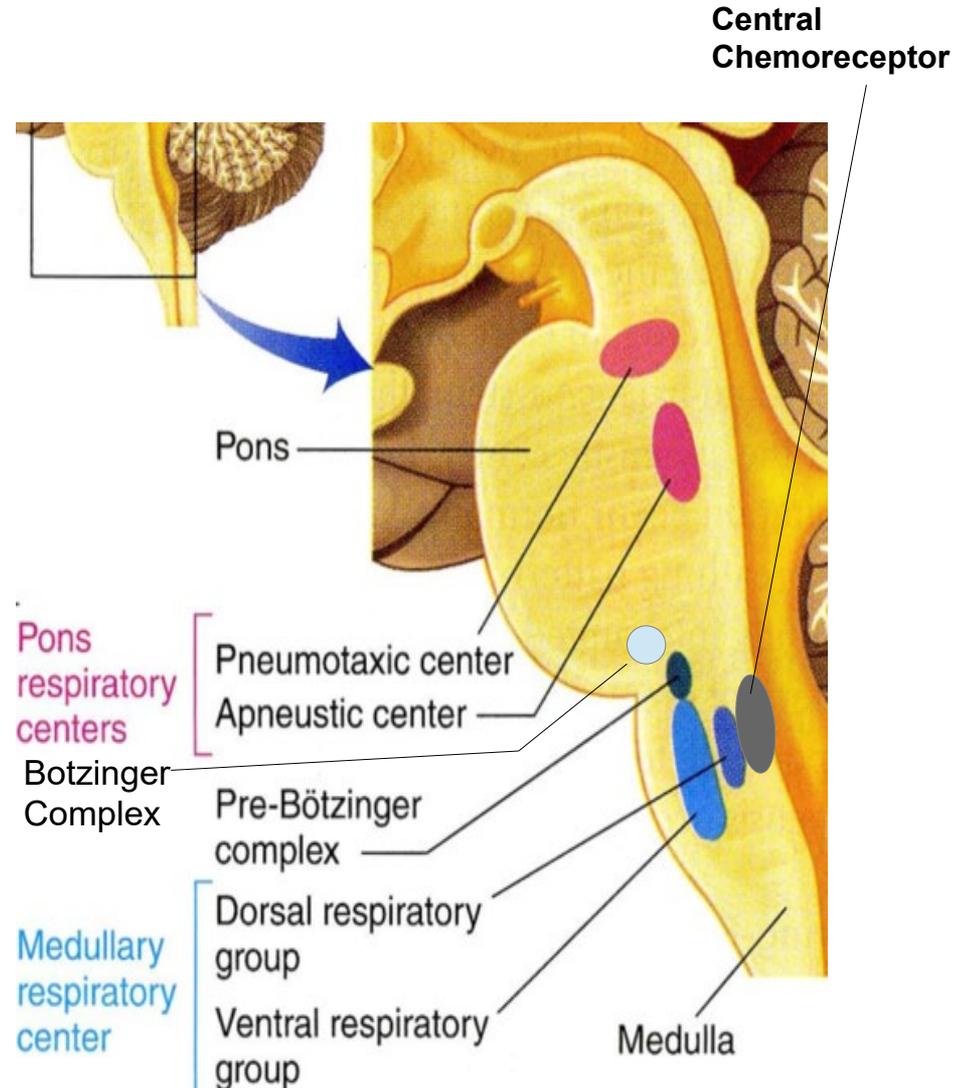
Just superior to the ventral respiratory center are the

pre-Botzinger complex //  
pacemaker // depolarize for two  
seconds

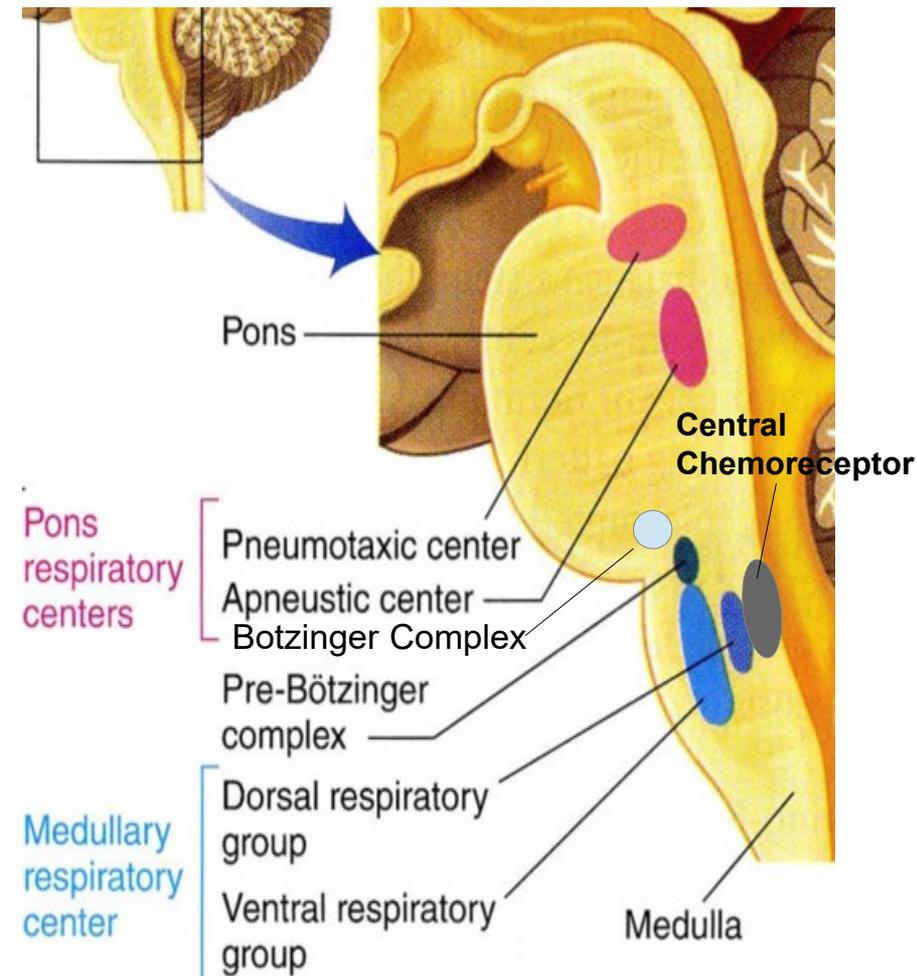
Botzinger complex // inhibits  
Inspiration for three seconds

**These two nuclei set the respiratory  
rate during the resting state** by  
sending action potentials to the  
ventral respiratory group

**Respiratory cycle is five seconds //  
12 cycles per min**



# Neural Control of Breathing



The **ventral respiratory group (VRG)** in the **medulla sets the respiratory rate** and rhythm for breathing. There are inspiratory nuclei and expiratory nuclei in the VRG.

**Inspiratory nuclei** (pre-Botzinger complex) leak sodium to reach threshold and depolarize to create an action potential. These nuclei are the **pacemaker** for breathing.

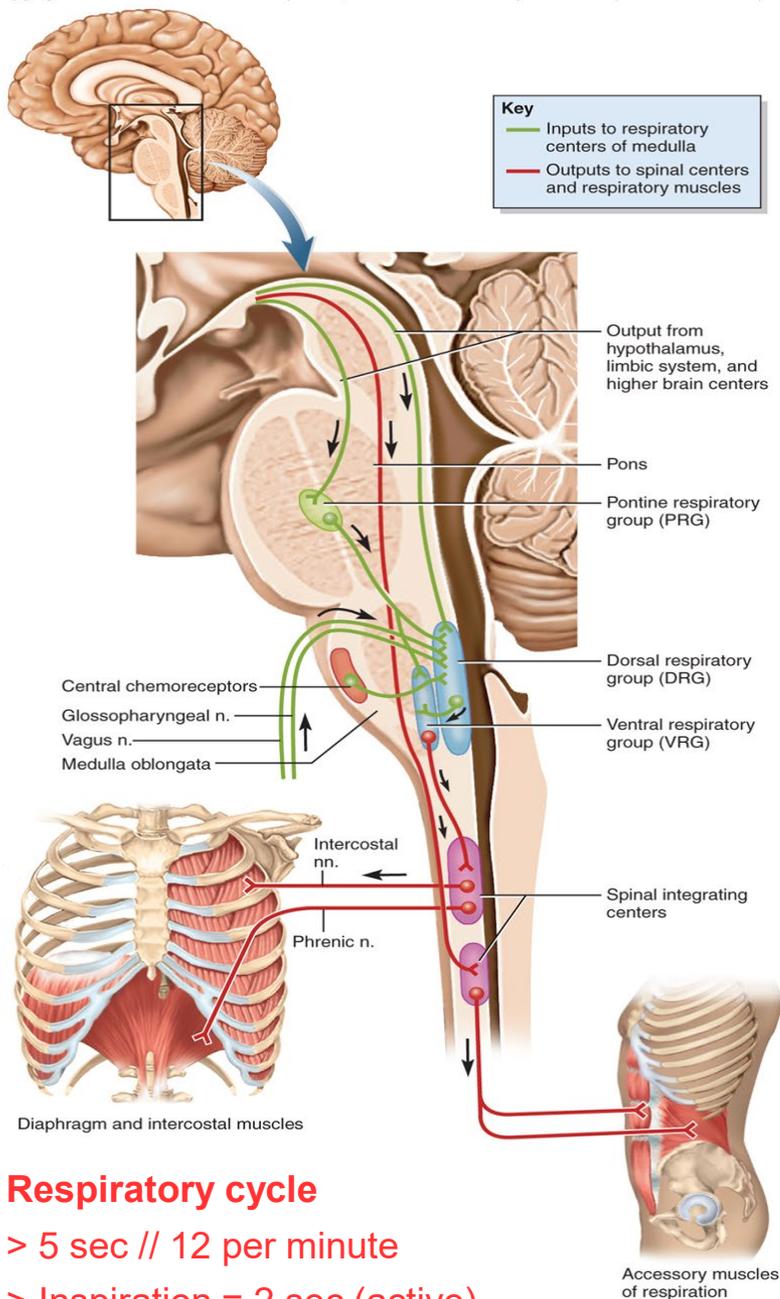
Expiratory nuclei in the VRG (Botzinger complex) inhibit the inspiratory nuclei to stop inspiration. The passive recoil of the lung's elastic tissue forces the air out of the lungs.

**Inspiration is active and expiration is passive.**

The pre-Botzinger complex influence the rate of depolarization of the VRG pacemaker to regulate inspiration

The Botzinger complex regulates expiration.

# Ventral Respiratory Group



## Respiratory cycle

- > 5 sec // 12 per minute
- > Inspiration = 2 sec (active)
- > Expiration = 3 sec (passive)

The VRG action potentials are sent to the **spinal cord integration center**. Action potentials travel along the phrenic nerve to the diaphragm.

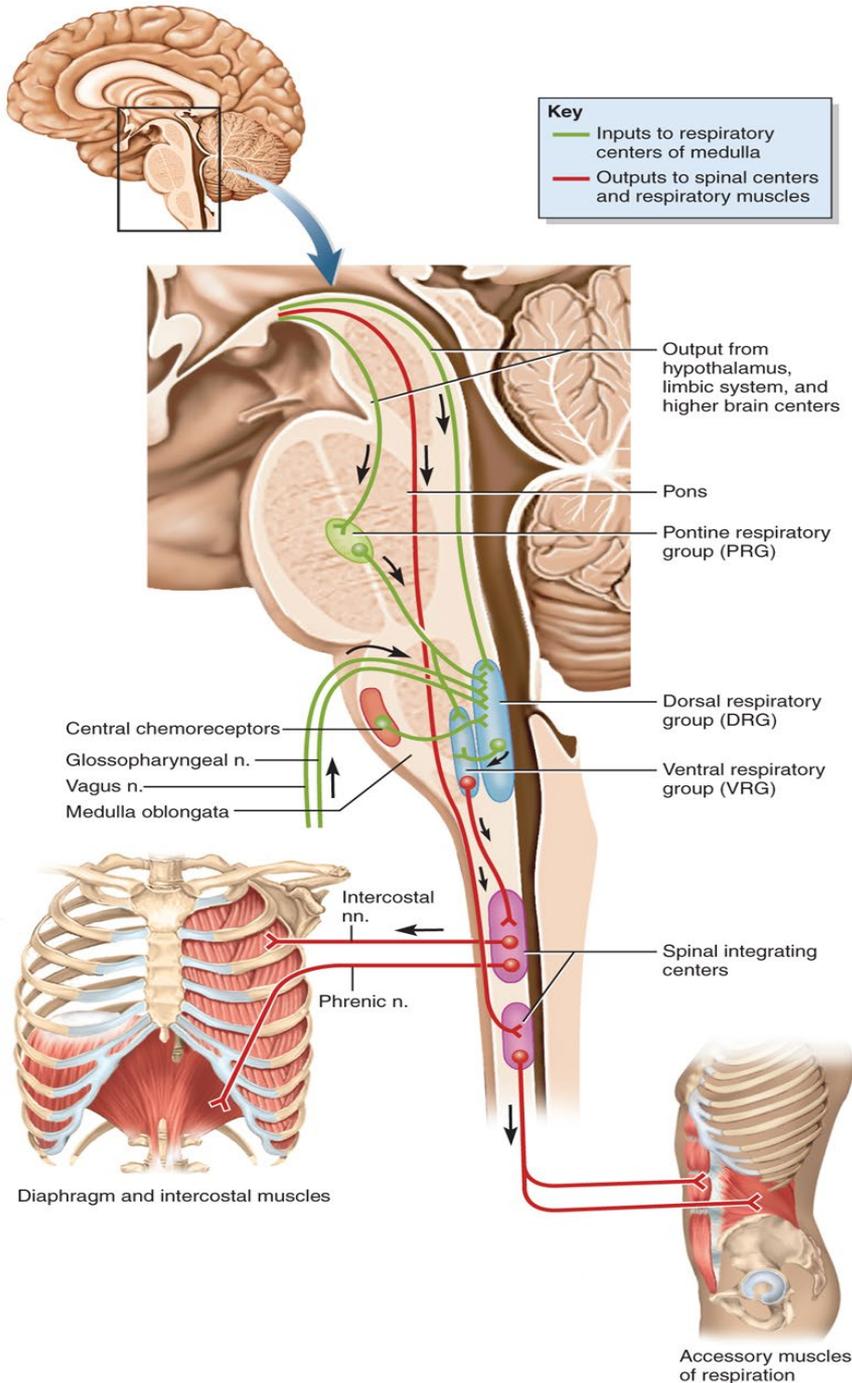
In a resting state, the phrenic nerve will cause the diaphragm to contract.

VRG inspiratory nuclei send action potentials for **2 seconds** to the diaphragm which results in inspiration.

VRG expiratory nuclei inhibit the inspiratory nuclei for **3 seconds** to cause the diaphragm and external intercostal muscles to relax

Passive elastic recoil of lung tissue moves air out of the lungs

In forced respiration the, the DRG send action potentials through the intercostal nerves will contract the external intercostal muscles to increase respiratory volume.



## Dorsal Respiratory Groups in the Medulla

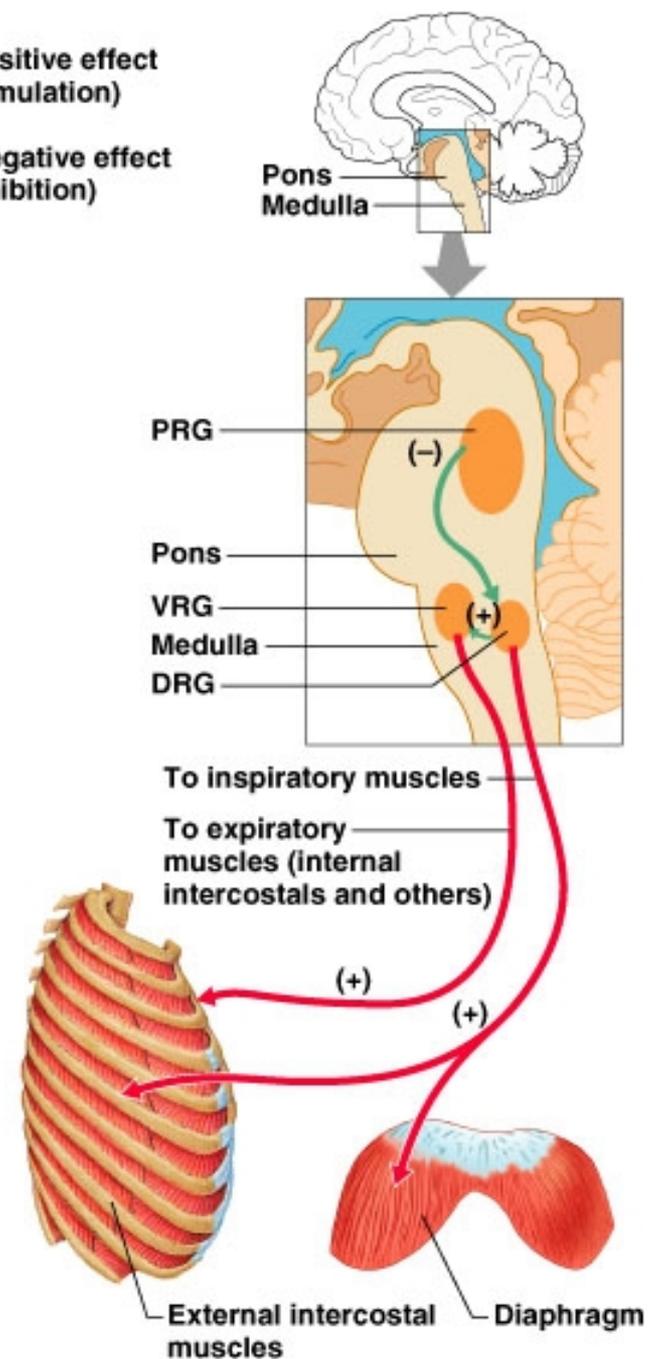
DRG receives signals from the pons respiratory center, central and peripheral chemo-receptors, stretch receptors, irritant receptors, cerebral cortex, and Limbic system.

DRG signals are used to modify the VRG respiratory rate. These signals adjust breathing to be faster, slower, shallower, or deeper.

DRG may also send motor pathways directly to spinal cord integration center that then continue to the intercostal muscles and the diaphragm by lower motor neurons.

# Respiratory Control Centers

**Key:**  
(+) = Positive effect (stimulation)  
(-) = Negative effect (inhibition)



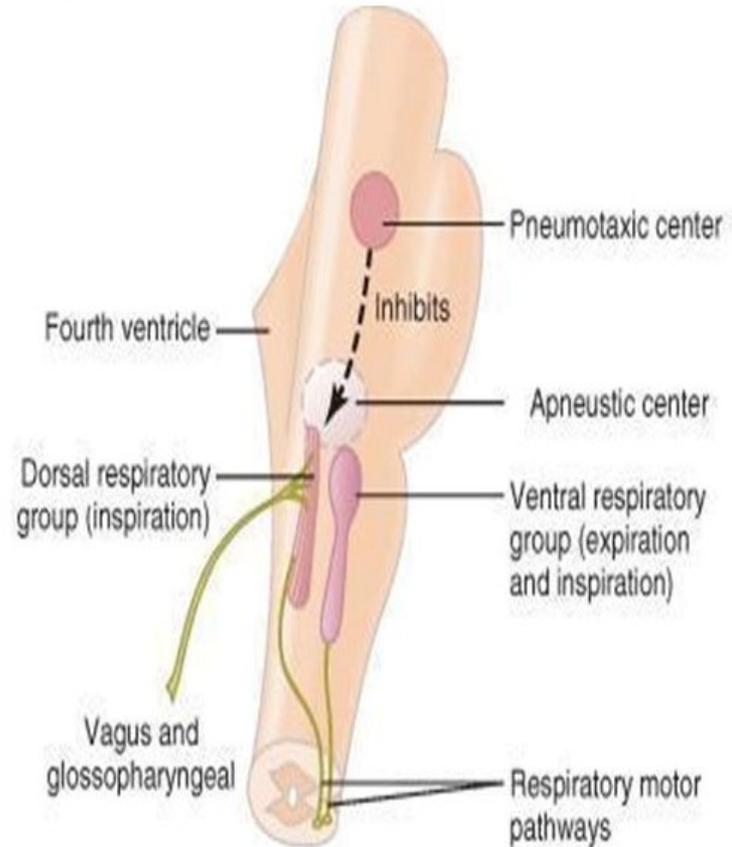
# Neural Control of Breathing

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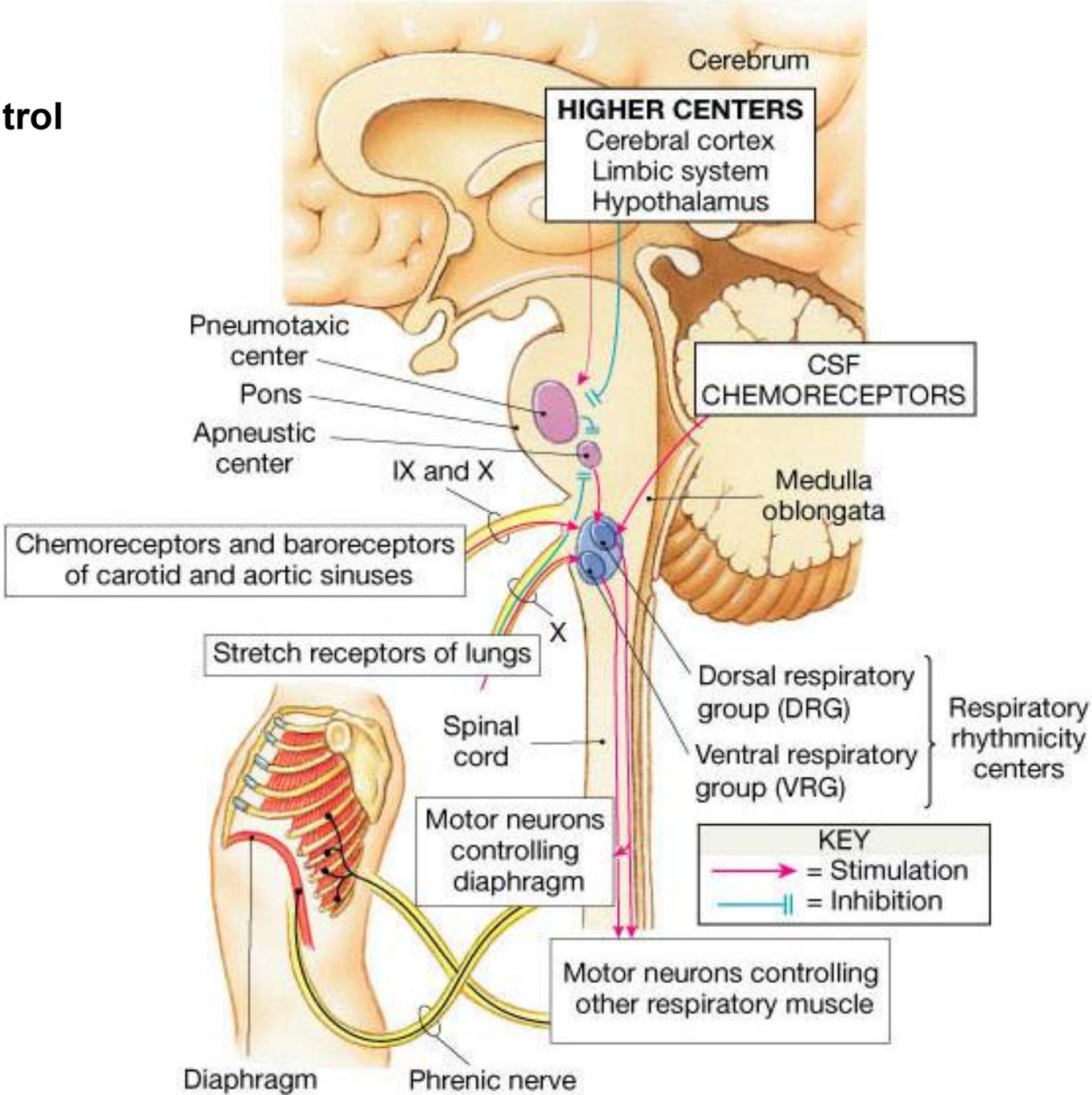
## Pneumotaxic Center

Strong stimulation may reduce inspiratory phase by 0.5 seconds and inspiratory rate increase to 30 to 40 per min

Weak stimulation may increase inspiratory phase by 5 seconds or more and respiratory rate drops to 3 to 5 per min



# Respiratory Control Centers



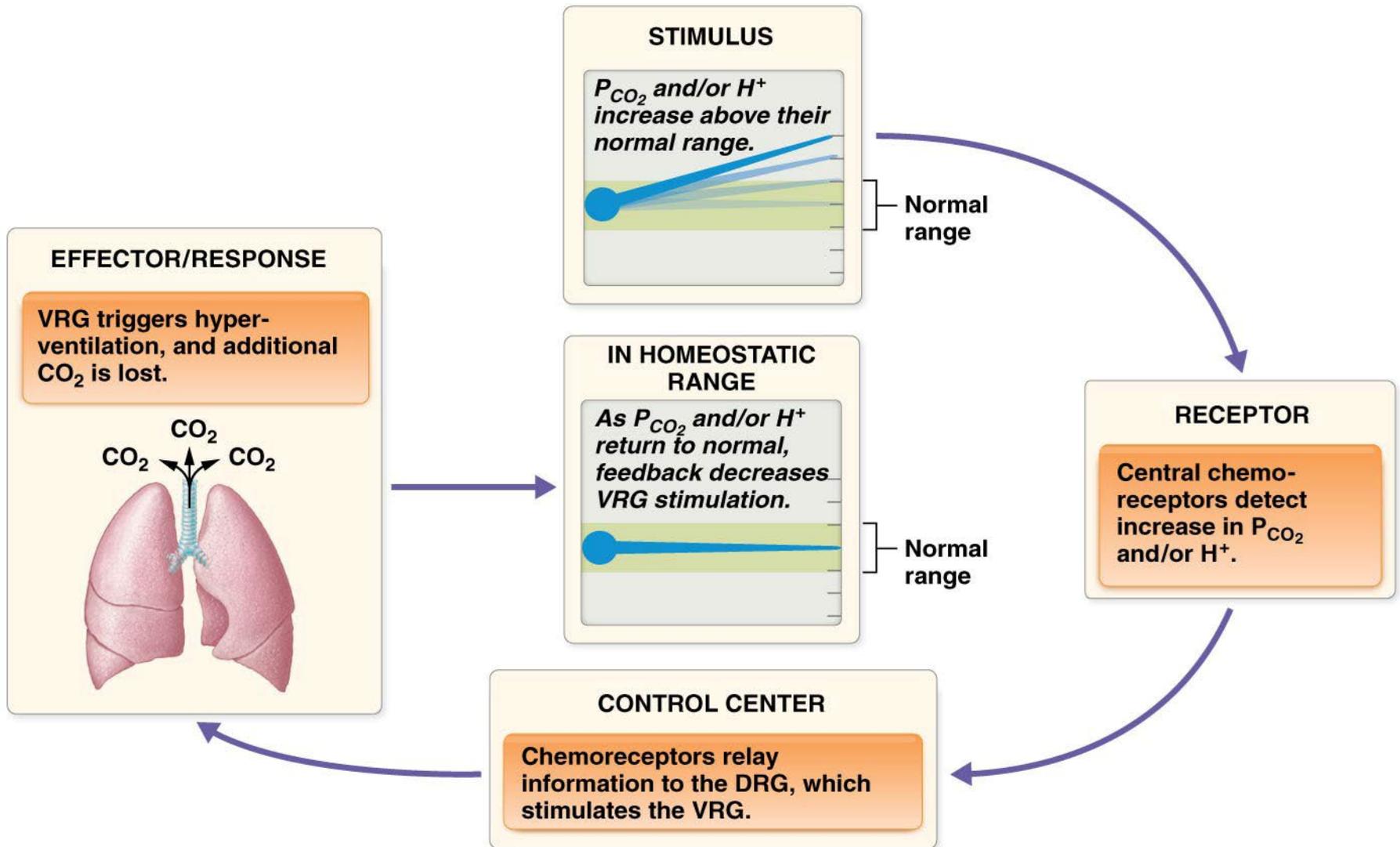
# Physiologic States May Influence Neural Control of Breathing

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Many factors may regulate breathing to match our physiological state

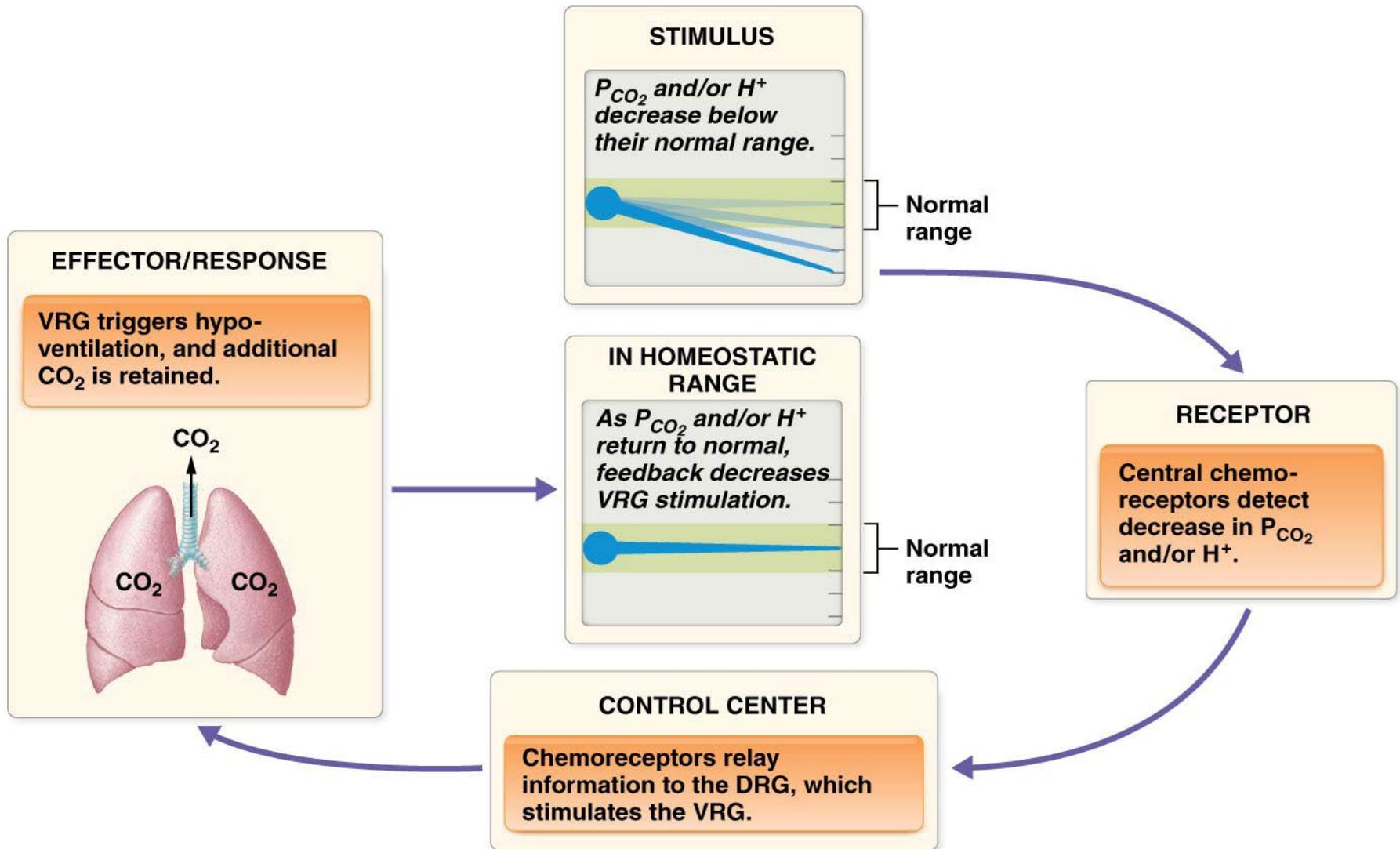
- central chemoreceptors
- peripheral chemoreceptors
- proprioceptors
- lung stretch receptors (Hering-Breuer reflex)
- irritant reflex receptors
- limbic system
- temperature
- pain
- stretching the anal sphincter
- blood pressure (minor)

# Role of the central chemoreceptors in regulation of blood pH via breathing rate.



(a) Response to increased arterial  $P_{CO_2}$  and/or  $H^+$  concentration by a negative feedback loop

# Role of the central chemoreceptors in regulation of blood pH via breathing rate.



(b) Response to decreased arterial  $P_{CO_2}$  and/or  $H^+$  concentration by a negative feedback loop

## Other Respiratory Regulators

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### Irritant receptors

nerve endings amid the epithelial cells of the airway

respond to smoke, dust, pollen, chemical fumes, cold air, and excess mucus

trigger protective reflexes //

bronchoconstriction, shallower breathing, breath-holding (apnea) followed by strong coughing

## Other Respiratory Regulators

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### Stretch receptors

found in the smooth muscles of bronchi and bronchioles, and in the visceral pleura

respond to inflation of the lungs

known as the inflation reflex or **Hering-Breuer Reflex**

triggered by excessive inflation

protective reflex

inhibits inspiratory neurons therefore able to stop inspiration

## Other Respiratory Regulators

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### Hyperventilation

anxiety triggered state in which breathing is so rapid that it expels  $\text{CO}_2$  from the body faster than it is produced

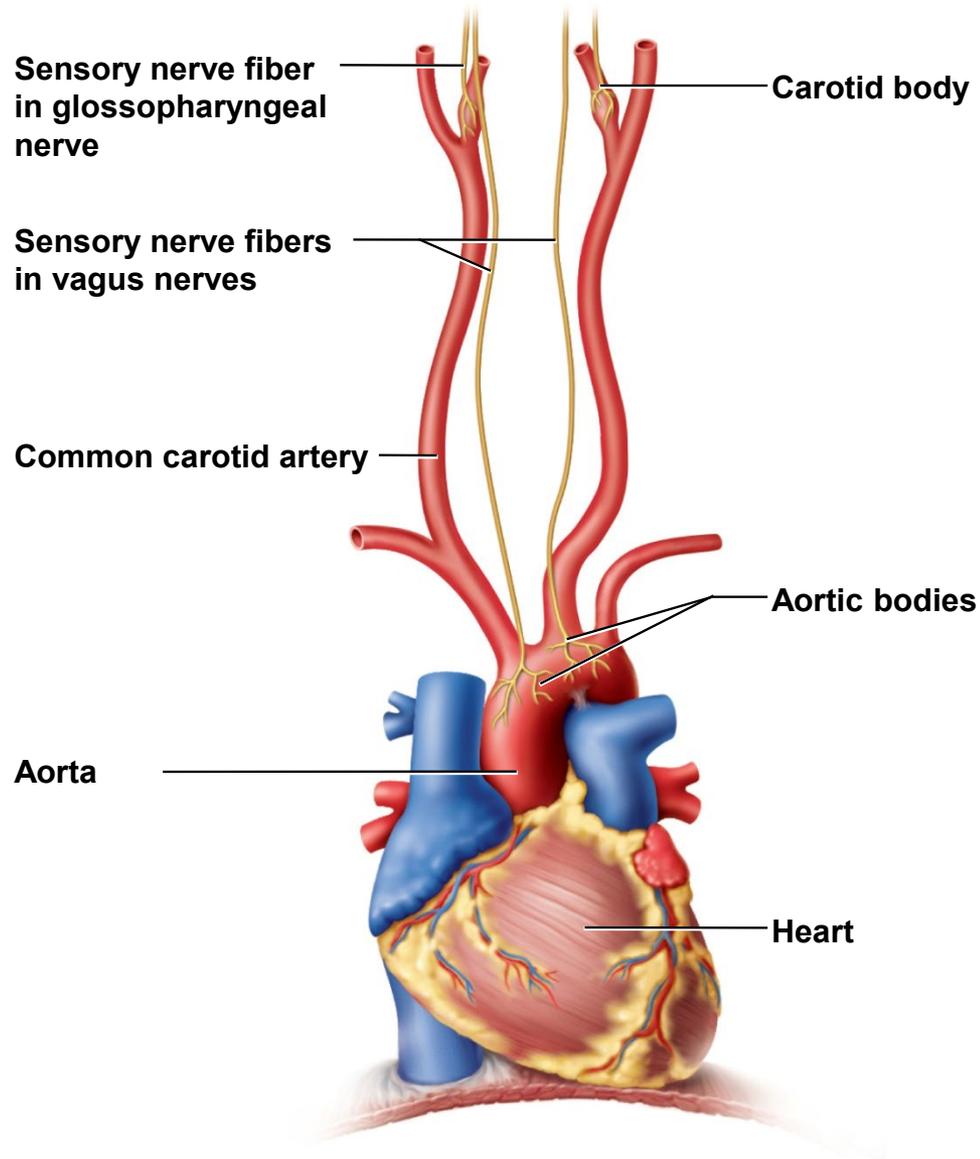
blood  $\text{CO}_2$  levels drop (i.e. fewer protons = higher pH number = more alkaline)

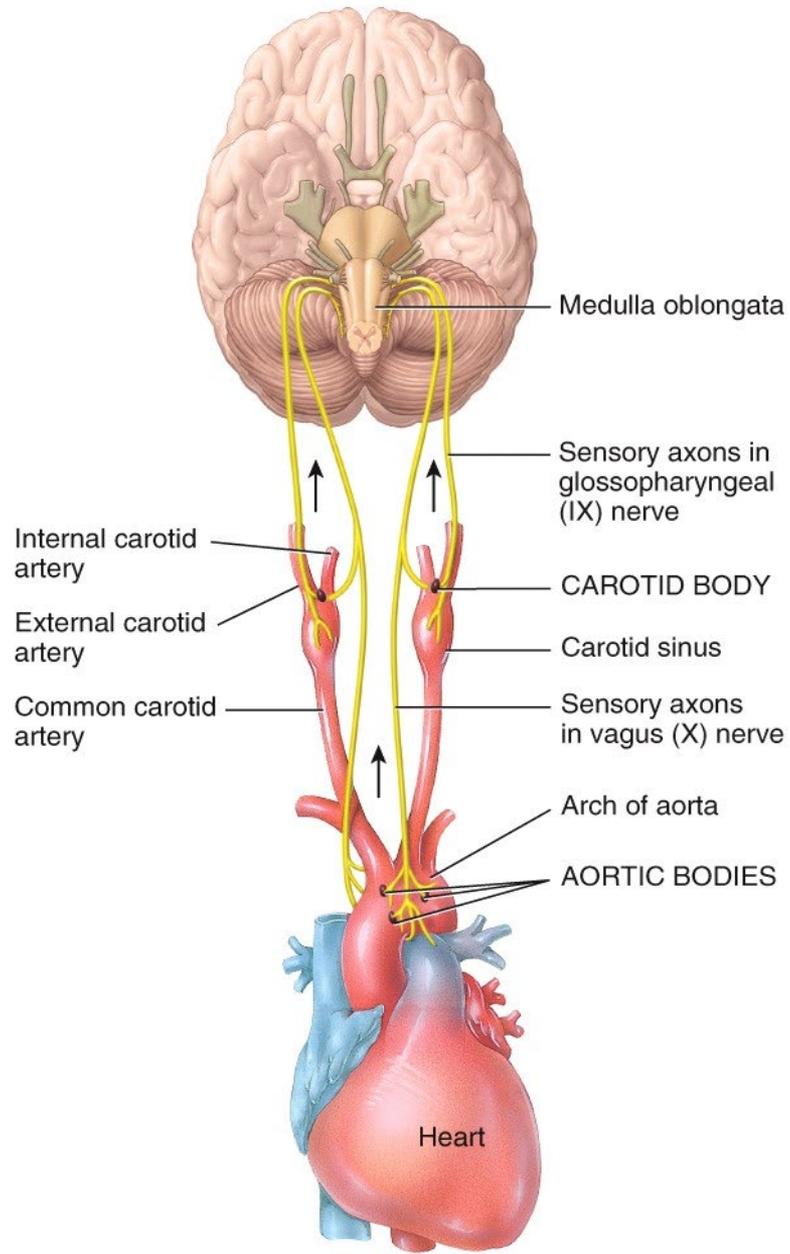
as pH number rises (i.e. more alkaline) this then **causes the cerebral arteries to constrict** /// reducing cerebral perfusion which may cause dizziness or fainting

can be brought under control by having the person re-breathe the expired  $\text{CO}_2$  from a paper bag

# Peripheral Chemoreceptors

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# Voluntary Control May Also Modify Breathing

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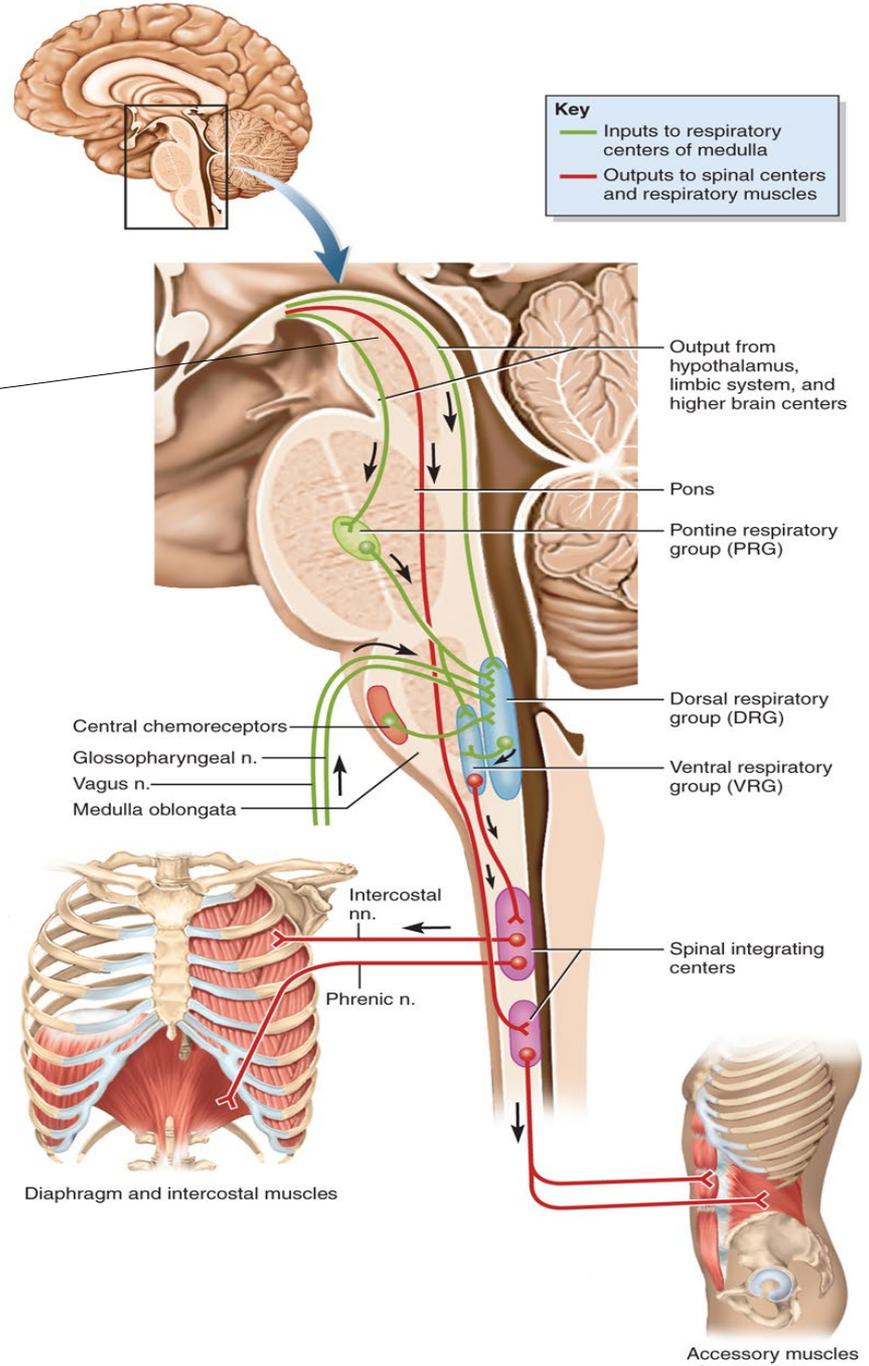
Originates in the motor cortex of frontal lobe of cerebrum

Descending signal sent via corticospinal tracts to respiratory neurons in spinal cord /// **Note: this track bypasses brain stem regulation**

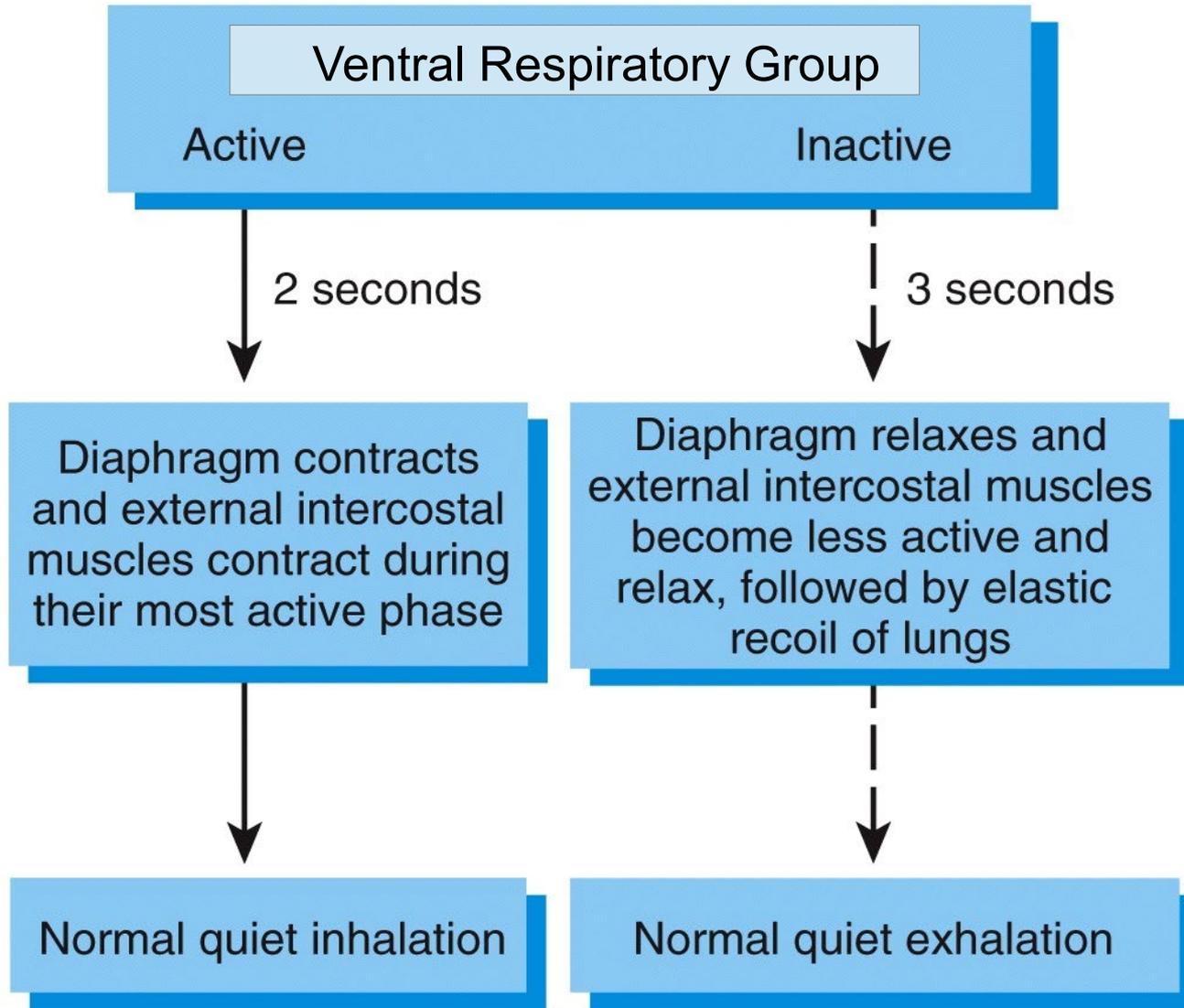
There is a limits to voluntary control /// called the **breaking point** // when CO<sub>2</sub> levels will rise to a point then the automatic controls override your will power not to breath

# Voluntary Control May Also Modify Breathing

Note: this track bypasses brain stem regulation

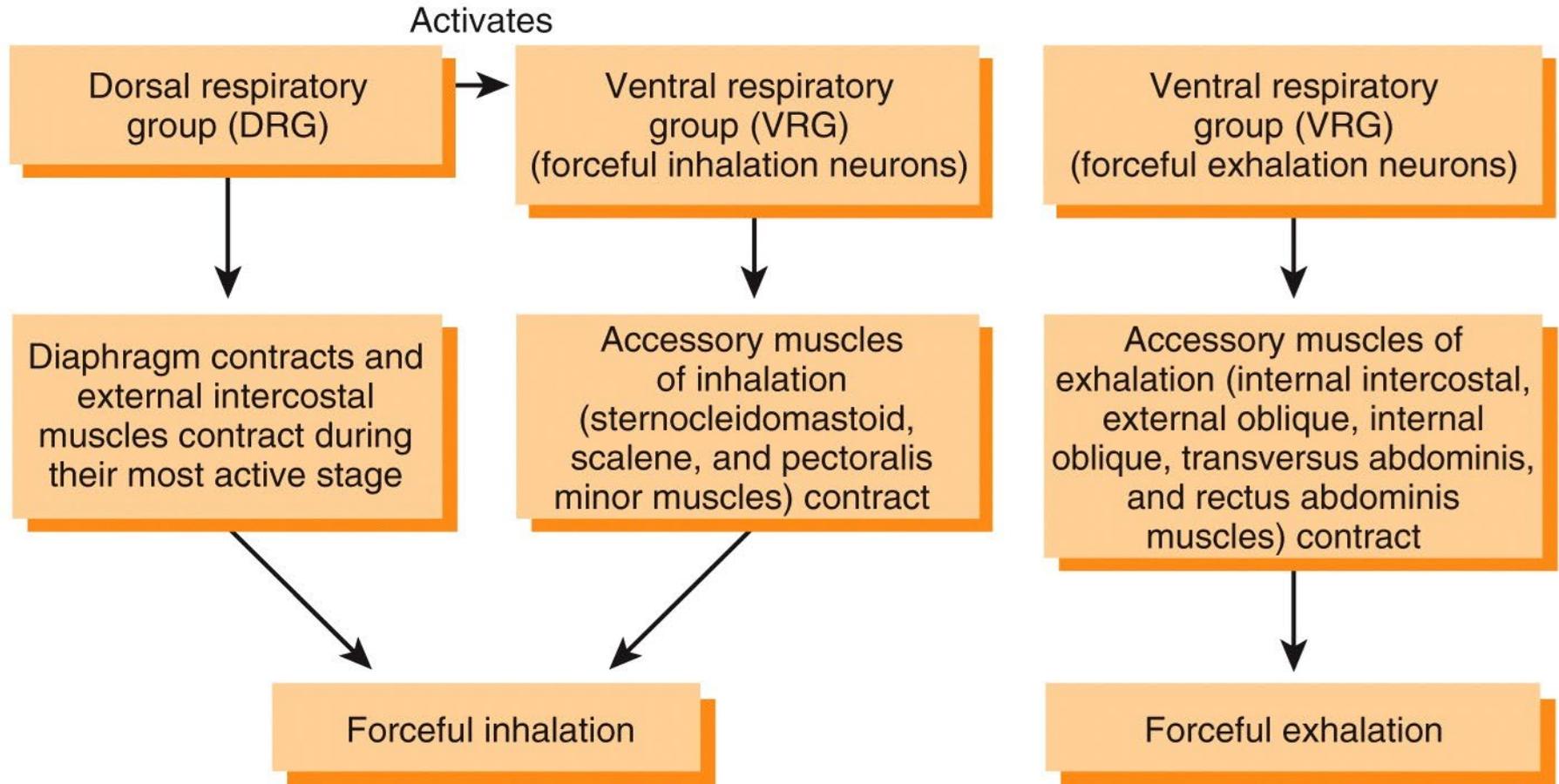


# Quiet Breathing





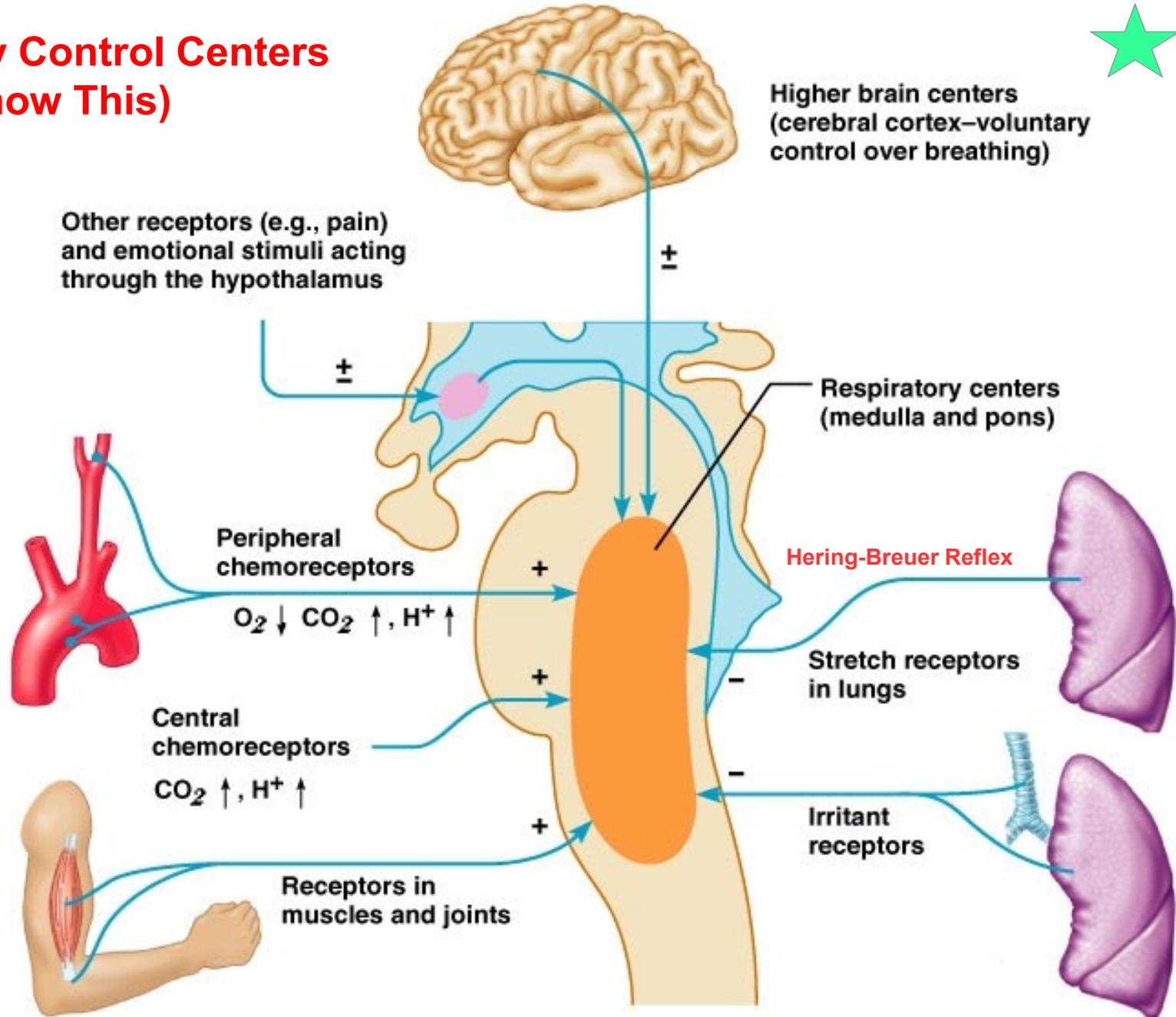
# Forceful Breathing or Breathing During Exercise



# Control mechanisms of ventilation.

STIMULI	CONTROL MECHANISM	EFFECT ON RESPIRATORY CENTERS	EFFECT ON VENTILATION
<b>Cerebral cortex inputs (e.g., emotion)</b>	<b>Voluntary control</b> 	+/-	<b>Varied</b>
<b>Changes in arterial <math>P_{CO_2}</math>, <math>H^+</math> concentrations</b>	<b>Central chemoreceptors</b> 	+/-	<b>Hyperventilation when <math>P_{CO_2}</math> and/or <math>H^+</math> concentrations increase; hypoventilation when <math>P_{CO_2}</math> and/or <math>H^+</math> decrease</b>
<b>Changes in arterial <math>P_{O_2}</math></b>	<b>Peripheral chemoreceptors</b> 	+	<b>Hyperventilation when arterial <math>P_{O_2}</math> decreases</b>

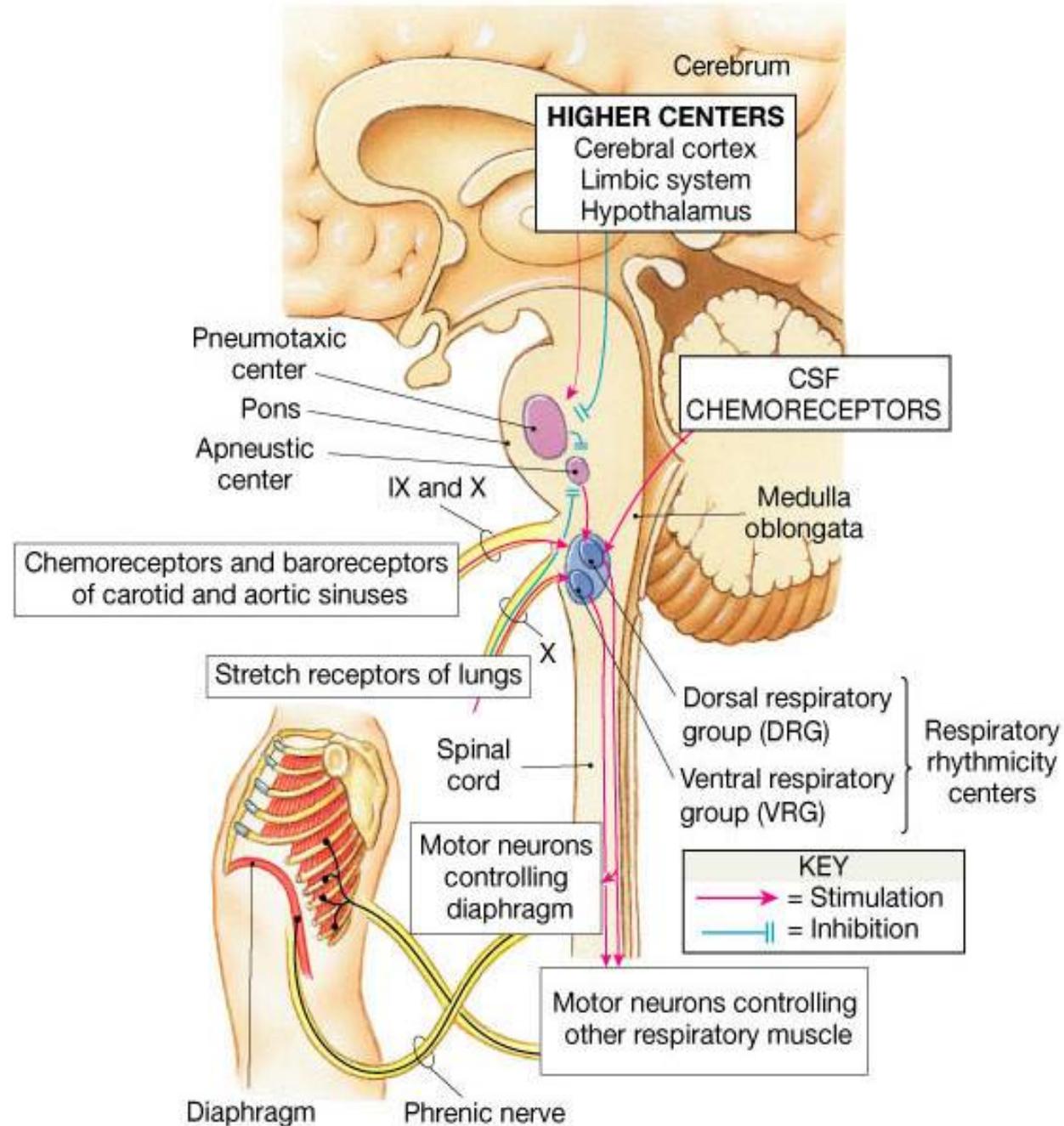
# Respiratory Control Centers (Know This)

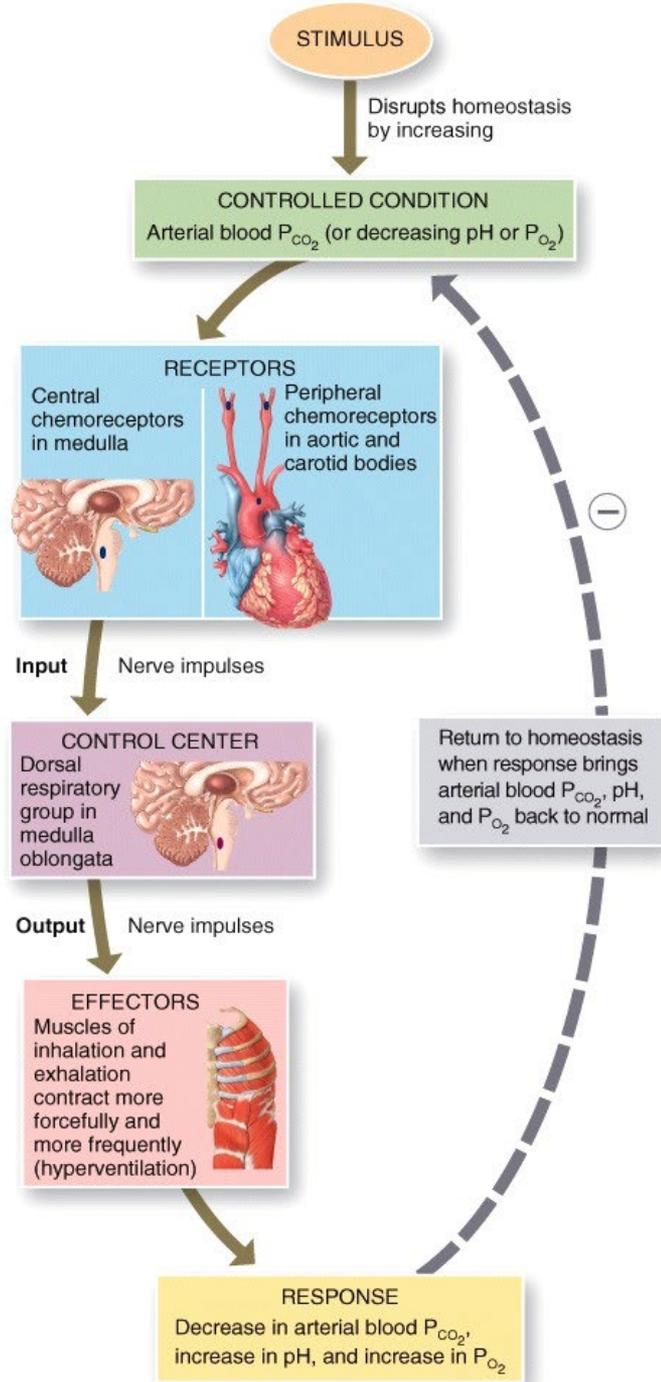


# Respiratory Control Centers

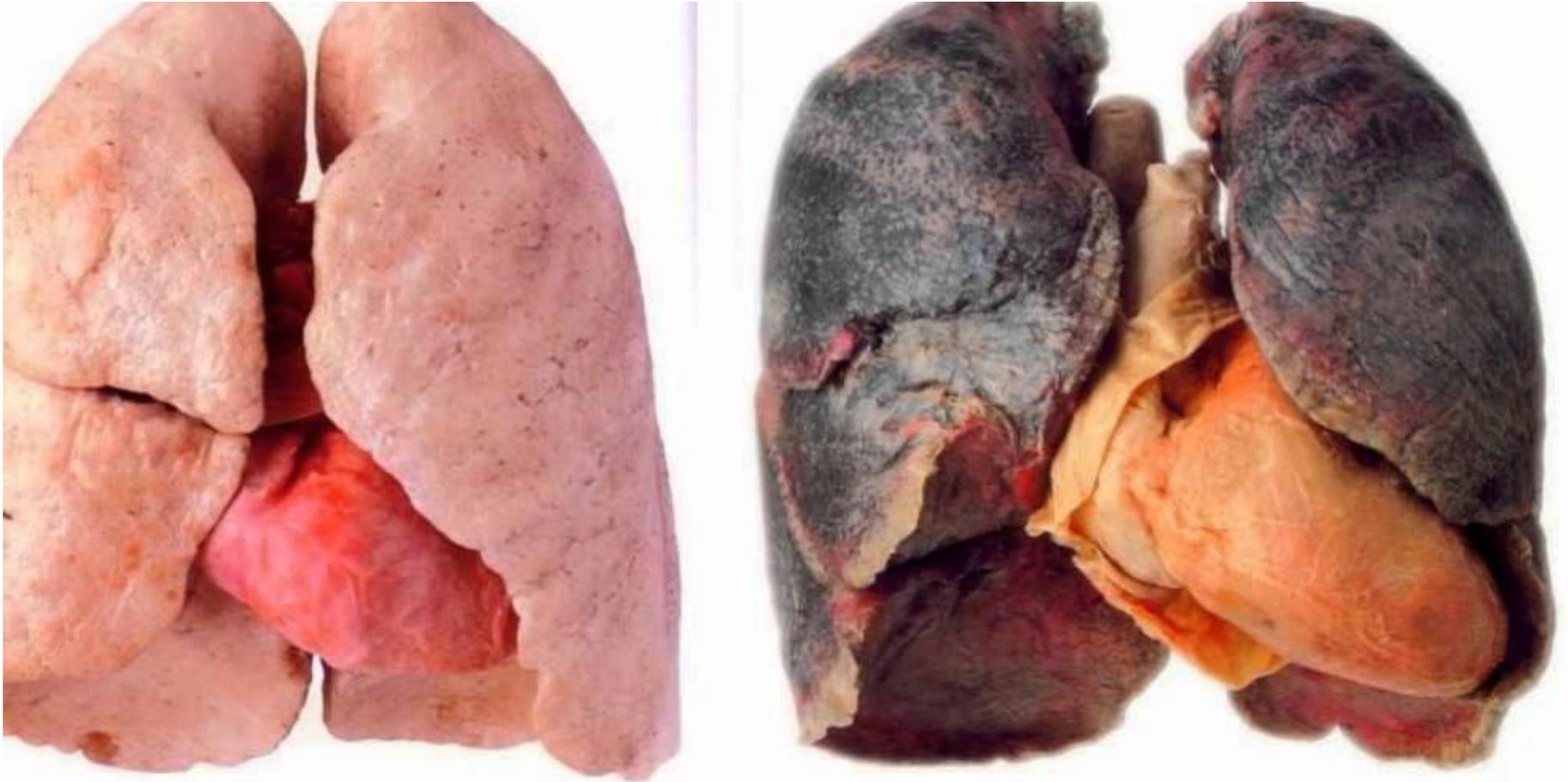
When you speak or sing the cerebral cortex must adjust breathing.

Your breathing rate will also change as you get ready to board a spaceship.





## Non-Smoker's Lungs VS Smoker's Lungs



# What Happens When A Smoker Quits

## 20 minutes after quitting

The heart rate and blood pressure drop back to normal levels.

## 1-3 months after quitting

Circulation improves and lung function increases

## 1 year after quitting

The risk of getting coronary heart disease is half as high as a smoker's. The risk of heart attack drops dramatically.

## 10 years after quitting

The risk of dying from lung cancer is about half that of a person who is still smoking. The risk of cancer of the larynx (voice box) and pancreas decreases.

## 12 hours after quitting

The level of carbon monoxide in the blood drops to normal.

## 1-9 months after quitting

Coughing and shortness of breath decrease. Tiny hair-like structures that move mucus out of the lungs (called cilia) start to regain normal function.

## 5 years after quitting

The risk of cancers of the mouth, throat, esophagus, and bladder are cut in half. Cervical cancer risk falls to that of a non-smoker.

## 15 years after quitting

The risk of coronary heart disease is that of a non-smoker's.



# Central and Peripheral Input to Respiratory Centers

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## Central chemoreceptors (responsible for 75% of CO<sub>2</sub> drive)

brainstem neurons respond to changes in pH of cerebrospinal fluid

pH of cerebrospinal fluid reflects the CO<sub>2</sub> level in the blood

by regulating respiration to maintain stable pH /// respiratory center also ensures stable CO<sub>2</sub> level in the blood

## Peripheral chemoreceptors (responsible for 25% of CO<sub>2</sub> drive)

located in the carotid and aortic bodies of the large arteries above the heart

peripheral chemoreceptors are sensitive to **both PCO<sub>2</sub> and low arterial PO<sub>2</sub>**

*under normal conditions central chemoreceptors primary regulator of respiration*

*Following chronic high CO<sub>2</sub> – PCR senses low PO<sub>2</sub> and this becomes primary stimulus for respiration*

# Hypoxic Drive

Under normal conditions,  $PCO_2$  is the stimulus used to start inspiration.

If high  $PCO_2$  becomes a chronic condition, then chemoreceptors in medulla become insensitive to  $PCO_2$

Then peripheral chemoreceptor (which monitor arterial  $PO_2$ ) stimulate inspiration if arterial blood has low  $PO_2$  levels

This theory also suggest that high  $PO_2$  will then inhibit inspiration.

*Therefore, apparent danger if you give oxygen to COPD patient if they are under hypoxic drive /// high  $PO_2$  will stop inspiration !!!!!*

# Hypoxic Drive

Chronic Elevation of CO<sub>2</sub> Levels

Medullary Chemoreceptors  
Become Insensitive to High PCO<sub>2</sub>

PCO<sub>2</sub> Increases  
PO<sub>2</sub> Decreases

Respiration Slows

PCO<sub>2</sub> Decreases  
PO<sub>2</sub> Increases

Remove CO<sub>2</sub> / Take in O<sub>2</sub>

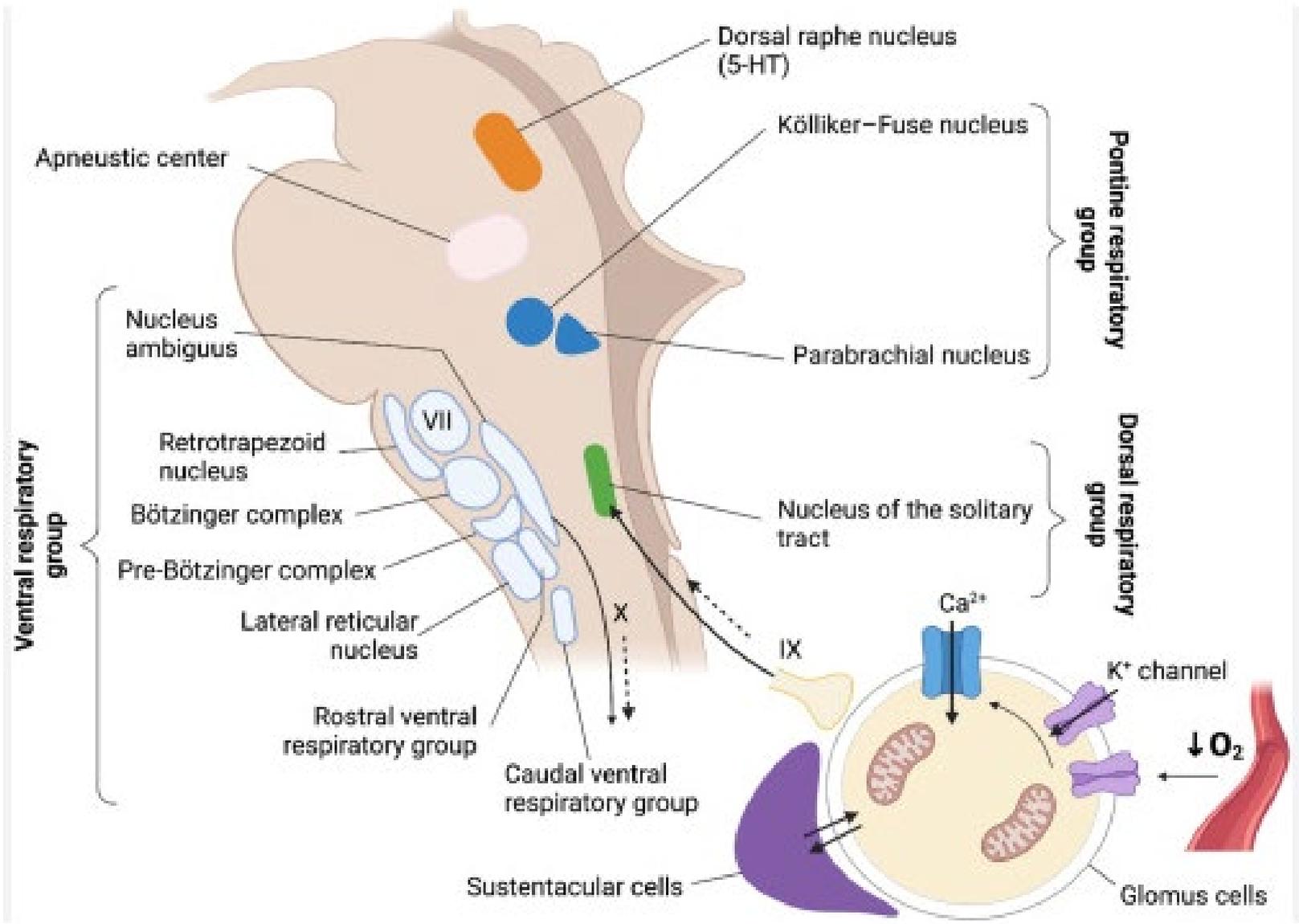
Increased Respiration

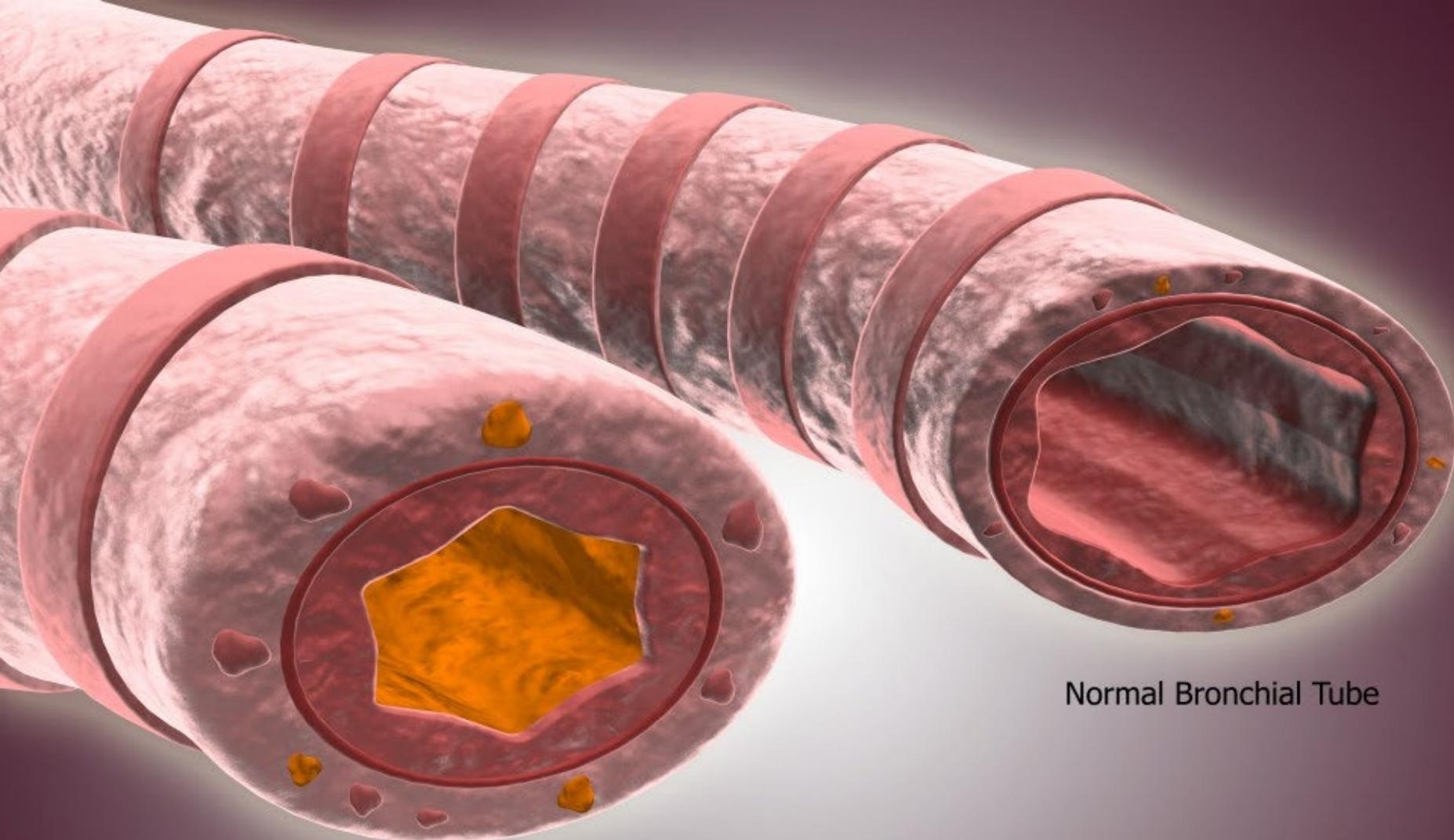
Inspiratory  
Muscles Stimulated

No Increase  
In Respiration

Marked Decrease  
In O<sub>2</sub> Levels

Very Low PO<sub>2</sub> Stimulates  
Peripheral Chemoreceptors





Normal Bronchial Tube

Inflamed Bronchial Tube  
of an Asthmatic