



CSIR-NET

LIFE SCIENCE

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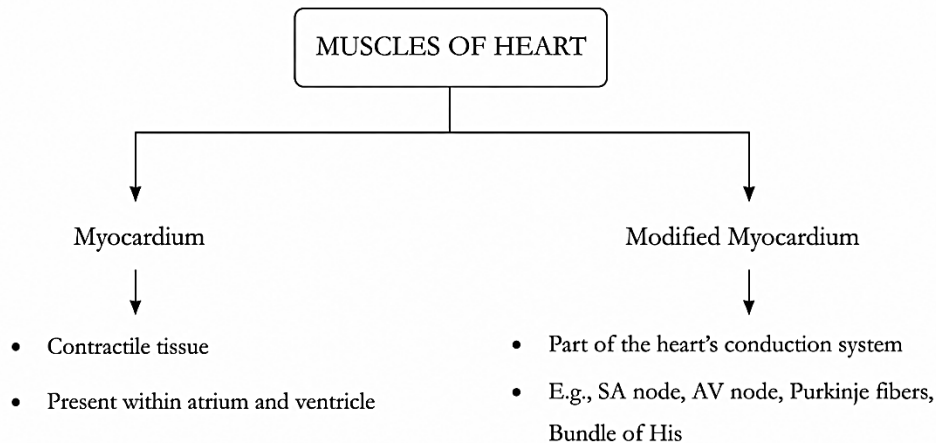


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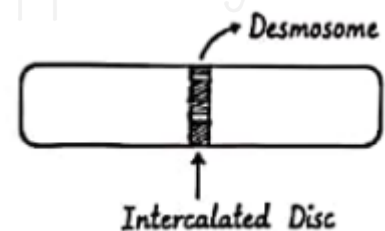
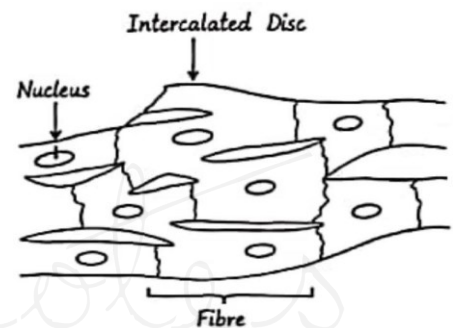
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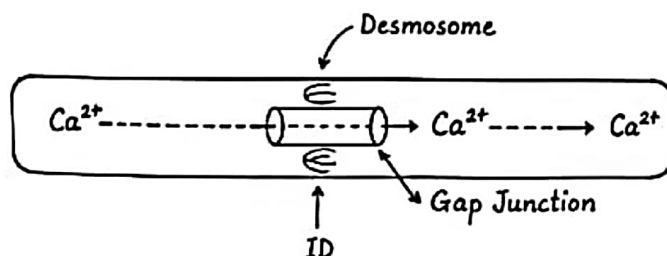
System Physiology - Animal



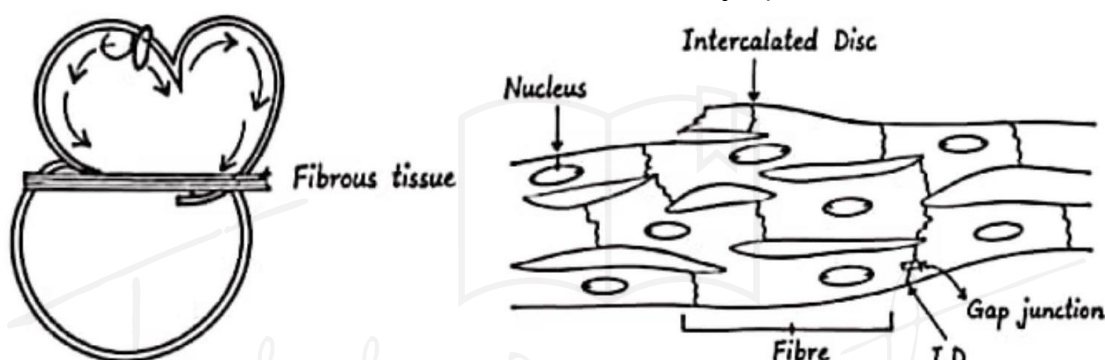
➤ Properties Of Myocardium

- ✓ Striated muscle - dark and light bands present under microscope *d/t* particular actin and myosin arrangement
- ✓ → Myosin
- ✓ Myosin II is responsible for muscle contraction in cardiac, skeletal and smooth muscle
- ✓ Myosin is composed of a heavy and light chain
- ✓ In the heart, myosin heavy chain is of two types
- ✓ α Myosin Heavy Chain (α MHC): Present in atrium
- ✓ β Myosin Heavy Chain (β MHC): Present in ventricle
 - Single nucleus per cell Presence of branching in the myocardium at regular intervals
 - Presence of Intercalated Disc
- ✓ Exclusive feature of the myocardium
- ✓ Intercalated disc - fusion of 2 plasma membrane to one another with help of desmosome protein
- ✓ Area connected by desmosome under microscope gives a dark colored line i.e., Intercalated disc
 - Gap Junction
- ✓ Presence of channel protein at level of intercalated disc connecting two cells
- ✓ Gap junctions are present in myocardial cells and smooth muscles but are absent in the skeletal muscle
- ✓ Gap junction is composed of Connexin (C_x) protein.
- ✓ Advantage
- ✓ Stimulation of myocardium cell → Action potential generated → Entry of Ca^{2+} in cytoplasm of myocardial cells → Contraction.
- ✓ Due to gap junction → Ca^{2+} moves from one cell to another within a fraction of a second through the gap junction.
- ✓ Net result - All myocardial cells connected with one another will contract simultaneously.





- ✓ Syncytium property
- ✓ When SA node is stimulated at single point of atrium muscle → Atrium contracts simultaneously (d/t gap junction present in between atrium muscle cells).
- ✓ When SA node is stimulated at one point near atrium → Ca^{2+} spread from one myocardial cell to other myocardial cells → both atria contracts simultaneously i.e., syncytium property.
- ✓ Fibrous tissue ring (electrical divider) divide atrium from ventricular muscles
- ✓ It does not contain gap junction.
- ✓ It is electrically inext hence Ca^{2+} cannot enter within ventricular muscles.
- ✓ Atrium behaves like a separate entity from ventricle.
- ✓ All chambers of the heart do not contracts simultaneously d/t electrical divider.



➤ Comparison Between Myocardium and Skeletal Muscles

Myocardium	Skeletal Muscles
- Branching present	- No branching
- Myocardium - striated muscle	- Skeletal muscle - striated muscle
- Single nucleus per cell	- Multinucleated cells
- Intercalated discs are present	- Intercalated discs are absent
- Gap junctions are present	- Gap junctions are absent
- Syncytium property present	- Cannot behave like single syncytium

➤ Modified Myocardium

- ✓ Modified myocardium-present in the conducting system of heart (SA node, AV node, Bundle of His, Purkinje fibers)

➤ Properties Of Modified Myocardium

- ✓ Less actin and myosin proteins
- ✓ Fewer striations compared to normal myocardium
- ✓ Gap junctions are present
- ✓ Less number of cell organelles
- ✓ Every part of modified myocardium contains small round cells i.e., P-cell (Pacemaker cell)
- ✓ Can generate spontaneous impulses without help of autonomic nervous system (sympathetic and parasympathetic system) - Automaticity property
- ✓ Also seen in a transplanted heart

RATE OF AUTOMATICITY

SA node
60-80 beats/min
(max: 100 beats/min)

AV node and Bundle of His
40-60 beats/min

Purkinje fibres
15-40 beats/min

- ✓ SA node (dominant/ normal pacemaker) → inhibitory effect on other pacemaker tissue in heart (AV node, Bundle of His and Purkinje fibers)
- ✓ This property is known as Overdrive suppression of SA node
- ✓ In normal heart, only SA node produces beats, other pacemaker cells (AV node, Bundle of His, and Purkinje fibers) remain silent
- ✓ Silent pacemakers are known as Latent pacemakers

➤ Complete Heart Block

- ✓ Complete heart block at the AV node → ventricle stop pumping (No impulse from SA node) → Asystole of the ventricle
- ✓ Can persist for 20 – 25sec
- ✓ If it persists for > 4-5 secs → Transient brain hypoxia
- ✓ Cause dizziness and fainting and fall
- ✓ Collectively known as Stokes-Adams Syndrome
- ✓ But after 20 – 25sec → Purkinje fibers start generating pulse and this is known as Idioventricular rhythm (rate 15-40 beats/minute)

➤ Heart Rate Of A Transplanted Heart

- ✓ Resting heart rate - 95-100 beats/min
- ✓ Normally during exercise, heart rate increases due to sympathetic system stimulation

In a person with a transplanted heart, during exercise

↓
Sympathetic system activation stimulates adrenal medulla

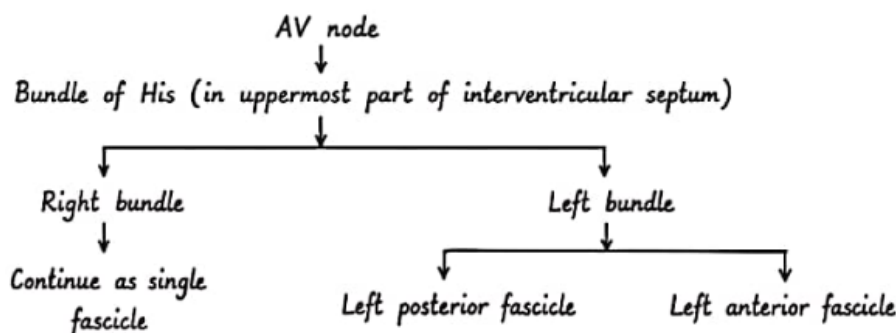
↓
Production of epinephrine/ norepinephrine

↓
Acts on the β_1 receptor of the transplanted heart

↓
Gradual increase in heart rate

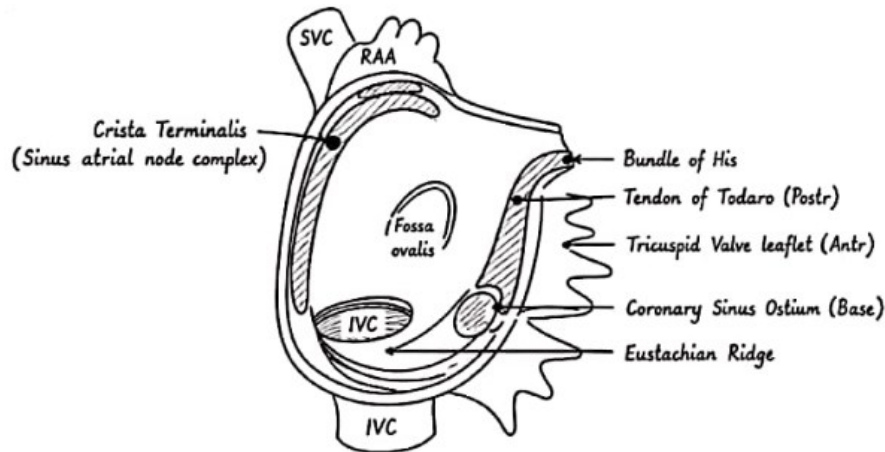
➤ Conducting System of the Heart

- ✓ SA node and AV node are connected via three internodal bundles
- ✓ Anterior internodal bundle - Bundle of Bachmann
- ✓ Middle internodal bundle - Bundle of Wenckebach
- ✓ Posterior internodal bundle - Bundle of Thorel
- ✓ Conduction velocity of impulse is slower when travelling through atrial muscle
- ✓ Internodal bundle-faster conducting bundle
- ✓ Bundle of His - only electrical connection between atrium and ventricle
- ✓ It penetrates fibrous tissue ring dividing the atrial and ventricular syncytium



- ✓ Fascicle → composed of multiple fibers
- ✓ Located in a subendocardial region
- ✓ Just beneath endocardium → fascicle separates out
- ✓ These separated out fibers from fascicles are known as Purkinje fibers
- ✓ Direction of Purkinje fibers → from endocardium to epicardium

➤ Sa Node and AV Node



Features	Sinoatrial node (sa)	Atrioventricular node (av)
Location	- Sub-epicardial structure	- Endocardial surface
Dimension	- 10-20 mm long, 5 mm thick, and 1-2 mm deep	- AV node is compact ($5 \times 3 \times 1 \text{ mm}$)
Physiological location	- Posterior wall of the right atrium near the superior vena cava opening	- Base of the right atrial septum
Anatomical location	- Groove between Crista terminalis and corresponding Sulcus terminalis	- Triangle of Koch - Coronary Sinus Ostium (base) - Tendon of Todaro - Septal leaflet of Tricuspid Valve

➤ Conduction Time and Velocity

AV Nodal Delay

- ✓ Time required for impulse conduction from the SA node to the AV node - 30 ms
- ✓ Now, Impulse travels through the AV node (5 mm length)
- ✓ AV nodal delay - Total time taken required to travel through AV node - 100 ms (60 – 125 ms, very slow)
- ✓ Faster heart rate (faster conduction) → AV nodal delay- 60 ms
- ✓ Slow heart rate → AV nodal delay- 125 ms

HIS Bundle Delay

- ✓ Time taken to cross Bundle of His –40 ms(35 – 55 ms)
- ✓ His bundle is located in the uppermost portion of the interventricular septum
- ✓ When an impulse crosses the Bundle of His → depolarization of ventricular muscle
- ✓ First part of ventricular muscle to depolarize - Middle part of the interventricular septum

➤ Impulse Conduction

- ✓ Time intervals for impulse conduction
- ✓ 30 milliseconds - to cross from SA node to the AV node
- ✓ 100 milliseconds - to cross AV node
- ✓ 40 milliseconds - to cross bundle of His
- ✓ Total time required to start ventricular depolarization for 1st time -170 ms

- ✓ Total time taken to reach the apex - 200 milliseconds
- ✓ Impulse travel from apex towards the base of heart → both ventricles depolarize simultaneously - Time taken to travel from apex to the base of the heart - 20 ms
- ✓ Total time taken for complete depolarization of the heart - 220 ms

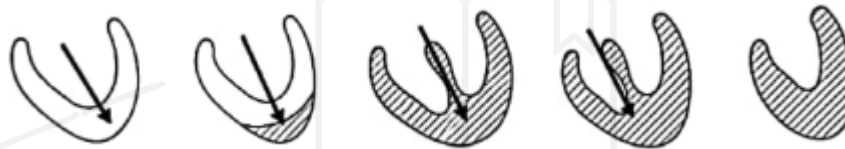
➤ Conduction Velocity of Impulses In Different Parts of the Heart

- ✓ Fastest impulse conduction - from apex to the base of the heart through Purkinje fibres
- ✓ Slowest conduction velocity - AV node

Part	Conduction Velocity
SA Node	0.05 m/sec
AV Node	0.02 – 0.05 m/sec
Purkinje fibers	4 m/sec
Atrium muscle / Ventricular muscle / Bundle of His	1 m/sec

- ✓ Reasons for the fastest conduction velocity of Purkinje Fiber
- ✓ ↑↑ gap junctions
- ✓ Thickest fibre diameter (more diameter, less resistance)
- ✓ Action potential generated in Purkinje fiber - fast type

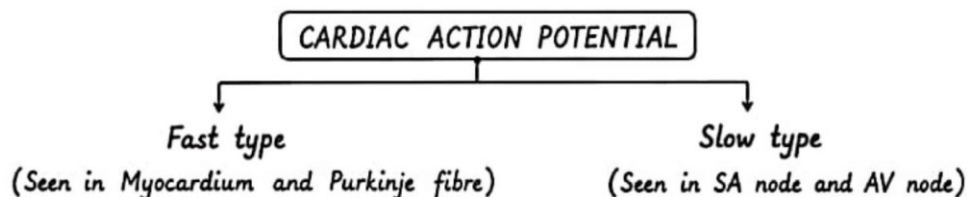
➤ Depolarization and Repolarization



- ✓ Black color arrow indicates the voltage factor pointing towards the apex of the heart (during depolarization and repolarization)

	Depolarization	Repolarization
Atria	Starts at the SA node	Starts near the SA node
Ventricular	- First part: Left endocardial surface of the interventricular septum at the middle portion - Last part: Uppermost part of the interventricular septum and postero-basal epicardial surface of the left ventricle	- Apical epicardial surface - first to repolarize - Repolarization web moves from apex to base of heart

➤ Action Potential in Cardiac Muscle



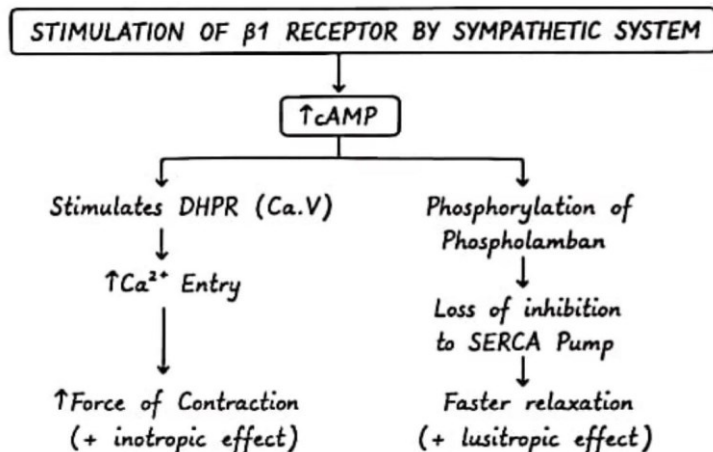
✓ Resting Membrane Potential Of Heart Muscles

- Myocardium: -90 mV
- Purkinje fibers: -80 mV
- SA node: -50 mV
- AV node: -60 mV
- SA node and AV node do not have fixed RMP
- Range for both → - 65 to -40 mV

Fast type of action potential	Slow type of action potential
➤ Phase 0 : Depolarisation phase: ✓ RMP of -90 mV suddenly shifting to $+35$ mV ➤ Phase 1 : Rapid transient / initial rapid repolarisation phase: ✓ Membrane potential returning to baseline, not completely ➤ Phase 2 : Stable phase (plateau phase): ✓ Stable membrane potential, not much fluctuations ➤ Phase 3 : Repolarization phase: ✓ Membrane potential returns to resting membrane potential ➤ Phase 4 : Resting membrane potential (RMP): ✓ Rapid rising phase of phase 0, hence fast type action potential	➤ Phase 4 (RMP phase) : Prepotential / Pacemaker Potential / diastolic depolarisation phase: ✓ Varies from -65 mV to -40 mV; ✓ Unstable RMP – pacemaker / pre-potential / diastolic depolarization phase; ✓ Threshold voltage for nodal tissue is -40 mV ➤ Phase 0 : Depolarization phase: ✓ Actual action potential starts ➤ Phase 3 : Repolarization phase: ✓ Membrane potential returning back ➤ Phases 1 and 2 Are absent; ✓ Phase 2 may occasionally be present; ➤ Slow rise in slopes in phase 0 seen, hence known as slow-type action potential

Phase	Description
Phase 2 (Plateau)	➤ Exit of K^+ (K delayed rectifier) ➤ Entry of Ca^{2+} by L-type Ca^{2+} channel
Phase 3 (Repolarization)	➤ Opening of K^+ ➤ Closure of L-type Ca^{2+} channel
RMP	➤ Na^+/K^+ ATPase ➤ NCX

➤ Effect of Sympathetic Stimulation



- ✓ Ca^{2+} is returned to sarcoplasmic reticulum via SERCA (Sarcoplasmic Endoplasmic Reticulum Calcium ATPase) transporter
- ✓ Phospholamban protein located close to SERCA pump causes its inhibition → slow relaxation of the heart
- ✓ Positive chronotropic effect ($\uparrow$ heart rate) and positive dromotropic effect ($\uparrow$ conduction velocity) effects occurs at nodal tissue level

➤ Important Information

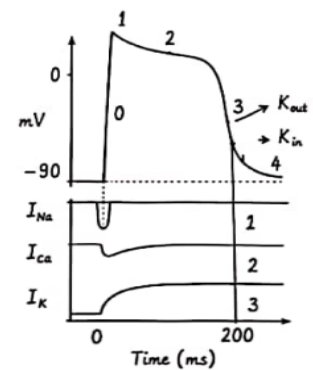
- ✓ Voltage-gated Na^+ channel (Phase 0) is blocked by Class I antiarrhythmic drug
- ✓ Slope of Phase 0 will be slow
- ✓ Potassium channel blocker (Class III antiarrhythmic drug) blocks exit of K^+
- ✓ Increases AP duration
- ✓ Calcium channel blocker (Class IV antiarrhythmic drug) blocks voltage gated calcium channel (phase 2)
- ✓ More effect on nodal tissues

➤ Summary

- ✓ 0: Depolarization phase
- ✓ 1: Initial rapid repolarization
- ✓ 2: Plateau phase
- ✓ 3: Late rapid repolarization
- ✓ 4: Baseline (RMP)

➤ Currents

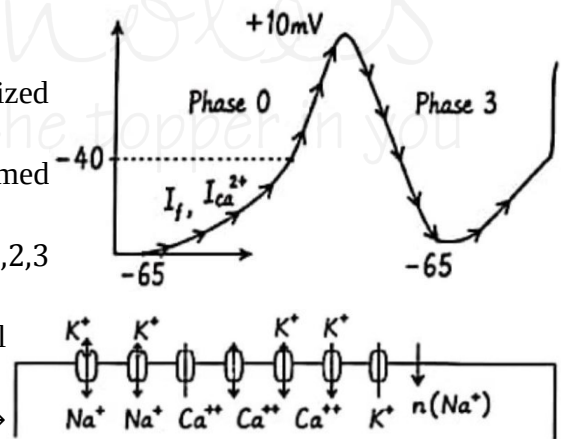
- ✓ Phase 0
 - I_{Na} (Sodium current) is in a downward direction - Entry of sodium
- ✓ Phase 1,2,3
 - I_K (potassium current) is in an upward direction - Efflux of potassium
- ✓ Phase 2
 - I_{Ca} (calcium current) is in a downward direction - Influx of calcium



➤ Ionic Basis Of Slow Type Of Action Potential

✓ Prepotential Phase / RMP Phase / Phase 4

- Membrane voltage at -65 mV
 - ☞ HCN - hyperpolarization induced cAMP nucleotidegated channel opens (slow)
 - ☞ Na^+ enters nodal tissue cell
- HCN 4 is permeable to $Na^+ > K^+$
 - ☞ Na^+ entry $\gg$ K^+ exit
- HCN - peculiar channel (opens in hyperpolarized state)
 - ☞ It is called Funny Channel, current via it is termed Funny Current
 - ☞ Four sub-varieties of HCN channel are HCN1,2,3 and 4
 - ☞ HCN 1,2,3 channels are present at basal ganglia and hippocampus regions
- T-type voltage-gated $\rightarrow$ Ca^{++} channel open $\rightarrow$ influx of Ca^{++} into the cell (Transient rapid)
 - ☞ Cause voltage to rise to -40 mV, (threshold voltage of nodal tissue)



✓ Phase 0: depolarisation phase

- L-type Ca^{++} channels (slow) opens, and the membrane voltage slowly rises
 - ☞ Voltage-gated Ca^{++} channels
 - ☞ Blocked by Class IV antiarrhythmic drug

✓ Phase 3 (Repolarization phase)

- In this phase,
 - ☞ Ca^{++} channel closes,
 - ☞ K^+ channel opens, causing K^+ to leak out of nodal tissue cells and return of membrane voltage to -65 mV

- Opening of the HCN channel → entry of Na^+ into the cell → membrane voltage begins to rise
- Closure of K^+ channels is necessary for prepotential
 - ☞ ↓ permeability of the K^+ channels is required for prepotential

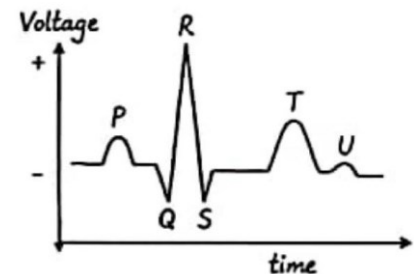
➤ Basics of ECG

✓ Electrocardiogram

- ECG/EKG
- Electrocardiogram: ECG Recording
- Electrocardiograph: Machine recording ECG
- ECG - recording of the change in voltage with respect to change in time

✓ Waves In ECG

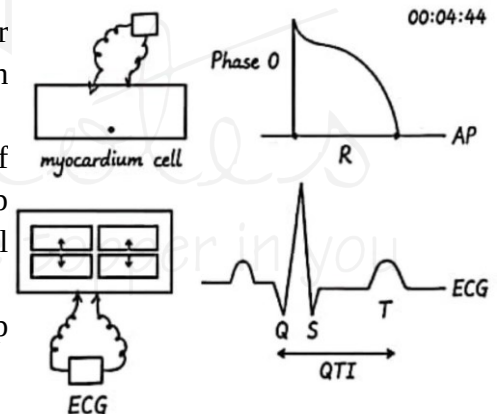
P wave	Atrial depolarization
QRS complex	Ventricular depolarization
T wave	Ventricular repolarization
U wave	Repolarization of the last part of the ventricle (Purkinje fiber)/ papillary muscles of the heart



- P wave - generates due to atrial depolarization
- Q wave - First negative wave that appears after the P wave
- R wave - First positive wave that appears after the P wave
- S wave - any negative wave after the R wave
- In some abnormal ECGs, 2nd positive wave is seen after the S wave – R' wave

➤ Basic Concept

- ✓ An electrode is placed inside a myocardium cell and another outside, with a recording instrument in between, which records action potential of cell → Monophasic
- ✓ 2 electrodes are placed on the surface of group of myocardial cells, electrically interconnected via gap junctions → give surface potential → Biphasic electrical recording i.e., ECG
- ✓ ECG - Summation of action potentials appearing in a group of myocardial cells
 - Surface recording
 - $ECG = \Sigma AP$ (summation of action potentials)



➤ Volume Conductor Principle

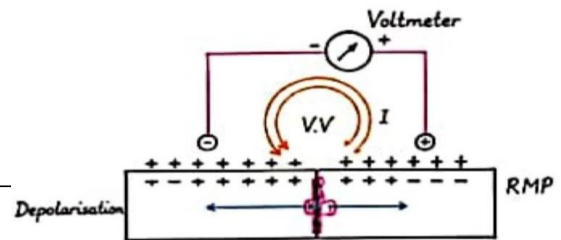
- ✓ Potential/current recorded on the surface of the heart is the same as the potential / current on the surface of the skin
- ✓ Tissue surrounding the heart are good electrical conductor

➤ Normal Action Potential

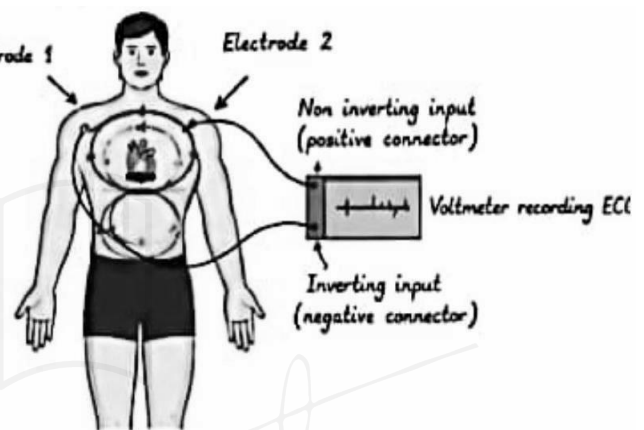
- ✓ Phase 0 - Depolarization phase
 - This phase coincides with appearance of QRS complex in ECG (Q wave)
 - Q wave indicates ventricular depolarization in an ECG
- ✓ Phase 3 -Repolarization phase coincides with the end of the T wave in ECG
 - T wave denote repolarization in ECG
- ✓ Action Potential Duration (APD) - Time duration of the entire action potential
 - Corresponds to the QT interval (QTT)
 - $APD = QT_T = 200 - 400$ milliseconds

- Any parameter that prolongs the AP duration will prolong the QT interval
 - Class III anti-arrhythmic drug (K^+ channel blocker) → slow repolarization → prolongs AP duration.
 - Class I anti-arrhythmic drug (Na^+ channel blocker) → ↓ in slope of phase 0 → prolongs QRS complex duration

- ✓ P wave - generation with the initial depolarization
- ✓ QT wave == the interval between the start of the QRS complex and the end of the T wave
- ✓ ECG electrodes are placed on the surface
 - In ECG, resting membrane potential → positive
 - For normal AP, RMP is negative



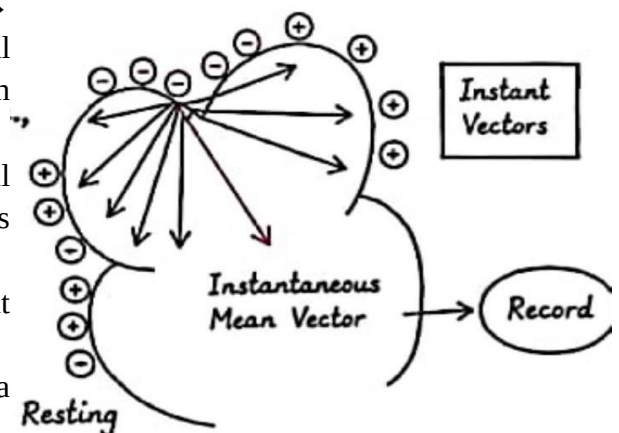
- ✓ In depolarization
 - Inside potential - positive, and the outside - negative
 - ECG electrodes record negative waves
- ✓ When depolarisation spread, some part is in resting condition (outside-positive), some part is depolarized (outside - negative)
- ✓ Separation of positive and negative charge on surface of myocardium → creation of an electrical dipole
 - A tiny current (I) flows from positive to negative charges on the surface of the myocardial cell
 - Connect voltmeter to the positive and negative part → measure potential difference (voltage recording)



- ✓ Voltage - vectorial quantity with a particular amplitude and direction
 - Amplitude of the voltage vector depends on muscle mass, depolarizing simultaneously
 - Direction of the voltage vector - negative → positive
- ✓ ECG - recording of the voltage vector

➤ Instantaneous Mean Vector

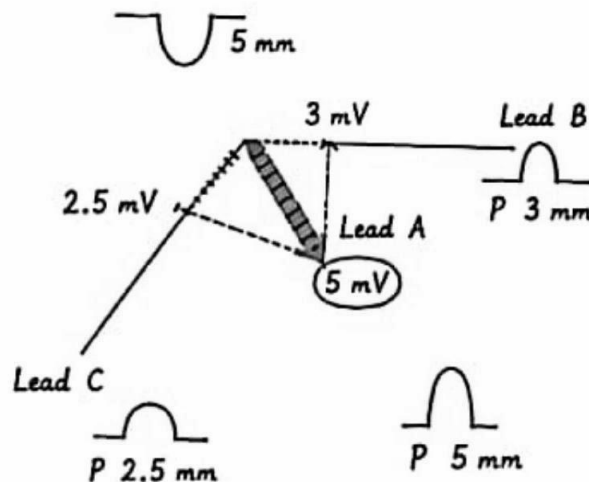
- ✓ When SA node starts generating impulses → depolarization of atria → negative surface potential
- ✓ Some part is still in resting condition → separation of negative and positive part →
- ✓ Multiple Voltage vector generated at any point will be directed negative to positive and are known as instant vectors
- ✓ ECG machine does not record all the instant vectors
- ✓ It records mean of all instant vector created at a point of time i.e., Instantaneous mean vector
- ✓ Instantaneous mean vector - created due to atrial depolarisation
- ✓ If instantaneous mean vector is created in the direction from SA node → AV node → Apex of heart
- ✓ Amplitude - depends upon muscle mass depolarizing simultaneously in both atrium



➤ Generation of P-wave

✓ Example

- Amplitude of Instantaneous mean vector is 5 mV, directed from SA node to AV node
- Place lead A directly facing instantaneous mean vector → give positive deflection with amplitude 5 mm
- Now place lead B in other direction, vector analysis can be done by drawing a perpendicular from the top of the voltage vector to the axis of that lead
 - ☞ Amplitude of voltage vector - 3 mV in direction of lead
 - ☞ Amplitude of waves created - 3 mm
- Place Lead C in another direction, draw perpendicular line from main voltage vector
 - ☞ Amplitude of voltage vector -2.5 mV
 - ☞ Positive wave with deflection - 2.5 mm
- Generation of an instantaneous mean vector is due to atrial depolarization
 - results in P-wave in the ECG
 - ☞ Amplitude of P-wave depends on the direction and amplitude of the instantaneous mean vector
 - ☞ If lead is placed just opposite to instantaneous mean vector → 5 mm deflection but in opposite (negative) direction
 - ☞ If voltage vector is directed towards the lead → Positive deflection
 - ☞ If voltage vector is directed against the lead → Negative deflection



➤ Leads: Standard 12 Lead Ecg

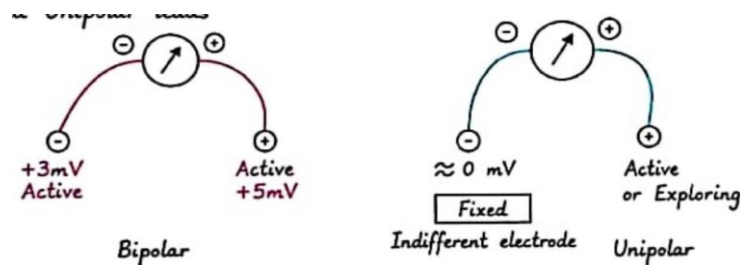
Name of leads	Numbers of leads	Discovered by
Standard Bipolar Limb Leads	Lead I, Lead II, and Lead III	W. Einthoven
Augmented Unipolar Limb leads	aVR, aVL, and aVF	E. Goldberger
Precordial Unipolar leads/ Chest lead	V1, V2, V3, V4, V5, and V6	Frank Wilson

- ✓ aVR - Augmented voltage of right arm
- ✓ aVL - Augmented voltage of left arm
- ✓ aVF - Augmented voltage of left foot
- ✓ Total - 3 bipolar leads & 9 Unipolar leads

➤ Bipolar & Unipolar Leads

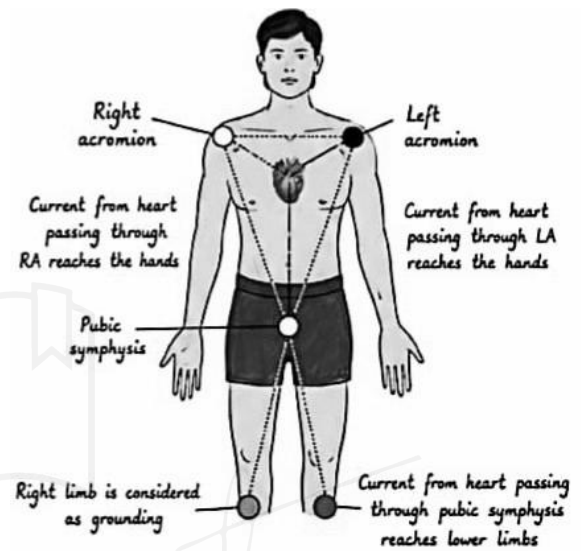
Bipolar lead electrodes	Unipolar leads electrodes
➤ Both leads (negative and positive) are active i.e., both leads are picking some voltage from skin ➤ Net voltage recorded by voltmeter is the difference between 2 active voltage points ➤ E.g., positive end: +5 mV Voltage ✓ Negative end: +3 mV voltage ✓ Net deflection = $+5 - (+3) = +2$	➤ The negative end and positive end are connected on surface of skin ➤ But negative end is connected in such way that the voltage/ potential is ~ 0 i.e., fixed voltage/ indifferent → known as indifferent / Inactive electrode ➤ Positive end records active voltage from surface → Known as the active or exploring electrode

➤ Bipolar leads and Unipolar leads

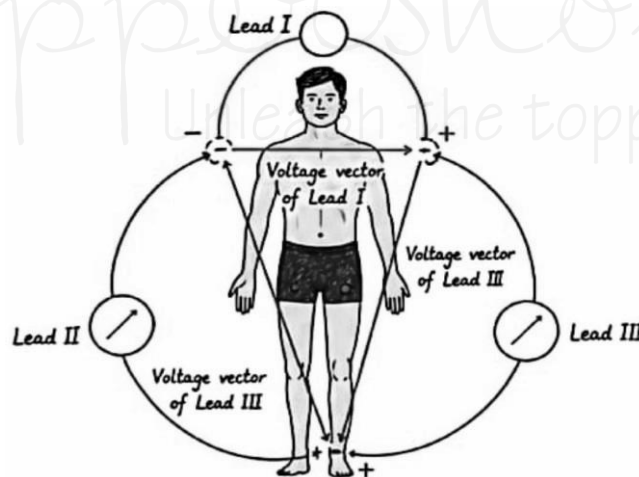


➤ Lead Placement

- ✓ 3 reference point: right acromion, left acromion, and symphysis pubis → Forming an equilateral triangle
- ✓ In a standard ECG, leads are placed above the wrist
- ✓ Human body acts as a volume conductor
 - Electrical activity flowing at point of left acromion → same electrical activity will flow throughout whole left arm point → same recording throughout whole arm
 - Electrical activity flowing through right acromion → same electrical activity throughout right arm → same voltage
 - Electrical current flowing through symphysis pubis → same electrical current will flow in left foot
- ✓ As per Einthoven's convention, the current from the left leg is measured, and the right leg is taken as the grounding electrode



➤ Standard Bipolar Limb Leads

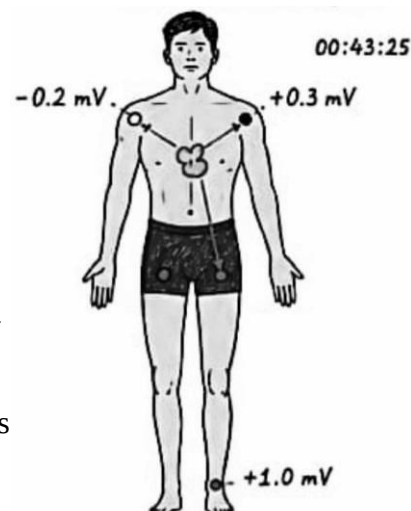


Leads	Lead I	Lead II	Lead III
Connection of voltmeter	➤ Negative end → Right arm ➤ Positive end → Left arm	➤ Negative end → Right arm ➤ Positive end → Left foot	➤ Negative end → Left arm ➤ Positive end → Left leg
Net voltage	➤ Potential difference between left arm - right arm	➤ Potential difference between left leg - right arm	➤ Potential difference between left leg - left arm
Voltage vector direction	➤ Negative → positive	➤ Negative → positive	➤ Negative → positive

➤ All three leads form an equilateral triangle with heart at its centre, known as Einthoven's triangle

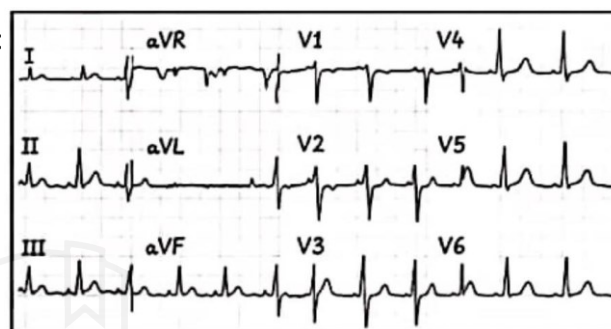
➤ Einthoven Law

- ✓ Assume
- ✓ Voltage spreading at left acromion: $+0.3 \text{ mV}$.
- ✓ Voltage flowing at right acromion: -0.2 mV
- ✓ Voltage flowing at left foot: $+1.0 \text{ mV}$
- ✓ Voltage recorded by each lead is calculated as below:
 - Lead I: Left arm - right arm = $(+0.3) - (-0.2) = +0.5 \text{ mV}$
 - Lead II: Left foot - right arm = $(+1.0) - (-0.2) = +1.2 \text{ mV}$
 - Lead III: Left leg - left arm = $(+1.0) - (+0.3) = +0.7 \text{ mV}$
 - Lead II voltage = Lead I + Lead III voltage, known as Einthoven law

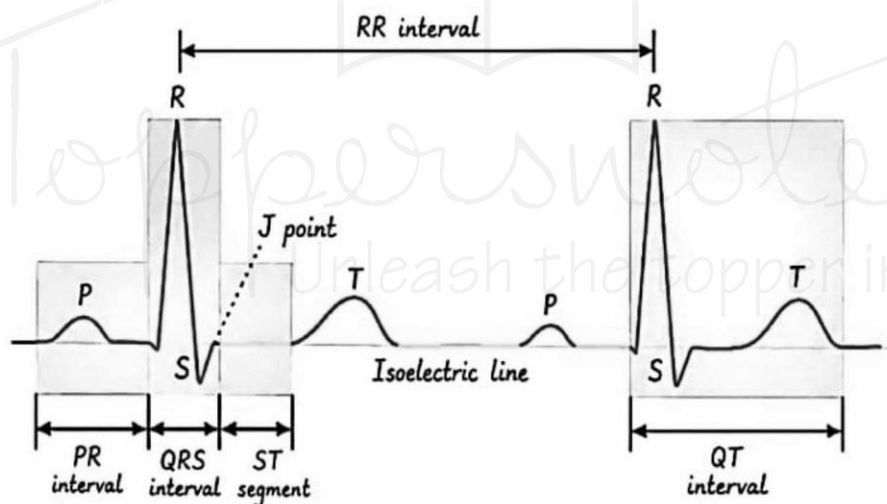


➤ Normal ECG

- ✓ Lead I, II, III: Bipolar, standard limb leads: Biphasic
- ✓ aVR: All negative deflection
- ✓ aVL/aVF: Biphasic
- ✓ V1, V2: No Q wave, deep S wave
- ✓ V3, V4: Biphasic
- ✓ V5, V6: Small Q wave, tall R wave



➤ Parametres In Normal Ecg



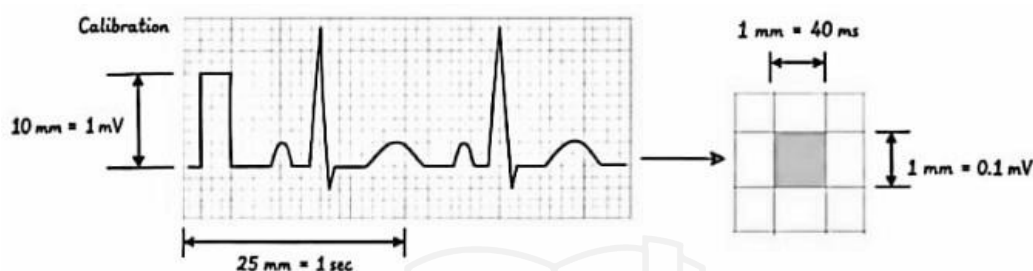
- ✓ Isoelectric line - no current is flowing on the surface of the heart
 - Seen after complete depolarization of ventricle (ST segment) and after complete repolarization (TP segment)
 - Baseline of the ECG

➤ Waves and Intervals

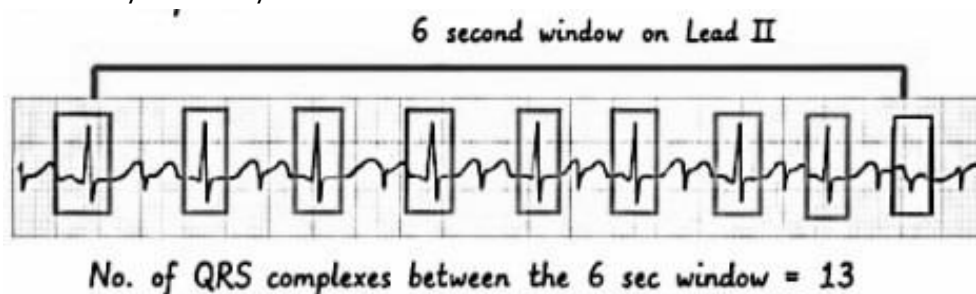
- ✓ PR interval starts from the onset of the P wave up to the onset of the Q wave
- ✓ If Q wave is absent, then the onset of the R wave is taken
- ✓ QRS interval/ QRS complex
- ✓ J point → End of the S wave, and an isoelectric line (ST segment) is starting
- ✓ RR interval → From peak of one R wave to the peak of the next R wave
- ✓ ST segment
- ✓ QT interval

Waves/Interval	Physiological correlates	Amplitude	Duration
P wave	Atrial depolarization	0.1 – 0.3mv	< 0.1Sec
PR interval	Time between the start of atrial depolarization to start of ventricular depolarization		0.12 to 0.2 Sec
Q wave	Septal depolarization		
R wave	Ventricular depolarization	1 mv	0.06 to 0.10 Sec
S wave	Depolarization of the base of ventricles		
ST segment	Depolarization complete. Isoelectric line		0.08 – 0.12Sec
T wave	Repolarization of both the right and left ventricles	0.2 to 0.3 mV	
QT interval	Ventricular depolarization and repolarization		0.2 to 0.4 Sec

➤ Calculation of the Heart Rate from ECG



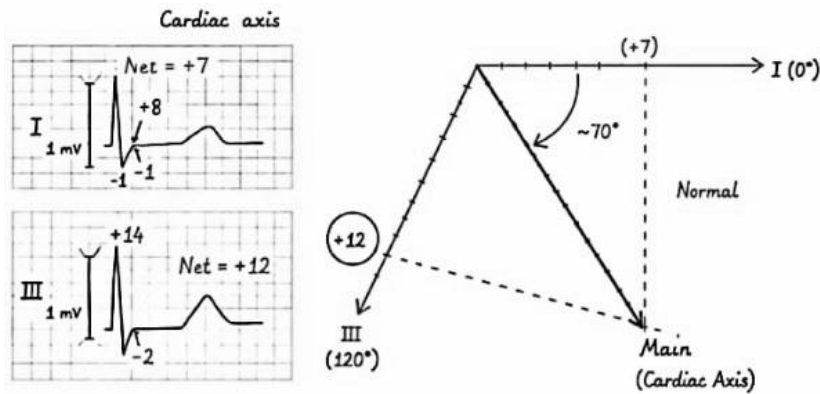
- ✓ ECG recording paper records at 25 mm/sec speed
- ✓ Smallest square boxes measure 1 mm × 1 mm
 - Duration of 1 mm/1 smallest square is $1/25 = 0.04\text{sec}$
- ✓ The X-axis is the time axis, and the Y-axis is the voltage axis on the ECG paper
 - 1 mV of voltage gives 10 mm of deflection on the Y-axis
- ✓ Voltage of each square on the Y-axis will be 0.1 mV
 - If speed of ECG paper is 25 mm/sec, then in 60 seconds
 - Distance covered = $25 \times 60 = 1500\text{ mm}$
- ✓ Heart rate is calculated by R – R interval in mm/smallest box
- ✓ Approximately RR interval - 20 small boxes / 20 mm
 - $HR = 1500/20 = 75/\text{min}$



- ✓ If ECG is irregular, particularly take a long lead II recording
- ✓ In long lead II calculate 6-second (1sec – 25 mm), i.e., 150 mm (6 seconds × 25 mm)
- ✓ In 6 sec window period, calculate how many QRS complex is there
 - In this ECG in 6 sec window period - 13 QRS complex
 - No. of QRS complex between 60 sec window = $13 \times 10 = 130$
 - So, heart rate – 130/min

➤ Cardiac Axis Calculation

- ✓ Any two leads are taken e.g., Take Lead I and III
- ✓ Lead I is directed at 0° and lead III is directed at 120°



- ✓ Calculation of the net deflection (positive and negative deflections) of the QRS complex
 - Net deflection in lead I = $+8 - 1 = +7$
 - Net deflection lead III = $+14 - 2 = +12$
- ✓ Cardiac axis - calculate direction of main cardiac vector
 - Plot perpendiculars on lead I and lead III axis
 - Connect centre with cutting point → Direction of main vector
 - Direction and amplitude of main cardiac vector is cardiac axis
 - Direction of cardiac axis is calculated with respect to 0°
 - Angle of this cardiac axis $\sim 70^\circ$
 - Amplitude is calculated in same scale used for calculation of Lead I and lead III

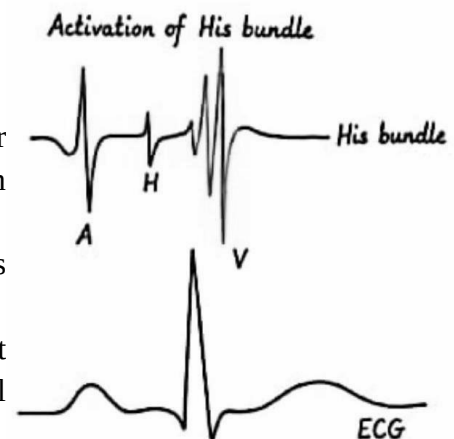
➤ Main Cardiac Axis

- ✓ Direction from -30° and $+100^\circ$
 - If main cardiac vector remains within this zone → Normal cardiac axis

Left axis deviation	Right axis deviation
➤ Cardiac axis is $> -30^\circ$ ➤ Causes ✓ Normal variant ✓ Left ventricular hypertrophy ✓ Left anterior fascicle block ✓ Inferior wall myocardial infarction ✓ Lateral infarction	➤ Cardiac axis is $> +100^\circ$ ➤ Causes ✓ Normal variant ✓ Right ventricular hypertrophy ✓ Left posterior fascicle block

➤ His Bundle Electrogram

- ✓ Invasive ECG recording
- ✓ Lead has to be placed close to the AV node
- ✓ Introduce catheter from peripheral vein with tip of catheter carrying this lead which should be introduced in left atrium close to AV node
- ✓ This lead gives better picture of AV nodal depolarization as well as Bundle of His depolarization
- ✓ When impulse travel from SA node to AV node, it first depolarizes the upper part of AV node i.e., AV nodal depolarization which will be picked up by this lead



A-wave	H-wave	V-wave
➤ AV nodal depolarization	➤ Bundle of His electrogram depolarization	➤ Ventricular muscle (septum) depolarization

- ✓ *P* wave (atrial depolarization) will start earlier than the *A* wave

PA interval (30 ms)	AH interval (100 ms)	HV interval (40 ms)
➤ Time interval between the onset of the <i>P</i> wave to the onset of <i>A</i> wave	➤ Time interval from <i>A</i> wave up to the <i>H</i> wave (AV nodal delay = 100 ms)	➤ Time interval between <i>H</i> and <i>V</i> waves ➤ His Bundle delay: Duration taken for transport of electrical activity through Bundle of His

- ✓ *PR* interval = *PA* interval + *AH* interval + *HV* interval
- ✓ Prolonged *PR* interval → heart block
- The block could either be at the level of the AV node or the bundle of His
 - His bundle electrogram is a must to pinpoint the site of the block

Cardiac Cycle

Introduction

- Cyclical events at the level of ventricles
- Depolarization occurs first in the left ventricle, it contracts earlier than the right ventricle
- Ejection of blood occurs first in the right ventricle than the left ventricle

Duration Of The Cardiac Cycle

- The total duration of the cardiac cycle is 0.8 seconds

$$\text{Duration} = \frac{60 \text{ seconds}}{\text{Heart Rate (70)}}$$

Duration of the ventricular cycle	Duration of the atrial cycle
- Systole - 0.3 seconds	- Systole - 0.1 seconds
- Diastole - 0.5 seconds	- Diastole - 0.7 seconds

Duration of Cardiac Cycle In A Person Who Is Exercising

- Exercise induces an immediate ↑ in the heart rate
- ↑ Heart rate - ↓ Total duration of the cardiac cycle
- When the total cardiac cycle duration ↓, both the systole & diastole duration will decrease (diastole >>> systole)

Phases of the Cardiac Cycle

Diastole	Systole
- Protodiastole - Isovolumic relaxation (IVR) - First rapid filling phase (FRFP) - Diastasis - Last rapid filling phase (LRFP)	- Isovolumic contraction (IVC) - Rapid ejection (RE) - Slow ejection (SE)

Heart Sounds

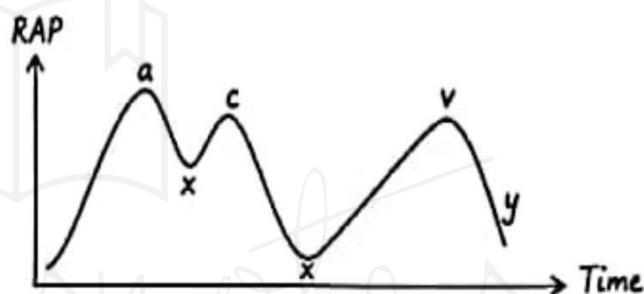
	First heart sound (1 ST HS)	Second heart sound (2 ND HS)
Phase	- The first heart sound is <i>d/t</i> the closure of the atrioventricular valve - Thus, the first heart sound occurs at the beginning of isovolumic contraction	- The second heart sound is <i>d/t</i> the closure of the semilunar valve - The second heart sound thus happens at the start of the isovolumic relaxation
Duration	- The first heart sound has a longer duration of 0.15 seconds	- The duration of this heart sound is shorter, 0.12 seconds

Frequency	- It is a low-frequency sound of 25 – 45 Hz	- It is sharp. The frequency of the sound is also very high, i.e. 50 Hz
Amplitude	- It is loud and has a high amplitude	- It is soft. The amplitude of the second heart sound is low
ECG	- At the peak of the R wave of the ECG	- At the end of the T wave

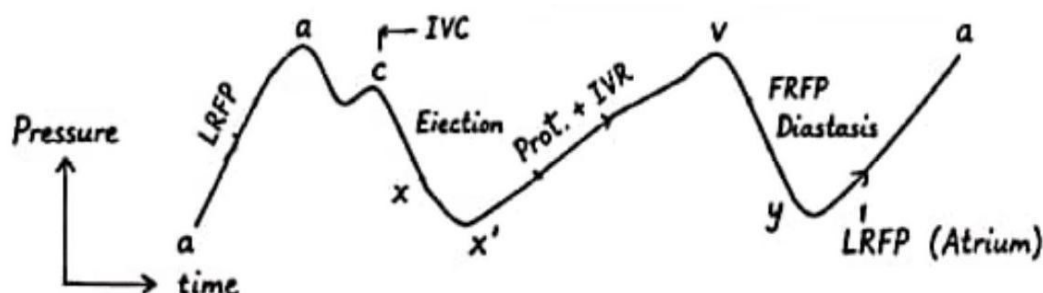
Audible third heart sound	Audible fourth heart sound
➤ Hyperdynamic Circulation: Blood runs in the heart's cavity with a higher velocity, which causes greater vibration and thus, audible third heart sound ✓ For example – In children, because of high heart rate, anemia, pregnancy ➤ Increased Volume of Blood: This is seen in patients with congestive cardiac failure, causing dilation of the chamber of the heart	➤ D/t the ↑ stiffness of the left ventricular wall. ✓ When blood rushes through the ventricular cavity, vibrations are caused. When the wall stiffness, the sound of vibration becomes louder ✓ Example : Fibrosis after myocardial infarction or even hypertrophy

Jugular Venous Pressure (Jvp)

- It is the right atrium pressure/ central venous pressure during different phases of the cardiac cycle - Clinically recorded from the internal jugular vein
- Left atrium pressure is recorded by pulmonary capillary wedge pressure technique
- X-axis: time
- Y axis: RAP change



Positive	<i>a</i> → Atrium Contraction	Last rapid filling phase.
	<i>c</i> → bulging of tricuspid valve into RA cavity	Isovolumic contraction phase
	<i>v</i> → Increase in pressure and volume of the right atrium	Protodiastole and isovolumic relaxation
	<i>x</i> → Relaxation of Atrium	Between the last rapid filling phase and isovolumic contraction phase
	<i>x'</i> → Pulling of the tricuspid valve towards RV during ventricular systole	Ejection phase
	<i>y</i> → Opening of tricuspid valve	First rapid filling phase and the diastasis phase



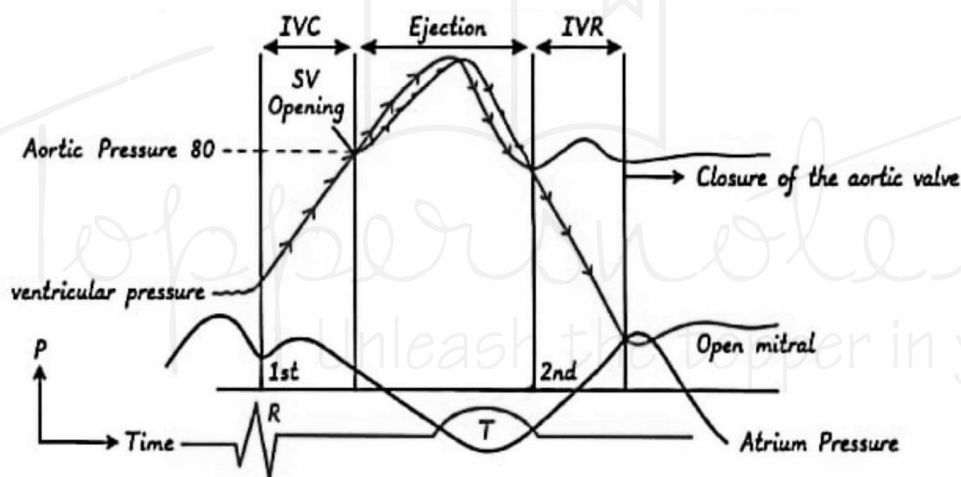
Abnormal JVP

- Prominent a wave:
 - ✓ Tricuspid stenosis
 - ✓ Pulmonary hypertension
- Canon a wave:
 - ✓ Complete heart block
 - ✓ Junctional rhythm
- Absent a wave: Atrial fibrillation
- Large v wave: Tricuspid regurgitation
- Sharp y descent: Constrictive pericarditis (Friedreich's sign)
- Sharp x descent: Tamponade & constrictive pericarditis

Changes In Pressure During Cardiac Cycle

- The X-axis denotes time & the Y-axis denotes pressure
- The gaps between the lines are time gaps between different events in a cardiac cycle

Red Line	Blue Line
➤ Denotes the ventricular pressure ✓ It is the end of diastole and the beginning of systole ✓ The end-diastolic pressure is 4–12 mm Hg in the left ventricle and 1–7 mm Hg in the right ventricle	➤ Denotes aortic pressure ✓ The first phase here is the beginning of isovolumic contraction ✓ The pressure at the arch of the aorta at the end-diastole (before IVC) is 80 mmHg



Aortic Ventricular Pressure Curve

- **IVC phase:**
 - ✓ On contraction, the ventricular pressure rapidly rises
 - ✓ The aortic pressure remains the same, and at a certain point, the ventricular pressure rises above the aortic pressure, thus opening the semilunar valve
- **Ejection phase:**
 - ✓ Blood is ejected out through semilunar valves while ventricular pressure continues to rise during contraction
 - ✓ Aortic pressure ↑ but remains lower than the ventricular pressure
 - ✓ When the ejection continues, the ventricular pressure will start falling, & the aortic pressure will also start falling proportionately
 - ✓ However, now the aortic pressure is more than the ventricular pressure, but the ejection continues
 - ✓ Now, the ventricular pressure is lower than the aortic pressure
 - ✓ This is when the blood will try to fall back from the arch of the aorta
 - ✓ The immediate closure of the aortic valve prevents this

➤ Isovolumic relaxation phase:

- ✓ The ventricle relaxes increasingly & pressure thus falls to near 0 mmHg
- ✓ This is the point where the filling of the ventricle will start again
- ✓ During the closure of the aortic valve, the aortic pressure rises & falls a while later & then remains constant, which is the diastolic pressure
- ✓ The mitral & tricuspid valves open, the ventricular filling starts again, & the pressure will rise again

Atrium Pressure Curve (Jvp)

- The "a" wave is followed by the "c" wave in the isovolumic contraction phase
- During the ejection, the descent continues due to low pressure
- The "v" wave is plotted at the end of isovolumic relaxation
 - ✓ Here, the ventricular pressure is 0 mmHg, and the atrium pressure is high
 - ✓ This leads to the opening of the mitral valve, causing a sudden decrease in atrial pressure

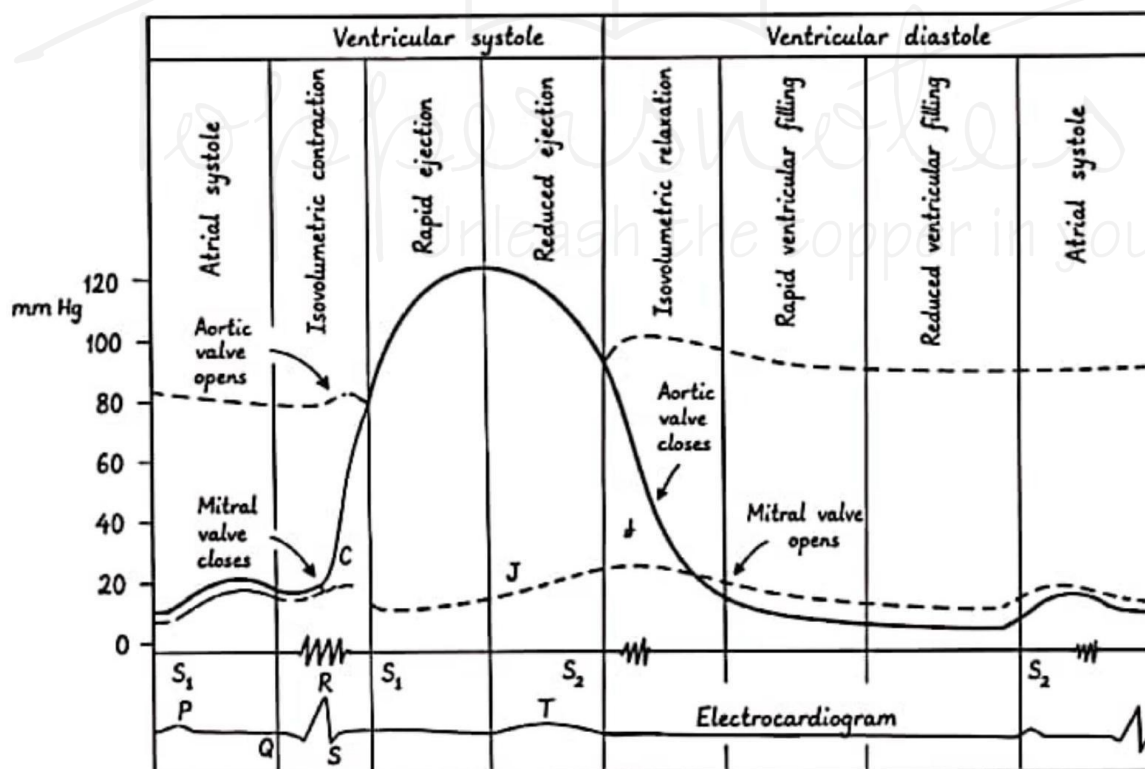
Heart Sounds

- First HS is heard at the beginning of IVC
- Second HS is heard at the end of diastole
- The instrument used is Phonocardiogram

ECG

- First HS corresponds to the peak of the R wave
- Second HS corresponds to the end of the T wave

Wiggers Diagram



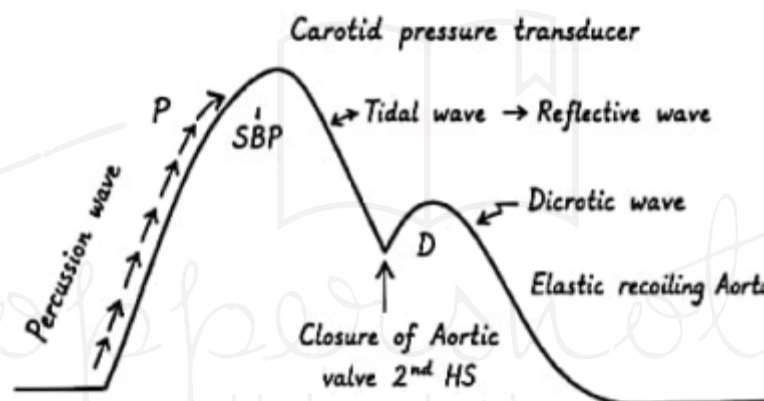
Electro-Mechanical Systole

- Beginning of electrical activity of ventricle (depolarization)
 - ✓ Q wave
- Electrical activity is followed by mechanical activity
 - ✓ Ending of mechanical activity is 2nd HS
 - ✓ Semilunar valves close

- QS^2 is known as the electromechanical systole
- The time duration between the onset point of ejection and end of ejection (2nd heart sound) of the dicrotic notch in the aortic pressure curve is known as the Left ventricular ejection period (LVEP)
 - ✓ It lasts from the opening to the closing of the semilunar valve
- The pre-ejection period (PEP) is from the onset of the Q wave to the onset of the left ventricular Ejection
 - ✓ $PEP = QS_2 - LVEP$
- Important Information
 - ✓ $\frac{PEP}{LVEP} = 0.35 \propto \frac{1}{EF}$
- It is to be noted that the pre-ejection period/left ventricular ejection period has a ratio of 0.35 and is inversely proportional to the heart's ejection fraction

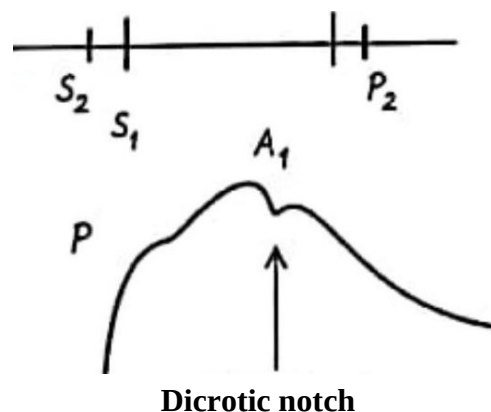
Aortic Pulse

- The aortic pressure curve is typically recorded at the level of the carotid artery by placing a carotid pressure transducer
- With the onset of ejection, there is a rapid rise in aortic pressure which is known as a percussion wave
- The fall in aortic pressure leads to the closure of semilunar valves, producing a notch in the curve known as the dicrotic notch

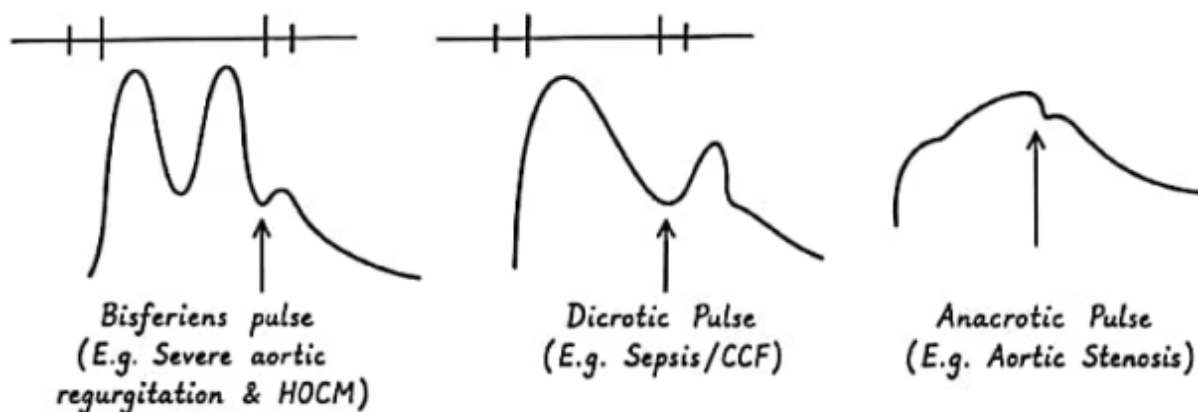


- Upon valve closure, due to elastic recoiling, the aortic arch returns to its original position, resulting in a notable elevation of aortic pressure
 - ✓ This is represented by the dicrotic wave
- From the beginning of ejection upto dicrotic notch - Systole
- Diastole starts after the dicrotic notch
- Following ejection, the aortic pressure drops, but elastic tissue in the aortic arch might cause its expansion & can lead to a temporary rise in pressure by reflecting the blood back from the wall
 - ✓ This reflective wave is called the tidal wave

Normal Carotid Pulse



Abnormal Pulse



Measurement of Cardiac Output

Methods	Explanation
Fick Principal method	- Best method for low cardiac output states of the body - Invasive method
Dye dilution and thermodilution methods (Both are based on the same principle)	- The principle for these were discovered by two scientists, Stewart and Hamilton - It is also known as the Stewart-Hamilton method. - Dye dilution is also called the indicator dilution method and is the older version. It was replaced by a newer thermodilution method - The thermodilution method is done by inserting a catheter at the level of the pulmonary artery, hence known as the pulmonary artery catheter method. - Cold saline solution is injected into the chamber of the heart - The thermodilution method is the gold-standard method - It works best for the high cardiac output state of the body. - Invasive method
Echocardiography	- It is a noninvasive method and is the most common method of measuring CO - It is also the least accurate method
Impedance cardiography or Thoracic Bioimpedance	- It is done by placing electrodes on the surface of the chest. - A low amplitude current is passed. - The error percentage by this method is 20%.
Velocity Encoded Phase Contrast MRI	- It is the most accurate method for measuring CO - Currently, it is not used clinically, only for research purposes

Fick's Principle

$$CO = \left\{ \frac{O_2 \text{ absorption at lung (min)}}{(Artery O_2 - Venous O_2) \text{ Content}} \right\}$$

- Oxygen consumption is measured using spirometer
- Blood sample from the artery and vein is taken
 - ✓ Oxygen content is measured.
 - ✓ Mixed venous blood sample is taken from the pulmonary artery
 - ✓ Brachial artery blood is taken for arterial blood

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Fick's Principle

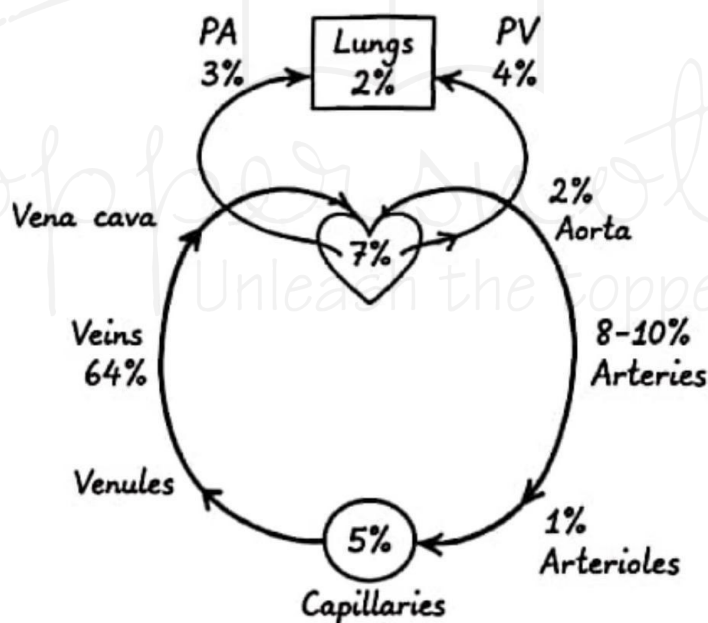
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- Oxygen consumption is measured using spirometer
- Blood sample from the artery and vein is taken
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Dye Dilution Method

- $CO = \left\{ \frac{\text{Amount dye (mg)} \times 60}{\text{time (sec)} \times \text{Avg. Conc. (mg/ml)}} \right\}$
- Dye is injected into the heart chamber → Dye circulates through systemic circulation → Dye concentration measured in multiple arterial blood samples
- After a certain point, the concentration of dye in the artery will be 0
 - ✓ The time interval is measured
 - ✓ Average concentration of dye in arterial blood is measured
 - ✓ Amount of dye injected is measured
- Since the cardiac output is measured in minutes, we multiply it by 60

Hemodynamic

Distribution of blood volume



- Total cardiac output in a person with 75 kg weight: ~ 5.4 L.

Central Circulation

Blood distribution	Blood volume %
Heart Chambers	7% of Total Blood volume
Pulmonary Artery (PA)	3% of Total Blood Volume
Pulmonary Capillaries	5% of Total Blood Volume
Pulmonary Vein (PV)	4% of Total Blood Volume

- Blood in the central circulation (i.e., the lungs & the heart)
- Contains 16% of the total blood volume
- Remaining part of circulation → peripheral circulation