

Cardiovascular system

Cardiac output & Regulation & Measurement

• Usually cardiac output is expressed in three ways

i) Stroke volume :- Stroke volume is the amount of blood pumped out by each ventricle during each beat.

Its normal value is 70 ml when heart rate is normal.

ii) Minute volume :- Amount of blood pumped out by each ventricle in one minute. It is the product of stroke volume & heart rate.

$$\text{Minute volume} = \text{Stroke volume} \times \text{Heart rate}$$

$$\text{Normal value} = 5 \text{ L / ventricle / min}$$

iii) Cardiac index :- Cardiac index is the min volume expressed in relation to square metre of body surface area. It is defined as the amount of blood pumped out per ventricle / min / sq. m of the body surface area.

$$\text{Normal value} : 2.8 \pm 0.3 \text{ L / sq. m of body surface area / min}$$

Ejection fraction :- It is the fraction of end diastolic volume that is ejected by each ventricle.

Cardiac reserve :- Maximum amount of blood that can be pumped out by each ventricle (by heart) above the normal value.

• Cardiac reserve plays an imp role in increasing cardiac output during the conditions like exercise. It is essential to withstand the stress of exercise.

- It is usually expressed in percentage.
- In cardiac diseases, cardiac reserve is min or nil.

Healthy adult - 300% - 400%.

Old age - 200% - 250%.

Athletes - 500% - 600%.

Factors maintaining Cardiac output :-

1. Venous return :- It is the amount of blood which is returned to heart from diff parts of the body.

• When it increases, the ventricular filling & cardiac output are increased.

• The cardiac output is directly proportional to venous return, provided the other factors remain const.

• Venous return in turn depends on 5 factors

i. Respiratory pump $\propto$ Venous return.

ii. Muscle pump $\rightarrow$ muscular activity $\propto$ venous return.

iii. Gravitational force $\propto \frac{1}{\text{Venous return}}$ (Venous pooling)

iv. Venous pressure

v. Sympathetic tone $\rightarrow$ causes vasoconstriction



2. Force of contraction :- Cardiac output is directly proportional to the force of contraction, provided other 3 factors remain const.
- According to Frank-Starling law, force of contraction of heart is directly proportional to initial length of muscle fibres, before onset of contraction.
- Force of contraction depends upon preload & afterload.
- * Force of contraction of heart & cardiac output are directly proportional to preload.
- * Force of contraction of heart & cardiac output are inversely proportional to afterload.

3. Heart Rate :- Cardiac output is directly proportional to heart rate provided other 3 are remain const. Moderate change in heart rate does not alter the cardiac output.

marked increase in heart rate $\rightarrow$ Cardiac output $\uparrow$
 " decrease " " " $\rightarrow$ " " $\downarrow$

4. Peripheral Resistance :- Resistance offered to blood flow at the peripheral blood vessels. So, the cardiac output is inversely proportional to peripheral resistance.

Resistance offered at arterioles $\rightarrow$ arterioles & called resistant vessels. In the body, max peripheral resistance is offered at splanchnic region.

Measurement of Cardiac Output by Using Oxygen Consumption :-

- Fick's principle is used to measure the cardiac output by determining the amount of oxygen consumed in the body in a given period of time & dividing its value by the arteriovenous difference across the lungs.

$$\text{Cardiac Output} = \frac{\text{O}_2 \text{ consumed (in mL/min)}}{\text{Arteriovenous O}_2 \text{ difference.}}$$

- Amount of O₂ consumed is measured by the respirometer or BMR apparatus.
- Oxygen content in arterial blood is determined by collecting blood from any artery. Oxygen content is determined by blood gas analysis.
- Oxygen content in venous blood :- Only mixed venous blood is used to determine the O₂ content of venous blood, since it is diff in diff veins.
- Mixed venous blood is collected from Rt atrium or pulmonary artery. It is done by introducing a catheter through basilic vein of forearm.
- Oxygen is determined in blood by blood gas analysis.

Measurement of cardiac output by using CO_2 :

- Cardiac output is also measured by knowing the arteriovenous diff of CO_2 & amount of CO_2 removed out of lungs.
- Since cardiac output is equal to the amount of blood passing through lungs, the cardiac output = 5 L/min
- Nitrous oxide is also used to measure cardiac output by applying Fick's principle.

Fick's principle:

$$\frac{\text{Amount of substance taken or given by organ in unit time}}{\text{unit time}} = \text{Amount of blood flow / minute} \times \text{Arteriovenous difference.}$$

Advantage of Fick's principle: Result are accurate

Disadvantage: It is an invasive method and involves the insertion of catheter through subject vein.

Cardiac cycle

- Events occurs from beginning of one heart beat to the beginning of next is called cardiac cycle.
- Each cycle is initiated by spontaneous generation of action potentials in SA node.
- The action potentials travels from SA node $\rightarrow$ AV Bundle.
- There is a delay of more than 0.1 sec during passage of cardiac impulse from atria to ventricles, allows atria to contract first.
- Atria - primer pumps for ventricles.

Diastole & Systole:-

Diastole $\rightarrow$ Relaxation - heart fills with blood.

Systole $\rightarrow$ Contraction.

- Duration of cardiac cycle is reciprocal of heart sound / rate = $\frac{1}{72} = 0.8 \text{ sec}$

$$\text{Heart rate} \propto \frac{1}{\text{Duration of Cardiac cycle.}}$$

- In normal resting state of 72 beats/min, during each cardiac cycle systole takes 0.4 sec.

Mechanical Events in Heart:-

a) Ventricular fill with blood during systole:-

- Period of rapid filling of ventricles, lasts for first $\frac{1}{3}^{\text{rd}}$ of diastole.
- During last $\frac{2}{3}^{\text{rd}}$ of ventricular diastole, atria contract & accommodate about 20% filling of ventricles.



b) Outflow of blood from ventricles during systole :-

Isovolumetric contraction :-

- Immediately after ventricular contraction begins, ventricular pressure rises abruptly causes AV valves to close.
- Addition 0.02 - 0.03 seconds are required to develop enough pressure to push semilunar valves open.
- Cardiac muscle tension is increased but little & no shortening of muscle fibres occurs.

Period of ejection :-

- When the L ventricular pressure rises above 80 mm Hg & R ventricular pressure above 8 mm Hg, the semilunar valves are open.
- 60% of blood in ventricles at end of diastole is ejected. At end of systole
- 70% of ejection occurs during $\frac{1}{3}$ rd ejection period - rapid ejection
- 30% " " " " next $\frac{2}{3}$ rd " " - slow ejection.

c) Isovolumetric relaxation :-

- End of systole, ventricles relax causing rapid decrease in ventricular pressure.
- Period during which there is a backflow of blood associated with closure of aortic & semi-lunar valves - protodiastole.
- The ventricular muscles continue to relax without any change in volume for 0.02 to 0.06 sec.

Phases of Cardiac cycle :-

1. Rapid filling of ventricles
2. Slow " " "
3. Atrial systole
4. Isovolumetric contraction
5. Rapid ejection
6. Slow ejection
7. Protodiastole
8. Isovolumetric relaxation.

Electrical Events :-

'P' wave :- Spread of depolarization of atria, followed by atrial contraction, atrial pressure rises.

QRS wave :- Appears after 0.16 sec after onset of depolarisation of ventricles, contraction, ventricular pressure rises, Begins slightly before onset of ventricular systole.

T wave :- Repolarisation of ventricles, occurs before end of ventricular contraction.

Heart Sounds :-

- When ventricles contract, sound is produced due to closure of A-V valves. Vibration pitch is low & long lasting. It is 1st heart sound.
- Sound due to aortic & pulmonary valves closure at end of systole is rapid snap & vibrates for short period. It is 2nd heart sound.
- 3rd heart sound - Turbulence of blood in rapid filling phase
- 4th heart sound - " " " during atrial systole.

Efficiency of Cardiac contraction :-

- The ratio of work output to total chemical energy expenditure is efficiency of cardiac contraction.

Max efficiency $\rightarrow$ 20-25%.

Heart failure $\rightarrow$ 5-10%.

Cardiac Murmurs :-

- Cardiac murmur is the abnormal or unusual heart sound. It is also called abnormal heart sound or cardiac bruit.
- Cardiac murmur is heard by stethoscope, along with normal heart sounds.
- Exactly cardiac murmur is heard by placing a chest piece of stethoscope over the auscultatory areas.
- Murmur due to disease of particular valve is heard well over the auscultatory area of that valve.
- Sometimes the murmur is felt by palpation as "thrills". In some patients murmur is heard without any aid, even at a dist of few feet away from the patient.

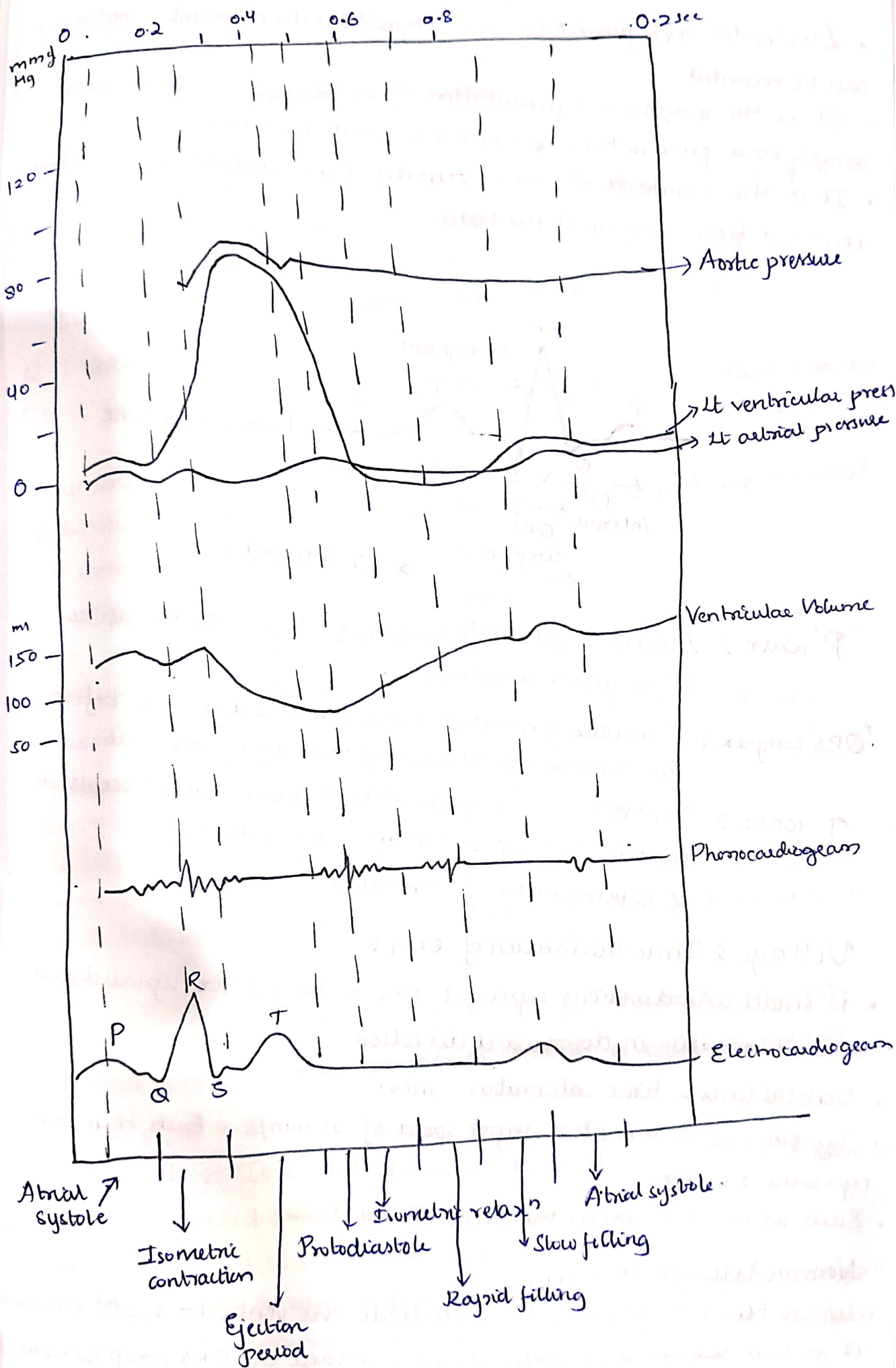
Systolic murmur :- Incompetence of AV valves, Stenosis of semilunar valves, anemia, septal defect, narrowing of aorta

Diastolic murmur :- Stenosis of AV valves

Incompetence of Semilunar valves.

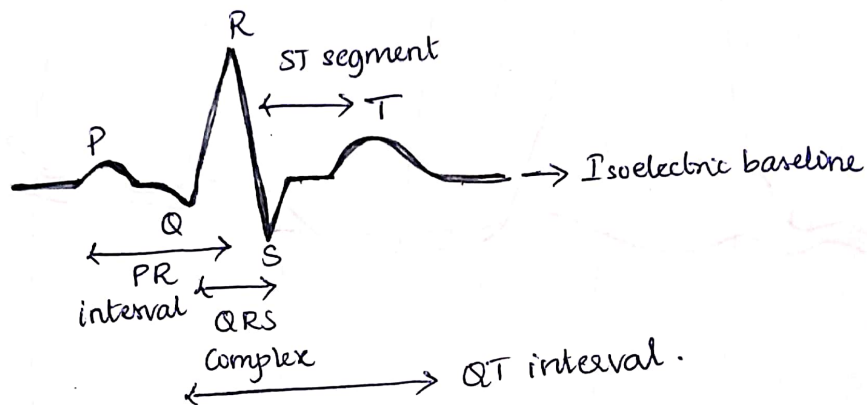
Continuous murmur :- Patent ductus arteriosus.





ECG

- Electrodes are placed on skin opposite sides of heart, potential can be recorded.
- It is the graphical representation of electrical activities of heart which occur prior to the onset of mechanical activities.
- It is the summed electrical activity of all cardiac muscle fibres recorded from surface of the body.



'P' wave :- Electrical potentials generated when atria depolarize before atrial contraction.

'QRS' complex :- Potentials generated when ventricles depolarize before contraction. Both are depolarising waves.

'T' wave :- Ventricles recover from state of depolarisation occurs in muscle 0.25 - 0.35 sec after depolarisation.
(Repolarisation of ventricles)

Voltage & Time Calibration of ECG :-

- 10 small line divisions represent 1mv. with positive upward direction and negative in downward direction.
- Vertical lines - time calibration lines.
- Typical ECG is run at a paper speed of 25 mm/s. Each thin line represent 0.04 sec.
- Each 25 mm in horizontal direction is 1 sec.

Normal Voltage in ECG :-

- When ECG is placed by 1 electrode over ventricles & 2nd electrode is placed elsewhere on body remote, voltage of QRS complex may be as great as 3-4 mv.

- When ECG's are recorded by placing electrodes on 2 arms or 1 arm & 1 leg, the voltage of QRS is $1 - 1.5 \text{ mV}$
P wave is b/w $0.1 - 0.3 \text{ mV}$
T wave is b/w $0.2 - 0.3 \text{ mV}$

P-Q interval :-

- Time b/w beginning of P wave & beginning of QRS complex is P-Q interval. Normally about 0.16 sec .

It shortens at faster heart rates - Sympathetic effect

It lengthens at slower heart rates - Para " "

Q-T interval :- Contraction of ventricles

It is from beginning of Q-wave to end of T-wave. About 0.35 sec .

Augmented Unipolar limb leads :-

- 2 limbs are connected through electrical resistance to the terminal
- 3rd limb is connected to -ve terminal.

+ve terminal on Rt arm $\rightarrow$ VR lead

+ve " of Lt arm $\rightarrow$ VL lead

-ve " of Lt leg $\rightarrow$ VF lead.

- Recordings from aVR are inverted compared to standard ECG.

Feature :-

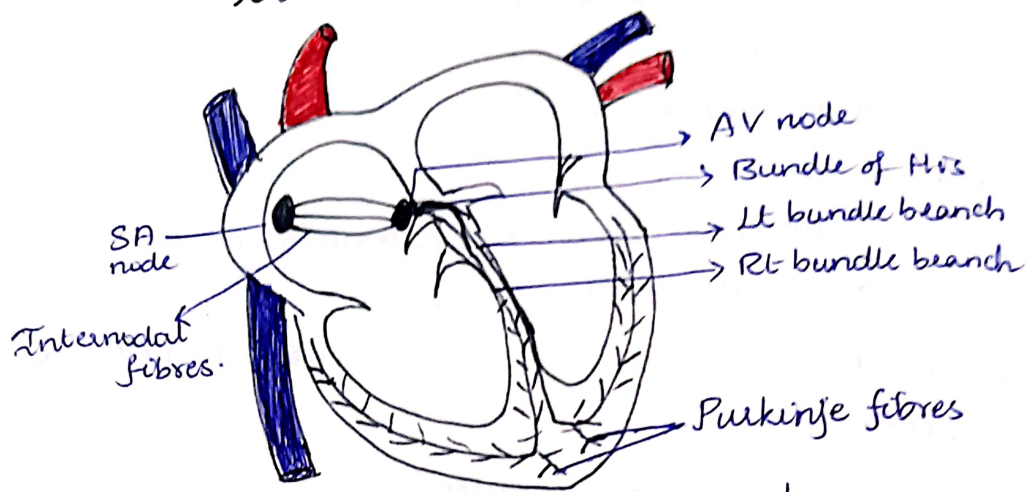
- Record electrical activity of heart in frontal plane.
- Unipolar
- Along with bipolar leads, they form hexagonal reference systems imp to determine direction of cardiac vectors & cardiac axis in frontal plane.

Enthoven's Law :- States that in ECG for standard limb leads potential of any wave or complex in lead II is equal to sum of potentials of I & III.

Enthoven's Δ :- It is defined as an equilateral Δ that is used as a model of standard limb leads to record electrocardiogram.

- Heart is presumed to lie on the centre of Enthoven Δ .
- Electrical potential generated from the heart appears simultaneously on the wires of 3 limbs, namely Lt arm, Rt arm & Lt leg. ECG is recorded on 12 leads, which are classified into 2 categories.

i) Bipolar leads ii) Unipolar leads.



Conducting system of heart & SA node.

- Conducting system of the heart is formed by the modified cardiac muscle fibres.
- These fibres are specialized cells, which conduct the impulses rapidly from SA node to ventricles.
- Conductive tissues of the heart are also called junctional tissues.

Components :-

1. AV node
2. Bundle of His
3. Rt & Lt bundle branches
4. Purkinje fibres.
5. SA node.

- Sino-atrial node is situated in Rt atrium, just below the opening of Superior Vena cava.
- AV node is situated in Rt post portion of intra-atrial septum.

Pathway :-

Action potential is initiated in the SA node



Action potentials are conducted from the SA node to the atrial muscle



Action potentials spread through the atria to the AV node, where conduction slows.



Action potentials travel rapidly through the

interventricular septum to the apex of the heart. from Bundle of His

↓
Impulses spread upwards through the ventricular muscle by Purkinjee fibres.

↓
The entire heart returns to the resting state, remaining there until another action potential is generated on SA node.

Velocity of Impulses at diff parts of Conducting system :-

1. Atrial muscle fibres - 0.3 m/s
2. Internodal " - 1.0 m/s
3. AV node - 0.05 m/s
4. Bundle of His - 0.12 m/s
5. Purkinjee fibres - 4.0 m/s
6. Ventricular muscle fibres - 0.5 m/s

Thus, the velocity of impulses is max in purkinjee fibres & min at AV node.

- Normal duration of 'R-R' interval is 0.18 sec & varies b/w 0.12 to 0.2 sec. If it is more than 0.2 s, it signifies the delay in the conduction of impulse from SA node to ventricles.
- Usually, the delay occurs in the AV node. So it is called AV nodal delay.

Clinical Significance :-

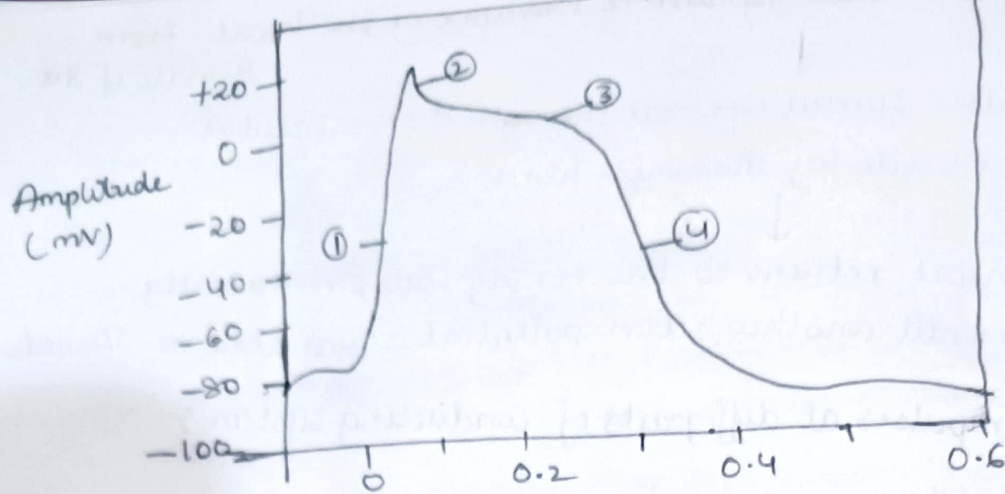
- Variation in the duration of 'P-R' intervals helps in diagnosis of several cardiac problems such as :
 1. It is prolonged in bradycardia & 1st degree heart block.
 2. It is shortened in tachycardia, Wolf Parkinson white syndrome, Duchene muscular dystrophy, & type II glycogen storage disease.

Cardiac action potential :-

Phases of Cardiac action potential :

Action potential in single cardiac muscle fibres occurs in 4 phases

1. Initial depolarisation
2. " repolarisation
3. A plateau of final depolarisation.
4. Final repolarisation



① Initial Depolarisation :-

- Initial depolarisation is very rapid & it lasts for about 2 msec.
- Amplitude of depolarisation is about +20 mV

② Initial Repolarisation :-

- Immediately after depolarisation, there is an initial rapid repolarization for a short period of about 2 msec.
- The end of rapid repolarization is represented by a notch.

③ Plateau or Final Depolarisation :-

- Afterwards, the muscle fibres remain in depolarized state for sometime before further repolarization.
- It forms the plateau in action potential curve. The plateau lasts for about 200 msec in atrial muscle fibres & for about 300 msec in ventricular muscle fibres.
- Due to long plateau in action potential, the contraction time is also longer in cardiac muscle by 5-15 times than in skeletal muscle.

④ Final Repolarization :-

- Final repolarization occurs after the plateau. It is a slow process and it lasts for about 50-100 msec before the re-establishment of Resting membrane potential (RMP).

① Windkessel effect :-

- Windkessel effect is the recoiling effect of blood vessels that converts the pulsatile flow of blood into a continuous flow.
- Blood vessels showing windkessel effect are known as Windkessel vessels.
- Thus, the flow of blood in aorta is a type of pulsatile flow and flow of blood through other blood vessels is continuous flow.
- The elastic recoiling of these blood vessels creates the continuous momentum of blood. So, the pulsatile flow is converted to continuous flow.
- Thus, the windkessel vessels play an imp role in maintaining the continuous flow of blood through the circulatory tree by acting as a second pump, the first pump being the heart.

② Intercalated disc :-

- Intercalated disc is a tough double membranous structure, situated at the junction b/w the branches of neighbouring cardiac muscle fibres.
- It is formed by the fusion of the membrane of the cardiac muscle branches.
- Intercalated discs form adherens junctions, which play an imp role in the contraction of cardiac muscle as a single unit.

③ Cardiac Pacemaker Potential :-

- The cardiac pacemaker potential refers to the unique electrical activity of specialized cells within the heart, that act as a natural pacemakers, initiating & regulating the heart rhythm's.
- These cells are primarily found in SA node located in Rt atrium.
- During pacemaker potential, these cells gradually depolarize, meaning the electrical charge across their cell membrane becomes less negative.
- This gradual depolarization brings the cell closer to its threshold potential, at which an action potential will be generated & propagated throughout the heart causing contraction.
- The pacemaker potential is driven by the various ion channels in the cell membrane.
- Specifically there are two types of ion channels involved funny channels & T-type calcium channels.
- The funny channels allow a slow influx of Sodium & Potassium ions.

- into the cell, which gradually depolarizes the cell.
- The T-type calcium channels on other hand, allow a small influx of calcium ions, further contributing to depolarization.
- Once the pacemaker potential reaches the threshold, a rapid depolarization occurs due to opening of voltage gated Ca channels, which allow a large influx of Ca^{2+} ions into the cell.
- The rapid depolarization triggers the generation of action potential, leading to the contraction of heart muscle & subsequent pumping of blood.
- The pacemaker potential allows the SA node to maintain control over the heart rhythm and ensures coordinated contraction of the chambers.

④ Sinus Arrhythmia :-

- Sinus arrhythmia is a normal rhythmical increase and decrease in heart rate in relation to respiration. It is also called respiratory sinus arrhythmia. (RSA)
- Normal sinus rhythm means the normal heart beat with SA node as the pacemaker. Normal heart rate is $72/\text{min}$.
- However under physiological conditions, in normal healthy person heart rate varies according to the phases of respiratory cycle.
- Heart rate $\uparrow$ s during inspiration & $\downarrow$ s during expiration.

⑤ Stokes Adams syndrome :-

- Stokes-Adams syndrome is the sudden attack of dizziness and unconsciousness caused by heart block. It may be accompanied by convulsions also.
- In many patients suffering from heart block, the complete heart block occurs intermittently.
- When the block occurs, the ventricles stop beating immediately.
- Ectopic pacemaker starts functioning only after 5 to 30 seconds.
- During this time, the blood circulation is affected because lack of ventricular output. Brain cannot withstand the stoppage of blood & oxygen supply even for 5 seconds.
- Before the onset of discharge from ectopic pacemaker, dizziness and fainting occurs. If the discharge of impulses from ectopic pacemaker is delayed beyond 20 secs, death occurs.

⑥ Ventricular fibrillation :-

- Ventricular fibrillation is the dangerous cardiac arrhythmia, characterized by rapid and irregular twitching of ventricles.
- Ventricles beat very rapidly & irregularly due to the circus movement of impulses within ventricular muscle. The rate reaches 400-500/min.
- This is triggered by ventricular extrasystole.
- This type of arrhythmia is serious, as it leads to death, since ventricles cannot pump blood.
- Ventricular fibrillation very common during electrical shock & during ischemia of conductive system.
- It also occurs in other conditions like coronary occlusion, chloroform anesthesia, cyclopropane anesthesia, trauma of heart & disturbances of heart during cardiac surgery.

⑦ Cardiac arrest :-

- Sudden, unexpected loss of heart function, breathing & consciousness.
- In cardiac arrest, the heart abruptly stops beating. Without prompt intervention, it can result in the person's death.
- The main symptom is loss of consciousness & unresponsiveness.
- The medical emergency needs immediate CPR or use of a defibrillator.
- Hospital care includes drugs, an implantable device or other procedures.

⑧ Paroxysmal Tachycardia :-

- Paroxysmal tachycardia is the sudden attack of increased heart rate due to ectopic foci arising from atria, AV node or ventricle.
- It is also called Bouvieret Hoffmann syndrome. Increase in heart rate due to ectopic foci arising from either atria or AV node is called supraventricular tachycardia.
- It differs from ventricular tachycardia, which does not depend upon atria or AV node. The attack lasts for a period of few seconds to few hours.
- It also stops suddenly. After the attack, heart functions normally.
- Symptoms include palpitations, chest pain, rapid breathing and dizziness.

It is of 3 types :

1. Atrial paroxysmal tachycardia.
2. AV nodal " "
3. Ventricular " "

⑨ Heart Block :-

- Heart block, also known as atrio-ventricular block, is a condition that affects the electrical conduction system of the heart.
- It occurs when there is a delay in transmission of impulses from atria to ventricles.
- The heart's electrical conduction system consists of specialized cells that generate and transmit electrical signals, coordinating the heart contractions.
- These signals regulate the heart rate & ensure that the atria & ventricles are contract in coordinated manner.

Based on severity heart block is 3 types

1. First degree heart block :- This is mildest form of heart block and usually doesn't cause any symptoms & no require treatment. The electrical signals are delayed as they pass through the AV node, but all impulses eventually reach the ventricles.

2. Second degree heart block :- This type of heart block is subdivided into

a. Mobitz type I :- In this subtype, there is a progressive delay in the electrical signals passing through AV node until one impulse is blocked and doesn't reach the ventricles. It typically causes no or minimal symptoms.

b. Mobitz type II :- The electrical signals are blocked without any progressive delay. It is considered more serious than type I, as it can lead to more significant symptoms & may require treatment.

3. Third degree heart block :- Also known as complete heart block, this is the most severe form of heart block.

- In this condition, the electrical signals from atria are completely blocked and do not reach the ventricles.
- As a result atria & ventricles are beat independently, with the ventricles relying on their own backup pacemaker cells.
- Third degree heart block usually requires medical intervention, such as pacemaker to regulate the heart rate.

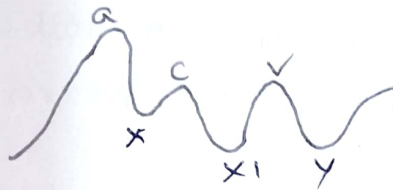
Symptoms :-

- Dizziness or lightheadedness
- Fainting
- Chest pain & shortness of breath.
- Irregular or slow heart rate.

- Causes of heart block can vary. Some common causes include
 - Aging :- Heart block is more common in older adults due to age related degenerative changes in the electrical conduction system.
 - Heart diseases :- Conditions such as coronary artery disease, heart attack, cardiomyopathy & myocarditis can damage the heart's electrical system & lead to heart block.
 - Medications :- Such as beta blockers, calcium channel blockers, & antiarrhythmic drugs can interfere with the heart's electrical conduction.
 - Congenital heart defects
 - Inflammatory conditions.
- Test :- ECG test

⑩ Jugular Venous pulse tracing :-

- Recording of jugular venous pulse is called phlebogram. It is similar to intra-atrial pressure curve, phlebogram also has 3 positive waves namely a, c, v and 3 negative waves names x, x1, y



'a' wave :- 'a' wave is the first positive wave. It is due to rise in atrial pressure during atrial systole. It precedes ventricular systole.

'x' wave :- 'x' wave is a negative wave due to fall in atrial pressure. It coincides with atrial diastole and beginning of ventricular systole.

'c' wave :- 'c' wave is a positive wave due to rise in atrial pressure during isometric contraction period. During this period, the av valves bulge into the atria and increase in pressure in the atria slightly.

• Earlier, it was thought that this wave was due to transmission of pulse from neighbouring carotid artery. Hence it was called 'c' wave.

'x1' wave :- It is a negative wave due to fall in atrial pressure during ejection period. During ejection period, AV ring is pulled towards ventricle causing distension of atria. So, the atrial pressure falls.

'v' wave :- 'v' wave is a positive wave due to rise in atrial pressure. The pressure increases because of venous return. It is obtained during isometric relaxation period or during atrial diastole.

'y' wave :- 'y' wave is the negative wave ^(due to rise in atrial pressure) which denotes fall in atrial pressure. The pressure increases because of pressure. Pressure falls due to the opening of AV valve and emptying of blood into the ventricle. This wave appears during rapid & slow filling periods. 'y' wave is followed by 'a' wave.

11) Phonocardiogram :-

- Phonocardiography is the technique used to record heart sounds.
- Phonocardiogram is the graphical record of heart sounds. It is done by placing an electronic sound transducer over the chest.
- This transducer is connected to a recording device like polygraph.
- All the four heart sounds can be recorded in phonocardiogram. It helps to analyze the frequency of the sound waves.

Appearance of Heart sounds in Phonocardiogram :-

- In phonocardiogram, the heart sounds are recorded in the following manner.

First heart sound :- First heart sound is recorded as single group of waves. The waves are of small amplitude to start with.

- Later the amplitude rapidly rises & falls to form crescendo & decrescendo series of waves. About 9-13 waves appear.

Second heart sound :- Second heart sound appears as single group of waves, which have same amplitude. About 4-6 waves are recorded.

Third heart sound :- Third heart sound is found in phonocardiogram with only 1-4 waves grouped together.

Fourth heart sound :- Mostly, the fourth heart sound merges with first heart sound. If it appears as separate form, it has 1-2 waves with very low amplitude.

12) Factors influencing Venous Return :-

- Venous return is the amount of blood which is returned to heart from different parts of the body.
- When it increases, the cardiac output & ventricular filling are increased.
- The cardiac output is directly proportional to venous return. Venous return in turn, depends on 5 factors:

i) Respiratory pump :- Respiratory pump is the respiratory activity that helps the return of blood, to heart during inspiration.

• It is also called abdominothoracic pump. Intrathoracic pressure increases the diameter of IVC, resulting in increased venous return.

• Increase in intra-abdominal pressure compresses abdominal veins & pushes the blood upwards towards the heart & thereby the venous return is increased.

ii. Muscle pump :- Muscle pump is the muscular activity that helps in return of blood to heart. During muscular activities veins are compressed and squeezed.

• Due to presence of valves in veins, during compression blood is moved towards the heart. When muscular activity increases, venous return ↑s.

iii. Gravity :- Gravitational force reduces the venous return. When a person stands for a long period, gravity causes pulling of blood in the legs, which is called venous pulling. Because of venous pulling, the amount of blood returning to heart ↓s.

iv. Venous pressure :- Venous pressure is inversely proportional to venous return. Increase in venous pressure decreases venous return.

v. Sympathetic tone :- Venous return is aided by sympathetic or vasomotor tone, which causes contraction of venules. Vasoconstriction pushes the blood towards heart.

⑬ Fick's Principle :- According to this principle, the amount of a substance taken up by an organ or given out in a unit of time is the product of amount of blood flowing through the organ & the arteriovenous diff of the substance across the organ.

$$\text{Amt of substance taken or given} = \text{Amt of blood flow/min} \times \text{Arteriovenous difference.}$$

⑭ Water-Hammer pulse :-

Water hammer pulse is the abnormal pulse, characterized by a rapid upstroke & equally rapid downstroke. It is also called collapsing or Corrigan pulse.

• It is seen in conditions like aortic regurgitation, patent ductus arteriosus and arteriovenous fistula.

• It is best felt by raising the arm of the subject & holding it by grasping the wrist with palm of the observer.

15) Korotkoff's sound :-

- The chest piece of the stethoscope is placed over the antecubital fossa and the arm cuff is slowly deflated. While doing so, series of sounds are heard through the stethoscope.
- These sounds are known as Korotkoff sounds. While reducing the pressure, Korotkoff sounds have 5 phases.
- As the pressure in the cuff is slowly decreased, blood flow through the artery is initially blocked by the inflated cuff, resulting in no sound being heard.
- However, the cuff pressure drops below the systolic pressure, blood flow is restored in spurts.
- These spurts of blood create vibrations on the artery, which can be detected as sounds with a stethoscope or through electronic sensors in automated devices.

- ① Phase I :- The first sound that is heard as the cuff pressure falls just below the systolic blood pressure. The sound is clear, rhythmic, repetitive thumping sound.
- ② Phase II :- A murmur or swishing sound follows the first sound. It is soft & less distinct than the initial thumping sound.
- ③ Phase III :- A crisp, louder & more intense thumping sound is heard during this phase. (Appearance of gong sound).
- ④ Phase IV :- The sound becomes muffled & softer. It is described as a distinct, abrupt muffling or a blowing sound.
- ⑤ Phase V :- The final sound heard before the sounds disappear completely. It is a complete cessation of sound, indicating the cuff pressure has fallen below the diastolic blood pressure.

16) Brain bridge reflex :-

- Brain bridge reflex is a cardioaccelerator reflex that increases the heart rate when venous return is increased.
- Since, this reflex arises from Rt atrium, it is also called Rt atrial reflex.
- Increase in venous return causes distension of Rt atrium & stimulation of stretch receptors, situated in the wall of Rt atrium.
- Stretch receptors, in turn send inhibitory impulses through Inferior vena cava to the vasodilator area of vasomotor centre.

- Vasoconstrictor area is inhibited, resulting in increase in heart rate.

⑦ Reynold's number :-

- Critical velocity at which the flow becomes turbulent is known as Reynolds number.

$$\text{Reynolds number} = \frac{PDV}{\eta}$$

P = Density of the blood

D = Diameter of the vessel

V = Velocity of the flow.

η = Viscosity of the blood.

⑧ Peripheral resistance :-

- Resistance offered to blood flow in peripheral blood vessels is called Peripheral resistance.
- Arterial branches become narrower & their walls become thinner while reaching the periphery. Aorta has got the max diameter of about 25 mm.
- Diameter of arteries gradually decreased & at the end arteries, it is about 4 mm. It further decreases to 30 μ m in the arterioles & ends up with 10 μ m in terminal arterioles.
- Peripheral resistance is offered to blood flow in the arterioles and so these vessels are called resistant vessels.

⑨ Tissue fluid formation & Edema :-

1. (Blood does not pass through capillary system continuously. It is because of the alternate constriction & dilatation of meta arterioles and the alternate opening & closure of precapillary sphincters.
2. Direction of blood flow through capillaries is not fixed as in the case of other blood vessels. Blood may flow in opp direction in two adjacent capillaries)

⑩ Tissue fluid formation :-

- Tissue fluid is also known as interstitial fluid, is the fluid that surrounds & bathes the cells in the body's tissues.
- It is derived from the blood plasma & plays a vital role in providing oxygen, nutrients and other essential substances to the cells while removing waste products.

- Tissue fluid formation occurs through a process called filtration. Filtration is driven by the balance of hydrostatic pressure & osmotic pressure across the capillaries.
- Hydrostatic pressure: This is the pressure exerted by the fluid within the capillaries. It tends to push fluid out of the capillaries into the surrounding tissues.
- Osmotic pressure: This is the pressure exerted by the conc of solutes, such as proteins, in the blood plasma. Osmotic pressure tends to draw fluid back into the capillaries.
- At the arterial end of capillaries, the hydrostatic pressure is higher than osmotic pressure. As a result, fluid is forced out of the capillaries into the interstitial spaces, forming tissue fluid.
- At the venous end of the capillaries, the osmotic pressure is higher than hydrostatic pressure, allowing the reabsorption of excess fluid back into the capillaries.

②① Edema :-

- Edema is the abnormal accumulation of fluid in the interstitial spaces of tissues, leading to swelling.
- It occurs when there is an imbalance b/w fluid filtration out of capillaries & fluid reabsorption back into capillaries.
- Edema can be localized, affecting a specific area or generalized, involving the whole body.

Causes :-

- Increased capillary permeability & Impaired lymphatic drainage.
- Elevated hydrostatic pressure & decreased osmotic pressure.
- Inflammation, injury, heart failure, kidney disorders & venous insufficiency.

Signs & Symptoms :- Depends on the location & severity, but often include

- Swelling, tightness or heaviness in the affected area & restricted movement.
- Edema can be diagnosed through physical examination, medical history and sometimes additional tests such as blood work / imaging studies.

Treatment :-

- Administering anti-inflammatory medications, managing heart or kidney disease, or employing measures to reduce fluid retention such as diuretic medications, compression therapy. Reducing Na intake. Healthy wt maintaining.

②1 Regulation of BP through Renin-Angiotensin Mechanism :-

Actions of Angiotensin II :-

- When blood pressure and ECF volume decrease, renin secretion from kidneys is increased. It converts angiotensinogen to angiotensin I.
- Angiotensin I is converted to Angiotensin II by ACE (Angiotensin converting enzyme).
- Angiotensin II acts in two ways to restore the blood pressure.
 - i. It causes constriction of arterioles in the body so that the peripheral resistance increased & blood pressure rises.
 - In addn, angiotensin II causes constriction of afferent arterioles in kidneys, so that glomerular filtration reduces. This results in retention of water & salts, increases ECF volume to normal level.
 - This in turn increases the blood pressure to normal.
 - ii. Simultaneously, angiotensin II stimulates adrenal cortex to secrete aldosterone. This hormone its reabsorption of sodium from renal tubule.
 - Sodium reabsorption is followed by water reabsorption, resulting in increased ECF volume & blood volume. It increases the blood pressure to normal level.

Actions of Angiotensin III & Angiotensin IV :-

Like angiotensin II, angiotensin III & IV also increase the blood pressure & stimulate adrenal cortex to secrete aldosterone.

②2 Marey's Law :-

- According to Marey Law, the pulse rate is inversely proportional to blood pressure. Baroreceptors induce the Marey reflex only during resting conditions.
- So, in many conditions such as exercise, there is an increase in both blood pressure & heart rate.

②3 Physiological basis of drug in Hypertension (HTN) :-

- These drugs act directly on vascular smooth muscle and relax them without contribution of cell receptors.
- By relaxing smooth muscle, vasodilation occurs. This reduces total peripheral resistance & venous return to the heart is reduced through it.
- As this lowers the blood pressure.

②4 Orthostatic hypotension :-

- Orthostatic hypotension also called postural hypotension - is a form

of low blood pressure that happens when you stand up from sitting or lying down.

- Orthostatic hypotension can make you feel dizzy, or lightheaded and may be even cause you to faint.
- Orthostatic hypotension may be mild, and episodes can last for less than a few minutes. However, long lasting orthostatic hypotension can signal more serious problems, so it's imp to see a doctor if you frequently feel lightheaded when standing up.
- Acute orthostatic hypotension is usually caused by dehydration or lengthy bed rest and is easily treated.
- Chronic orthostatic hypotension is usually a sign of another health problem, so treatment varies.

25) Baroreceptor reflex :-

- The fall in arterial blood pressure or the occlusion of common carotid arteries decreases the pressure in carotid sinus.
- This causes inactivation of baroreceptors. Now, there is no inhibition of vasoconstrictor centre or excitation of vasodilator centre.
- Therefore, the blood pressure rises.
- Information regarding blood pressure within the range of 50-200 mm Hg reaches the vasomotor centre through carotid baroreceptors.
- Information about the blood pressure range of 100-200 mm Hg goes through aortic baroreceptors.
- Both carotid & aortic baroreceptors are stimulated by rising pressure than the steady pressure and their response depends upon the rate of increase in blood pressure.
- Since the baroreceptor mechanism acts against the rise in arterial blood pressure, it is called pressure buffer mechanism or system & the nerves from baroreceptors are called buffer nerves.

26) Sinoaortic Reflex & Its importance :-

- Sinoaortic reflex is a physiological reflex mechanism that plays a vital role in regulating blood pressure & heart rate.
- It involves the communication b/w the baroreceptors located in the aortic arch and carotid sinuses & the cardiovascular control centres in brain.
- The baroreceptors are specialized sensory receptors that detect changes in blood pressure. When blood pressure increases, these

receptors are stretched, leading to an increase in the firing rate of these nerve impulses.

- Conversely, when blood pressure decreases, the firing rate of these receptors decreases.
- The sensory information from the baroreceptors is transmitted to cardio-vascular centres in medulla oblongata.
- In response to increased BP, the sinoaortic reflex triggers a parasympathetic response and inhibits the sympathetic nervous system. This leads to decrease in heart rate & a dilation of blood vessels, resulting in a decrease in BP.
- Conversely, when blood pressure drops, the reflex triggers a sympathetic response, leading to increase in heart rate & constriction of blood vessels, which increases BP.

Importance :-

- It maintains homeostasis & ensure the stability of blood pressure. It helps to regulate blood pressure within a normal range, allowing the body's organs & tissues to receive an adequate blood supply.
- It plays a critical role in adapting to various physiological & environmental changes that effect blood pressure, such as changes in posture, physical activity, stress & blood loss.
- Disruptions in the sinoaortic reflex can contribute to conditions such as
 - a) HTN
 - b) Hypotension.

(27) Regulation of Cerebral circulation :-

- The regulation of cerebral circulation is crucial for maintaining adequate blood flow to the brain, which is necessary for its metabolic demands & overall functioning.
- The regulation involves interplay of various mechanisms to ensure a constant & appropriate supply of oxygen, glucose & other nutrients to brain cells.

@ Autoregulation :-

- The brain has an intrinsic ability to regulate its own blood flow to ensure a relatively const supply of oxygen & nutrients.
- Autoregulation allows for stable cerebral blood flow despite changes in systemic blood pressure.
- It is primarily mediated by the dilation or constriction of cerebral

arterioles in response to change in local metabolic factors such as oxygen, carbon dioxide levels, pH and adenosine contraction.

② Chemical factors :-

Chemical factors which increase cerebral blood flow are

- i. Decreased O_2 tension
- ii. Increased CO_2 "
- iii. " hydrogen ion concentration.

- Carbon dioxide is the most imp factor, as it causes dilatation of cerebral blood vessels, leading to increase in blood flow.
- A moderate increase in CO_2 tension does not alter the blood flow due to autoregulation.
- When arterial partial pressure of CO_2 rises above 45 mm Hg, the cerebral blood flow actually increases.
- CO_2 combines with water to form carbonic acid, which dissociates into bicarbonate ions & hydrogen ion.
- The hydrogen ion causes dilatation of blood vessels in brain. Hypoxia increases cerebral blood flow by vasodilation.

③ Nervous factors :-

- Cerebral blood vessels are supplied by sympathetic vasoconstrictor fibres.
- But these fibres do not play any role in regulating cerebral blood flow under normal conditions.
- In pathological conditions like HTN, the sympathetic nerves cause constriction of cerebral blood vessels, leading to reduction in blood flow.
- It prevents cerebral vascular hemorrhage & cerebral stroke.

Other factors

④ Cerebral perfusion pressure

⑤ Metabolic regulation

⑥ Autoregulation

⑦ Blood gases & pH.

②8 Triple Response :-

- The triple response is a physiological phenomenon that occurs in response to a localized injury or irritation of the skin.
- The triple response consists of 3 distinct changes in the affected area.

1. Redness (Flare) :- The initial response to the injury is the dilation of small blood vessels in the surrounding area, leading to increased

blood flow. This results in redness & swelling at the site of injury.

- The redness occurs due to the release of chemical mediators such as histamine, which cause vasodilation.

2. Swelling (Wheal): Following the vasodilation, there is an increased permeability of local blood vessels.

• This allows fluids & proteins to leak into the surrounding tissues, leading to swelling or edema.

- The accumulation of fluid causes skin to appear raised & puffy, forming a wheal.

3. Itching & Flattening: In addⁿ to redness & swelling, the triple response often includes itching or pruritus.

- This sensation is thought to be caused by release of histamine & other chemical mediators.
- As the response progresses, the wheal may gradually flatten out & returning to normal & the itching subsides.

- Triple response is commonly used in dermatology & allergy testing.

②⑨ Coronary circulation & Special features:-

Coronary arteries:-

- Heart muscle is supplied by two coronary arteries, namely rt and lt coronary arteries, which are the first branches of aorta.
- Arteries encircle the heart in the manner of a crown, hence the name coronary arteries.

Rt & Lt Coronary arteries:-

- Rt coronary artery supplies whole of the Rt ventricle & post portion of lt ventricle.
- Lt coronary artery supplies mainly the ant & lat parts of lt ventricle.

Variations in Coronary arteries:-

1. In 50%-60% of human beings, the rt coronary artery is larger and supplies more blood to heart than lt coronary artery.
2. In 15%-20% of human beings, the lt coronary artery is larger.
3. In 20%-30% of human beings, both arteries supplies almost equal amount of blood.

Branches of Coronary artery:-

- Coronary artery divide & subdivide into smaller branches, which run along the surface of heart.
- Smaller branches are called epicardiac arteries & gives rise to further smaller branches known as fial arteries or intramural vessels.

Venous drainage :-

• Venous drainage from heart muscle is by 3 types of vessels.

1. Coronary sinus :- Coronary sinus is larger vein draining 75% of total coronary flow. It drains blood from lt side of the heart & opens into rt atrium near tricuspid valve.
2. Ant coronary veins :- Ant coronary veins drain blood from rt side of heart & open directly into rt atrium.
3. Thebesian veins :- Thebesian veins drain deoxygenated blood from myocardium, directly into ~~rt atrium~~ concerned chambers of heart.

Physiological Shunt :-

- Physiological shunt is the diverted route, through which the venous blood is mixed with arterial blood.
- Deoxygenated blood flowing from thebesian veins into cardiac chambers makes up the part of normal physiological shunt.
- Other part of physiological shunt is the drainage of deoxygenated blood from bronchial circulation into pulmonary vein, without being oxygenated.

Phasic changes in Coronary blood flow :-

- Blood flow through coronary arteries is not const. It decreases during systole & increases during diastole.
- Intramural vessels of fial arteries supplying myocardium are perpendicular to the cardiac muscles.
- So, during systole, intramural vessels are compressed & blood flow is reduced.
- During diastole compression is released & the blood vessels are distended, so, the blood flow increases.

Factors regulating Coronary blood flow :-

- 1) Autoregulation
- 2) Need for oxygen
- 3) Metabolic factors
- 4) Coronary perfusion pressure
- 5) Nervous factors.

③① Cardiovascular changes during Exercise :-

- During exercise, the CVS undergoes several changes to meet the increased demands of the body.
- These changes are aimed at delivering oxygen & nutrients to the working muscles and removing metabolic waste products efficiently.
- Here are some of the key cardiovascular changes that occur during exercise :

a. Increased Heart Rate :- One of the primary change during exercise is an increase in heart rate. The heart beats faster to pump a greater volume of blood to the muscles & organs. This helps to deliver O_2 & nutrients more rapidly & increases the removal of waste products.

b. Increased stroke volume :- Stroke volume is the amount of blood pumped by heart with each contraction. During exercise, stroke volume typically increases. This means that with each heart beat the heart ejects a large vol of blood, allowing for more efficient delivery of O_2 with blood to the muscles.

c. Elevated Cardiac output :- Cardiac output is the volume of blood pumped by heart per min. It is the product of stroke volume & heart rate. During exercise, both heart rate & stroke volume increases, resulting in significant rise in cardiac output. This increase ensures an adequate supply of O_2 & nutrients to the muscles.

d. Vasodilation :- Exercise leads to dilation of blood vessels, primarily arterioles in working muscles. This process is called vasodilation. Vasodilation allows increased blood flow to muscles, improving O_2 & nutrient delivery. Additionally, helps to dissipate heat generated during exercise through skin, promoting body temp regulation.

e. Increased Venous Return :- Venous return is the flow of blood back to heart from the body tissues. During exercise, venous return increases due to the muscle contractions & pumping action of surrounding veins.

f. Increased blood pressure :- Blood pressure tends to rise during exercise. This is mainly due to the increased cardiac output & the vasoconstriction of non-essential blood vessels. The elevated blood pressure ensures an adequate perfusion of blood to the muscles & organs.

31) Critical Closing Pressure (CCP) :-

- CCP is the minimum pressure required to keep a blood vessel open and maintain blood flow. It represents the balance b/w the intravascular pressure & the extravascular forces acting on the muscle.
- In simple terms, CCP is the point at which a blood vessel collapses or closes off due to ext compression.
- It is influenced by the interaction b/w the pressure inside the vessel & the surrounding tissue pressure.
- When intravascular pressure is higher than the external compressive forces, the blood vessels remains open, allows blood flow.
- When the ext compressive forces exceed the intravascular pressure the vessel collapses, leading to cessation or reduction of blood flow.
- If the the intravascular pressure falls below the CCP, blood flow to tissue is compromised, resulting in inadequate O_2 & nutrient delivery.

32) Laplace law :-

- Laplace law describes the relation b/w the pressure inside a spherical structure, such as blood vessel or hollow organ & tension in its wall.
- According to Laplace's law, the tension (T) in the wall of structure is directly proportional to the pressure (P) inside the structure & the radius of the structure & inversely proportional to wall thickness (h).

$$T = \frac{P \times r}{h}$$

33) Cushing's Reflex :-

- Cushing reflex also known as the Cushing response. It is a physiological response of the body to increased intracranial pressure (ICP).
- When there is an increase in ICP, it indicates compression or damage to the brain, typically due to conditions like head injury, brain tumor, or bleeding within the skull.

- The Cushing reflex is an autoregulatory response to restore cerebral blood flow & oxygenation to brain.
- The Cushing reflex involves 3 main components:
 - i) Increased Blood Pressure :- This response helps to maintain cerebral perfusion pressure in the face of increased ICP.
 - ii) Bradycardia :- Bradycardia helps to decrease cerebral blood flow & lower the volume of blood entering the brain, thus reducing the overall ICP.
 - iii) Irregular respirations :- Abnormal respiratory patterns further assist in maintaining cerebral blood flow & reducing ICP.

The Cushing reflex is considered as a pathological response because it indicates severe brain injury or increased ICP. It is an attempt by the body to protect the brain by ensuring an adequate supply of O_2 & nutrients despite the compression.

34) Hypovolemic & Distributive Shocks :-

- Shock due to decreased blood volume is called hypovolemic shock or cold shock.
- It occurs when there is acute loss of at least 10%-15% of blood.
- Loss of blood less than 10% may not produce any significant effect because of a rapid compensatory mechanism.

Manifestations of Hypovolemic shock :-

- i) Decrease in cardiac output
- ii) Low blood pressure
- iii) Thin thready pulse
- iv) Pale & cold skin
- v) Increase in respiratory rate
- vi) Restlessness or lethargy.

Pathological conditions when Hypovolemic shock occurs :-

- a) Hemorrhage - Hemorrhagic shock
- b) Trauma - Traumatic shock
- c) Surgery - Surgical shock
- d) Burns - Burn shock
- e) Dehydration - Dehydration shock.

Treatment

- Blood transfusion
- Plasma "
- Administration of plasma substitutes
- " " Glucocorticoids.
- Oxygen therapy
- By changing the posture.

⇒ Distributive shock also known as vasodilatory shock, occurs when there is widespread & excessive dilation of the blood vessels throughout the body.

- This dilation leads to a decrease in systemic vascular resistance resulting in poor blood flow & inadequate O_2 & nutrient delivery to organs & tissues.

Types :-

- Septic shock
- Anaphylactic shock
- Neurogenic shock.

- Treatment of distributive shock involves administration of epinephrine & antihistamines in anaphylactic shock. Additionally IV fluids & medications to support blood pressure may be administered.

③ Regulation of Blood Pressure :-

- Blood pressure is regulated by a complex interplay of physiological mechanisms involving the ANS, hormonal systems & local factors.

i. The autonomic nervous system, specifically the sympathetic & parasympathetic divisions, plays a central role in blood pressure regulation.

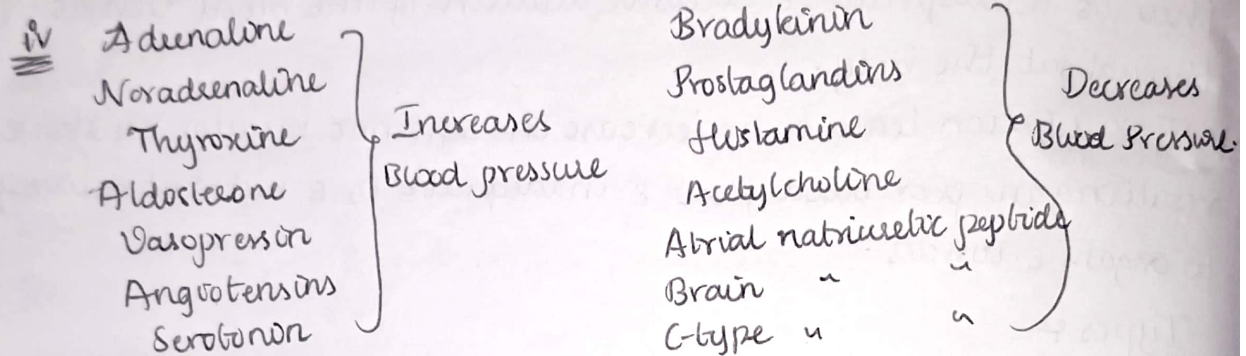
Sympathetic activation ↑ blood pressure through vasoconstriction & increased heart rate.

Parasympathetic activation ↓ blood pressure through vasodilation & decreased heart rate.

ii. The renin-angiotensin-aldosterone system (RAAS) is a hormonal

system that helps to regulate blood pressure. It involves the release of renin, conversion of angiotensinogen to angiotensin I & then angiotensin II, which causes vasoconstriction & stimulates aldosterone release, leading to increased sodium & water retention.

iii. Baroreceptors located in the carotid sinus & aortic arch, are pressure sensors that detect changes in blood pressure. They initiate reflex responses through the cardiovascular control centres in the medulla oblongata, leading to adjustments in sympathetic & parasympathetic activity, heart rate & vascular tone.



v. Local factors within blood vessels, such as endothelium-derived nitric oxide, also influence blood pressure regulation by promoting vasodilation & vasoconstriction.

vi Kidneys play a crucial role in long term blood pressure regulation through the control of fluid volume & sodium balance. They regulate blood pressure by adjusting the excretion or reabsorption of sodium & water.

vii Blood pressure regulation is dynamic process, with constant adjustments occurring in response to various stimuli, such as changes in body position, physical activity, stress & metabolic demands.

Hypertension — Increased BP

Hypotension — Decreased BP.

(36) Myocardial Infarction :-

- Commonly known as heart attack, occurs when the blood supply to a part of heart muscle is significantly reduced or completely blocked, leading to tissue damage or death.
- It is usually caused by the occlusion of coronary artery, which supplies O₂ rich blood to the heart muscle.

Causes:-

- The most common cause of MI is the formation of blood clot (thrombus) within a coronary artery.
- This clot often develops in a blood vessel that has already been narrowed by atherosclerosis, a condition characterized by a buildup of plaque on the arterial walls.
- Other causes may include coronary artery spasm, embolism or dissection of a coronary artery.

Symptoms:-

- Symptoms may include chest pain or discomfort, often described as squeezing, pressure, heaviness or tightness in the chest.
- The pain may radiate to the left arm, jaw, neck or back.
- Other accompanying symptoms may include shortness of breath, sweating, nausea, lightheadedness & anxiety.

Diagnosis:-

- ECG changes
- Blood tests (ex: cardiac troponins)
- Imaging techniques - angiography & echocardiography.

Treatment:-

- Medications - Aspirin, nitroglycerin & antiplatelet drugs may be given to improve blood flow & reduce clotting.
Thrombolytic drugs or anti-coagulants may be administered to dissolve or prevent further clot formation.
- Percutaneous Coronary Intervention
- Coronary Artery Bypass Grafting (CABG)
- Cardiac rehabilitation & medications.

Complications:-

- Complication of MI leads to heart failure, arrhythmia, cardiogenic shock, rupture of the heart muscle, development of aneurysms in the heart wall.