Neural BP regulation

Fatima Ryalat, MD, PhD

- MAP = CO * TPR

- MAP can be changed by altering the CO (or any of its parameters: SV, HR), altering the TPR (or any of its parameters), or altering both CO and TPR.

- CO and TPR are not independent variables.

Mechanisms regulating BP

- Pa is regulated by two major systems.
- The first system is **neurally mediated** and known as the **baroreceptor reflex**. The baroreceptor reflex attempts to restore Pa to its set-point value in a matter of seconds.

- The second system is **hormonally mediated** and includes the **renin-angiotensin-aldosterone system**, which regulates Pa more slowly, primarily by its effect on blood volume.

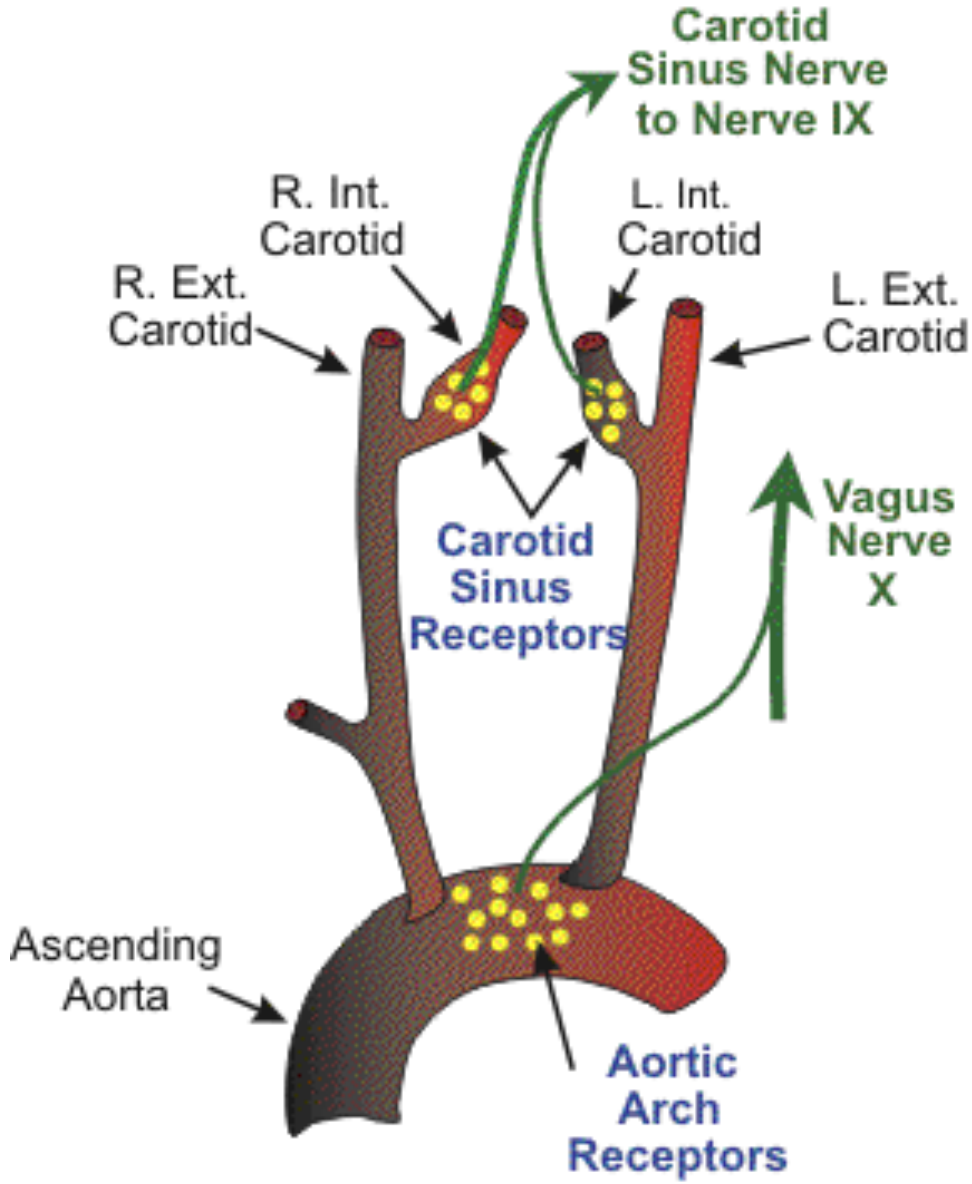


Figure 1. Location and innervation of arterial baroreceptors.

Baroreceptors

- Information from the carotid sinus baroreceptors is carried to the brain stem on the carotid sinus nerve, which joins the glossopharyngeal nerve (CN IX). Information from the aortic arch baroreceptors is carried to the brain stem on the vagus nerve (CN X)

Baroreceptors

- Located within the walls of the carotid sinus and the aortic arch.
- The baroreceptors are **mechanoreceptor**s, which are sensitive to pressure or stretch.
- The carotid sinus baroreceptors are responsive to increases or decreases in arterial pressure,
- whereas the aortic arch baroreceptors are primarily responsive to increases in arterial pressure.

Baroreceptors

- Increases in arterial pressure cause increased stretch on the baroreceptors and increased firing rate in the afferent nerves.

- Decreases in arterial pressure cause decreased stretch on the baroreceptors and decreased firing rate in the afferent nerves.

Baroreceptors

- Although the **baroreceptors are sensitive to the absolute level of pressure, they are even more sensitive to changes in pressure and the rate of change of pressure**. The strongest stimulus for the baroreceptors is a rapid change in arterial pressure.

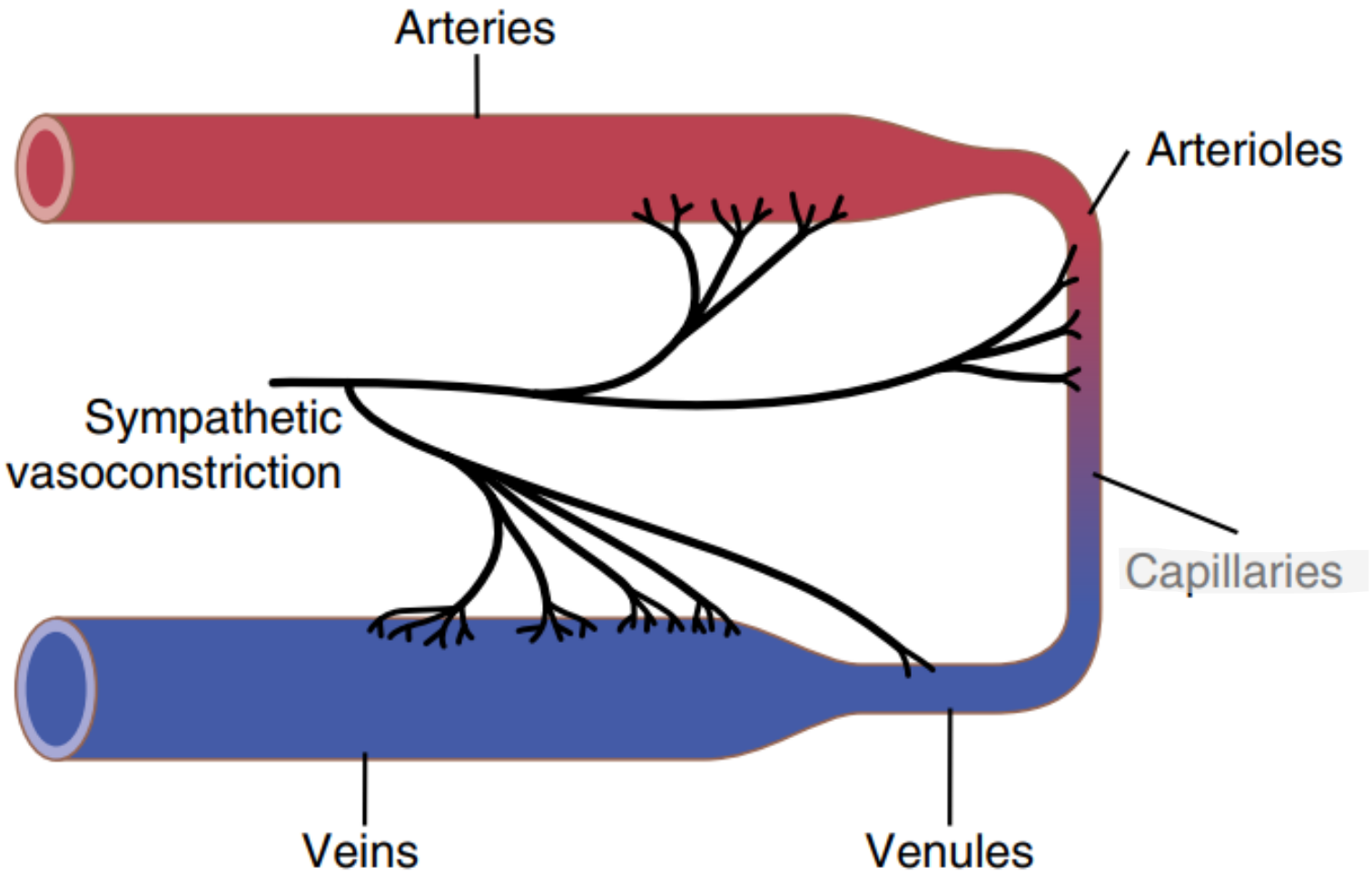
Parasympathetic cardiovascular effects

-
- The **parasympathetic** outflow is the effect of the vagus nerve on the SA node to decrease the heart rate.

Sympathetic cardiovascular effects

- The **sympathetic** outflow has four components:

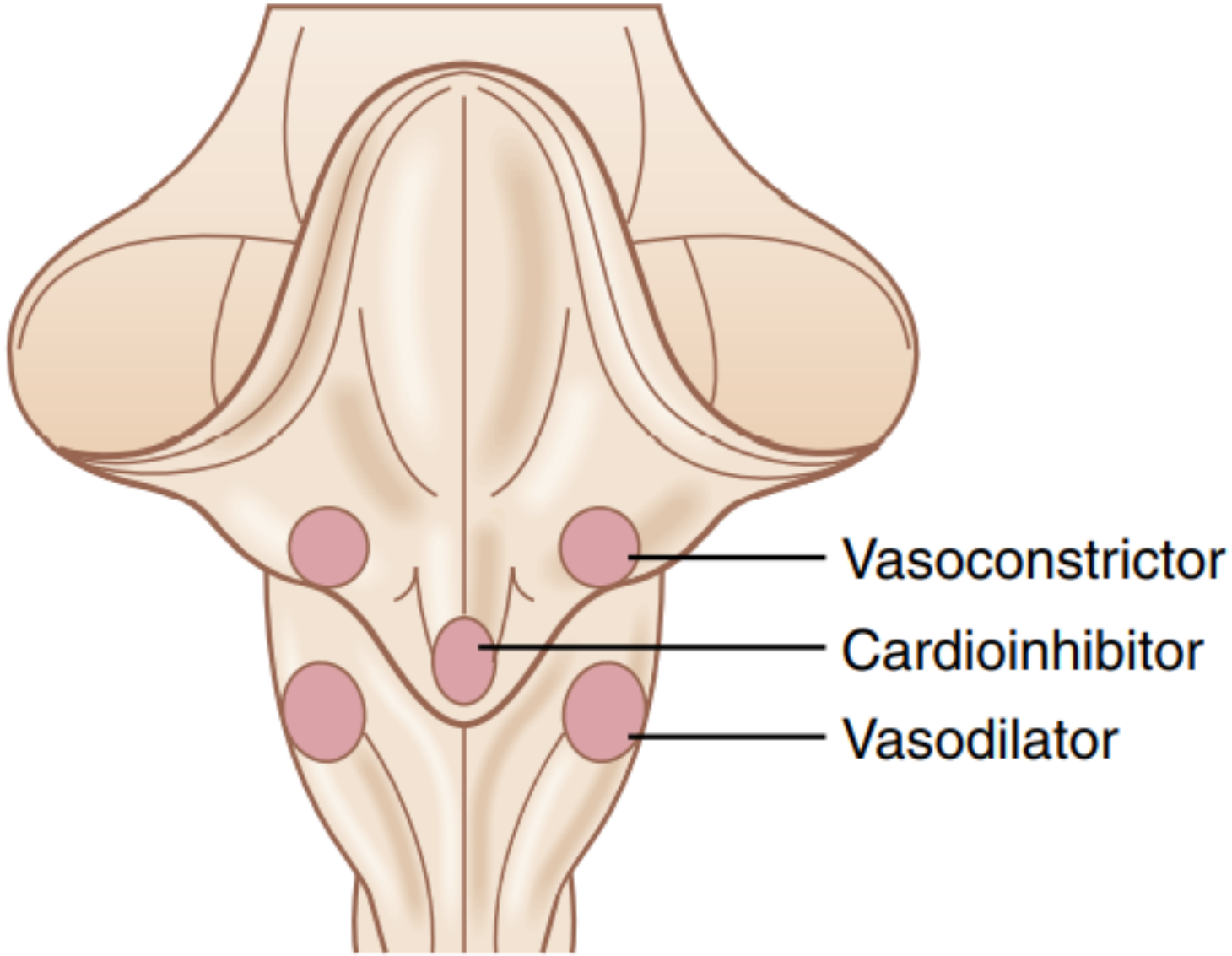
- an effect on the SA node to increase heart rate,
- an effect on cardiac muscle to increase contractility and stroke volume,
- an effect on the arterioles to produce vasoconstriction and increase TPR,
- and an effect on veins to produce venoconstriction and decrease unstressed volume.


Arteries
Arterioles
Sympathetic
vasoconstriction
Capillaries
Veins
Venules

Vasoconstriction

- The vasoconstrictor fibers are distributed to essentially all segments of the circulation (except capillaries), but more to some tissues than to others.

- This **sympathetic vasoconstrictor effect is especially powerful in the kidneys, intestines, spleen, and skin** but is much less potent in skeletal muscle, heart, and the brain.


Vasoconstrictor
Cardioinhibitor
Vasodilator

Brain stem cardiovascular centers

- Located in the reticular formations of the medulla and in the lower one-third of the pons.

- Information is integrated in the **nucleus tractus solitarius**, which then directs changes in the activity of several cardiovascular centers.

- These **cardiovascular centers are tonically active**, and the nucleus tractus solitarius simply directs, via the centers, increases or decreases in outflow from the sympathetic and parasympathetic nervous systems.

Brain stem cardiovascular centers

- The **vasoconstrictor center (area)** (also called C1) is located in the anterolateral portions of the upper medulla.

- Efferent neurons from this vasomotor center are part of the sympathetic nervous system and synapse in the spinal cord, then in sympathetic ganglia, and finally on the target organs, producing vasoconstriction in the **arterioles and venules and veins**.

Vasomotor tone

- Under normal conditions, the vasoconstrictor area of the vasomotor center **transmits signals continuously to the sympathetic vasoconstrictor nerve fibers over the entire body**, causing slow firing of these fibers at a rate of about 0.5 to 2 impulses per second. This continual firing is called sympathetic vasoconstrictor tone. These impulses normally maintain a partial state of constriction in the blood vessels, called **vasomotor tone**.

Brain stem cardiovascular centers

- The **vasodilator center (area)** is located in the anterolateral portions of the lower half of the medulla.

- The fibers from these neurons project upward to the vasoconstrictor area just described, **inhibiting the vasoconstrictor activity** of this area and causing vasodilation.

Brain stem cardiovascular centers

- The **sensory area** is located bilaterally in the **nucleus tractus solitarius** in the posterolateral portions of the medulla and lower pons.

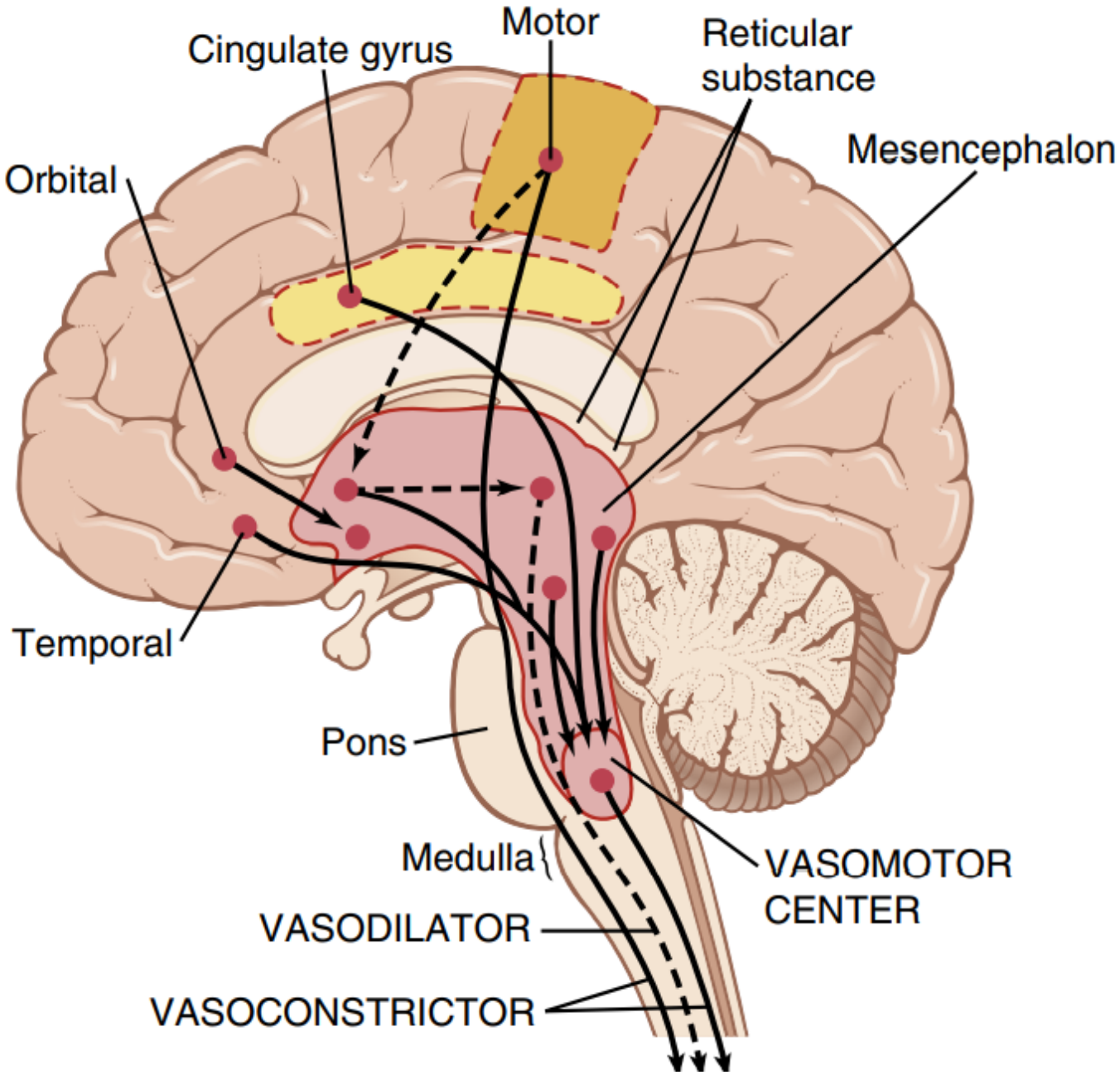
- The neurons of this area receive sensory nerve signals from the circulatory system mainly through the vagus and glossopharyngeal nerves, and output signals from this sensory area then help control activities of the vasoconstrictor and vasodilator areas of the vasomotor center, thus providing reflex control of many circulatory functions.

Brain stem cardiovascular centers

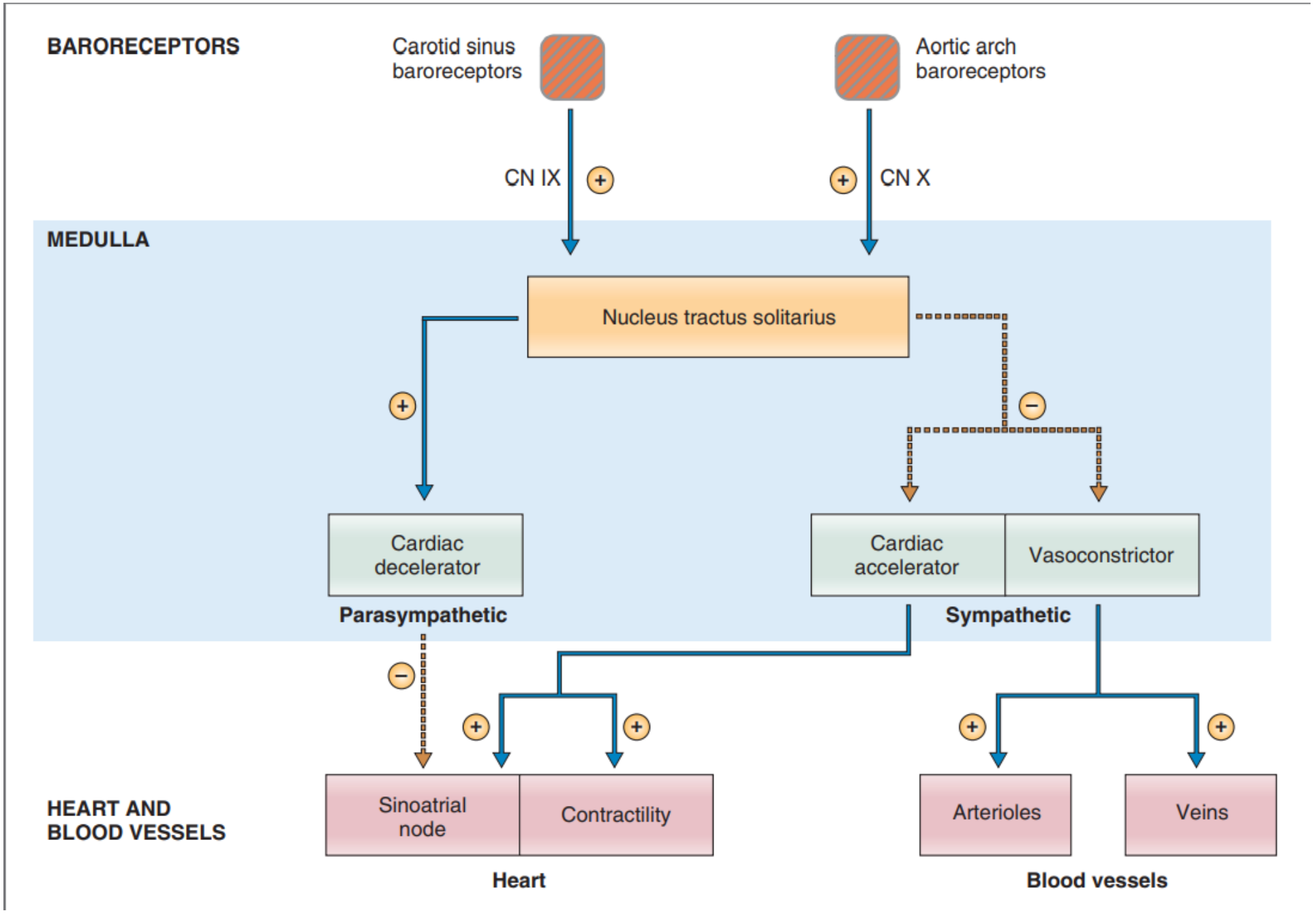
- The **cardiac accelerator center**.
- Efferent neurons from the cardiac accelerator center are also part of the sympathetic nervous system and synapse in the spinal cord, in sympathetic ganglia, and finally in the heart.
- In the heart, the effects of this activity are an increased firing rate of the SA node (to increase heart rate), increased conduction velocity through the AV node, and increased contractility.

Brain stem cardiovascular centers

- The **cardiac decelerator center**.
- Efferent fibers from the cardiac decelerator center are part of the parasympathetic nervous system:
- They travel in the vagus nerve and synapse on the SA node to decrease heart rate

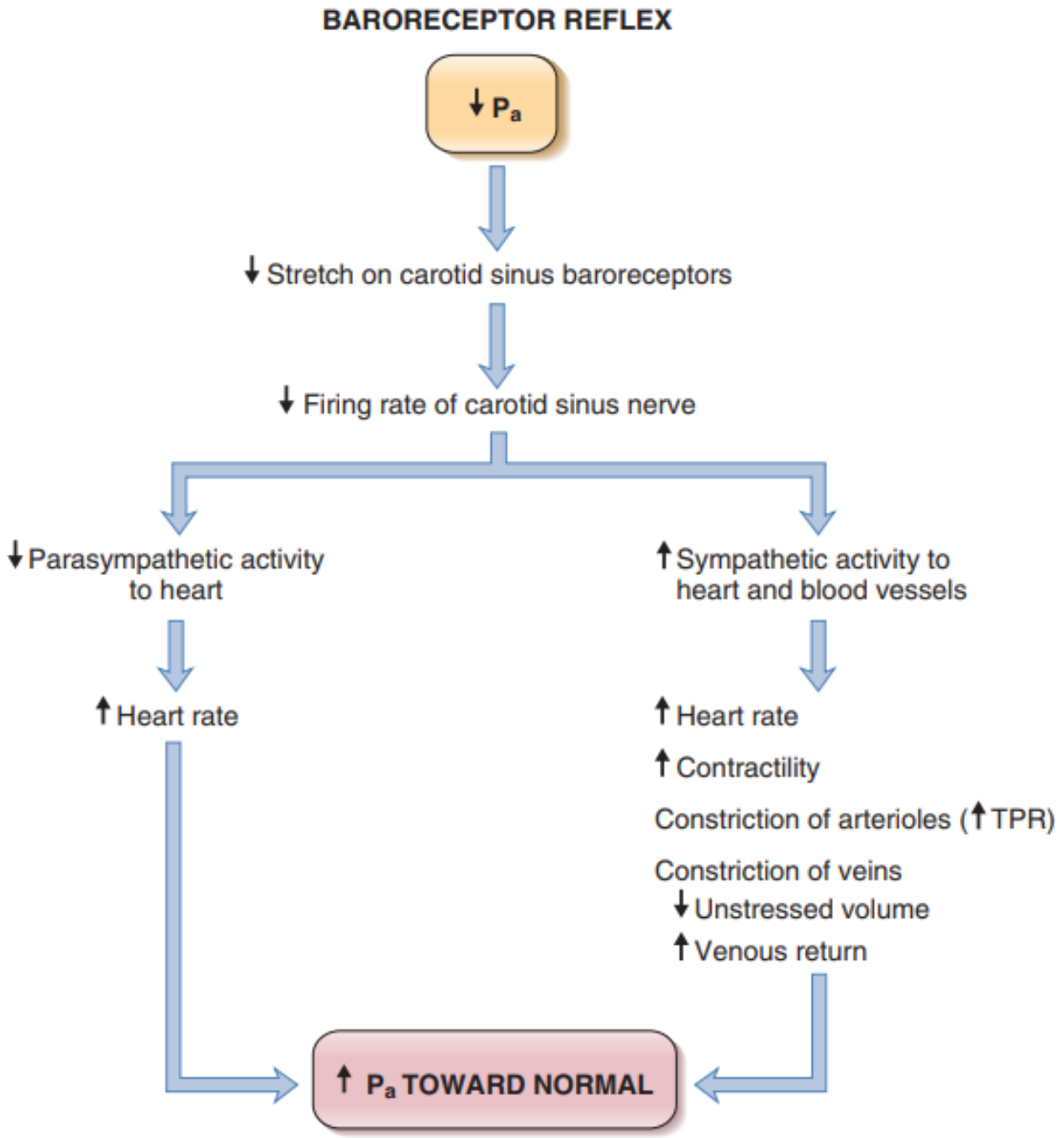

Cingulate gyrus
Motor
Reticular substance
Mesencephalon
Orbital
Temporal
Pons
Medulla
VASOMOTOR CENTER
VASODILATOR
VASOCONSTRICTOR

Baroreceptor reflex response to increase in arterial pressure


BARORECEPTORS
Carotid sinus baroreceptors
Aortic arch baroreceptors
CN IX
CN X
MEDULLA
Nucleus tractus solitarius
Cardiac decelerator
Cardiac accelerator
Vasoconstrictor
Parasympathetic
Sympathetic
HEART AND BLOOD VESSELS
Sinoatrial node
Contractility
Arterioles
Veins
Heart
Blood vessels

Baroreceptor reflex response to decrease in arterial pressure

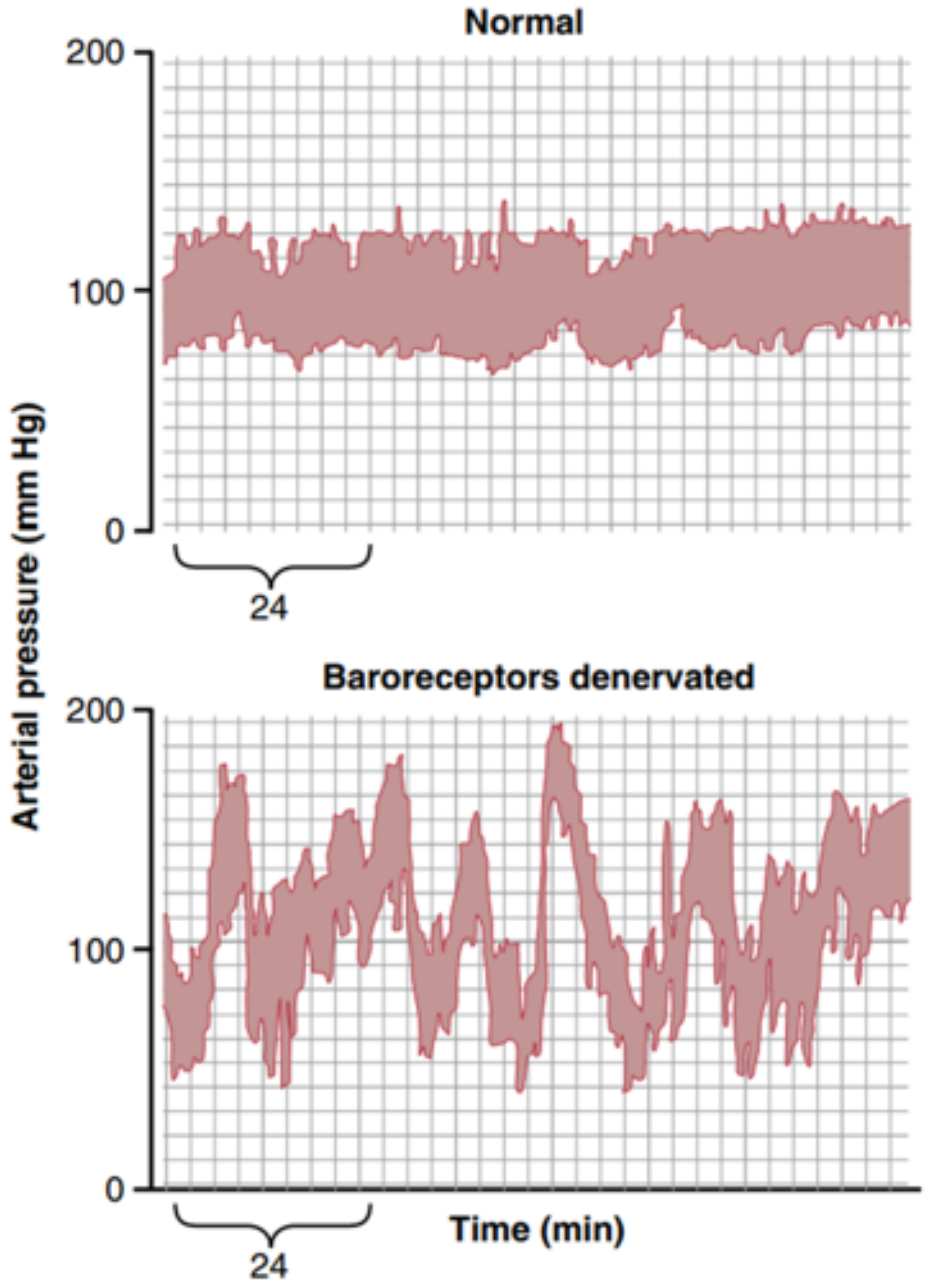
- **Hemorrhage** produces a decrease in Pa because, as blood volume decreases, stressed volume also decreases.
- In response to an acute reduction in Pa, the baroreceptor reflex is activated and attempts to restore blood pressure back toward normal.

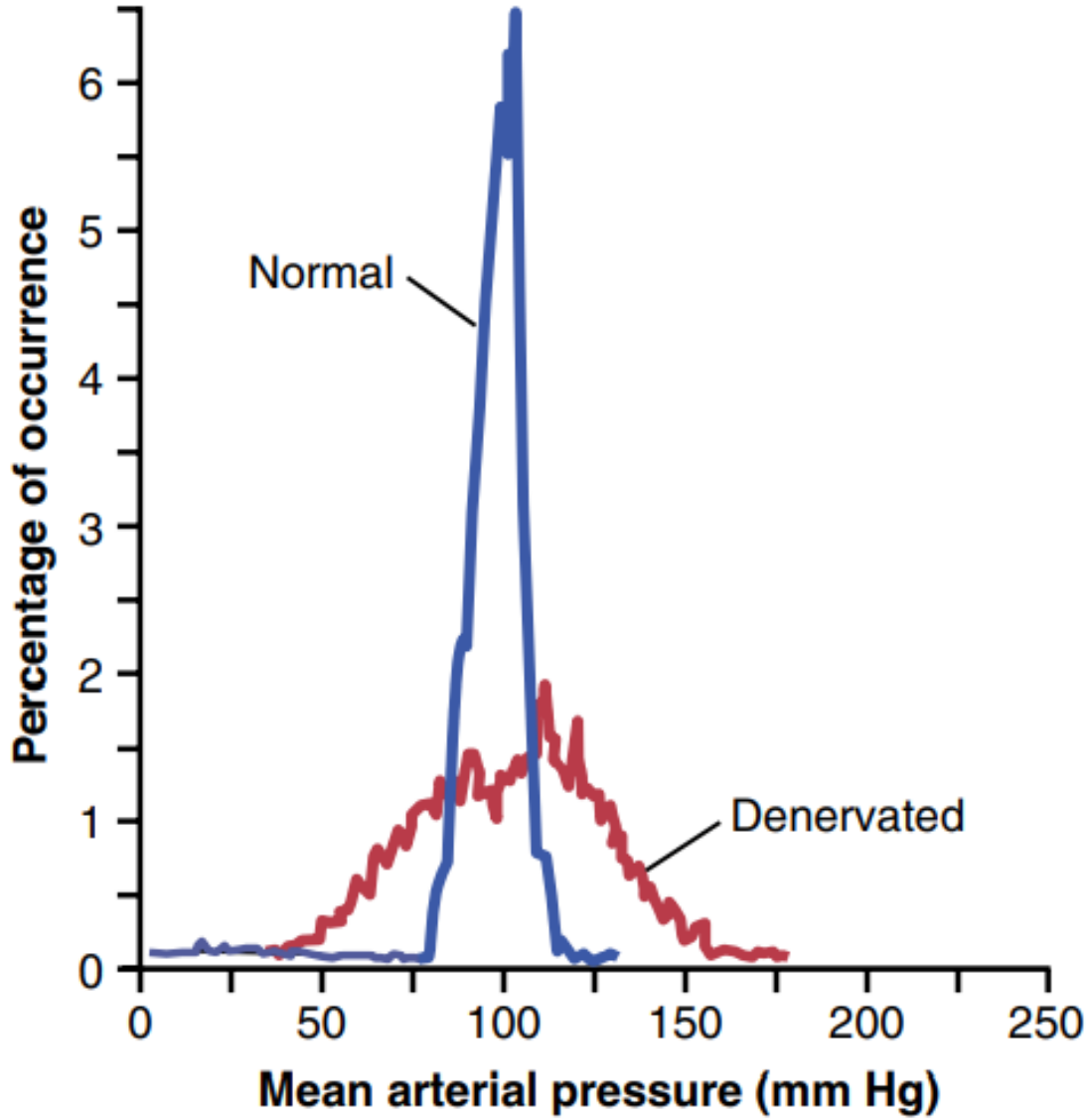

BARORECEPTOR REFLEX
↓ P_a
↓ Stretch on carotid sinus baroreceptors
↓ Firing rate of carotid sinus nerve
↓ Parasympathetic activity to heart
↑ Heart rate
↑ Sympathetic activity to heart and blood vessels
↑ Heart rate
↑ Contractility
Constriction of arterioles (↑ TPR)
Constriction of veins
↓ Unstressed volume
↑ Venous return
↑ P_a TOWARD NORMAL

Baroreceptors Attenuate Blood Pressure Changes During Changes in Body Posture.

- The ability of the baroreceptors to maintain relatively constant arterial pressure in the upper body is important when a person stands up after lying down. Immediately on standing, the arterial pressure in the head and upper part of the body tends to fall and marked reduction of this pressure could cause loss of consciousness.
- However, the falling pressure at the baroreceptors elicits an immediate reflex, resulting in strong sympathetic discharge throughout the body that minimizes the decrease in pressure in the head and upper body.

Pressure Buffer Function of the Baroreceptor Control System.




Percentage of occurrence
0
1
2
3
4
5
6
Normal
Denervated
0
50
100
150
200
250
Mean arterial pressure (mm Hg)

Are the Baroreceptors Important in Long-Term Regulation of Arterial Pressure?

Test of Baroreceptor Reflex: Valsalva Maneuver

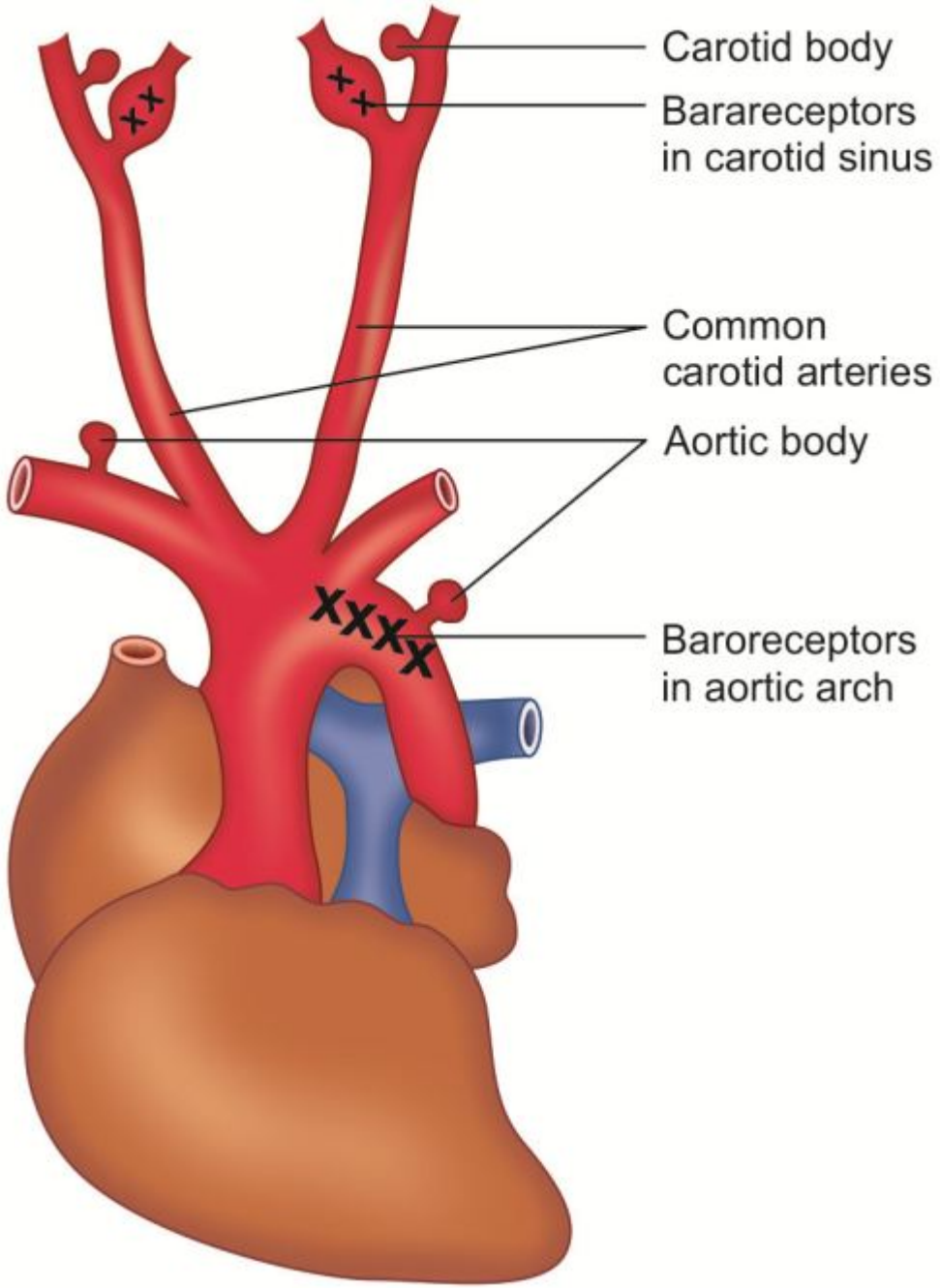
- Exhalation against closed epiglottis-→ increase intrathoracic pressure → decrease VR and CO then BP → activate baroreflex → increase sympathetic and decrease parasympathetic.

Peripheral Chemoreceptors in Carotid and Aortic Bodies

- Peripheral chemoreceptors **for O2** are located in the **carotid bodies** near the bifurcation of the common carotid arteries and in the **aortic bodies** along the aortic arch.

- Their chemoreceptors are primarily sensitive to decreases in (PO2). The chemoreceptors also are sensitive to increases in (PCO2) and decreases in pH, particularly when PO2 is simultaneously decreased.

- **The response of the peripheral chemoreceptors to decreased arterial PO2 is greater when the PCO2 is increased or the pH is decreased**.



Peripheral Chemoreceptors in Carotid and Aortic Bodies

- When **arterial PO2 decreases**, there is an increased firing rate of afferent nerves from the carotid and aortic bodies that **activates sympathetic vasoconstrictor centers**. As a result, there is arteriolar **vasoconstriction** in skeletal muscle, renal, and splanchnic vascular beds.

Central Chemoreceptors

- The brain is intolerant of decreases in blood flow, and therefore it is not surprising that chemoreceptors are located in the medulla itself.
- These chemoreceptors are **most sensitive to CO2 and pH** and less sensitive to O2.

- **Changes in PCO2 or pH stimulate the medullary chemoreceptors**, which then direct changes in outflow of the medullary cardiovascular centers.

Central Chemoreceptors

- If **the brain becomes ischemic** (i.e., there is decreased cerebral blood flow), cerebral PCO2 immediately increases and pH decreases.

- The medullary chemoreceptors detect these changes and direct an **increase in sympathetic** outflow that causes intense arteriolar **vasoconstriction** in many vascular beds and an increase in TPR.

- Blood flow is thereby redirected to the brain to maintain its perfusion. As a result of this vasoconstriction, **BP increases dramatically**, even to life-threatening levels.

Central Chemoreceptors

- **The Cushing reaction:**
- When intracranial pressure increases (e.g., tumors, head injury), there is compression of cerebral arteries, which results in decreased perfusion of the brain.
- There is an immediate increase in PCO2 and a decrease in PH.
- The medullary chemoreceptors respond to these changes in PCO2 and pH by directing an increase in sympathetic outflow to the blood vessels to increase TPR and dramatically increase BP.

Thank you