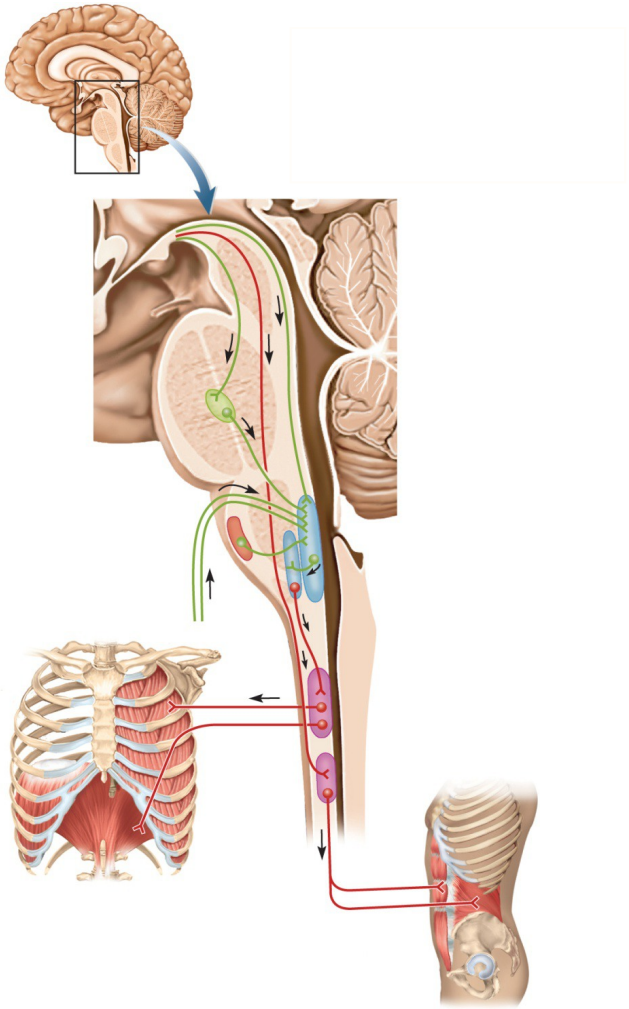
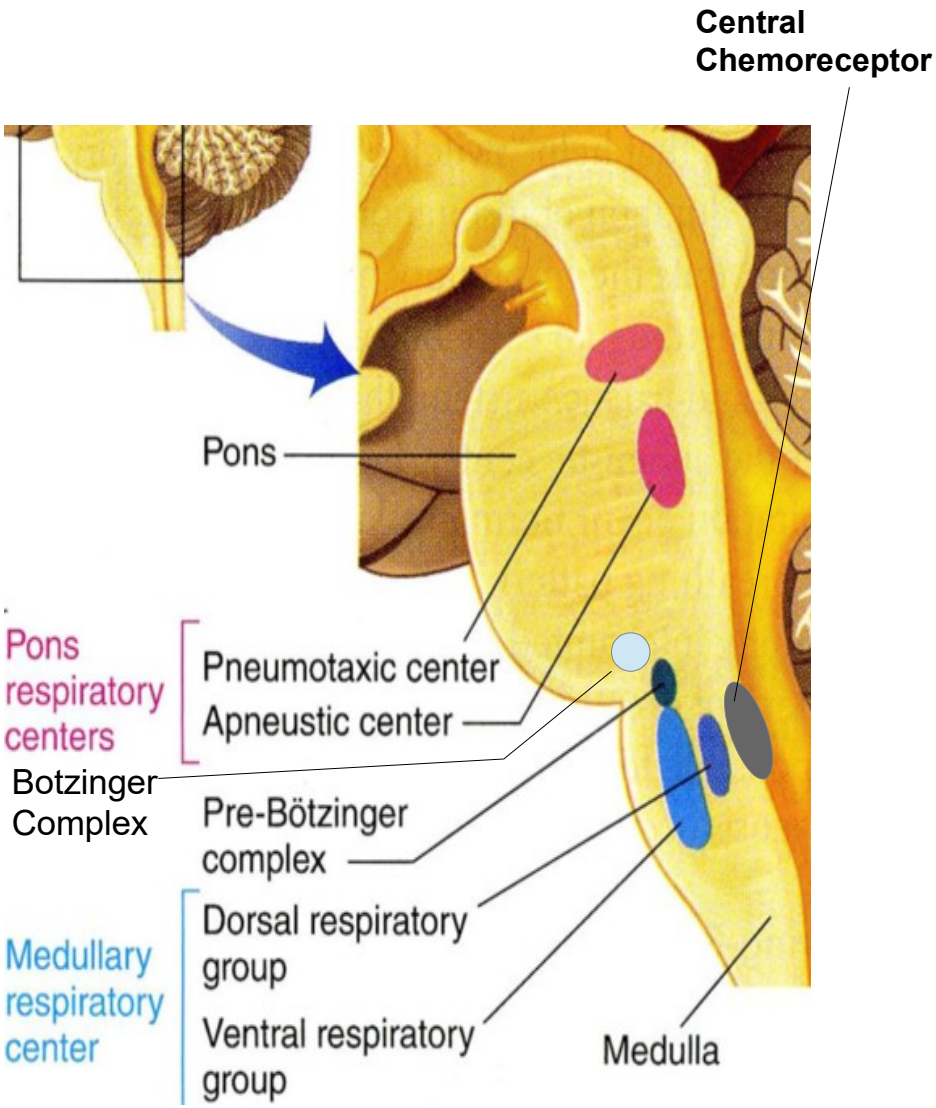
Chapter 22.3

Neural Control of Breathing



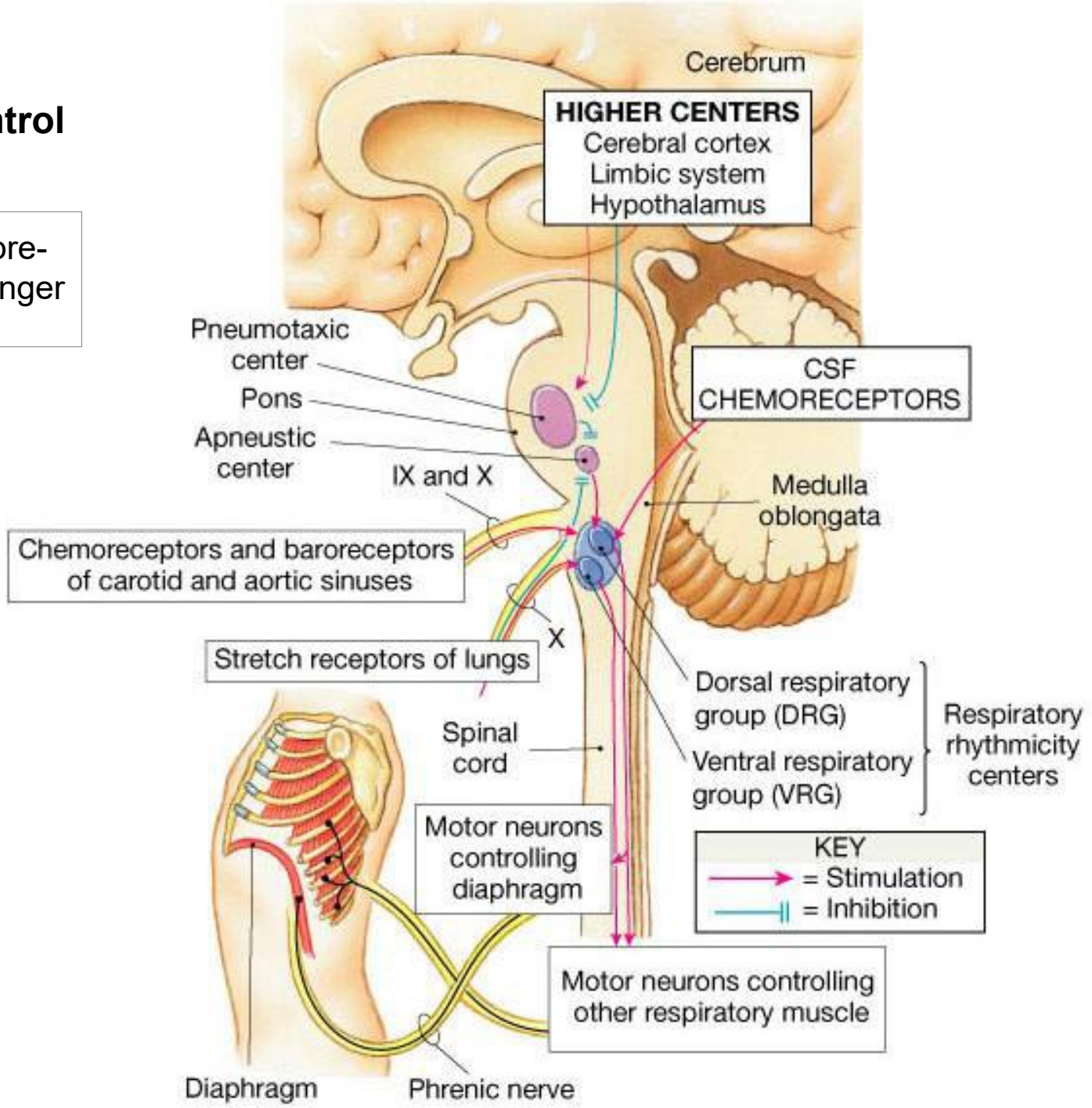
Neural Control of Breathing

- Breathing is the movement of air in and out of the lungs
- No air moves in the transition between inspiration (in) vs expiration (out)
- Two centers in the brain stem regulate breathing
  - Pons Respiratoy Center
  - Medullary Respiratory Center (MRC)
- *Breathing is influenced by higher brain centers, chemo-receptors, stretch receptors, irritant receptors, and proprioreceptors.*



Respiratory Control Centers

Not shown are the pre-Botzinger and Botzinger centers.



Neural Control of Breathing

- The medullary respiratory center and the pons respiratory center work together to control breathing.

- Each center have two nuclei
  - The pons respiratory center
    - Pneumotaxic center
    - Apneustic center
  - The medullary respiratory center (MRC)
    - Ventral respiratory group (sets breathing rate sent to diaphragm) /// MRC's ventral respiratory group is a motor pathways to control diaphragm contraction.
    - Dorsal respiratory group (collects stimulus from higher brain centers then relay signals to VRG and motor pathways to intercostal muscles and other respiratory muscles) /// MRC dorsal respiratory group is a motor pathway to control intercostal and other respiratory muscles

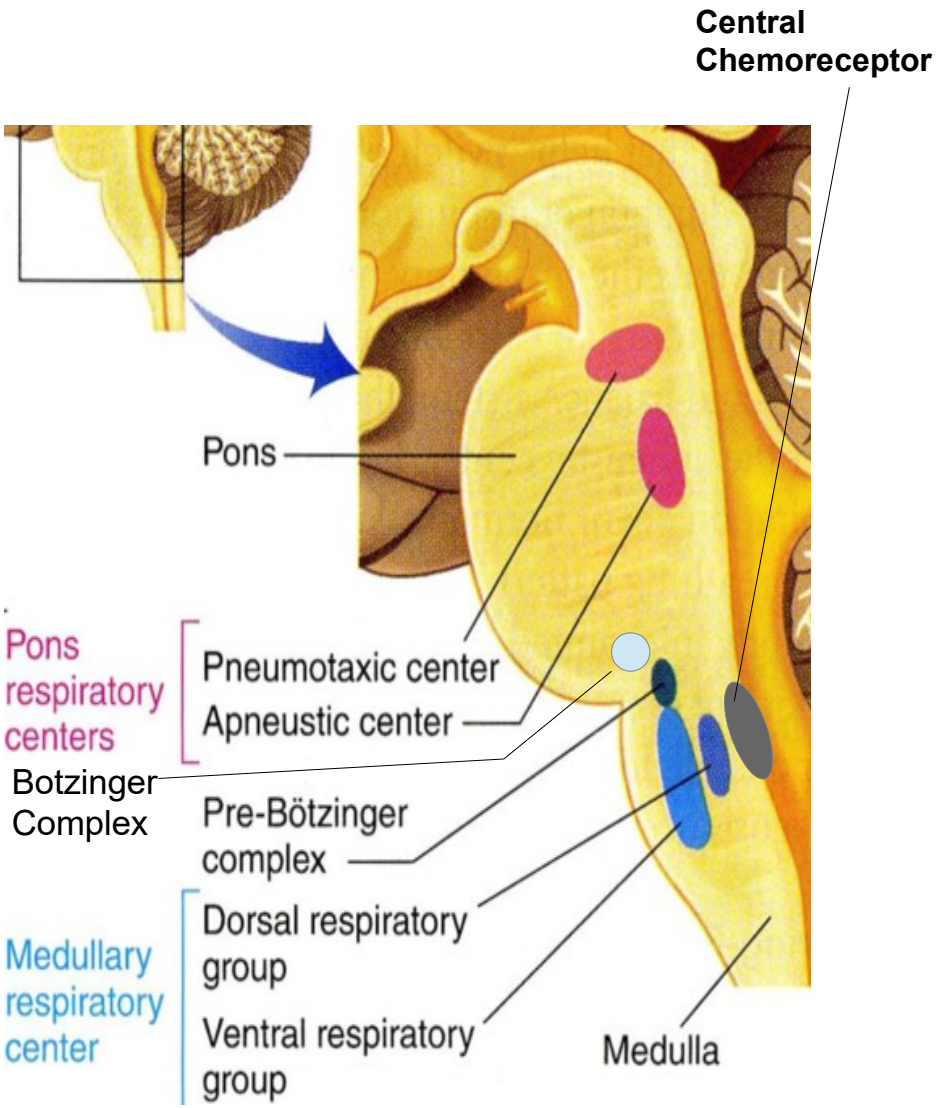
Neural Control of Breathing

Just superior to the ventral respiratory center are two more nuclei

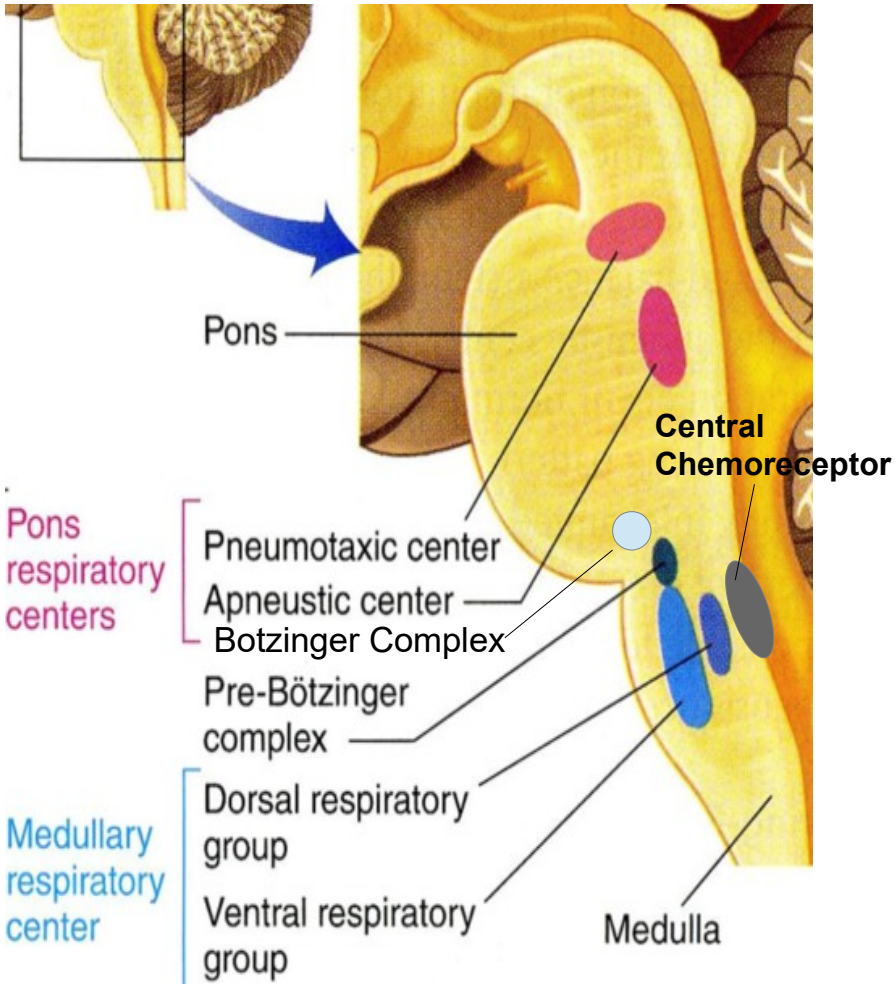
- pre-Botzinger complex
- Botzinger complex

These two nuclei influence the ventral respiratory group function.

The central chemorecepors will monitor CO2 and pH to also modify the ventral respiratory group's breathing rates.



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 leak sodium to reach threshold and depolarize to create an action potential. These nuclei are the pacemaker for breathing.

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

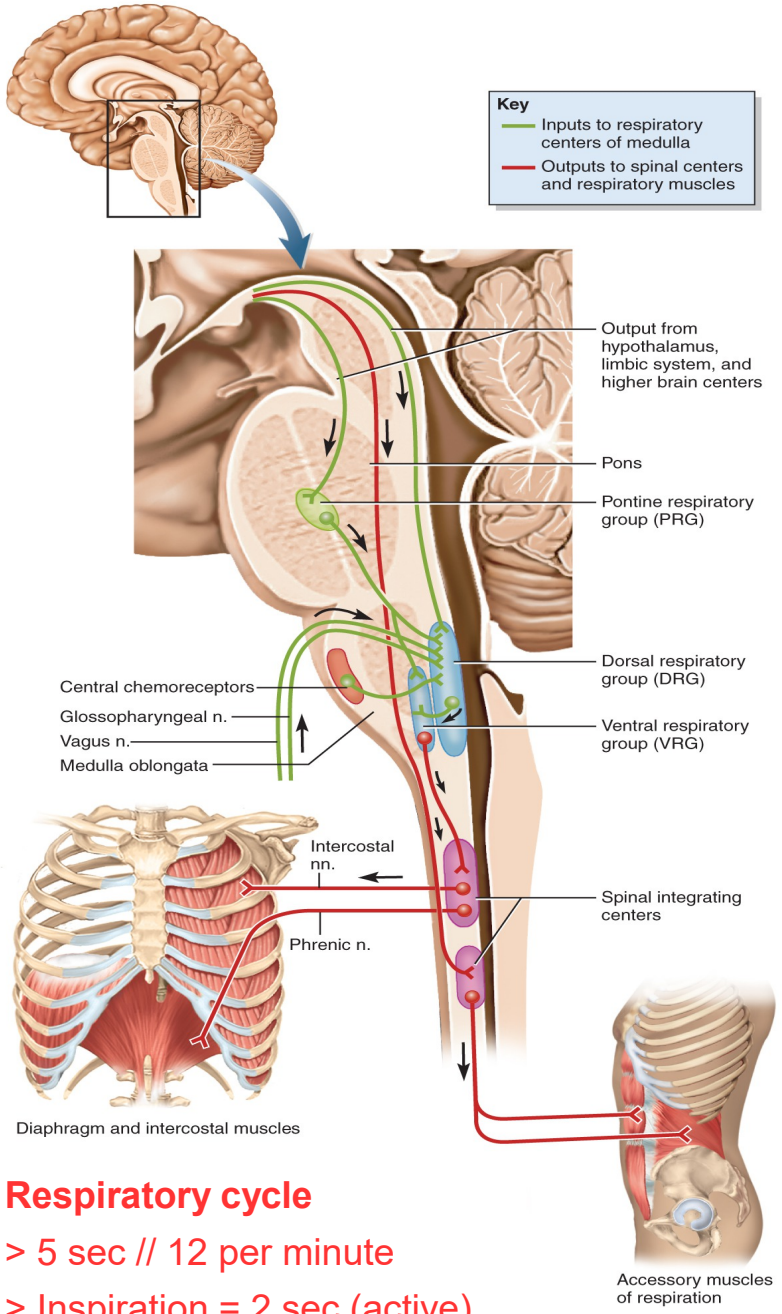
Inspiration is active and expiration is passive.

The pre-Botzinger complex influence the rate of depolarization of the VRG pacemaker.

The pre-Botzinger complex regulates 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.

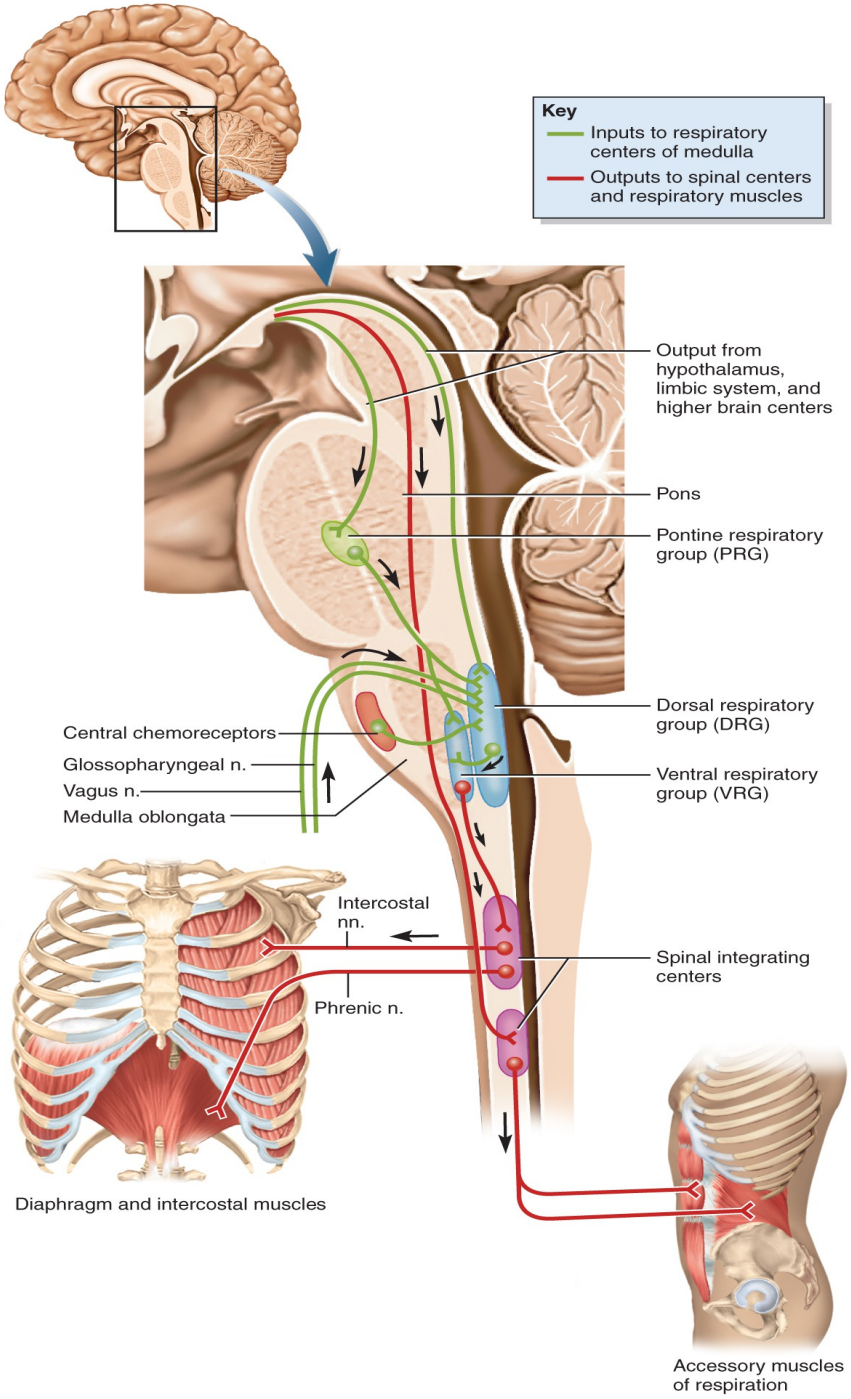
In a resting state, the phrenic nerve will cause the diaphram to contract.

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

VRG expiratory nuclei inhibit the inspiratory neuclei 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.



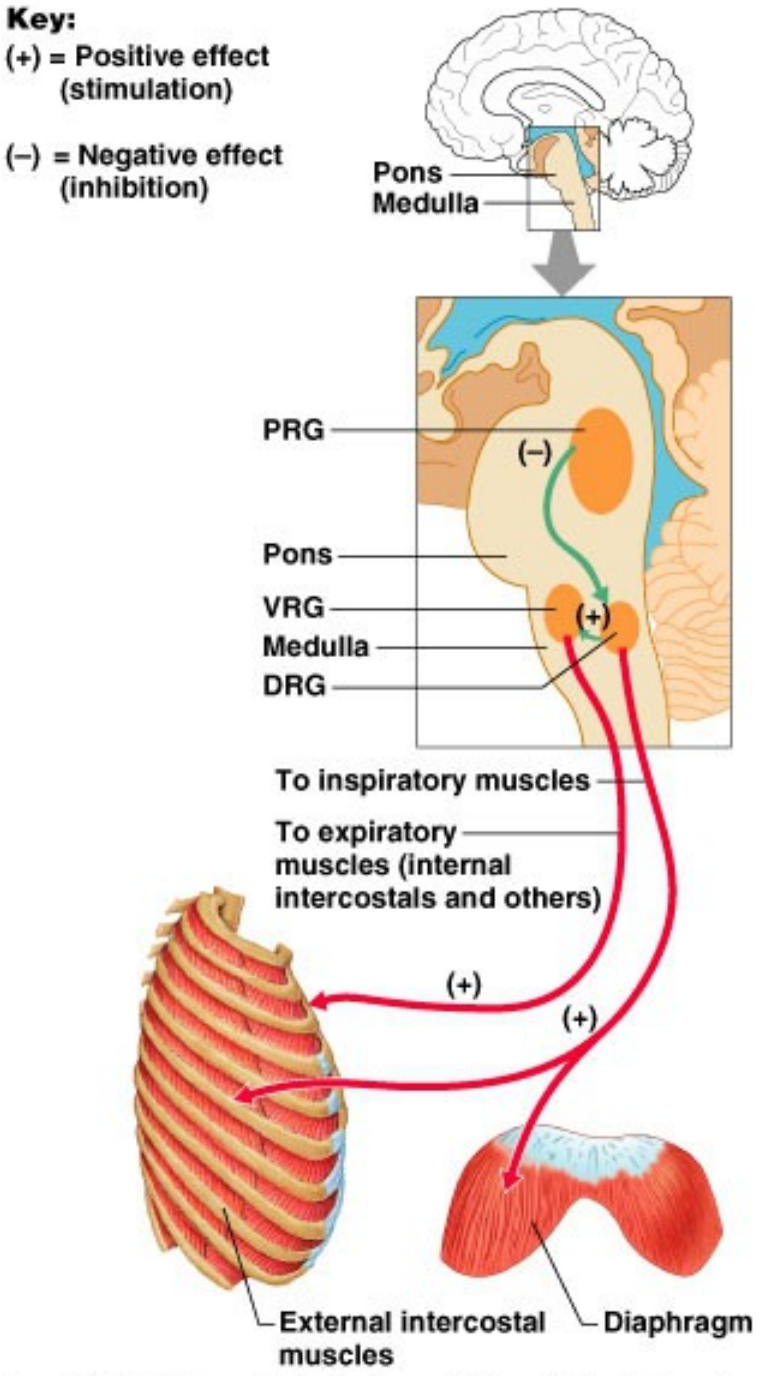
Dorsal Respiratory Groups in the Medulla

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 and continue to the intercostal muscles and the diaphragm.

Respiratory Control Centers

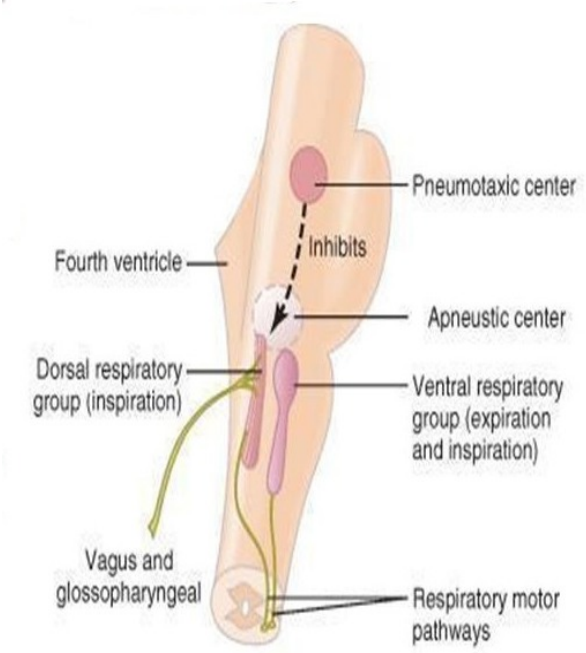


Neural Control of Breathing

Pontine Respiratory Group

- Pneumotaxic center
  - Controls transition between inspiration and expiration by inhibiting inspiration.
  - Senses expansion of lungs and allow expiration to begin
  - Role in fine tuning the respiratory rate of the VRG

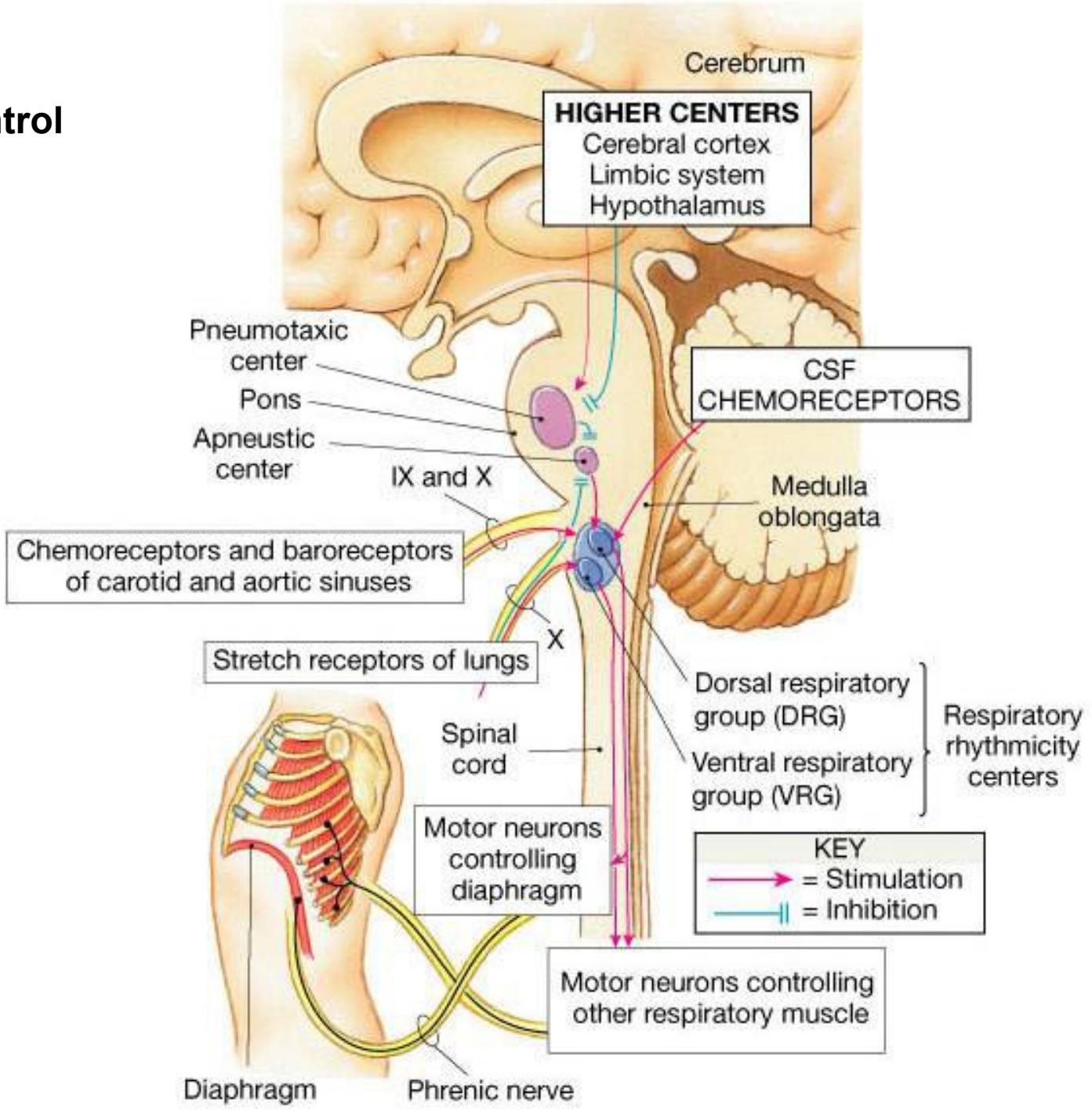
- Apneustic center
  - Sends inspiration signals to both VRG and DRG
  - If pneumotaxic center is damaged the loss of inhibition results in longer periods of inspiration



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

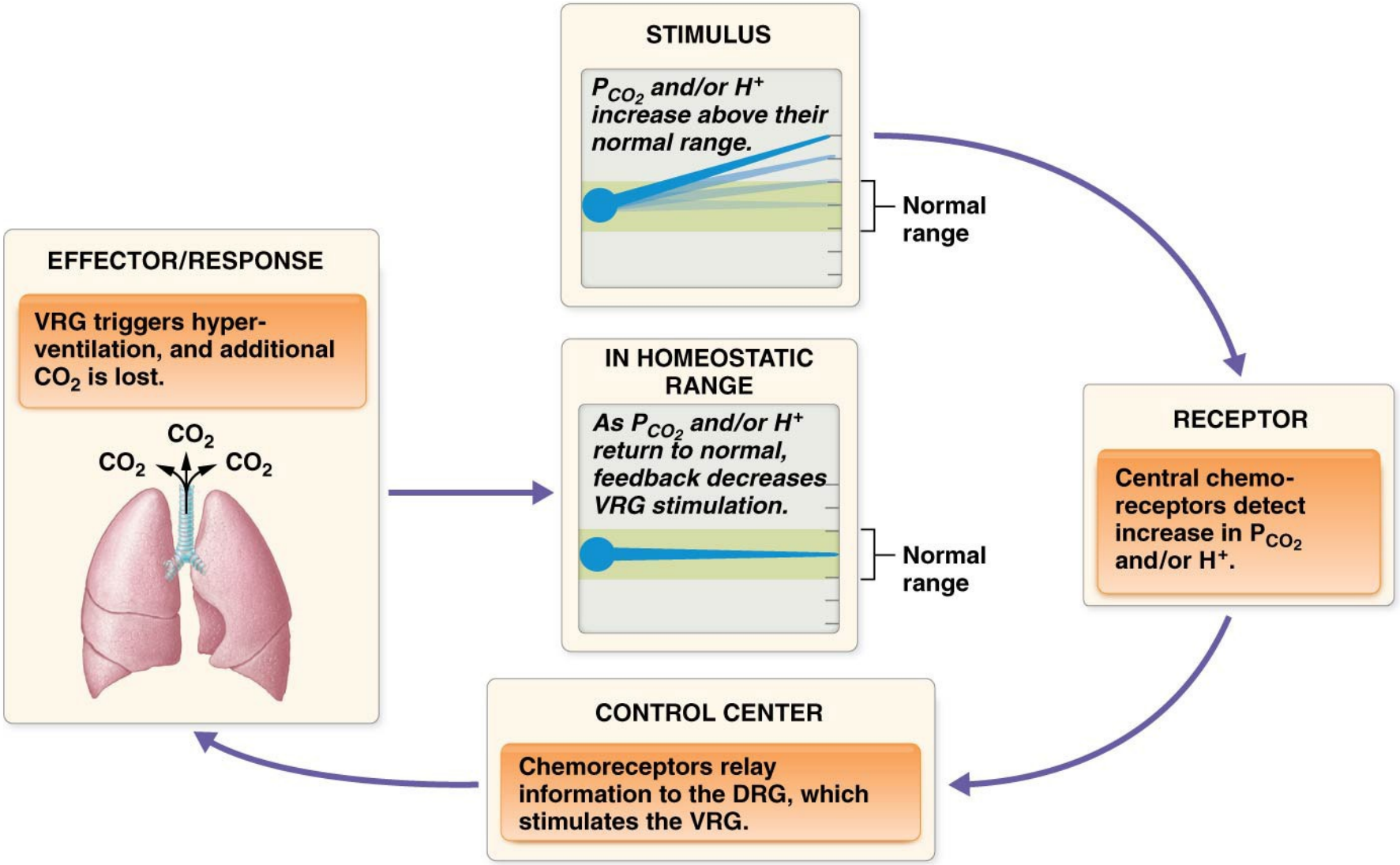


Physiologic States May Influence Neural Control of Breathing

- Many factors may regulate breathing to match our physiological state

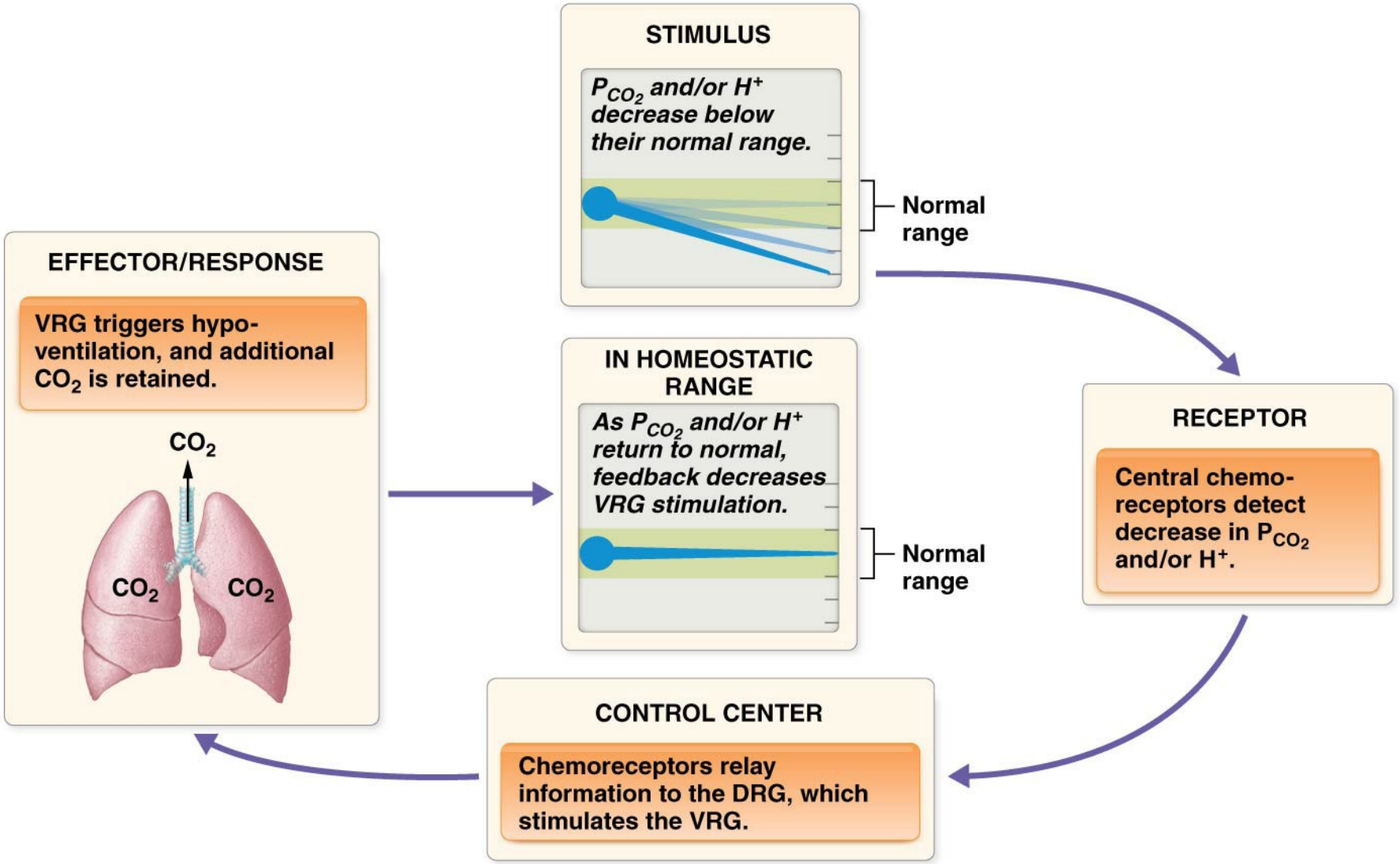
  - central chemoreceptors
  - peripheral chemoreceptors
  - proprioreceptors
  - lung stretch receptors (Hering-Breuer reflex)
  - irritant reflex receptors
  - limbic system
  - temperature
  - pain
  - stretching the anal spincter
  - blood pressure (minor)

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

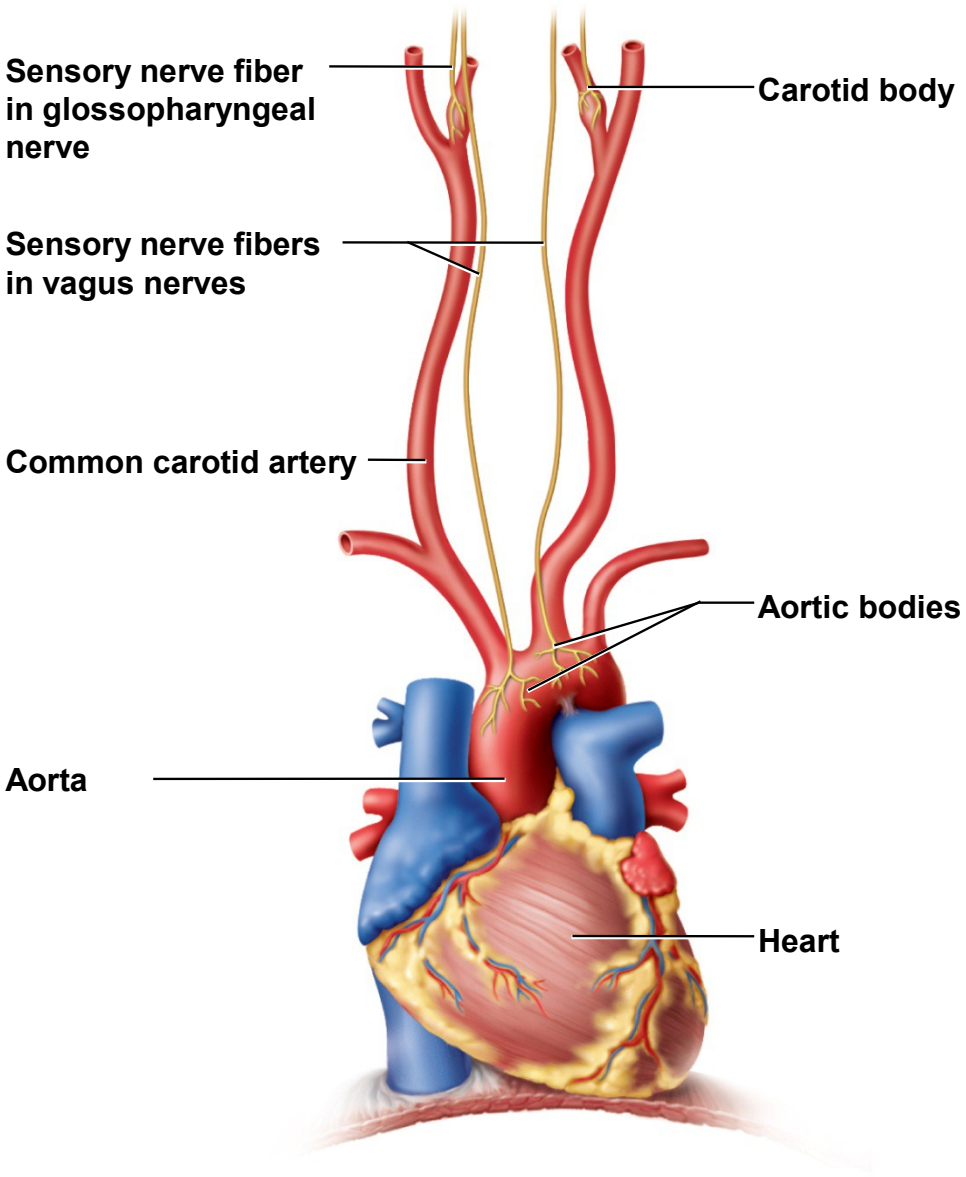
Other Respiratory Regulators

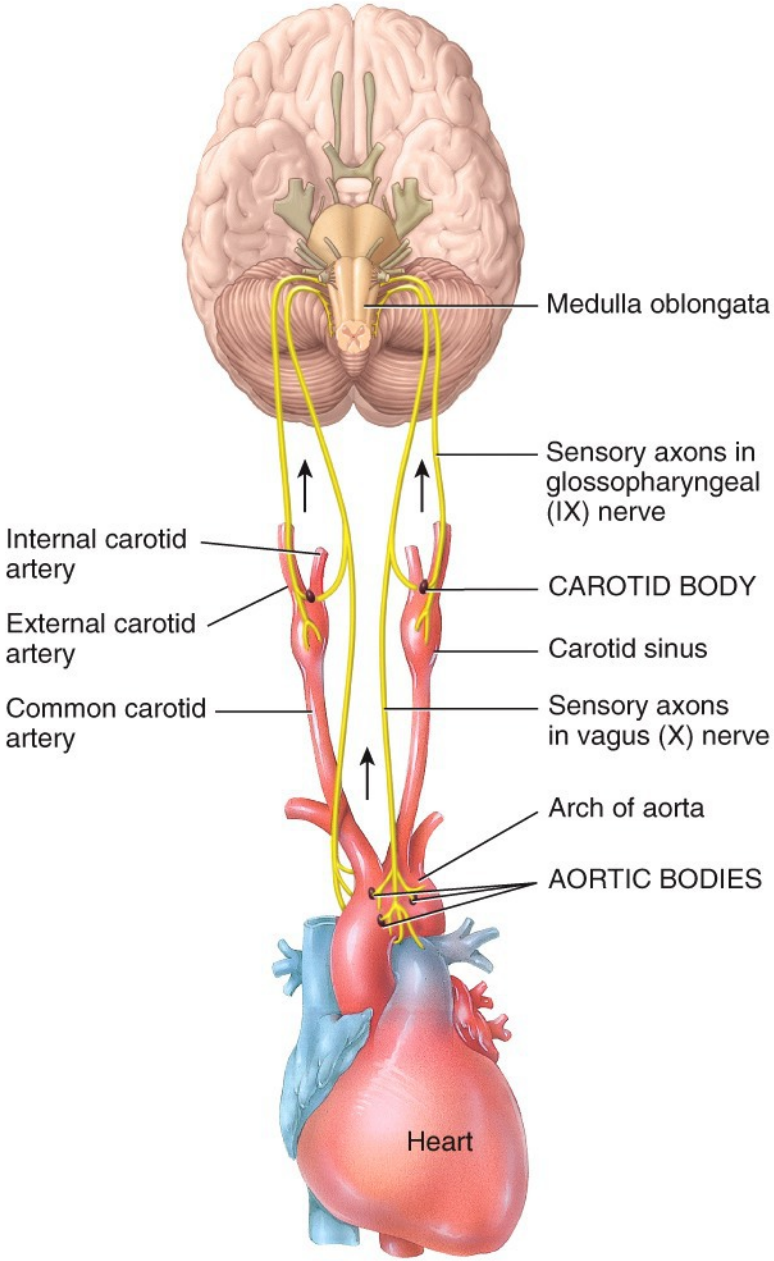
- Stretch receptors
  - found in the smooth muscles of bronchi and bronchioles, and in the visceral pleura
  - respond to inflation of the lungs
  - known as the inflation reflex or Hering-Breuer Reflex
    - triggered by excessive inflation
    - protective reflex
    - inhibits inspiratory neurons 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



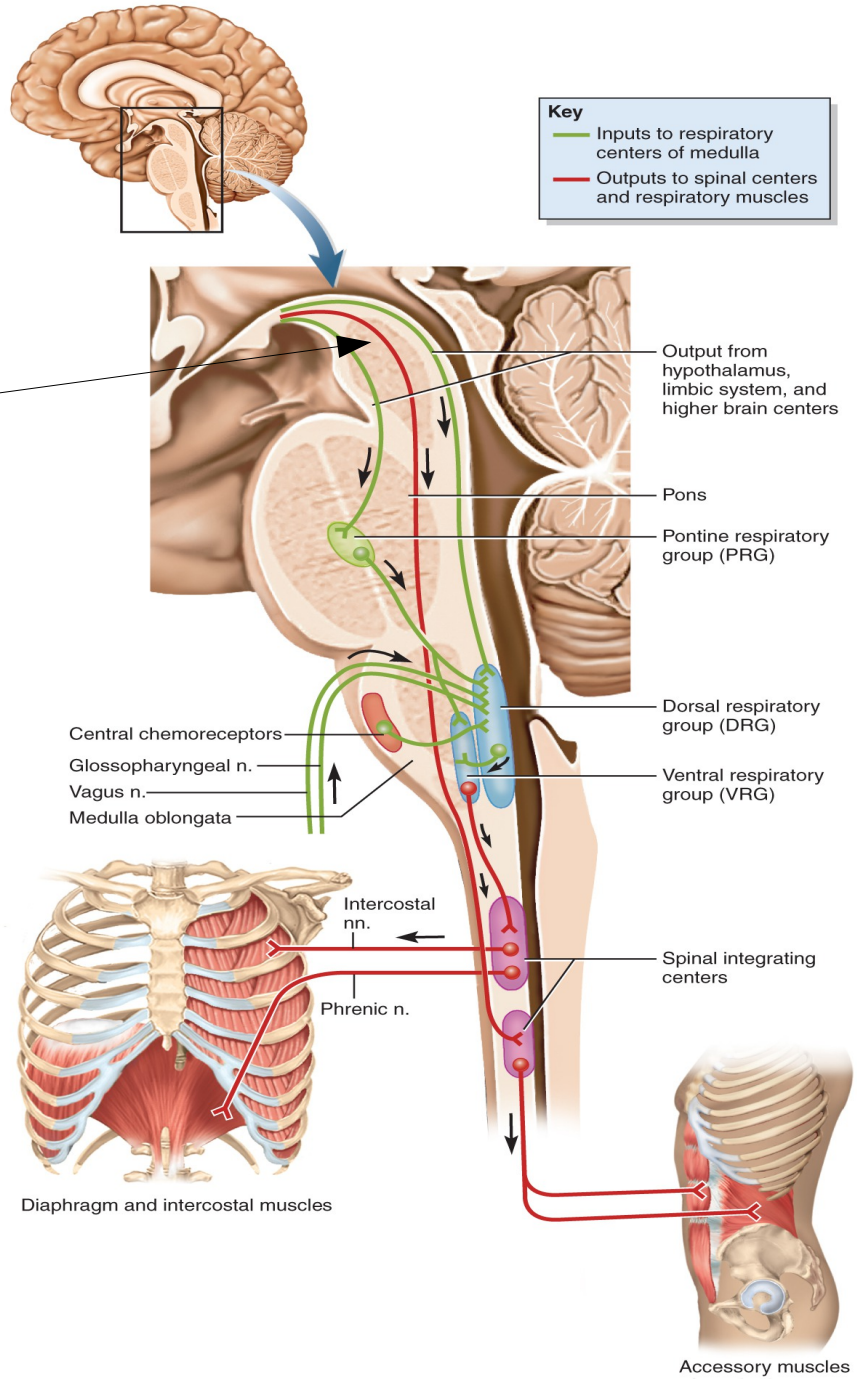

Medulla oblongata
Sensory axons in glossopharyngeal (IX) nerve
Internal carotid artery
CAROTID BODY
External carotid artery
Carotid sinus
Common carotid artery
Sensory axons in vagus (X) nerve
Arch of aorta
AORTIC BODIES
Heart

Voluntary Control May Also Modify Breathing

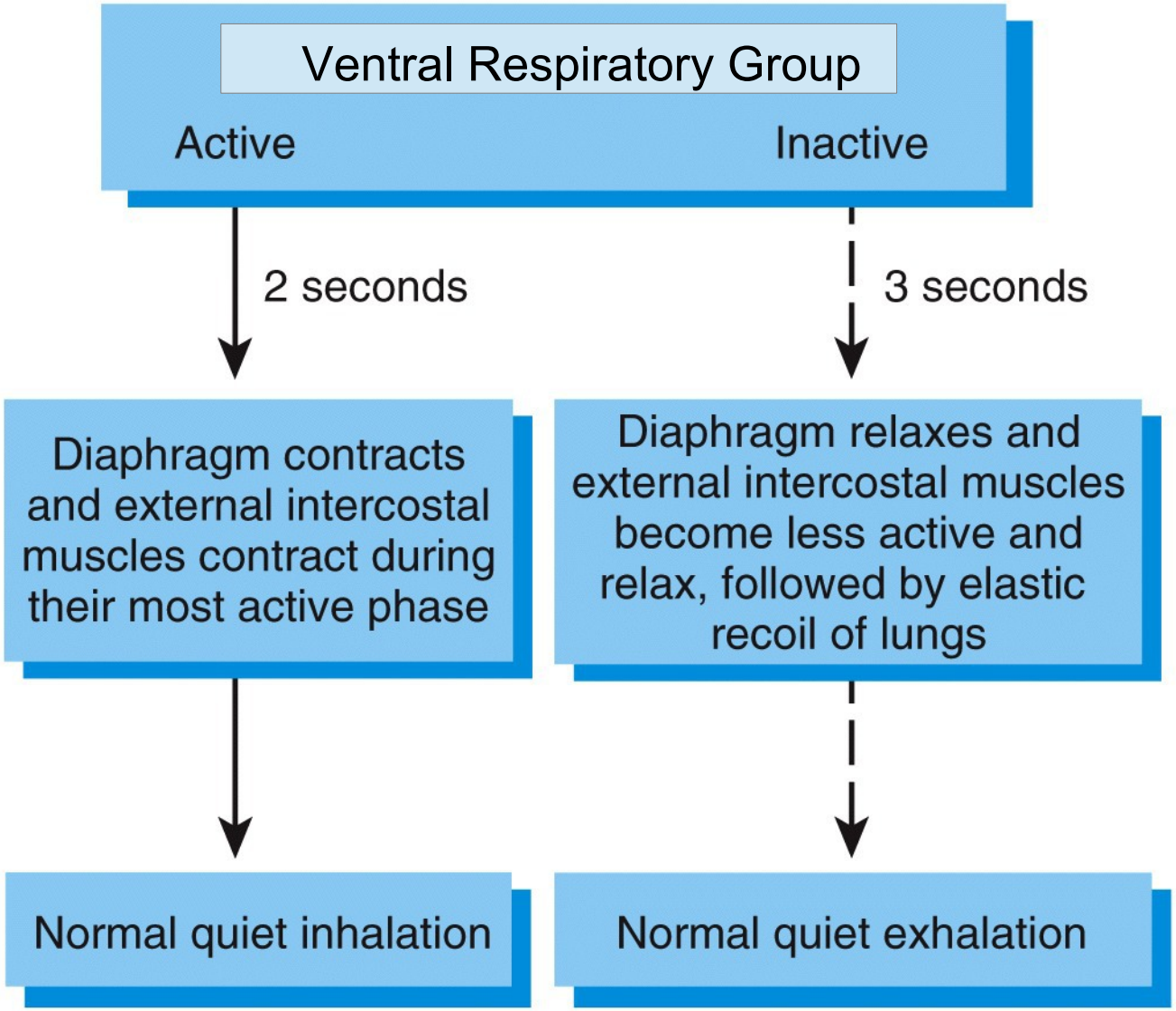
- Originates in the motor cortex of frontal lobe of cerebrum
- Descending signal sent via corticospinal tracts to respiratory neurons in spinal cord /// Note: this track bypasses brain stem regulation
- There is a limits to voluntary control /// called the breaking point // when CO_2 levels will rise to a point then the automatic controls override your will power not to breath

Voluntary Control May Also Modify Breathing

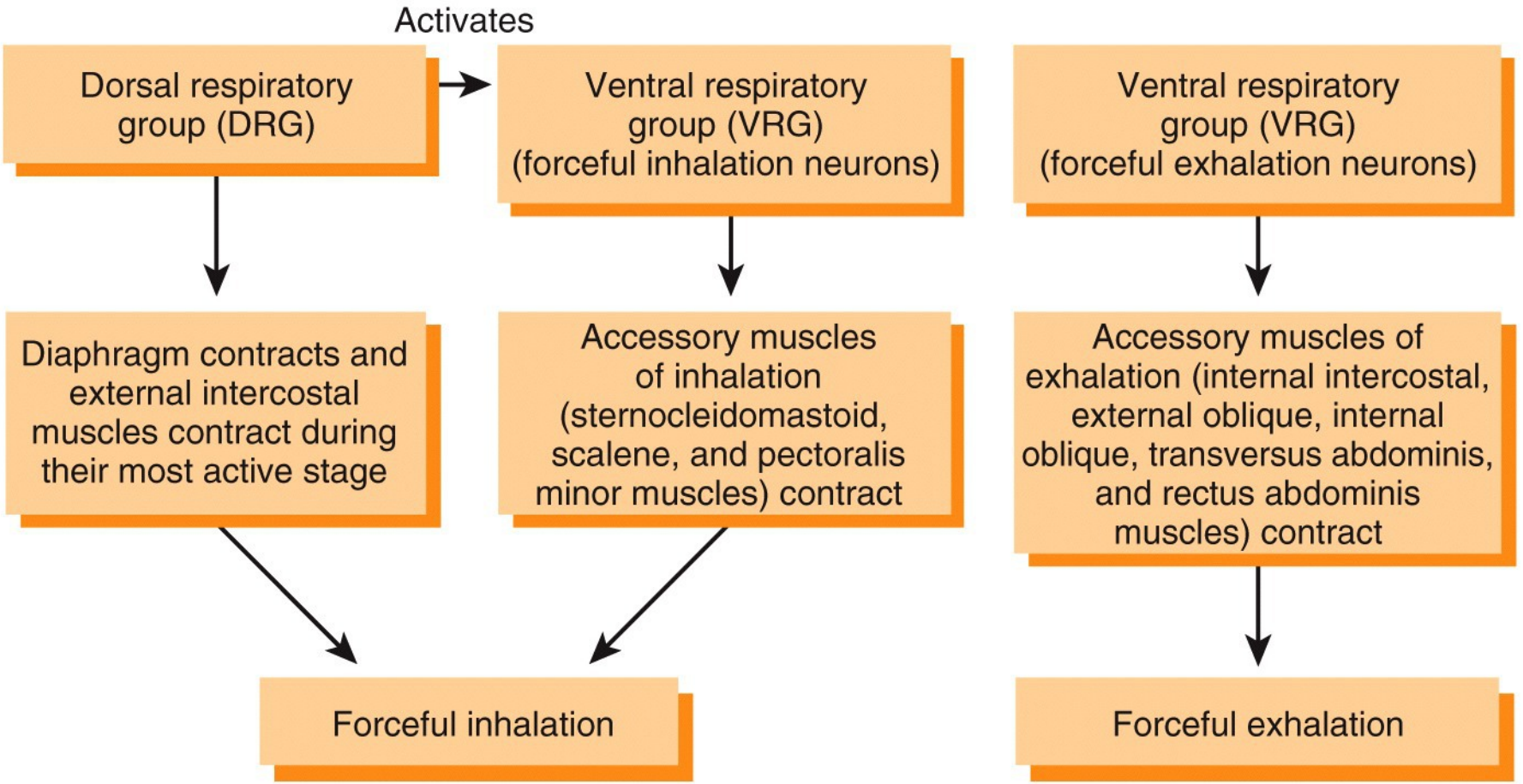
Note: this track bypasses brain stem regulation



Quiet Breathing

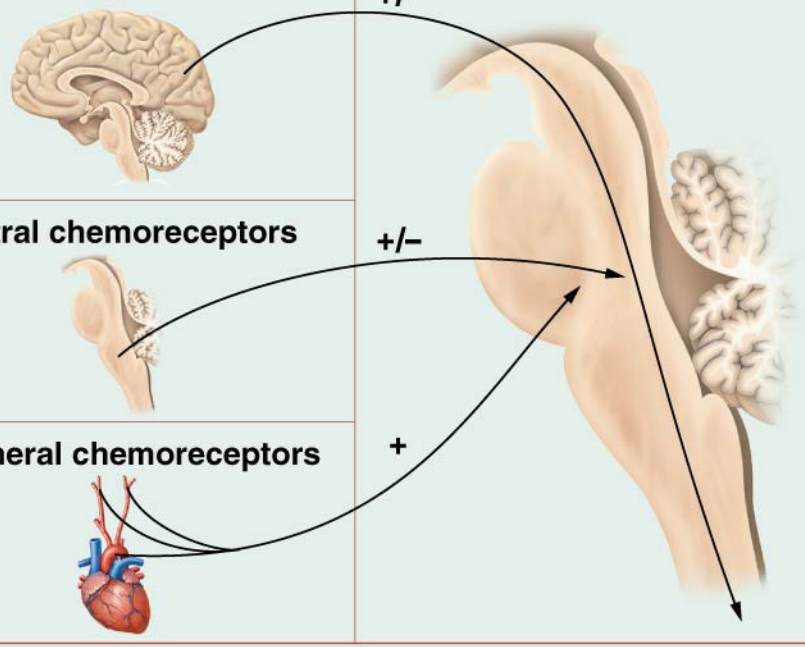


Forceful Breathing or Breathing During Exercise

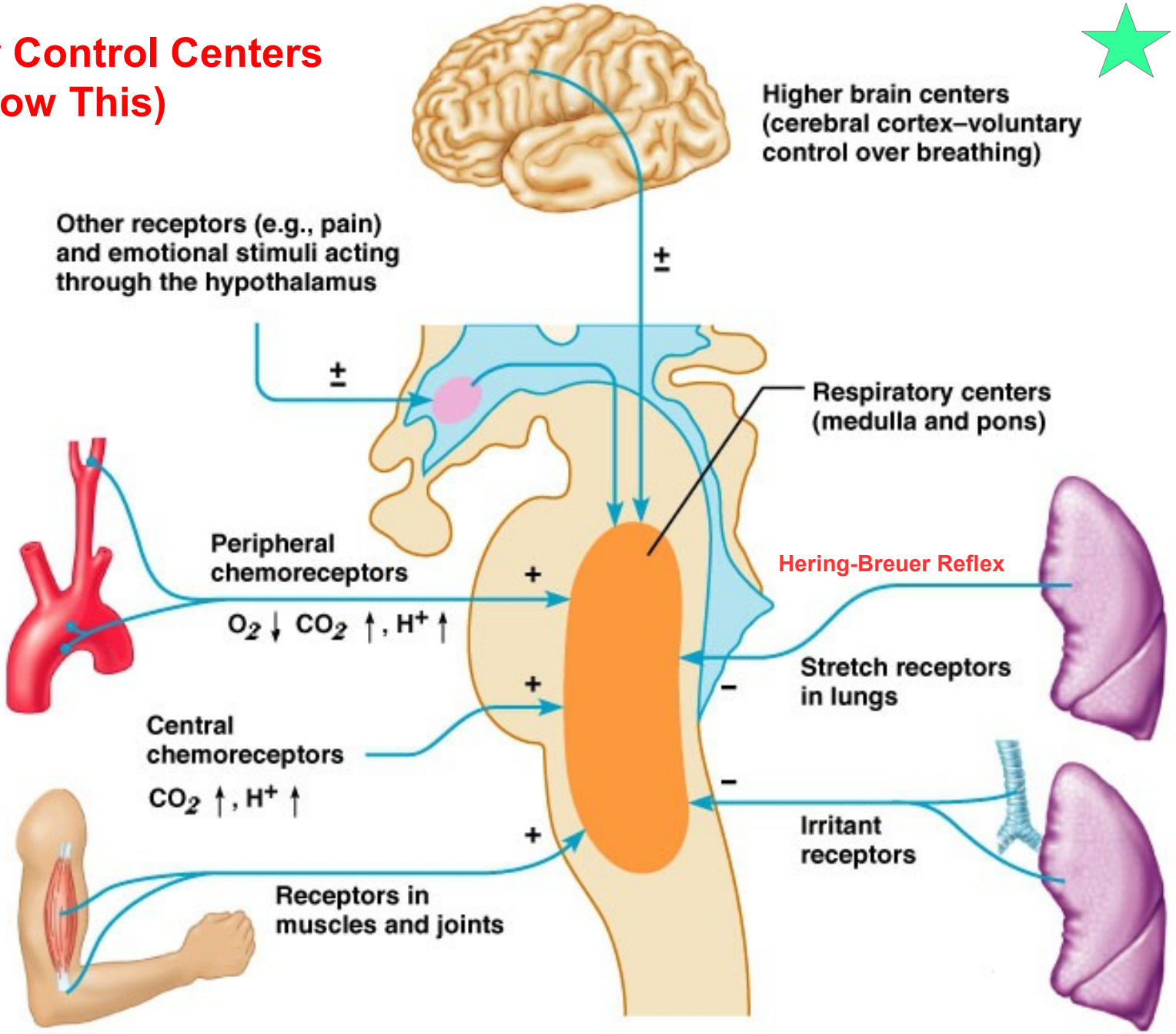


Control mechanisms of ventilation.

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



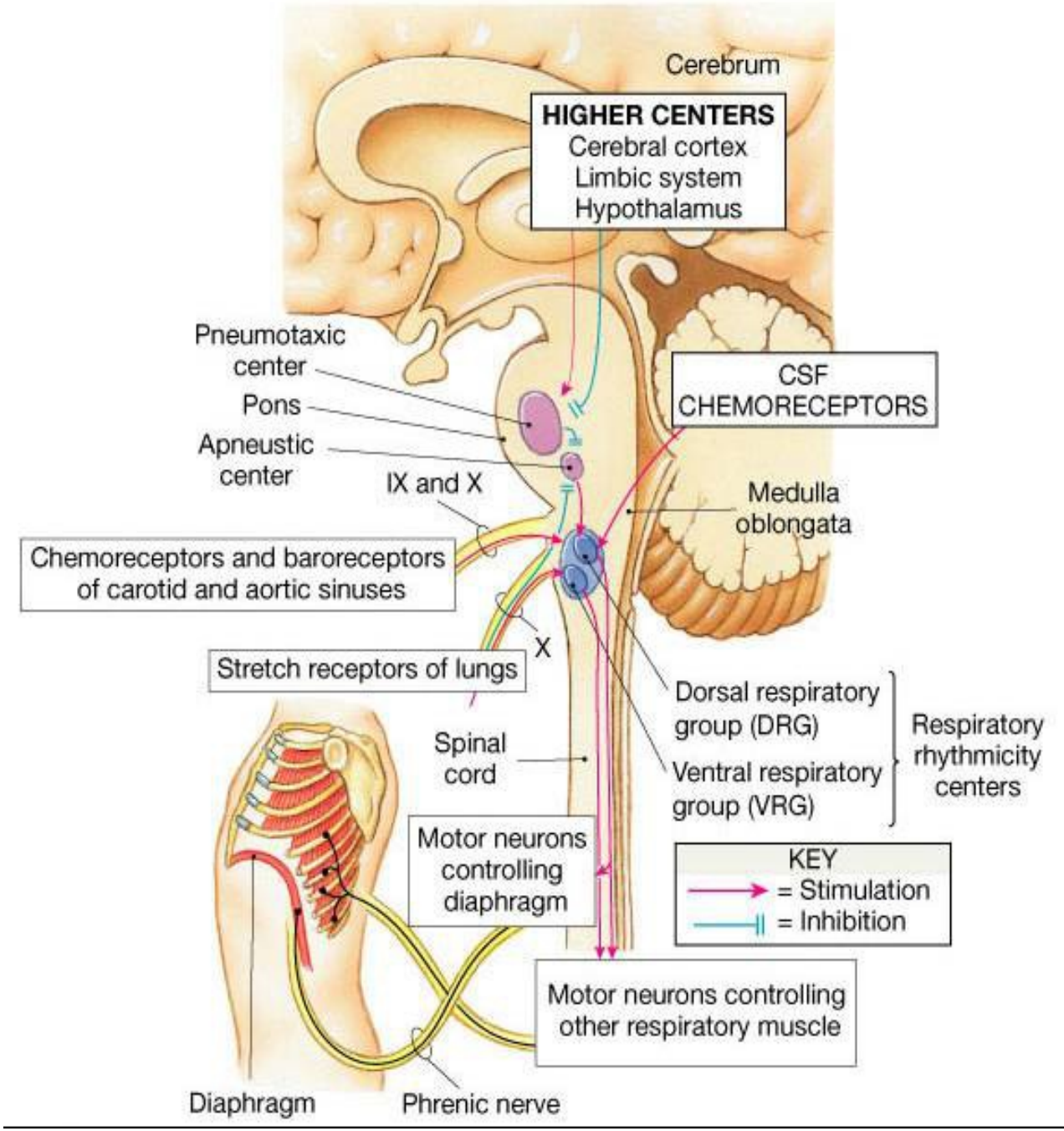
Respiratory Control Centers (Know This)

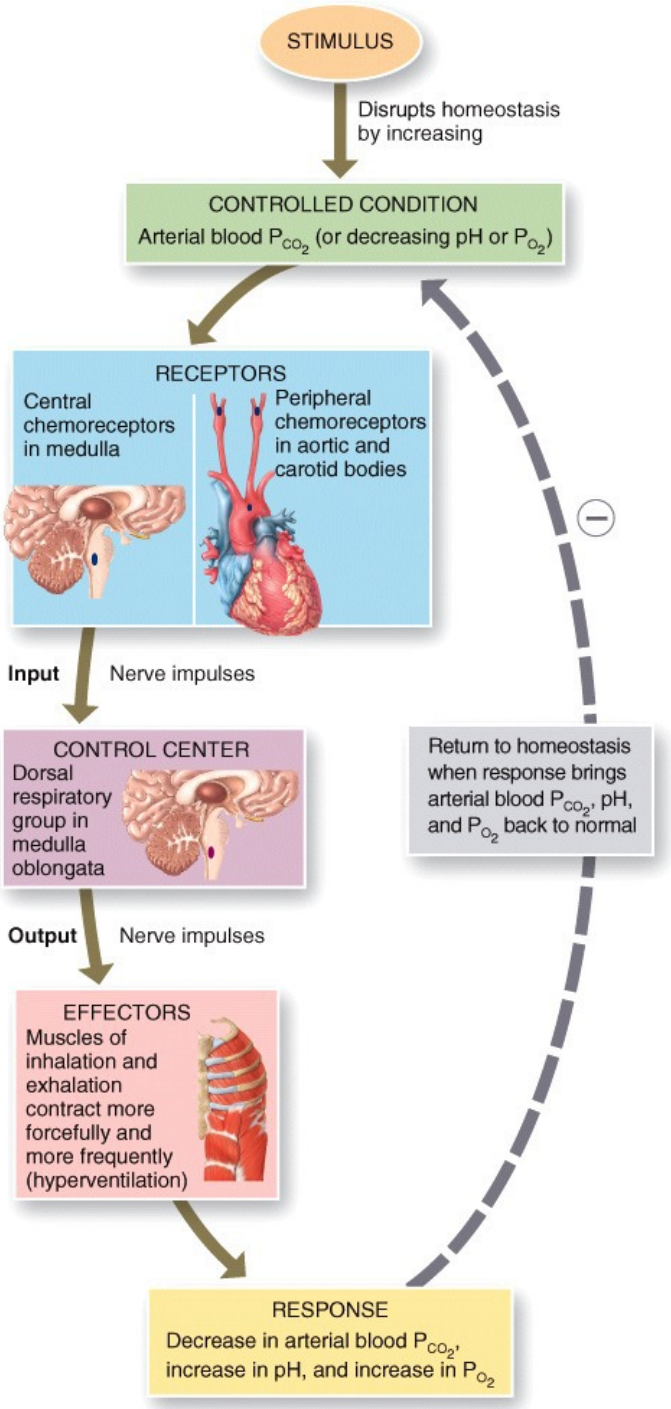


Respiratory Control Centers

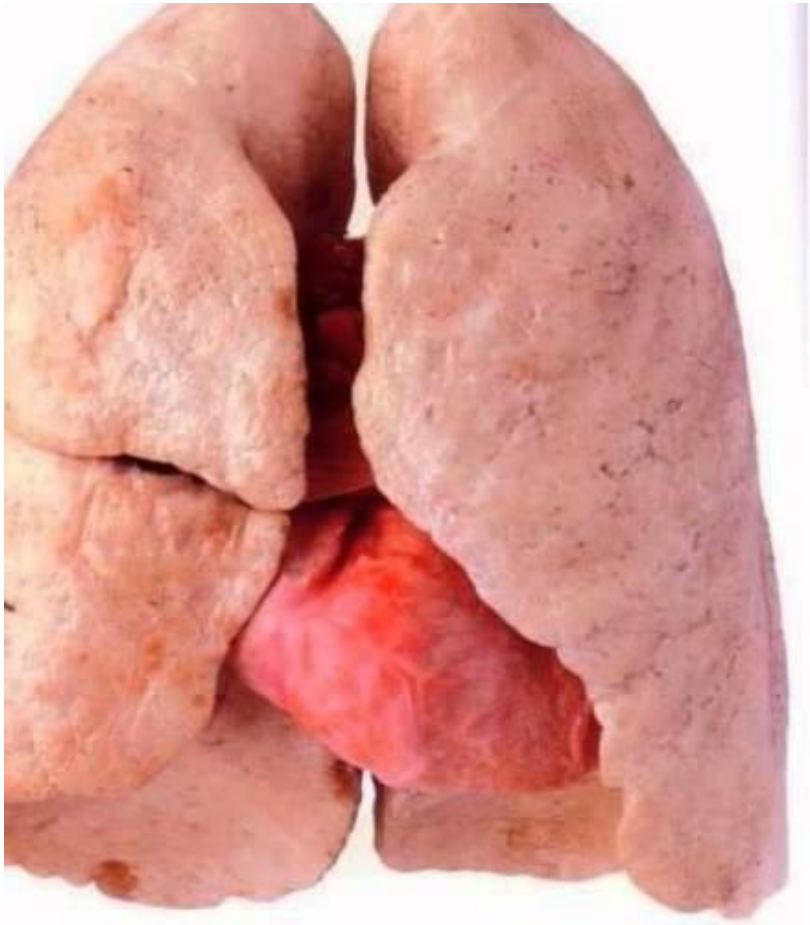
When you speak or sing the cerebral cortex must adjust breathing.

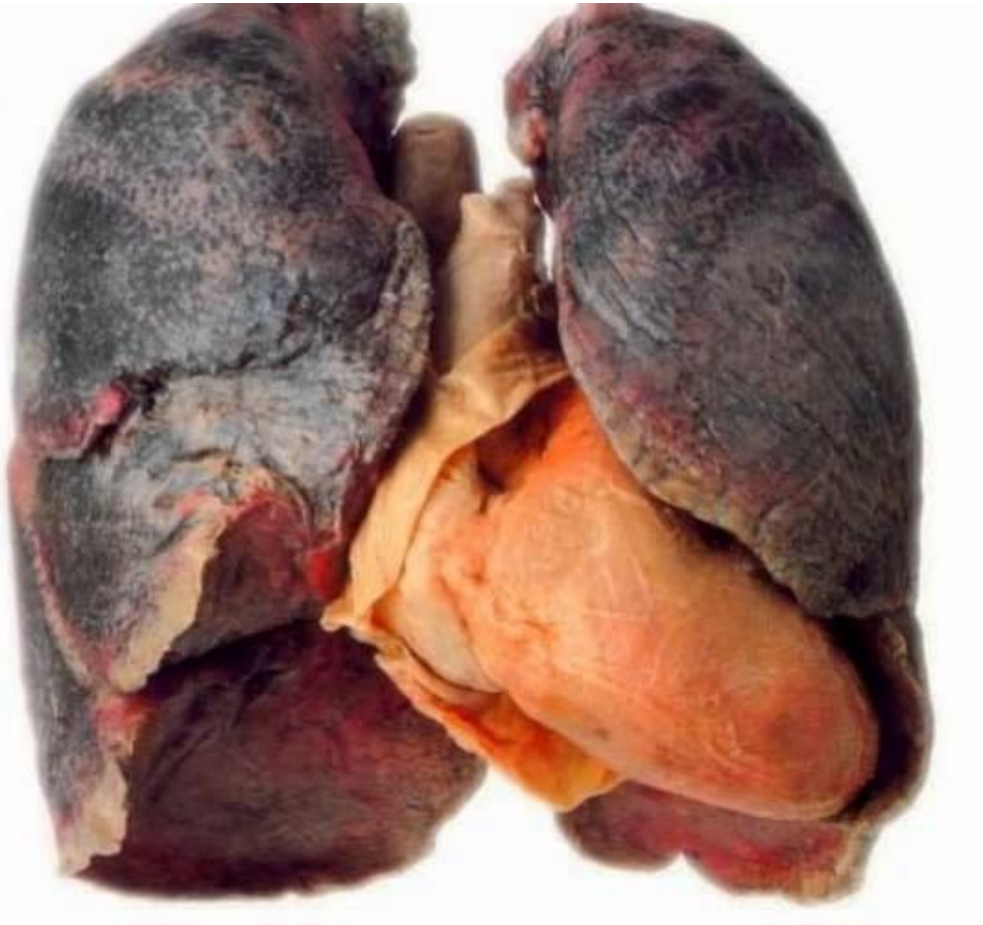
Your breathing rate will also change as you get ready to board a spaceship.




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

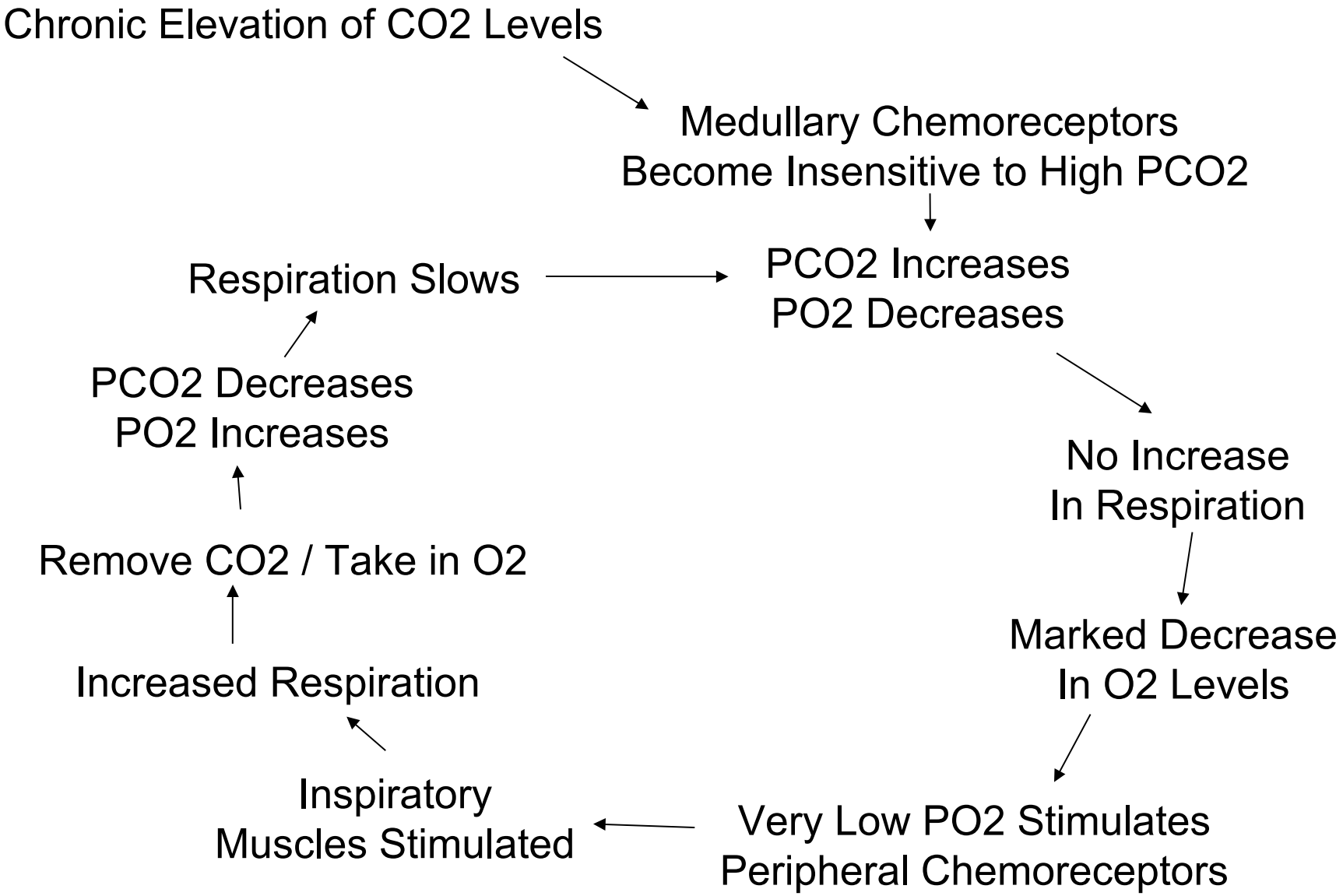
Peripheral chemoreceptors (responsible for 25% of CO_2 drive)

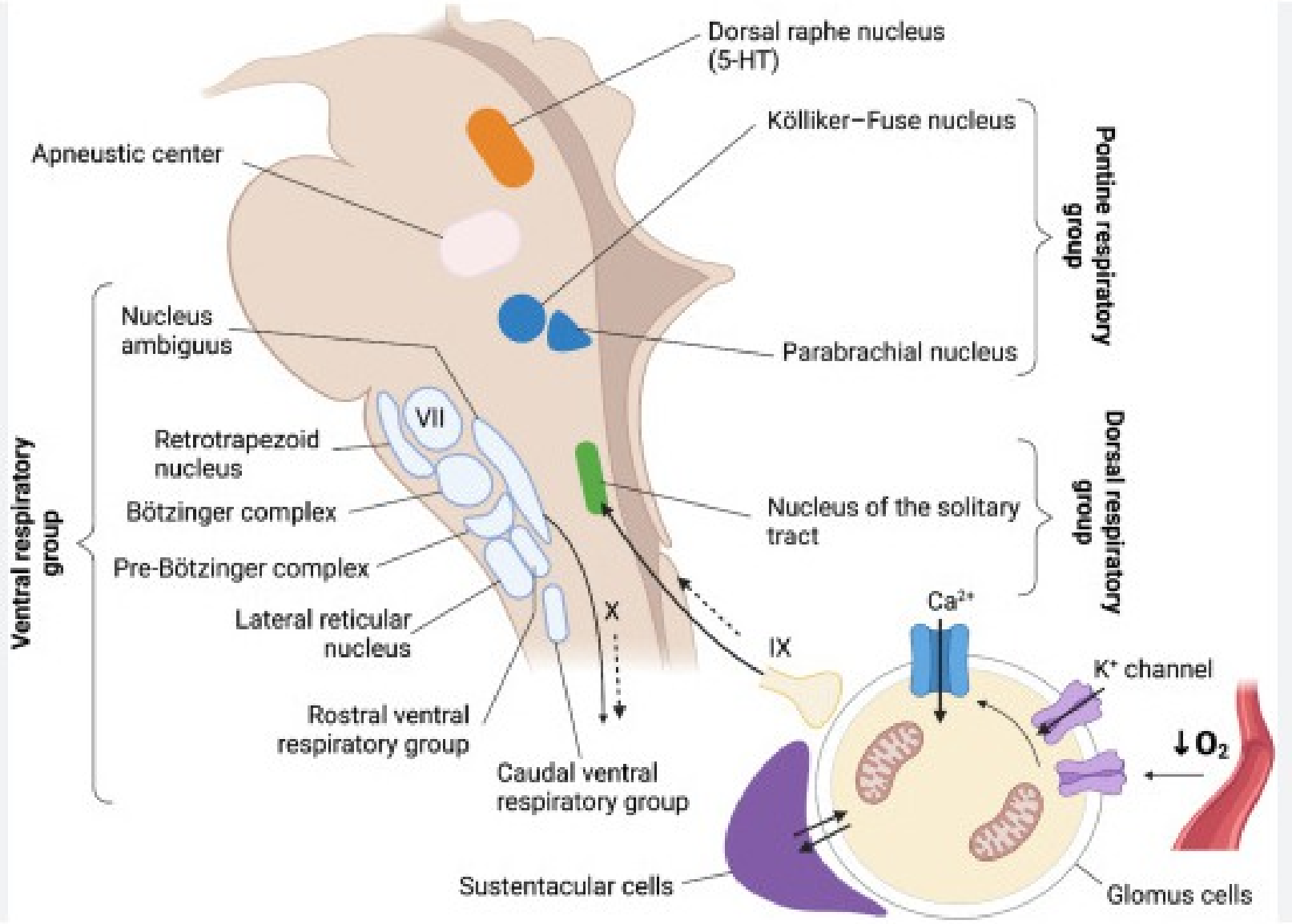
- located in the carotid and aortic bodies of the large arteries above the heart
- peripheral chemorecptors also sensitive to PCO_2
- *NOTE: PCR are also sensitive to low arterial PO_2*
  - *under normal conditions central chemoreceptors primary regulator of respiration*
  - *following chronic high CO_2 – 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