Emergency Medicine Reports
The Practical Journal for Emergency Physicians
Volume 30, Number 13 / June 8, 2009
www.emreports.com

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Statement of Financial Disclosure

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Missed Myocardial Infarction: ECG Strategies to Reduce the Risk

Does the electrocardiograph in your emergency department include a computer-generated interpretation on every electrocardiogram printed? Like me, do you often find them less than helpful? Occasionally troubling? How do you react when the computer says "consistent with acute ischemia" and the patient is perfectly fine? As in most situations in medicine, the utility of any ancillary test requires judgment within the clinical context. So it is with ECGs. While getting better, computer analysis of chest wall voltages cannot replace the human analysis of the entire patient, including the ECG. That said, studies indicate that emergency physicians do miss important ECG patterns. This issue of EM Reports *covers important concepts in the ECG diagnosis of myocardial infarction.*

—J. Stephan Stapczynski, MD, Editor

ECG Interpretation

Of clinical features useful in MI diagnosis, the ECG is the most important bedside finding to diagnose acute MI.[1] The ECG is the branch point in treatment of acute MI, as patients with STEMI are taken for emergent reperfusion therapy, and those with non-STEMI are treated medically. The ECG also gives data on the location and extent of injury. (*See Table 1.*)

Several studies have found that the sensitivity of the initial ECG for detection of acute MI can be as low as 50%.[2] Specificity also can be less than desired, as noted from a recent study in which 15 experienced cardiologists were given 116 ECGs to evaluate; percutaneous coronary intervention was recommended in 7.8-33% of cases even though only 8% actually had STEMI.[3] Two other important observations are that a normal ECG is present in up to 4% of acute MI cases,[4] and patients having acute MI who present with normal or non-diagnostic ECGs are twice as likely to present atypically, i.e. without chest pain as well (11-14% vs. 24%).[5]

Patients with initially non-diagnostic ECGs often will develop ECG findings of MI in the first few hours after hospital arrival.[6] Thus, the importance of a second ECG or continuous ST segment monitoring to help detect the evolving acute MI in the ED. (*See Figure 1.*) Depending on the clinical situation, ECGs can be repeated every 30 minutes to 4 hours.[7]

Prior studies on missed MI in the ED found that 25% of these patients were discharged with ST segment elevation on their ECG.[8] More recent work by Pope in 2000 found 11% of missed MI patients were still being discharged with ST segment elevations of 1-2 mm on their ECGs.[9] The most common misdiagnoses of ST segment elevation on the ECG were left ventricular aneurysm, benign early repolarization, and left ventricular hypertrophy.[10] There are several useful principles on interpretation of difficult ECGs. (*See Table 2.*)

ECG Criteria for Diagnosis of MI

STEMI. The ECG criteria for STEMI diagnosis, are ≥ 1 mm ST segment ele-

Executive Summary

- In AMI patients, the initial ECG has diagnostic changes (ST segment elevation) in about half, new ischemic ST-T wave changes in about one third, non-diagnostic ST-T changes in about one fifth, and is truly normal in less than 5%.
- In ED patients, ST segment elevation usually is not due to AMI.
- ECG evidence of AMI is difficult to detect with either an LBBB or ventricular-paced rhythm.
- Left ventricular hypertrophy and benign early repolarization variant are the most common ECG mimics of AMI.

vation in two contiguous leads for limb leads (I, II, III, aVF, aVL) and ≥ 2 mm ST segment elevation in precordial leads (V_1-V_6). Posterior infarcts are the major exception to this. The ST segment elevation can be rather variable in appearance. (*See Figure 2.*) In most cases of MI, the actual slope is flat or concave upward. In some cases the slope is downward (convex), which can be confused with early repolarization. While ST segment elevation is the typical finding in STEMI, the earliest changes are hyperacute T waves or T waves that are much higher and wider than normal. (*See Figure 3.*) These are seen most often in anterior MIs (leads V_2-V_5).

STEMI result from larger, more proximal blockages in the major coronary vessels (right, LAD, circumflex). Occlusion of these vessels causes a full thickness ischemia of a portion of the heart. The ST segments "elevate" on the ECG is because ischemia changes the electrical properties (elevated resting membrane potential, lengthens action potential) of the affected myocytes. These changes produce a voltage gradient between normal and ischemic regions (the injury current). (*See Figure 4.*) Likewise, ST segment depressions (reciprocal changes) also are seen in STEMIs, as this represents the same voltage differences seen on lead sensing the infarct from the opposite side of the heart. For example, ST segment elevation with anterior MI will produce inferior ST segment depression. Reciprocal changes from an inferior MI are best seen in lead aVL. For reasons that are not well understood, reciprocal changes are only seen in about 80% of inferior MIs, and only 33% of anterior MIs.[10,11] The ST segment depression of posterior MIs actually represents the reciprocal changes from elevation present leads V_8-V_9 (when additional leads are used). The presence of reciprocal changes increases the likelihood of acute MI with a positive predictive value of > 90%.[12]

Table 1: Anatomic Location of Acute MI and the Corresponding ECG Changes*

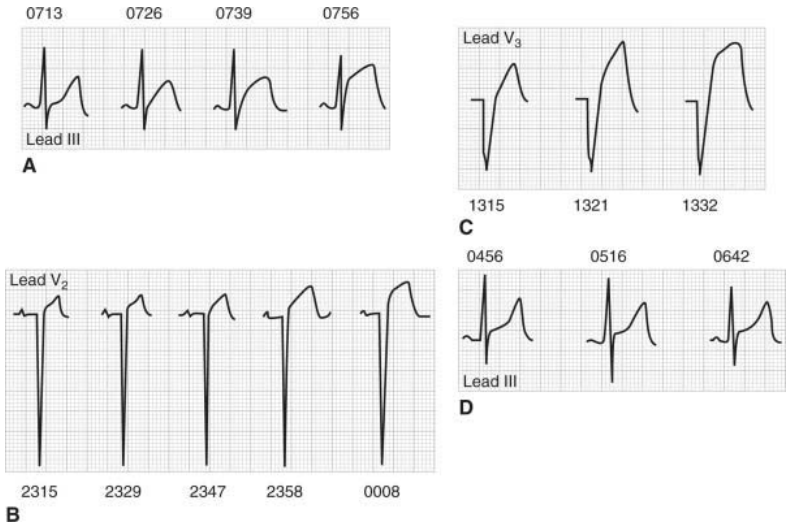
Anatomic Location	ECG Leads	ECG Findings
Anterior	V_2–V_4 II, III, aVF	ST Elevation ≥ 2 mm Reciprocal depression (only in 33%)
Inferior	II, III, aVF aVL	ST Elevation ≥ 1 mm Reciprocal depression (in ~ 80 %)
Right ventricle	V_4R V_1–V_3	ST Elevation diagnostic ST Elevation indicative
Lateral	I, aVL, V_5, V_6	ST Elevation ≥ 2 mm (in precordial leads)
Posterior	V_1, V_2 V_8, V_9 when used	Reciprocal depression ONLY ST Elevation ≥ 2 mm

*Adapted from: Reference 12

Natural Evolution of ECG Changes in STEMI. The ECG undergoes predictable changes during the course of an untreated STEMI. The very first signs on the ECG are hyperacute T waves (*see Figure 3*), which appear in the first few minutes after vessel occlusion. However, they are transient and are not often seen because most patients usually have not arrived at the hospital. Following a variable period of time (hours to days), the ST segments will return to the normal baseline. Up to 95% of inferior but only 40% of anterior ST segment elevations are resolved in 2 weeks.[13] Persistence of elevation for more than 2 weeks is associated with greater morbidity, as 60% of these patients develop a ventricular aneurysm.[13]

Later in the course of a STEMI when the ST segments are no longer elevated, deep T wave inversions usually appear in the leads where the ST segments were elevated, sometimes as early as 72 hours after MI. The T wave inversions can resolve over time (days, weeks, even months) or remain permanent. Q waves also appear in these leads, but are not always seen in all STEMIs. They can develop in as

Figure 1: Serial ECG Changes in MI



Example of serial ECG changes of acute MI (A-C) and benign early repolarization (D). Note that in A-C the first tracings are non-diagnostic for ST elevation, but that in only 30 minutes the ST changes have dramatically worsened. This illustrates the importance of obtaining serial ECGs in some patients. Used with permission from: Marx JA, ed. *Rosen's Emergency Medicine*, 6th edition. Elsevier: Oxford, UK; 2006: fig 77-20.

little as 1-2 hours after onset of symptoms or may take 8-12 hours.[12] Q waves also can be produced by some subendocardial (non-transmural) infarct. The presence of Q waves is not always pathologic and up to 12% of healthy young men can have inferior Q waves.[14] Normal Q waves are narrow with a duration of less than 0.04 seconds, and of low amplitude of less than one-third the size of the accompanying R wave.

Non-STEMI. In non-STEMI, the area of infarcting muscle is smaller, in most cases because a smaller or branch coronary vessel is occluded instead of a primary vessel. Subendocardial ischemia/infarction also can occur without vessel occlusion because this area of the heart is the most distant from coronary supply. When chronic hypertension leads to a thickened left ventricle, the combination of increased blood pressure and increased distance from coronary flow can produce areas of ischemia without vessel occlusion. The ECG shows ST segment depression instead of elevation. In some cases, deep T wave inversions are seen instead of ST segment depression, which is thought to be due more to ischemia (unstable angina) than infarction. The term Wellen's syndrome is used to describe deep T wave inversions in leads V_1-V_4, and this pattern correlates with LAD stenosis.[15]

Non-ischemic conditions also can cause ST segment depression, including LVH, left or right bundle branch blocks, digitalis effect, hypokalemia, and cardiomyopathy.

ECG Diagnosis of MI—Classic Patterns

In patients with normal coronary anatomy, the infarct-related vessel's location on the heart will produce typical changes on the ECG. (*See Table 1.*) (*See Table 3.*)

Anterior MI. Anterior STEMI is produced by a thrombus in the left anterior descending artery (LAD), which supplies the anterior surface of the left ventricle. ST segment elevations are seen in leads V_2-V_4, with reciprocal changes in inferior leads (II, III, and aVF). The elevations can be quite large (4-6 mm or more) and have a rounded appearance. The rounded appearance and increased mortality associated with anterior MI gives the term "tombstones" for the ST segment elevations. Variations on this pattern include anterior-lateral MI, anterior-septal MI, and anterio-inferior MI. Lateral involvement is seen when elevations include leads V_5-V_6, and septal extension is noted with elevation in V_1-V_2 primarily. When the LAD wraps around the apex of the heart, it supplies the inferior wall (typically territory of the right coronary artery, and therefore ST segment elevation may be seen in inferior leads instead of reciprocal depression.

Anterior STEMIs can be more subtle. One example is when an RBBB is produced by occlusion of the first septal branch off the LAD. The patient will have a new RBBB, which can make the ST segment elevations much more difficult to see. (*See Figure 5.*) Another example is when the ECG is taken early in the course of the MI and only hyperacute T waves are present. (*See Figure 3.*) If the ECG is not repeated, the later ST elevation will be missed.

Inferior MI. In the majority of cases (80-90%), inferior STEMI occurs by thrombus in the right coronary artery.[16] The right coronary is said to be dominant, i.e., supplies the right ventricle and 20-25% of the left ventricle, in about 80-90% of people. In the remainder of people, the circumflex is dominant and wraps around the apex of the heart to supply the right ventricle as well. Therefore, a clot in the circumflex also can produce inferior STEMI changes.

ST segment elevations are easiest to see in lead III followed by lead aVF and lead II. Reciprocal depression usually is seen in lead aVL, and if this is absent, the diagnosis of acute inferior MI should be reconsidered. ST depression also seen in V_1-V_4 is indicative of posterior wall injury. Bradycardia is common in inferior MI due to ischemia of the AV node.

ST segment elevation in V_1-V_3 with inferior MI suggests right ventricle involvement, which occurs in about 30% of inferior MIs.[16] Right ventricu-

Table 2: ECG Interpretation and Missed MI[2,6,10]

11–25% of missed MI patients are discharged with 1–2 mm ST segment elevation on their ECG

Most common ECG misdiagnoses of MI are:
- Left ventricular hypertrophy
- Benign early repolarization
- Left ventricular aneurysm

Sensitivity of presenting ECG for acute MI can be as low as 50%
- Do not place too much emphasis on a normal/non-specific ECG in a patient who is pain-free at the time.

Serial ECGs can be essential for accurate diagnosis of acute MI
- If available, compare pre-hospital ECG with one(s) done in the ED.
- Can repeat every 30 minutes to 4 hours
- Look for expected evolutionary changes (*Figure 1*).
- 20% of MI patients with non-diagnostic ECG will develop MI findings in first few hours after arrival.

Reciprocal changes increase likelihood of acute MI when present
- Absent in 66% of anterior MIs
- Absent in only 20% of inferior MIs
- Do not discount acute MI if reciprocal changes are absent.

Besides ischemia, ST segment depression can also arise from:
- LVH with strain
- Left or right BBB
- Digitalis effect
- Hypokalemia
- Cardiomyopathy

Consult a cardiologist for assistance interpreting difficult or questionable ECGs

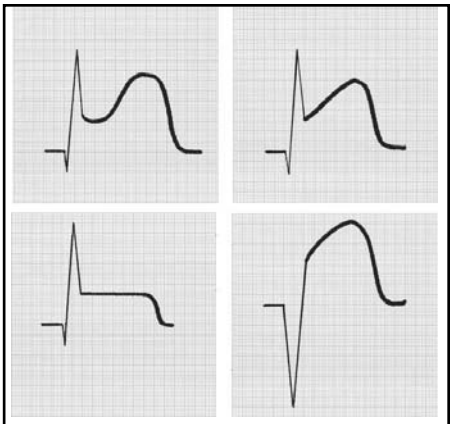
lar involvement almost always is due to occlusion of the right coronary proximal to the RV branch.[17] Rarely, RV infarction can occur from occlusion of a dominant left circumflex that supplies the right ventricle as well. One recent study suggests the possibility of using ST depression in lead aVL as a sign of RV infarction,[18] finding that ≥ 1 mm ST depression in aVL was 87% sensitive, 91% specific, with a diagnostic accuracy of 89% for detecting right ventricle infarction among inferior MI patients.[18]

Ultimately, right ventricle MI is confirmed by taking a right-sided ECG and finding ST elevation in V_4R. ST elevation in V_4R has a sensitivity and specificity of > 90% for right ventricle infarctions.[19] However, ST elevation in V_4R is often transient in inferior STEMI, may be very subtle (just 1 mm), and may fade quickly after only a few hours.

Inferior STEMI can be difficult to detect because the degree of ST elevation can vary from 8 mm to barely 1 mm. Comparison with old ECGs when available may help highlight these subtle changes, and one also can obtain serial ECGs more frequently (every 10-15 minutes) to look for evolution to more obvious STEMI findings.

Lateral MI. Isolated lateral STEMI manifests with ST segment elevation in V_5, V_6, aVL, and lead I, and usually results from blockage of the left circumflex artery. Lateral MIs also can appear simultaneously with posterior MI or as posterior/inferior MI with occlusion of a dominant circumflex. Blockage of a dominant right coronary also will give an inferior/posterior/lateral MI pattern. The ECG is less sensitive for detecting lateral MIs than anterior or inferior MIs. ST elevation in lateral leads is generally small (> 2 mm in only 5%).[20] Only 66% have both ST elevation and reciprocal depression, 30% have isolated ST depression, and 33% have neither ST elevation nor depression.[21] Although not common, about 10% of lateral MIs also will have right ventricle infarction from occlusion of a dominant circumflex.[17]

Figure 2: Examples of Different ST Morphology Seen in Acute MI



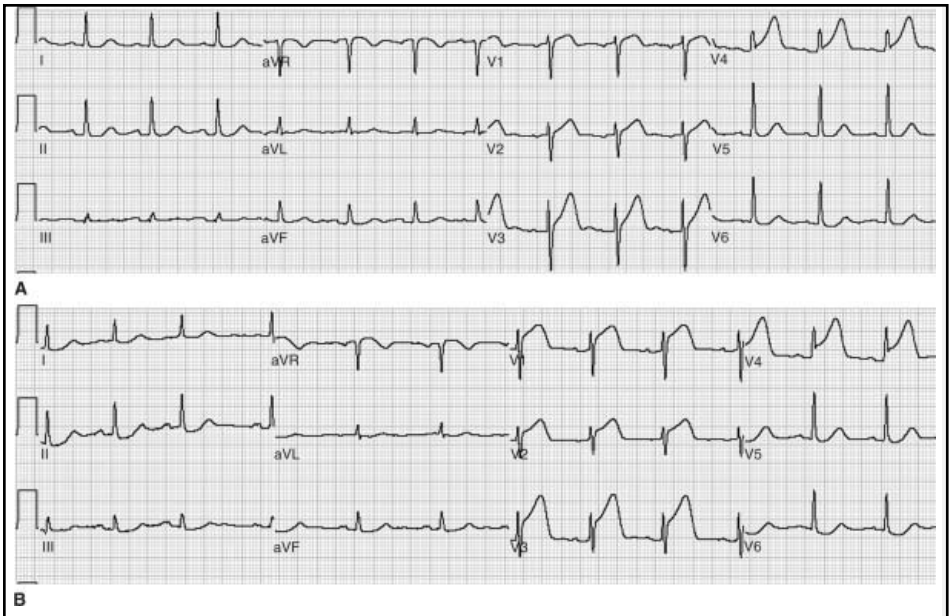
Note that the ST segment can be concave upward (top left), flat (bottom left), or convex upward (bottom right). Used with permission from: Goldberger AL. *Clinical Electro-cardiography: A Simplified Approach*, 7th ed. Elsevier: Oxford, UK:2006:fig 8-6.

Difficult-to-Detect MIs on ECG

Several MI patterns are more difficult to diagnose. They include posterior MI, MI in the presence of LBBB, and MI with a paced rhythm. (*See Table 4.*)

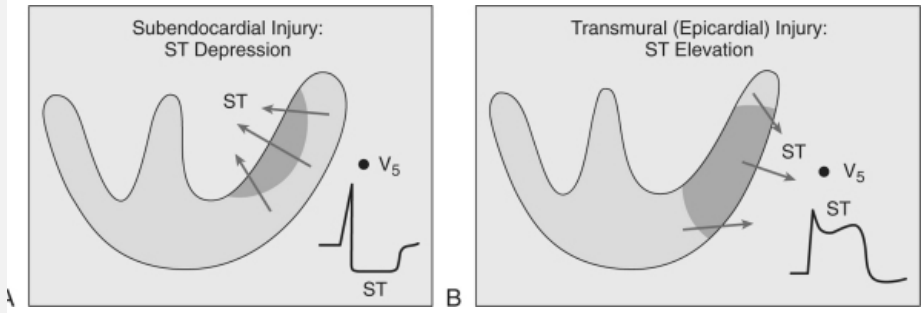
Posterior MI. A posterior STEMI

Figure 3: Hyperacute T Waves



The top ECG shows very early findings of broad, large "hyperacute" T waves in leads V_3 and V_4. Compare this to the bottom ECG, which shows the same patient 30 minutes later. ST elevation is now obvious in V_1-V_4 and reciprocal ST depression is evident in leads V6, II and III. Used with permission from: Marx JA, ed. *Rosen's Emergency Medicine*, 6th edition. Elsevier: Oxford, UK; 2006: Fig. 77-1.

Figure 4: Pathogenesis of ST Segment Changes



Anatomic differences in the location of myocardial ischemia and their corresponding ECG findings. Note how vectors align to produce ST segment depression with subendocardial injury and elevation with transmural injury. The ST depression seen in subendocardial injury is not the same as reciprocal changes that occur with STEMI. With STEMI, the reciprocal depression is seen because the ECG leads are looking at the ST elevation from the opposite side causing vectors to be moving away from the lead instead of toward it. Used with permission from: Libby P, Bonow RO, Mann DL. *Braunwald's Heart Disease: A Textbook*, 8th ed. Elsevier: Oxford, UK; 2008: fig 12-36.

is unique in that it is the only STEMI that does not produce ST elevation on the standard 12-lead ECG. As noted, posterior MI will have reciprocal ST segment depression in leads V_1-V_4.

The ST elevation from isolated posterior MI will be seen only when the 15-lead ECG is used, adding leads V_7-V_9. The additional leads are placed on the fifth intercostal space; V_7 at the posterior axillary line, V_8 at the scapular tip, and V_9 at the left paraspinal border. The criteria for posterior MI is only 1 mm of ST elevation in these leads, not 2 mm as in precordial leads. Some even have suggested using 0.5 mm of ST elevation to increase sensitivity.[22]

Posterior MIs are much less common than other STEMIs, making up only 3-8% of STEMIs.[23] The ECG pattern can be similar to that of anterior ischemia and, as expected, posterior MIs are missed much more often than other STEMIs.[22] However, on close evaluation, the anterior ST segment depression in posterior MI often is upsloping compared to the downsloping segments in anterior ischemia. Normally, there also is an upright R wave and an upright T wave with posterior MI, whereas with anterior ischemia the T wave often is flipped. In addition, the ST changes in anterior ischemia often extend throughout precordial leads to include V_6, whereas in posterior MI, V_5 and V_6 are not usually affected.

LBBB with MI. MI in the presence of LBBB is a well-known example of a difficult ECG diagnosis. Although LBBB is seen only in about 1% of the general population, the incidence increases with age, and roughly 6-9% of acute MI patients will have LBBBs.[24] LBBB naturally produces ST-T wave changes that, as a general rule, mimic or hide the presence of acute MI. (*See Insert Figure 6.*) However, in some cases, STEMI still can be detected using the Sgarbossa criteria.[25]

The Sgarbossa criteria consist of three ECG findings used to detect STEMI in the presence of LBBB. They are, in order of decreasing sensitivity, ST elevation of $\geq$ 1 mm in more than one lead concordant (in the same direction) with the QRS complex; ST depression of $\geq$ 1 mm in leads V_1-V_3; and ST elevation of $\geq$ 5 mm discordant with the QRS complex. (*See Insert Figure 6.*) Concordant ST elevation should be present in the leads in which the QRS is predominantly positive (typically leads V_5, V_6, I, II, and aVL). More recent studies have found that discordant 5 mm ST elevation was seen in only

Table 3: Tips to Diagnose STEMI by ECG[20,65]

Inferior MI
- Lead III is best location to see ST elevation
- Lead aVL is best location to see reciprocal depression
- Reciprocal depression is seen in 80% of inferior MIs
- Right ventricle involvement in 30% of inferior MIs
- Only requires 1 mm ST elevation in lead V_4R

Anterior MI
- When present, hyperacute T waves are seen more often with anterior MI
- May need comparison with old ECG to identify them
- Pseudo-elevation from LVH, BER, LBBB will affect anterior MI identification most

Lateral MI
- Can occur with inferior or anterior MI or in isolation
- Isolated lateral MI often has small ST elevation (< 2 mm)
- 10% also will have right ventricle involvement

Posterior MI
- STEMI that does not produce ST elevation on standard 12-lead ECG
- ST elevation only seen in leads V_7–V_9 and only 1 mm elevation is required
- Look for an upright R wave, upsloping ST depression with an upright T wave to distinguish from anterior ischemia

26% of patients with acute MI, and this finding alone may not be useful.[26] However, the other two Sgarbossa criteria remained effective in a recent meta-analysis.[27]

RBBB with MI. The presence of a RBBB does not impede the diagnosis of acute MI nearly as much as an LBBB, but it still can be problematic as it may be difficult to see where the QRS complex ends and the ST segment begins. (*See Figure 5.*) One can determine the length of the QRS in unaffected leads and apply that duration to the lead in question. In STEMI patients with RBBB, the T wave inversion and ST segment depression normally seen in leads V_1 to V_3 due to the RBBB can hide the ST segment elevation due to the infarction.[28]

Paced Rhythm with MI. Standard right ventricle pacemakers produce an ECG similar in appearance to that of an LBBB and, as a result, acute MI is hard to diagnose in a paced rhythm. Leads V_1-V_3 in some patients can show conspicuous ST elevation simply as a result of the pacing. The Sgarbossa criteria can aid in detection of acute MI in paced rhythms.[29] However, the Sgarbossa criteria in paced rhythms are not as accurate as for LBBB. The most accurate marker is discordant $\geq$ 5 mm ST elevation most often seen in leads V_2-V_4.[30] This was found to be only 53% sensitive, but had a higher specificity of 88%. Two other criteria were suggested and may be helpful in some cases, but were not found to be statistically significant.[29] Some physicians suggest it may not be possible to accurately identify an acute MI with a ventricular paced rhythm.

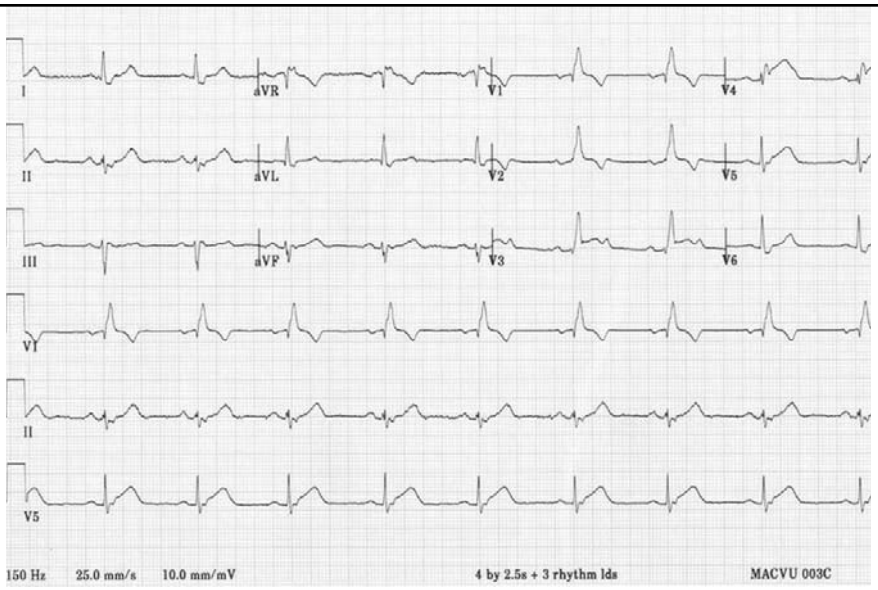
ECG Patterns that Mimic STEMI

The other side of the coin in ECG diagnosis of acute MI is being aware of conditions that can simulate acute MI when it is not present. The sensitivity of ST elevation alone for acute MI is not particularly high, largely due to these other conditions that also produce ST changes. One series found that in ED patients with chest pain and ST elevation, only 15% were from acute MI while the other 85% were from non-MI diagnoses: 25% were from LVH, 15% from LBBB, 12% from early repolarization, and the remainder were from RBBB, LV aneurysm, pericarditis, with 17% undefined.[31] Given the pressures on ED staff to reduce door-to-drug times[32] and the pay-for-performance incentives in place at many hospitals to meet pre-set timelines in treatment of acute MI, one must be extra careful to keep the number of patients misdiagnosed with acute MI to a minimum as well. While these patients do not experience the same increased mortality rates as do MIs that are missed and sent home, they still may undergo unnecessary risk from invasive procedures and/or thrombolytic drugs.

The incidence of patients who are misdiagnosed with acute MI is unknown. One older study of 93 patients who were all given thrombolytic therapy for presumed acute MI found that 11% did not actually have an acute MI.[33] Of these 9 patients, 30% instead had early repolarization, 30% had LVH, and the other 30% had intraventricular conduction delays (i.e., bundle branch blocks).[33] A more recent study of 820 patients referred for urgent catheterization who initially were diagnosed with acute MI found that 2.3% were not having an MI.[34] A third larger study of 1345 patients referred for emergent angioplasty found 14% had no coronary lesion identified.[35]

Benign Early Repolarization. Benign early repolarization (BER) is a variant of the normal ECG, seen in 1-5% of healthy adults, often in young men (< 40 years), especially athletes or those with natural bradycardia.[36] It also has been associated with black men age 20-40 years, but some have disputed this connection.[37] The exact basis of the changes seen in BER is not proven, but one theory is that they arise from an early repolarization of the subepicardium relative to the subendocardial area. As a result of the

Figure 5: **Acute MI in RBBB**



Thanks to Dr. James V. Ritchie, MD, for use of the ECG tracing.

rapid repolarization, mild ST elevation occurs. The following criteria are used to define BER: ST elevation with upward concavity of the initial segment, notching or slurring of the terminal QRS complex (J point), symmetric and concordant large T waves, widespread or diffuse ST elevation, and temporal stability.[38] (*See Insert Figure 7.*)

Correct identification of BER is important in ED chest pain patients not only to keep from confusing this with ACS, but also because of the relatively high frequency with which it is seen in ED populations. Although only seen in 1-5% of the general population, several series have found anywhere from 13% to 48% of ED chest pain patients have BER on their ECG.[39] One useful feature to identify BER is J point elevation. The J point refers to the junction where the QRS ends and the ST segment begins. In BER, J point elevation usually is < 3.5 mm, and the ST segment appears to be evenly lifted from the baseline, thus preserving the normal upward sloping initial ST segment shape.[39] A J point that is notched or irregular also is suggestive of BER. The tall T waves always should be concordant with the QRS complex as well. ST segment elevation usually is present in leads V_2-V_5, and elevation in limb leads is less common. The up-sloping or concave ST segment is only a guideline, as the ST segment also can be concave early in acute MI.[40]

Left Ventricular Hypertrophy (LVH). LVH is a common finding on ECGs, seen in roughly 3% of men and 1.5% of women.[40] ECG criteria for LVH include increased height of the QRS complex with tall R waves (11-13 mm in aVL) in leads I, aVL, V_5, and V_6 and deep S waves in leads V_1 and V_2. Altogether, 70% of patients with LVH changes on their ECG will have ST-T wave changes.[41] ST segments can be elevated or depressed, and T waves can be tall or inverted. (*See Insert Figure 8.*) The abnormal repolarization changes in LVH naturally produce baseline ECG changes that can hide or mimic acute MI. One study found misinterpretation of the LVH changes was responsible for 70% of patients initially being misdiagnosed with ACS who later were found not to have acute MI.[42]

There are two useful features one can use to distinguish acute MI from LVH in the ECG. First, the shape of the ST segment in LVH tends to be concave upward, while in acute MI it tends to be convex downward or flat. (*See Figure 8.*) The strain pattern of lateral ST depression with inverted T waves (strain pattern) often is seen in leads V_3-V_6 as a "mirror image" of the ST elevation in leads V_1-V_2. (*See Insert Figure 8.*) When present together, this combination can be reassuring that ECG changes are due to LVH and not acute MI.[43] The second method is to use serial ECGs. The changes of LVH will not progress over the next several hours like those of an acute MI do.

Acute Pericarditis. Patients with acute pericarditis and ST elevation make up about 1% of ED patients with ST elevation.[28] Their clinical presentation can either be with pleuritic chest pain or with pain similar to that of acute cardiac ischemia. Substernal chest pressure was the presenting complaint in 36% of patients with pericarditis in one recent series.[44] Although not always present, the chest pain classically is worse when supine and improved when sitting up. Viral prodromes are noted in about 50% of patients, and up to 60% of patients present with fever.[45] Patients also can present with dyspnea and have pericardial friction rubs. Cardiac biomarkers often are elevated in acute pericarditis. One recent study found that troponin I levels were elevated in 32% of patients with pericarditis but, unlike acute MI, higher troponin levels were not associated with worse outcomes.[46]

ECG changes seen in acute pericarditis can be confused with those of acute MI, and the distinction is important to avoid treating these patients with anticoagulants or thrombolytics that theoretically could induce bleeding in the pericardial space and lead to tamponade. Cardiac tamponade after systemic thrombolytics has been documented in large series to occur in about 1% of cases.[47]

The well-known ECG changes seen in pericarditis are those of diffuse ST segment elevation and PR segment depression. However, a variety of other ECG changes also can occur, including T wave inversions, ST segment depression, prolonged QT interval, bundle branch blocks, and even Q-waves.[44] These changes result from injury currents caused by general irritation of the myocardial surface and can appear similar to STEMI on first glance. ECG findings in acute pericarditis also tend to evolve in four

Table 4: Difficult ECG Patterns[25,44,46]

Difficult to Detect MIs
LBBB with MI — Sgarbossa criteria, in order of decreasing sensitivity:
• ST elevation ≥ 1 mm in more than one lead concordant (in the same direction) with the QRS complex
• ST depression of ≥ 1 mm in leads V_1–V_3
• ST elevation of ≥ 5 mm discordant with the QRS complex
• Non-STEMI cases will not have any of these changes
Paced Rhythm with MI
• Sgarbossa criteria not sensitive in this situation
• Studies suggest most accurate marker is discordant ≥ 5 mm ST elevation most often seen in leads V_2–V_4
• Some suggest ECG in pace rhythm is always non-diagnostic of MI
ECG Patterns that Mimic STEMI
Left ventricular hypertrophy
• Common reason for misdiagnosis of ACS/acute MI when not actually present
• ST segment usually concave upward in LVH, convex downward or flat in MI
• ST elevation with LVH only in leads V_1–V_2
• Inverted T waves only in leads V_3–V_6 with LVH
Benign early repolarization
• ST elevation with upward concavity of the initial segment
• Notching or slurring of the terminal QRS complex (J point)
• J point elevation is < 3.5 mm
• Symmetric and concordant large T waves
• Widespread or diffuse ST elevation
• Temporal stability — no reciprocal changes
• Narrow QRS
Pericarditis
• Troponin elevations seen in 32% of patients with acute pericarditis
• Diffuse ST elevation is common but can be regional (inferior distribution most common)
• PR depression is "almost" diagnostic
• Reciprocal PR elevation may be easier to see in lead aVR
• Without PR segment changes, may not be able to distinguish from acute MI
Left ventricular aneurysm
• Most commonly seen in anterior distribution but can occur in inferior and posterior areas too
• May be very hard to distinguish from acute MI changes
• Look for lack of reciprocal changes, presence of Q waves, previous ECG comparison, lack of rising biomarkers
Sub-arachnoid hemorrhage
• Inverted T waves with prolonged QT interval
Brugada syndrome
• ST elevation downsloping or upsloping, but only in leads V_1–V_3
• Also has inverted T in same leads
• Much more common in South Asian men
• Can lead to spontaneous ventricular fibrillation

classic stages: ST elevation seen in Stage 1, resolution of ST elevation in Stage 2, T-wave inversions in Stage 3, and normalization of the ECG in Stage 4.[44] These stages can occur in an unpredictable fashion. Some patients pass through Stages 1-3 in only a few hours, while others may take weeks to do so. Yet others will not exhibit all four stages and may progress directly from Stage 1 to Stage 4.

Fortunately, several useful clues can help differentiate acute pericarditis from STEMI. The single most useful finding is that of PR segment depression. PR depression arises from inflammation of the atria and is considered "almost diagnostic" of acute pericarditis.[41] PR depression is best seen in leads V_5, V_6, II, III, and aVF, while reciprocal PR elevation can be seen in aVR and may be more obvious than PR depression.[48] ST elevation usually is diffuse and essentially is seen in every lead except aVR and V_1. Thus, the elevation overlaps the territory of all three coronary vessels, which is atypical for STEMI. Reciprocal ST depression also can occur in lead aVR. More focal ST elevation also can occur with more localized inflammation[49,50] and, when present, is seen most often in inferior leads.[44] ST elevation can vary from 1-4 mm, and is rarely > 5 mm. The ST segment most often is concave, but can be convex or flat as in elevation in acute MI. T wave inversion can follow the ST elevation as in acute MI and is seen in leads where ST elevation occurred.

In the absence of PR depression, the ECG alone may not distinguish between acute pericarditis and acute STEMI.

Persistent ST Elevation After MI (LV Aneurysm). In some cases, typically after a large transmural MI, ST segment elevation remains present for more than six months.[51] The majority of cases with persistent ST elevation seem to follow anterior MI but also can develop on the inferior and posterior walls. In the past, persistent ST elevation on the ECG commonly was associated with an LV aneurysm, and even some recent studies found that 30% of patients with persistent elevation did

show evidence of LV aneurysm by echocardiography.[52] However, others suggest this is not the case.[53]

LV aneurysm is a complication that can follow large transmural MIs when a larger area of cardiac muscle dies and is replaced by a relatively weak, thin layer of necrotic muscle and fibrous scar that bulges out during systole. The LV aneurysms range from smaller ones only 1 cm in diameter to large ones up to 8 cm.[53] Unlike pseudoaneurysms, which actually are contained myocardial wall ruptures, true cardiac aneurysms rarely lead to rupture. Even if aneurysm is not present, persistent ST elevation is associated with large areas of damage from acute MI. Only 10% of patients with anterior wall scars < 30% developed persistent ST elevation, but 78% of those with scars ≥ 50% did.[50] Thus, the risk of persistent ST elevation correlates with the size of the previous infarct, but the mechanism for the persistent elevation remains unclear.

In some patients, persistent ST elevation after MI can appear very similar to an acute anterior MI. In other cases, the ST segments only partially return to normal, and the T waves can stay inverted, giving the suggestion of a late presentation of acute MI. Three features can be used to help differentiate LV aneurysm from STEMI. First, reciprocal changes will not be present, and the expected evolution of the ST segments/T waves also will not occur. In addition, Q waves will be present in the same distribution as the elevation. The T wave amplitude/QRS amplitude did help distinguish LV aneurysm from acute MI.[54] A ratio > 0.36 in any single lead likely was caused by acute MI, while a ratio < 0.36 in all leads was associated with LV aneurysm.[54]

Subarachnoid Hemorrhage. ECG changes occur in 60-70% of patients with intracerebral hemorrhage and 40-70% of those with subarachnoid hemorrhage (SAH).[55] These changes also have been described less often with ischemic stroke and head trauma. Inverted T waves associated with prolonged QT intervals are the most common finding, but ST segment changes and large U waves occur as well.[56] Deeply inverted T waves due to cardiac sources are associated with normal QT intervals, while those from stroke or SAH present with prolonged QT intervals.[57] When ST segment elevations are present, regional wall motion defects not due to coronary disease are seen.[56] These cardiac changes often spontaneously resolve over several days in patients who survive their CNS pathology. This recovery is similar to Takotsubo syndrome, and catecholamine release is hypothesized as a cause for both conditions.[58] Elevated troponin also can be seen and, as expected, is associated with increased mortality.[58]

In most cases, the patient's history (chest pain vs. headache) would keep one from going astray, but patients who present with mental status changes may not be able to give this key information.

Brugada Syndrome. The Brugada syndrome is a genetic disorder with autosomal dominant transmission associated with sudden cardiac death.[59] The patients have normal hearts and no coronary disease but have several abnormalities on their ECGs: an RBBB pattern with ST segment elevation on the right precordial leads.[59] The syndrome has been traced to a defect in the cardiac sodium channel, which increases risk of spontaneous ventricular tachycardia/fibrillation.[60] The sodium channel defect is not always evident, but can be worsened by higher body temperatures and type I antidysrhythmics.[60] The syndrome also has been uncovered in patients with cocaine toxicity[61] or fever.[62] Brugada syndrome is associated with ethnic populations (e.g., Southeast Asians), where it is estimated to cause 40-50% of cases of spontaneous ventricular fibrillation[63] and 4-12% of all sudden unexpected deaths. Although less common, Brugada syndrome also has been described in Caucasians and Hispanics.[62,64]

Brugada syndrome is relevant to this discussion because the ST elevation seen in the baseline ECG can simulate acute MI. (*See Insert Figure 9.*) More importantly, the Brugada pattern is not static and can be a transient finding[64] perhaps leading to confusion with serial ECG interpretation. In many patients, the pattern can appear and completely disappear, or it can change between the two variants. (*See Insert Figure 9.*) Patients often self-terminate from their ventricular dysrhythmias and present only with syncope. The pattern often is first detected in young adults but also is seen in school-age children. Other than syncope or cardiac arrest due to ventricular fibrillation, no other symptoms are ascribable to Brugada syndrome. Therefore, the presence of ST elevation in leads V_1-V_3 in an otherwise asymptomatic patient should prompt concern for the diagnosis. Consultation with a cardiologist for assistance with ECG interpretation and disposition of the patient is recommended.

Conclusion

While important, the ECG is only an imperfect tool for the diagnosis of acute coronary syndrome. Understand its limitations and learn to use the ECG as an adjunct to, and not a replacement for, your clinical judgment.

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Physician CME Questions

121. What percentage of AMI patients have a diagnostic (ST segment elevation) initial ECG?
   A. about 30%
   B. about 50%
   C. about 70%
   D. almost 100%
122. According to recent data, how many patients are discharged from the ED with ST elevation on their ECGs?
   A. 2%
   B. 5%
   C. 11%
   D. 15%
123. Acute posterior MIs:
   A. do not produce ST elevation on standard 12-lead ECGs even though they are considered a STEMI
   B. can be diagnosed by 1 mm ST elevation on leads V_7-V_9
   C. are missed more often than other MIs (inferior, anterior, lateral)
   D. usually do not show ST changes in V_5 and V_6
   E. all of the above
124. The most accurate of the Sgarbossa criteria to diagnose acute MI in a LBBB ECG is:
   A. ST elevation of ≥ 1 mm in more than one lead concordant (in the same direction) with the QRS complex
   B. ST depression of ≥ 1 mm in leads V_1-V_3
   C. ST elevation of ≥ 5 mm discordant with the QRS complex
   D. All are equally sensitive
125. The most common cause of ST elevation on ECGs in ED patients is:
   A. acute MI
   B. pericarditis
   C. LVH
   D. LBBB
   E. early repolarization
126. The best way to tell acute MI from LVH on the ECG alone is:
   A. the shape of the ST segment is concave upward
   B. the shape of the ST segment is convex upward
   C. presence of flipped T waves
   D. ST elevation is only in leads V_1 and V_2
   E. expected evolution of ECG changes over time
127. The most sensitive ECG finding to diagnose pericarditis is:
   A. diffuse ST elevation
   B. lack of reciprocal changes
   C. lack of T wave inversions
   D. PR depression
128. Reciprocal ECG changes are:
   A. more common in anterior compared to inferior MIs
   B. about equal in anterior and inferior MIs
   C. less common in anterior compared to inferior MIs
   D. not seen in acute MIs
129. Which of these neurologic conditions is more likely to produce T wave changes on the ECG?
   A. head trauma
   B. subarachnoid hemorrhage
   C. ischemic stroke
   D. intracerebral hemorrhage
130. All of the following statements regarding the Brugada syndrome are true *except*:
   A. It has a stable ECG pattern.
   B. It is more common in Southeast Asians.
   C. It has ST segment elevation on the anterior precordial leads.
   D. It can produce syncope.

CME Answer Key: 121. B; 122. C; 123. E; 124. A; 125. C; 126. E; 127. D; 128. C; 129. D; 130. A

Emergency Medicine Reports™ (ISSN 0746-2506) is published biweekly by AHC Media LLC, 3525 Piedmont Road, N.E., Six Piedmont Center, Suite 400, Atlanta, GA 30305. Telephone: (800) 688-2421 or (404) 262-7436.

Associate Publisher: Russ Underwood

Specialty Editor: Shelly Morrow Mark

Director of Marketing: Schandale Kornegay

GST Registration No.: R128870672

Periodicals Postage Paid at Atlanta, GA 30304 and at additional mailing offices.

POSTMASTER: Send address changes to Emergency Medicine Reports, P.O. Box 740059, Atlanta, GA 30374.

Back issues: $31. Missing issues will be fulfilled by customer service free of charge when contacted within one month of the missing issue's date.

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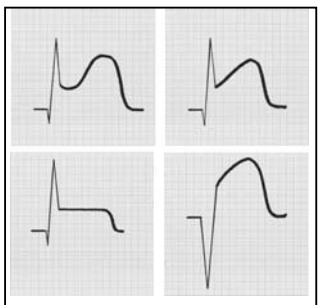
Missed MI: ECG Strategies to Reduce the Risk

Anatomic Location of Acute MI and the Corresponding ECG Changes*

Anatomic Location	ECG Leads	ECG Findings
Anterior	V_2–V_4 II, III, aVF	ST Elevation ≥ 2 mm Reciprocal depression (only in 33%)
Inferior	II, III, aVF aVL	ST Elevation ≥ 1 mm Reciprocal depression (in ~ 80 %)
Right ventricle	V_4R V_1–V_3	ST Elevation diagnostic ST Elevation indicative
Lateral	I, aVL, V_5, V_6	ST Elevation ≥ 2 mm (in precordial leads)
Posterior	V_1, V_2 V_8, V_9 when used	Reciprocal depression ONLY ST Elevation ≥ 2 mm

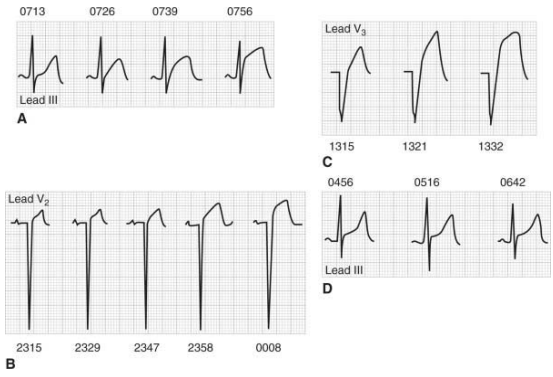
*Adapted from: Reference 12

Examples of Different ST Morphology Seen in Acute MI



Note that the ST segment can be concave upward (top left), flat (bottom left), or convex upward (bottom right). Used with permission from: Goldberger AL. *Clinical Electro-cardiography: A Simplified Approach*, 7th ed. Elsevier: Oxford, UK:2006:fig 8-6.

Serial ECG Changes in MI



Example of serial ECG changes of acute MI (A-C) and benign early repolarization (D). Note that in A-C the first tracings are non-diagnostic for ST elevation, but that in only 30 minutes the ST changes have dramatically worsened. This illustrates the importance of obtaining serial ECGs in some patients. Used with permission from: Marx JA, ed. *Rosen's Emergency Medicine*, 6th edition. Elsevier: Oxford, UK; 2006: fig 77-20.

ECG Interpretation and Missed MI

11–25% of missed MI patients are discharged with 1–2 mm ST segment elevation on their ECG

Most common ECG misdiagnoses of MI are:
- Left ventricular hypertrophy
- Benign early repolarization
- Left ventricular aneurysm

Sensitivity of presenting ECG for acute MI can be as low as 50%
- Do not place too much emphasis on a normal/non-specific ECG in a patient who is pain-free at the time.

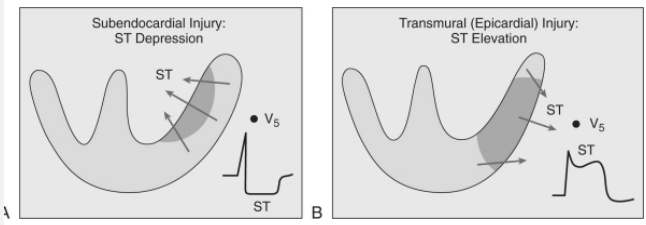
Serial ECGs can be essential for accurate diagnosis of acute MI
- If available, compare pre-hospital ECG with one(s) done in the ED.
- Can repeat every 30 minutes to 4 hours
- Look for expected evolutionary changes (*Figure 1*).
- 20% of MI patients with non-diagnostic ECG will develop MI findings in first few hours after arrival.

Reciprocal changes increase likelihood of acute MI when present
- Absent in 66% of anterior MIs
- Absent in only 20% of inferior MIs
- Do not discount acute MI if reciprocal changes are absent.

Besides ischemia, ST segment depression can also arise from:
- LVH with strain
- Left or right BBB
- Digitalis effect
- Hypokalemia
- Cardiomyopathy

Consult a cardiologist for assistance interpreting difficult or questionable ECGs

Pathogenesis of ST Segment Changes



Anatomic differences in the location of myocardial ischemia and their corresponding ECG findings. Note how vectors align to produce ST segment depression with subendocardial injury and elevation with transmural injury. The ST depression seen in subendocardial injury is not the same as reciprocal changes that occur with STEMI. With STEMI, the reciprocal depression is seen because the ECG leads are looking at the ST elevation from the opposite side causing vectors to be moving away from the lead instead of toward it. Used with permission from: Libby P, Bonow RO, Mann DL. *Braunwald's Heart Disease: A Textbook*, 8th ed. Elsevier: Oxford, UK; 2008: fig 12-36.

Tips to Diagnose STEMI by ECG

Inferior MI
- Lead III is best location to see ST elevation
- Lead aVL is best location to see reciprocal depression
- Reciprocal depression is seen in 80% of inferior MIs
- Right ventricle involvement in 30% of inferior MIs
- Only requires 1 mm ST elevation in lead V_4R

Anterior MI
- When present, hyperacute T waves are seen more often with anterior MI
- May need comparison with old ECG to identify them
- Pseudo-elevation from LVH, BER, LBBB will affect anterior MI identification most

Lateral MI
- Can occur with inferior or anterior MI or in isolation
- Isolated lateral MI often has small ST elevation (< 2 mm)
- 10% also will have right ventricle involvement

Posterior MI
- STEMI that does not produce ST elevation on standard 12-lead ECG
- ST elevation only seen in leads V_7–V_9 and only 1 mm elevation is required
- Look for an upright R wave, upsloping ST depression with an upright T wave to distinguish from anterior ischemia

Difficult ECG Patterns

Difficult to Detect MIs

LBBB with MI — Sgarbossa criteria, in order of decreasing sensitivity:
- ST elevation ≥ 1 mm in more than one lead concordant (in the same direction) with the QRS complex
- ST depression of ≥ 1 mm in leads V_1–V_3
- ST elevation of ≥ 5 mm discordant with the QRS complex
- Non-STEMI cases will not have any of these changes

Paced Rhythm with MI
- Sgarbossa criteria not sensitive in this situation
- Studies suggest most accurate marker is discordant ≥ 5 mm ST elevation most often seen in leads V_2–V_4
- Some suggest ECG in pace rhythm is always non-diagnostic of MI

ECG Patterns that Mimic STEMI

Left ventricular hypertrophy
- Common reason for misdiagnosis of ACS/acute MI when not actually present
- ST segment usually concave upward in LVH, convex downward or flat in MI
- ST elevation with LVH only in leads V_1–V_2
- Inverted T waves only in leads V_3–V_6 with LVH

Benign early repolarization
- ST elevation with upward concavity of the initial segment
- Notching or slurring of the terminal QRS complex (J point)
- J point elevation is < 3.5 mm
- Symmetric and concordant large T waves
- Widespread or diffuse ST elevation
- Temporal stability — no reciprocal changes
- Narrow QRS

Pericarditis
- Troponin elevations seen in 32% of patients with acute pericarditis
- Diffuse ST elevation is common but can be regional (inferior distribution most common)
- PR depression is "almost" diagnostic
- Reciprocal PR elevation may be easier to see in lead aVR
- Without PR segment changes, may not be able to distinguish from acute MI

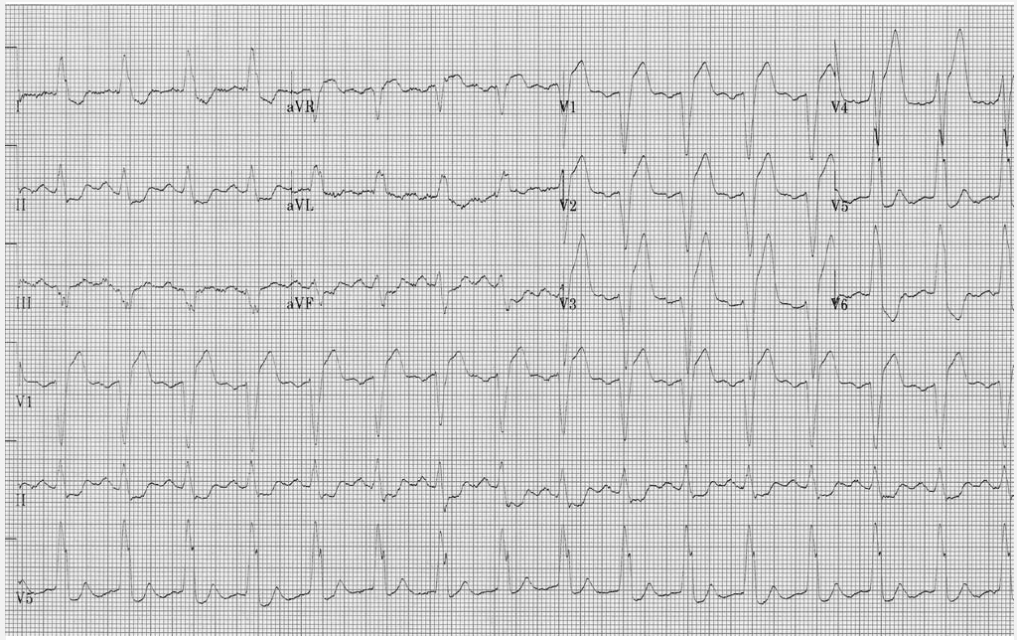
Left ventricular aneurysm
- Most commonly seen in anterior distribution but can occur in inferior and posterior areas too
- May be very hard to distinguish from acute MI changes
- Look for lack of reciprocal changes, presence of Q waves, previous ECG comparison, lack of rising biomarkers

Sub-arachnoid hemorrhage
- Inverted T waves with prolonged QT interval

Brugada syndrome
- ST elevation downsloping or upsloping, but only in leads V_1–V_3
- Also has inverted T in same leads
- Much more common in South Asian men
- Can lead to spontaneous ventricular fibrillation

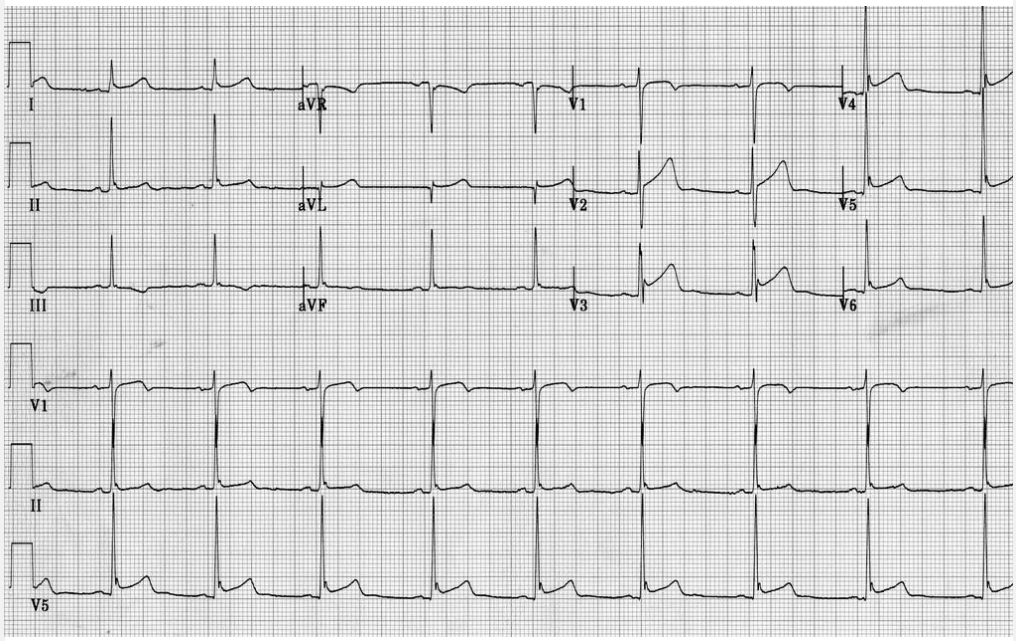
Supplement to *Emergency Medicine Reports*, June 8, 2009: "Missed Myocardial Infarction: ECG Strategies to Reduce the Risk." *Author:* **Gary Hals, MD, PhD**, Attending Physician, Department of Emergency Medicine, Palmetto Richland Memorial Hospital, Columbia, SC.

Figure 6: Acute MI in LBBB



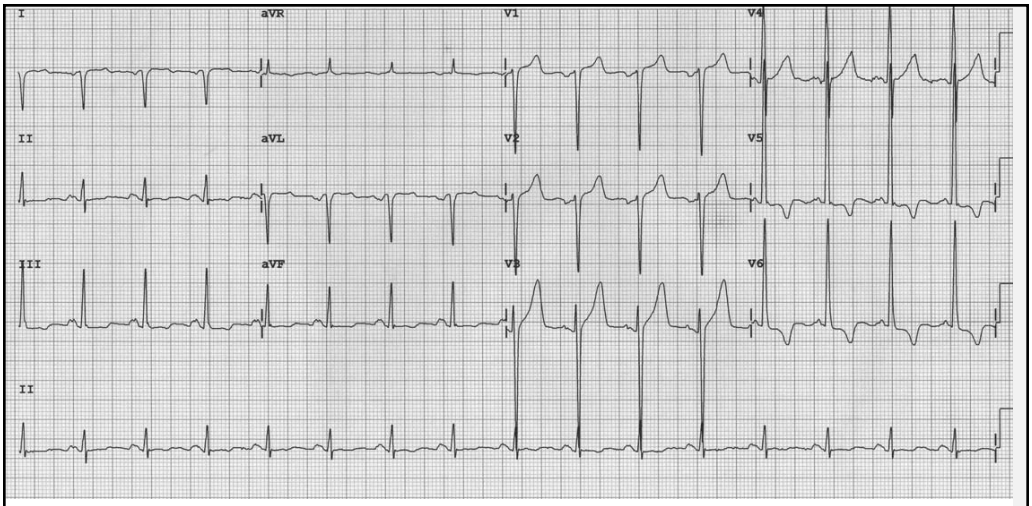
This ECG shows examples of Sgarbossa criteria for > 5 mm ST elevation discordant with the QRS complex (seen in V_2-V_4). Thanks to Dr. James V. Ritchie, MD, for use of the ECG tracing.

Figure 7: Example of Benign Early Repolarization



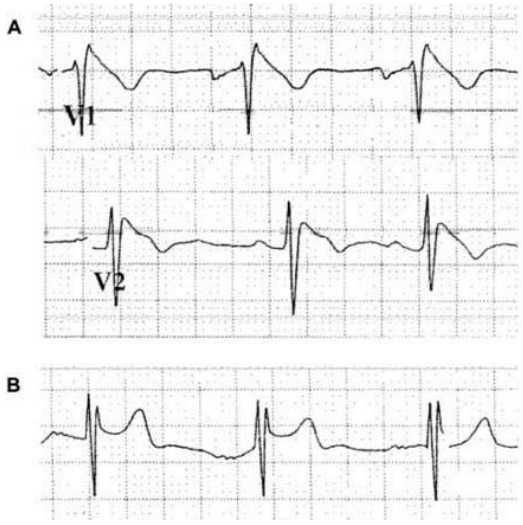
ST segment elevation is seen with concavity of initial segment and widespread ST elevation across the precordial leads.

Figure 8: Examples of LVH with Strain Pattern



There are flipped T waves in V_5 and V_6 with ST elevation in V_1-V_3. The tall R waves and deep S waves in V_1-V_3 and the fact that the T waves are only flipped in V_5 and V_6 are helpful to identify this as LVH with strain.

Figure 9: ST Segment Variants in Brugada Syndrome



This shows example of the "coved" type of ST elevation in the top panel, with the "saddle" type ST elevation in the lower panel. Serial ECGs would be helpful to show lack of evolution of the ECG changes. Used with permission from: Mattu A, Rogers RL, Kim H, et al. The Brugada syndrome. *Am J Emerg Med* 2003;21:146-151.

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CORRECT ● **INCORRECT** ⊘ ✓ ✗ ⚠

1. If you are claiming physician credits, please indicate the appropriate credential: ○ MD ○ DO ○ Other _____

	Strongly Disagree	Disagree	Slightly Disagree	Slightly Agree	Agree	Strongly Agree
After participating in this program, I am able to:						
2. Recognize or increase index of suspicion for specific conditions.	○	○	○	○	○	○
3. Understand the epidemiology, etiology, pathophysiology, and clinical features of the entity discussed.	○	○	○	○	○	○
4. Apply state-of-the-art diagnostic and therapeutic techniques (including the implications of pharmacologic therapy discussed) to patients with the particular medical problems discussed.	○	○	○	○	○	○
5. Understand the differential diagnosis of the entity discussed.	○	○	○	○	○	○
6. Understand both likely and rare complications that may occur.	○	○	○	○	○	○
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12. This activity has changed my clinical practice.	○	○	○	○	○	○

If so, how?_____

13. How many minutes do you estimate it took you to complete this entire semester (13 issues) activity? Please include time for reading, reviewing, answering the questions, and comparing your answers with the correct ones listed. _____ minutes.
14. Would you prefer to test with each issue or by semester?_____ by issue_____by semester (check one)
15. Do you have any general comments about the effectiveness of this CME program?

_____

I have completed the requirements for this activity.

Name (printed) _____ **Signature**_____

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1. A B C D 2. A B C D 3. A B C D 4. A B C D 5. A B C D 6. A B C D 7. A B C D
8. A B C D 9. A B C D 10. A B C D

UNCOMMON BUT IMPORTANT INFECTIOUS DISEASES

11. A B C D E F 12. A B C D 13. A B C D 14. A B C D 15. A B C D 16. A B C D 17. A B C D
18. A B C D 19. A B C D 20. A B C D

EMERGENCY DEPARTMENT CROWDING

21. A B C D 22. A B C D 23. A B C D 24. A B C D 25. A B C D 26. A B C D 27. A B C D
28. A B C D 29. A B C D 30. A B C D

A PAIN IN THE BACK

31. A B C D 32. A B C D E 33. A B C D 34. A B C D 35. A B C D 36. A B C D E 37. A B C D E
38. A B C D E 39. A B C D E 40. A B C D E

ACCIDENTAL HYPOTHERMIA

41. A B C D 42. A B C D 43. A B C D 44. A B C D 45. A B C D 46. A B C D 47. A B C D
48. A B C D 49. A B C D 50. A B C D

ACUTE APPENDICITIS: DIAGNOSIS AND TREATMENT IN 2009: PART I

51. A B C D E 52. A B C D E 53. A B C D E 54. A B C D E 55. A B C D E 56. A B C D 57. A B C D
58. A B C D E 59. A B C D 60. A B C D

ACUTE APPENDICITIS: DIAGNOSIS AND TREATMENT IN 2009: PART II

61. A B C D 62. A B C D 63. A B C D 64. A B C D 65. A B C D 66. A B C D 67. A B C D
68. A B C D 69. A B C D 70. A B C D

ONCOLOGIC EMERGENCIES

71. A B C D E 72. A B C D E 73. A B C D E 74. A B C D E 75. A B C D E 76. A B C D E 77. A B C D E
78. A B C D E 79. A B C D E 80. A B C D E

ATYPICAL PRESENTATIONS OF ISCHEMIC CEREBROVASCULAR DISEASE: PART I

81. A B C D E 82. A B C D 83. A B C D E 84. A B C D 85. A B C D E 86. A B C D E 87. A B C D
88. A B C D 89. A B C D 90. A B C D

ATYPICAL PRESENTATIONS OF ISCHEMIC CEREBROVASCULAR DISEASE: PART II

91. A B C D 92. A B C D E 93. A B C D E 94. A B C D E 95. A B C D 96. A B C D 97. A B C D E
98. A B C D E 99. A B C D E 100. A B C D E

MISSED MYOCARDIAL INFARCTION IN THE ED: STRATEGIES TO REDUCE THE RISK

101. A B C D 102. A B C D 103. A B C D E 104. A B C D 105. A B C D E 106. A B C D 107. A B C D E
108. A B C D 109. A B C D 110. A B C D

COMMON MISDIAGNOSES OF MYOCARDIAL INFARCTION

111. A B C D E 112. A B C D E 113. A B C D 114. A B C D 115. A B C D 116. A B C D 117. A B C D E
118. A B C D 119. A B C D E 120. A B C D

MISSED MYOCARDIAL INFARCTION: ECG STRATEGIES TO REDUCE THE RISK

121. A B C D 122. A B C D 123. A B C D E 124. A B C D 125. A B C D E 126. A B C D E 127. A B C D
128. A B C D 129. A B C D 130. A B C D