

REGULATION OF BLOOD PRESSURE



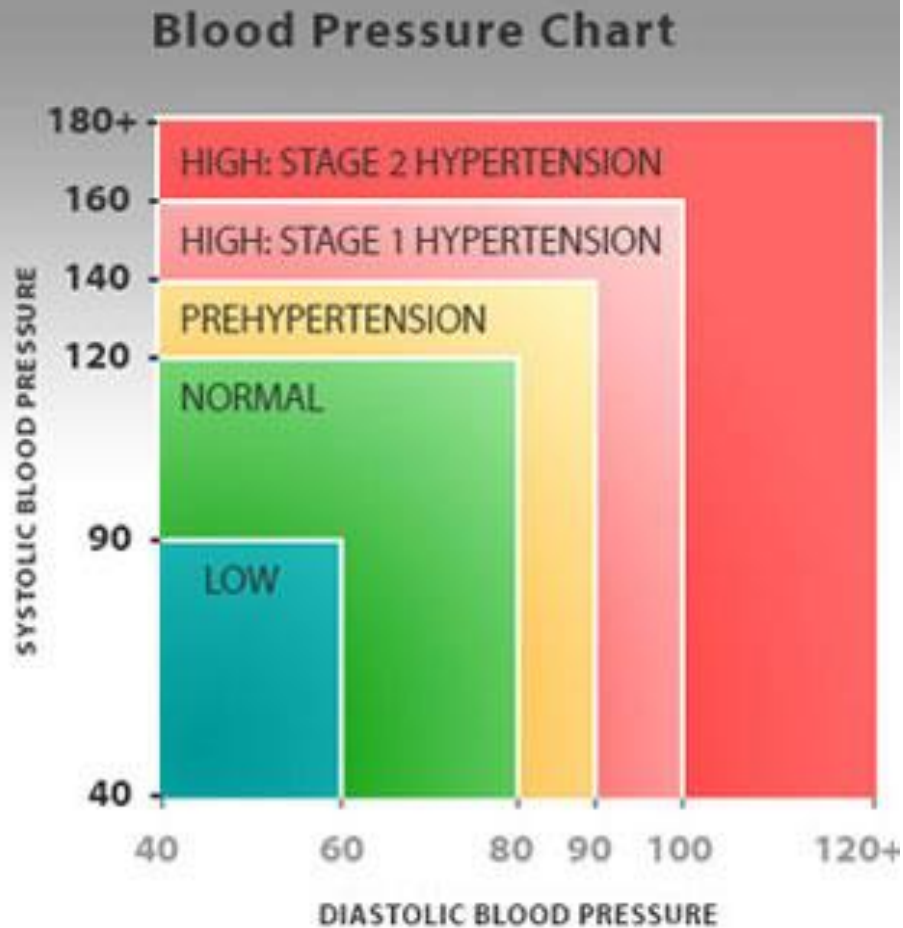
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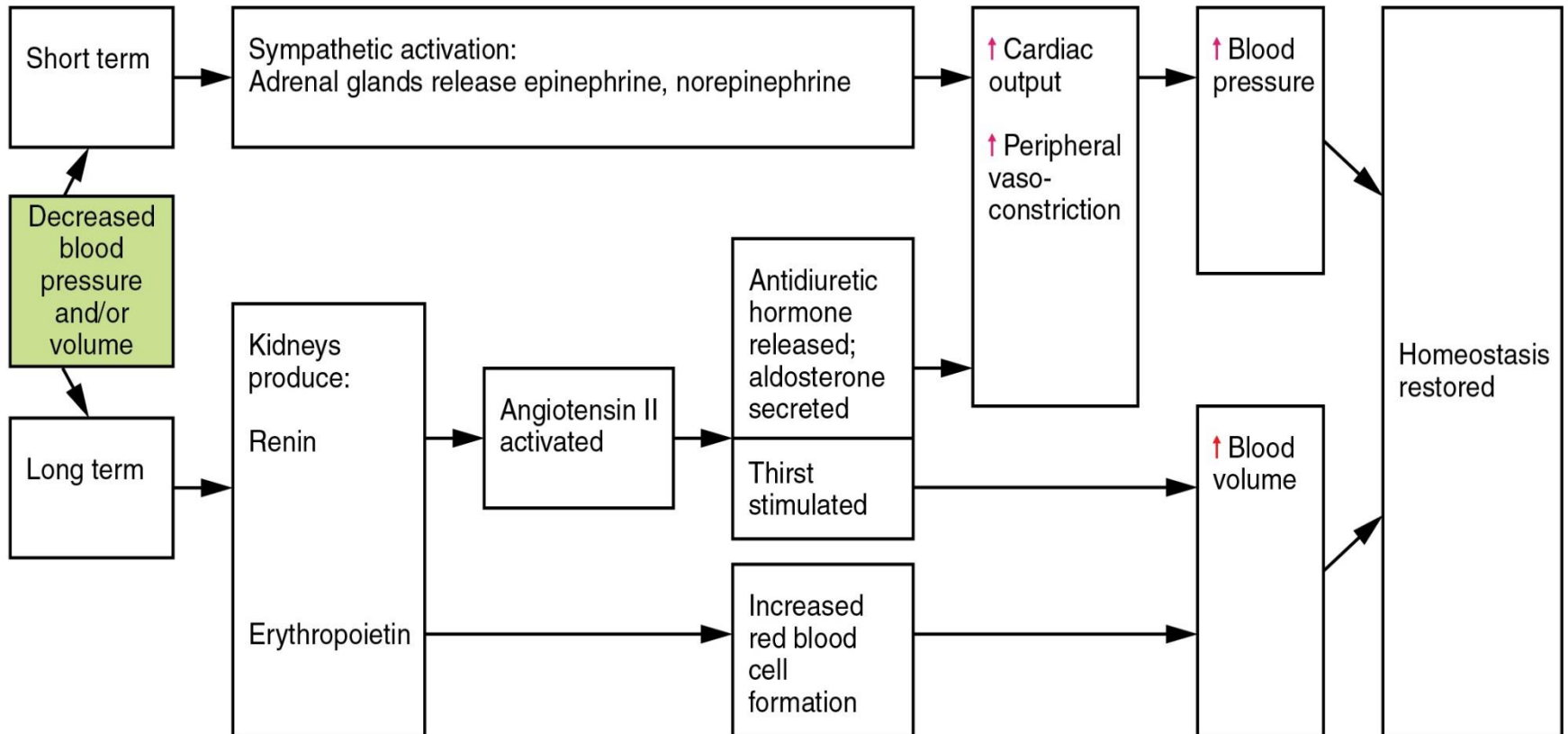
LECTURE OBJECTIVES

- Factors regulating arterial blood pressure
- Explain how they influence arterial blood pressure.
- Physiological importance of regulating arterial blood pressure
- Discuss short term, intermediate and long-term regulation of blood pressure; nervous, hormonal and renal regulation of arterial blood pressure.

REGULATION OF BLOOD PRESSURE



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SHORT TERM REGULATION OF BLOOD PRESSURE

Rapidly acting within seconds to minutes

1. Baroreceptors Reflex Mechanism
2. Chemoreceptors Mechanism
3. CNS Ischemic Response Mechanism
4. Atrial Stretch Volume Receptors

1. THE BARORECEPTORS

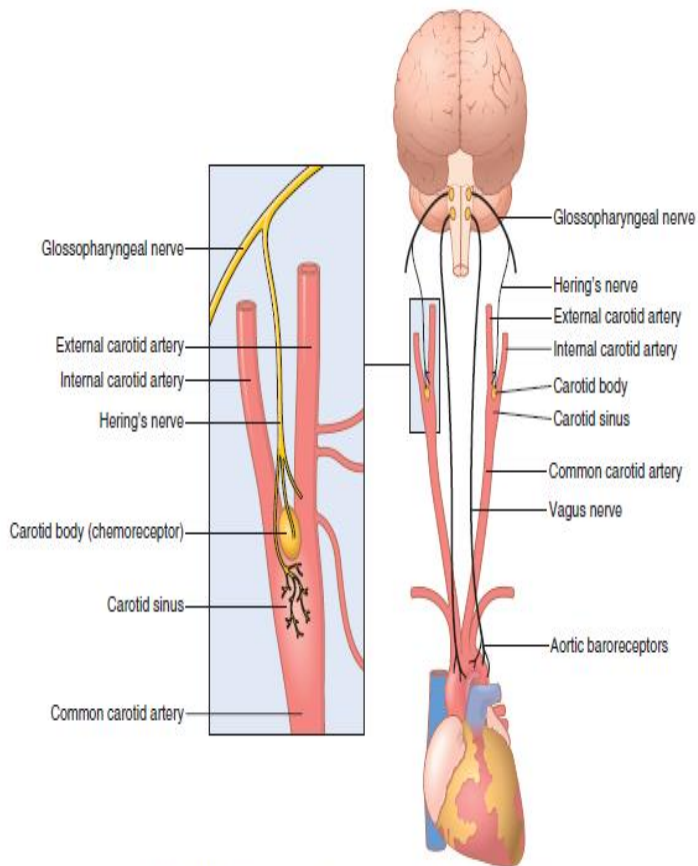


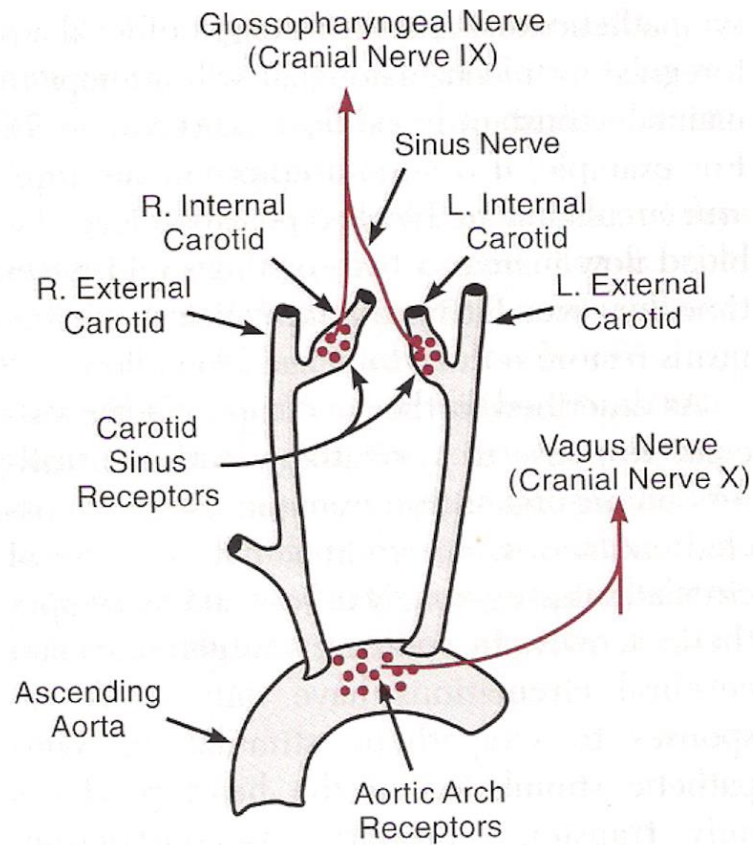
Figure 18-5. The baroreceptor system for controlling arterial pressure.

Changes in MAP are detected by **baroreceptors** (pressure receptors) in the carotid and aortic arteries.

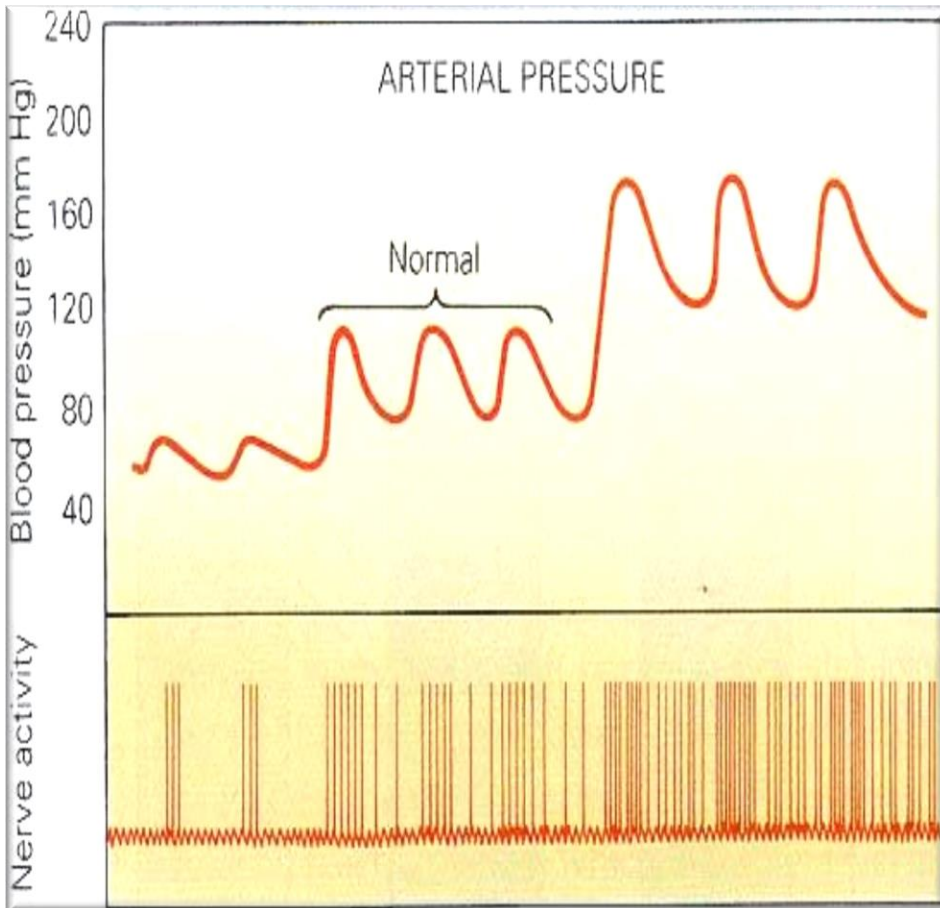
Carotid baroreceptors are located in the carotid sinus, both sides of the neck. Aortic baroreceptors are located in the aortic arch.

These receptors provide information to the cardiovascular centres in the medulla oblongata about the degree of stretch with pressure changes.

1. THE BARORECEPTORS

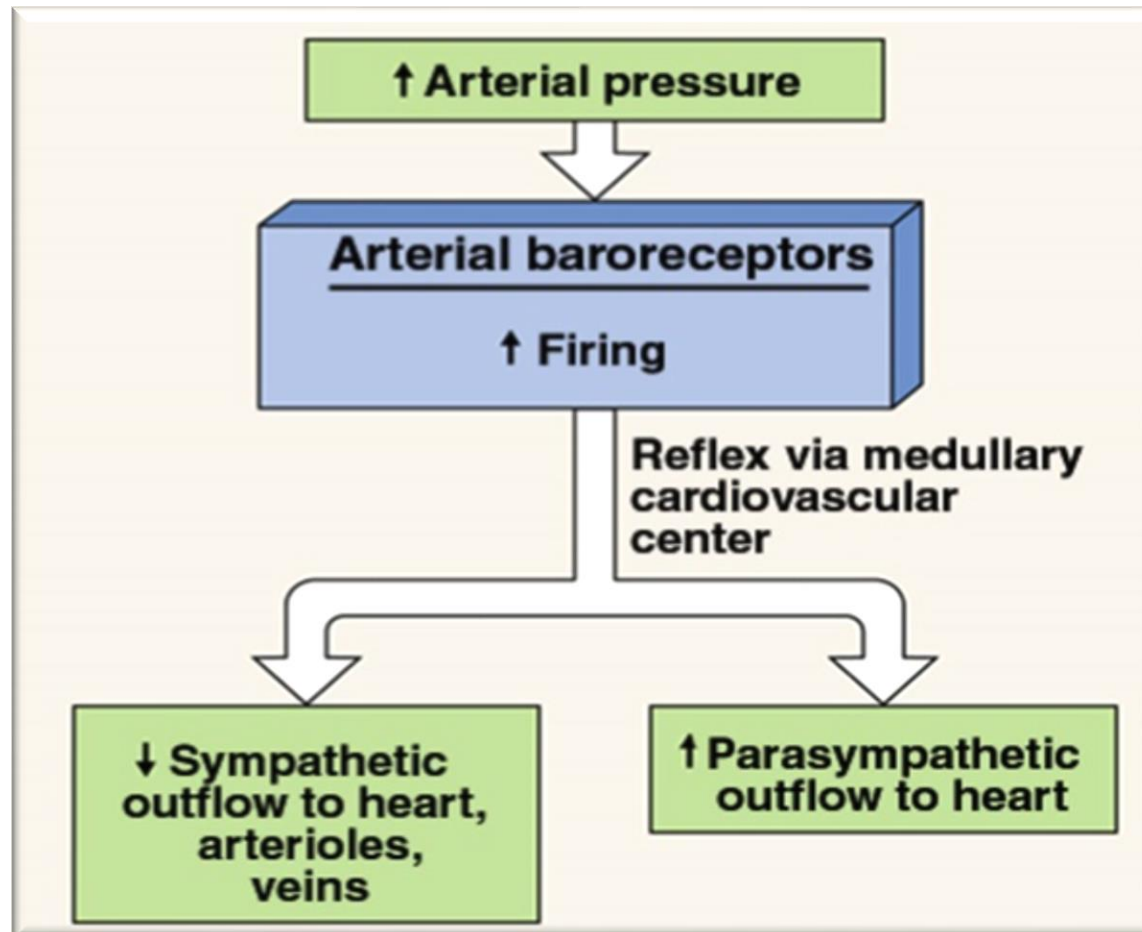


1. THE BARORECEPTORS

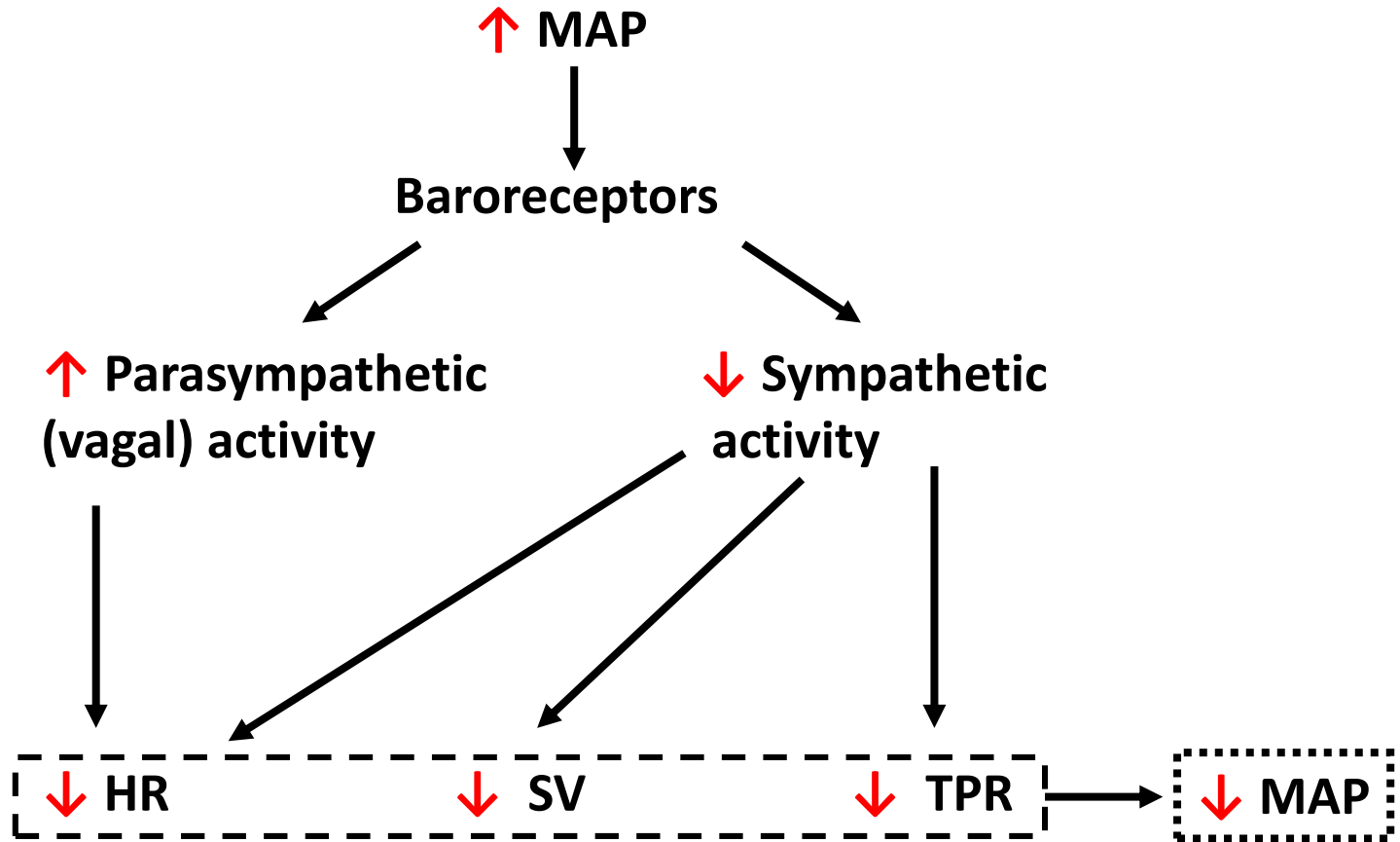


- ❑ At normal arterial pressure the baroreceptors are active.
- ❑ Increased blood pressure increases their rate of activity, while decreased pressure decreases the rate of firing (activity).
- ❑ They play an important role in maintaining relatively constant blood flow to vital organs such as brain during rapid changes in pressure such as standing up after lying down. That is why they are called “pressure buffers”.

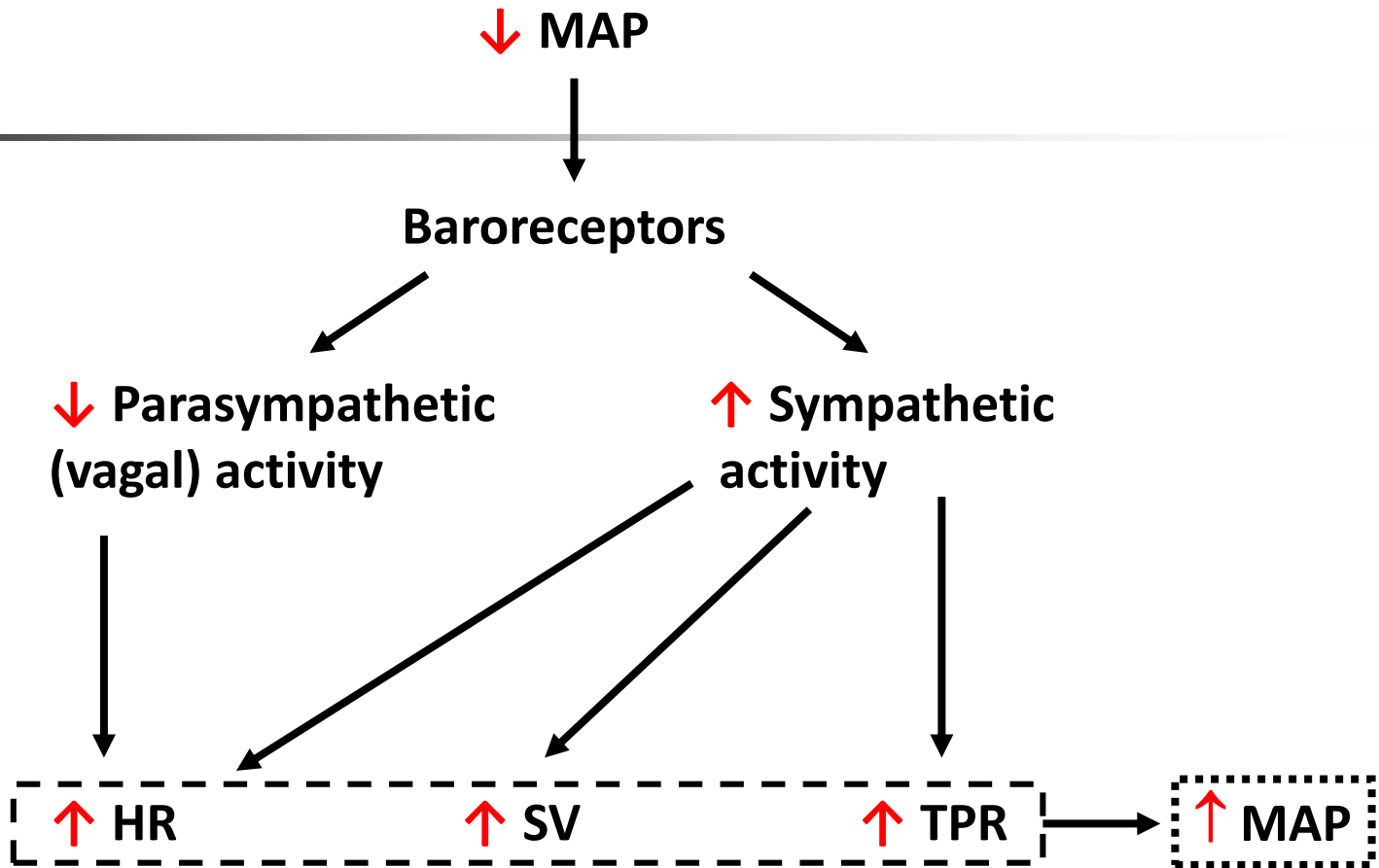
1. THE BARORECEPTORS



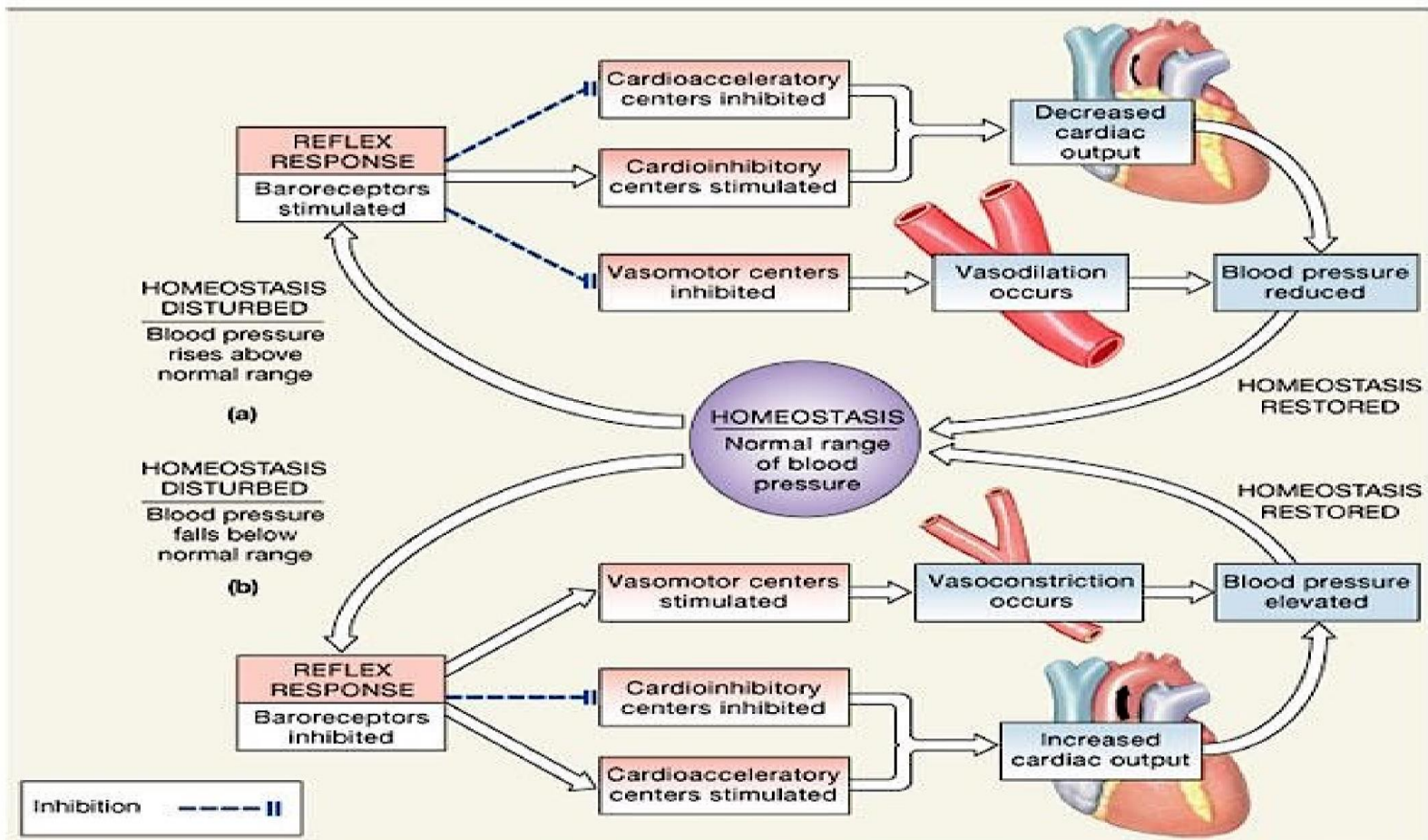
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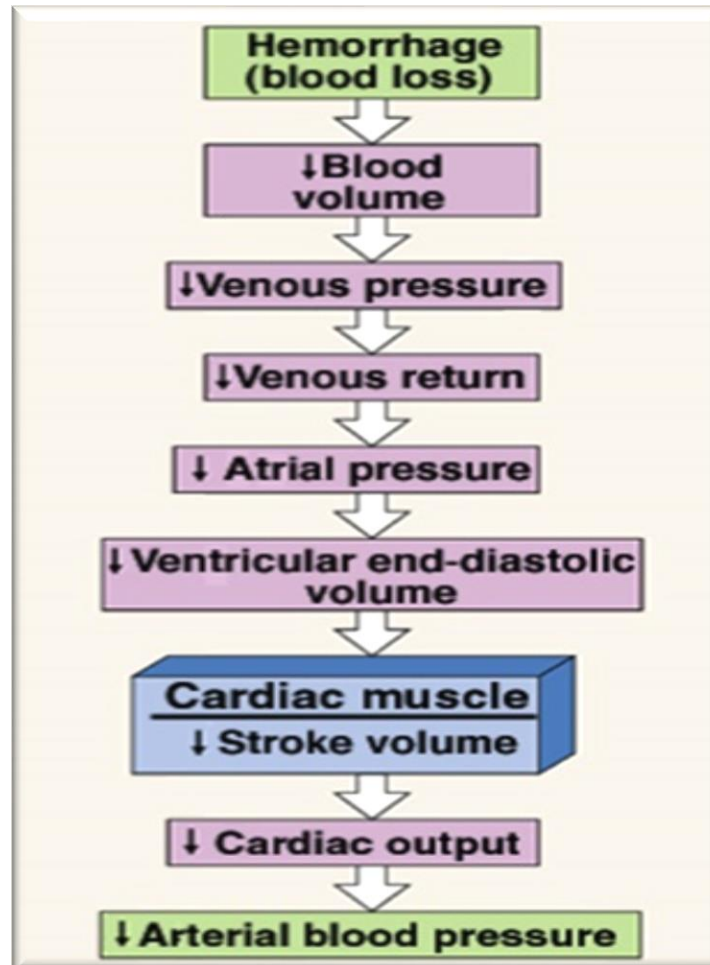




1. THE BARORECEPTORS

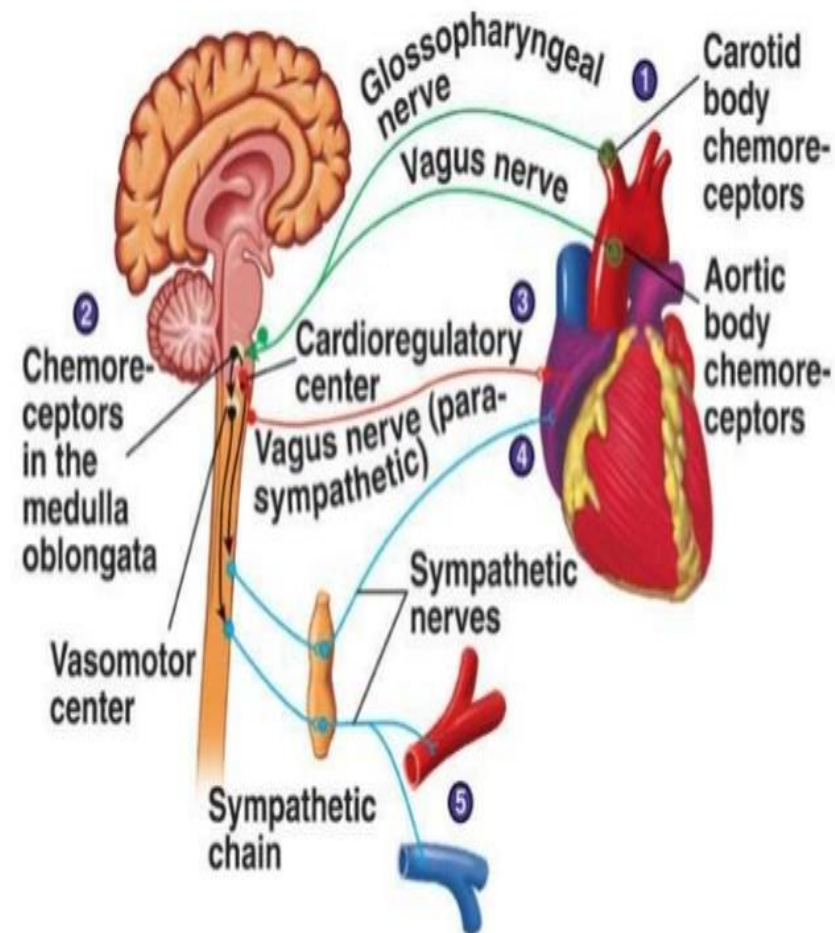
- ❑ Baroreceptors are important in maintaining MAP constant during changes in body posture.
- ❑ When change of posture from supine to erect, drop in the MAP in the head and upper part of the body.
- ❑ The baroreceptor reflex is activated → strong sympathetic impulses → vasoconstriction. This minimizes the drop in MAP.

ARTERIAL BARORECEPTOR REFLEX ROLE IN HEMORRAHAGE



2. THE CHEMORECEPTOR REFLEX

- ❑ Chemoreceptors have high blood flow (1200 ml/min/g tissue). This makes it easy for these cells to **detect changes in O_2 , CO_2 , and H^+** .
- ❑ Reduced blood flow (due to reduced MAP) stimulates the **chemoreceptors** through oxygen lack, increased hydrogen ions or carbon dioxide.
- ❑ **Chemoreceptors are stimulated when the MAP is lower than 60 mmHg.**
- ❑ Response is excitatory, NOT inhibitory; mainly through activation of sympathetic nervous system.
- ❑ They reduce blood flow to unessential areas and protect vital tissues like brain and heart.





3. CNS ISCHEMIC RESPONSE

- ❑ CNS ischemic response operates rapidly to prevent further decrease in MAP whenever blood flow to the brain decreases.
- ❑ It is one of the most powerful activators of the sympathetic vasoconstrictor system.
- ❑ When $MAP < 20$ mmHg → cerebral ischemia of vasomotor center → strong excitation of vasomotor center (due to accumulation of CO_2 , lactic acid,) → strong vasoconstriction of blood vessels including the kidney arterioles.



4. ATRIAL STRETCH VOLUME RECEPTORS

Receptors in large veins close to heart, walls of the atria (response of blood volume).

An increased blood volume → stretch of atria → activate atrial volume receptors → sensory afferent nerves to medulla → inhibiting the cardiovascular centre → **This results into decreased blood volume through:**

(a) → ↓ sympathetic drive to kidney:

- → dilate afferent arterioles → ↑ glomerular capillary hydrostatic pressure → ↑ GFR → ↓ blood volume (towards normal).
- ↓ renin secretion (Renin is an enzyme which activates angiotensinogen in blood). Inhibition of renin secretion → inhibit RAAS → inhibit aldosterone production → ↓ Blood volume (towards normal)

(b) → ↓ ADH secretion → ↓ blood volume (towards normal).

(c) → ↑ Atrial Natriuretic Peptide (ANP) causes loss of blood volume.



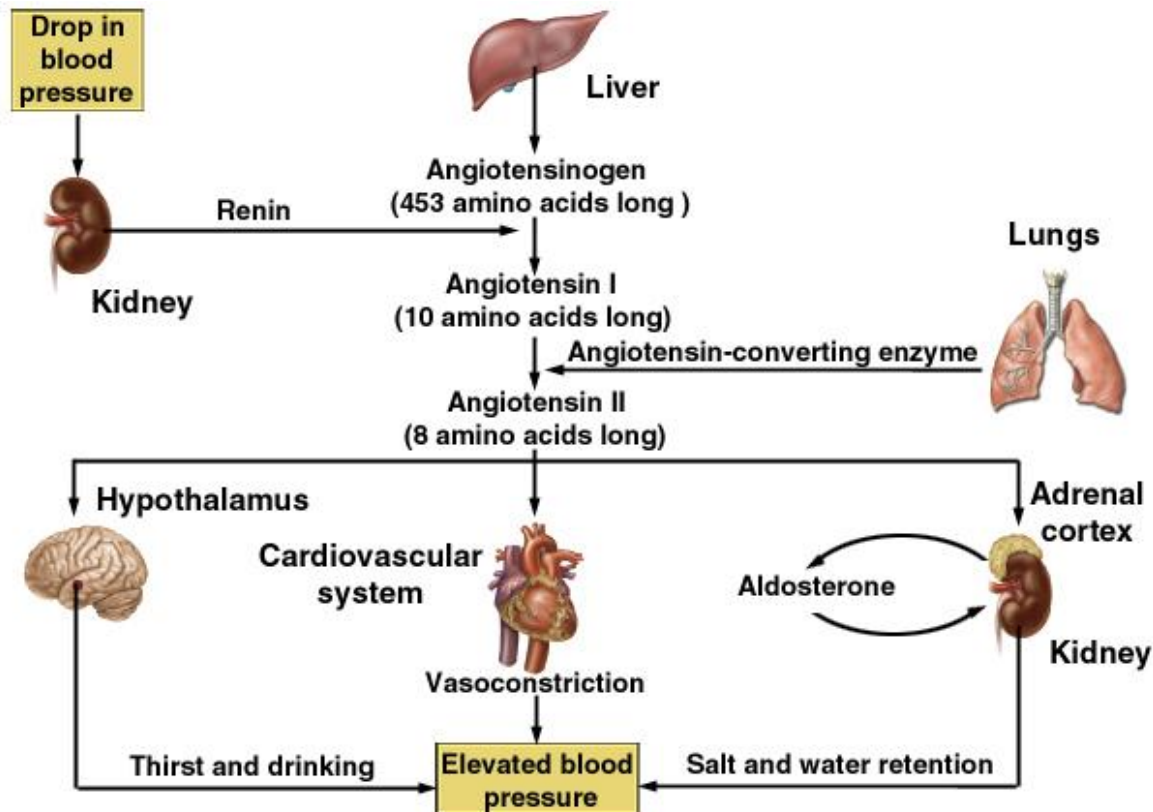
INTERMEDIATE TERM REGULATION OF BLOOD PRESSURE

Respond from 30 min to several hrs

1. Renin – Angiotensin system
2. Capillary shift mechanism
3. Stretch relaxation of vessels

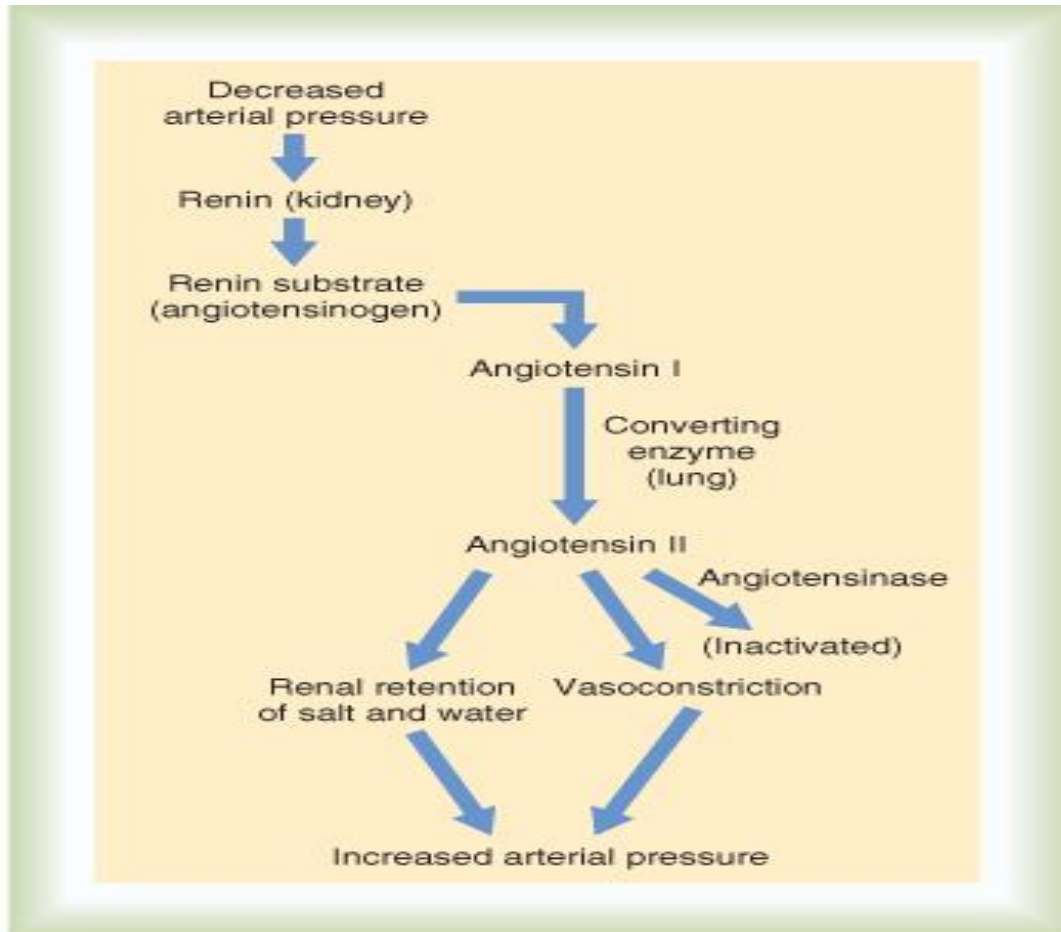
INTERMEDIATE TERM REGULATION OF BLOOD PRESSURE

1. Renin – Angiotensin system



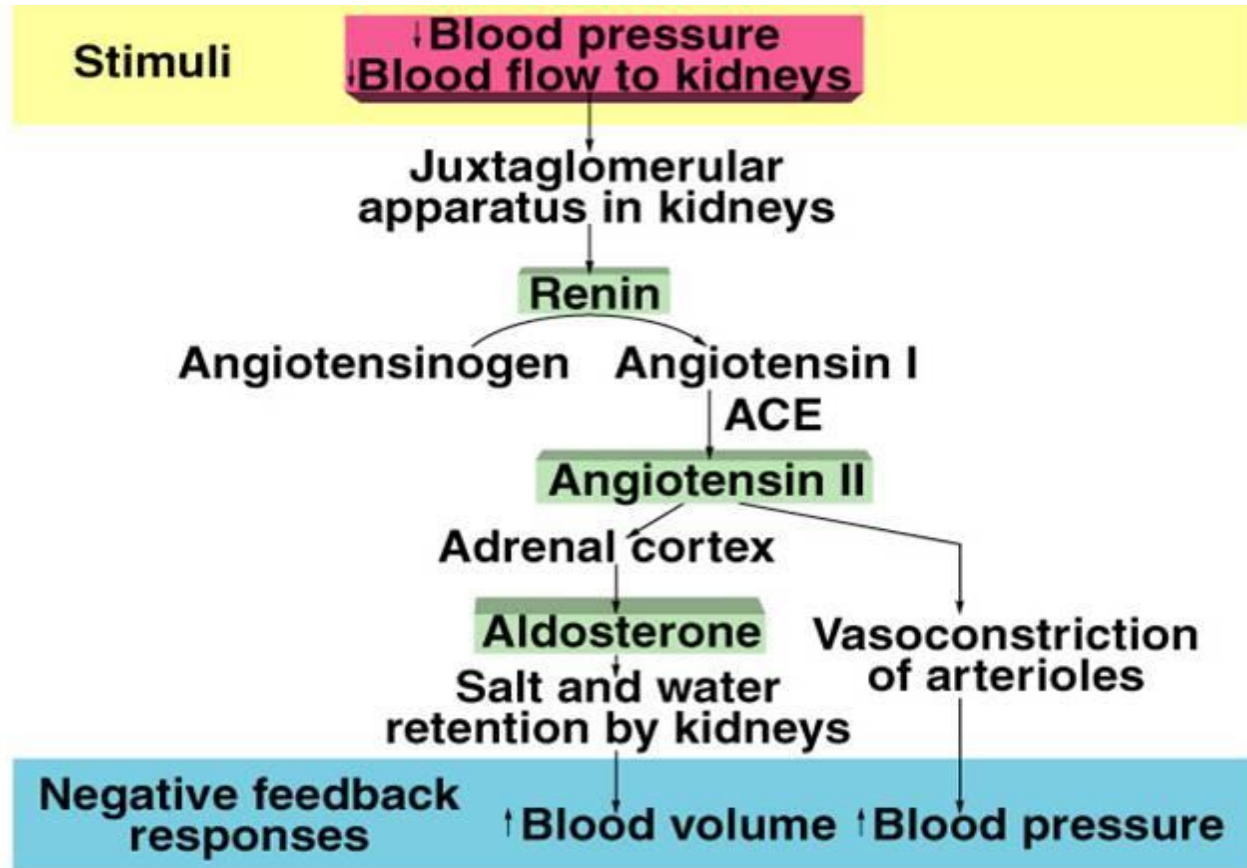
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1. Renin – Angiotensin system



INTERMEDIATE TERM REGULATION OF BLOOD PRESSURE

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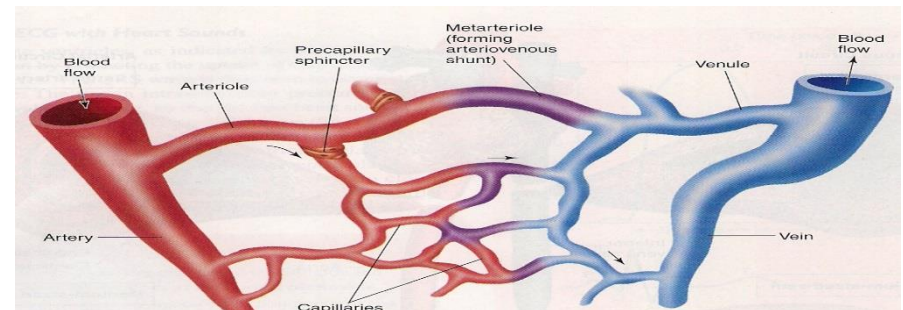


INTERMEDIATE TERM REGULATION OF BLOOD PRESSURE

2. Capillary shift mechanism

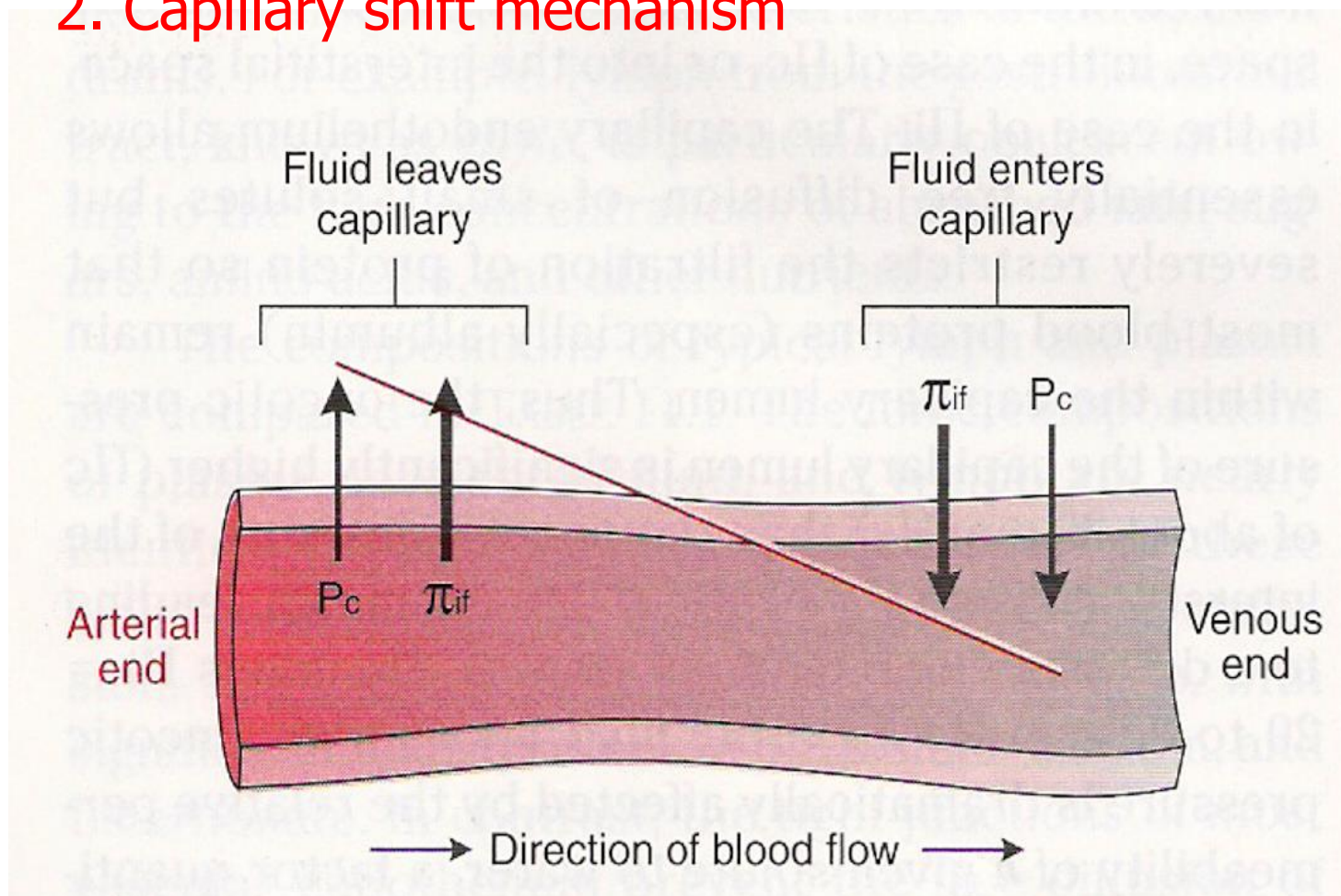
Movement of fluid from interstitial spaces into capillaries in response to \downarrow BP to maintain blood volume.

Conversely, when capillary pressure \uparrow too high, fluid is lost out of circulation into the tissues, reducing blood volume as well as all pressures throughout circulation



INTERMEDIATE TERM REGULATION OF BLOOD PRESSURE

2. Capillary shift mechanism





INTERMEDIATE TERM REGULATION OF BLOOD PRESSURE

3 Stretch Relaxation of Vessels

Blood vessel smooth muscle respond to changes in blood volume.

When pressure in blood vessels is “**too high**”, vessels stretched and stretching more and more for minutes or hours; resulting in decrease blood pressure in vessels toward normal.

The continuing stretch response of the vessels can serve as an intermediate-term pressure “buffer.”

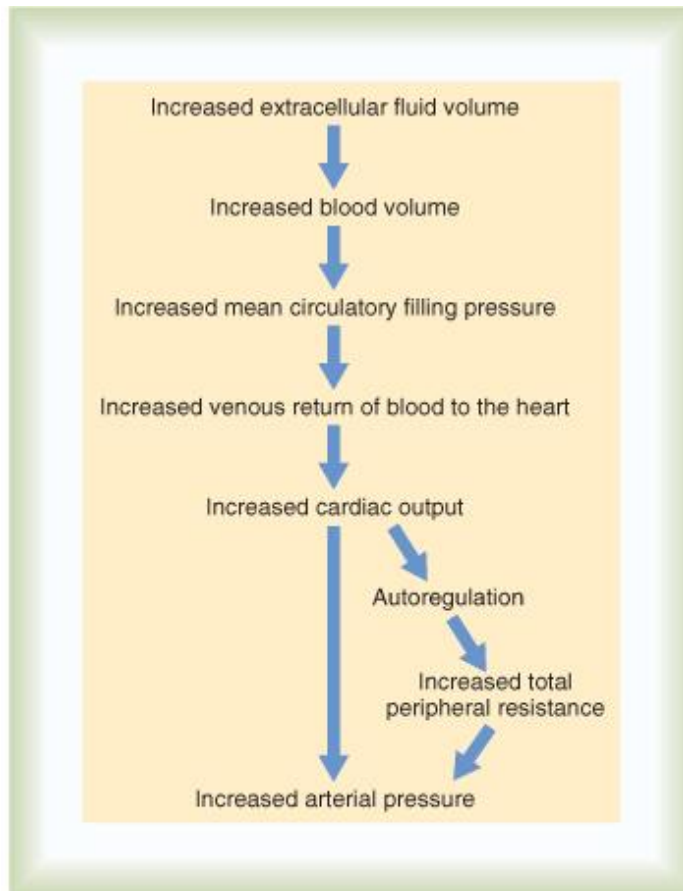


LONG TERM REGULATION OF BLOOD PRESSURE (acting within days to months)

1. Renal Body fluid control mechanism
2. Aldosterone Mechanism

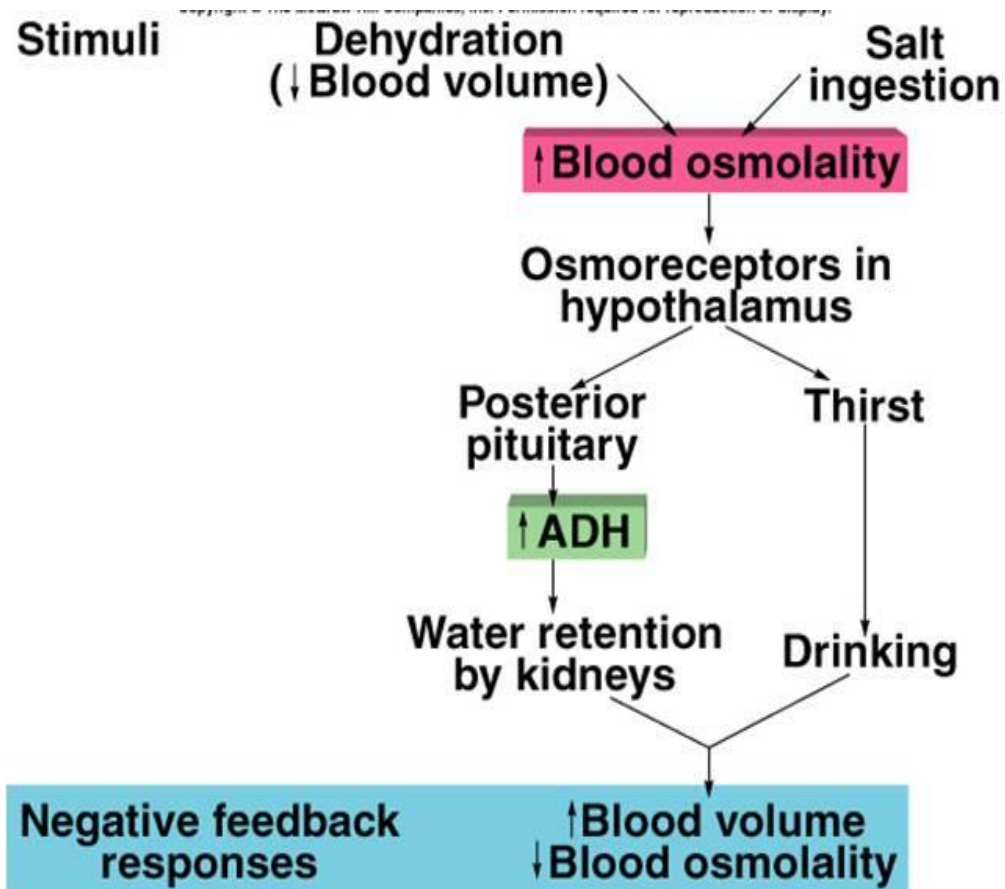
LONG TERM REGULATION OF BLOOD PRESSURE

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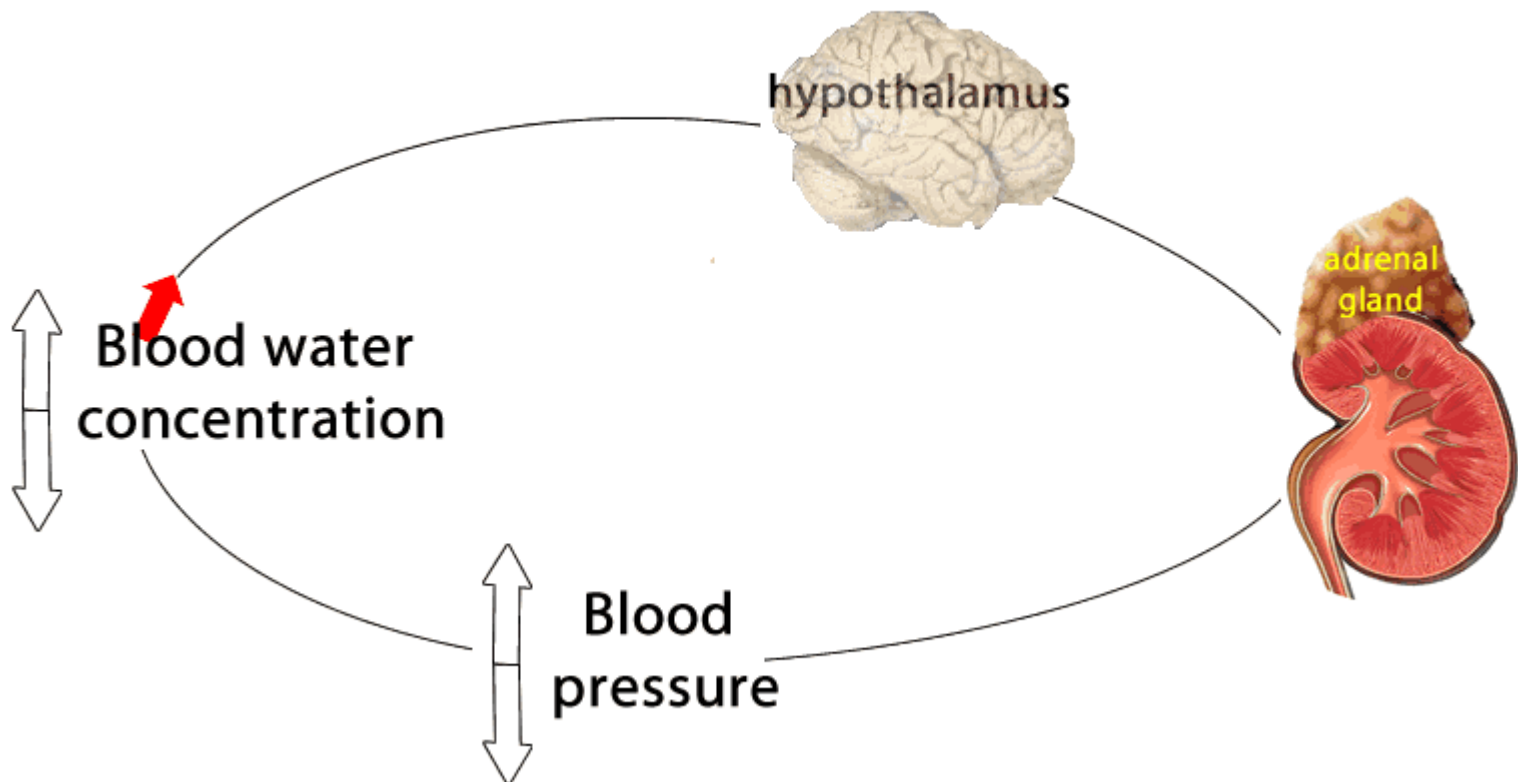
LONG TERM REGULATION OF BLOOD PRESSURE

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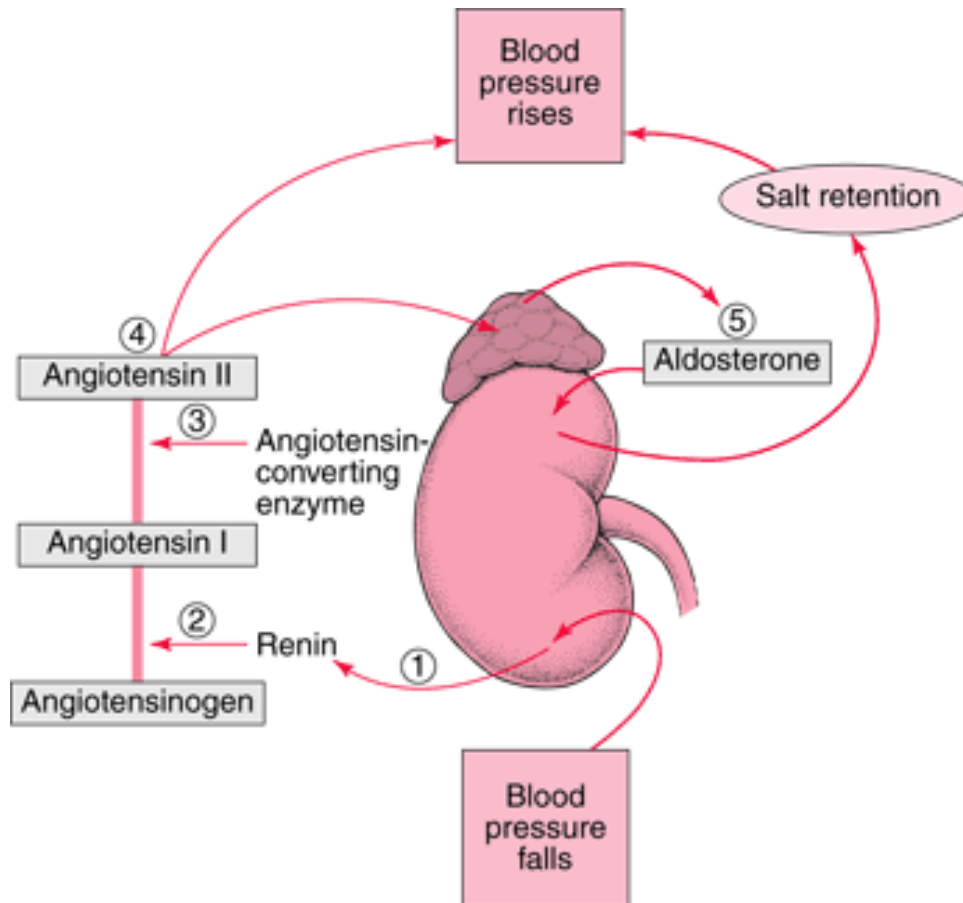
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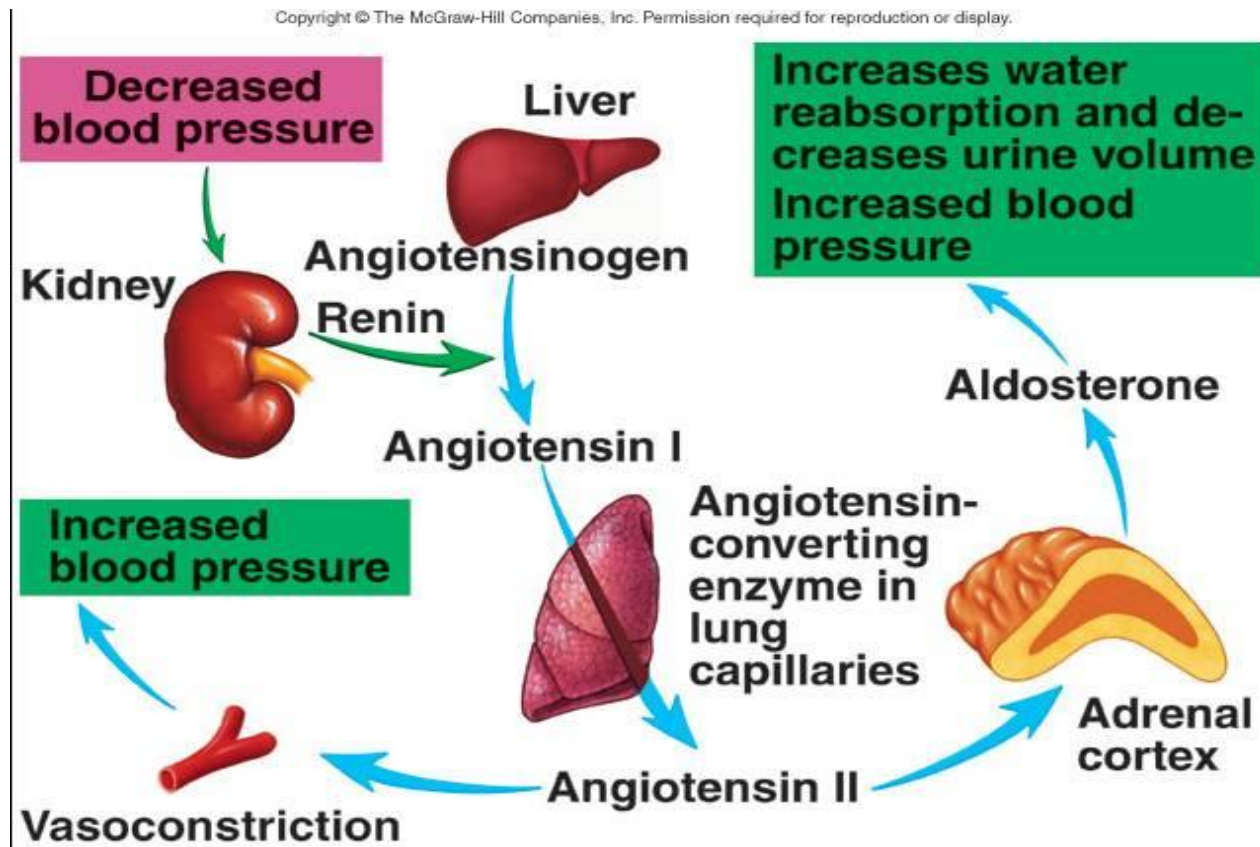
LONG TERM REGULATION OF BLOOD PRESSURE

2. Aldosterone Mechanism



LONG TERM REGULATION OF BLOOD PRESSURE

2. Aldosterone Mechanism





THANK YOU