The cardiovascular system

***Functional anatomy of the heart*:-**

The heart is actually two separate pumps, (right heart) that pump blood through the lungs and (left heart) that pumps blood through the peripheral organs.

Each of these two pumps is in turn comprised two chambers, an (atrium) and (ventricle). Located between atria and ventricles on both sides of the heart are (atrio-ventricular A-V valve), which normally allow blood to flow from atrium to the ventricle but prevent backward flow from ventricles to the atria. The right A-V valve is called (tricuspid valve) and the left is called the (mitral valve).

Blood exits from right ventricle through the (pulmonary valve) into the pulmonary artery and from the left ventricle through the (Aortic valve) into the aorta. The pulmonary and aortic valves allow blood to flow into the arteries during ventricular contraction (systole) but prevent blood from moving in the opposite direction during ventricular relaxation (diastole).

The wall of the heart called (myocardium) are composed primarily of cardiac muscle (myocytes). The inner surface of the myocardium, which comes in contact with blood within the cardiac chambers, is lined with a layer of cells called (endothelial cell). Which provide a smooth surface throughout the cardiovascular system, including the blood vessel, and help to prevent blood clotting?

The cardiac muscle is arranged in layers that completely encircle the chambers of the heart. When the walls of the chamber contracts, this exerts pressure on the blood that the chambers enclose and propels the blood forward. Cardiac muscle cells are striated and have typical myofibrils containing (actin) and (myocin) filaments, similar to those found in skeletal muscle, which slide along each other during the process of contraction. Adjacent cardiac cell are joined end to end at structured called (intercalated discs) which are actually cell membrane that have a very low electrical resistance. This permits ions, and therefore action potentials to move with ease

from one cardiac muscle cell to another. Therefore, cardiac muscle is a (syncytium) of many myocytes that are interconnected, when one of these cells become excited, the action potential spread rapidly throughout the interconnection.

Rhythmical excitation of the heart:

Action potential in cardiac muscle:

Contraction of cardiac muscle, similar to contraction of other muscles, is triggered by depolarization of the cell membrane and development of action potential, which spread from one cell to another. The resting membrane potential of normal ventricular muscle is approximately (- 90 mv)

Time = 400ms
Ventricular action potential

time =150 ms
atrial action potential

Stage number	stage name	stage cause	ionic movement
0	Rapid Depolarization	open of (voltage- gated fast Na^+ channel)	Na^+ influx
1	Early repolarization	open(Cl^- channel)	Cl^- influx
2	Plateau	open (transient outward K^+ current) close (voltage-gated fast Na^+ channel) influx open (L- type slow voltage gate ca^{++} channel)	K^+ efflux prevent Na influx Ca^{++} influx
3	Late Repolarization	open (inward rectifying K^+ current) open outward (delayed) rectifing K^+ current	K^+ efflux K^+ efflux
4	Resting membrane potential		

myocardial cell resting membrane potential as most of the cell of the body are characterized by: first it has high K^+ , low Na^+ and Ca++ ions intracellular. Second, it has negative action potential (⁻ 90 mv) i.e. intracellular has lower voltage by 90 mv comparing to outside the cell. The negative action potential is caused by two factors : the high concentration of K ions in the intracellular fluid and high permeability of cell membrane to K ions compared with other ions. Theoretical resting membrane potential can be calculated according to wernst equation :

$$\text{equilibrium potential} = 61 . \text{Log} \frac{\text{concentration of ion outside the cell}}{\text{concentration of ion inside the cell}}$$

for K^+ equilibrium potential = 61 . log 4/140

= ⁻ 94 mv.

Action potential of SA node (sinoatrial node):

Pre - potential or pacemaker potential:

AV + SA node are called (pacemaker) because they can generate action potential while atria and ventricle not. The resting membrane potential in pacemaker tissue is ⁻ 60 mv (i.e. easily excitable) while atria and ventricle is ⁻ 90 mv. The action potential of the atria and ventricle is stable while in pacemaker tissue decline from ⁻60 mv to ⁻40 mv (called pre potential or pacemaker potential) the first 2/3 of the pacemaker potential is caused by K+ influx by (inward rectifying K+ current) and last 1/3 is due to Ca influx through T-type Ca channel (T: transient).

Stage 0: is due to opening of L-type Ca channel (L: Long lasting)

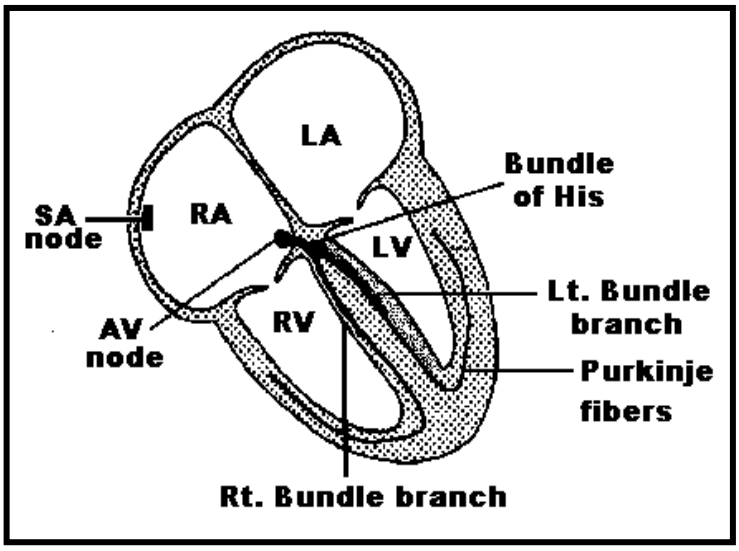
Stage 1 and Stage 2: Stage 1 (spike) is not seen, while stage 2 (plateau) is much shorter.
Stage 3 : Is caused by K^+ efflux through outward (delay) rectifier K^+ current.

Refractory period of action potential:

-Absolute refractory periods: It is the period during which membrane cannot be re-excited by an outside stimulus, regardless of level of external voltage applied, usually is due to prolonged plateau.

-Relative refractory period: Is the time during which propagated action potential can be generated but with a depolarizing stimulus that is larger than normal.

Spread of cardiac excitation:



1/Atria: Sinoatrial node (SA node) is located in the wall of the atria and has the ability to generate impulse that spread by the wall of the atria and make them contract, and then move to atrio-ventricular node (AV node) at speed of (0.3 m/sec),

besides that the impulses will be spread at a faster speed (1 m/sec) by 3 inter-nodal (as it connects SA node with AV node) bundles: 1- anterior internodal tract of Backman. 2- middle internodal tract of Wekeback. 3- posterior internodal tract of Thorel.

2/A-V node : The atria is completely separated from ventricle by atrio-ventricular fibrous tissue so that impulses can be transmitted only through A-V node. There is a delay in the A-V node (A-V nodal delay) which occurs in the AV node. This delay allows the atria to pump blood into the ventricles, before the ventricles begins their pumping cycles. The cause of this delay is due to slow conduction of impulse (as their size is smaller than the size of normal atrial muscle fibers). The slow conduction is caused by: 1- All these fibers have resting membrane potential that are less negative than normal resting potential of other cardiac muscle. 2- Few gap junctions connect the successive muscle cell in the pathway so there is a great resistance to impulse. In normal heart the only pathway by which the impulse can travel from atria to the ventricle is through A-V bundle.

The purkinje system begins in the SA node and from here passes through the atria by the way of the (internodal pathway to the A-V node). From the A-V node, a large bundle of purkinje fiber called (AV bundle or bundle of his). Bundle of his divided into right and left bundle branch. Right and left branch now run on both sides of the septum subendocardially. At the apex they separate where right branch continue to supply right ventricle while left branch supply left ventricle. The system characterize by:

1. It is not nerve but modified myocardial cell.
2. Impulse pass in one direction to prevent re-entry pathway.

Conduction disorder caused by damage to AV node or AV bundle:

occasionally, pathological condition destroy or damage the AV bundle or AV node so that impulse no longer pass from the atria to the ventricle. When this occurs, the atria continue to beat at

the normal rate of the SA node, while the ventricles establish their own rate of rhythm. This condition is called (heart block). Usually the purkinje fiber in the AV bundle or one of the bundle branches of the ventricles become the (ventricular pacemaker), because these fibers have a higher rate of rhythm than the muscle fiber of of the ventricle. The natural rate of the rhythm in the ventricle after heart block can be as slow as 15 beats per minute or as rapid as 60 beat per minute. In heart block, the atrial contraction are not coordinated with ventricular contraction, which prevent the ventricle from becoming as well filled before contraction as in the normal heart. Loss of this coordinated atrial function impairs maximal heart pumping about 30%, however a person with heart block live for many years because the normal heart has tremendous reserve capacity for pumping blood.

Cardiac flutter and fibrillation – The circus movement:

Occasionally an impulse in the heart continues all the way around the heart, and when arriving back at the starting point, re-excites the heart muscle to cause still another impulse that goes around the heart, continuing indefinitely. This is called (circus movement). This does not occur in the normal heart for two reasons:

1/ Normal cardiac muscle has long absolute refractory period usually about 0.25 second, which means the muscle fiber can not be re-excited.

2/ the impulse of the normal heart travel so rapid that it will normally pass over the entire atrial or ventricular muscle mass in about 0.06 second, and therefore disappears before the heart muscle become re-excitable. In the abnormal heart, however, the circus movement can occur in the following conditions: 1. When the refractory period of the cardiac muscle become much less than 0.25 second. 2. when perkinje system is destroyed so that impulses take a far longer time to travel through the ventricles because of the slow conduction in the muscle fiber. 3. when the atria or the ventricle become dilated so that the length of the pathway around the heart is greatly increased, thereby increasing the time required to the impulse to travel around the

heart. When the impulse dose not travel directly around the heart, but instead travels in a zigzag direction, which lengthen the pathway to as much as times the direct distance around the heart, this obviously prolongs the time for transmission of the impulse and can result in re-excitation of the cardiac muscle.

In the atria, a regular movement around and around the atria causes (atrial flutter) whereas zigzag impulse cause (atrial fibrillation). The zigzag impulses in the fibrillation also divide into multiple impulses so that there may be as many as fire to ten impulses traveling in different directions at the same time. As a result, the atria partially contracted all the time, but they never contract rhythmically to provide any pumping action. Flutter only very rarely occurs in the ventricles, but (ventricular fibrillation) with many zigzag impulses spreading in all directions at once, is a major cause of cardiac failure and death. Either electric shock to the ventricle or ischemia of the ventricular muscle as a result of coronary thrombosis can initiate ventricular fibrillation.

Electrocardiogram (ECG):

Indirect recording of electrical potentials of the heart.

QT interval
PR interval

QRS duration
ST segment

Interval	Time average second	Event in heart
PR	0.16	Atrial depolarization and conduction through AV node
QRS	0.08	ventricular depolarization and atrial repolarization
ST	0.32	ventricular repolarization
QT	0.4	ventricular depolarization plus ventricular repolarization

Normal ECG consist of :

P wave: Which is caused by the depolarization process in the atria.

QRS complex of waves : which is caused by depolarization process of the ventricles.

And T wave: which is caused by repolarization of the ventricles.

The PQ interval : The time between atrial and ventricular depolarization.

Normally, depolarization begins in the atria approximately 0.16 second before beginning of the P wave and the Q wave, called [PQ interval or PR interval when Q wave is absent] and is normally 0.16 second. Abnormal slow conduction of the impulse through the AV bundle occurs when the bundle become ischemic following coronary thrombosis. When PQ interval become very long, conduction to the AV bundle will eventually cease, causing heart block.

Abnormal QRS waves:

Since the QRS wave represent passage of the depolarizing process through the ventricles, any condition that causes abnormal impulse transmission will alter the shape, the voltage, or the duration of the QRS complex. For example hypertrophy of one ventricle will cause increased voltage and is likely to increase predominantly R or S wave depending of the electrocardiographic lead and the ventricle affected. Also, damage to any portion of the Purkinje system will delay transmission of the impulse through the heart and therefore causes abnormal shape of QRS complex as well as prolongation of the complex.

Abnormal T wave:

Some diseases damage the ventricular muscle just enough that it becomes difficult for the muscle to re establish normal membrane potentials after each heart beat. As a result, some of the ventricle fibers may continue to emit electrical current longer than usual, which causes a bizarre pattern to the T wave such as a biphasic pattern, or sometime inversion of the T wave. Thus, an abnormal T wave ordinarily means mild to sever damage to at least a portion of ventricular muscle.

Elevated or depressed ST segment : current of injury:

Occasionally the ECG segment between the S and T wave is displaced either above or below the major level of ECG. This is caused by failure of some of cardiac muscle fibers to repolarize between each two heart beat.

As a result, between heart beats these fibers continue to emit large quantities of electrical current , called (current of injury) which cause an elevated or depress S-T segment is observed, one can be certain that at least some portion of the ventricle muscle is severely damaged.

Abnormal rhythm:

In atrial fibrillation, no true P wave can be discerned, but many very fine wave continue indefinitely in the ECG. Finally in extra systole of the heart, occasional QRS appear in the record point completely out of rhythm.

1. Normal ECG

2. Prolonged PR intervalin first degree heart block

3. high voltage of QRS in left ventricular hypertrophy

4.wide & prolong QRS due to damage in purkinje System seen in right bundle branch block.

5. Biphasic T wave

6. Inverted T wave
Seen in ischemic
Heart disease

7. elevated T wave

8. Depressed T wave

9. atrial fibrillation with no true P wave

10. ventricular ectopic

Mechanics and regulation of heart pumping:

Cardiac cycle:

Two major phases of cardiac cycles occurring in the ventricles:

1- Period of ventricular relaxation called, ***diastole*** lasting for about 0.5 second, in which the ventricle fill with blood.

2- A period of ventricular contraction and blood ejection called ***systole*** lasting about 0.3 second, thus, at a normal heart rate of about 72 beat/min, the entire cardiac cycle lasts about 0.8 second. As the heart rate increase, the fraction of cardiac cycle in diastole decrease, which means that the heart beating very fast may not remain relaxed long enough to allow complete filling of the ventricles before the next contraction.

Closure of AV valve (mitral + tricuspid) causes first heart sound. Closure of aortic and pulmonary valve causes second heart sound.

The largest volume of ventricle is end diastolic volume . The smallest volume of the ventricle is end systolic volume. Stroke volume amount of blood ejected by each beat. Stroke volume = end diastolic volume ⁻ end systolic volume. Ejection fraction = (stroke volume)/ (end diastolic volume). When increase amount of blood flow into the heart from vein and distend it's chambers, the stretched cardiac muscle automatically contract with increase force. This increased force in turn, pumps the extra blood through the heart into the arterial system. This is called: Frank-Starling law of the heart.

Autonomic nervous control of the heart:

There are two types of nerve fibers that supply the heart:

1- **Parasympathetic fibers:** Carry by the vagus, stimulation of Parasympathetic nerve cause release of acetylcholine (Ach) , stimulation cause decrease in heart rate.

2- Sympathetic fibers: carry by sympathetic nerve, stimulation of sympathetic nerve cause release of norepinephrin, stimulation cause increase in heart rate and increase in force of contraction.

Blood volume distribution:

About 84% of total blood volume is in the systemic circulation. 9% is in the pulmonary circulation, and 7% is in the heart. Within each of these circulations, about 3/4 of the blood is in the vein and about 1/6 in the arteries and 1/12 in the arterioles and capillaries. Thus although the capillary blood exchange nutrients and waste products in peripheral tissue and gases in the lungs, only a small part of the total blood volume is in the capillaries at any given time.

Systemic arterial pressure:

The left ventricle normally pumps about 5 litters of blood into the aorta each minute. Each heart beat ejects approximately 70 ml of blood into the aorta: this is called (stroke volume output).

As a result, the arteries become greatly distended during cardiac systole, and during diastole the recoil of the arteries causes blood stored in the arterial tree to (run off) through the systemic vessels to the vein. Thus the aortic pressure rises to its highest point, the (systolic pressure) during systole and fall to its lowest point, the (diastolic pressure) at the end of diastole. In the normal adult, the systolic pressure is approximately 120 mmHg and diastolic pressure is 80 mm Hg. This is usually written (120/80).

The difference between systolic and diastolic pressure (120– 80 = 40 mmHg. Is called (pulse pressure). The two most important factors that can increase pulse pressure are:

1- Increased stroke volume.

2- Decreased arterial compliance.

Decrease arterial compliance can result from hardening of the arteries that occurs with aging and/or arteriosclerosis.

Arterial pressure changes through out the cardiac cycle. The (mean arterial pressure), however, is not simply the value halfway between systolic and diastolic pressure, because diastolic usually lasts longer than systole.

True mean arterial pressure can be measured with catheter placed in arteries, but a fairly accurate estimate of mean arterial pressure can also be obtained from the systolic and diastolic pressure as follows:

Mean arterial pressure = diastolic pressure + 1/3 pulse pressure

For example mean arterial pressure = 80 + 1/3 (120 ⁻ 80) =93.3 mmHg. Because resistance to blood flow through the major arteries is so slight, the mean arterial pressure does not change markedly until the arteries become very small.

In the arterioles, the major site of resistance to blood flow along the vasculature, there is a large drop in pressure to about 30 mmHg at the juncture of the arterioles and capillaries.

The capillaries also contribute a moderate amount of resistance causing the mean arterial pressure to fall about 10 mmHg at the juncture of the capillaries and veins. Resistance in the venous system is relatively slight, and pressure falls only 10 mmHg in the entire system: pressure in the right atrium is approximately 0 mmHg.

Regulation of systemic arterial pressure:

Because systemic arterial pressure is the driving force for blood flow through the tissue of the body, it is not surprising that it is carefully regulated. Under resting conditions, the mean arterial pressure is approximately 100 mmHg, but for short periods of time, such as during strenuous exercise, mean arterial pressure may rise to as high as 150 mmHg in the normal person. In chronic hypertension, the pressure remains elevated indefinitely, or until treatment is instituted to lower pressure.

Blood pressure regulation is so important for homeostasis that the body is endowed with multiple short-term and long-term control mechanisms that keep mean arterial pressure relatively constant.

A- Cardiovascular autonomic reflexes (mechanism work within seconds to minutes):

I. Baroreceptor reflexes (Baro: pressure).

Are initiated by changes in mechanical stretch of receptor called (Baroreceptor) located in the wall of the internal caroted arteries, the aorta and in other regions of the circulation. When arterial pressure becomes excessively high in these vessels, these receptors are stimulated and impulses are transmitted to the brain to inhibit the sympathetic nervous system, as a result, the normal sympathetic impulses through the body are reduced, causing decreased heart rate, decreased strength of heart contraction, and decreased peripheral vascular resistance, which together help to reduce the blood pressure back toward normal. Conversely, a fall in blood pressure decrease the number of impulses transmitted by the baroreceptor; these impulses then no longer inhibit the sympathetic nervous system so that it become very active, causing the blood pressure to increase back toward normal.

There are also stretch receptor located in the other regions of circulation such as atria, the ventricles, and the pulmonary artery. These receptors called, (cardio pulmonary baroreceptors) also function in a manner similar to the arterial baroreceptors to keep the cardiovascular control center informed about pressure in the venous side of the systemic circulation as well as in the pulmonary circulation. Increased pressure in these regions inhibits sympathetic activity, where as decreased pressure stimulates sympathetic activity.

II. Chemoreceptors: (Chemo: chemical):

Chemoreceptors also exist in the brain and in the peripheral circulation, for example, an increase in carbon dioxide concentration excites the neurons of the vasomotor center of the brain stem, resulting in strong sympathetic stimulation through the body and an increase in blood pressure. This mechanism helps to ensure adequate pressure during stressful conditions, since physical stress to the body often increase the basal level, of metabolism and production of carbon dioxide. There are also small structure known as (carotid and aortic bodies) located in the arch of the aorta, that respond to changes in arterial blood oxygen when blood oxygen tension decreases, these

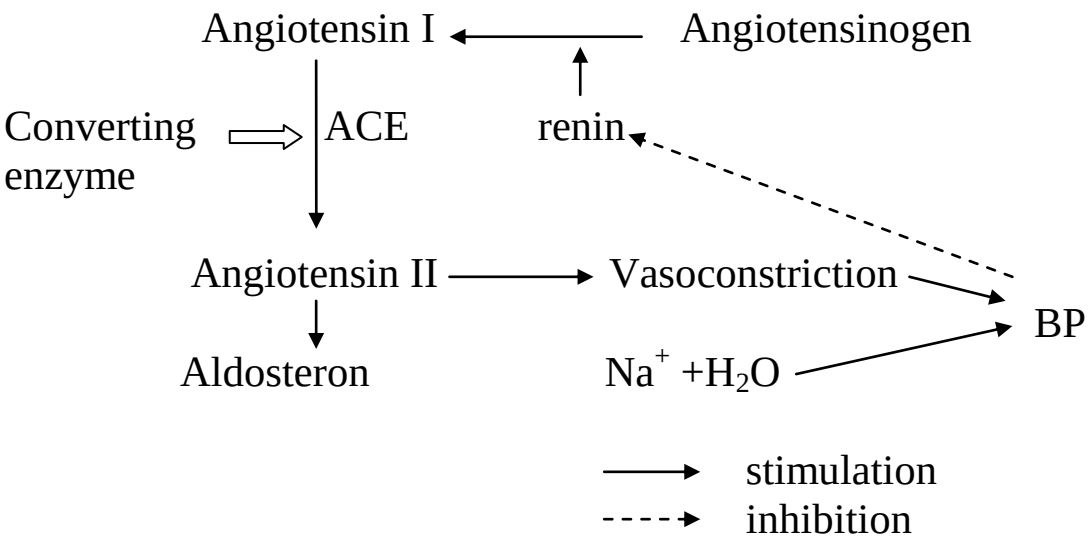
chemoreceptors cause reflex activation of the sympathetic nervous system, thereby raising blood pressure.
The increased blood pressure in turn helps to maintain adequate delivery of oxygen to vital organs, especially the brain, in which sympathetic stimulation does not markedly increase vascular resistance. However, these receptor are much more important for control of respiration than for blood pressure regulation.

III. Cerebral ischemia:
The lack of adequate blood flow to the brain (ischemia); also a potent stimulus for activation of sympathetic nervous system and increased blood pressure. In brain ischemia, the vasomotor center (center in the brain control changes in the heart and blood vessels) of the brain automatically becomes highly exited, probably because of failure of blood to carry carbon dioxide out of the vasomotor center rapidly enough. As a result central nervous system ischemic reflex) initiate strong sympathetic stimulation throughout the body, immediately elevating the arterial pressure; this in turn increase cerebral blood flow back towerd normal and helps to relieve the effect of ischemia.

B – Hormonal control of arterial pressure (Mechanism work within minutes to hours):

I. Norepinephrine and epinephrine:
Sympathetic stimulation to adrenal medulla causes release norepinephrine and epinephrine which add to vasoconstrictor effect of increased sympathetic stimulation.

II. Renin-angiotensin–aldosterone system:



III. Vasopressin (also called antidiuretic hormone: ADH):
Vasopressin has a direct vasoconstriction effect on peripheral blood vessels and also decrease renal excretion of water, there by increasing blood volume.

C. long–term regulation by the kidney (Mechanism work within hours or days).

The role of kidneys in long-term regulation of arterial pressure and circulatory volume:

The most important mechanism for long term control of arterial pressure is linked to control of circulatory volume by the kidney, a mechanism known as (renal-body fluid feedback mechanism). When arterial pressure rises too high, the kidney excrete increased quantities of sodium and water. As a result, the extracellular fluid volume and blood volume, both decreases, and continue to decrease until arterial pressure returns back to normal and the kidney excrete normal amount of sodium and water. Conversely, when arterial pressure falls too low, the kidney reduce their rate of sodium and water excretion and over a period of hours to days, if the person drinks enough water and eats enough salt to increase blood volume, arterial pressure will return to its previous level. This mechanism for blood pressure control is very slow to act, sometimes requiring several days or perhaps as long as week or more to come to

equilibrium. Therefore, it is not of major importance in acute control of arterial pressure. On the other hand, it is by far the most potent of all long term arterial pressure controllers.

This basic mechanism for long-term control of blood volume and arterial pressure is enhanced by some of the hormonal mechanisms discussed above, especially the rennin-angiotensin-aldosteron system. For example, increasing intake of salt tends to rise blood volume and arterial pressure, which in tern increase renal salt and water excretion through (pressure natruresis). The increase renal excretion eliminates the extra salt, with relatively small changes in blood volume and arterial pressure, as long as the rennin-angiotensin-aldosteron system are function normally. Most persons can easily eliminate extra salt intake, with very small increase in arterial pressure and blood volume, because increased salt intake also reduce the formation of angiotensin II and aldosteron, which helps to eliminate the additional sodium. As long as the rennin-angiotensin-aldosteron systemare fully operative, salt intake can be as low as 1/10 normal or as high as 10 times normal with only few millimeters of change in blood pressure. However, when the rennin-angiotensin-aldosteron system are not functioning change in salt intake have a much greater effect on blood volume and arterial pressure.

Hypertension:

Hypertension is a syndrome characterized by elevated systemic arterial pressure, a person usually considered to be clinically hypertension if the arterial pressure is greater than (140/90 mmHg). The incidence of hypertension is much higher in elderly subjects.

Hypertension is one of the principle risk factor for development of stroke, myocardial infarction, and kidney disease. Yet despite the great incidence of hypertension in the population and its important consequences, its precise cause in most people is still unknown. This type of hypertension is called (essential hypertension). In the remaining cases, the cause is usually renal disease, or nervous or hormonal disorders.

1. Renal hypertension:

a. Renal artery stenosis ⟶ ↓glomerular filtaration⟶ H_2O + electrolyte retention ⟶↑blood pressure ⟶↑ glomerular filtration ⟶normal water and electrolytes excretion

b. Kidney damage as in glumerulonephritis:

Kidney damage ⟶↓glomerular filtration ⟶ H_2O + electrolyte retention ↑ blood pressure due to increase cardiac output ⟶ increase blood flow to tissue ⟶vasoconstriction ⟶ (autoregulation) ⟶ increase blood pressure due to increase peripheral resistance

Blood pressure = Cardiac output X Peripheral resistance

Any factor increase cardiac output and/or peripheral resistance will increase blood pressure. Peripheral resistance affected by blood viscosity, and arterial radius.

c. Ischemia of the kidney:

Ischemia of the kidney⟶ release of angiotensinII⟶ ↑ Blood pressure while cardiac output is affected by stroke volume and heart rate.

2. Hormonal hypertension:

A. Adrenal medulla hormone.

Adrenal medulla tumor (pheochromocytoma)⟶ ↑ secretion of epinephrine & norepinephrine ⟶ ↑ Blood pressure with mechanism similar to that when there is sympathetic stimulation.

B. Adrenal cortex hormone:

Adrenal cortex tumor (primary aldosteronism) ⟶↑ aldosteron ⟶↑ Blood pressure.

Summary for control of blood Pressure:

Rapidly acting pressure control mechanism acting within seconds or minute:

1. The baroreceptor feed back mechanism
2. Central nervous system ischemic mechanism
3. Chemoreceptor mechanism

After an acute fall in pressure, as might be caused by severe hemorrhage the nervous mechanisms combine:

1. constriction of veins and provide transfer of blood into the heart.
2. Increase heart rate and contractility of the heart provide greater pumping capacity by the heart.
3. Constriction of the arteriols to impede the flow of blood out of the artery

Circulatory shock:

Circulatory shock occurs when the cardiac output is so greatly reduced that tissue throughout the body begin to deteriorate for lack of adequate nutrition. Any circulatory abnormality that greatly reduce cardiac output can cause circulatory shock. There are major classification of shock:

1. Cardiogenic shock:

This occurs most frequently in acute heart failure, in which the cardiac output falls because of impaired pumping ability of heart itself.

2. Hypovolemic shock:

This type of shock results from reduced blood volume caused by:

a. Blood loss (which is called hemorrhagic shock).

b. Plasma loss: caused by intestinal obstruction and sever burn.

c. Dehydration: caused by excessive sweating, severe diarrhea and vomiting, inadequate intake of fluid and electrolytes, kidney disease.

Hypovolemic shock decrease venous return to the heart thereby reducing cardiac output.

3. Septic shock: This condition, known as (blood poisoning) refers to widely disseminated infection to many area of the body, with the infection being born through the blood from one

tissue to another causing high fever, marked vasodilation, increase cardiac output, sludging of blood and disseminated intravascular coagulation (which is development of microclot in wide spread area). Some of typicalcauses are:

a. Peritonitis

b. Generalized infection from skin, or kidney, or from gangrene infection.

c. Endotoxin shock: which occur when large segment of gut loses much of its blood supply and results in proliferation of bacteria in gut release a toxin called (endotoxin)?

4. Neurogenic shock:

This is a circulatory shock that results from sudden inhibition of the sympathetic nervous system throughout the body caused by general anesthesia or brain damage. This allows all the systemic vessels to dilate and the blood to (pool) in the lower part of the body rather than returning to the heart. If a person with loss of sympathetic tone is kept in the standing position this can actually cause death but if the person is placed in a horizontal or head down position, sufficient blood will usually still flow back to the heart to allow survival.