ECG INTERPRETATION FOR THE EMERGENCY DEPARTMENT

Electrocardiographic abnormalities encountered in acute myocardial infarction

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The evaluation of patients with chest pain is a challenging area of accident and emergency (A&E) work. In the clinical assessment of such patients, interpretation of the electrocardiogram (ECG) is an essential adjunct to the history and examination. Approximately 20% of patients presenting with chest pain will have acute myocardial infarction (AMI), 35% angina/unstable angina, and 45% non-cardiac chest pain.[1] [2] Reaching a rapid and accurate clinical diagnosis is of great importance, particularly as urgent reperfusion treatments such as thrombolytic agents and coronary angioplasty have been shown to improve prognosis in patients with AMI—if applied appropriately and early.

The electrocardiographic criteria identifying the group of patients likely to benefit from urgent reperfusion treatments are: (1) ST segment elevation in at least two anatomically contiguous leads and (2) *new* left bundle branch block (LBBB); further, ST segment depression in the right precordial leads (V1–V3), indicative of posterior AMI with posterior lead ST segment elevation, may represent yet another electrocardiographic indication for urgent coronary reperfusion. The ST segment elevation associated with an evolving myocardial infarction is usually readily identifiable. A&E physicians responsible for the administra-tion of thrombolysis, however, need to be aware of the common pseudoinfarct ST elevation patterns that are associated with non-AMI ECG syndromes in the chest pain patient as LBBB and left ventricular hypertrophy. This article will review the ECG changes associated with AMI. Others in this series review the ECG changes associated with posterior wall and right ventricular wall infarction, the diagnosis of AMI in the presence of LBBB, non-infarction ST segment elevation syndromes, and the patient with chest pain and a non-diagnostic ECG.

Case reports

CASE 1

A 56 year old man presented to the A&E department giving a three hour history of central chest pain. The pain had started at rest while at work and was associated with short-ness of breath and sweating. Clinical examina-tion revealed that he was pale, sweaty, and clammy. His ECG revealed prominent T waves in the anterior leads, likely consistent with early anterior myocardial infarction (fig 1). He was given an aspirin and opiate analgesia but his pain continued. Thirty minutes after arrival, a second ECG was recorded (fig 2) demonstrat-ing extensive ST segment elevation in the antero-lateral distribution consistent with AMI; he

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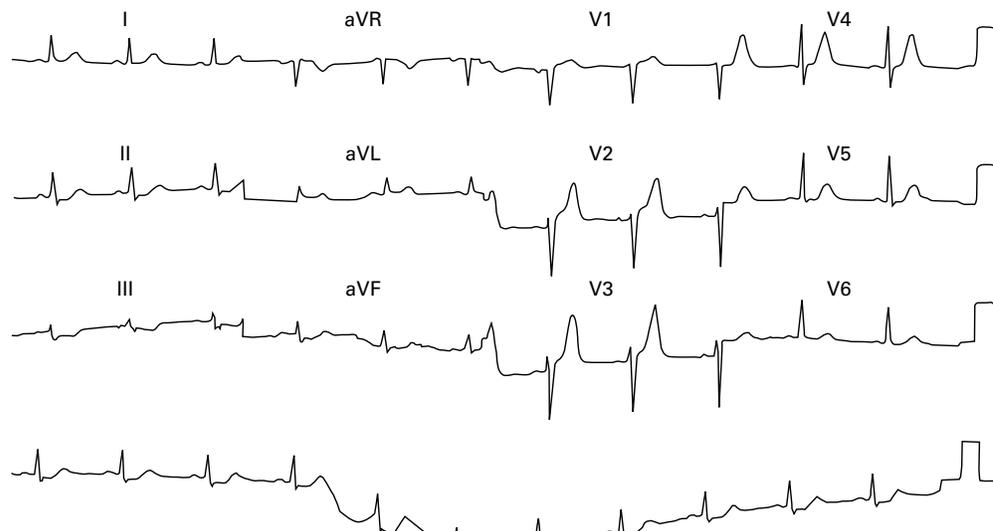
Figure 1   AMI with hyperacute T wave (case 1). Normal sinus rhythm with prominent T waves is seen in leads V2–V4. There is also a suggestion of ST segment elevation in lead aVL and ST segment depression in the inferior leads. This ECG tracing is compatible with the early stages of AMI, primarily manifested by the anterior hyperacute T waves.
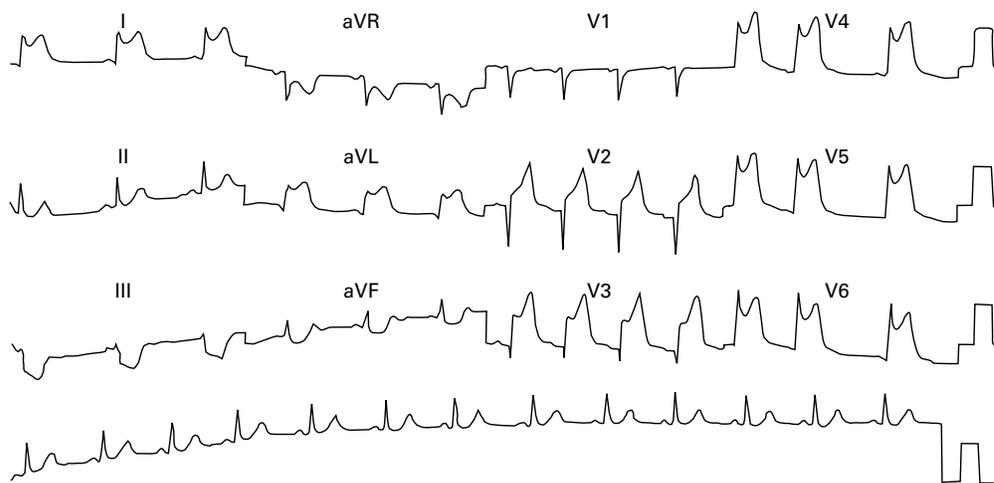
Figure 2   AMI with serial change (case 1). This ECG was taken 30 minutes after the ECG depicted in fig 1. Normal sinus rhythm with widespread ST segment elevation is seen in the anterolateral leads while reciprocal changes is noted in leads III and aVF—confirming the diagnosis of AMI.
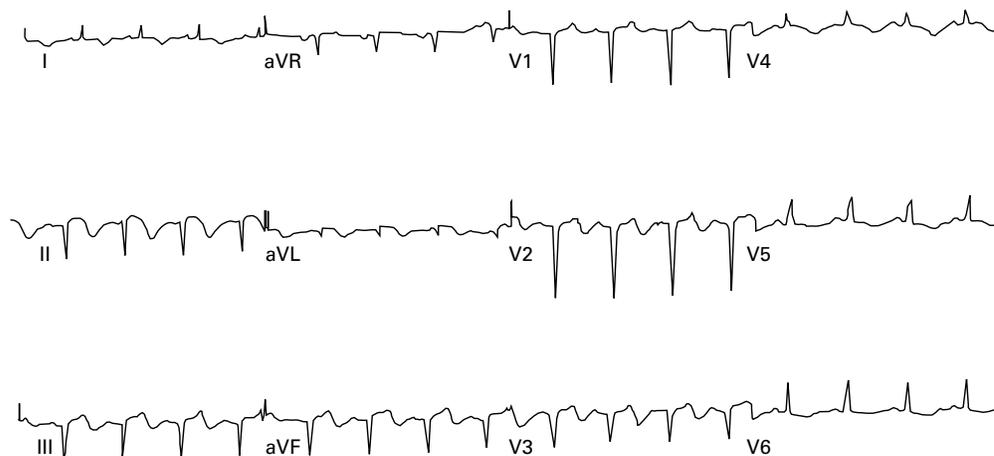

Figure 3   ST segment elevation in a chest pain patient with left ventricular aneurysm (case 2)—ST segment elevation is seen in the inferior leads. Serial ECGs did not reveal change in the ST segment abnormality. Further, a rapid review of past medical records revealed similar ECGs as well as a history of left ventricular aneurysm of the inferior wall.
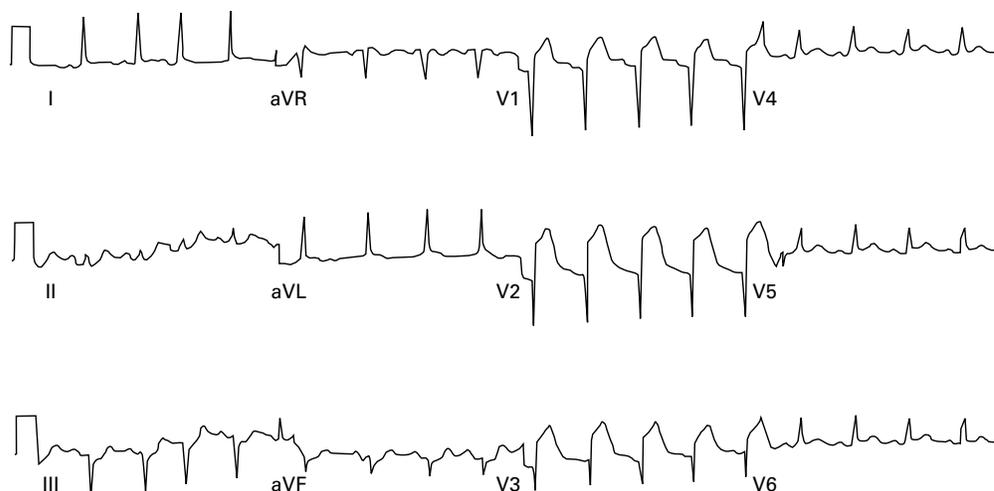

Figure 4   AMI with simultaneous ST segment elevation and pathological Q waves (case 3). The clinical history suggested that the AMI had initiated within the past two hours despite the pathological Q waves.
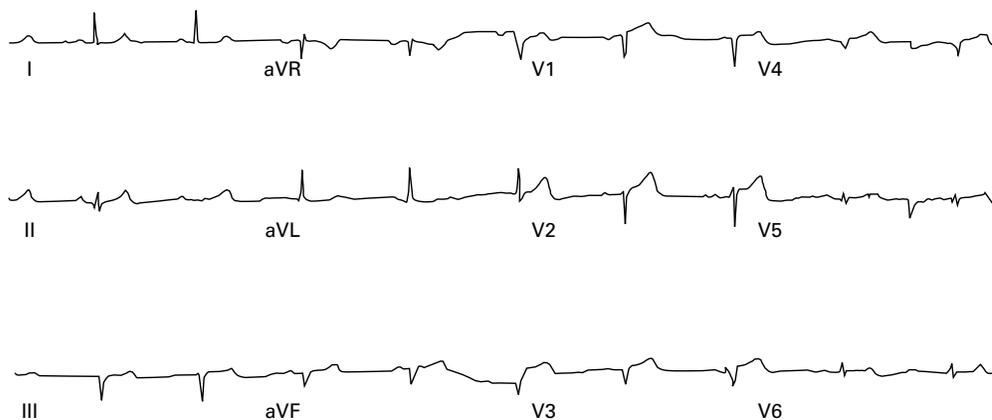
Figure 5   Atypical ST segment elevation in a patient with missed, early AMI (case 4)—ST segment elevation is seen in leads V1–V4 as well as in leads II, III, and aVF. The initial, up-sloping portion of the ST segment is concave, suggestive but not diagnostic of a non-infarction cause of the electrocardiographic abnormality. This patient presented with chest pain and the depicted ECG, which was misread initially, lead to a diagnostic error and perhaps an unnecessary death.
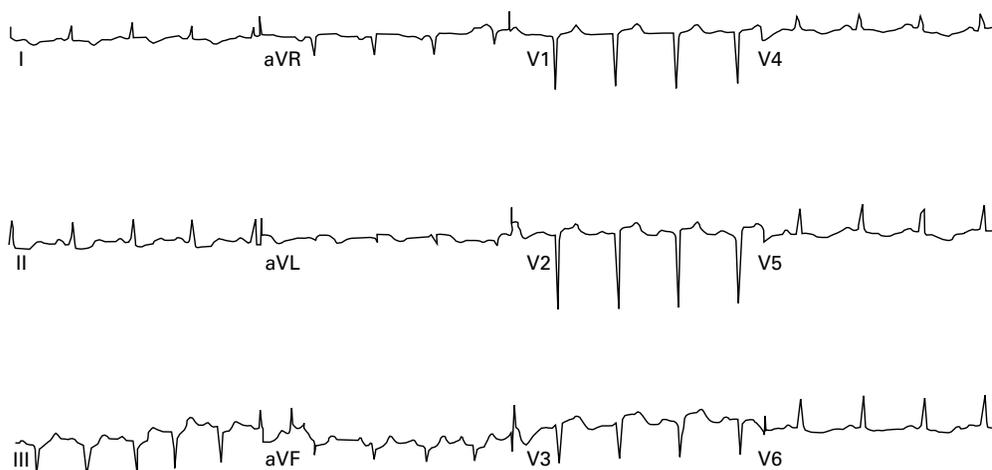
Figure 6   Early AMI with reciprocal ST segment depression (case 5)—the ST segment changes seen in leads V1–V3 are not highly suggestive of AMI: minimal ST segment elevation and concave morphology of the elevation. The presence of ST segment depression in the inferior leads (particularly leads III and aVF), however, strongly supports an electrocardiographic diagnosis of AMI.

was given thrombolysis. The patient's pain initially lessened with some reduction in the ST segment elevation; however, the pain worsened and was followed soon after with the development of ventricular fibrillation, which did not respond to multiple defibrillation attempts. He was not resuscitated.

CASE 2
A 66 year old man presented to the A&E department with central chest pain of two hours' duration. He had suffered from exertional chest pain for the last six months after an inferior myocardial infarction. His pain usually responded to sublingual nitrates, though on this occasion it had not. Clinical examination was unremarkable. The ECG is shown in fig 3, revealing prominent ST segment elevation inferiorly worrisome for AMI. A rapid review of his past ECG, however, revealed similar ST segment changes in a previous study as well as documented inferior wall aneurysm. His pain settled with opiate analgesia and buccal nitrates. The patient was admitted to hospital for further care; subsequent evaluation did not reveal evidence of AMI.

CASE 3
A 46 year old man presented to the emergency department with chest pain of 20 minutes duration. The pain was accompanied by nausea and diaphoresis. The patient appeared pale and diaphoretic. The ECG (fig 4) revealed prominent ST segment elevation in the anterior leads with pathological Q waves in the
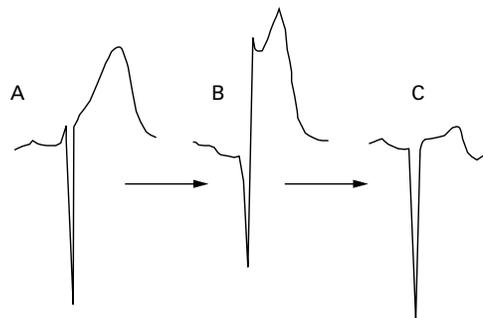
Figure 7   The electrocardiographic evolution of AMI. (A) Hyperacute T wave 15 minutes after the onset of chest discomfort. (B) Marked ST segment elevation with prominent Q wave three hours into AMI. (C) Persistent ST segment elevation with Q wave six weeks after AMI, indicative of an aneurysm.
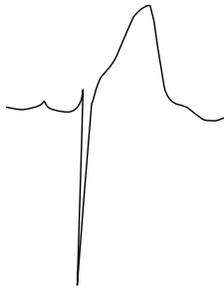
Figure 8 Hyperacute T waves of early AMI are often asymmetric with a broad base.
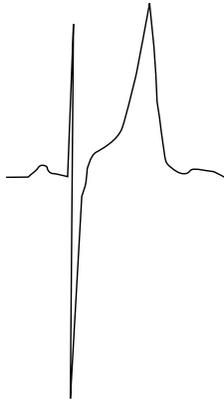

Figure 9 Hyperkalaemic T waves tend to be tall, narrow, and peaked with prominent or sharp apex. Also, these T waves tend to be symmetric in morphology; if "split down the middle" the resulting portions would be mirror images. As the serum potassium concentration increases, the T waves tend to become taller, peaked, and narrowed in a symmetric fashion in the anterior distribution. This T wave morphology may be confused with the hyperacute T wave of early transmural myocardial infarction.

same leads; the diagnosis was AMI. After careful questioning, it was felt that the AMI had only recently initiated despite the appearance of the fully developed Q waves. The patient was urgently transferred to the catheterisation laboratory where a proximal left anterior descending artery occlusion with thrombus was noted. The lesion was successfully opened with stent placement. Cardiac enzymes were raised, confirming the diagnosis of AMI. The patient had an uneventful hospital course and was discharged on hospital day number five.

CASE 4
A 58 year old woman presented to the emergency department with chest discomfort. The patient described this as an ache with associated nausea. The medical history was unremarkable. The clinical examination was unrevealing. The ECG demonstrated ST segment elevation in the anterior leads with a concave morphology, felt at that time to be consistent with benign early repolarisation (fig 5). The patient was given antacids for a presumed gastrointestinal dyspeptic syndrome. She was sent home but returned 20 hours later with marked dyspnoea. Repeat evaluation revealed pulmonary oedema. The ECG indicated obvious completed anterior wall AMI with pathological Q waves. Sudden pulseless ventricular tachycardia developed. Despite aggressive care, the patient died soon after arrival at the emergency department.

CASE 5
A 39 year old man with hypertension and diabetes mellitus was transported to the emergency department by ambulance with chest pain, shortness of breath, and emesis. On arrival, the patient was diaphoretic and appeared ill. The ECG (fig 6) demonstrated ST segment elevation in leads V1–V3 of questionable aetiology as well as ST segment depression in leads III and aVF. The ST segment depression was felt to represent reciprocal ST segment depression; its presence suggested that the ST segment elevation was due to AMI. A repeat ECG demonstrated more pronounced ST segment elevation, confirming the diagnosis of AMI. The patient received thrombolysis and made an uneventful recovery from the myocardial infarction.

Discussion
Patients with AMI presenting shortly after the onset of their symptoms may have a normal or near normal ECG. It is estimated that approximately 10% of patients with proved AMI never

develop significant electrocardiographic changes, including major ST segment or T wave abnormalities. This situation tends to occur in the context of subendocardial myocardial infarction as well as in patients with infarction of the posterior or lateral walls of the left ventricle. It must also be appreciated that only 50% of patients who sustain a full thickness myocardial infarction will have obvious ST segment elevation on the initial ECG on presentation to the A&E department; the initial ECG in these remaining non-diagnostic patients will range from entirely normal to non-specifically abnormal.[3] "Non-specifically abnormal" describes the ECG with ST segment changes less than 1 mm in magnitude and T wave abnormalities other than inversion or peaking.

The ECG undergoes a well established temporal evolution after the onset of and persistence of coronary artery occlusion (fig 7).[4] The earliest electrocardiographic finding resulting from AMI is the hyperacute T wave which may appear minutes after the interruption of blood flow; the R wave also increases in amplitude at this stage. The hyperacute T wave is noted as early as 30 minutes after the onset of coronary occlusion and transmural infarction. It tends to be a short lived structure which evolves rapidly on to ST segment elevation. The hyperacute T waves of early AMI are often asymmetric with a broad base (fig 8). Such a finding on the ECG is short lived in the AMI patient; progressive ST segment elevation is usually encountered.

The electrocardiographic differential diagnosis of the hyperacute T wave includes both transmural AMI and hyperkalaemia as well as early repolarisation, left ventricular hypertrophy, and acute myopericarditis. The principle entity to exclude is hyperkalaemia; this T wave morphology is also described as "hyperacute" and may be confused with the hyperacute T wave of early transmural myocardial infarction. Hyperkalaemic hyperacute T waves tend to be tall, narrow, and peaked with a prominent or sharp apex. Also, these T waves tend to be symmetric in morphology; if "split down the middle", the resulting portions would be mirror images. As the serum potassium concentration increases, the T waves tend to become taller, peaked, and narrowed in a symmetric fashion in the anterior distribution (fig 9).

Widening of the T waves then occurs, in association with ST segment elevation. The initial change seen in the ST segment is a relative flattening, associated with loss of the ST-T wave angle followed by the development of ST segment elevation. The normal concavity of the ST segment is lost and, as the ST segment elevates further, it tends to become convex (fig 10). The range of ST segment elevation varies from very subtle changes of less than 1 mm to gross elevation exceeding 10 mm (fig 11). With massive ST elevation ("tombstones"), the QRS complex and T wave are often indistinguishable, forming the giant R wave configuration, a single monophasic deflection (fig 2). This type of ST segment elevation is usually evident within hours of the onset of coronary occlusion
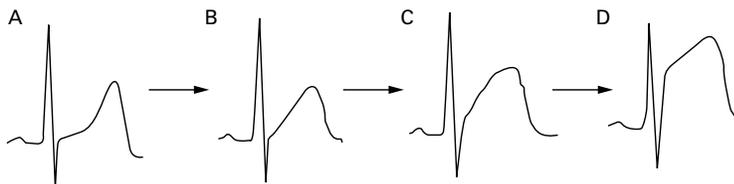

Figure 10 Electrocardiographic complexes in lead II of an early inferior wall AMI. (A) show a normal complex while (B) through (D) reveal changes consistent with AMI. (B) reveals early changes with flattening of the originally concave ST segment which becomes convex (C) as the infarction progresses. Pathological ST segment elevation is then seen (D).
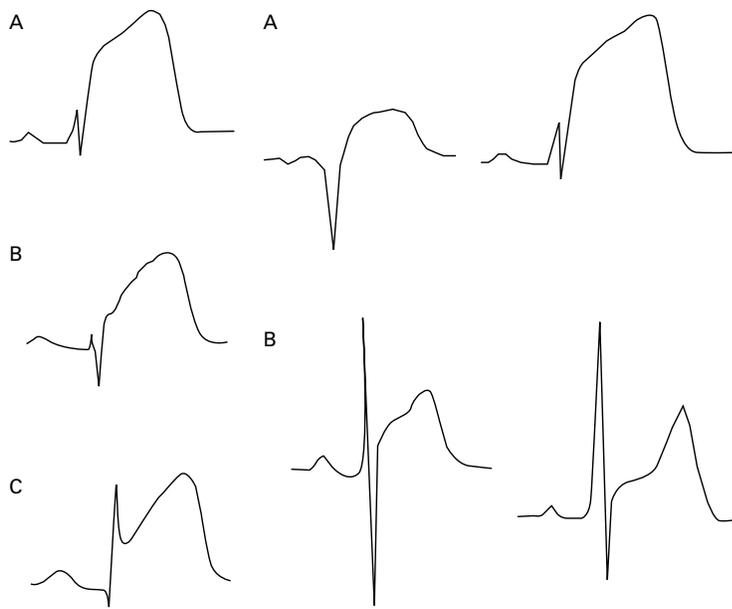
Figure 11   Various forms of ST segment elevation seen in the AMI patient. (A) and (C) reveal obliquely flat segments while (B) demonstrates a convex form of ST segment elevation.
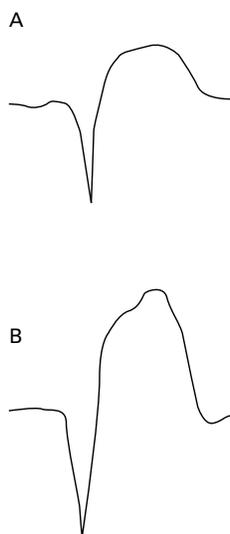

Figure 12   An analysis of the ST segment waveform (from the beginning at the J point to the end at the apex of the T wave) may be particularly helpful in distinguishing among the various causes of ST segment elevation and identifying the AMI case. (A) The initial up-sloping portion of the ST segment usually is either flat (horizontally or obliquely) or convex in the AMI patient. (B) The initial up-sloping portion of the ST segment in the non-AMI patient is frequently convex, as seen in these two non-infarction examples (pericarditis on the left and benign early repolarisation on the right). (C) The initial up-sloping portion of the ST segment usually is either flat or convex in the AMI patient. This morphological observation, however, should only be used as a guideline—it is not infallible. Patients with ST segment elevation due to AMI may demonstrate concavity of the waveform, as seen in examples anterior wall AMI (left upper) and inferior wall AMI (right lower).

Figure 13   A Q wave is seen, indicating past myocardial infarction.

Figure 14   Prominent Q wave with marked ST segment elevation in two patients with AMI. The chest discomfort started approximately two to three hours before presentation in each case. The simultaneous presence of ST segment elevation and pathological Q wave in a patient with a short history of chest discomfort does not rule out the need for nor benefit of acute revascularisation treatments, such as thrombolysis.

distinguishing ST segment elevation associated with spasm from that which occurs with AMI is rarely possible in the acute setting.[4 5–6]

A knowledge of the morphology of the ST segment elevation is important; such knowledge will assist in the identification of both AMI and non-AMI causes of such ST segment changes in the chest paint patient. In the AMI patient, the initial up-sloping portion of the ST segment usually is either convex or flat; if the ST segment elevation is flat, it may be either horizontally or obliquely so (fig 12A). An analysis of the ST segment waveform may be particularly helpful in distinguishing among the various causes of ST segment elevation and identifying the AMI case. This technique uses the morphology of the initial portion of the ST segment/T wave. This portion of the cardiac electrical cycle is defined as beginning at the J point and ending at the apex of the T wave. Patients with non-infarctional ST segment elevation tend to have a concave morphology of the waveform (fig 12B). Conversely, patients with ST segment elevation caused by AMI have either obliquely flat or convex waveforms. This morphological observation should only be used as a guideline. As with most guidelines, it is not infallible; patients with ST segment elevation due to AMI may demonstrate concavity of this portion of the waveform (fig 12C).[7]

After the ST segment elevation reaches its maximum extent, there follows a gradual fall over the next 12 hours culminating in a return to the isoelectric line. Changes in the QRS complex then develop, including loss of R wave height and development of pathological Q waves—an initial negative deflection of the QRS complex (fig 13). Both changes result from the loss of viable myocardium beneath the recording electrode. The development of Q waves is the only firm electrocardiographic evidence of myocardial death. However, Q waves do not necessarily develop in every lead in which ST segment elevation occurred. With relatively small infarctions, scar tissue may contract during the healing process and render the area electrically inert, causing a disappearance of the Q waves. Q waves are frequently seen within 12 hours (and occasionally as early as one to two hours) of the onset of symptoms, though they may not appear for over 24 hours. A portion of AMI patients, however, will develop Q waves as early as two hours after the onset of transmural AMI. A reliable two hour history of chest discomfort in the patient with electrocardiographic ST segment elevation and prominent Q waves (figs 4 and 14A/B) in the same anatomic distribution does not preclude the patient from consideration of acute, urgent revascularisation treatment.

As the process evolves, the ST segment elevation diminishes and the T waves invert. With an inferior myocardial infarction, it may require two weeks for the ST segments to return to the isoelectric line. ST segment elevation in the anterior chest leads may take even longer to resolve. T waves may remain inverted for months and, occasionally, the change is permanent. When the T waves

and symptom development. However, it must be remembered that ST segment elevation indicates myocardial injury, rather than established infarction. These early ST segment elevation morphologies may be completely reversible as demonstrated by the ECGs of patients with coronary artery spasm. However, coronary artery spasm is uncommon;
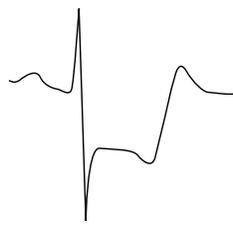
Figure 15 Reciprocal ST segment depression, also known as reciprocal change, is defined as ST segment depression in leads separate and distinct from leads reflecting ST segment elevation. The ST segment depression is either horizontal or down-sloping. Reciprocal ST segment depression in the setting of ST segment elevation on other electrocardiographic leads strongly supports the diagnosis of AMI.
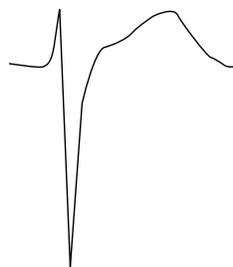

Figure 16 12 lead ECG demonstrating ST segment elevation and prominent Q waves in the anterior distribution. The patient had experienced an extensive anterior myocardial infarction three months ago. The persistent ST segment elevation resulted from a left ventricular aneurysm confirmed by echocardiography.

initially invert they do so in a characteristic symmetrical fashion. If the T wave remains inverted indefinitely, the shape tends to become asymmetric.

ST segment depression may be apparent in leads remote to the area of acute infarction with ST segment elevation; this ST segment depression is termed reciprocal change (fig 15). Reciprocal changes occur in as many as 80% of patients with ST segment elevation and the degree of depression is frequently proportional to the degree of ST segment elevation. The reciprocal depression is usually most marked early in the disease process and resolves within 24 hours of the onset of symptoms in at least 50% of patients. The pathogenesis of reciprocal change is uncertain, perhaps involving inverse or reciprocal ST segment change from the area with acute infarction manifested by ST segment elevation. When seen in leads orientated at approximately 180 degrees to the area of infarction, these changes are thought to represent a "mirror image" of the injury pattern, hence the term "reciprocal". This observation may be the explanation for the ST segment depression seen in lead aVL (−30 degrees) in association with ST segment elevation in lead III (+120 degrees), as these electrodes are directed opposite to each other. Reciprocal changes are most commonly seen with inferior myocardial infarction[4] where ST segment depression may be found in the right precordial chest leads (V1–V4). Here, the "mirror image" explanation is less convincing, especially when it is appreciated that the mean frontal plane (limb leads) and the horizontal plane (chest leads) have vectors which are oriented 90 degrees to each other. The pathogenesis of reciprocal changes has yet to be fully explained. "Mirror image" change may be one explanation, but it has also been shown that ST segment depression may occur as a result of ischaemia in an unrelated arterial territory; alternatively, reciprocal change may also result from extension of the infarction. Thus, ST segment depression in the right sided chest leads associated with an acute inferior myocardial infarction may be the result of posterior extension of the injury or ischaemia in the territory supplied by the left anterior descending artery.

Regardless of their cause, patients with an ECG demonstrating reciprocal changes have higher complication rates during the acute phase of their AMI and may gain particular benefit from thrombolysis.[8–11] Furthermore, the presence of reciprocal changes on the ECG is a highly sensitive indicator of AMI, with positive predictive values greater than 90%. An awareness of the significance of reciprocal changes can lead to greatly improved diagnostic accuracy, and is especially useful in patients with chest pain and ST segment elevation of uncertain cause, as seen in case 5 (fig 6).

In a normal ECG of a healthy patient without ischaemic heart disease, the ST segment in V2 and V3 merges with the T wave and can give the impression of ST segment elevation. In some patients, it is difficult to determine whether this is a normal variant ("high take off") or true pathological ST segment elevation. Caution is advised when minimal ST segment elevation is apparent in these leads alone. Serial ECGs performed over a short period of observation will assist in answering this question in a patient with chest pain. The early ST segment and T wave changes seen in AMI are dynamic and are likely to change over the short term; evolving changes or the development of reciprocal changes would indicate the changes are of significance.

The ECG in a patient with a previous myocardial infarction can also give rise to diagnostic difficulty. ST segment elevation in association with Q waves may persist for a number of months particularly after anterior myocardial infarction—indicating a left ventricular aneurysm (figs 3 and 16). In the acute setting, it can be difficult to differentiate new ST segment elevation from previous, persisting ST segment change in patients with previous anterior myocardial infarction (case 2, fig 3). Serial ECGs and comparison with an old recording are helpful in these cases. Other conditions, such as left ventricular hypertrophy, benign early repolarisation and LBBB, can all give rise to electrocardiographic changes which can be confused with those of AMI. This fact is often not highlighted when the cardinal electrocardiographic features of these conditions are interpreted by inexperienced clinicians who may erroneously diagnose AMI. As a consequence, thrombolysis may be inappropriately prescribed. The electrocardiographic changes associated with the conditions mentioned above will be the subject of future articles.

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