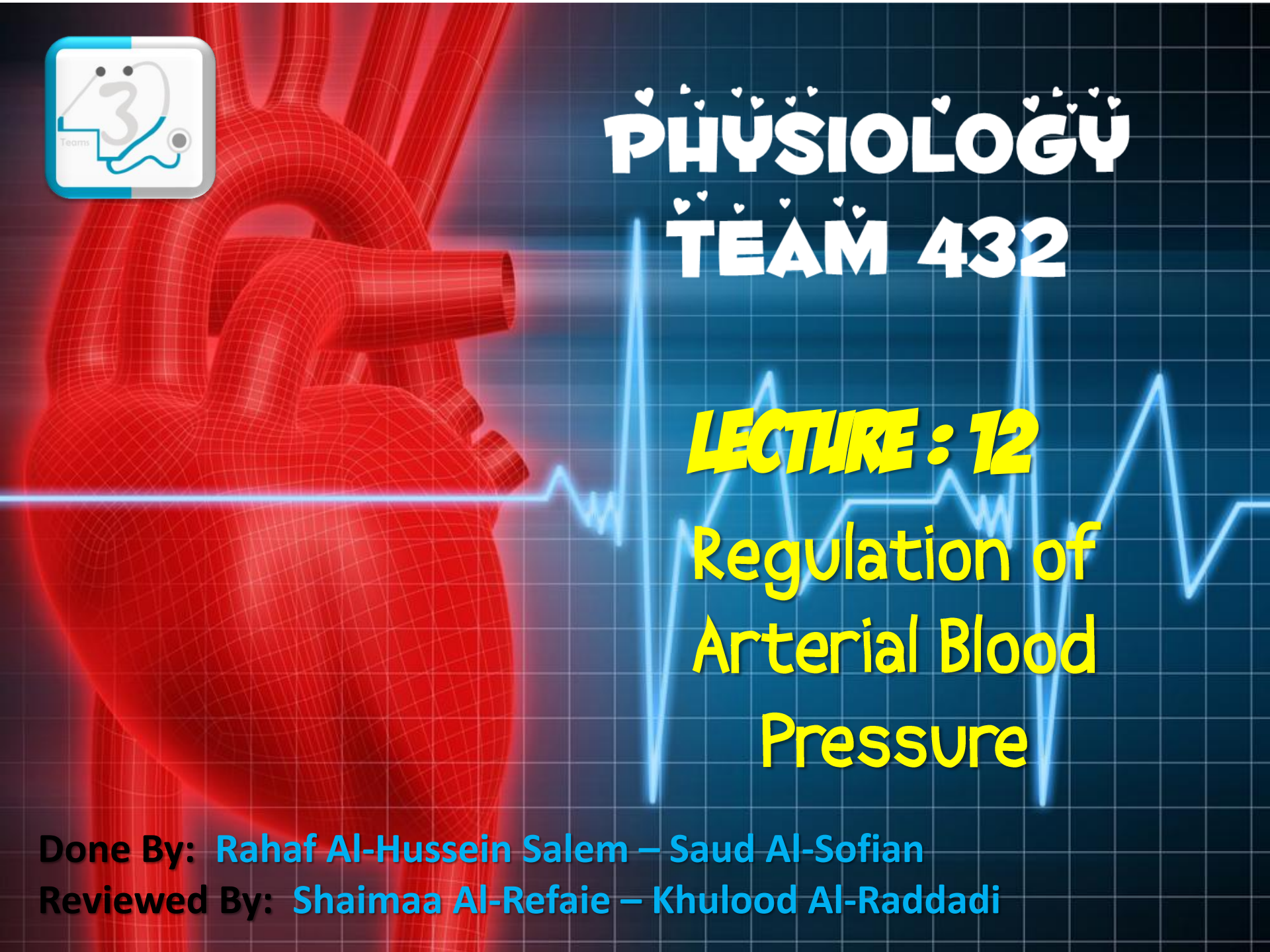


PHYSIOLOGY TEAM 432

LECTURE : 12

Regulation of Arterial Blood Pressure

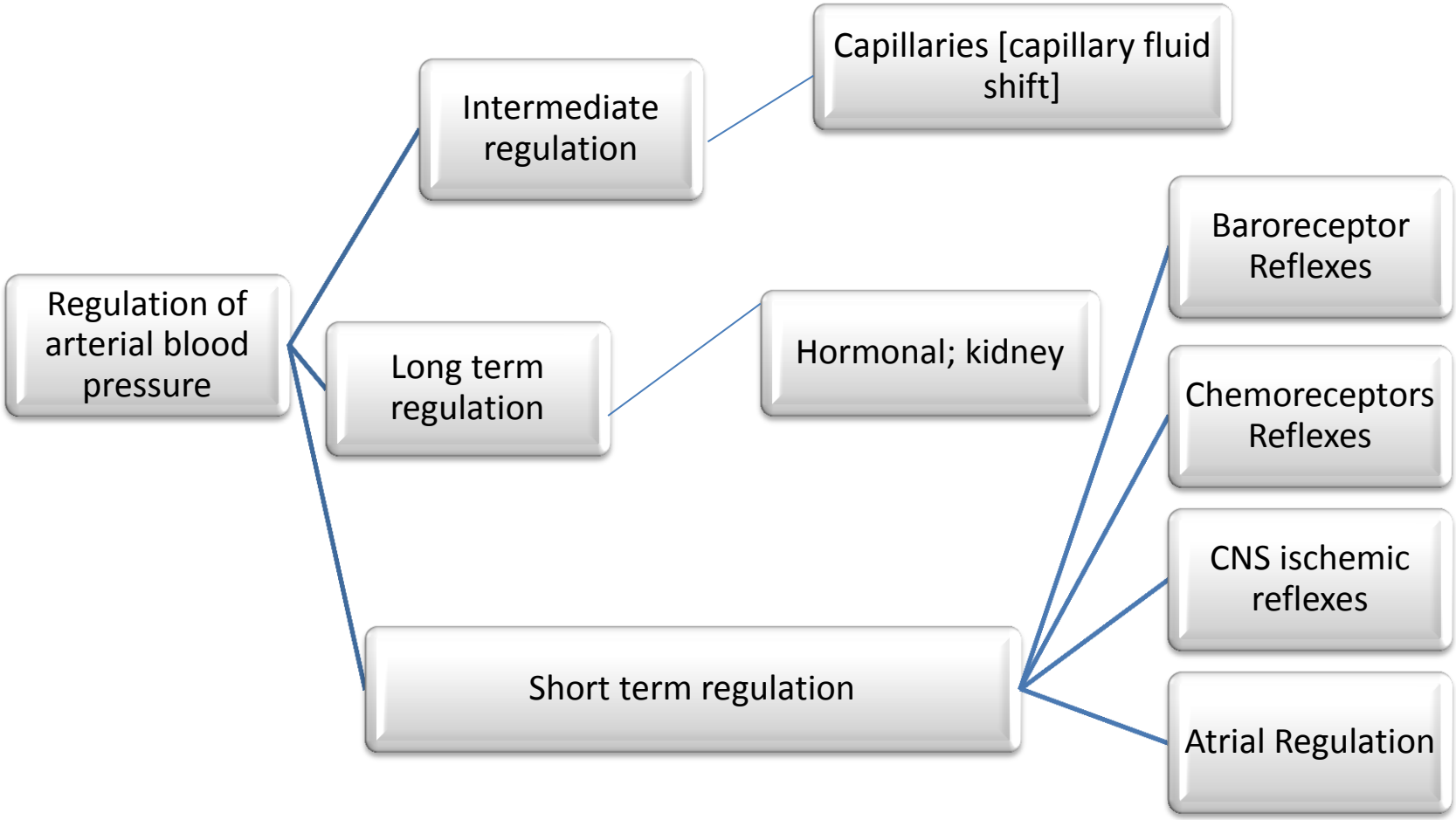
Done By: Rahaf Al-Hussein Salem – Saud Al-Sofian

Reviewed By: Shaimaa Al-Refaie – Khulood Al-Raddadi

OBJECTIVES

1) To understand the regulation of arterial blood pressure and its importance
2) List the intermediate and long-term mechanisms regulating ABP.
3) Explain the intermediate mechanisms in the regulation of ABP; Capillary fluid shift.
4)Explains the role of the kidney in the long-term regulation of ABP.: the renin –angiotensin system and its components.

MIND MAP


Regulation of arterial blood pressure
Intermediate regulation
Capillaries [capillary fluid shift]
Long term regulation
Hormonal; kidney
Short term regulation
Baroreceptor Reflexes
Chemoreceptors Reflexes
CNS ischemic reflexes
Atrial Regulation

Regulation of Arterial Blood Pressure

Short term regulation

a. Baroreceptors

b. Chemoreceptor reflex

c. CNS ischemic reflex

d. Atrial reflexes

Intermediate regulation

Capillary fluid shift

Long -term regulation

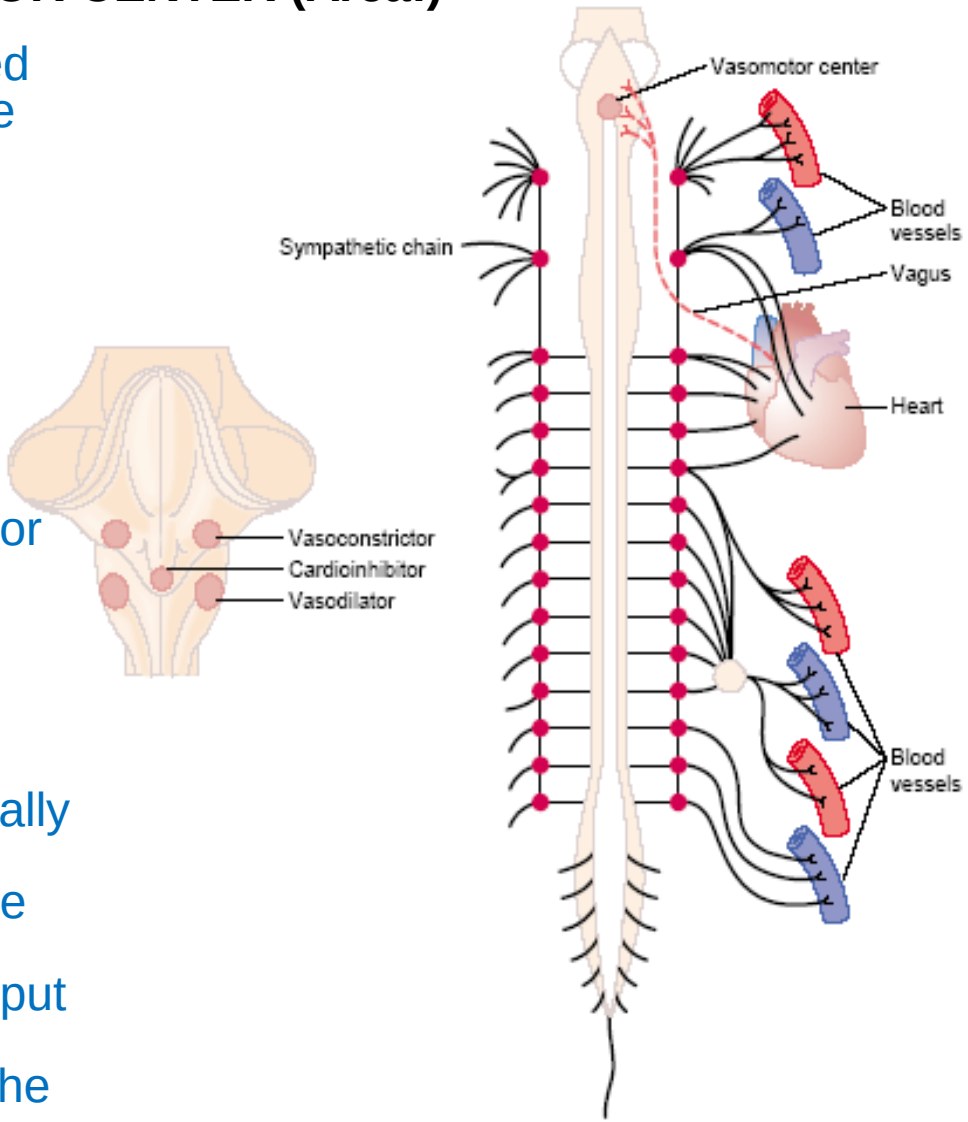
Example : hormonal – role of the kidney and the RAS system

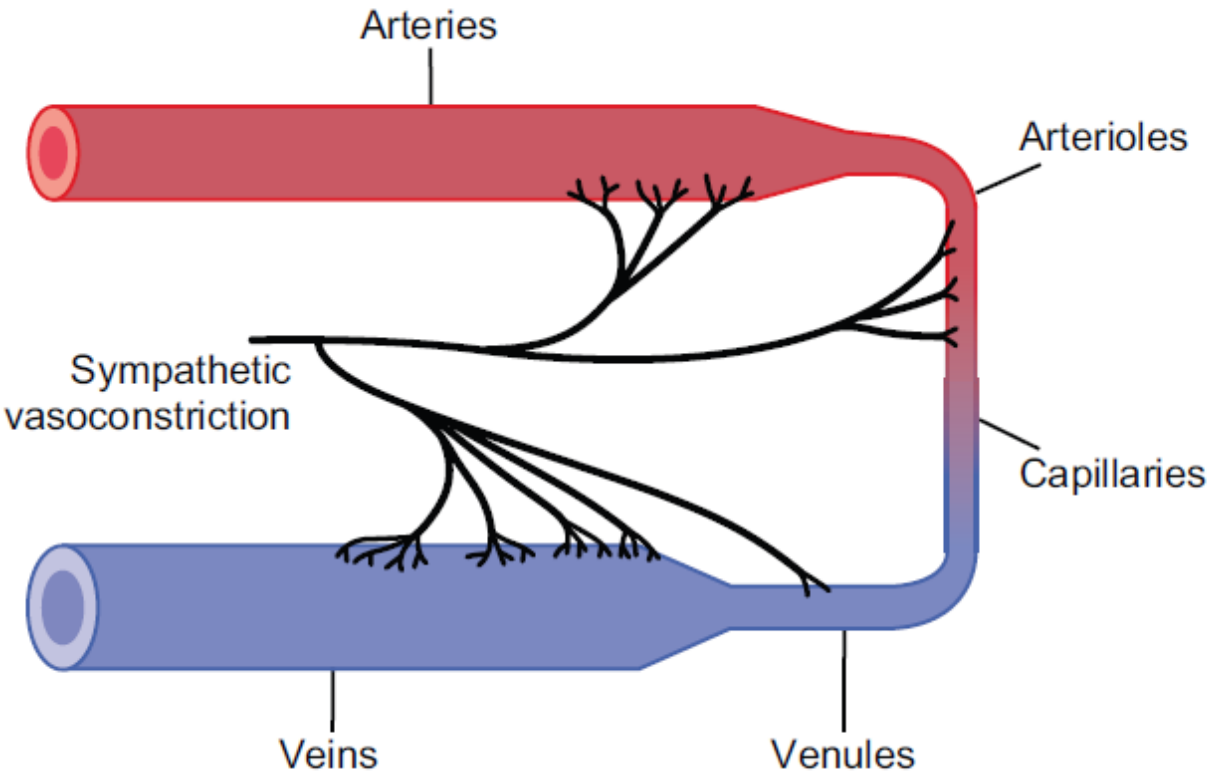
VASOMOTOR CENTER (Area!)

1. A vasoconstrictor area located bilaterally in upper medulla. excite vasoconstrictor neurons of the sympathetic nervous system.

2. A vasodilator area located bilaterally in the lower half of the medulla. inhibit the vasoconstrictor area, thus causing vasodilation.

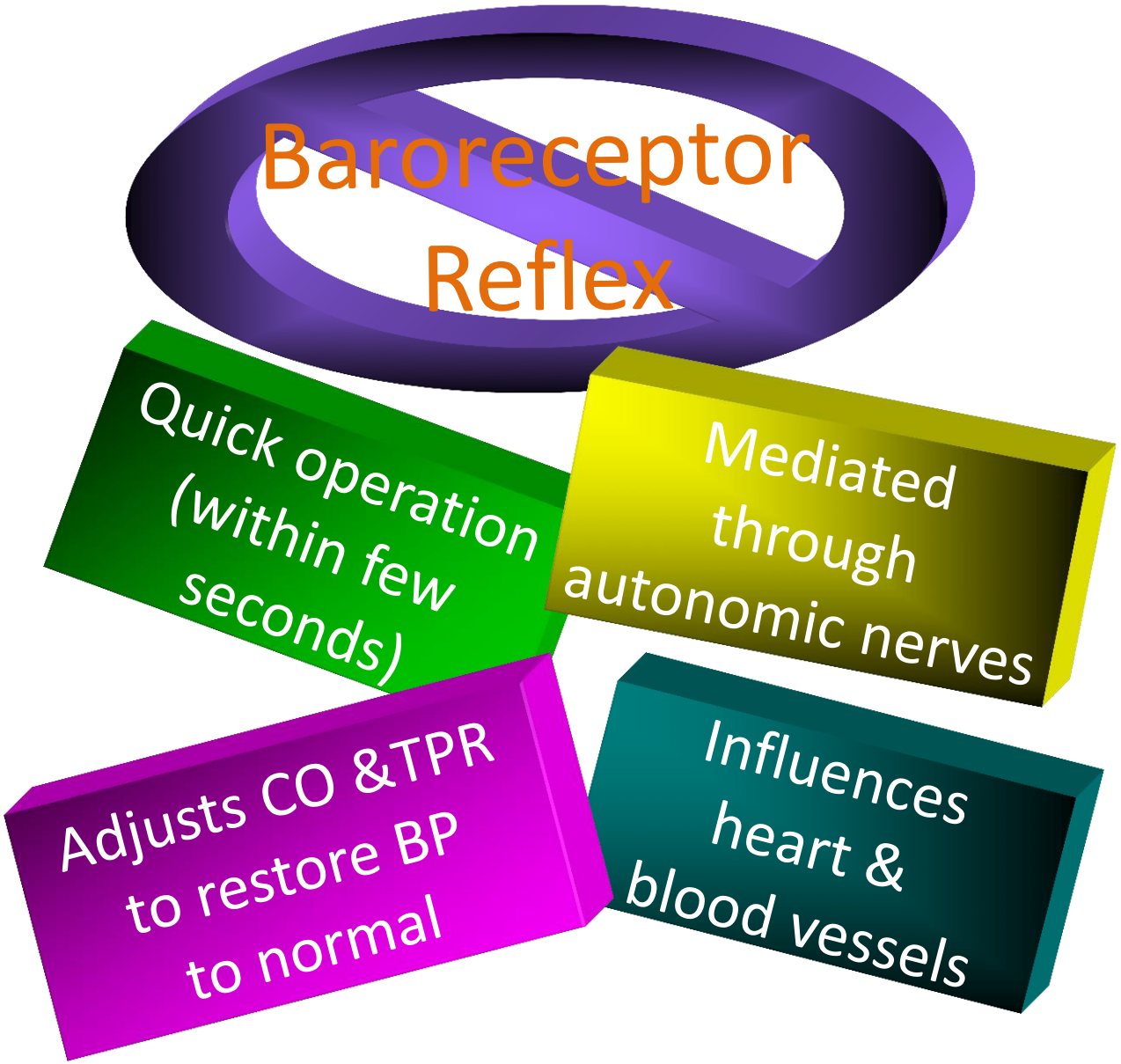
3. A sensory area located bilaterally in medulla and pons (tractus solitarius). Receive sensory nerve signals from vagus and glossopharyngeal nerves and output control the vasoconstrictor and vasodilator areas An example is the baroreceptor reflex





Continuous Partial Constriction of the Blood Vessels Is Normally Caused by Sympathetic Vasoconstrictor Tone.

in most tissues all the vessels *except the capillaries, precapillary sphincters, and metarterioles* are innervated


Baroreceptor Reflex
Quick operation (within few seconds)
Mediated through autonomic nerves
Adjusts CO &TPR to restore BP to normal
Influences heart & blood vessels

Renal Control

It is perfect
100 %

Slow operation
(within hours to Days)

Mediated
through
Kidneys Renin Angiotensin
aldosterone mechanism,

Adjusts urinary output and TPR
to restore BP
to normal

Influences
Kidneys &
blood vessels

CONTROL OF VMC

Reticular Substance of Brain Stem

Hypothalamus

Posterolateral portions Cause Excitation. Anterior part can cause Excitation or Inhibition

Cerebral Cortex

Motor Cortex Cause Excitation

VMC=Vasomotor center

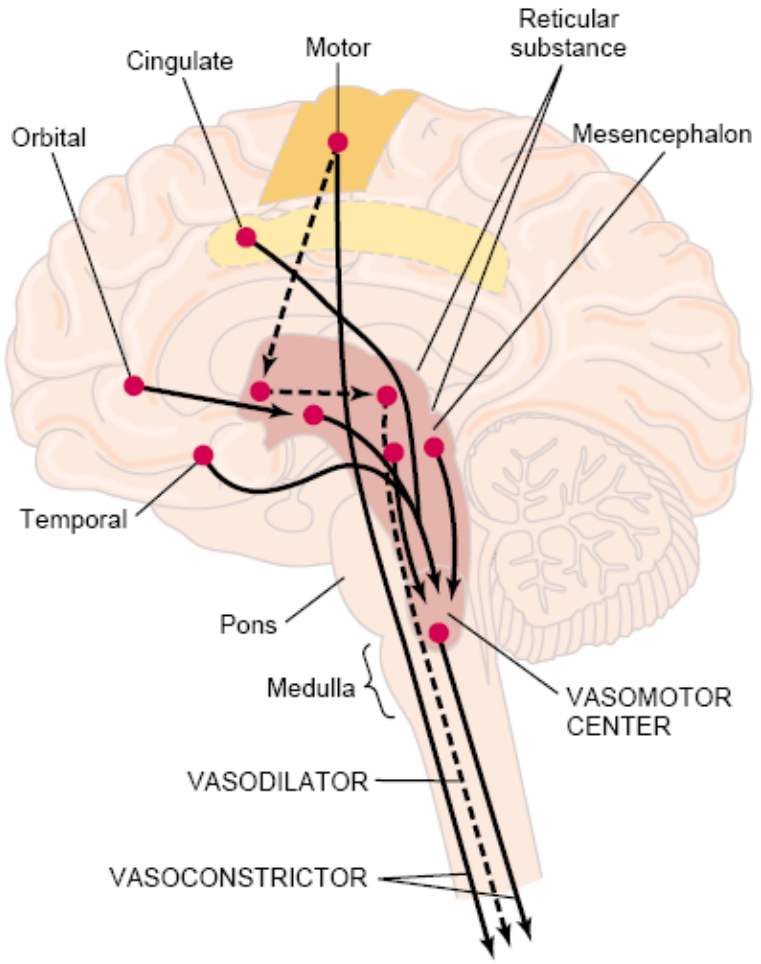


Figure 18–3

A. Baroreceptor Reflexes:

Baroreceptors:

Stretch receptors.

Located in: Carotid sinus and aortic arch .

They sense the blood pressure in the aortic arch and internal carotid → send signal to the vasomotor center in the medulla oblongata along vagus and glossopharyngeal Nerves (afferent)

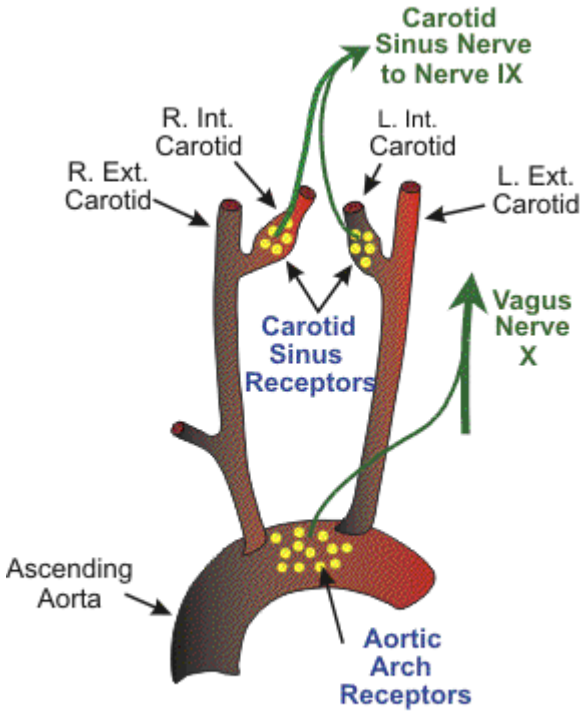


Figure 1. Location and innervation of arterial baroreceptors.

They respond to a rapidly changing BP. In the range 60-180 mmHg and therefore is responsible for regulation of pressure in acute changes not chronic [because of the resetting of baroreceptors] also in between the heart beats. When you change your posture from superior to erect, a drop in ABP in the head and upper part of the body will occur.

As barorecptor reflex becomes activated, strong sympathetic impulses lead to Vasoconstriction and minimize the decrease in BP.

http://www.youtube.com/watch?v=zqcnExm3xdY

Stretch of receptors → increase the rate of firing and impulses travel along vagus & glossopharyngeal to the medullary CVCs:

(+) vagal center : decrease HR.

(-)vasoconstrictor center: VD (vasodilation will occur)

Increase in ABP

Resetting of baroreceptors: This makes baroreceptors not suitable for long term regulation of ABP, as they are rapidly reset to the new pressure. Adaptation of a receptor means decrease in impulse discharge from the receptor despite persistence of the stimulus.

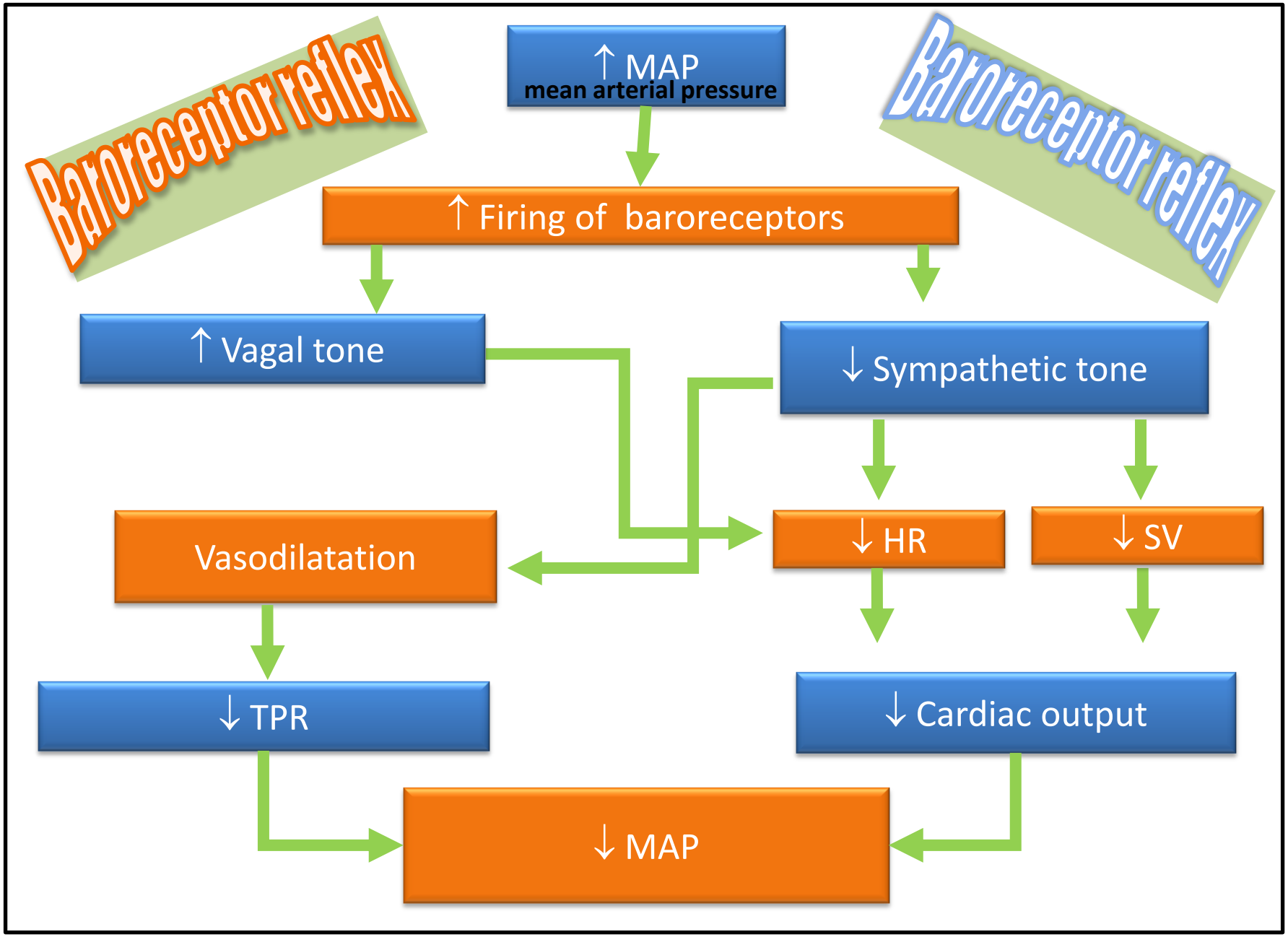
---

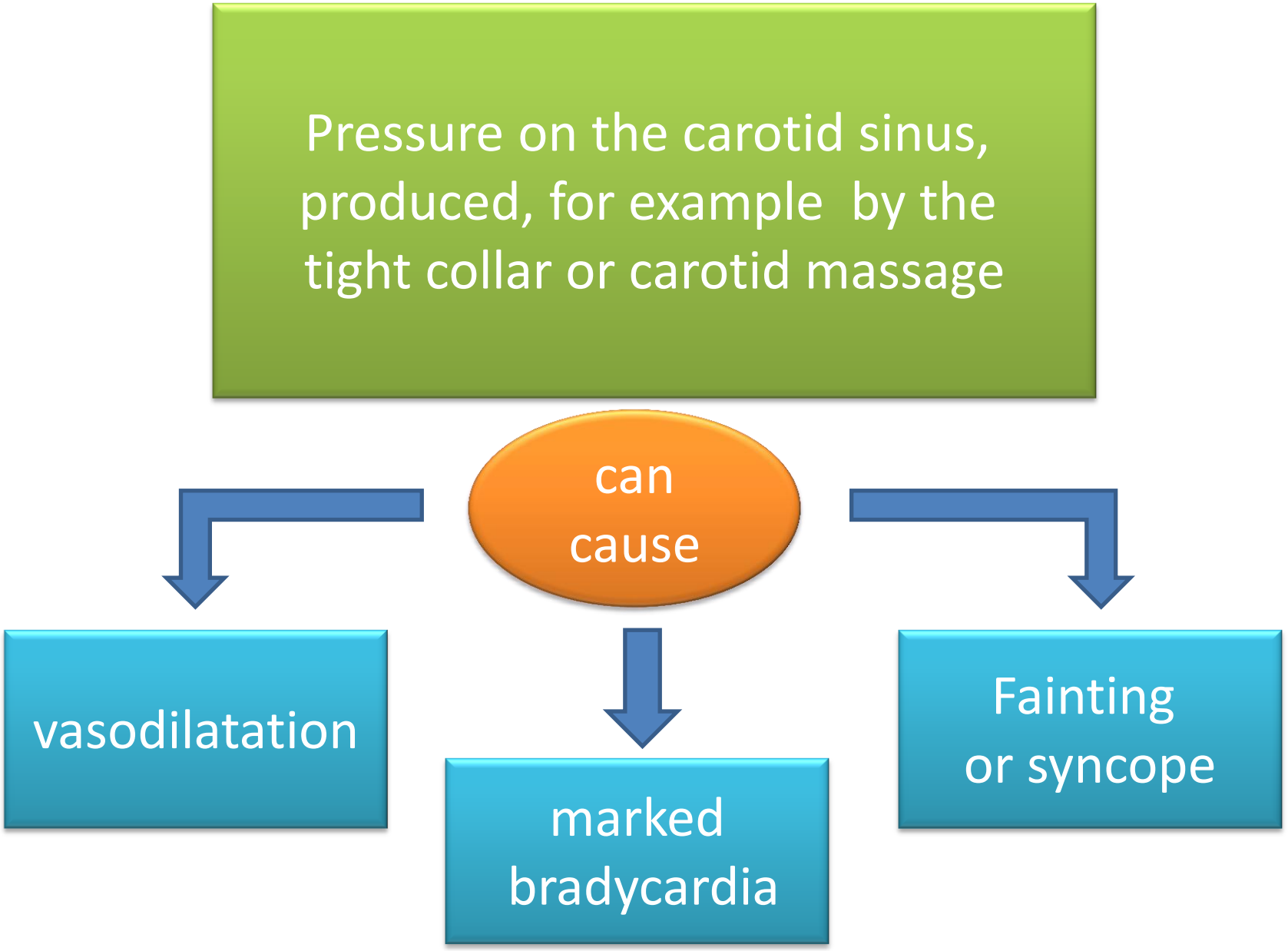
will decrease the inhibitory impulse discharge from baroreceptors → vasomotor center is released from inhibition resulting in:

(+) heart: Heart Rate & contractility.

(+) sympathetic vasoconstriction tone: Vasoconstriction

Decrease in ABP


Baroreceptor reflex
↑ MAP
mean arterial pressure
Baroreceptor reflex
↑ Firing of baroreceptors
↑ Vagal tone
↓ Sympathetic tone
Vasodilatation
↓ HR
↓ SV
↓ TPR
↓ Cardiac output
↓ MAP


Pressure on the carotid sinus, produced, for example by the tight collar or carotid massage
can cause
vasodilatation
marked bradycardia
Fainting or syncope

Syncope

Transient loss of consciousness

Associated with

Abrupt vasodilatation

Inadequate cerebral blood flow

Hypotension and bradycardia

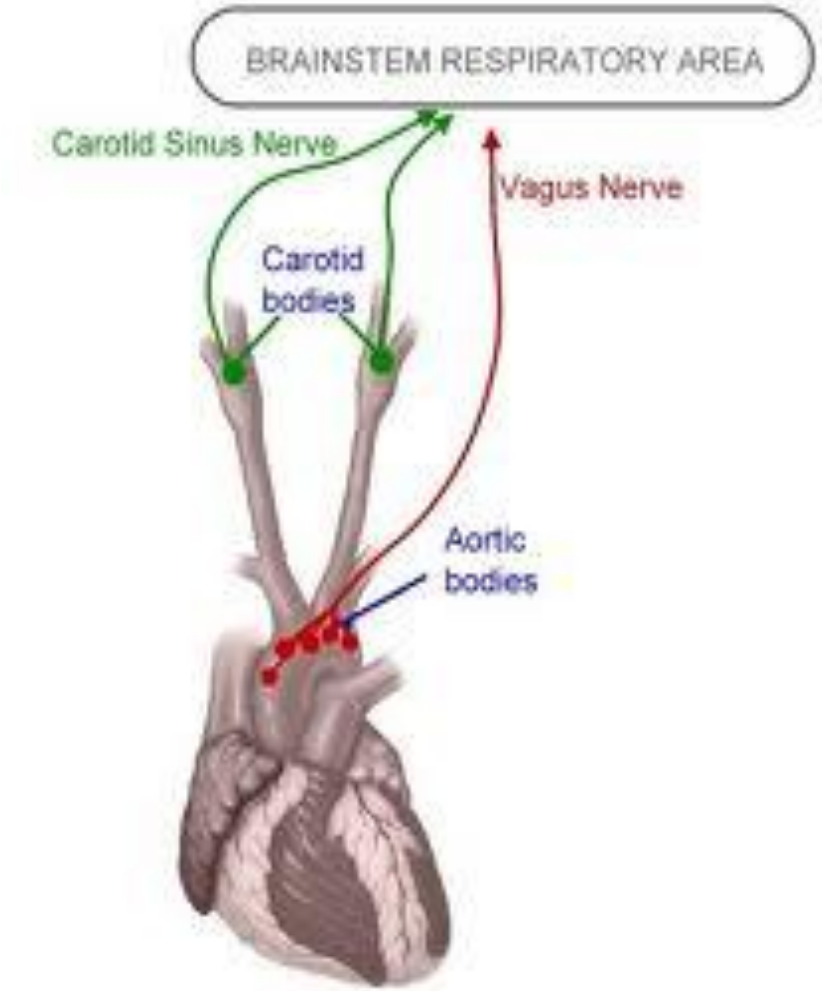
B. Chemoreceptors Reflex:

Chemosensitive cells, are stimulated in response to: **lack of oxygen , CO2 excess, H+ excess.** Carotid and Aortic **BODIES** are responsible for it.

They have a very high blood flow (1200 ml/min/g tissue). This makes it easy for these cells to detect **changes in O2, CO2, and H+.** Become **activated** when ABP becomes less than 60 mmHg (tissue perfusion will decrease). So, they are not involved in ABP control at normal range.

***1st emergency reflex to regulate ABP ***

When blood flow to chemoreceptors decreases it leads to decrease in O2, and increase in CO2 & H+ → (+) chemo. Signals (+) CVS which will result in , tachycardia , venoconstriction and vasoconstriction.



http://www.youtube.com/watch?v=AawfvwY2Muk

C. CNS Ischemic Response:

- It is one of the most powerful activators of the sympathetic vasoconstrictor system.
- Operates as an emergency arterial pressure control system, which acts rapidly and powerfully in order to prevent further decrease in ABP whenever blood flow to the brain decreases to a lethal level.
- When the **B.P. <20** due to an accident or increase in intracranial pressure [which will compress on the blood vessels entering the brain (due to a tumor or other reasons)] → cerebral ischemia, the vasomotor center responds directly to ischemia and become strongly excited to raise the blood pressure (STRONG VASOCONSTRICTION of blood vessels including Kidneys) as high as the heart can possibly pump

D. Atrial Reflexes:

if blood volume is increased? e.g infusing 500 ml into a person:
Increase in blood volume→ stretch of the atria leading to:
a. (+) ANP release causing vasodilation of renal vessels, diuresis, natriuresis.
b. Hypothalamus:
1. (-) ADH (vasopressin) → water diuresis.
2. (-) sympathetic discharge → vasodilation of renal vessels
c. stretch SAN and increase HR(the tachycardia is not as a result of sympathetic rather than the stretch receptors)

Norepinephrine will increase the TPR while water will increase the blood volume.

if blood volume is decreased? e.g an accident or dehydration:
Decrease in blood volume→
a. (-) ANP release causing vasoconstriction of renal vessels, diuresis, natriuresis.
b. Hypothalamus:
1. (+) ADH → water retention.
2. (+) sympathetic discharge → Vasoconstriction of renal vessels

Anti-diuretic hormone = ADH (released by the hypothalmus)
It is secreted by the posterior pituitary in response to ↑blood osmolarity (often due to dehydration).

- Action: Promote water reabsorption by the kidney tubules → H_2O moves back into the blood → less urine formed

Measurement of arterial blood pressure

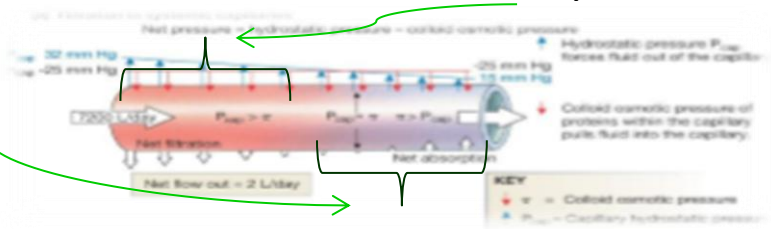
If you put a stethoscope on an artery you don't hear any sound because of the stream line flow in the arteries .. Unlike the turbulent flow; it will be heard , turbulent flow is heard when the artery is partially constricted or if there were a connection between the artery and vein (AV shunt or AD current).

Note: Dr. asked if we knew the **kortkoff sounds**.. So makes sure you know them

http://en.wikipedia.org/wiki/Korotkoff_sounds

Intermediate mechanism

- **Intermediate : works within hours ; by the capillary fluid shift**
- ✓ **Function of the capillary mainly is for tissue perfusion**
- ❑ **The arterial side , Fluids are moving from arterial side to the tissue [bulk flow out], which contain molecules such as oxygen and glucose ; this is because of the high capillary hydrostatic pressure**
- ❑ **Excess fluids return through the venous pathway [bulk flow in] as a result of low capillary hydrostatic pressure**
- Note :In the picture its important to differentiate between the arterial part of the capillary and the venous side of it



<u>Early signs of dehydration :</u>

Dryness of the mouth and lips , pinch the skin near the bone .. If it doesn't go back normally.. It means there is loss of the elasticity , as the fluids have left the skin and went to the circulation to maintain blood pressure and volume (compensatory mechanism), when this occurs it is called fluid shift ..
This pinching is witnessed after at least 12 hours of dehydration

Capillary fluid shift mechanism:

- ✓ **Activated within 30 minutes up to hours. This effect lasts for long period as days.**

- ➢ **Any time capillary pressure falls too low**

Fluid is absorbed by osmosis from the tissues [skin] into circulation

[patient has been dehydrated for at least 12 hours]

Build up blood volume and Increase PB toward normal

- ➢ **If capillary pressure increases too high (such as over hydration , heart failure , liver failure)**

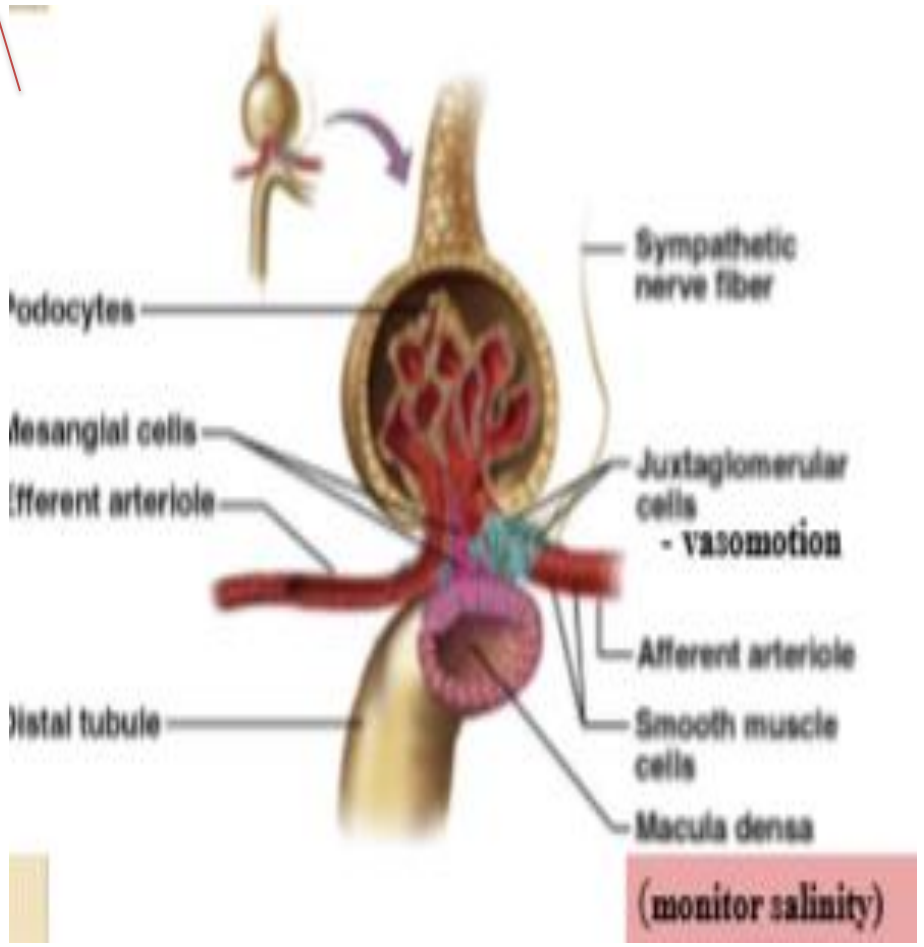
Fluid moves out of the circulation into the ECF-compartment [skin; edema is seen]

Blood volume & pressure back to normal

In water poisoning the ADH or vasopressin is not inhibited. So you're drinking water and there is no drainage of the water because of the water retention caused by the ADH

Long-term Regulation of ABP: Role of the kidney & Renin-angiotensin system

The picture represents a distal convoluted tubules (juxtaglomerular apparatus) of the kidney which contains sensors called macula densa , it monitors the salinity (salinity= Soudium concentration



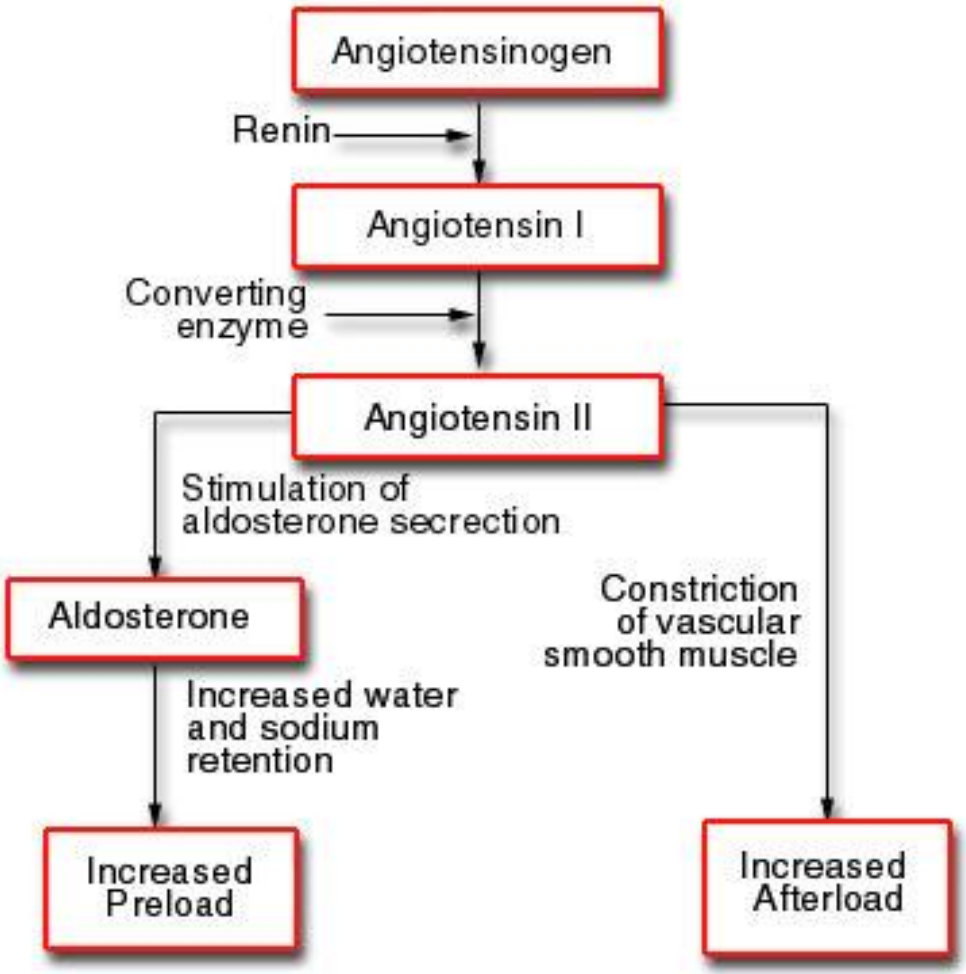
If the concentration of the sodium in the macula densa is high (too much sodium in the body) ; the kidney will lose sodium as a result natriuses occur and we will get rid of it in urine. If the concentration is low , salt and water retention a will occur – this mechanism is a result of the renin angiotensin system

Renin Angiotensin System

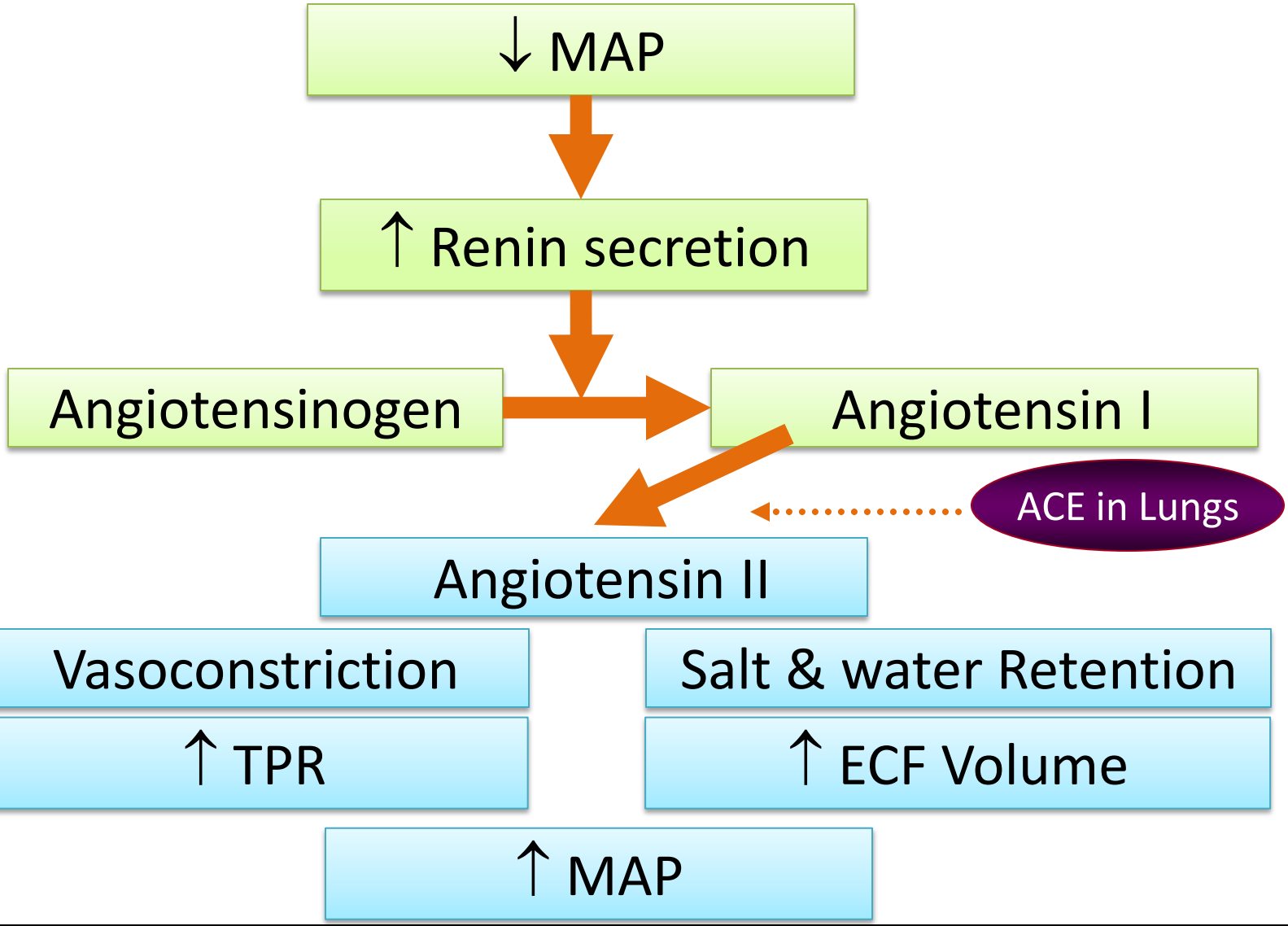
Renin is synthesized and stored in an *inactive form called prorenin* in the juxtaglomerular cells (JG cells)of the kidneys.

The JG cells are modified smooth muscle cells located in the walls of the afferent arterioles immediately proximal to the glomeruli.

Thus, the renin-angiotensin system is an automatic feedback mechanism that helps maintain the arterial pressure at or near the normal level even when salt intake is increased or decreased.



Renal control

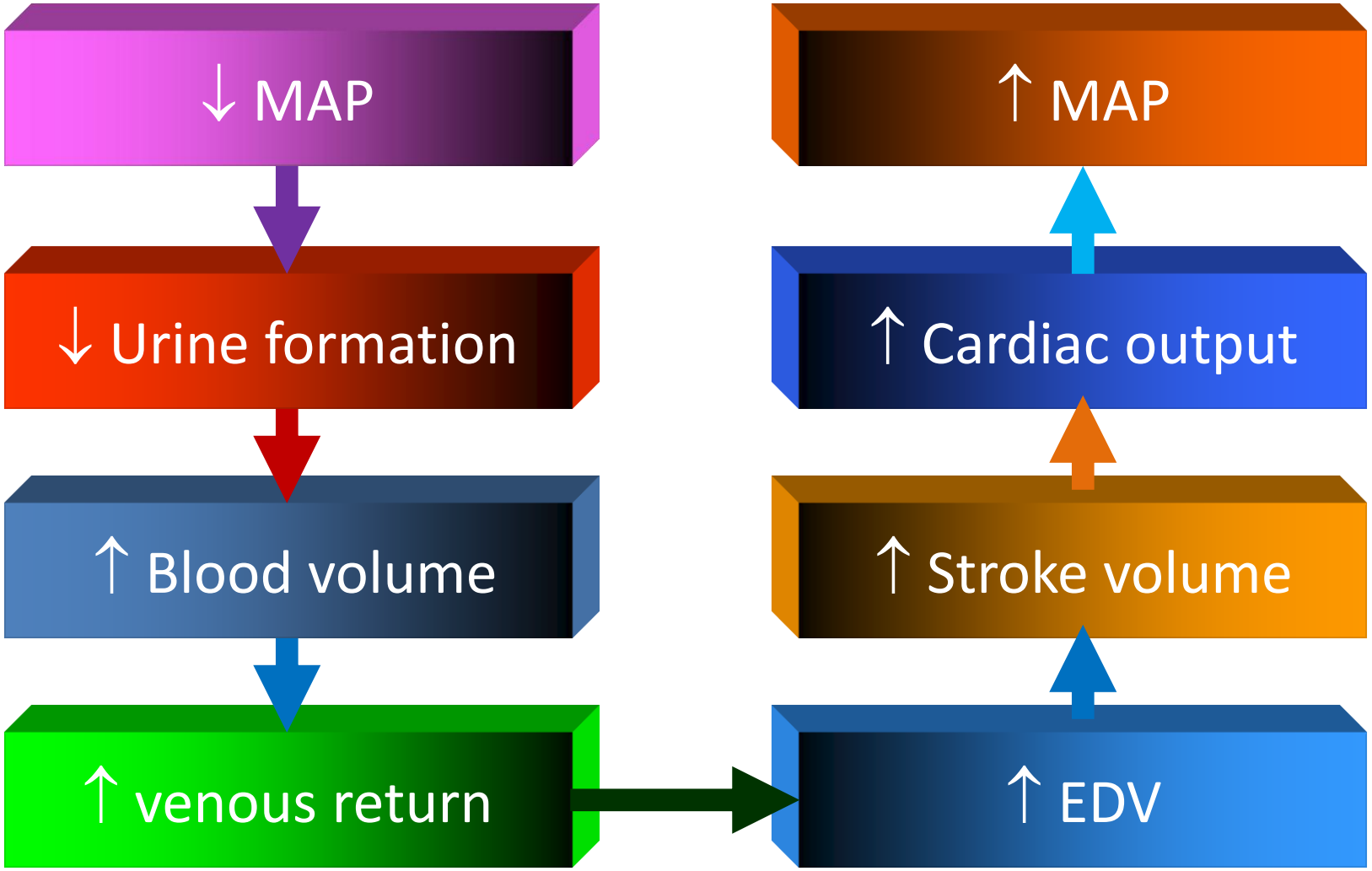


Role of the Renin-Angiotensin System in Maintaining a Normal Arterial Pressure Despite Wide Variations in Salt Intake:

One of the most important functions of the renin-angiotensin system is to allow a person to eat either very small or very large amounts of salt without causing great changes in either extracellular fluid volume or arterial pressure.

The initial effect of increased salt intake → elevate the extracellular fluid volume → elevate the arterial pressure → increased blood flow through the kidneys, → reduces the rate of secretion of renin to a much lower level → decreased renal retention of salt and water = return of the extracellular fluid volume almost to normal, and, finally, return of the arterial pressure also almost to normal.

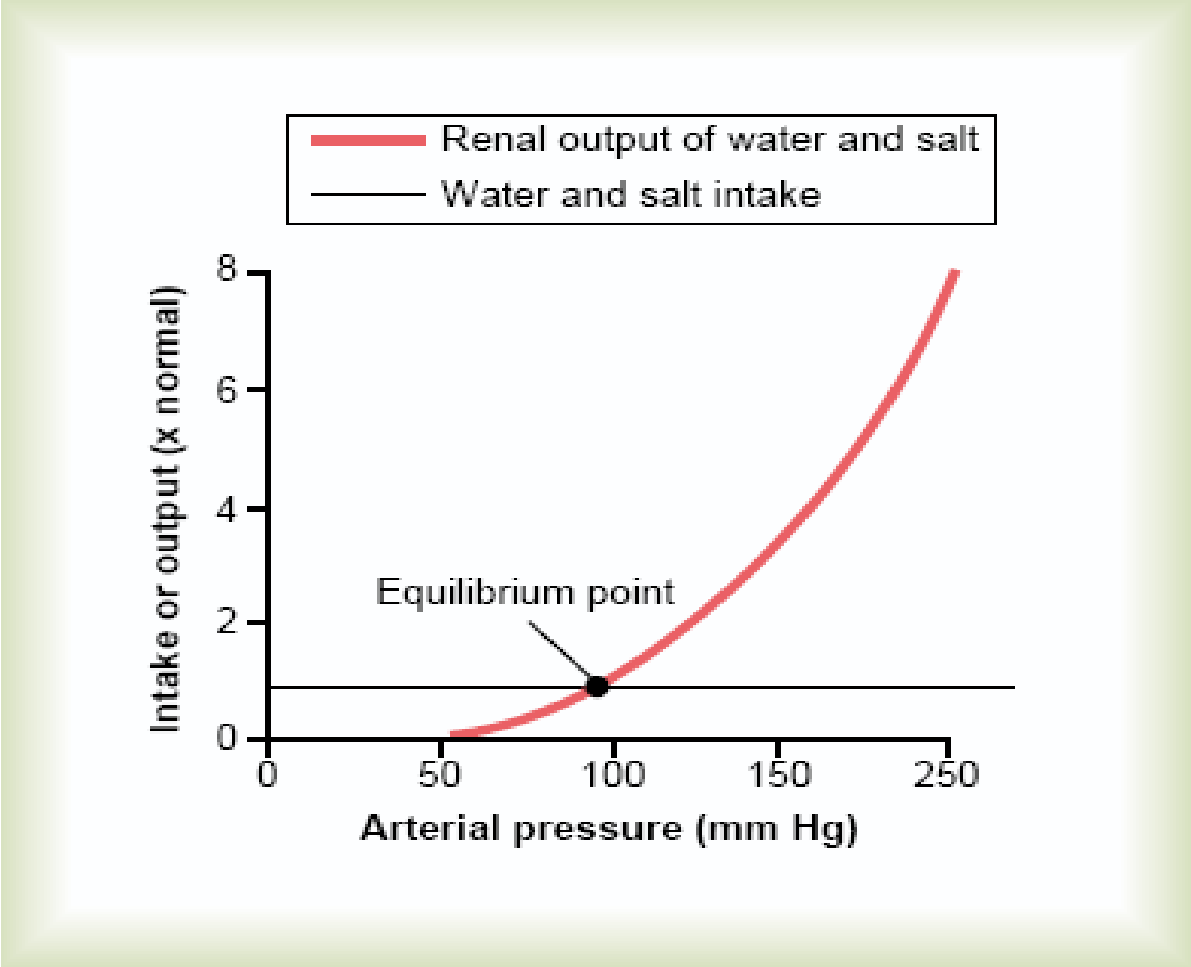
When the arterial pressure falls, intrinsic reactions in the kidneys themselves cause many of the prorenin molecules in the JG cells to split and release renin. → renin enters the renal blood and then passes out of the kidneys to circulate throughout the entire body → increase of renal water and salt retention causing an increase in the pressure


↓ MAP
↓ Urine formation
↑ Blood volume
↑ venous return
↑ EDV
↑ Stroke volume
↑ Cardiac output
↑ MAP

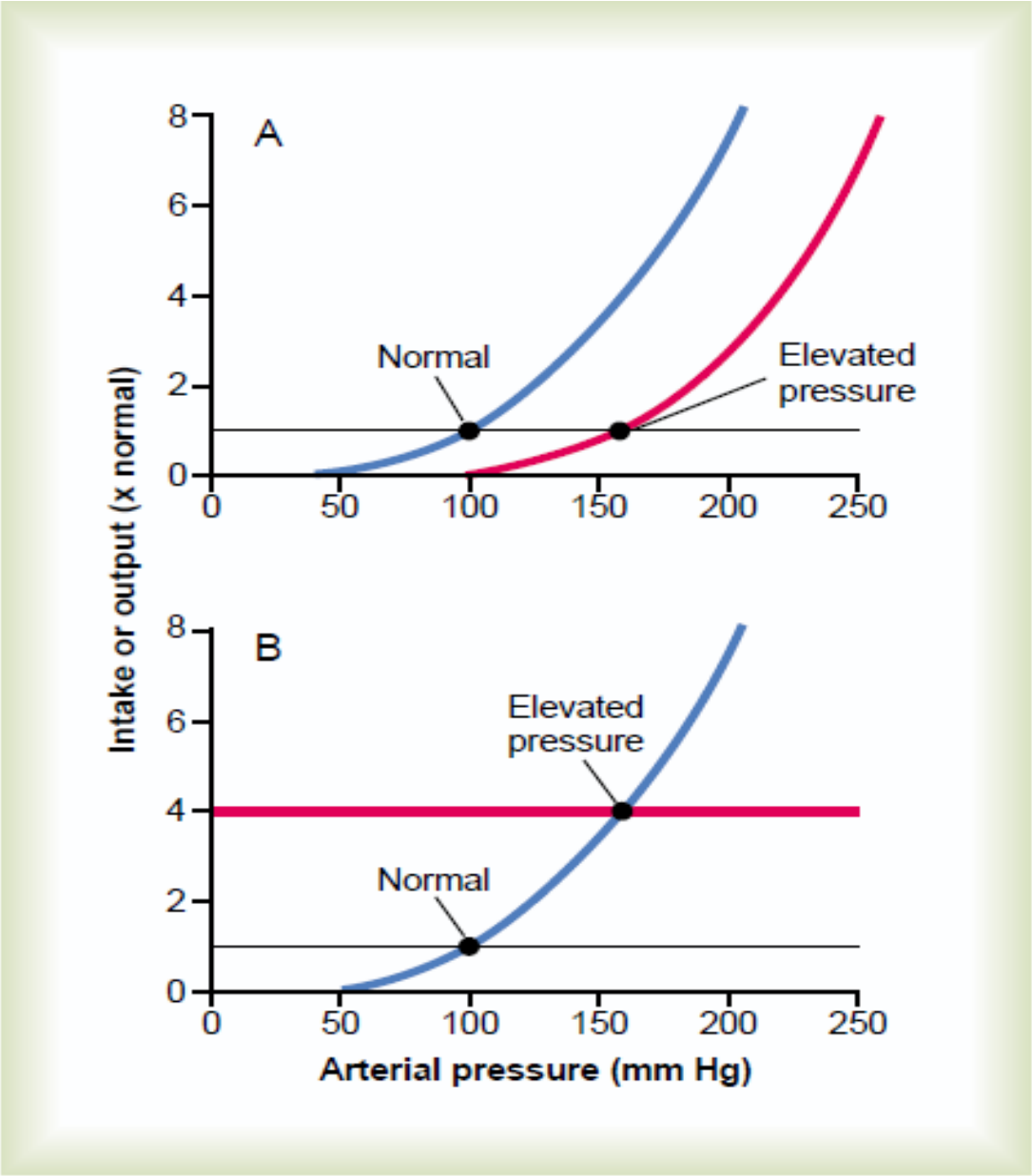
Effect of Salt Intake on the body

Increased Salt Intake	Decreased Salt Intake
Increased extracellular volume	Decreased extracellular volume
Increased arterial pressure	Decrease in arterial pressure
Decreased renin and angiotensin	Increased renin and angiotensin
Decreased renal retention of salt and water	Increased renal retention of salt and water
Return of extracellular volume almost to normal	Return of extracellular volume almost to normal
Return of arterial pressure almost to normal	Return of arterial pressure almost to normal

Pressure Natriuresis and Pressure Diuresis



Two ways in which the arterial pressure can be increased: *A, by* shifting the renal output curve in the right-hand direction toward a higher pressure level or *B, by increasing the intake level of salt* and water.



Functions of Angiotensin II:

1. Vasoconstriction.
2. Decreases excretion of both salt and water by the kidneys (main mechanism for long-term regulation).

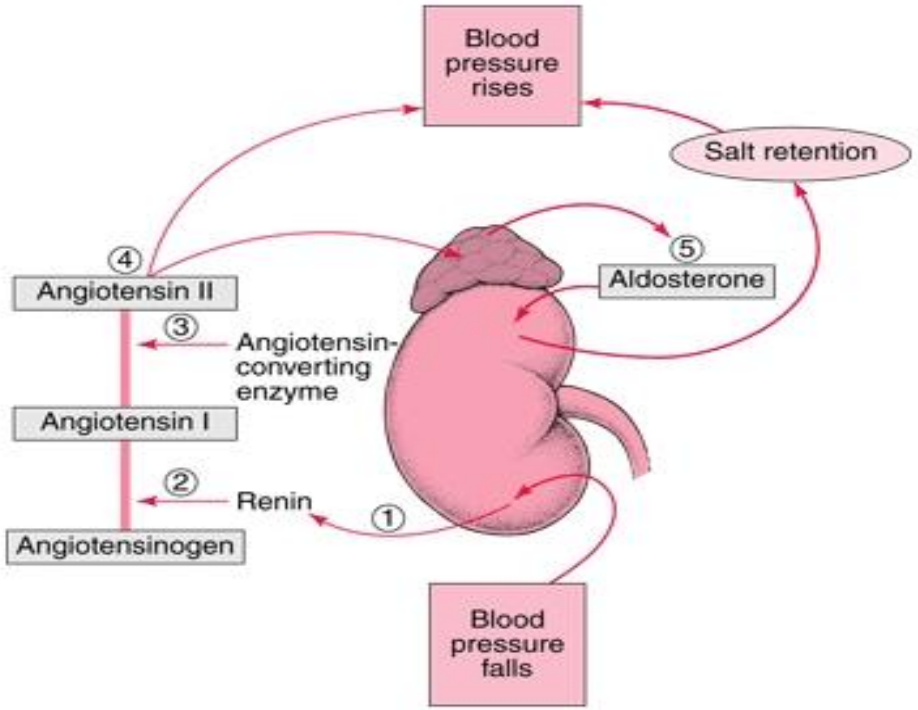
Angiotensin causes the kidneys to retain both salt and water in two major ways

Angiotensin acts directly on the kidneys to cause salt and water retention.

Angiotensin causes the adrenal glands to secrete aldosterone, and the aldosterone in turn increases salt and water reabsorption by the kidney tubules. Thus, whenever excess amounts of angiotensin circulate in the blood, the entire long-term renal–body fluid mechanism for arterial pressure control automatically becomes set to a higher arterial pressure level than normal

Control of blood volume

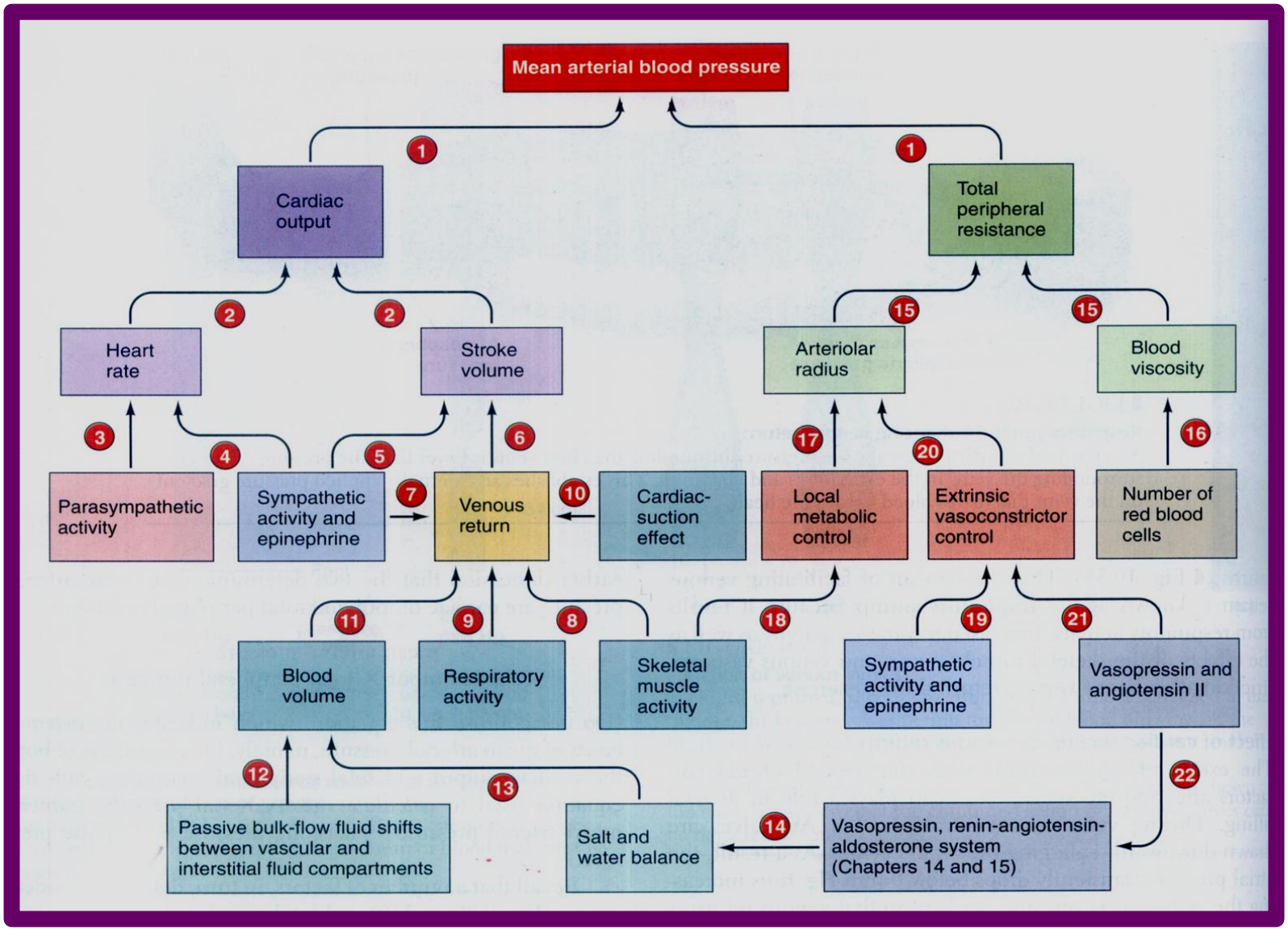
Aldosterone: - Secreted by the adrenal cortex triggered by angiotensin II → Promotes sodium reabsorption by the kidney tubules (Na+ moves back into the blood and - H2O follows by osmosis)
Whereas ADH promotes H2O reabsorption only (in response to dehydration)→ aldosterone promotes reabsorption of both H2O and salt (in response to ↓ BP)



Salt sensitive hypertensive patients ; there's a problem in the kidney , can't excrete excess load of sodium so increase blood volume and blood pressure those people are not allowed to have salt

This is just an extra piece of information mentioned in the lecture by the Dr.

SUMMARY


Mean arterial blood pressure
1
1
Cardiac output
Total peripheral resistance
2
2
15
15
Heart rate
Stroke volume
Arteriolar radius
Blood viscosity
3
4
5
6
17
20
16
Parasympathetic activity
Sympathetic activity and epinephrine
7
Venous return
10
Cardiac-suction effect
Local metabolic control
Extrinsic vasoconstrictor control
Number of red blood cells
11
9
8
18
19
21
Blood volume
Respiratory activity
Skeletal muscle activity
Sympathetic activity and epinephrine
Vasopressin and angiotensin II
12
13
22
Passive bulk-flow fluid shifts between vascular and interstitial fluid compartments
Salt and water balance
14
Vasopressin, renin-angiotensin-aldosterone system (Chapters 14 and 15)

SUMMARY

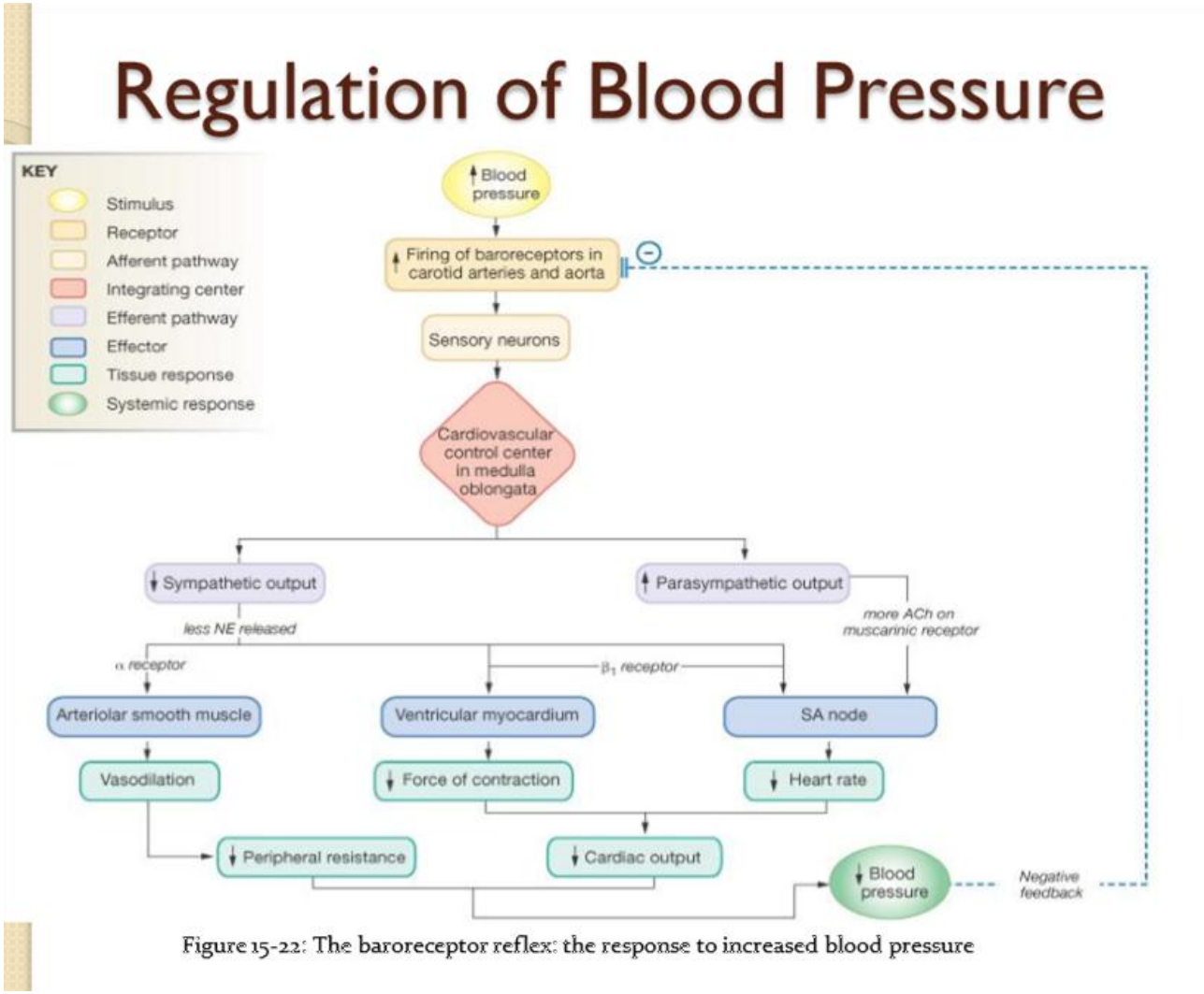
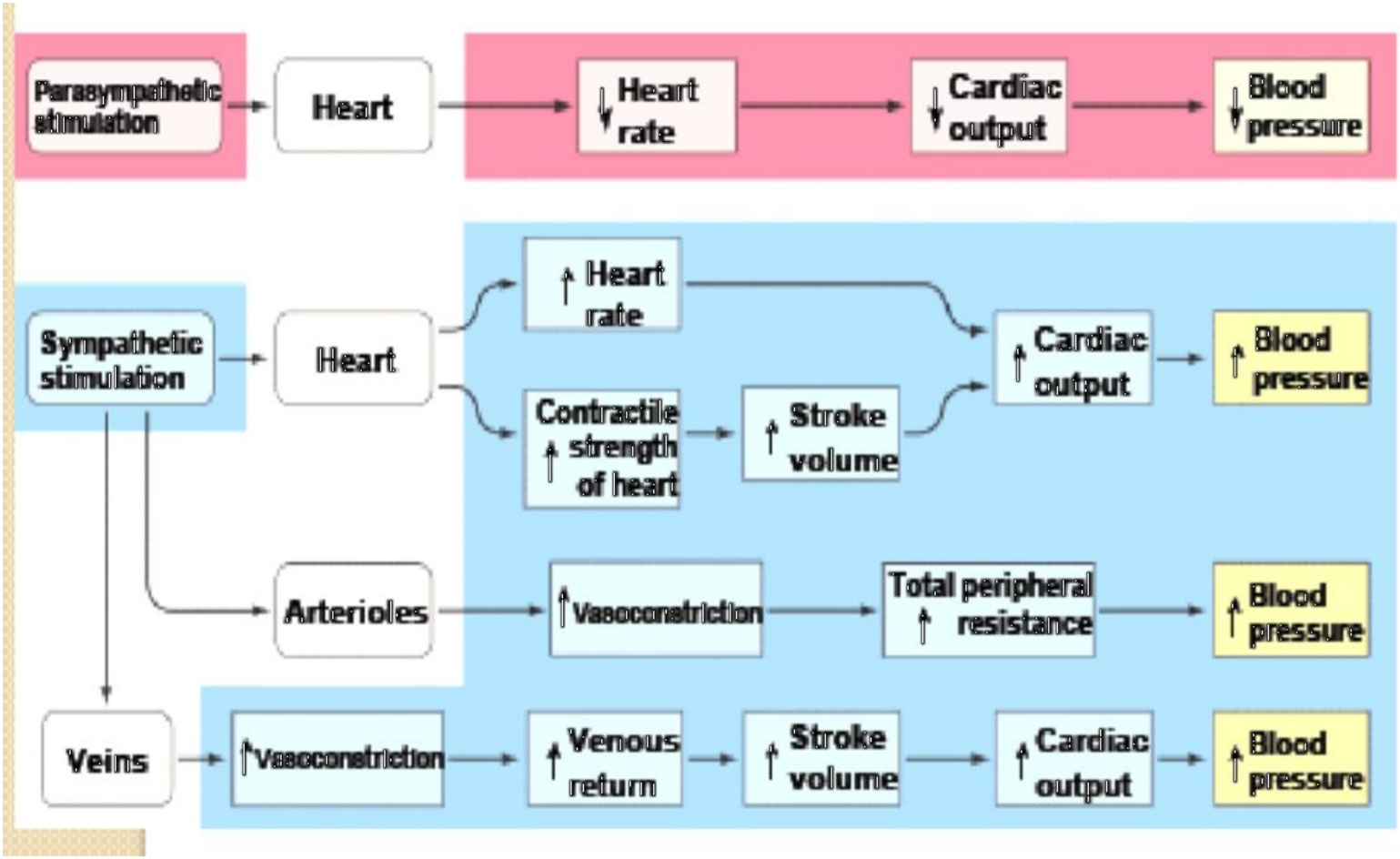


Figure 15-22: The baroreceptor reflex: the response to increased blood pressure

SUMMARY


Parasympathetic stimulation
Heart
↓ Heart rate
↓ Cardiac output
↓ Blood pressure
Sympathetic stimulation
Heart
↑ Heart rate
↑ Cardiac output
↑ Blood pressure
Contractile ↑ strength of heart
↑ Stroke volume
Arterioles
↑ Vasoconstriction
Total peripheral ↑ resistance
↑ Blood pressure
Veins
↑ Vasoconstriction
↑ Venous return
↑ Stroke volume
↑ Cardiac output
↑ Blood pressure

Useful Videos

- Regulation of Arterial Blood Pressure.
- 12 Ways to Lower Blood Pressure Naturally Without Drugs.

THE END

If there are any problems or suggestions
Feel free to contact:

Physiology Team Leaders
Mohammed Jameel & Khulood Al-Raddadi

432100187@student.ksu.edu.sa
432200235@student.ksu.edu.sa

THANK YOU

Actions speak louder than Words