ABC of clinical electrocardiography
Acute myocardial infarction–Part I

Francis Morris, William J Brady

In the clinical assessment of chest pain, electrocardiography is an essential adjunct to the clinical history and physical examination. A rapid and accurate diagnosis in patients with acute myocardial infarction is vital, as expeditious reperfusion therapy can improve prognosis. The most frequently used electrocardiographic criterion for identifying acute myocardial infarction is ST segment elevation in two or more anatomically contiguous leads. The ST segment elevation associated with an evolving myocardial infarction is often readily identifiable, but a knowledge of the common "pseudo" infarct patterns is essential to avoid the unnecessary use of thrombolytic treatment.

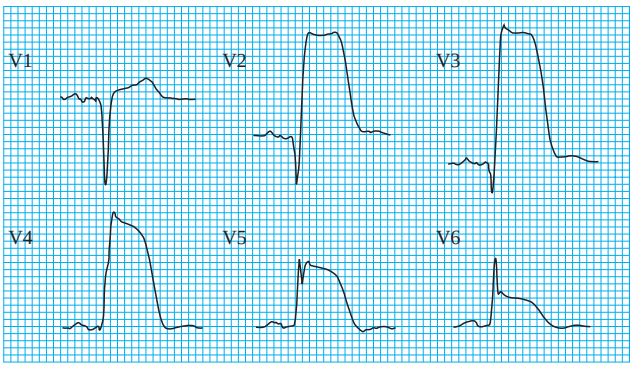
In the early stages of acute myocardial infarction the electrocardiogram may be normal or near normal; less than half of patients with acute myocardial infarction have clear diagnostic changes on their first trace. About 10% of patients with a proved acute myocardial infarction (on the basis of clinical history and enzymatic markers) fail to develop ST segment elevation or depression. In most cases, however, serial electrocardiograms show evolving changes that tend to follow well recognised patterns.

Hyperacute T waves

The earliest signs of acute myocardial infarction are subtle and include increased T wave amplitude over the affected area. T waves become more prominent, symmetrical, and pointed ("hyperacute"). Hyperacute T waves are most evident in the anterior chest leads and are more readily visible when an old electrocardiogram is available for comparison. These changes in T waves are usually present for only five to 30 minutes after the onset of the infarction and are followed by ST segment changes.

ST segment changes

In practice, ST segment elevation is often the earliest recognised sign of acute myocardial infarction and is usually evident within hours of the onset of symptoms. Initially the ST segment may straighten, with loss of the ST-T wave angle . Then the T wave becomes broad and the ST segment elevates, losing its normal concavity. As further elevation occurs, the ST segment tends to become convex upwards. The degree of ST segment elevation varies between subtle changes of <1 mm to gross elevation of >10 mm.

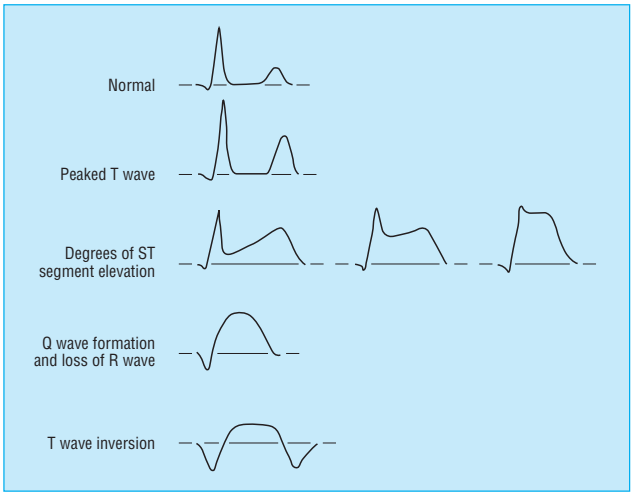


Anterior myocardial infarction with gross ST segment elevation (showing "tombstone" R waves)

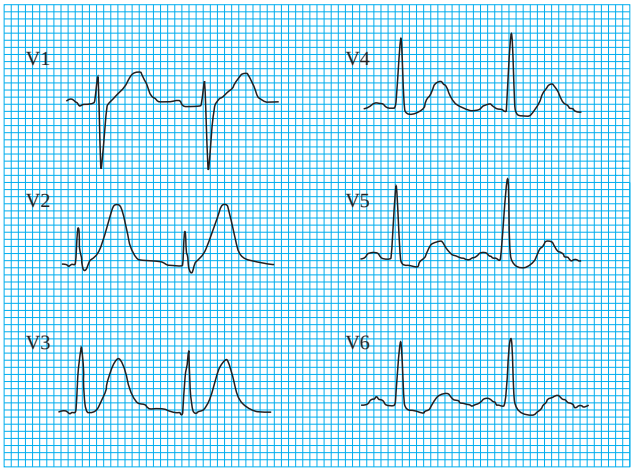
Indications for thrombolytic treatment

- ST elevation >1 mm in two contiguous limb leads or >2 mm in two contiguous chest leads
- Posterior myocardial infarction
- Left bundle branch block

ST segment depression or enzymatic change are not indications for thrombolytic treatment



Sequence of changes seen during evolution of myocardial infarction



Hyperacute T waves

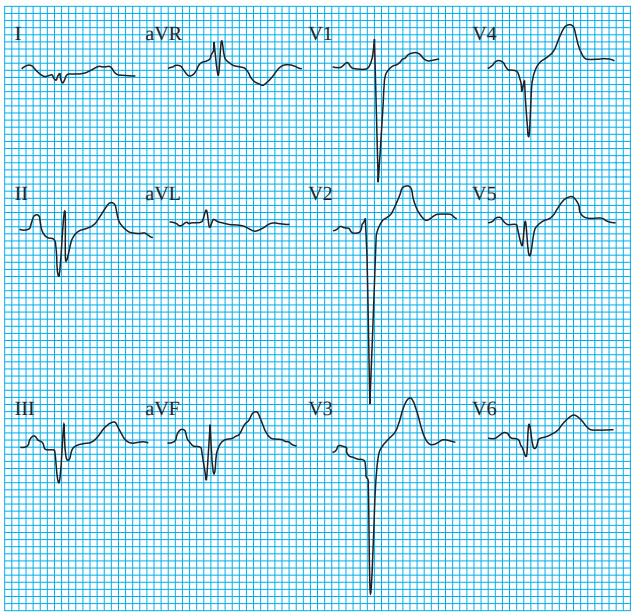
Sometimes the QRS complex, the ST segment, and the T wave fuse to form a single monophasic deflection, called a giant R wave or "tombstone"

Pathological Q waves

As the acute myocardial infarction evolves, changes to the QRS complex include loss of R wave height and the development of pathological Q waves.

Both of these changes develop as a result of the loss of viable myocardium beneath the recording electrode, and the Q waves are the only firm electrocardiographic evidence of myocardial necrosis. Q waves may develop within one to two hours of the onset of symptoms of acute myocardial infarction, though often they take 12 hours and occasionally up to 24 hours to appear. The presence of pathological Q waves, however, does not necessarily indicate a completed infarct. If ST segment elevation and Q waves are evident on the electrocardiogram and the chest pain is of recent onset, the patient may still benefit from thrombolysis or direct intervention.

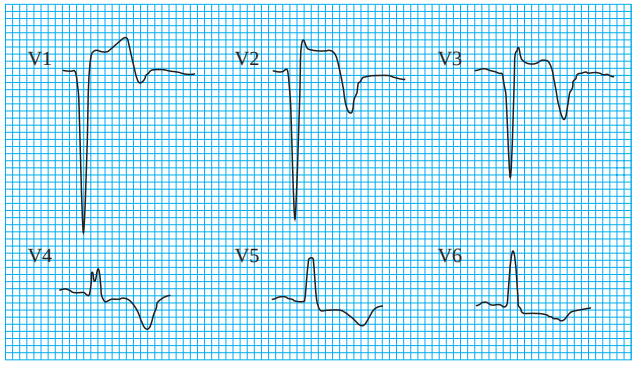
When there is extensive myocardial infarction, Q waves act as a permanent marker of necrosis. With more localised infarction the scar tissue may contract during the healing process, reducing the size of the electrically inert area and causing the disappearance of the Q waves.



Pathological Q waves in inferior and anterior leads

Resolution of changes in ST segment and T waves

As the infarct evolves, the ST segment elevation diminishes and the T waves begin to invert. The ST segment elevation associated with an inferior myocardial infarction may take up to two weeks to resolve. ST segment elevation associated with anterior myocardial infarction may persist for even longer, and if a left ventricular aneurysm develops it may persist indefinitely. T wave inversion may also persist for many months and occasionally remains as a permanent sign of infarction.

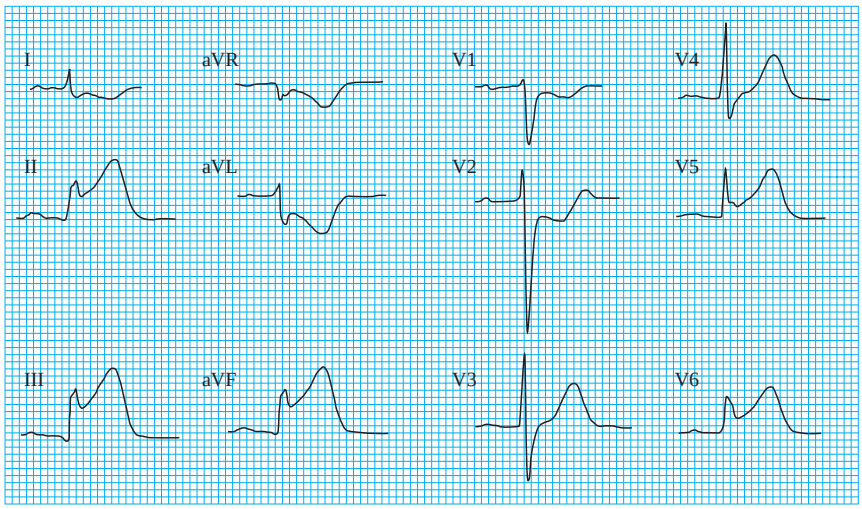


Long standing ST segment elevation and T wave inversion associated with a previous anterior myocardial infarction (echocardiography showed a left ventricular aneurysm)

Reciprocal ST segment depression

ST segment depression in leads remote from the site of an acute infarct is known as reciprocal change and is a highly sensitive indicator of acute myocardial infarction. Reciprocal changes are seen in up to 70% of inferior and 30% of anterior infarctions.

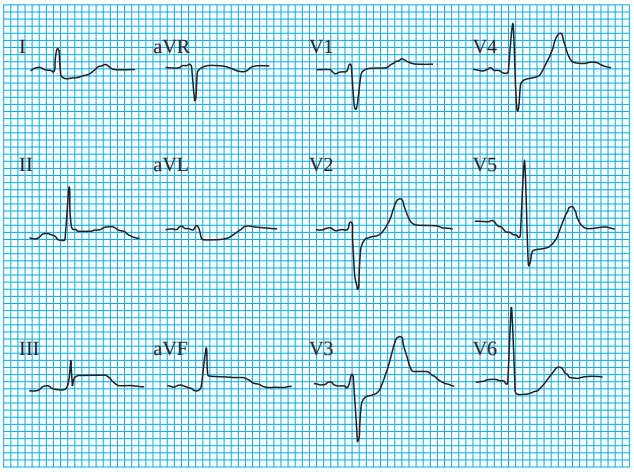
Typically, the depressed ST segments tend to be horizontal or downsloping. The presence of reciprocal change is particularly useful when there is doubt about the clinical significance of ST segment elevation.



An inferolateral myocardial infarction with reciprocal changes in leads I, aVL, V1, and V2

Reciprocal change strongly indicates acute infarction, with a sensitivity and positive predictive value of over 90%, though its absence does not rule out the diagnosis.

The pathogenesis of reciprocal change is uncertain. Reciprocal changes are most frequently seen when the infarct is large, and they may reflect an extension of the infarct or occur as a result of coexisting remote ischaemia. Alternatively, it may be a benign electrical phenomenon. The positive potentials that are recorded by electrodes facing the area of acute injury are projected as negative deflections in leads opposite the injured area, thus producing a "mirror image" change. Extensive reciprocal ST segment depression in remote regions often indicates widespread arterial disease and consequently carries a worse prognosis.



Reciprocal changes: presence of widespread ST segment depression in the anterolateral leads strongly suggests that the subtle inferior ST segment elevation is due to acute infarction

Localisation of site of infarction

The distribution of changes recorded in acute myocardial infarction allows the area of infarction to be localised, thus indicating the site of arterial disease. Proximal arterial occlusions tend to produce the most widespread electrocardiographic abnormalities. The anterior and inferior aspects of the heart are the areas most commonly subject to infarction. Anteroseptal infarcts are highly specific indicators of disease of the left anterior descending artery. Isolated inferior infarcts—changes in leads II, III, and aVF—are usually associated with disease in the right coronary or distal circumflex artery. Disease in the proximal circumflex artery is often associated with a lateral infarct pattern—that is, in leads I, aVL, V5, and V6.

Anatomical relationship of leads

Inferior wall—Leads II, III, and aVF
Anterior wall—Leads V1 to V4
Lateral wall—Leads I, aVL, V5, and V6

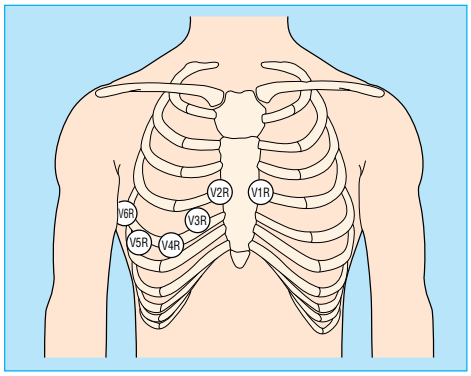
Non-standard leads

Right ventricle—Right sided chest leads V1R to V6R
Posterior wall—Leads V7 to V9

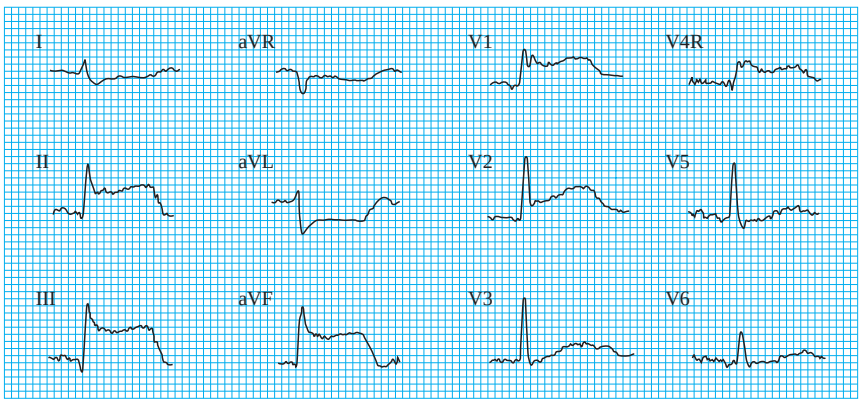
Right ventricular infarction

Right ventricular infarction is often overlooked, as standard 12 lead electrocardiography is not a particularly sensitive indicator of right ventricular damage. Right ventricular infarction is associated with 40% of inferior infarctions. It may also complicate some anterior infarctions but rarely occurs as an isolated phenomenon. On the standard 12 lead electrocardiogram right ventricular infarction is indicated by signs of inferior infarction, associated with ST segment elevation in lead V1. It is unusual for ST segment elevation in lead V1 to occur as an isolated phenomenon.

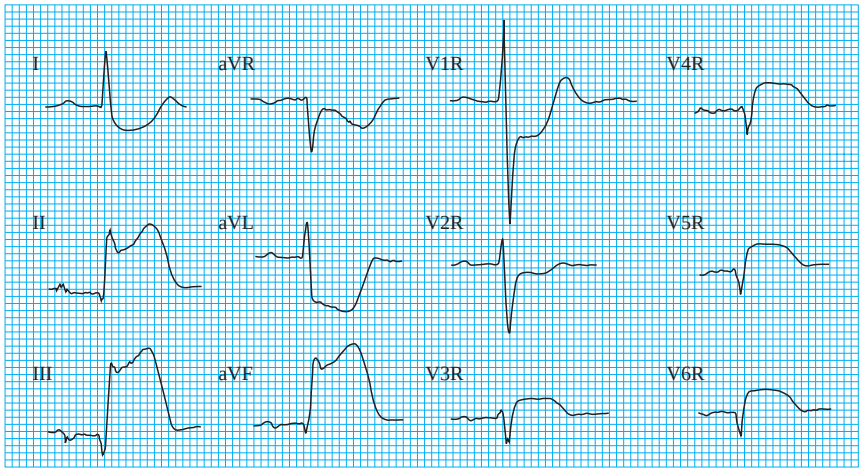
Right sided chest leads are much more sensitive to the presence of right ventricular infarction. The most useful lead is lead V4R (an electrode is placed over the right fifth intercostal space in the mid-clavicular line). Lead V4R should be recorded as soon as possible in all patients with inferior infarction, as ST segment elevation in right ventricular infarction may be short lived.



Placement of right sided chest leads



Acute inferior myocardial infarction with associated right ventricular infarction



Acute inferior myocardial infarction with right ventricular involvement

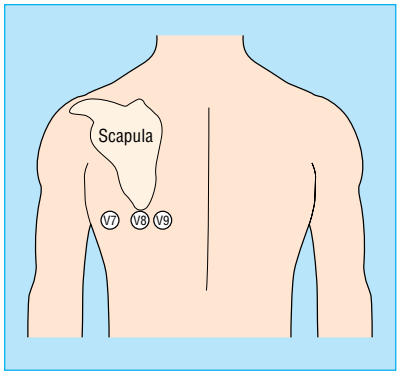
The diagnosis of right ventricular infarction is important as it may be associated with hypotension. Treatment with nitrates or diuretics may compound the hypotension, though the patient may respond to a fluid challenge

Right ventricular infarction usually results from occlusion of the right coronary artery proximal to the right ventricular marginal branches, hence its association with inferior infarction. Less commonly, right ventricular infarction is associated with occlusion of the circumflex artery, and if this vessel is dominant there may be an associated inferolateral wall infarction.

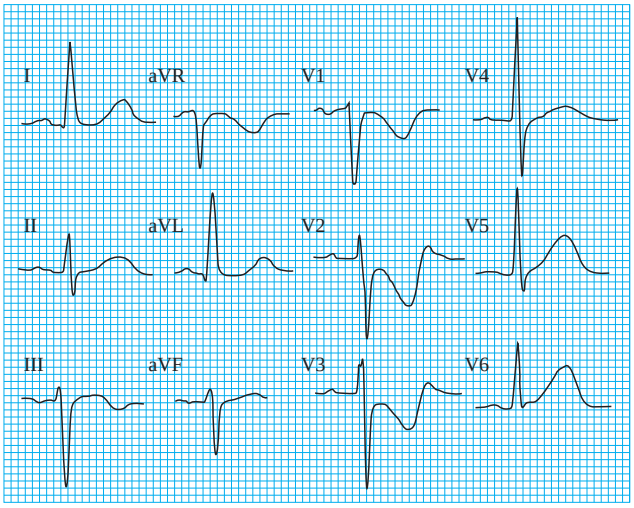
Posterior myocardial infarction

Posterior myocardial infarction refers to infarction of the posterobasal wall of the left ventricle. The diagnosis is often missed as the standard 12 lead electrocardiography does not include posterior leads. Early detection is important as expeditious thrombolytic treatment may improve the outcome for patients with posterior infarction.

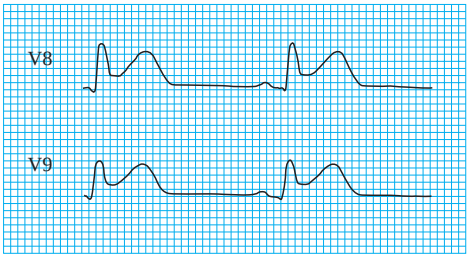
The changes of posterior myocardial infarction are seen indirectly in the anterior precordial leads. Leads V1 to V3 face the endocardial surface of the posterior wall of the left ventricle. As these leads record from the opposite side of the heart instead of directly over the infarct, the changes of posterior infarction are reversed in these leads. The R waves increase in size, becoming broader and dominant, and are associated with ST depression and upright T waves. This contrasts with the Q waves, ST segment elevation, and T wave inversion seen in acute anterior myocardial infarction. Ischaemia of the anterior wall of the left ventricle also produces ST segment depression in leads V1 to V3, and this must be differentiated from posterior myocardial infarction. The use of posterior leads V7 to V9 will show ST segment elevation in patients with posterior infarction. These additional leads therefore provide valuable information, and they help in identfying the patients who may benefit from urgent reperfusion therapy.



Position of V7, V8, and V9 on posterior chest wall



Isolated posterior infarction with no associated inferior changes (note ST segment depression in leads V1 to V3)



ST segment elevation in posterior chest leads V8 and V9

The ABC of clinical electrocardiography is edited by Francis Morris, consultant in emergency medicine at the Northern General Hospital, Sheffield; June Edhouse, consultant in emergency medicine, Stepping Hill Hospital, Stockport; William J Brady, associate professor, programme director, and vice chair, department of emergency medicine, University of Virginia, Charlottesville, VA, USA; and John Camm, professor of clinical cardiology, St George's Hospital Medical School, London. The series will be published as a book in the summer.

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