The cardiovacular 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) 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.

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).

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.

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.

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, thesechemoreceptors 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.
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.

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