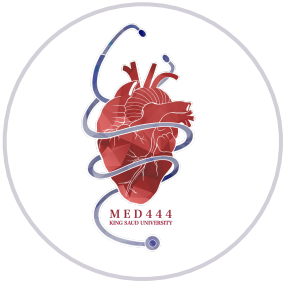
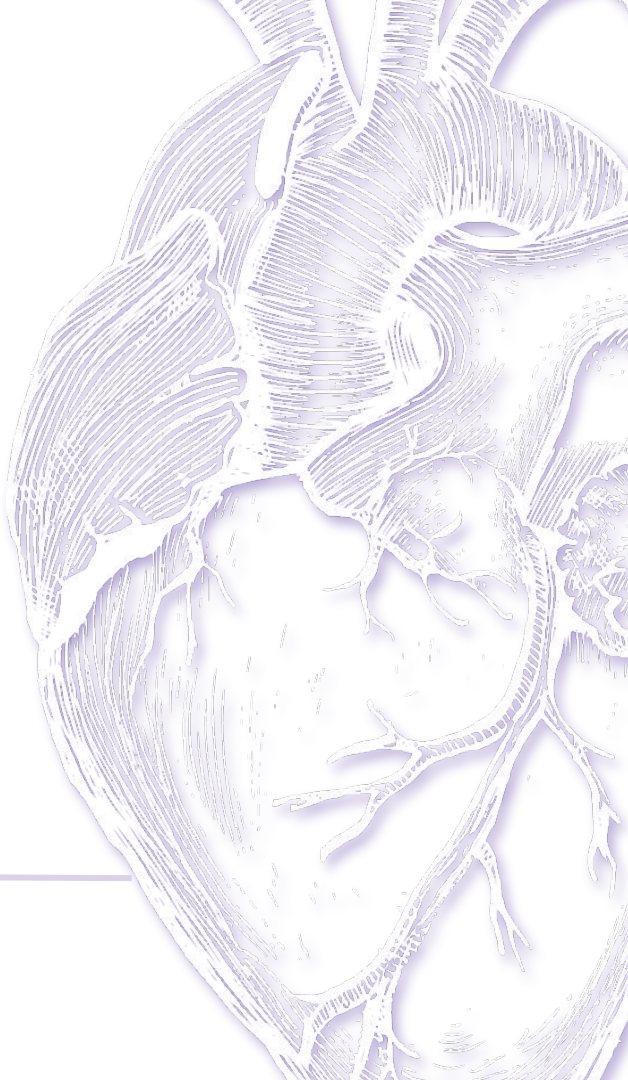




Regulation of blood pressure

Editing File

Main text Female's slide Male's slide Important text Doctor's note Extra

Objectives:

- To identify different regulatory mechanisms of arterial blood pressure.
- To understand Baroreceptors' short- term reflex regulatory mechanism of arterial blood pressure.
- To understand Chemoreceptors' short- term reflex regulatory mechanism of arterial blood pressure.
- To understand hormonal long- term regulatory mechanisms of arterial blood pressure.
- 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.

تيمنا هيل وفستقة وأحلى تيم بالمنطقة❤❤
أووووعاااااك يا أدم هذا تيم الفزيو 44 !!!

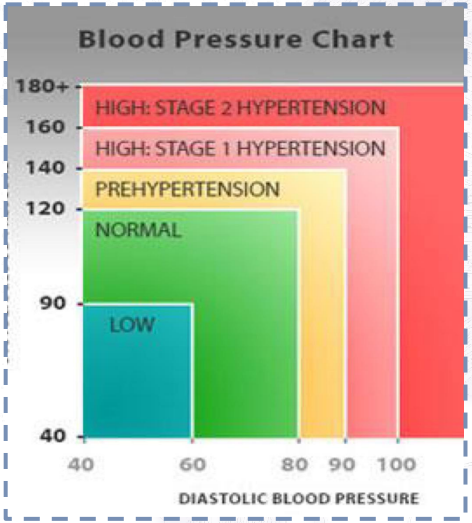
Regulation of Arterial Blood Pressure

Importance of ABP Regulation:

- Maintaining BP is important to ensure a steady blood flow & perfusion to the tissues.
- Inability to regulate blood pressure can contribute to diseases.

In order to regulate the blood pressure, the determining factors should be regulated:

- Cardiac output (Flow.)
- Peripheral Resistance.
- Blood volume.



Blood pressure =Cardiac output X Peripheral Resistance

BP= CO X PR

Important slide

Mechanisms Regulating The Mean Arterial Pressure

Don't be afraid this table will be clear if you check the next slides
لا تخافون لو ماقدرتوا تحفظونها بنتكلم عنها بالتفصيل بالسلايدات الجاية
(أهم شيء اعرفوا أنه في 3 responses بتشتغل لما يكون فيه انخفاض أو ارتفاع بالضغط) وهي التايتلز هنا ..

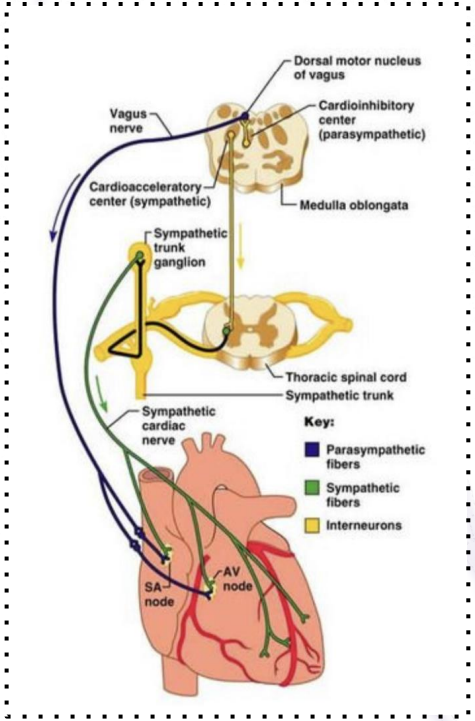
1)Naturally-mediated Fast response **(short-term)**	2)Intermediate	3)Hormonally- mediated Long term **(slow response)**
1A) Baroreceptor reflex (excitatory or inhibitory)	2A) Fluid (capillary) shift mechanism (inhibitory & excitatory)	3A) RAAS system (excitatory)
1B) Chemoreceptor reflex (excitatory)	2B) Stretch (Stress) - relaxation mechanism (inhibitory)	3B) ADH/Vasopressin (excitatory)
1C) CNS ischemic response (excitatory)	ADH Vasoconstriction system	3D) Natriuretic peptide (Inhibitory)
1D) Atrial stretch volume receptor reflex (inhibitory)	Renin - Angiotensin vasoconstriction	3E) Erythropoietin (Excitatory)
1E) Thermoreceptors (inhibitory & excitatory)	-	3F) Renal body fluid control
1F) Pulmonary receptors (excitatory)	-	-

1) Rapidly Acting Control Mechanisms

لو يكون فيه ارتفاع أو انخفاض بالضغط مباشرة هالسستم يبدأ يشتغل بثواني

بمعنى أنه يشتغل سواء ارتفاع أو انخفاض بالضغط (excitatory or inhibitory)

- Acts Within sec/min (short-term).
- Concerned in regulating the Cardiac Output (CO) & the Peripheral Resistance (PR).
- Reflex mechanisms act through the autonomic nervous system (ANS):



Centers in Medulla Oblongata

Cardio-inhibitory center(CIC)

Parasympathetic nervous system

لا يكون ارتفاع يشتغل عشان يقلل الضغط

Cardio-accelerators center(CAC) or vasomotor center

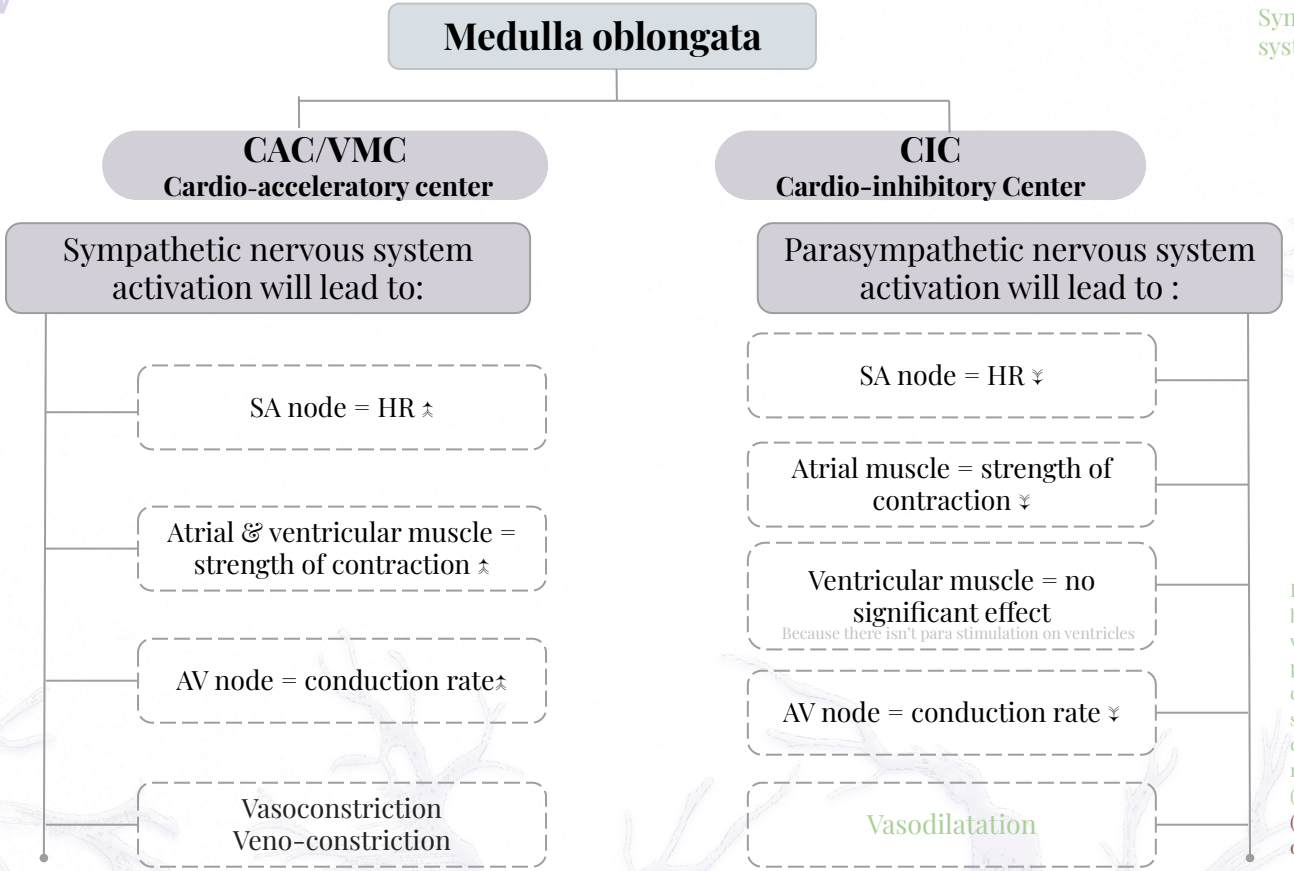
Sympathetic nervous system

لا يكون انخفاض يشتغل عشان يرفع الضغط

Female's slide

Effects of Autonomic Nervous System Stimulation

Sympathetic and Parasympathetic systems don't work in the same time



Parasympathetic only affect the HR directly, but conduction and contractility and vasodilatation are affected indirectly. When parasympathetic is on, the sympathetic is off, so there will be inhibition of sympathetic actions (vasoconstriction, contractility), when those actions inhibited, normally it will be the opposite which is (vasodilatation, decrease contractility)... (the parasympathetic only have direct effect on HR, other effects are indirect)

This slide is mix between male and female slide

Here is the start of short term and rapidly acting mechanism

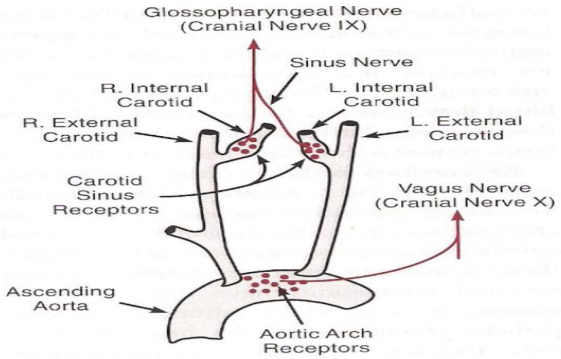
1A)The Baroreceptors(short term)

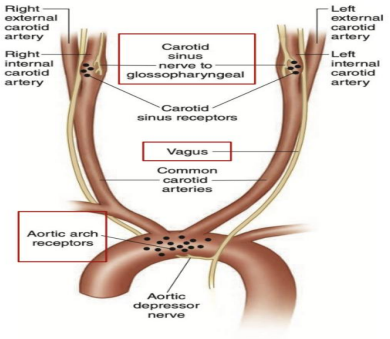
Baroreceptors are Mechano-stretch receptors high- pressure receptors..
Fast & neurally mediated

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

Changes in MAP are detected by baroreceptors (pressure receptors) in the carotid and aortic arteries & stimulated in response to blood pressure changes

These receptors provide powerful moment-to-moment control of arterial blood pressure and on stimulation will send sensory signals (information) through Glossopharyngeal nerve fibers to the required cardiovascular centres in the medulla oblongata about the degree of stretch with pressure changes.





Here is the start of short term and rapidly acting mechanism

Male's slide

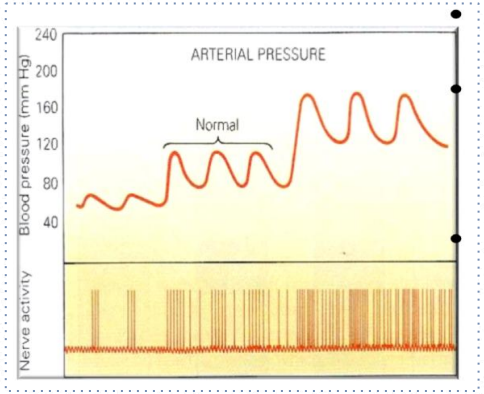
1A)The Baroreceptors(short term)

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).

فَلْيَنْظُرِ الإِنْسانُ مِمَّ خُلِق

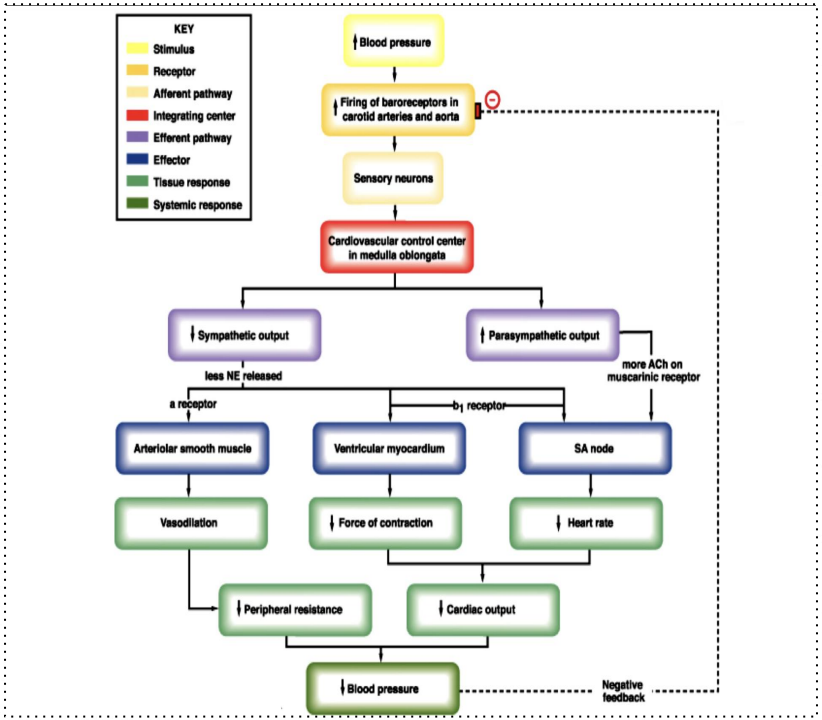
They play an important role in and maintaining relatively constant blood flow to vital organs such as brain during rapid changes in pressure such as standing up after lying down. That is why they are called **"pressure buffers"**.



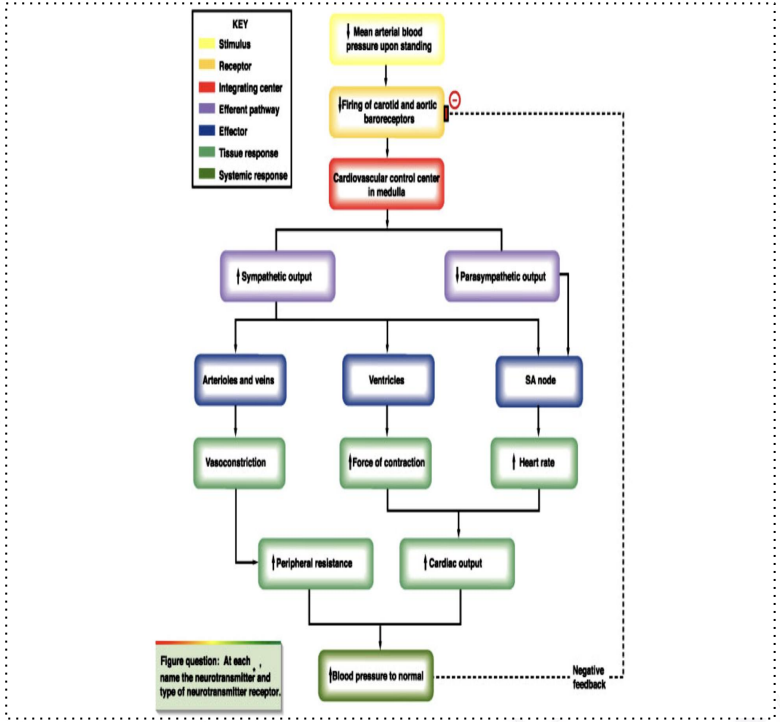
Important slide

Baroreceptors reflex to changes in arterial pressure

Increase in arterial pressure



Decrease in arterial pressure



Baroreceptor Reflex Mechanism During Changes in Body Posture

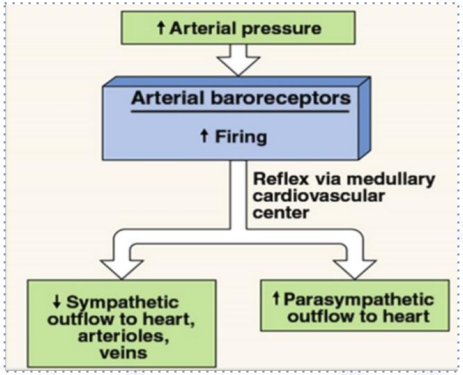
Baroreceptors are important in maintaining MAP constant during changes in body posture.

On immediate standing, from supine to erect , (MAP) Arterial Pressure in the head & upper parts of the body tends to fall, which may cause loss of consciousness.

Falling pressure will elicit an immediate reflex at the Baroreceptors, resulting in strong sympathetic discharge throughout the body.

This immediate reflex mechanism minimizes the drop in the arterial pressure that occured in the head & upper parts of the body

baroreceptor reflex ↠ Inhabited ↠ strong sympathetic impulses ↠ vasoconstriction. This minimize the drop in MAP.



1B/Chemoreceptor reflex (Rapidly Acting)

هو نفس الي قبل بس يختلفبمواقع الرسبتورس وكلش بس بدل لا يستجيب sterach يستجيب للتغيرات الي تصير بال02 ,H, co2

- **Chemoreceptors** are closely associated with the baroreceptors' control pressure system.
- Chemoreceptor reflex **operates in much same way** as the baroreceptor reflex, EXCEPT that chemoreceptors are **chemo-sensitive cells instead of stretch receptors**.
- Reduced blood flow & reduced Mean Arterial Pressure MAP, **will stimulates the chemoreceptors chemically** through oxygen lack(↓[O2]), increased hydrogen ions(↑[H+]) & / or increase carbon dioxide(↑[CO2]).
- Chemoreceptors have high blood flow (1200 ml/min/g tissue), which make it easy for these cells to detect changes in O2, CO2, & H+.
- Chemoreceptors are stimulated when the MAP is lower than 60 mmHg. Their response is **excitatory, NOT inhibitory**; **acts mainly through activation of sympathetic nervous system.**
  يعني يرفع الضغط فقط ماينزله
- They reduce blood flow to unessential areas and protect vital tissues like brain and heart

Important slide

Female's slide

Type of Chemoreceptor reflex

Chemoreceptors

Peripheral (arterial) Chemoreceptors

- **Sensory receptors** located in **carotid & aortic bodies.**

- Sensitive to **O2** lack (↓), **CO2** (↑or↓), & **pH** changes (↑or↓).

- Their stimulation will excite the sensory nerve fiber, along with the baroreceptor excitatory fibers

- They are stimulated when the MAPis lower than **60 mmHg**.

Central Chemoreceptors

- **Sensory receptors** located in the **medulla oblongata itself**.

- Very sensitive to **CO2** excess (↑) & drop (↓) **pH** in medulla.
- Not sensitive to peripheral **O2** lack (↓)

- They are stimulated when the **MAP** is **lower than 20 mmHg** with high accumulation of local CO2& lactic acid.

Baroreceptor and chemoreceptors reflexes role in HEMORRHAGE :

Peripheral Chemoreceptor Reflexes

ARTERIAL BARORECEPTOR REFLEX ROLE IN HEMORRAHAGE

- Hemorrhage
- ↓ Blood volume
- ↓Venous pressure
- ↓ Venous return
- ↓ Atrial pressure
- ↓Ventricular EDV
- Cardiac muscle ——— ↓SV
- ↓ Cardiac output
- ↓ Arterial blood pressure

Chemoreceptors reflexes usually act to BP

↓↓BP ← Hemorrhage

Hypoxia

Peripheral Chemoreceptors

++ vasomotor Center (VMC)

=Cardioinhibitory center (CIC)

+Sympathetic Nerve Fibers

=parasympathetic nerve fibers

+Adrenal Medulla

↑↑Sympathetic Effect

Vasoconstriction

↑Cardiac Contractility

↑Heart Rate (HR)

↑Total Peripheral Resistance (TPR)

↑Cardiac Output (CO)

Rapidly Acting: 3C) CNS Ischemic Response

"Last Ditch Stand" Pressure Control Mechanism

1. It is not one of the normal regulatory mechanisms of ABP. It operates principally/rapidly as an Emergency Pressure Control system to prevent further decrease in arterial blood pressure/(MAP).

2. It's one of the most powerful activators of the sympathetic vasomotor (vasoconstrictor system) nervous control areas in medulla oblongata.

3. It acts rapidly & very powerfully.

4. It acts whenever blood flow to the brain ↓ dangerously close to the lethal level Mean arterial pressure (MAP) < 20 mmHg ↠ cerebral ischemia of vasomotor center ↠ strong excitation of vasomotor center due to high accumulation of local CO_2 & lactic acid), in order to prevent further drop in the MAP ↠ strong vasoconstriction of blood vessels including the kidney arterioles.

Rapidly Acting: Other Vasomotor Reflex:

1D) atrial stretch receptor reflex

Are low-pressure Receptors found in large veins close to heart & in the atria wall (response of ↑ blood/Plasma volume). من اسمها تعرفون موقعها

↑ Venous Return (increase blood volume) ↠ ++ stretch atria & activate atrial stretch/volume receptors ↠ sensory afferent nerves to medulla ↠ **inhibiting** sympathetic in **the cardiovascular center** ↠ reflex vasodilation, more inhibition of Renin & anti diuretic hormone (ADH) secretion, leads to reflex increase urine excretion to decrease in blood volume towards normal & ↓ABP through:

1. ↓sympathetic drive to kidney (reflex vasodilation) :

- ↠ dilate afferent arterioles ↠ ↑ glomerular capillary hydrostatic pressure ↠↑glomerular filtration rate (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 ↠↓Na & water retention &↑urine excretion ↠↓ Blood volume (towards normal).

2. ↓ ADH secretion :
↓Water retention, ↓ blood volume (towards normal).

3. ↑ Atrial Natriuretic Peptide (ANP) :
causes ↑urine excretion (↓ of blood volume).

Rapidly Acting: Other Vasomotor Reflexes

- 1E)Thermoreceptors: (in skin/hypothalamus)
  - Exposure to heat ↠ vasodilatation (team 438: Allows fluid to exit and absorb the heat).
  - Exposure to cold ↠vasoconstriction (team 438 : Allows the heat to be trapped in the system).
- 1F)Pulmonary receptors:
  - Lung inflation ↠ vasoconstriction

2)Intermediate Mechanisms Regulating ABP

Activated/ respond from within 30 min to several hrs

During this time, the nervous mechanisms usually become less & less effective يعني
على مايخلص الأول مباشرة يبدا يشتغل ذا ويكون الاول خلص

Intermediate regulatory mechanisms is mediated by vasoactive compounds which will have effects on vasculature.

Vasoactive compounds will modify the amount of resistance in the systemic circulation(systemic vascular resistance – SVR) by targeting arterioles.

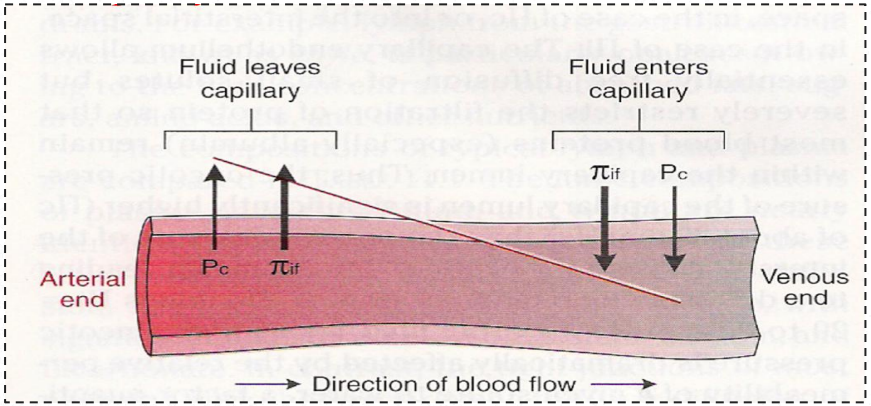
1. Fluid (capillary)-Shift mechanism. (M+F slide)
2. Stress-relaxation of the vasculature.(M+F slide)
3. Biogenic amines.
4. ADH vasoconstriction mechanism.
5. Atrial natriuretic peptide (ANP)
6. Renin Angiotensin-II vasoconstriction mechanism.(M+F slide)

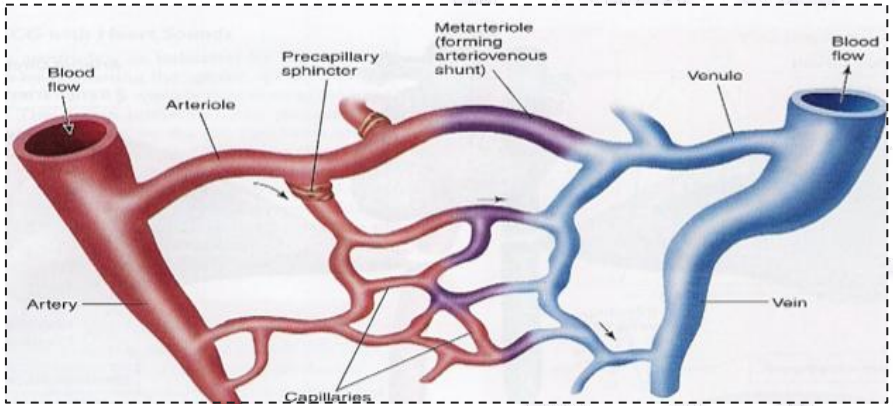
will discuss these(5 ,6) in long term regulation
بتقولون كيف؟ الميقانزم حقتها طويلة وتحتاج وقت فنبدا من عند الانترميديت بس ماتوقف مثل الباقين لا تكمل برضو وتدخل على اللونق

Intermediate Mechanisms Regulating BP

Fluid (Capillary) - Shift mechanism

- Movement of fluid from interstitial spaces into capillaries in response to ↓ BP to maintain blood volume,& ↑ pressure in the circulation.
- Conversely, when capillary pressure ↑ too high, capillary pressure ↑fluid is lost out of circulation into the tissues, reducing blood volume as well as all pressures throughout circulation.





Important slide

Intermediate Mechanisms Regulating BP

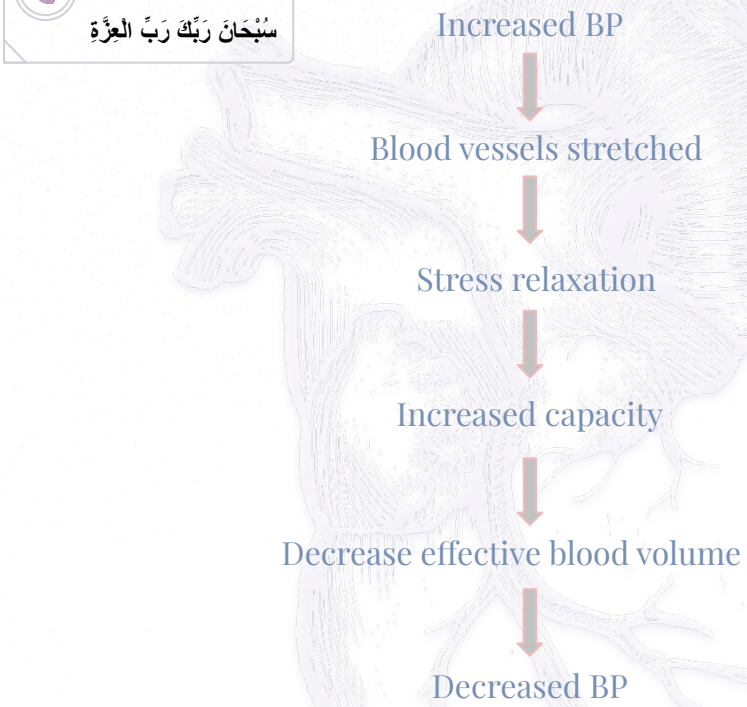
Stretch-stress relaxation of vessels

سُبْحَانَ رَبِّكَ رَبِّ الْعِزَّةِ

1. Adjustment of blood vessel smooth muscle to respond to changes in blood volume.

2. When pressure in blood vessels becomes too high, they become stretched & keep on stretching more & more for minutes or hours; resulting in fall of pressure in the vessels toward normal.

3. This continuing stretch of the vessels can serve as an intermediate-term pressure "buffer."



Biogenic Amines

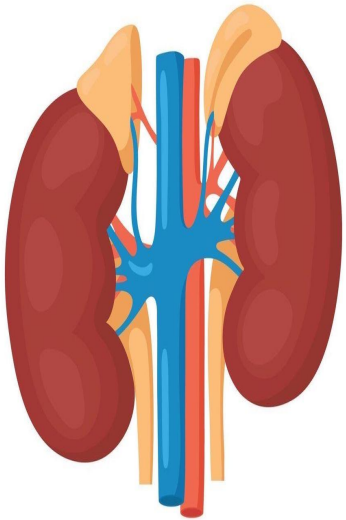
Vasoconstrictors	Vasodilators
Epinephrine via α1 Serotinin, etc.	Epinephrine via β2 Histamine ANP, etc.

3)Long term (slow response) regulation:

- Hormonally mediated.
- Takes few hours to begin showing significant response.
- acting within days to months
- **Mainly renal** Body fluid control mechanism **:** Acts if BP is too low

A. Renin-Angiotensin-Aldosterone System.
B. Vasopressin Anti-diuretic hormone (ADH) Mechanism.

Others:
C. Atrial Natriuretic Peptide Mechanism (Low-pressure volume receptors.)
D. Erythropoietin (EPO).



Important slide

3A)Renal body fluid control mechanism
(Renin-Angiotensin Aldosterone System)

1.A decrease in blood pressure causes a decrease in renal perfusion pressure, which is sensed Juxtaglomerular apparatus of kidneys (considered volume receptors) these cells will secrete renin into the bloodstream.

2. Renin is an enzyme. In plasma, renin catalyzes the conversion of **angiotensinogen** to **angiotensin I.**

3. In the lungs, angiotensin I is converted to **angiotensin II**, catalyzed by **angiotensin-converting enzyme (ACE).**

Angiotensin II acts on the cells of the **adrenal cortex** to stimulate the synthesis and secretion of aldosterone. **Aldosterone** then acts on the principal cells of the renal distal tubule and collecting duct to **increase Na^+ reabsorption** and, thereby, to increase ECF volume and blood volume. The actions of aldosterone require gene transcription and new protein synthesis in the kidney. These processes require hours to days to occur and account for the slow response time of the renin–angiotensin II–aldosterone system.

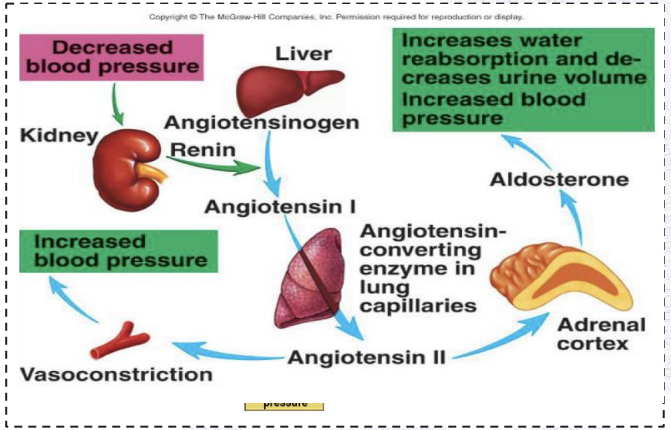
Thx to 443

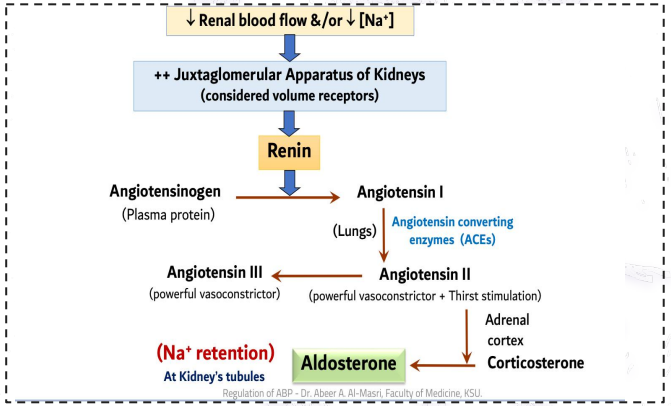
Angiotensin II also has its own direct action on the **kidney**, independent of its actions through aldosterone. Angiotensin II **stimulates Na^+–H^+ exchange** in the renal proximal tubule and increases the reabsorption of Na^+.

Angiotensin II acts on the hypothalamus to increase **thirst** and water intake. It also stimulates secretion of **antidiuretic hormone (ADH)**, which increases water reabsorption in collecting ducts. By increasing total body water, these effects complement the increases in Na^+ reabsorption (caused by aldosterone and Na^+–H^+ exchange), thereby increasing ECF volume, blood volume, and blood pressure.

Angiotensin II also acts directly on the **arterioles** to cause vasoconstriction. The resulting **increase in TPR** leads to an increase in blood pressure

It also can be converted to angiotensin III, which is also a powerful vasoconstrictor.





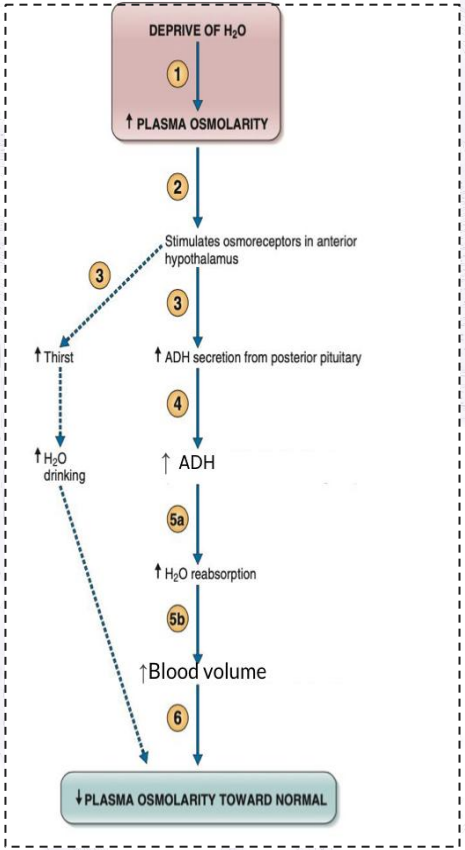
Female's slide

Renal Body fluid control mechanism

3B) (Anti-Diuretic Hormone (ADH), or Vasopressin)

ADH secretion from the posterior pituitary is increased by two types of stimuli: by increases in serum osmolarity (high salt intake) and by hypovolemia (dehydration) and blood pressure.

1. Hypovolemia & dehydration stimulates Hypothalamic Osmoreceptors
2. ADH will be released from posterior Pituitary gland:
   Causes vasoconstriction, in order to ↑ arterial blood pressure (ABP).
   Promotes water reabsorption at kidney tubules to ↑ blood volume.
3. Stimulates thirst & drinking.
4. Usually, when ADH is secreted, Aldosterone is secreted.



Renal Body fluid control mechanism

Negative feedback response

Increased extracellular fluid

Increased blood volume

Increased mean circulatory filling pressure

Increased venous return of blood to heart

Increased cardiac output

Increased arterial pressure

Increased urine output

Autoregulation

Increased TPR

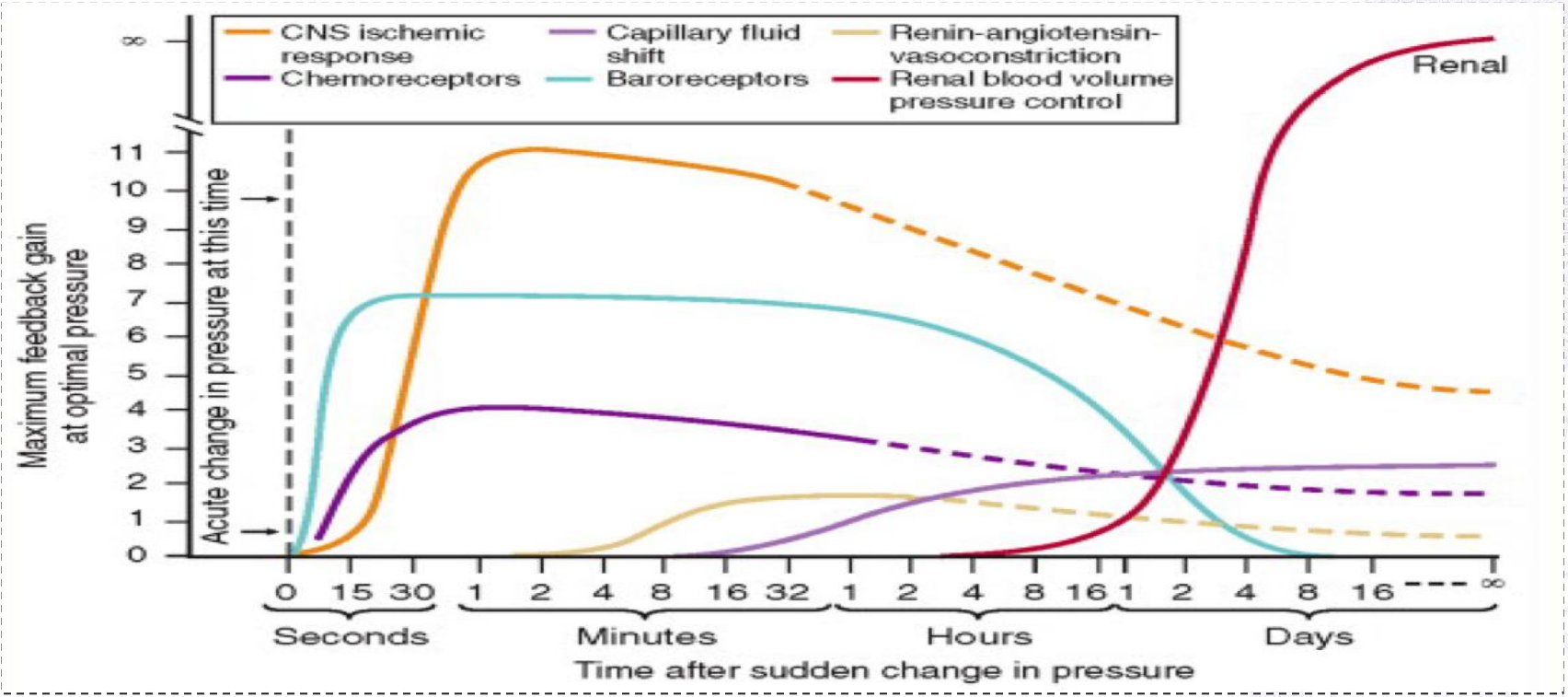
Note from 443: especially in this schema the two ways in which an increase in cardiac output can increase the arterial pressure. One of these is the direct effect of increased cardiac output to increase the pressure, and the other is an indirect effect to raise total peripheral vascular resistance through autoregulation of blood flow. The second effect can be explained as follows.

Referring back, let us recall that whenever an excess amount of blood flows through a tissue, the local tissue vasculature constricts and decreases the blood flow back toward normal. This phenomenon is called "autoregulation," which means simply regulation of blood flow by the tissue itself. When increased blood volume increases the cardiac output, the blood flow increases in all tissues of the body, so that this autoregulation mechanism constricts blood vessels all over the body. This in turn increases the total peripheral resistance. Finally, because arterial pressure is equal to cardiac output times total peripheral resistance, the secondary increase in total peripheral resistance that results from the autoregulation mechanism helps greatly in increasing the arterial pressure.

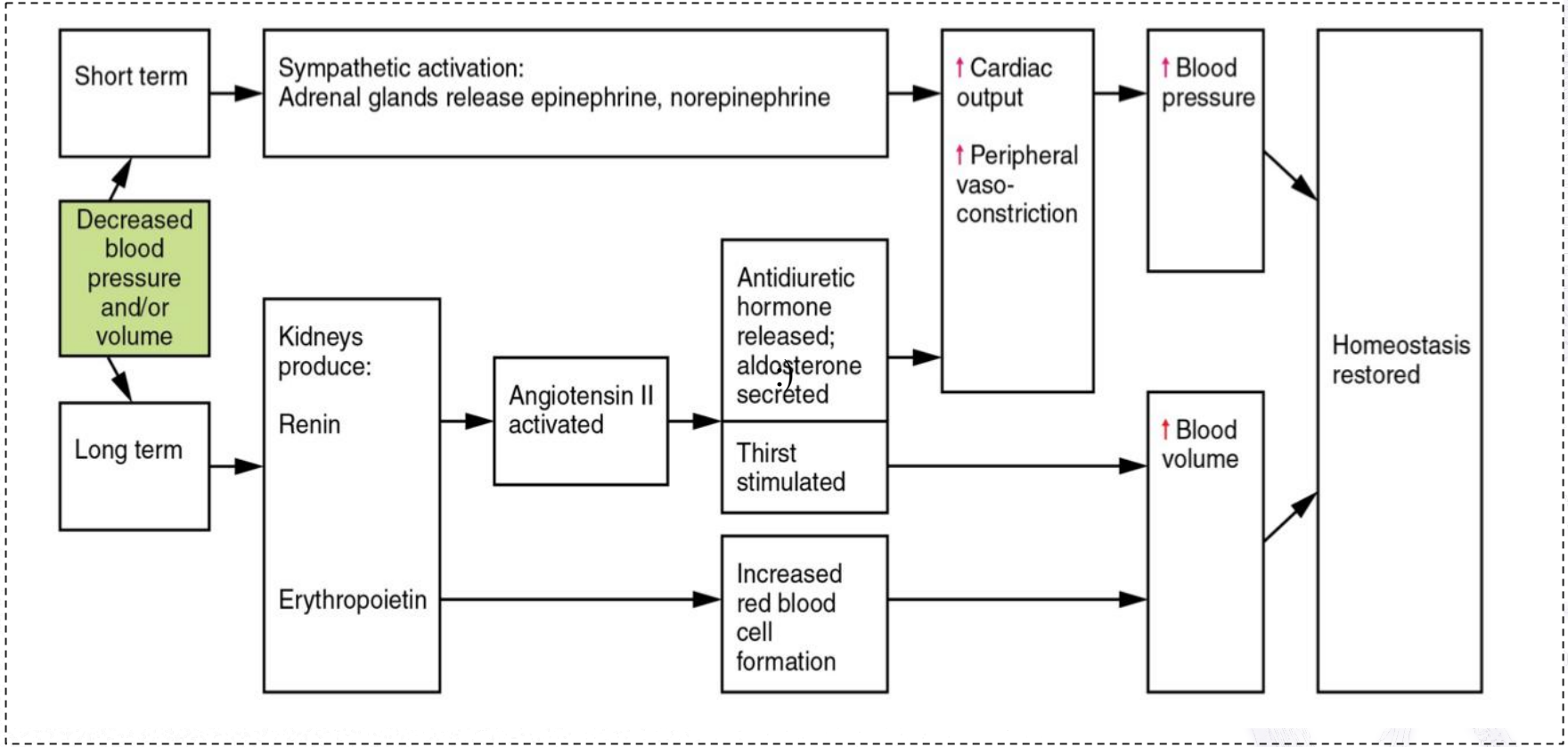
Other Long- Term (Slow Response) Regulation:

3C) Low-pressure volume receptors	3D) Erythropoietin (EPO)
Atrial Natriuretic Peptide (ANP) hormone: **1.**ANP is a vasoactive peptide released from the atria in response to a rise in atrial pressure, which in turn linked to an () in venous pressure. **2.** ANP lowers blood pressure by vasodilatation & inhibition of sodium reabsorption by the kidneys through inhibition of renin & aldosterone release (opposing renin-angiotensin-aldosterone system). This will have a diuretic effect where urinary production will ↑, causing a ↓ in blood volume & arterial blood pressure. 3.ANP has inhibitory effects on vasopressin.	1.A hormone Secreted by the kidneys when blood volume is too low 2.Acts in the bone marrow & leads to formation of Red blood cells (RBCs) → to ↑ blood volume.

Control mechanisms at different time intervals after onset of a disturbance to the arterial pressure:



Summary of regulation of arterial blood pressure



Check here for our summary
Highly recommended !!!!!

Sorry but if you will not cheack it راحت عليك المليون

MCQs:

Answers

For more question check our summary file!

1/D
2/C
3/C

1 Which of the following would be expected to occur during brain ischemia?

A	Increase in parasympathetic activity	B	Decrease in arterial pressure	C	Decrease in heart rate	D	Increase in sympathetic activity

2 Chemoreceptors are located in all the following except?

A	Aorta	B	Carotid bodies	C	Right atrium	D	Medulla

3 Which response is excitatory, NOT inhibitory?

A	Pulmonary receptors	B	Thermoreceptors	C	Chemoreceptors	D	Baroreceptor

SAQ

Enumerate the short compensatory mechanisms that help in increasing blood pressure?

1-Baroreceptor reflex
2-Chemoreceptor reflex
3-Atrial stretch receptor
4-Thermo receptor
5-pulmonary receptor

Name 2 different mechanisms for regulating mean aterial blood pressure, 2 in each time period?

Short term:Baroreceptor reflex ,Chemoreceptor reflex
long term:RAAS,Erythropoietin

Enumerate types of chemoreceptors?

1-Peripheral chemoreceptors
2-Central Chemoreceptor

What the difference between chemoreceptors and baroreceptor?

baroreceptors are mechanoreceptors responding to blood pressure changes while chemoreceptors are cells sensing the concentration of chemicals in the surrounding extracellular fluid.

Finally you have arrived , we have been waiting for you !!

Meet our team !

Team leaders

Rimaz Alhammad

Noreen Almaraba

Rayan Alshehri

Omar Albaqami

Aljoharah Alyahya

Heroes of the lecture :

omar alhumaidi

Shahad Alzenaidy

Alanoud Alnajawi

Did you like the lecture ? we mean our work :)

Contact with us! physiology.444ksu@gmail.com