ECG in ACS

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ECG in NSTEMI

- In NSTEMI, patients with ST depression have higher overall mortality than isolated inversed T-wave.
- Normal T-wave:
  - Upright and asymmetric.
  - Frontal plane: tallest T-wave is 5 mm, sometimes up to 8mm
  - Horizontal plane: tallest T-wave is 10 mm, sometimes up to 12 mm.

ECG in NSTEMI – Normal T-wave

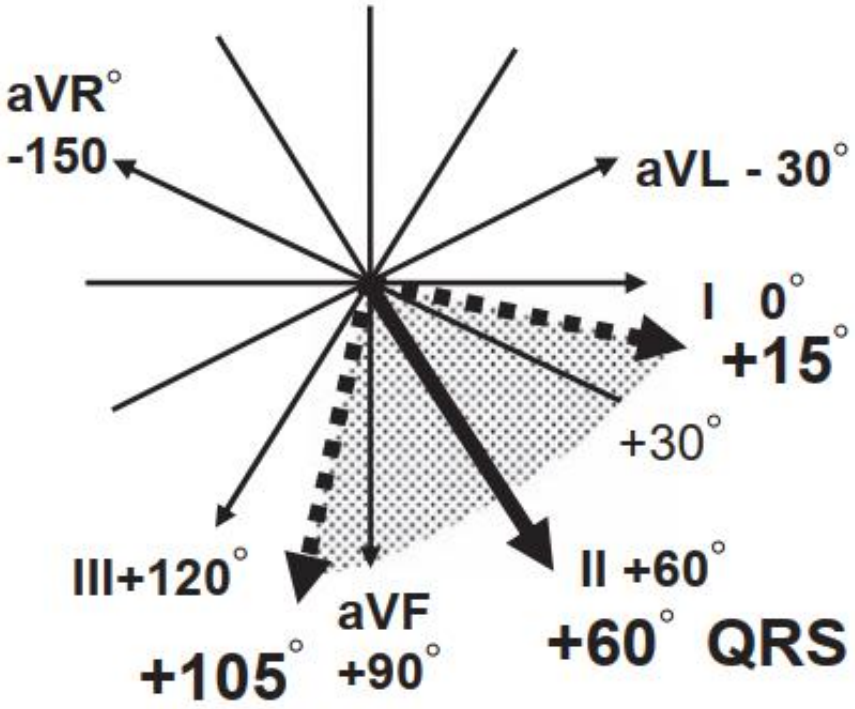


Figure 24.1: Axis of the T Wave in the Frontal Plane. If the axis of the QRS is 60°, the T wave should be within 45° (*shaded*) to the left or right of the axis of the QRS complex as shown.

ECG in NSTEMI – Abnormal T-wave

- According to the shapes of T-wave, there are 4 types:
  - Taller than normal.
  - Depression
    - > 2mm
    - < 2mm.
  - Biphasic
  - Flat
- The causes:
  - Myocardial ischemia
  - Metabolic and ionic abnormalities.
  - Unspecific.

ECG in NSTEMI – Abnormal T-wave

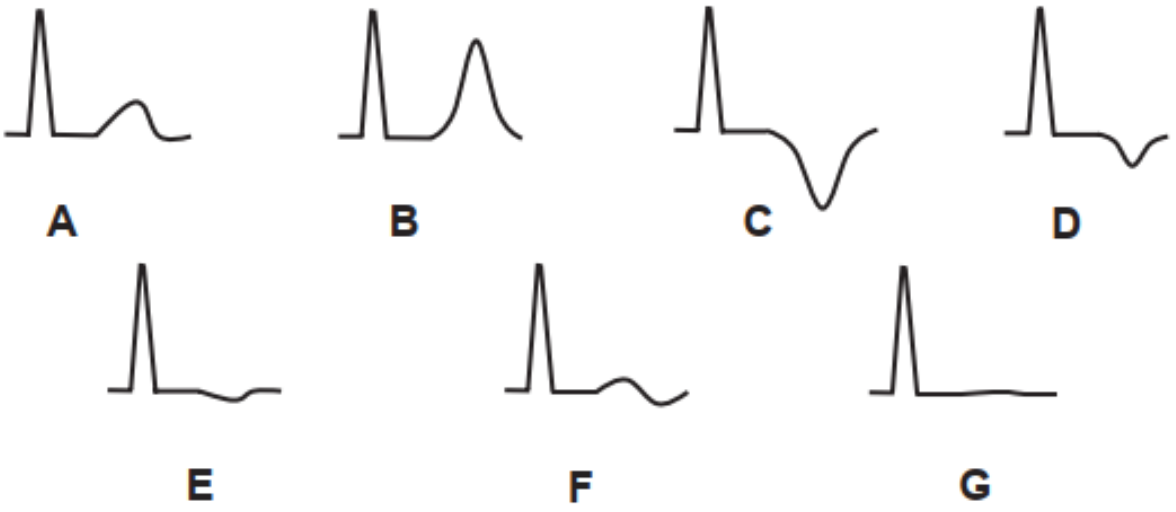
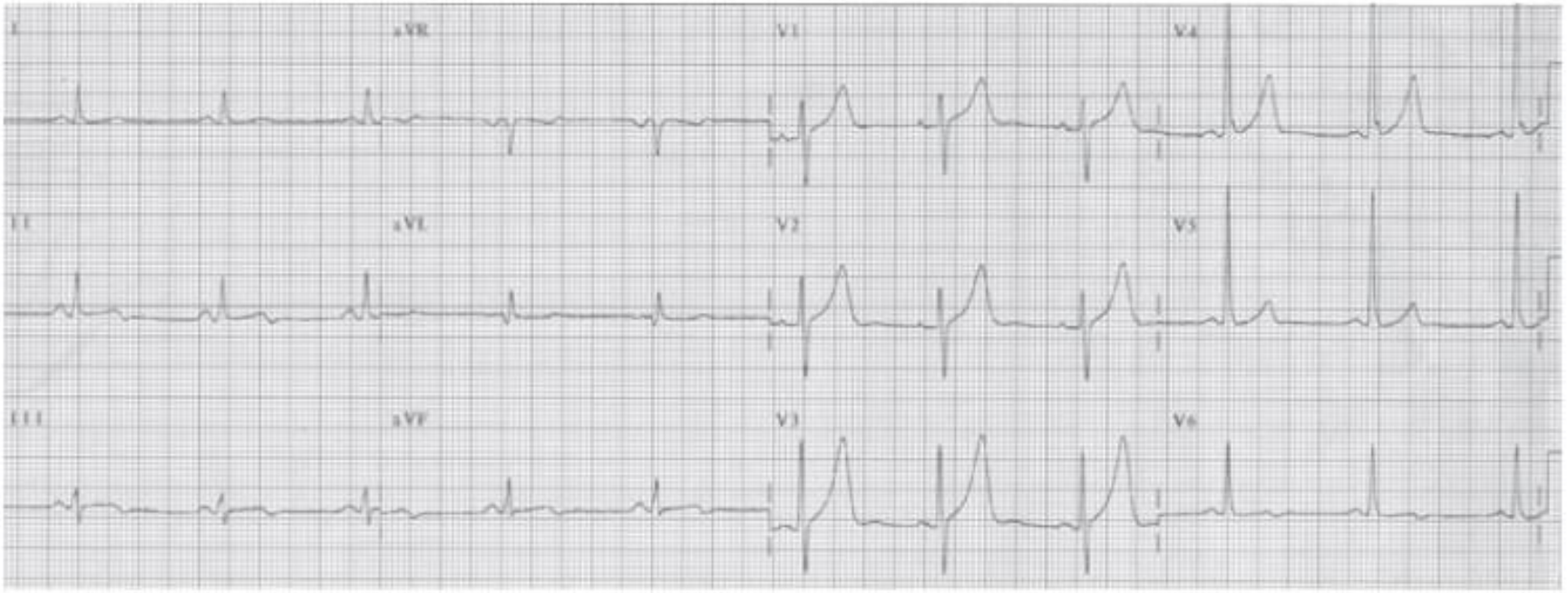


Figure 24.4: T Waves. **(A)** Normal T wave. **(B)** Peaked T waves from subendocardial ischemia. **(C)** Classical deep T-wave inversion due to transmural ischemia. **(D)** Symmetrically but less deeply inverted T wave also due to transmural ischemia. **(E)** Shallow T-wave inversion **(F)** Biphasic T wave. **(G)** Low, flat, or isoelectric T wave. Although the T-wave configuration of **B, C,** and **D** suggests myocardial ischemia, these T-wave abnormalities may also be due to other causes.

ECG in NSTEMI – Abnormal T-wave

- Peaking of T-wave:
  - Subendocardial ischemia.
    - The base of T-wave is broad, prolonged QT-interval.
  - Hyperkalemia:
    - Small U-wave.
    - Generalized.
  - Fluoride intoxication
  - Left ventricular volume overload such as aortic regurgitation

ECG in NSTEMI – Abnormal T-wave



Peaking of T-wave because of subendocardial ischemia

ECG in NSTEMI – Abnormal T-wave

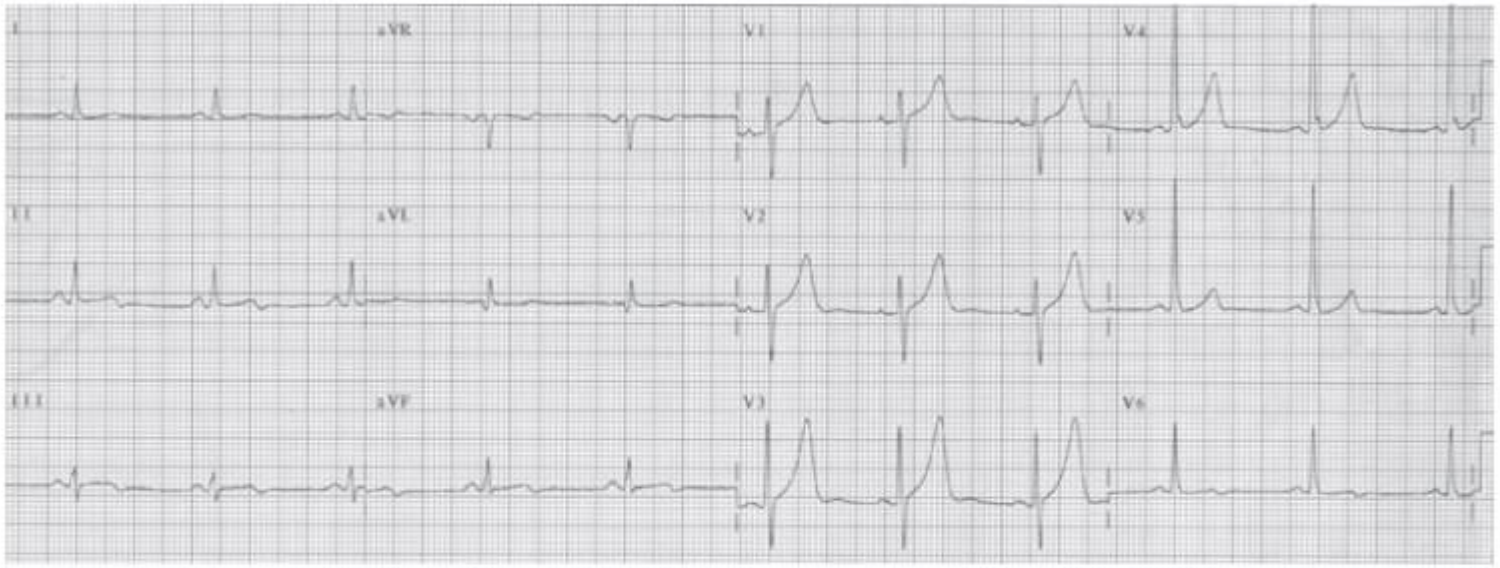
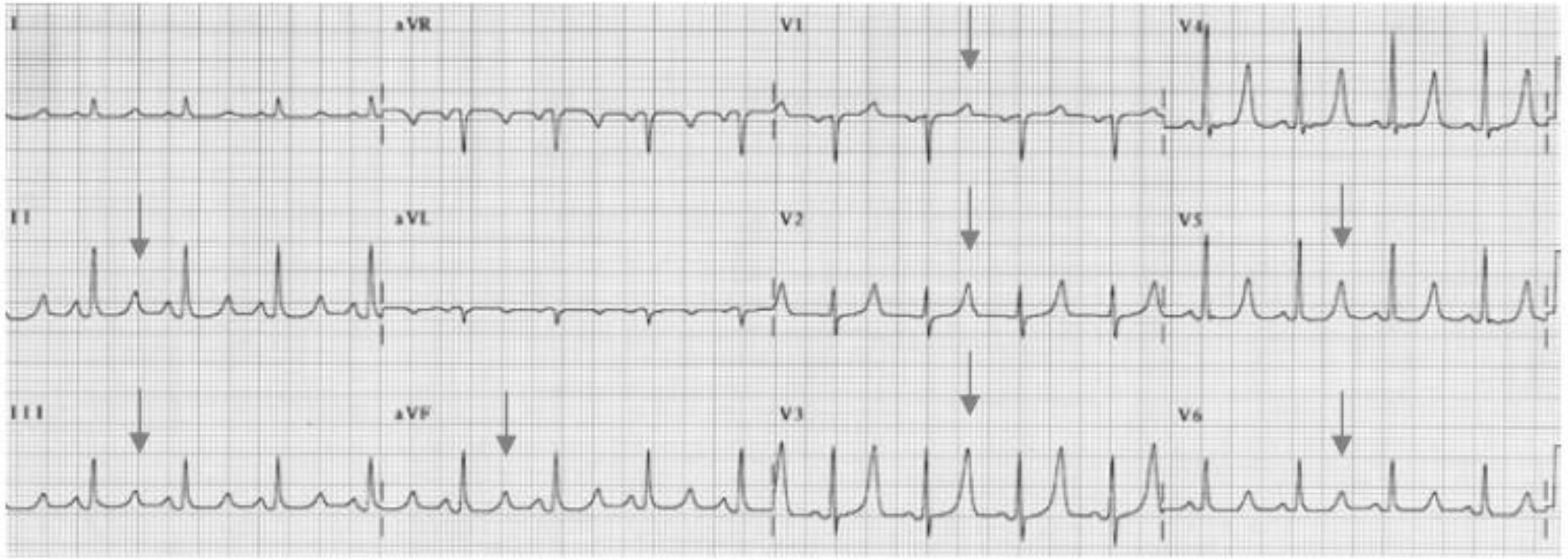


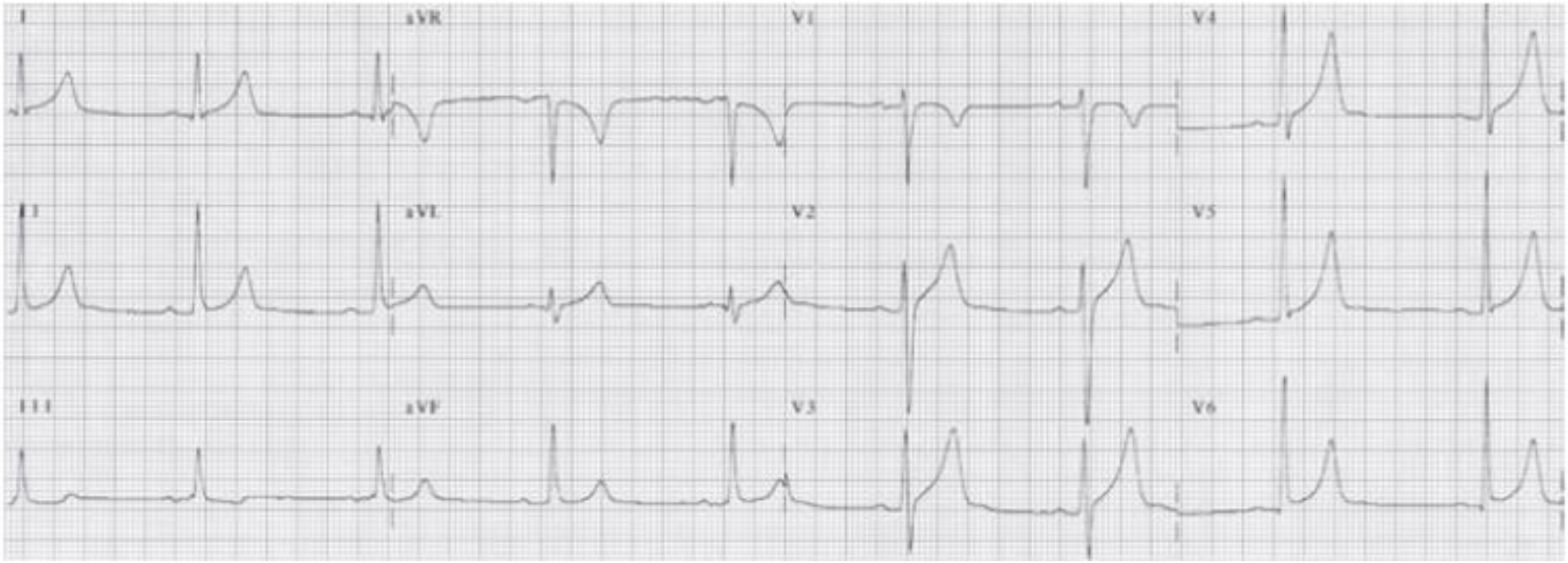
Figure 24.5: Subendocardial Ischemia. Peaking of the T waves is confined to V_1 to V_4 consistent with subendocardial ischemia involving the anterior wall. Note also that the T waves are taller in V_1 than in V_6 and are biphasic in leads II, III, and aVF. Peaking of the T waves mark the area of ischemia and can occur as the initial manifestation of acute coronary syndrome before the onset of ST segment elevation.

ECG in NSTEMI – Abnormal T-wave



Peaking of T-wave because of hyperkalemia

ECG in NSTEMI – Abnormal T-wave



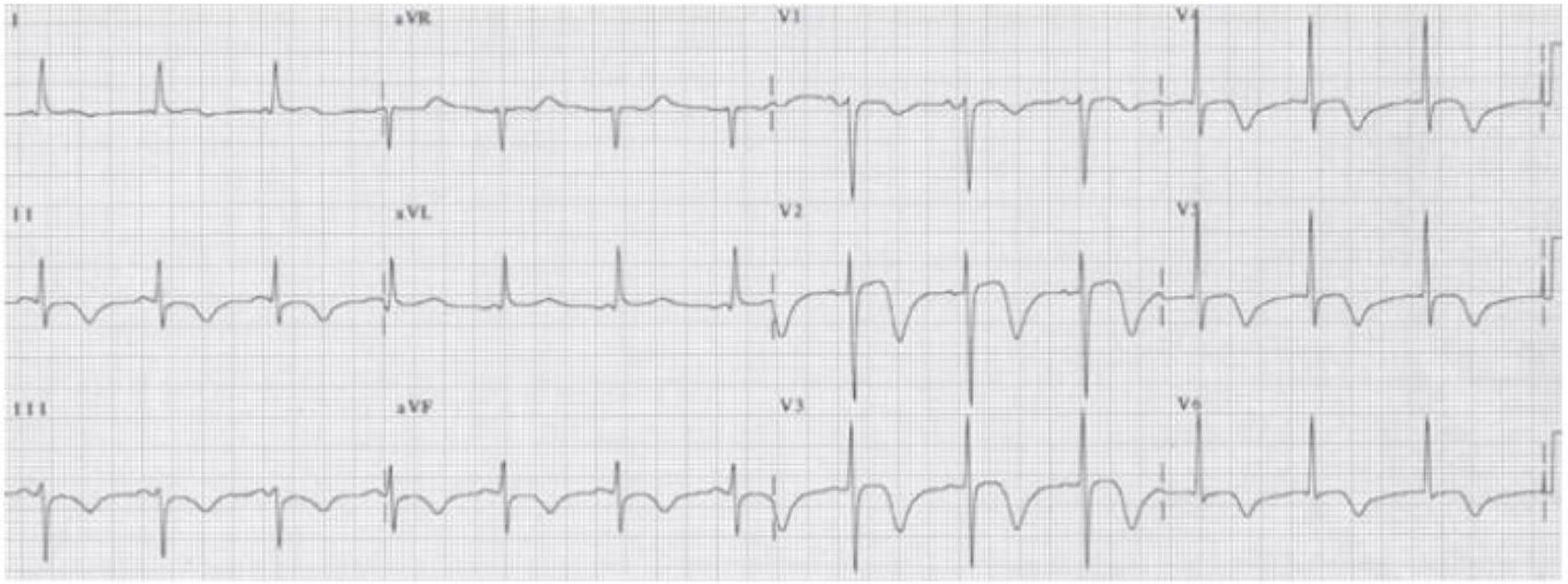
Normal variant, usually associated with early repolarization

ECG in NSTEMI – Abnormal T-wave

- Symmetrical T-wave inversion:
  - Transmural ischemia
    - T-wave infarct.

Other causes of deep T-wave inversion: There are several other causes of deep and symmetrical T-wave inversion other than myocardial ischemia. These include hypertrophic cardiomyopathy especially the apical type, pericarditis, pulmonary embolism, mitral valve prolapse, metabolic conditions, electrolyte disorders, and effect of drugs such as tricyclic antidepressants and antiarrhythmic agents. It can also be due to noncardiac conditions such as cerebrovascular accidents or other craniocerebral abnormalities, peptic ulcer perforation, acute cholecystitis, and acute pancreatitis. It may even be a variant of normal especially in young African American males. Deep symmetrical inversion of the T wave, therefore, is not specific and does not necessarily imply that the T-wave abnormality is due to transmural myocardial ischemia.

ECG in NSTEMI – Abnormal T-wave



Symmetrical T-wave inversion in NSTEMI

ECG in NSTEMI – Abnormal T-wave

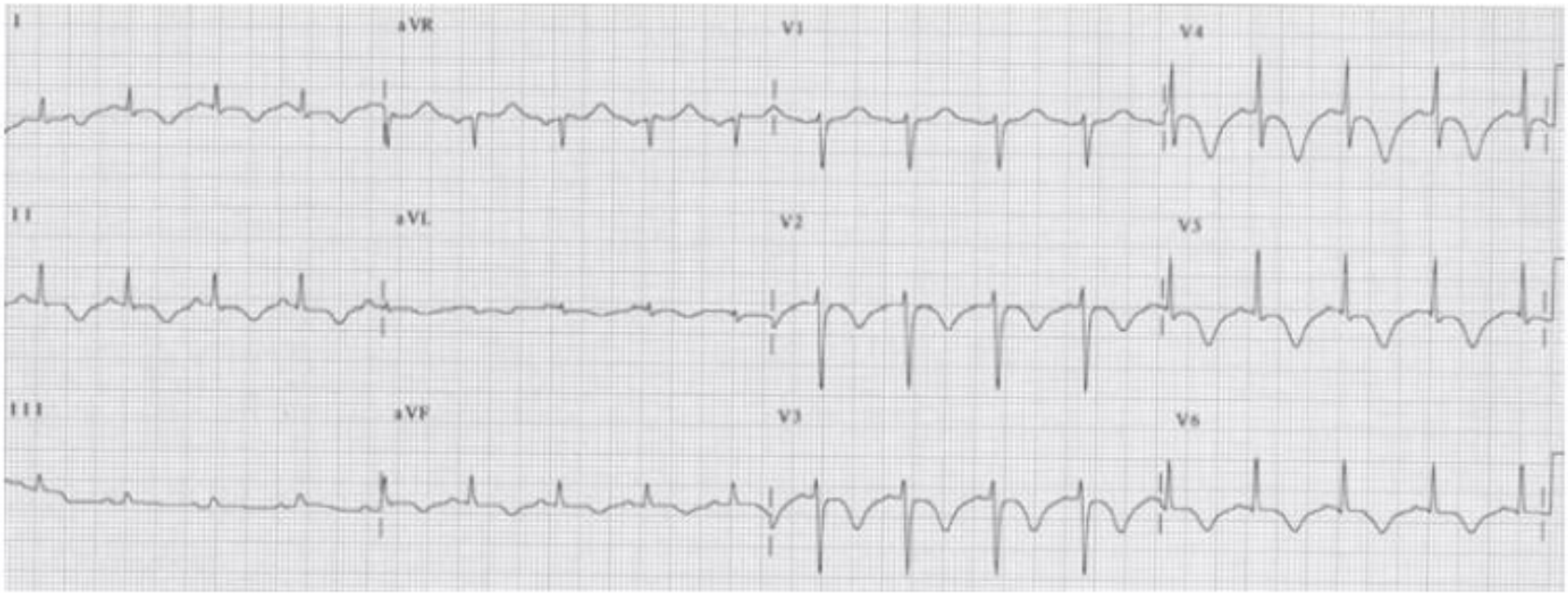


Figure 24.11: T-Wave Inversion from Cerebrovascular Accident Hemorrhage.

ECG in NSTEMI – Abnormal T-wave

- Secondary T-wave inversion:
  - Left ventricular hypertrophy.
  - Block LBBB.

ECG in NSTEMI – Mechanism of T-wave

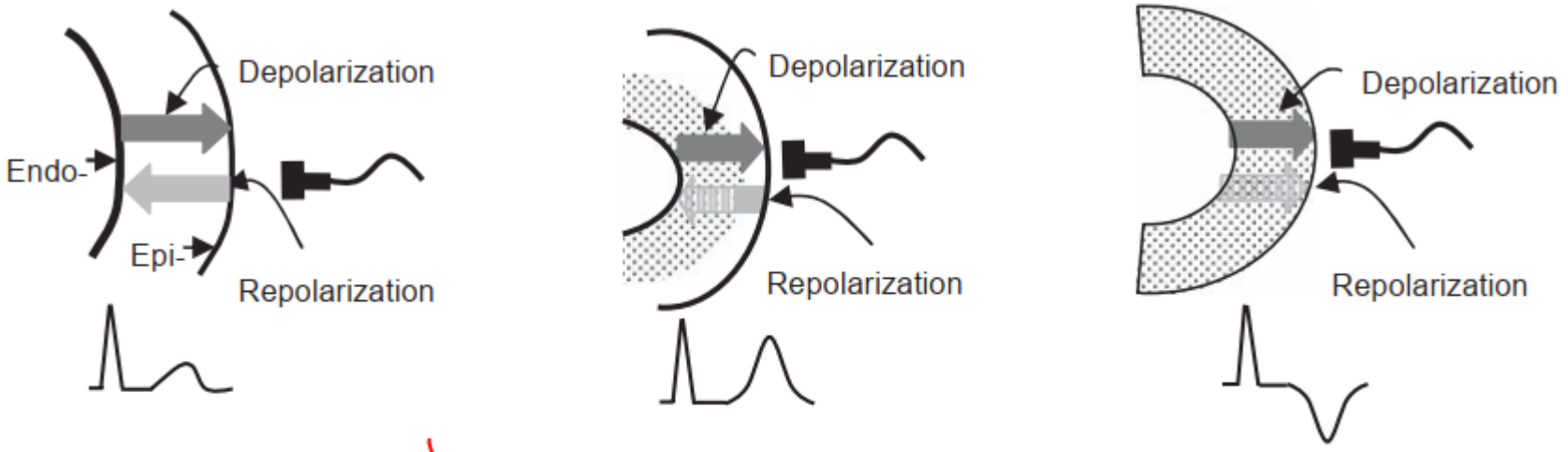


Figure 24.14: **(A) The T Wave** Normal myocardium. Depolarization starts from endocardium to epicardium since the Purkinje fibers are located subendocardially. Repolarization is reversed and is epicardial to endocardial, thus the T wave and QRS complex are both upright. **(B) Subendocardial Ischemia.** The shaded portion represents the area of ischemia. The direction of depolarization and repolarization is similar to normal myocardium. After the repolarization wave reaches the ischemic area, the repolarization wave is delayed causing the T wave to be tall and symmetrical. **(C) Transmural Ischemia.** The direction of repolarization is reversed that of normal and is endocardial to epicardial resulting in deeply and symmetrically inverted T waves.

ECG in NSTEMI – ST-segment

- ST-segment change indicates a more advance stage in myocardial ischemia, called myocardial injury.
- Horizontal or depressed ST-segment is associated with NSTEMI.

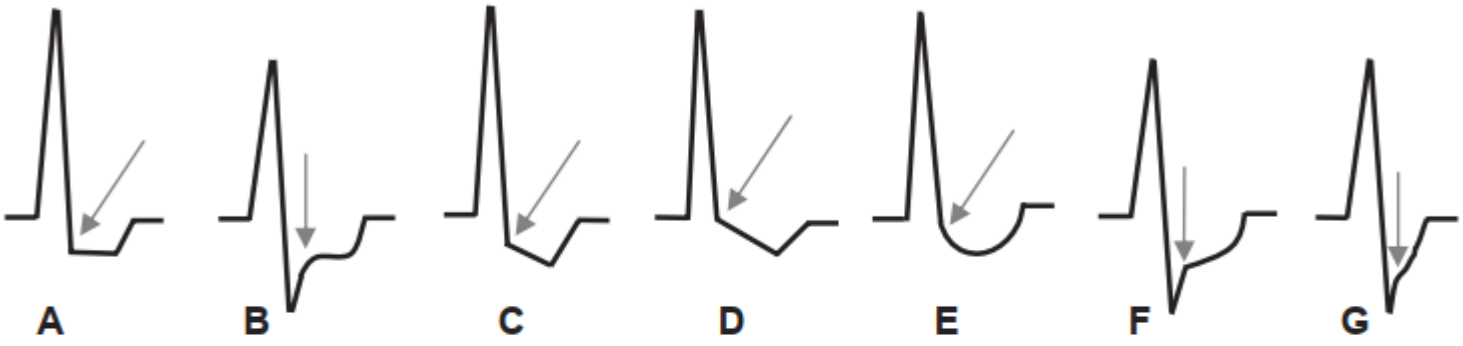
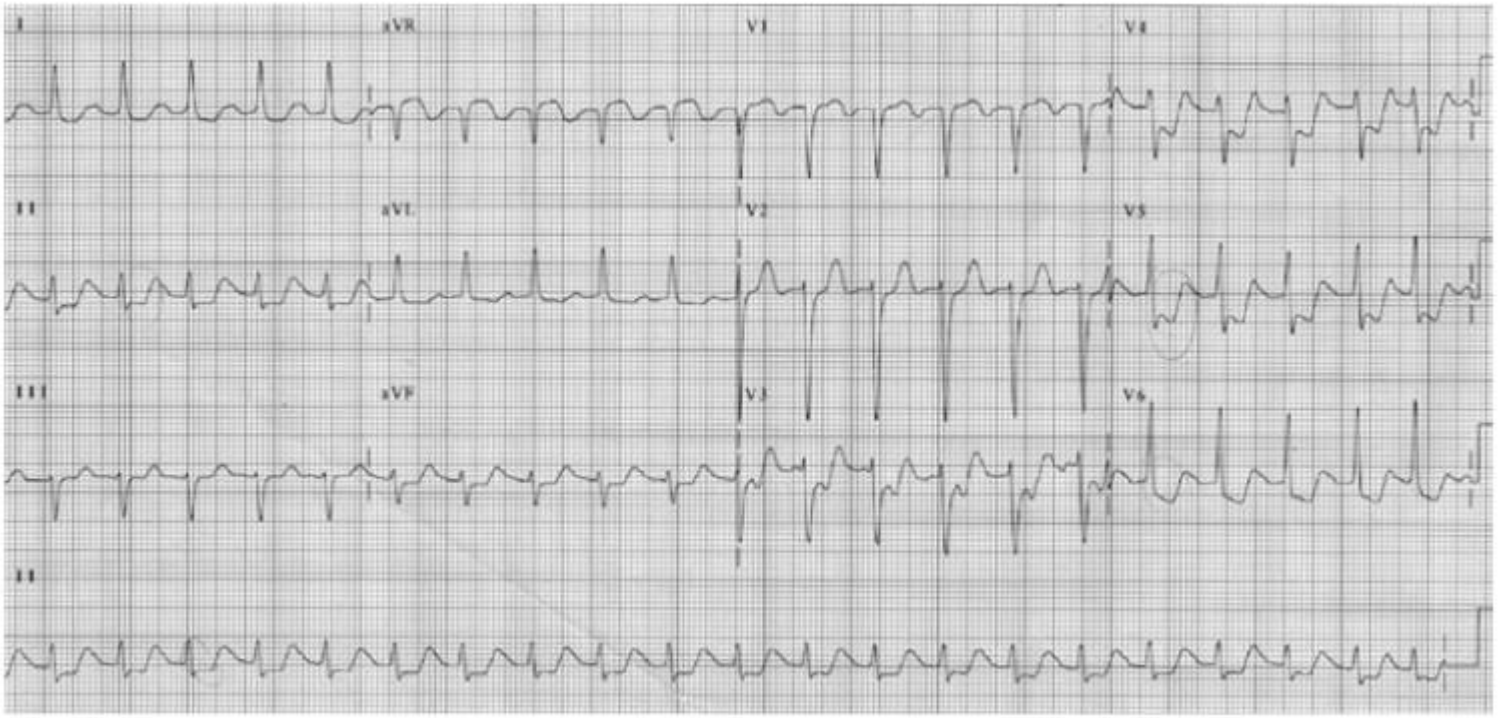


Figure 24.15: ST Segment Depression. **(A, B)** Horizontal ST depression. **(C, D)** Downsloping ST segment depression. **(E)** Scooping ST segment depression frequently from digitalis effect. **(F)** Slow upsloping ST segment depression. **(G)** Fast upsloping ST segment depression frequently a normal finding. **(A, B, C, F)** Typical ischemic ST depression. **(D)** Left ventricular strain frequently associated with left ventricular hypertrophy. Arrows indicate the J point.

ECG in NSTEMI – Abnormal T-wave



Generally ST-segment Depression

ECG in NSTEMI – ST-segment

- ST segment depression:
  - Myocardial injury
  - Digitalis: scooping depression.
  - Secondary ST-segment depression: left ventricular hypertrophy.

ECG in NSTEMI – ST-segment

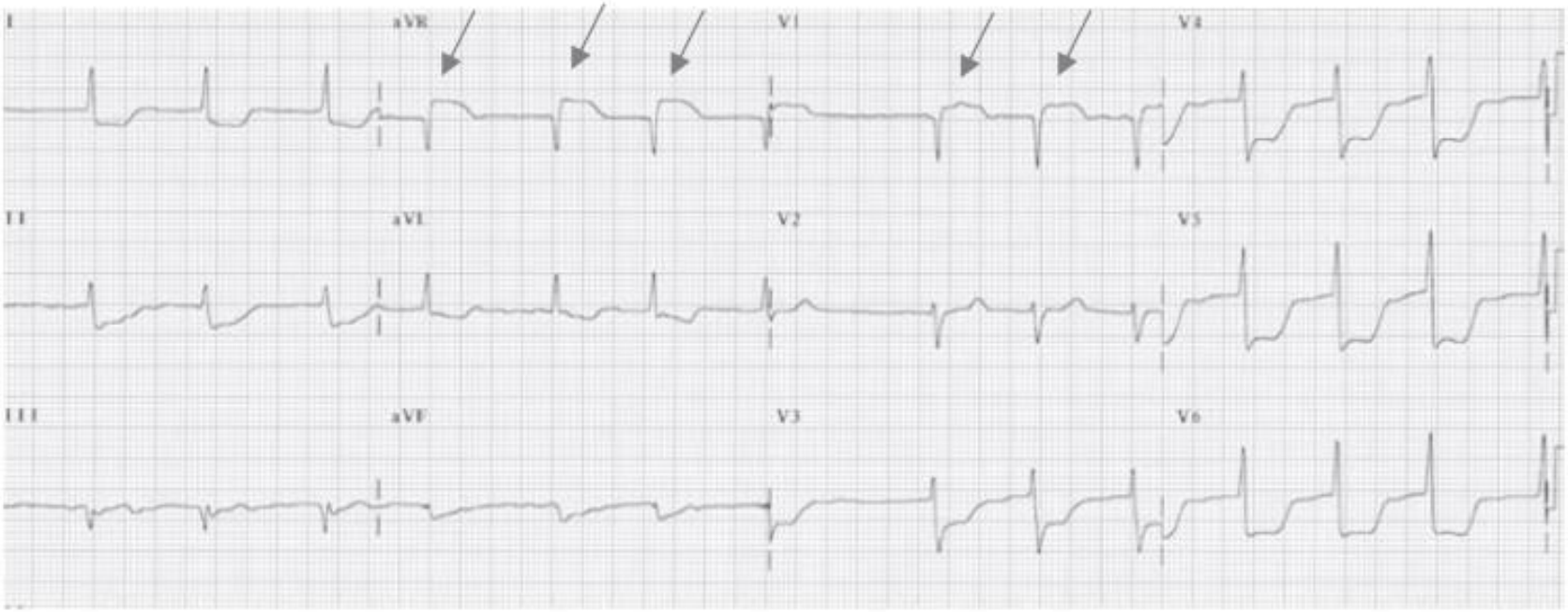
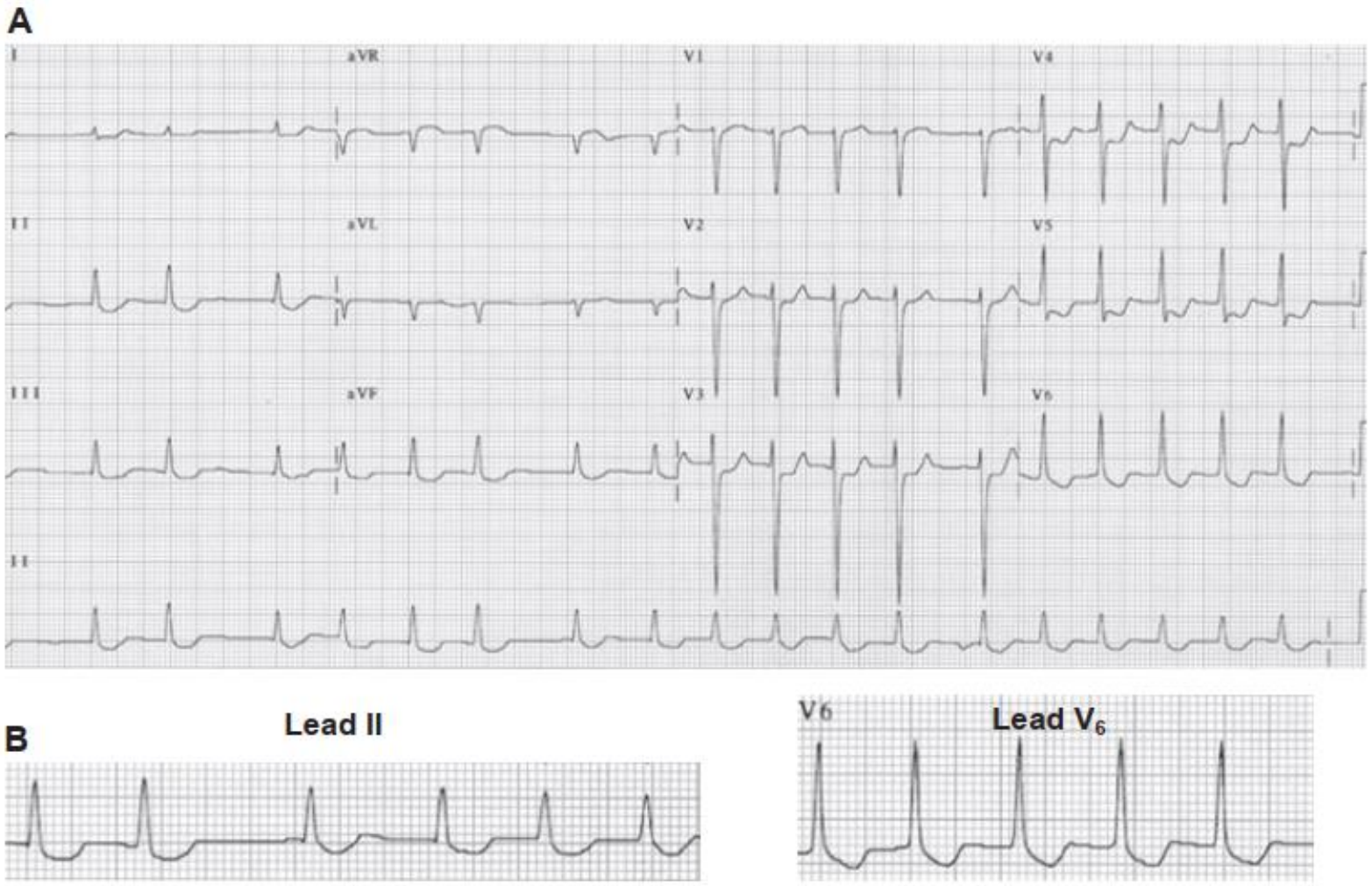


Figure 24.18: ST Depression from Subtotal Occlusion of the Left Main Coronary Artery. Twelve-lead electrocardiogram (ECG) shows atrial fibrillation and marked ST depression in multiple leads including V_3 to V_6 and leads I, II, aVL, and aVF. There is also elevation of the ST segment in leads V_1 and aVR. The ST elevation in aVR is higher than the ST elevation in V_1 (*arrows*). This type of ECG is frequently associated with subtotal occlusion of the left main coronary artery or its equivalent.

ECG in NSTEMI – Abnormal T-wave



Scooping ST-segment Depression in digitalis

ECG in NSTEMI – Abnormal T-wave

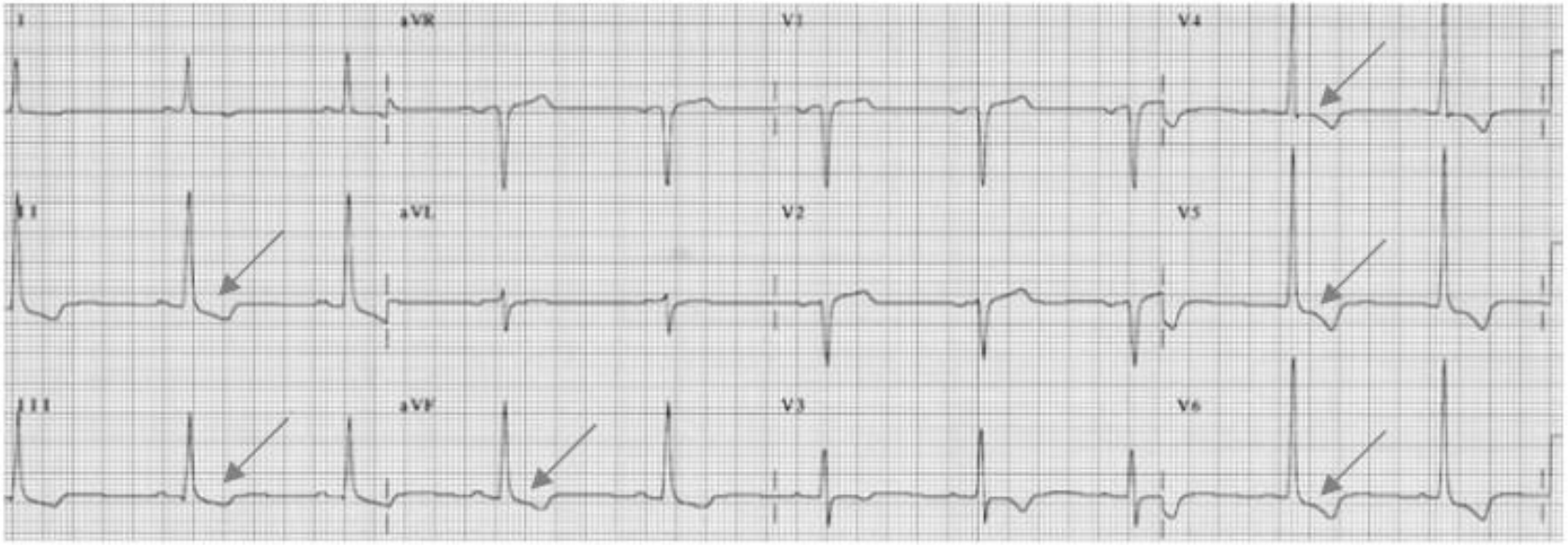


Figure 24.20: Left Ventricular Hypertrophy: Twelve-lead electrocardiogram showing left ventricular hypertrophy (LVH) with downsloping ST depression from left ventricular strain. The J point is not depressed and the ST segments have a downsloping configuration with upward convexity (*arrows*). This type of LVH is usually seen in patients with hypertension and is often described as pressure or systolic overload.

Strain pattern in left ventricular hypertrophy

ECG in NSTEMI – Mechanism ST changes

- There are two different mechanisms:
  - Systolic current of injury
  - Diastolic current of injury

ECG in NSTEMI – Mechanism of ST changes

- Systolic current of Injury:
  - The resting potential is less negative in injured myocardial cells.
  - The height, amplitude and duration of action potential is diminished.
  - The current of injury during electrical systole (phase 1-3) is directed to the injured myocardium.

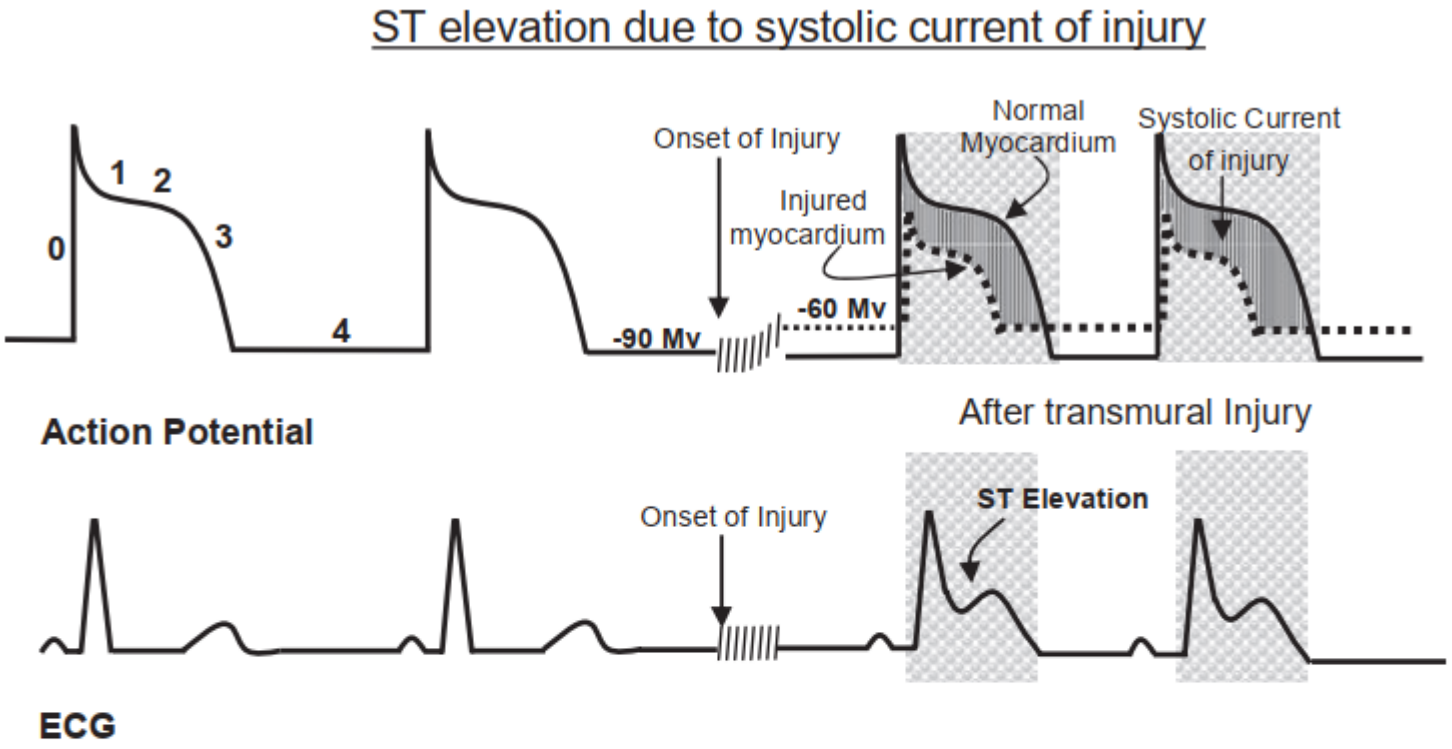


Figure 24.21: Systolic Current of Injury. The upper row represents transmembrane action potentials before and after myocardial injury and the lower row the corresponding electrocardiogram (ECG). A change in resting potential from –90 to approximately –60 mV will occur when cells are injured. A less negative resting potential causes the amplitude and duration of the action potential to diminish when compared to normal cells. This difference in potential creates a current of injury during electrical systole (corresponding to phases 1–3 of the action potential equivalent to the ST segment and T wave in the ECG) between normal and injured myocardium. This current flows from normal myocardium toward the injured myocardium. Thus, if the injury is subepicardial or transmural, the current of injury is directed toward the overlying electrode resulting in ST elevation. (0 to 4 represent the different phases of the action potential.)

ECG in NSTEMI – Mechanism of ST changes

- Diastolic current of Injury:
  - This mechanism is according to phase 4, corresponding to TQ segment.
  - The resting potential is also less negative than normal cells.
  - So, the extracellular charge is more negative (or less positive) than normal cells.
  - The diastolic current is directed away the injured myocardial cells, then in systolic current, ECG measurer will compensate for this phenomena, so leads to ST depression or elevation.

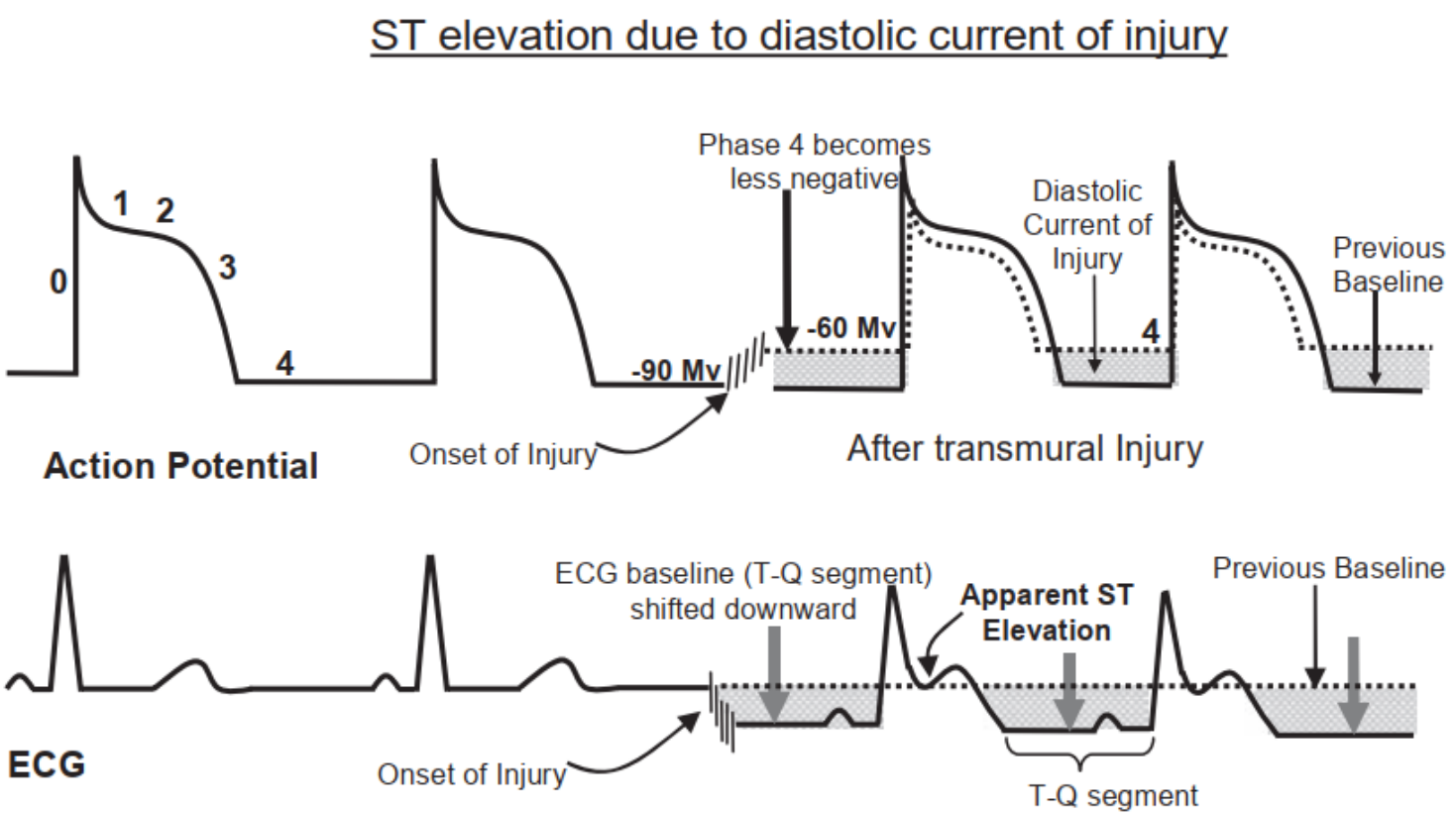
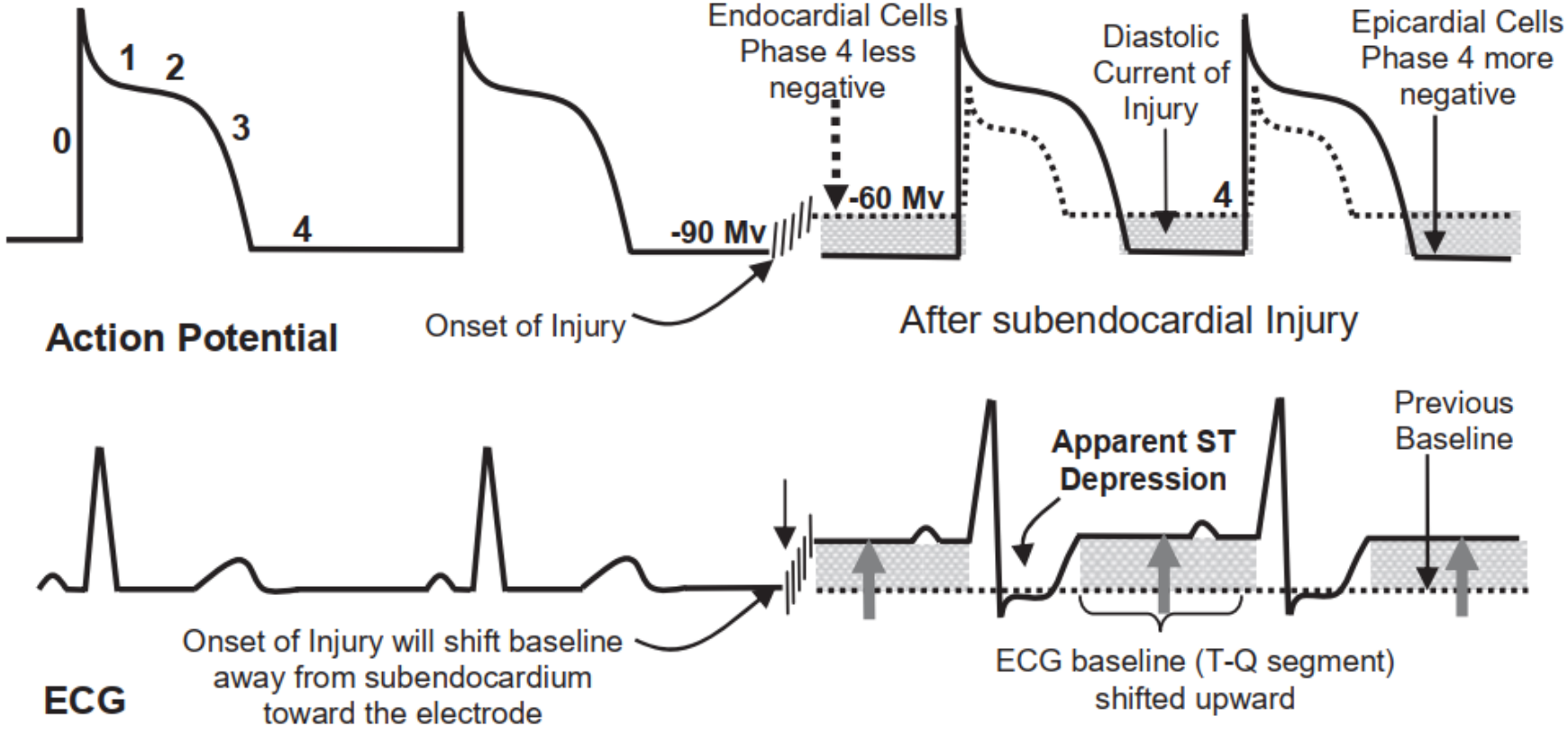


Figure 24.22: Diastolic Current of Injury. The yellow shaded areas in the upper and lower diagrams represent electrical diastole showing a change in resting potential from –90 to –60 mV after myocardial injury. Because the resting potential of injured cells is less negative, the cells are relatively in a state of partial depolarization. Thus, the extracellular membrane of the injured cells is more negative (less positive) compared with that of normal myocardium causing a diastolic current of injury directed away from the injured myocardium. This diastolic current of injury causes the TQ segment to be displaced downward away from the overlying electrode. When all cells are discharged during systole, the potential gradient between injured and normal cells is diminished, shifting the electrocardiogram baseline to its original position, resulting in apparent ST elevation.

ST depression due to diastolic current of injury



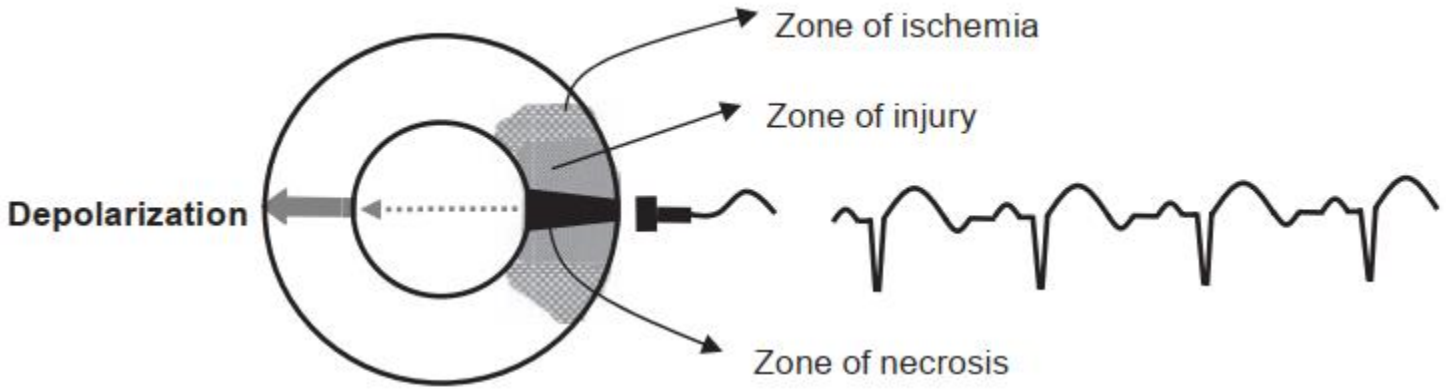


Figure 24.24: Q Waves. Diagrammatic representation of a transmural infarct. The necrotic area (*black*) involves the whole thickness of the myocardium and consists of cells that cannot be depolarized. Thus, an electrode overlying the necrotic area will normally record the electrical activity on the opposite side of the myocardium (*red arrow*), resulting in q waves.