NAME: NWEKE TOCHI PRAISE

MATRIC NO: 19/MHS01/447

LEVEL: 200

COURSE: PHYSIOLOGY

1. Discuss the long term regulation of mean arterial blood pressure?
   The arterial blood pressure is regulated by four[4] regulatory mechanisms to maintain the blood pressure within normal limits;
   a. Nervous mechanism or short term regulatory mechanism
   b. Renal mechanisms or long term regulatory mechanism
   c. Hormonal mechanism
   d. Local mechanism
      The long term regulation of mean arterial ; the kidneys play an important role in the long term regulation of mean arterial blood pressure. When the blood pressure alters slowly in several days/ months/ years, the neural mechanism adapts to the altered pressure and loses the sensitivity for the changes. It cannot regulate the pressure any more.in such conditions the renal mechanisms operates efficiently to regulate the blood pressure. Therefore it is called long term regulation.
      Kidney regulate arterial blood pressure by two ways:
      - By regulation of ECF volume
      - Through renin-angiotensin mechanisms
        By regulation of extracellular fluid volume
        When blood pressure increase, kidneys excrete large amounts of water and salt, particularly sodium by means of pressure diuresis and pressure natriuresis [pressure diuresis/pressure natriuresis means the excretion of large quantity of water and sodium in urine respectively]. Because of natriuresis and diuresis there is decrease in the ECF volume and blood volume, which in turn brings the barterial blood pressue back to normal.
        When blood pressure decreases, there is reabsorption of water from the renal tubules is increased. This in turn increases ECF volume, blood volume and cardiac output resulting in restoration of blood pressure.

        Through renin- angiotensin mechanisms
        When blood pressure and ECF volume decrease the juxtaglomerular apparatus in the kidney is stimulated to increase its secretion of renin

Renin converts angiotensin into angiotensin 1 and angiotensin 1 is converted into angiotensin 2 by ANGIOTENSIN CONVERTING ENZYME
Angiotensin 2 acts in two ways to restore the blood pressure
It causes constriction of arterioles I the body so that the peripheral resistance is increased is increased and blood pressure rises.
In addition angiotensin 2 causes constriction of afferent arterioles in the kidneys so that the glomerular filtration reduces which results in retention of water and salts. This in turn increases the blood pressure to normal level
Simultaneously, angiotensin 2 stimulates the adrenal cortex to secrete aldosterone. This hormone increases reabsorption of sodium from renal tubules, the sodium reabsorption is followed by water absorption resulting in increased ECF volume and blood volume. It increases the blood pressure to normal level

1. Write short notes on the following :
    - Pulmonary circulation
    - Circle of willis
    - Splanchnic circulation
    - Coronary circulation
    - Cutaneous circulation

Pulmonary circulation: it is the portion of the circulatory system which carries deoxygenated blood away from the right ventricles, to the lungs and returns oxygenated blood to the left atrium and ventricle of the heart. The vessels of the pulmonary circulation are pulmonary veins and pulmonary arteries. Deoxygenated blood leaves the heart, goes to the lungs and then re-enters the heart; deoxygenated blood leaves through the right ventricle through the pulmonary artery. From the right atrium, the blood is pumped through the tricuspid valve (or right atrioventricular valve), into the right ventricle. Blood is then pumped from the right ventricle through the pulmonary valve and into the main pulmonary artery. The oxygenated blood then leaves the lungs through pulmonary veins, which return it to the left part of the heart, completing the pulmonary cycle. The pulmonary circulation loop is virtually bypassed in fetal circulation. The fetal lungs are collapsed, and blood passes from the right atrium directly into the left atrium through the foramen ovale: an open conduit between the paired atria, or through the ductus arteriosus : a shunt between the pulmonary artery and the aorta. When the lungs expand at birth, the pulmonary pressure drops and blood is drawn from the right atrium into the right ventricle and through the pulmonary circuit. Over the course of several months, the foramen ovale closes, leaving a shallow depression known as the fossa ovalis.

Clinical significance

A number of medical conditions can affect the pulmonary circulation. Pulmonary hypertension describes an increase in resistance in the pulmonary arteries. Pulmonary embolus is a blood clot , usually from a deep vein thrombosis that has lodged in the pulmonary vasculature. It can cause difficulty breathing or chest pain, is usually diagnosed through a CT pulmonary angiography or V/Q scan, and is often treated with anticoagulants such as heparin and warfarin.

Circle of willis: The Circle of Willis is the joining area of several arteries at the bottom (inferior) side of the brain. At the Circle of Willis, the internal carotid arteries branch into smaller arteries that supply oxygenated blood to over 80% of the cerebrum.

The circle of willis is an anastomotic system of arteries that sits at the base of brain. It is also a circulatory anastomosis that supplies blood base to the brain and surrounding structures. The structure encircles the middle area of the brain, including the stalk of the pituitary gland. The circle of Willis is a ring of interconnecting arteries located at the base of the brain around the optic chiasm or chiasma (partial crossing of the optic nerve – CNII; this crossing is important for binocular vision), infundibulum of the pituitary stalk and the hypothalamus. This arterial ring provides blood to the brain and neighbouring structures. Polygonal anastomotic shape offers the possibility of alternate pathways for the blood flow, which is essential for the brain functioning, since it is the structure that is mostly sensitive to hypoxia. Hypoxia of the brain tissue that lasts longer than 6 minutes results with the irreversible changes in the brain parenchyma, and depending on the location of the lesion, the functional damages vary widely. Anastomosis between the anterior and posterior circulation. Provides arterial branches that vascularize the brain.

Anterior circulation

Common carotid -> internal carotid -> anterior cerebral artery (-> anterior communicating artery), middle cerebral artery

Posterior circulation

Subclavian arteries -> vertebral arteries -> unite forming the basilar artery -> anterior inferior cerebellar, superior cerebellar, posterior cerebral arteries(->posterior communicating artery)

Circle of Willis

Polygonal anastomosis between:

Internal carotid artery (branch of the common carotid)

Anterior cerebral artery (branch of the internal carotid)

Anterior communicating artery (branch of the anterior carotid, connects left and right anterior cerebral arteries)

Posterior cerebral artery (branch of the basilar artery)

Posterior communicating artery (branch of the posterior cerebral, connects the three cerebral arteries on the same side)

Functions: The circle of willis plays an important role, as it allows for proper blood flow from the arteries to both the front and back hemispheres of the brain. The arteries that stem off from the arteries that stem off from the circle of willis supply much of the blood to the brain.

Clinical significance

Thrombosis, occlusion, aneurysm, rupture, infarction, ischemic attack, cerebral hemorrhage.

Splanchnic circulation: It is also called Mesenteric Circulation. The splanchnic circulation consists of the blood supply to the gastrointestinal tract, liver, spleen, and pancreas. It consists of two large capillary beds partially in series. The small splanchnic arterial branches supply the capillary beds, and then the efferent venous blood flows into the PV. The splanchnic circulation is composed of gastric, small intestinal, colonic, pancreatic, hepatic, and splenic circulations, arranged in parallel with one another. The three major arteries that supply the splanchnic organs, cellac and superior and inferior mesenteric, give rise to smaller arteries that anastomose extensively. The circulation of some splanchnic organs is complicated by the existence of an intramural circulation. Redistribution of total blood flow between intramural vascular circuits may be as important as total blood flow. Numerous extrinsic and intrinsic factors influence the splanchnic circulation. Extrinsic factors include general hemodynamic conditions of the cardiovascular system, autonomic nervous system, and circulating neurohumoral agents. Intrinsic mechanisms include special properties of the vasculature, local metabolites, intrinsic nerves, paracrine substances, and local hormones. The existence of a multiplicity of regulatory mechanisms provides overlapping controls and restricts radical changes in tissue perfusion.

Coronary circulation: Is the circulation of blood in the blood vessels that supply the heart muscle (myocardium). Coronary arteries supply oxygenated blood to the heart muscle and cardiac veins drain away the blood once it has been deoxygenated. Coronary circulation, part of systemic circulatory system that supplies blood to and provides drainage from the tissues of the heart. In the human heart, two coronary arteries arise from the aorta just beyond the semilunar valves; during diastole, the increased aortic pressure above the valves forces blood into the coronary arteries and thence into the musculature of the heart. Deoxygenated blood is returned to the chambers of the heart via coronary veins; most of these converge to form the coronary venous sinus, which drains into the right atrium. The heart normally extracts 70 to 75 percent of the available oxygen from the blood in coronary circulation, which is much more than the amount extracted by other organs from their circulations—e.g., 40 percent by resting skeletal muscle and 20 percent by the liver. Obstruction of a coronary artery, depriving the heart tissue of oxygen-rich blood, leads to death of part of the heart muscle (myocardial infarction) in severe cases, and total heart failure and death may ensue. The coronary circulation is that which supplies the heart muscle itself. It is of crucial importance in

striated muscle in the human heart

human cardiovascular system: Wall of the heart

Coronary vessels supplying arterial blood to the heart penetrate the epicardium before entering the myocardium heart.

Cutaneous circulation: The cutaneous circulation is the circulation and the blood supply of the skin. The skin is not a very metabolically active tissue and has relatively small energy requirements, so its blood supply is different to that of other tissues. The cutaneous circulation, particularly in the hands and feet,

has been subjected to extensive investigation, using a variety of techniques. This shows that humoral and neural influences which operate elsewhere in the body control skin blood flow, the latter being mediated- through the sympathetic nervous system. Apart from its nutritive function, the circulation in skin is involved in the control of systemic blood pressure and, probably more important, plays an essential role in man's thermal homeostasis.

2. Discuss the cardiovascular adjustment that occurs during exercise?

   During exercise, more blood is sent to the active skeletal muscles, and, as body temperature increases, more blood is sent to the skin. This process is accomplished both by the increase in cardiac output and by the redistribution of blood flow away from areas of low demand, such as the splanchnic organs. During exercise, increases in cardiac stroke volume and heart rate raise cardiac output, which coupled with a transient increase in systemic vascular resistance, elevate mean arterial blood pressure (60). However, long-term exercise can promote a net reduction in blood pressure at rest. With exercise, there are increases in metabolic rate, heart rate, blood flow (hyperemia), respiration, and heat production.

The integrated response to severe exercise involves fourfold to fivefold increases in cardiac output, which are due primarily to increases in cardiac rate and to a lesser extent to augmentation of stroke volume. The increase in stroke volume is partly due to an increase in end-diastolic cardiac size (Frank-Starling mechanism) and secondarily due to a reduction in end-systolic cardiac size. The full role of the Frank-Starling mechanism is masked by the concomitant tachycardia. The reduction in end-systolic dimensions can be related to increased contractility, mediated by beta adrenergic stimulation. Beta adrenergic blockade prevents the inotropic response, the decrease in end-systolic dimensions, and approximately 50% of the tachycardia of exercise. The enhanced cardiac output is distributed preferentially to the exercising muscles including the heart. Blood flow to the heart increases fourfold to fivefold as well, mainly reflecting the augmented metabolic requirements of the myocardium due to near maximal increases in cardiac rate and contractility. Blood flow to the inactive viscera (e.g., kidney and gastrointestinal tract) is maintained during severe exercise in the normal dog. It is suggested that local autoregulatory mechanisms are responsible for maintained visceral flow in the face of neural and hormonal autonomic drive, which acts to constrict renal and mesenteric vessels and to reduce blood flow. However, in the presence of circulatory impairment, where oxygen delivery to the exercising muscles is impaired as occurs to complete heart block where normal heart rate increases during exercise are prevented, or in congestive right heart failure, where normal stroke volume increases during exercise are impaired, or in the presence of severe anemia, where oxygen-carrying capacity of the blood is limited, visceral blood flows are reduced drastically and blood is diverted to the exercising musculature. Thus,, visceral flow is normally maintained during severe exercise as long as all other compensatory mechanisms remain intact. However, when any other compensatory mechanism is disrupted (even the elimination of splenic reserve in the dog), reduction and diversion of visceral flow occur.

The changes which occurs in cardiovascular system during exercise are:

I. Heart and Exercise:

Prolonged and systematic exercise causes enlargement of the heart, and this is happens only to cope with the excessive work load imposed upon the heart during work. There is a lot of misunderstanding that prolonged exercise may cause dilatation of the heart similar to that happens in heart disease. But the hypertrophy of the heart in athletes is caused by physiological processes.

II. Heart Rate Changes during exercise :

The acceleration of the heart is observed immediately following exercise. It has been observed that the heart rater is increased slightly even before onset of exercise and it is presumably due to influence of the cerebral cortex on the medullary cardiac centre. A short rise of heart rate is observed at first minute of exercise but after that this rate of rise is slight decreased.

Within 4 to 5 minutes of exercise the maximal rise is more or less achieved. A ‘plateau’ is observed if the exercise is further continued. But the time is variable from individual to individual and even with different degrees of work load. In athletes, the rate of rise of the heart will be slower.

Besides these, maximal heart rate that is reached during exercise and the rapidity with which the maximal value is attained depends upon several factors which are:

(a) Emotional,

(b) Environmental temperature and humidity, and

(c) Physical conditions of the subjects.

There is no satisfactory explanation of the increase of heart rate in man during exercise. The explanation is mostly based on the animal experimentation. It is claimed that both nervous and chemical factors are playing in such process. Initial rise of heart rate (anticipatory heart rate) just before exercise is due to the influence of cerebral cortex and other higher brain centres.

With the onset of exercise the rise of heart rate may be due to:

(a) Reflexes originating in the receptors of moving joints or contracting muscle,

(b) Stimulation of chemoreceptors in muscles by the acid metabolites,

(c) Sympathetico-adrenal activation causing section of much larger amounts of epinephrine in the blood,

(d) Rise of body temperature, and

(e) Stimulation of stretch receptors in atrium by the rapid venous return in heart thus causing Bainbridge reflex.

There is controversial opinion regarding the Bainbridge reflex. None does believe that the increase of heart rate during exercise is due to the effect of such reflex, because during the right atrial pressure does not rise and if it is so then instead of rise there is possibility of increase of heart rate.

Regarding the return of heart rate to initial resting level depends upon the intensity of work load and also on the physical condition of the individual. The rapidity with which the heart rate returns to the resting level following cessation of exercise is considered as a test for physical fitness. In trained individual or in physically fit person the recovery period is very short.

III. Cardiac Output:

During exercise the cardiac output is greatly increased. In trained athletes, it may achieve a maximal output of 30 litres per minute, at an O2 uptake of 4 litres per minute but in non-athletes, the output may be average 22 litres at an O2 uptake of 3.3 litres per minute. The exercise in cardiac output during exercise is the result of the increase in stroke volume and heart rate.

It has been claimed for a long time that the increased stroke volume during exercise is due to functioning of Starling law of heart. But Starling law of heart cannot hold good because modern technique claims that the diastolic size of the heart is not increased during exercise. Instead, the diastolic size of the heart is decreased during exercise so that the increased stroke volume cannot be caused by greater stretching.

Besides this, Rushmer (1959) has claimed that the increased cardiac output during exercise does not necessarily involve increase in stroke volume and heart rate. He claimed that the stroke volume during exercise is increased no doubt, but by about the same amount on changing from standing to the supine position. He claimed that increase in cardiac output is mostly due to increase of heart rate.

IV. Venous Return:

Venous return is greatly increased during exercise for the following reason:

(a) Milking or Massaging Action of Skeletal Muscles:

During exercise, the alternate contraction and relaxation of the muscle act as a booster pump for flowing blood towards the heart. Due to presence of valves in the veins, the blood is squeezed out from the vein towards the heart during contraction and allowed to fill blood during relaxation of the muscle. This pumping mechanism depends upon intensity and type of exercise

(b) Respiratory Movements:

Respiratory movements exert a sucking effect over the right heart and great veins so that greater venous return may occur. Visa fronte is the consequence causing of the above effect during respiratory effort. During inspiration the thoracic cavity is enlarged causing fall of intrathoracic pressure. This fall of intrathoracic pressure as well as increase of pressure on the anterior abdominal wall due to descent of diaphragm cause rapid return of blood into the heart. Expiration has got the opposite effect, and

(c) Contraction of Limb Veins:

It is claimed that limb veins undergo reflex vasoconstriction during exercise thus facilitating rapid venous return to the heart.

V. Blood Pressure:

Blood pressure is raised with the onset of exercise. There may be an anticipatory blood pressure due to nerve impulses originating from the cerebral cortex to the medullary cardiac and vasoconstrictor centres. Other factors that may participate in the rise of blood pressure during exercise are due to activation of sympathetico adrenal systems causing shifting of blood from the splanchnic beds to the other parts of the body.

So the rise of arterial blood pressure during exercise is due to:

(a) Increase of cardiac output, causing greater distention of aorta and large arteries,

(b) Increase of heart rate, and

(c) Compensatory vasoconstriction in the non-active organs (splanchnic beds and skin) and vasodilatation in the active organs so as to perfuse the active organs with a greater pressure.

The nature of blood pressure rise cannot be generalised because the pressure changes mostly depend upon the type, speed and duration of the activity and also of the physical condition of the subject.

VI. Circulatory Status During Exercise:

During exercise, the circulation is adjusted in such a way that the active muscles as well as the vital organs get blood supply to a greater proportion than that of the inactive organs and the non-vital organs. It has been observed that the active muscle gets more blood supply during exercise and the circulation is increased more than about 30 times

It is claimed that this greater supply is due to decrease of vascular resistance caused by locally accumulated metabolites. During exercise sudden lack of O_2 caused the increased accumulation of CO_2, lactic acid, adenosine, intracellular K^+ and histamine. These substances may cause hyperaemia (reactive hyperaemia) and thus the resistance to blood flow is decreased.

As the work load of the heart is increased tremendously during exercise, the coronary flow is increased accordingly to its own nourishment, otherwise hypoxia may prevail. So in moderate exercise, coronary flow is increased according to the O_2 requirement of the cardiac muscle. But in severe exercise, the coronary flow may be increased no doubt, but the cardiac muscle due to tremendous increase of heart rate, will fail to maintain its O_2 according to its need and the subject may feel anginal pain.

Pulmonary circulation during exercise is increased in proportion to the increase in venous return to the heart. But with the increase of pulmonary circulation, the pulmonary arterial pressure is insignificantly

increased possibly due to distensibility of its blood vessels. Blood flow to the brain is relatively under normal state and remains mostly unaltered during exercise.

During exercise the blood flow in the active muscle, lung, heart is increased, but the same in the abdominal organ, kidneys and in the skin; (initially) is greatly decreased due to compensatory vasoconstriction. This happens possibly through the chemoreceptor reflex initiated by the accumulated metabolites during exercise so as to cause redistribution of blood from abdominal organs to the exercising muscle, heart, lung and skin (later stage). Skin blood flow is initially decreased but as the work is continued and the body temperature is increased the skin blood flow is also increased only to eliminate excess heat produced by the contracting muscle.