

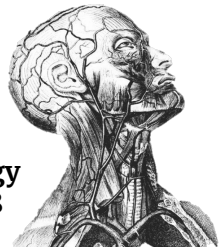
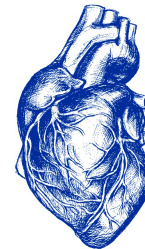


# Lecture 10

## Regulation of BP

- **Red: important**
- **Black:** in male / female slides
- **Pink:** in female slides only
- **Blue:** in male slides only
- **Gray:** extra information

[Editing file](#)



Physiology  
MED438

# Objectives:

- Factors regulate the 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.

# Blood pressure regulatory mechanisms

## Short term Sec - mins

Baroreceptors  
(excitatory + inhibitory)

Chemoreceptors  
(excitatory)

CNS ischemic response  
(excitatory)

Other vasomotor  
reflexes:

- Atrial stretch receptors (Inhibitory)
- Thermoreceptors (excitatory + inhibitory)
- Pulmonary receptors (excitatory)

## Intermediate Mins - hrs

Nervous mechanisms become less effective.

Renin-Angiotensin  
(excitatory)

Capillary Fluid shift  
(excitatory + inhibitory)

Stress-relaxation of  
vessels  
(inhibitory)

## Long term Days - months

Aldosterone  
(excitatory)

Vasopressin (or ADH)  
(excitatory)

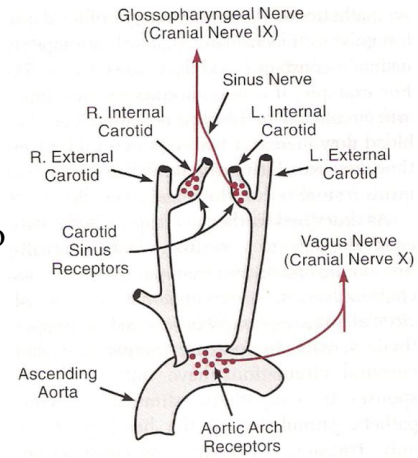
Natriuretic peptide  
(Inhibitory)

Erythropoietin  
(excitatory)

# Short term mechanisms

## 1. Baroreceptors:

- They are pressure receptors located in the **arch of aorta** and **carotid sinus**.
- They help maintain blood flow during **rapid** pressure changes i.e: standing up
- They are NOT used in long-term regulation of BP due to their tendency to get accustomed to the high pressure levels in 1-2 days.
- When MAP increases, the baroreceptors are stimulated, which will in turn **increase** parasympathetic activity and **decrease** sympathetic activity.
- The vagal activity decreases MAP back to the normal range.
- The opposite effect is seen when MAP decreases below normal.



Initial cause	Change in receptor	neural activity	Effect of neural activity	Final effects
Increase in MAP	1.Receptors are stretched 2. Increase firing rate	↑ vagal activity	↓ heart rate	- ↓ cardiac output - ↓ peripheral resistance - which decreases arterial pressure
		↓ sympathetic activity	1. ↓ heart rate 2. ↓ stroke volume 3. vasodilation	
Decrease in MAP	1. Decrease in stretch So, decrease firing rate	↓ vagal activity	↑ heart rate	- ↑ cardiac output - ↑ peripheral resistance - which increases arterial pressure
		↑ sympathetic activity	1. ↑ heart rate 2. ↑ stroke volume 3. vasoconstriction	

Receptors in the aorta and carotid sinus send impulses through the **vagus** nerve and the **glossopharyngeal** nerve respectively to the medulla to activate or inhibit the sympathetic and parasympathetic activity

**1**

## Body posture

When someone suddenly stands up from a supine position, arterial pressure **decreases**, causing the **baroreceptor** reflex to be **activated**, leading to **vasoconstriction**.

This is **important** to make sure the **brain** is supplied with enough oxygen.

## 2. Atrial stretch volume receptors:

- These receptors are found on **the walls of the atria**.
- As blood volume in the atria **increases**, the atrial wall is **stretched**, which activates the receptors.
- The receptors cause inhibition of the cardiovascular center in the medulla, which **decreases the blood volume** through the following:
  1. ↓ Sympathetic drive to the kidney → vasodilation of vessels → more fluid is lost → ↓ blood volume.
  2. ↓ Antidiuretic hormone (**ADH**) secretion → ↓ water & Na retention → ↓ blood volume.
  3. ↑ Atrial Natriuretic Peptide (**ANP**) causes loss of blood volume (reflex vasodilation of renal vessels, diuresis and natriuresis).
  4. Stretch of SA node → ↑ HR (Bainbridge Reflex).

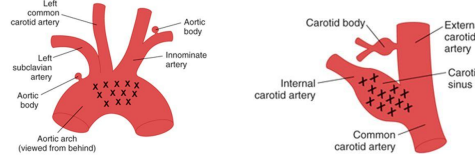
**2**

## Hemorrhage

Loss of blood causes a **decrease in volume and pressure**, which follows the same sequence of events as a decrease in MAP.

### 3. Chemoreceptors:

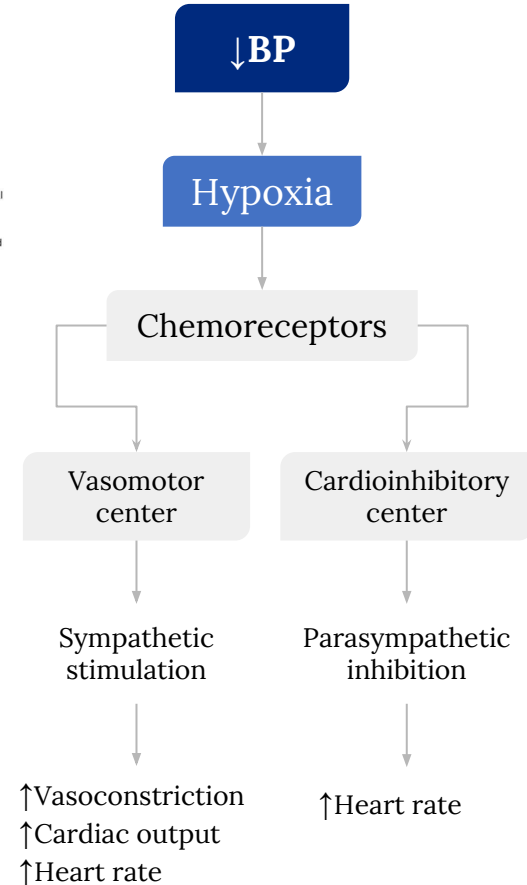
- Receptors that detect change in  $O_2$ ,  $CO_2$ , and  $H^+$ .
- They have high blood flow (1200 ml/min/g tissue) to make detection easier.
- Reduced blood flow causes receptors to sense:
  - Decrease in  $O_2$
  - Increase in  $CO_2$
  - Increase in  $H^+$
- Actions are **excitatory** (sympathetic activation), **NOT** inhibitory.
- Receptors are activated when Mean Arterial Pressure is **less than 60 mmHg**.
- They reduce blood flow to unessential areas and **protect vitals like brain and heart**.



#### Types of chemoreceptors:

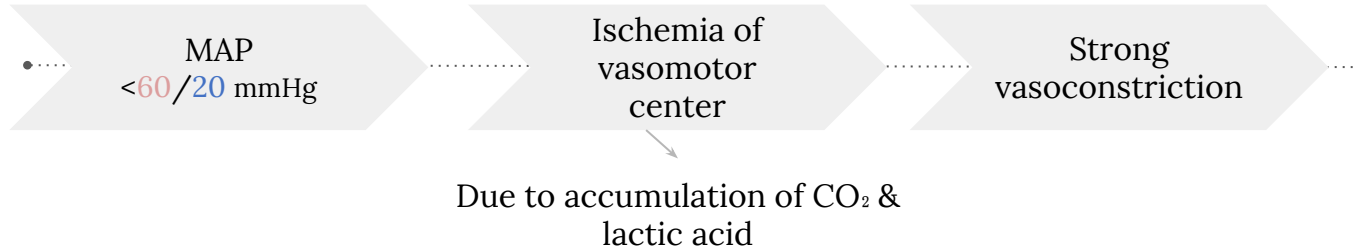
Peripheral	Central
Found in carotid & aortic bodies	Found in the medulla itself
Sensitive to $\downarrow O_2$ (hypoxia), $\uparrow CO_2$ , & $\uparrow H^+$	Highly sensitive to excess in $CO_2$ , and decrease in pH (increase $H^+$ )

- Since pressure has an effect on change in  $O_2$ ,  $CO_2$ , and  $H^+$ , and as mentioned previously volume is proportional to pressure, **hemorrhage has a similar effect on chemoreceptors**.



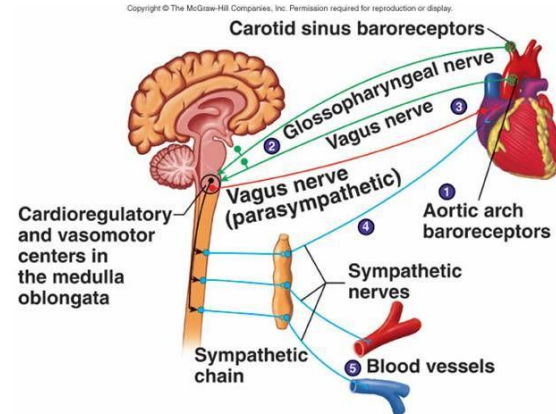
## 4. CNS ischemic response:

- Emergency pressure control mechanism that acts **rapidly with very powerful sympathetic activity** when blood flow to the brain ↓ dangerously .
- Prevents **further** decrease in MAP, acts at < 60/20 mmHg.



## 5. Other vasomotor (fast) mechanisms:

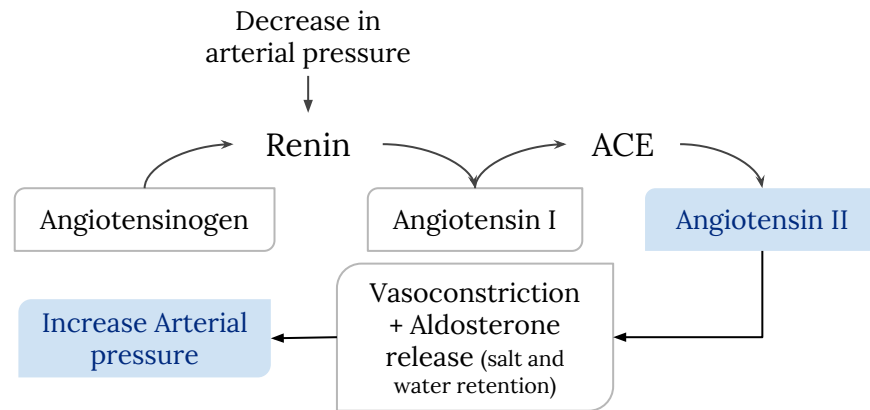
- **Thermoreceptors**
  - Heat → Vasodilation
    - Allows fluid to exit and absorb the heat.
  - Cold → Vasoconstriction
    - Allows the heat to be trapped in the system
- **Pulmonary receptors**
  - Lung inflation → vasoconstriction



# Intermediate mechanisms

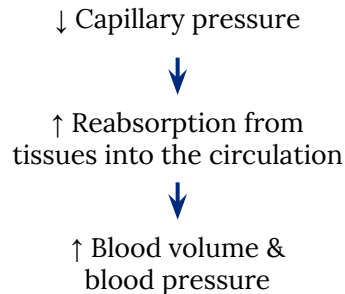
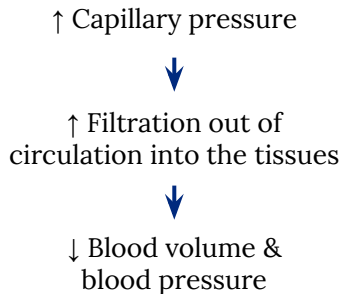
## 1. Renin-Angiotensin mechanism:

- **Low arterial BP triggers kidneys to release renin.**
- Renin converts Angiotensinogen → Angiotensin I, then into Angiotensin II by ACE in the lungs.
- Renin also **increases aldosterone** release, both lead to **increased arterial blood pressure.**



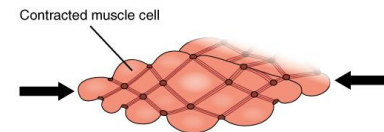
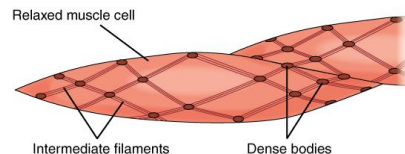
## 2. Capillary fluid shift mechanism (from high to low P)

- Shift of fluid through the tissue capillary walls in and out of the circulation to readjust the blood volume as needed.



## 3. Stretch Relaxation of Vessels

- Blood vessel smooth muscles can respond to changes in **blood volume.**
- When pressure is too high, they become stretched, causing a fall in pressure.
- This serves as a **buffer** for change in pressure.





# Long term mechanisms

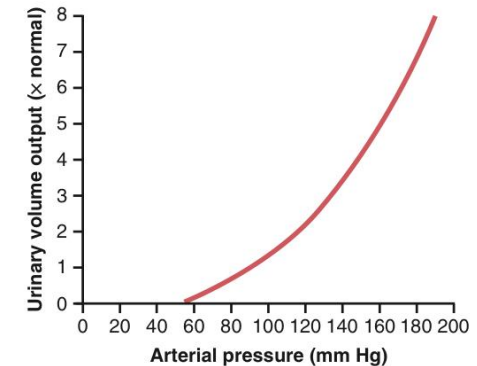
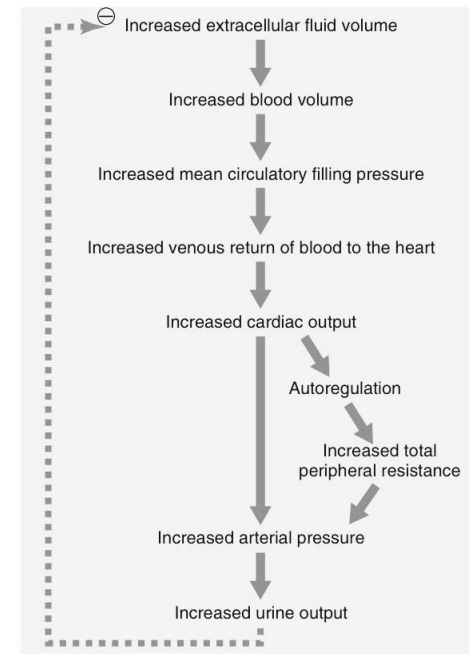
- **Hormonally-Mediated** regulation of arterial blood pressure.
- Concerned in regulating blood volume (mainly renal), acts if **BP is too low**.
- The **fundamental mechanisms** for long-term arterial pressure control are:
  - a. Renal body fluid control mechanism.
  - b. Aldosterone mechanism

## 1. Renal body fluid control mechanism:

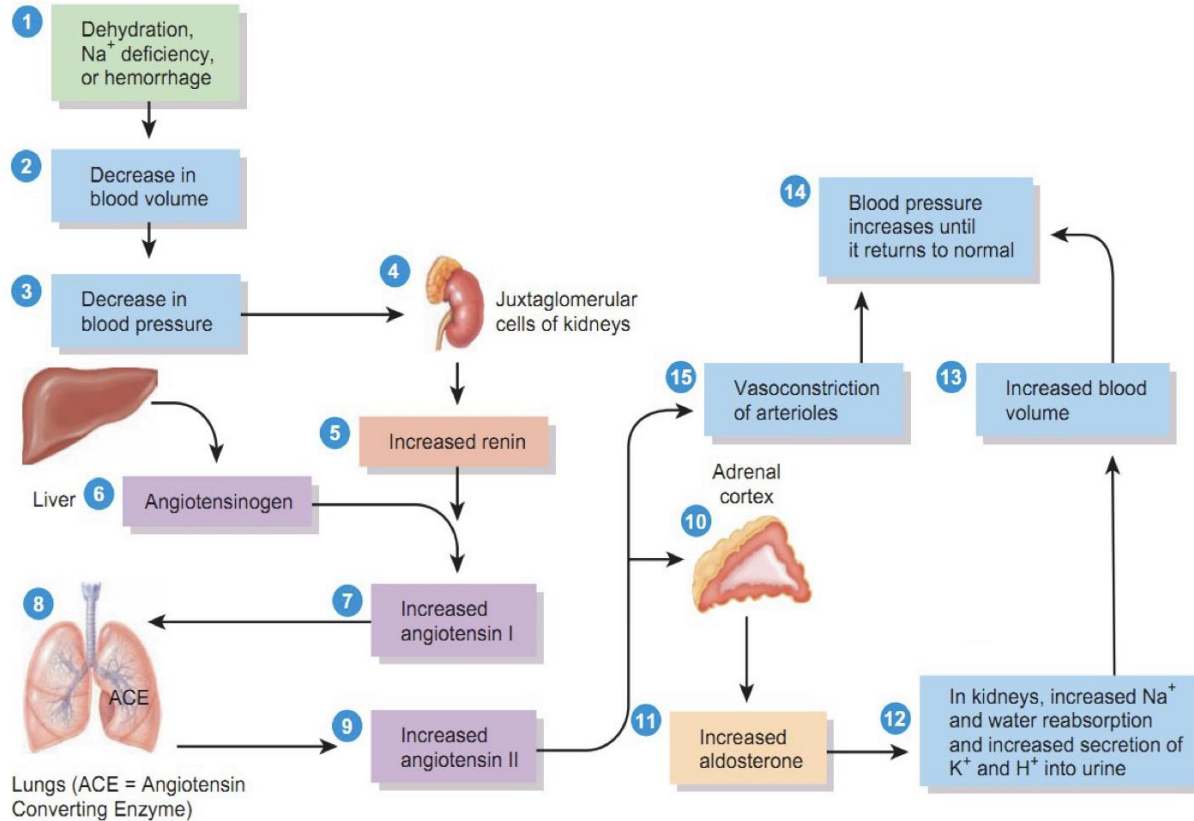
- When blood pressure falls, the kidneys retain water, increasing **blood volume** and returning the pressure back to normal.
- An increase in arterial pressure of only a few mmHg can **double renal output of water (pressure diuresis)**, as well as **double the output of salt (pressure natriuresis)**, thus returning the pressure back to normal.

## 2. Aldosterone mechanism:

- The Angiotensin II released during the intermediate mechanism stimulates the adrenal gland to secrete aldosterone.
- Aldosterone increases the kidney's Na<sup>+</sup> and water reabsorption, leading to an increase in blood volume.



# Aldosterone Mechanism:



### 3. Antidiuretic hormone (ADH), or vasopressin

↓ Blood volume  
(Hypovolemia & dehydration)  
↓ Blood pressure

↓  
Stimulates Hypothalamic  
Osmoreceptors

↓  
ADH will be released into bloodstream  
from storage in **posterior pituitary gland**

Promotes **water reabsorption**  
at kidney tubules

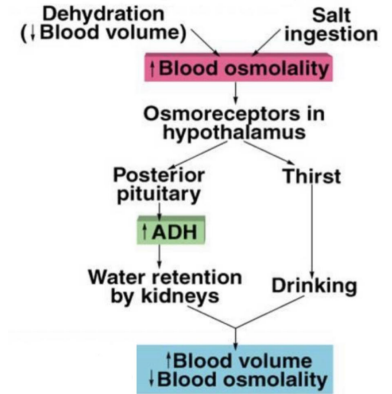
↓  
↑ Blood volume

**Vasoconstriction**

↓  
↑ ABP

**Thirst stimulation**

Usually when ADH is secreted,  
**aldosterone** is also secreted



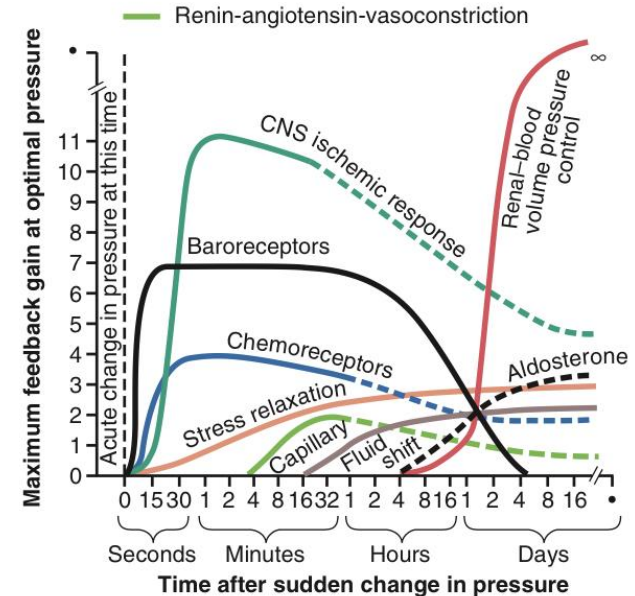
## 4. Atrial Natriuretic Peptide (ANP) hormone

- Hormone released from cardiac muscle cells (wall of the right atrium) in response to an increase in blood volume (**Low-pressure volume receptors**).
- Simulates an  $\uparrow$  in urine output (diuresis) and natriuresis, thus  $\downarrow$  blood volume & blood pressure.

## 5. EPO (Erythropoietin)

- Hormone secreted by the kidneys when blood volume is **too low**.
- It Leads to RBCs formation  $\rightarrow \uparrow$  blood volume.

## Potency of Various Arterial Pressure Control Mechanisms at Different Time Intervals After The Onset of a Disturbance to ABP



**Figure 19-17.** Approximate potency of various arterial pressure control mechanisms at different time intervals after the onset of a disturbance to the arterial pressure. Note especially the infinite gain ( $\infty$ ) of the renal body fluid pressure control mechanism that occurs after a few weeks' time. CNS, central nervous system.

# Quiz

1. Which of the following mechanisms acts when the arterial pressure reaches 20 mmHg?

- A. Baroreceptors reflex
- B. Capillary shift fluid
- C. CNS ischemic response
- D. Atrial stretch mechanisms

2. Which of the following hormones is released in response to a decrease in blood volume?

- A. Atrial Natriuretic Peptide (ANP) hormone
- B. Erythropoietin
- C. Renin
- D. Angiotensin II

3. Which of the following is true if the blood pressure increases?

- A. decrease firing of the baroreceptors
- B. increase firing of the baroreceptors
- C. increase sympathetic output
- D. decrease parasympathetic output

4. In the central chemoreceptors the sensory receptors located in the?

- A. Carotid bodies
- B. Aortic bodies
- C. Medulla
- D. Vagus nerve

5. Which one of the following mechanism play an important role in maintaining constant blood flow to vital organs?

- A. capillary shift mechanism
- B. renal body fluid control mechanism
- C. Chemoreceptors
- D. Baroreceptors

## SAQ:

1. Explain how vasopressin effects arterial blood pressure?

Slide 11

2. How does temperature affect the aperture of blood vessels?

Slide 7

# Leaders

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Abdulrahman Alhawas

# Members

- Lama AlZamil
- Arwa AlEmam
- Noura AlTurki
- Ghada AlSadhan
- Nouf AlShammari
- Nouf AlHumaidhi
- Taibah AlZaid
- Ajeed AlRashoud
- Reem AlGarni
- Raghad AlKhashan
- Leen AlMazroa
- Sara Alarifi
- **Maha AlNahdi**
- Badr Almuhana
- Abdulrahman Almezaini
- Omar Aldosari
- Omar Alghadir
- Ibrahim Alshaqrawi
- Abdullah Aldawood
- Abdullah Shadid
- Meshari Alzeer
- Mohammed Alhamad
- Abdullah Alassaf
- **Khalid Alkhani**
- Amjad Albaroudi
- Mohammed Alhuqbani

Thank you!