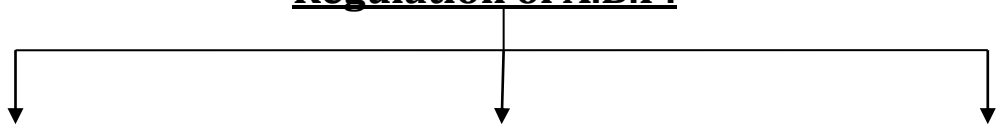
Components of CVS and functional parts of systemic circulation

1. **The left side of the heart**: It acts as a Pressure pump driving blood to various tissues through the system of blood vessels.
2. **Elastic arteries**: It contains a lot of elastic fibers in their wall and shows the phenomenon of compliance and elastic recoil. As compliance means that the arterial wall is stretched in front of the ejected blood and prevents marked increase in pressure during systole. While, its recoil over the moving blood column prevents marked drop of pressure during diastole. That prevents marked change of pressure and the blood flow continues both during systole and diastole (throughout the Cardiac cycle).
3. **Arterioles (resistance vessels):** They contain a thick layer of smooth muscles in their wall, and their diameter is under the control of autonomic nervous system which acts on the smooth muscles. Thus with sympathetic stimulation V.C of their wall occurs with marked increase in the resistance to blood flow leading to marked increase in arterial blood pressure. The reverse occurs with parasympathetic stimulation which causes V.D of the arterioles and decrease in the peripheral resistance so arterial blood pressure markedly drops.
4. **The Capillary (Exchange vessels)**: The capillary network is the site of contact between the blood and the interstitial cell spaces. So, it is the only site of CVS through exchange between blood and surrounding tissue occur.
5. **The Venous Vessels (Capacitance vessels):** They have highly distensible walls. So, they can accommodate a large volume of blood with little change in pressure. The veins can either distend and store or contract and mobilize blood within C.V.S.
6. **The right heart (Volume pump):** The right side of the heart receives venous return which carries high concentration of CO_2 and waste products and Pumps it to the pulmonary circulation.
7. **Lymphatic Vessels:** Act as a drainage system preventing collection of fluids in the interstitial tissue spaces

Regulation of A.B.P:



A. Nervous regulatory mechanism (Rapidly acting)

B. Capillary fluid shift mechanism (Intermediately acting)

C. Hormonal mechanism (Slowly acting)

A. Nervous Regulation

(Rapidly acting)

Medullary Cardiovascular Centers: They are

1- Vasoconstrictor center (V.C.C):

- This center discharge continuously at a definite rate under basal condition (it has a tone, which can be increased or decreased).
- The V.C.C sends its impulses to the arterioles through sympathetic V.C fibers.
- Stimulation of the V.C.C ⇒
    a. V.C of the arterioles → ↑ P.R → ↑ A.B.P.
    b. V.C of the veins → ↑ venous return → ↑ S.V → ↑C.O.P → ↑ A.B.P

2- Cardio-stimulatory Center

- This center has a low tone under basal conditions.
- Stimulation of this center → ↑ H.R & ++ force of myocardial contraction
- Both lead to increased A.B.P
- Impulses from this center reach the heart through cardiac sympathetic fibers.

N.B: V.C.C & C.S.C are called together the pressor area.

3- Cardio-inhibitory Center(C.I.C)

- This center has a strong basal tone, which continuously inhibits the inherent high rhythm of the S.A.N
- Impulses from this center reach the heart through the vagal fibers.
- Stimulation of this center → ↓ H.R → ↓ C.O.P → ↓ A.B.P

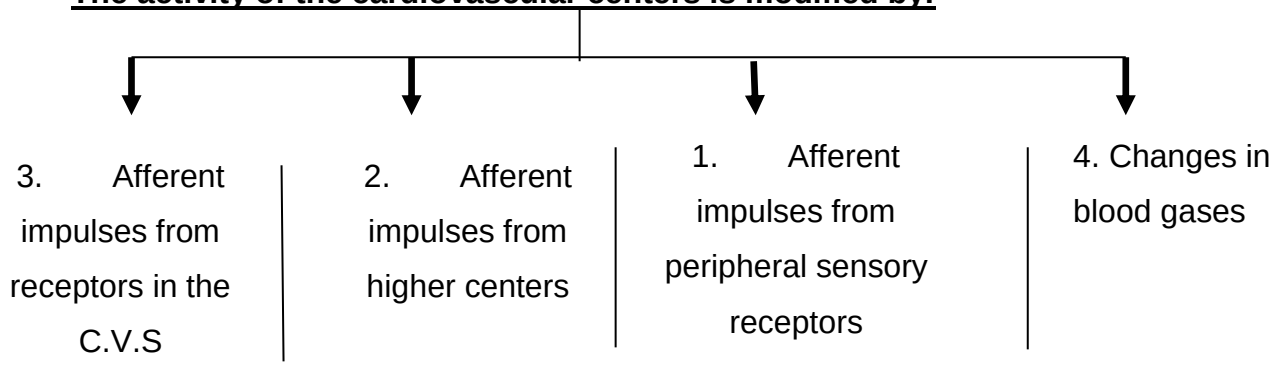
4- Vasodilator Center (V.D.C)

- Stimulation of this center → ↓ A.B.P as it send impulses which inhibit the V.C.C

N.B: V.D.C and C.I.C are called together the depressor area.

There is reciprocal enervation between the pressor and the depressor areas i.e. stimulation of the pressor area leads to inhibition of the depressor area and vice versa.

The activity of the cardiovascular centers is modified by:



1. **Afferent impulses from the cardiovascular receptors:** These receptors are
   a) Arterial pressure receptors (baroreceptors).
   b) Atrial receptors.
   c) Left ventricular receptors.
   d) Pulmonary baroreceptors.

a) Arterial pressure receptors (baroreceptors)

- Site: Carotid sinus & Aortic arch.
- Stimulus:
  - These receptors are stimulated by **stretching** the arterial wall
  - They begin to discharge at a pressure of 60 mmHg, and the frequency of discharge is increased with increased pressure.
  - **The sensitivity of these receptors reaches its maximum at the normal level of A.B.P.**

- When the pressure > 180 mmHg there is little increase in the frequency of discharge.

▪ Function

The arterial baroreceptors are very important for regulating the functions of the cardiovascular centers as:

i. At normal level of arterial blood pressure

- The baroreceptors discharge inhibitory impulses continuously through the carotid sinus nerve & the vagus nerve to the medullary C.V.C ⇒

- Inhibition of the V.C.C to decrease its rate of discharge to the level that maintains the V.C tone of the arterioles at the level which keep the normal A.B.P.
- Inhibition of the C.S.C → ↓ rate of sympathetic discharge to the heart.
- Stimulation of the C.I.C →inhibition of the inherent high rhythm of the S.A.N
- So the arterial blood pressure is kept at the normal level.

ii. When the arterial blood pressure is increased.

- When A.B.P is increased above normal, the rate of inhibitory impulse discharge from the carotid sinus & aortic arch is increased → inhibition of the pressor area and stimulation of the depressor area → ↓ A.B.B.P to the normal value as follow:
  - Inhibition of the V.C.C: ⇒ arteriolar V.D & ↓ P.R ⇒ ↓ A.B.P
  - Inhibition of the C.S.C ⇒ ↓H.R &
    ↓ Force of ventricular contraction
    ⇒ ↓ C.O.P ⇒ ↓ A.B.P
  - Stimulation of the C.I.C: ⇒ ↓H.R &
    ↓ Force of ventricular contraction
    ⇒ ↓ C.O.P ⇒ ↓ A.B.P
  - Stimulation of the V.D.C⇒ inhibition of the V.C.C ⇒ V.D& ↓ P.R ⇒ ↓ A.B.P

iii. When the arterial blood pressure is decreased.

The reverse will occur

N.B: Carotid sinus syndrome

Some peoples have a sensitive carotid sinus reflex, so when a tight collar is turned around the neck of such peoples, it stimulates the receptors in the carotid sinus leading to discharge of impulses which cause inhibition of the pressor area and stimulation of the depressor area leading to marked drop of the arterial blood pressure and decreased cerebral blood flow leading to cerebral hypoxia and loss of consciousness.

b) Atrial receptors (Volume receptors)

- Site: They are present in the walls of both atria near their junction with the superior vena cava on the right atrium and the pulmonary veins on the left atrium.
- Stimulus: The rate of discharge from these atrial receptors is proportional to the degree of filling of the atria (with the central venous pressure)
- Innervations: Impulses from these receptors are carried along afferent fibers in the vagus nerve to reach the medullary C.V.C

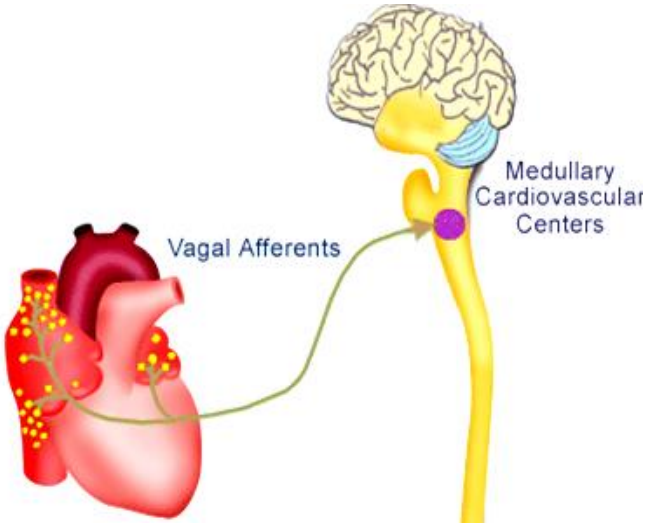


Fig.(51): Atrial volume receptors

- Function: These receptors send inhibitor impulses to cardiovascular centers as:

2. Afferent impulses from the higher centers

a- From the cerebral cortex.

- Emotions and muscular exercise leads to stimulation of the cerebral cortex.
- Impulses originate from the limbic lobe and the premotor area of the cerebral cortex and passes to the medullary C.V.C through the hypothalamus.

- This leads to stimulation of the pressor area and inhibition of the depressor area → ↑ A.B.P which can occur before the beginning of exercise.

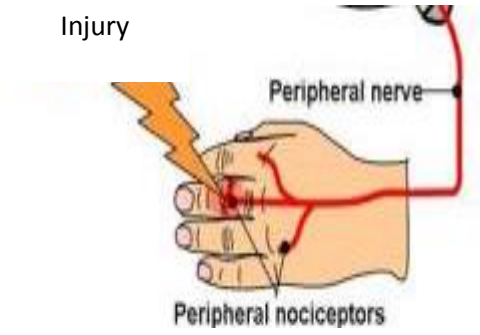
b- From the Hypothalamus.

The hypothalamus is considered a center of the autonomic nervous system which sends impulses to modify the activity of the medullary cardiovascular centers in case of

- Regulation of the body temperature, as during exposure to hot environment there is V.D of the skin blood vessels to increase heat loss.
- During emotions.

3. **Afferent impulses from the peripheral sensory areas**

- Stimulation of the cutaneous pain receptors leads to reflex acceleration of the heart and rise of arterial blood pressure due to increased secretion of adrenaline and noradrenaline.
- Stimulation of the visceral pain receptors → ↓ ABP due to stimulation of the depressor area and inhibition of the pressor area leading to decreased C.O.P and P.R.



Cutoneous pain

Visceral pain

N.B: Alam – Smirk reflex

Stimulation of the proprioceptors in the skeletal muscles during exercise leads to reflex accleration of the heart and rise of arterial blood pressure.

4. **Effect of changes in blood gases.**

Changes in blood gases affect the medullary C.V.C by two different mechanisms

a- Direct mechanism:

- Mild Hypoxia or hypercapnia will stimulate the pressor area & inhibit depressor area → ↑ A.B.P.
- Sever hypoxia or hypercapnia has a toxic effect on the C.V.C and result in hypotension and death.

N.B: Cushing reflex(viiiiiiiiiiiiip)

- Sudden rapid rise of intracranial tension → compression of the medullary blood vessels → hypoxia and hypercapnia which results in stimulation of the V.C.C, C.I.C and stimulation of the respiratory center.
- All these → ↑ A.B.P to overcome the intracranial resistance and maintain the cerebral blood flow…… (Significance).
- The decreased heart rate is a reflex due to increased A.B.P

b- Indirect mechanism.

- This is carried out through the peripheral chemoreceptors which are small highly vascularized bodies located along the aortic arch and near the carotid sinus so they are called aortic and carotid bodies.
- The aortic body sends its impulses along the vagus nerve to the medullary centers, while the carotid bodies send their impulses through the carotid sinus nerve, which is a branch of the glossopharyngeal nerve.

B. Capillary fluid shift mechanism
(Intermediate acting mechanism

- The volume of the tissue fluids is 4 times greater than the plasma volume
- So, it acts as a reservoir for the plasma (when the plasma volume is decreased it is corrected by replacement of tissue fluids to the intravascular compartment).

a- When A.B.P is decreased

- This leads to decreased hydrostatic pressure which is filtering force.

- So, the absorption force is increased and fluids are transported from the tissue spaces to the intravascular compartment → ↑ plasma volume & ↑ C.V.P
- So, S.V is increased → ↑ C.O.P → ↑ ABP.

b- When A.B.P is increased

- The reverse occurs.

. **Hormonal Mechanism**

(Slowly acting Mechanism) This regulatory mechanism occurs through:

a. Impulses from atrial mechanoreceptors

b. Renin-angiotensin system

c. Atrial natriuretic peptide. (A.N.P)

a- Impulses from the atrial mechanoreceptors.

- When the plasma volume is increased this → ↑ C.V.P → stimulation of the volume receptors in atrial wall.
- That sends impulses to the Hypothalamus along the vagus nerve → ↓ release of the A.D.H secretion → ↑ excretion of H2O &Na+ ions in urine → reduction of the plasma volume & ↓ A.B.P.
- On the other hand when the plasma volume is decreased as in vomiting, diarrhea, or blood loss as in hemorrhage: The reverse occurs

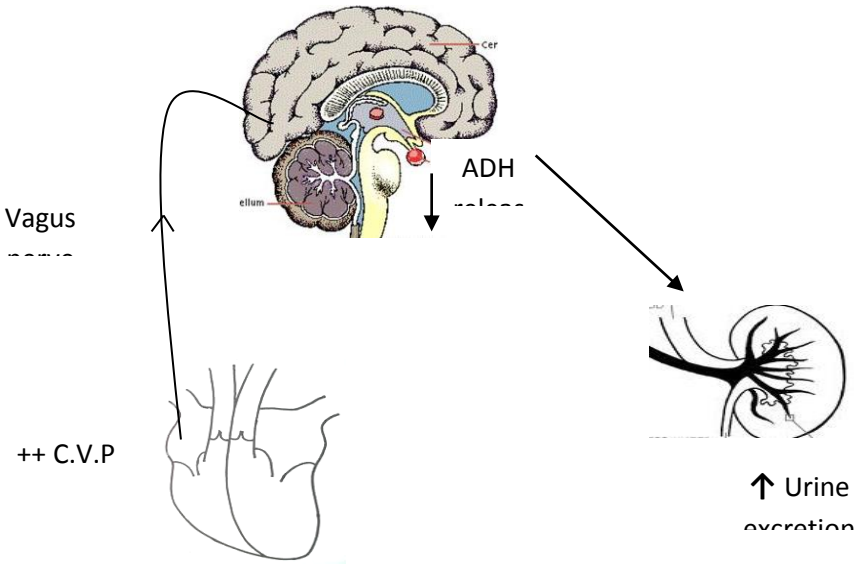


Fig.(54): Atrial mechanoreceptors

b- <u>Renin- Angiotensin system:</u>

- ↓ ABP
- Renal ischemia
- Sympathetic stimulation

} ++ Renin secretion

<u>Action of renin:</u>

- Renin passes to the blood stream acting on α globulin in the plasma called angiotensinogen converting it to Angiotensin I which is converted into Angiotensin-II by Angiotensin converting enzyme (ACE) present in the pulmonary circulation.
- Angiotensin II lead to ↑ A.B.P by:
  - Powerful V.C of the arterioles as the action of Angiotensin II is 50 times greater than that of adrenaline.
  - Angiotensin II stimulate the release of aldosterone → salt and water retention → ↑ V.R , C.V.P , C.O.P and A.B.P
  - Stimulation of A.D.H release leading to water retention.

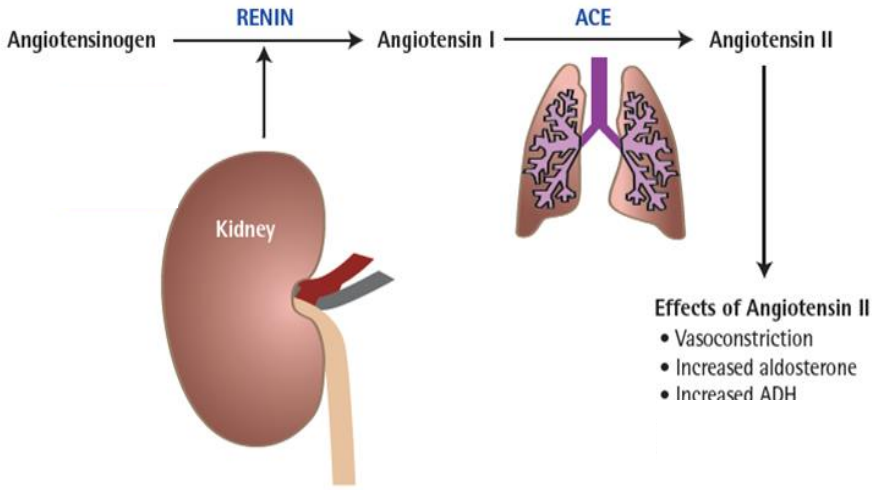


Fig.(55): Renin Angiotensin system

C- <u>Atrial Natriuretic peptide (A.N.P)</u>

Over stretch of the atria by increased blood volume stimulate its release of ANP which → ↑ excretion of excess sodium and water by the kidney → ↓ blood volume back to normal.

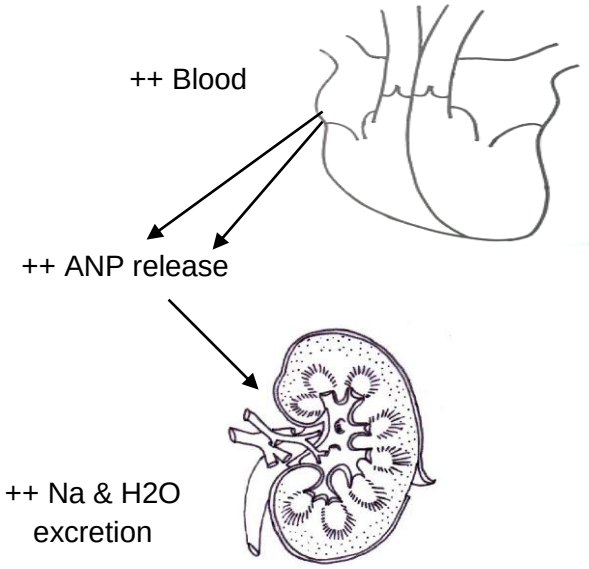


Fig.(56): A.N.P role in A.B.P regulation

Arterial pulse

- The blood forced in the aorta during systole sets a pressure wave which travels down the arteries expanding the arterial wall and felt as a pulse.
- The velocity of the pressure is about 7 meters / sec.
- The rate of transmission of the pulse wave depends on the elasticity of the wall of the arteries.
- In atherosclerosis the arteries becomes rigid and the rate of transmission becomes faster than the normal elastic arteries.

Site: The arterial pulse can be felt in superficial arteries as radial, ulnar & femoral. The carotid pulse is most important and should be searched whenever cardiac arrest is suspected.

Physiological importance of arterial pulse: It is important for:

a. Determining the heart rate.

b. Rhythm (regular or not)
c. Force of blood ejection: as when the stroke volume is increased the pulse becomes full e.g. in muscular exercise, when it is decreased as in hemorrhage the pulse becomes weak.
d. To detect the elasticity of the artery as normally the wall of the artery is not felt because it is soft. In atherosclerosis this elasticity is lost and the artery is felt as a cord like.

Arterial pulse wave☹viiiiiiiiiiiiiiiiiiiiiiiiiiiiiiiiiiiiiiiiiiiiiiip)!!!! مهمه جد

- The arterial pulse wave is formed of an ascending limb or (anacrotic limb) and a descending limb (Catacrotic limb).
- The ascending limb is caused by ejection of blood from the left ventricle to the aorta and the pressure in the aorta rises to its systolic level.
- While the descending limb is caused by gradual decline of pressure to the diastolic level.
- The Catacrotic limb contain the dicrotic notch or incisura
- Dicrotic notch or incisura: it is caused by sudden closure of the aortic valve in the beginning of left ventricle relaxation which causes back flow of the blood and reduction of pressure.
- Dicrotic wave: it follows the incisura and is produced by elastic recoil of the aortic wall over the moving blood.