

Neural BP regulation-5

- $MAP = CO * TPR$
- 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.

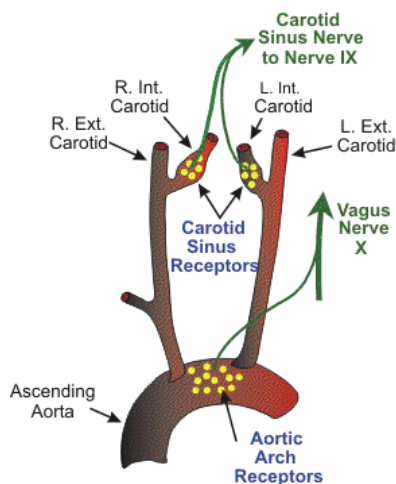


Figure 1. Location and innervation of arterial baroreceptors.

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)

- Located within the walls of the carotid sinus and the aortic arch.
 - The baroreceptors are **mechanoreceptors**, 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.
- 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.
 - 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.

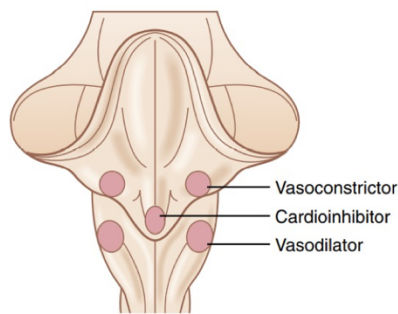
Parasympathetic cardiovascular effects

- 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.
- **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.

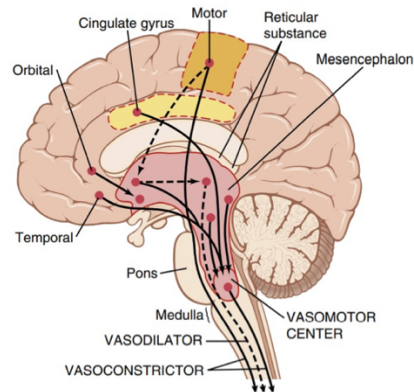


Brain stem cardiovascular centers

- 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.
- 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.**
 - 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.
 - **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.
- 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.**

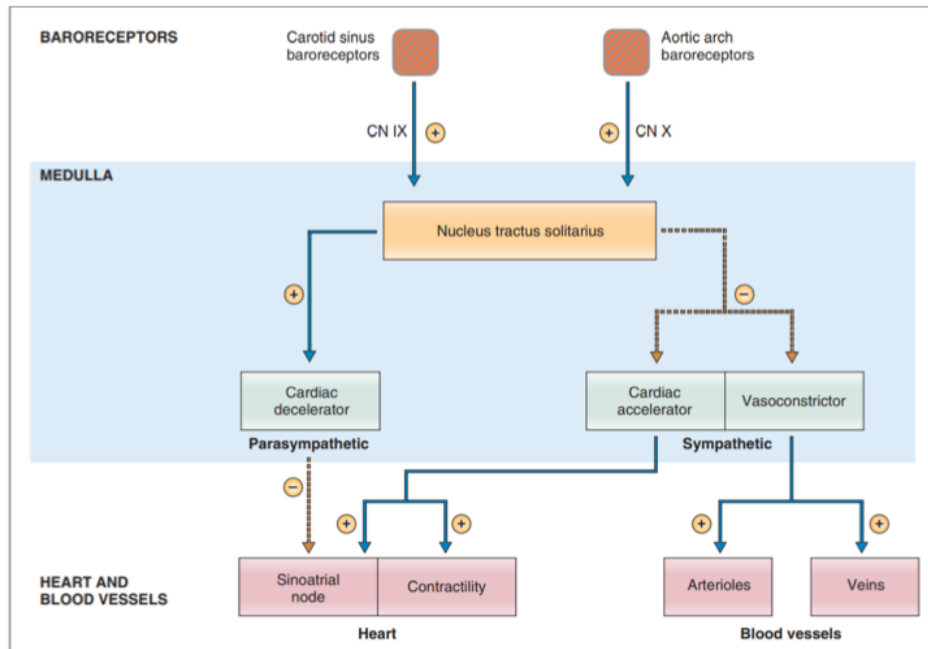
- The **cardiac decelerator center**.
- Efferent fibers from the cardiac decelerator center are part of the **parasympathetic nervous system**:
- They travel in the vagus nerve and synapse on the SA node to decrease heart rate



Vasomotor tone

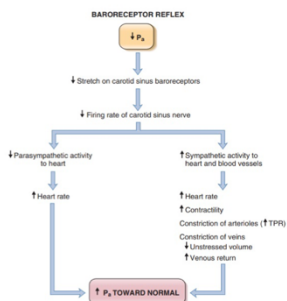
- Under normal conditions, the vasoconstrictor area of the vasomotor center **transmits signals continuously to the sympathetic vasoconstrictor nerve fibers over the entire body**, causing slow firing of these fibers at a rate of about 0.5 to 2 impulses per second. This continual firing is called **sympathetic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

Baroreceptor reflex response to increase in arterial pressure



Baroreceptor reflex response to decrease in arterial pressure

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

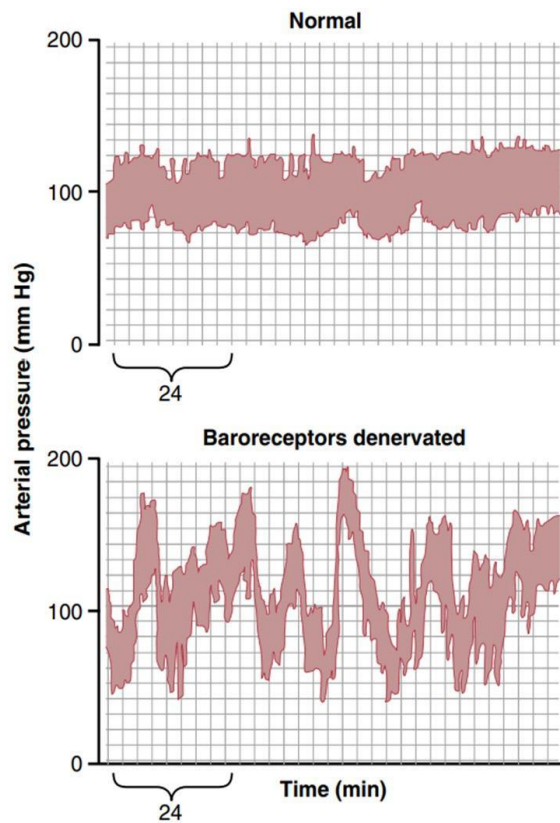


Baroreceptors Attenuate Blood Pressure Changes During Changes in Body Posture.

- 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**

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- **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.**
- 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.
- 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.
- **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.

Goodluck, Rayyan shtewe ☺ .