Physiology

L-30

Dr.Hind

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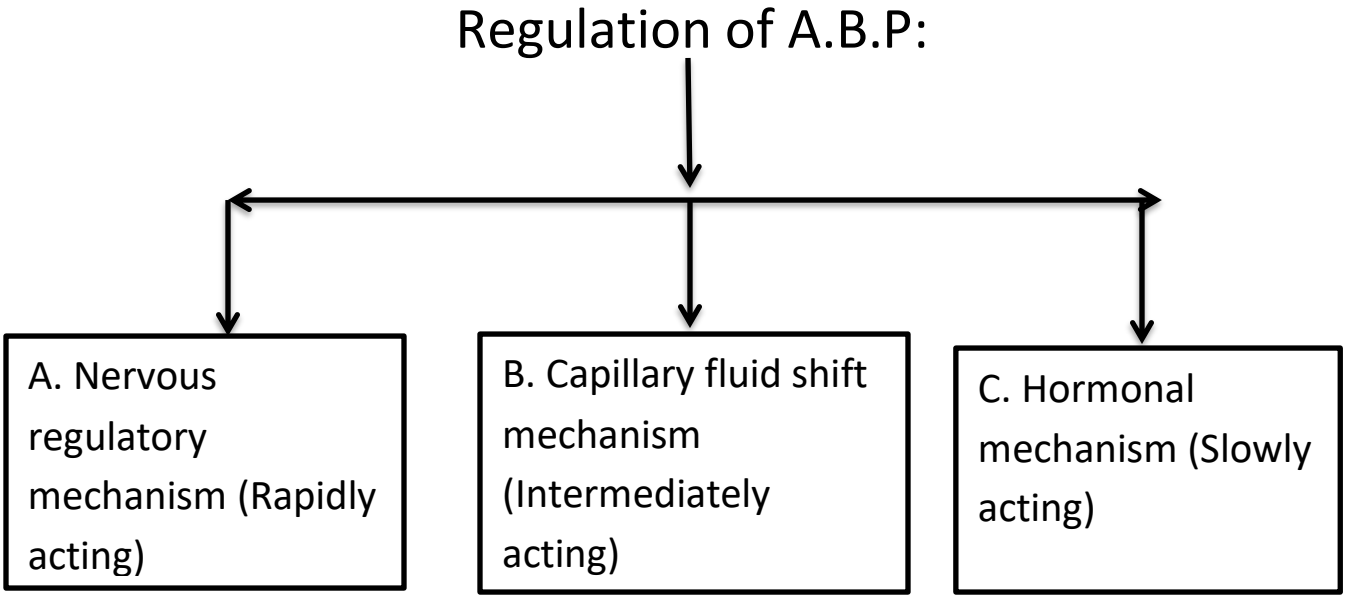
*pressure= flow * resistance

* ABP= COP * total peripheral resistance

= H.R * stroke volume * total peripheral resistance



-Any stimulus or factor that increases syst olic blood will also increase the pulse pressure.

-And any stimulus or factor will increase the diastolic blood pressure will decrease the pulse pressure.

-By maintaining the blood pressure we maintain adequate tissue perfusion thus maintaining homeostasis.

A. Nervous Regulation

(Rapidly acting)

Medullary Cardiovascular Centers:(all of them are found in the medulla oblongata), They are:

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

-In the medulla oblongata.

-It's active all the time, so it has a basal tone.

- 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.

(P.R is the function of arterioles)

b. V.C of the veins → ↑ venous return → ↑ S.V → ↑C.O.P → ↑ A.B.P

-The vasoconstrictor center is active all the time so it has a basal tone.

-So if we have a blood vessel it's lumen radius at rest 7mm and his wall can dilate at a maximum of 10 mm.

-In vasoconstriction our body makes sure that there is an adequate blood supply reaching the body so it's lumen radius will decrease from 7mm to 5mm (more than this no adequate blood supply reaches the tissue)

-And in vasodilatation our body makes sure that the blood will keep moving at the blood vessels and it's not pooling in the lumen of the blood vessel so its lumen radius will increase from 7 mm to 9 mm (more than this blood pooling will occur)

-If a vasoconstrictor center was not active all the time(even at rest) the blood will stay in veins and it will not complete the circulation.

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

(arterioles → vasodilatation → P.R decrease → A.B.P decrease).

-Arteriole with vasodilation will decrease the peripheral resistance and will decrease the ABP.

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

-So if our body needs to increase its arterial blood pressure for any reason like exercise or if we lost a high amount of blood that pressor area will be activated.

-And if our body needed to decrease its arterial blood pressure for any reason like in hypertension the depressor area will be activated.

-Hypertension is very Dangerous as it could cause the raptures of blood vessels and this will cause severe internal bleeding and even death.

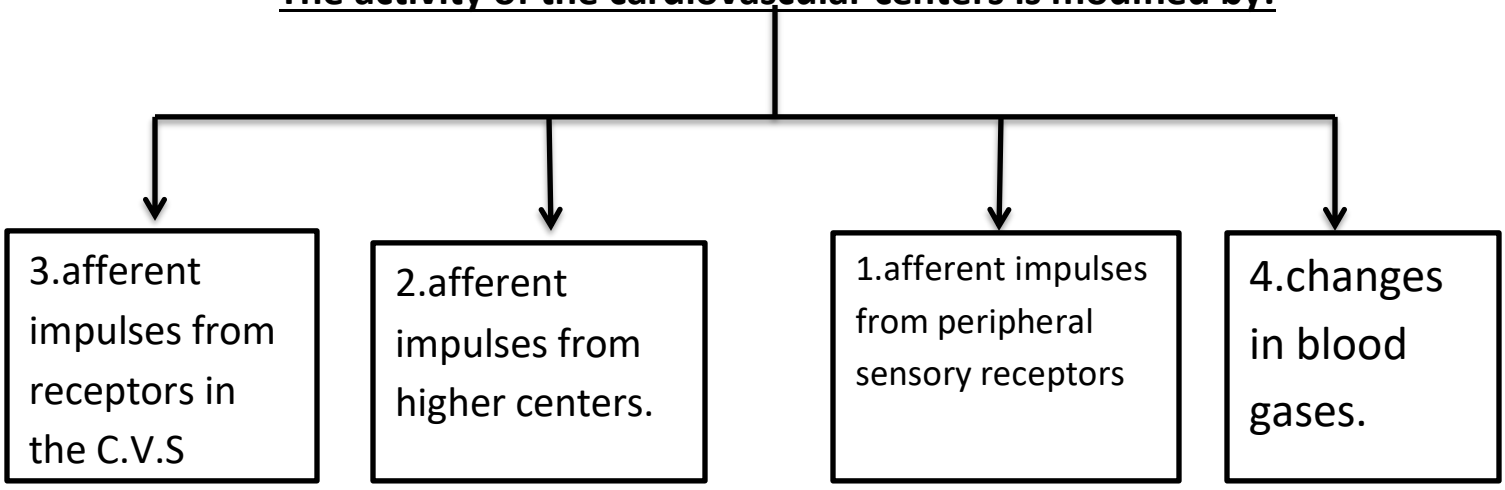
-Any stimulus that activates our pressor area will deactivate or inhibit our depressor area and vice versa.

-So any stimulus that activates our cardio stimulatory center will inhibit our cardio inhibitory center and vice versa except Cushing reflex.

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: active all the time (baroreceptors)

* Site: Carotid sinus **in the blood vessel wall** & Aortic arch **(modification to the wall of blood vessels to make it very sensitive to stretch).**

* 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.

Arterial pressure receptors are active all the time.

These baroreceptors are some type of modifications of the wall of blood vessels to make it very sensitive to stretch.

- The sensitivity of these receptors reaches its maximum at the

normal level of A.B.P.

-nearly at 140 mm Hg or higher there is no increase in action potential discharge

-At normal blood pressure120/80 baroreceptors send inhibitory impulses to the pressor area because there is no need to increase arterial blood pressure.

-At high blood pressure150/100 The baroreceptors will increase the discharge of action potential thus inhibiting the pressor area and activating the depressor area to decrease the arterial blood pressure .

-At low arterial blood pressure like in hypovolemic shock the blood volume will be so small so it will not activate the baroreceptors so There will be no discharge of action potential's so the pressor area will be activated and the depressor area will be inhibited to increase the arterial blood pressure.

- 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.

-Any stretching of the carotid sinus like wearing a tight collar or shaving close to the neck will stimulate the carotid sinus and will cause loss of consciousness and the other symptoms of the syndrome.

-One of the ways used to treat this syndrome is by cutting the nerve that feeds the carotid sinus .

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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.

The pressure at the superior Venna cava is called the central venous pressure (CVP)

Which indicates the pressure at the right atrium which also indicates the pressure of the right side of the heart.

* Stimulus: The rate of discharge from these atrial receptors is proportional to the degree of filling of the atria (with the central venous pressure)

So if the volume of blood in the atria increases the central venous pressure will also increase this will activate the depressor area and inhibit the pressor area

And if the volume of blood in the atria is decreased like in the hypervolemic shock the reverse will occur

Hypovolemic shock causes severe drop in the arterial blood pressure and a severe decrease in the blood volume

* Innervations: Impulses from these receptors are carried along afferent fibers in the vagus nerve to reach the medullary C.V.C

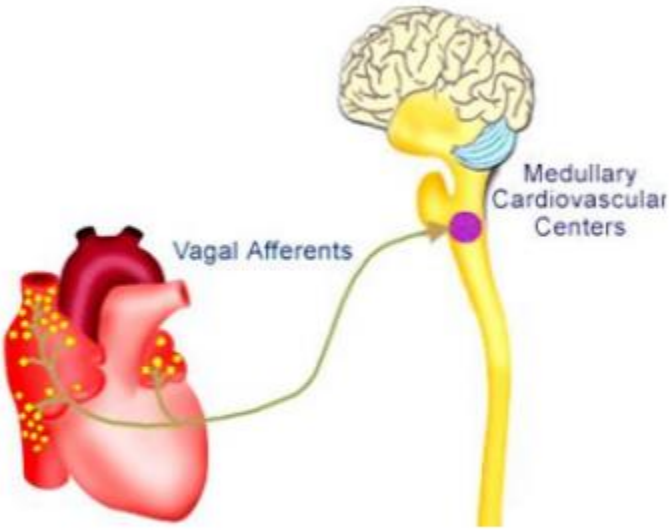


Fig.(51): Atrial volume receptors

* 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.

-When someone is willing to excersice the pressor area will be activated to give more blood supply to muscles(adequate) during the exercise.

- 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.

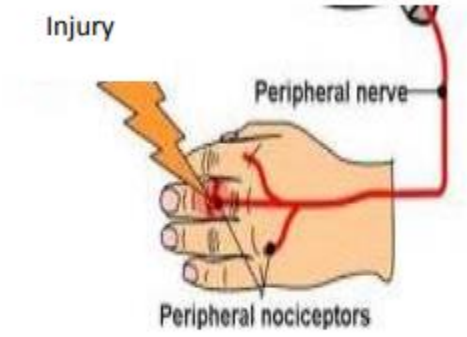
3. Afferent impulses from the peripheral sensory areas

* Stimulation of the **cutaneous pain receptor** **(mild to moderate pain)** 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 **(severe pain)** → ↓ ABP due to stimulation of the depressor area and inhibition of the pressor area leading to decreased C.O.P and P.R.

-Cutaneous pain receptors reacts to mild to moderate pain but visceral pain receptors react only to severe pain.

-Mild to moderate pain will activate the pressor area and the respiratory centers the arterial blood pressure will increase and the heart rate will increase too.



Cutoneous pain

Visceral pain

N.B: Alam – Smirk reflex Stimulation of the proprioceptors in the skeletal muscles during exercise leads to reflex acceleration of the heart and rise of arterial blood pressure.

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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)

-Rapid , Acute rise of intracranial pressure (CSF pressure) stimulates vasoconstrictor center and cardiac inhibitory center.

- Sudden rapid rise of intracranial tension → compression of the medullary blood vessels → hypoxia and hypercapnia which results in stimulation ofthe 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.

-So hypoxia hypercapnia and acidosis signals will go from carotid and aortic bodies to the medulla oblingata and stimulate the pressor area and respiratory centers so the arterial blood pressure will rise to make oxygen and nutrients reach the body tissues faster

- 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

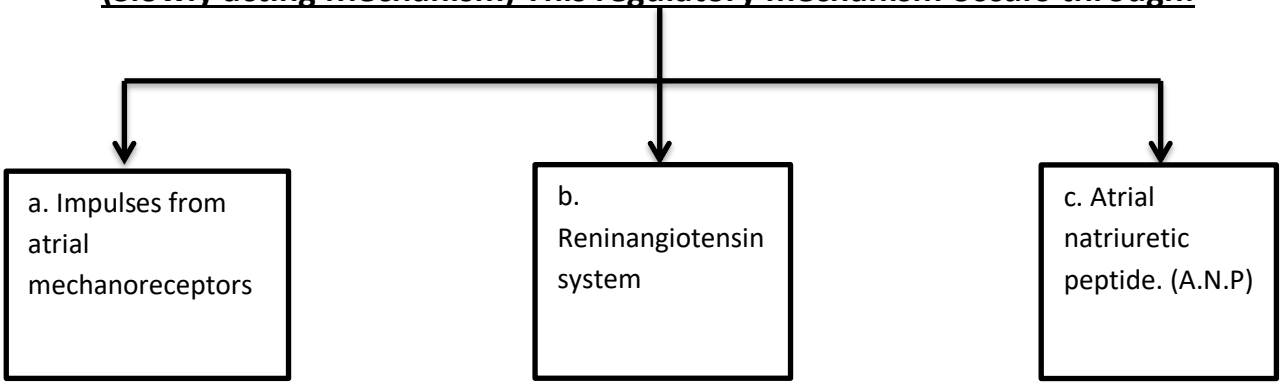
- So, S.V is increased → ↑ C.O.P → ↑ ABP.

b- When A.B.P is increased

- The reverse occurs.

Hormonal Mechanism

(Slowly acting Mechanism) This regulatory mechanism occurs through:

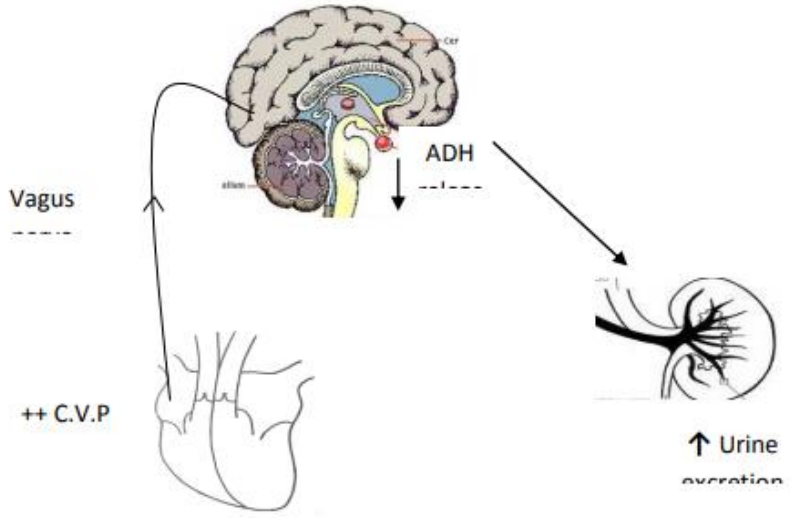


a- Impulses from the atrial mechanoreceptors.

- 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



b- Renin- Angiotensin system:

- ↓ ABP
- Renal ischemia
- Sympathetic stimulation

→ ++ Renin secretion

Action of renin:

- 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.

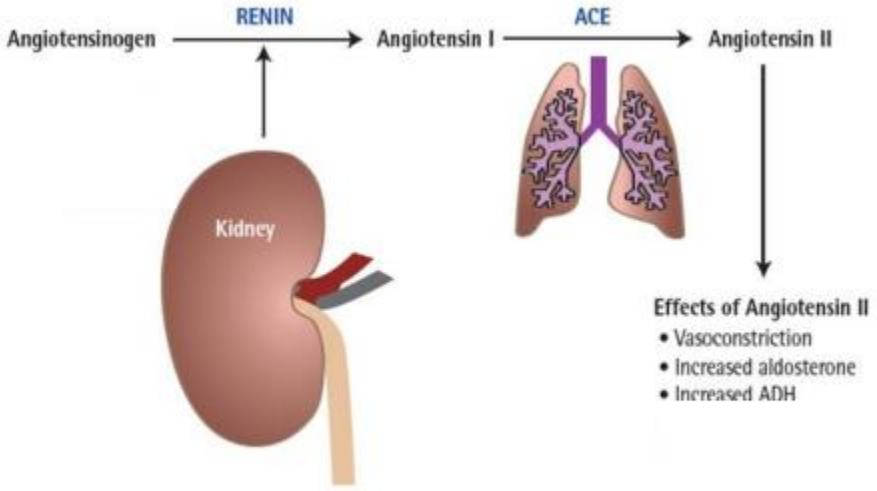


Fig.(55): Renin Angiotensin system

C- Atrial Natriuretic peptide (A.N.P)

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

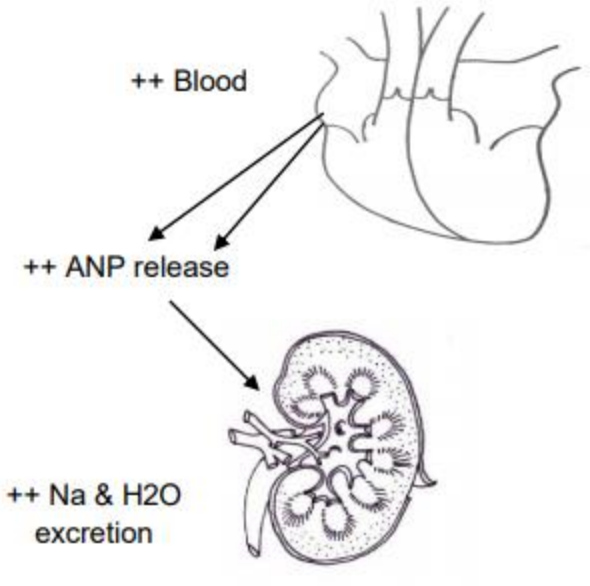


Fig.(56): A.N.P role in A.B.P regulation

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