

ELIAS B. HANNA, MD

Assistant Professor of Medicine,
Department of Medicine, Cardiovascular Section,
Louisiana State University Health Sciences Center,
New Orleans

DAVID LUKE GLANCY, MD

Emeritus Professor of Medicine,
Department of Medicine, Cardiovascular Section,
Louisiana State University Health Sciences Center,
New Orleans

ST-segment elevation: Differential diagnosis, caveats

ABSTRACT

The differential diagnosis of ST-segment elevation includes four major processes: ST-segment elevation myocardial infarction (STEMI); early repolarization; pericarditis; and ST elevation secondary to an abnormality of the QRS complex (left bundle branch block, left ventricular hypertrophy, or preexcitation). Other processes that may be associated with ST elevation include hyperkalemia, pulmonary embolism, and Brugada syndrome. The clinical setting and specific electrocardiographic criteria often allow identification of the cause. This article reviews ST-T and QRS configurations specific to each diagnosis.

KEY POINTS

Features of STEMI: (1) ST elevation that is straight or convex upward and blends with T to form a dome; (2) wide upright T or inverted T waves; (3) Q waves; (4) ST elevation or T waves that may approximate or exceed QRS height; and (5) reciprocal ST depression.

Features of early repolarization include a notched J point and ST elevation not exceeding 3 mm.

Features of pericarditis include PR depression greater than 1 mm and ST elevation less than 5 mm.

Features of left bundle branch block, left ventricular hypertrophy, and preexcitation: both ST and T are discordant to QRS; ST elevation is less than 25% of QRS height (and less than 2.5 mm in left ventricular hypertrophy); and delta waves, short PR, and pseudo-Q waves are seen in preexcitation.

Features of hyperkalemia include narrow-based, peaked T waves “pulling” the ST segment.

doi:10.3949/ccjm.82a.14026

WHEN THE ST SEGMENT is elevated on the electrocardiogram, our first concern is whether the patient is having an ST-segment elevation myocardial infarction (STEMI). However, a number of other conditions can cause ST elevation, and to complicate matters, some of these can coexist with STEMI.

Nevertheless, careful attention to the ST-T and QRS-complex configurations often allows diagnosis of the cause of ST elevation (**FIGURE 1, TABLE 1**). This paper discusses the differential diagnosis of ST elevation.

■ MEASURED AT THE J POINT OR LATER

ST-segment deviation is usually measured at its junction with the end of the QRS complex, ie, the J point, and is referenced against the TP or PR segment.¹ Some authors prefer measuring the magnitude of the ST deviation 40 to 80 msec after the J point, when all myocardial fibers are expected to have reached the same level of membrane potential and to form an isoelectric ST segment.^{2,3}

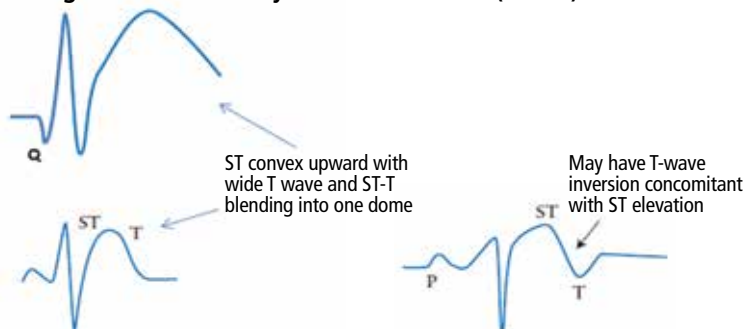
■ ST-SEGMENT ELEVATION MYOCARDIAL INFARCTION

A diagnosis of STEMI that mandates emergency reperfusion requires ST elevation equaling or exceeding the following cut-points, in at least two contiguous leads (using the standardization of 1.0 mV = 10 mm)^{4,5}:

- 1 mm in all standard leads other than V₂ and V₃
- 2.5 mm in leads V₂ and V₃ in men younger than age 40, 2 mm in leads V₂ and V₃ in men age 40 and older, and 1.5 mm in these leads in women
- 0.5 mm in the posterior chest leads V₇

Patterns of ST-segment elevation

ST-segment elevation myocardial infarction (STEMI)



Early repolarization

Concave ST elevation

Notched, slurred, or otherwise well-demarcated J point

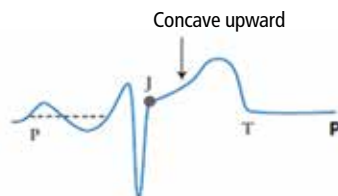


Pericarditis

Concave ST elevation

PR depression

ST/T ratio > 25%



Left bundle branch block



Hyperkalemia

ST segment pulled by T wave

T tall and narrow

(not wide as in STEMI)

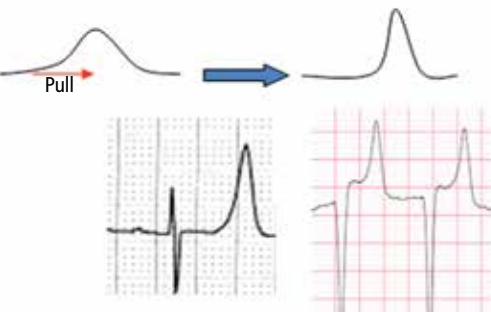


FIGURE 1

to V_9 ; ST elevation is attenuated in the posterior leads because of their greater distance from the heart, explaining the lower cut-point.⁶

While ST elevation that falls below these cut-points may be a normal variant, any ST elevation or depression (≥ 0.5 mm) may be abnormal and may necessitate further evaluation for ischemia, particularly when the clinical setting or the ST morphology suggests ischemia or when other signs of ischemia such as T-wave abnormalities, Q waves, or reciprocal ST-segment changes are also present on the electrocardiogram.

Conversely, ST elevation that exceeds these cut-points may not represent STEMI. In an analysis of patients with chest pain manifesting ST elevation, only 15% were eventually diagnosed with STEMI.⁷ In addition to size, careful attention to the morphology of the ST segment and the associated features is critical (FIGURE 1).

Other features of STEMI

In STEMI, the ST elevation is typically a convex or a straight oblique line, blending with a wide T wave to form a dome.⁸ But ST elevation may be concave in up to 40% of anterior STEMI, especially in the early stage.^{3,9,10} The nonconcave morphology is highly specific but not sensitive for the diagnosis of anterior STEMI.^{3,8,9}

Four other features characteristic of STEMI may be present (FIGURES 2 AND 3):

- Concomitant T-wave abnormalities (wide, ample, or inverted T waves)
- Q waves
- ST depression in the reciprocal leads. Reciprocal ST depression is seen in all inferior STEMI and in 70% of anterior STEMI.^{11,12} Diffuse ST elevation mimicking pericarditis may be seen with midvessel occlusion of a left anterior descending artery that wraps around the apex and supplies part of the inferior wall.
- ST or T-wave amplitude may approximate or exceed the QRS amplitude in at least one lead.^{3,13,14} This finding is characteristic of STEMI, in which the QRS “shrinks” as the infarcted area becomes electrically neutral, whereas the ST-T segments become ample.^{3,13} In fact, early STEMI may

be characterized by a small R wave that seems to be “pulled up” by the elevated ST segment. A small or absent R wave along with an ample, convex ST segment that fuses with the T wave and exceeds the height of the remaining R wave is called “tombstoning” (FIGURE 3). Tombstoning is most commonly seen with anterior infarction and implies more extensive myocardial damage and a worse prognosis than STEMI without tombstoning.¹⁵

Note that ST elevation may not be acute STEMI but an old STEMI with a chronically dysfunctional myocardium (dyskinetic or aneurysmal myocardium). In fact, an old STEMI may manifest as a chronic, persistent ST elevation along with Q waves, and T waves may be inverted or upright, but not ample.¹⁴ A history of an old MI, old electrocardiograms, if available, and quick bedside echocardiography may allow the diagnosis. In the case of an old dyskinetic infarct, echocardiography shows a thin, bright (scarred), and possibly aneurysmal myocardium, whereas in acute STEMI, the myocardium is neither thin nor scarred yet. If the patient does not report a history of MI, if the T wave is ample (> 75% the size of QRS), or if the patient presents with atypical ongoing angina, presume it is acute STEMI.

■ EARLY REPOLARIZATION

Early repolarization is a normal variant of ST elevation that equals or exceeds 1 mm (measured at the J point). It is highly prevalent in people under age 40 and remains prevalent in middle-aged people.

Two distinct and sometimes coexistent forms of early repolarization have been described: (1) ST elevation in the anterior leads V_1 to V_3 ,^{16–19} and (2) ST elevation in the lateral leads (V_4 to V_6 , I, aVL) or inferior leads.^{18–22} The prevalence of the first form—ie, ST elevation of 1 mm or more in any of the leads V_1 through V_3 —is 60% to 90% in men age 45 and younger, 20% to 40% in men over age 45, and about 10% in women of any age.¹⁶ Thus, this form of early repolarization is called “normal male pattern.”

Even early repolarization that involves the lateral or inferior leads is common, with a

TABLE 1

Differential diagnosis of ST-segment elevation

ST-segment elevation myocardial infarction (STEMI)^a

1. ST-segment elevation straight or convex upward, blends with T to form a dome
2. Wide upright T or inverted T
3. Q waves
4. ST-segment elevation or T wave may approximate or exceed QRS height
5. Reciprocal ST-segment depression

Early repolarization

Notched J point

ST-segment elevation ≤ 3 mm

Pericarditis

PR depression > 1 mm^b

ST-segment elevation < 5 mm

Left ventricular hypertrophy, left bundle branch block, preexcitation

Both ST segment and T wave are directed opposite to QRS

ST-segment elevation $< 25\%$ of QRS height (and ST-segment elevation < 2.5 mm in left ventricular hypertrophy)

ST-segment elevation of left bundle branch block may be straight or convex upward

Delta wave, short PR, and pseudo-Q waves are seen in preexcitation

Hyperkalemia

Narrow-based, peaked T wave pulling the ST segment

^a The presence of only one of the first four STEMI features makes the diagnosis of STEMI. Conversely, the lack of all STEMI features does not necessarily rule out STEMI. The fifth STEMI feature, reciprocal ST-segment depression, may be seen with other diagnoses: ST-segment depression in the lateral leads usually accompanies anterior ST-segment elevation of left bundle branch block and left ventricular hypertrophy; ST-segment depression may be seen in leads aVR and V_1 in pericarditis, and in lead aVR in early repolarization. While the corrected QT interval may be prolonged in STEMI, it is normal in pericarditis and early repolarization.

^b PR depression < 1 mm may be seen in early repolarization, and PR depression of any degree may be seen with the atrial injury coinciding with STEMI.

STEMI or pericarditis?

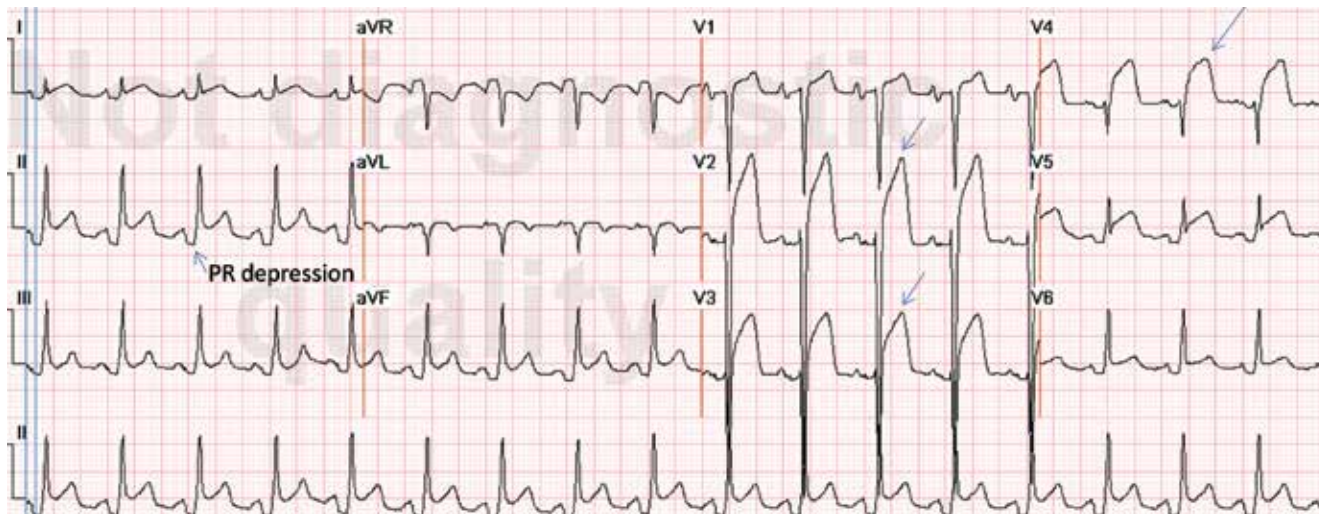


FIGURE 2. Diffuse ST-segment elevation with ST-segment depression in lead aVR. This initially suggests pericarditis. PR depression in leads II, aVF, V₅, and V₆ further suggests pericarditis. But the presence of features of pericarditis does not necessarily rule out STEMI. The five STEMI features must be ruled out. In this case, the ST-segment morphology and the abnormally wide T wave are features of STEMI. The ST elevation has an upwardly convex shape with a wide and high T wave fused with the ST segment, typical of STEMI (leads V₂–V₄, arrows). Also, the size of the ST elevation (ie, > 5 mm in V₂–V₄ and larger than the QRS complex in V₄, a feature called “tombstoning”) is more consistent with STEMI than with pericarditis. In this patient, the left anterior descending artery was found to be occluded on coronary arteriography.

prevalence of about 15% in people ages 30 to 40 and about 5% to 10% in those 40 to 65.^{20–23} It is two to four times more prevalent in men and three times more prevalent in African Americans. It is also highly prevalent in athletes younger than 25 (about 30% to 40%).²²

Either way, early repolarization closely resembles the ST elevation of pericarditis and has the following features (**FIGURE 4**):

- The ST segment is concave upward, and the J point is well demarcated and may be notched or slurred (**FIGURE 1**).
- ST elevation is usually no more than 3 mm.
- ST elevation may be limited to the anterior leads or, in many instances, may extend to the inferior or lateral leads. Early repolarization is very rarely limited to the limb leads, and involvement of some precordial leads is the rule.^{18,19} The ST segment is depressed in lead aVR in 50% of patients.^{18,19}
- The T wave is usually ample and may be more than 10 mm tall in the precordial leads in one-third of patients,¹⁷ but as opposed to the ample T wave of STEMI, it is not broad and remains smaller than the

QRS complex. The ample T wave distinguishes early repolarization from pericarditis, and explains the low ST-T ratio in lead V₆. In up to 10% of young black men, the T wave has a terminal inversion in leads V₃ to V₅, and occasionally in V₁ and V₂, mimicking infarction (**FIGURE 5**).²⁴

- The QRS complex tends to have prominent precordial voltage, in sharp contrast to STEMI, in which QRS shrinking occurs.^{3,17,22}

The early repolarization pattern may be intermittent, may vary among serial electrocardiograms, may decrease with a rise in sympathetic tone, as observed during exercise, and may increase with a rise in vagal tone.^{18,19,25,26} Although it is usually a benign finding, the early repolarization pattern in leads other than V₁ to V₃ has been associated with an increased risk of sudden death, particularly when the ST elevation is horizontal-descending rather than upsloping and, possibly, when early repolarization involves the inferior leads with a J point that is notched or elevated 2 mm or more.^{20,22}

'Tombstoning' and 'shrinking'



FIGURE 3. In a patient with lung cancer, sinus tachycardia is seen with diffuse ST-segment elevation, along with ST-segment depression in aVR. The QRS voltage is low, particularly when compared with the electrocardiogram recorded a few days earlier (left lower panel). PR depression is seen in lead II. The combination of these findings may suggest pericarditis with a pericardial effusion. However, the ST-T morphology in lead V₂, where the ST and T are blended to form one dome, is characteristic of STEMI (top arrow). Moreover, the ST elevation and T wave in leads V₂–V₄ are larger than the QRS, the QRS voltage is “shrinking” (arrowhead), and the R wave is pulled up by the ST segment (star); this is called “tombstoning.” All these features are characteristic of STEMI, wherein the R wave and the QRS complex shrink before forming a deep Q wave. In fact, an electrocardiogram recorded 1 hour later (right lower panel) shows a fully developed Q wave in lead V₂ (bottom arrow).

PERICARDITIS

In pericarditis, ST elevation is concave upward and is widespread to more than one region without reciprocal ST depression, except for the frequent ST depression in leads aVR and V₁ (64%)²⁷; ST elevation is seldom greater than 4 to 5 mm (FIGURE 6).^{27,28} Since the subepicardial injury is diffuse in pericarditis, the axis of the ST segment follows the anatomic axis of the heart and is generally +45° in the frontal plane. Thus, ST depression is seen in leads aVR and V₁; ST elevation is highest in leads II, V₅, and V₆ and is less in leads III and aVL, where the ST segment may occasionally be depressed.²⁹

Transient PR depression greater than 1 mm is often seen, particularly in leads II, aVF, and V₄ to V₆, and represents atrial subepicardial injury. PR depression in those leads is always associated with PR elevation in lead

aVR and sometimes V₁. PR changes often coexist with ST changes but may be isolated and may precede ST changes.³⁰ PR depression is characteristic of pericarditis but may be seen in early repolarization, where it is less marked than in pericarditis (< 0.8 mm) and implies early repolarization of the atrial tissue,³¹ and in MI, where it implies atrial infarction with atrial injury pattern.

Classically, it is said that in pericarditis, unlike in STEMI, the T wave does not invert until the ST elevation subsides. In reality, up to 40% of patients develop a notched or biphasic positive-negative T wave before full return of the ST segment to the baseline.^{27,32} And if T-wave inversion antedates pericarditis, concomitant ST elevation and T-wave inversion may be seen once pericarditis develops. However, the T wave inverts less deeply and less completely than in STEMI, and the corrected QT interval

Early repolarization

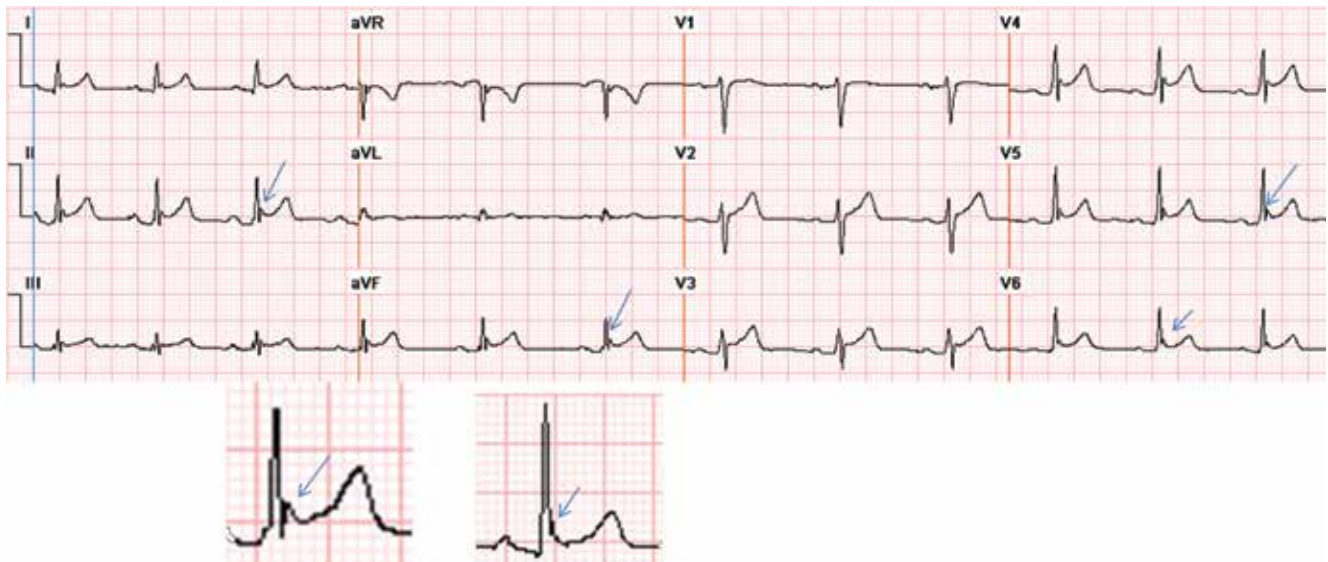


FIGURE 4. Early repolarization with ST-segment elevation is seen in the inferior leads and in the anterolateral leads V_2 to V_6 . ST elevation is most prominent in lead V_4 and lead II, with a concavely upward ST morphology and a notch at the J point (arrows and left magnified image). In half of early repolarization cases, the J point is smooth but well demarcated (right magnified image). Note the slight PR depression in leads II and V_5 . Slight PR depression may be seen in normal individuals and corresponds to the normal atrial repolarization.

remains normal even when the T wave inverts.

Three criteria distinguish pericarditis from early repolarization (but not from STEMI):

- PR depression greater than 1 mm
- ST-segment depression in lead V_1
- A ratio of ST-segment height to T-wave height of at least 25% in lead V_6 , V_5 , V_4 , or I. This feature distinguishes pericarditis from early repolarization with a high sensitivity and specificity. In pericarditis, the T waves have normal or reduced amplitude, and the ST-T ratio is therefore high,³³ whereas in early repolarization the T waves are tall, so the ST-T ratio is less than 25%.

Widespread ST elevation may be seen with both pericarditis and early repolarization. ST elevation limited to the anterior leads is more likely to be early repolarization than pericarditis.

LEFT BUNDLE BRANCH BLOCK

In left bundle branch block, a deep and wide S wave is seen in leads V_1 to V_3 and sometimes in the inferior leads, with ST elevation and T waves that are discordant with the QRS complex—ie, directed opposite to the QRS (FIGURES 7–9). The ST elevation is typically

concave upward.^{8,34} Occasionally, ST elevation may be straight or convex, mimicking the dome of STEMI. In the lateral leads, the discordant ST segment is depressed, mimicking a reciprocal ST change.

The following findings imply MI:

- ST elevation or depression that is concordant with the QRS complex. Moreover, since ST deviation is mandatory with left bundle branch block, a “normal-looking” ST segment implies ischemia.
- Inverted T waves concordant with the QRS in more than one lead, or biphasic T waves in more than one lead (eg, V_1 to V_3). Across the precordial leads, T waves may transition from positive to negative one lead earlier or later than the QRS and ST transition. Therefore, even in the absence of ischemia, the T wave may be inverted in lead V_3 , in which the QRS is deeply negative and the ST is still elevated (negative T-wave concordance in one lead). Also, the T wave may be upright in leads V_5 , V_6 , and I where QRS is upright and the ST segment is depressed (positive T-wave concordance does not imply ischemia).

- In addition to concordance, a discordant ST segment or T wave that is very large may imply ischemia. For example, a discordant ST segment or T wave that is larger than the QRS height implies ischemia. A discordant ST elevation greater than 5 mm has been suggested by Sgarbossa et al³⁵ as a diagnostic feature of STEMI; however, this feature is seen in 10% of control patients with left bundle branch block and no STEMI, and it is thus poorly specific and also poorly sensitive, frequently missing STEMI.^{35–37} Smith et al³⁶ have suggested that a discordant ST elevation of at least 25% of the S-wave depth is a far more sensitive and accurate feature but one that may still be found in up to 10% of control patients.³⁶

■ LEFT VENTRICULAR HYPERTROPHY

In left ventricular hypertrophy, a deep S wave is seen in leads V₁ to V₃, with ST elevation and T waves that are discordant with the QRS complex. Rarely, ST elevation may be straight or convex. The following findings imply MI:

- ST elevation or depression that is concordant with the QRS.
- Inverted T waves that are concordant with the QRS in more than one lead, or biphasic T waves in more than one lead (eg, V₁ to V₃).
- A discordant ST segment or a T wave that is very large may imply ischemia. In left ventricular hypertrophy, ST elevation is usually less than 2.5 mm in leads V₁ to V₃ and is rarely seen in the inferior leads, where it would be less than 1 mm.³⁴ When ST elevation is seen in leads V₁ to V₃ in left ventricular hypertrophy, an ST magnitude of 25% or more of the total QRS voltage has a 91% specificity for STEMI.³⁴

On another note, right ventricular hypertrophy and right bundle branch block may lead to ST-segment depression and T-wave inversion, but not to ST elevation. Thus, ST elevation occurring with right ventricular hypertrophy or right bundle branch block implies STEMI. While only left bundle branch block poses a diagnostic challenge, both types of bundle branch block, if secondary to STEMI, represent equally high-risk categories.³⁸

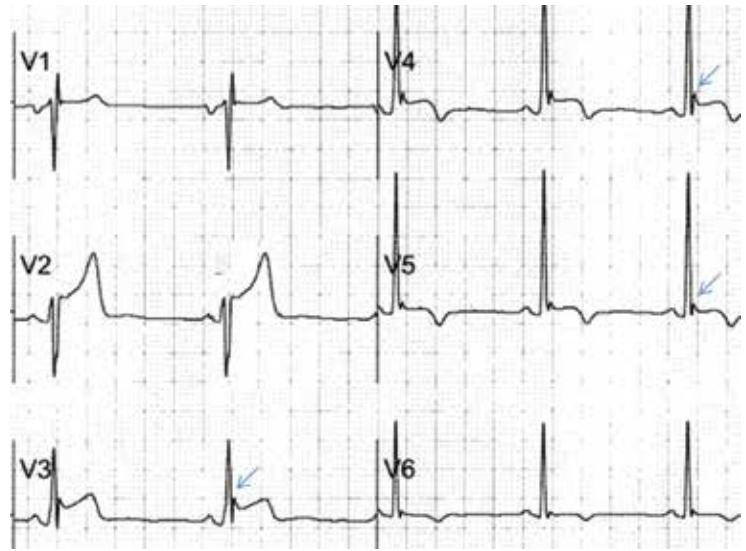


FIGURE 5. Early repolarization with a normal variant T-wave inversion in a 33-year-old black man. The ST segment is elevated with a notched J point in leads V₂ to V₅ (arrows). The T wave is inverted in V₄ and V₅. Depending on the autonomic tone, the T waves may at times become upright.

■ PREEXCITATION

Preexcitation may be associated with negative delta waves that mimic Q waves, and with ST elevation in the leads where the negative delta waves are seen, ie, ST elevation discordant with the delta wave (**FIGURE 10**). The QRS morphology and the delta wave allow preexcitation to be distinguished from STEMI.

■ HYPERKALEMIA

The most common finding in hyperkalemia is a peaked, narrow-based T wave that is usually, but not necessarily, tall. ST elevation may be evident in leads V₁ to V₃ (**FIGURE 11**). In contrast with hyperkalemia, the T wave of STEMI is typically wide.

■ OTHER CAUSES OF ST-SEGMENT ELEVATION

Takotsubo cardiomyopathy

Takotsubo cardiomyopathy mimics all electrocardiographic features of anteroapical STEMI. ST elevation may extend to the inferior leads but cannot be isolated in the inferior leads.³⁹ As in apical STEMI, reciprocal ST depression is uncommon. Within 24 to 48 hours, ST

Pericarditis

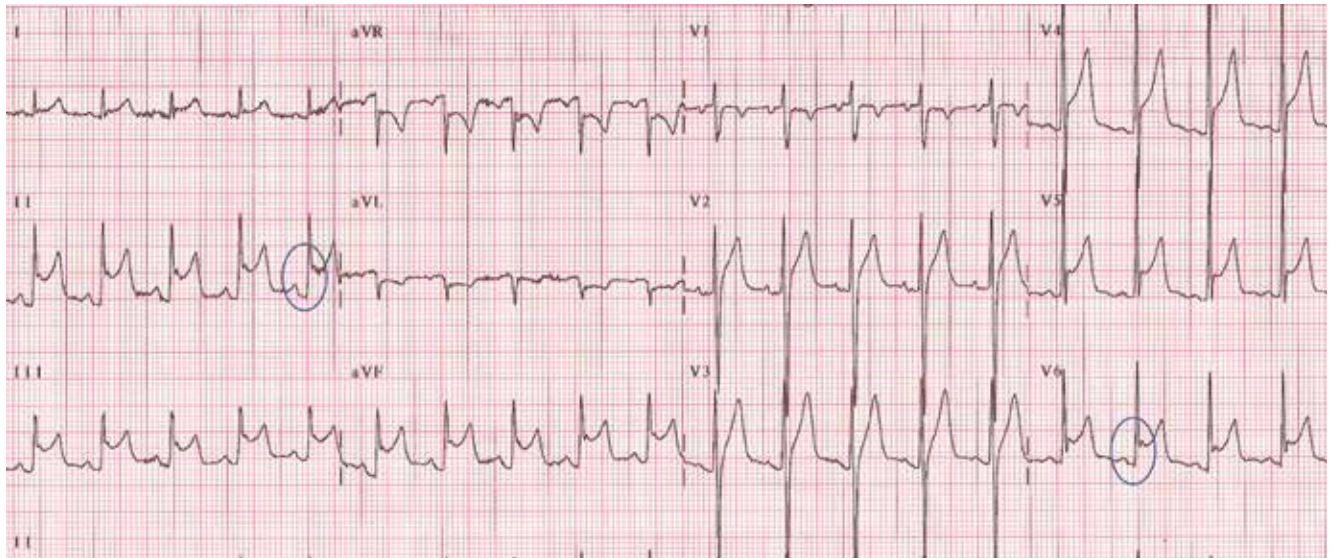


FIGURE 6. Diffuse ST-segment elevation in most leads, with ST depression in lead aVR and an isoelectric ST segment in V₁. None of the STEMI features are present: ST elevation is concave upward, no reciprocal ST depression is seen except in lead aVR; the T wave is not wide, inverted, or ample (in relation to the QRS complex); and no Q wave is seen. Furthermore, ST elevation does not exceed 5 mm; ST and T heights are smaller than QRS height; and PR depression is present (circled areas). As opposed to early repolarization, the ratio of ST to T in leads V₅ and V₆ exceeds 25%. This is consistent with pericarditis, and the hospital course of this patient confirmed this diagnosis.

Supraventricular tachycardia with left bundle branch block and STEMI

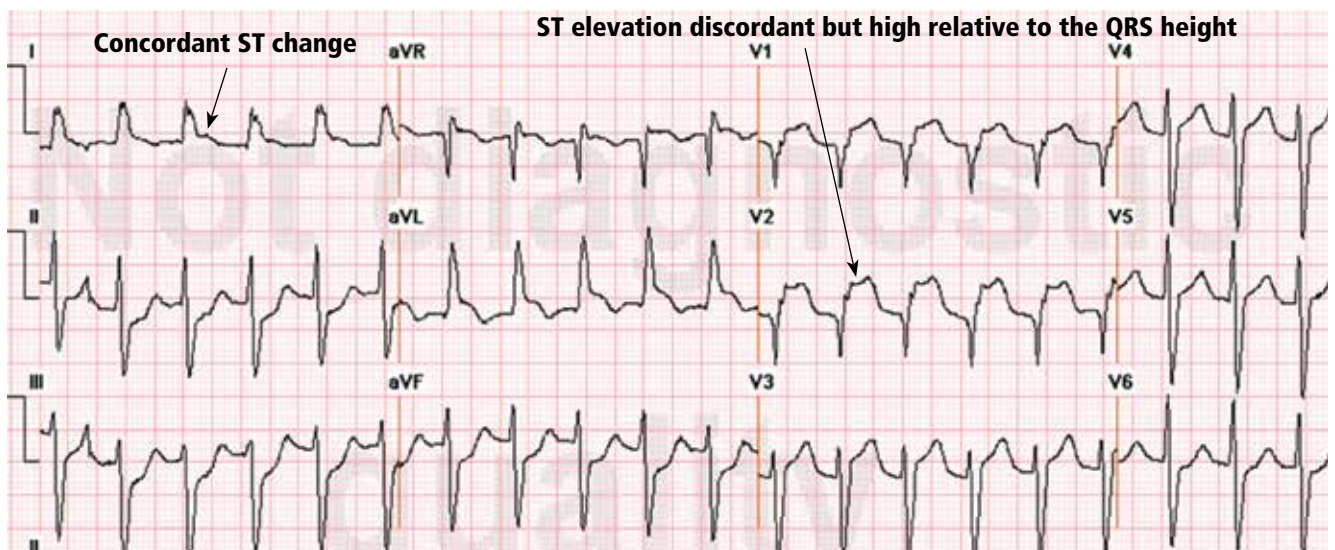


FIGURE 7. Supraventricular tachycardia with a typical left bundle branch block pattern in leads I and aVL. Concordant ST-segment elevation is seen in leads I and aVL, while concordant ST depression is seen in the inferior leads (arrows). The ST elevation in lead V₂ is discordant but is disproportionately high in relation to the QRS (well above 25% of the QRS height). All these features are diagnostic of STEMI.

Left bundle branch block and STEMI

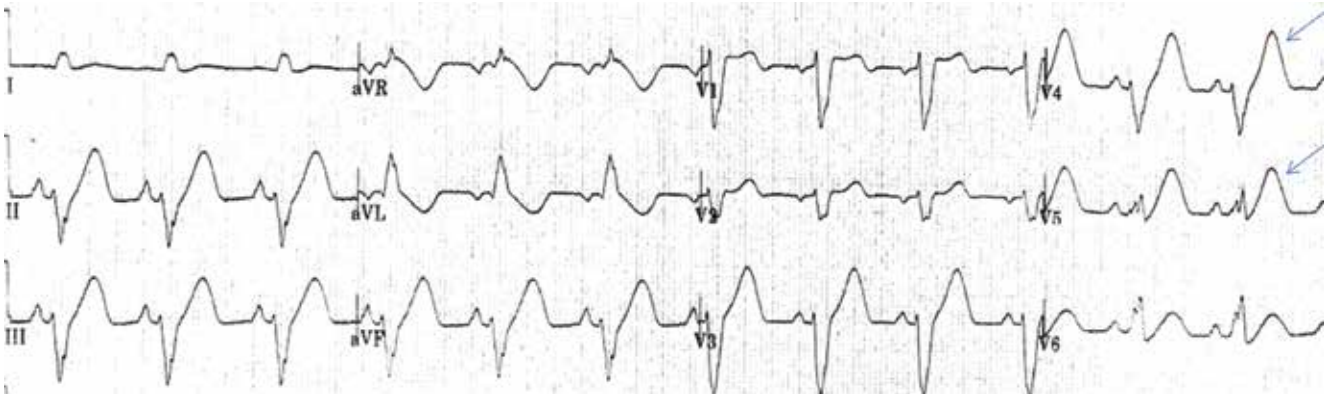


FIGURE 8. Left bundle branch block with discordant ST-segment changes. However, the T wave is wide and fused with the ST segment in a domed morphology, and the T wave is larger than the QRS in leads V_4 , V_5 , and II (arrows). This implies the diagnosis of STEMI with hyperacute T waves. This patient had an occluded left anterior descending coronary artery.

Left bundle branch block and abnormal T waves

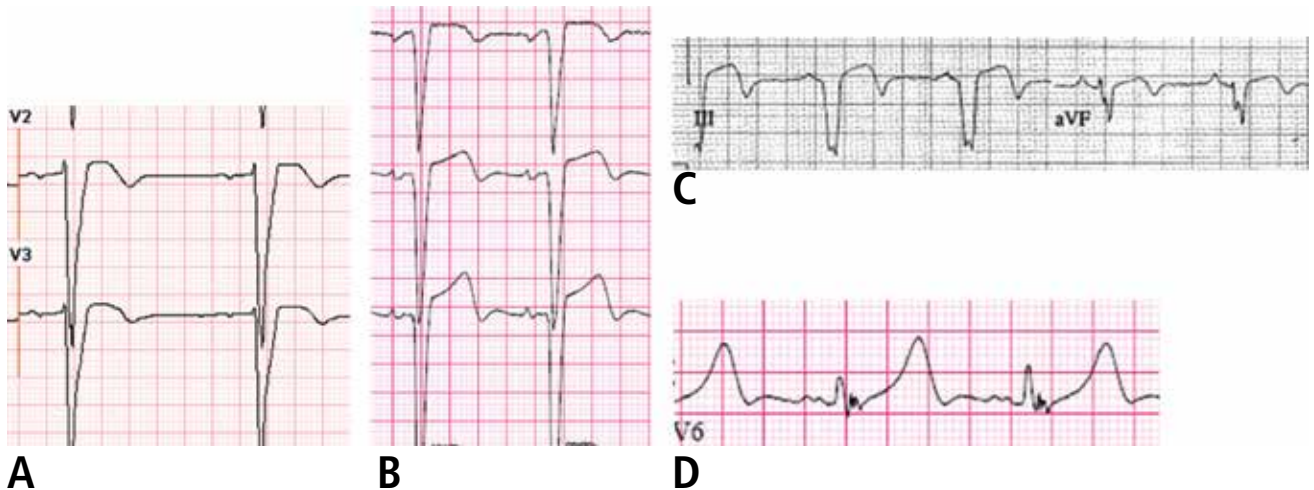


FIGURE 9. Left bundle branch block with abnormal T waves. Panels A and B show discordant ST-segment elevation in V_1 to V_3 but concordant T wave inversion (A) or biphasic T wave (B). This is consistent with an anterior injury pattern. Panel C shows concordant T-wave inversion in the inferior leads, consistent with inferior injury. Panel D shows a large concordant T wave in lead V_6 , larger than the QRS, consistent with injury.

elevation evolves into deep anterior T-wave inversion and a prolonged QT interval. Transient Q waves may be seen.

Myocarditis

Myocarditis may have one of two electrocardiographic patterns: a pericarditis pattern, or a typical STEMI pattern with Q waves some-

times localized to one area.⁴⁰

Atrial flutter waves

Atrial flutter waves, particularly of 2:1 atrial flutter, may deform the ST segment so that it mimics an injury pattern on the electrocardiogram. Flutter waves may mimic ST elevation or ST depression (**FIGURE 12**).

STEMI or preexcitation?

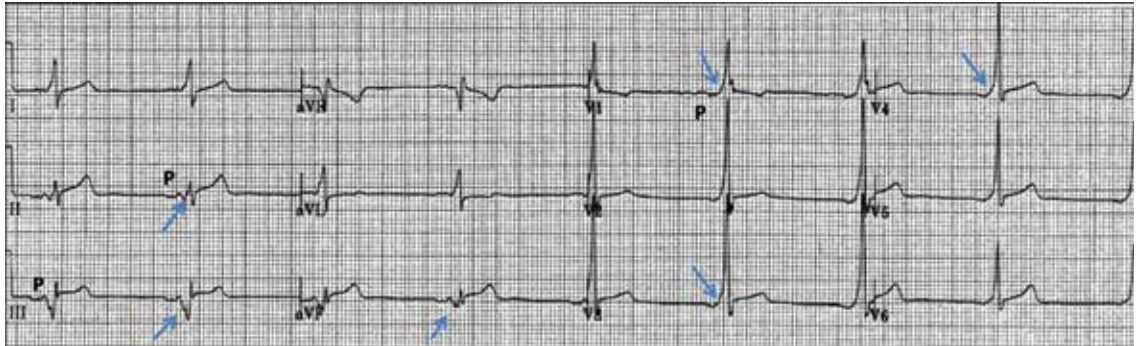


FIGURE 10. At first glance, it seems there is ST-segment elevation in the inferior leads II, III, and aVF, with a wide Q wave. Moreover, there is a wide and tall R wave in lead V₁ suggesting an associated posterior infarction. All this is consistent with acute inferoposterior STEMI. On further analysis, however, a slur is seen on the upslope of QRS in leads V₁ to V₆ (arrows), and the P wave is “riding” this slur. In the inferior leads, the P wave is riding the Q wave, which is in fact a negative delta wave. Thus, this electrocardiogram represents preexcitation. The ST deviations are secondary to the preexcitation and have an orientation opposite to the delta wave.

Hyperkalemia

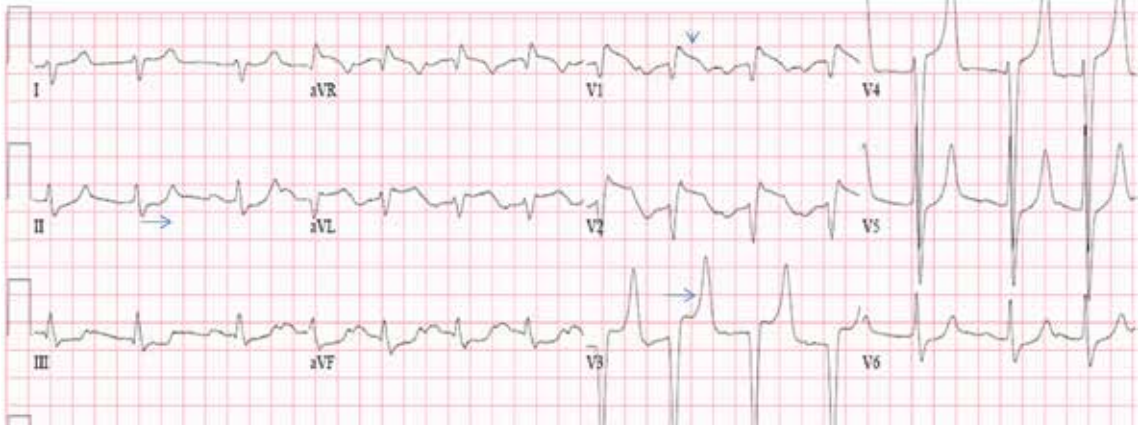


FIGURE 11. There are ST-segment elevations in leads V₁–V₄, ST-segment depressions in the inferior leads, and peaked T waves in leads V₃–V₅. These T waves have a narrow base and seem to “pull” the ST segment, creating ST elevation in the anterior leads and ST depression in the inferior leads (arrows). This shape is consistent with hyperkalemia. In addition, the downsloping ST elevation seen in V₁ and V₂ is consistent with hyperkalemia (arrowhead). Occasionally, STEMI may have a similar ST-T shape. An rSR’ pattern is seen in V₁–V₂; this is consistent with STEMI but also with hyperkalemia, in which conduction blocks are common. The serum potassium level was 7.4 mmol/L (normal 3.5–5), and coronary angiography revealed normal coronary arteries.

The most common finding in hyperkalemia is a peaked, narrow-based T wave that is usually, but not necessarily, tall

Large pulmonary embolism

A large pulmonary embolism may be associated with T-wave inversion in the anterior leads or the inferior leads, or both, reflective of cor pulmonale. Less commonly, ST elevation in the anterior or inferior leads is seen. In fact, changes of both anterior and inferior ischemia should always suggest a

pulmonary embolism.^{41,42}

Brugada syndrome

Brugada syndrome is characterized by ST elevation and a right bundle branch block or pseudo-right bundle branch block pattern in at least two of the leads V₁ to V₃. In pseudo-right bundle branch block, the QRS adopts

an rSR' morphology in the anterior leads but is normal in the lateral leads. Type 1 Brugada pattern, the pattern that is most specifically associated with sudden death, is characterized by a coved, downsloping ST elevation of 2 mm or more with T-wave inversion (**FIGURE 13**).⁴³ The Brugada pattern can be transient, triggered by fever, cocaine, or class I antiarrhythmic drugs.

Hyperkalemia, Brugada syndrome, and sometimes pulmonary embolism are characterized by an ST elevation that slopes downward (**FIGURES 11 AND 13**), which contrasts with the upsloping, convex ST elevation of STEMI.

Type 1 Brugada pattern

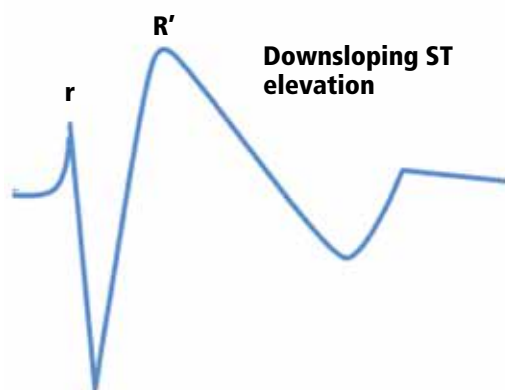


FIGURE 13. Type 1 Brugada pattern in V_1 and V_2 , with a downsloping ST-segment elevation that creates a pseudo-R' wave (pseudo-right bundle branch block). The QRS does not have a right bundle branch block morphology in leads V_5 and V_6 .

Atrial flutter

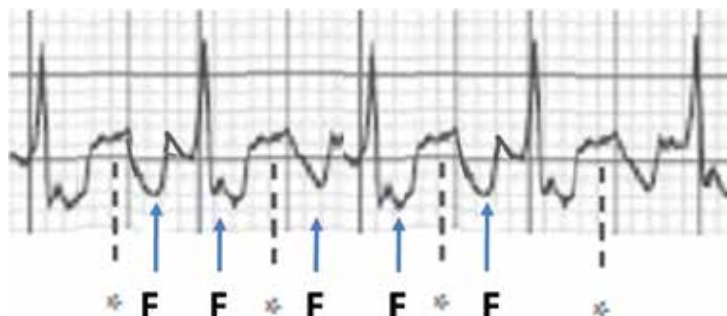
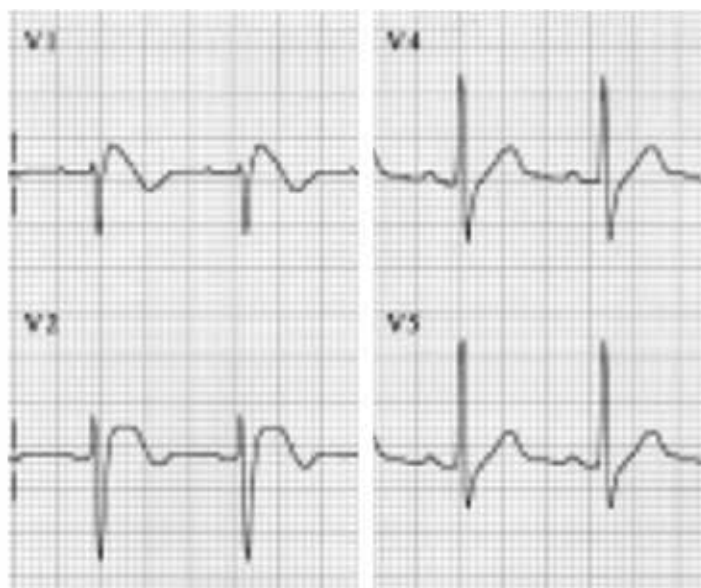


FIGURE 12. Atrial flutter that simulates ST-segment elevation. An "F" indicates the negative flutter wave; an asterisk indicates the upslope of the flutter wave that is superimposed on the ST segment, mimicking ST elevation.



REFERENCES

1. Rautaharju PM, Surawicz B, Gettes LS, et al; American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; American College of Cardiology Foundation; Heart Rhythm Society. AHA/ACCF/HRS recommendations for the standardization and interpretation of the electrocardiogram: part IV: the ST-segment, T and U waves, and the QT interval: a scientific statement from the American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; the American College of Cardiology Foundation; and the Heart Rhythm Society. Endorsed by the International Society for Computerized Electrocardiology. *J Am Coll Cardiol* 2009; 53:982–991.
2. Surawicz B, Nilans TK. Chou's Electrocardiography in Clinical Practice: Adult and Pediatric. 5th ed. Philadelphia, PA: WB Saunders; 2001:194–207.
3. Smith SW, Khalil A, Henry TD, et al. Electrocardiographic differentiation of early repolarization from subtle anterior ST-segment elevation myocardial infarction. *Ann Emerg Med* 2012; 60:45–56.e2.
4. American College of Emergency Physicians; Society for Cardiovascular Angiography and Interventions; O'Gara PT, Kushner FG, Ascheim DD, et al. 2013 ACCF/AHA guideline for the management of ST-elevation myocardial infarction: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. *J Am Coll Cardiol* 2013; 61:e78–e140.
5. Thygesen K, Alpert JS, Jaffe AS, et al; Joint ESC/ACCF/AHA/WHF Task Force for the Universal Definition of

- Myocardial Infarction.** Third universal definition of myocardial infarction. *Circulation* 2012; 126:2020–2035.
6. **Matetzky S, Freimark D, Chouraqui P, et al.** Significance of ST segment elevations in posterior chest leads (V7 to V9) in patients with acute inferior myocardial infarction: application for thrombolytic therapy. *J Am Coll Cardiol* 1998; 31:506–511.
 7. **Brady WJ, Perron AD, Martin ML, Beagle C, Aufderheide TP.** Cause of ST-segment abnormality in ED chest pain patients. *Am J Emerg Med* 2001; 19:25–28.
 8. **Brady WJ, Syverud SA, Beagle C, et al.** Electrocardiographic ST-segment elevation: the diagnosis of acute myocardial infarction by morphologic analysis of the ST segment. *Acad Emerg Med* 2001; 8:961–967.
 9. **Smith SW.** Upwardly concave ST-segment morphology is common in acute left anterior descending coronary occlusion. *J Emerg Med* 2006; 31:69–77.
 10. **Kosuge M, Kimura K, Ishikawa T, et al.** Value of ST-segment elevation pattern in predicting infarct size and left ventricular function at discharge in patients with reperfused acute anterior myocardial infarction. *Am Heart J* 1999; 137:522–527.
 11. **Birnbaum Y, Sclarovsky S, Mager A, Strasberg B, Rechavia E.** ST segment depression in a VL: a sensitive marker for acute inferior myocardial infarction. *Eur Heart J* 1993; 14:4–7.
 12. **Engelen DJ, Gorgels AP, Cheriex EC, et al.** Value of the electrocardiogram in localizing the occlusion site in the left anterior descending coronary artery in acute anterior myocardial infarction. *J Am Coll Cardiol* 1999; 34:389–395.
 13. **Collins MS, Carter JE, Dougherty JM, Majercik SM, Hodsdon JE, Logue EE.** Hyperacute T-wave criteria using computer ECG analysis. *Ann Emerg Med* 1990; 19:114–120.
 14. **Smith SW.** T/QRS ratio best distinguishes ventricular aneurysm from anterior myocardial infarction. *Am J Emerg Med* 2005; 23:279–287.
 15. **Balci B.** Tombstoning ST-elevation myocardial infarction. *Curr Cardiol Rev* 2009; 5:273–278.
 16. **Surawicz B, Parikh SR.** Prevalence of male and female patterns of early ventricular repolarization in the normal ECG of males and females from childhood to old age. *J Am Coll Cardiol* 2002; 40:1870–1876.
 17. **Klatsky AL, Oehm R, Cooper RA, Udaltsova N, Armstrong MA.** The early repolarization normal variant electrocardiogram: correlates and consequences. *Am J Med* 2003; 115:171–177.
 18. **Mehta M, Jain AC, Mehta A.** Early repolarization. *Clin Cardiol* 1999; 22:59–65.
 19. **Mehta MC, Jain AC.** Early repolarization on scalar electrocardiogram. *Am J Med Sci* 1995; 309:305–311.
 20. **Rollin A, Maury P, Bongard V, et al.** Prevalence, prognosis, and identification of the malignant form of early repolarization pattern in a population-based study. *Am J Cardiol* 2012; 110:1302–1308.
 21. **Tikkanen JT, Anttonen O, Junttila MJ, et al.** Long-term outcome associated with early repolarization on electrocardiography. *N Engl J Med* 2009; 361:2529–2537.
 22. **Tikkanen JT, Junttila MJ, Anttonen O, et al.** Early repolarization: electrocardiographic phenotypes associated with favorable long-term outcome. *Circulation* 2011; 123:2666–2673.
 23. **Noseworthy PA, Tikkanen JT, Porthan K, et al.** The early repolarization pattern in the general population: clinical correlates and heritability. *J Am Coll Cardiol* 2011; 57:2284–2289.
 24. **Wasserburger RH.** Observations on the juvenile pattern of adult negro males. *Am J Med* 1955; 18:428–437.
 25. **Kralios FA, Martin L, Burgess MJ, Millar K.** Local ventricular repolarization changes due to sympathetic nerve-branch stimulation. *Am J Physiol* 1975; 228:1621–1626.
 26. **Spratt KA, Borans SM, Michelson EL.** Early repolarization: normalization of the electrocardiogram with exercise as a clinically useful diagnostic feature. *J Invasive Cardiol* 1995; 7:238–242.
 27. **Surawicz B, Lasseter KC.** Electrocardiogram in pericarditis. *Am J Cardiol* 1970; 26:471–474.
 28. **Hull E.** The electrocardiogram in pericarditis. *Am J Cardiol* 1961; 7:21–32.
 29. **Spodick DH.** Diagnostic electrocardiographic sequences in acute pericarditis. Significance of PR segment and PR vector changes. *Circulation* 1973; 48:575–580.
 30. **Spodick DH.** Acute pericarditis: current concepts and practice. *JAMA* 2003; 289:1150–1153.
 31. **Charles MA, Bensinger TA, Glasser SP.** Atrial injury current in pericarditis. *Arch Intern Med* 1973; 131:657–662.
 32. **Noth PH, Barnes HR.** Electrocardiographic changes associated with pericarditis. *Arch Intern Med* 1940; 65:291–320.
 33. **Ginzton LE, Laks MM.** The differential diagnosis of acute pericarditis from the normal variant: new electrocardiographic criteria. *Circulation* 1982; 65:1004–1009.
 34. **Armstrong EJ, Kulkarni AR, Bhavé PD, et al.** Electrocardiographic criteria for ST-elevation myocardial infarction in patients with left ventricular hypertrophy. *Am J Cardiol* 2012; 110:977–983.
 35. **Sgarbossa EB, Pinski SL, Barbagelata A, et al.** Electrocardiographic diagnosis of evolving acute myocardial infarction in the presence of left bundle-branch block. GUSTO-1 (Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries) Investigators. *N Engl J Med* 1996; 334:481–487.
 36. **Smith SW, Dodd KW, Henry TD, Dvorak DM, Pearce LA.** Diagnosis of ST-elevation myocardial infarction in the presence of left bundle branch block with the ST-elevation to S-wave ratio in a modified Sgarbossa rule. *Ann Emerg Med* 2012; 60:766–776.
 37. **Madias JE, Sinha A, Agarwal H, Ashtiani R.** ST-segment elevation in leads V1–V3 in patients with LBBB. *J Electrocardiol* 2001; 34:87–88.
 38. **Sgarbossa EB, Pinski SL, Topol EJ, et al.** Acute myocardial infarction and complete bundle branch block at hospital admission: clinical characteristics and outcome in the thrombolytic era. GUSTO-I Investigators. Global Utilization of Streptokinase and t-PA [tissue-type plasminogen activator] for Occluded Coronary Arteries. *J Am Coll Cardiol* 1998; 31:105–110.
 39. **Bybee KA, Kara T, Prasad A, et al.** Systematic review: transient left ventricular apical ballooning: a syndrome that mimics ST-segment elevation myocardial infarction. *Ann Intern Med* 2004; 141:858–865.
 40. **Magnani JW, Dec GW.** Myocarditis: current trends in diagnosis and treatment. *Circulation* 2006; 113:876–890.
 41. **Sreeram N, Cheriex EC, Smeets JL, Gorgels AP, Wellens HJ.** Value of the 12-lead electrocardiogram at hospital admission in the diagnosis of pulmonary embolism. *Am J Cardiol* 1994; 73:298–303.
 42. **Glancy DL, Mikdadi GM.** Syncope in a 67-year-old man. *Proc (Bayl Univ Med Cent)* 2005; 18:74–75.
 43. **Wilde AA, Antzelevitch C, Borggrefe M, et al; Study Group on the Molecular Basis of Arrhythmias of the European Society of Cardiology.** Proposed diagnostic criteria for the Brugada syndrome: consensus report. *Circulation* 2002; 106:2514–2519.

ADDRESS: Elias B. Hanna, MD, Department of Medicine, Cardiovascular Section, Louisiana State University Health Sciences Center, 1542 Tulane Avenue, 3rd Floor, Room 323, New Orleans, LA, 70112; e-mail: ehanna@lsuhsc.edu