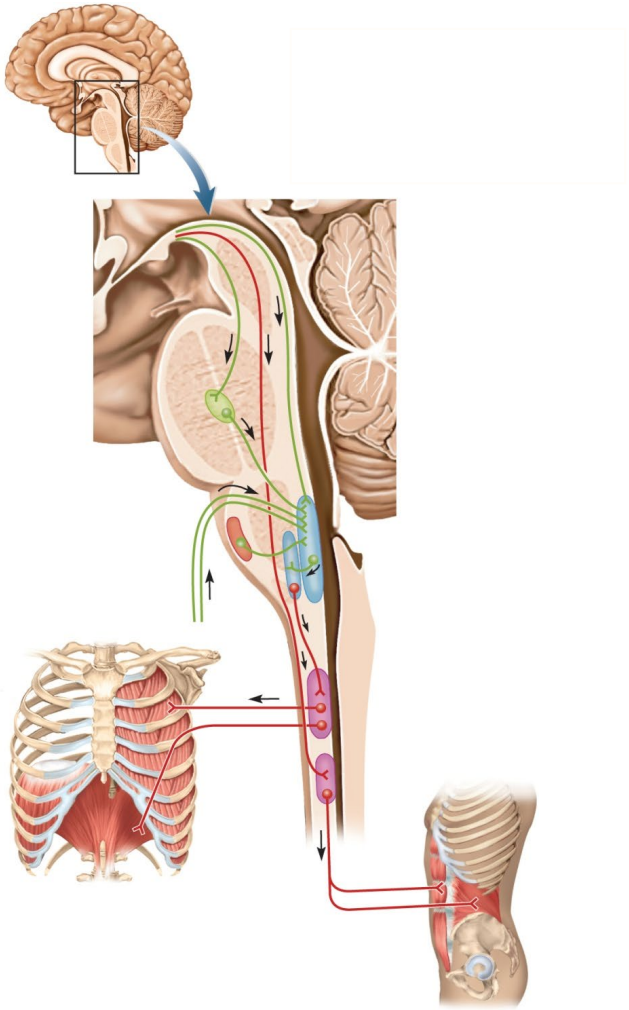
Chapter 22.2

Neural Control of Breathing



Neural Control of Breathing

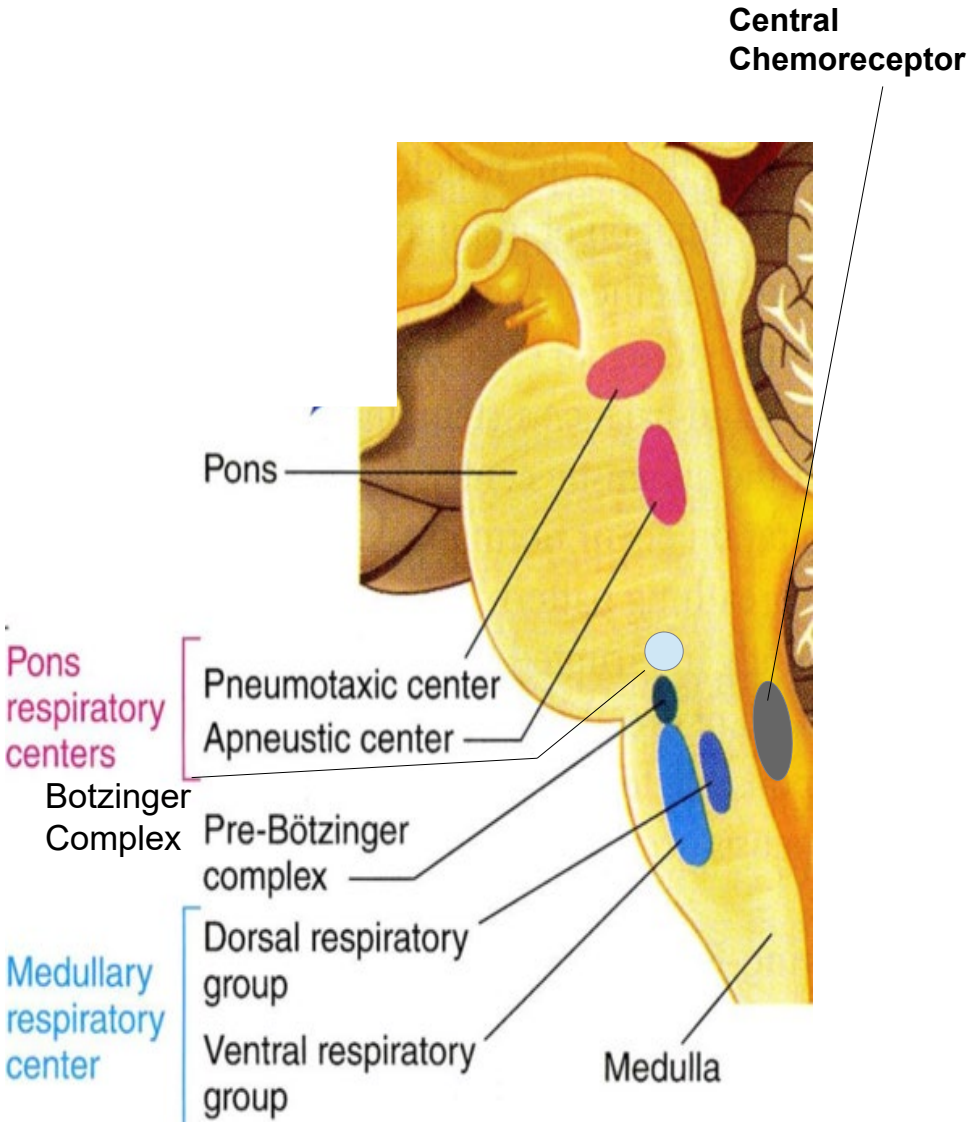
Breathing is the movement of air in and out of the lungs

Nuclei in the brain stem help to regulate rhythmic breathing

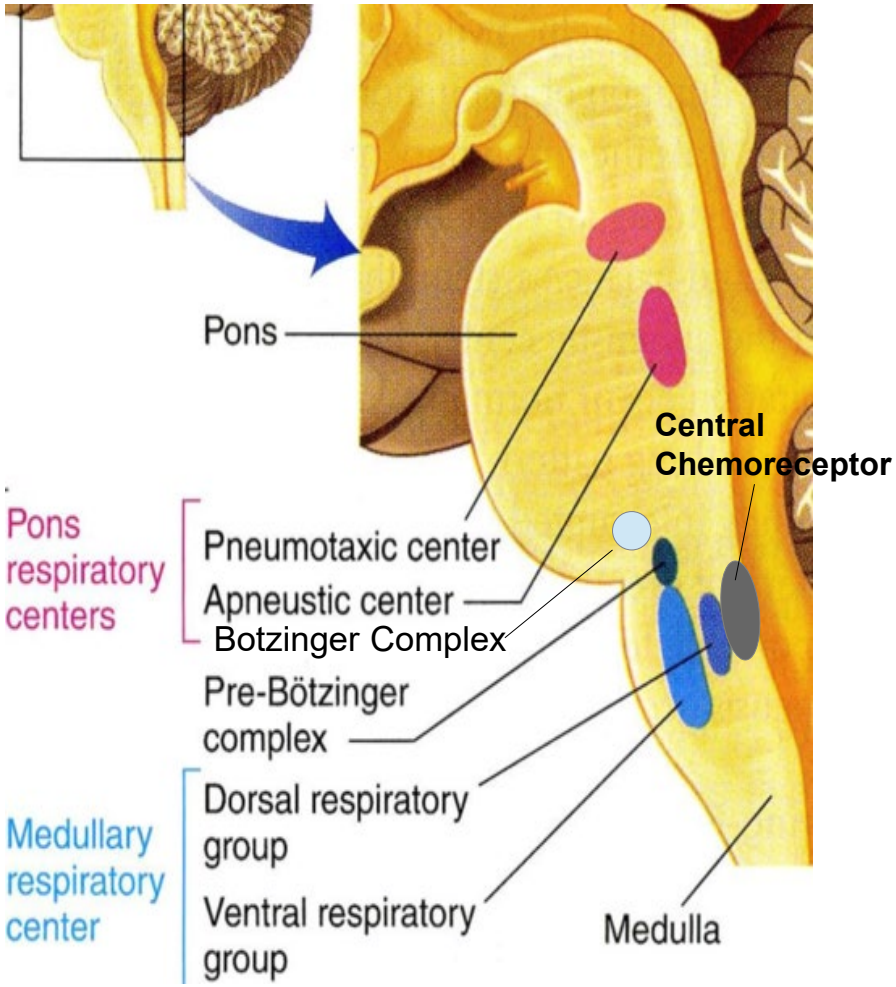
Two primary control centers:
> Pons Respiratory Center
> Medullary Respiratory Center

Breathing also influenced by higher brain centers, central and peripheral chemo-receptors, stretch receptors, irritant receptors, proprioceptors,and voluntary motor pathways.

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



Ventral Respiratory Group Sets the Respiratory Rate

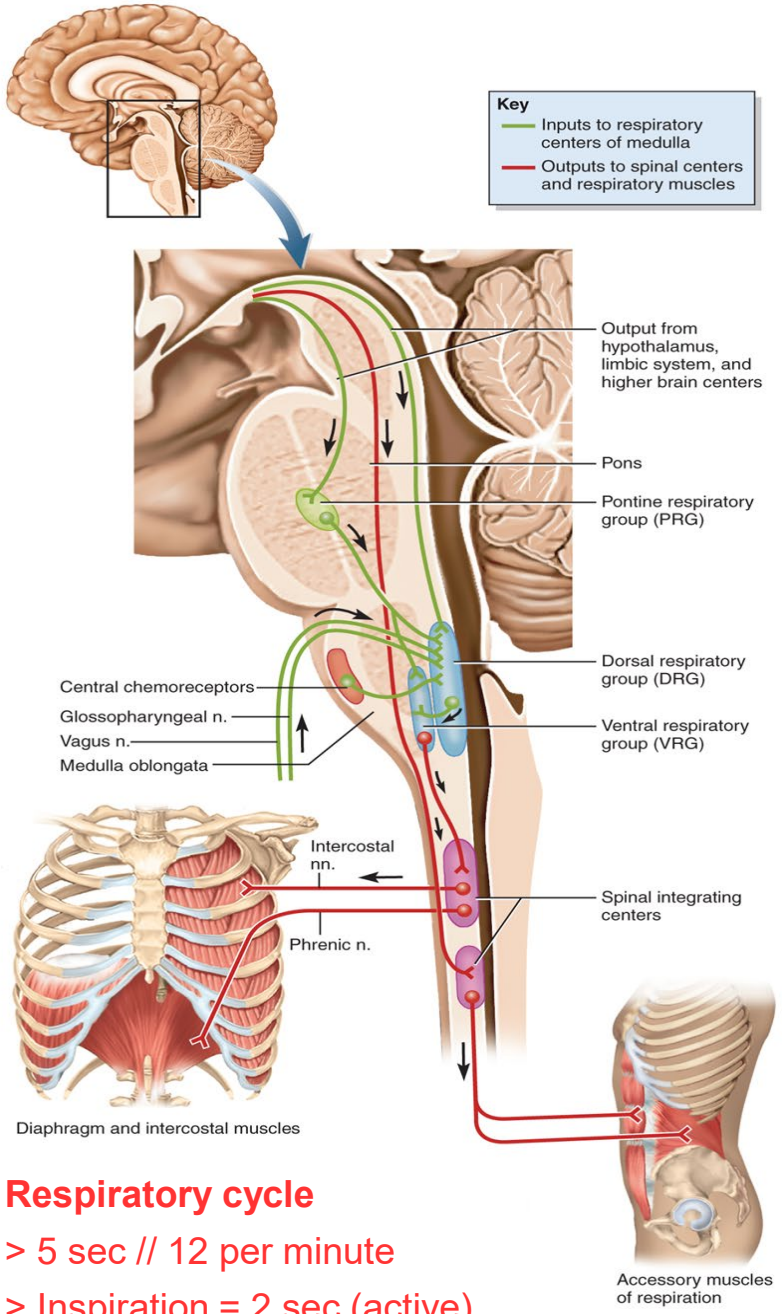


The ventral respiratory group (VRG) in the medulla sets the respiratory rate and rhythm for breathing. There are inspiratory nuclei and expiratory nuclei in the VRG.

Inspiratory nuclei (pre-Botzinger complex) leak sodium to reach threshold, depolarize, and send signal to ventral respiratory group to initiate inspiration. The pre-Botzinger 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. The Botzinger complex regulates expiration.

Spinal Cord Integration Center



Respiratory cycle

> 5 sec // 12 per minute

> Inspiration = 2 sec (active)

> Expiration = 3 sec (passive)

The VRG send action potentials to the spinal cord integration center. Action potentials travel along the phrenic nerve to the diaphragm and intercostal nerves to the external intercostal muscles.

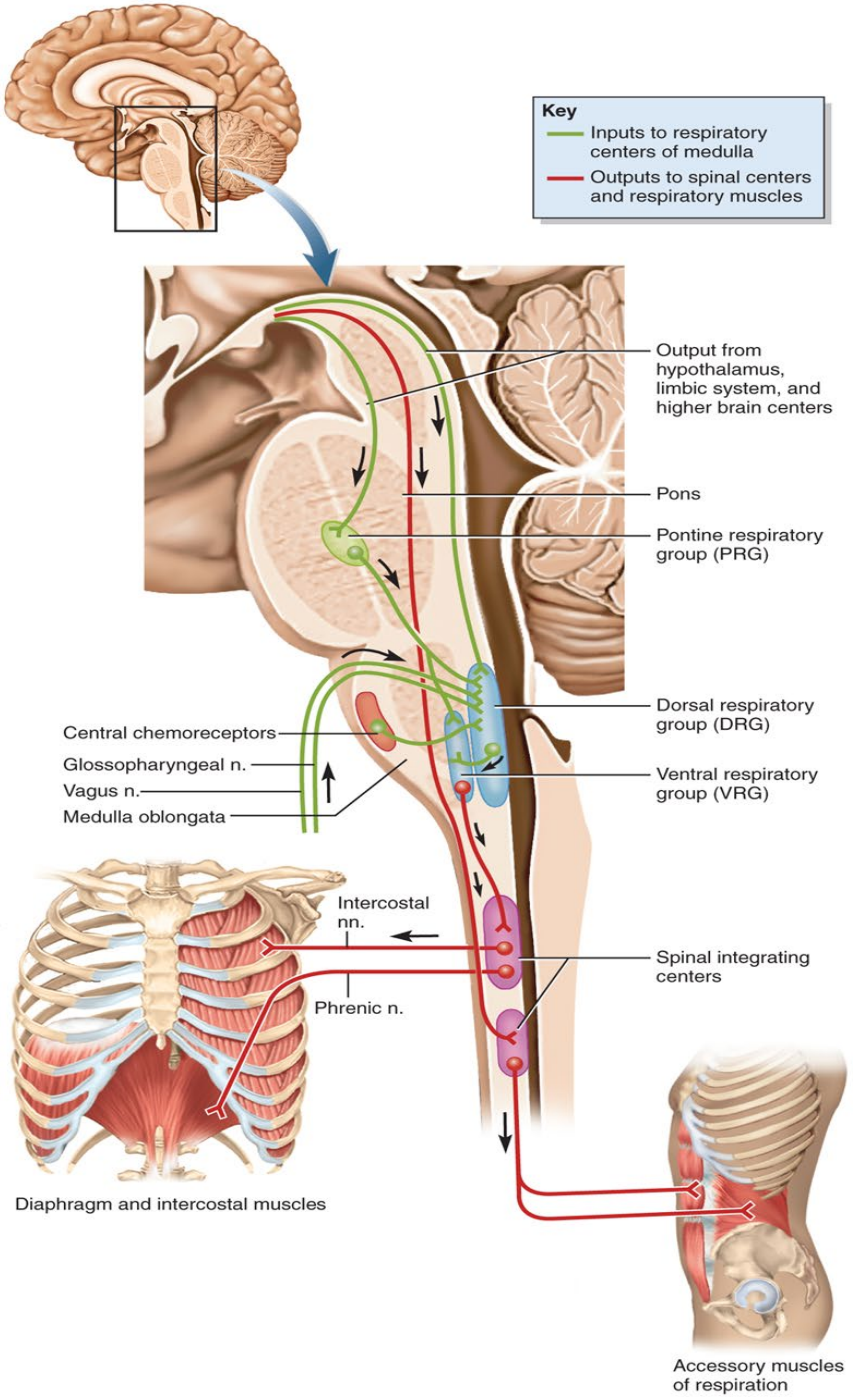
In a resting state, the phrenic nerve will only cause the diaphragm to contract

In forced respiration VRG send action potentials through the intercostal nerves to contract the external intercostal Muscles, increasing inspiration.

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



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.

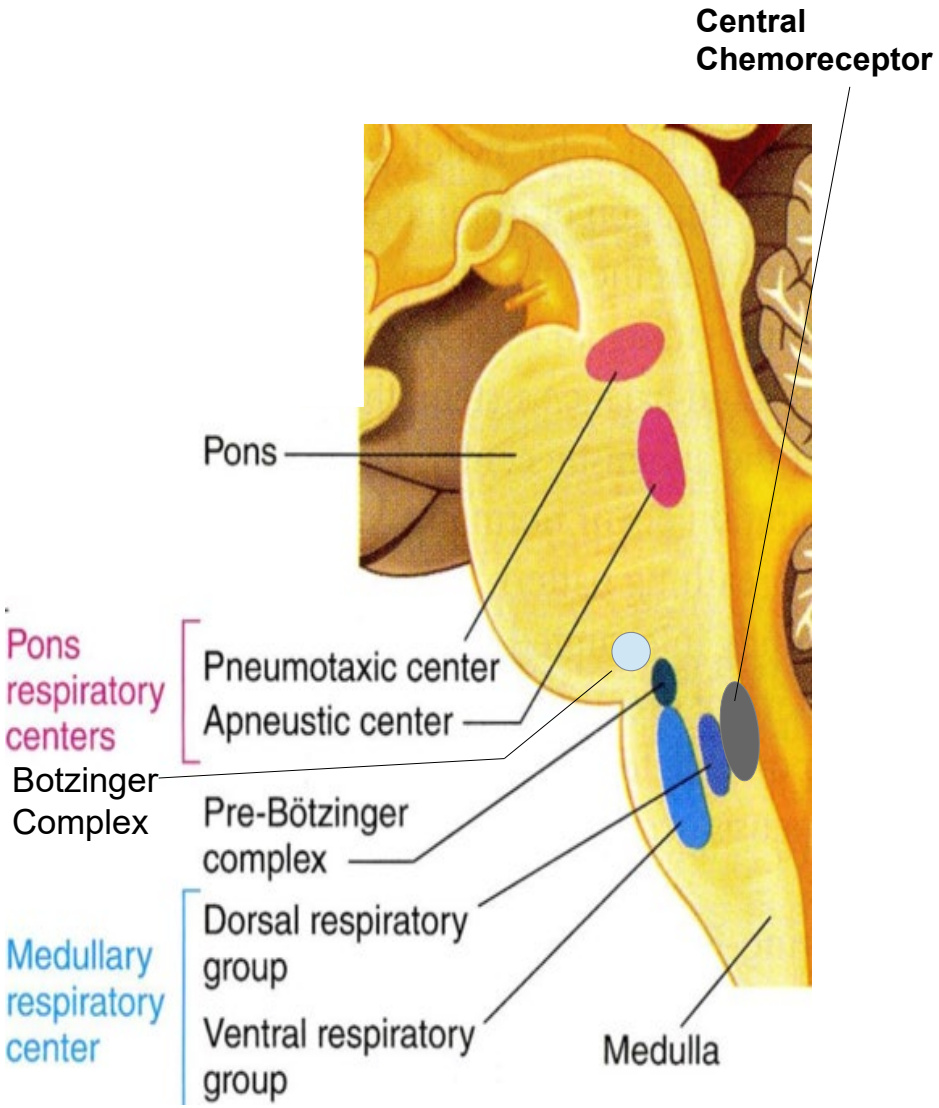
More Neural Control of Breathing

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 prolong inspiration // called apneustic breathing.

Central chemoreceptors monitors CO2 in cerebral spinal fluid // increase in CO2 stimulates inspiration // decrease C02 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

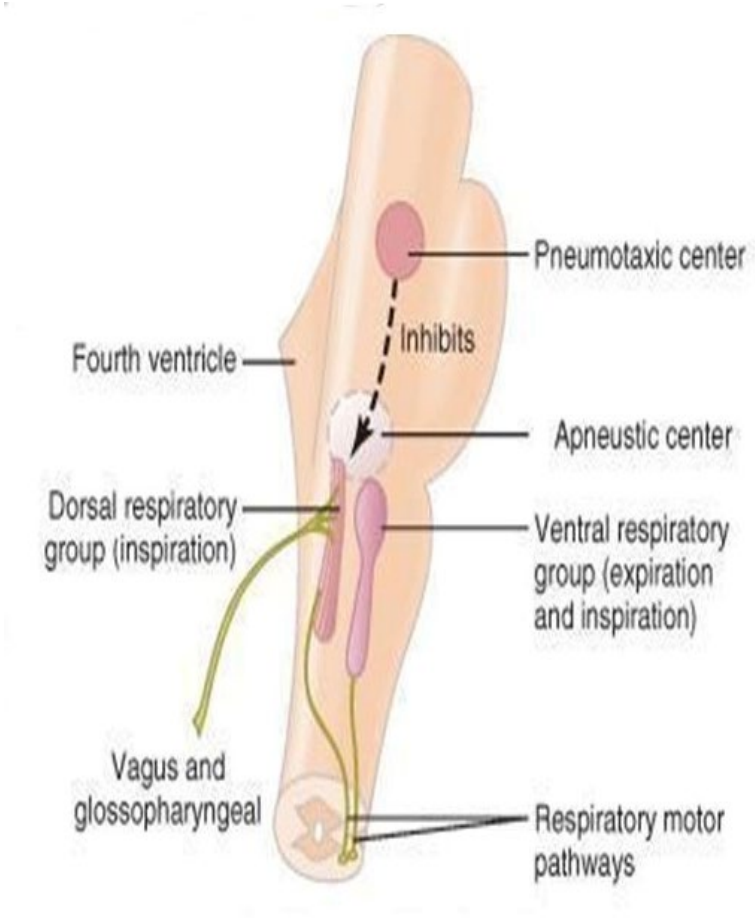


Neural Control of Breathing

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

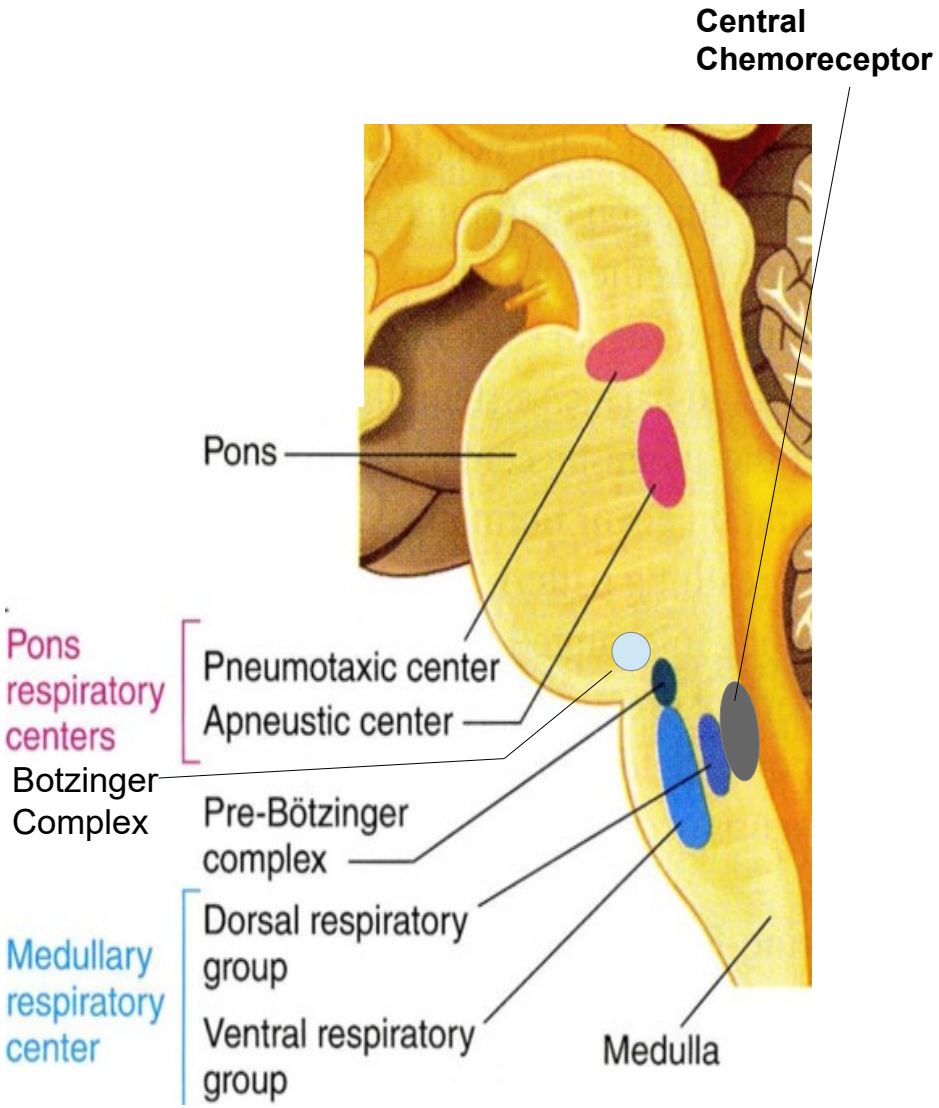


More Neural Control of Breathing

Dorsal and ventral respiratory group send Inspiratory neurons to the spinal cord's anterior Horn

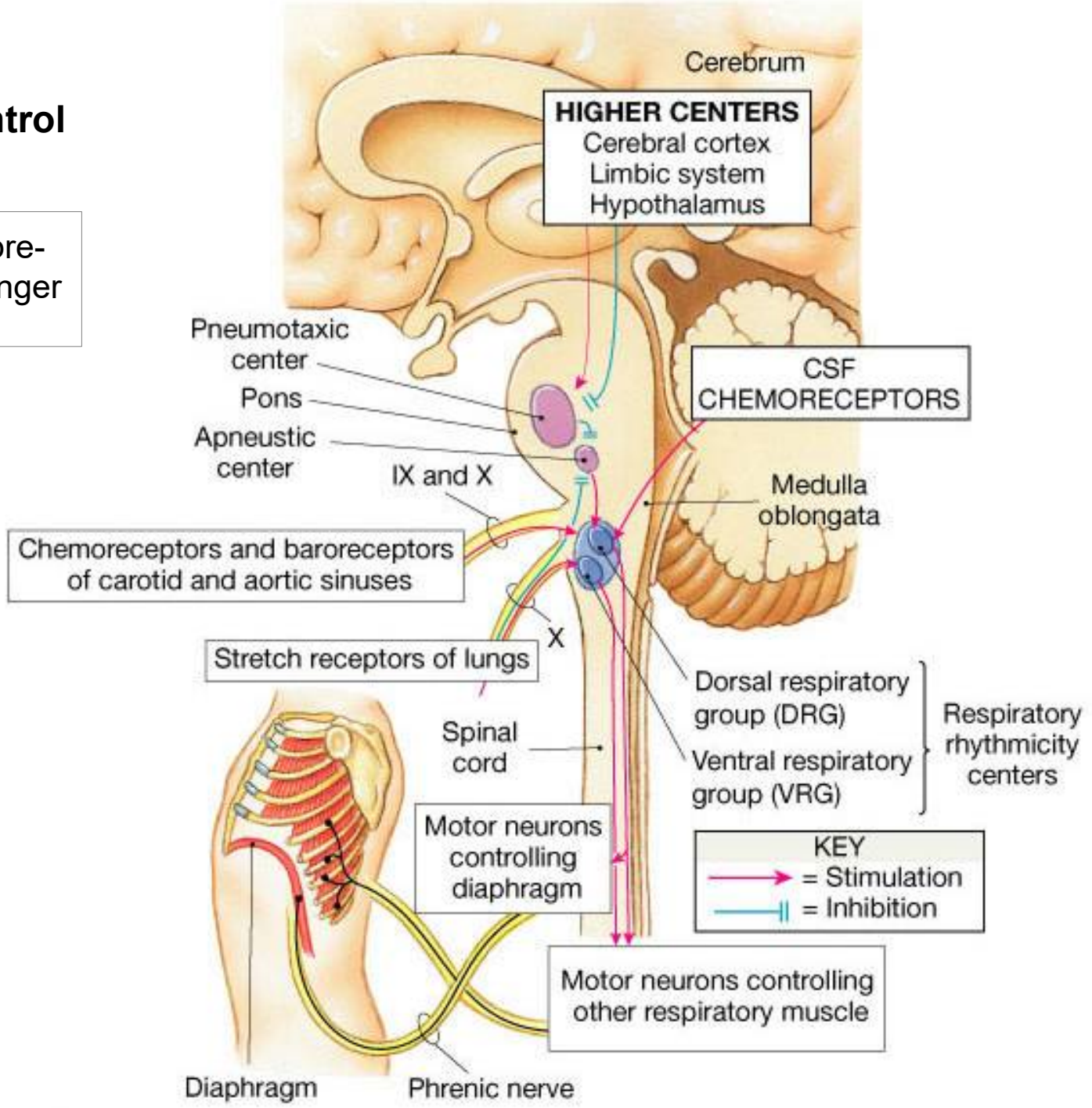
C3 to C5 lower motor pathway to phrenic nerve (diaphragm)

T1 to T11 lower motor pathway to intercostal muscles

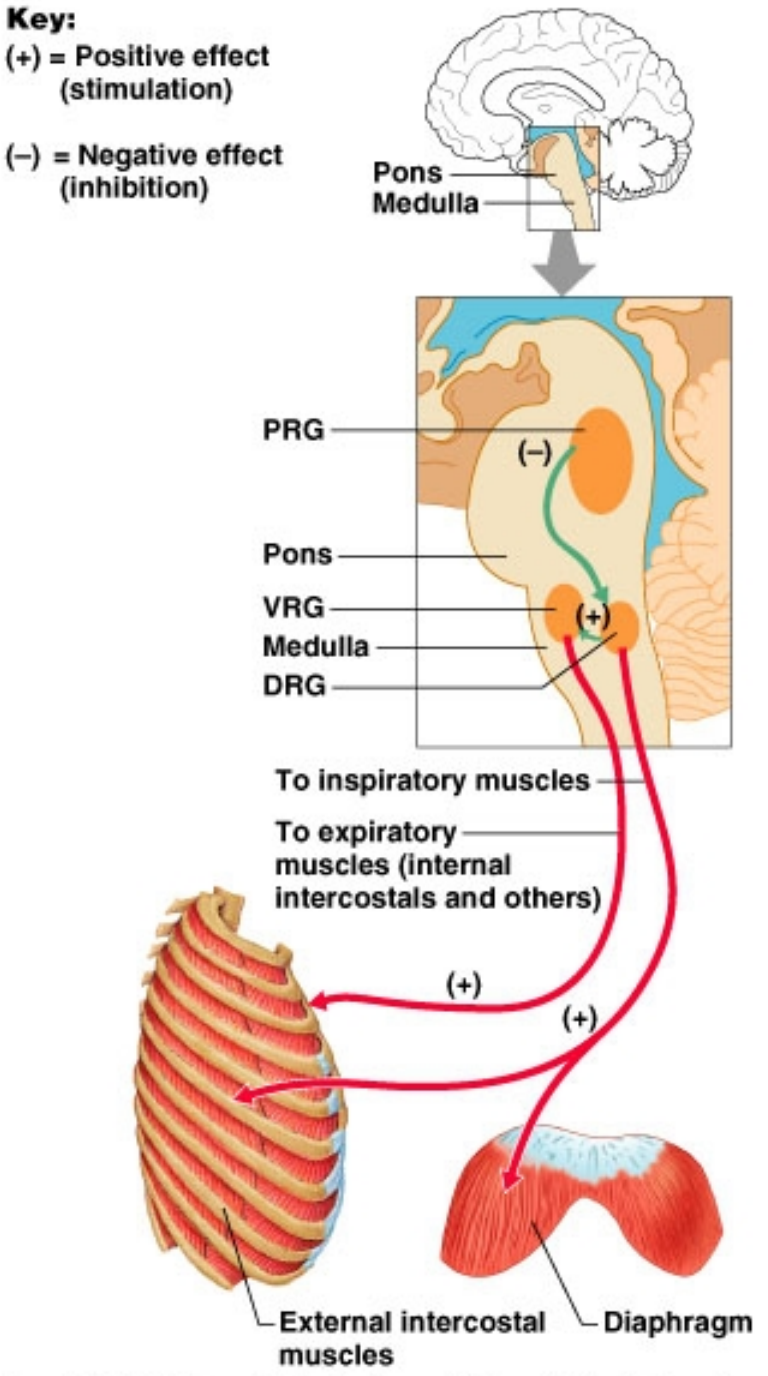


Respiratory Control Centers

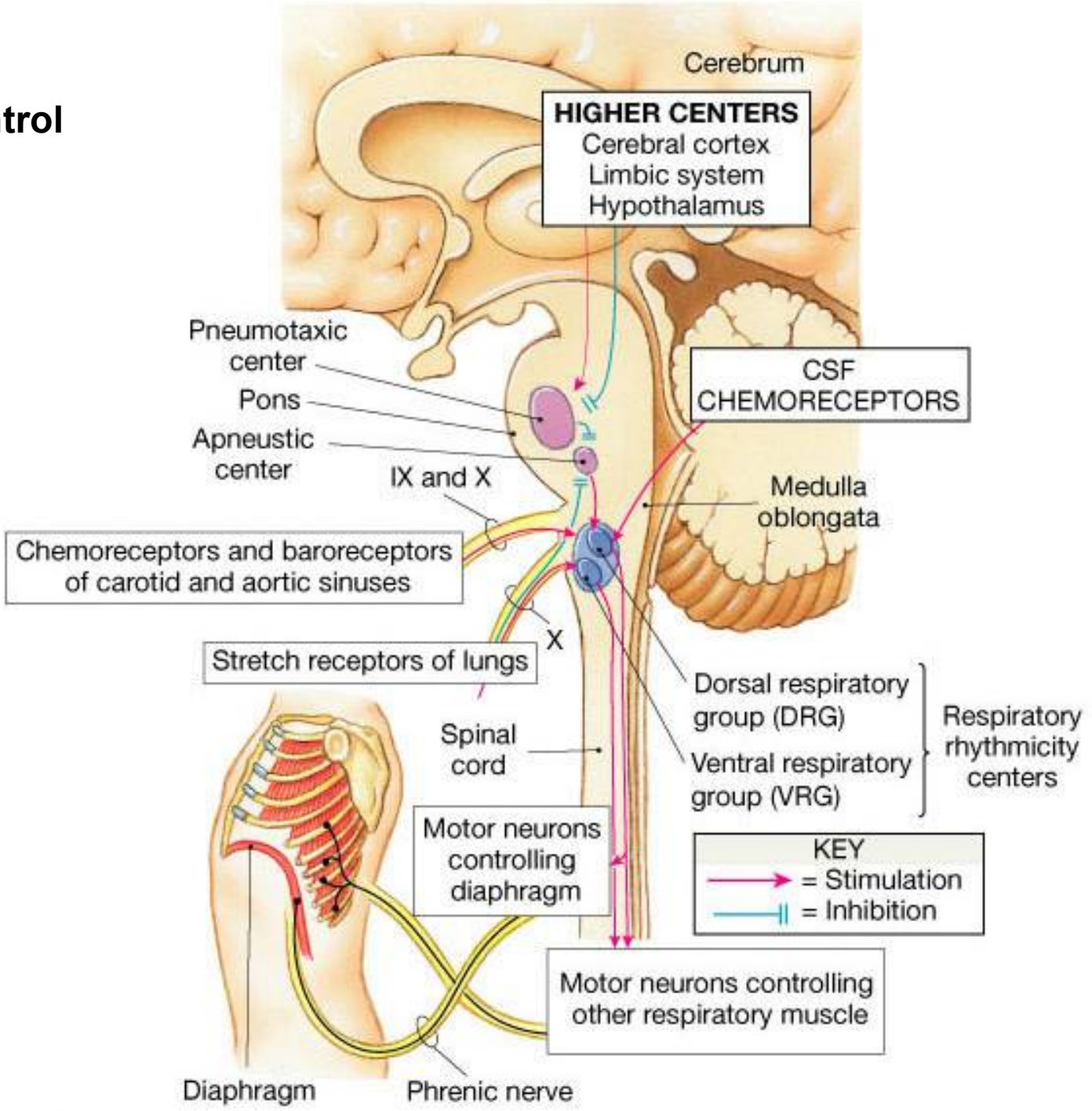
Not shown are the pre-Botzinger and Botzinger centers.



Respiratory Control Centers



Respiratory Control Centers

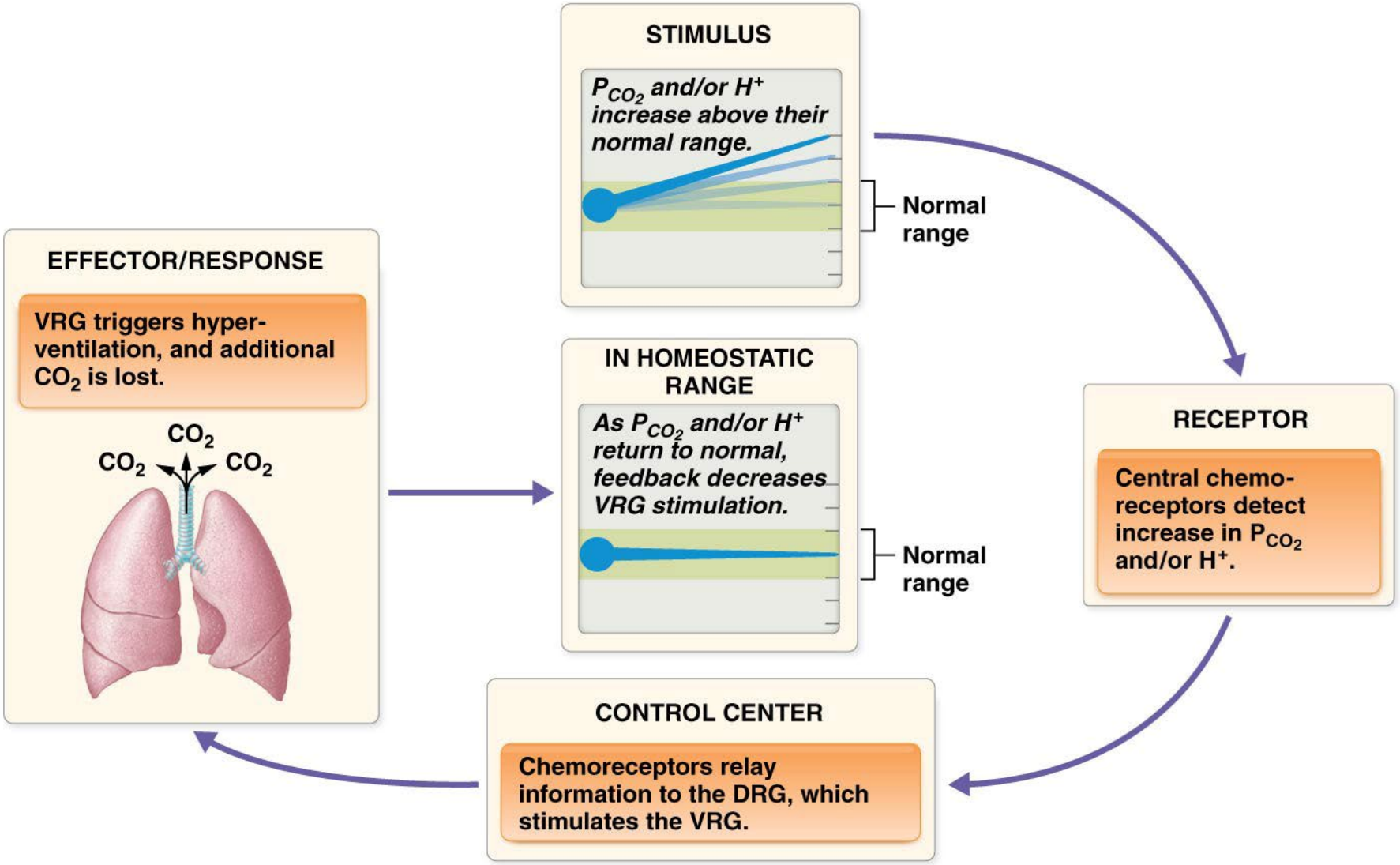


Physiologic States May Influence Neural Control of Breathing

Many factors may regulate breathing to match our physiological state

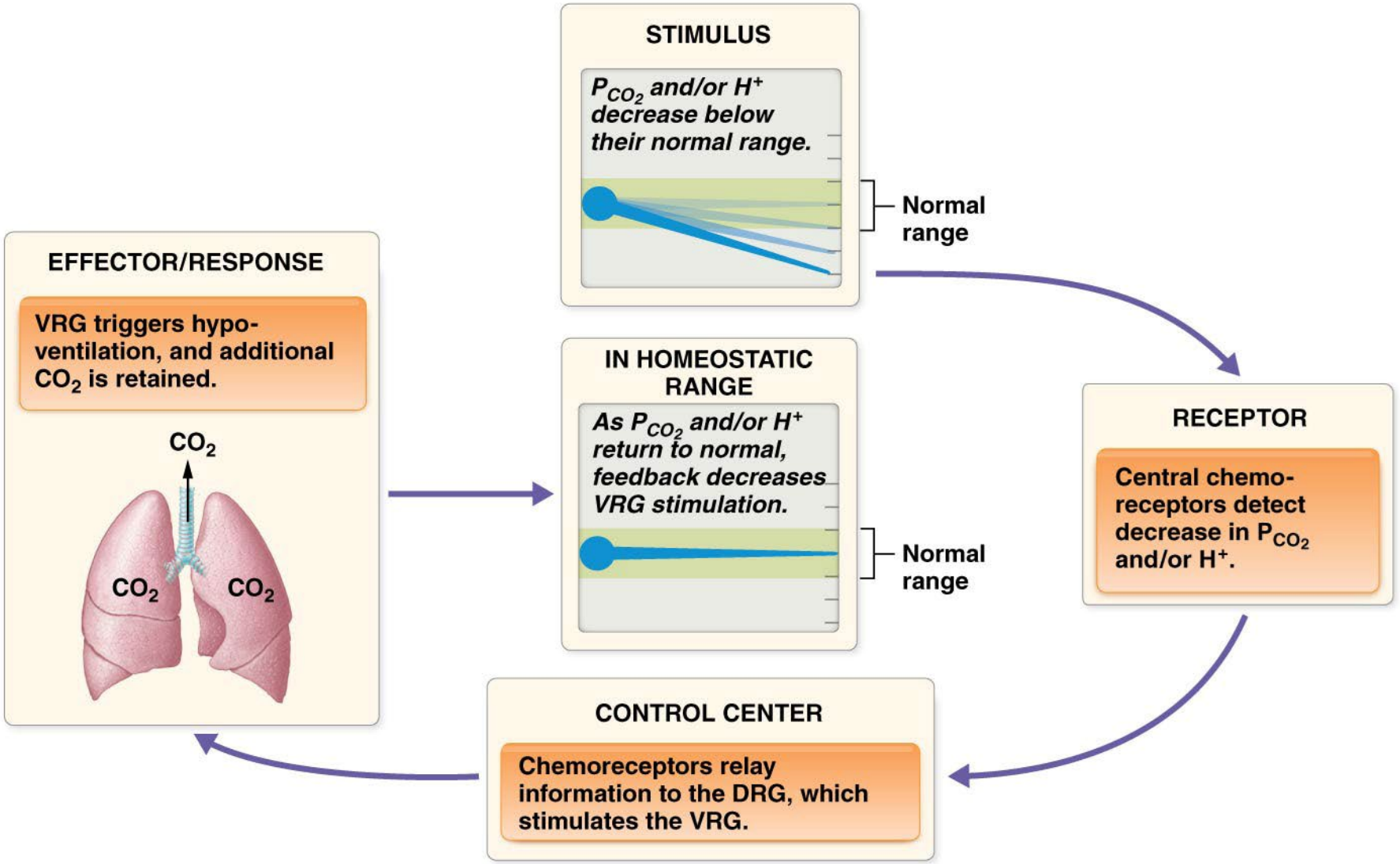
- central chemoreceptors
- peripheral chemoreceptors
- proprioreceptors
- lung stretch receptors (Hering-Breuer reflex)
- irritant reflex receptors
- limbic system
- temperature
- pain
- stretching the anal spincter
- blood pressure (minor)

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

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

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

Hyperventilation

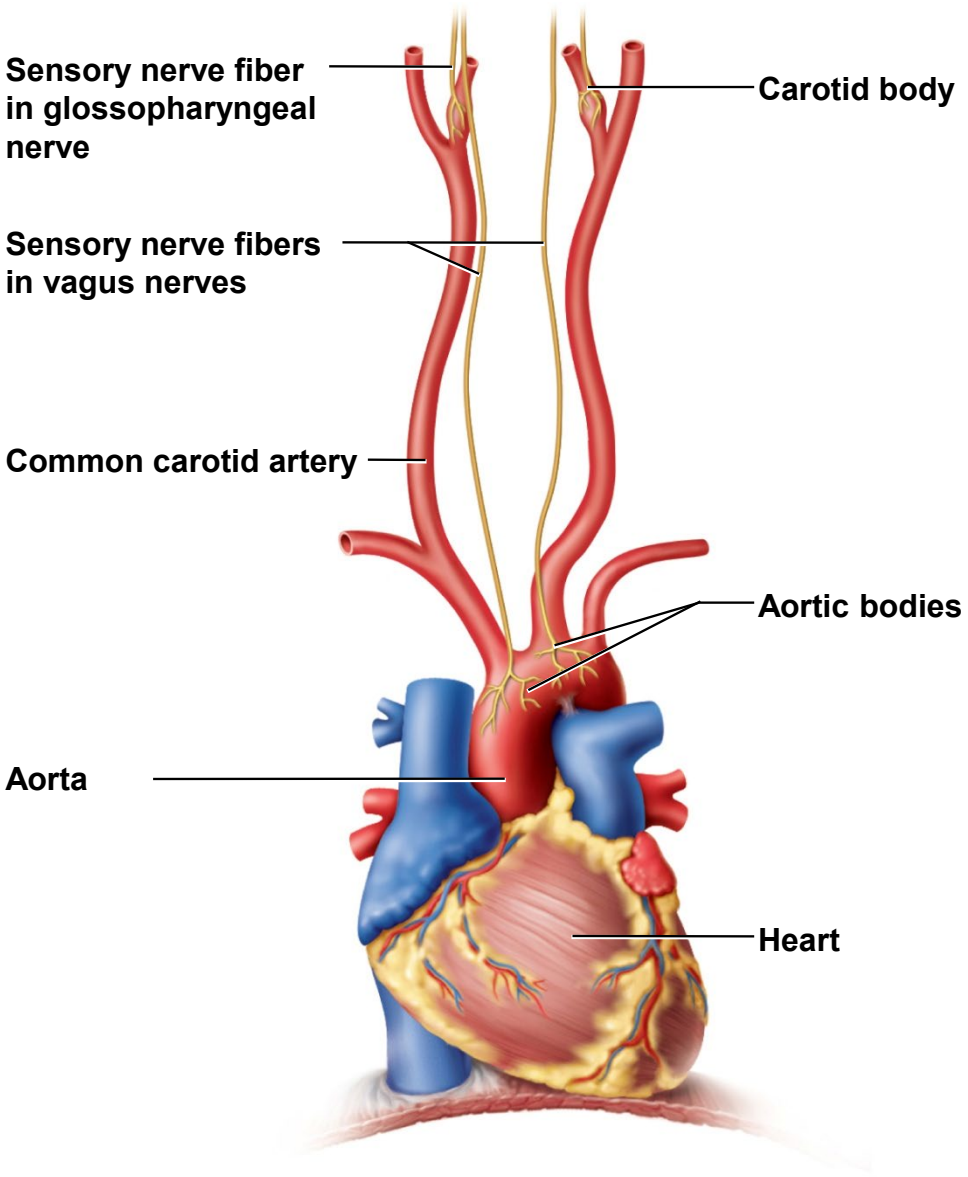
anxiety triggered state in which breathing is so rapid that it expels CO_2 from the body faster than it is produced

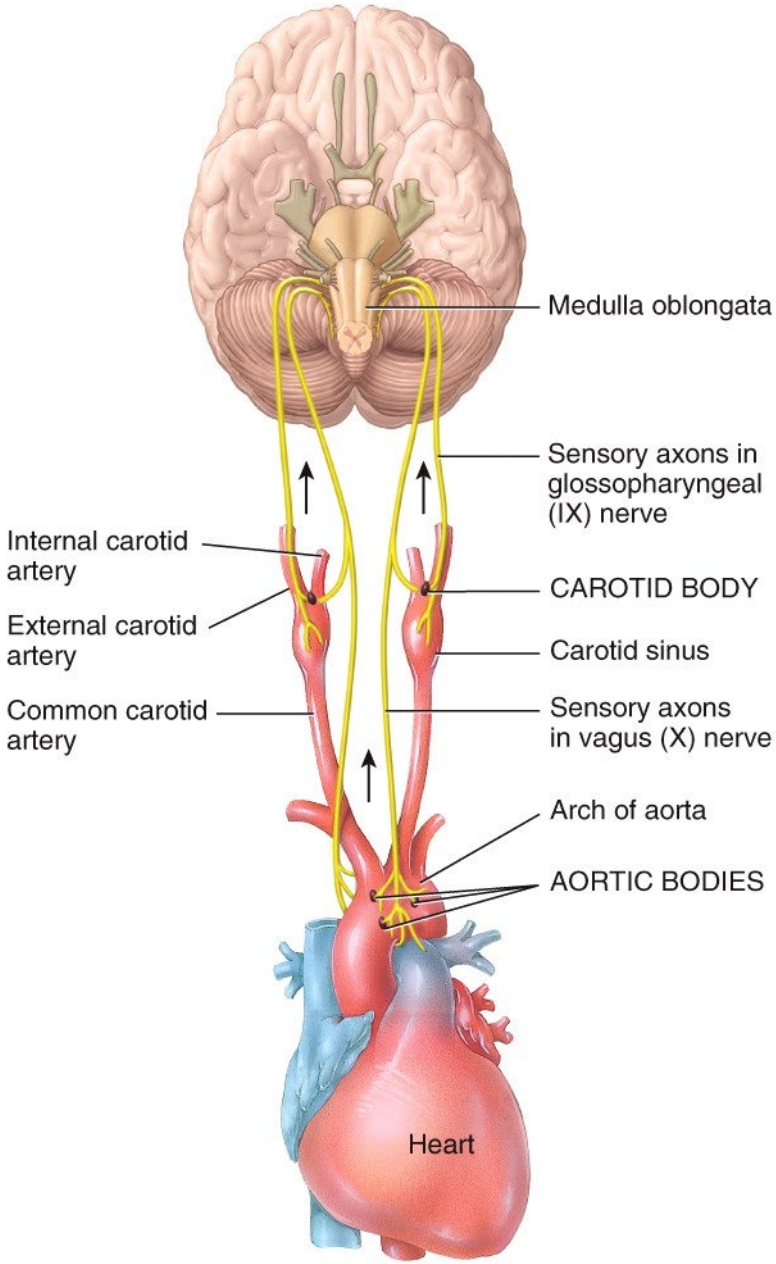
blood CO_2 levels drop (i.e. fewer protons = higher pH number = more alkaline)

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 CO_2 from a paper bag

Peripheral Chemoreceptors




Medulla oblongata
Sensory axons in glossopharyngeal (IX) nerve
Internal carotid artery
CAROTID BODY
External carotid artery
Carotid sinus
Common carotid artery
Sensory axons in vagus (X) nerve
Arch of aorta
AORTIC BODIES
Heart

Voluntary Control May Also Modify Breathing

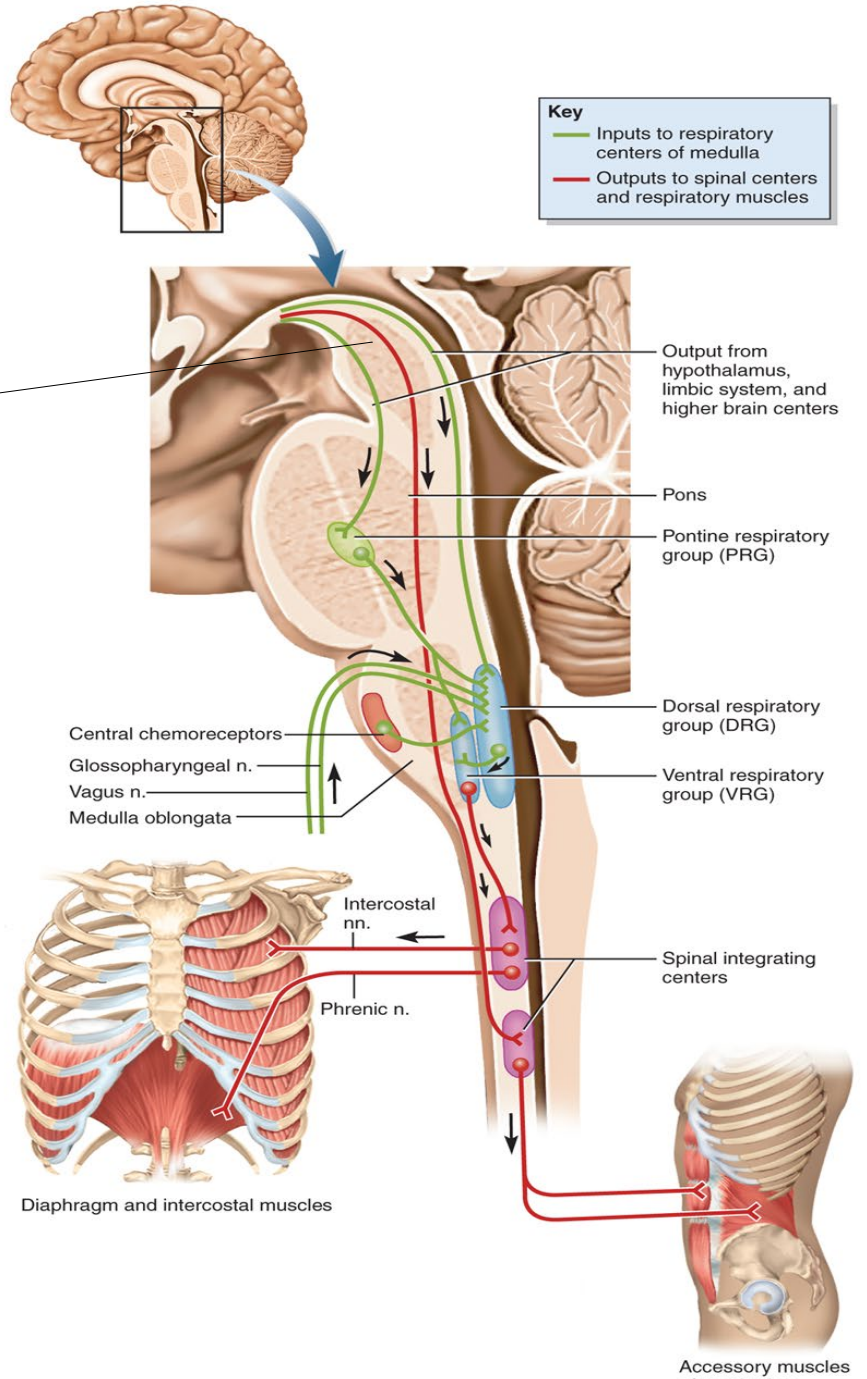
Originates in the motor cortex of frontal lobe of cerebrum

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

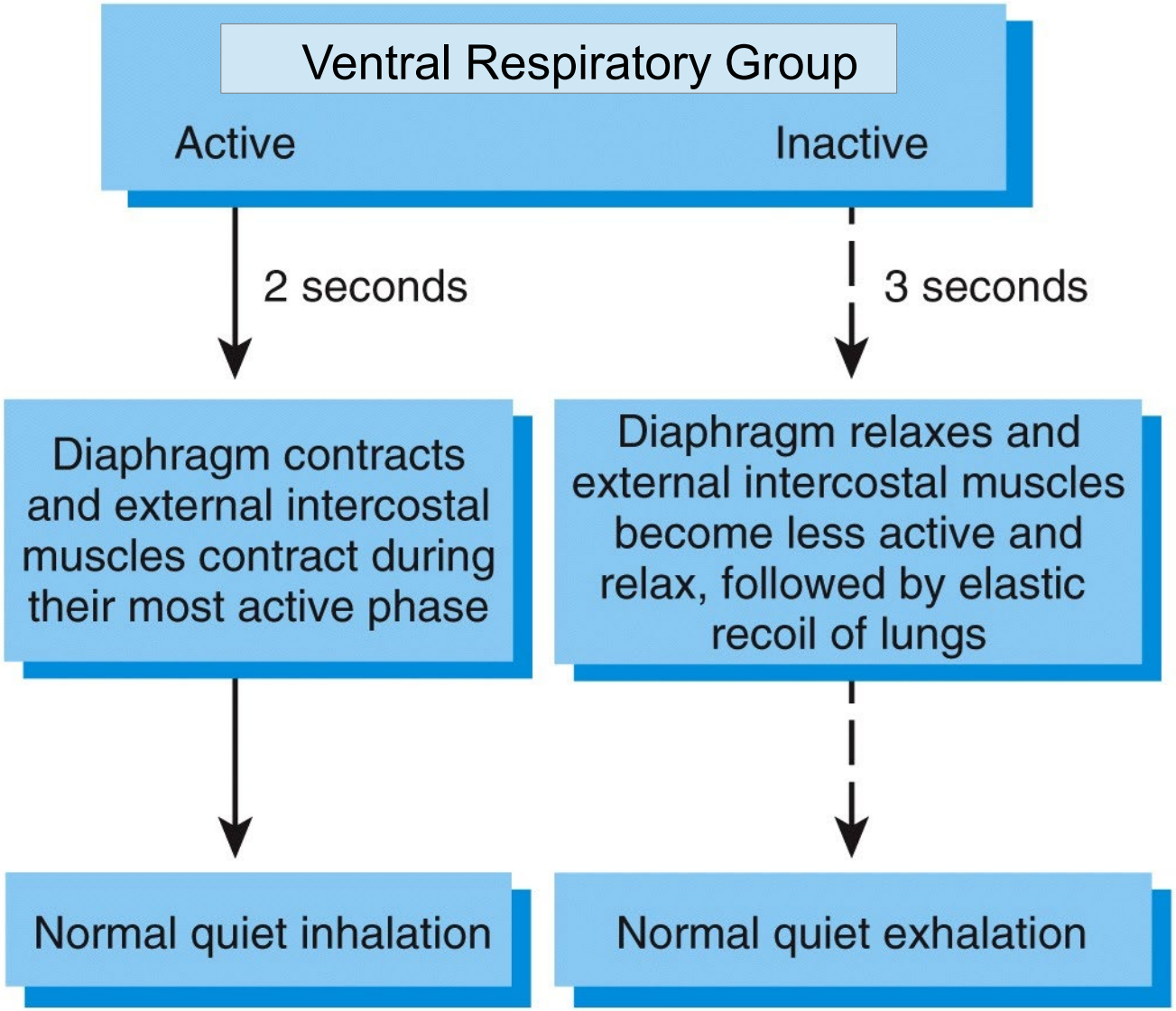
There is a limits to voluntary control /// called the breaking point // when CO_2 levels will rise to a point then the automatic controls override your will power not to breath

Voluntary Control May Also Modify Breathing

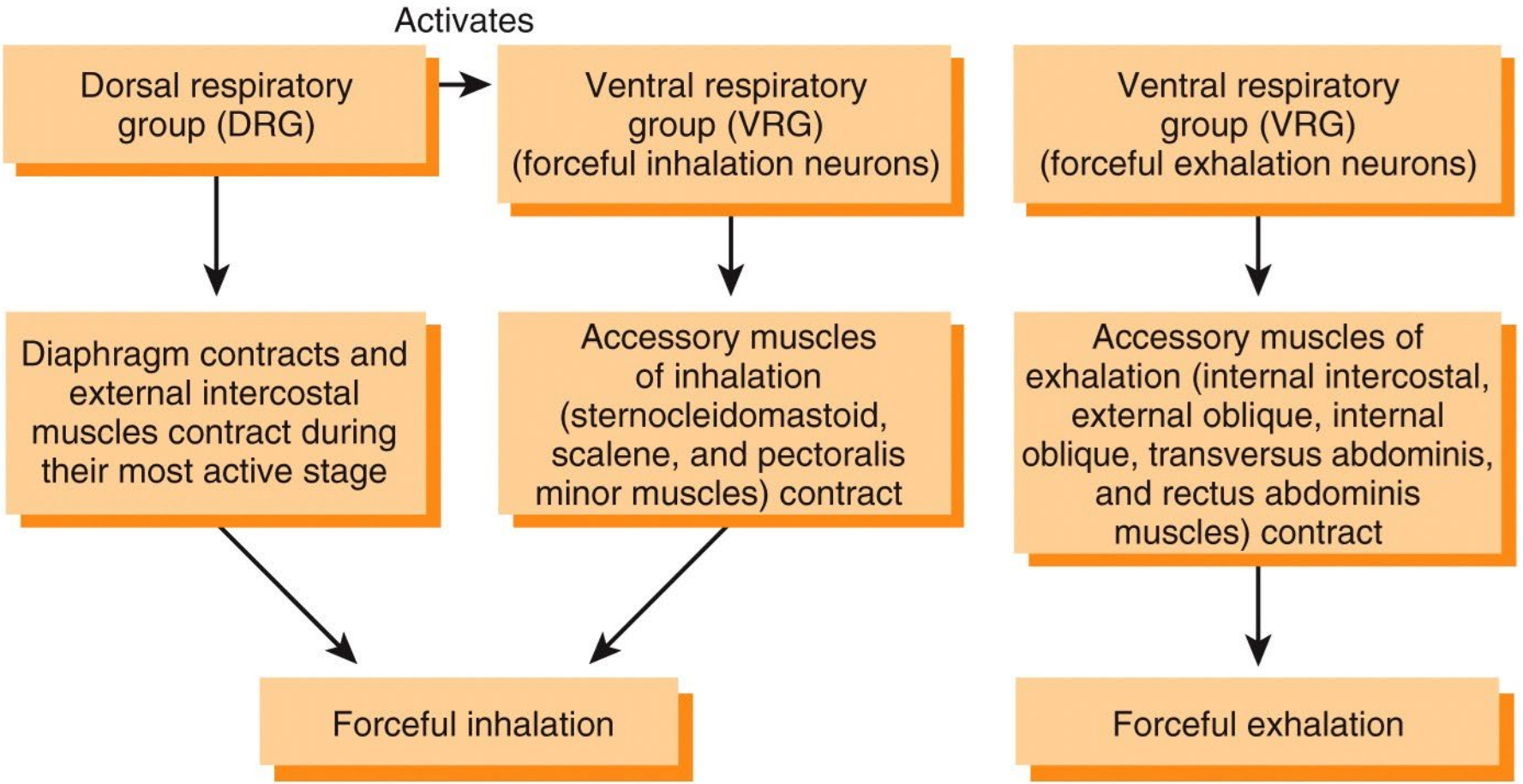
Note: this track bypasses brain stem regulation



Quiet Breathing

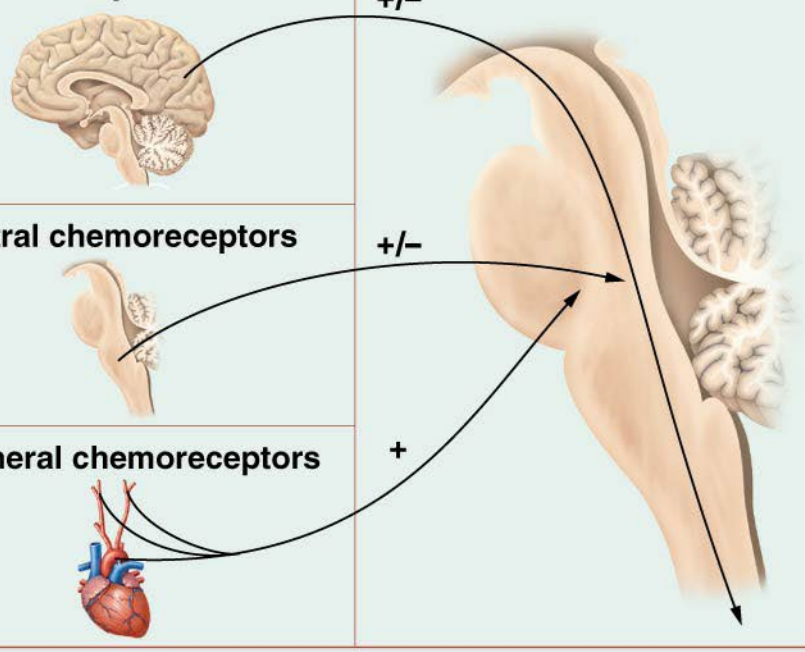


Forceful Breathing or Breathing During Exercise

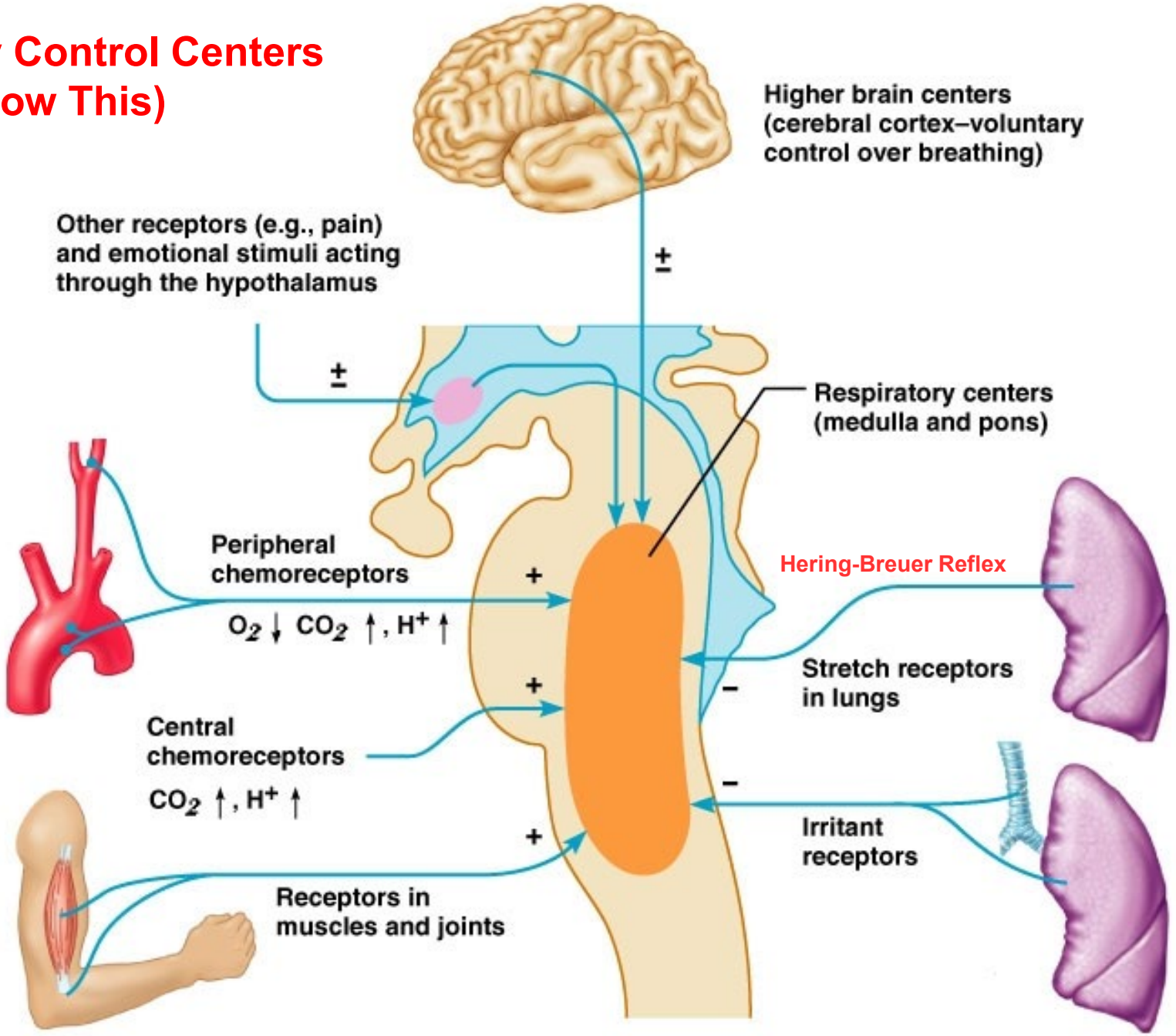


Control mechanisms of ventilation.

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



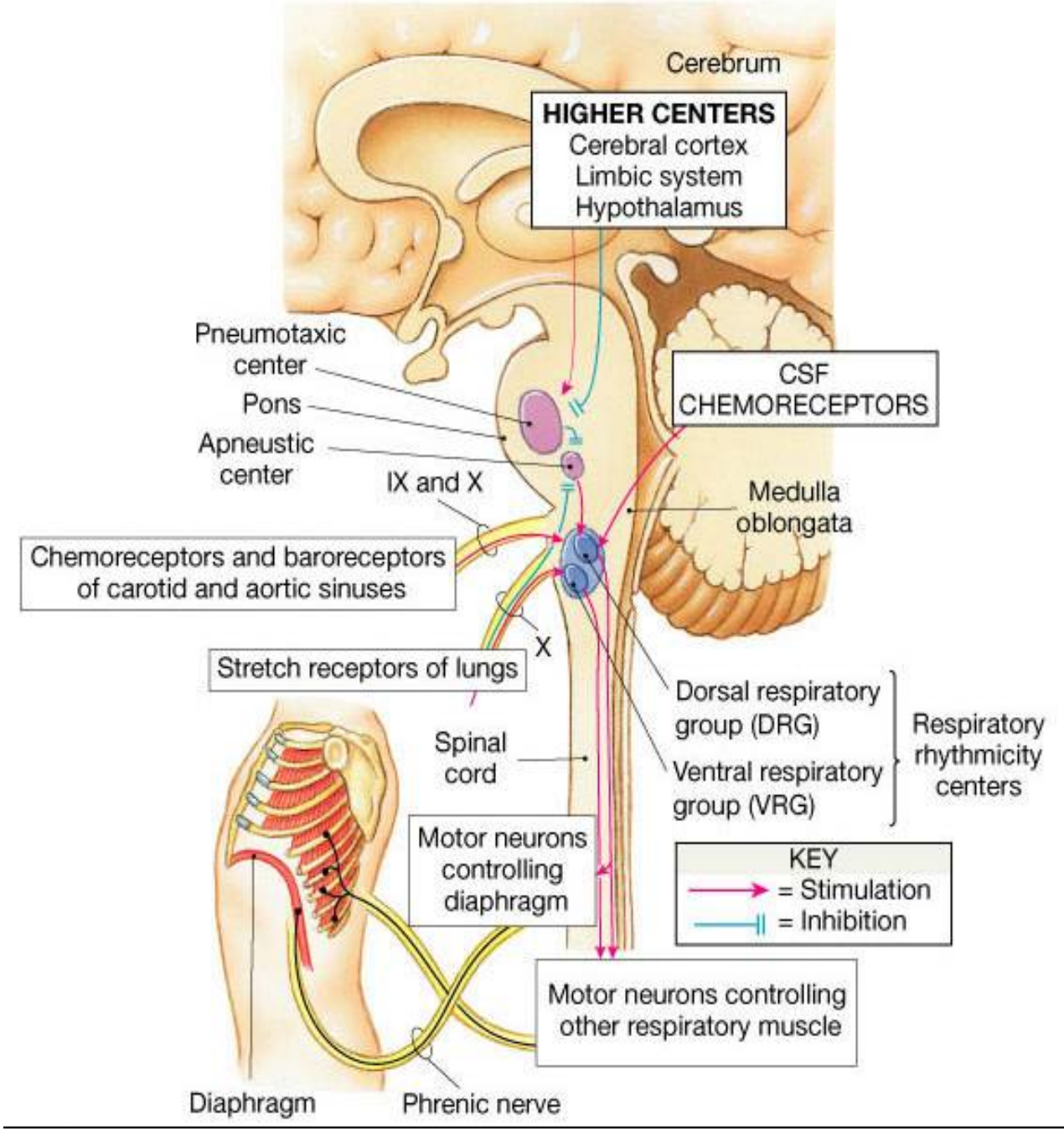
Respiratory Control Centers (Know This)

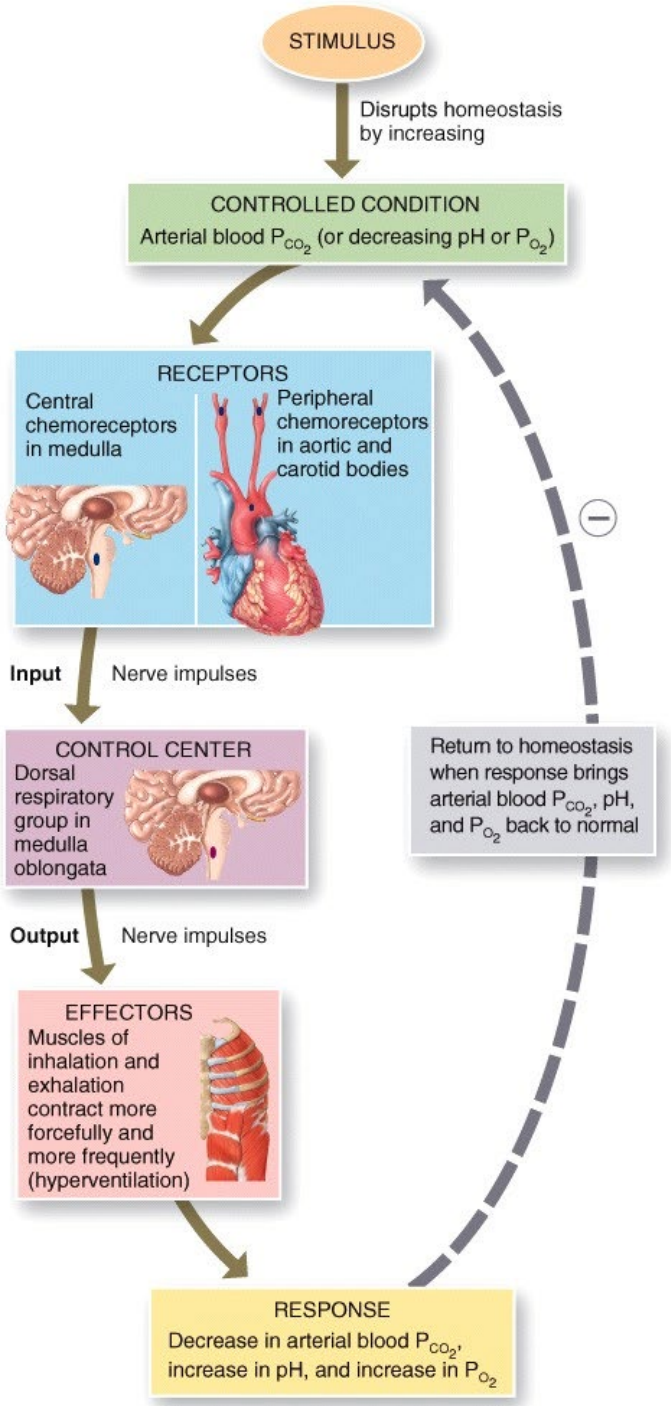


Respiratory Control Centers

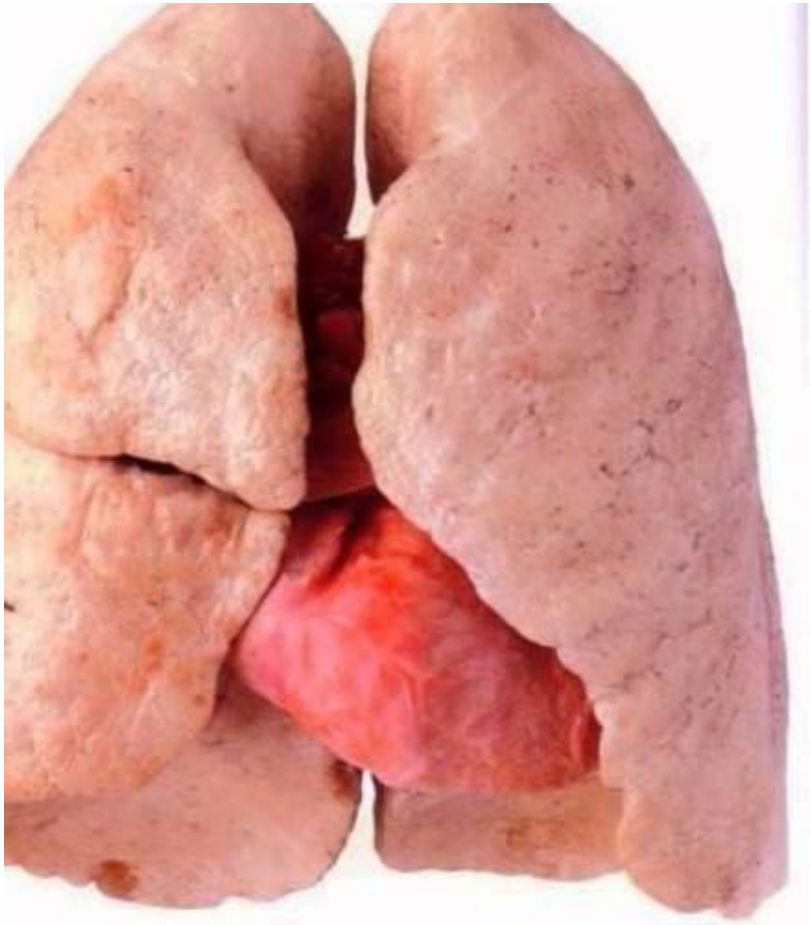
When you speak or sing the cerebral cortex must adjust breathing.

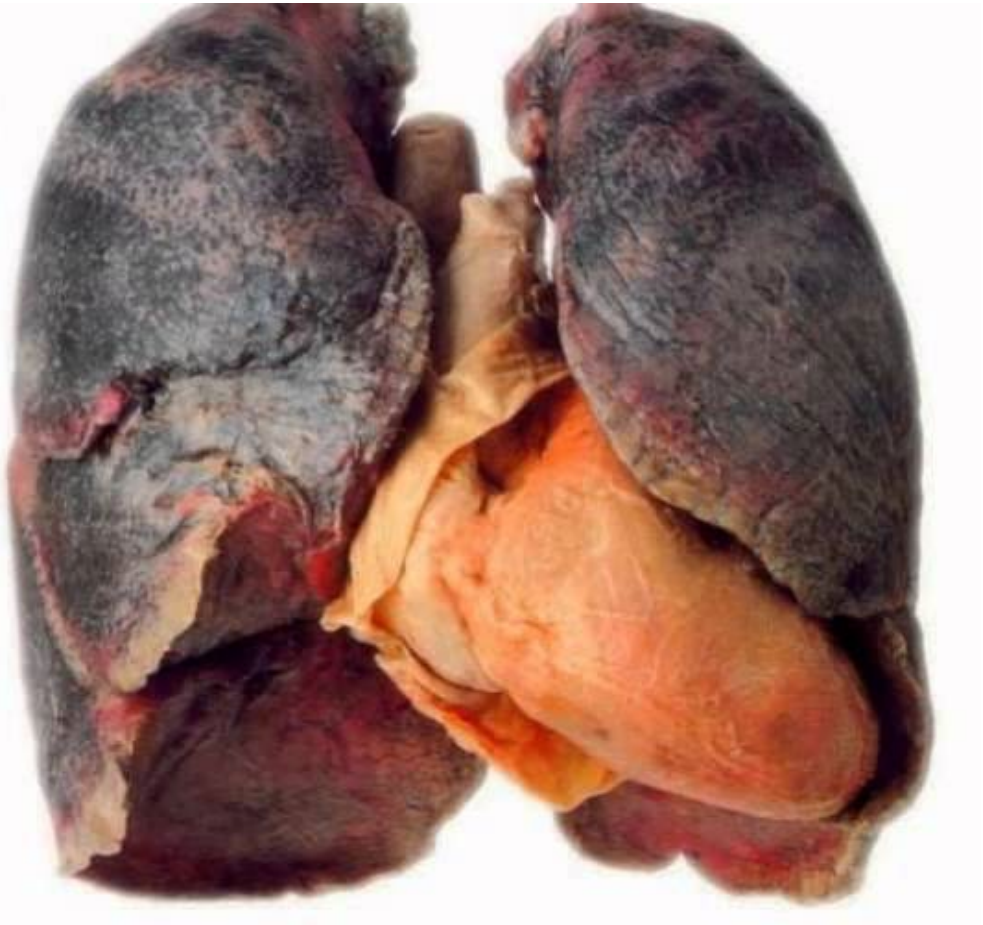
Your breathing rate will also change as you get ready to board a spaceship.




STIMULUS
Disrupts homeostasis by increasing
CONTROLLED CONDITION
Arterial blood P_{CO_2} (or decreasing pH or P_{O_2})
RECEPTORS
Central chemoreceptors in medulla
Peripheral chemoreceptors in aortic and carotid bodies
Input
Nerve impulses
CONTROL CENTER
Dorsal respiratory group in medulla oblongata
Output
Nerve impulses
EFFECTORS
Muscles of inhalation and exhalation contract more forcefully and more frequently (hyperventilation)
RESPONSE
Decrease in arterial blood P_{CO_2}, increase in pH, and increase in P_{O_2}
Return to homeostasis when response brings arterial blood P_{CO_2}, pH, and P_{O_2} back to normal
−

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.

Source: The American Cancer Society (www.cancer.org)

15▫40 connection

NewCancerConversation.org

Central and Peripheral Input to Respiratory Centers

Central chemoreceptors (responsible for 75% of CO_2 drive)

brainstem neurons respond to changes in pH of cerebrospinal fluid

pH of cerebrospinal fluid reflects the CO_2 level in the blood

by regulating respiration to maintain stable pH /// respiratory center also ensures stable CO_2 level in the blood

Peripheral chemoreceptors (responsible for 25% of CO_2 drive)

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

peripheral chemorecptors are sensitive to both PCO_2 and low arterial PO_2

under normal conditions central chemoreceptors primary regulator of respiration

Following chronic high CO_2 – PCR senses low PO_2 and this becomes primary stimulus for respiration

Hypoxic Drive

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

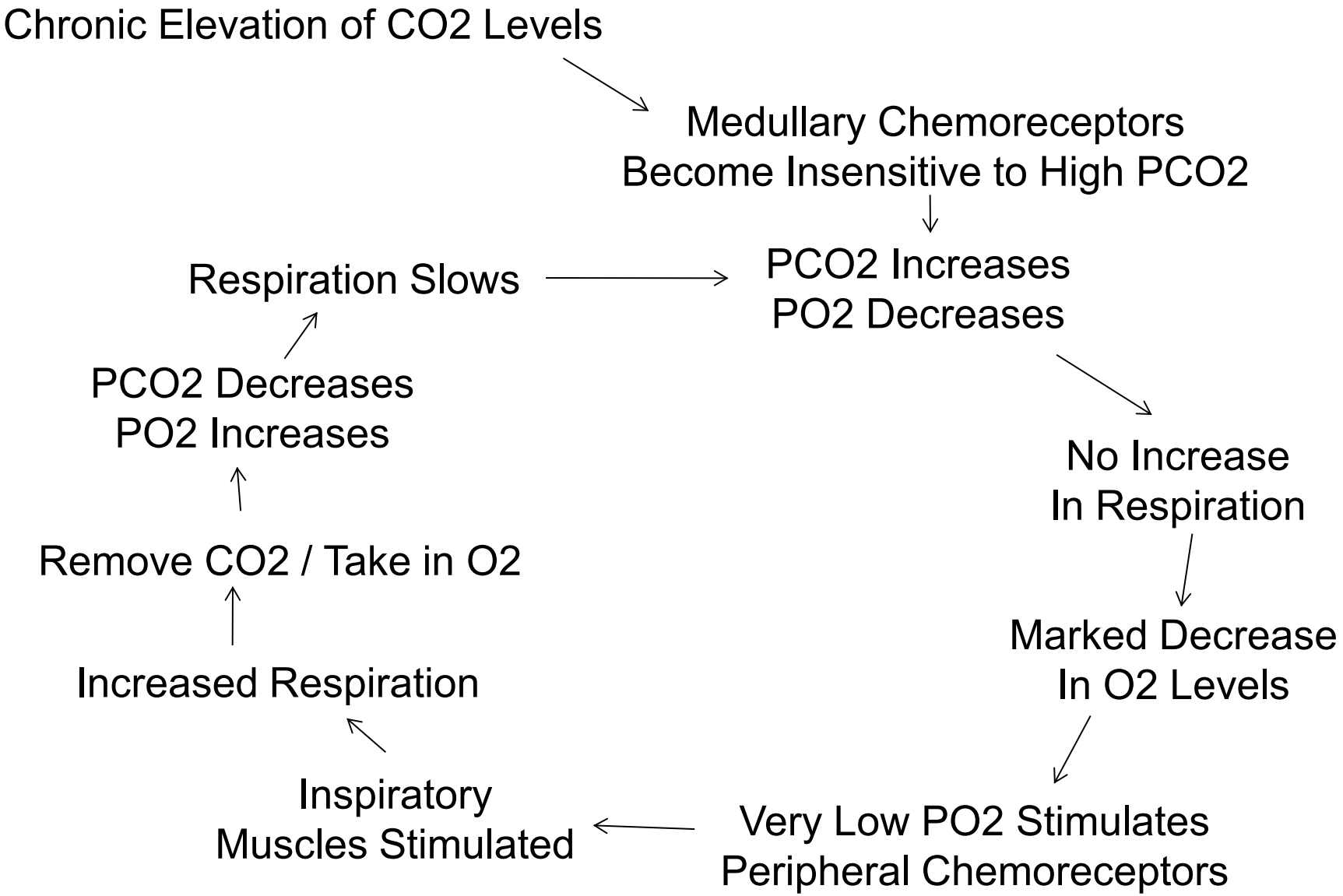
If high PCO_2 becomes a chronic condition, then chemoreceptors in medulla become insensitive to PCO_2

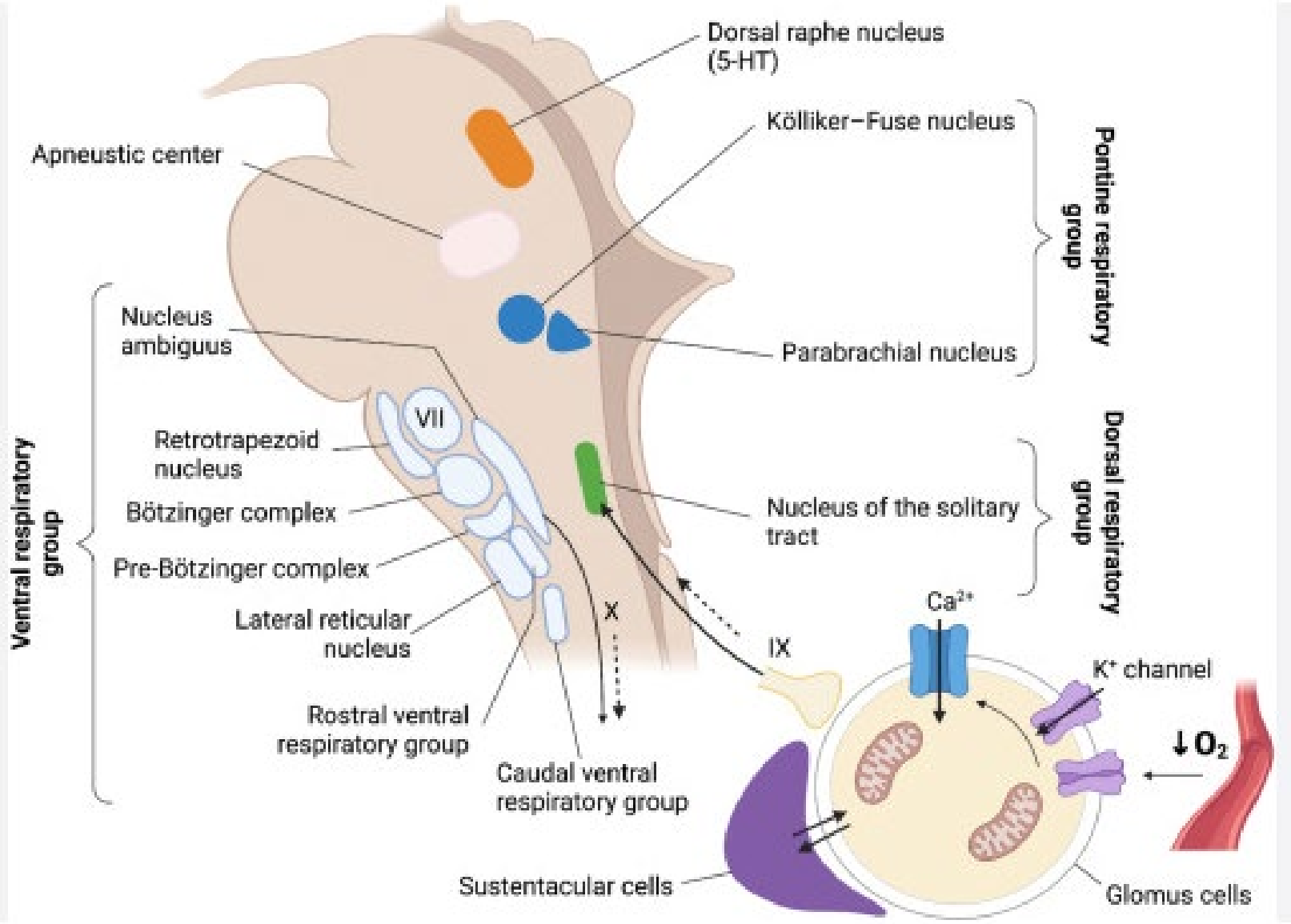
Then peripheral chemoreceptor (which monitor arterial PO_2) stimulate inspiration if arterial blood has low PO_2 levels

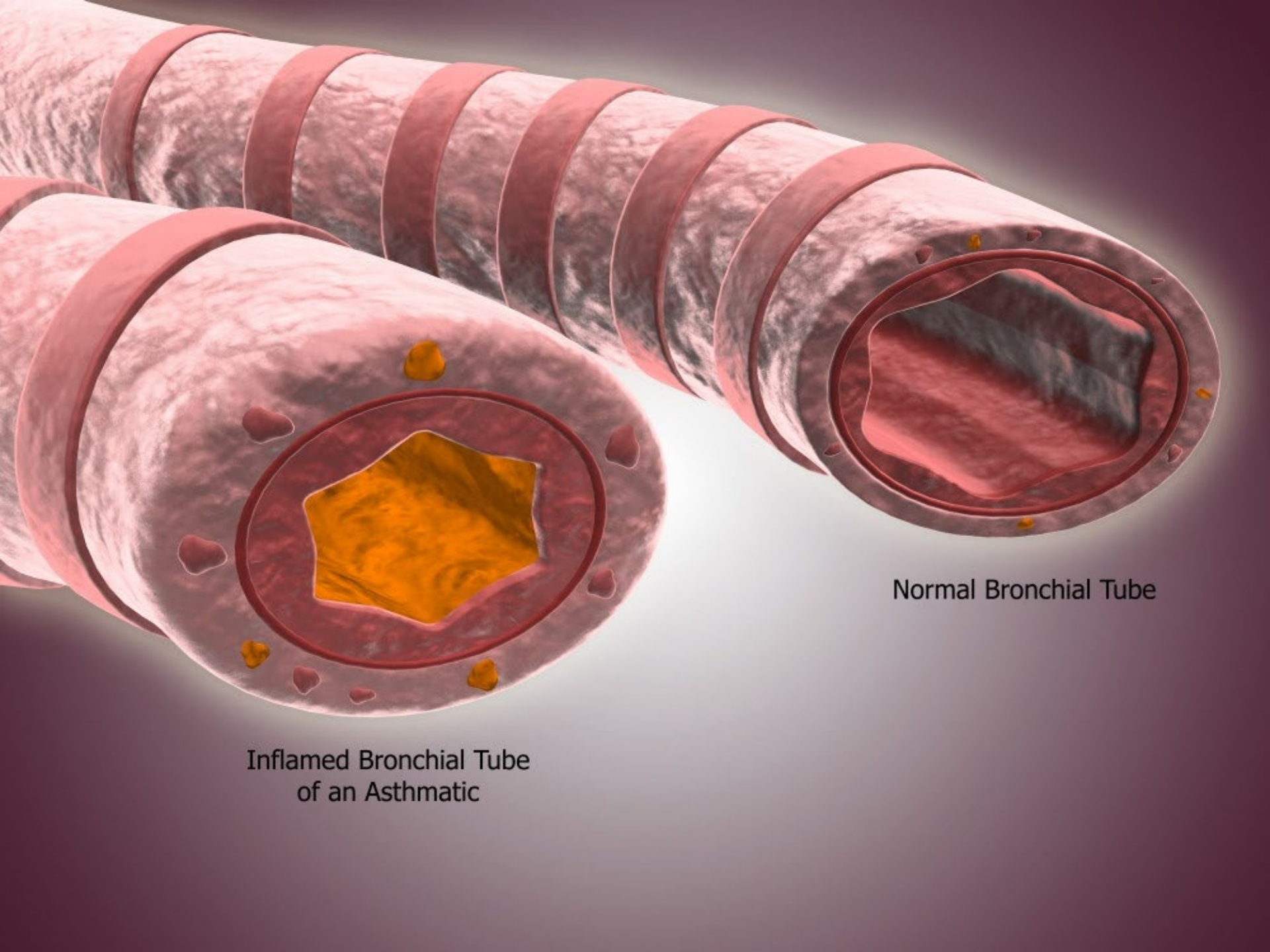
This theory also suggest that high PO_2 will then inhibit inspiration.

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

Hypoxic Drive




Dorsal raphe nucleus
(5-HT)
Apneustic center
Kölliker–Fuse nucleus
Pontine respiratory group
Nucleus ambiguus
Parabrachial nucleus
Ventral respiratory group
VII
Retrotrapezoid nucleus
Bötzinger complex
Nucleus of the solitary tract
Dorsal respiratory group
Pre-Bötzinger complex
Lateral reticular nucleus
X
IX
Ca2+
K+ channel
↓O2
Rostral ventral respiratory group
Caudal ventral respiratory group
Sustentacular cells
Glomus cells


Normal Bronchial Tube
Inflamed Bronchial Tube
of an Asthmatic