ECG Changes In Myocardial Infarction

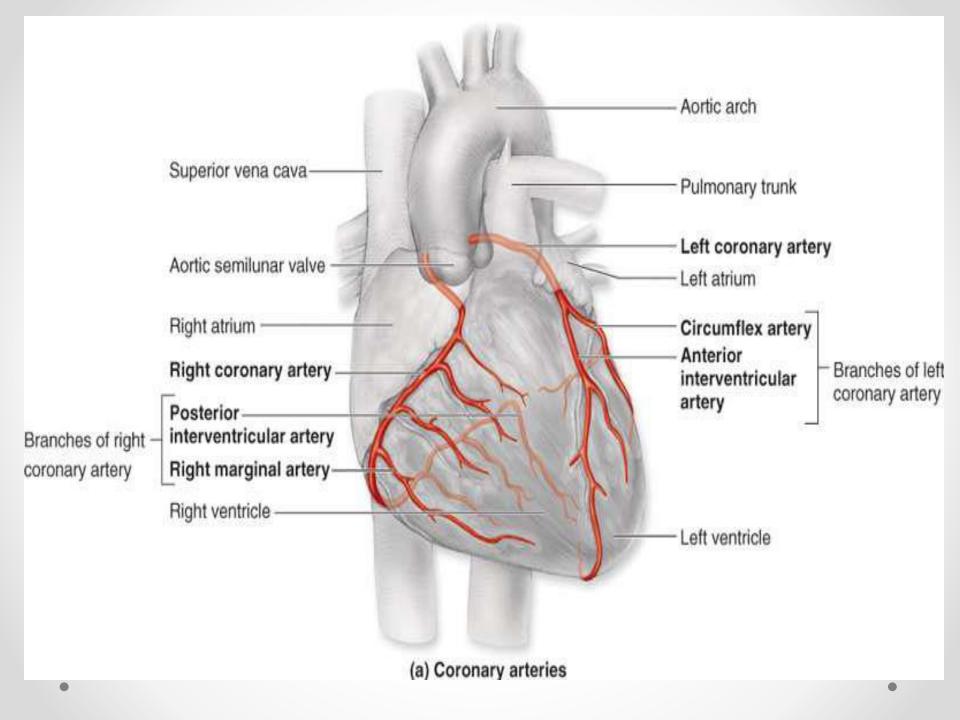
AFSHEEN MAHMOOD NOOR

CORONARY CIRCULATION

CONSIST OF

- 1) Arterial supply
- 2) Venous drainage
- 3) Lymphatic drainage

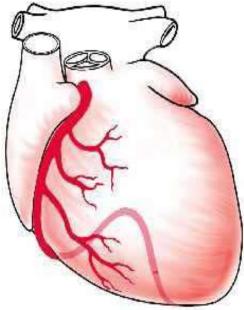
- The cardiac muscle is supplied by two coronary arteries the right and left coronary arteries.
- Both arteries arises from the sinuses behind the cusps of the aortic valves at the root of the aorta.



Right Coronary artery

Smaller than left coronary artery.

• Arises from anterior coronary sinus.



Course

- Emerges from the surface of heart between pulmonary trunk and right auricle.
- Winds round the inferior border to reach the diaphragmatic surface to reach the posterior inter-ventricular groove.
- Terminates by anastomising with left coronary artery

Branches

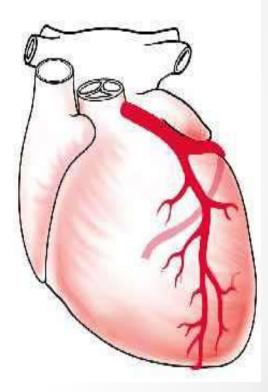
- Large Branches
 - marginal
 - Post-interventricular
- Small branches:
 - Right atrial
 - Infundibular
 - Nodal in 60% cases
 - Terminal

Areas of Distribution

- Right atrium
- Ventricles
 - Greater part of right ventricle, except the area adjoining the anterior interventricular groove.
 - A small part of the left ventricle adjoining the posterior interventricular groove.
- Posterior part or the inter-ventricular septum
- Whole of the conducting system of the heart except a part of the left branch of AV bundle. The SA node is supplied by left coronary artery in 40% cases

Left Coronary Artery

- Larger than the right coronary artery.
- Arises from left posterior aortic sinus.





- Runs forward and to the left and emerges between the pulmonary trunk and the left auricle.
- Here the anterior inter-ventricular branch is given .
- The further continuation of the left coronary artery is sometimes called the circumflex artery.
- After giving off the anterior interventricular branch it runs into the left anterior coronary sulcus.
- It winds around the left border and near posterior interventriular groove it terminates by anastomosing with the right coronary artery.

Branches

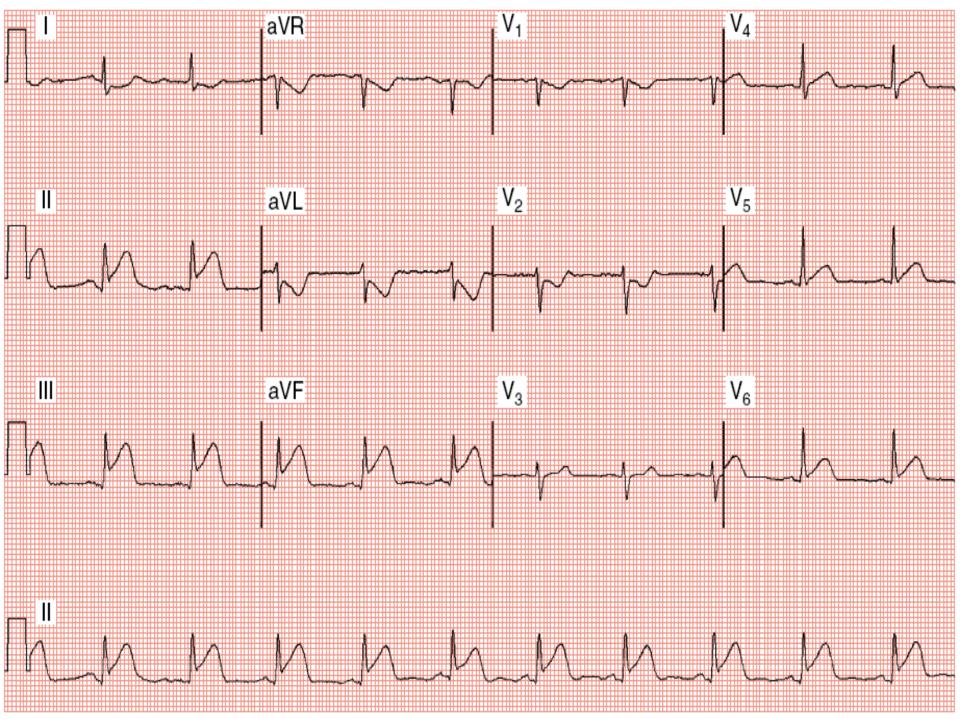
- Large Branches:
 - Anterior interventricular
 - Branch to the diaphragmatic surface of the left ventricle
- Small Branches:
 - Left atrial
 - Pulmonary
 - Terminal

Areas of Distribution

- Left atrium
- Ventricles:
 - Greater part of left ventricle, except the area adjoing the posterior interventricular groove.
 - A small part of right ventricle adjoining the anterior interventricular groove.
- Anterior part of interventricular septum.
- Part of left branch of AV bundle

Clinical Scenario

- A 65-year-old gentleman, Smoker, Diabetic & Hypertensive presented to the emergency with central chest pain, of half an hour duration, it was of severe intensity, crushing in nature, radiating to the left arm, accompanied with mild sweating, on examination, he was looking pale with sweating, pulse of 40 bpm, Bp of 90/60 mm Hg, rest of the examination was unremarkable.
- His ECG was done which showed marked ST segment elevation in lead II, III, AVF & complete AV Block.
- He was managed with SK, LMWH, Aspirin & a Pacemaker was inserted.

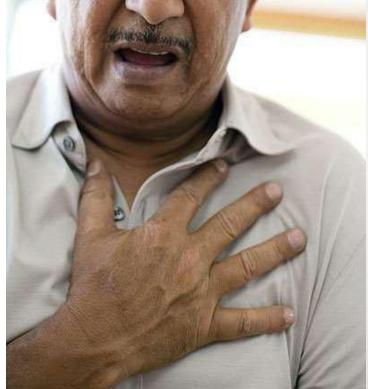


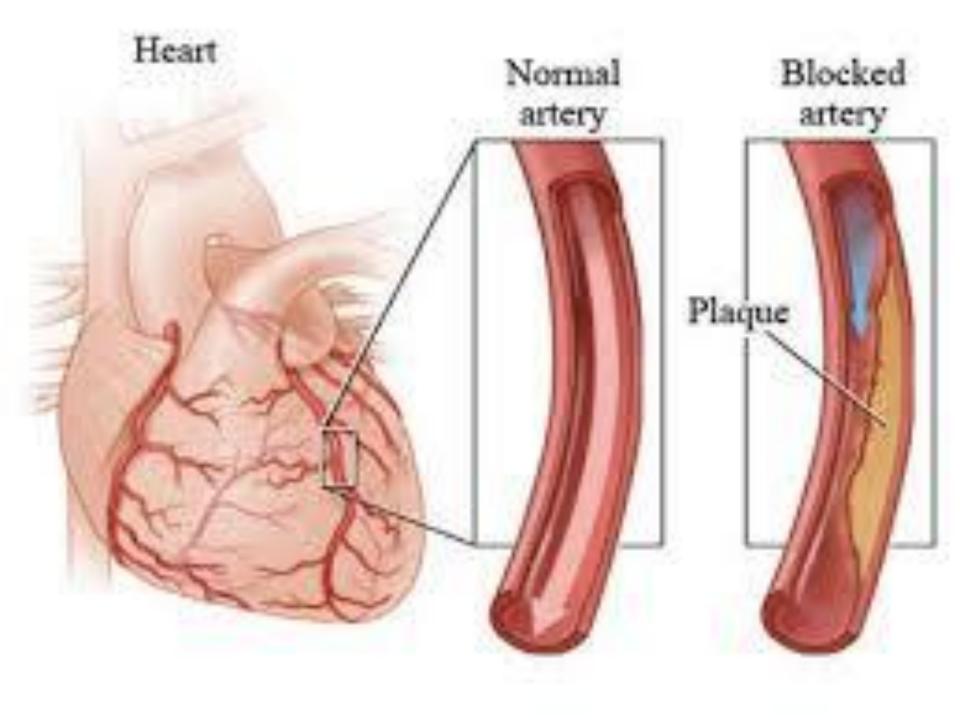
DIAGNOSIS???

Myocardial Infarction (MI) commonly

referred to as heart attack.

 Occurs when blood flow decreases or stops to a part of the heart, causing damage to the heart muscle.





How atheroma can cause a myocardial infarction

Coronary artery magnified

Endothelial cells

Atheroma – fatty material within the wall Below the endothelial cella

Myocardial infarction – blood cannot reach the cells → not enough oxygen → stop respiration → ventricular systole stops

SOME IMPORTANT RISK FACTORS FOR CORONARY ARTERY DISEASE

Fixed

Modifiable

- Age
- Male sex
- Family history

- Smoking
- Hypertension
- Lipid disorders
- Diabetes mellitus
- Haemostatic variables
- Sedentary lifestyle
- Obesity
- Dietary deficiencies of antioxidant vitamins and polyunsaturated fatty acids





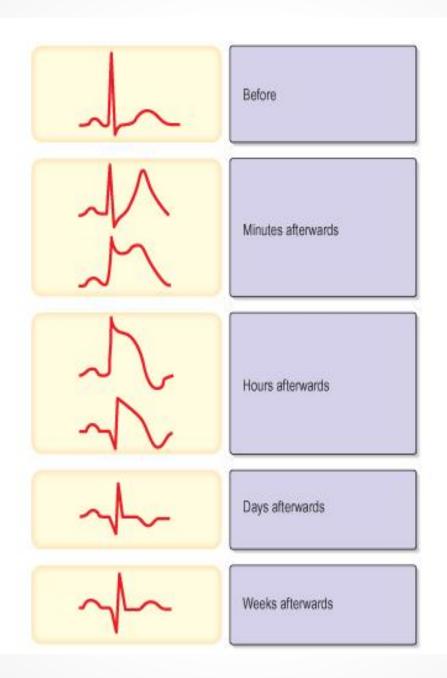


Angina Pectoris

- This is due to myocardial ischemia and presents as a central chest tightness or heaviness, which is brought on by exertion and relieved by rest.
- It may radiate to one or both arms, the neck, jaw or teeth. Other precipitants: emotion, cold weather, and heavy meals.
- Associated symptoms: dyspnoea, nausea, sweatiness, faintness.

Angina vs MI

- LOCATION
- TIMING
- AGGRAVATING OR RELIVING FACTORS
- ASSOCIATED SYMPTOMS
- ECG CHANGES



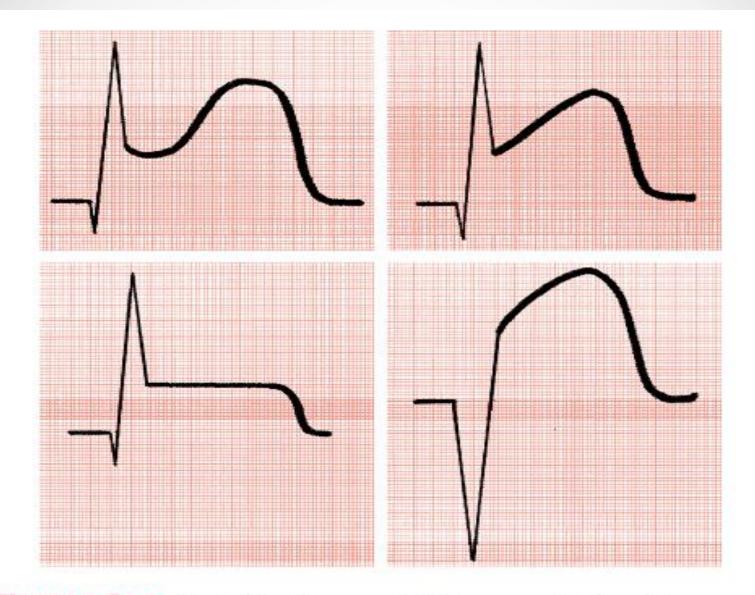
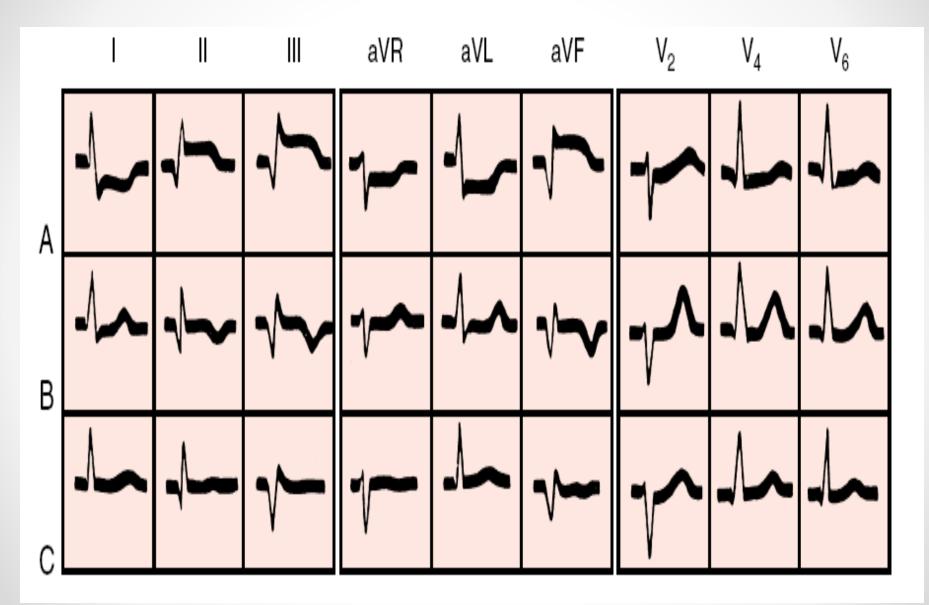
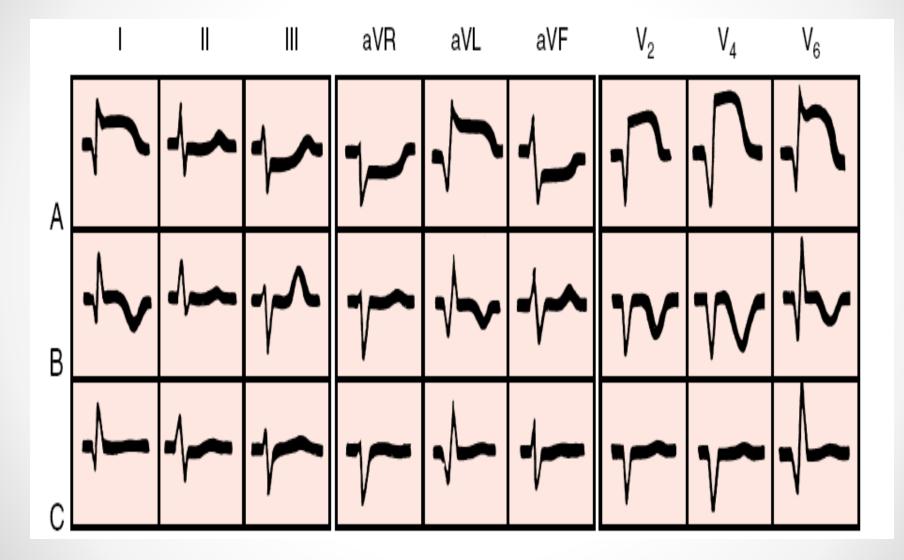


Figure 8-6. Variable shapes of ST segment elevations seen with acute myocardial infarctions.



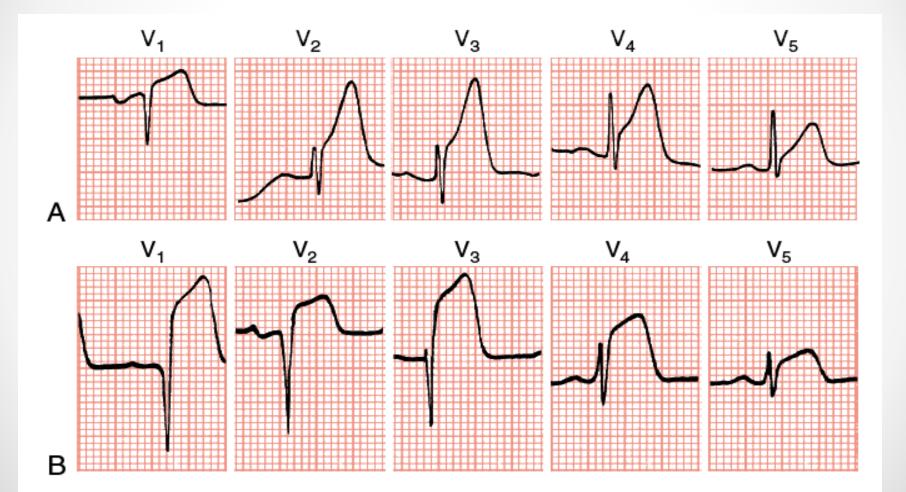
Inferior wall MI

- A, Acute phase of an inferior wall myocardial infarction: ST segment elevations and new Q waves.
- **B**, Evolving phase: deep T wave inversions.
- C, Resolving phase: partial or complete regression of ST-T changes (and sometimes of Q waves). In A and B, notice the reciprocal ST-T changes in the anterior leads (I, aVL, V2, V4).



Anterior wall MI

- **A,** Acute phase of an anterior wall infarction: ST segment elevations and new Q waves.
- **B**, Evolving phase: deep T wave inversions.
- **C,** Resolving phase: partial or complete regression of ST-T changes (and sometimes of Q waves). In *A* and *B*, notice the reciprocal ST-T changes in the inferior leads (II, III, and aVF).



ECG Localization of MI

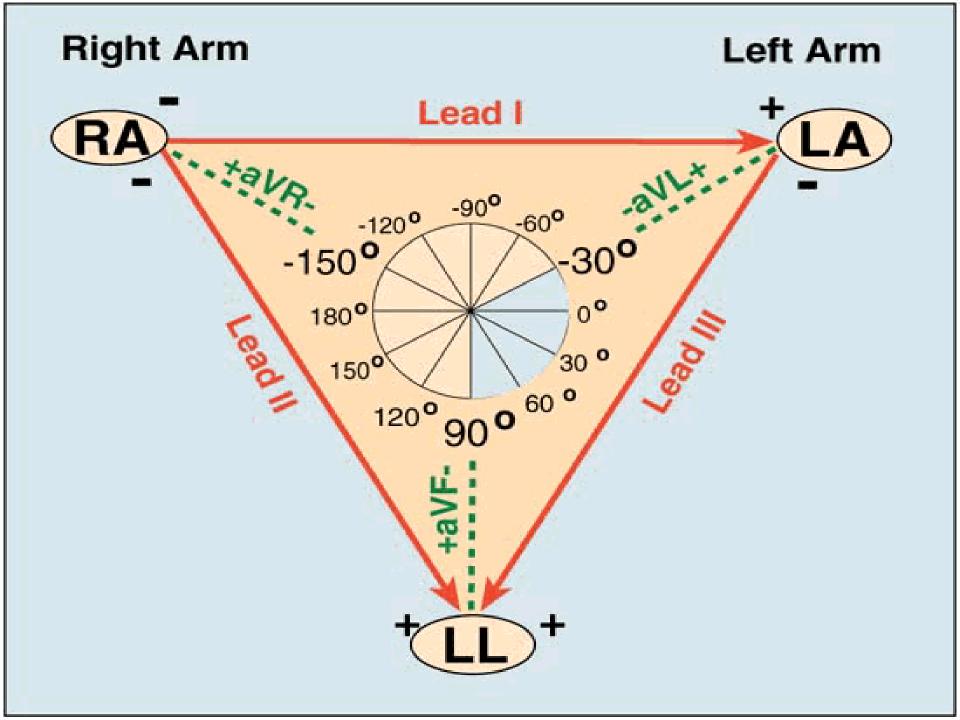
- Chest leads from a patient with acute anterior ST segment elevation myocardial infarction (STEMI).
- A, In the earliest phase of the infarction, tall, positive (hyperacute) T waves are seen in leads V2 to V5.
- B, Several hours later, marked ST segment elevation is present in the same leads (current of injury pattern), and abnormal Q waves are seen in leads in V1 and V2.

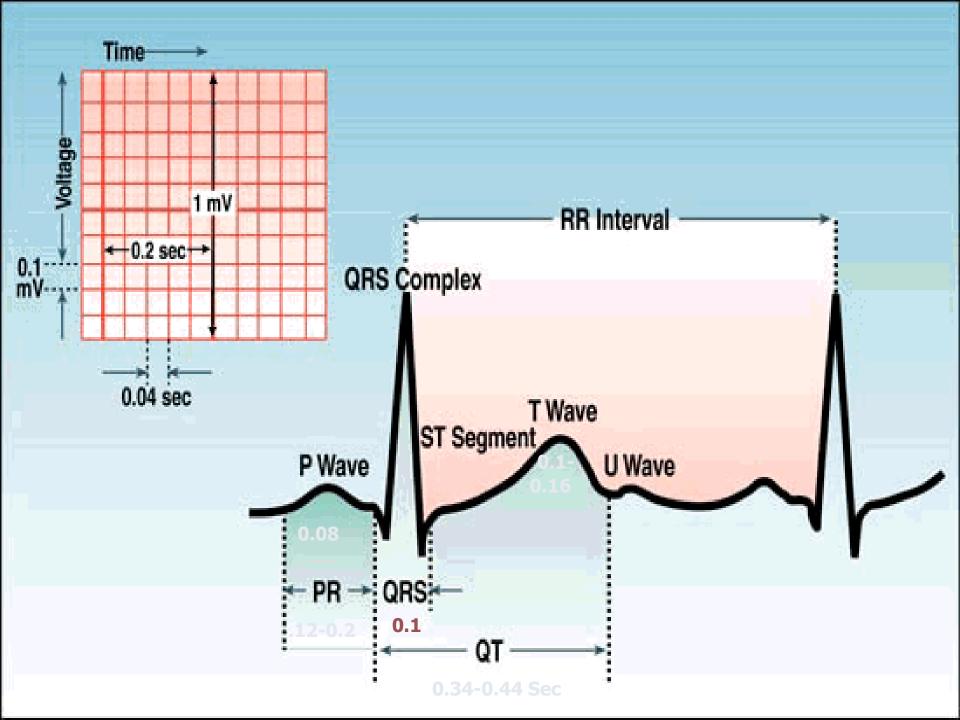
- MIs are generally localized to a specific portion of the left ventricle, affecting either the anterior or the inferior wall.
- Anterior infarctions are sometimes designated as anteroseptal, strictly anterior, or anterolateral/anteroapical, depending on the leads that show signs of the infarction

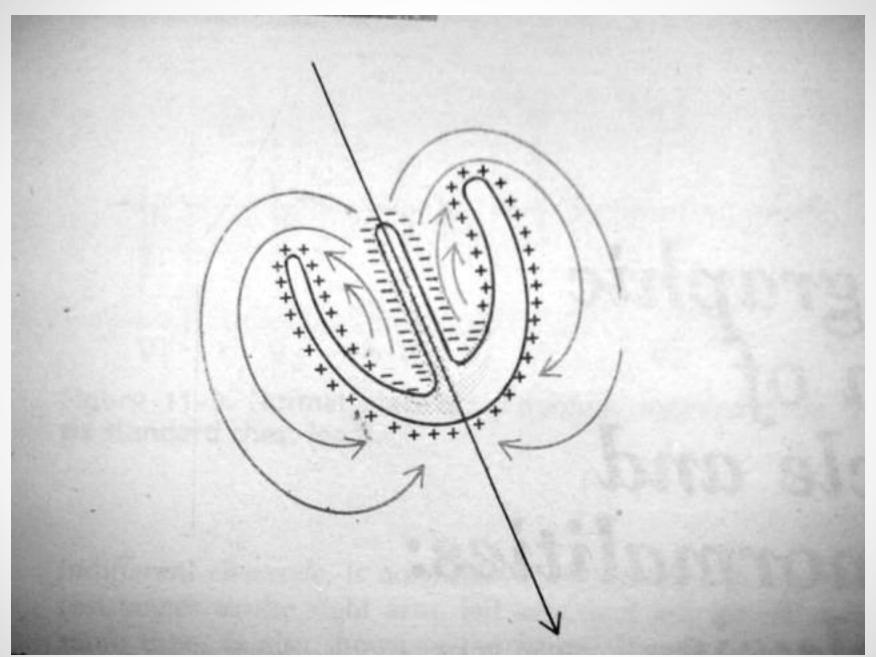
LOCALIZATION OF CORONARY CIRCULATION IN M.I.

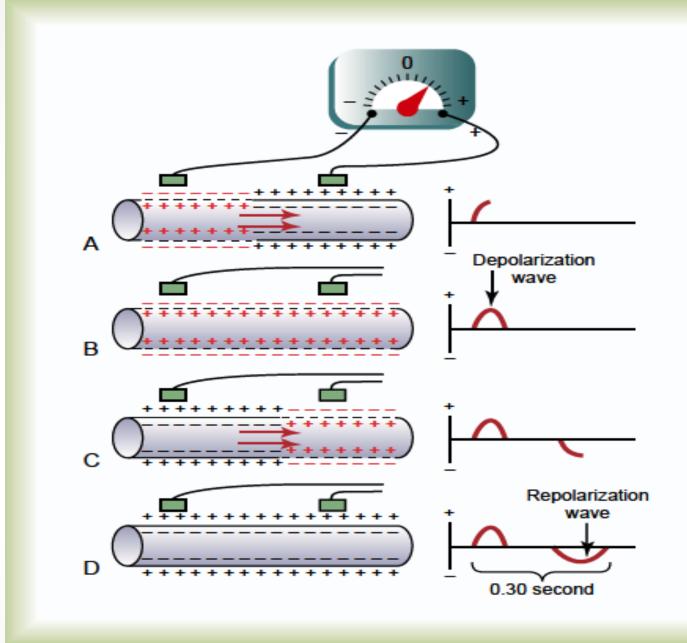
ANATOMIC	ECG LEADS COR	ONARY ARTERY
Septal	V1-v2	Proximal LAD
Anterior	V3-V4	LAD
Apical	V5-V6	Distal LAD, LCx, or RCA
Lateral	I, aVL	LCx
Inferior	II, III, aVF	RCA(85%), LCx (15%)
RV	V1-V2 & V4R	Proximal RCA
Posterior	ST depression V1-V3	RCA or LCx

Current of Injury



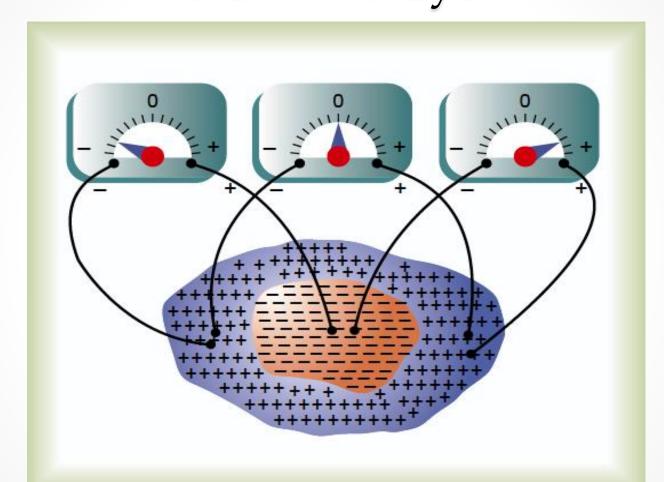




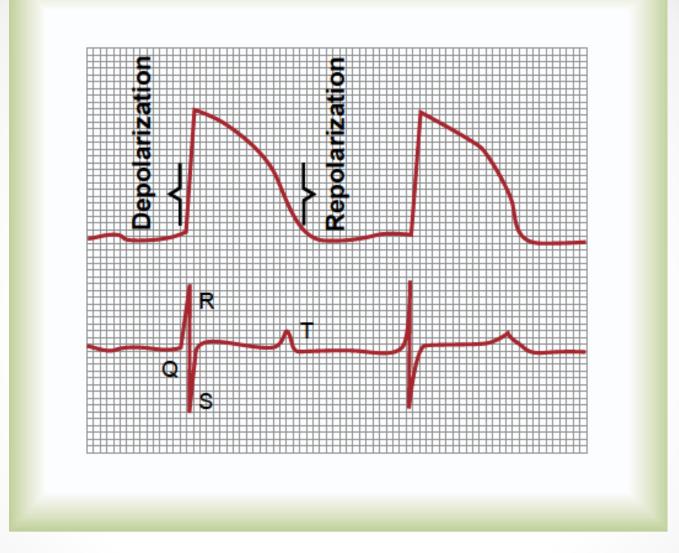


Recording the depolarization wave (A and B) and the repolarization wave (C and D) from a cardiac muscle fiber

Flow of Current Around the Heart During the Cardiac Cycle



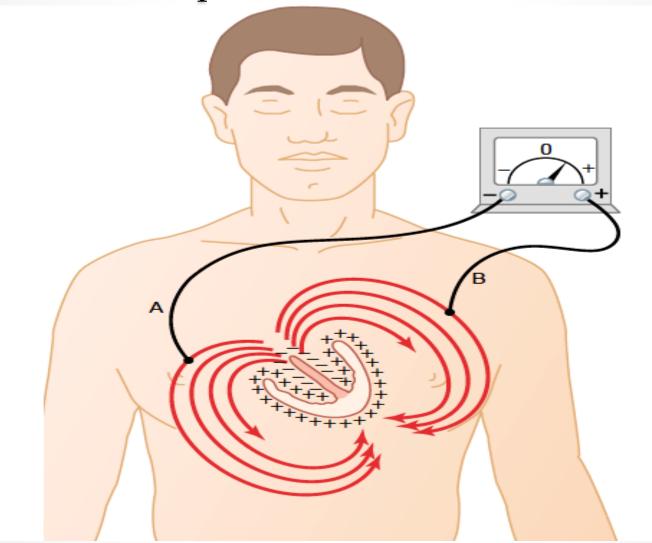
Instantaneous potentials develop on the surface of a cardiac muscle mass that has been depolarized in its center



Above, Monophasic action potential from a ventricular muscle fiber during normal cardiac function, showing rapid depolarization and then repolarization occurring slowly during the plateau stage but rapidly toward the end. *Below*, *Electrocardiogram recorded* simultaneously

Flow of current in the chest around partially

depolarized ventricles



Electrodes present near the base of the heart are -ve and near the apex are +ve.

Basic Concepts

- When current moves towards recording electrode positive deflection
- Movement away from recording electrode causes negative deflection
- Biphasic deflection when recording electrode positioned midway

Resting state (polarized)

- Membrane interior negative
- Exterior positive
- Balanced state mentioned by equal positive and negative charges

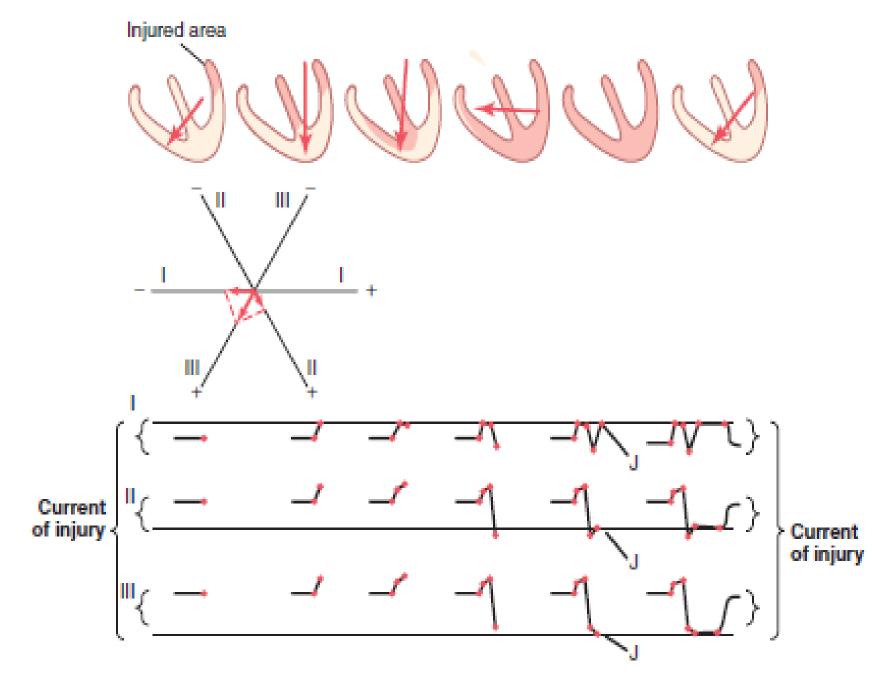
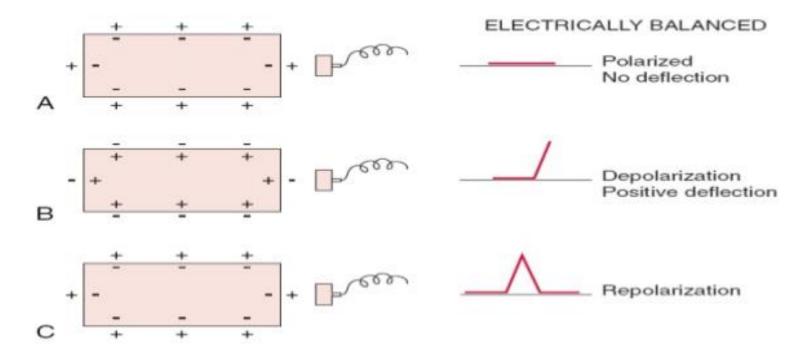


Figure 12-17. Effect of a current of injury on the electrocardiogram.

Action Potential in cardiac tissue



A, Resting cell: Positive ions on the outer surface and negative ions inside equal an electrically balanced or polarized cell. **B**, Depolarized cell: Negative ions on the outer surface and positive ions inside. **C**, Repolarization of cell Positive ions return to the outside.

Current of Injury

- Ischemia, infection& trauma cause myocytes to remain partially or completely depolarized emitting negative charges into surrounding fluids.
- Injured part is negative while healthy remains positive

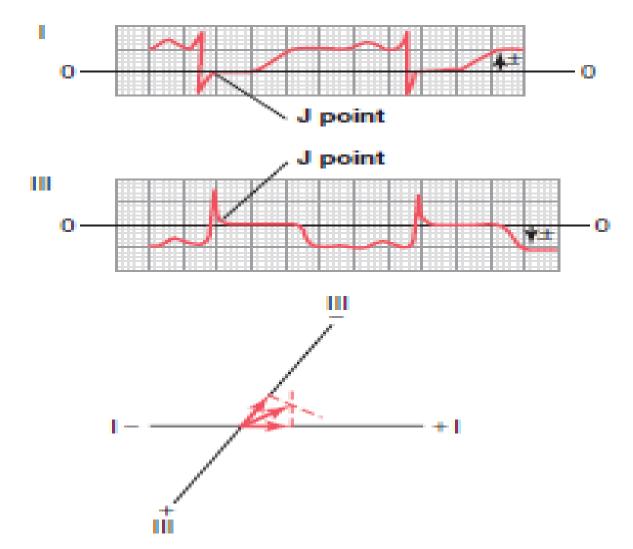


Figure 12-18. J point as the zero reference potential of the electrocardiograms for leads I and III. Also, the method for plotting the axis of the injury potential is shown in the lowermost panel.

J point

- Zero reference point, where myocardium is completely depolarized (injured/ normal) so no current flow.
- Located at end of QRS
- Even current of injury is absent now.
- ECG potential is at zero volt
- Isoelectric line from end of S to beginning of T wave or horizontal line joining the two j points represents zero potential level from which all potentials caused by current of injury are measured

ST Segment Shift

- Current of injury will always cause ST/TP segment shift
- Actually TP not ST segment is shifted from zero reference.
- Commonly TP segment is taken as zero reference with appearance of ST segment shift

