HUMAN ANATOMY AND PHYSIOLOGY-1(BP101T)

UNIT: 5 CARDIOVASCULAR SYSTEM

CLASS: 7

TOPIC: DISORDERS OF HEART

1. HYPERTENSION: The term hypertension is used to describe blood pressure that is sustained at a higher than the generally accepted 'normal' maximum level for a particular age group, e.g.:

• At 20years-140/90mmhg

• At 50years-160/95mmhg

• At 75 years -170/105 mmhg

Def: hypertension is defined as the persistant raise in blood pressure. Clinically when the systolic blood pressure above 150 mm of Hg. and diastolic pressure elevated above 90 mm of Hg it is considered as hypertension. Types of hypertension:

1) Primary hypertension

2) Secondary hypertension

1) Primary hypertension: It is also called as Essential hypertension.

• This means hypertension of unknown cause.

• It accounts for 85 to 90% of all cases and is subdivided according to the rate at which the disease progresses.

a) **Benign (chronic) hypertension** The rise in blood pressure is usually slight to moderate and continues to rise slowly over many years. Sometimes complications are the first indication of hypertension, e.g. heart failure, cerebrovascular accident, myocardial infarction. Occasionally the rate of progress increases and the hypertension becomes malignant.

Predisposing factors include:

- inherited tendency
- obesity
- excessive alcohol intake
- cigarette smoking
- lack of exercise.

b) Malignant (accelerated) hypertension • The blood pressure is already elevated and continues to rise rapidly over a few months. Diastolic pressure in excess of 120 mmHg is common. The effects are serious and quickly become apparent,

• E.g. haemorrhages into the retina, papilloedema (oedema around the optic disc), encephalopathy (cerebral oedema) and progressive renal disease, leading to cardiac failure.

Secondary hypertension Hypertension resulting from other diseases accounts for 10 to 15% of all cases. a) Kidney diseases/renal hypertension: Raised blood pressure is a complication of many kidney diseases. Those are:

i) Stenosis of renal arteries

ii) Tumors of juxta glomerular cells leading to formation of extra angiotension- ii

iii) Glomerulonephritis

b) Endocrine disorders/endocrine hypertension:

i. Pheochromocytoma: tumors in adrenal gland

ii. Hyperaldosteronism: excess secretion of aldosterone from adrenal cortex

iii. Cushings's syndrome: excess secretion of glucocorticoid from adrenal cortex.

c) Structure of the aorta/cardio vascular hypertension:

i) Coarchidation of aorta: Hypertension develops in branching arteries proximal to the site of a stricture. In congenital coarctation the stricture is between the ductus arteriosus and the left subclavian artery causing hypertension in the head, neck and right arm.

ii) Atherosclerosis

3) Drugs:

Hypertension may be a complication of some drug treatment, e.g.: • corticosteroids • non-steroidal anti-inflammatory drugs • oral contraceptives.

1) **Neurogenic hypertension:** the nerve disorder produce hypertension. i) Lesions in nerve cell ii) Sectioning of nerve fibre.
2) **Hypertension during pregnancy:** some pregnant women develop hypertension because of toxemia of pregnancy.

Effects and complications of hypertension: Heart • The rate and force of cardiac contraction are increased. • This is followed by back pressure and accumulation of blood in the lungs (pulmonary congestion), hypertrophy of the right ventricle and eventually to right ventricular failure. Hypertension also predisposes to ischaemic heart disease and aneurysm formation, Stroke, caused by cerebral haemorrhage, is common, the effects depending on the position and size of the ruptured vessel. When a series of small blood vessels rupture, • e.g. microaneurysms, at different times, there is progressive

disability. Rupture of a large vessel causes extensive loss of function or possibly death.

Hypertensive encephalopathy:

Hypertensive encephalopathy is a rare condition in which hypertension is accompanied by neurological disturbance, e.g. papilloedema, difficulty with speech, paraesthesia, convulsions and loss of consciousness. It is usually reversed when hypertension is controlled.

Kidneys

• Essential hypertension causes kidney damage. If sustained for only a short time recovery may be complete. Otherwise the kidney damage causes further hypertension owing to activation of the renin-angiotensin-aldosterone system.

• progressive loss of kidney function and kidney failure.

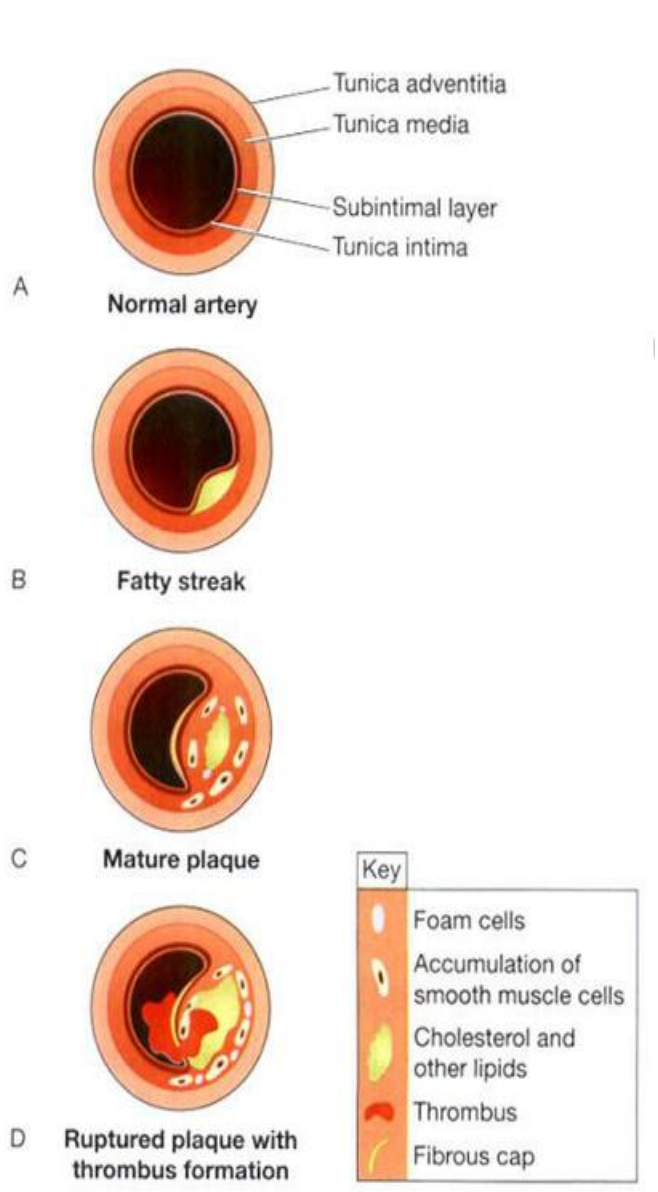
Pulmonary hypertension Raised blood pressure in the pulmonary circulation is secondary to: changes in blood vessels, described above chronic diseases of the respiratory system diseases of the heart, e.g. congenital defects of the septum, stenosis and incompetence of the mitral or aortic valve, heart failure diseases of other organs that cause raised pressure in the left side of the heart, e.g. cirrhosis of the liver, thrombosis of the portal vein.

2. ATHEROSCLEROSIS:

Atheroma Formation: Pathological changes Patchy changes (atheromatous plaques) develop in the tunica intima of large and medium-sized arteries. Thesconsist of accumulations of cholesterol and other lipid compounds, excess smooth muscle and fat-filled monocytes (foam cells). The plaque is covered with a fibrous cap. As plaques grow they spread along the artery wall forming swellings that protrude into the lumen. Eventually the whole thickness of the wall and long sections of the vessel

may be affected Plaques may rupture, exposing subintimal materials to the blood. This may cause thrombosis and vasospasm and will compromise blood flow. Arteries most commonly involved are those in the heart, brain, kidneys, small intestine and lower limbs.

Causes of atheroma Fatty streaks: The origin of atheromatous plaques is uncertain. Fatty streaks present in artery walls of infants are usually absorbed but their incomplete absorption may be the origin of atheromatous plaques in later life. Age: Atherosclerosis is considered to be a disease of older people because it is usually in these age groups that clinical signs appear. Plaques, however, start to form in childhood in developed countries. The incidence of atheroma is widespread in developed countries. Why atheromatous plaques develop is not yet clearly understood but the predisposing factors appear to exert their effects over a long period. This may mean that the development of atheroma can be delayed or even arrested by a change in lifestyle.



<u>Predisposing factors include:</u>

• Heredity — family history

• Gender — males are more susceptible than females until after the menopause

• increasing age

• Hypertension

• Diabetes mellitus

• Smoking, especially cigarettes

• Excessive emotional stress in work or home environment

• Diet, e.g. high intake of refined carbohydrates and/or cholesterol and saturated fatty acids (from animal fats)

• Obesity

• Sedentary lifestyle

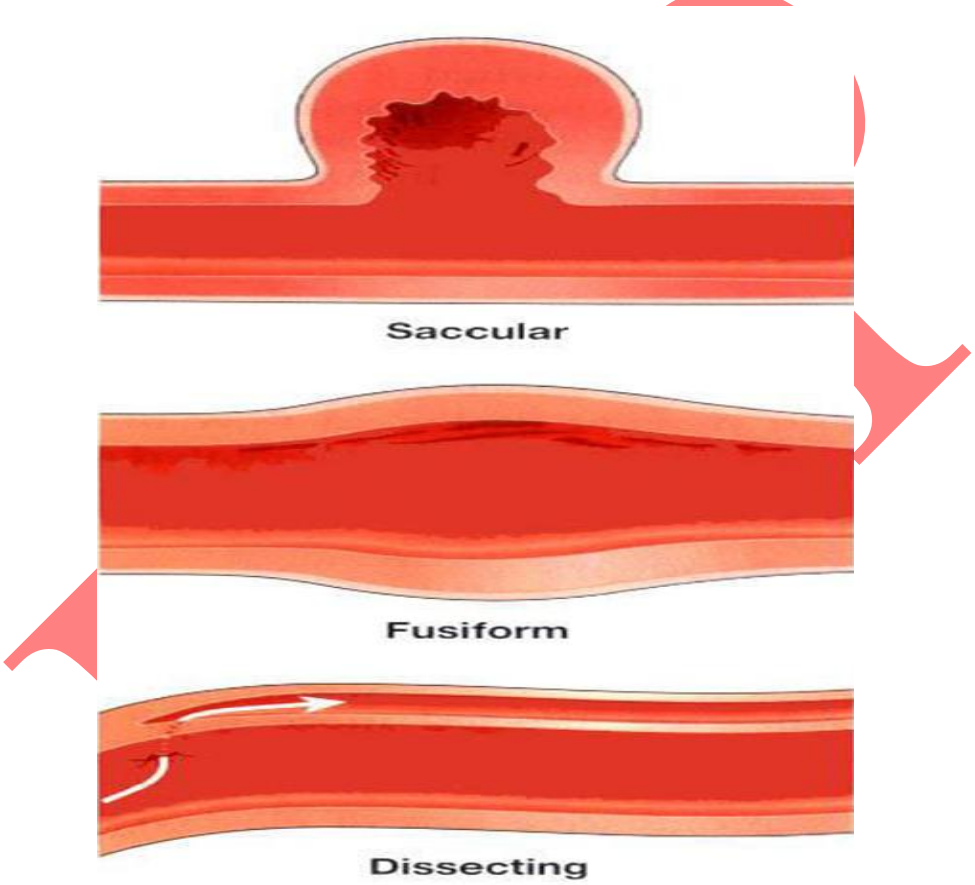
• Excessive alcohol consumption.

Effects of atheroma: Arteries may be partially or completely blocked by atheromatous plaques alone, or by plaques combined with a thrombus. This may reduce or completely block the blood supply. The effects depend on the site and size of the artery involved and the extent of collateral circulation. Commonly the arteries affected are those in the heart, abdomen and pelvis. 1) Narrowing of an artery The tissues distal to the narrow point become ischaemic. The cells may receive enough blood to meet their minimum needs, but not enough to cope with an increase in metabolic rate, e.g. when muscle activity is increased.This causes acute cramp like ischaemic pain. Cardiac muscle and skeletal muscles of the lower limb are most commonly affected. Ischaemic pain in the heart is called angina pectoris and in the lower limbs, intermittent claudication. 2) Occlusion of an artery When an artery is completely blocked, the tissues it supplies rapidly undergo degeneration and die from ischaemia which leads to infarction. The extent of tissue damage depends on: • The size of the artery occluded • The amount and type of tissue involved • The extent of collateral circulation, e.g. in the brain the circulus arteriosus (circle of Willis) provides extensive collateral blood vessels while in the heart there are very

few. When a coronary artery is occluded myocardial infarction occurs. Occlusion of arteries in the brain causes cerebral ischaemia and this leads to cerebral infarction (stroke).

Complications of atheroma: 1) Thrombosis and infarction If the fibrous cap overlying a plaque breaks down, platelets are activated by the damaged cells and a blood clot (thrombus) forms, blocking the artery and causing ischaemia and infarction. Pieces of the clot (emboli) may break off, travel in the bloodstream and lodge in small arteries distal to the clot, causing small infarcts (areas of dead tissue). 2) Haemorrhage When calcium salts are deposited in the plaques, the artery walls become brittle, rigid and unresponsive to rises in blood pressure and may rupture, causing haemorrhage. 3) Aneurysm formation When the arterial wall is weakened by spread of the plaque between the layers of tissue, a local dilatation (aneurysm) may develop (see below). This may lead to thrombosis and embolism, or the aneurysm may rupture causing severe haemorrhage. The most common sites affected are the aorta and the abdominal and pelvic arteries.

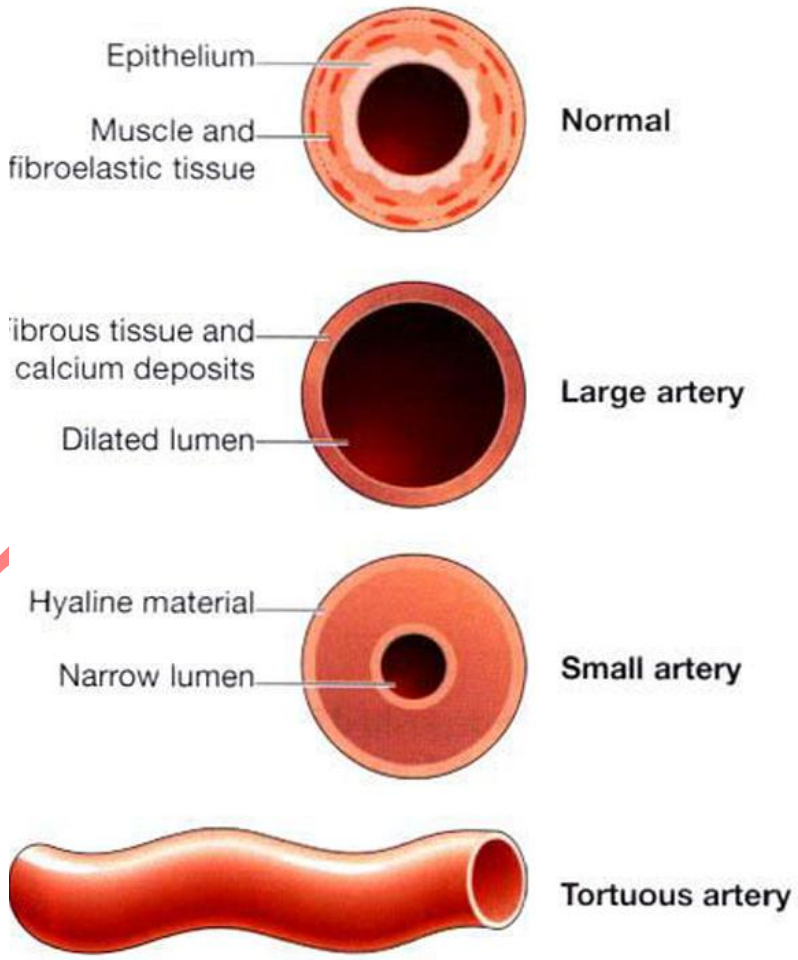
Complications of aneurysms a) Haemorrhage b) Pressure A ruptured aneurysm may cause sudden death or disability of varying severity, depending on the size and site of the artery. Localised swelling may cause pressure affecting adjacent tissues including organs, blood vessels and nerves. c) Thrombosis and embolism A blood clot (thrombus) may form in an artery where the endothelium has been damaged by an aneurysm. A piece of clot (embolus) may break off and travel in the bloodstream



until it lodges in a small artery distal to the aneurysm and obstructs the blood flow, causing ischaemia and infarction.

ARTERIOSCLEROSIS: This is a progressive degeneration of arterial walls, associated with ageing and accompanied by hypertension. Large and medium arteries The tunica media is infiltrated with fibrous tissue and calcium. This causes the vessels to lose their elasticity. The lumen dilates and they become tortuous. Loss of elasticity increases systolic blood pressure, and the pulse pressure (the difference

between systolic and diastolic pressure). Small arteries and arterioles Hyaline thickening of the tunica media and tunica intima causes narrowing of the lumen and they become tortuous These arteries are the main determinants of peripheral resistance and narrowing of their lumens increases peripheral resistance and blood pressure. Ischaemia of tissues supplied by affected arteries may occur. In the limbs, the resultant Ischaemia predisposes to gangrene which is particularly serious in people with diabetes mellitus.



Senile arteriosclerosis. This is a condition affecting elderly people in which the progressive loss of elasticity and reduced arterial lumen leads to cerebral ischaemia and loss of mental function. There may or may not be evidence of hypertension.

CARDIAC FAILURE:

❖ The heart is described as failing when the cardiac output is unable to maintain the circulation of sufficient blood to meet the needs of the body. In mild cases, cardiac output is adequate at rest and becomes inadequate only when increased cardiac output is required, e.g. in exercise.

❖ Heart failure may affect either side of the heart, but since both sides of the heart are part of one circuit, when one half of the pump begins to fail it frequently leads to increased strain on, and eventual failure of, the other half. The main clinical manifestations depend on which side of the heart is most affected.

COMPENSATORY MECHANISMS IN HEART FAILURE: When heart failure happens acutely, the body has little time to make compensatory changes, but if the heart fails over a period of time the following changes are likely to occur in an attempt to maintain cardiac output and tissue perfusion, especially of vital organs.

The cardiac muscle fibres enlarge and increase in number, which makes the walls of the chambers thicker the heart chambers enlarge decreased renal blood flow activates the renin angiotensin aldosterone system, which leads to salt and water retention. This increases blood volume and cardiac workload. The direct vasoconstrictor action of angiotensin 2 increases peripheral resistance and puts further strain on the failing heart.

Acute cardiac failure:

A sudden decrease in output of blood from both ventricles causes acute reduction in the oxygen supply to all the tissues. Recovery from the acute phase may be followed by chronic failure, or death may occur due to anoxia of vital centres in the brain.

The commonest causes are:

❖ severe damage to an area of cardiac muscle due to ischaemia caused by sudden occlusion of one of the larger coronary arteries by atheroma or atheroma with thrombosis pulmonary embolism

❖ acute toxic myocarditis

❖ severe cardiac arrhythmia

❖ rupture of a heart chamber or valve cusp

❖ severe malignant hypertension. Chronic cardiac failure This develops gradually and in the early stages there may be no symptoms because certain compensatory changes occur as described above. When further compensation is not possible there is a gradual decline in myocardial efficiency. causes include:

• Chronic hypertension, myocardial fibrosis, valvular disease, lung diseases, anaemia

• Previous acute cardiac failure

• Degenerative changes of old age.

Types of cardiac failure: 1) Right-sided (congestive) cardiac failure The right ventricle fails when pressure developed within it by the contracting myocardium is less than the force needed to push blood through the lungs. When compensation has

reached its limit, and the ventricle is not emptying completely, the right atrium and venae cavae become congested with blood and this is followed by congestion throughout the venous system. The organs affected first are the liver, spleen and kidneys. Oedema of the limbs and ascites (excess fluid in the peritoneal cavity) usually follow. This problem may be caused by increased vascular resistance in the lungs, weakness of the myocardium and/or stenosis and incompetence of valves in the heart or great vessels. Resistance to blood flow through the lungs When this is increased the right ventricle has more work to do.

It may be caused by: ❖ The formation of fibrous tissue following inflammation or chronic disease of the lungs ❖ Back pressure of blood from the left side of the heart, e.g. in left ventricular failure, when the mitral valve is stenosed and/or incompetent. ❖ Weakness of the myocardium This may be caused by ischaemia following numerous small myocardial infarcts

2) Left-sided or left ventricular failure:

This occurs when the pressure developed in the left ventricle by the contracting myocardium is less than the pressure in the aorta and the ventricle cannot then pump out all the blood it receives. Causes include: • Excessively high systemic (aortic) blood pressure • Incompetence of the mitral and/or the aortic valve • Aortic valve stenosis • Myocardial weakness. ❖ Failure of the left ventricle leads to dilatation of the atrium and an increase in pulmonary blood pressure. ❖ This is followed by a rise in the blood pressure in the right side of the heart and eventually systemic venous congestion. ❖ Congestion in the lungs leads to pulmonary oedema and dyspnoea, often most severe at night. This paroxysmal nocturnal dyspnoea may be due to raised

blood volume as fluid from peripheral oedema is reabsorbed when the patient slips down in bed during sleep.

ISCHAEMIC HEART DISEASE:

❖ Ischaemic heart disease is due to the effects of atheroma, causing narrowing or occlusion of one or more branches of the coronary arteries. The narrowing is caused by atheromatous plaques.

❖ Occlusion may be by plaques alone, or plaques complicated by thrombosis.

❖ The overall effect depends on the size of the coronary artery involved and whether it is narrowed or occluded.

❖ Narrowing of an artery leads to angina pectoris, and occlusion to myocardial infarction, i.e. an area of dead tissue.

❖ When atheroma develops slowly, a collateral arterial blood supply may have time to develop and effectively supplement or replace the original.

❖ This consists of the dilatation of normally occurring anastomotic arteries joining adjacent branch arteries. When sudden severe narrowing or occlusion of an artery occurs the anastomotic arteries dilate but may not be able to supply enough blood to meet the needs of the myocardium. Angina pectoris This is sometimes called angina of effort because increased cardiac output required during extra physical effort causes severe ischaemic pain in the chest. The pain may also radiate to the arms, neck and jaw. Other factors which may precipitate angina include:

• cold weather • exercising after a heavy meal • strong emotions.

❖ A narrowed coronary artery may supply sufficient blood to the myocardium to meet its needs during rest or moderate exercise but not when greatly increased cardiac outputis needed, e.g. walking may be tolerated but not running.

The thick, inflexible atheromatous artery wall is unable to dilate to allow for the increased blood flow needed by the more active myocardium which then becomes ischaemic.

❖ In the early stages of development of the disease the chest pain stops when the cardiac output

❖ Returns to its resting level soon after the extra effort stops.

Myocardial infarction An infarct is an area of tissue that has died because of lackof oxygenated blood. The myocardium is affected when a branch of a coronary artery is occluded.

Common causes:

❖ The commonest cause is an atheromatous plaque complicated by thrombosis.

❖ The extent of myocardial damage depends on the size of the blood vessel and site of the infarct.

❖ The damage is permanent because cardiac muscle cannot regenerate and the dead tissue is replaced with non-functional fibrous tissue.

❖ Speedy restoration of blood flow through the blocked artery using clot-dissolving (thrombolytic)

❖ drugs can greatly reduce the extent of the permanent damage and improve prognosis, but treatment must be started within a few hours of the infarction occurring.

❖ The effects and complications are greatest when the left ventricle is involved. Myocardial infarction is usually accompanied by: Very severe crushing chest pain behind the sternum Which, unlike angina pectoris, continues even when the Individual is at rest.

Complications: These may be fatal and include:

• pericarditis

• angina pectoris

• recurrence

• Severe arrhythmias, especially ventricular fibrillation, due to disruption of the cardiac conducting system

• Cardiac failure, caused by impaired contraction of the damaged myocardium and, in severe cases cardiogenic shock

• rupture of a ventricle wall, usually within 2 weeks of the original episode

• pulmonary or cerebral embolism originating from a mural clot within a ventricle, i.e. a clot that forms inside the heart over the area of dead tissue

CARDIAC ARRHYTHMIAS:

❖ The heart rate is normally initiated by intrinsic impulses generated in the SA node. The rhythm is determined by the route of impulse transmission through the

conducting system. The heart rate is usually measured as the pulse, but to determine the rhythm, an electrocardiogram ❖ (ECG) is required.

❖ A cardiac arrhythmia is any disorder of heart rate or rhythm, and is the result of abnormal generation or conduction of impulses.

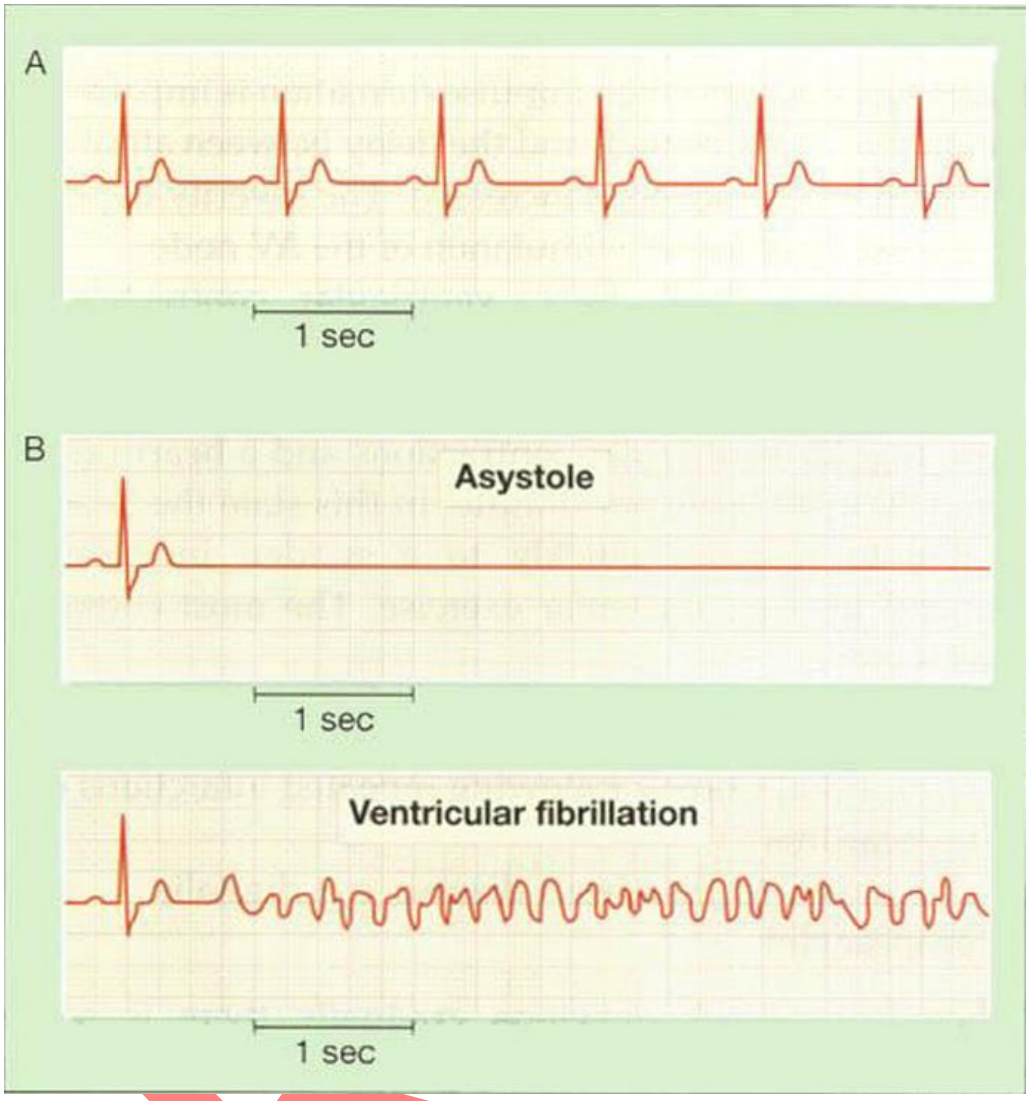
❖ The normal cardiac cycle gives rise to normal sinus rhythm

❖ Which has a rate between 60 and 100 beats per minute.

Abnormal rhythms include: Sinus bradycardia. This is sinus rhythm below 60 beats per minute. This may occur during sleep and is common in athletes. It is an abnormality when it follows myocardial infarction or accompanies raised intracranial pressure.

Sinus tachycardia: This is sinus rhythm above 100 beats per minute when the individual is at rest. This accompanies exercise and anxiety; but is an indicator of some disorders, e.g. fever, hyperthyroidism, some cardiac conditions.

Asystole: This occurs when there is no electrical activity in the ventricles and therefore no cardiac output. The ECG shows a flat line. Ventricular fibrillation and asystole cause sudden and complete loss of cardiac output, i.e. cardiac arrest and death.



Fibrillation This is the contraction of the cardiac muscle fibres in a disorderly sequence. The chambers do not contract as a whole and the pumping action is disrupted. In atrial fibrillation contraction of the atria is uncoordinated and rapid, pumping is ineffective and stimulation of the AV node is disorderly. Ventricular contraction becomes rapid and rhythm and force irregular; although an adequate cardiac output and blood pressure may be maintained, the pulse is irregular. The causes are: Increased excitability and disorganized activity are not always clear but predisposing conditions include: • Ischaemic heart disease • Degenerative changes in the heart due to old age • thyrotoxicosis • rheumatic heart disease. In ventricular

fibrillation there is disorganised and very rapid contraction causing disruption of ventricular function.

Blood is not pumped from the heart into either the pulmonary or the systemic circulation. No pulses can be felt, consciousness is lost and breathing stops. The ECG shows an irregular chaotic trace with no recognizable wave pattern. If normal heart action cannot be restored quickly, death follows due to cerebralanoxia.

HEART BLOCK:

❖ Heart block occurs when impulse formation is Heart block Heart block occurs when impulse formation is impaired or conduction is prevented, and the delay between atrial and ventricular contraction is increased.

❖ The severity depends on the extent of loss of stimulation of the AV node.

❖ In complete heart block, ventricular contraction is entirely independent of impulses initiated by the SA node. Impulses generated by the AV node result in slow, regular ventricular contractions and a heart rate of about 30 to 40 beats per minute. In this state the heart is unable to respond quickly to a sudden increase in demand by, e.g., muscular exercise.

The most common causes are:

• Acute ischaemic heart disease

• Myocardial fibrosis following repeated infarctions or myocarditis

• Drugs used to treat heart disease, e.g. digitalis, propranolol.

When heart block develops gradually there is some degree of adjustment in the body to reduced cardiac output but, if progressive, it eventually leads to death from cardiac failure and cerebral anoxia.