ST Segment and T Wave Abnormalities Not Caused by Acute Coronary Syndromes

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The evaluation of the chest pain patient suspected of acute coronary syndrome (ACS) represents the major indication for electrocardiograph (ECG) performance in the emergency department (ED) and prehospital settings [1]. The ECG demonstrates significant abnormality in a minority of these patients, ranging from minimal nonspecific ST segment/T wave changes to pronounced STE and T wave abnormalities, including the prominent T wave, the inverted T wave, and the nonspecific T wave (Figs. 1 and 2). The ECG syndromes responsible for these various abnormalities include potentially malignant entities, such as ACS and cardiomyopathy, and less concerning patterns, such as benign early repolarization (BER) or ventricular paced rhythms (VPR) [2–4].

In a study considering all chest pain patients with electrocardiographic ST segment depression (STD), the following clinical syndromes were responsible for the ECG abnormality: ACS, 26%; left ventricular hypertrophy (LVH), 43%; bundle branch block (BBB), 21%; VPR, 5%; left ventricular aneurysm, 3%; and other patterns, 1% [5]. Similarly, STE is a fairly common finding on the ECG of the chest pain patient and frequently does not indicate STE acute myocardial infarction (AMI). One prehospital study of adult chest pain patients revealed that, of patients manifesting STE who met criteria for fibrinolysis, most were not diagnosed with AMI; rather, LVH and left BBB were found more frequently [6]. Furthermore, in two reviews of adult ED chest pain patients with STE on ECG, the ST segment abnormality resulted from AMI in only 15%–31% of these populations; LVH, seen in 28%–30% of these patients, was a frequent cause of this STE. Other findings responsible for this STE included BER, acute
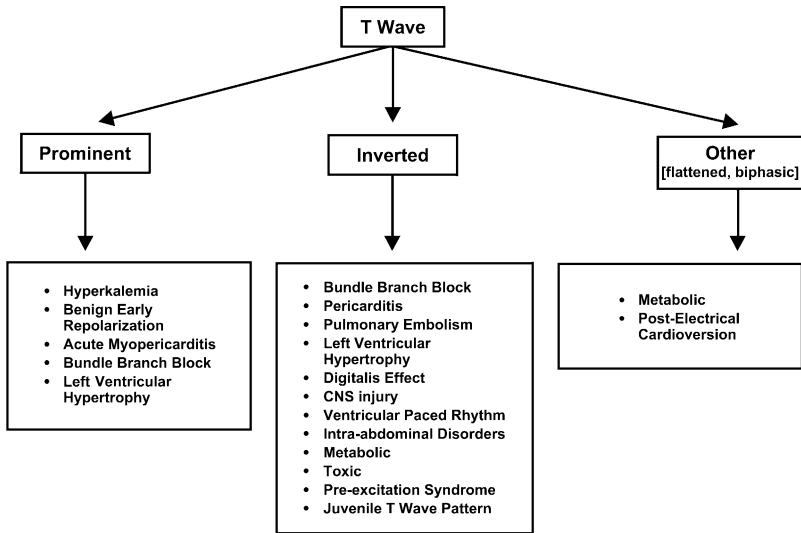
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Fig. 1. Electrocardiographic differential diagnosis of T wave abnormalities—prominent, inverted, and nonspecific in non-ACS syndromes.

myopericarditis, BBB, VPR, and ventricular aneurysm [7,8]. In a critical care unit setting, Miller et al [9] showed that STE was noted frequently, yet was responsible for AMI in only 50% of patients.

This article discusses the non-ACS causes of ST segment/T wave abnormalities, highlighting differentiation from STE associated with ACS.

Benign early repolarization

BER is a normal electrocardiographic variant with no known association with cardiac dysfunction or disease. BER describes a pattern of STE with prominent T waves most often seen in the precordial leads. A recent investigation demonstrated a BER prevalence of 29% among patients undergoing a screening health examination. Patients who had early repolarization were more likely to be male, were younger (less than age 40 years), and tended to be more athletically active compared with those individuals without the early repolarization pattern. The long-term health of these patients who had BER was equivalent to the control population [10]. In another large study of BER, the mean age of patients was 39 years (range, 16–80 years); although the pattern was seen across this rather broad age range, it was encountered predominantly in patients less than age 50 years and rarely seen in individuals older than age 70 years [11]. The BER pattern is seen much more often in men than in women. BER is encountered most frequently in younger black men (20–40 years of age) [12].

The electrocardiographic characterization of the BER pattern (Figs. 3–5) includes the following features: STE [1]; concavity of the initial, upsloping

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                          ┌──────────────┐
                          │  ST Segment  │
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                 ┌──────────┐        ┌───────────┐
                 │ Elevated │        │ Depressed │
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┌───┐   ┌──┐
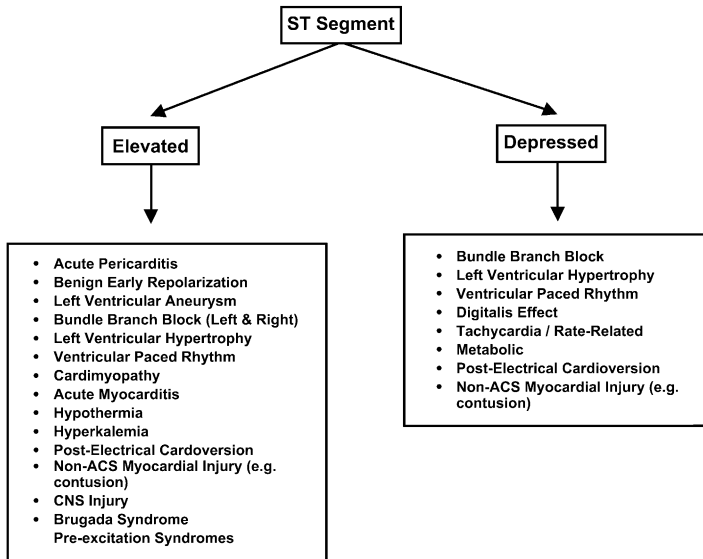| • **Acute Pericarditis** | | • **Bundle Branch Block** |
• **Benign Early Repolarization**		• **Left Ventricular Hypertrophy**
• **Left Ventricular Aneurysm**		• **Ventricular Paced Rhythm**
• **Bundle Branch Block (Left & Right)**		• **Digitalis Effect**
• **Left Ventricular Hypertrophy**		• **Tachycardia / Rate-Related**
• **Ventricular Paced Rhythm**		• **Metabolic**
• **Cardimyopathy**		• **Post-Electrical Cardioversion**
• **Acute Myocarditis**		• **Non-ACS Myocardial Injury (e.g.**
• **Hypothermia**		**contusion)**
• **Hyperkalemia**		
• **Post-Electrical Cardoversion**		
• **Non-ACS Myocardial Injury (e.g.**		
contusion)		
• **CNS Injury**		
• **Brugada Syndrome**		
Pre-excitation Syndromes		

Fig. 2. Electrocardiographic differential diagnosis of ST segment elevation and depression in non-ACS syndromes.

portion of the ST segment [2]; notching or slurring of the J point [3]; symmetric, concordant, prominent T waves [4]; widespread distribution of the electrocardiographic abnormalities [5]; and temporal stability [6,13,14].

In the normal state, the ST segment is neither elevated nor depressed; it is located at the isoelectric baseline as defined by the TP segment. The ST segment itself begins at the J or juncture point. The ST segment is elevated in the BER pattern, usually less than 3.5 mm. The contour of the elevated ST segment is an important characteristic of the pattern; the ST segment seems to have been lifted off the baseline starting at the J point (Figs. 3–5). The normal concavity of the initial, upsloping portion of the ST segment is preserved. Eighty percent to 90% of individuals demonstrate STE less than 2 mm in the precordial leads and less than 0.5 mm in the limb leads; only 2% of cases of BER manifest STE greater than 5 mm [13,14]. In the BER pattern, the J point itself frequently is notched or irregular. This finding, although not diagnostic of BER, is highly suggestive of the diagnosis [11,13,15].

Prominent T waves also are encountered (see Figs. 3 and 4). These T wave are often of large amplitude and slightly asymmetric morphology. The T waves are concordant with the QRS complex (ie, oriented in the same direction as the major portion of the QRS complex) and usually are found in the precordial leads. The height of the T waves in BER ranges from approximately 6 mm in the precordial leads to 4–6 mm in the limb leads [11,13,16].

[1] ST segment elevation
[2] upward concavity of initial portion of ST segment
[3] notching or slurring of terminal QRS complex
[4] slightly asymmetric, concordant T waves of large amplitude
[5] widespread or diffuse distribution of ST segment elevation
[6] relative temporal stability

Fig. 3. ECG criteria for benign early repolarization.

These abnormalities are greatest in the precordial leads, particularly the precordial leads (leads V2–V5). STE in the limb leads, if present, is usually less pronounced. In fact, this isolated STE in the limb leads is seen in less than 10% of BER cases and should prompt consideration of another explanation for the observed ST segment abnormality, such as AMI. The T waves tend to follow the QRS complex in the BER pattern; essentially, pronounced STE usually is associated with prominent T waves in the same distribution.

Acute myopericarditis

Acute pericarditis is better termed acute myopericarditis in that both the pericardium and the superficial epicardium are inflamed. This epicardial inflammation produces the ST segment and related electrocardiographic changes; the pericardial membrane is electrically silent in a direct effect on the ST segment and T wave.
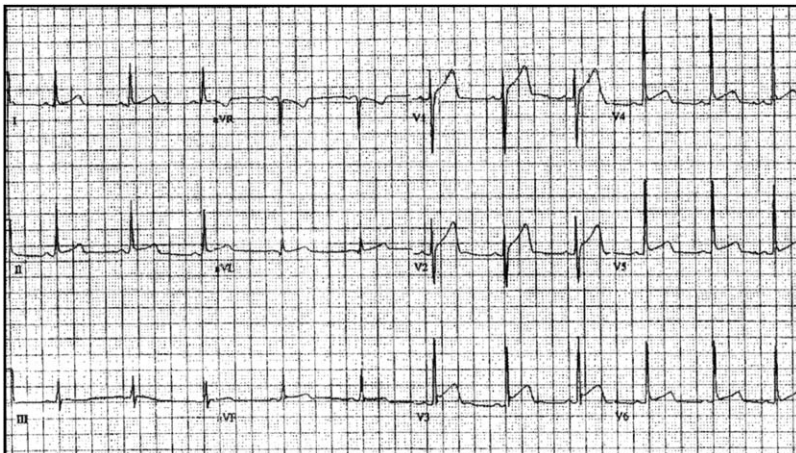

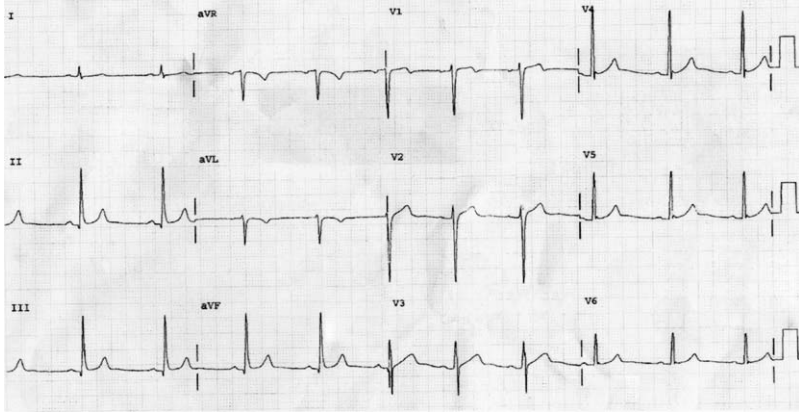Fig. 4. Benign early repolarization.

Fig. 5. Benign early repolarization.

The electrocardiographic abnormalities evolve through four classic stages (Fig. 6) [16]. Stage I (Figs. 6 and 7) is characterized by STE, prominent T waves, and (in most cases) PR segment depression. Stage II is characterized by a normalization of the initial abnormalities, namely a resolution of the STE. Stage III involves T wave inversion, usually in the same distribution where STE was encountered. Finally, stage IV is a normalization of all changes with a return to the baseline ECG. Persistent STE and pathologic Q waves are not encountered in patients who have myopericarditis—these electrocardiographic findings suggest another etiology.

These electrocardiographic stages usually occur in an unpredictable manner. In a general sense, stages I through III develop over hours to days. Conversely, changes related to stage IV myopericarditis may not develop for many days to many weeks. Furthermore, patients may not manifest all characteristic features. Finally, patients may present for medical care at a later stage of the process; for instance, the patient may present after a delay of

[1] ECG stages
    [a] stage 1 – ST segment elevation &
          PR segment changes
    [b] stage 2 – ST segment normalization
    [c] stage 3 – T wave inversion
    [d] stage 4 – normalization
[2] ST segment elevation (stage1)
    [a] upward concavity of initial
          ST segment < 5 mm
    [b] widespread
[3] PR segment (stage 1)
    [a] depressed in inferior & lateral leads
    [b] "reciprocal" elevation in lead aVr
[4] T wave (stage 1)
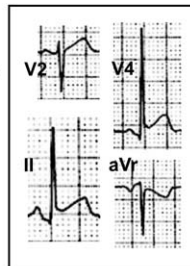   [a] often prominent, particularly in precordial leads
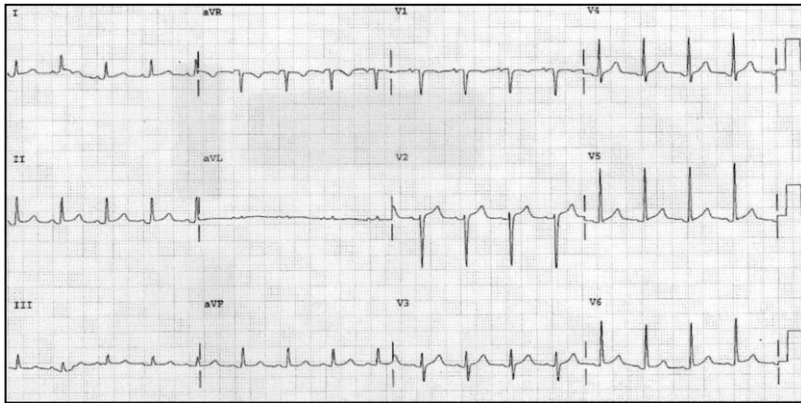
Fig. 6. ECG criteria for myopericarditis.

Fig. 7. Myopericarditis.

a week or more with chest discomfort and manifest electrocardiographic T wave inversion—the clinician having "missed" the STE.

Stage I abnormality—that is, STE, prominent T wave, and PR segment depression—is often electrocardiographically obvious with STE the most prominent electrocardiographic feature (Figs. 6 and 7). The magnitude of elevation usually ranges from 2–4 mm, with greater than 5 mm unusual for myopericarditis. The morphology of the elevated ST segment is most frequently concave in shape. In other cases, STE can also be obliquely flat or convex in contour; these morphologies, however, are suggestive of AMI [7]. STE resulting from myopericarditis is usually widespread, noted in the following electrocardiographic leads: I, II, III, aVL, aVF, and V2–V6—essentially all leads except the more rightward-oriented leads aVR and V1; reciprocal ST segment depression is seen in lead aVR and occasionally in lead V1. The STE is seen most often in many leads simultaneously, though it may be limited to a specific anatomic segment if the process is focal; if focal inflammation is present, the inferior wall most often is involved.

PR segment abnormality (Figs. 6 and 7) resulting from atrial inflammation and irritation is a highly suggestive feature of stage I myopericarditis. PR segment depression is described as "almost diagnostic" [16] and is best observed in the lateral precordial (V5 and V6) and inferior (II, III, and aVF) leads. Reciprocal PR segment elevation is seen in lead aVR; in many cases, this finding is in fact more obvious to the clinician compared with PR segment depression [17,18].

T wave inversion, a stage III feature, is usually transient and most often occurs in leads that had recently manifested stage I STE. The magnitude and morphology of the inverted T wave are nonspecific. The inverted T waves are usually of normal amplitude with symmetric initial (downsloping) and final (upsloping) limbs, which can be confused with an ACS presentation.

Additional electrocardiographic findings may be noted in the patient who presents with diseases associated with pericarditis: myocarditis and pericardial effusion. Myocarditis may manifest Q waves, bundle branch block, and dysrhythmias (Figs. 8 and 9). Electrocardiographic changes suggestive of pericardial effusion include widespread low voltage (resulting from increased resistance to injury current flow with the accumulated fluid) and electrical alternans (a beat-to-beat alteration in QRS complex size caused by shifting of the heart within the fluid-filled pericardium).

Left ventricular hypertrophy

In patients who have the electrocardiographic LVH pattern, ST segment/ T wave changes are encountered in approximately 70% of cases; these changes result from altered repolarization of the ventricular myocardium caused by LVH [16,19] and are collectively and incorrectly referred to as the strain pattern The electrocardiographic abnormalities seen in this scenario most often involve the ST segment and T wave. ST segment abnormalities (depression and elevation) and T wave changes (prominence or inversion) are encountered. These ST segment/T wave abnormalities are the new norm in many patients who have the electrocardiographic LVH pattern. In a prehospital setting, most chest pain patients manifesting electrocardiographic STE did not have AMI as a final hospital diagnosis; rather, LVH accounted for a significant portion of these patients [6]. The ED population demonstrates a similar trend [7,8]. Furthermore, Larsen and colleagues have shown that the electrocardiographic pattern consistent with LVH is encountered in approximately 10% of adult chest pain patients initially diagnosed in the ED with ACS, of whom only one quarter were found to have ACS. In this study, clinicians frequently attributed the ST segment/ T wave changes seen to ACS, when in fact the observed changes resulted from repolarization abnormality associated with LVH pattern [20].

LVH is associated with poor R wave progression and loss of the septal R wave in the right to mid precordial leads, most commonly producing a QS pattern. In general, these QS complexes are located in leads V1 and V2, rarely extending beyond lead V3. STE is encountered in this distribution, together with prominent T waves. The STE seen in this distribution may be

[1] ST segment
   [a] depression
   [b] elevation
[2] other findings
   [a] T wave inversion
   [b] Q wave
   [c] brady- & tachydysrhythmia
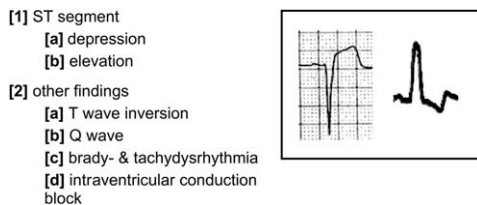   [d] intraventricular conduction block

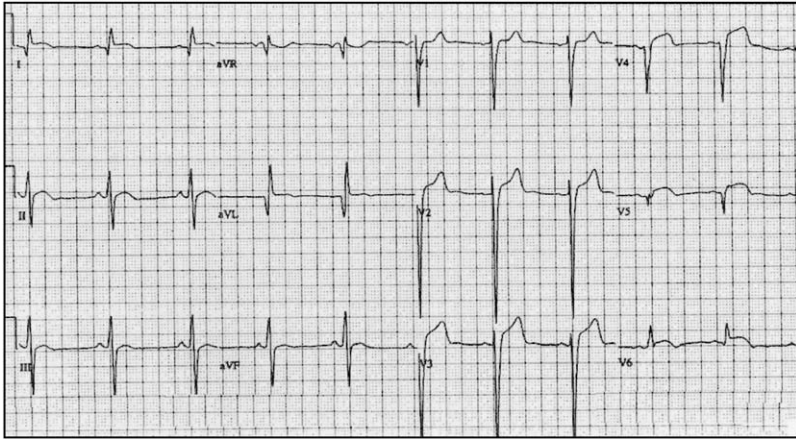Fig. 8. ECG findings for acute myocarditis.

Fig. 9. Acute myocarditis.

greater than 5 mm in height. The initial, upsloping portion of the ST segment/T wave complex is frequently concave in LVH compared with the flattened or convex pattern observed with AMI. This morphologic feature is imperfect; early AMI may reveal such a concave feature (Figs. 10 and 11) [7].

Leftward-oriented leads I, aVL, V5, and V6 frequently demonstrate large, monophasic R waves; these leads typically reveal STD with inverted T waves. This ST segment/T wave complex has been described in the following manner: initially bowed upward (convex upward) followed by a gradual downward sloping into an inverted, asymmetric T wave with an abrupt return to the baseline [21]. The T wave, however, may assume other morphologies, including minimally inverted or inversion greater than 5 mm. These T wave abnormalities also may be encountered in patients lacking prominent

[1] voltage criteria for LVH *
   [a] S (V1 or V2) + R (V5 or V6) > 35 mm
   [b] age > 35 years
[2] poor precordial R wave progression
   [a] QS complex (V1 - V3)
   [b] transition complex (V2 - V4)
   [c] RS or R wave (V4 - V6)
[3] leads V1 - V3
   [a] ST segment elevation
   [b] upward concavity of initial
      ST segment ~ 5 mm
   [c] prominent upright T wave
[4] leads V4 – V6 (ST segment – T wave complex)
   [a] initially convex upward
   [b] followed by gradual downward sloping into inverted,
      asymmetric T wave
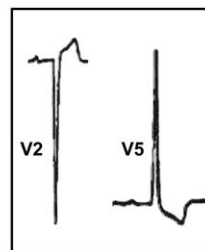   [c] with abrupt return to baseline

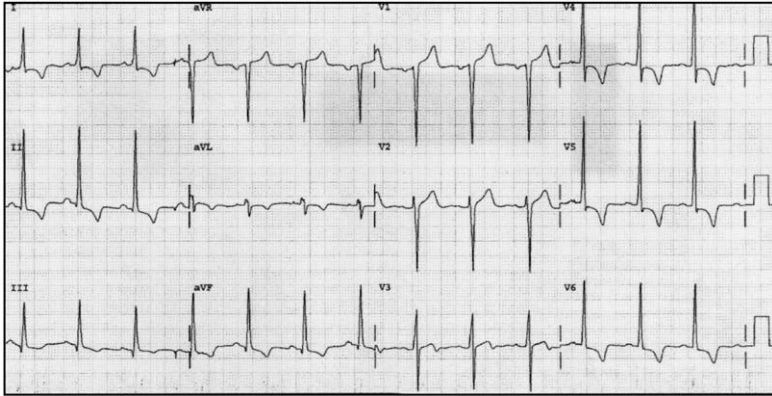Fig. 10. ECG criteria for left ventricular hypertrophy.

Fig. 11. Left ventricular hypertrophy.

QRS voltage (ie, large S and R waves) typical of LVH [16,19]. Other features of this portion of the ST segment/T wave complex suggestive of LVH-related change include the following: (1) J point depression, (2) T wave asymmetry with rapid return to the baseline, (3) "overshoot" of the terminal T wave at the baseline (terminal positivity), (4) T wave inversions in leads V4, V5, and V6 with the inversion greatest in lead V6, and (5) prominent T wave inversion in lead V6 (greater than 3 mm) (Figs. 10 and 11).

Bundle branch block

Unlike left bundle branch block (LBBB), right bundle branch block (RBBB) does not obscure the electrocardiographic diagnosis of ACS. In BBB, the QRS complex duration is prolonged—greater than 0.12 seconds. Perhaps the most obvious and distinctive electrocardiographic feature in RBBB is a prominent R wave in lead V1. This R wave is broad and may assume any of several morphologies: monophasic R wave, biphasic rSR', or qR formation. In lead V6, a wide RS wave is seen (Figs. 12 and 13).

Significant ST segment/T wave changes are encountered in the patient who has uncomplicated BBB [16]. In general, the correct and appropriate position of the ST segment/T wave complex is dictated by the major, terminal portion of the QRS complex—the rule of appropriate discordance. Using this concept, the ST segment/T wave complex is located on the opposite side of the isoelectric baseline from the major, terminal portion of the QRS complex. As such, leads with predominantly positive QRS complex would present with STD and T wave inversion—discordant STD and T wave inversion. Conversely, a primarily negative QRS complex would be associated with STE and prominent, upright T wave—discordant STE. This concept holds true not only for right and left BBB, but also for VPR, and, to a lesser extent, LVH.

[1] RBBB criteria
    [a] QRS complex > 0.12 sec
    [b] R complex (V1 & V2)
    [c] R, Rs, QS or Qr complex (V5, & V6)
[2] leads V1 – V2 (ST segment – T wave complex)
    [a] depressed ST segment
    [b] inverted T wave
    [c] gradual downward sloping into inverted,
        asymmetric T wave with abrupt
        return to baseline
[3] leads V5, & V6
    [a] variable dependent upon QRS complex configuration
    [b] ST segment – T wave directed opposite of major, terminal portion
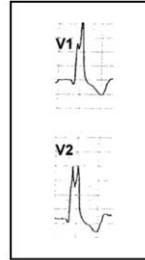   of QRS complex

Fig. 12. ECG criteria for right bundle branch block.

LBBB is found commonly among ED chest pain patients, which is unfortunate for several reasons: (1) LBBB is a marker of significant heart disease with extreme risk for acute cardiovascular complication and death in patients who have ACS, (2) LBBB, if new or presumably new and occurring within an appropriate clinical context suggesting AMI, represents an indication for fibrinolysis, and (3) LBBB markedly reduces the diagnostic power of the ECG in the evaluation of potential AMI.

As with RBBB, the rule of appropriate discordance predicts the normal ST segment/T wave findings in LBBB. In the right precordial leads (leads V1 and V2), broad, mainly negative QS or rS complexes are found (Figs. 14 and 15). In these leads, STE with a prominent T wave is seen. The STE in these leads ranges from minimal (1–2 mm) to prominent (>5 mm), although STE >5mm in these leads should spark consideration of AMI [22]. Moving from the right to left precordial leads, poor R wave progression or QS complexes are noted, rarely extending beyond leads V4 or V5. In leads V5 and V6, a positive, monophasic R wave is encountered; the ST segment is depressed in these leads, whereas the T wave is inverted. Similar morphologies are found in leads I and aVL (Figs. 14 and 15).
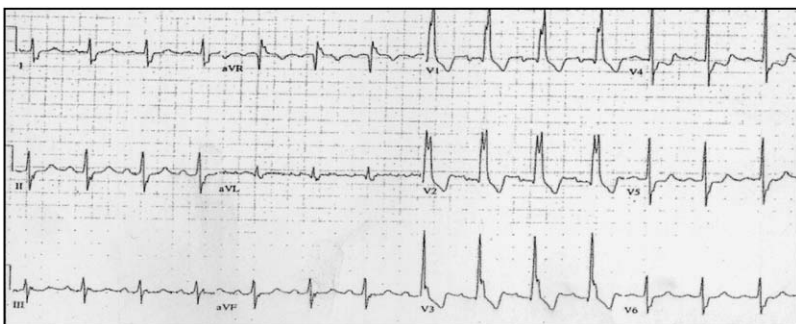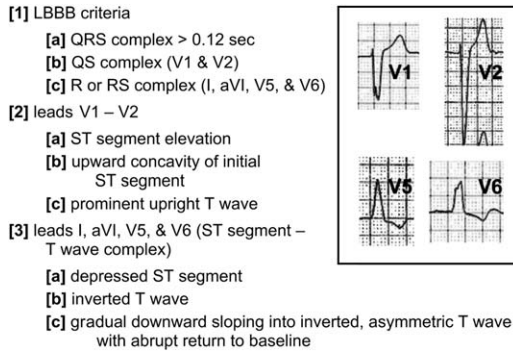
Fig. 13. Right bundle branch block.

[1] LBBB criteria
    [a] QRS complex > 0.12 sec
    [b] QS complex (V1 & V2)
    [c] R or RS complex (I, aVl, V5, & V6)
[2] leads V1 – V2
    [a] ST segment elevation
    [b] upward concavity of initial
       ST segment
    [c] prominent upright T wave
[3] leads I, aVl, V5, & V6 (ST segment –
    T wave complex)
    [a] depressed ST segment
    [b] inverted T wave
    [c] gradual downward sloping into inverted, asymmetric T wave
       with abrupt return to baseline

Fig. 14. ECG criteria for left bundle branch block.

Ventricular paced rhythm

As with the LVH and LBBB patterns, right-VPR confounds the ability of the physician to detect ACS on the ECG. Right-VPR not only confounds the electrocardiographic diagnosis of ACS but also imitates ECG findings of acute coronary ischemic events. In right-VPR, the ECG displays a broad, mainly negative QRS complex with a QS configuration in leads V1 to V6; if an R wave is present, it is usually small and does not appear until the left precordial leads, resulting in poor R wave progression. A large monophasic R wave is encountered in leads I and aVL and, on occasion, in leads V5 and V6. QS complexes also may be encountered in the inferior leads (Figs. 16–18).

The anticipated or expected ST segment/T wave configurations are discordant and directed opposite from the terminal portion of the QRS complex—the rule of appropriate discordance—similar to the
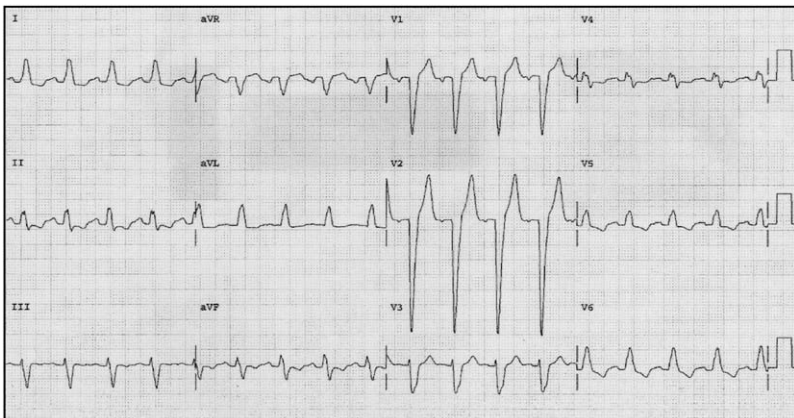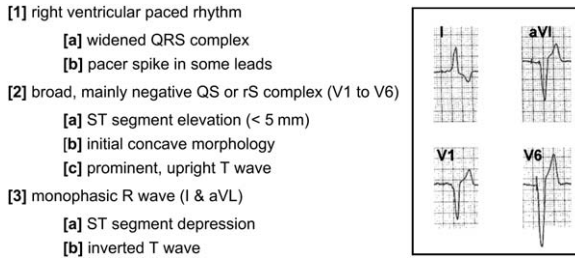
Fig. 15. Left bundle branch block.

[1] right ventricular paced rhythm
    [a] widened QRS complex
    [b] pacer spike in some leads
[2] broad, mainly negative QS or rS complex (V1 to V6)
    [a] ST segment elevation (< 5 mm)
    [b] initial concave morphology
    [c] prominent, upright T wave
[3] monophasic R wave (I & aVL)
    [a] ST segment depression
    [b] inverted T wave

Fig. 16. ECG criteria for right ventricular paced rhythm.

electrocardiographic principles applied to BBB [16,23]. Accordingly, leads with QS complexes demonstrate STE with an upright T wave; this STE is seen in leads II, III, and aVF as well as leads V1 to V6, depending on the positioning of the pacemaker electrode. Leads with a large monophasic R wave demonstrate STD with T wave inversion (Figs. 16–18) [24].

Left ventricular aneurysm

One structural complication of extensive myocardial infarction is ventricular aneurysm, most often arising from the left ventricle after a large transmural infarction. The most frequent electrocardiographic manifestation of ventricular aneurysm is STE (Figs. 19 and 20). Because of the frequent anterior location of ventricular aneurysm, STE is observed most often in leads I, aVl, and V1 to V6. An inferior wall ventricular aneurysm would reveal STE in the inferior leads, though usually less pronounced than that seen with the anterior left ventricular aneurysm. The actual ST segment abnormality may manifest varying morphologies, ranging from obvious, convex STE to minimal, concave elevations.
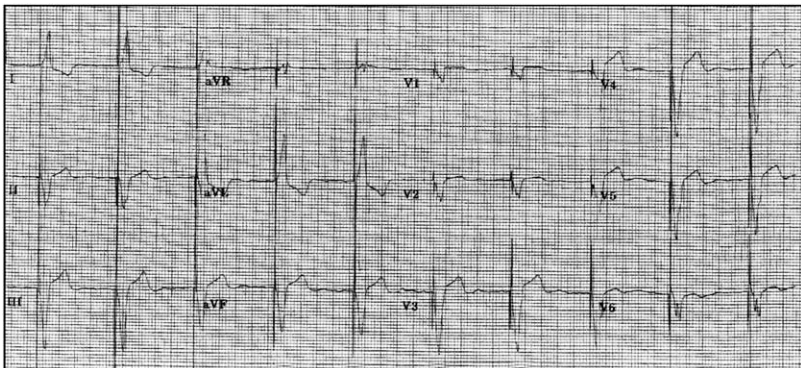

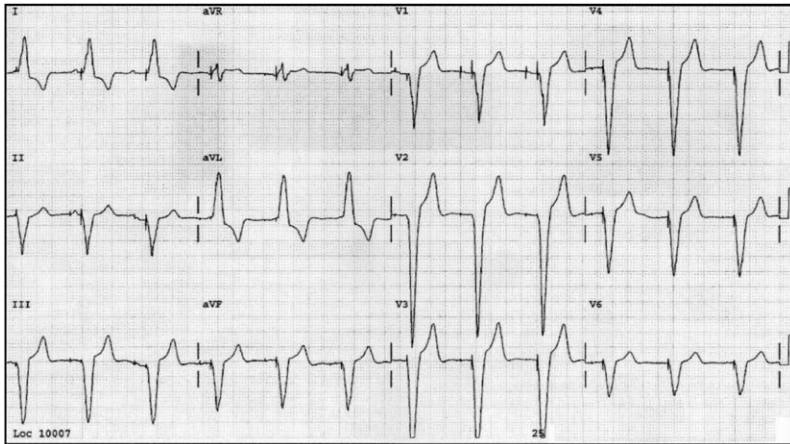Fig. 17. Right ventricular paced rhythm, with ventricular pacing spikes.

Fig. 18. Right ventricular paced rhythm with atrial and ventricular pacing spikes.

In patients who have ventricular aneurysm, significant Q waves are observed in the same distribution as the STE. The calculation of the ratio of the amplitude of the T wave to that of the QRS complex may help distinguish anterior AMI from ventricular aneurysm. If the ratio of the amplitude of the T wave/QRS complex exceeds 0.36 in any single lead, the ECG likely reflects AMI. If this ratio is less than 0.36 in all leads, however, the findings are likely caused by ventricular aneurysm (Fig. 20) [25].

Other non-ACS causes of ST segment/T wave abnormalities

Cardiomyopathy may produce electrocardiographic patterns that simulate findings associated with acute and chronic coronary syndromes [16]. These findings include significant Q waves, ST segment changes, T wave

[1] past MI
    [a] Q wave
    [b] poor R wave progression
[2] persistent ST segment elevation
    [a] varying magnitudes
    [b] varying morphologies
[3] variable T wave (normal to inverted)
[4] relationship of QRS complex to T wave
    [a] predominant, prominent QRS complex relative to T wave
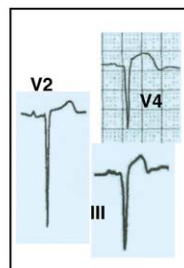   [b] T wave / QRS complex amplitude ratio < 0.36

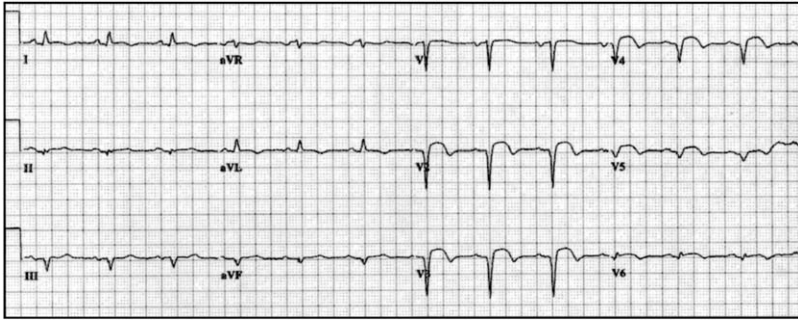Fig. 19. ECG findings for left ventricular aneurysm.

Fig. 20. Left ventricular aneurysm.

abnormalities, and BBB patterns. Cardiomyopathy may produce significant Q waves in the inferior and right precordial distributions; in certain cases, such Q waves may be seen across the precordium, involving the entire ante-rolateral region of ECG. These leads also can demonstrate STE with prominent T waves. In most instances, the STE is usually less than 5 mm in height with a concave, initial upsloping portion of the ST segment/T wave complex.

In 1992, Pedro and Josep Brugada described a new syndrome that was associated with sudden death in individuals who have a structurally normal heart and no evidence of atherosclerotic coronary disease [26]. Patients who have this syndrome were noted to have a distinct set of electrocardiographic abnormalities, characterized by an RBBB pattern with STE in the right precordial leads. Patients who have this Brugada syndrome have a tendency for sudden cardiac death resulting from polymorphic ventricular tachycardia [27]. ECG abnormalities (Fig. 21) that suggest the diagnosis include RBBB (complete or incomplete) and STE in leads V1, V2, or V3. Two types of STE (Fig. 21) morphologies have been described: convex upwards (coved) and concave upwards (saddle-type) [28–30]. The ECG morphologies may transform from one type to the other or may normalize completely.
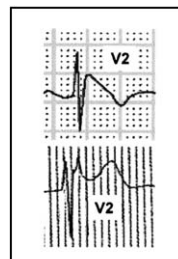

Fig. 21. ECG findings for Brugada syndrome-related ST segment/T wave abnormalities.

Left ventricular apical ballooning syndrome, also known as Takastubo syndrome, is a recently described disorder in which patients develop anginal symptoms with acute congestive heart failure during times of stress. At cardiac catheterization, these patients are found to have abnormal left ventricular function but normal coronary arteries [31,32]. Classic electrocardiographic findings (Fig. 22) encountered in this syndrome include STE, T wave inversion, and abnormal Q waves [33]. These findings are most often transient, presenting only when the patient is symptomatic and resolving during physiologically normal periods. The STE itself has a similar morphology to that seen in the patient who has AMI [33].

At therapeutic levels, digitalis produces characteristic electrocardiographic changes, referred to as the digitalis effect. The electrocardiographic manifestations (Fig. 23) of digitalis are as follows: (1) "scooped" STD, most prominent in the inferior and precordial leads (those with the largest R wave) and usually absent in the rightward leads; (2) flattened T waves; (3) increased U waves; and (4) shortening of the QT interval.

The earliest sign of hyperkalemia is the appearance of tall, symmetric T waves, described as hyperacute, which may be confused with the hyperacute T wave of early STE AMI. As the serum potassium level increases, the T waves tend to become taller, peaked, and narrowed in a symmetric fashion in the anterior distribution (Fig. 24). Hyperkalemic 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 (Fig. 24). Conversely, the hyperacute T waves of early AMI are often asymmetric with a broad base. As the serum level continues to increase, the QRS complex widens (Fig. 24), which can make the ST segment seem elevated. This pseudo-STE associated with hyperkalemia is characterized by J point elevation and prominent T waves.

The Wolff Parkinson White syndrome (WPW) frequently presents with evidence of ventricular pre-excitation or actual dysrhythmic events. Such evidence of pre-excitation includes the classic electrocardiographic triad of PR interval shortening, a delta wave, and QRS complex widening. The patient

[1] ST segment elevation
[2] T wave inversion
[3] pathologic Q waves

Fig. 22. ECG findings for Takastubo cardiomyopathy.

[1] ST segment
    [a] depression
    [b] "scooped" ST segment
        depression
    [c] most prominent in inferior
        & precordial leads
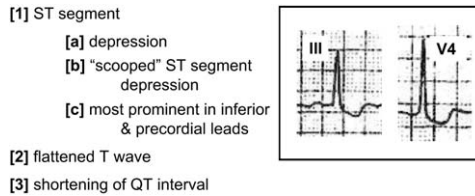[2] flattened T wave
[3] shortening of QT interval

Fig. 23. ECG Findings for digoxin effect.

may present with paroxysmal supraventricular tachycardia that does not reflect the underlying WPW syndrome, rapid, bizarre atrial fibrillation, broad-complex tachycardia, or sudden cardiac death. Various pseudoinfarction findings may be observed that, if not recognized, may once again lead the uninformed clinician to the wrong diagnostic conclusion. Q waves can be seen in leads II, III, and aVF, mimicking past inferior myocardial infarction, and tall R waves in the right precordial leads are suggestive of a posterior wall AMI. T wave inversions can be seen in leads with prominent R waves (Fig. 25).

With the development of hypothermia, the ECG manifests numerous ECG abnormalities, particularly an unusual form of STE (Fig. 26). This hypothermia-related electrocardiographic change involves the juncture between the terminal portion of the QRS complex and the initial ST segment—the J point. The J point itself and the immediately adjacent ST segment seem to have lifted unevenly off the isoelectric baseline, producing the J wave (also know as the Osborn J wave). In general, the amplitude of the J wave is directly proportional to the degree of hypothermia. Other electrocardiographic features associated with hypothermia include (1) bradycardia, (2) tremor artifact, (3) prolongation of the PR and QT intervals, (4) T wave inversions in leads with preeminent J waves, and (5) dysrhythmias such as atrial fibrillation and ventricular fibrillation (Fig. 26).
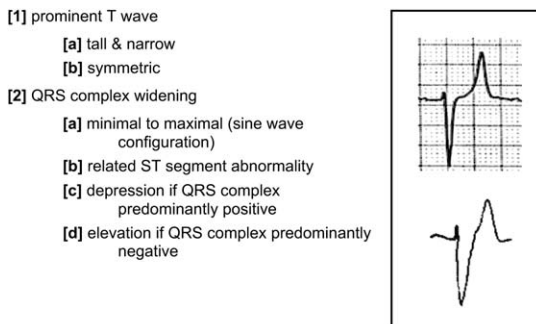
[1] prominent T wave
    [a] tall & narrow
    [b] symmetric
[2] QRS complex widening
    [a] minimal to maximal (sine wave
        configuration)
    [b] related ST segment abnormality
    [c] depression if QRS complex
        predominantly positive
    [d] elevation if QRS complex predominantly
        negative

Fig. 24. ECG findings for hyperkalemia-related ST segment/T wave abnormalities.

[1] ventricular pre-excitation syndrome
    [a] shortened PR interval
    [b] delta wave
    [c] widened QRS complex
[2] T wave inversion
[3] ST segment
    [a] depression
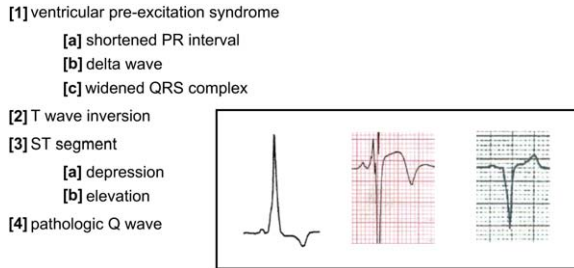   [b] elevation
[4] pathologic Q wave

Fig. 25. ECG findings of Wolff-Parkinson-White syndrome-related ST segment/T wave abnormalities.

Central nervous system events such as subarachnoid and intraparenchymal hemorrhage can present with ST segment/T wave abnormalities. These CNS disasters may manifest electrocardiographic abnormality, most often significant T wave inversion in the precordial leads; other electrocardiographic abnormalities such as STD and STE, however, also are seen.

ST segment/T wave abnormalities—electrocardiographic distinction from acute coronary syndrome

If the ECG reveals ST segment or T wave changes (Fig. 27), the clinician then is confronted with the challenge of identifying the source of the electrocardiographic abnormality. In addition to clinical correlation, specific attributes of these ECG findings aid in reaching a diagnosis. QRS complex magnitude (a criterion for LVH pattern diagnosis) and the width (a criterion for BBB or VPR) should be considered first. If either of these abnormalities is present, a confounding electrocardiographic pattern is present, complicating the analysis. Furthermore, the presence of one of these patterns serves to forewarn that ST segment or T wave abnormalities will be encountered;

[1] J wave or Osborn wave
    [a] positive deflection at
        J point
    [b] associated ST segment
        elevation
[2] other findings
    [a] bradydysrhythmia
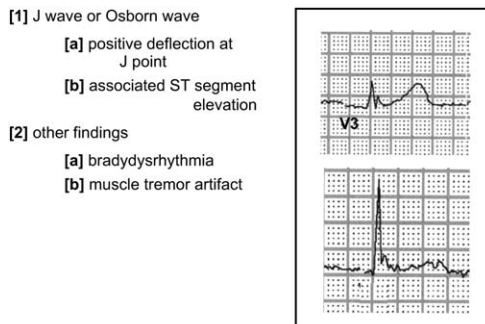   [b] muscle tremor artifact

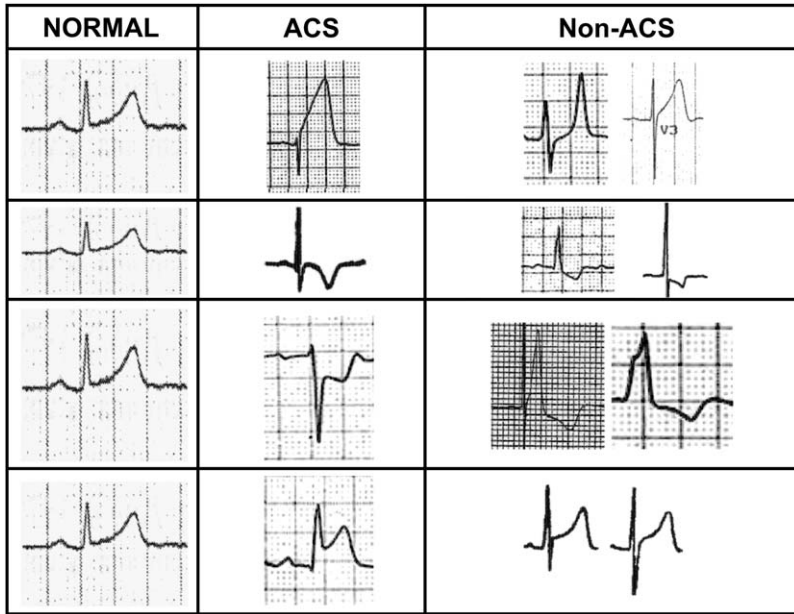Fig. 26. ECG findings for hypothermia.

Fig. 27. Specific ECG findings (prominent T wave, T wave inversion, ST segment depression, and ST segment elevation) in normal, ACS, and non-ACS presentations.

these abnormalities can represent the normal findings associated with these patterns or, alternatively, ACS changes superimposed on the confounding pattern.

If a confounding pattern is not seen, the ST segment should be scrutinized, considering the presence of either STE or STD. From the anatomic perspective, the location of the elevation is suggestive of the electrocardiographic diagnosis in two circumstances. First, widespread anterior STE most often is caused by a non-AMI process, including LVH, BBB, and BER [34]. These patterns, when considered as a whole, are encountered much more frequently than AMI occurring in the anterior area [34].

Second and perhaps more important, inferior STE, particularly when isolated, results from AMI in most instances [34]. Again, the most commonly encountered non-infarction causes of STE usually have electrocardiographically widespread or diffuse STE [23,35,36]. Isolated STE is a rare finding in BER [35], whereas isolated STE is not found in LVH and BBB presentations [23,35]. Similarly, lateral wall STE is an electrocardiographic finding more often the result of AMI [34].

The morphology of the elevated ST segment is a predictor of etiology. The use of ST segment waveform analysis has been reported as a useful adjunct in establishing the electrocardiographic diagnosis of AMI [7]. Using this analysis, a concave ST segment pattern is seen significantly more often in the non-AMI patient, whereas the non-concave morphology is seen

almost exclusively in the patient who has AMI. As with most guidelines, it is not infallible; patients who have STE caused by AMI may demonstrate concavity of this portion of the waveform.

STD can occur as the sole finding on the ECG or as a component of a more complicated electrocardiographic presentation. If present as the major electrocardiographic abnormality, the morphology of STD is an important consideration. Horizontal (flat) or downsloping ST segment depression is associated more often with ACS, although nonischemic causes of STD also may present with similar morphologies [37]. Reciprocal STD is defined as depression in leads anatomically opposite, or near-opposite, to those with STE (eg, STD in lead aVL in inferior STE AMI). Its presence on the ECG supports the diagnosis of AMI with high sensitivity, and positive predictive values greater than 90%. The use of reciprocal change in prehospital and ED chest pain patients retrospectively increased the diagnostic accuracy in the electrocardiographic recognition of AMI [6]. It is perhaps most useful in patients who have chest pain and STE of questionable cause. The STD occurring in an LVH, BBB, or VPR does not meet criteria for reciprocal ST segment depression.

Considering the magnitude of ST segment changes, the total amount of STE is greater in the patient who has AMI compared with the non-infarction patient. Furthermore, the total quantity of ST segment deviation, ie, the sum of STE and STD, is significantly greater in the patient who has AMI [34].

Summary

The 12-lead ECG furnishes invaluable information in patients who present with chest pain or other symptomatology suggestive of ACS. The ECG can demonstrate abnormalities in a subset of these individuals, ranging from minimal nonspecific ST segment/T wave changes to pronounced STE. The electrocardiographic differential diagnosis of these abnormalities includes not only AMI and ACS, but also BER, myopericarditis, LVH, BBB, VPR, ventricular aneurysm, and other non-ACS entities. A sound review of the 12-lead ECG, together with knowledge of the expected electrocardiographic abnormalities associated with these common diseases, assists the clinician in differentiating ACS and non-ACS ST segment and T wave abnormalities on the 12-lead ECG.

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