

Very important

Extra information

Physiology

OF THE CARDIOVASCULAR SYSTEM

Regulation of blood pressure

Objectives :

- List short, intermediate & long-term mechanisms regulating ABP.
- Chemoreceptors regulatory mechanism of ABP.
- Baroreceptors regulatory mechanism of ABP.
- Role of the kidney in the long-term regulation of ABP.

Regulation of ABP^{” arterial blood pressure”}

- Maintaining BP is important to ensure a steady blood flow (perfusion) to tissues.
- Inability to regulate blood pressure can contribute to diseases.
- In order to regulate the blood pressure, the determining factors have to be regulated:
 - **Cardiac output.**
 - **Peripheral resistance.**
 - **Blood volume.**

- If the blood pressure drops suddenly, 2 problems confronts the pressure control system :

1- The first if Survival :

To return the arterial pressure immediately to near a normal level that the person can live through the acute episode.

2- The second is to return the blood volume eventually to its normal level :

So that the circulatory system can re-establish full normality including return of the arterial pressure back to its normal value.

EXTRA Regulation of ABP” arterial blood pressure”

- **Guyton corner:**

Several systems contribute to the regulation of arterial blood pressure. One of these, the *baroreceptor system*, is a simple and excellent example of a rapidly acting control mechanism. In the walls of the bifurcation region of the carotid arteries in the neck, and also in the arch of the aorta in the thorax, are many nerve receptors called *baroreceptors* that are stimulated by stretch of the arterial wall. When the arterial pressure rises too high, the baroreceptors send barrages of nerve impulses to the medulla of the brain. Here these impulses inhibit the *vasomotor center*, which in turn decreases the number of impulses transmitted from the vasomotor center through the sympathetic nervous system to the heart and blood vessels. Lack of these impulses causes diminished pumping activity by the heart and also dilation of the peripheral blood vessels, allowing increased blood flow through the vessels. Both of these effects decrease the arterial pressure, moving it back toward normal. Conversely, a decrease in arterial pressure below normal relaxes the stretch receptors, allowing the vasomotor center to become more active than usual, thereby causing vasoconstriction and increased heart pumping. The decrease in arterial pressure also raises arterial pressure, moving it back toward normal.

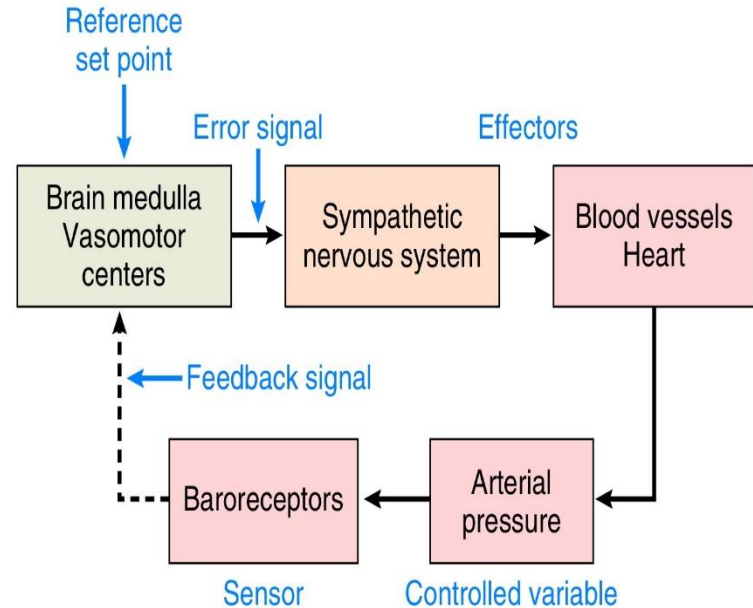
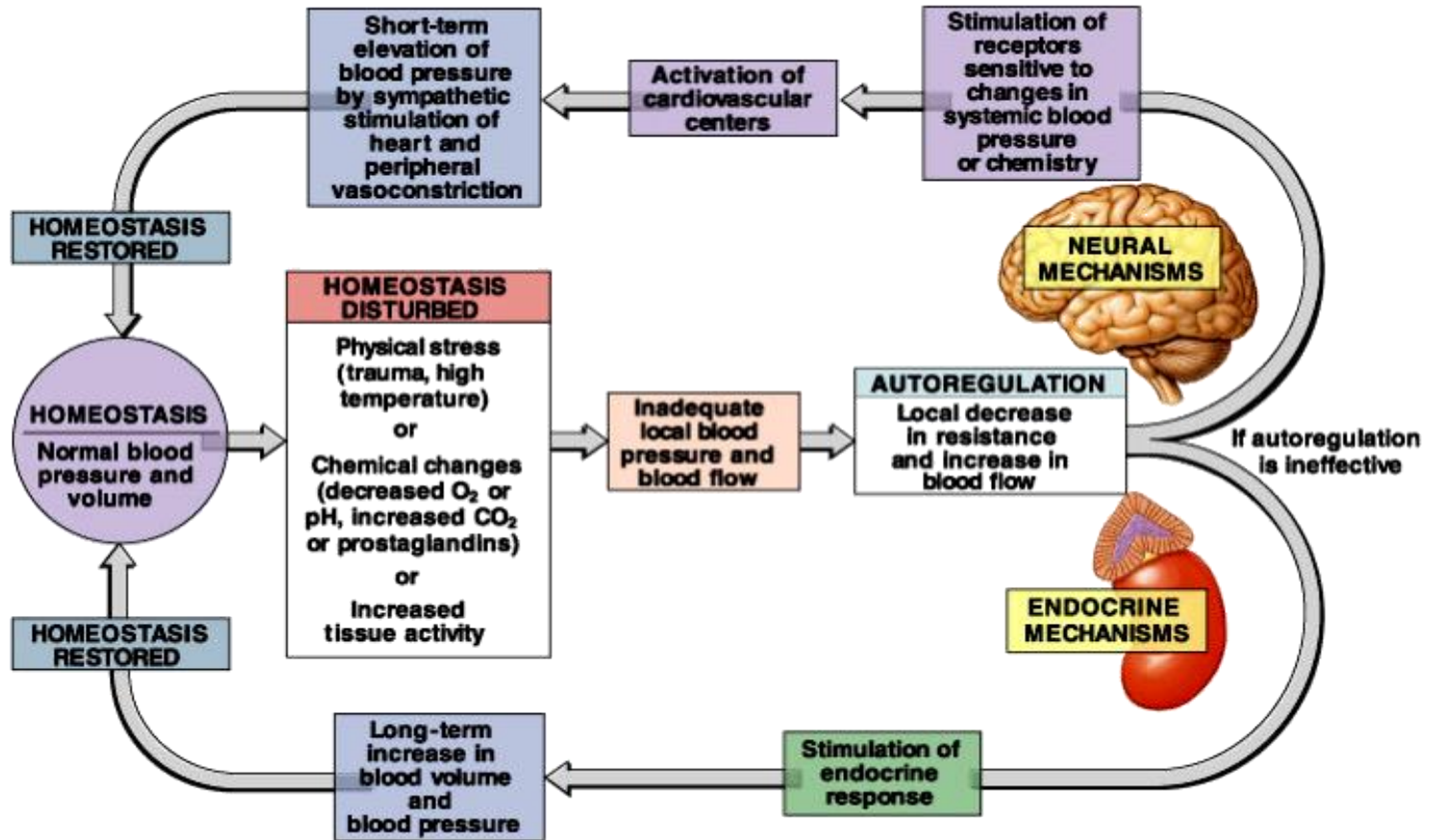


Figure 1-3. Negative feedback control of arterial pressure by the arterial baroreceptors. Signals from the sensor (baroreceptors) are sent to medulla of the brain, where they are compared with a reference set point. When arterial pressure increases above normal, this abnormal pressure increases nerve impulses from the baroreceptors to the medulla of the brain, where the input signals are compared with the set point, generating an error signal that leads to decreased sympathetic nervous system activity. Decreased sympathetic activity causes dilation of blood vessels and reduced pumping activity of the heart, which return arterial pressure toward normal.

Mechanisms Regulating Mean Arterial Pressure



when there's homeostasis disturbed the body will start autoregulation, the body automatically will regulate this disturbance, with one of two ways: "this doesn't mean if one of them work the other should stop"

- Neural mechanisms > very short within seconds-minutes - responsible for regulating cardiac output & peripheral resistance
- Endocrine (hormonal mechanisms) > long period takes hours-days-weeks - responsible for regulating blood volume
- Intermediate regulation > minutes-hours

Neurally- Mediated Regulation of ABP

Rapidly Acting Control Mechanisms:

- Acts within. **seconds / minutes**. [Fast Response (Short - Term)]
- Concerned by **regulating Cardiac output & Peripheral resistance**.
- Reflex mechanisms that act through autonomic nervous system:
Centers in medulla oblongata:
 - Vasomotor Center (**VMC**) ... *Sympathetic* nervous system.
 - Cardiac Inhibitory Center (**CIC**) .. *Parasympathetic* nervous system

Neural rapidly acting. In order to work it needs receptors, these receptors send the signals to medulla oblongata "to the two center VMC & CIC"

Effects of Rapidly Acting Pressure Control Mechanisms :

- To cause constriction of the veins and provide transfer of blood into the heart.
- To cause increased heart rate and contractility of the heart and provide greater pumping capacity by the heart
- To cause constriction of the peripheral arterioles to impede the flow of the blood out of the arteries.
- All these effects occur almost instantly to raise the arterial pressure back into a survival range.

Short Term Reflex mechanisms for Maintaining Normal ABP

Short Term Reflex mechanisms for Maintaining Normal ABP

Baroreceptors reflex.

Chemoreceptors reflex.

Atrial stretch receptor reflex.

Thermo-receptors.

Pulmonary receptors.

- "Baro " means relating to pressure.
- Baroreceptors reflex > the most important & very powerful "this one moment to moment" (على طول شغال)
إذا ارتفع ضغط الدم ينزله وإذا نازل يرفعه
- Chemoreceptors reflex > work with baroreceptors only in the situation of low blood pressure "it doesn't work if the blood pressure is high" "in case of emergency, such as hemorrhage , ischemia"

Baroreceptor Reflex

Mechano-stretch receptors located in the wall of carotid sinus & aortic arch

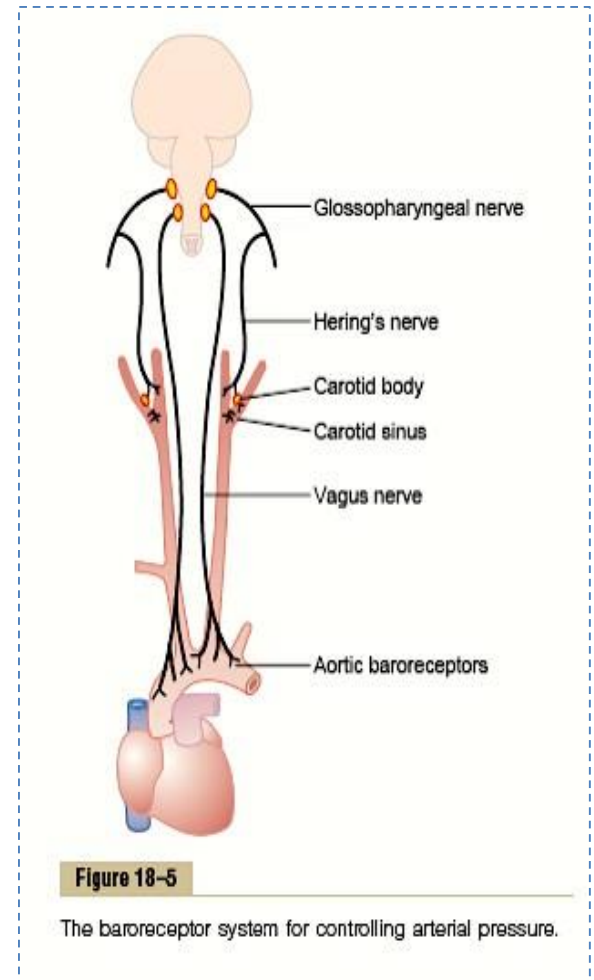
Fast, neurally mediated

Baroreceptor Reflex

Provide powerful moment-to-moment control of arterial pressure

Stimulated in response to BP changes

- Location of baroreceptors: They're found in aortic arch & carotid bodies
- "they're mechano-stretch receptors which means they will be stimulated according to the pressure changes which mainly act on the vessel wall.



Baroreceptor Reflex

➤ Importance of the baroreceptor reflex :

- To keep the arterial pressure relatively constant in the range of (70 mmHg - 150 mmHg), maintain the **mean** blood pressure at about 100 mmHg
- Pressure buffer system – reduce the blood fluctuation during the daily events, such as changing of the posture, respiration, excitement.

➤ Baroreceptor Resetting :

- Baroreceptor will adapt to the long term change of blood pressure.
- If the blood pressure is elevated for a long period of time, several days or years, the set point will transfer to the elevated mean blood pressure.
- That makes the baroreceptor system unimportant for long-term regulation of arterial pressure.

EXTRA-Baroreceptor Reflex

- Guyton corner:**

By far the best known of the nervous mechanisms for arterial pressure control is the *baroreceptor reflex*. Basically, this reflex is initiated by stretch receptors, called either *baroreceptors* or *pressoreceptors*, located at specific points in the walls of several large systemic arteries. A rise in arterial pressure stretches the baroreceptors and causes them to transmit signals into the CNS. “Feedback” signals are then sent back through the autonomic nervous system to the circulation to reduce arterial pressure downward toward the normal level.

Baroreceptors are spray-type nerve endings that lie in the walls of the arteries and are stimulated when stretched. A few baroreceptors are located in the wall of almost every large artery of the thoracic and neck regions, but as shown in **Figure 18-5**, baroreceptors are extremely abundant in (1) the wall of each internal carotid artery slightly above the carotid bifurcation, an area known as the *carotid sinus*, and (2) the wall of the aortic arch. Page 219

Figure 18-5 shows that signals from the “carotid baroreceptors” are transmitted through small *Hering’s nerves* to the *glossopharyngeal nerves* in the high neck, and then to the *nucleus tractus solitarius* in the medullary area of the brain stem. Signals from the “aortic baroreceptors” in the arch of the aorta are transmitted through the *vagus nerves* to the same nucleus tractus solitarius of the medulla. Page 219

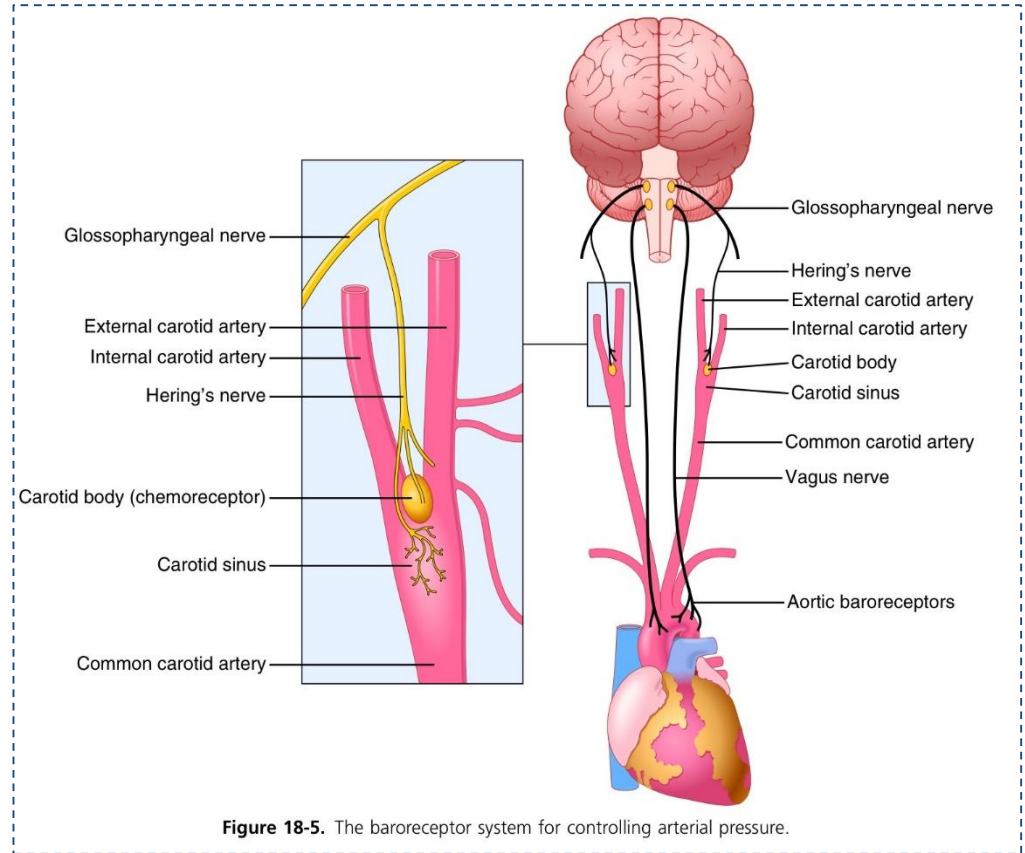
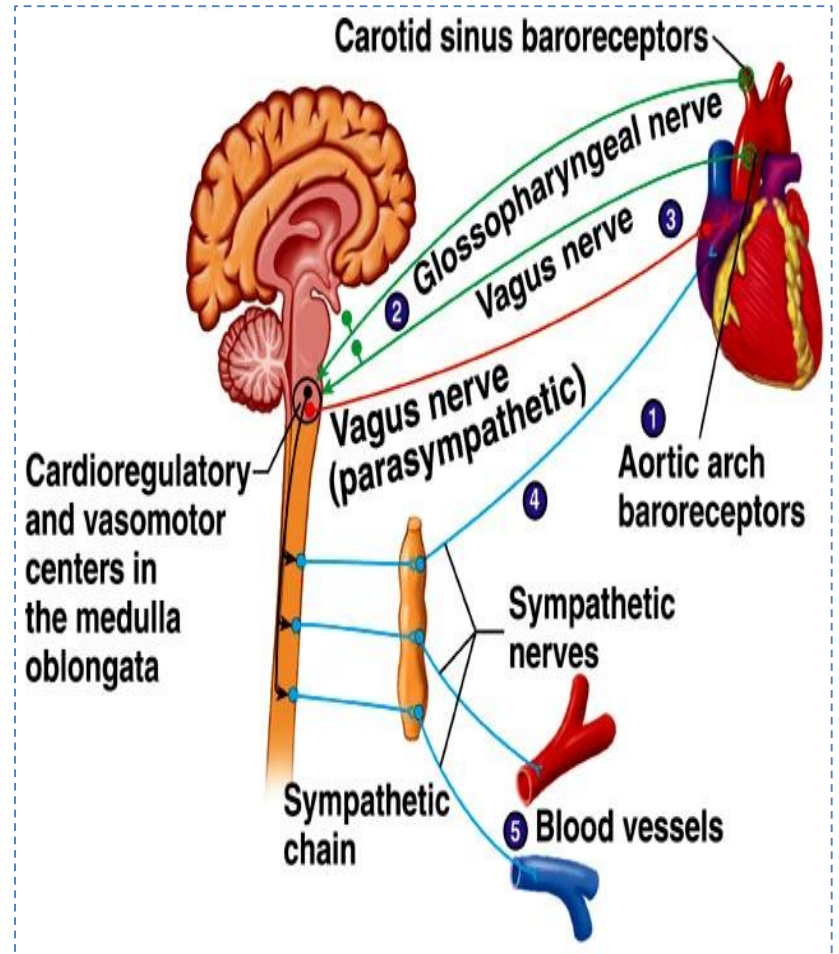
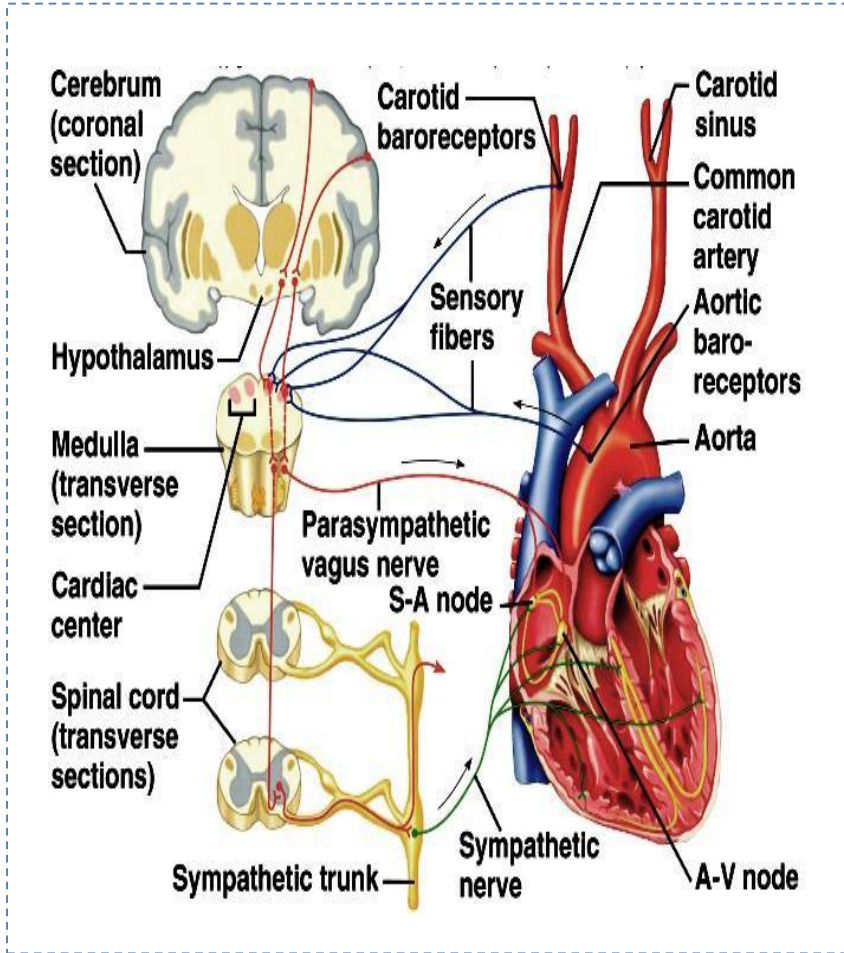


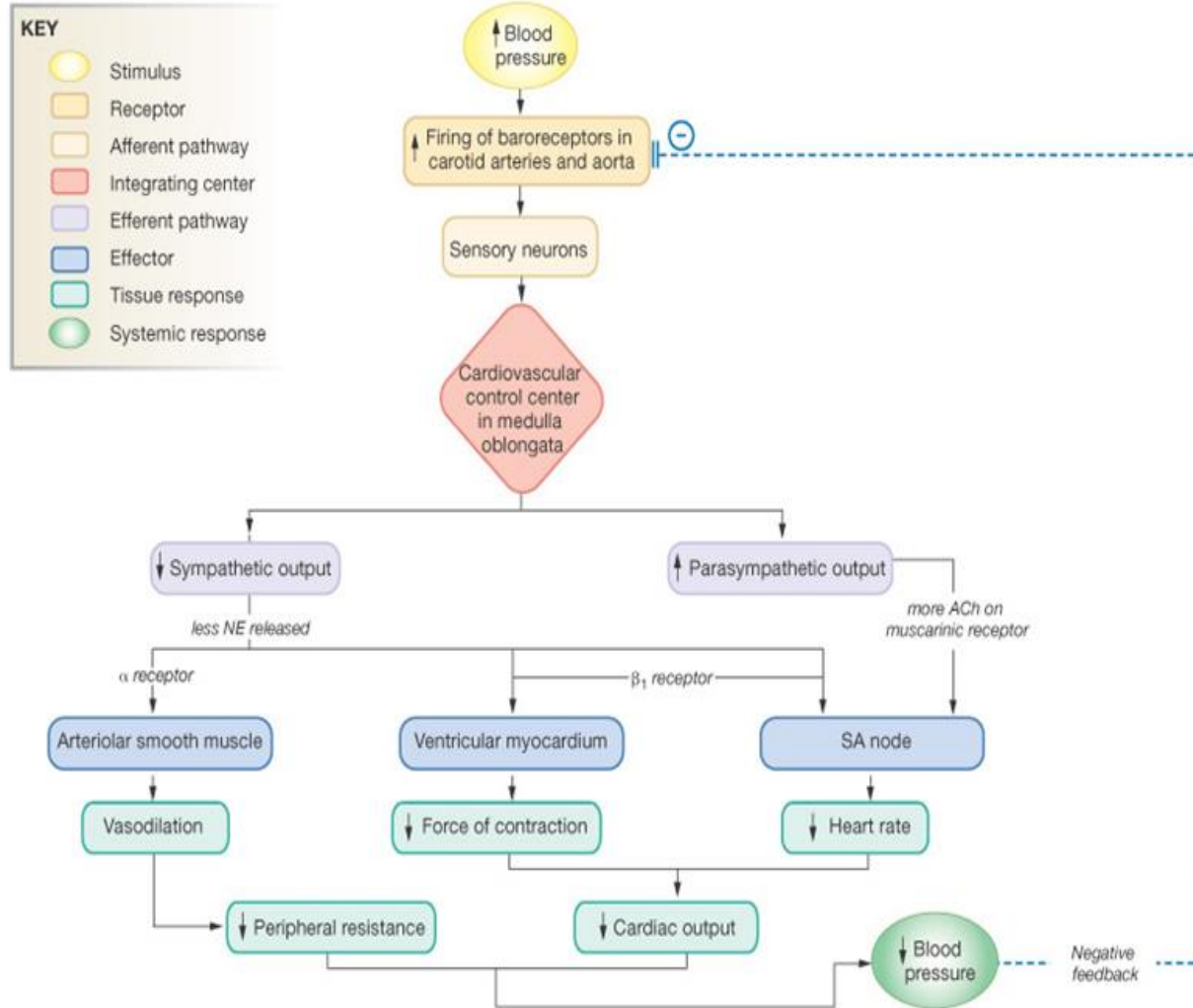
Figure 18-5. The baroreceptor system for controlling arterial pressure.

Baroreceptor Reflex



Stretch receptors in large systemic arteries (particularly the carotid artery and aorta).

Baroreceptor Reflex



- Sudden increase in BP :**
 ↑ activity of the baroreceptors which produces:
- 1) ↓ H.R. & ↓ C.O. due to ↓ sympathetic & ↑ parasympathetic activity.
 - 2) Widespread peripheral vasodilatation due to inhibition of the vasomotor center.

Baroreceptor Reflex

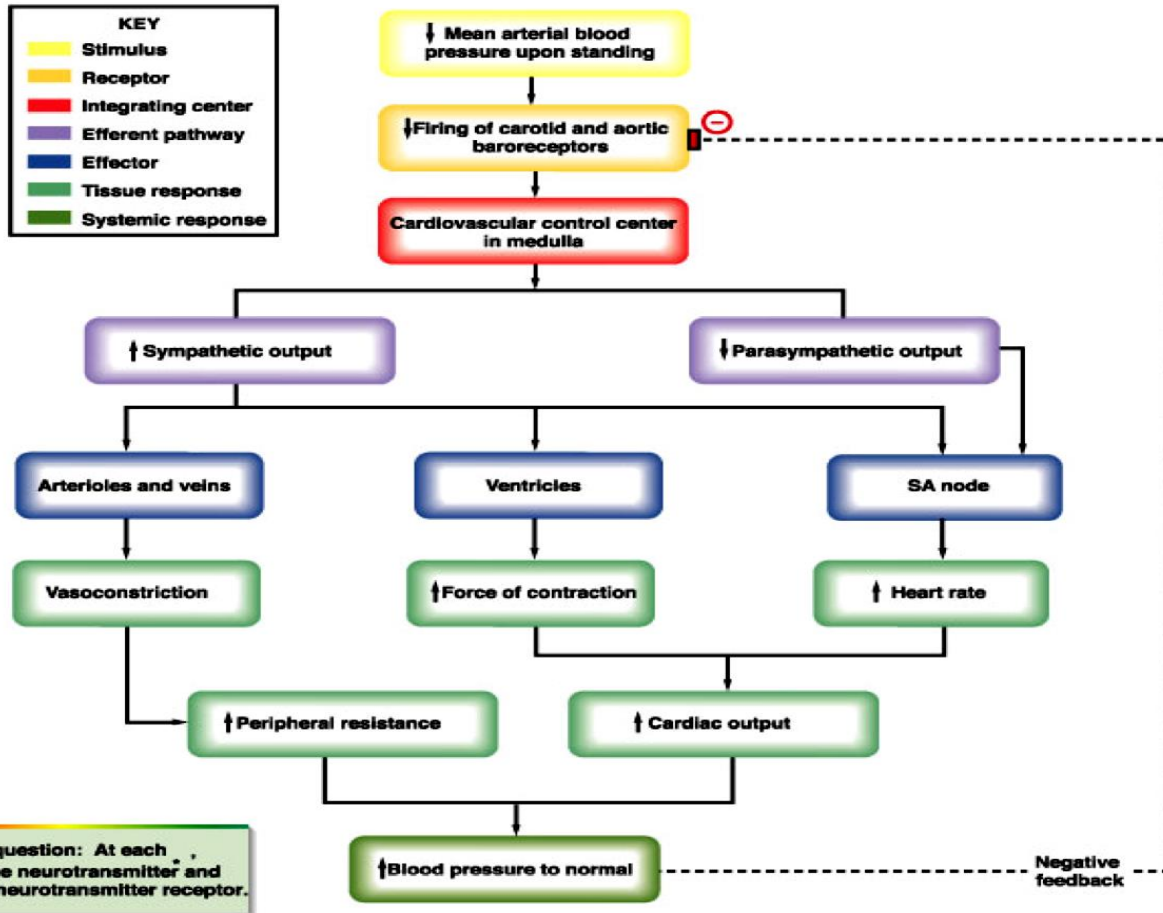


Figure question: At each , name the neurotransmitter and type of neurotransmitter receptor.

Sudden decrease in BP :

↓ activity of the baroreceptors which produces:

1) ↑ H.R. & ↑ C.O. due to ↑ sympathetic & ↓ parasympathetic activity.

2) Widespread peripheral vasoconstriction due to stimulation of the vasomotor center.

Baroreceptor Reflex Mechanism During Changes in Body Posture

- Immediately on standing, Arterial pressure in the head & upper part of the body tends to fall ,cause loss of consciousness.
- Falling pressure at the **baroreceptors** elicits an immediate reflex, resulting in **strong sympathetic discharge** throughout the body.
- This **minimizes** the decrease in pressure in the head & upper body

- **Denervation** of the baroreceptors can lead to *paroxysmal hypertension*.
“ is episodic **high blood pressure**”

Chemoreceptor Reflex

- Closely associated with the baroreceptor pressure control system.
- Monitor changes of oxygen, carbon dioxide, and hydrogen ions.
- **Chemoreceptor reflex** operates in much same way as the baroreceptor reflex, EXCEPT that chemoreceptors are *chemo-sensitive* cells instead of stretch receptors.

- **Guyton corner:**

The chemoreceptors are chemo sensitive cells sensitive to oxygen lack, carbon dioxide excess, and hydrogen ion excess. They are located in several small chemoreceptor organs about 2 millimeters in size (two carotid bodies, one of which lies in the bifurcation of each common carotid artery, and usually one to three aortic bodies adjacent to the aorta). The chemoreceptors excite nerve fibers that, along with the baroreceptor fibers, pass through Herring's nerves and the vagus nerves into the vasomotor center of the brain stem.

Chemoreceptor Reflexes: Two Types :

1-Peripheral chemoreceptors:

- Sensory receptors located in **carotid & aortic bodies**
- Sensitive to
 - 1- O_2 lack (\downarrow) (*hypoxia*)
 - 2- CO_2 (\uparrow or \downarrow)
 - 3- pH (\downarrow or \uparrow)
(*alkalosis or acidosis*)
- Chemoreceptors' stimulation excite nerve fibers that, along with baroreceptor fibers

2-Central Chemoreceptors:

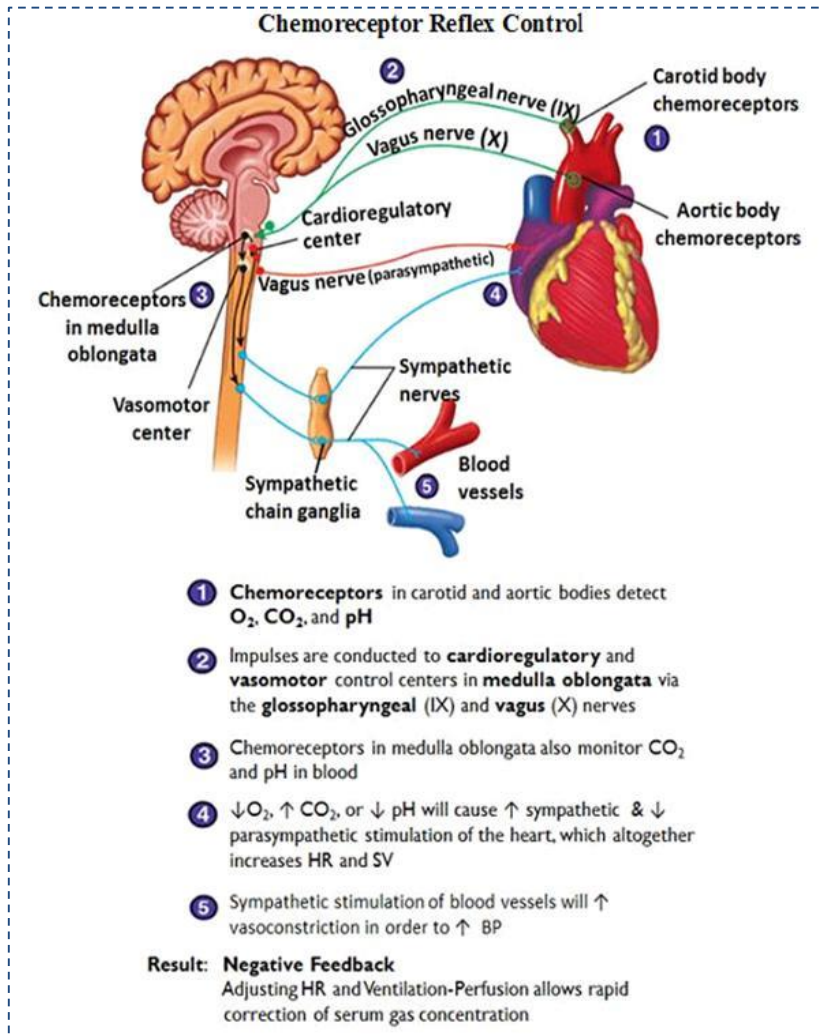
- Sensory receptor located in the **medulla itself**.
- Very sensitive to CO_2 excess (\uparrow) & pH (\downarrow) in medulla
(*It works in severe emergency "severe hypoxia". It is not sensitive to O_2 because it can not cross blood brain barrier, but it works due to excess amount of CO_2*)

The signals transmitted from the chemoreceptors **excite the vasomotor center**, and this **elevates** the arterial pressure back toward normal. However, this chemoreceptor reflex is not a powerful arterial pressure controller until the arterial pressure falls below 80 mm Hg. Therefore, it is at the lower pressures that this reflex becomes important to help prevent further decreases in arterial pressure

• **Guyton corner:**

Each carotid or aortic body is supplied with an abundant blood flow through a small nutrient artery, so the chemoreceptors are always in close contact with arterial blood. Whenever the arterial pressure falls below a critical level, the chemoreceptors become stimulated because diminished blood flow causes decreased oxygen, as well as excess buildup of carbon dioxide and hydrogen ions that are not removed by the slowly flowing blood.

Peripheral Chemoreceptor Reflex



- Stimulation of chemoreceptors leads to a reflex **increase** in vasomotor tone.
- which causes **generalized vasoconstriction** and hence a rise in blood pressure.
- Chemoreceptor mechanism is important in regulation of blood pressure when it *fall* below the range in which baroreceptors act (70 mmHg).
- Chemoreceptor reflex is useful in regulation of blood pressure when it falls to a level between **(40 and 70 mmHg)**.
- But if the blood pressure **below 40 mmHg**, the last ray of hope for survival is the central nervous system (CNS) ischemia response.
- So it sometimes called the “last ditch stand” pressure control mechanism.

Peripheral Chemoreceptor Reflex

Chemoreceptor reflexes usually act to increase BP

Blood pressure ↓

Hemorrhage

Hypoxia

+Chemoreceptors

vasomotor center
++

- C.I.C

+ Sympathetic

- parasympathetic

+ Adrenal medulla

↑ CO

Vasoconstriction
& ↑ TPR

↑ HR

CNS Ischemic Response

“Last ditch stand” pressure control mechanism

It is **not** one of the normal mechanisms for regulating ABP.

It operates principally as an emergency pressure control system to prevent further decrease in arterial pressure.

It acts rapidly & very powerfully whenever blood flow to the brain ↓ dangerously close to the lethal level.

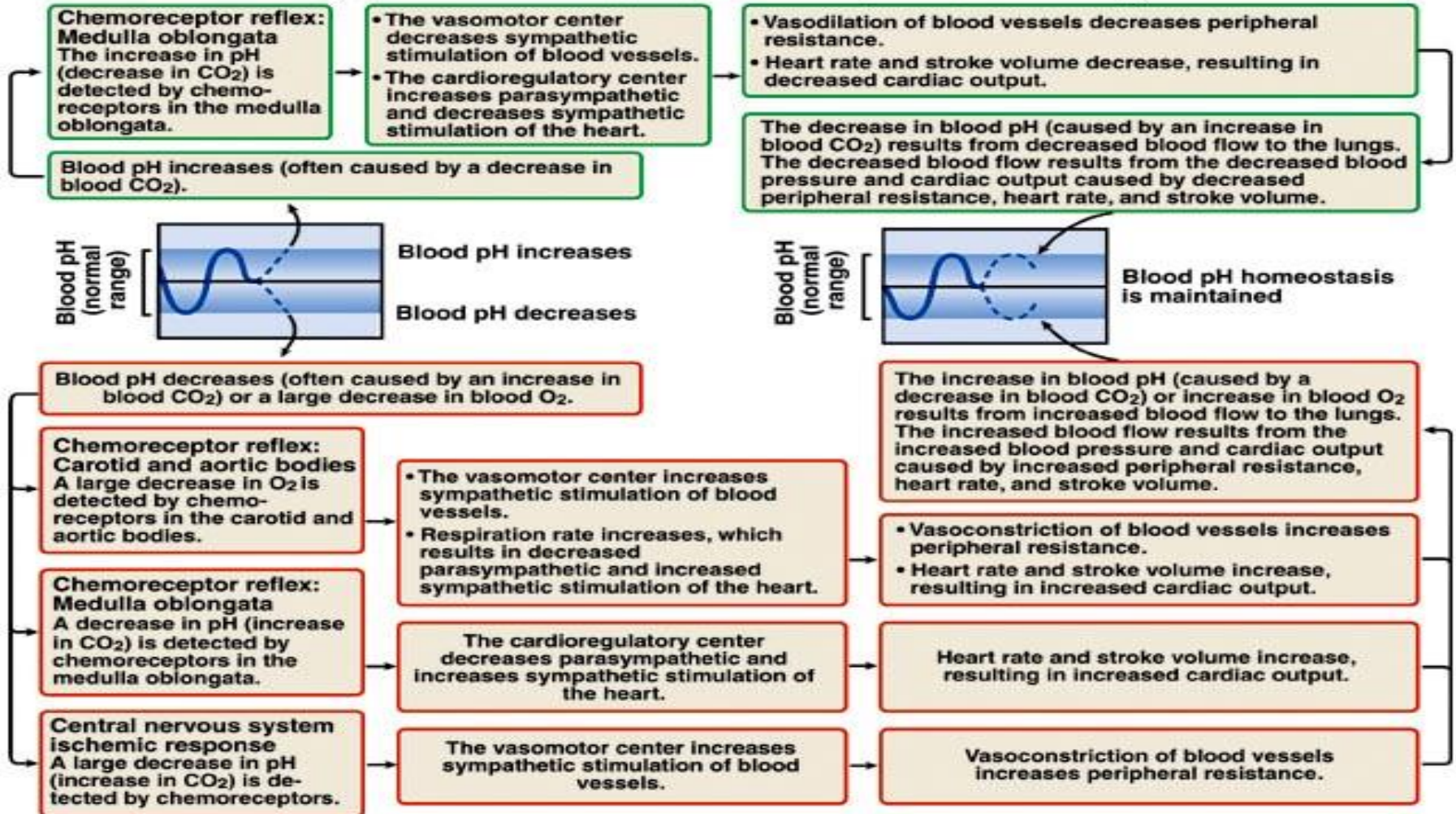
Local concentration of CO_2 ↑ greatly.

This has an extremely potent effect in stimulating the sympathetic vasomotor nervous control areas in the brain's medulla.

- **Guyton corner : P 209 12TH EDITION** when blood flow to the vasomotor center in the lower brain stem becomes decreased severely enough to cause nutritional deficiency—that is, to cause cerebral ischemia—the vasoconstrictor and cardioaccelerator neurons in the vasomotor center respond directly to the ischemia and become strongly excited. When this occurs, the systemic arterial pressure often rises to a level as high as the heart can possibly pump. This effect is believed to be caused by failure of the slowly flowing blood to carry carbon dioxide away from the brain stem vasomotor center, At low levels of blood flow to the vasomotor center, the local concentration of carbon dioxide increases greatly and has an extremely potent effect in stimulating the sympathetic vasomotor nervous control areas in the brain's medulla. This arterial pressure elevation in response to cerebral ischemia is known as the central nervous system (CNS) ischemic response. Despite the powerful nature of the CNS ischemic response, it does not become significant until the arterial pressure falls far below normal, down to 60 mm Hg and below, reaching its greatest degree of stimulation at a pressure of 15 to 20 mm Hg. Therefore, it is not one of the normal mechanisms for regulating arterial pressure. Instead, it operates principally as an emergency pressure control system that acts rapidly and very powerfully to prevent further decrease in arterial pressure whenever blood flow to the brain decreases dangerously close to the lethal level.

Effects of pH and Gases on Chemoreceptors' Stimulation

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Other Vasomotor Reflexes

Atrial stretch receptor reflex

↑ Venous Return ⇒ ++ atrial stretch receptors ⇒ reflex vasodilatation & ↓ ABP.

it works during swimming

الأطفال بعد السباحة يريدون الذهاب للمرحاض لأن الـvenous return يزداد تحت الماء.

Thermo-receptors (in skin/ hypothalamus)

Exposure to heat ⇒ vasodilatation.

Exposure to cold ⇒ vasoconstriction.

Pulmonary receptors

Lung inflation
↓
vasoconstriction.

first thing to do after being dizzy is to lie down & take deep inspiration

Hormonally- Mediated Regulation of ABP

➤ Long – term regulation of ABP :

- Hormonally mediated.
- Takes few hours to begin showing significant response. [slow response (Long-term).
- Concerned in regulating **blood volume**.

1- Renin-Angiotensin-
Aldosterone System.

2- Vasopressin [Anti-diuretic
hormone (ADH)] Mechanism

3- Atrial Natriuretic Peptide
Mechanism (Low-pressure
volume receptors.)

4- EPO (erythropoietin.)

Mainly renal: acts if **BP** is too **low**

The point at we reach aldosterone (sodium and water retention) is considered as **long-term** regulation.
The steps before we reach aldosterone (angiotensin) are considered as **intermediate** regulation.

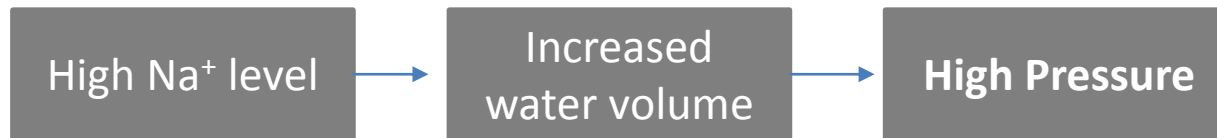
- **Guyton corner :**

experimental studies have shown that an increase in salt intake is far more likely to elevate the arterial pressure than is an increase in water intake. The reason for this is that pure water is normally excreted by the kidneys almost as rapidly as it is ingested, but salt is not excreted so easily. As salt accumulates in the body, it also indirectly increases the extracellular fluid volume for two basic reasons:

1- When there is excess salt in the extracellular fluid, the osmolality of the fluid increases, and this in turn stimulates the thirst center in the brain, making the person drink extra amounts of water to return the extracellular salt concentration to normal. This increases the extracellular fluid volume.

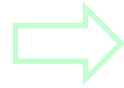
2- The increase in osmolality caused by the excess salt in the extracellular fluid also stimulates the hypothalamic-posterior pituitary gland secretory mechanism to secrete increased quantities of *antidiuretic hormone*. The antidiuretic hormone then causes the kidneys to reabsorb greatly increased quantities of water from the renal tubular fluid, thereby diminishing the excreted volume of urine but increasing the extracellular fluid volume.

Thus, for these important reasons, the amount of salt that accumulates in the body is the main determinant of the extracellular fluid volume. Because only small increases in extracellular fluid and blood volume can often increase the arterial pressure greatly if the vascular capacity is not simultaneously increased, accumulation of even a small amount of extra salt in the body can lead to considerable elevation of arterial pressure.



Anti-diuretic hormone (ADH), or vasopressin

Hypovolemia
(decreased blood
fluid volume) &
dehydration



Stimulation of
“Hypothalamic
Osmoreceptors” +
Thirst centers.



Release of ADH
from posterior
pituitary gland

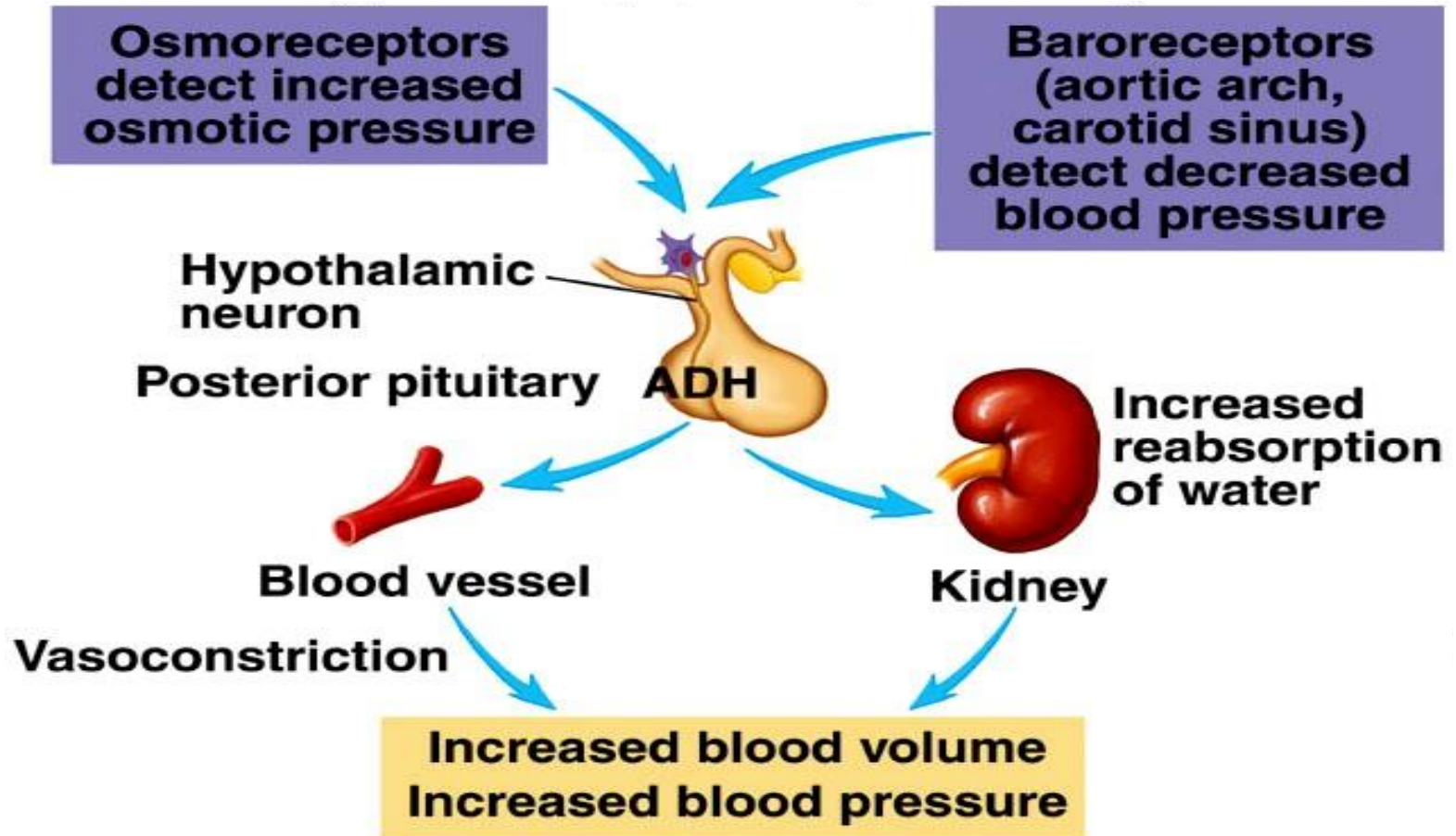


Finally, ADH will:

- 1- Promote water reabsorption at kidney tubules > **increasing blood volume.**
- 2- Cause vasoconstriction > **increasing arterial blood pressure (ABP)**

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

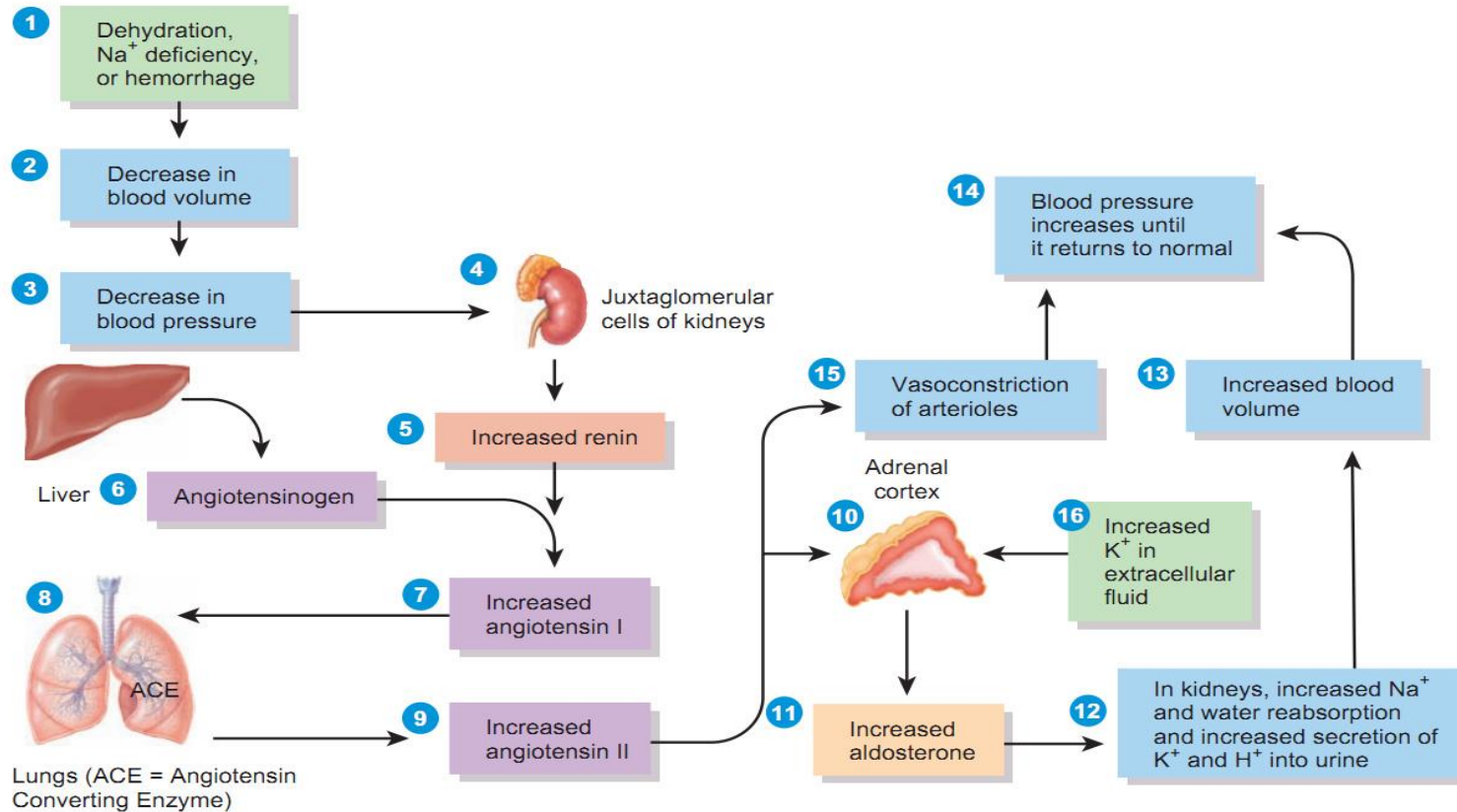
Anti-diuretic hormone (ADH), or vasopressin



Renin-Angiotensin Aldosterone system (RAA)

Regulation of aldosterone secretion by the renin–angiotensin–aldosterone (RAA) pathway.

Aldosterone helps regulate blood volume, blood pressure, and levels of Na⁺, K⁺, and H⁺ in the blood.



Angiotensinogen (Plasma protein)
 Angiotensin II (powerful vasoconstrictor)
 Angiotensin III (powerful vasoconstrictor)

- **Guyton corner :**

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

1. Angiotensin II acts directly on the kidneys to cause salt and water retention.
2. Angiotensin II causes the adrenal glands to secrete aldosterone, and the aldosterone in turn increases salt and water reabsorption by the kidney tubules.

- **Guyton corner :**

- 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*. 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 itself is an enzyme, not a vasoactive substance.

➤ Atrial Natriuretic Peptide (ANP) hormone:

- A hormone that is released from cardiac myocytes of **right atrium** in response to the increase in ABP.

- **Function :**

Stimulates an **increase** in urinary production.

Thus, causes a **decrease** in blood volume and blood pressure.

Respond to stretch of the wall of the right atrium :

- \uparrow atrial pressure \rightarrow stimulate CV center \rightarrow \uparrow H.R. & \uparrow C.O (Bainbridge reflex) \rightarrow prevent damming of blood in veins, atria & pulmonary Circulation.
- Atrial stretch also \rightarrow dilate afferent arterioles \rightarrow \uparrow Glomerular filtration rate (GFR) \rightarrow \downarrow ADH & \uparrow ANP hormone secretion \rightarrow \uparrow urine output. \rightarrow \downarrow B.P.

- **Guyton corner :**

Specific cells of the cardiac atria, when distended because of plasma volume expansion, secrete a peptide called *atrial natriuretic peptide (ANP)*. Increased levels of this peptide in turn directly inhibit the reabsorption of sodium and water by the renal tubules, especially in the collecting ducts. ANP also inhibits renin secretion and therefore angiotensin II formation, which in turn reduces renal tubular reabsorption. This decreased sodium and water reabsorption increases urinary excretion, which helps to return blood volume back toward normal. ANP levels are greatly elevated in congestive heart failure when the cardiac atria are stretched because of impaired pumping of the ventricles. The increased ANP helps to attenuate sodium and water retention in heart failure.

Erythropoietin

- It is also known as *hematopoietin*, or *hemopoietinis*.
 - It's a glycoprotein hormone that controls erythropoiesis.
 - Secreted by the kidneys when blood volume is too low.
 - It increases blood volume since it increases RBCs formation

Intermediate Mechanisms Regulating Arterial Blood Pressure

➤ Intermediate Mechanisms Regulating Arterial Blood Pressure:

1. Renin-angiotensin vasoconstrictor mechanism.
2. Stress-relaxation of the vasculature.
3. Fluid Shift mechanism.

*During this time, the nervous mechanisms usually become less and less effective.
The mechanisms are activated within 30 minute to several hours.*

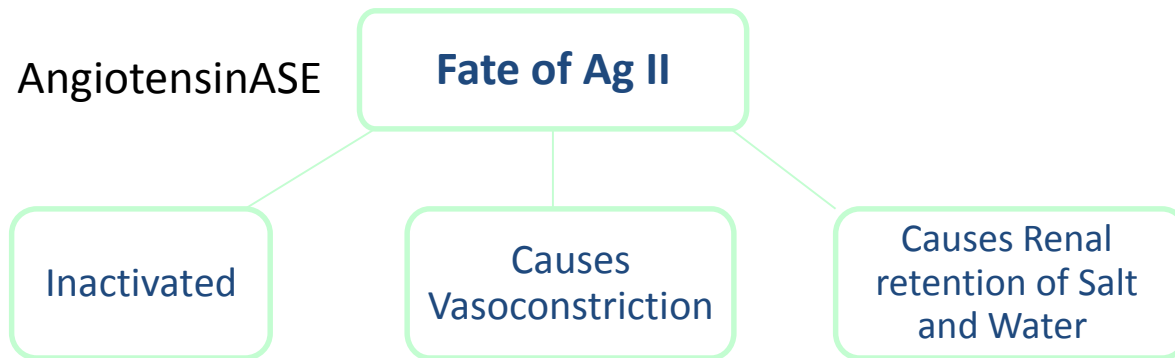
intermediate mechanisms:

- Renal angiotensin vasoconstriction mechanism occurs before the beginning of aldosterone effect (The steps before we reach aldosterone (angiotensin) are considered as **intermediate** regulation. while aldosterone (long-term))
- Fluid shift mechanism > in both situation high or low blood pressure, if it's high the capillaries release the fluid to the interstitial space to decrease the pressure in the vessels "which will cause edema", if it's low the opposite will occur "from the interstitial space to the capillaries"

1. The Renin-Angiotensin System

➤ In Short:

- If the arterial pressure is decreased, in response kidneys release **Renin**.
- **Renin** cleaves Angiotensinogen (In the liver) to produce Angiotensin I (AgI).
- Ag I is subsequently converted to **angiotensin II** by the enzyme angiotensin-converting enzyme (ACE) found in the lungs.

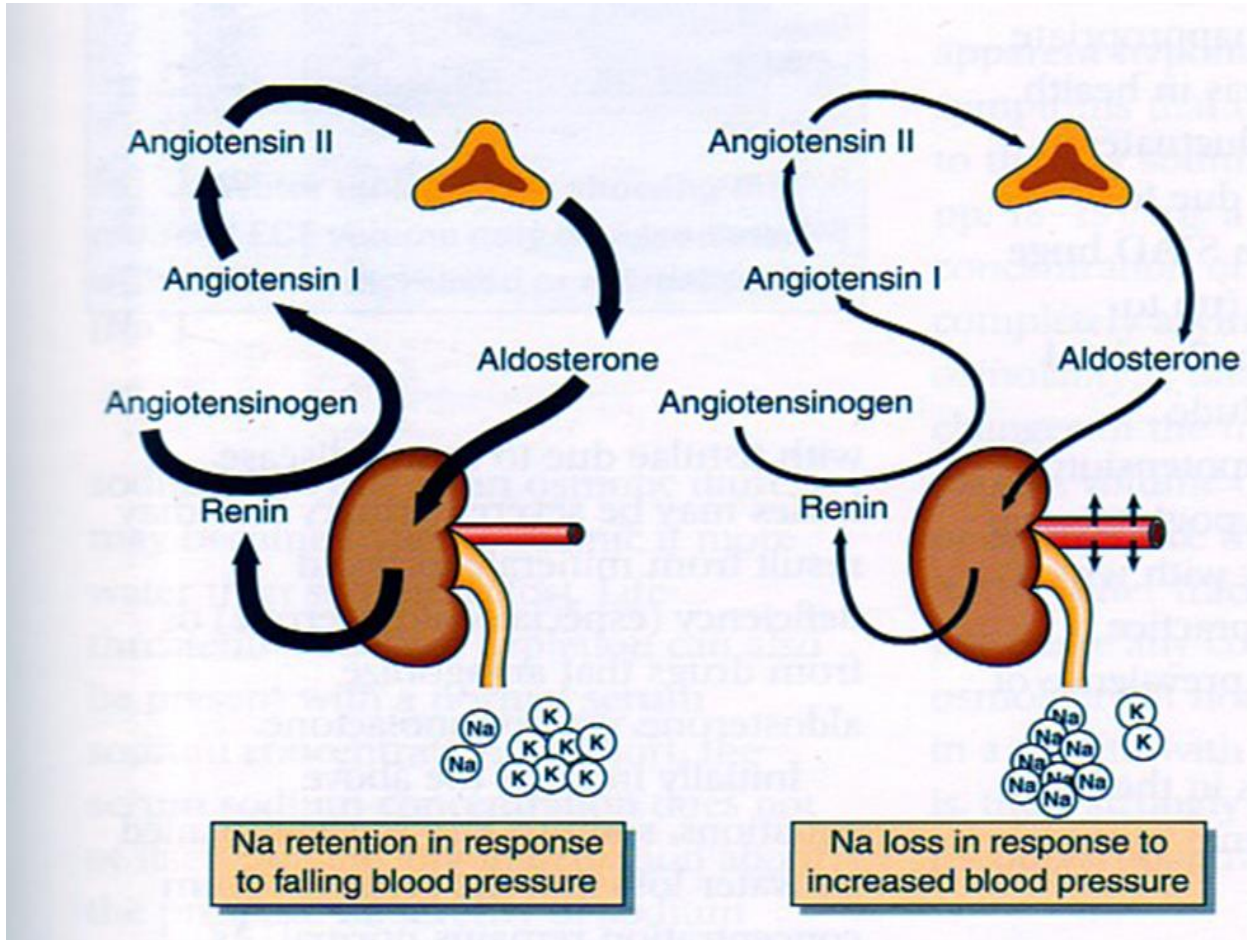


- As a result arterial blood pressure will increase.

Angiotensin II stimulates the secretion of the hormone aldosterone from the adrenal cortex. Aldosterone causes the tubules of the kidneys to increase the reabsorption of sodium and water into the blood.

Stay tuned *Renal block*

1. The Renin-Angiotensin System



2. Fluid Shift Mechanism

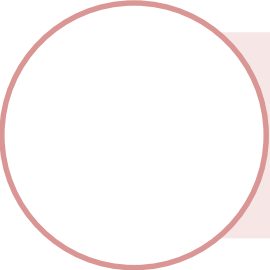
- In response to **decreased blood pressure**, fluids move from interstitial spaces into **Capillaries**.

WHY? In order to maintain Blood Volume.

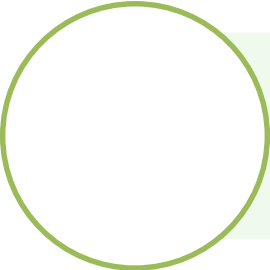
- Conversely, when **capillary pressure** ↑ **too high**, fluid is lost out of circulation into the tissues, reducing blood volume as well as all pressures throughout circulation.

No memorization needed, Just Common sense 😊

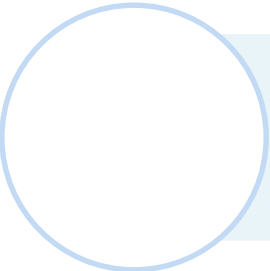
3. Stress-Relaxation Mechanism



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



When pressure in blood vessels becomes **too high** They become stretched (*vasodilatation*) & keep on stretching more & more for minutes or hours Resulting in fall of pressure in the vessels toward normal.



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

Summary

- **In brief :**

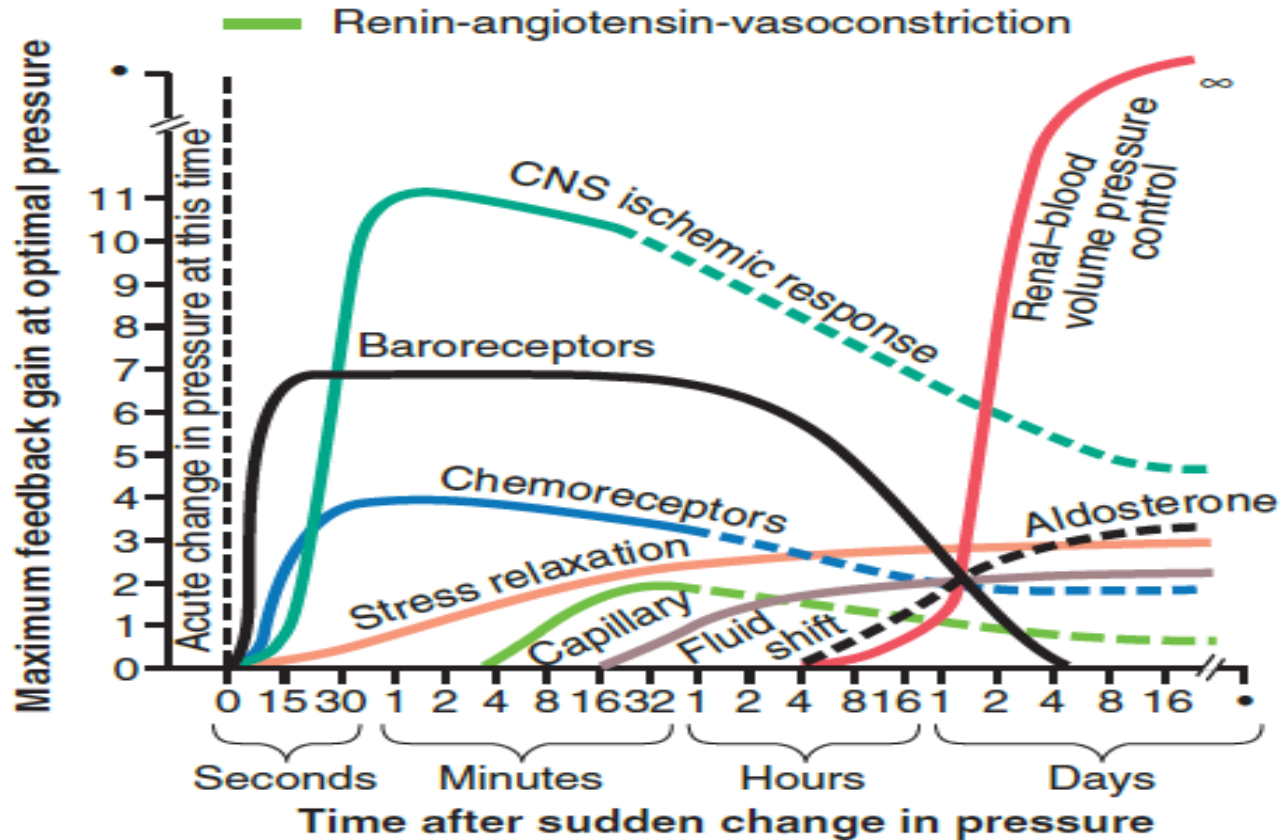
High BP Leads to:

- 1- ↓ Antidiuretic hormone (ADH) secretion.
- 2- ↓ Angiotensin II hormone secretion
- 3- ↓ Aldosterone hormone secretion
- 4- ↓ Erythropoietin hormone secretion.
- 5- ↑ Atrial natriuretic peptides (ANP) hormone secretion.

Low BP Leads to:

- 1- ↑ Antidiuretic hormone (ADH) secretion.
- 2- ↑ Aldosterone hormone secretion
- 3- ↑ Angiotensin II hormone secretion .
- 4- ↓ Natriuretic peptides (ANP) hormone secretion
- 5- ↑ Erythropoietin hormone secretion → ↑ RBCS, take few days.

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



* Extra explanation next slides

Rapidly Acting

- **Guyton corner :**

shows the approximate immediate (seconds and minutes) and long-term (hours and days) control responses, expressed as feedback gain, of eight arterial pressure control mechanisms. These mechanisms can be divided into three groups:

1. Those that react rapidly, within seconds or minutes;
2. Those that respond over an intermediate time period, that is minutes or hours
3. Those that provide long-term arterial pressure regulation for days, months, and years.

Guyton corner : That Act Within Seconds or Minutes. The rapidly acting pressure control mechanisms are almost entirely acute nervous reflexes or other nervous responses. the three mechanisms that show responses within seconds.

1. The baroreceptor feedback mechanism,
2. The central nervous system ischemic mechanism, and
3. The chemoreceptor mechanism.

Not only do these mechanisms begin to react within seconds, but they are also powerful. After any acute fall in pressure, as might be caused by severe hemorrhage, the nervous mechanisms combine to cause

- I. Constriction of the veins and transfer of blood into the heart,
- II. Increased heart rate and contractility of the heart to provide greater pumping capacity by the heart, and
- III. Constriction of most peripheral arterioles to impede flow of blood out of the arteries.

All these effects occur almost instantly to raise the arterial pressure back into a survival range.

When the pressure suddenly rises too high, as might occur in response to rapid transfusion of excess blood, the same control mechanisms operate in the reverse direction, again returning the pressure back toward normal.

Intermediate Term

Guyton corner :Pressure Control Mechanisms

That Act After Many Minutes. Several pressure control mechanisms exhibit significant responses only after a few minutes following acute arterial pressure change. Three of these mechanisms are

- (1) The renin-angiotensin vasoconstrictor mechanism,
- (2) Stress-relaxation of the vasculature, and
- (3) Shift of fluid through the tissue capillary walls in and out of the circulation to readjust the blood volume as needed.

We have already described at length the role of the renin-angiotensin vasoconstrictor system to provide a semi-acute means for increasing the arterial pressure when this is necessary. The stress-relaxation mechanism is demonstrated by the following example: When the pressure in the blood vessels becomes too high→ they become stretched and keep on stretching more and more for minutes or hours→ as a result, the pressure in the vessels falls toward normal. Can serve as an intermediate-term pressure “buffer.” (Function) The capillary fluid shift mechanism means simply that any time capillary pressure falls too low, fluid is absorbed from the tissues through the capillary membranes and into the circulation, thus building up the blood volume and increasing the pressure in the circulation. Conversely, when the capillary pressure rises too high, fluid is lost out of the circulation into the tissues, thus reducing the blood volume, as well as virtually all the pressures throughout the circulation. These three intermediate mechanisms become mostly activated within 30 minutes to several hours. During this time, the nervous mechanisms usually become less and less effective, which explains the importance of these non-nervous, intermediate time pressure control measures.

Long Term

Guyton corner :To the far right in Figure 19-17 is shown

the renal–blood volume pressure control mechanism (which is the same as the renal–body fluid pressure control mechanism), demonstrating that it takes a few hours to begin showing significant response. Yet, it eventually develops a feedback gain for control of arterial pressure nearly equal to infinity. This means that this mechanism can eventually return the arterial pressure nearly all the way back, not merely partway back, to the pressure level that provides normal output of salt and water by the kidneys. Many factors can affect the pressure-regulating level of the renal–body fluid mechanism. One of these, shown in Figure 19-17, is aldosterone. A decrease in arterial pressure leads within minutes to an increase in aldosterone secretion, and over the next hour or days, this effect plays an important role in modifying the pressure control characteristics of the renal–body fluid mechanism. Especially important is interaction of the renin-angiotensin system with the aldosterone and renal fluid mechanisms. For instance, a person’s salt intake varies tremendously from one day to another. We have seen in this chapter that salt intake can decrease to as little as one-tenth normal or can increase to 10 to 15 times normal and yet the regulated level of the mean arterial pressure will change only a few mm Hg if the renin-angiotensin-aldosterone system is fully operative. However, without a functional renin-angiotensin-aldosterone system, blood pressure becomes very sensitive to changes in salt intake.

Thus, arterial pressure control begins with the lifesaving measures of the nervous pressure controls, then continues with the sustaining characteristics of the intermediate pressure controls, and, finally, is stabilized at the long-term pressure level by the renal–body fluid mechanism

Physiology

OF THE CARDIOVASCULAR SYSTEM

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