

Electrocardiogram in the diagnosis of myocardial ischemia and infarction

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INTRODUCTION

The electrocardiogram (ECG) is an essential diagnostic test for patients with possible or established myocardial ischemia, injury, or infarction. Abnormalities are manifest in the ST segment, T wave, and QRS complex. However, the ECG may be normal or nonspecific in these patients.

In addition, findings thought typical of acute myocardial infarction (MI) due to atherosclerosis may occur in other conditions, such as myocarditis or stress cardiomyopathy. (See "[Clinical manifestations and diagnosis of myocarditis in adults](#)" and "[Clinical manifestations and diagnosis of stress \(takotsubo\) cardiomyopathy](#)".)

The use of the ECG in patients with suspected or proven myocardial ischemia, injury, or MI will be reviewed here. Other relevant topics include:

- (See "[ECG tutorial: Myocardial ischemia and infarction](#)".)
- (See "[Electrocardiogram in the prognosis of myocardial infarction or unstable angina](#)".)
- (See "[Electrocardiographic diagnosis of myocardial infarction in the presence of bundle branch block or a paced rhythm](#)".)

INDICATIONS

All patients with a presentation consistent with myocardial ischemia should receive one or more standard 12-lead ECGs.

The details on the evaluation of suspected ACS are described elsewhere. (See "[Initial evaluation and management of suspected acute coronary syndrome \(myocardial infarction, unstable angina\) in the emergency department](#)", section on '[Electrocardiogram assessment](#)' and "[Initial evaluation and management of suspected acute coronary syndrome \(myocardial infarction, unstable angina\) in the emergency department](#)", section on '[Importance of serial electrocardiograms](#)'.)

WHEN TO PLACE NONSTANDARD LEADS?

For many patients, standard ECG lead placement will reveal the severity and location of ischemia ([figure 1](#)). However, ECGs recorded with nonstandard ECG lead placement (eg, right-sided leads) can identify right ventricular infarction and posterior wall infarction that standard ECG lead placement cannot identify ([figure 2](#)). The need for nonstandard lead placement is determined by the findings from the standard ECG:

- **Patients with evidence of inferior wall MI** – In patients with ST elevation in leads II, III, and aVF, right-sided leads should be placed to assess for the presence of right ventricular infarction ([waveform 1](#) and [figure 2](#)). (See '[Inferior and right ventricular MI](#)' below.)
- **Patients with ST depression in leads V1 and V2** – In patients with ST depression in leads V1 and V2, posterior ECG leads should be placed to identify the presence of a posterior wall ST-elevation MI (STEMI) ([figure 3](#)). (See '[Posterior wall MI](#)' below.)

ECG CRITERIA FOR MYOCARDIAL ISCHEMIA/INFARCT

According to the 2018 European Society of Cardiology/American College of Cardiology Foundation/American Heart Association/World Health Federation Universal Definition of Myocardial Infarction, the following are the classic ECG criteria for the two major categories of ECG manifestations of acute myocardial ischemia [1]:

- **Findings consistent with ST-elevation myocardial infarction (STEMI)** – New ST-segment elevation at the J-point in two contiguous leads with the cut-points: ≥1 mm in all leads other than leads V2 to V3. For leads V2 to V3: ≥2 mm in males ≥40 years, ≥2.5 mm in males

<40 years, or ≥ 1.5 mm in females regardless of age. This assumes usual calibration of 1 mV/10 mm.

- **Findings consistent with non-ST-elevation MI (NSTEMI) or unstable angina** – New horizontal or downsloping ST depression ≥ 0.5 mm in two contiguous leads and/or T inversion > 1 mm in two contiguous leads with prominent R wave or R/S ratio > 1 .

The findings on the ECG depend upon several characteristics of the ischemia or infarction including:

- Duration – Hyperacute, acute, evolving, or chronic.
- Size – Amount of myocardium affected.
- Location – Anterior, lateral, inferior-posterior, or right ventricle. These criteria should be used as guidelines in the context of other clinical information. Further, false positive and negative ECG findings may appear and the ECG changes of acute ischemia/injury/MI may rapidly evolve.

SIGNIFICANCE OF Q WAVES

Q waves are not required for the ECG diagnosis of acute MI, as discussed above (see '[ECG criteria for myocardial ischemia/infarct](#)' above). When present, they may suggest the portion of the left and right ventricles that have been affected. Loss of electromotive forces due to infarcted myocardial tissue leads to R-wave loss. Delayed conduction through an ischemic area or conduction around it results in recording potentials from the opposite ventricular wall, which manifest as Q-wave formation in the relevant leads.

Q waves can be seen during the evolution of STEMI and, less commonly, in NSTEMI. Studies that compared ECG findings to pathologic specimens or magnetic resonance imaging have shown that Q waves correlate more with the size of an infarction than with the extent of infarction (ie, transmural or subendocardial) [2,3]. Thus, it is preferable to describe the ECG features of an infarct as Q wave or non-Q wave along with any abnormalities of the ST segment, rather than with the terms "transmural" or "subendocardial." (See "[Pathogenesis and diagnosis of Q waves on the electrocardiogram](#)".)

The ECG findings related to Q waves may be somewhat different with posterior or lateral MI. Loss of depolarization forces in these regions can reciprocally **increase** R-wave amplitude in leads V1 and V2 without causing diagnostic Q waves in any of the conventional leads. (See '[Posterior wall MI](#)' below.)

According to the Fourth Universal Definition, any one of the following three ECG criteria are associated with prior MI (in the absence of left bundle branch block or left ventricular hypertrophy) [1]:

- Any Q wave in leads V2 to V3 ≥ 0.02 sec or QS complex in V2 and V3.
 - Q wave ≥ 0.03 sec and ≥ 0.1 mV deep or QS complex in leads I, II, aVL, aVF; or V4 to V6 in any two leads of a contiguous lead grouping (I, aVL; V1 to V6; II, III, aVF).
 - R wave ≥ 0.04 sec in V1 to V2 and R/S ≥ 1 with a concordant positive T wave in the absence of a conduction defect.
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LOCATION OF ISCHEMIA OR INFARCTION

The portion of the left ventricle that is ischemic or infarcted may be predicted by which ECG leads show ST-segment, T-wave, or Q-wave abnormalities [1]. Clinicians may be able to use this information to adjust the management approach and help forecast potential complications. For instance, those with inferior MI should have right-sided ECG leads placed to look for ECG evidence of right ventricular involvement ([figure 2](#)). These patients should not be given nitrates, as this therapy may excessively decrease ventricular preload, thereby causing hypotension and worsening ischemia. If there is evidence of low cardiac output in the absence of signs of elevated left ventricular end-diastolic pressures, management may include judicious administration of intravenous fluids to enhance preload. (See "[Right ventricular myocardial infarction](#)".)

The location of acute infarction also has implications for the mechanism of atrioventricular (AV) heart blocks associated with different ECG presentations. Of note, AV heart block (second or third degree) in patients with acute or evolving inferior MI is usually localized to conduction abnormalities in the AV node (ie, above the bundle of His). In contrast, high degree AV block with an acute or evolving anterior MI is typically associated with infra-nodal block, especially when associated with a bundle branch block pattern. (See "[Conduction abnormalities after myocardial infarction](#)".)

Multiple factors can affect the amplitude of acute ischemic ST deviations. Marked ST elevation or depression in multiple leads usually indicates severe localized ischemia or ischemia affecting large regions of the myocardium. Conversely, substantial (≥ 70 percent) resolution of ST elevation promptly following fibrinolytic therapy is a robust predictor of both target vessel patency and outcomes [4-6]. However, these relationships are not universal since severe ischemia or even MI can occur with slight or even absent ST-T changes.

In the chronic post-infarction stage, localization of prior ischemic events has been determined by the distribution of Q waves when present. (See '[Significance of Q waves](#)' above.) Q waves usually follow a similar distribution to the acute ST and T-wave abnormalities.

Anterior, lateral, and apical MI — ST-segment elevation or Q waves in one or more of the precordial leads (V1 to V6) and leads I and aVL has traditionally been used to suggest anterior wall ischemia or infarction ([waveform 2A-B](#)). Although characteristic ECG changes in leads V1 to V3 are considered typical of anteroseptal ischemia, they may be more indicative of apical ischemia. This was illustrated in a review of 50 patients with new Q waves in leads V1 to V3 [7].

Echocardiography and angiography showed that the apex was affected in all patients and was the only involved site in 26 of the 50 patients (52 percent); the septum and anterior and lateral walls were also affected in the remaining 24 patients (48 percent), but the degree of involvement was less severe than in the apex.

Similar findings were noted in another analysis in which 48 of 52 patients with acute ST-segment elevations in leads V1 to V3 had an antero-apical infarct and a normal septum [8]. Changes in leads V4 to V6 suggest anterolateral ischemia or infarction.

Inferior and right ventricular MI — ST-segment shifts or Q waves in leads II, III, and aVF suggest inferior wall ischemia or infarction ([waveform 1](#)). If there is evidence of inferior wall ischemia, right-sided leads, especially V3R and V4R, should be obtained to assess for a possible right ventricular ischemia/infarction ([waveform 1](#) and [figure 2](#)) [2]. (See "[Right ventricular myocardial infarction](#)".)

Posterior wall MI — Acute posterior wall MI induces ST elevations in leads placed over the back of the heart, eg, leads V7 to V9 ([waveform 3](#) and [figure 3](#)) [9-12]. This is usually associated with reciprocal ST-segment depression in leads V1 to V2, V3, or V4. Similar ST changes can also be the primary ECG manifestation of anterior subendocardial ischemia that may occur in combination with inferior infarction. Posterior inferior wall MI can usually be differentiated from anterior wall ischemia by the presence of ST-segment elevation in the inferior leads (II, III, aVF) in addition to posterior leads V7 to V9 ([table 1](#)) [9,11,13]. Relatively tall R waves may also appear in leads V1 to V3 ([waveform 4](#)), corresponding to the appearance of pathologic Q waves (loss of depolarization forces) in the posterior leads.

Multiple regions — In some cases, ischemia affects more than one region of the myocardium. In this setting, the ECG should show the characteristic findings of involvement in each region ([waveform 5](#)). However, partial normalization may result from cancellation of opposing vectorial forces.

IDENTIFICATION OF THE INFARCT-RELATED ARTERY

The ECG may also provide information about the site of arterial occlusion in patients with STEMI [14].

Inferior MI on the ECG — Patients presenting with an inferior wall MI generally have occlusion of either the right or the left circumflex coronary artery.

The presence of ST-segment elevation in lead III exceeding that in lead II, particularly when combined with ST depression in leads I and aVL, is reported as a very useful predictor of an occlusion in the proximal or mid portion of the right coronary artery, with a relatively high sensitivity and specificity [14-16]. Data in a larger cohort suggest that the sensitivity of this sign may only be 70 percent, with specificity of about 72 percent [16]. The presence of ST-segment elevation in lead II, which is equal to that of lead III, especially when combined with ST depression in leads V1 to V3 or ST elevation in leads I and aVL, is a useful but not absolute predictor of a left circumflex coronary artery occlusion. These findings may also be seen in distal occlusion of a dominant right coronary artery [14,16].

In a series of 109 patients presenting with an inferior wall MI who underwent angiography, the presence of concomitant precordial ST-segment depression was a sensitive, but not specific, indicator of left circumflex occlusion; however, the absence of precordial ST-segment depression had a high negative predictive value for excluding the left circumflex artery as the culprit vessel [13].

Some patients with an inferior MI have right-sided ST elevation in leads V1 and V4R ([figure 2](#)); this finding is indicative of acute right ventricular injury [14,17-19] and correlates closely with occlusion of the proximal right coronary artery. In one report, ST elevation in V4R had 88 percent sensitivity and 78 percent specificity for concurrent right ventricular infarction [18]. ST elevation may sometimes extend from V1 to V2 or V3.

Anterior MI on the ECG — Patients presenting with an anterior wall MI usually have occlusion of the left anterior descending coronary artery (LAD). The presence of ST elevation in lead aVR, complete right bundle branch block, ST depression in lead V5, and/or ST elevation in V1 greater than 2.5 mm strongly predicts a LAD artery occlusion proximal to the first septal perforator [20].

The following additional observations with acute MI due to LAD occlusion have been made:

- Abnormal Q waves in only V4 to V6 are associated with an occlusion distal to the first septal perforator [20].

- An abnormal Q wave in aVL is associated with an LAD occlusion proximal to the first diagonal branch, while ST depression in aVL suggests an occlusion distal to the first diagonal [20].
- Inferior ST depression ≥ 1 mm plus ST elevation in aVL predicts an LAD occlusion proximal to both the septal perforator and diagonal while the absence of inferior ST depression is associated with a distal occlusion [21].
- Deep "coronary" T-wave inversions in multiple precordial leads (eg, V1 to V4) are typically caused by a tight stenosis in the LAD [22-25]. These patients often present without Q waves. (See '[Evolution of the ECG](#)' below.)
- Simultaneous ST elevations in both the anterior and inferior leads raises consideration of occlusion of an LAD that is long, wrapping around the apex to supply the distal inferior wall. This variant has been referred to as a "wrap-around" LAD [26]. However, ST elevation in leads II, III, and aVF may also occur with more distal occlusion of the LAD, distal to the first diagonal branch such that the injury current vector points in a more inferior direction [27].
- Depression of the early part of the ST segment (J point) associated with symmetrically tall positive (hyperacute) T waves in multiple precordial leads [28].

Abnormal findings in lead aVR — ST-segment elevation in lead aVR that is greater than or equal to ST elevation in lead V1 may be useful for diagnosing an acute left main coronary artery obstruction. In a report in which 16 such patients were compared to 46 with left anterior coronary artery obstruction and 24 with right coronary artery obstruction, the above finding distinguished left main coronary occlusion from an LAD occlusion with a sensitivity and specificity of 81 and 80 percent, respectively [29]. In addition, mortality was associated with a greater degree of ST elevation in aVR. Diffuse ST-segment depressions may be seen in other leads.

EXTENT OF THE INFARCT

The pattern of abnormalities can also provide information about the extent of the infarct. As an example, the presence of significant ST-segment elevation (>2 mm) in leads V5 to V6 in association with an inferior wall myocardial infarction is a sensitive and specific (94 and 98 percent, respectively) sign of a very large infarct-related artery and a large area of involved myocardium (inferior and lateral walls) [30]. (See "[Electrocardiogram in the prognosis of myocardial infarction or unstable angina](#)", section on 'Extent of myocardial injury'.)

A subanalysis of the CARDINAL study examined predictors of infarct size in 1622 patients undergoing fibrinolysis or primary percutaneous intervention [31]. The only predictor of having a large infarction (creatinine kinase MB fraction >3000) in those undergoing fibrinolysis or primary percutaneous intervention was a greater amount of ST elevation.

EVOLUTION OF THE ECG

A series of characteristic ECG changes are often seen after significant ischemia or infarction. (See "[ECG tutorial: Myocardial ischemia and infarction](#)", section on '[ST-elevation MI evolution](#)').

STEMI — In the very early stages, the initial ECG may be normal or show so-called "hyperacute T waves." Such T waves may precede or accompany ST-segment shifts, are located in leads that correspond to the area of infarction, and are characterized by a relative increase in positive amplitude ([waveform 6](#)). There are no generally accepted amplitude criteria for hyperacute T waves [32]. Thus, comparison with prior ECGs or serial ECGs may help to identify the presence of hyperacute T waves. This definition excludes tall T waves caused by normal variants, hyperkalemia, left ventricular hypertrophy, left bundle branch block, and cerebrovascular accidents.

Perhaps the most extreme examples are those associated with marked ST elevations that arise from the peak of the preceding R wave. These are formally referred to as "monophasic currents of injury" and are informally referred to as "tombstone T waves" because of their association with severe ischemia ([waveform 7](#)).

In a retrospective analysis of data from the emergency department, hyperacute T waves defined by statistical criteria applied to a single ECG did not appear to be specific or sensitive markers of acute MI [33].

When ischemic ST-segment elevation occurs as the earliest sign of acute STEMI, it is typically followed within a period ranging from hours to days by evolving T-wave inversions, and sometimes Q waves, in the same lead distribution ([waveform 2A-B](#)). T-wave inversions due to evolving or chronic ischemia are often associated with QT prolongation. The T-wave inversions may resolve after days or weeks or persist indefinitely. The extent of the infarct may be an important determinant of T-wave evolution. In one series, T waves that were persistently negative for more than one year in leads with Q waves were associated with a transmural MI; in contrast, T waves that were positive in leads with Q waves were indicative of a nontransmural MI [34]. The resolution of negative T waves also predicts recovery of regional left ventricular dysfunction [35].

Complete normalization of the ECG following STEMI is uncommon but can occur, particularly with smaller infarcts and when left ventricular ejection fraction and regional wall motion improve. Normalization is usually associated with spontaneous recanalization or good collateral circulation [36]. In contrast, persistent Q waves and ST elevations several weeks or more after an infarct correlate strongly with a severe underlying wall motion disorder (akinetic or dyskinetic zone), although not necessarily a frank ventricular aneurysm ([waveform 8](#)).

NSTEMI — Patients with NSTEMI typically present with ST-segment depressions and/or T-wave inversions in two or more leads. As noted above, reciprocal ST-segment depression can occur during an STEMI. For this reason, ST-segment elevation, which is sometimes subtle, should be sought in "opposing" (contralateral) leads whenever ST-segment depression is noted (eg, if ST depression is seen in leads V1 through V3, then the lateral/posterior chest leads should be evaluated for concomitant ST-segment elevation). Note also that the clinical term "opposing" here refers more technically to pairs of leads for which positive poles are oriented at >90 degrees to each other. Another set of "opposing leads" is constituted by lead III (positive pole at +120 degrees) and lead aVL (positive pole at -30 degrees).

As noted above, the association between Q waves and transmural infarction has been questioned and the presence or absence of Q waves may be more closely correlated with the size of the MI than with its transmural extent [2]. (See '[Location of ischemia or infarction](#)' above and "[Pathogenesis and diagnosis of Q waves on the electrocardiogram](#)".)

Patients may have other ECG patterns besides ST elevation or depression or T-wave inversions that can reflect ischemia or infarction.

OTHER ECG MANIFESTATIONS OF ISCHEMIA

Left anterior descending coronary T-wave inversion pattern — Some patients with ischemic chest pain present with deep "coronary" T-wave inversions in multiple precordial leads (eg, V1 to V4) with or without cardiac enzyme elevations and with minimal or no ST elevations. This pattern, often referred to as the "Wellens pattern" or as the "LAD-T-wave inversion pattern," is typically caused by high-grade stenosis in the LAD coronary artery system ([waveform 9](#)). The natural history of this pattern is unfavorable, with a high incidence of recurrent symptoms and myocardial infarction [22-24]. Prominent symmetrical T-wave inversions in the anterior precordial leads may also indicate a regional wall motion abnormality that may be reversible.

de Winter sign — This ECG pattern consists of upsloping ST-segment depression in (usually two or more of) leads V2 to V6 in concert with relatively tall, symmetric T waves, along with possible

loss of precordial R-wave progression [37]. The ST-segment depression and T-wave height are frequently maximal in V3. There may also be 1 to 2 mm ST-segment elevation in aVR. This ECG finding has been associated with occlusion of the LAD, although it may occur with other coronary lesions [38]. An earlier description of this sign classified it as an atypical variant of "hyperacute T waves," possibly reflecting combined transmural and subendocardial ischemia [32]. This finding appears to be highly specific but not sensitive for acute and, usually, rapidly evolving ischemia.

Pseudonormalization of T waves — Patients whose baseline ECG already shows abnormal T-wave inversions may develop paradoxical T-wave normalization (pseudonormalization) during episodes of acute transmural ischemia [39], often associated with chest discomfort or other symptoms or signs of ischemia. A related clinical issue is whether normalization of T-wave inversions during exercise (stress) tests is a form of paradoxical (pseudo-) normalization, and hence a marker of ischemia. We believe that normalization of inverted T waves during stress testing is nondiagnostic and should be considered in the overall clinical context. However, this finding, by itself, is not a specific or sensitive sign of ischemia and should not be labeled as "pseudonormalization," which implies an ischemic mechanism, but simply as "normalization."

UNEXPECTED ABSENCE OF DIAGNOSTIC FINDINGS

The greatest value of the ECG as a diagnostic test is that the results are immediately available. However, the clinician needs to recognize that it has limitations in both sensitivity and specificity for the diagnosis of myocardial ischemia. As mentioned above, the diagnosis of myocardial infarction (MI) does not require an abnormal ECG. (See "[Diagnosis of acute myocardial infarction](#)", section on '[Definitions](#)').

It is important to emphasize that the ECG should always be evaluated in clinical context; elements of the history and physical examination, as well as other laboratory tests, are essential in evaluating patients for myocardial ischemia. In addition, most patients should receive serial ECGs to increase the likelihood of capturing relevant information.

An initially normal ECG does not exclude ischemia or infarction. However, a normal ECG throughout the course of acute MI is distinctly uncommon. Thus, if the initial ECG is not diagnostic, but the patient remains symptomatic and there is a high clinical suspicion for MI, it is recommended that the ECG be repeated at 5- to 10-minute intervals [11]. (See "[Initial evaluation and management of suspected acute coronary syndrome \(myocardial infarction, unstable angina\) in the emergency department](#)").

Absence of Q waves — Pathologic Q waves may be absent in patients with MI. There are a number of mechanisms that could explain the absence of Q waves in such patients [40]:

- Small infarcts may not cause abnormal Q waves to appear.
- Infarction in areas that are electrically "silent," that is, in areas that project potentials to regions on the body surface on which there are no electrodes.
- Chronically ischemic or "hibernating" but noninfarcted myocardium [41,42]. (See "[Clinical syndromes of stunned or hibernating myocardium](#)".)
- Resolution of the Q wave. Approximately 10 percent of anterior and 25 percent of inferior MIs revert to a nondiagnostic pattern within two years after the infarct, and a higher percentage have a diminution in Q-wave area [43-45]. Functional recovery of stunned myocardium contributes to Q-wave resolution [45].
- Other concomitant conduction disorders such as left bundle branch block, electronic ventricular pacemaker patterns, and Wolff-Parkinson-White syndrome that obscure the emergence of new Q waves.

Left bundle branch block — The presence of left bundle branch block often obscures the classical ECG diagnosis of acute MI. Assessment of ST-segment changes may be useful in this setting. ST-segment shifts that occur in the same direction as the major QRS vector (so-called "primary" or concordant ST changes) can indicate ischemia or infarction. Such shifts may include ST depression of at least 1 mm in leads V1, V2, or V3 or in leads II, III, or aVF, with elevation of at least 1 mm in lead V5. Extremely discordant ST changes (changes in the opposite direction of the major QRS vector of >5 mm) were also reported to be suggestive of MI [14,46], although exceptions occur, importantly limiting the specificity of this sign. This issue is discussed in greater detail separately. (See "[Electrocardiographic diagnosis of myocardial infarction in the presence of bundle branch block or a paced rhythm](#)".)

ST elevation is not affected by the presence of a right bundle branch block and the ECG should be interpreted as if the right bundle branch block was not present. ST elevation can occur in numerous other conditions ([table 2](#)) [47].

DIFFERENTIAL DIAGNOSIS OF ECG ABNORMALITIES

ECG abnormalities that appear to represent myocardial ischemia or infarction may be present for other reasons.

Early repolarization — Early repolarization is present on the ECG when there is J-point elevation of ≥ 0.1 mV in two adjacent leads with either a slurred or notched morphology ([waveform 10](#)). While patients with acute myocardial injury due to STEMI can initially have elevation of the J-point with concave ST-segment elevation, the ST-segment elevation typically becomes more pronounced and convex (rounded upward) as the infarction persists. However, the primary distinguishing factor between early repolarization and acute myocardial injury is the presence of clinical symptoms such as chest pain or dyspnea. Early repolarization is discussed in detail elsewhere. (See "[Early repolarization](#)".)

This pattern may be seen in healthy subjects, particularly young males. The ECG shows normal variant ST-segment elevations (2 to 3 mm) that are usually best seen in the mid-chest leads, that is V3 to V4. Reciprocal ST depression may be present but limited to lead aVR. ST elevations may be seen in the limb leads, but are less than 1 mm.

ST-segment elevation or depression — Atherosclerotic narrowing of one or more coronary artery is the most common cause of myocardial ischemia or infarction and ST-segment abnormalities. However, other distinct clinical entities can lead these changes. Common associations include coronary artery spasm, microvascular coronary artery disease, myopericarditis, and stress cardiomyopathy. (See "[Vasospastic angina](#)" and "[Microvascular angina: Angina pectoris with normal coronary arteries](#)" and "[Myopericarditis](#)" and "[Clinical manifestations and diagnosis of stress \(takotsubo\) cardiomyopathy](#)".)

Acute pericarditis, in contrast to acute MI, typically induces diffuse ST-segment elevations, usually in most of the chest leads and leads I, II, aVL, and aVF ([table 3](#)). Reciprocal ST depression is seen in lead aVR. An important clue to pericarditis in addition to the diffuse nature of the ST elevations is the presence of PR segment elevation in aVR with PR segment depressions in other leads due to a concomitant atrial current of injury ([waveform 11](#)). Abnormal Q waves do not occur, and the ST elevation is followed by T-wave inversion after a variable time period. (See "[Acute pericarditis: Clinical presentation and diagnosis](#)", section on '[Electrocardiogram](#)'.)

Myocarditis can, in some patients, simulate the ECG pattern of acute pericarditis or acute MI. Like acute MI, myocarditis may be associated with regional ST elevations and Q waves, elevated serum concentrations of creatine kinase MB fraction, and regional wall motion abnormalities on echocardiography [48,49]. Myocarditis should be suspected in young patients who present with a possible MI but have a normal coronary angiogram. In one study of 45 such patients, 35 (78 percent) had a diffuse or focal myocarditis on myocardial imaging [49]. Complete recovery of left ventricular function occurred at six months in 81 percent. (See "[Clinical manifestations and diagnosis of myocarditis in adults](#)".)

ST elevation also occurs in the early phase of acute stress-induced (takotsubo) cardiomyopathy, sometimes called "transient left ventricular ballooning syndrome," which is marked by reversible left ventricular (apex and mid-ventricle) wall motion abnormalities. (See "[Clinical manifestations and diagnosis of stress \(takotsubo\) cardiomyopathy](#)".)

The Brugada pattern, with ST elevations in V1 to V3 associated with a right bundle branch-like pattern, is another cause of nonischemic ST-segment elevations. (See "[Brugada syndrome: Clinical presentation, diagnosis, and evaluation](#)".)

Digitalis, ventricular hypertrophy, hypokalemia, and a variety of other factors can cause ST-segment depression mimicking subendocardial ischemia. (See "[ECG tutorial: Miscellaneous diagnoses](#)", section on '[Digitalis toxicity](#)' and "[Clinical manifestations and treatment of hypokalemia in adults](#)", section on '[Cardiac arrhythmias and ECG abnormalities](#)' and "[Left ventricular hypertrophy: Clinical findings and ECG diagnosis](#)", section on '[Electrocardiographic findings: General](#)'.)

Abnormal T waves — Tall positive T waves do not invariably represent hyperacute ischemic changes but can reflect normal variants, hyperkalemia, cerebrovascular injury, left ventricular volume loads due to mitral or aortic regurgitation, among other causes. ST elevations and tall positive T waves are also common findings in leads V1 and V2 with left bundle branch block or left ventricular hypertrophy patterns ([table 2](#)).

Prominent T-wave inversions can occur in a number of conditions, including ventricular hypertrophy, cardiomyopathy, myocarditis, cerebrovascular injury, particularly intracranial hemorrhage ([waveform 12](#)), intermittent ventricular pacing, intermittent left bundle branch block, or intermittent ventricular preexcitation (the latter three are referred to as "memory T waves") [25,50].

Q waves — The differential diagnosis of Q waves, which are often seen with MI, includes physiologic or positional variants, ventricular hypertrophy, acute or chronic noncoronary myocardial injury, Wolff-Parkinson-White preexcitation pattern, and ventricular conduction disorders, especially left bundle branch block ([table 4](#)). The definition of abnormal Q waves has evolved. Classical teachings indicate that a Q wave must be at least 40 ms or more to be considered abnormal. Subsequent studies, however, suggest that Q waves with durations of over 30 ms in leads I, II, aVL, aVF, or V4 to V6 may be equally diagnostic [51]. (See '[Significance of Q waves](#)' above and "[Pathogenesis and diagnosis of Q waves on the electrocardiogram](#)".)

INFORMATION FOR PATIENTS

UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient info" and the keyword(s) of interest.)

- Basics topics (see "[Patient education: ECG and stress test \(The Basics\)](#)")
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SUMMARY

- **Indications** – All patients with suspected or confirmed myocardial ischemia or infarction (MI) should have an ECG performed soon after consideration of the diagnosis. Most patients will require serial ECGs. (See '[Indications](#)' above.)
- **When to place nonstandard leads** – Nonstandard leads should be placed in patients who have evidence of an inferior MI or lateral wall MI ([figure 2](#) and [figure 3](#)). (See '[When to place nonstandard leads?](#)' above.)
- **Findings in ST-elevation MI (STEMI)** – New abnormalities in the ST segment and T waves may represent myocardial ischemia (and sometimes infarction) and may be followed by the formation of Q waves. However, the ECG may be normal or nonspecific in a patient with either ischemia or infarction. (See '[Introduction](#)' above and '[Unexpected absence of diagnostic findings](#)' above.)
 - **Location of myocardial injury**
 - **Anterior or lateral wall injury** – ST-segment elevation or Q waves in one or more of the precordial leads (V1 to V6) and leads I and aVL has traditionally been used to suggest anterior or lateral wall ischemia or infarction ([waveform 2A](#) and [waveform 2B](#) and [waveform 9](#)). (See '[Anterior, lateral, and apical MI](#)' above and '[Anterior MI on the ECG](#)' above.)

- **Inferior wall injury** – ST-segment shifts or Q waves in leads II, III, and aVF suggest inferior wall ischemia or infarction ([waveform 5](#)). Right-sided ECG leads should be performed with inferior ST elevation to evaluate for right ventricular infarction ([figure 2](#)). (See '[Inferior and right ventricular MI](#)' above and '[Inferior MI on the ECG](#)' above.)
 - **Posterior wall injury** – The ECG diagnosis of acute posterior (or postero-lateral) wall MI is associated with ST-segment elevation in leads placed over the back of the heart, eg, leads V7 to V9, often with reciprocal ST depressions in leads V1 and V2 ([waveform 4](#)). (See '[Posterior wall MI](#)' above.)
- **Findings in non-ST-elevation myocardial infarction** – Patients with non-ST-elevation MI (NSTEMI) typically present with ST-segment depression and/or T-wave inversion in two or more leads but may have no ECG changes. (See '[NSTEMI](#)' above.)
 - **Other signs of myocardial ischemia or infarction** – Other signs of ischemia or infarction include:
 - **Left anterior descending coronary T-wave inversion pattern (Wellens pattern)** – Some patients with myocardial ischemia present with deep "coronary" T-wave inversions in multiple precordial leads (eg, V1 to V4) and with minimal or no ST elevations ([waveform 9](#)).
 - **de Winter sign** – Some patients with multivessel disease may present with hyperacute T waves ("de Winter sign"). (See '[de Winter sign](#)' above.)
 - **Pseudonormalization of T waves** – Patients whose baseline ECG shows abnormal T-wave inversions may develop paradoxical T-wave normalization (pseudonormalization) during episodes of acute transmural ischemia. (See '[Pseudonormalization of T waves](#)' above.)
 - **Other conditions causing ST elevation** – ST elevation can occur in other conditions ([table 2](#) and [waveform 8](#) and [waveform 11](#) and [waveform 12](#)). (See '[ST-segment elevation or depression](#)' above.)

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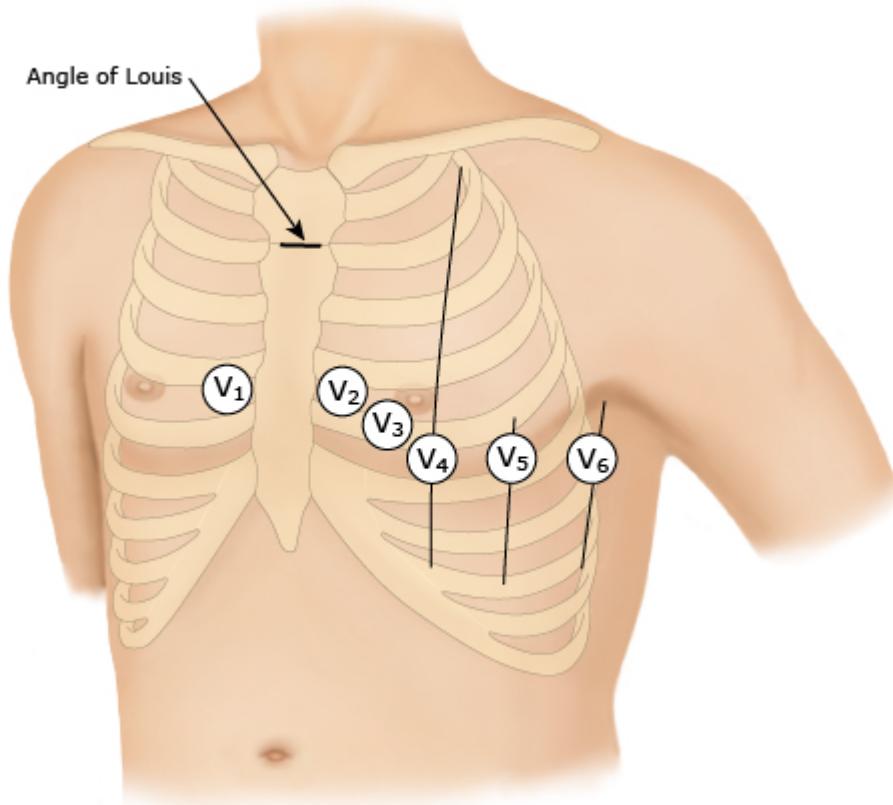
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Topic 2095 Version 36.0

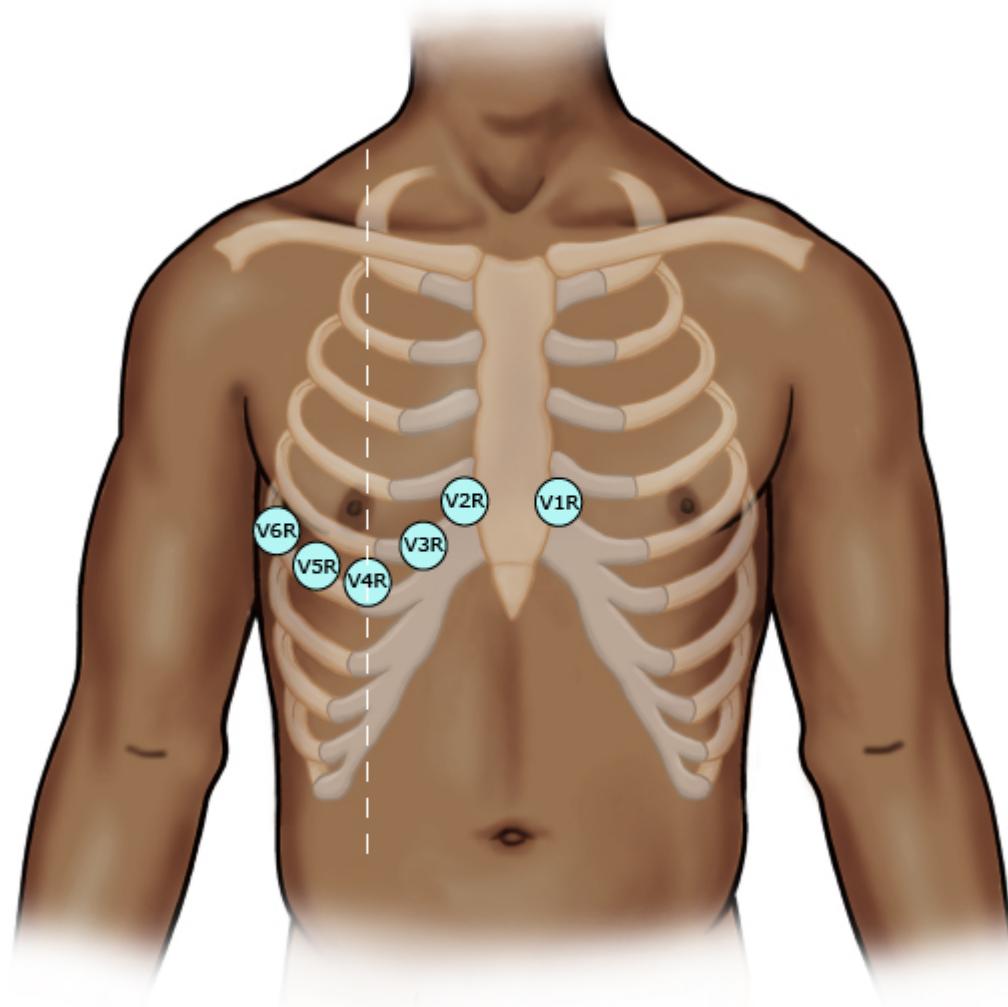
GRAPHICS

Chest lead placement



Graphic 122989 Version 1.0

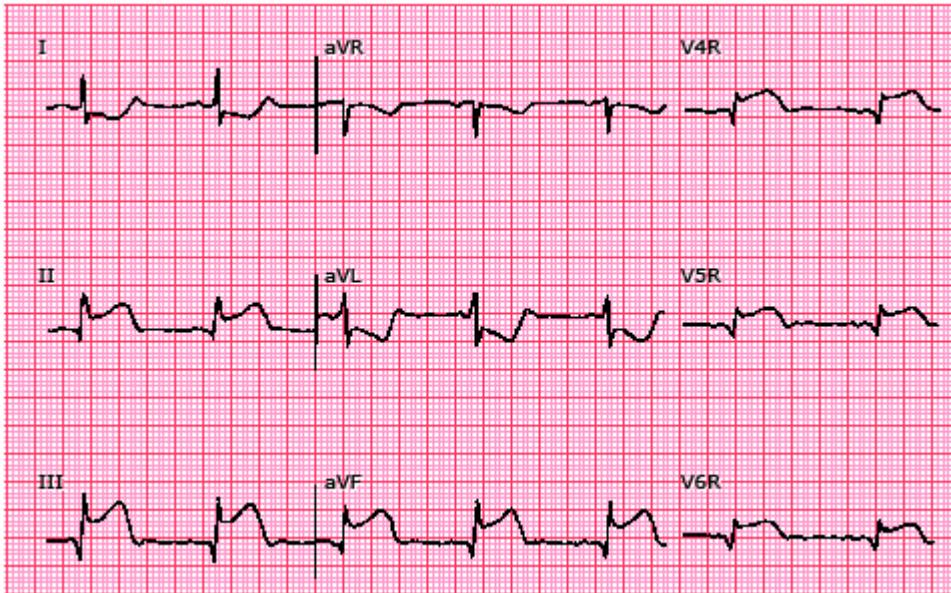
Lead placement for right-sided ECG



Right-sided (V1R to V6R) chest lead placements are mirror the traditional chest lead placements (V1 to V6). Specific positions for right-sided leads are as follows: V1R, 4th intercostal space at the left sternal border; V2R, 4th intercostal space at the right sternal border; V3R, midway between V2R and V4R; V4R, 5th intercostal space in the right midclavicular line; V5R, in the horizontal plane of V4R at the right anterior axillary line (or if the anterior axillary line is ambiguous, midway between V4R and V6R); and V6R in the horizontal plane of V4R at the right midaxillary line. Of note, V1R is the same as the standard V2 lead placement, and V2R is the same as the standard V1 lead placement.

ECG: electrocardiogram.

ECG of acute inferior and right ventricular myocardial infarction



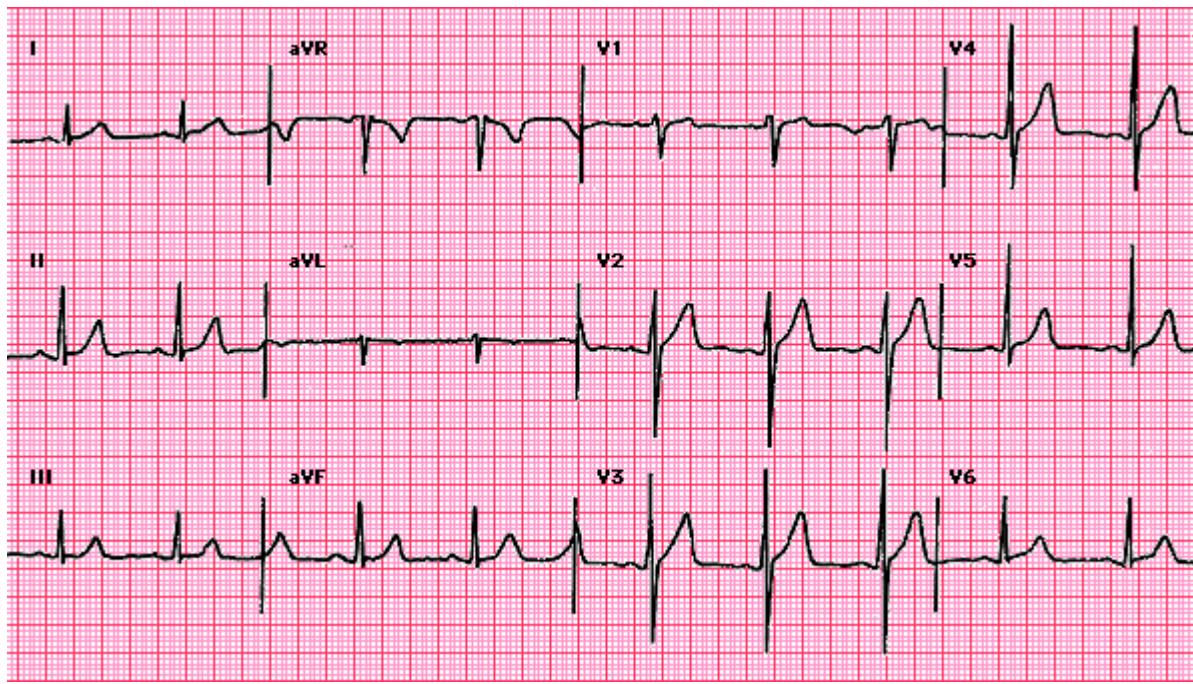
ECG shows Q waves and prominent doming ST-segment elevation in II, III, and aVF, findings which are characteristic of an acute inferior myocardial infarction. ST elevation in the right precordial leads - V4R, V5R, and V6R - indicates right ventricular involvement as well. The ST depressions in leads I and aVL represent reciprocal changes.

ECG: electrocardiogram.

Courtesy of Ary Goldberger, MD.

Graphic 82739 Version 4.0

Normal ECG

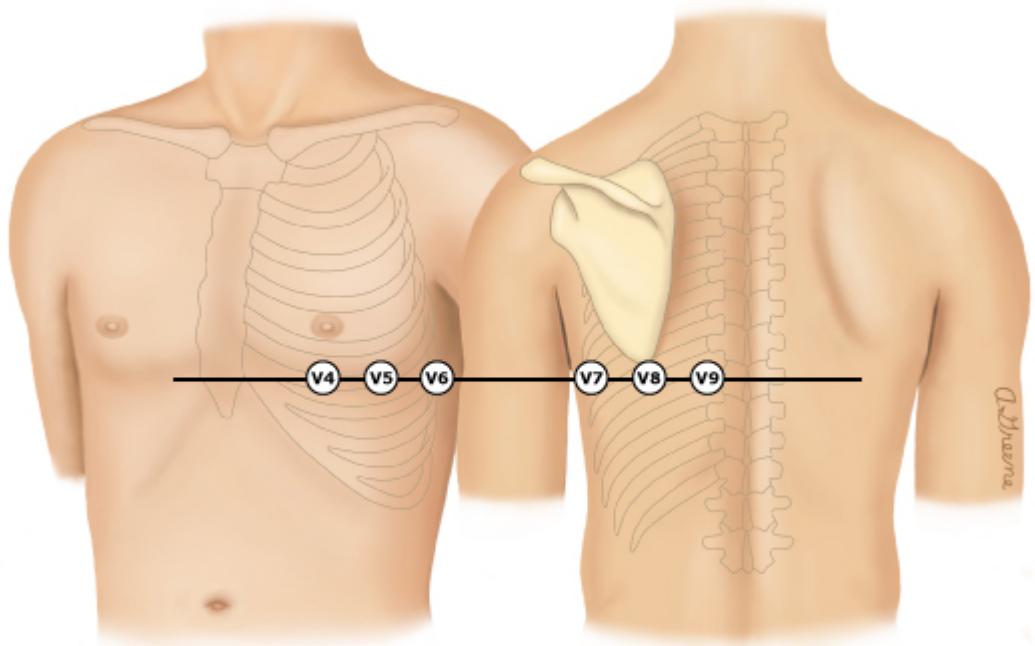


Normal electrocardiogram showing normal sinus rhythm at a rate of 75 beats/minute, a PR interval of 0.14 seconds, a QRS interval of 0.10 seconds, and a QRS axis of approximately 75°.

Courtesy of Ary Goldberger, MD.

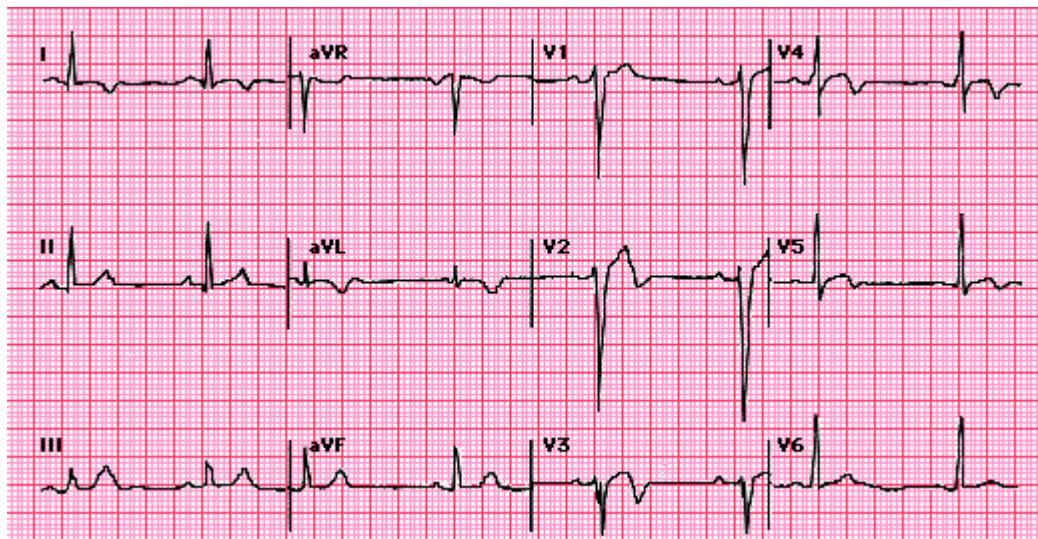
Graphic 76183 Version 4.0

Posterior leads V7 to V9



Graphic 94900 Version 1.0

Electrocardiogram (ECG) in an evolving anterior myocardial infarction

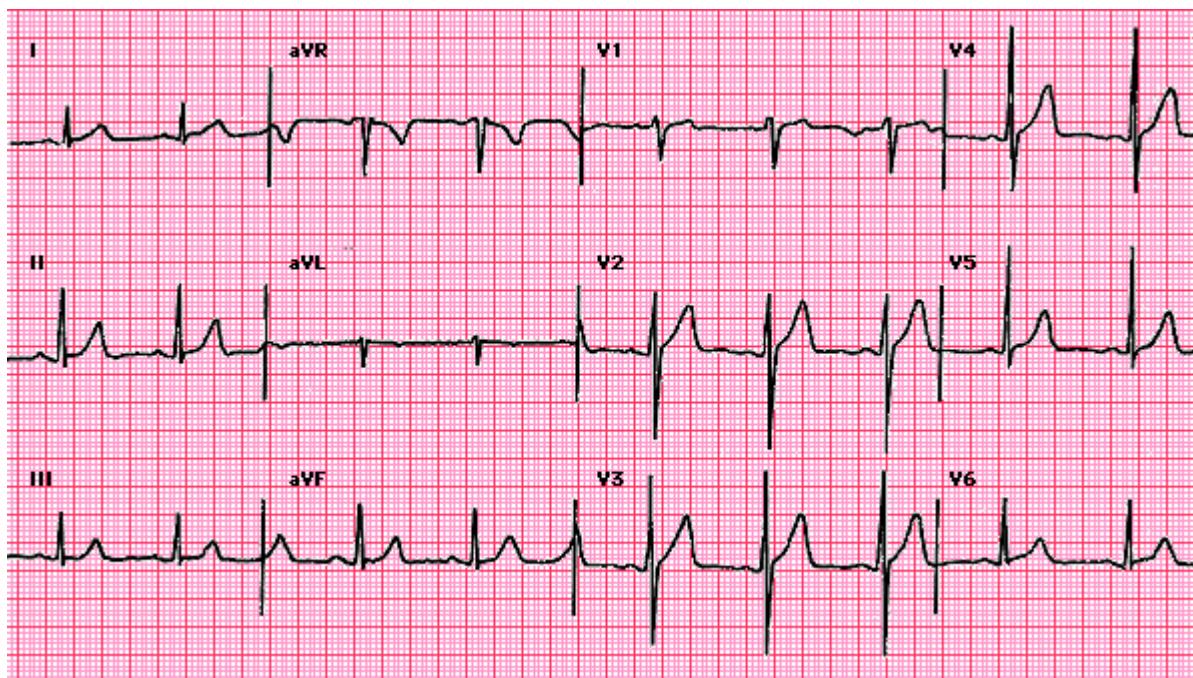


Electrocardiogram shows findings typical of an evolving Q-wave anterior MI: loss of R waves in leads V1 to V3, ST segment elevations in V2 to V4, and T wave inversions in leads I, aVL, and V2 to V5. Sinus bradycardia (55 beats/min) is present due to concurrent therapy with a beta blocker.

Courtesy of Ary Goldberger, MD.

Graphic 81914 Version 3.0

Normal ECG

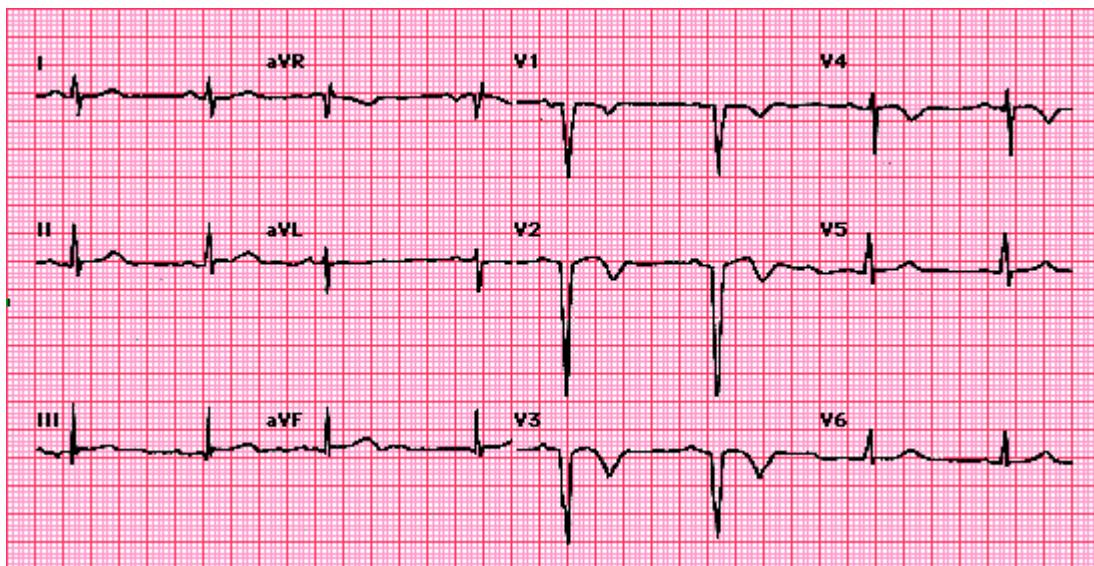


Normal electrocardiogram showing normal sinus rhythm at a rate of 75 beats/minute, a PR interval of 0.14 seconds, a QRS interval of 0.10 seconds, and a QRS axis of approximately 75°.

Courtesy of Ary Goldberger, MD.

Graphic 76183 Version 4.0

Electrocardiogram (ECG) late in the evolution of an anterior myocardial infarction

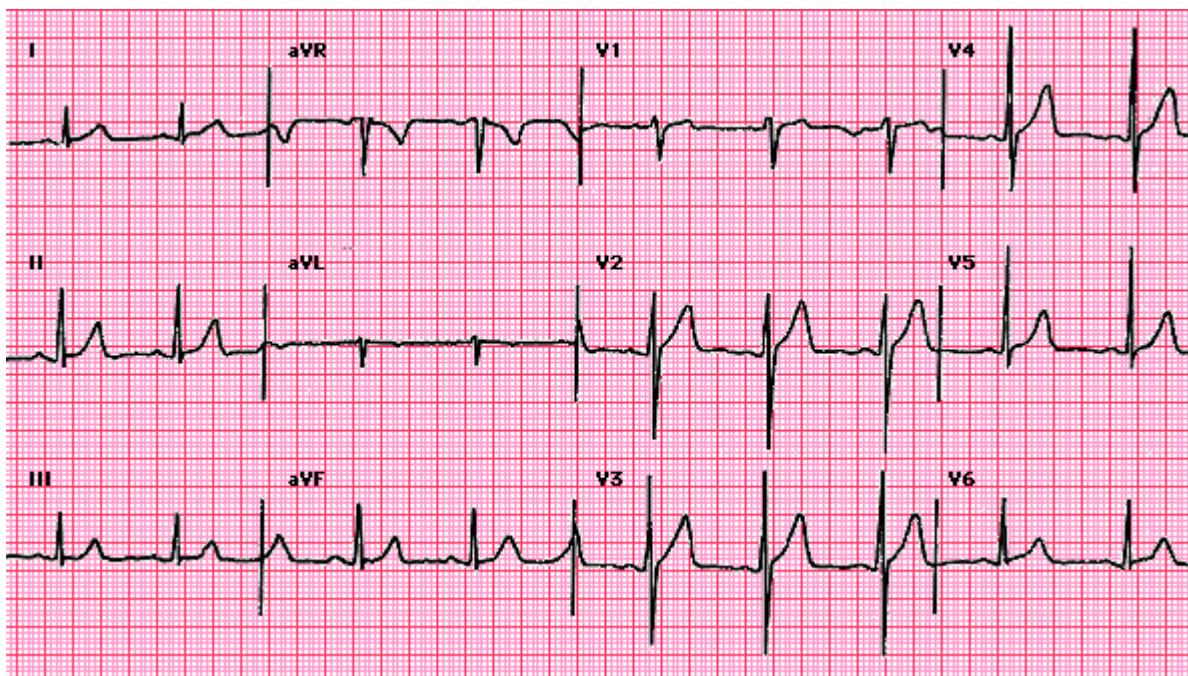


Later stage in the evolution of an acute anterior myocardial infarction. There is a QS pattern in leads V1 to V3 and T wave inversion in leads V2 to V4. The ST segment elevations in these leads have almost disappeared.

Courtesy of Ary Goldberger, MD.

Graphic 62059 Version 3.0

Normal ECG

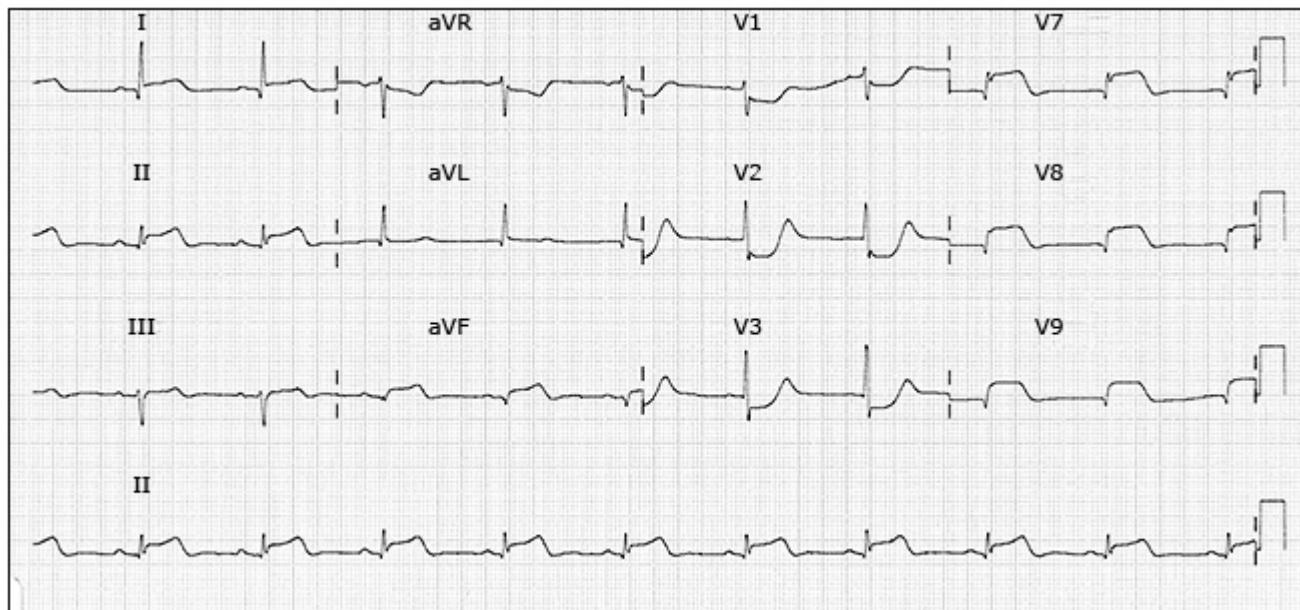


Normal electrocardiogram showing normal sinus rhythm at a rate of 75 beats/minute, a PR interval of 0.14 seconds, a QRS interval of 0.10 seconds, and a QRS axis of approximately 75°.

Courtesy of Ary Goldberger, MD.

Graphic 76183 Version 4.0

Posterior myocardial infarction leads V7 to V9



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Graphic 94928 Version 2.0

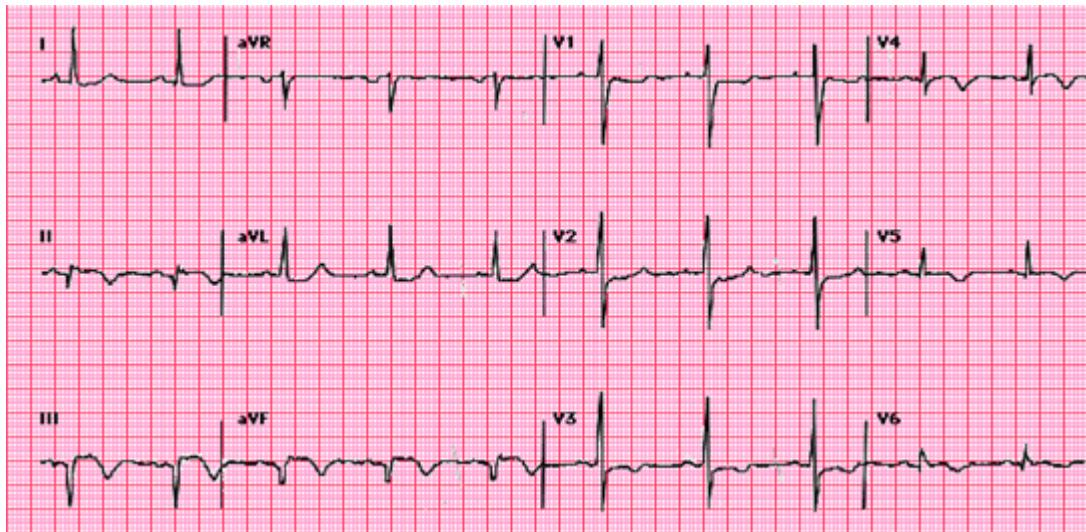
Causes and diagnosis of tall R waves in V1

Diagnosis	Confirmatory clues
True posterior infarct	ST ↓, T ↑ in V1-V2; Q waves and ST ↑ V7-V9
Right ventricular hypertrophy	RAD; RAE; secondary ST-Ts; V7-V9 normal
Ventricular septal hypertrophy	Associated Q waves; LVH; V7-V9 normal or deep narrow Q waves
Right bundle branch block	Wide QRS; broad S in V1, V6; R peaks late in V1; V7-V9 normal or broad S waves
Wolff-Parkinson-White syndrome	Short PR; delta wave; V7-V9 normal or delta wave
Normal variant	No other abnormalities

LVH: left ventricular hypertrophy; RAD: right-axis deviation; RAE: right atrial enlargement.

Graphic 72039 Version 2.0

ECG acute infero-postero-lateral myocardial infarction

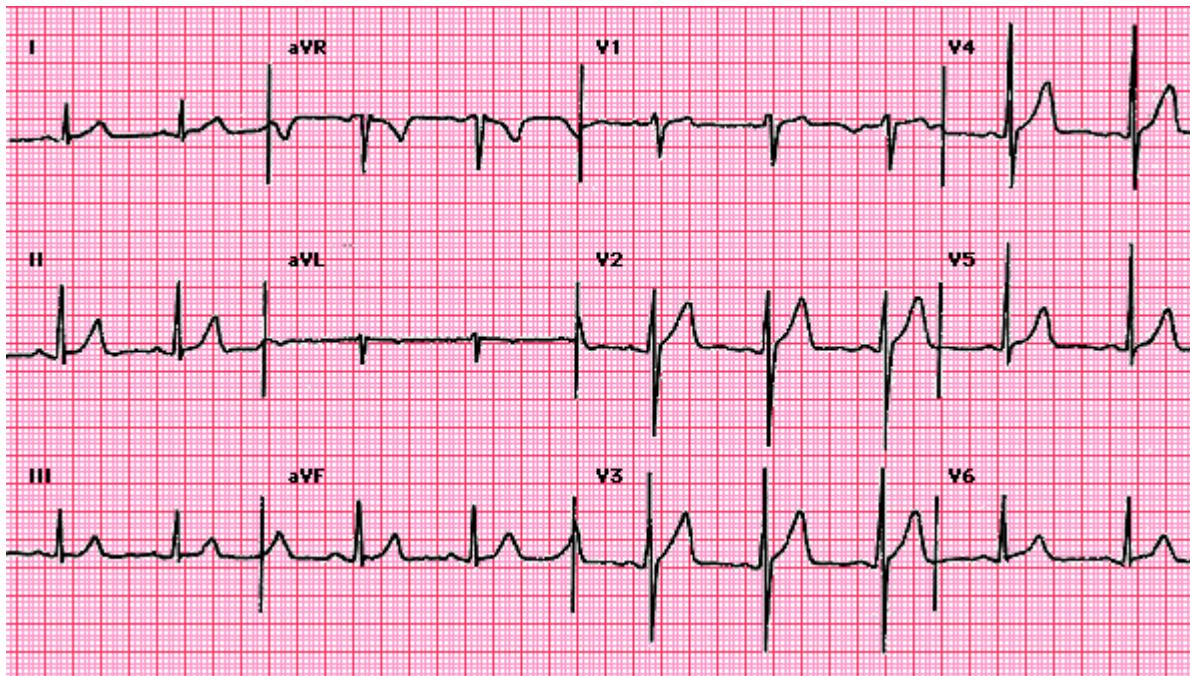


Electrocardiogram showing the major features of an inferior (Q waves, ST elevations, and T wave inversions in II, III, and aVF), posterior (tall R waves in V1 and V2), and lateral (T wave inversions in V4 to V6) myocardial infarction.

Courtesy of Ary Goldberger, MD.

Graphic 70036 Version 3.0

Normal ECG

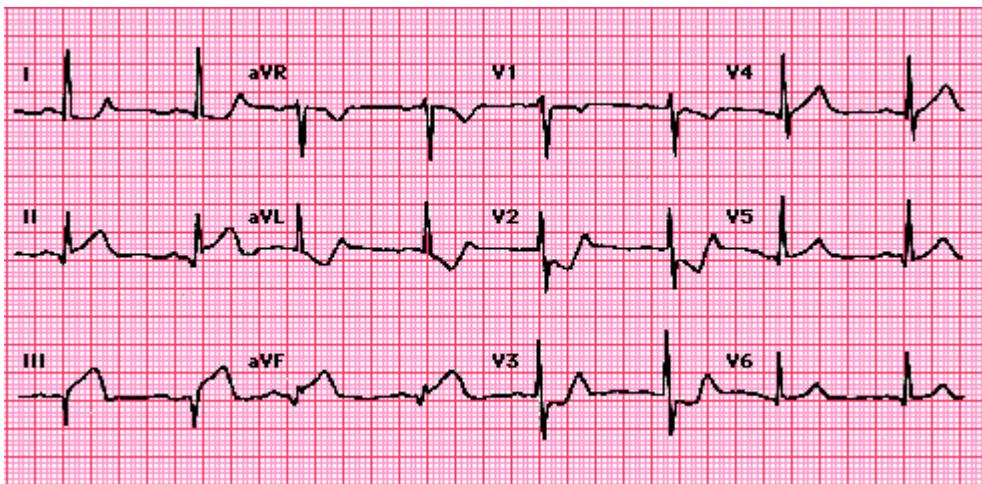


Normal electrocardiogram showing normal sinus rhythm at a rate of 75 beats/minute, a PR interval of 0.14 seconds, a QRS interval of 0.10 seconds, and a QRS axis of approximately 75°.

Courtesy of Ary Goldberger, MD.

Graphic 76183 Version 4.0

ECG of inferior MI with anterior ischemia

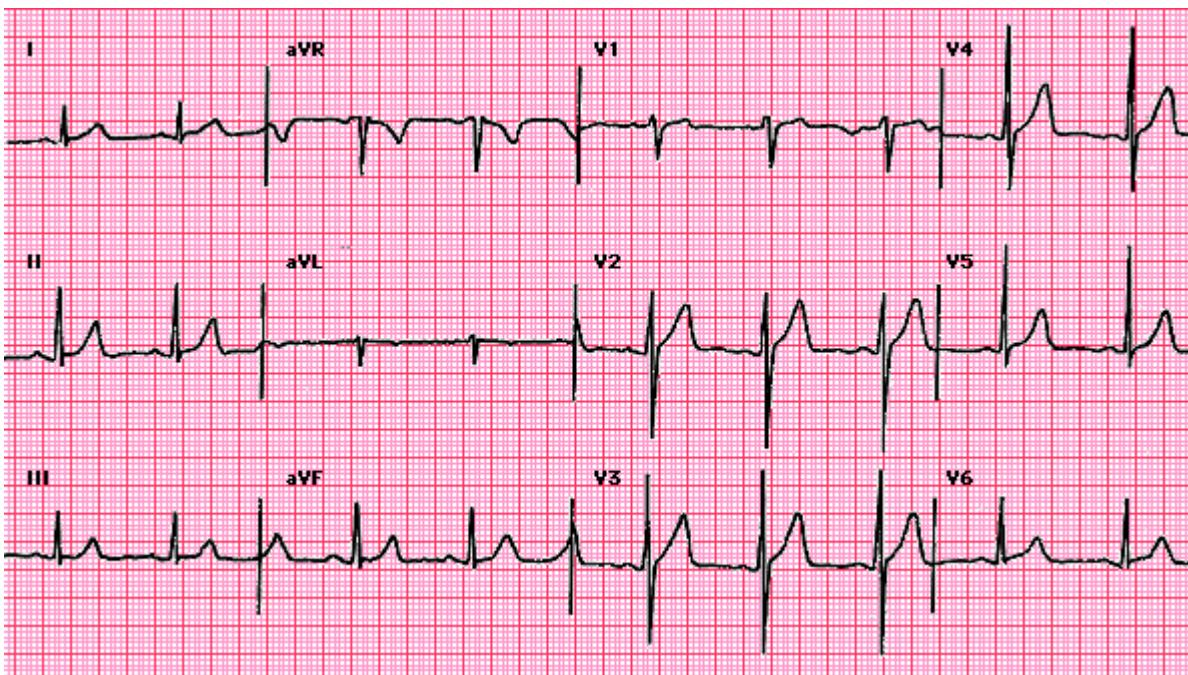


Electrocardiogram showing ischemic changes in two areas of the myocardium: inferior myocardial infarction (Q waves and ST elevations in leads II, III, and aVF); and anterior ischemia (ST depressions in leads V2 and V3).

Courtesy of Ary Goldberger, MD.

Graphic 54339 Version 4.0

Normal ECG

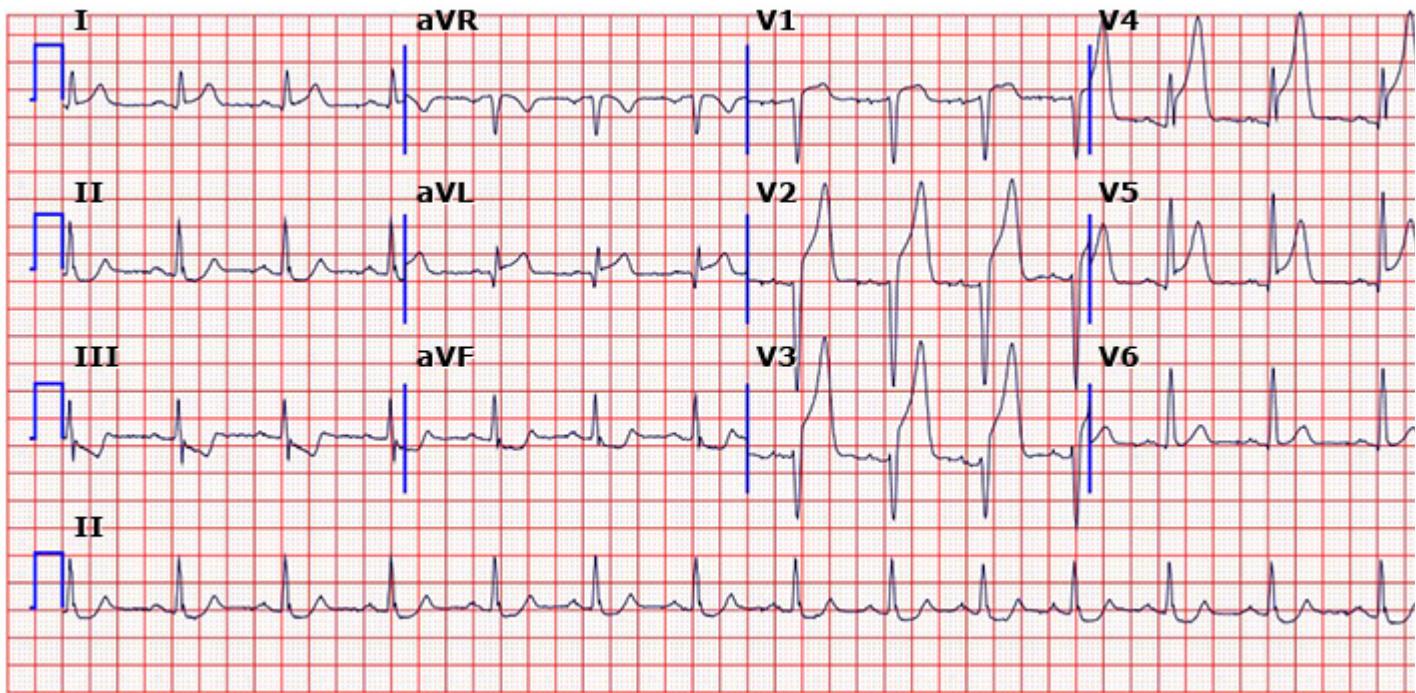


Normal electrocardiogram showing normal sinus rhythm at a rate of 75 beats/minute, a PR interval of 0.14 seconds, a QRS interval of 0.10 seconds, and a QRS axis of approximately 75°.

Courtesy of Ary Goldberger, MD.

Graphic 76183 Version 4.0

Evolving ST elevation and hyperacute T waves 1



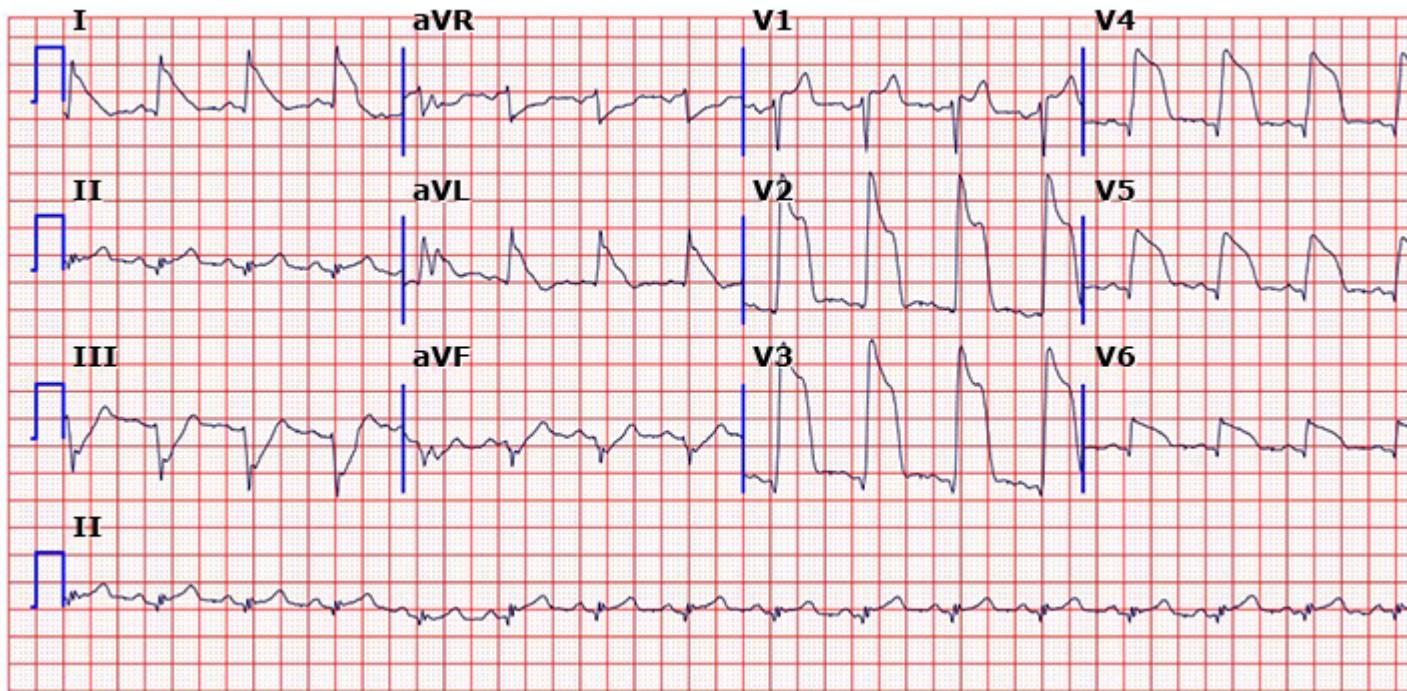
Acute anterior STEMI with hyperacute T waves. ECG shows sinus at about 80 bpm with marked ST elevations in I, aVL, and VI to V5. ST depressions, consistent with reciprocal changes, are seen in leads II, III and aVF. Very slow R-wave progression is present in leads VI to V3. Markedly positive (hyperacute) T waves accompany the precordial ST elevations. Coronary angiography revealed a total proximal occlusion of the left anterior descending coronary artery, successfully treated with a percutaneous coronary intervention.

STEMI: ST-elevation myocardial infarction; ECG: electrocardiogram; bpm: beats per minute.

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Graphic 142416 Version 1.0

Evolving ST elevation and hyperacute T waves 2



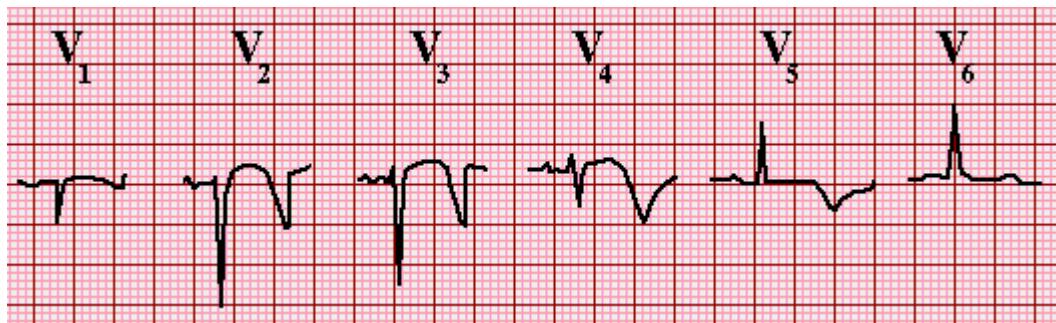
Acute anterior STEMI. What may appear to be wide QRS complexes in leads V2 to V6 are actually marked ST elevations, with hyperacute T waves, due to acute transmural anterior ischemia with myocardial infarction. The inferior leads show reciprocal ST depressions. Q waves are starting to appear in the precordial leads. This ST-T pattern, sometimes called "tombstones" in clinical parlance, is more technically known as a "monophasic current of injury." Coronary angiography showed a proximal occlusion of the left anterior descending coronary artery, treated with a percutaneous coronary intervention.

STEMI: ST-elevation myocardial infarction.

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Graphic 142417 Version 1.0

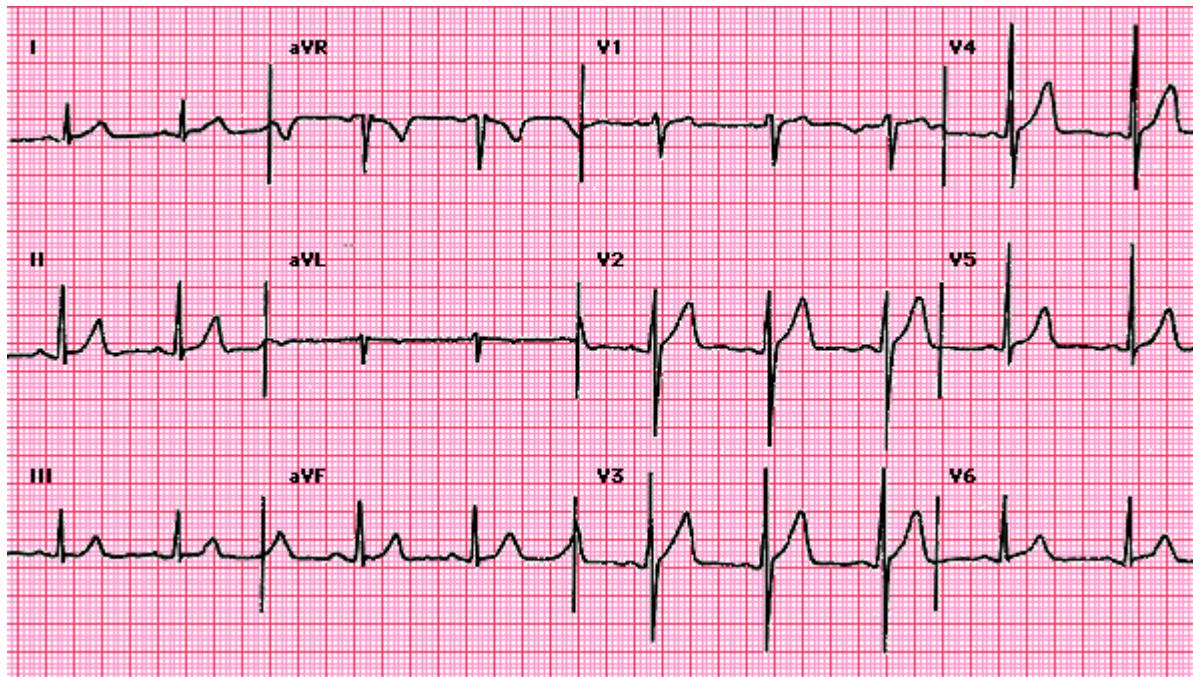
Persistent ST-segment elevation post-MI



The presence of an anterior wall aneurysm following an acute myocardial infarction is suspected because of persistent ST elevation in leads V₂ to V₄. Inverted T waves are evidence of the old infarct.

Graphic 74283 Version 6.0

Normal ECG

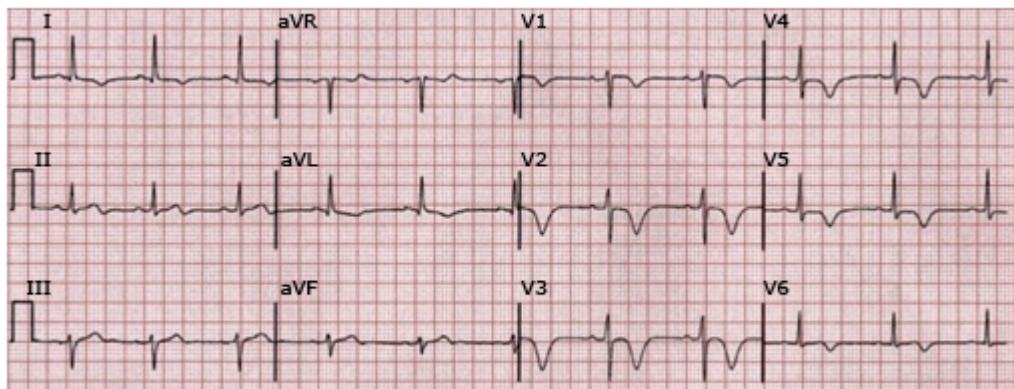


Normal electrocardiogram showing normal sinus rhythm at a rate of 75 beats/minute, a PR interval of 0.14 seconds, a QRS interval of 0.10 seconds, and a QRS axis of approximately 75°.

Courtesy of Ary Goldberger, MD.

Graphic 76183 Version 4.0

ECG LAD deep T-wave (wellens) inversion pattern



Courtesy of Dr. Ary Goldberger.

Graphic 67865 Version 4.0

Causes of ST-segment elevation

Myocardial ischemia/infarction

Noninfarction, transmural ischemia (eg vasospastic angina and takotsubo cardiomyopathy)

Acute MI especially due to acute epicardial coronary occlusion; may be due to takotsubo cardiomyopathy and other nonatherosclerotic coronary occlusion or severe hypoperfusion syndromes

Post-MI (ventricular aneurysm pattern)

Acute pericarditis

Abnormal early repolarization syndromes

Normal variants (including benign early repolarization)

Left ventricular hypertrophy or left bundle branch block (V1 to V2 or V3)

Other

Myocarditis (including COVID-19 infections)

Massive pulmonary embolism (leads V1 to V2 in occasional cases)

Brugada-type patterns (V1 to V3 with right bundle branch block-appearing morphology)

Myocardial tumor

Myocardial trauma

Hyperkalemia (only leads V1 and V2)

Hypothermia (J wave/Osborn wave)

Hypercalcemia (rarely)

Post-DC cardioversion (rarely)

Neurogenic stress cardiomyopathy

MI: myocardial infarction; COVID-19: coronavirus disease 2019; DC: direct current.

Graphic 81757 Version 8.0

Electrocardiogram (ECG) in early repolarization



J point and ST segment elevation is most prominent in V4 to V6. The ST segment maintains its normal configuration and is slightly concave.

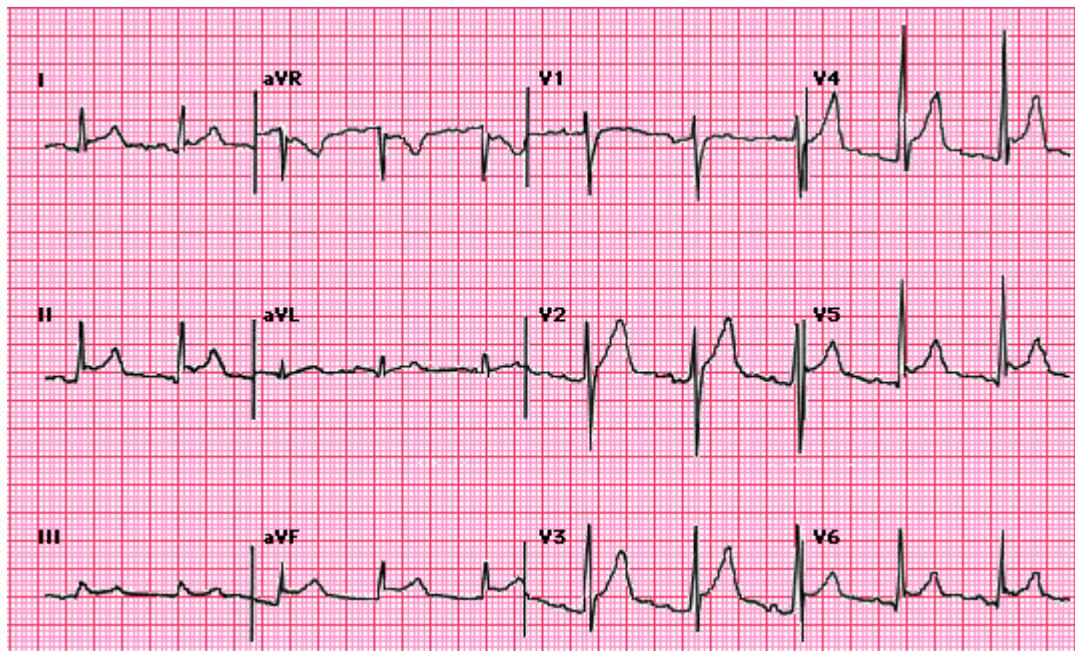
Graphic 79999 Version 3.0

Electrocardiogram features of acute pericarditis versus acute myocardial infarction

ECG features	Findings in acute pericarditis	Findings in acute MI
ST-segment elevation morphology	<ul style="list-style-type: none">▪ ST-segment elevation begins at J point, rarely exceeds 5 mm, normal concavity	<ul style="list-style-type: none">▪ ST-segment elevation begins at J point, often exceeds 5 mm in height, abnormal concavity (convex or "dome-shaped")
ST-segment elevation distribution	<ul style="list-style-type: none">▪ Widespread ST-segment elevation in most/all leads▪ Typically most prominent in inferolateral leads	<ul style="list-style-type: none">▪ Anatomical groupings of lead show ST-segment elevation, which corresponds to vascular territory of infarction
Reciprocal ST-segment changes	<ul style="list-style-type: none">▪ Usually not seen	<ul style="list-style-type: none">▪ ST-segment depressions usually seen in reciprocal leads
Concurrent ST elevation and T-wave inversion	<ul style="list-style-type: none">▪ Unusual unless concomitant myocarditis	<ul style="list-style-type: none">▪ Common
PR segment changes	<ul style="list-style-type: none">▪ PR elevation in aVR▪ PR depression in most/all other leads	<ul style="list-style-type: none">▪ Rare
Hyperacute T waves	<ul style="list-style-type: none">▪ Rare; if seen, due to fusion of elevated ST segment and T wave	<ul style="list-style-type: none">▪ Commonly seen at onset of acute infarction/ischemia
Q waves	<ul style="list-style-type: none">▪ Not usually new from acute pericarditis	<ul style="list-style-type: none">▪ Seen late in course of MI due to transmural injury
QT prolongation	<ul style="list-style-type: none">▪ Unusual	<ul style="list-style-type: none">▪ Can be seen

ECG: electrocardiogram; MI: myocardial infarction.

Electrocardiogram (ECG) in pericarditis

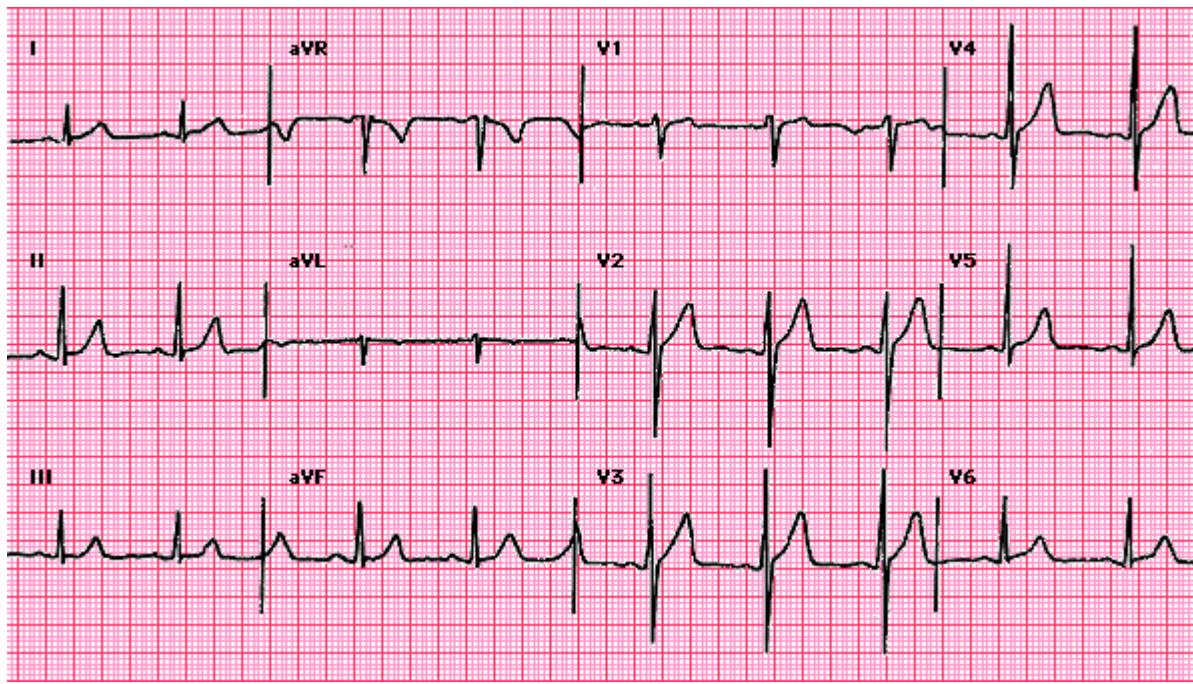


Electrocardiogram in acute pericarditis showing diffuse upsloping (concave up) ST-segment elevations seen best here in leads II, III, aVF, and V2 to V6. There is also subtle PR-segment deviation (positive in aVR, negative in most other leads). ST-segment elevation is due to a ventricular current of injury associated with epicardial inflammation; similarly, the PR-segment changes are due to an atrial current of injury, which, in pericarditis, typically displaces the PR segment upward in lead aVR and downward in most other leads.

Courtesy of Ary Goldberger, MD.

Graphic 77572 Version 5.0

Normal ECG

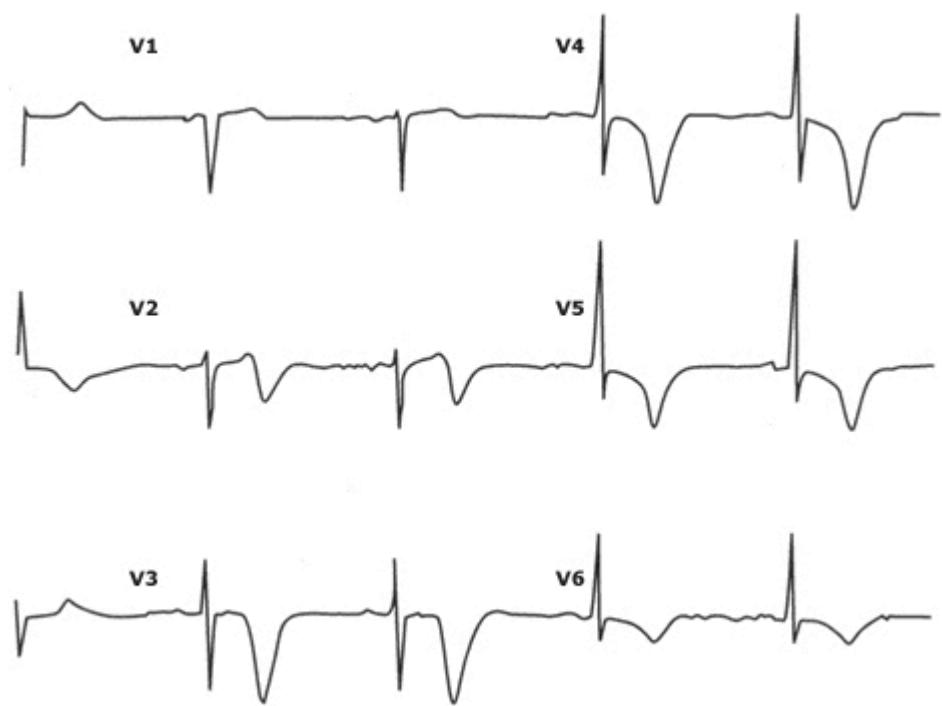


Normal electrocardiogram showing normal sinus rhythm at a rate of 75 beats/minute, a PR interval of 0.14 seconds, a QRS interval of 0.10 seconds, and a QRS axis of approximately 75°.

Courtesy of Ary Goldberger, MD.

Graphic 76183 Version 4.0

T wave abnormalities following stroke



Inverted, symmetric T waves are seen on the EKG of a patient with subarachnoid hemorrhage. Normal serum cardiac enzymes concentrations as well as subsequent normalization of the T waves attest to the neurogenic origin of the EKG pattern.

Graphic 56660 Version 3.0

Causes of Q waves on the electrocardiogram

Physiologic or positional factors

Normal variant "septal" q waves

Normal variant Q waves in leads V1,V2, aVL, III, and aVF

Left pneumothorax or dextrocardia: loss of lateral precordial R wave progression

Myocardial injury or infiltration

Acute processes: myocardial ischemia or infarction, myocarditis, hyperkalemia

Chronic processes: myocardial infarction, idiopathic cardiomyopathy, myocarditis, amyloidosis, tumor, sarcoid, scleroderma, Chagas disease, echinococcus cyst

Ventricular hypertrophy or enlargement

Left ventricle: slow R wave progression in which there are small or absent R waves in the mid-precordial leads

Right ventricle: reversed R wave progression in which there is a progressive decrease in R wave amplitude from V1 to the mid-lateral precordial leads, or slow R wave progression, particularly with chronic obstructive lung disease or acute pulmonary embolism

Hypertrophic cardiomyopathy - may simulate anterior, inferior, posterior, or lateral infarcts

Conduction abnormalities

Left bundle branch block - slow R wave progression in which there are small or absent R waves in the mid-precordial leads

Wolff-Parkinson-White patterns

