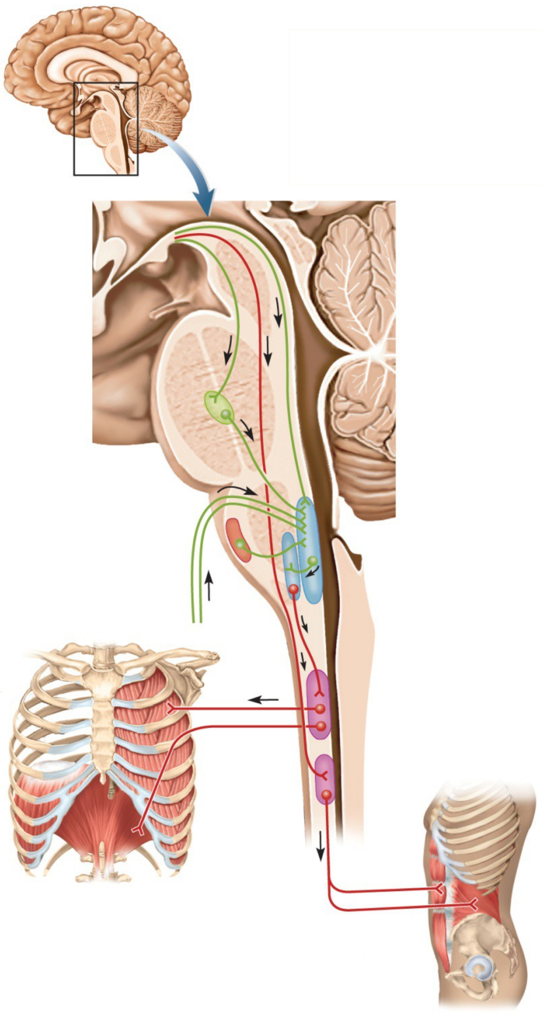
Chapter 22.2

Control of Breathing



Neural Control of Breathing

- This topic is still considered "unsettled science"

- *The exact mechanism for setting the rhythm of respiration remains unknown*

- Currently, we understand that there are three neural circuits (nuclei) within the brain stem which directly influence breathing

  - Ventral respiratory group (with Botzinger complex)
  - Dorsal respiratory group
  - Pontine respiratory group

- *Higher brain centers may also influence the dorsal respiratory group in the brain stem to further modify breathing //* cerebral cortex, limbic system, hypothalamus. // E.g. - When speaking or singing the cerebral cortex must adjust breathing rhythm

Neural Control of Breathing

Medula oblongata site for two of the respiratory control centers: Ventral and Dorsal Respiratory Groups

- Ventral respiratory group (also location for the pre-Botzinger complex)
  - primary generator of the respiratory rhythm
  - two neuron networks: (one inspiratory nuclei and one expiratory nuclei)
  - inspiratory nuclei fire first - 2 seconds – associated with inspiration // active
  - during inspiration
    - the action potentials are transmitted to “spinal integrating centers”
    - phrenic nerve cause diaphram to contract // increase lung volume
    - intercostal nerve cause external intercostal muscles to contract // increase lung volume
  - At same time the inspiratory neurons inhibit the expiratory nuclei for 2 seconds

Neural Control of Breathing

- expiratory nuclei (second nuclei) will produce action potential for 3 seconds
  - Action potential inhibis inspiratory nuclei, diaphragm, and external intercostal muscles
  - *allows for passive elastic recoil to occur* and as lung's recoil air is forced out of the lungs
  - respiratory cycle = 5 seconds // 12 per minute
    - Inspiration = 2 seconds
    - Expiration = 3 seconds

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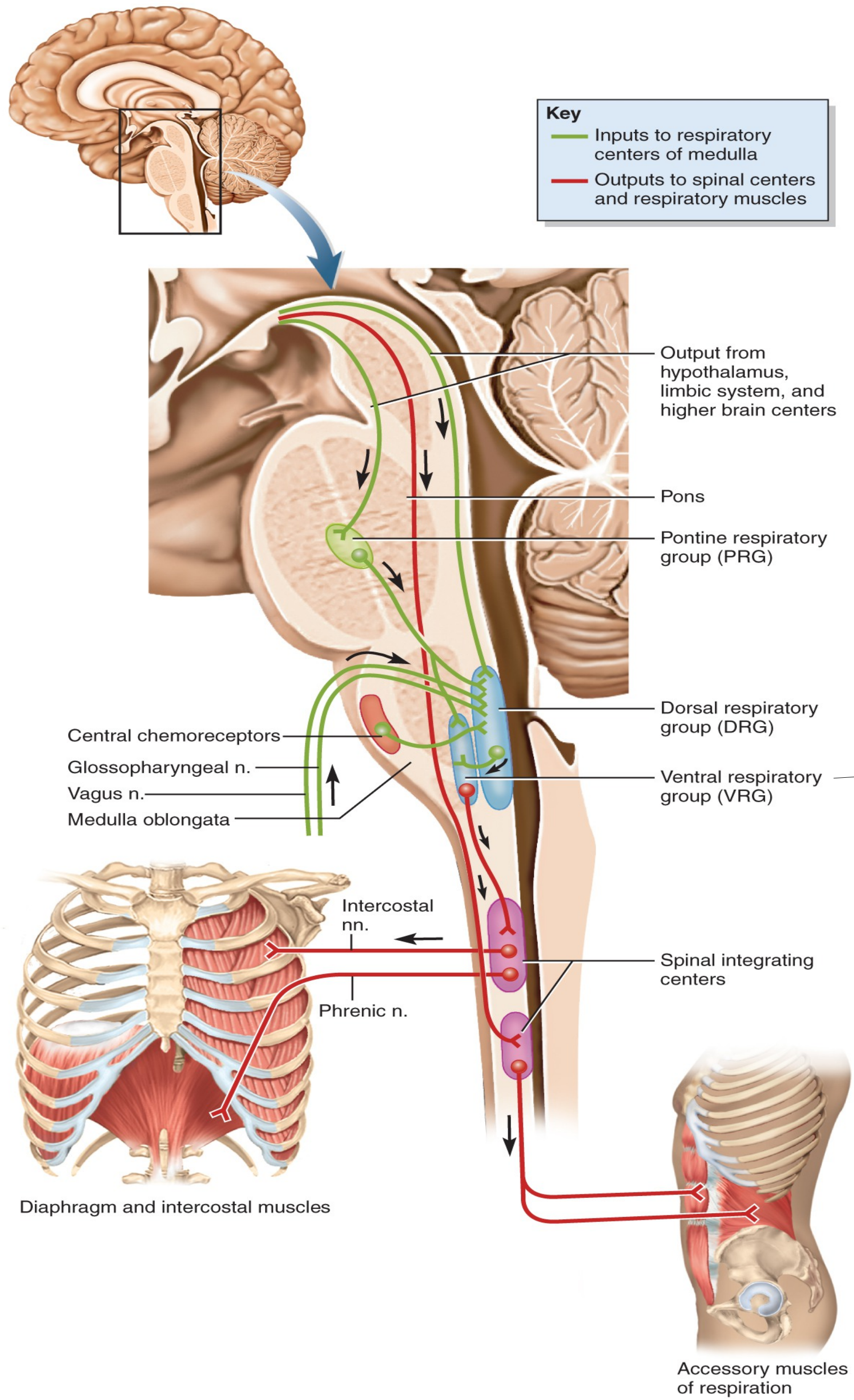

Neural control of the basic pattern of ventilation.

Two neuron networks in the Ventral Respiratory Group set respiratory rhythm.

First neuron network fires for two seconds which result in inspiration.

Second neuron network fires for three seconds which inhibit inspiratory neurons.

This initiates expiration via passive recoil of the lung's elastic fibers in connective tissue

Neural Control of Breathing

- Dorsal respiratory group
    - this is an integrating center
    - receives action potentials from the pons, chemosensitive areas of the medulla, chemoreceptors from major arteries, irritant receptors in lungs, and higher brain centers (i.e. emotions).
    - modify basic respiratory rhythm set by VRG
    - allows for breathing to be faster, slower, shallower, deeper.

Pons Also May Influence Respiratory Control Center

- Pontine respiratory group (PRG) /// nuclei that sends action potentials to dorsal respiratory group

  - modifies rhythm by sending signals to both the VRG and DRG

  - adapts breathing to special circumstances such as sleep, exercise, vocalization, and emotional responses

  - also receives input from limbic system and cerebrum.

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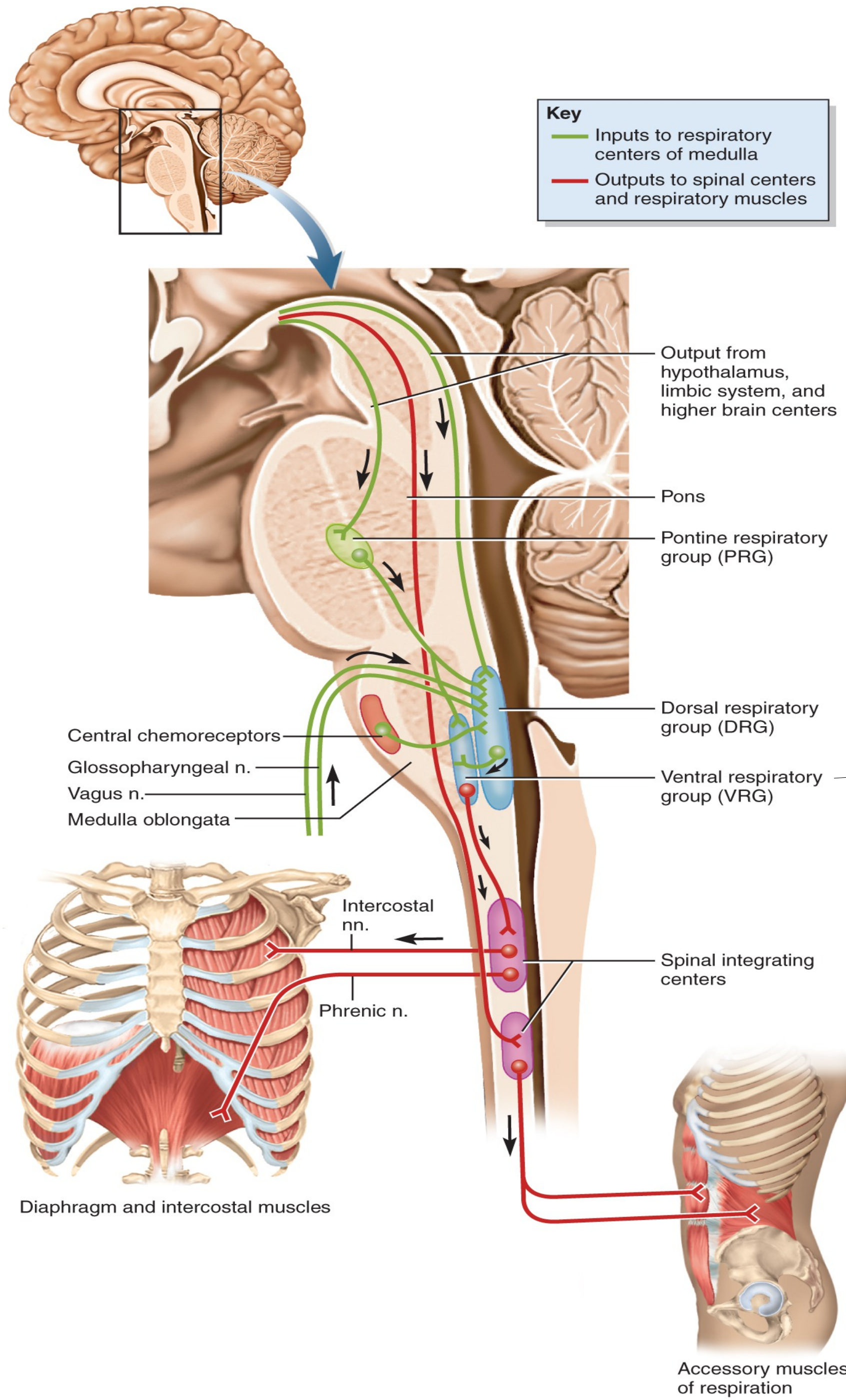

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DRG integrates afferent stimulus to modify VRG cycle.

Neural Control of Breathing

- Many factors -- used to regulate breathing to our physiological state

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

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)

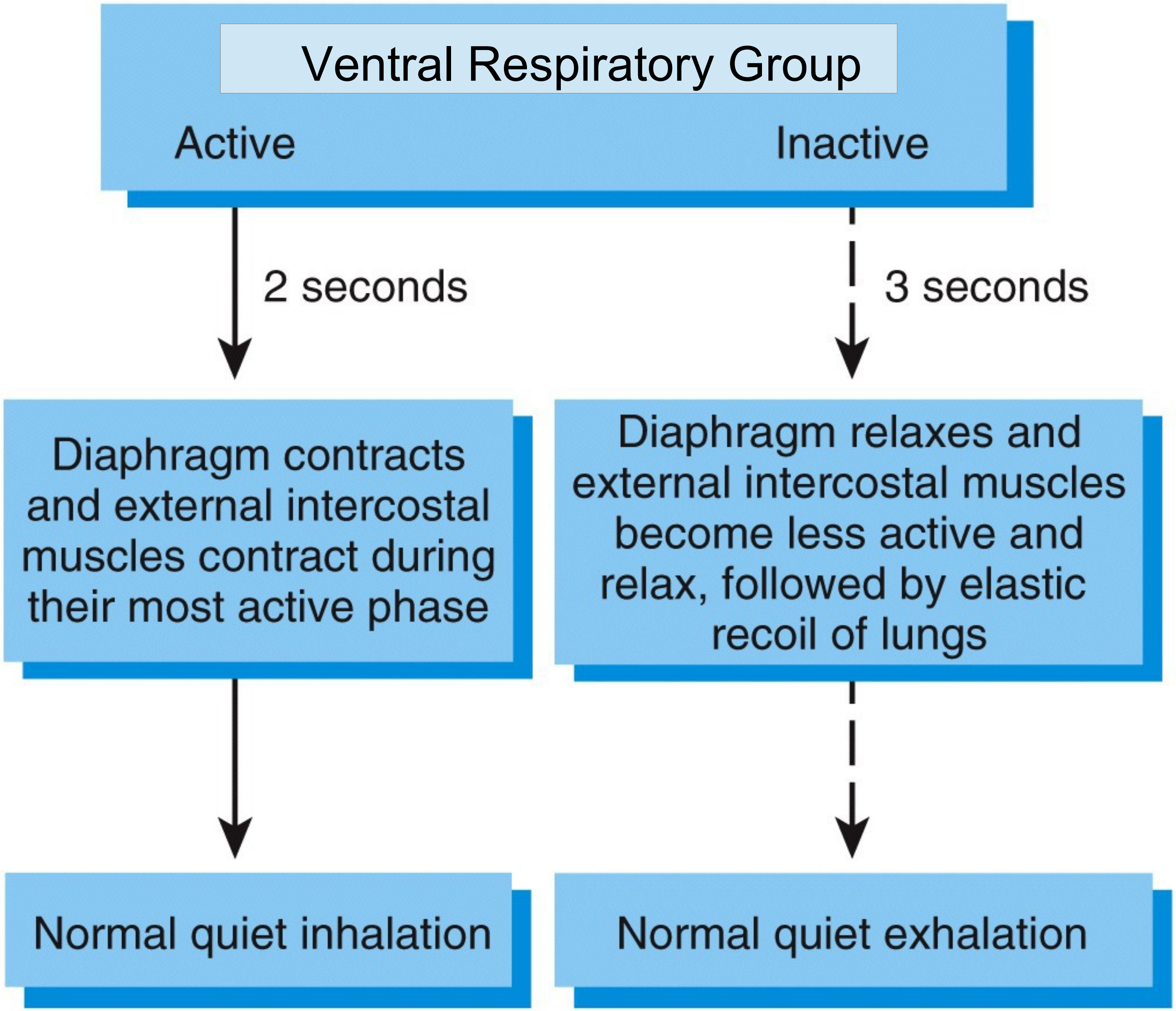
- located in the carotid and aortic bodies of the large arteries above the heart
- peripheral chemorecptors also sensitive to PCO_2
- *NOTE: PCR also sensitive to low arterial PO_2*
  - *under normal conditions central chemoreceptors primary regulator of respiration*
  - *following chronic high CO_2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

Voluntary Control of Breathing

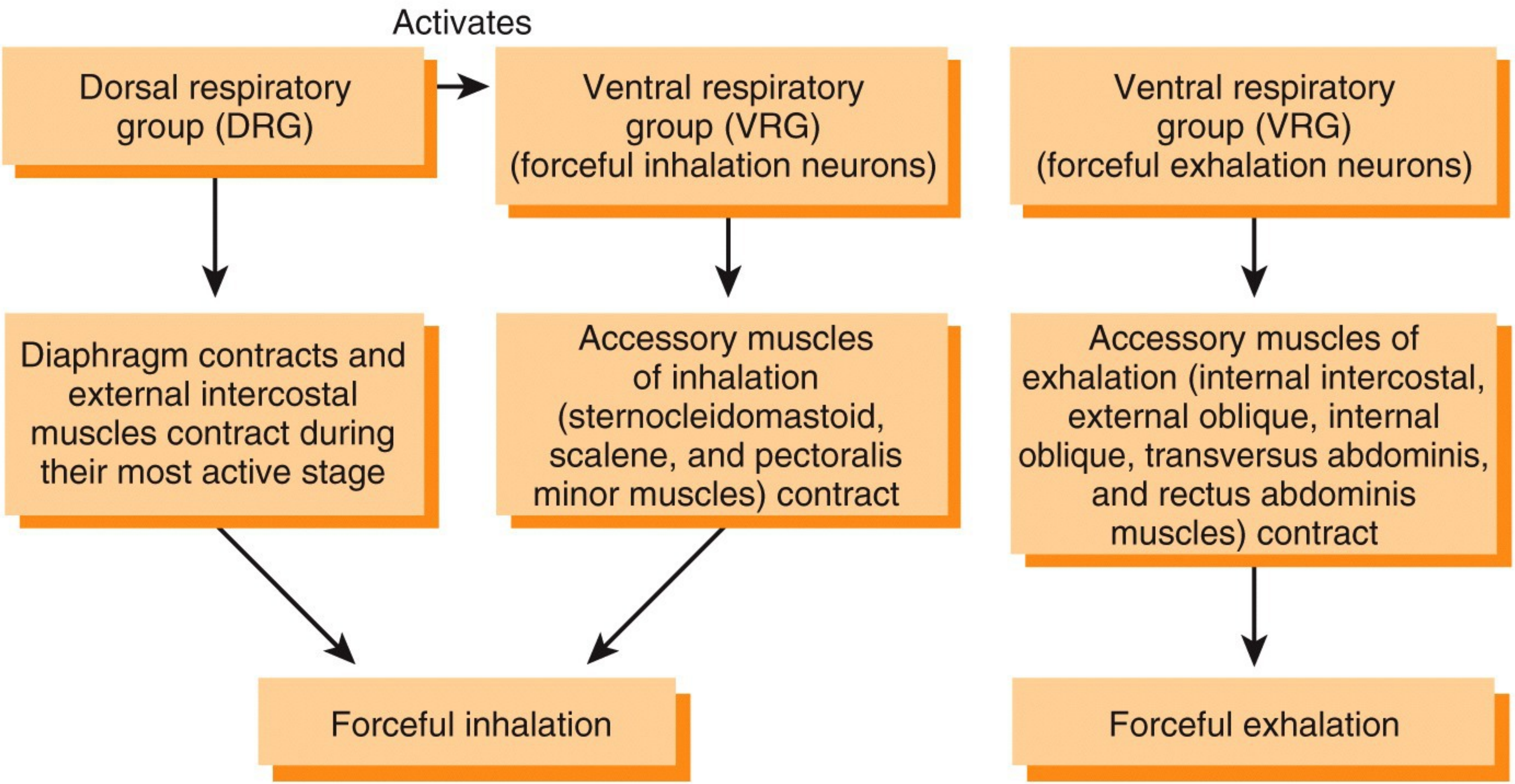
- Originates in the motor cortex of frontal lobe of cerebrum /// descending signal sent via corticospinal tracts to respiratory neurons in spinal cord /// Note: this track bypasses brain stem regulation

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

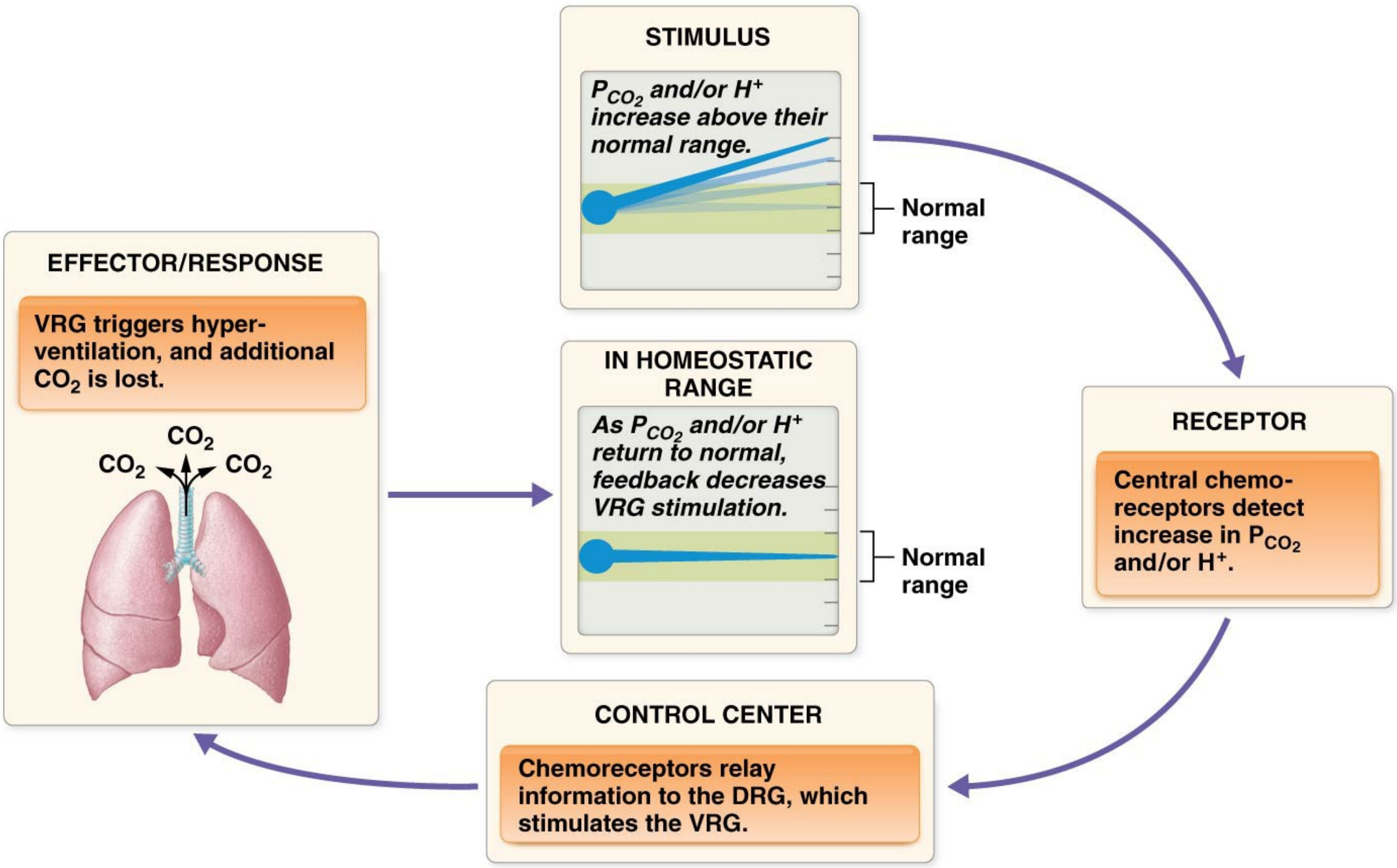
Quiet Breathing (Know This)



Forceful Breathing or Breathing During Exercise

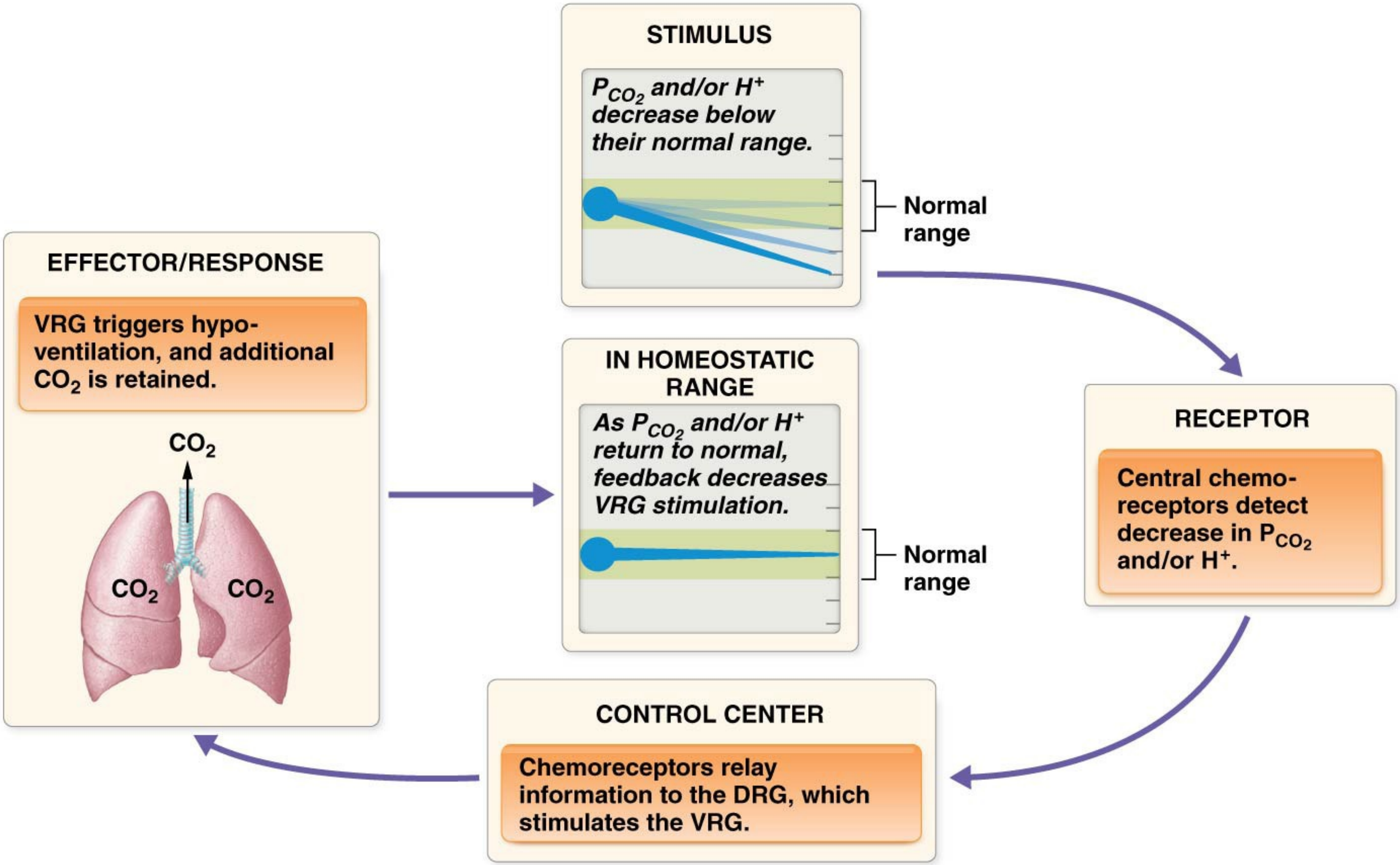


Role of the central chemoreceptors in regulation of blood pH via breathing rate.



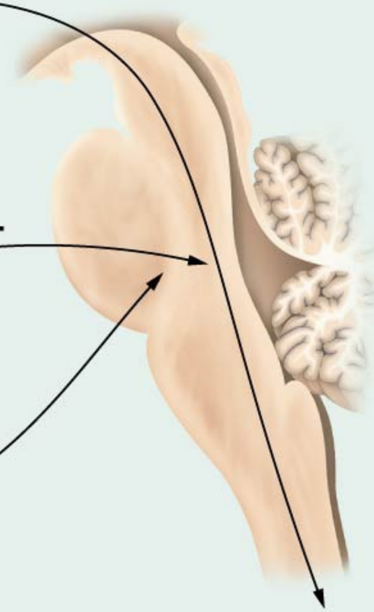
(a) Response to increased arterial P_{CO_2} and/or H^+ concentration by a negative feedback loop

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

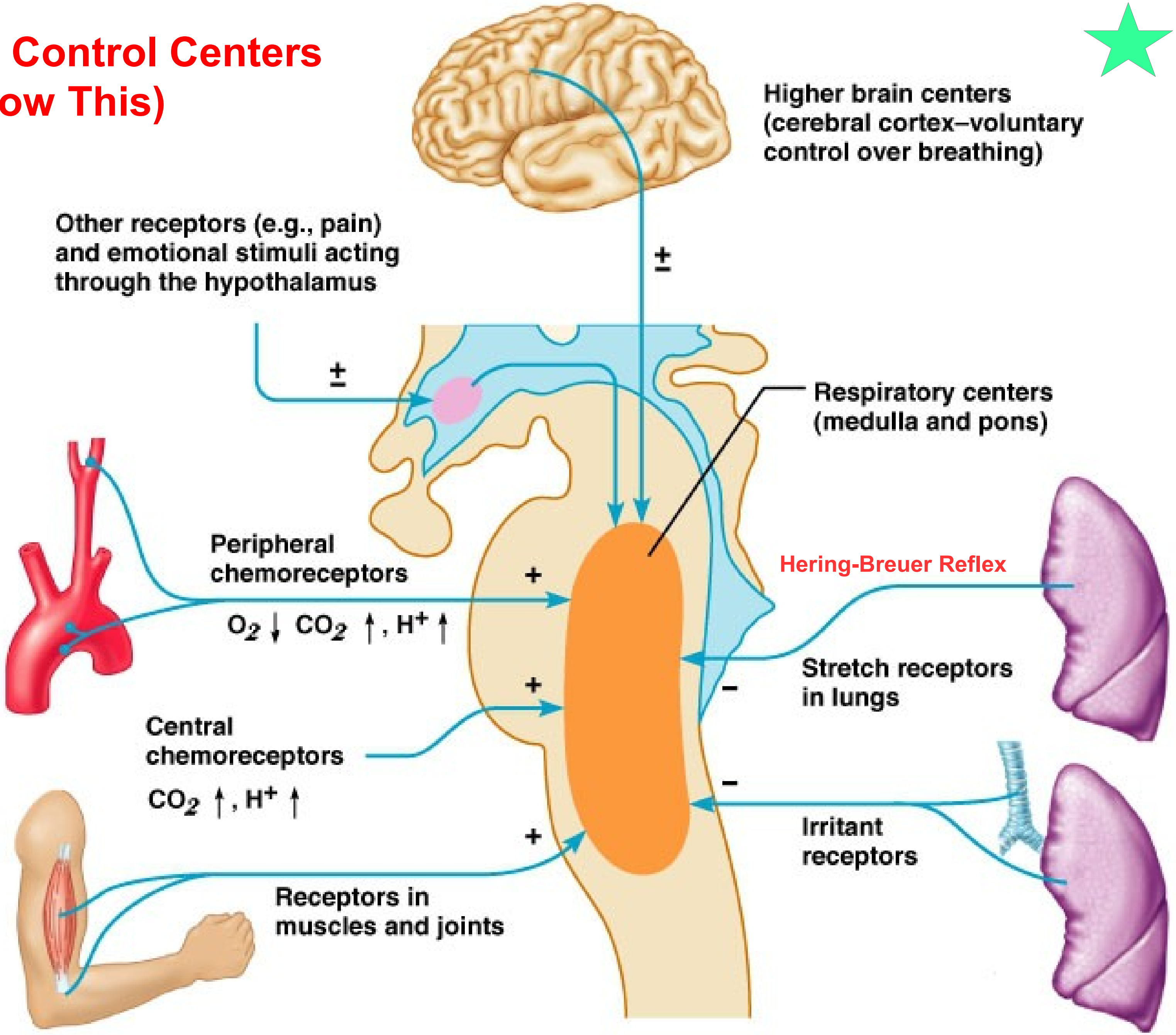


(b) Response to decreased arterial P_{CO_2} and/or H^+ concentration by a negative feedback loop

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)



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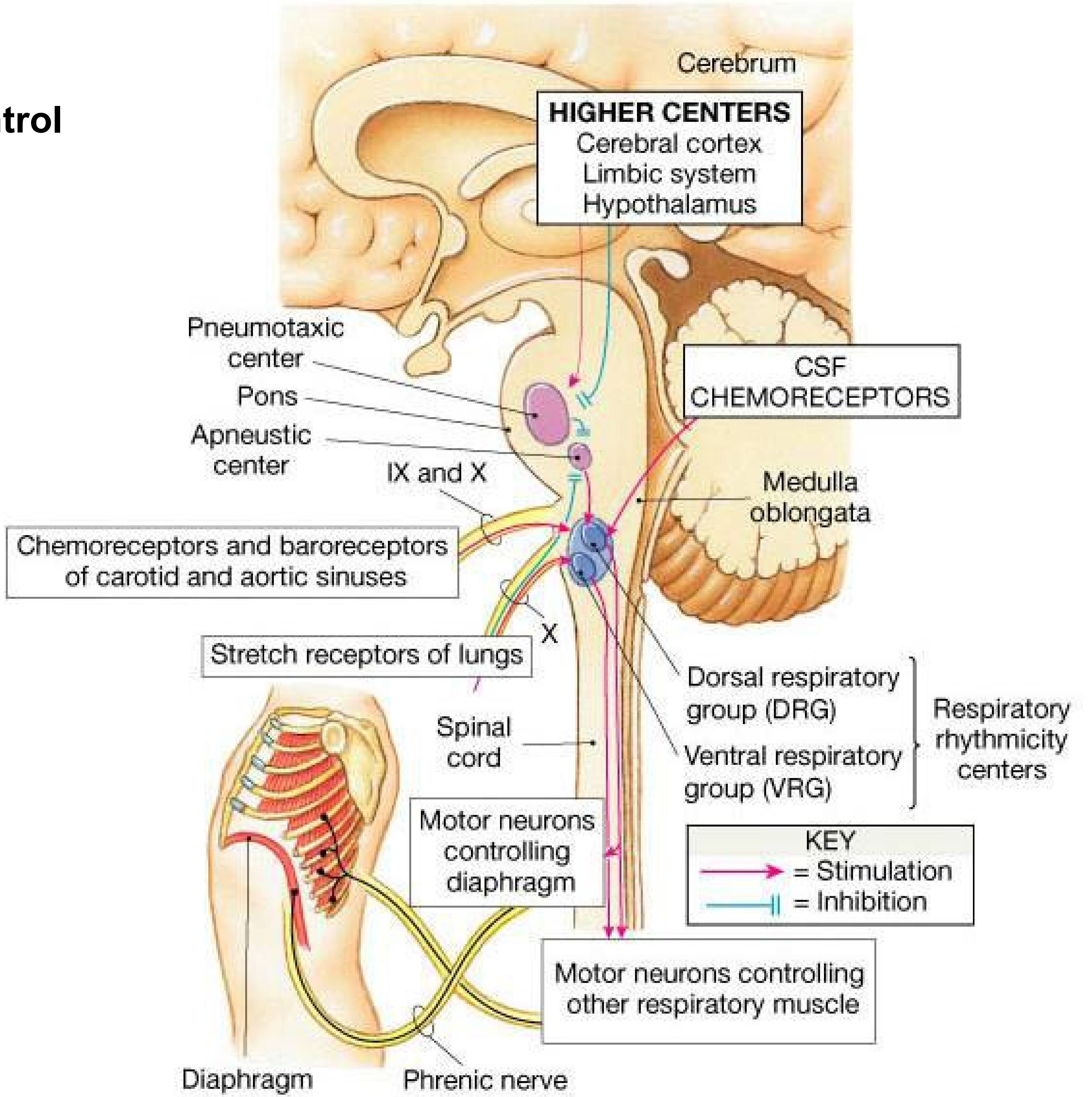
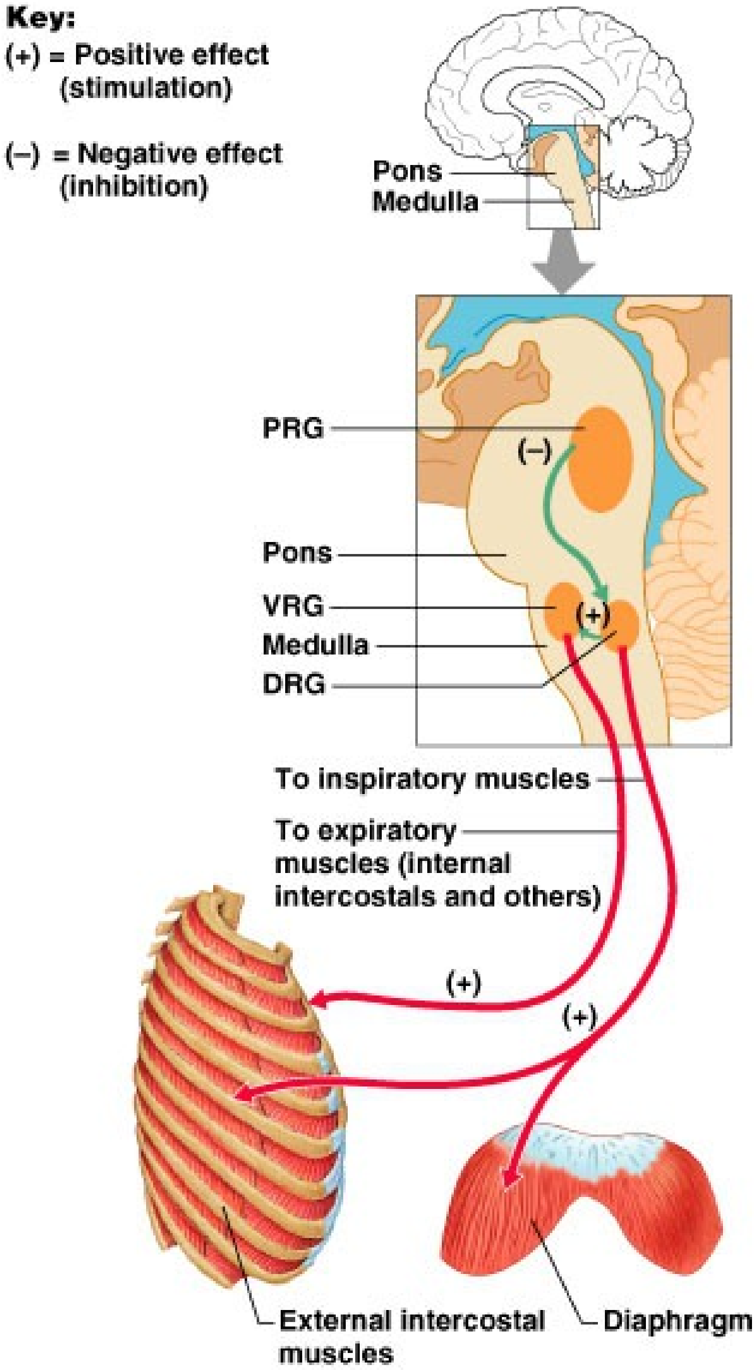
Respiratory Control Centers



Respiratory Control Centers



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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

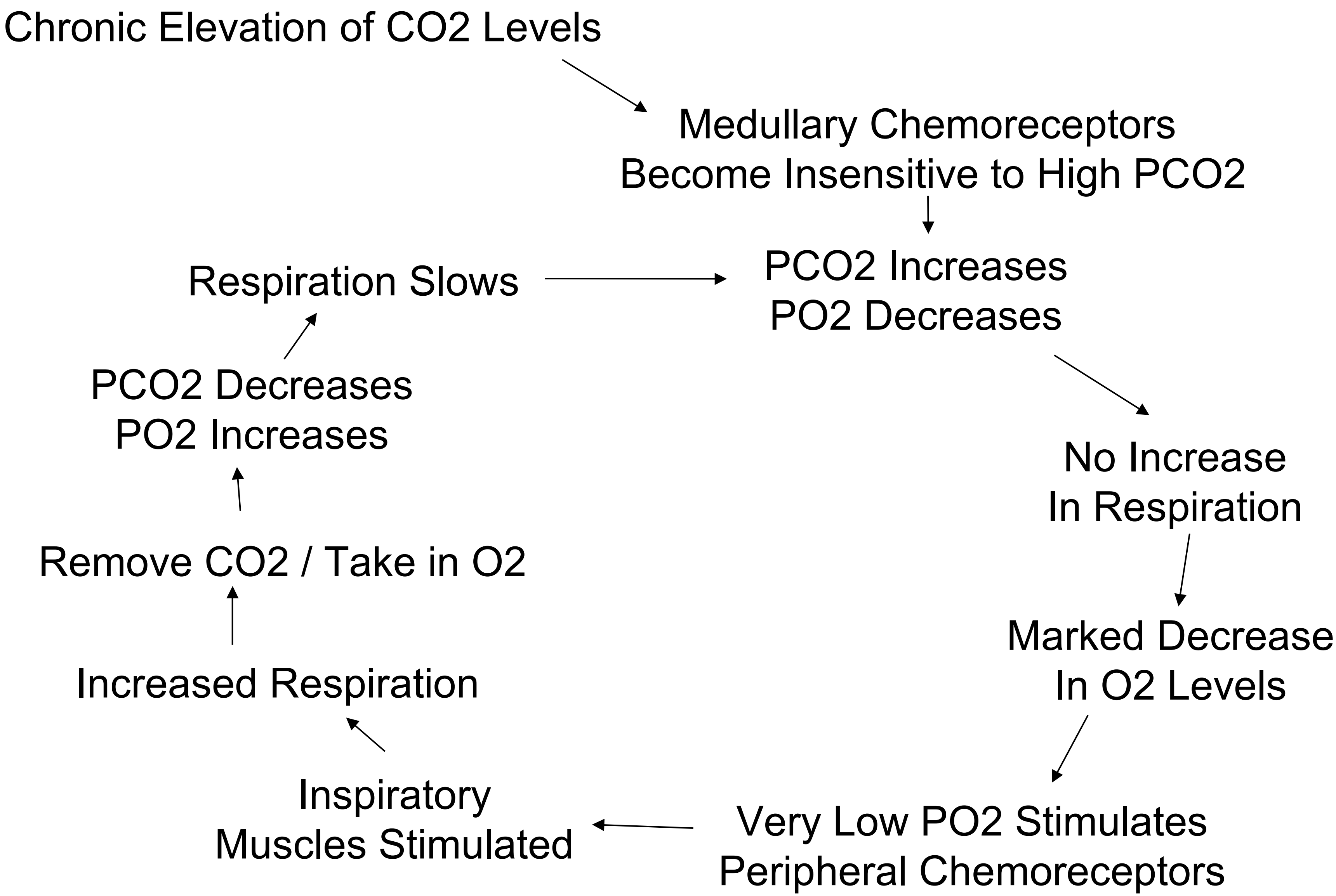
How Breathing Rate Influences Respiratory Centers

- 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

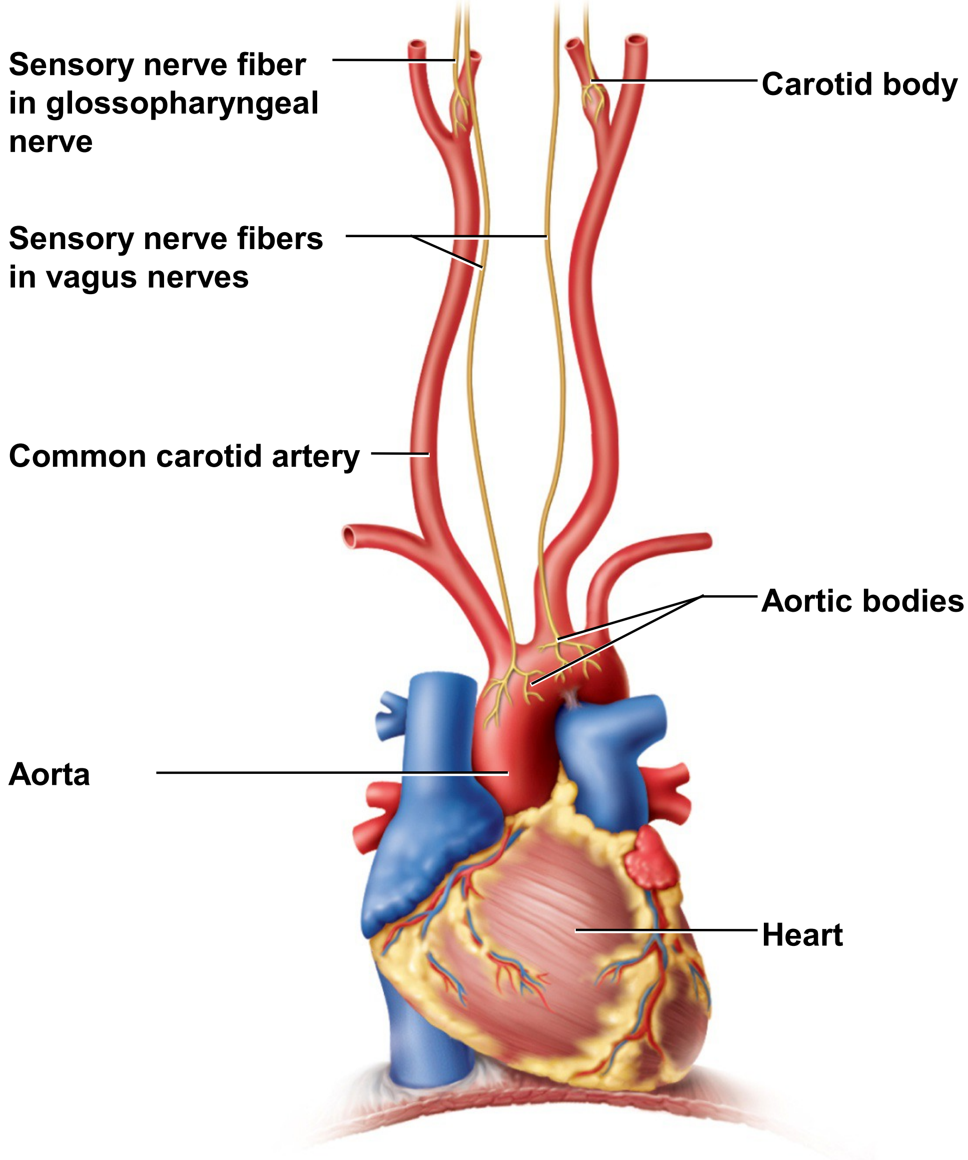
Hypoxic Drive

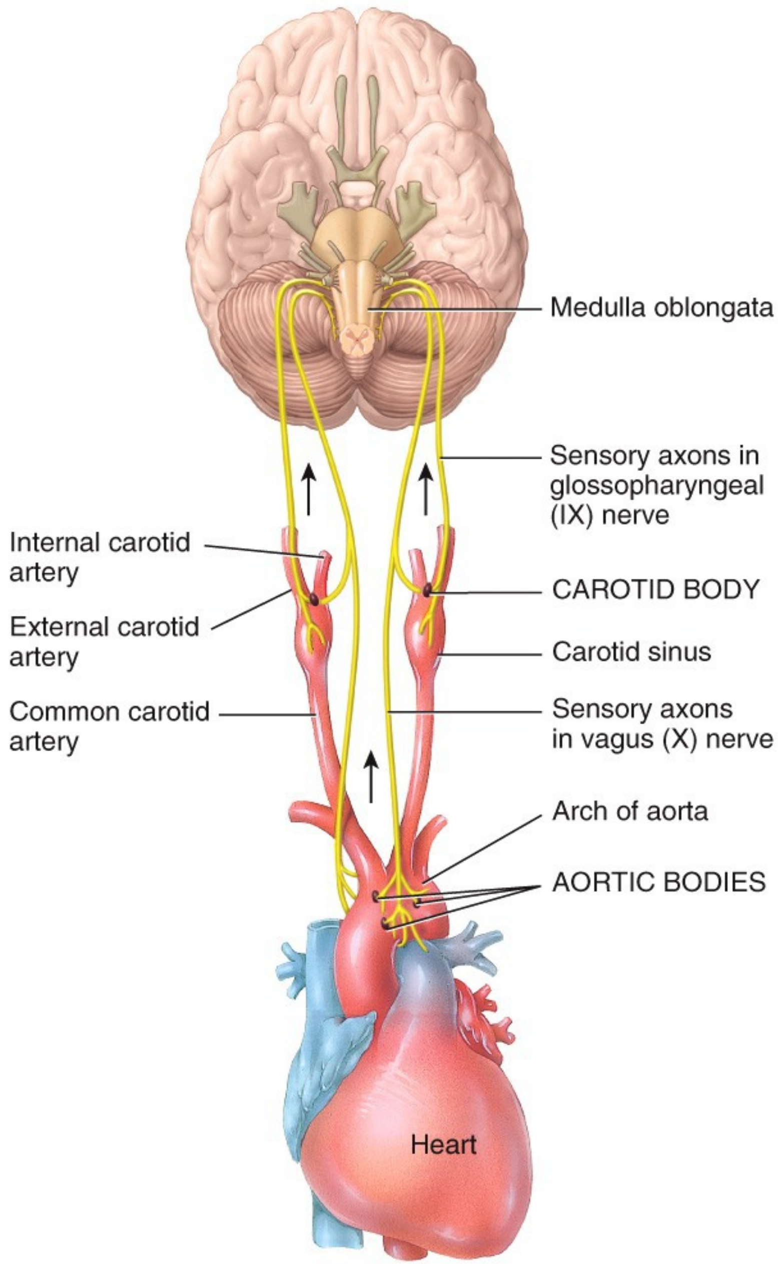
- Under normal conditions, PCO2 is the stimulus used to start inspiration.
- If high PCO2 becomes a chronic condition, then chemoreceptors in medulla become insensitive to PCO2
- Then peripheral chemoreceptor (which monitor arterial PO2) stimulate inspiration if arterial blood has low PO2 levels
- This theory also suggest that high PO2 will then inhibit inspiration.
- *Therefore, apparent danger if you give oxygen to COPD patient if they are under hypoxic drive /// high PO2 will stop inspiration !!!!!*

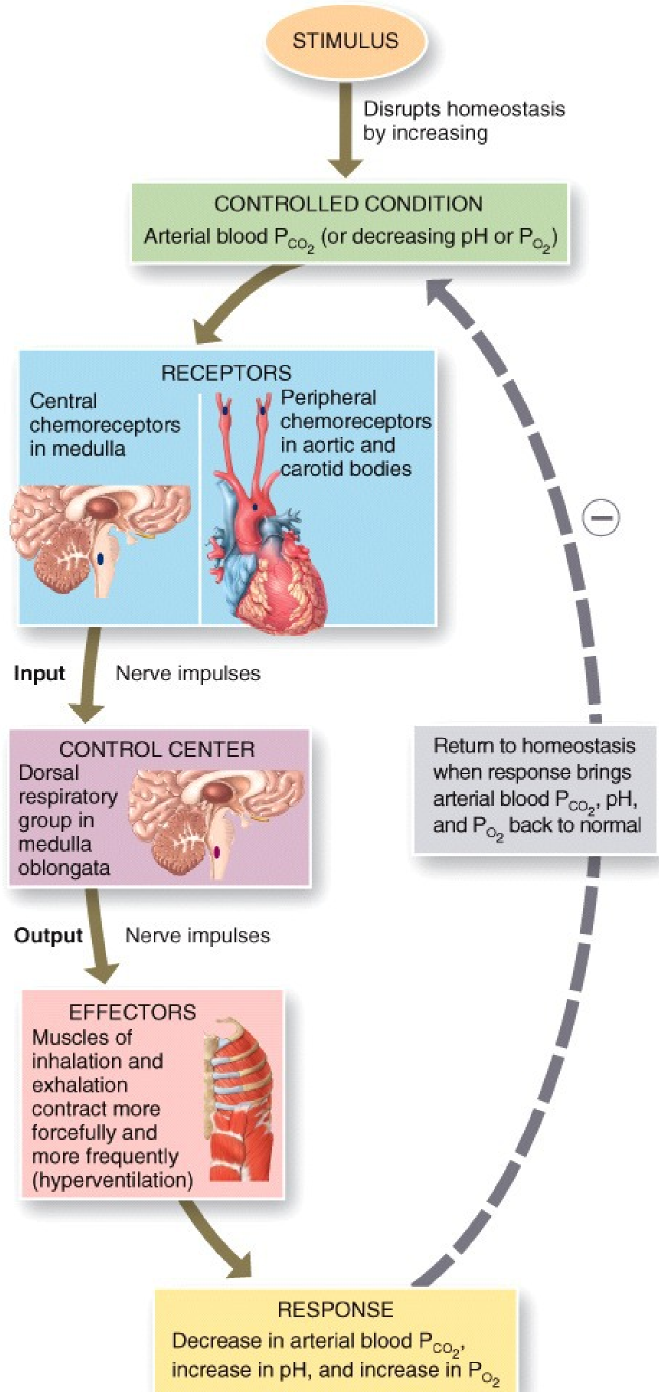
Hypoxic Drive



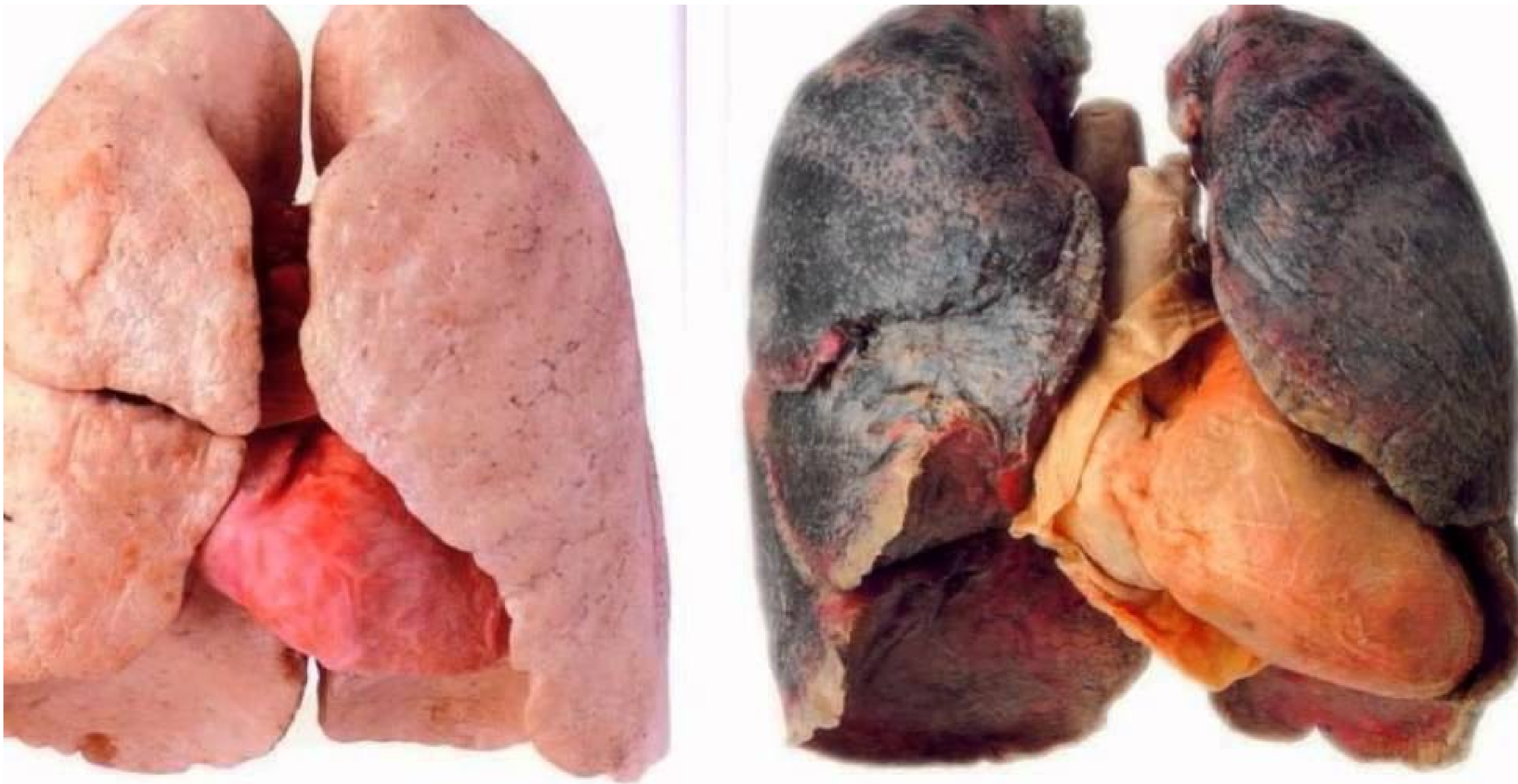
Peripheral Chemoreceptors




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


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.

12 hours after quitting

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

1-3 months after quitting

Circulation improves and lung function increases

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.

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.

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.

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.

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