ELECTRO CARDIO GRAM [ECG]

ECG :-

Electrical activity of ht is graphically presented.

— ECG machine works on the principle of galvanometer.

(1) When no electrical activity → Galv needle at O position in myocardium
↳ when all cells are at RMP - no Action pot.
or when all are completely depolarised

(2) Whenever positive charges move toward +ve electrode needle is deflected towards +ve side.

(3) When myocardium thickness is ↑ → more +ve charges enter on depolarisation → A longer vector ie: a larger EMF.
+ve deflection larger

(4) When +ve charges (wave of depolarisation) move away from +ve electrode — -ve deflection.

☆☆ Last pt to depolarise is first to repolarise

(5) When -ve charges move towards -ve electrode
+ve deflection of needle.

— **Moderate conductor**
deflection is with moderate vel.

Fast conductor
Needle is deflected with high velocity.

Electrical events during one cardiac cycle ⟹ Cardiac Vectors ⟹ ECG

① Atrial depolarisation → Atrial Vector downwards & left
Small depolarising vector ← Weak and deflects moderately.

Note :- The only electrical window B/W Atria & ventricles is AVN.

② Stimulation of AVN :- Spl slow conduction tiny tissue

Note :- ~~Purk~~ Purkinje fibre - fast conduction

	AVN	PF
i)	Cells are placed at right angle to direction of current	opp to AVN
ii)	Many cells & small cells	Large cell & less cell
iii)	No of gap junct is very less	More [too many]
iv)	depolarisation dependent on ~~Ca~~ Ca^{2+}	Na^+
v)	Cells - less diameter More resistance	opp to AVN
vi)	RMP -60 mV	-90 mV

Cation influx is fast.

When depolarising current is passing thru AV node
- It is very slow
- Electrical Vector is very small it being a tiny tissue.

ECG does not pick it up.
It is not detected.

③ Depolarisation of Ventricles :-

3 stages
- a) Rapid Inter ventricular depo
- b) Major vent depo
- c) Basal vent depo.

a) Septum is stimulated by Lft bundle branch.
∴ Electrical current moves upwards & towards right
Vector is small upward & rightward.
but it is fast.

b) Inner myocardium is first stimulated
The wave then moves outwards from inner to outward myocardium
Lft ventricle thick ⇒ vector is stronger
→ strong
Lft — vector is down & left
Rt — " " & right
→ weak
} Both add up
↓
Net vector is directed down & left

c) Basal vectors — small upward & rightward for both.

☆ Repolarisation Activity in the Atria is masked by depolarisation of ventricles.

Electrical events of Hrt → conducted to body surface.
Hence electrodes can be applied on body surface to record ECG

+ve electrode — Left foot
-ve " — Right hand

① Atrial vector — Moves towards +ve electrode
+ve deflection with moderate vel
P wave

② AVN depolarisation — Needle straight
electrically silent.

③ Septal depo vector — towards -ve electrode
-ve deflection small & fast
Q wave

④ Major vent depo vector — Moves towards +ve electrode
+ve deflection strong & fast
R wave

⑤ Basal Vent depo vector — towards -ve electrode
-ve deflection small & fast
S wave.

Note :- At end of QRS → Ventricles completely depolarised.
No current → No deflection of needle.

ST segment

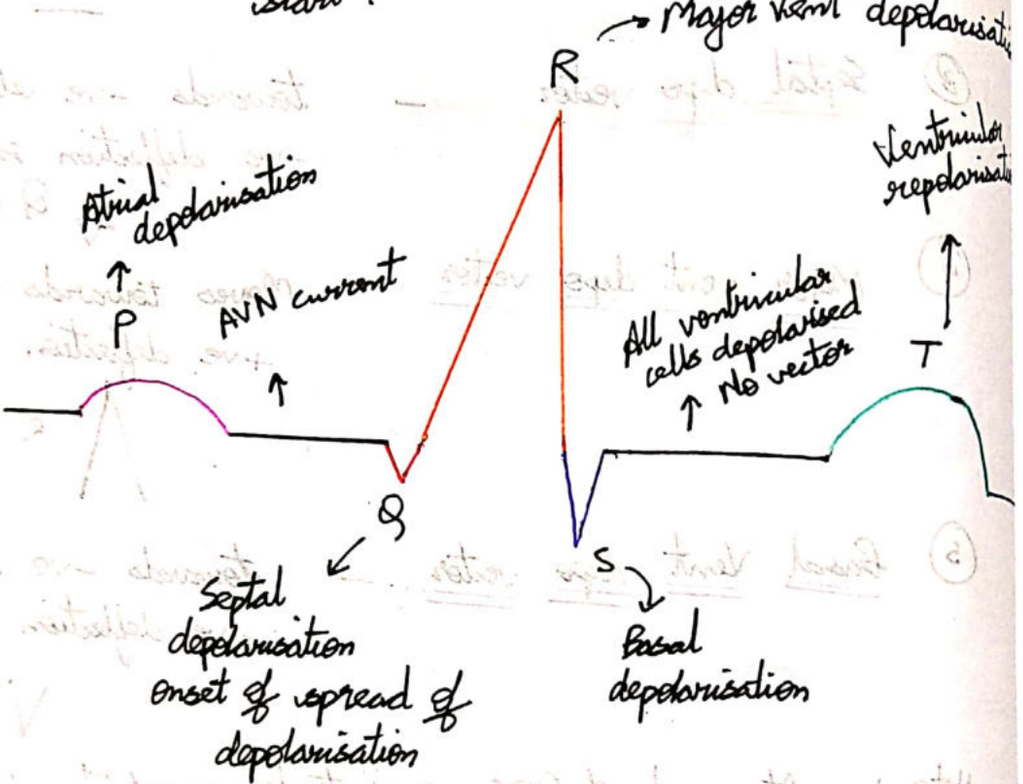
⑥ Ventricular repolarisation :- Repolarisation moves from out to inwards.
Net Repolarisation vector is directed right & upward. T wave
↙ slow
Slow process - gradual.
↳ +ve deflection

Note :- Ventricular depolarisation is represented by 3 waves since its a fast process. Septal Vectors are separate.
Whereas repolarisation is rep by one wave since its a gradual process. the vectors overlap.

PR segment :- Isoelectric line starting at P wave & ending at beginning of Q wave.

→ Rep AV nodal activity

ST segment :- Time during which whole ventricle is depolarised and Repolarisation is yet to start.



Phase 0 – QRS complex

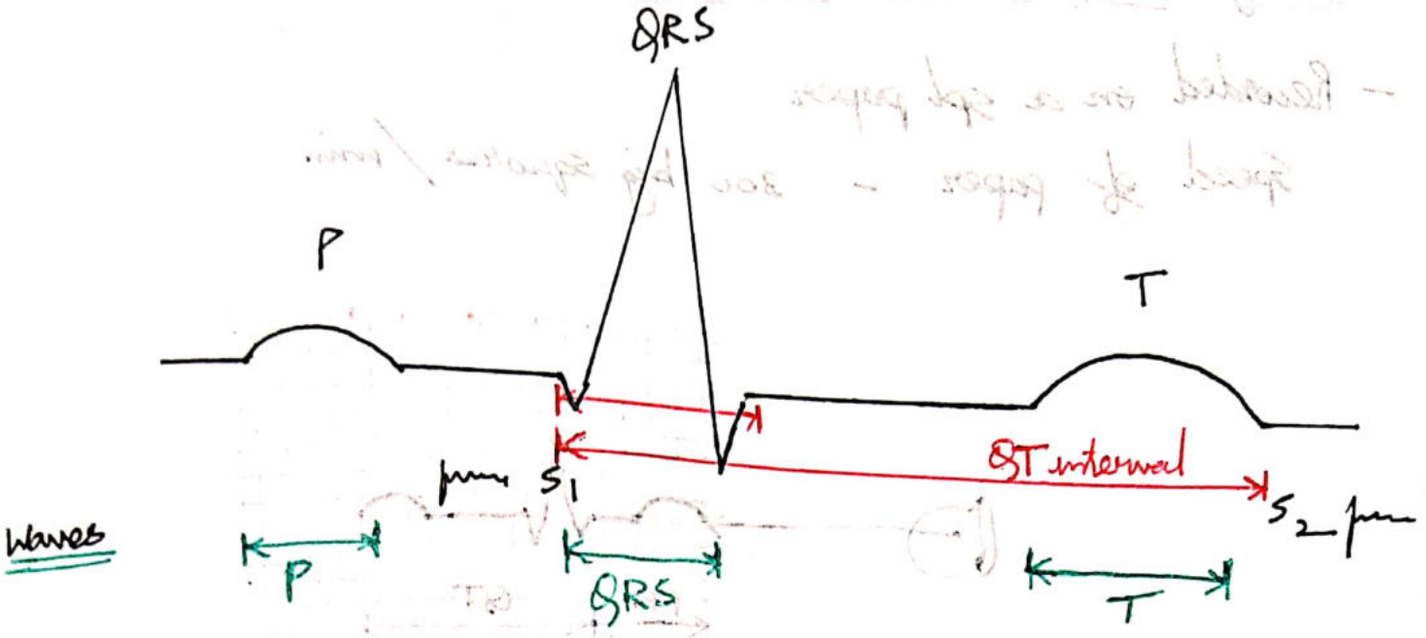
Plateau phase – ST segment

Phase 3 – T wave

Waves :- deflections of the needle during ECG recording
Patterns prod on ECG due to deflection of needle.

Intervals :- combination of waves or segments eg QRS

Segment :- Isoelectric event when needle is not moving



↳ It shows beginning of vent depolarisation and ends with end of vent repo.

Beginning of Action pot to terminating AP

Rep Ventricular systole.

Note :- After T wave sometimes we see 'U' wave

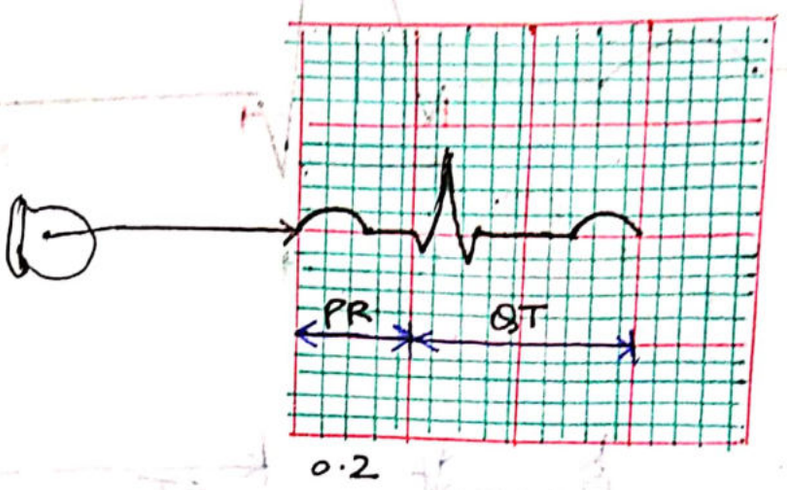
A small wave rep electrical activity in papillary muscle.

Sometimes elec activity of Papillary muscle is out of phase with the rest of muscle

Timings and duration of ECG :-

- Recorded on a spl paper

Speed of paper - 300 big squares / min



300 big squares — 60 sec.

1 big square = $\frac{60}{300}$ = 0.2 sec

1 big sq = 5 small sq = 0.2 sec

1 small sq = $\frac{0.2}{5}$ = 0.04 sec

P wave - 2½ small sq = 0.1 sec

PR segment - 2½ small sq = 0.1 sec

PR interval = 0.2 sec.

QRS complex - 2½ small sq = 0.1 sec.

QT interval = 0.4 sec.

MASTERING ECG LEADS :-

Bipolar limb leads :-

- **ECG leads :-** Basically a set of electrodes with wires connected on one side to ECG machine and other side the electrodes are attached to body surface.
  Purpose is to record the electrical activity of hrt.
- Body is a vol conductor ∴ it can conduct currents. Hence the electrical currents generated by the ~~body~~ hrt spred thru out the body.
- We can put electrodes at any two pts of the body ∴ when the current is flowing, these electrodes will measure the pot diff B/w the # two pts.

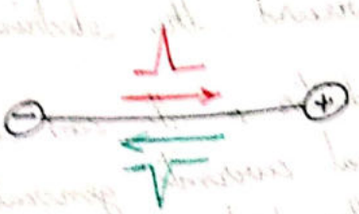
  +ve electrode ⇒ Exploring electrode
  -ve " ⇒ ~~Reference~~ "
  zero potential ⇒ Reference "
- **Circuit of lead :-** the +ve ∴ -ve electrode along with their wires are attached on one side to ECG mach ∴ the other the electrodes are applied on the body surface forming a circuit.
- The leads are applied on the wrist for convinience
- Since the lead is having one +ve electrode ∴ other -ve electrode which form two poles of the lead it is called Bipolar lead
- **Electrical Axis of the lead :-** It is determined by the placement of electrode on body
  An imaginary line connecting the two electrodes drawn from -ve / Ref electrode towards +ve electrode

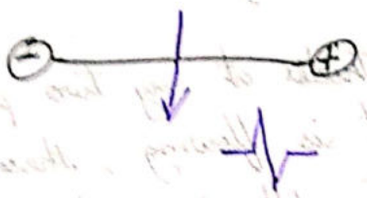
- Pattern of the lead :-

① when electrical vector moves towards exploring electrode => deflection upward ↑

② when electrical vector moves ⊥ to axis of lead => Net deflection is zero
+ve & -ve deflection in eq magnitude

The electrical activity recorded by the lead. is called Pattern of the lead.





- Limb leads scan the electrical activity in frontal plane
Chest " " " " " horizontal plane.

- When there are cardiac pathologies which result in alteration of electrical activity of the hrt., to find where is the pathology we need to look the cardiac vector at different angles.
We need to look from multiple leads.

- When multiple leads are used :-
different leads are having different electrical voltage projected from the QRS cardiac vector to the electrical axis of the lead.

- I lead system :- +ve on left arm -ve on right arm
II " " :- +ve on left leg -ve " " "
III lead " :- +ve " " " -ve on left arm

Einthoven' arranged this system so that major QRS complex should be +ve.

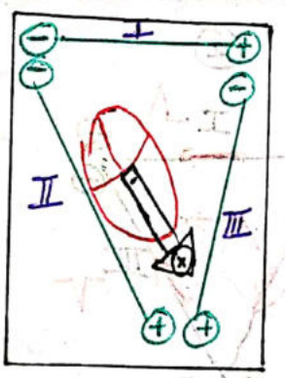
- To get mainly +ve deflection for QRS complex the +ve electrode must be placed downward or leftward
  ∵ QRS vector is directed downwards & leftwards.

- Limb leads < 3 bipolar limb leads
  3 Unipolar limb leads. } Conventional leads.
  Chest leads - 6

- Einthoven's triangle :-

Imaginary Δle around the brt.

Δle on the frontal plane of the body's surface.



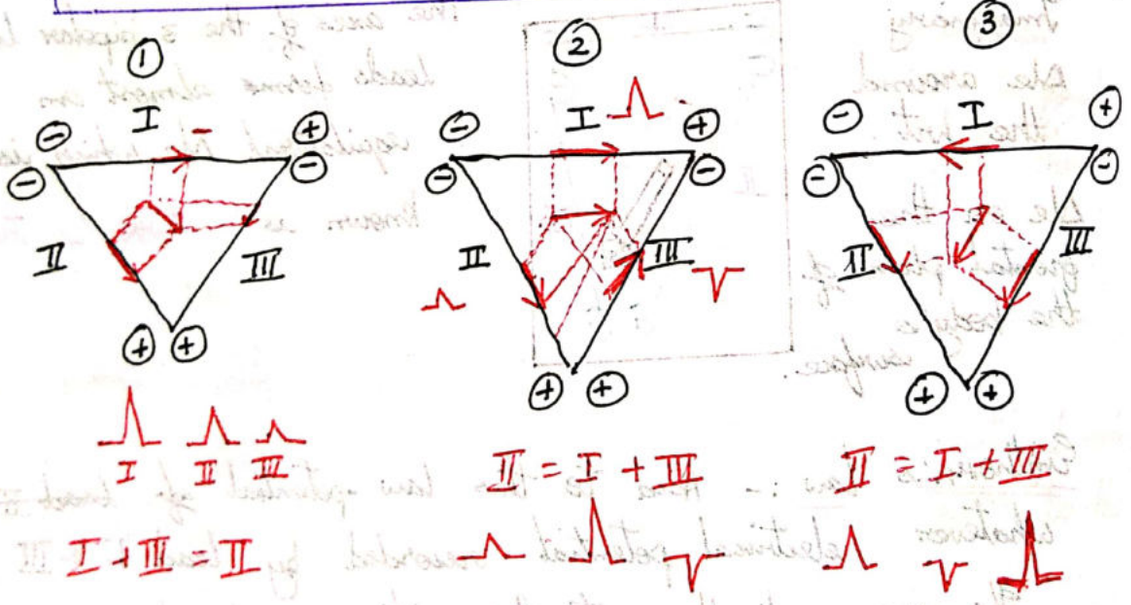
The axes of the 3 bipolar limb leads forms almost an equilateral Δle. which is known as Einthoven's triangle

Einthoven's Law :- According to this law ~~potential of lead II is~~ whatever electrical potential recorded by lead I & III, if you put them together it should become eq to that recorded by lead II.

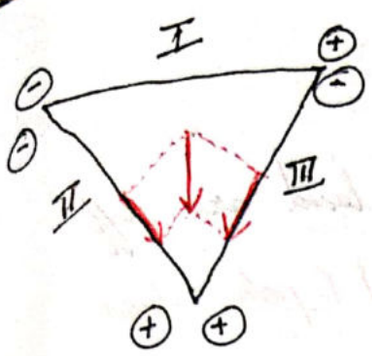
$$I + III = II$$

- As compared to the body potential the potential is -ve on the right & upwards, & +ve on the left & down ∴ the ventricular depolarisation is moving downwards & left.
- Voltage of any two leads is known, the third can be determined.
- If the position of cardiac vector is changed, the pattern recorded by the leads changes but the Einthoven law remains unchanged ie I + III = II.

- Einthoven's Δle is a hypothetical Δle made by the 3 bipolar limb leads around the brt in the frontal plane of body's surface.



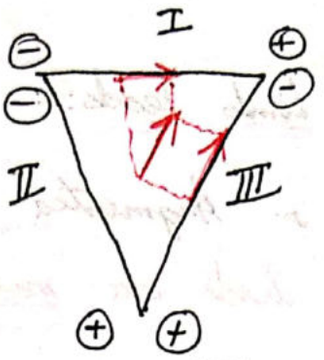
Einthoven's law states that electrical activity of brt which results into a cardiac vector that will prod deflections in such a manner into lead I, II & III when their voltages are measured potential of lead II shuld be eq to the sum of the pot of I & III if all 3 leads are recorded simultaneously



$II = I + III$

$II = III$

$\because I = 0$



$I =$ 

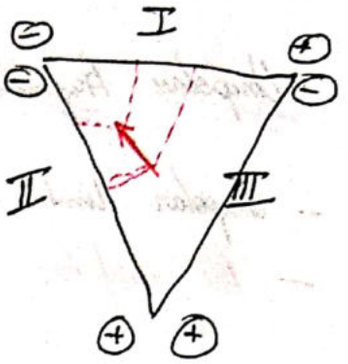
$III =$ 

$II = 0$

$II = I + III$

$0 = I - III$

$I = III$



$III = 0$

$II = I + III$

$\because III = 0$

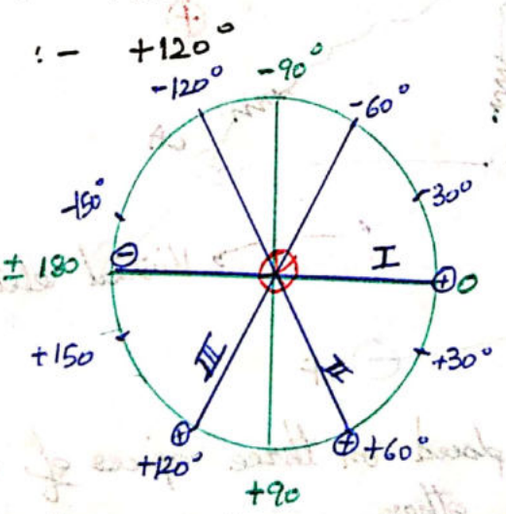
– Triaxial Reference System :-

With Reference to +ve electrode the electrical axes in frontal plane are oriented with the center of hrt as :-

Lead I :- 0°

Lead II :- +60°

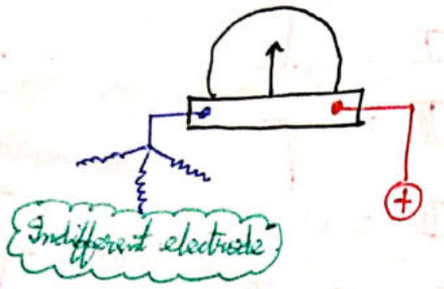
Lead III :- +120°



Lead I axis – reference axis

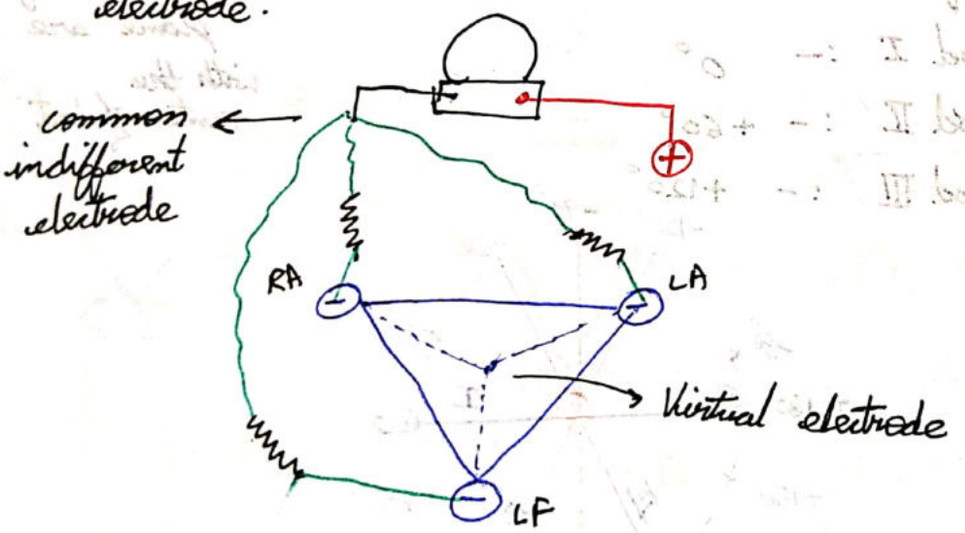
Unipolar Augmented limb leads:-

- Unipolar limb lead & Augmented Uni lead are diff
- All unipolar limb leads are modified Bipolar leads



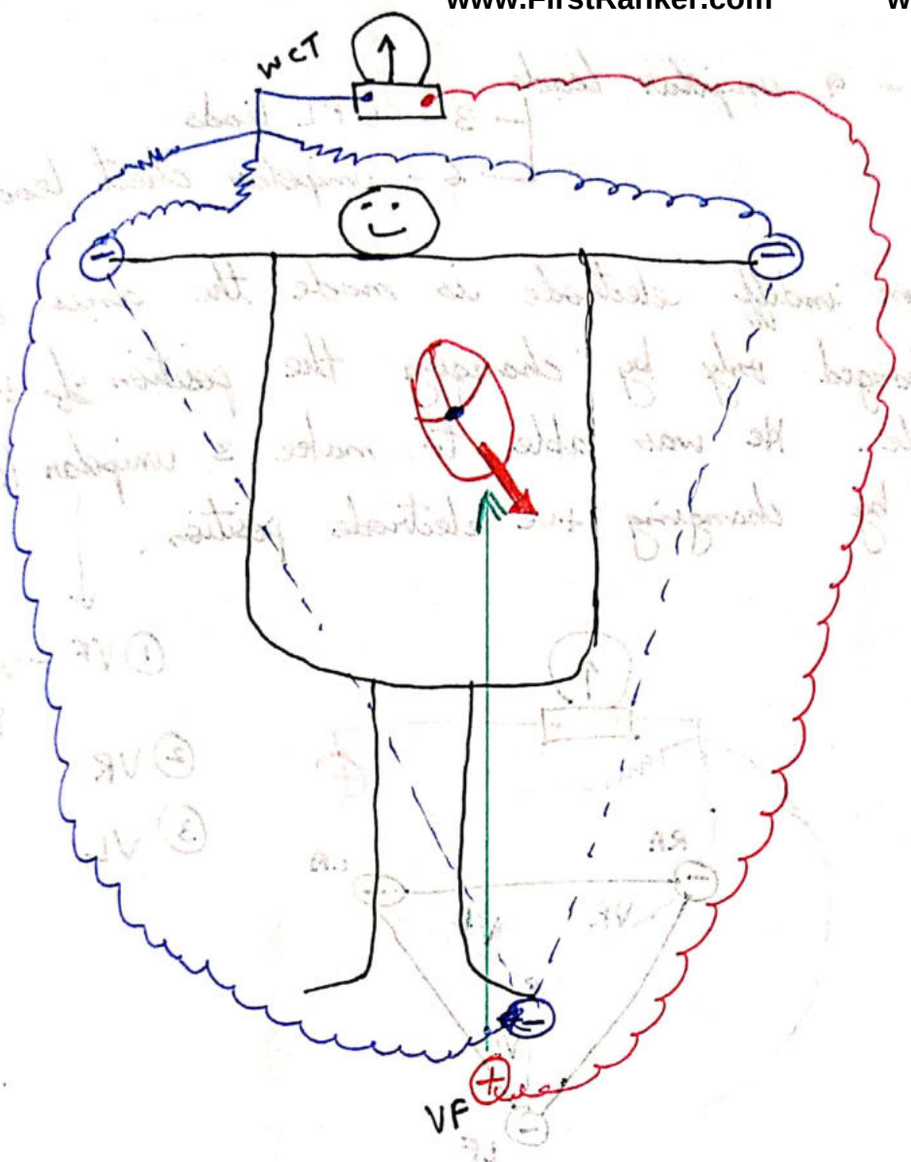
-ve terminal thru multiple connections & resistances is converted into indifferent electrode / Near zero electrode
↳ It senses almost zero potential
Also called Null / virtual electrode

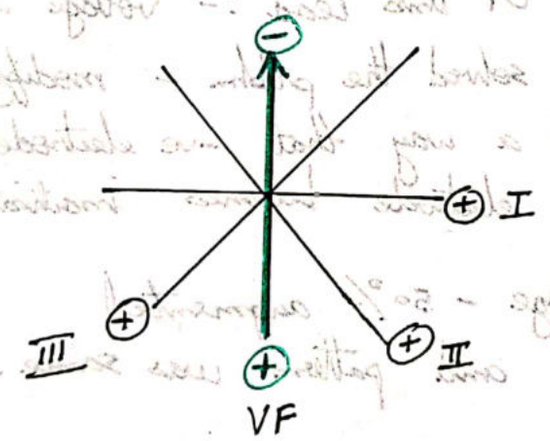
∴ Unipolar limb lead is constructed with +ve electrode as exploring electrode & -ve electrode as indiff electrode.



-ve electrodes placed on three apices of eq Δle cancel each other.

Wilson converted -ve electrode by a set of multiple -ve electrodes into virtual electrode & achieved near zero potential virtual electrode placed at centre of the hrt





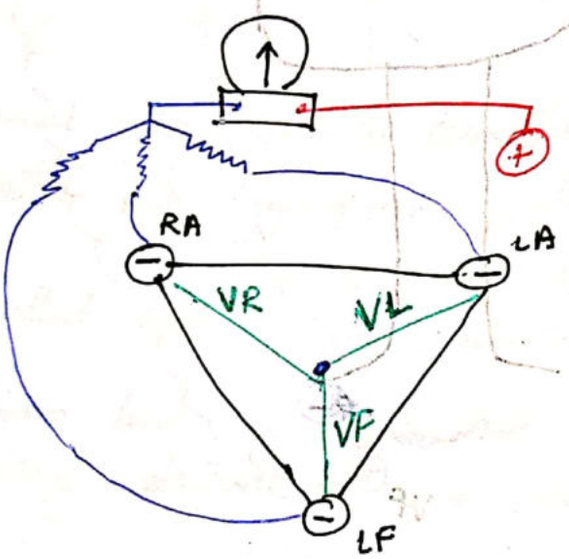
VF = voltage from foot

- Whenever +ve electrode is exploring the voltage rel to the indiff electrode in the body this lead is called V lead.
  - ↳ Tol voltage sensed by this is significantly reduced. as compared to bipolar leads.

- Wilson - 9 unipolar leads
  - 3 - UPL leads
  - 6 - unipolar chest leads.

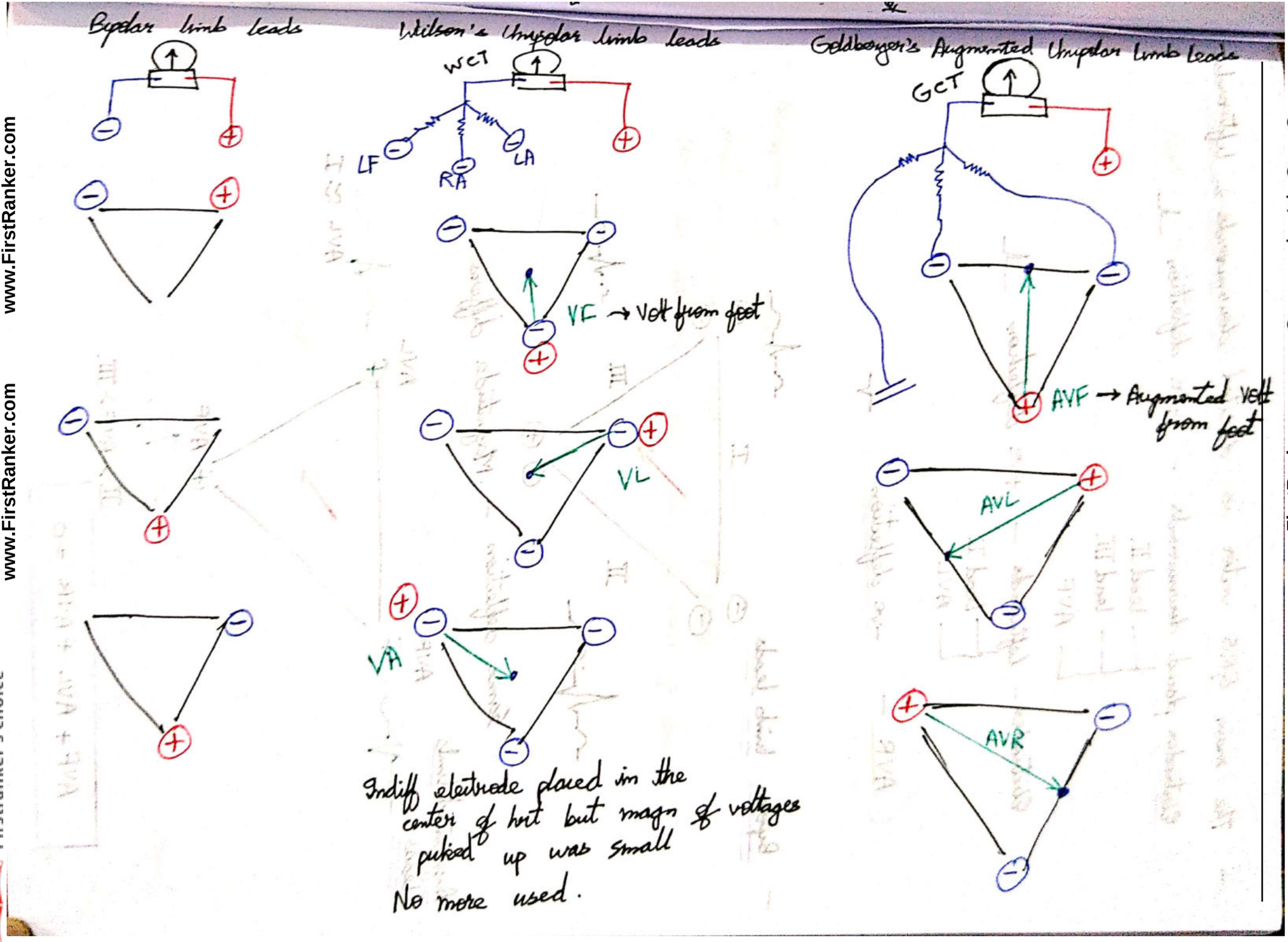
- One an indiff electrode is made the axes can be changed only by changing the position of +ve electrode. He was able to make 3 unipolar leads only by changing +ve electrode position.

① VF - voltage from foot
② VR
③ VL



- Disadvantage of UP limb lead :- voltage recorded was v. low
  - ↳ Goldberger solved the prblm - modify the WCT in such a way that -ve electrode near the exploring electrode becomes inactivated.

Augmented unipolar lead → voltage - 50% augmented and pattern was same.



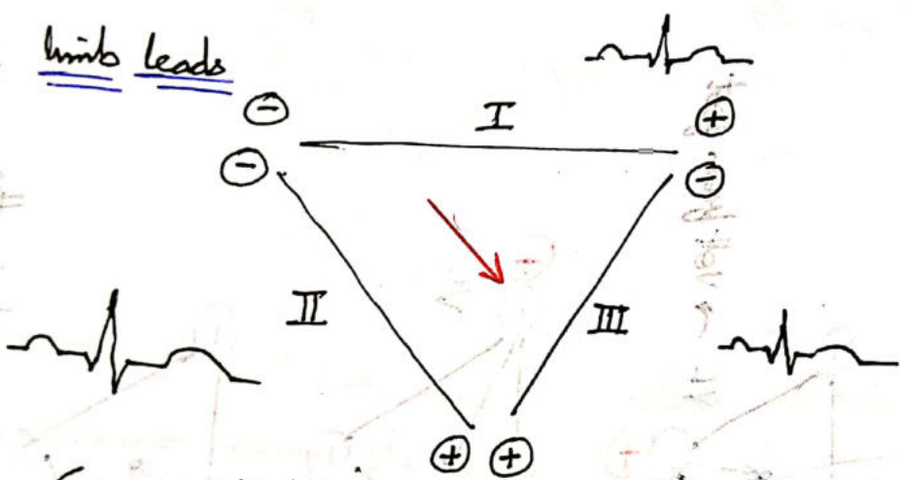
- The main QRS vector is directed downwards & leftwards

Electrodes placed downwards — +ve deflection
- Lead II
- Lead III
- AVF

Electrodes - leftwards — +ve deflection
- Lead I
- AVL

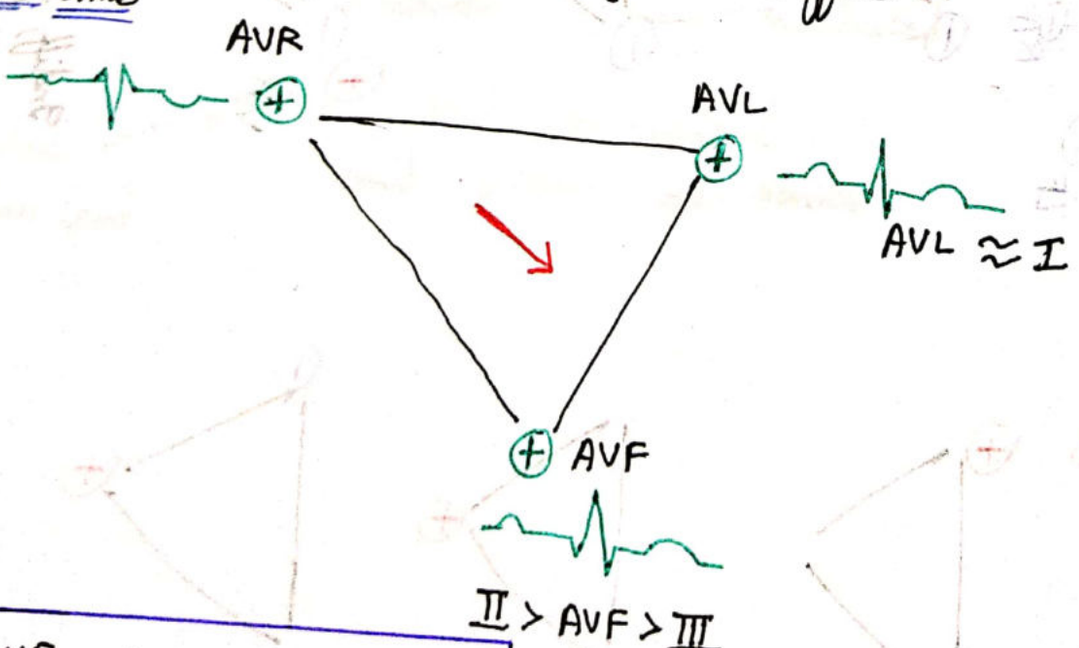
AVR — -ve deflection

- Bipolar limb leads



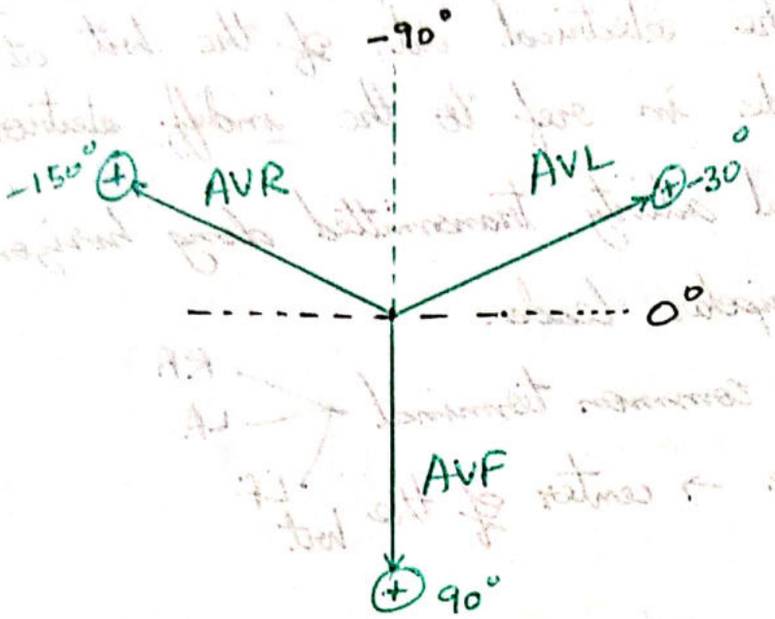
Same deflection - Magnitude differs

Augmented limb leads

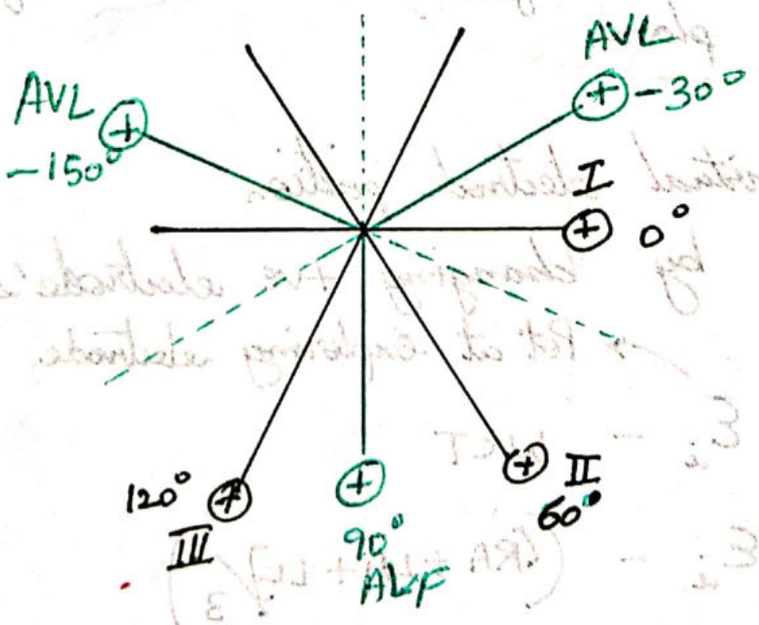


AVF + AVL + AVR = 0

- Triaxial Reference System (AUPL) :-
  Lead I axis is used as reference

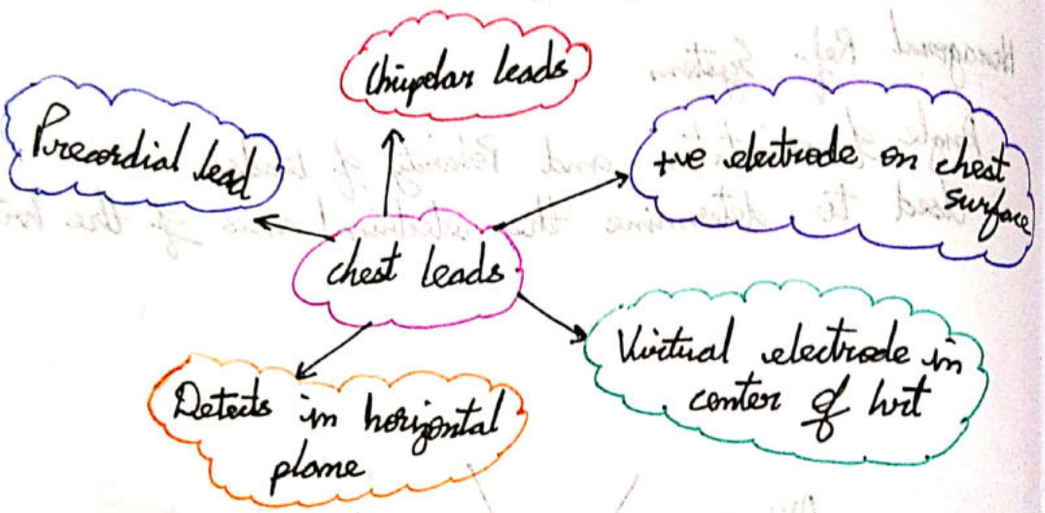


- Hexagonal Ref System
  Angle of orientation and Polarity of leads.
  Used to determine the electrical axis of the hrt.



Chest leads :- Also called Precordial ECG leads

- The exploring electrode :- on chest wall
  ↳ which determines the electric pot at the site of electrode prod by the electrical act. of the hrt at the site of electrode in ref to the indiff electrode.
  detects electrical activity transmitted along horizontal plane
- They are also unipolar leads.
- -ve terminal → common terminal ← RA, LA, LF
  virtual electrode → center of the hrt



- No change in virtual electrode position
  Recording done by changing +ve electrode's position.
- $V_i = E_i - WCT$ → Pot at exploring electrode
  $= E_i - \left(\frac{RA + LA + LL}{3}\right)$
  Pot detected by chest lead

- ✳ Potential of the virtual electrode is not only near zero but it is constant through out the cardiac cycle or it does not fluctuate significantly.

But exp electrode records the electric activity of the hrt

be pred during diff phases of cardiac cycle in a diff way

- Six chest leads :- standard.

V_1 => +ve on 4th intercostal space right

V_2 => .. .. left

V_4 => on the 5th intercostal space lft on midclavicular line.

V_3 => midway B/W position of V_2 & V_4

V_5 => At the same horizontal level of V_4 Laterally on Anterior axillary line

V_6 => At the same horizontal level of V_4 & V_5 more laterally on mid axillary line.

- Orientation of chest leads :-

I Anatomical orientation
- V_1, V_2 } oriented over the right side of hrt
- V_5, V_6 } .. .. .. lft .. .. ..
- V_3, V_4 } Over inter ventricular septum

II Functional
- V_1, V_2 } Most sensitive to Septal depo => Septal leads
- V_5, V_6 } .. .. .. Major Ventricular depo which is leftwards => Left leads
- V_3 & V_4 .. .. .. Anterior MI

MI on septal area => V_1 & V_2

MI on left side => V_5 & V_6

Anterior MI => V_3 & V_4

Antero Septal MI => V_1 V_2 V_3 V_4 => Antero Septal leads

Antero lateral MI => V_3 V_4 V_5 V_6 => Antero lateral leads

Large Anterior MI => V_2 V_3 V_4 V_5

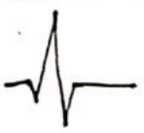
- Angle of axis of one lead with ref to the the axis of the other lead => 30° [approx]

QRS complex - Nomenclature :-

↳ Graphical Rep of electrical activity of hrt when it is undergoing ventricular depolarisation.

- **Isoelectric line** :- extends B/W T wave & P wave
  ↳ TP segment — No electrical act. of hrt.

☆ PR & ST segment can be elevated in pathological cond so cannot be used as ref. Hence not called Isoelectric line.

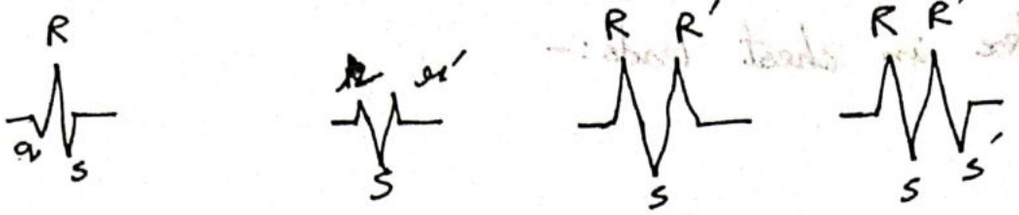


① Initial -ve wave => 'q' wave or 'Q' wave

Note :- small letter → small amplitude
Capital letter → large amplitude

② 1st +ve wave => 'r' or 'R' wave

③ -ve wave after R wave => 's' or 'S' wave



④ If we 2nd +ve wave in QRS complex => r' or R'

Note :- First +ve wave → R wave
Any -ve before R wave → Q wave
" " after " " → S wave

⑤ -ve wave after R' => S' wave

Note :- When deflection > 3 small squares => Large
deflection < 3 small sq => Small.

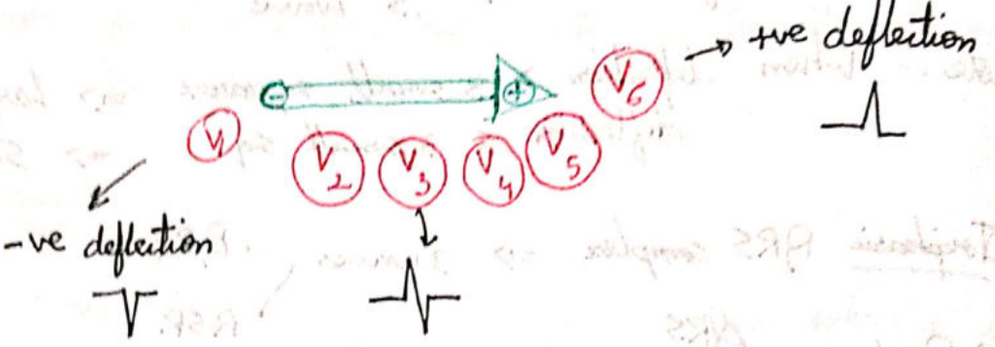
① Triphasic QRS complex => 3 waves — QRS, RSR'

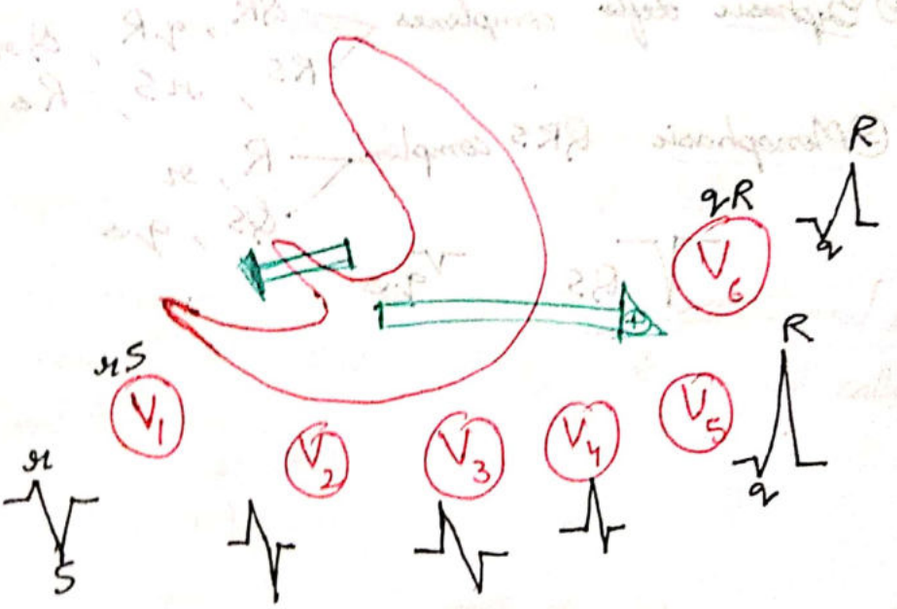
② Biphasic QRS complexes — QR, qR, Qr, qr, RS, rS, Rs, rs

③ Monophasic QRS complex — R, r, QS, qs

QRS complex in chest leads:-

- Chest leads record the electrical activity projected along horizontal plane.
- The 6 chest leads record the electrical activity of hrt differently.
- WRT major ventricular vector the position of +ve electrode is moved from tail end of the vector progressively to its front.





$V_1 \Rightarrow rS$

$V_6 \Rightarrow qR$

V_5 has taller R than V_6 bcoz lung is B/W V_6 & hrt so the amplitude is reduced

As we move from rt to lft → QRS converts from very -ve to very +ve.

- R wave progression :- The ↑ in amplitude of R wave progressively from right sided chest lead to lft sided chest lead.
  - ↑ in amplitude from V_1 to V_4 / V_5 of R wave
  - ↓ in amplitude of S wave from V_1 to V_4 / V_5

- In either V_3 / V_4 R and S becomes eq. This lead is called transitioning lead.

  V_3 & V_4 - Transition zone.

  V_2 - Early transition zone - if R=S become eq at V_2

  V_5/V_6 - delayed " " - if R>S become eq here.

- Abnormalities of R wave progression

① Accentuated R wave progression ⇒ Increased more than normal.
- progressively the ht of R wave ↑ but increase is more than normal.

Accentuated means ⇒ more depolarising currents more from lft → Powerful vectors rt to lft

- Rt sided leads - S wave deeper than normal
- lft " " - R - taller " "

Powerful vector moving from ~~left~~ rt to lft and its power is more than normal., and is more leftward

Conditions it is seen in :- a) lft ventricle becomes hypertrophied
↳ lft ventricle vector more powerful vector more towards left side

Hence the resultant vector is more towards left ventricle
↳ forces are added on left side – more powerful.
↙
This extra power – taller R wave

~~b)~~

Whenever major ventricular dep vector is more powerful & more leftward it will prod accentuated R wave progression

b) When Rt ventricular vector & Lft vent vector become out of phase due to some abnormality in conduction system:

Rt – occurs earlier & Lft is delayed
then Rt fails to partially neutralise the Lft vector.
← Since they're not simultaneous

It happens in Lft bundle branch block.
↓
The depo of left side is delayed and it'll be out of phase with rt one.
The delayed QRS vector – leftward
QRS complex – wide & taller

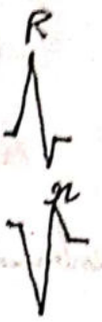
c) Rt vent vector is reduced – Rt vent MI
↳ Rare.

Lft Ventricular hypertrophy
↑
Accentuated R wave
Right Ventricular MI
Lft bundle branch block

② Reversed R wave progression :-
- When the major ventricular depo vector is reversed in direction. - Resultant vector towards right.
- Progressive ↓ in R waves

V_1 & V_2 => tall R waves

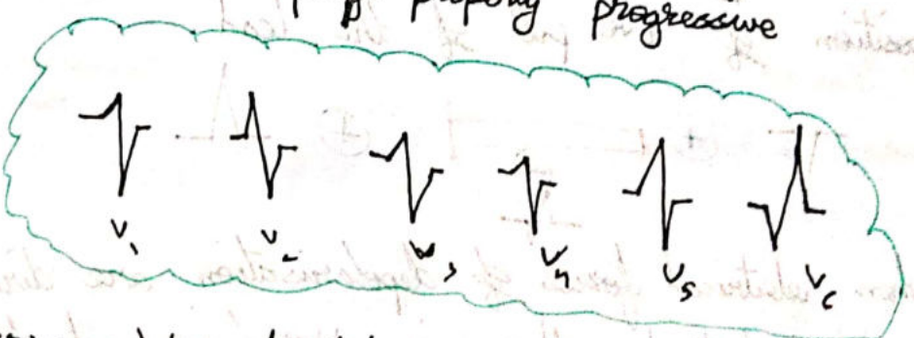
V_5 & V_6 => short R waves



Causes :- a) Rt ventricular hypertrophy
b) Rt bundle branch block => delayed depolarisation of Rt Ventricle & Also prolonged
↳ Large Rt vector.
c) Posterior MI => Forces leftward - Reduced unable to cancel Rt Vent depo vector

∴ The R waves progressively ↓ from V_1 to V_5

③ Poor R wave progression :-
- R wave not properly progressive



Causes :- a) Loss of Anterior forces - Ant MI
↳ This does not allow Ant leads V_3 & V_4 to form tall R waves

Note :- Poor R wave progression is not diagnostic but is suggestive of Anterior MI

b) Left Vent hypertrophy

Hence in Lft hypertrophy two kinds of abnormalities are seen

Accentuated & Poor R wave progression depending on degree and rotation of electrical vector

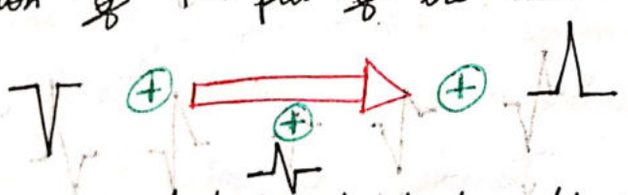
If not ⊥d to V_3 & V_4 → Poor R wave.

If purely leftward & more amplitude → Accentuated

c) Rt Ventricular hypertrophy – If the vector moves too much Rightward

QRS complex in limb leads:-

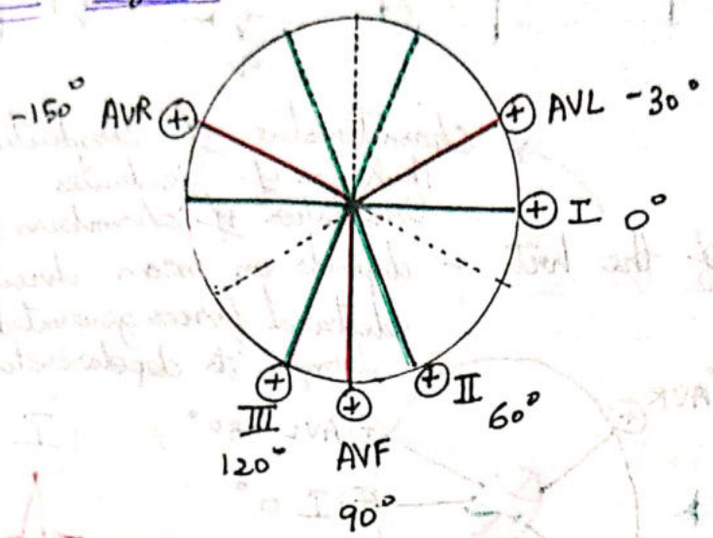
- There are 18 different normal variants of QRS complex in limb leads.
- Most of leads pick up the electrical act. of Septal & Major Ventricular depolarisation.
- Law of ECG :- The same electrical activity vector will produce diff patterns in diff leads depending upon the position of +ve pole of the lead.



☆ When electrical forces of depolarisation are directed towards +ve electrode there is +ve / upward deflection.
When vector is ⊥d to +ve electrode => No net change

Biphasic 

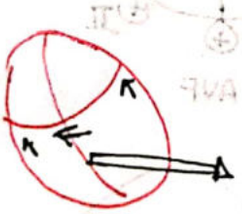
- Hexaxial diagram



- QRS pattern recorded depends on
  - Placement of +ve electrod
  - Direction of movt of the depolarisation

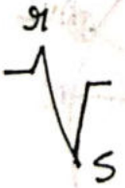
- Patterns by AVR
  - AVR is oriented upwards & Rightwards

QRS pattern in AVR - predominantly -ve

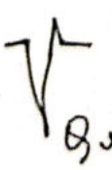


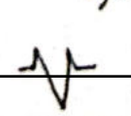
Septal towards AVR +ve
Major Vent opposite

All of them have -ve deep deflection
Major Vent is always recorded

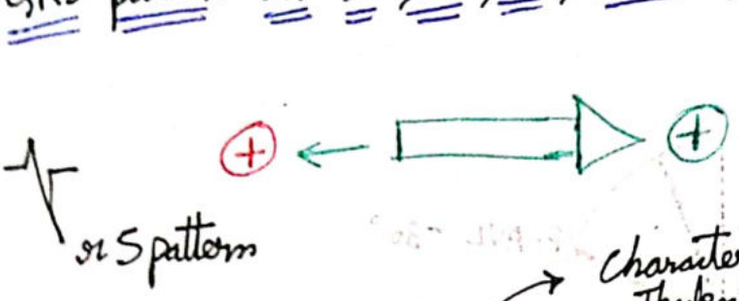
①  => R S pattern septal recorded

②  => Septal > basal not recorded QS pattern

③  => Q R pattern
Basal is recorded
Septal not ,,

Rare ← ④  => R S R' - All are recorded - rare

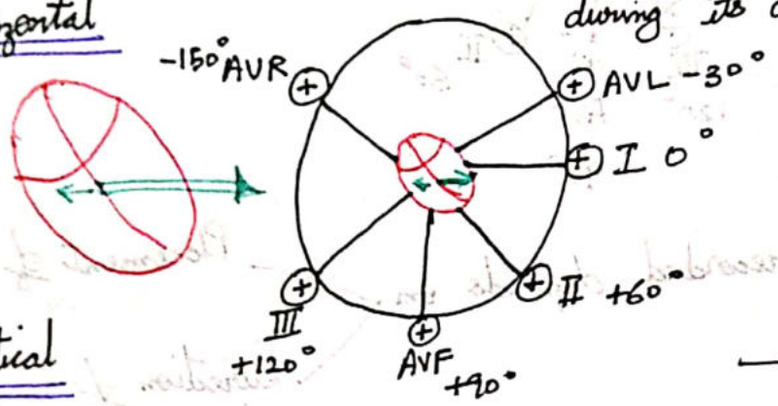
- QRS pattern in I, II, III, AVF and AVL leads :-



Electrical Position of the Ventricles - depends on mean direction of electrical forces generated by ventricles during its depolarisation

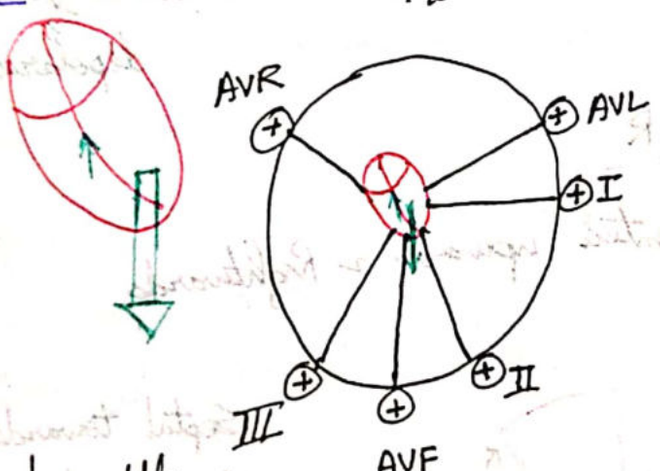
Characteristics of conduction Sys
Thickness of Ventricles
Orientation of chambers of heart

① Horizontal

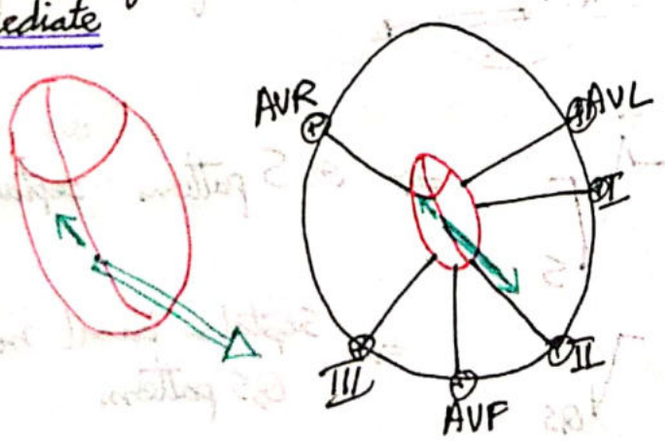


large as parallel

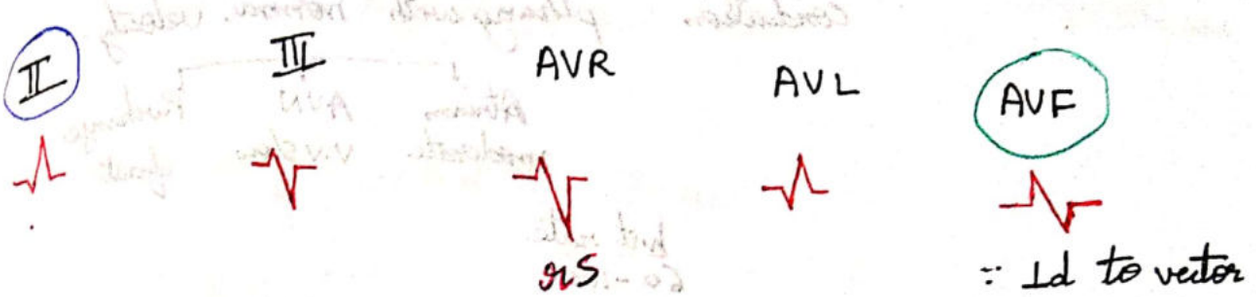
② Vertical

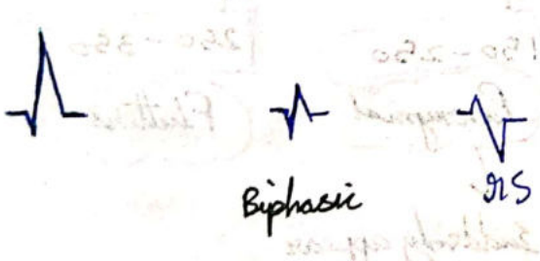


③ Downward & Lft Intermediate

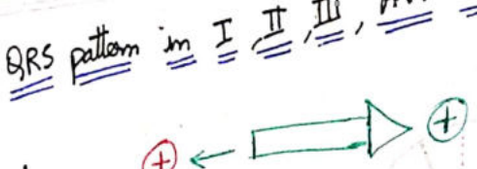


AVR - will always be -ve





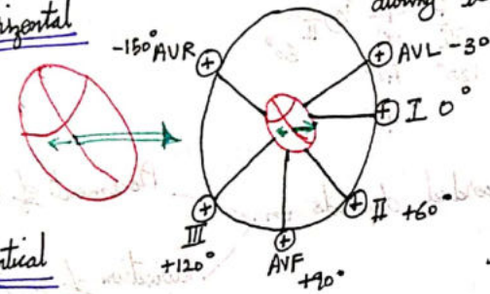
- QRS pattern in I, II, III, AVF and AVL leads :-



Electrical Position of the heart - depends on mean direction of electrical forces generated by ventricles during its depolarisation

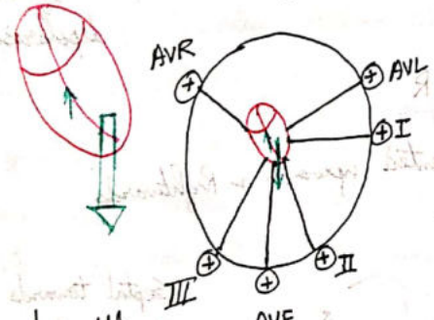
Characteristics of conduction System
Thickness of ventricles
Orientation of chambers of heart

① Horizontal

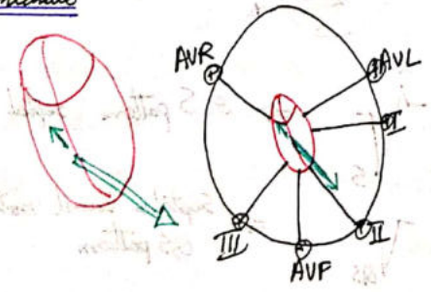


large as parallel

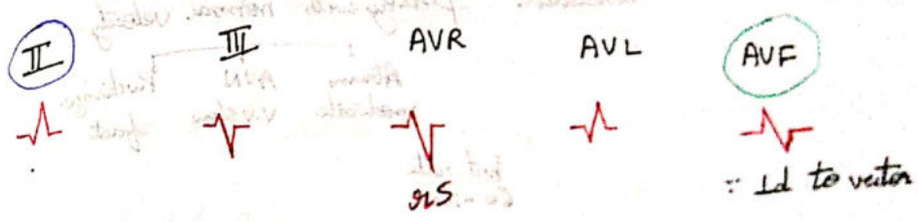
② Vertical

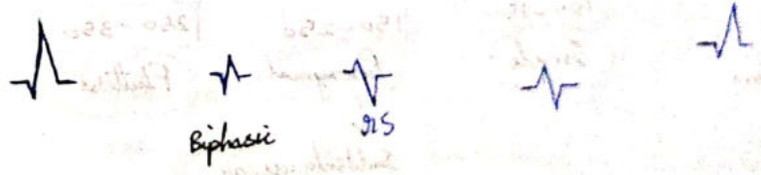


③ Downward & left Intermediate



AVR - will always be -ve

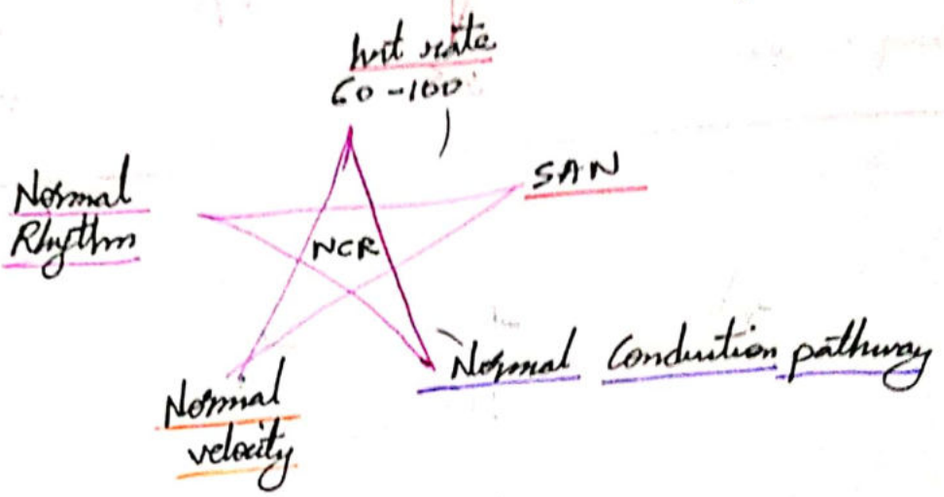




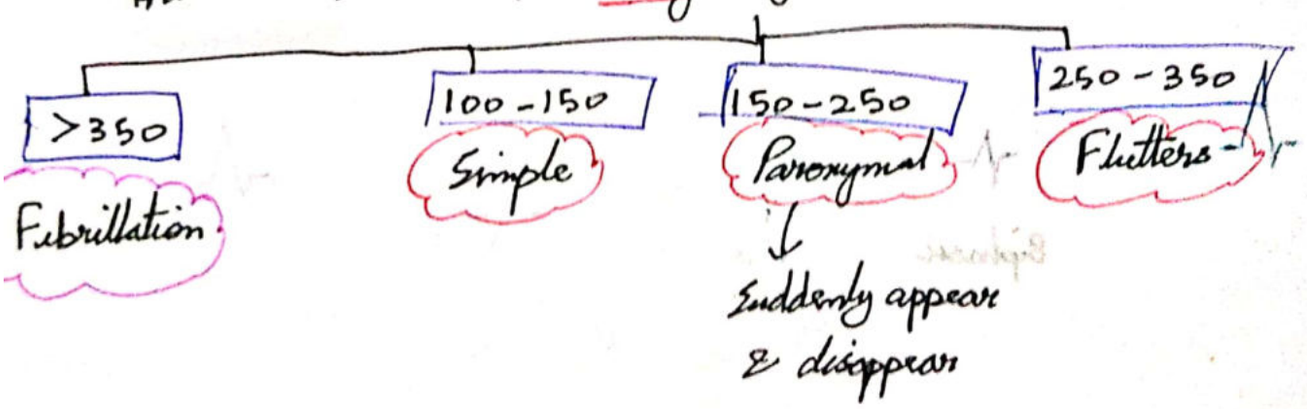
Cardiac Arrhythmias :-

- Deviation from normal rhythm of the heart.

- Normal cardiac rhythm
  1. Heart rate :- 60 to 100
  2. Every heart beat must originate from SAN
  3. Cardiac impulse shud propagate thru normal conduction pathway with normal velocity.
     - Atrium — moderate
     - AVN — V.V slow
     - Purkinje — fast



- Heart rate < 60 => Brady arrhythmias
  Heart rate > 100 => Tachy arrhythmias



Heart rate 40 - 60 => Mild BA
20 - 40 => Moderate BA
< 20 => Severe BA

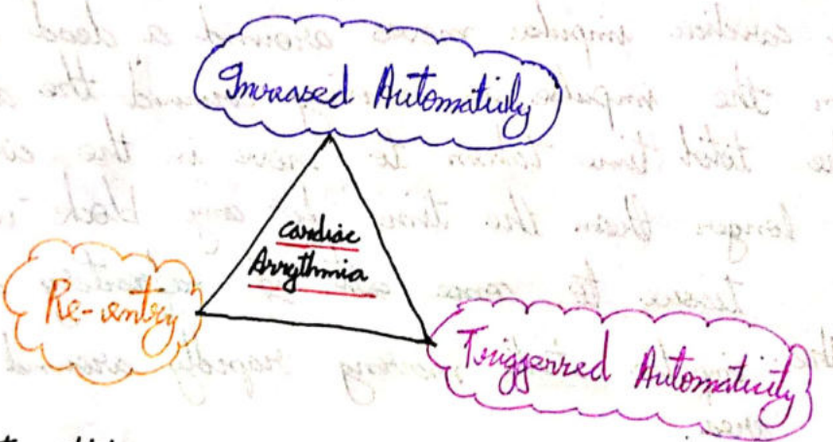
Supra ventricular Tachyarrhythmias

Add to site

1. Sinus Arrhythmia => Abnormal Rhythms in SAN
2. Atrial Arrhythmia => .. .. in Atria
3. Junctional Arrhythmia => .. .. .. AV node.
   or nodal arrhythmia
4. Ventricular Arrhythmia => .. .. .. Ventricles

- Mechanisms of Cardiac Arrhythmias :-

1. ↑ Automaticity :-
2. Triggered Automaticity
3. Phenomenon of Reentry / Circus movt



1. ↑ Automaticity :-

- SAN generates potentials spontaneously — Pacemaker.
  ie shows automaticity.
- ↑ automaticity → stimulation of β_1 adrenergic receptors by epinephrine
  → Threshold attained rapidly and there are more frequent action pot. firing More frequently

② Triggered Automaticity :-

- Ventricular cell - Na^+ dependent depo

① Early After depolarisation => depo starts early
② fluctuating RMP => delayed after depolarisation

Triggered automaticity induced Tachy arrhythmias are p

If the cells
- Ischemic
- Injured
- Catecholamines
- others factors which load it with cations

=> RMP cannot Rest
↓
fluctuating RMP
↓
Arrhythmia

③ Re entry / Circus movt :-

If a cardiac impulse moves around a dead area when the impulse is moving around the dead area the total time taken to move in the circle is longer than the time for any block in the tissue to come out of refratory area., the impulse starts moving rapidly around the dead area.

Ischemic hrt disease

Sinus Arrhythmias and ECG patterns.

① Physiological :- Normal phenomenon – very mild

- Vagus controls the automaticity of SAN & AVN
- Inhibitory influence of Vagus
- Vagal activity fluctuates during Respiratory cycle

Vagal tone fluctuations

Inspiratory phase → Vagus inhibited → Hrt rate ↑ slightly

Expiration → opposite → Hrt rate ↓ slightly

- Diabetic patient → Autonomic neuropathy → Vagus not func → Hrt rate will not fluctuate during Resp cycle
- Transplanted Hrt → denervated → No Vagal influence → does not undergo physiological sinus arrhythmia
- During Inspiration R to R distance ↓ → short RR distance → small time → more complexes

→ In short time more QRS complexes

During expiration → Vagal influence ↑ → Hrt rate ↓
R to R distance larger

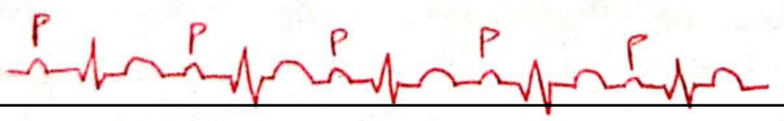
② Sinus Tachycardia :-

→ Hrt rate more than 100 due to ↑ activity in SAN → overstimulation of SAN

- exercise
- fever
- Thyrotoxicosis

P waves prod more frequently

RR distance will be less



(3) Sinus Bradycardia :-

↳ slow hrt rate < 60

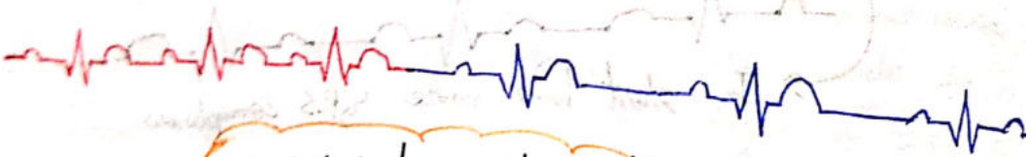
- → Athletes → due (↑) vagal tone
- → Hyperthyroidism
- → Hypothermia
- → Obstructive Jaundice → Bile aid accumulates in SAN.

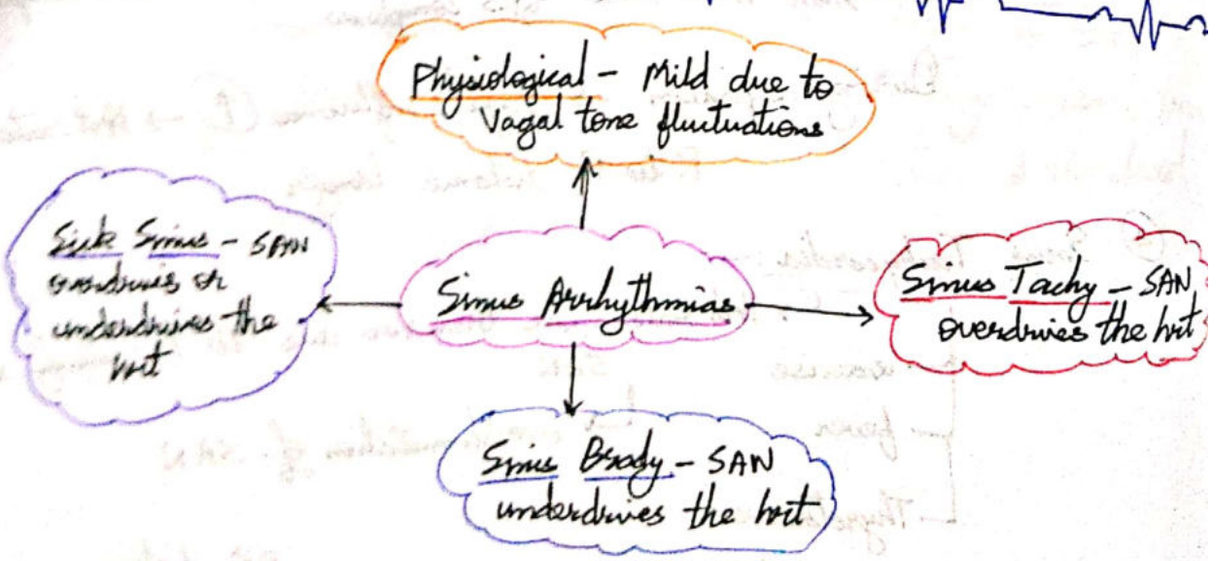
P wave frequency is less

R-R distance larger than normal

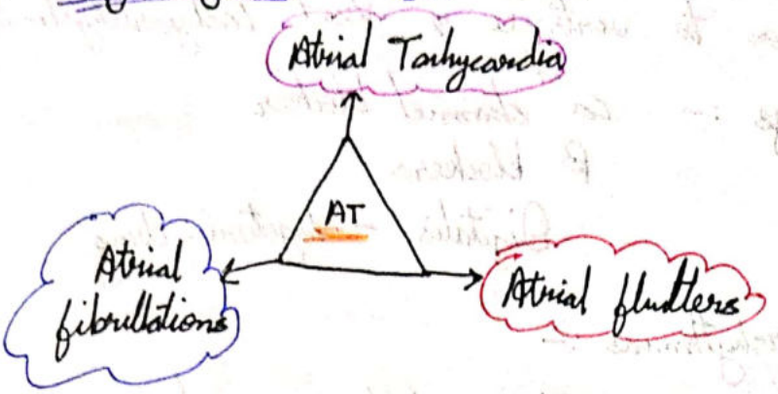
(4) Sinus Tachy brady syndrome / Sick Sinus Syndrome :-

SAN behaves pathologically - sometimes it is firing faster than normal & at some other times slower than normal.



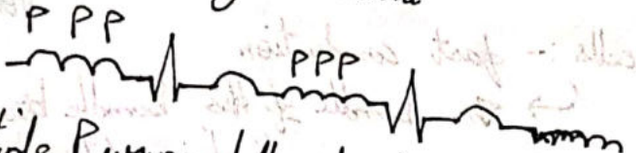


Atrial Tachyarrhythmias and ECG :-



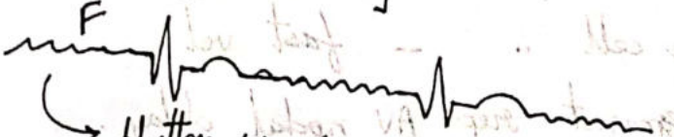
① Atrial Tachy :- [125 - 250/min - electrical activity]

↳ Atria is abnormally irritated



↳ Multiple P waves followed by one QRS
↳ well defined & recognisable

② Atrial flutter :- [250 - 350]

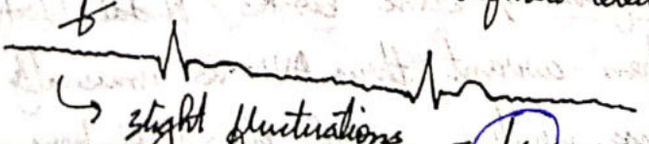


↳ flutter waves - very rapid atrial depo present in atrial tissue.

Saw tooth Base line. (F) waves

③ Atrial fibrillation :- More than 1 ectopic foci - simultaneously firing.

Small Multiple Vectors directed in diff directions
↳ dyssynchronised
No well defined electrical vector in atria



↳ slight fluctuations - (f) waves

If Atrial tachyarrhythmias are transmitted to Ventricles then they can precipitate Vent Tachyarrhythmias which are life threatening

- Management of Atrial Tachy :- AV node is slowed to avoid transmission to ventricles & Vent tachyarrhythmias

drugs :- Ca^{2+} channel blocker
β blockers
Digitalis - Vagotonic drug

- Junctional Arrhythmias :-

- AVN :- only area thru which current can pass from atria to ventricle.
  ↳ delays the impulse → RMP => -60 mV
  voltage gate Na^+ are not

- Purkinje cells :- fast conduction
  ↳ In bundle of His, bundle branches & Purkinje fibres

∴ Junc tissue rspl in slow conduction.

- Atrial tissue depo - moderate vel
  AVN " - slow vel
  Purkinje cell " - fast vel.

- PR segment rep AV nodal delay.

PR interval = P wave + PR segment

If conduction thru AVN slower than normal => PR interval / segment is prolonged

If cond thru AVN faster than normal => PR interval / segment shor

↳ drug like caffeine, sympathometic drugs.

(1) Junctional bradycardia / Junc blocks / Nodal blocks / Hrt bl

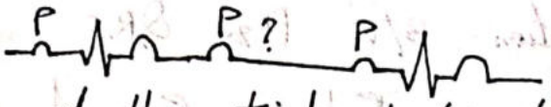
↳ when current thru AVN is unusually delayed

↳ drugs which can slow AVN => Ca^{2+} channel blockers
β blakers
Adenosine

↳ unusual delay
PR interval is prolonged → 1st degree

- 1st degree block :- Every impulse / current from atrium is allowed to enter the ventricle but is held in the AVN for usually longer duration.

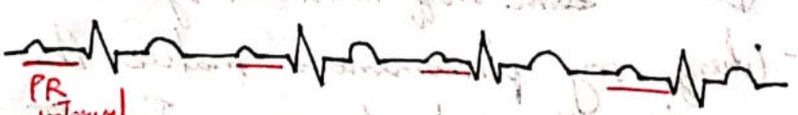
- 2nd degree block :-



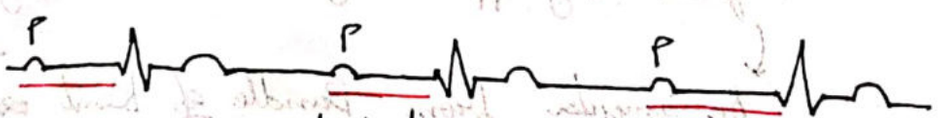
Some of the atrial depolarisations are suppressed / aborted in the AVN.
ie Some P are not followed by QRS

- 3rd degree block :- drug like Adenosine completely suppresses the AV node.
  - None of the Atrial depo are trans to Ventricles
  - As a result a new pacemaker dev for Ventricle.
  - Atrial & Vent activity dissociated from each other.
  - Complete hrt block.

(N)



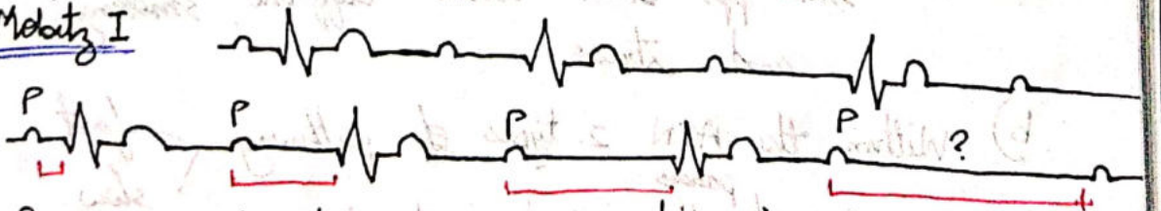
I° HB



PR prolonged but every P followed by QRS

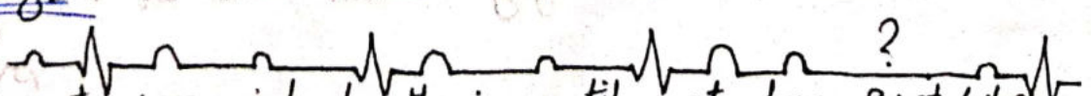
II° HB

Mobitz I



Progressive elongation of PR interval until one P wave is not fol by QRS complex

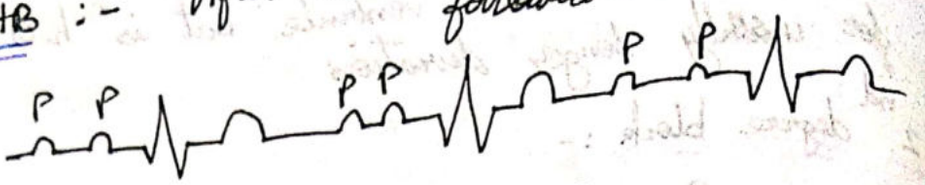
Mobitz II



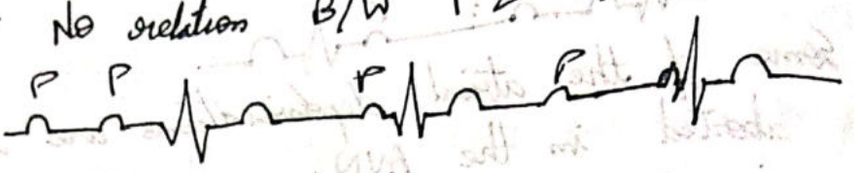
PR is not progressively lengthening until a pt where P not fol QRS

~~III° HB~~

2:1 II° HB :- After two P waves a QRS complex followed



III° HB :- No relation B/W P & QRS



② Junc Tachycardias :-

a)- Abnormal / Pathological connection B/W atria & vent is called Bundle of Kent
↳ faster than AVN.

→ when no Junc Tachy

↓

~~Junc~~ Wolff Parkinson Syndrome -

- When any phenomenon makes the AVN condu faster they ppt dangerous Junc Tachy.

↓

An impulse from Bundle of Kent reenters the A ↳ cirus most

Hrt rate is doubled.

- Such ppl shud avoid caffeine, smoking, Alcohol, and stress.

b) Within the AVN 2 types of pathway (fast, slow).

Current passes thru fast and then reenters thru the slow - intranodal re entrant phenomenon.

Treatment :- Massaging Carotid Sinus → Vagal outflow ↓ AVN inhibi

Ventricular Arrhythmias :-

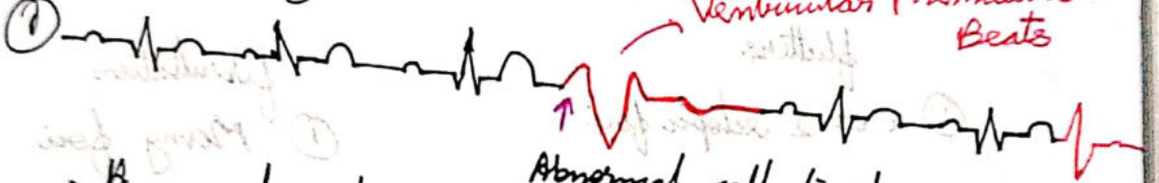
- Common cause :- irritable foci in the ventricles - fire in a pathological way.

eg :- Ischemic cell ⇒ O_2 supply ↓ ⇒ less ATP ⇒ Na/K^+ ATPase does not func ⇒ Na^+ entrapped in cell. ⇒ RMP is less -ve ⇒ fluctuating RMP

Injured Ischemic cell ⇒ leaky to cations.

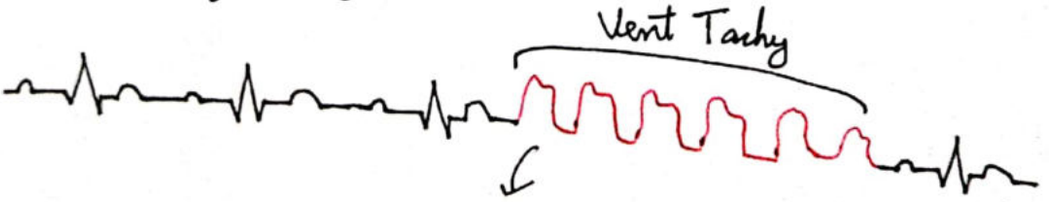
- ✱ Biventricular arrhythmia ∵ both ventricles are electrically connected.

- ①



- ✱ normal vector is not made
  - Moderate vel hence wider
  - current not coming thru Bundle of His
  - ✱ it is thru an ectopic focus
- If they are occasional - Not dangerous
  .. .. .. occuring more frequently - dangerous.

②



In ① diff ectopic foci have fired.

- flutter
  ↳ If cont for long time cardiac output will ↓ ← Multiple areas of Severe Ischemia appear in heart ← many area starts firing on its own