

بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ



CVS Module

Small Group Discussion (SGD)

FIRST YEAR MBBS BATCH 50



Topic

Regulation of Blood Pressure

Dr. Ali Zain

FCPS PGT

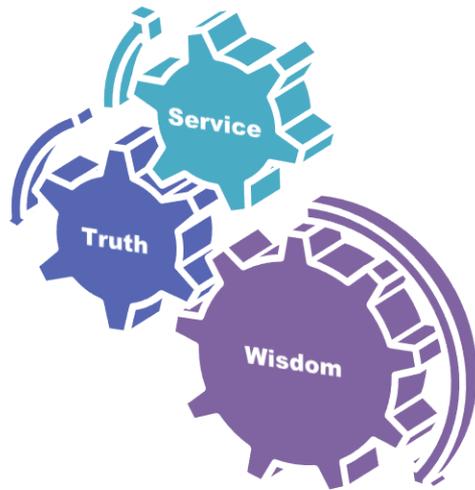
Dept. Of Physiology

Date: 16 Sep 2023

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Motto

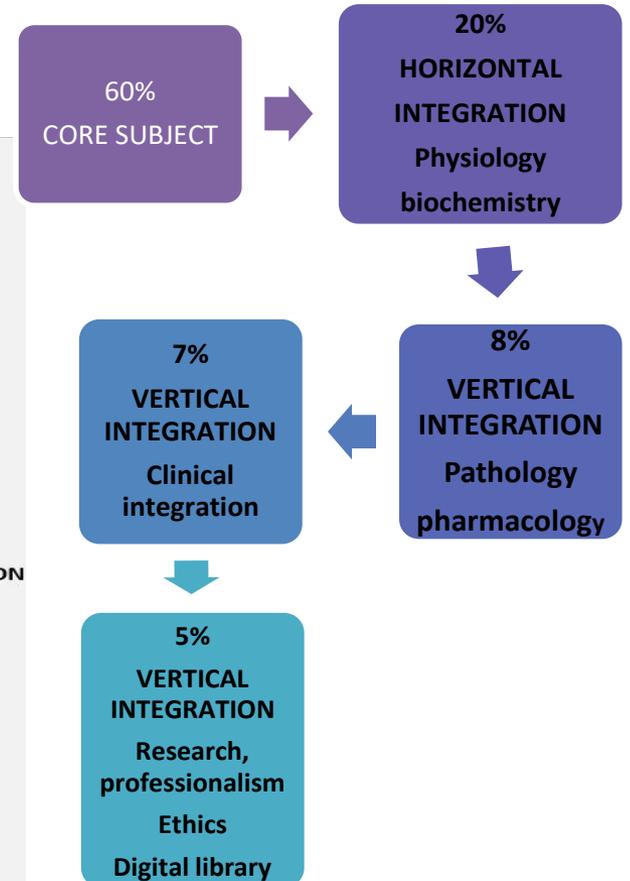
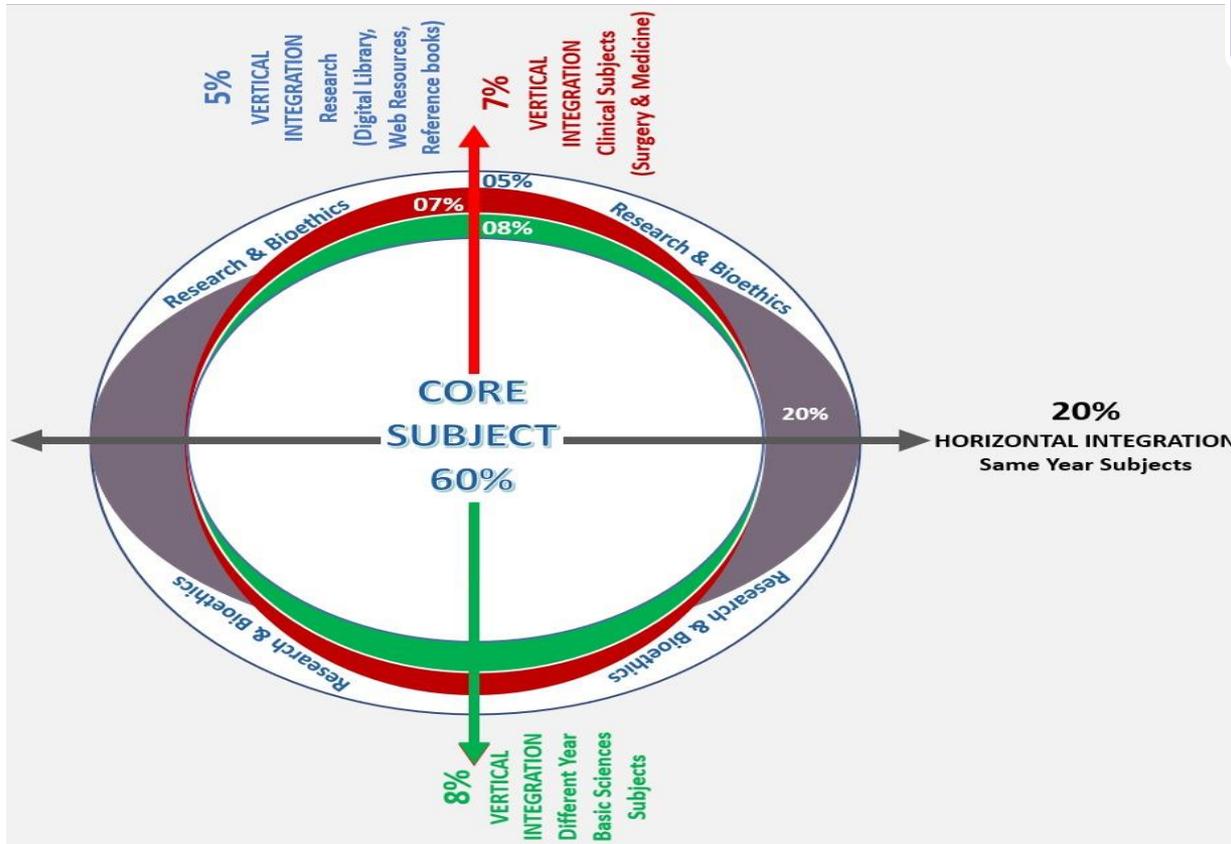


Vision; The Dream/Tomorrow

- To impart evidence based research oriented medical education
- To provide best possible patient care
- To inculcate the values of mutual respect and ethical practice of medicine



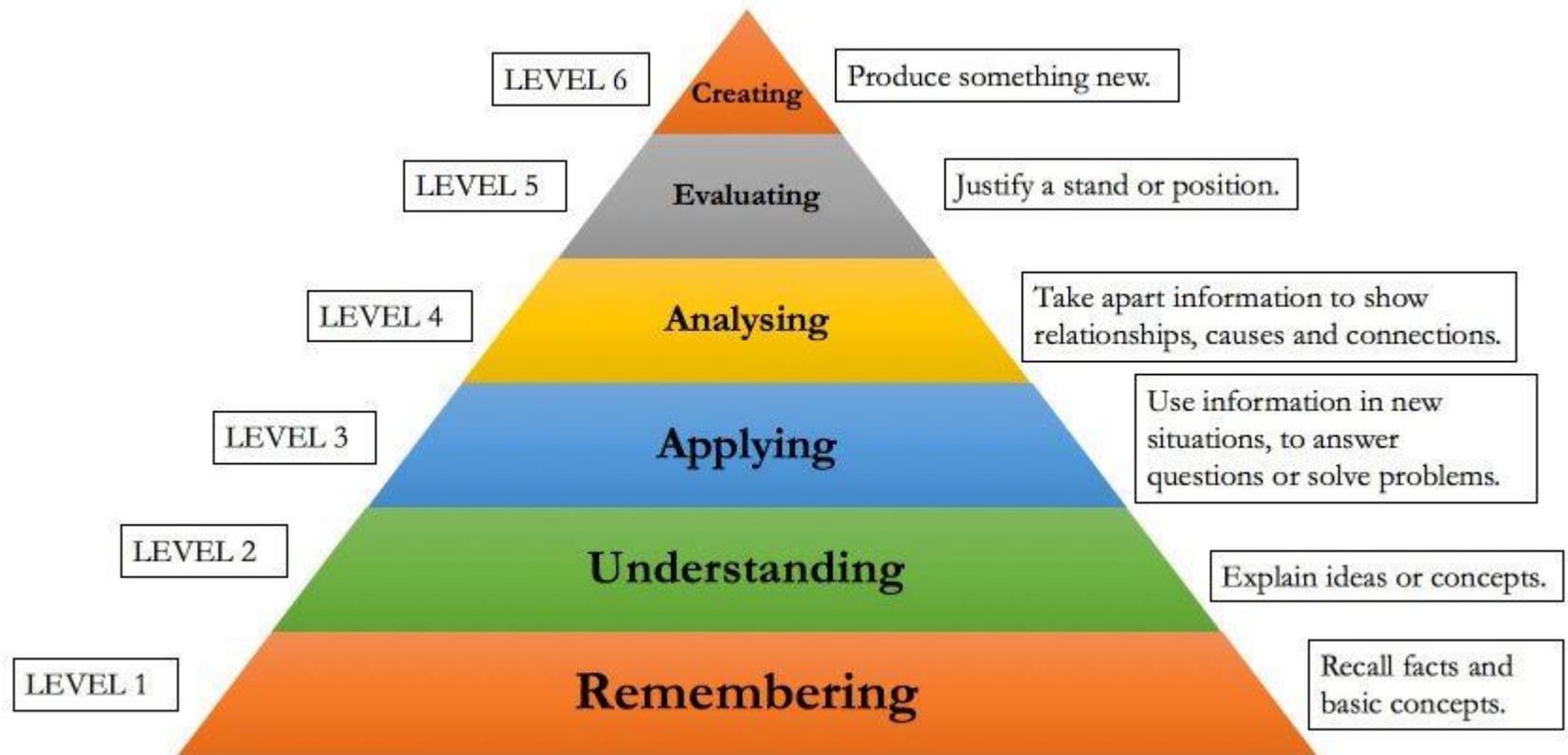
Professor Umar Model of Integrated Lecture



BLOOM'S TAXONOMY : DOMAINS OF LEARNING

Sr. #	Domain of learning	Abbreviation	Levels of the domain	Meaning
1	cognition	C	C1	Recall / Remembering
2			C2	Understanding
3			C3	Applying / Problem solving
4	Psychomotor	P	P1	Imitation / copying
5			P2	Manipulation / Follows instructions
6			P3	Precision / Can perform accurately
7	Attitude	A	A1	Receiving / Learning
8			A2	Respond / Starts responding to the learned attitude
9			A3	Valuing / starts behaving according to the learned attitude

BLOOM'S TAXONOMY OF THE COGNITIVE DOMAIN





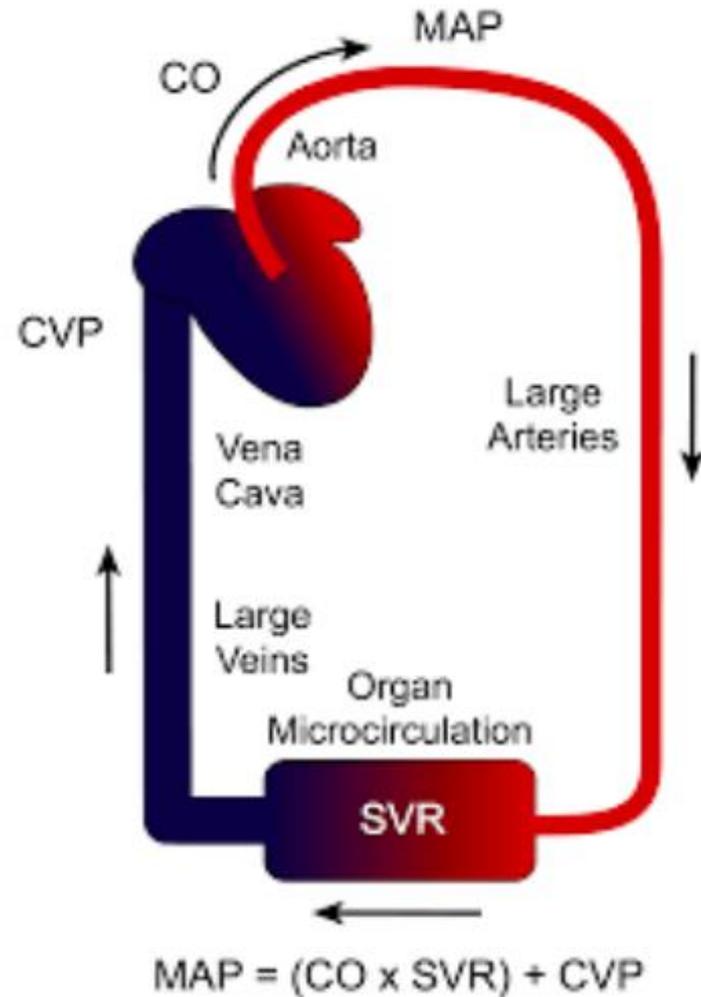
LEARNING OBJECTIVES

Sr. #	Learning Objective	Domain of Learning
1	To study different short and long term regulation of blood pressure.	C1
2	To Describe Baroreceptor and chemoreceptor mechanism.	C2
3	To understand Renin–Angiotensin Aldosterone system.	C2
4	To understand Hormonal control mechanism.	C2
5	To relate Vasomotor reflexes at different time intervals after the disturbance.	C3
6	To diagnose hypertension.	C3
7	To assess case study of active blood loss presented in trauma centre.	C3

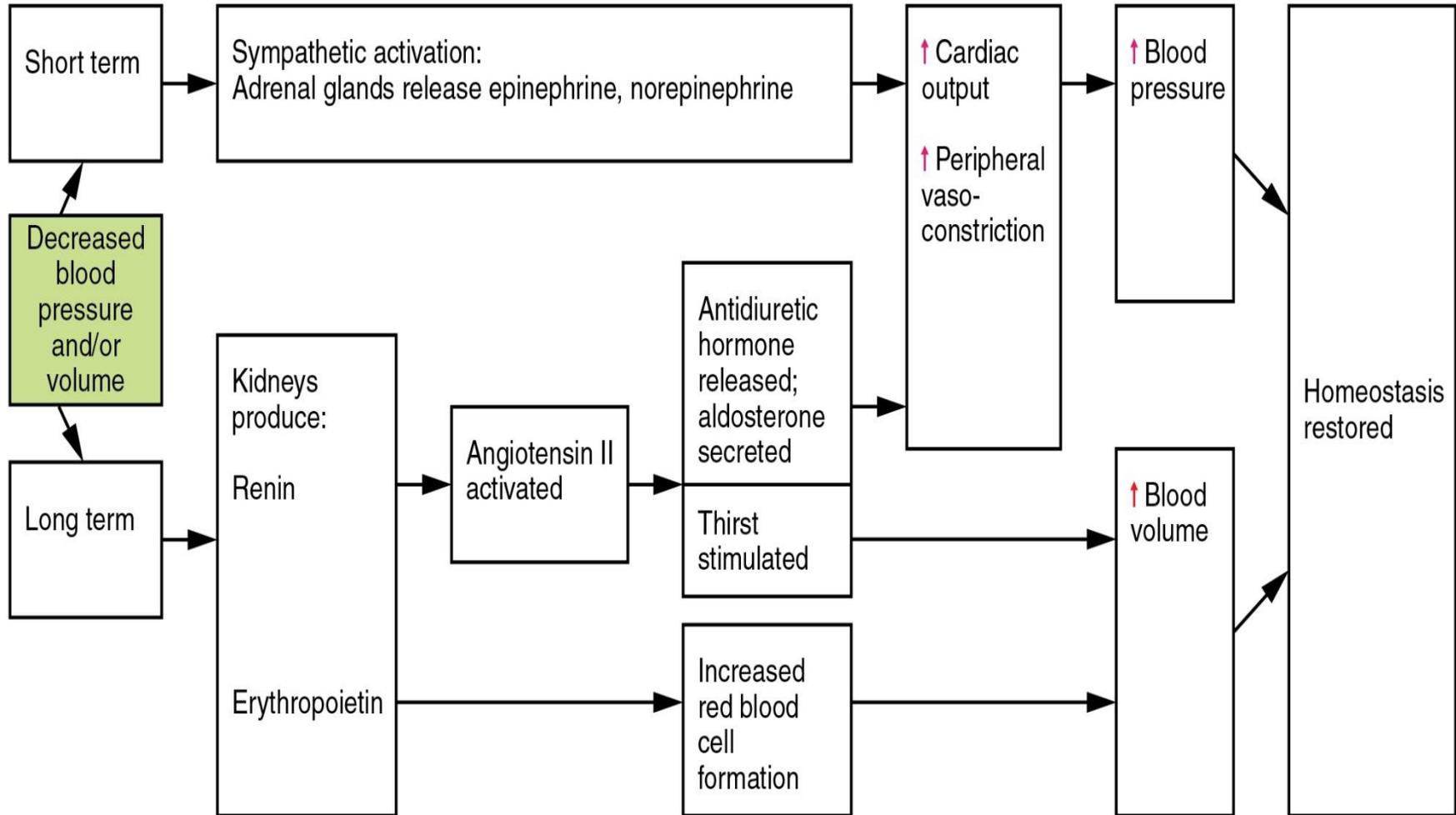
Blood Pressure:-

Blood pressure is the force or pressure of blood on the walls of Blood vessels.

- ❖ Blood pressure is mentioned in terms of systolic blood pressure over diastolic blood pressure.



Regulation Of Blood Pressure:-





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:-

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

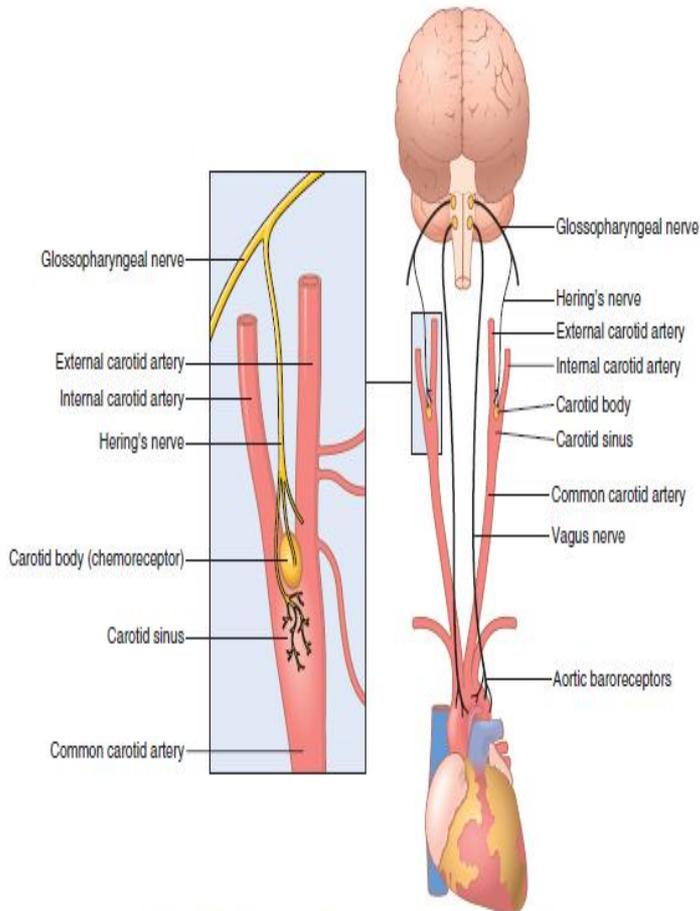
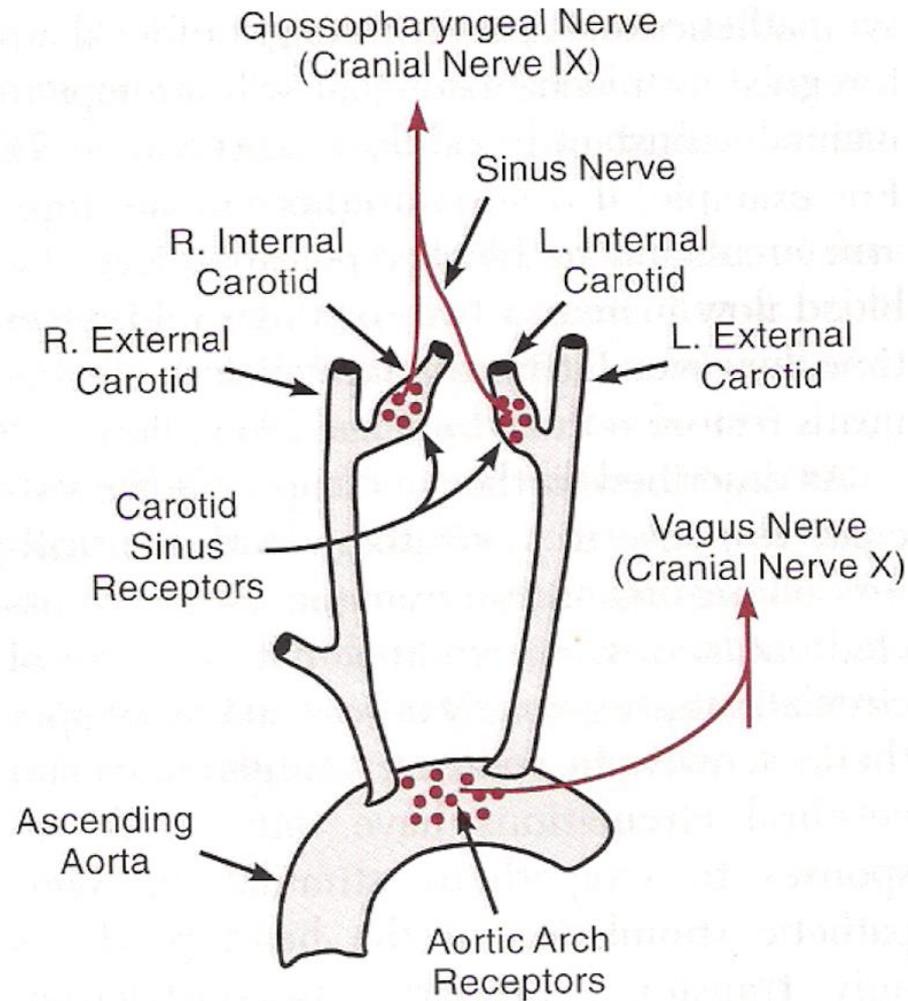


Figure 18-5. The baroreceptor system for controlling arterial pressure.

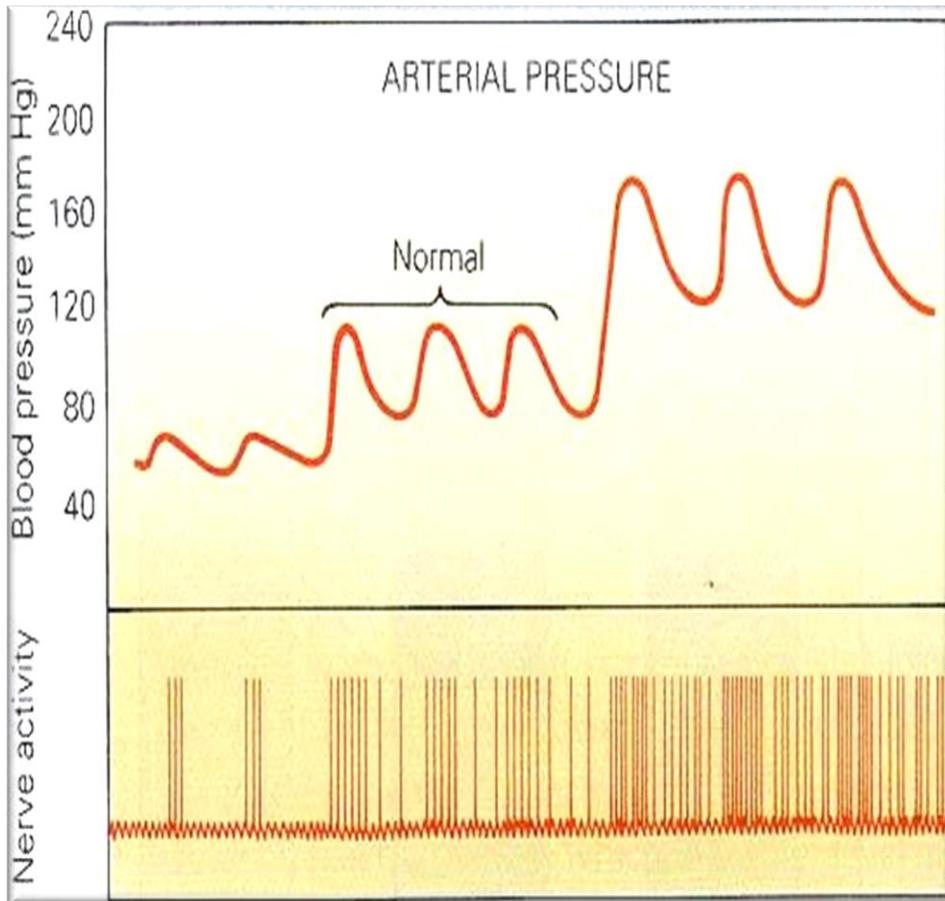
Carotid baroreceptors are located in the carotid sinus, both sides of the neck. Aortic baroreceptors are located in the aortic arch.

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

1. The Baroreceptors:-

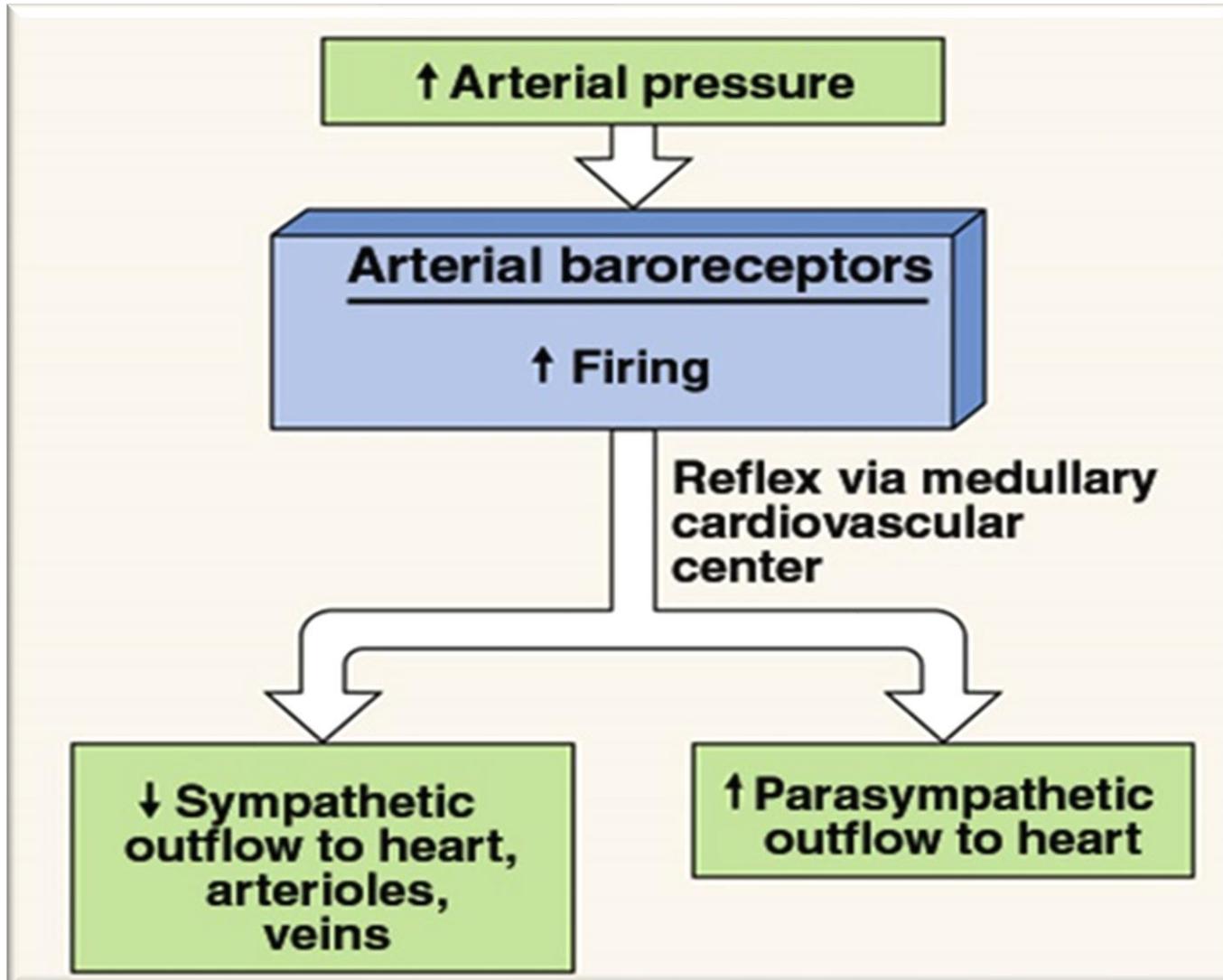


1. The Baroreceptors:-

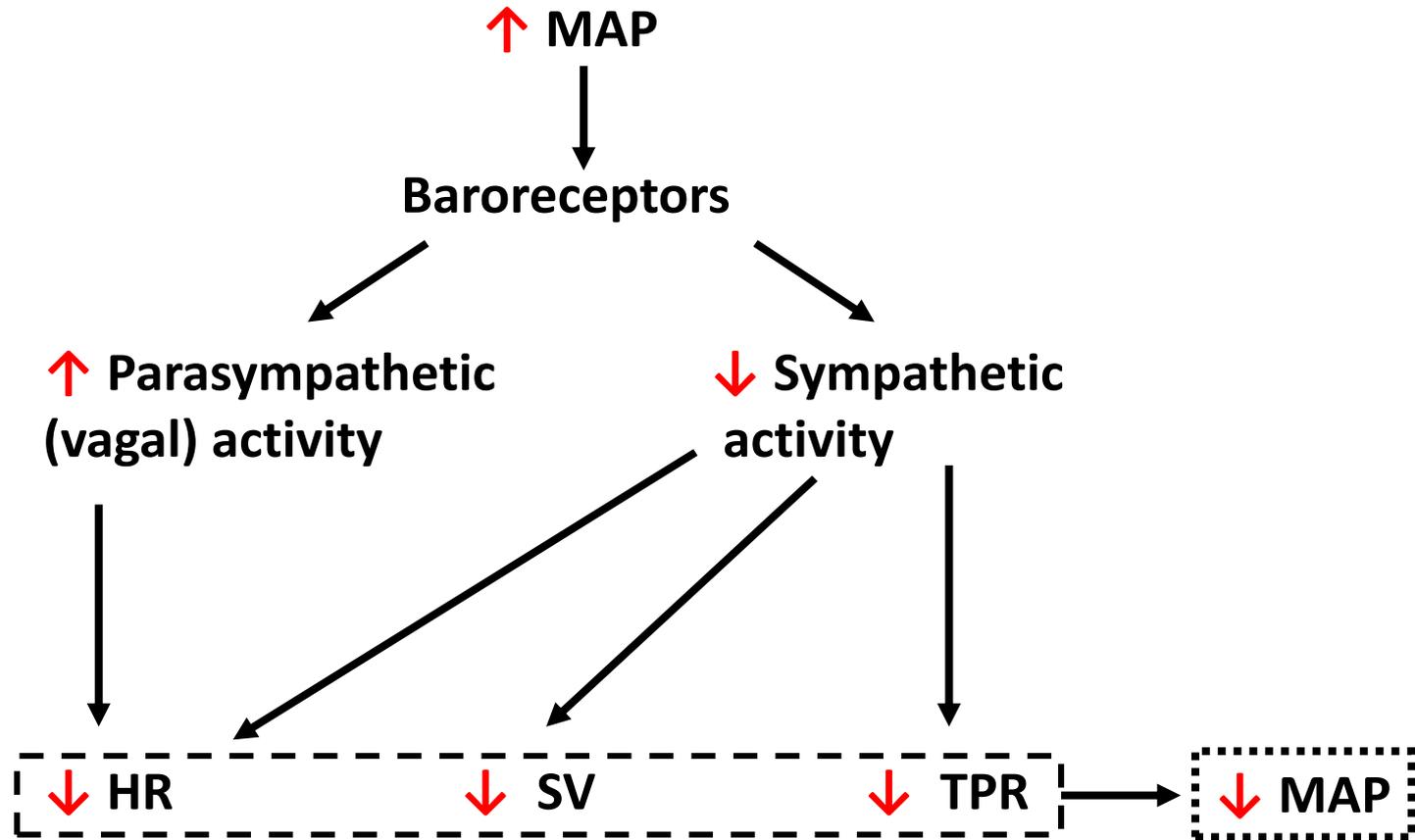


- ❑ At normal arterial pressure the baroreceptors are active.
- ❑ Increased blood pressure increases their rate of activity, while decreased pressure decreases the rate of firing (activity).
- ❑ They play an important role in maintaining relatively constant blood flow to vital organs such as brain during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

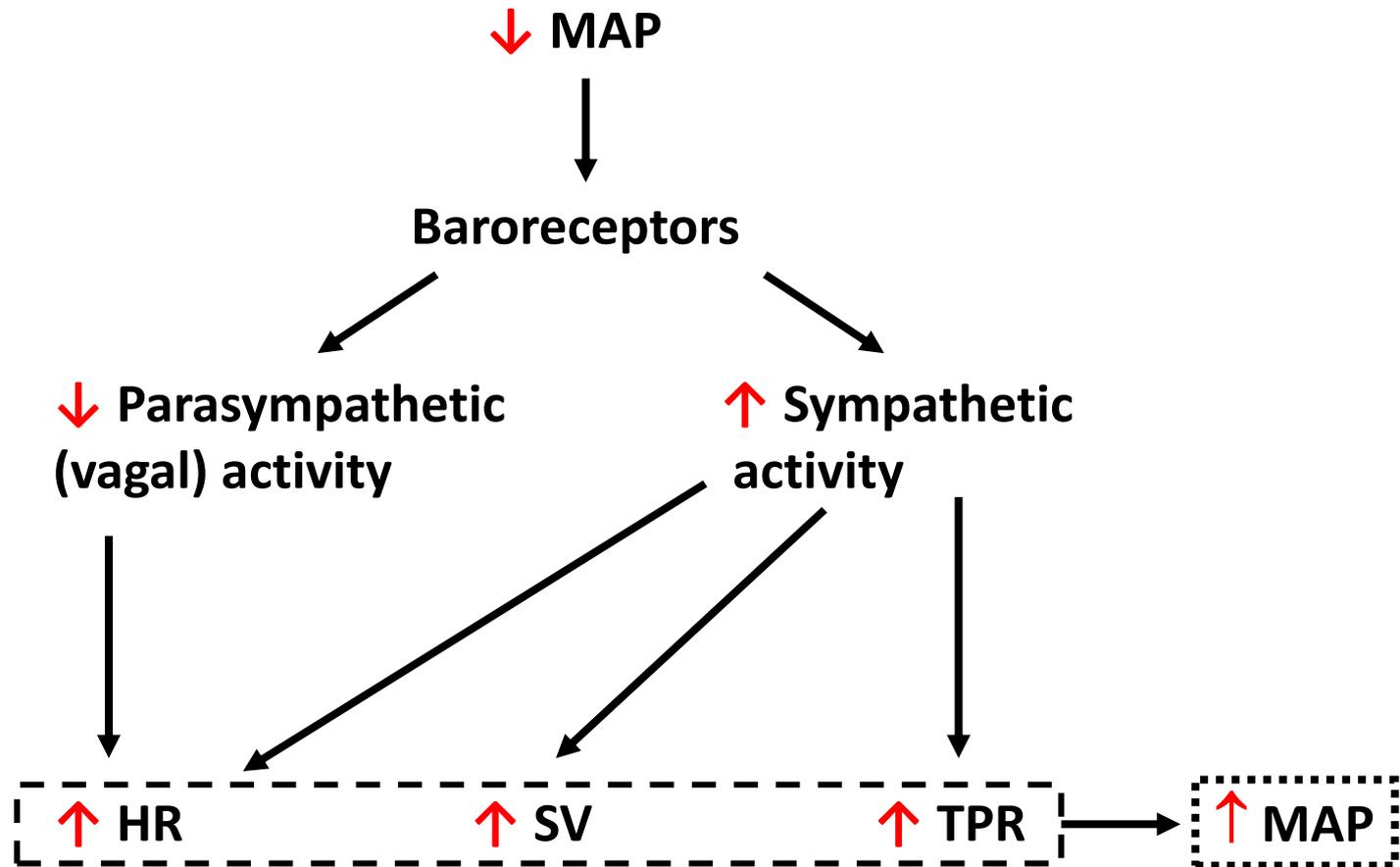
1. The Baroreceptors:-



Increase In Mean Arterial Pressure:-

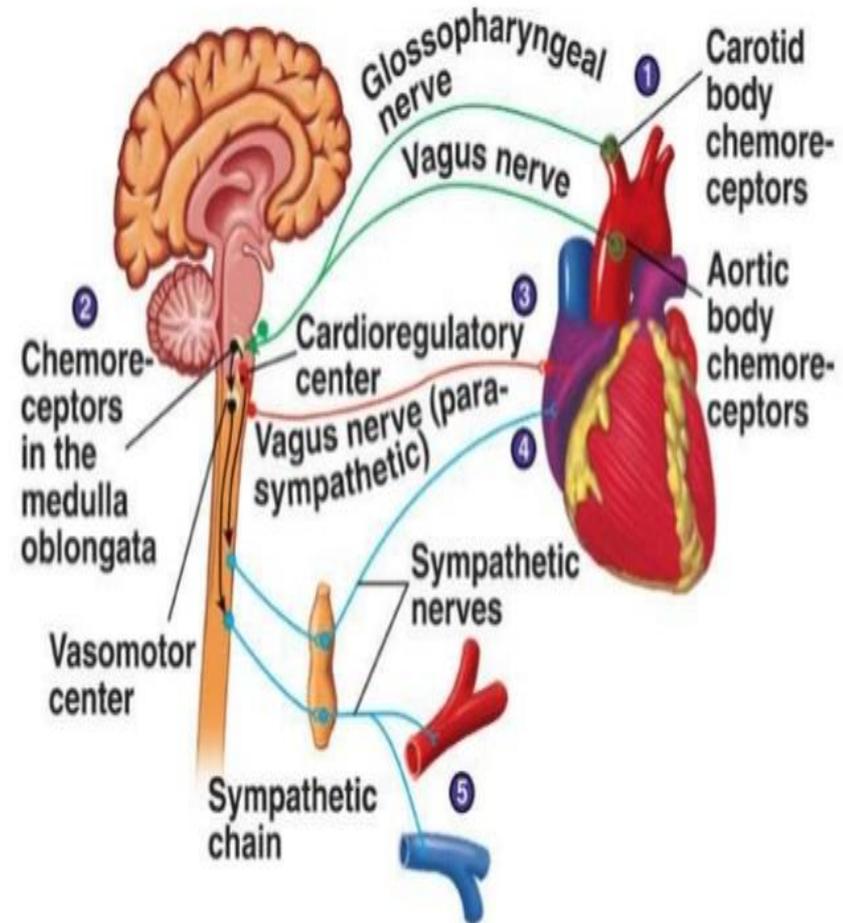


Decrease In Mean Arterial Pressure:-



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 $MAP < 20 \text{ mmHg}$ → 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.



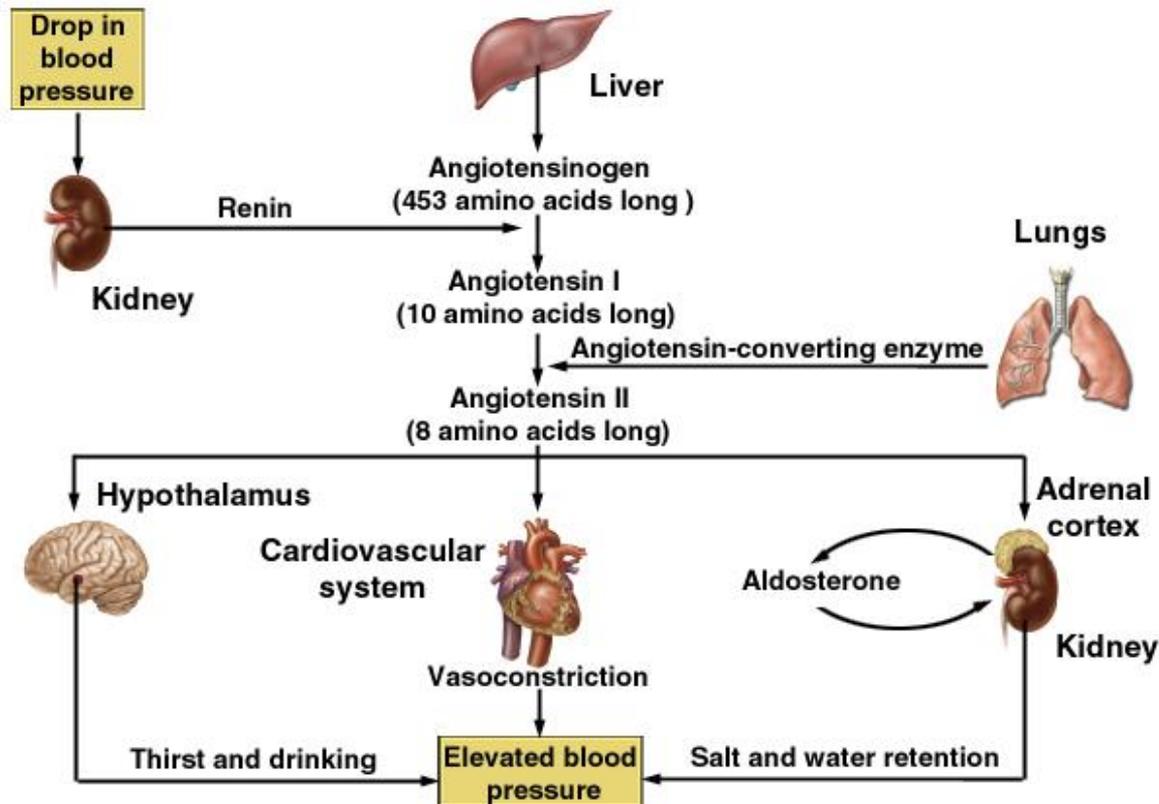
Intermediate Term Regulation Of Blood Pressure:-

Respond from 30 min to several hours:-

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

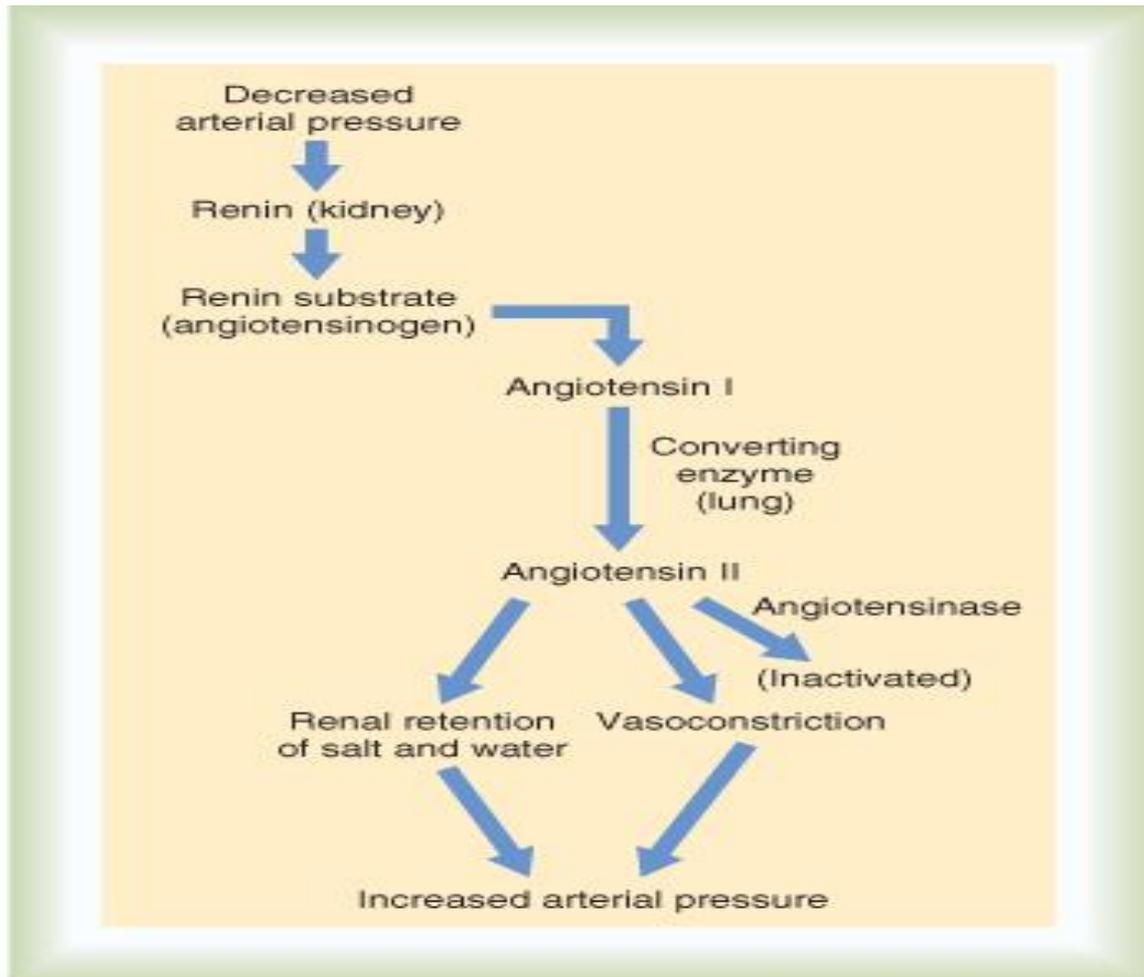
Intermediate Term Regulation Of Blood Pressure:-

1. Renin – Angiotensin system



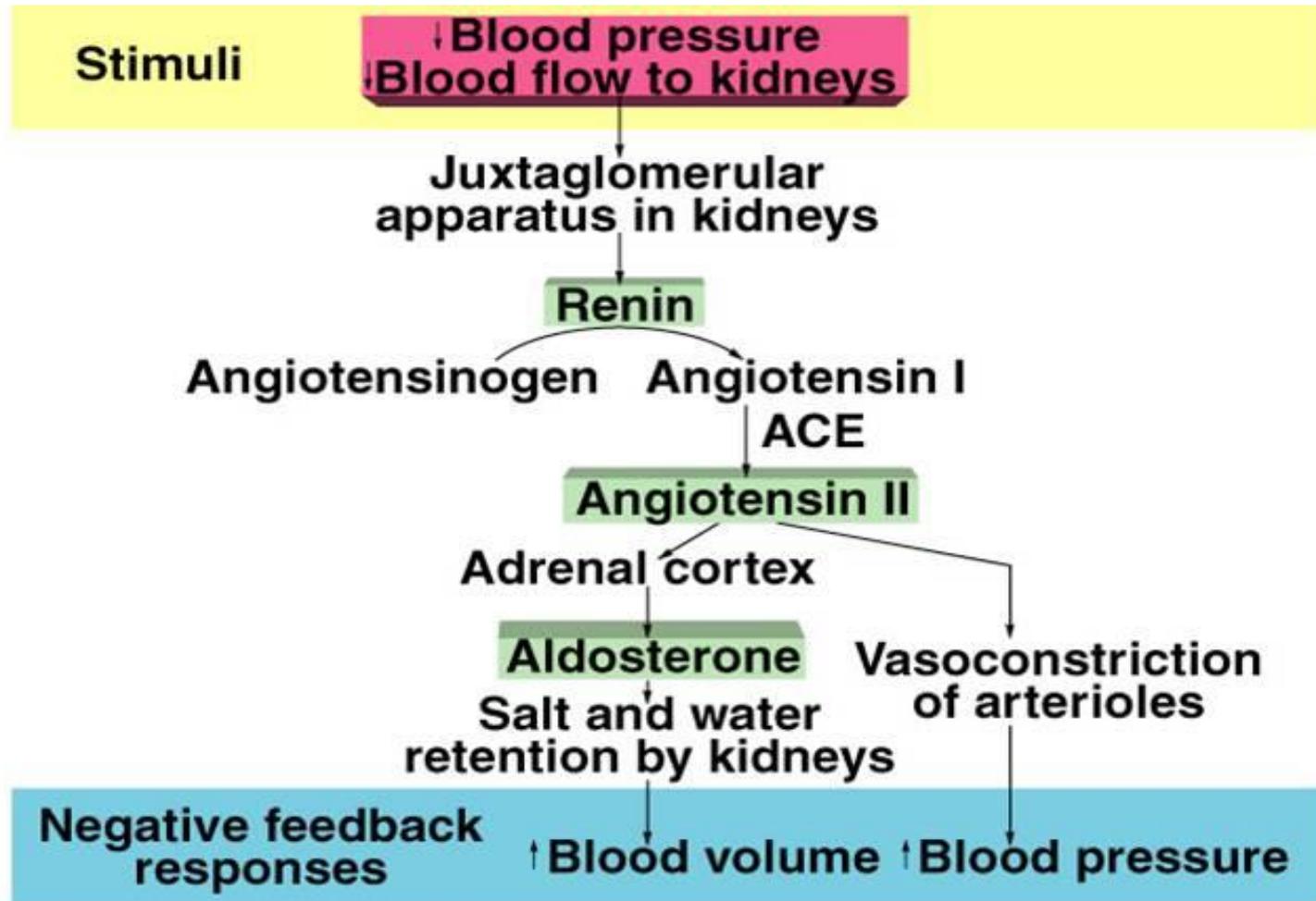
Intermediate Term Regulation Of Blood Pressure:-

1. Renin – Angiotensin system



Intermediate Term Regulation Of Blood Pressure:-

1. Renin – Angiotensin system

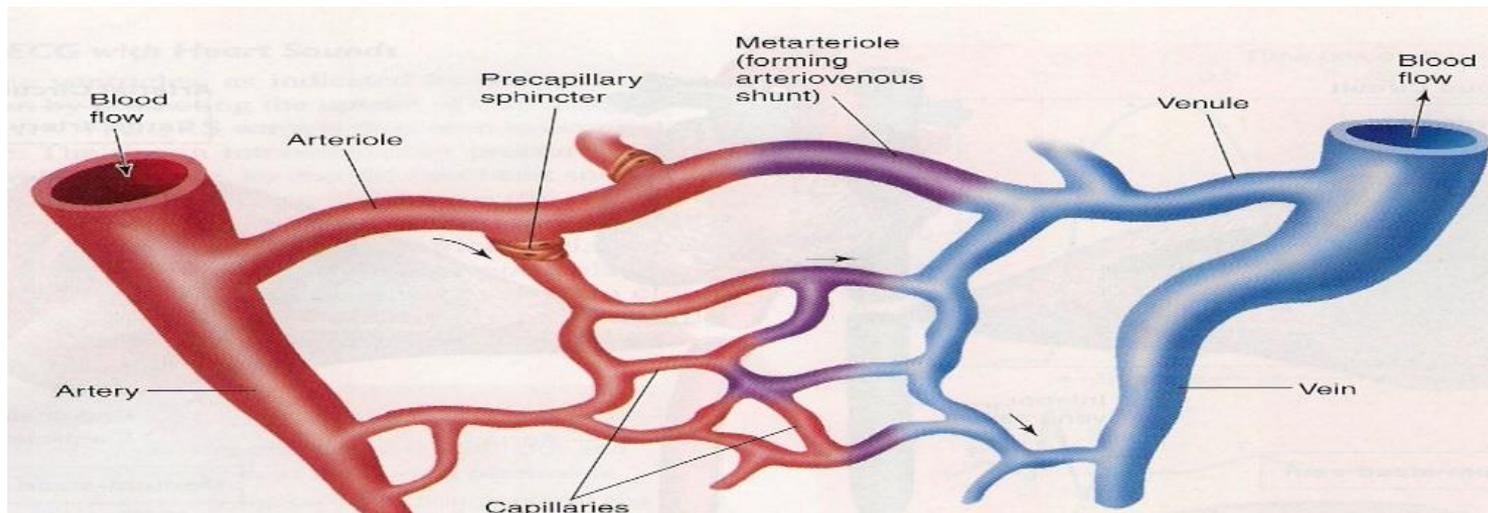


Intermediate Term Regulation Of Blood Pressure:-

2. Capillary shift mechanism

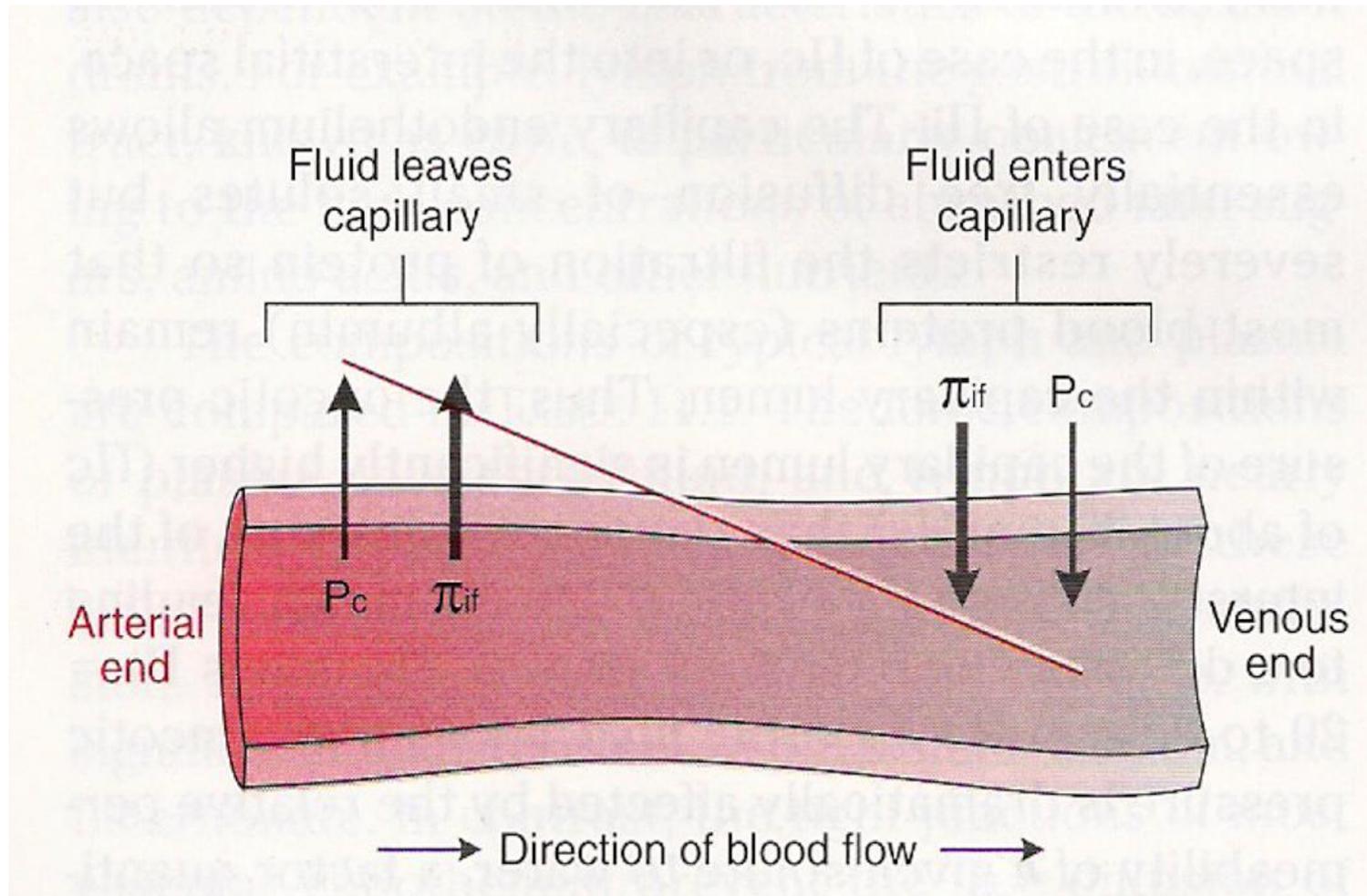
Movement of fluid from interstitial spaces into capillaries in response to \downarrow BP to maintain blood volume.

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.”



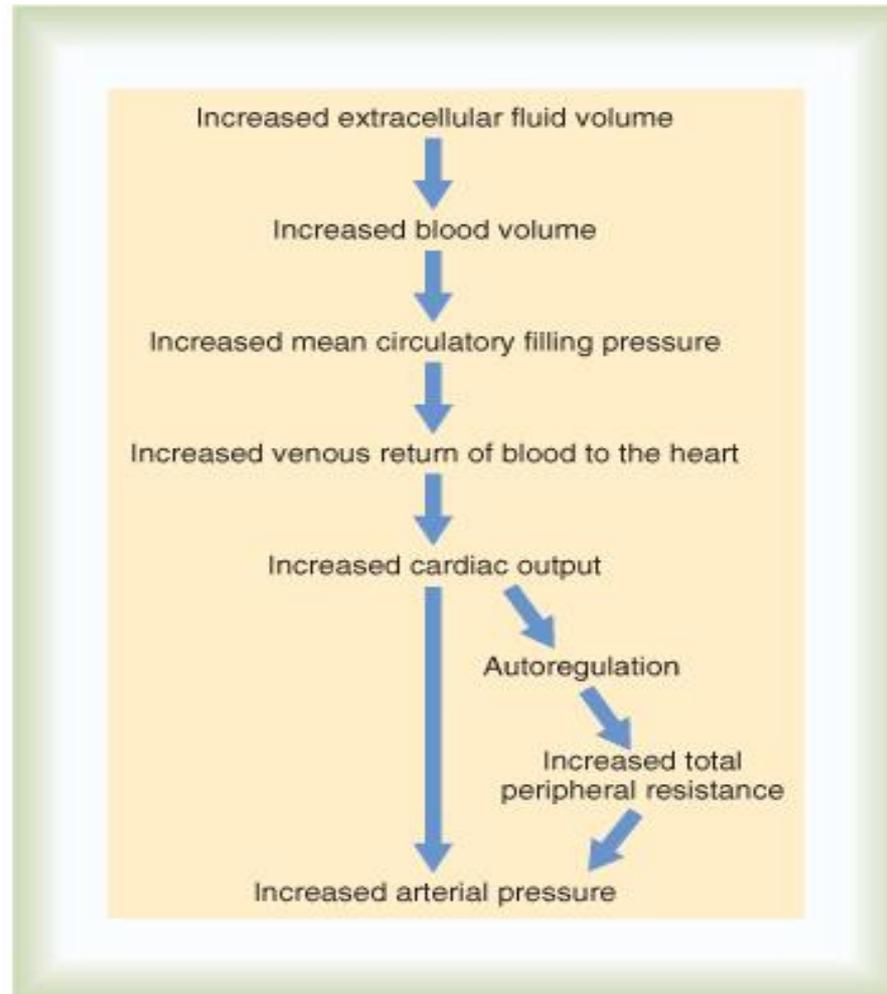
Long Term Regulation Of Blood Pressure:-

(acting within days to months)

1. Renal Body fluid control mechanism
2. Aldosterone Mechanism
3. Hormonal mechanism

Long Term Regulation Of Blood Pressure:-

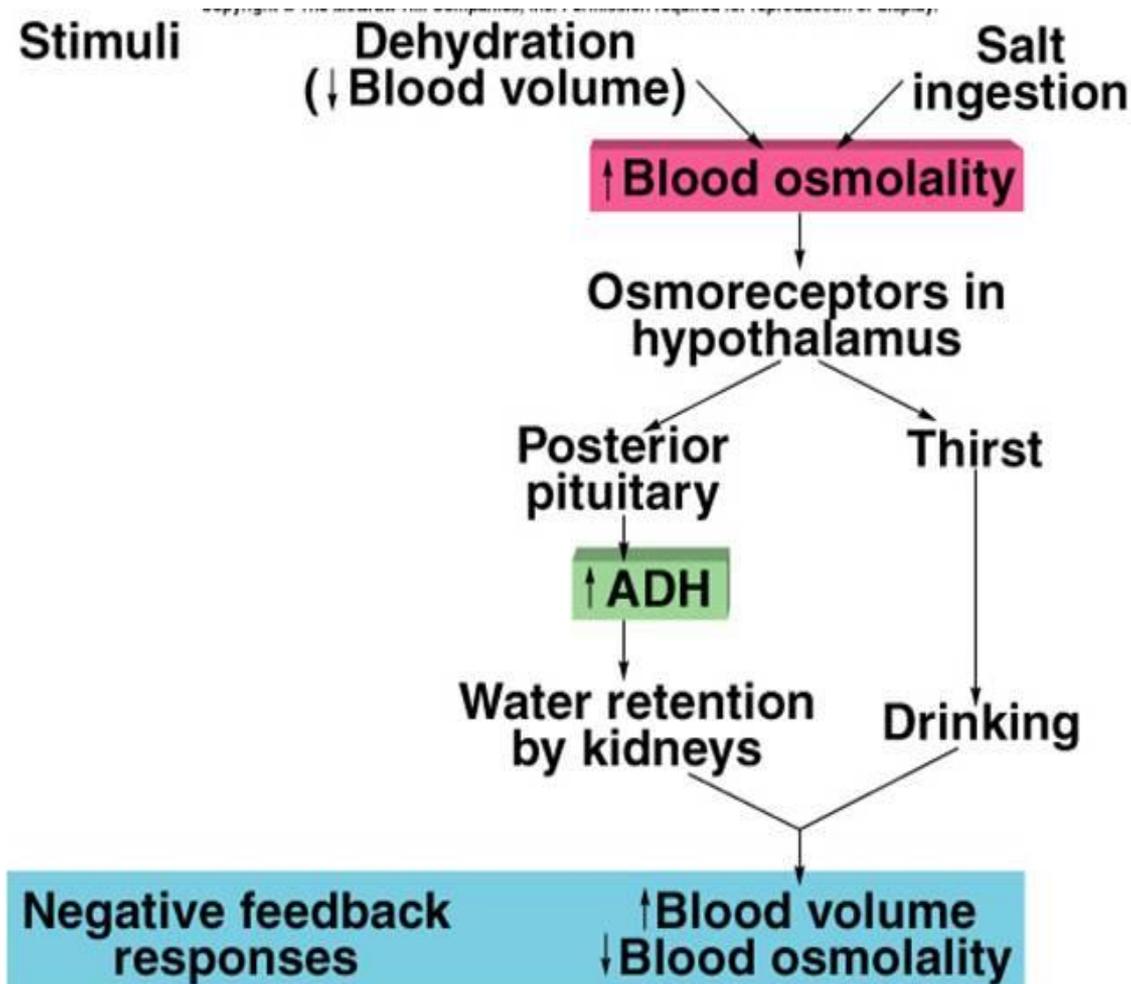
1. Renal Body fluid control mechanism



Long Term Regulation Of Blood

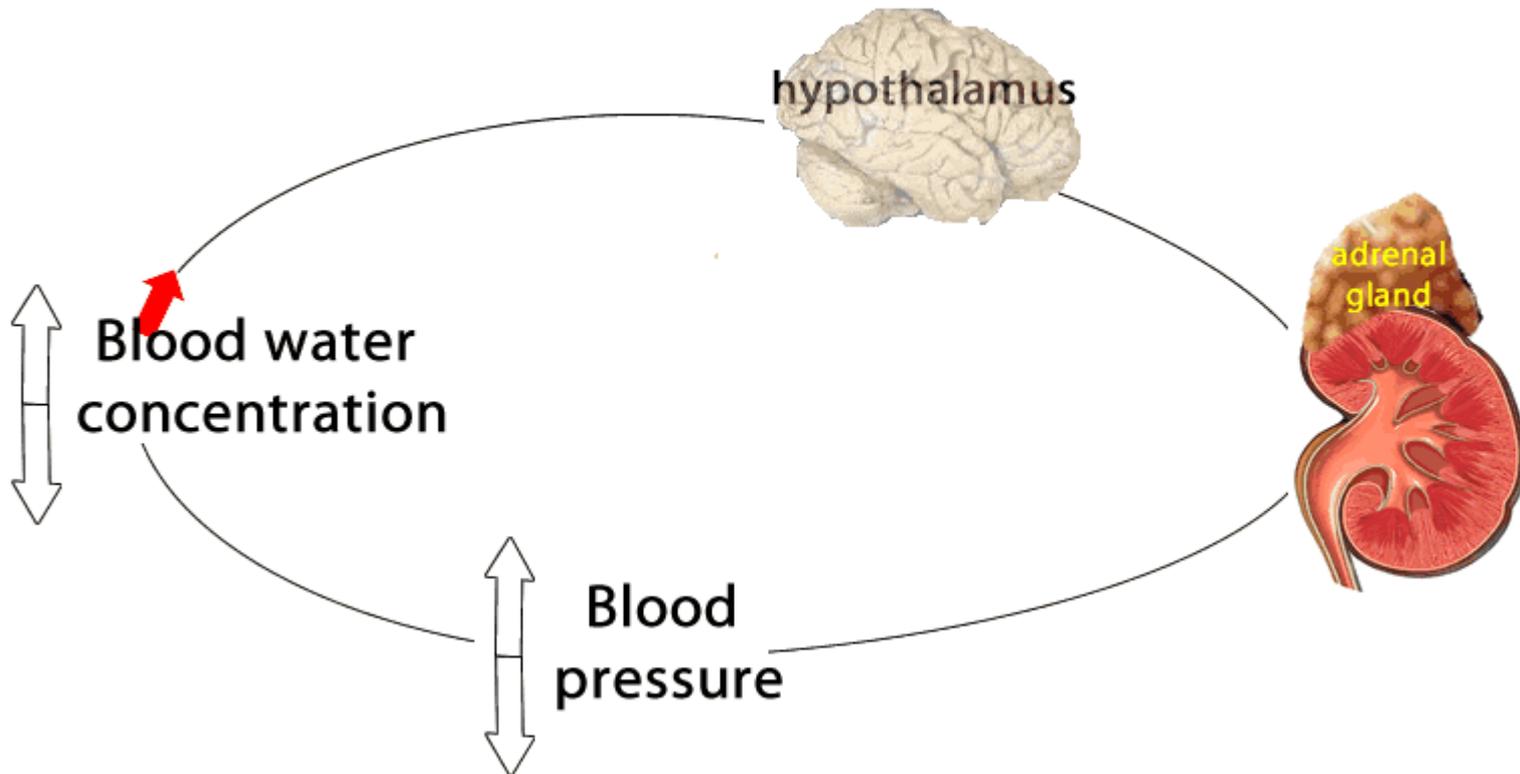
Pressure:-

1. Renal Body fluid control mechanism



Long Term Regulation Of Blood Pressure:-

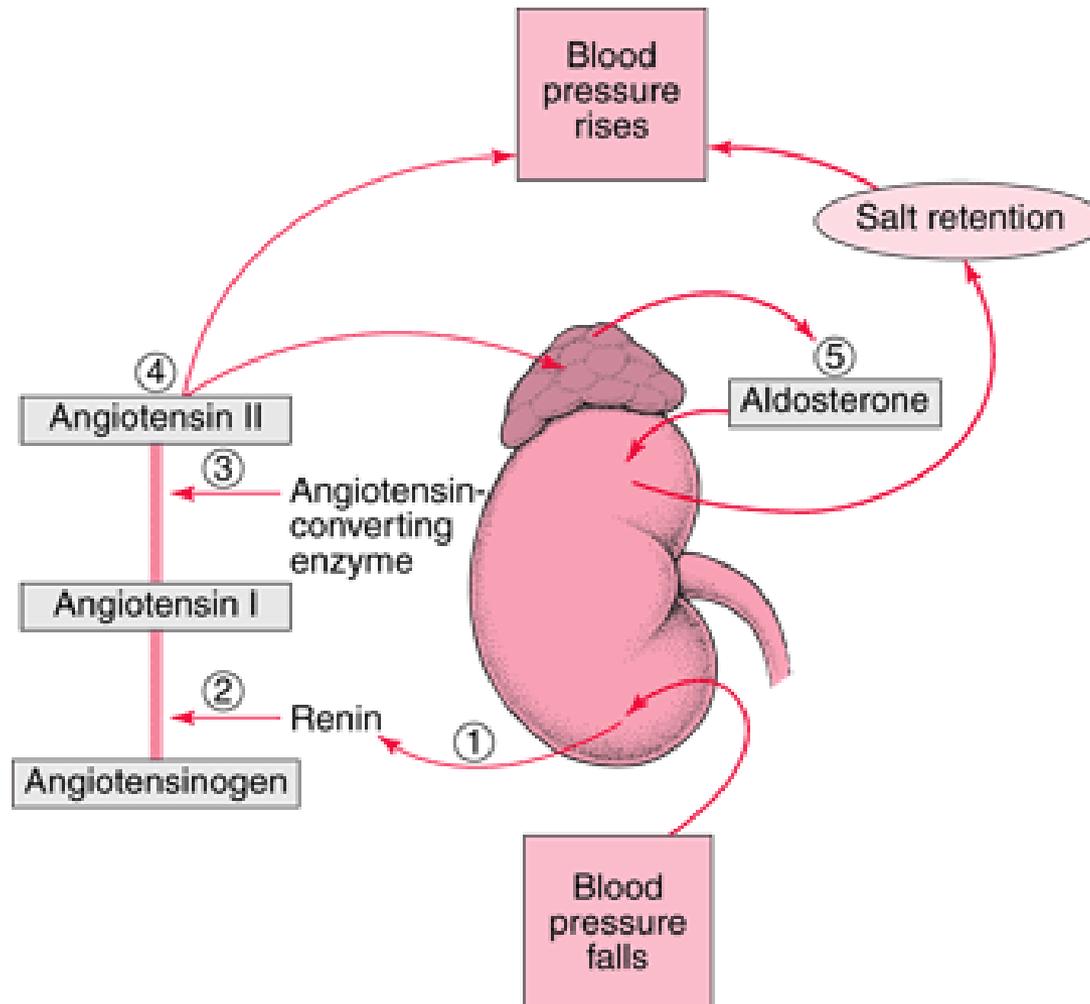
1. Renal Body fluid control mechanism



Long Term Regulation Of Blood

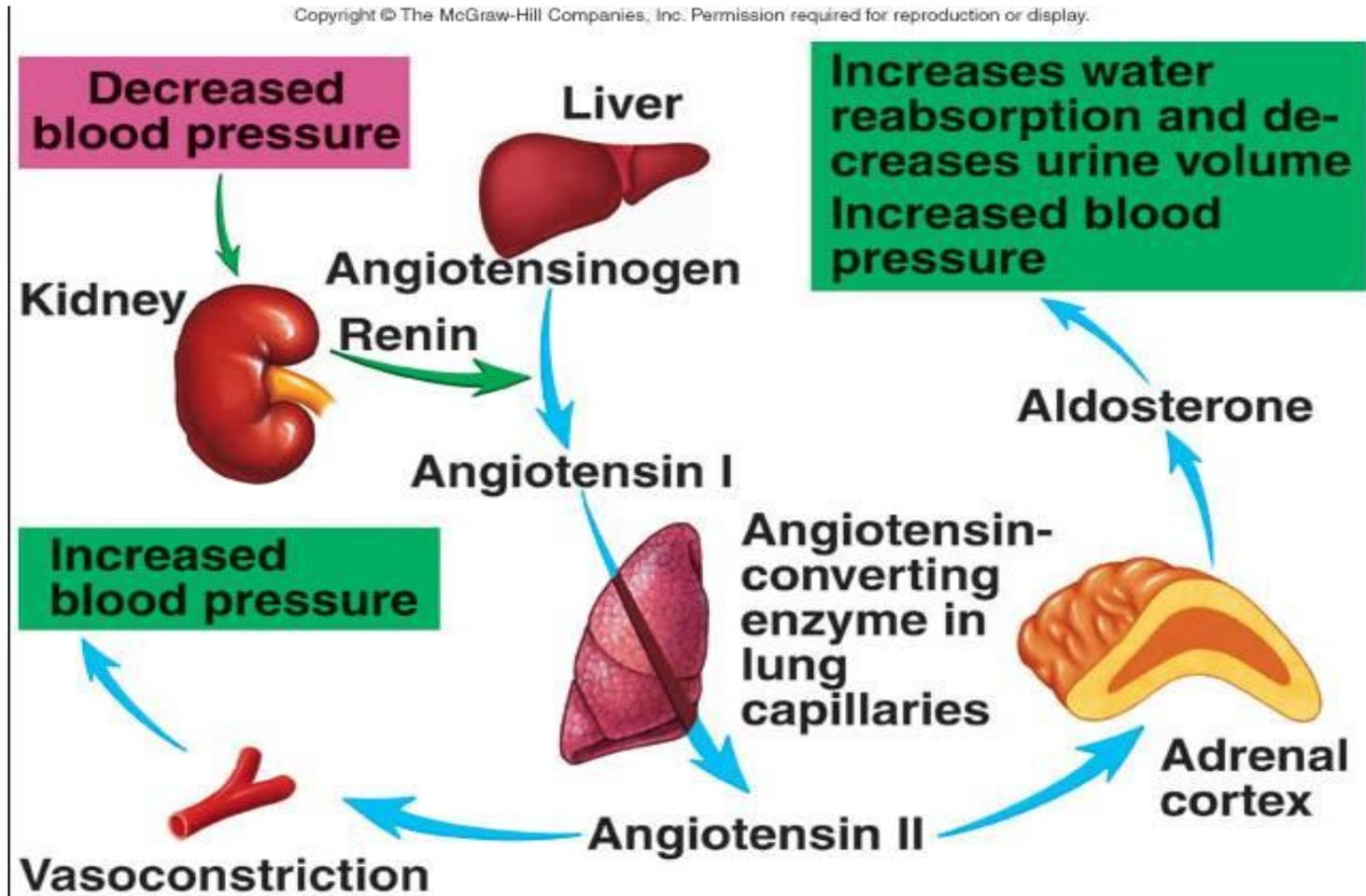
Pressure:-

2. Aldosterone Mechanism



Long Term Regulation Of Blood Pressure:-

2. Aldosterone Mechanism



Renin – Angiotensin Aldosterone System (RAAS)

Decreased renal blood flow (or/and) decrease in sodium activates Juxtaglomerular apparatus of kidneys (volume receptors)



Renin system is activated and converts angiotensin (plasma protein) to angiotensin I (lungs)

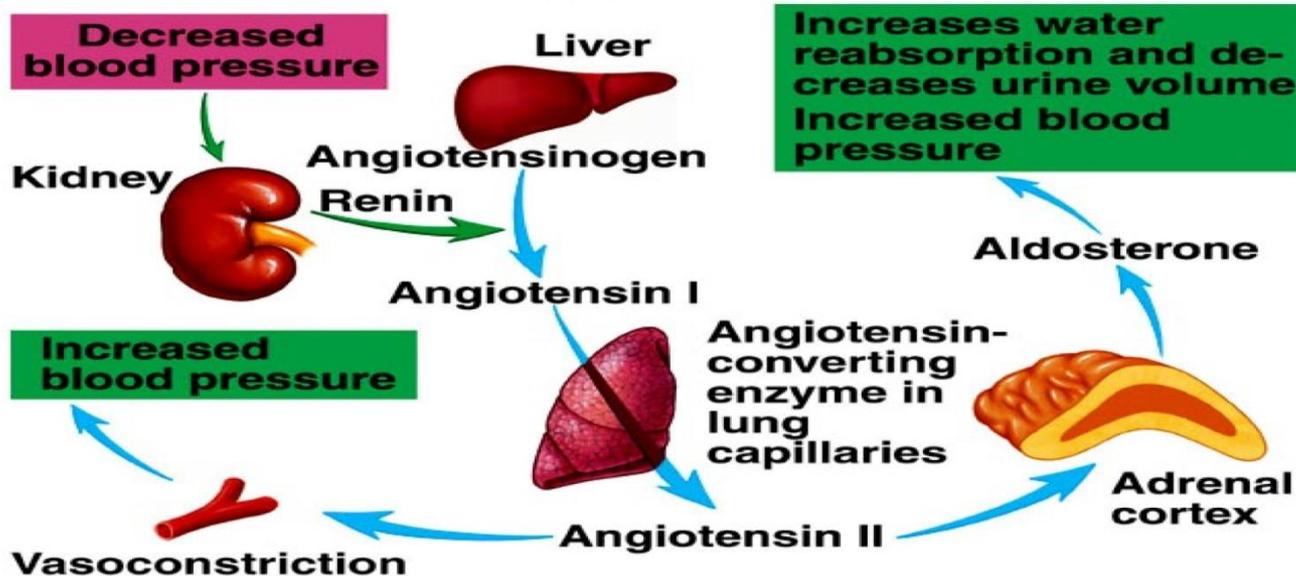


Through converting enzymes Angiotensin I is converted to II (vasoconstrictor) to Ag III



In adrenal cortex corticosterone is converted to aldosterone which promotes Na retention

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Angiotensin II stimulates the secretion of the hormone aldosterone from the adrenal cortex.



Long Term Regulation Of Blood Pressure:-

3. Hormonally Mediated:-

- Takes few hours to begin showing significant response.
- Mainly renal (acts if BP is too low):
 - Renin-Angiotensin-Aldosterone System.
 - Vasopressin [Anti-diuretic hormone (ADH)] Mechanism.
- Others:
 - Atrial Natriuretic Peptide Mechanism (Low-pressure volume receptors).
 - EPO (erythropoietin).



Hormonal Regulation of Blood Pressure:-

1. Catecholamines (Adrenaline and Noradrenaline)

Alpha adrenoceptor stimulation promotes vasoconstriction

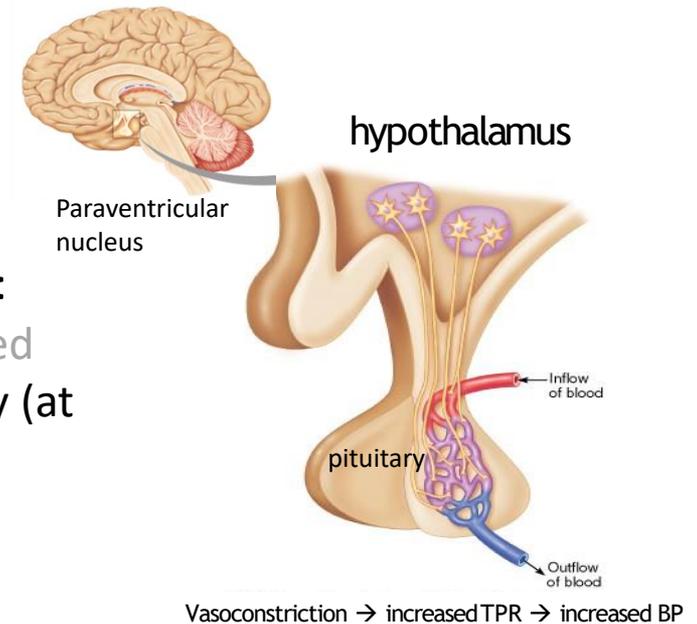
Beta adrenoceptor stimulation promotes vasodilation

- Adrenaline released from the adrenal medulla circulates in the blood and can bind to both beta and alpha adrenoceptors.
- Noradrenaline released from the sympathetic nerves binds primarily to adrenoceptors.

Hormonal Regulation of Blood Pressure:-

2. Vasopressin (Antidiuretic hormone; ADH)

ADH (vasopressin) is synthesized in the Paraventricular nucleus of the hypothalamus, then it is stored in the posterior pituitary.



Vasoconstriction → increased TPR → increased BP

It has two major functions which will lead to Increase in BP:

1- Vasoconstriction, in order to ↑ ABP (that is why it is called vasopressin) 2- Promotion of water retention by the kidney (at kidney tubules to ↑ blood volume + thirst stimulation

This is how it is secreted.

1- Increased Osmolarity.

This happens with dehydration, salt intake, or hypovolemia.

Osmolarity means increased conc. of solutes in the blood.

2- Osmoreceptor Stimulation.

Receptors in the hypothalamus sense the increased osmolarity in the blood.

3- ADH release.

The pituitary gland releases ADH. Usually, when it is secreted aldosterone is secreted.



Other Vasomotor Reflexes:- (Not as strong)

Atrial stretch receptor reflex:

- increase in venous return stimulates atrial stretch receptors which in turn produces reflex vasodilatation & decrease in ABP.

Thermo-receptors (in skin / hypothalamus):

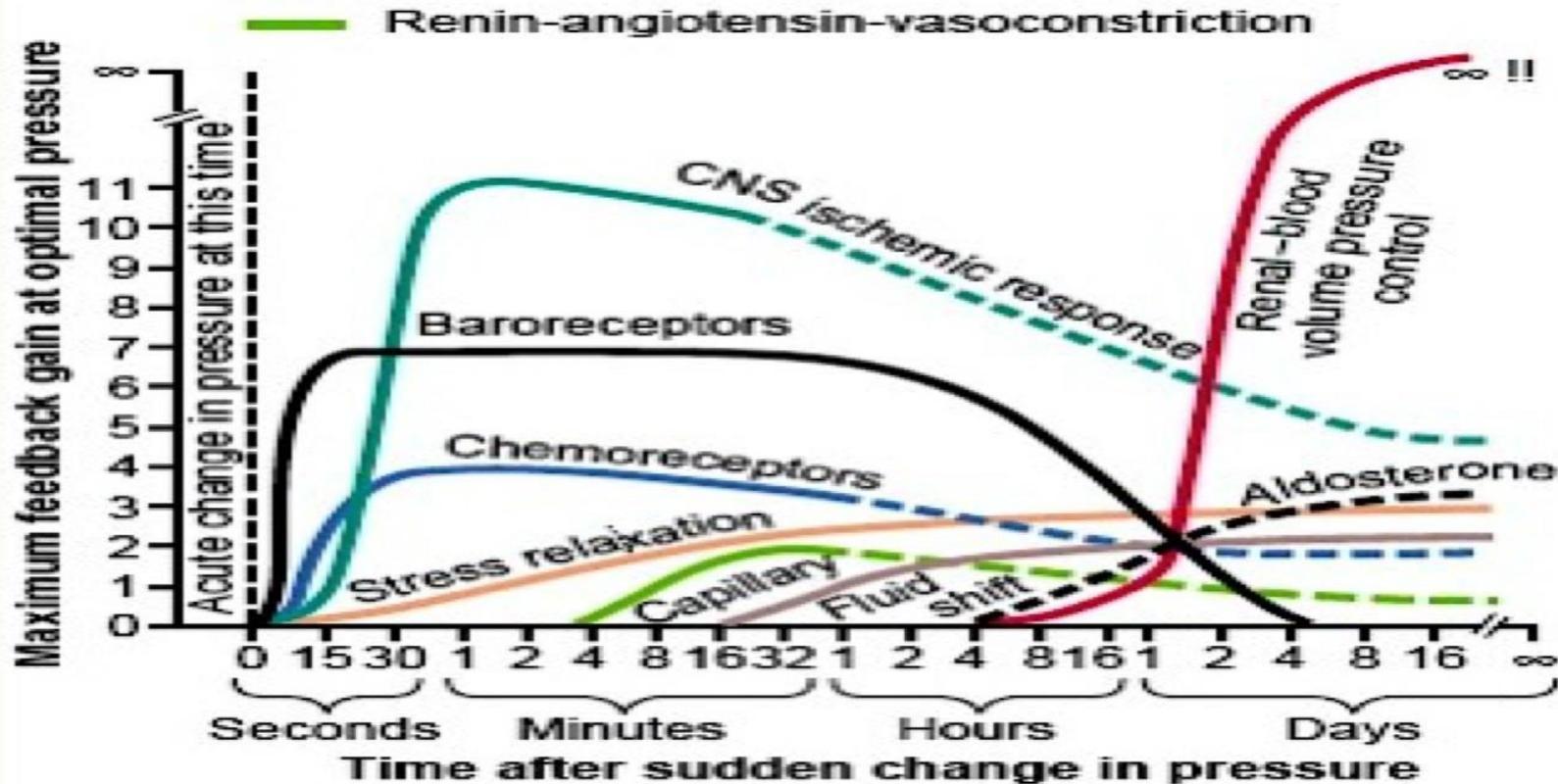
- Exposure to heat > vasodilatation.
- Exposure to cold > vasoconstriction.

Pulmonary receptors:

- Lung inflation > vasoconstriction.

Control Mechanisms at Different Time Intervals After the Onset of Disturbance:-

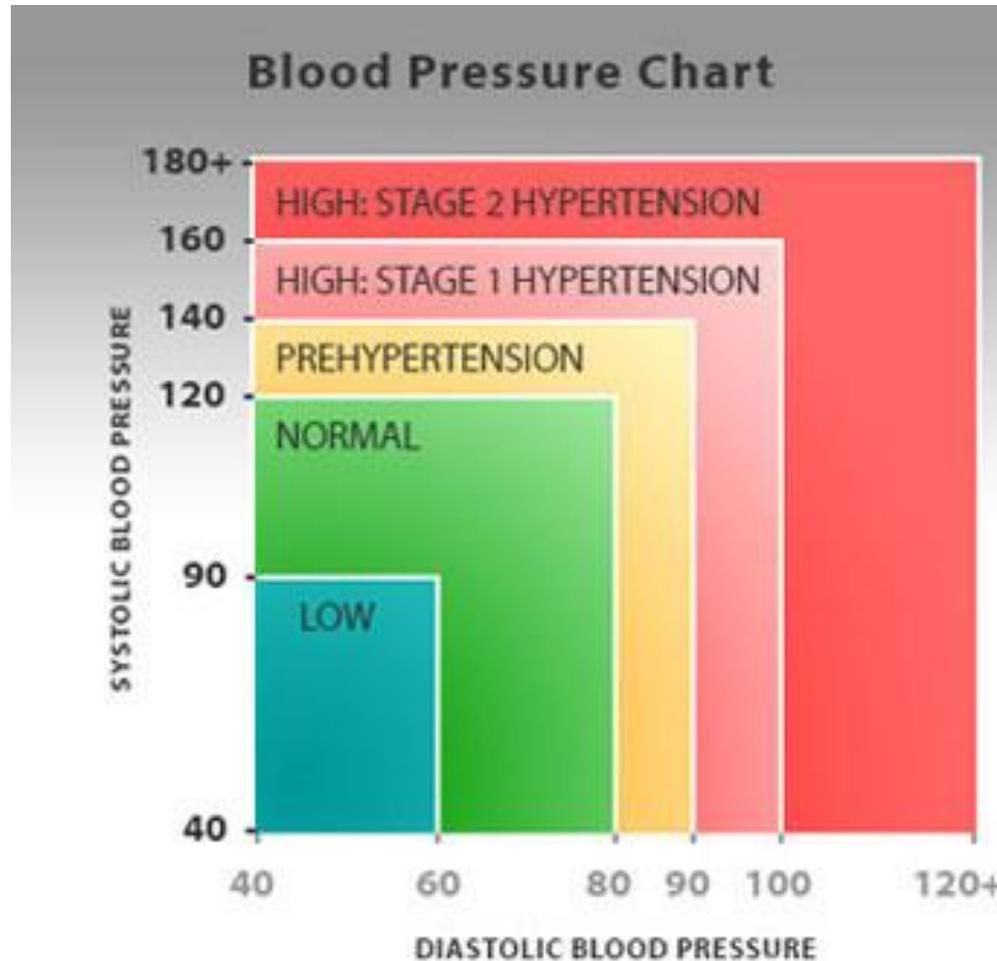
*Not important





Vertical integration

Regulation Of Blood Pressure:-



Complications of Hypertension:- (Mentioned in the Objectives)

What was mentioned in class:

- Cardiac remodeling.
- LV hypertrophy.
- Heart failure.

Further complications and their explanation:

The excessive pressure on your artery walls caused by high blood pressure can damage your blood vessels, as well as organs in your body. The higher your blood pressure and the longer it goes uncontrolled, the greater the damage.

Uncontrolled high blood pressure can lead to:

- **Heart attack or stroke.** High blood pressure can cause hardening and thickening of the arteries (atherosclerosis), which can lead to a heart attack, stroke or other complications.
- **Aneurysm.** Increased blood pressure can cause your blood vessels to weaken and bulge, forming an aneurysm. If an aneurysm ruptures, it can be life-threatening.
- **Heart failure.** To pump blood against the higher pressure in your vessels, your heart muscle thickens. Eventually, the thickened muscle may have a hard time pumping enough blood to meet your body's needs, which can lead to heart failure.
- **Weakened and narrowed blood vessels in your kidneys.** This can prevent these organs from functioning normally.
- **Thickened, narrowed or torn blood vessels in the eyes.** This can result in vision loss.
- **Metabolic syndrome.** This syndrome is a cluster of disorders of your body's metabolism, including increased waist circumference; high triglycerides; low high-density lipoprotein (HDL) cholesterol, the "good" cholesterol; high blood pressure; and high insulin levels. These conditions make you more likely to develop diabetes, heart disease and stroke. **Trouble with memory or understanding.** Uncontrolled high blood pressure may also affect your ability to think, remember and learn. Trouble with memory or understanding concepts is more common in people with high blood pressure.

COMPLICATIONS OF HYPERTENSION

"THE 5C'S"

C	CORONARY ARTERY DISEASE Can lead to narrowing of blood vessels making them more likely to block from blood clots.	
C	CHRONIC RENAL FAILURE Constant high blood pressure can damage small blood vessels in the kidneys making it not to function properly.	
C	CONGESTIVE HEART FAILURE Pumping blood against the higher pressure in the vessels causes the heart muscles to thicken.	
C	CARDIAC ARREST High blood pressure can cause CAD, damaged arteries cannot deliver enough oxygen to other parts of the body eventually leading to heart attack.	
C	CEREBROVASCULAR ACCIDENT Hypertension leads to atherosclerosis and hardening of the large arteries. This, in turn, can lead to blockage of small blood vessels in the brain.	

LEARN MORE: HYPERTENSION COMPLICATIONS

The excessive pressure on the artery walls caused by hypertension or high blood pressure can damage the blood vessels, as well as organs in the body. The higher the blood pressure and the longer it goes uncontrolled, the greater the damage. With time, hypertension increases the risk of heart disease, kidney disease, and stroke.





Case Study

Clinical Scenario:-

In the emergency department, and a 45-year-old male patient is brought in by ambulance following a motor vehicle accident. The patient was the driver of the vehicle and sustained multiple injuries, including a fractured femur and a deep laceration on his thigh. On initial assessment, you notice signs of significant blood loss, and the patient's blood pressure is 80/50 mm Hg, with a heart rate of 120 beats per minute. The patient is conscious but appears pale, diaphoretic, and anxious.





Question Related to Scenario

Question 1 : What is the primary physiologic compensatory mechanism that the body employs to maintain mean arterial pressure?

Answer: The primary physiologic compensatory mechanism for maintaining mean arterial pressure in response to acute blood loss is the activation of the sympathetic nervous system. This system releases norepinephrine, which constricts blood vessels, raising peripheral resistance and increasing blood pressure.



Question Related to Scenario

Question 2 : Explain the role of heart rate in compensating for acute blood loss and its impact on mean arterial pressure?

Answer:

An elevated heart rate (tachycardia) helps compensate for acute blood loss by increasing cardiac output. This ensures that a sufficient volume of blood is circulated to maintain tissue perfusion and mean arterial pressure.



Question Related to Scenario

Question 3 : How does the body's adjustment of peripheral resistance contribute to the maintenance of mean arterial pressure?

Answer:

Adjustment of peripheral resistance involves vasoconstriction, which occurs as a response to acute blood loss. Factors influencing this process include sympathetic nerve activity, circulating catecholamines, and local factors like tissue oxygen demand.



Biomedical Ethics

Euthanasia:-

The term “Euthanasia” is derived from Greek, literally meaning “**good death**”. taken in its common usage, however, euthanasia refers to the **Termination of a person’s life, to end their suffering, usually from an incurable or terminal condition.** it is for this reason that euthanasia was also coined the name “**Mercy Killing**”.



EUTHANASIA:-

Types of Euthanasia



Active

deliberate act,
usually
through the
intentional
administration
of lethal drugs,
to end an
incurably or
terminally ill
patient's life.

Passive

deliberate
withholding or
withdrawal of
life-prolonging
medical
treatment
resulting in the
patient's death.



Suggested Research Article



Related Research Article

[The Journal of Physiological Sciences](#)

<https://www.ahajournals.org/doi/10.1161/01.RES.32.5.564>

Review | [Published: 12 November 2019](#)

Sex differences in baroreflex function in health and disease

[Qi Fu](#) ✉ & [Shigehiko Ogoh](#)

The Journal of Physiological Sciences **69**, 851–859 (2019) | [Cite this article](#)

4989 Accesses | **28** Citations | [Metrics](#)

Abstract

This brief review summarizes the current knowledge on sex differences in baroreflex function, with a major focus on studies in humans. It has been demonstrated that healthy women have blunted cardiovagal baroreflex sensitivity during a rapid (i.e., within seconds) hypertensive stimulus, but baroreflex sensitivity is similar between the sexes during a hypotensive stimulus. Normal aging decreases cardiovagal baroreflex sensitivity and the rate of decline is similar in men and women. Cardiovagal baroreflex sensitivity is reduced in pathological conditions such as hypertension and type II diabetes, and the reduction is greater in female patients than male patients. There is no clear sex difference in sympathetic baroreflex sensitivity among young individuals, however, with women of more advanced age, sympathetic baroreflex sensitivity decreases, which appears to be associated with greater arterial stiffness compared with



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Research:-

<https://journals.sagepub.com/doi/10.1177/0268355520938283>

Images and Links:-

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Thank You!