Chapter 4

Electrocardiogram

ECG Genesis

Excursion of trans-membrane potential that occurs in the atria and ventricles when they become active and recover

Every portion has different shape of bioelectric potential

- **SA node: half-circle shape**
- **Atrium: Quick rise and fall**
- **Ventricle: quick rise but slow fall**

Three theories:

- **Membrane theory**
- **Interference theory**
- **Dipole theory**

Membrane Theory

Refractory period is long

Heart is small compared to the cardiovascular system

Can explain the direction of action and recovery

Can't explain the shape of ECG

- **Active cells are electro-negative viewing from outside**
- **If excitation and recovery in the same direction, we have alternate potential curve**
- **Curve of same polarity is obtained when recovery is in opposite of excitation**
- **Knowing the polarity, direction can be understood**

Limitations:

- **0 potential for**
  - **electrodes placed perpendicular to excitation**
  - **if excitation starts midway between electrodes**

Placement of electrodes is very important!!!

Interference Theory

Better bridge between ECG and trans-membrane potential
Can explain the shape of ECG

Potential seen by two recording electrodes is the instantaneous algebraic sum of the potential under each electrode (monophasic action potential, MAP)

$$V = V_1 + V_{2(\text{inverted and delayed})}$$

Consider Ventricular potential

The shape is similar to QRS and T waves

T is inverted due to rich coronary circulation

Dipole Theory

- Viewed from surface → active cells are electronegative wrt resting or recovered cells
- Possible to express boundary between two regions as an array of dipoles
- Extensively used to explain ECG patterns obtained from body surface leads

ECG paper: self-study

ECG Lead

10 electrodes: 4 in limbs, 6 in chest
ECG collected as potential difference between combination of electrodes
Lead: signal between any two electrode or combination
6 limb leads and 6 chest leads

Limb leads: only limb electrodes are used, 2 types

Bipolar limb leads: potential in 2 limbs, third limb is kept connected with ground (I, II, III)

Unipolar limb leads: also known as augmented leads, one limb to +ve and rest 2 are jointly in –ve (aVL, aVR, aVF)

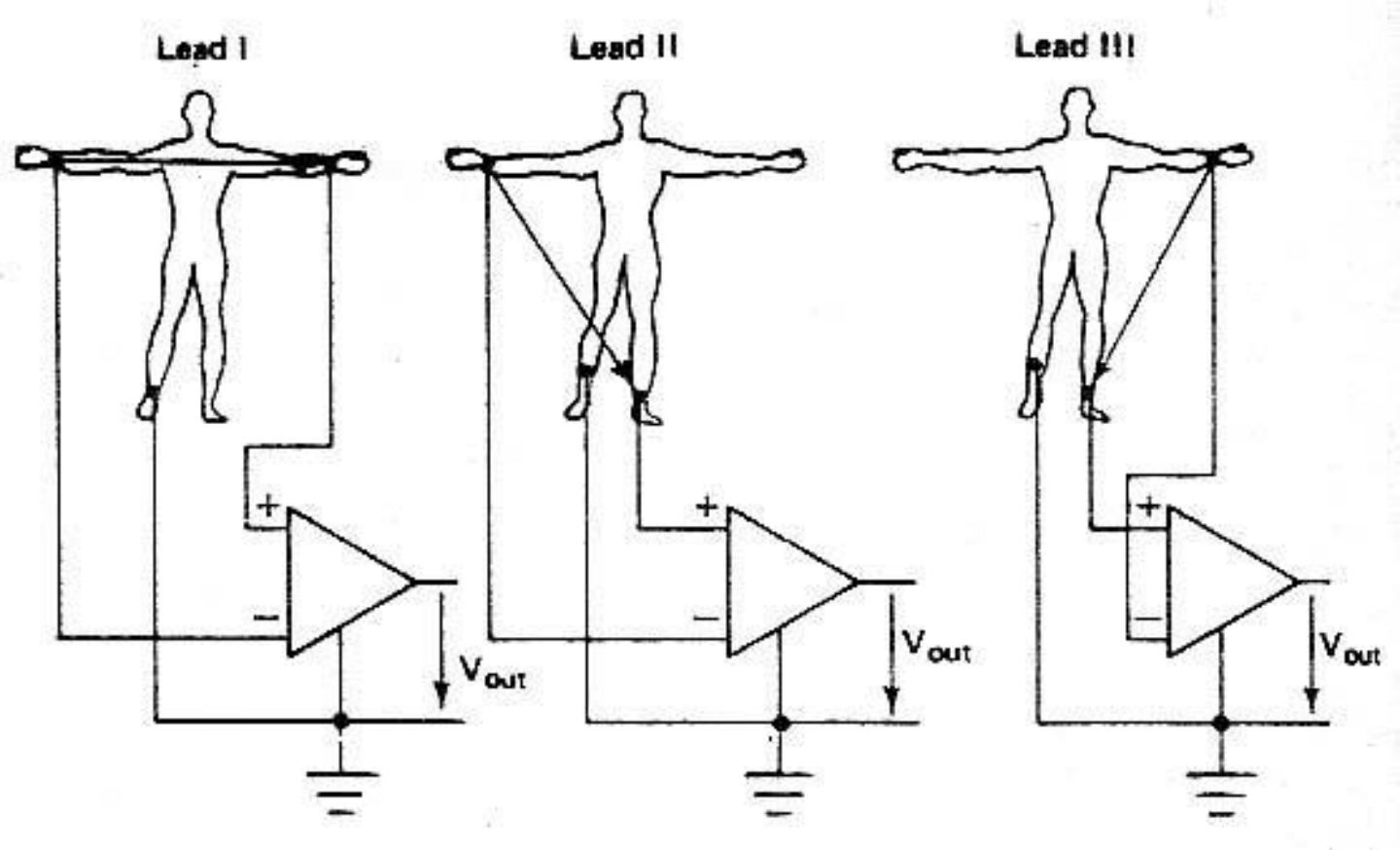
Chest Leads: one chest electrode to +ve, 3 limb electrodes are jointly to –ve. Also known as V-leads (V_1, V_2, V_3, V_4, V_5, V_6)

Why so many leads?
Information available in one lead is not available in others
Total information is obtained by combined information of all leads

Lead Diagrams: Bipolar limb leads

Lead I:
RA to LA
Lead II:
RA to LL
Lead III:
LA to LL

RL is always grounded



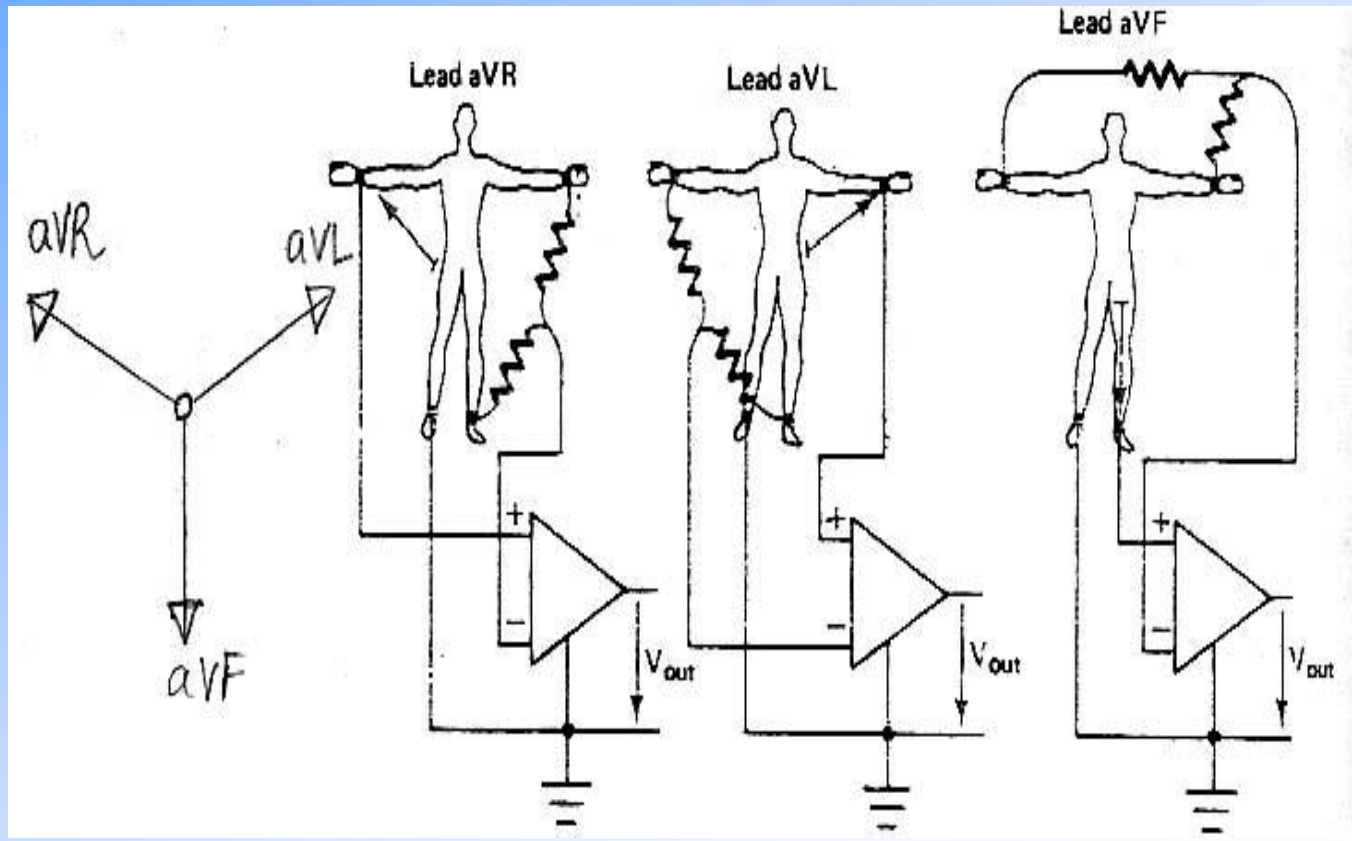
Lead Diagrams: Unipolar limb leads

aVR:
RA to +ve
LA & LL to -ve

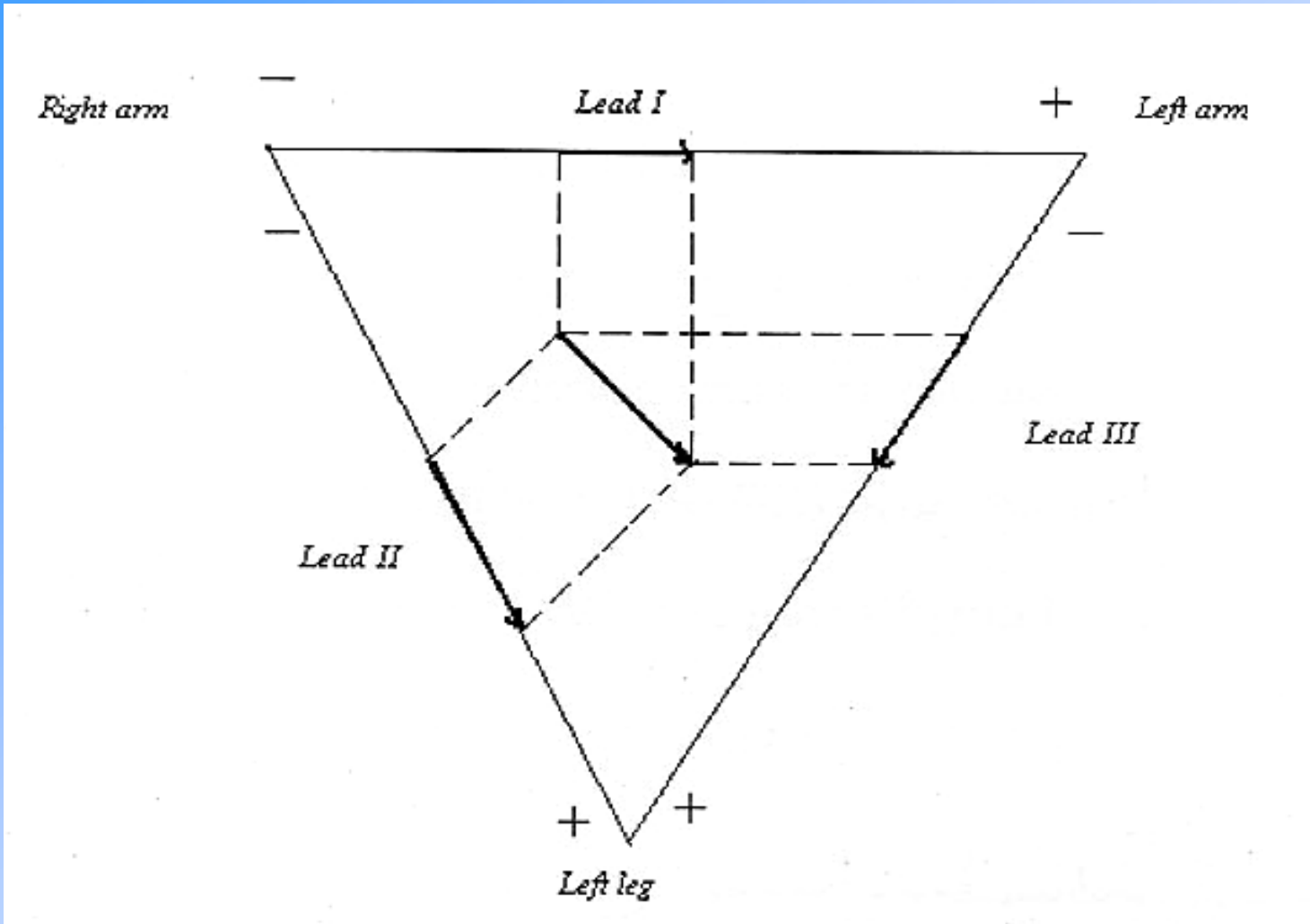
aVL:
LA to +ve
RA & LL to -ve

aVF:
LL to +ve
RA & LA to -ve

RL is always grounded



Lead Diagrams: Einthoven triangle



Lead Diagrams: V leads

V_1 Fourth intercostal space, at right sternal margin.

V_2 Fourth intercostal space, at left sternal margin.

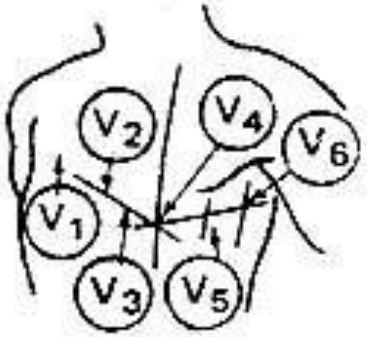
V_3 Midway between V_2 and V_4.

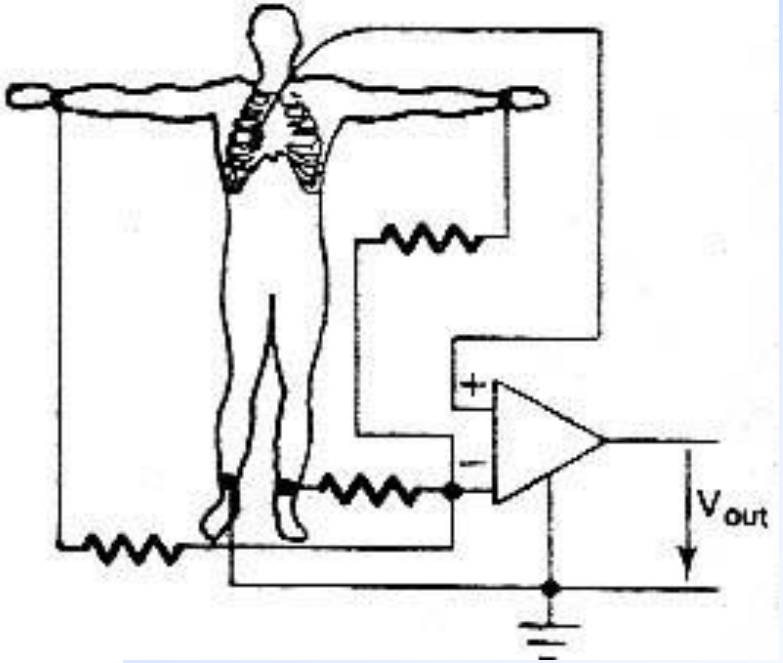
V_4 Fifth intercostal space, at mid-clavicular line.

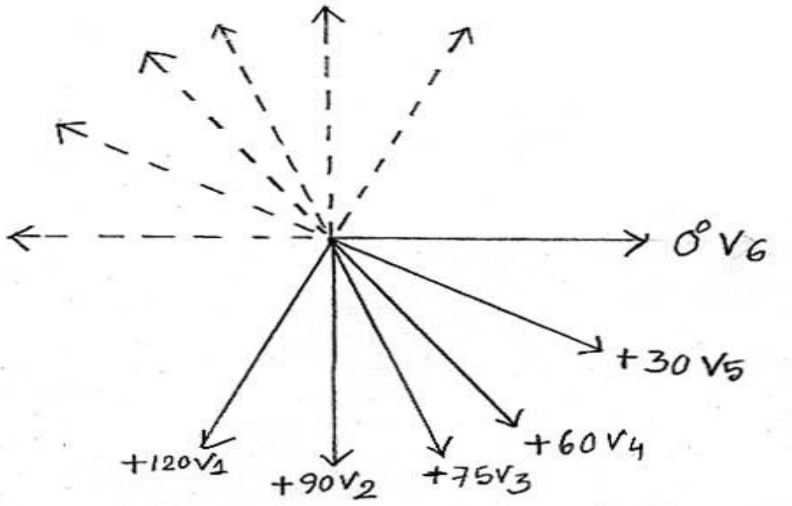
V_5 Same level as V_4, on anterior axillary line.

V_6 Same level as V_4, on mid-axillary line.

$V_1 - V_6$







Normal ECG

P wave

- **inverted in aVR**
- **most prominent in I &II and almost equal**
- **less in III, aVL and aVF**

QRS complex

- **large and inverted in aVR**
- **largest in II**
- **less and equal in I and aVF**
- **least in III and aVL**

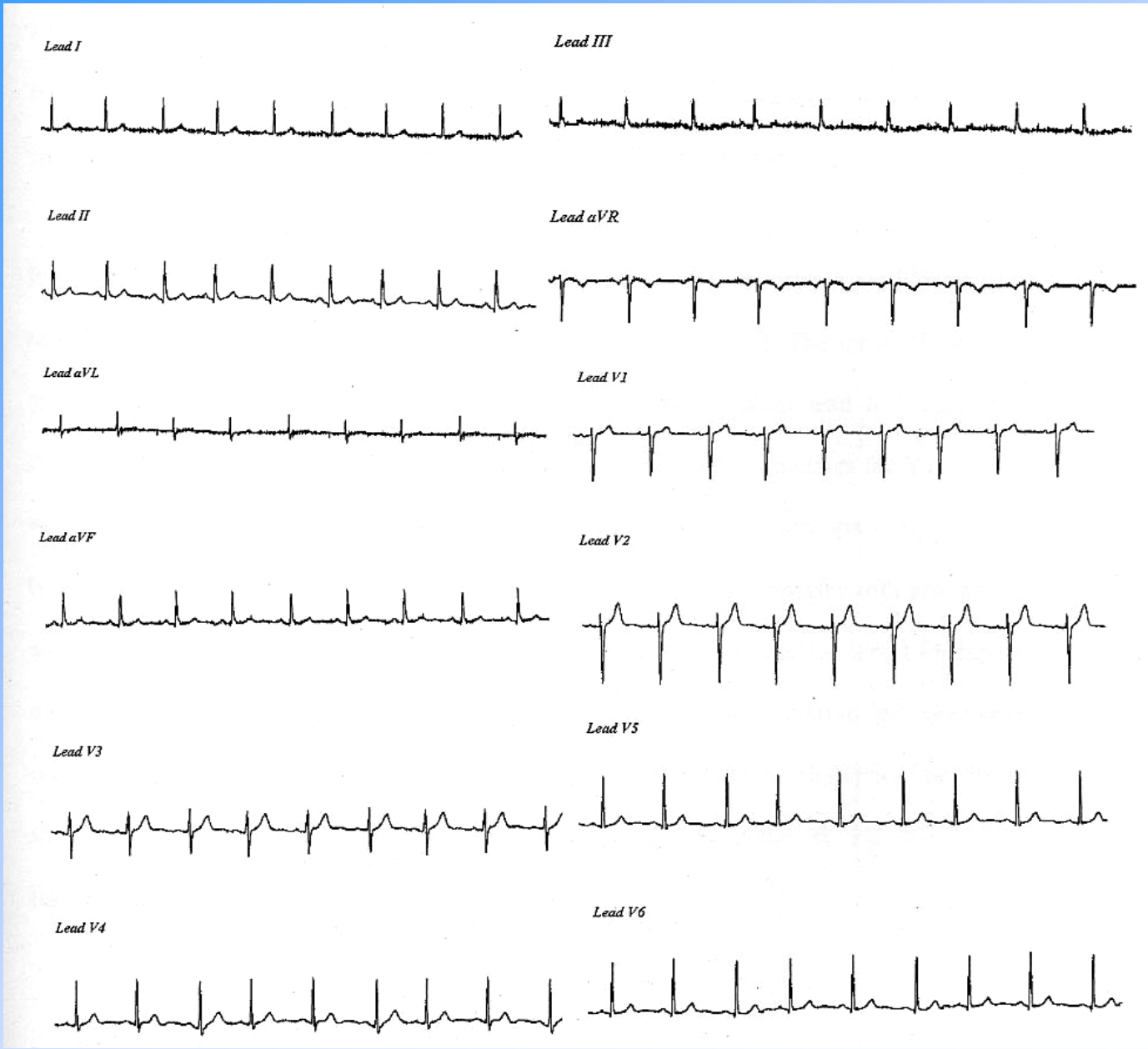
T wave

- **almost 0 in III**
- **almost equal in I and II**

V leads

- **excitation starts at V_3 and moves both ways**

Normal ECG


Lead I
Lead III
Lead II
Lead aVR
Lead aVL
Lead V1
Lead aVF
Lead V2
Lead V3
Lead V5
Lead V4
Lead V6

Changes in ECG

Amplitude and duration of wave and/or complex
Interval and/or segment length
Phase reversal
Absence and/or multitude of some waves
Base line
Heart rate: too high (Tachycardia) or too low (Bradycardia)

Why?

change in ion due to

less concentration in normal fluid flow
less fluid with normal concentration
obstruction in pathways

Cardiac Abnormality

Not all are detectable by ECG

Only five are discussed: detectable in ECG

- **Atrio-ventricular (AV) Block**
- **Atrial Arrhythmias**
- **Ventricular Recovery and Injury**
- **Ventricular Fibrillation (VF)**
- **Bundle Branch Block (BBB)**

AV Block

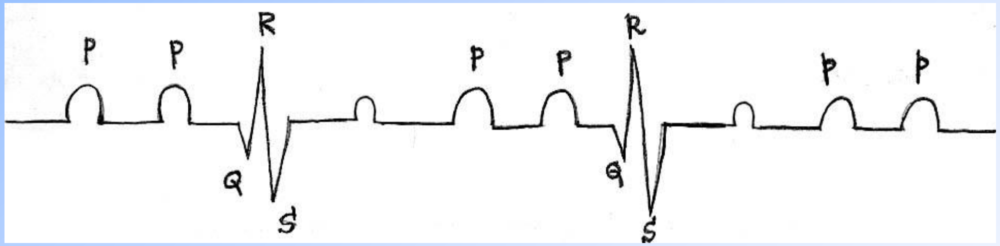
Disturbance in the conduction of excitation from atria to ventricles

Normal T_{PR}= 0.2 sec

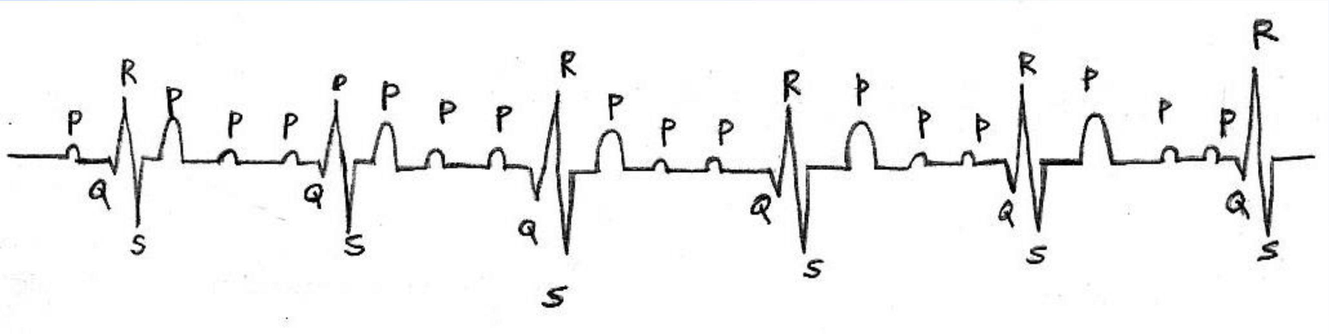
1st degree (1D) block → T_{PR}> 0.2 sec and ECG organized

2nd degree (2D) block → multiple of P wave for each QRS

(2:1, 3:1, etc 2D)



3D or Total Block → atrial pulse does not propagate to ventricles
AV node starts giving pulse (QRS)
no fixed relation between P and QRS



Atrial Arrhythmias

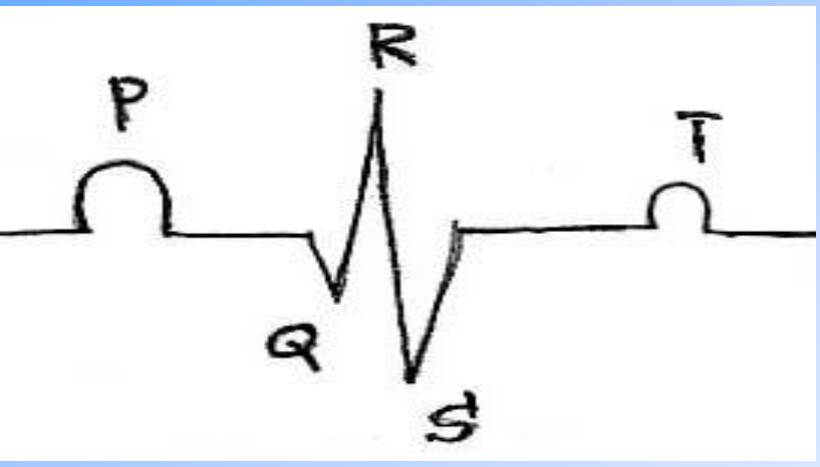
Change in metabolism in atria

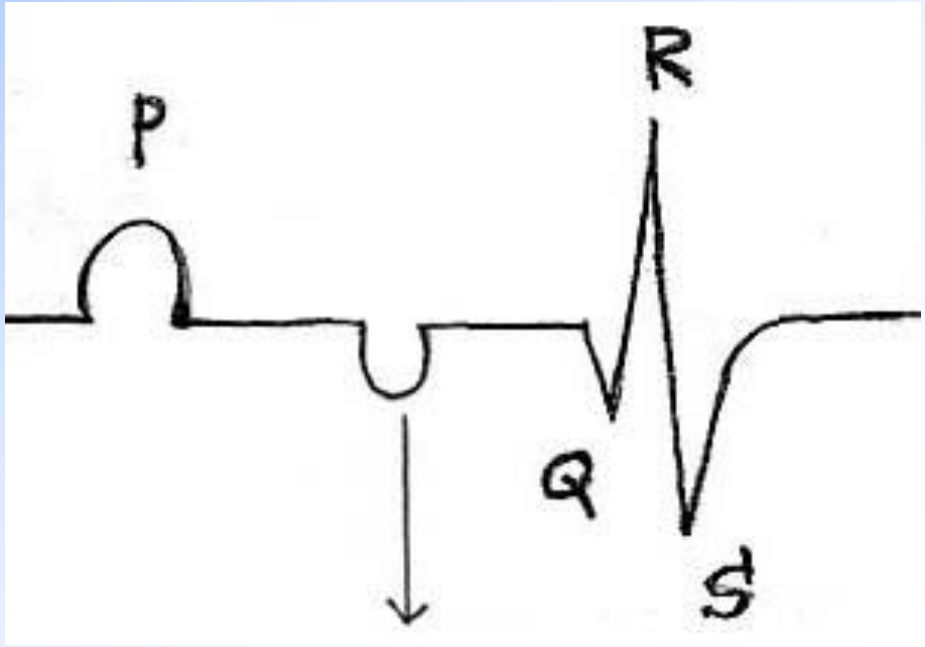
faster ion movement

high rhythmicity of atria (atrial tachycardia)

HR > 200 bpm but organized, atrial flutter

Recovery of atria seen in ECG





Ventricular Recovery and Injury

Ventricular recovery: T-wave and S-T segment

Less oxygenated blood in ventricles

→ full repolarization may not occur

→ S-T segment shifts upwards

Coronary artery becomes blocked

→ myocardial cells cannot sustain normal metabolism and their cell membranes depolarize and remain in this state as the cells die and are replaced by scar tissue (Myocardial Infarction, MI)

Net result

Early shift in the S-T segment

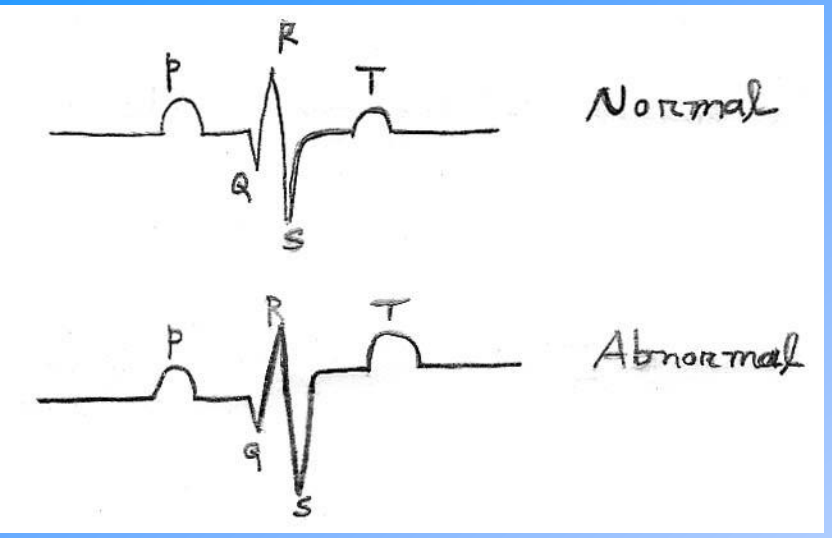
A change in the magnitude of the Q wave.

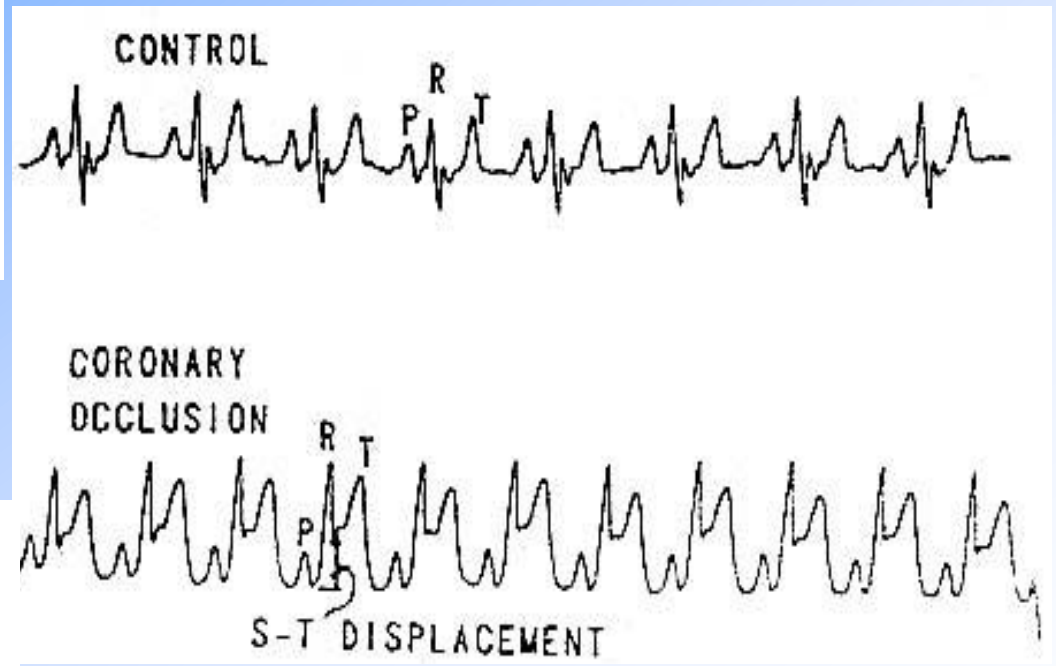
Problems

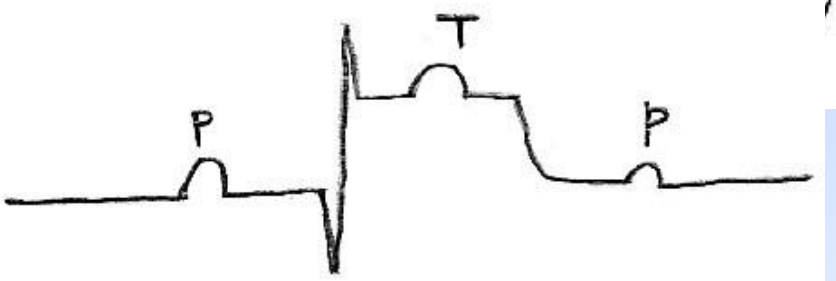
Breathing difficulty (Paraxysmal Disposal)

Lung diseases

Ventricular Recovery and Injury

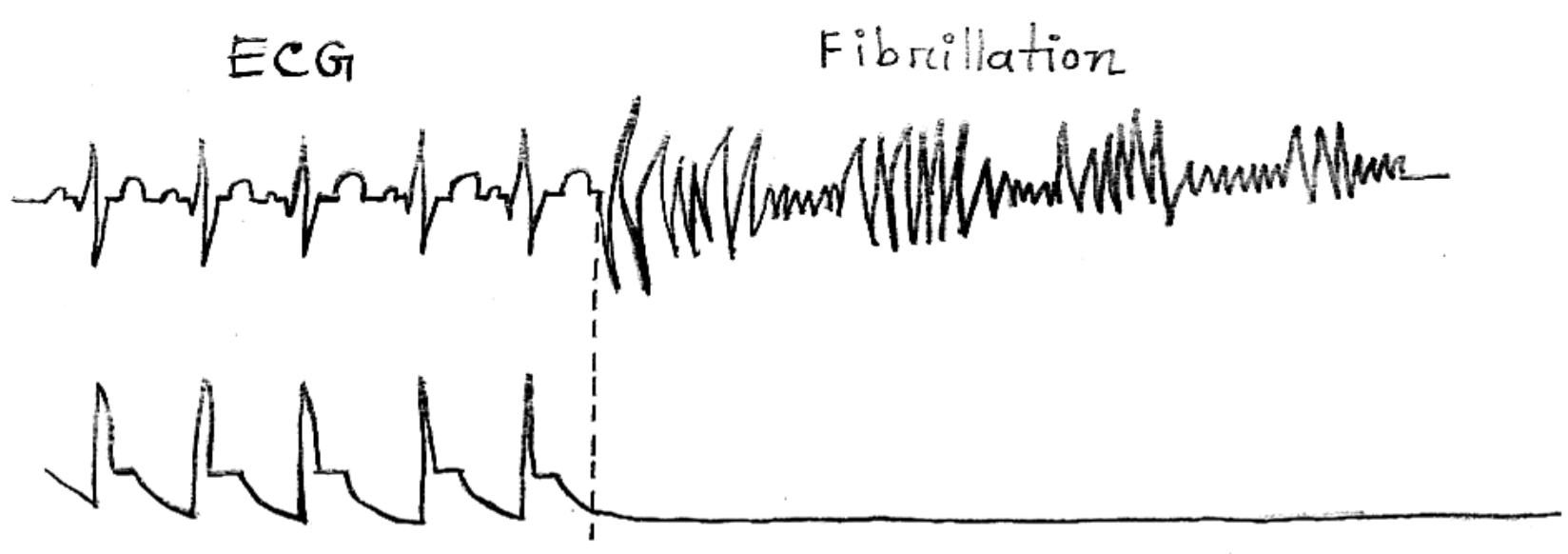






Ventricular Fibrillation

Loss in synchronism of action between atria and ventricles
Normal rhythm is replaced by rapid irregular twitching of muscular wall
Loss of pumping in the ventricles
Fall in blood pressure to a near-zero level
Cardiac output is zero
Activity of ventricle is reduced
Magnitude of QRS decreases
Conduction of A-V node, His bundle and Purkinje Fibers absent
QRS-T waves replaced by fibrillation waves
Patients may die within some minutes
Not self-correcting



Ventricular Fibrillation

Remedy

- **Cardiopulmonary resuscitation (CPR)**
  - **chest is rhythmically and forcefully compressed to squeeze blood out of the heart.**
  - **lungs are inflated rhythmically by mouth-to-mouth breathing**
- **Ventricular defibrillation (VD)**
  - **passing a pulse of current through the heart**
  - **failure of VD means end of life**

BBB

Failure of His bundle (main or branch) to transmit excitation

Ventricular Conduction: AV node, His bundle (with left and right branches) and Purkinje fibers where propagation velocity is higher than in ventricular muscle

both ventricular contract simultaneously with maximum force

Block of excitation in any branch

Late excitation (depolarization)

Prolonged QRS

Excitation first appears on the surface of right ventricles due to

The nature of the conduction system

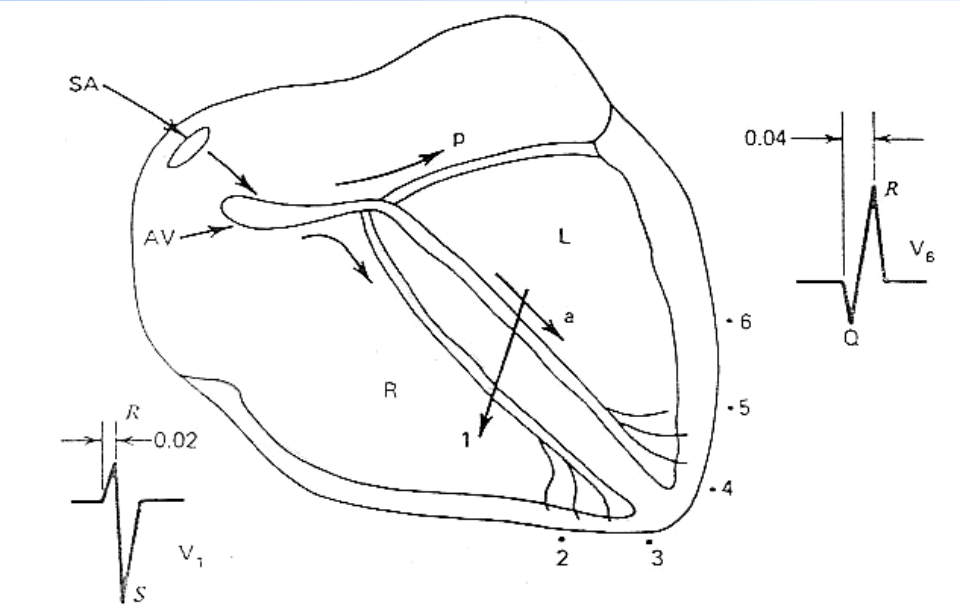
Differing thicknesses of the ventricular myocardium

Normal activation of ventricle produces

R-S wave in V1 , duration 0.02 sec

Q-R wave in V6 , duration 0.04 sec

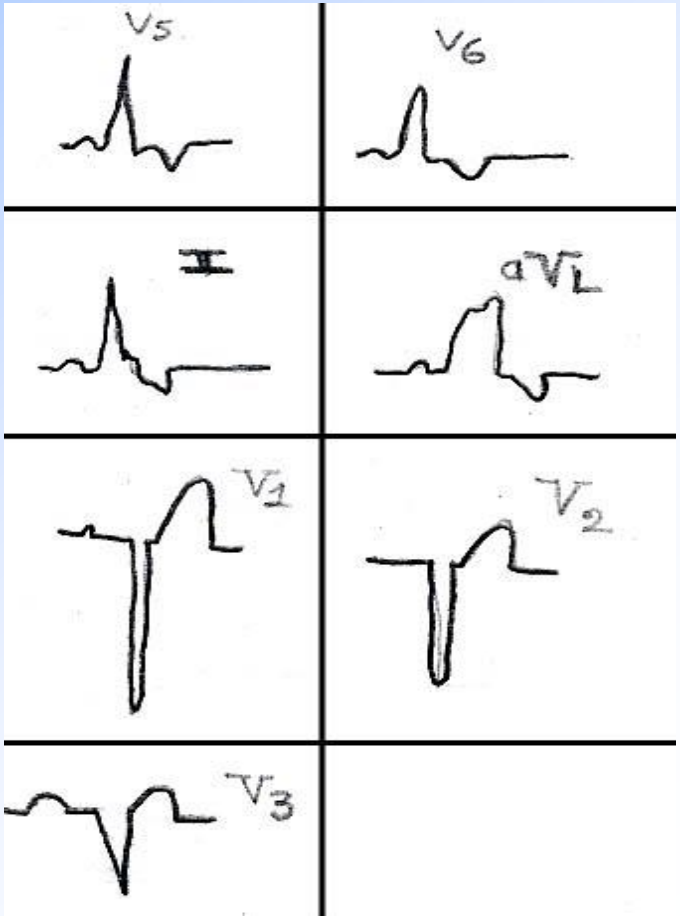
R is prominent in V_6



LBBB

Failure of left bundle to transmit excitation

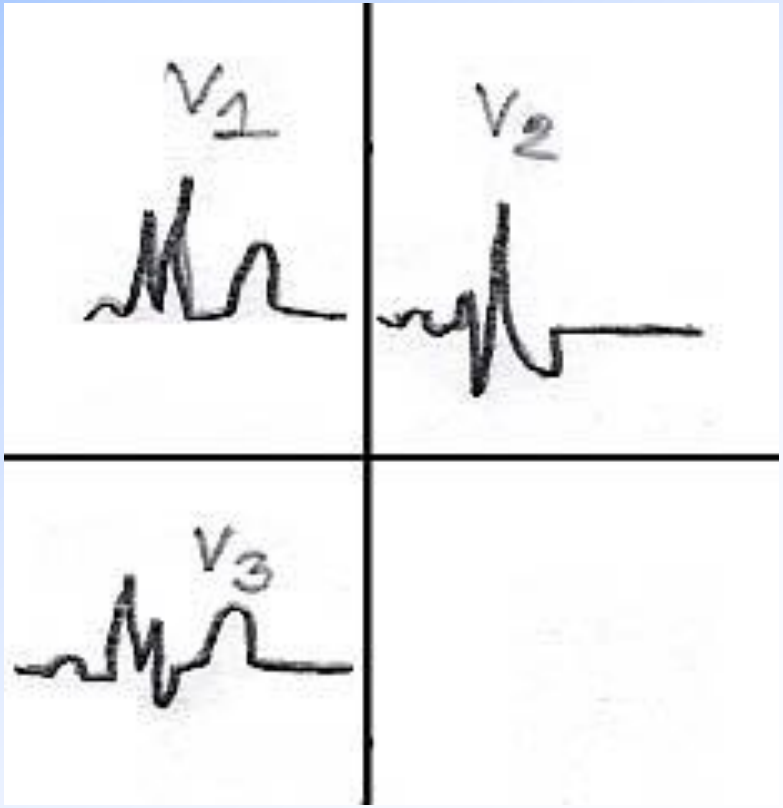
Right-sided V leads show a large, broad, downward wave
Left-sided V leads show a large, broad, upward wave
V_5-V_6: QRS is prolonged downward
V_{1-3}: little evidence of delayed conduction



RBBB

Failure of right bundle to transmit excitation

Left-sided V leads show a large, broad, downward wave
Left-sided V leads show a prominent S wave
V_{1-3}: QRS is prolonged and M-shaped



Questions?
Comments!

Thank You !!!