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Mimics of ST-Elevation Myocardial Infarction (STEMI)

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STATEMENT OF FINANCIAL DISCLOSURE

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Introduction

One common adage in emergency medicine is that ST-segment elevation in a patient with chest pain should be considered an acute myocardial infarction (AMI) until proven otherwise. Like many axioms in emergency medicine, it embodies an "assume the worst and rule it out" approach to patient evaluation. While such a principle concerning ST-segment elevation may be prudent, the electrocardiogram (ECG) is only one factor to consider when assessing patients with chest pain. A patient's history, physical examination findings, and other features of the ECG must be considered when determining the cause of ST-segment elevation. In fact, the majority of ST-segment elevations seen in emergency department (ED) patients with chest pain are not the result of AMI.[1] Thus, it is important for EM physicians to have an understanding of the differential diagnosis of ST-segment elevation.

In 2013, the American College of Cardiology Foundation and the American Heart Association (ACCF/AHA) revised the electrocardiographic definition of ST-elevation myocardial infarction (STEMI) to "ST elevation at the J point in at least two contiguous leads of ≥ 2 mm (0.2 mV) in men (≥ 2.5 mm in men under 40 years old) or ≥ 1.5 mm (0.15 mV) in women in leads V2-V3 and/or of ≥ 1 mm (0.1 mV) in other contiguous chest leads or the limb leads."[2] In the updated guidelines, a new left bundle branch block (LBBB) in isolation no longer is considered a STEMI equivalent. Moreover, the American College of Cardiology (ACC) emphasized that AMI is a syndrome: a constellation of clinical findings, including, but not limited to, findings on the 12-lead ECG that are concerning for an acute infarct, but also including the subsequent release of biomarkers indicative of myocardial necrosis.

The mechanism by which ST-segment elevation occurs in an AMI is incompletely understood; however, what is known is that ST elevation occurs reliably with transmural and subepicardial myocardial infarctions.[3] In a classic study conducted in 1960, ST-segment elevation was described as an "injury current," after observing its presence in a canine myocardium after ligating its supplying coronary artery.[4] In this experiment, the injured myocardium displayed simultaneous areas of depolarized and repolarized tissue, which resulted in ST elevation. A competing theory suggests that the surface of injured myocytes becomes more negatively charged, inducing a positive charge in the surrounding (uninjured) myocytes, which produces ST elevation.[5] Regardless of the mechanism, the final common pathway for ST elevation is the irregularity in repolarization.

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EXECUTIVE SUMMARY

- Early repolarization is the most common reason for false-positive catheterization lab activation in emergency department patients with chest pain.
- New onset left bundle branch block (LBBB) is no longer considered a STEMI equivalent.
- The modified Sgarbossa criteria are useful in identifying an acute myocardial infarction in the presence of LBBB.
- The ratio of the ST segment to the dominant QRS wave > 0.25 suggests an acute infarct in the presence of left ventricular hypertrophy.
- The ST elevation associated with acute pericarditis is diffuse and most prominent in lateral and inferior leads.

There are other conditions that alter repolarization and produce ST elevation but are not STEMI. The purpose of this article is to discuss several of these STEMI mimics, and to help make them memorable with the "ELEVATION" mnemonic: Early repolarization, Left bundle branch block, Electrolytes, Ventricular enlargement, Aneurysm, Thailand (representative of South Asia, where Brugada syndrome has the highest reported incidence), Inflammation (myopericarditis), Osborn (J) waves, and Non-thrombotic vasospasm.

Early Repolarization

Historically, ECGs with an early repolarization pattern have been regarded as a benign variant associated with persistent ST-segment elevation in individuals with no evidence of cardiac disease. (*See Figure 1.*) In addition to ST-segment elevation, slurring or notching on the downstroke of a dominant R wave is common. Early repolarization was initially associated with young healthy athletes, but increasingly is found in a wider variety of individuals.[6] The dramatic appearance of ST segment in multiple contiguous leads results in early repolarization being cited as the most common cause of false-positive catheterization laboratory activations in patients without elevated cardiac biomarkers.[7] Over the years, different authors have used different criteria for the diagnosis of early repolarization. To provide consistency, in 2015, Hancock et al proposed three criteria that are required for the diagnosis of benign early repolarization:[8]

- The QRS slur or notch (termed a J wave) must be on the downslope of the R wave and be above the isoelectric line.
- The peak of the J point must be elevated ≥ 0.1 mV in two or more contiguous leads, except V1-V3.
- The QRS duration must be < 120 ms.

This definition helps clinicians differentiate a normal electrocardiographic variant from ischemic ST elevation. In benign early repolarization, the ST elevation, if present, is described as concave (*see Figure 2, left*), in contrast to a STEMI, which typically has a convex (*see Figure 2, right*) ST elevation morphology. However, the ST-segment convexity only confers a 77% sensitivity for infarction and, therefore, should not be used as the sole discriminating finding between early repolarization and infarction.[9] Smith et al published predictors that help differentiate between subtle anterior wall STEMI over benign early repolarization. These include low R wave amplitude (best measured in V4), greater degrees of ST-segment elevation, and longer computer-measured QTc.[10] These criteria are relatively complex to implement in a clinical setting; however, they offer highly sensitive (86%) and specific (90%) discrimination between anterior wall STEMI and early repolarization.[11]

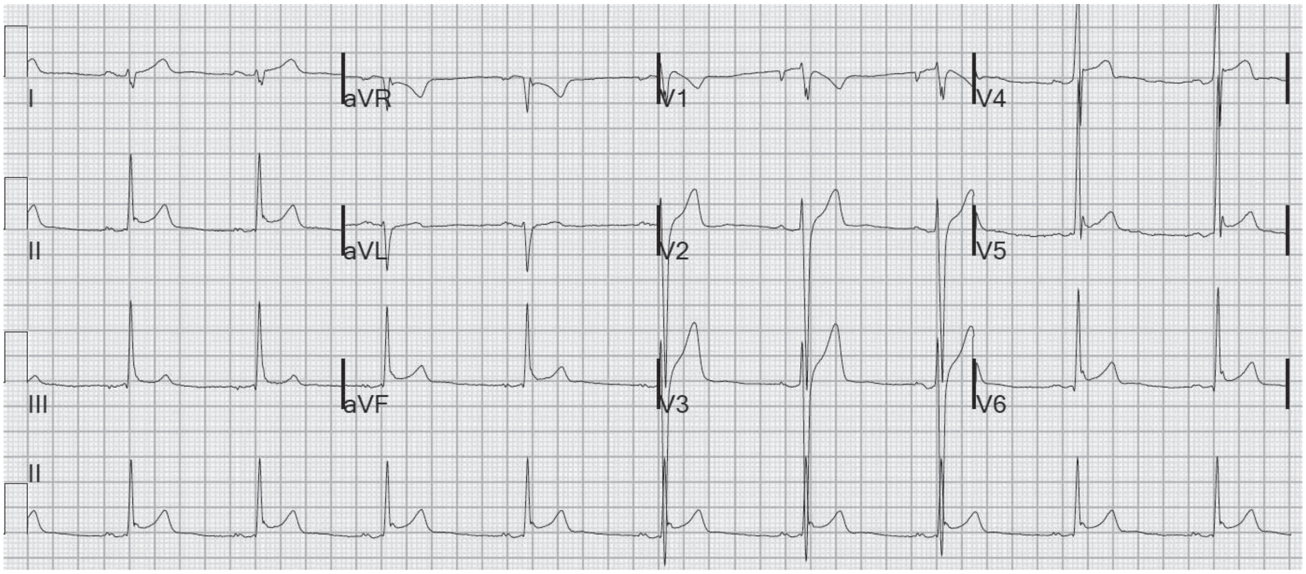
Electrophysiology literature suggests that early repolarization is associated, in some cases, with sudden cardiac death (idiopathic ventricular fibrillation) earning the term "early repolarization syndrome" (ERS) or "J wave syndrome."[12] The J waves seen in ERS may be indistinguishable from those that are true Osborn waves in hypothermia, since the underlying mechanism is identical. In addition, because ERS and Brugada syndrome (covered later in this article) are believed to be part of the same continuum, it is not surprising that both have been associated with polymorphic ventricular tachycardia and ventricular fibrillation (VF).[12] Although the absolute risk of sudden death with ERS remains unclear, it is estimated to be small (lower than that of Brugada syndrome). It is important to differentiate early repolarization pattern from ERS; the former is an ECG variant seen in asymptomatic individuals, whereas ERS is applied only after a documented VF arrest.

Left Bundle Branch Block

LBBBs are characterized by a QRS duration > 120 ms with features suggestive of depolarization from the right to the left ventricle: a dominant Q or predominant S in V1-V3 (QRS will be net negative) and a broad, dominant R wave in the lateral leads (I, aVL, V5, V6). (*See Figure 3.*) This QRS configuration is present in an LBBB band in a right ventricular paced pattern (since both of these cause ventricular depolarization to happen from right to left). A new LBBB once was believed to be a STEMI equivalent; however, this recommendation was removed from the 2013 ACCF/AHA STEMI Management Guidelines because of low frequency of acutely obstructing coronary lesions on cardiac catheterizations.[2] In hemodynamically stable patients with a presumed new LBBB, evaluation of their symptoms requires both measurement of cardiac biomarkers and observation. In patients with hemodynamic compromise (including acute heart failure), revascularization should be emergently considered.[13]

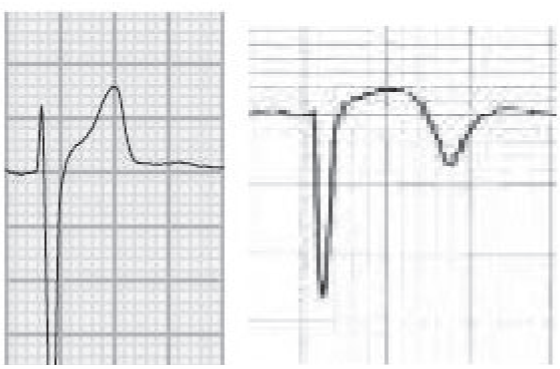
The Sgarbossa criteria can help guide the decision for emergent catheterization and coronary intervention in the presence of both new and old LBBBs.[14] The normal state of an LBBB is described by the "rule of appropriate

Figure 1. Early Repolarization



Early repolarization showing J-point elevation in multiple contiguous leads and slurring and/or notching on the downstroke of the R wave in leads II, III, aVF, V5, and V6. Image used with permission from Life in the Fast Lane (LITFL.com).

Figure 2. ST-Segment Concave and Convex



Concave or sagging ST segment on the left and convex (or non-concave) ST segment on the right

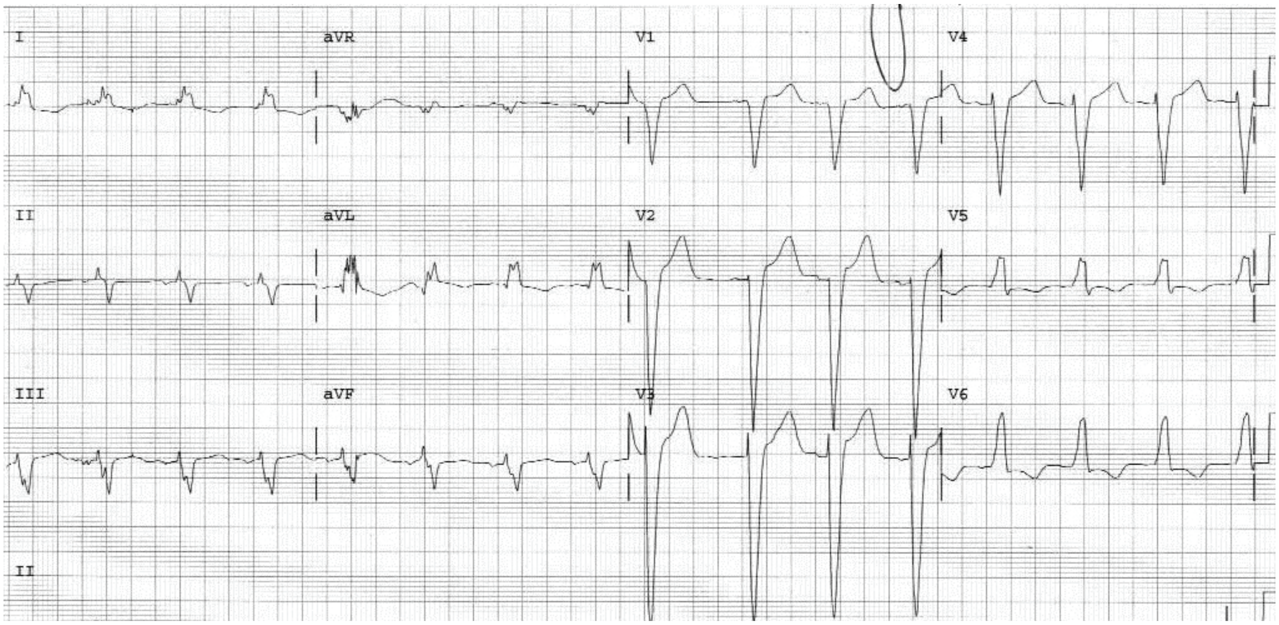
discordance." This idea is that ventricular repolarization (ST-T) occurs in the opposite direction of most of the ventricular depolarization (QRS), which manifests itself as the net polarity of the QRS and T wave being opposite from each other. Thus, an ST segment in the same direction as the QRS (also known as "concordant") is indicative of ischemia/infarction. Conversely, in an LBBB, the QRS in V1-V3 is always negative; therefore, the normal condition of the ST segment in these leads is ST elevation. Thus, excessive discordant ST-segment elevation in leads V1-V3 is indicative of an anterior MI. The modified Sgarbossa criteria determine "excessive discordance" by a proportion rather than an absolute number (these criteria have been validated):[15,16]

1. Concordant ST elevation ≥ 1 mm in any single lead (*see Figure 4*);
2. Concordant ST depression ≥ 1 mm in just one of leads V1-V3;
3. Proportionally excessive discordant ST elevation as defined by a ratio of ST elevation at the J-point, relative to the depth of the S wave (ST/S ratio), of ≥ 0.25 (this has replaced the original third criterion of ST elevation, which was an absolute number [≥ 5 mm]). (*See Figure 4.*)

The original Sgarbossa decision tool assigned points to each criterion: 5 points for concordant ST elevation > 1 mm in any lead; 3 points for concordant ST depression > 1 mm in leads V1 to V3; and 2 points for discordant ST elevation > 5 mm in any lead. With a threshold of 3 or more points, the initial article describing Sgarbossa criteria reported a sensitivity of about 80% and a specificity of about 90% for detecting AMI in the presence of an LBBB.[14]

In subsequent studies, other authors reported lower sensitivities, and a 2008 meta-analysis of 11 studies reported a summary sensitivity of only 20% and a summary specificity of 98% using a score of 3 or more.[17] To increase sensitivity, the modified criteria have been proposed, and although they have not been subjected to extensive validation, the reported sensitivity from a few studies is about 80%.[16] Also observed in the setting of LBBB, Chapman's sign (a notching of the R wave seen in leads I, aVL, and sometimes V6) occasionally is seen in anterior wall MIs, as well as the analogous Cabrera's sign (a notch in the S wave, seen mostly in V3 and V4).

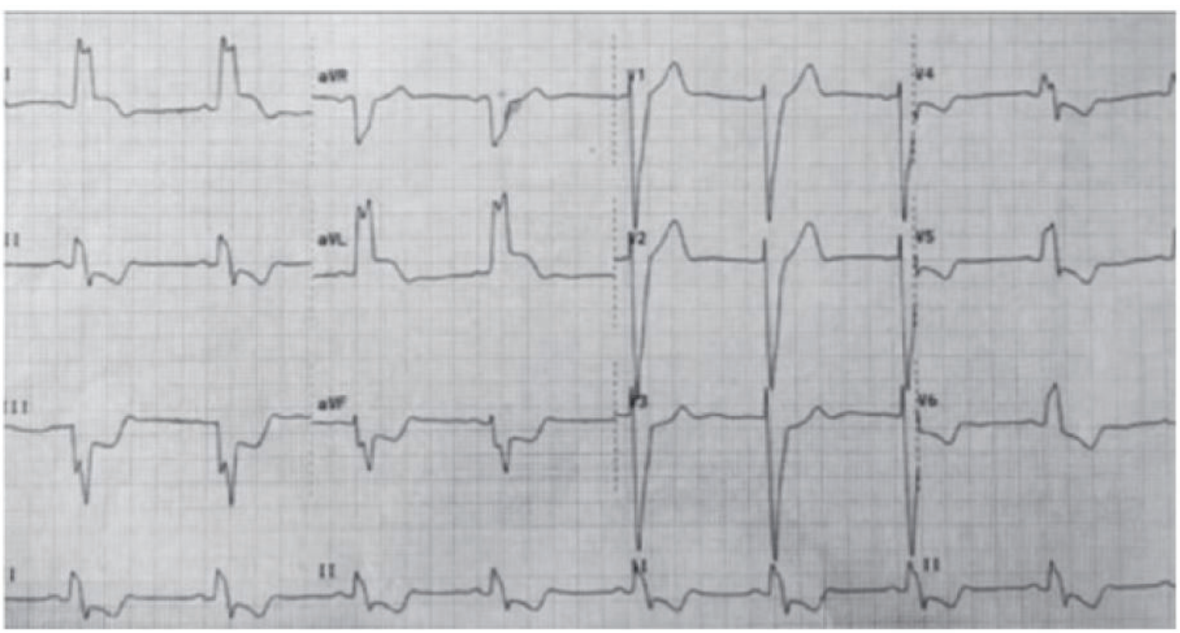
Figure 3. Left Bundle Branch Block



QRS duration > 120 msec, dominant WS wave in V1-V3, bodard R wave in lateral leads I, aVL, V5, and V6.

Figure courtesy of J. Stephan Stapczynski, MD.

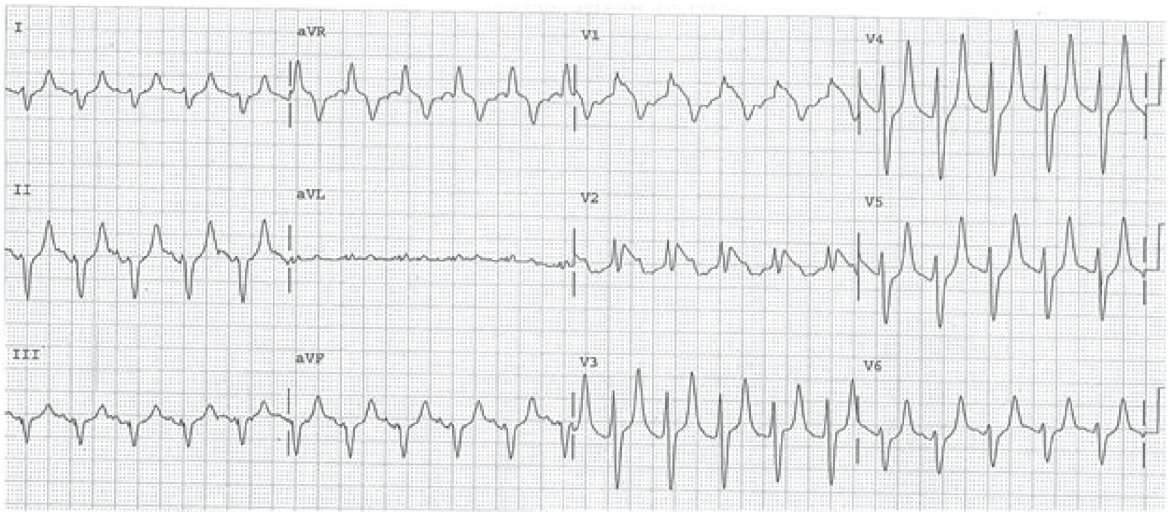
Figure 4. Left Bundle Branch Block Satisfying the First Sgarbossa Criterion



ECG contributed by Larissa Velez, MD.

Both of these signs, although easier to appreciate than the Sgarbossa criteria, are less sensitive. Very importantly, both Chapman's and Cabrera's signs are Q wave equivalents and, therefore, are only indicative of a completed infarct and do not indicate ischemic or viable myocardium. The most clinically useful Sgarbossa criteria pertaining to patients with right ventricular paced rhythms was the ST-segment elevation > 5 mm discordant with the QRS complex — this finding had sensitivity of 10% and specificity of 99% for identifying acute MI.[18]

Figure 5. Hyperkalemia



Note the prominent, narrow, symmetric, peaked T waves in leads V3-V6.

ECG contributed by Larissa Velez, MD.

Electrolytes

Electrolyte derangements of potassium, calcium, magnesium, and sodium alter the cardiac action potential, resulting in ECG changes. Hyperkalemia frequently can cause ST elevation, most commonly in leads V1 and V2 (*see Figure 5*) and should be suspected when there is any QRS widening, especially when associated with some symmetric peaking of the T waves ("T waves that will poke you if you touch them"). Other ECG changes in hyperkalemia include shortening of the QT interval, shortening of the PR interval, flattening of the P waves, loss of sinoatrial conduction resulting in a wide-complex ("sine-wave" or "sinoventricular") rhythm, and, ultimately, ventricular fibrillation and asystole. The ECG changes might not occur in a stepwise fashion and are more dependent on the rate of potassium elevation than on the absolute value.[19] The easiest way to diagnose hyperkalemia is by measuring serum potassium. After hyperkalemia has been adequately treated, the ECG findings improve.

Similarly, calcium plays an important role in phase 2 (the plateau phase) of the cardiac action potential. Calcium maintains a balance between inward calcium flow through the L-type calcium channels, coupled with outward potassium flow through the delayed rectifier potassium channels. This balance is affected by the presence of excess serum calcium because hypercalcemia (*see Figure 6*) slows ventricular conduction velocity and shortens the refractory period of myocytes. Characteristic ECG changes include shortening of the QT interval. This shortening of the QT interval is what may mimic ST elevation. Hypercalcemia also can be arrhythmogenic, including atrioventricular blocks, and can induce a variety of T wave changes, including flattening, inversion, and notching.[20] Hypercalcemia also can cause the appearance of J waves (discussed later).

Sodium channel blocker (SCB) toxicity may manifest as ST elevation, particularly in lead aVR. The most well-studied SCBs are the tricyclic antidepressants (TCAs). The classic electrocardiographic findings of TCA toxicity are sinus tachycardia, a QRS duration of more than 100 ms, and a rightward shift of the terminal 40 ms of the QRS, best demonstrated by a dominant R wave in lead aVR.[21,22] Although cardiology literature considers a QRS duration up to 120 ms as normal, in the presence of SCB toxicity, QRS durations greater than 100 ms are associated with increased chances of seizures, and QRS durations greater than 160 ms are associated with increased chances for ventricular dysrhythmias.[23]

Ventricular Enlargement

Left ventricular hypertrophy (LVH) is known to cause many false-positive cardiac catheterization lab activations.[7] (*See Figure 7.*) The electrocardiographic diagnostic criteria for LVH all have poor sensitivity.[24] The most specific and widely used criteria are the Sokolow-Lyon criteria, which confer a specificity of 100% and consist of the following: the amplitude of the S wave in V1 plus R wave in V5 or V6 that is more than 35 mm, or R wave amplitude in V5 or V6 that is more than 26 mm.[25,26]

The repolarization abnormality, which can cause ST elevation in leads V1-V3 as well as T wave abnormalities (formerly known as "strain") in lateral leads, occurs as a result of the anatomical and electrical remodeling of the left ventricle in the setting of hypertrophy.[27] The ST-segment elevation seen in leads V1 to V3 in patients with LVH may mimic an anterior STEMI. It has been noted that the ST elevation seen in a STEMI usually is greater than that seen with LVH. Rather than a specific value in mm, a ratio of the ST elevation to the size of the dominant wave (either R or S) in the QRS complex in the same

Figure 6. Hypercalcemia

Shortened QT interval (QTc = 392 ms) with serum calcium of 19.0 mg/dL

Figure courtesy of J. Stephan Stapczynski, MD.

lead can be used to distinguish when ST elevation is within typical values for LVH. A threshold value of < 0.25 for the ST to R/S ratio has been proposed for what is expected with LVH.[28] However, no validated guidelines exist to address an appropriate amount of ST elevation for LVH with "strain."[29] LVH with repolarization abnormality may be indistinguishable from myocardial ischemia if no previous ECG is available for comparison.

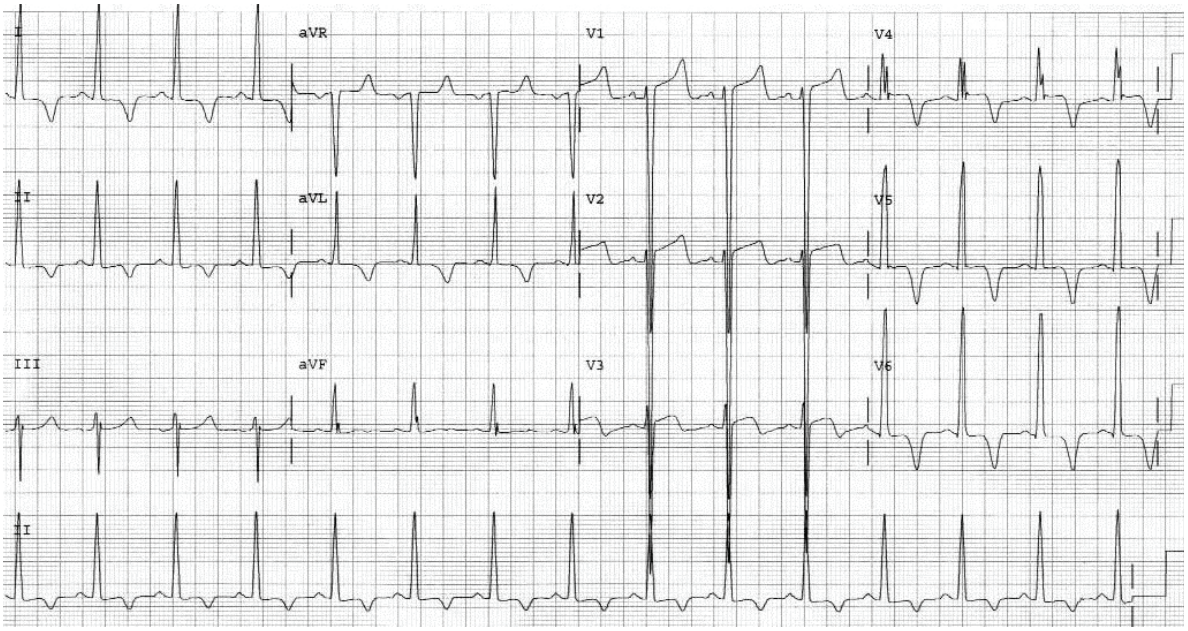
In adults, chronic RV hypertrophy most often is attributed to long-standing pulmonary hypertension (e.g., pulmonary hypertension). Similar to LVH, no sensitive criteria exist for the diagnosis of RVH. The simplest and most specific criteria is an R wave in V1 > 7 mm (or R/S in V1 > 1) and right axis deviation.[25] Acute RV enlargement ("right heart strain") does not have a large R wave in V1 but may have right axis deviation. RVH by itself rarely causes ST elevation, except in some cases of acute pulmonary embolism (PE). The ST elevation seen in acute PE typically is seen in leads V1 and aVR.[30] A meta-analysis of ECG features in acute PE found that six ECG findings had significant predictive value for circulatory collapse: a heart rate > 100 beats/min, an S1Q3T3 pattern, a complete right bundle branch block (RBBB), inverted T waves in V1-V4, ST elevation in aVR, and atrial fibrillation.[31] (*See Figure 8.*) The mechanism behind this is unclear. It has been reported that RVH can cause ST depression in V1-V3 and can mimic a posterior STEMI.

Aneurysm

When a transmural infarct is not aborted by therapeutic intervention and the AMI completes itself, the myocardium is replaced by a thin, fibrous layer, which is called an LV aneurysm. On the ECG, a left ventricular aneurysm may manifest as persistent ST elevation in the territory of a prior infarct, commonly concomitant with Q waves. (*See Figure 9.*) Most of these aneurysms occur at the left ventricular apex. Smith and colleagues have derived and validated a rule for differentiation of anterior LV aneurysm from acute anterior STEMI.[32,33] The rule states that when the differential diagnosis is acute LAD occlusion vs. anterior LV aneurysm, if any of leads V1-V4 has a T wave amplitude to QRS amplitude ratio of > 0.36, then STEMI is likely. In general, aneurysm is favored by prominent Q waves in leads V1-V4 with corresponding diminished T wave amplitudes.

Takotsubo cardiomyopathy, also termed "the broken heart syndrome" or stress cardiomyopathy (SCM), refers to an apical LV motion abnormality in the absence of an identifiable coronary artery occlusion or prior scar. As many as 80% of these cases also will have abnormal troponin values.[34] The syndrome is called "Takotsubo" because the apical ballooning that occurs at the LV apex resembles a Japanese octopus trap, called a "takotsubo." First described in 1990, Takotsubo mainly is a disease of older women (mean age 66 years and 89.8% women).[34] Usually, it occurs after emotional or physical stress and is thought to be the result of catecholamine surges. Takotsubo cardiomyopathy has been reported to be the cause of troponin-positive ST elevation in 1% to 2% of cases. Unfortunately, Takotsubo's ECG is identical to an anterior STEMI and may include diffuse T wave inversions and prolonged QT. The revised Mayo Clinic criteria assist with the diagnosis of SCM:

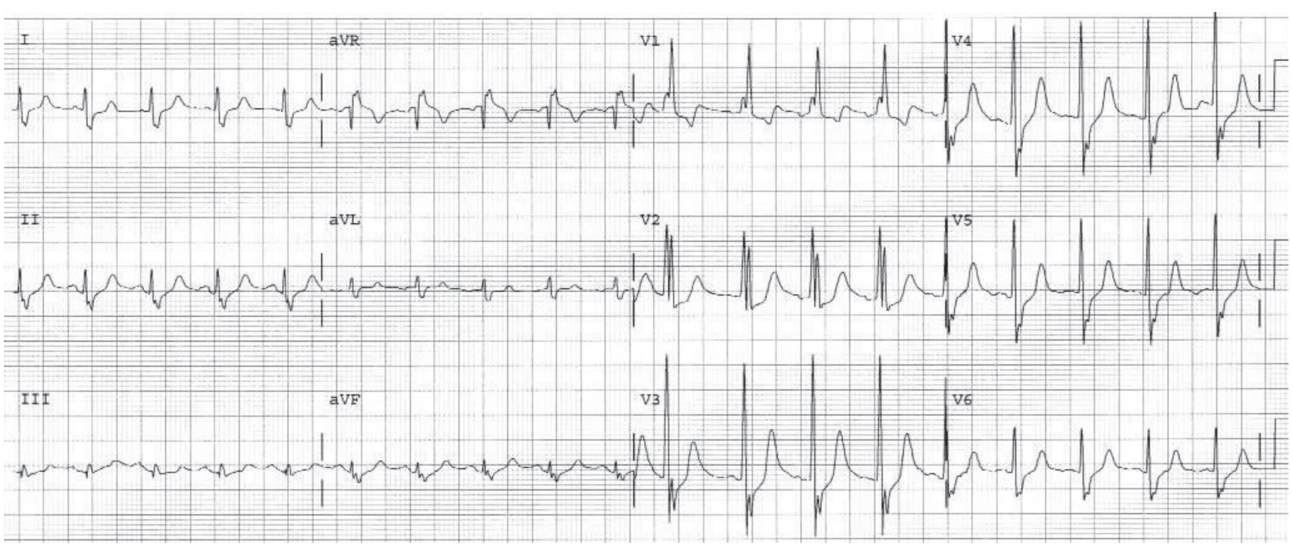
Figure 7. Left Ventricular Hypertrophy



The depth of the S wave in V1 plus the height of the R wave in V5/6 is > 35 mm with ST segment and T wave changes indicative of "strain."

Figure courtesy of J. Stephan Stapczynski, MD.

Figure 8. Acute Pulmonary Embolism



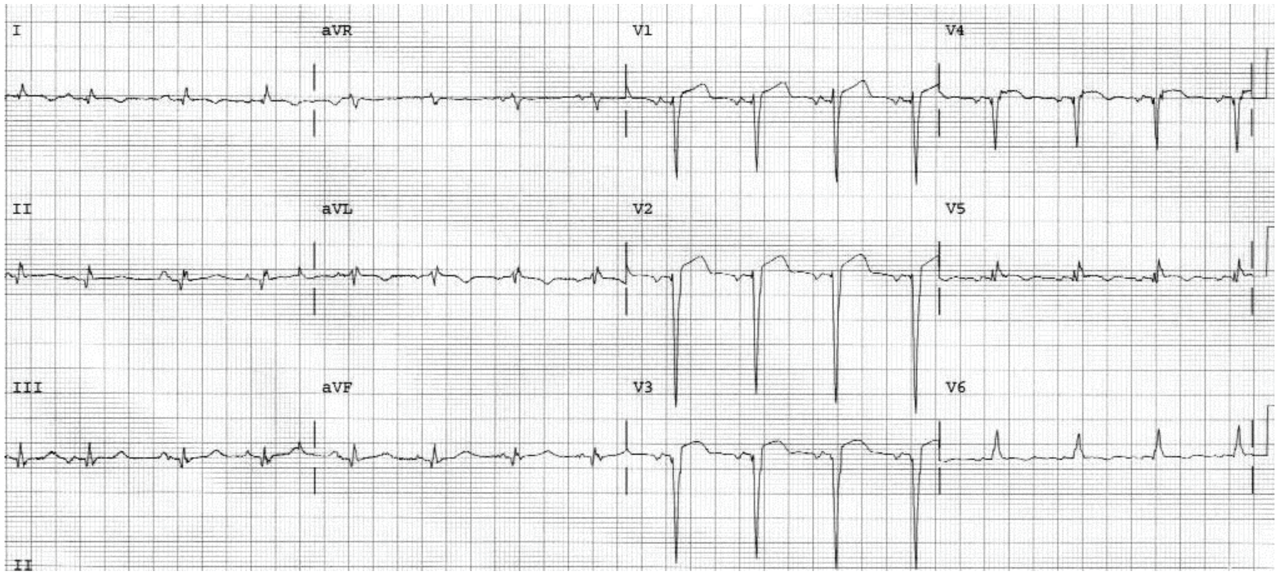
Note the sinus tachycardia (heart rate 111 beats/min), incomplete RBBB pattern, and ST elevation in lead aVR.

Figure courtesy of J. Stephan Stapczynski, MD.

- Transient dyskinesis of the LV midsegments;
- Regional wall motion abnormalities beyond a single epicardial vascular distribution;
- Absence of obstructive coronary artery disease or acute plaque rupture;
- New electrocardiographic abnormalities or modest troponin elevation;
- Absence of pheochromocytoma and myocarditis.[35]

Note that these criteria include the results of a left heart catheterization. In the emergency department, these patients must receive a cardiology evaluation and an emergent LHC.

Figure 9. Left Ventricular Aneurysm



Persistent ST elevation due to LV aneurysm that developed after an anterior infarction indicated by Q waves in V2 to V4.

Figure courtesy of J. Stephan Stapczynski, MD.

Thailand (Brugada) Syndrome

Brugada syndrome is considered a part of the ERS spectrum.[36] For many years, it was called a "sleeping sickness" in Southeast Asia (known as "Lai Lai" in Thailand) because it referred to young men who would scream in the middle of the night and suddenly die. In 1992, Brugada syndrome was described initially by the Brugada brothers as an RBBB with persistent ST elevation in patients presenting with syncope and dysrhythmic events.[37]

However, it should be noted that Brugada syndrome is defined as the ECG pattern in addition to symptoms (syncope, palpitations, sudden death). If seen in isolation, the Brugada ECG pattern is referred to as Brugada sign, and its significance is dependent on the patient's clinical context. It is thought that the Brugada syndrome is the cause of death in about 4% to 5% of sudden death cases, particularly in young males. After genotyping, the most common mutation observed was in an SCN5A sodium channel.[25] Although this channel's predominant contribution is during phase 0 and 1 of the fast myocyte action potential, it is postulated that this mutation leads to an unopposed outward potassium current that leads to dysrhythmias.[38]

The ECG pattern in Brugada syndrome looks like an R wave in which the ST segment has a gradual downslope such that at 40 ms after the ST takeoff the increase in amplitude is < 4 mm.[39] This abnormal ST segment may be mistaken for ST elevation, particularly in patients who present with ventricular dysrhythmias. There are two (previously considered to be three) ECG patterns in Brugada syndrome. (*See Figure 10.*) The most recognized Brugada ECG phenotype, Type 1, has ST elevation in V1-3 of a "coved" variety and frequently is identified as an RBBB.[25] Type 2 has "saddle back" ST morphology with > 2 mm ST elevation. The Brugada ECG phenotype may be transient and exacerbated by the presence of sodium channel blockers, fever, or have no identifiable precipitant.[40] Patients with Brugada syndrome must be evaluated for internal cardioverter defibrillator (ICD) placement to prevent sudden death.[39] Since the absolute risk of death in patients with isolated Brugada sign is unknown, emergency providers should recommend follow-up with cardiology.

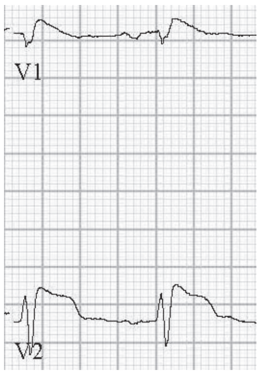
Inflammation (Myopericarditis)

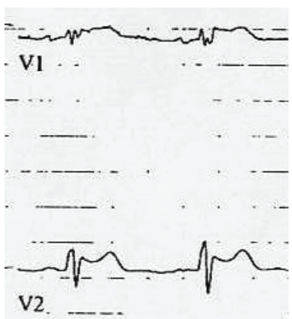
The classic ECG changes associated with acute pericarditis, a relatively rare clinical entity, are diffuse ST elevation and PR depression. When evaluating for these two findings, it is crucial to remember that the isoelectric line on the ECG is the TP segment. The morphology of ST elevation of pericarditis can mimic that of an infarct and can cause both confusion and consternation to the emergency physician.

The diagnosis of AMI is favored if any of the following two ECG findings are present: any ST depression (other than V1 or aVR), or ST elevation in lead III that is greater than the amplitude of ST elevation in lead II.[41] In pericarditis, ST-segment elevation is seen most commonly and most prominently in the inferior and high lateral leads. Notably, if there is inferior ST elevation, the existence of ST depression in aVL favors inferior wall MI over pericarditis.[42] In other words, any suggestion of reciprocal changes must raise the clinician's suspicion for STEMI.

In general, PR depression only is reliably seen in viral acute pericarditis, is transient, and must be seen in multiple

Figure 10. Morphology of Both Types of Brugada Syndrome





Upper figure: Type I Brugada pattern with coved ST segment.

Lower image: Type II Brugada pattern with saddle back ST segment.

Figures courtesy of J. Stephan Stapczynski, MD.

leads. The "checkmark" or "RT sign" seen in some cases of pericarditis describes the appearance of a checkmark-like deflection at the terminal portion of the QRS joining the T wave. Spodick's sign, a down-sloping of the TP segment and best seen in lead II and the lateral precordial leads, is seen in about 80% of pericarditis patients.[43] In addition, the ECG findings of STEMI often are dynamic compared to those of pericarditis, which are unlikely to change in the ED. Prolongation of the QRS complex and shortening of the QT interval, which can be seen in patients with STEMI, usually do not occur with pericarditis.[44] One final way to differentiate between pericarditis and early repolarization is to calculate the ratio of the height of the onset of the ST segment and the T wave amplitude in V6. If this ratio is > 0.25, acute pericarditis is likely.[45]

Osborn (J) Waves

In 1953, Osborn performed an experiment on dogs, cooling them to 23°C, during which he noted a pattern on the ECG that conferred a bad prognosis and a likelihood of progression to VF.[46] Initially, it was postulated that the Osborn wave was not an injury current similar to an infarct, but rather caused by impeded elimination of CO_2. Osborn waves, also known as "J waves," are brief positive deflections at the junction of the QRS complex and ST segment. Osborn waves are most commonly observed in hypothermia, but may be seen in hypercalcemia, vasospastic myocardial ischemia, and brain injury. J waves are thought to be caused by the difference in the action potential propagation between epicardial M cells and endocardial cells.[47] It is the same mechanism thought to cause ERS, which is why the clinical implications of J waves may be more far-reaching than considered previously. Once thought to be "simply" an RBBB pattern, the J wave associated with Brugada syndrome may carry a risk of sudden death.

Non-Thrombotic Vasospasm

ST elevation caused by vasospasm has an identical morphology to that of a true STEMI, even though it is not caused by coronary artery endoluminal plaque rupture or intraluminal thrombosis. Vasospastic angina (once termed "Prinzmetal," or variant angina) occurs as the result of coronary artery vasospasm, which produces ST elevation along the involved coronary territory. In vasospastic angina, administration of nitrates or benzodiazepines usually resolves the chest pain and the ST-segment elevations. In the ED it often is difficult to distinguish vasospastic angina from STEMI and as such, patients with chest pain and ST elevation should always undergo an evaluation for ischemia.

An example of vasospastic angina occurs after cocaine use. Cocaine causes ischemia because of its adrenergic stimulation, causing vasospasm as well as causing increased myocardial oxygen demand. Chest pain is a common complaint in patients with cocaine toxicity and the Cocaine Associated Chest Pain (COCHPA) study group determined that there was no clinical parameter that could identify patients with very low risk for cocaine-induced MI.[48] In addition to cocaine-induced MI, chronic cocaine users are at an increased risk for accelerated atherosclerosis, which further predisposes them to acute coronary syndrome.[49,50]

Conclusion

Chest pain is a common cause of ED visits. In patients with ECG findings consistent with a STEMI, the primary goal is to salvage myocardium through rapid reperfusion. However, before activating the cardiac catheterization lab for ECG changes, it is important to consider non-ischemic causes of ST-segment elevation. The goal of this review was to provide clinicians with tools for ECG evaluation that will help identify some of the most common STEMI mimics.

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CME/CE Questions

1. Which of the following is true regarding early repolarization?
   a. ST-segment convexity is more than 90% sensitive for infarction ST-elevation myocardial infarctions (STEMIs).
   b. There is no association with sudden death.
   c. It causes QRS prolongation.
   d. The J waves are similar to those seen in hypothermia.

2. Which of the following patients with a typical left bundle branch block (LBBB) should be considered for emergent percutaneous coronary intervention (PCI)?
   a. Anyone with risk factors and "typical" chest pain
   b. Any new or presumed new LBBB
   c. ST elevation in leads I, aVL, and V4-V6
   d. ST elevation in leads V1 and V2

3. A 24-year-old woman presents to the emergency department (ED) with altered mental status, tachycardia, and hypotension. An electrocardiogram (ECG) is performed, and the QRS complex is 0.16 seconds wide. The polarity of that QRS complex is net negative in lead I and net positive in lead aVF (right axis deviation). You also note that there is a prominent R wave in lead aVR. Which of these toxic or metabolic events is most likely?
   a. Hyperkalemia
   b. Hypothermia
   c. Hypercalcemia
   d. Tricyclic antidepressant toxicity

4. Which of the following is true regarding ST-segment elevation caused by left ventricular hypertrophy (LVH)?
   a. LVH can cause ST-segment elevation in leads II, III, and aVF.
   b. The T waves are not affected.
   c. Electrical remodeling is the cause of the ST-segment elevation.
   d. The ratio of the ST-segment elevation to the size of the dominant QRS wave is > 0.25.

5. In the setting of Takotsubo cardiomyopathy, which of the following is true?
   a. Troponins may be elevated.
   b. Troponins will be normal.
   c. Patients uniformly develop LV wall rupture.
   d. It most commonly occurs in younger women.

6. A young Asian male from Thailand presents by emergency medical service after a ventricular fibrillation (VF) arrest at home. His post-defibrillation ECG suggests a right bundle branch block with a coved ST segment pattern. Which of the following is true?
   a. This disease is frequently accompanied by a delta wave.
   b. Identified aggravating factors for this condition include fever.
   c. Further arrhythmias should be treated with sodium channel blockade.
   d. The ECG pattern does not vary with time.

7. A homeless man was found passed out under a bridge and is brought to the ED. His ECG shows irregular bradycardia at a rate of 55, a short QT segment, and a notch at the end of the R wave. Which of the following should be considered in the management of this patient?
   a. Active warming
   b. Emergent reperfusion
   c. Aspirin
   d. Ionized calcium

8. One hour after doing "a little bit" of cocaine, a 35-year-old woman complains of chest pain. The ECG shows ST elevation in leads V1-V4. Which of the following is true regarding this patient?
   a. Cocaine use is associated with decreased myocardial oxygen demand.
   b. The patient should be treated as a potential acute coronary syndrome.
   c. The patient is not at risk for accelerated atherosclerosis.
   d. Chest pain is an uncommon complaint in ED patients who present after cocaine use.

9. When evaluating an ED patient with ST elevation, which of the following is true?
   a. The most likely cause is acute infarction.
   b. The most common cause of false-positive catheterization lab activations in patients without troponin elevation is early repolarization.
   c. The updated American College of Cardiology Foundation/American Heart Association definition for a STEMI requires the same degree of ST elevation in leads V2-V3 in men and women.
   d. ST-segment elevation occurs reliably in both transmural and subendocardial infarction.

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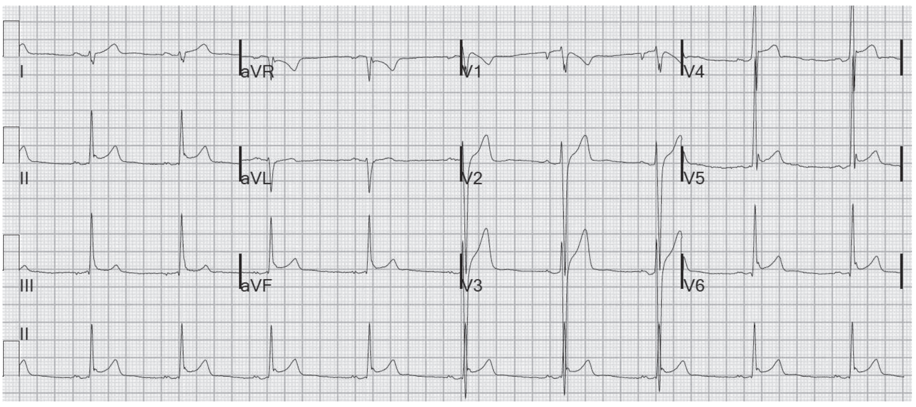
EMERGENCY MEDICINE REPORTS

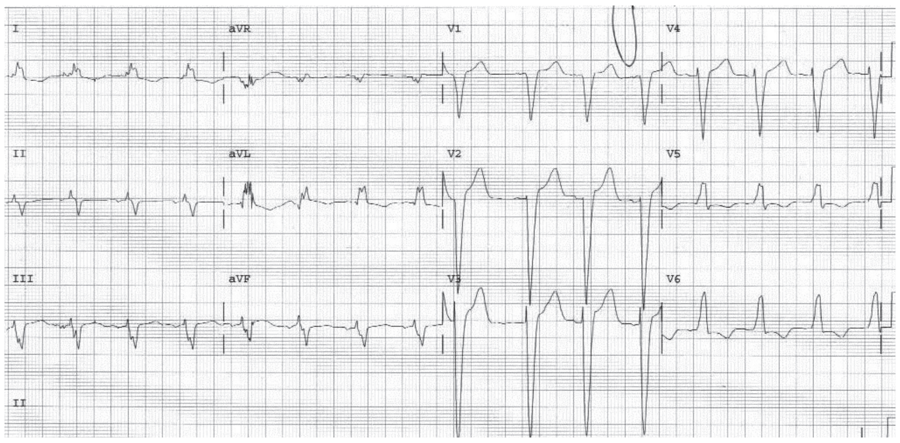
Mimics of ST-Elevation Myocardial Infarction (STEMI)

Early Repolarization



Early repolarization showing J-point elevation in multiple contiguous leads and slurring and/or notching on the downstroke of the R wave in leads II, III, aVF, V5, and V6. Image used with permission from Life in the Fast Lane (LITFL.com).

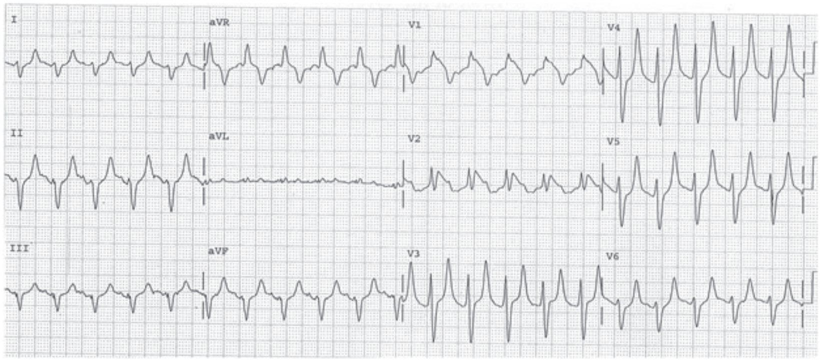
Left Bundle Branch Block



QRS duration > 120 msec, dominant WS wave in V1-V3, bodard R wave in lateral leads I, aVL, V5, and V6.

Figure courtesy of J. Stephan Stapczynski, MD.

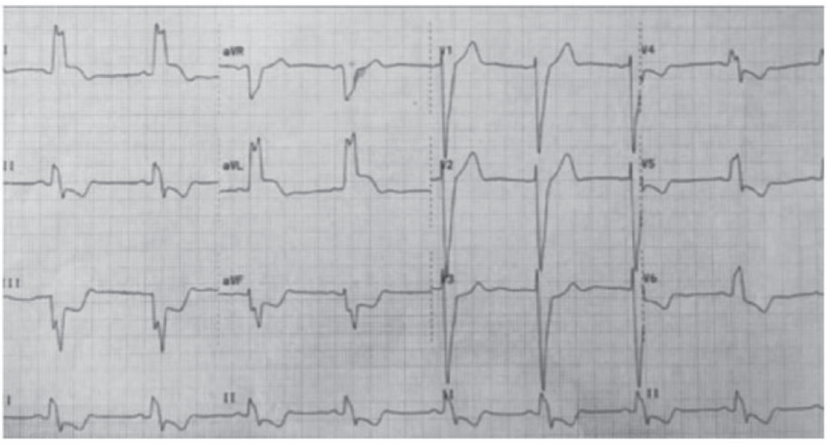
Hyperkalemia



Note the prominent, narrow, symmetric, peaked T waves in leads V3-V6.

ECG contributed by Larissa Velez, MD.

Left Bundle Branch Block Satisfying the First Sgarbossa Criterion



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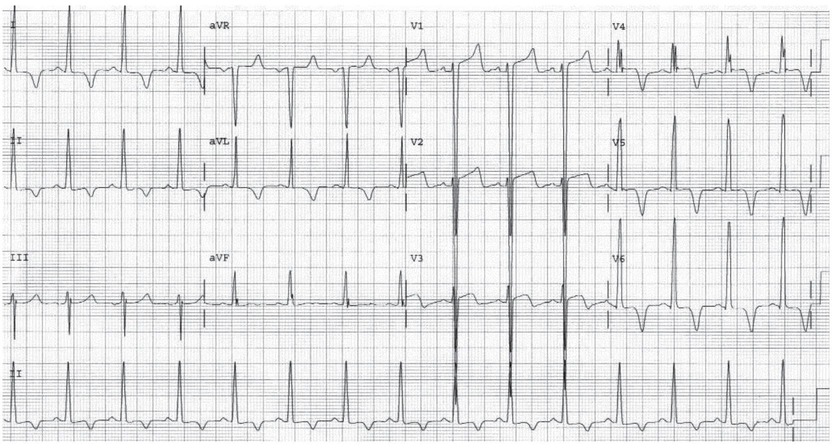
Hypercalcemia

Shortened QT interval (QTc = 392 ms) with serum calcium of 19.0 mg/dL

Figure courtesy of J. Stephan Stapczynski, MD.

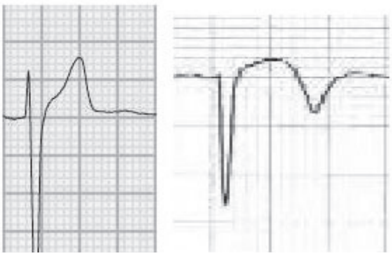
Left Ventricular Hypertrophy



The depth of the S wave in V1 plus the height of the R wave in V5/6 is > 35 mm with ST segment and T wave changes indicative of "strain."

Figure courtesy of J. Stephan Stapczynski, MD.

ST-Segment Concave and Convex



Concave or sagging ST segment on the left and convex (or non-concave) ST segment on the right

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