

Electrocardiographic Patterns Mimicking ST Segment Elevation Myocardial Infarction

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KEYWORDS

• Electrocardiogram • ECG • ST segment elevation • STEMI • Mimic

KEY POINTS

- The 12-lead surface electrocardiogram (ECG) is inexpensive, portable, and transmittable; it remains the cornerstone of prompt diagnosis of and primary indication for the management of ST elevation myocardial infarction (STEMI).
- Although the ECG is reasonably reliable, it remains an imperfect diagnostic tool. Some patients do present with classic symptoms and findings; however, approximately 60% to 80% of patients with ST segment elevation on the presenting ECG are ultimately found to not be associated with STEMI.
- In certain difficult cases, a patient's ECG can resemble STEMI yet manifest ST segment elevation from a non-acute coronary syndrome entity, the so-called *STEMI mimics*. In other situations, the patient's ECG makes it difficult or impossible to determine whether STEMI is present, the so-called *STEMI confounders*; these *confounders* to STEMI diagnosis are also *mimickers* of AMI.
- The ultimate goal with both the STEMI mimics and the confounders is to maximize rapid, accurate diagnosis while avoiding delays in treatment of alternative causes of ST segment elevation.

INTRODUCTION

Cardiovascular disease is the leading cause of death worldwide. In fact, 7 million patients present annually to emergency departments in the United States with symptoms concerning for myocardial ischemia.¹ Prompt reperfusion in the setting of ST segment elevation myocardial infarction (STEMI) can dramatically reduce the associated mortality and morbidity. Unfortunately, the benefit of reperfusion therapies in acute myocardial infarction (AMI) decays quickly over time. Improvements in care systems, such as the door-to-balloon initiative and the American Heart Association's Mission Lifeline have altered the landscape of STEMI management over the past decade, minimizing delays to reperfusion and significantly improving outcomes. These systems

require the rapid mobilization of large teams of practitioners and resources, involve not insignificant risk to patients, and rely heavily on the clinician to quickly and accurately determine whether an electrocardiographic finding represents acute closure of a coronary artery and related STEMI. The 12-lead surface electrocardiogram (ECG) is inexpensive, portable, and transmittable; it remains the cornerstone of the prompt diagnosis of and the primary indication for management of STEMI. Although reasonably reliable, the ECG remains an imperfect diagnostic tool. Some patients do present with classic symptoms and findings; however, approximately 60% to 80% of patients with ST segment elevation on the presenting ECG ultimately are found to not be associated with STEMI.^{2,3} Refer to **Fig. 1** for a depiction of the

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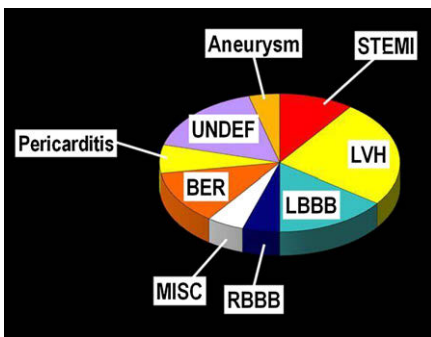


Fig. 1. Causes of ST segment elevation in adult patients with chest pain. BER, benign early repolarization; LBBB, left bundle branch block; LVH, left ventricular hypertrophy; MISC, miscellaneous; RBBB, right bundle branch block; UNDEF, undefined.

causes of ST segment elevation in adult patients with chest pain.^{2,3}

In certain difficult cases, a patient's ECG can resemble STEMI, yet manifest ST segment elevation from a non-acute coronary syndrome (ACS) entity, the so-called *STEMI mimics*. In other situations, the patient's ECG makes it difficult or impossible to determine whether STEMI is present, the so-called *STEMI confounders*; these *confounders* to STEMI diagnosis are also *mimickers* of AMI.

All such cases can leave a practitioner wondering whether to initiate reperfusion therapy, either via administration of a fibrinolytic agent or activation of STEMI alert process, in essence, whether to expose patients to both the benefits and the risks of fibrinolysis or invasive coronary angiography. In some cases, the astute clinician can detect an alternative diagnosis masquerading as an STEMI. Failure to recognize these mimics can lead to inappropriate use of resources, exposure of patients to unnecessary risk, and increased rather than decreased morbidity and mortality. The ultimate goal with both the STEMI mimics and the confounders is to maximize rapid, accurate diagnosis while avoiding delays in the treatment of alternative causes of ST segment elevation. Because the risk of cerebral hemorrhage from fibrinolysis is not insignificant, careful consideration of the ECG, looking for the STEMI mimics, is required in patient-care situations in which primary percutaneous coronary intervention (PCI) is not an option. More importantly, fibrinolysis given in the setting of certain STEMI mimics, such as acute myopericarditis, is associated with high mortality.

Although each of the conditions discussed here is unique, a common issue that must not be overlooked is the interpretation of the ECG within the context of the patient's presentation; in other words, does the patient look like he or she is

experiencing a STEMI? The STEMI mimics and confounders more often imitate the ECG findings of AMI than the clinical syndrome, so the patient *with* ST segment elevation but *without* a convincingly clinical picture of STEMI should prompt the provider to suspect a non-AMI presentation. At times, these diagnoses are very challenging, which will understandably impact the rapid application of reperfusion therapy and likely increase the door-to-therapy time.

STEMI MIMICKING PATTERNS *Myocarditis and Myopericarditis*

Inflammation of the pericardium and heart muscle is a common cause of chest pain with ST segment elevation. Seventy-three percent of patients diagnosed with acute myopericarditis will have ST segment elevation on initial ECG.⁴ Additionally, 44% of patients with chest pain and positive troponin but who do not have obstructive coronary disease by angiography demonstrate evidence of myocarditis by cardiac magnetic resonance imaging (MRI) using late gadolinium enhancement to reveal areas of myocardial necrosis in a noncoronary distribution (ie, midwall or subepicardial rather than subendocardial).⁵

Myocarditis affects patients of all ages and has a wide spectrum of clinical severity, ranging from incidental chest discomfort to fulminant heart failure with cardiogenic shock. The term *myocarditis* refers to an inflammatory process of the heart muscle (as reflected by the presence of biomarkers and ECG changes), whereas *pericarditis* refers to isolated inflammation of the lining around the heart. The pericardium is electrically silent, thus, when patients present with a clinical picture suggestive of pericarditis and demonstrate ST segment changes, the myocardium is also affected. Frequently, inflammation involves both components, hence, the term *myopericarditis* is used.

The cause is not frequently elucidated, but a viral cause is thought to be most common, with a minority of cases stemming from toxins or autoimmune processes. Patients may remember a prodrome of viral illness in the previous week or two. The pain is classically pleuritic in nature and changes in severity with position (sitting forward or lying back).

Biomarkers of myocardial necrosis, such as troponin and creatinine kinase, are positive in the setting of myocarditis, although they may be negative in the setting of pure pericarditis. The absolute value of troponin elevation is associated with the extent of myocardial cell injury but only very roughly correlates with clinical severity. A rub on cardiac auscultation is a highly specific but very

insensitive physical examination finding and is frequently absent when even a small effusion is present.⁶ Although a small pericardial effusion is not uncommon, it is quite rare for pericarditis to present with cardiac tamponade.

The electrocardiogram (Figs. 2 and 3) generally evolves in patients with myocarditis/myopericarditis, yet this evolution occurs less rapidly than that seen with STEMI; thus, serial ECGs, which demonstrate changes in the ST segment and T-wave morphologies over minutes, are frequently useful in making the distinction. ST segment elevation in multiple coronary distributions, especially when seen in patients who are clinically stable, favors inflammation over STEMI in large part because patients who simultaneously occlude multiple coronary arteries typically present with shock or death.⁷ The presence of ST segment depression is less likely with myocarditis/myopericarditis (with the exception of lead AVR); when present, ST segment depression likely represents reciprocal ST segment depression and suggests STEMI. The presence of PR segment depression

in leads with ST segment elevation as well as PR segment elevation in lead AVR favors inflammation over STEMI. It is possible for myocarditis to be isolated anatomically in a single coronary artery distribution, leaving distinction from STEMI more challenging. In cases involving a coexistent pericardial effusion, electrical alternans and/or diminished electrical forces of the QRS complex can also be seen.

Early Repolarization (Also Known as Benign Early Repolarization)

Benign early repolarization (BER) is a normal variant electrocardiographic pattern; it manifests as ST segment elevation at the J point, predominantly in the precordial leads. It is frequently present in the general population, particularly in younger patients and male gender. BER is associated with young athletic men and is seen across all races; it has been linked to the black race, although some have disputed this association. It is not itself a pathologic finding and has not been

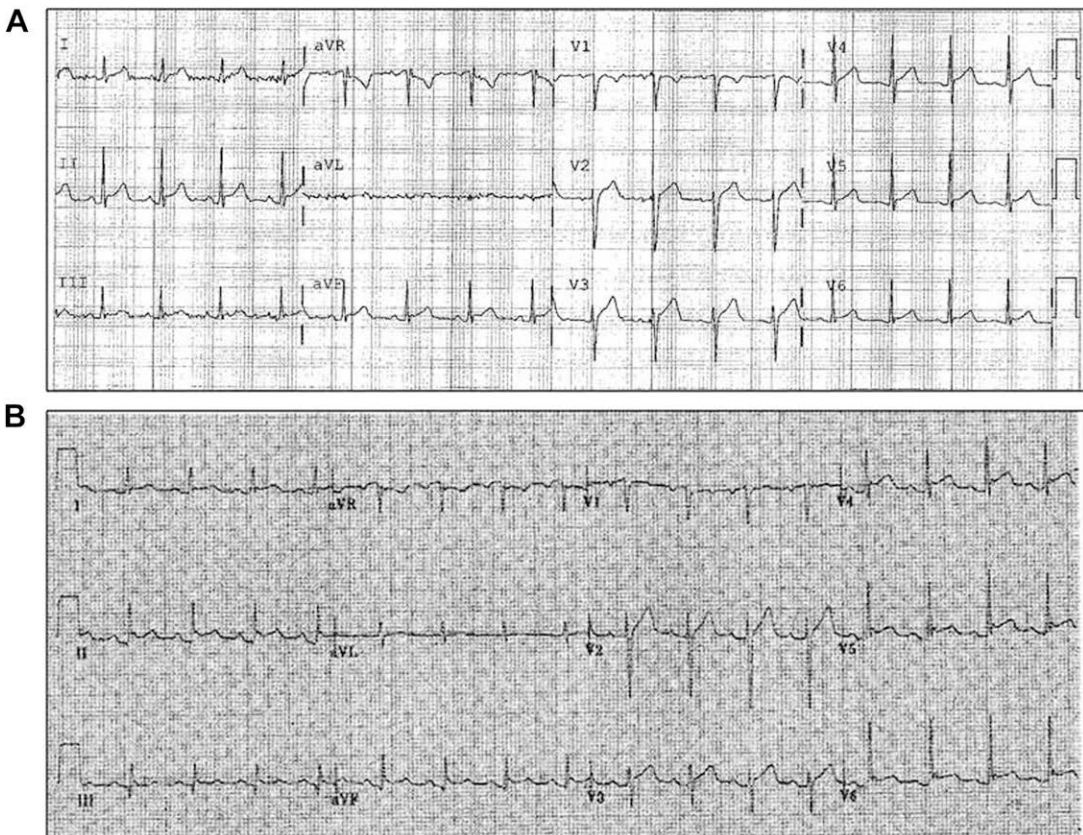


Fig. 2. Myopericarditis: Two examples of myopericarditis. (A) Myopericarditis with diffuse ST segment elevation. (B) Myopericarditis with diffuse ST segment elevation and PR segment changes (depression in leads II, III, aVF, and V6 and elevation in lead aVR).



Fig. 3. Myopericarditis versus STEMI. (A) ST segment elevation with PR segment depression of myopericarditis. (B) STEMI.

tied to a disease process; for unknown reasons, BER is seen frequently in patients with chest pain who have used cocaine.

The electrocardiographic description (Figs. 4 and 5) of BER focuses on ST segment elevation with the following associated features: ST segment elevation with an upward concavity of its initial portion, notching or slurring of the terminal QRS complex (the J point), prominent T waves that are symmetric and concordant with the QRS complex (except in leads V1-V2), widespread distribution of ST segment elevation, and relative temporal stability of the pattern. The degree of ST segment elevation, beginning at the J-point elevation, is usually less than 3.0 mm, with a range of 0.5 to 5.0 mm. The ST segment elevation morphologically seems as if the ST segment has been evenly lifted upwards from the

isoelectric baseline at the J point with preservation of the normal concavity of the initial, up-sloping portion of the ST segment. The J point itself is frequently notched or irregular in contour and is considered highly suggestive of BER. Prominent T waves are seen and are of large amplitude, slightly asymmetric morphology, and concordant with the QRS complex; the height of the T waves in BER ranges from approximately 6.5 mm in the precordial distribution to 5.0 mm in the limb leads. The ST segment and T-wave abnormalities of BER are most often seen in leads V1 to V4. At times, coexistent changes are also seen in leads II, III, AVF, V5, and V6; importantly, BER-related changes noted only in the limb leads are unusual and likely result from some other pathologic process, such as STEMI. Lastly, the chronic nature of the ST segment elevation is helpful in the

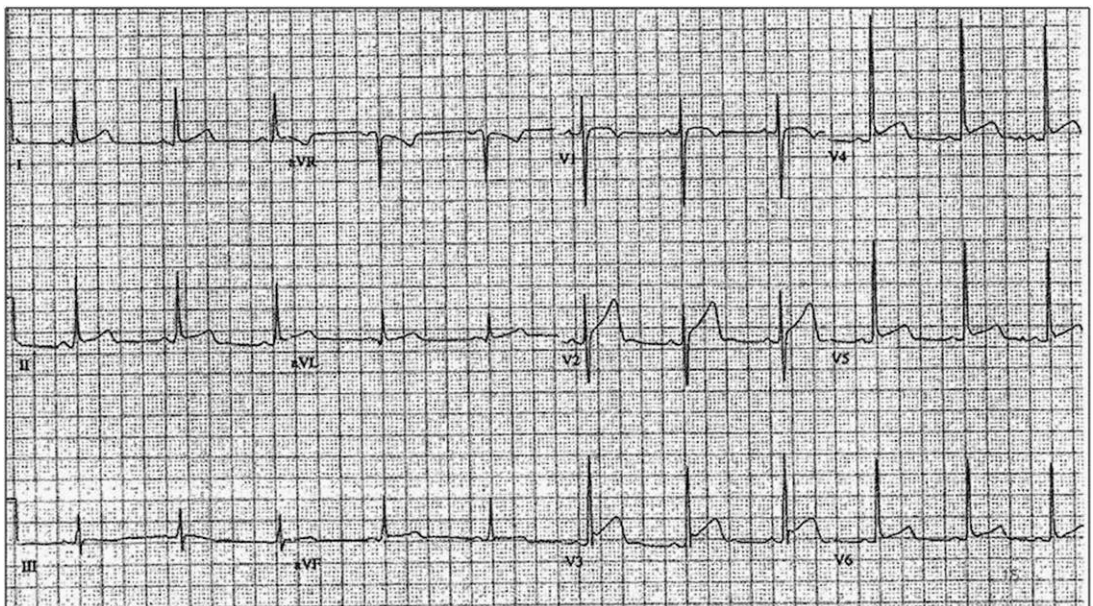


Fig. 4. Benign early repolarization with widespread, concave ST segment elevation.

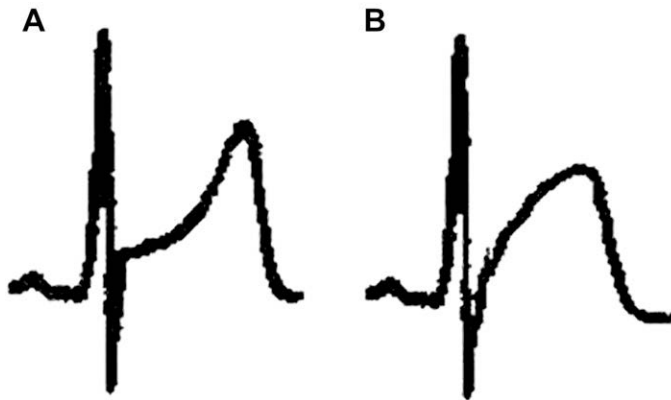


Fig. 5. ST segment elevation in benign early repolarization (A) and STEMI (B).

diagnosis of BER. The ST segment and T-wave abnormalities seen in BER will change very slowly over time with a diminution of ST segment elevation over decades; STEMI-related changes, on the other hand, evolve over minutes to hours.

Previous Infarction with Ventricular Aneurysm

The presence of ST segment elevation on ECG in patients with known prior myocardial infarction presents a special diagnostic challenge; these patients have known coronary disease and remain at an increased risk of recurrent STEMI. Patients who have had a past myocardial infarction can present with the various mechanical complications and have lingering ST segment elevation that has not resolved. Other patients, after a past myocardial infarction with remodeling, can present with persistent ST segment elevation attributable to aneurysmal dilatation of the infarcted segment of myocardium. In the prereperfusion era, rates of

left ventricular (LV) aneurysm approached 10%, but they have decreased dramatically since the introduction of fibrinolysis and mechanical reperfusion. Approximately one-quarter of patients with persistent ST segment elevation following STEMI will have an LV aneurysm as determined by echocardiography.⁸

When considered together, the patients' history of present illness, past medical history, and prior ECG are the most useful tools to identify LV aneurysm and distinguish the ST segment elevation from that of STEMI. Patients should be able to clarify whether they had a prior heart attack and to what degree their current presentation is similar or dissimilar. In this case, the bedside echocardiogram is generally unhelpful because the preexisting wall motion abnormality is difficult to distinguish from a new one.

The most frequent electrocardiographic manifestation (Figs. 6 and 7) of ventricular aneurysm is ST segment elevation, most often in the anterior

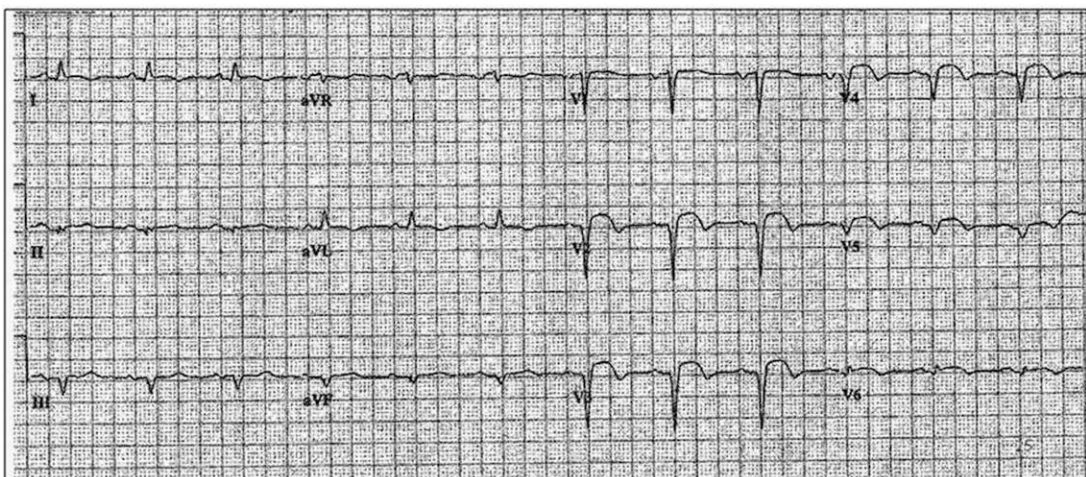


Fig. 6. LV aneurysm. ST segment elevation in leads V1 to V5 along with Q waves and T-wave inversion.

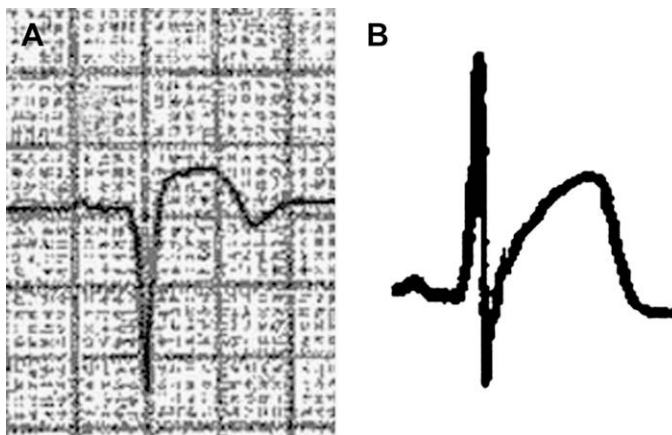


Fig. 7. ST segment elevation in LV aneurysm (A) versus STEMI (B).

distribution; inferior and lateral aneurysms are also encountered. The 12-lead electrocardiogram demonstrates ST segment elevation of varying morphologies and magnitudes, ranging from obvious, convex ST segment elevation to minimal, concave elevations. Pathologic Q waves are usually observed in leads with ST segment elevation. Inverted T waves of minimal magnitude are also seen in these same leads. Of course, a comparison with a past ECG since the myocardial infarction, if available, can determine if any change has occurred. Reciprocal ST segment depression is usually not present and this is helpful in distinguishing aneurysm from true STEMI. Lastly, the performance of serial ECGs can be invaluable, with dynamic changes suggesting STEMI and the absence of evolution suggesting aneurysm.

Coronary Vasospasm (Prinzmetal Angina)

Coronary vasospasm presents along a wide spectrum of disease severity, ranging from intermittent chest pain (so-called variant angina) to clinically severe STEMI equivalents with concomitant heart failure and ventricular fibrillation. It is exceptionally common among patients presenting to emergency departments with chest pain syndromes. The Clopidogrel and Acetyl Salicylic Acid in Bypass Surgery for Peripheral Arterial Disease (CASPAR) trial found 49% of patients presenting with acute chest pain who did not have a culprit lesion by angiography did have coronary vasospasm by acetylcholine challenge.⁹ Coronary vasospasm can occur in patients across a broad demographic without clear gender or race associations; moreover, it can occur along the length of any of the coronary arteries. Coronary vasospasm is associated with tobacco, cocaine use, and ergonovine derivatives and can occur in both atherosclerotic

as well as angiographically normal vessels. There is a form of coronary vasospasm caused by allergic histamine release, called Kounis syndrome.¹⁰ The final diagnosis of coronary vasospasm is made in the heart catheterization laboratory. Prior use of provocation tests with acetylcholine and methylergonovines has largely fallen out of favor.

Because acute coronary vasospasm can lead to near complete cessation of blood flow in the affected coronary artery, its presentation and pathophysiology parallels STEMI with the important distinction of absent vessel thrombosis. Rapid response to nitrates is a hallmark of coronary vasospasm. Treatment of normotensive patients with ST segment elevation remains a useful strategy to identify those with rapidly resolving syndromes when vasospasm is more likely. When considering reperfusion treatment, it is important to consider the possibility of coronary vasospasm.

The association of coronary vasospasm with cocaine (and other amphetamines) ingestion presents another challenge. Cocaine is both vasoconstrictive as well as thrombogenic, leaving users at an increased risk of both mechanisms of coronary flow obstruction and resultant infarction. In patients with a clear amphetamine toxidrome (eg, hypertension, agitation, and so forth) as well as ST segment elevation, it is prudent to treat the patients with intravenous benzodiazepines and oral or topical nitrates with repeat performance of the ECG to determine if persistent ST segment elevation is present. It must be stressed that STEMI remains the working diagnosis and, if unable to demonstrate resolution of the ST segment elevation within a short period of time, reperfusion therapy should follow. Refer to **Figs. 8** and **9** for a depiction of ST segment elevation as seen in coronary vasospasm.

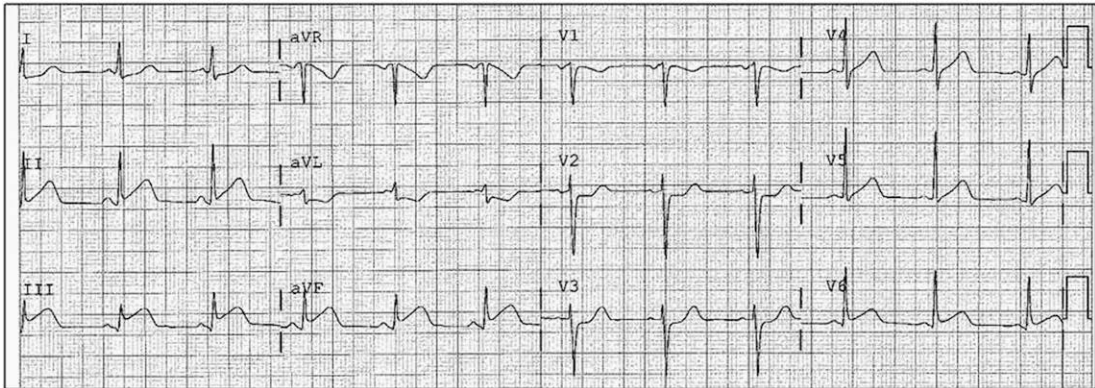


Fig. 8. Coronary vasospasm: ST segment elevation in the inferior leads along with ST segment depression in leads aVL, V2, and V3. This patient presented with chest pain and was ultimately diagnosed with coronary vasospasm (Prinzmetal angina). This form of ST segment elevation is indistinguishable from that seen in STEMI.

Other Non-STEMI ST Segment Elevation Syndromes

Takotsubo cardiomyopathy (apical ballooning syndrome)

Apical ballooning syndrome (ABS) is a recently identified disease in cardiology. In its short history, it has had different names, including *Takotsubo cardiomyopathy* and the *broken heart syndrome*, and it falls along the spectrum of acute stress-induced cardiomyopathies. Although the name and classic presentation involve dyskinesia at the LV apex, multiple variations, including basal and midcavitary dyskinesia, have also been reported. The clinical presentation of ABS is frequently indistinguishable from ACS and is well known to mimic STEMI.¹¹ Patients can present with the full spectrum of chest pain equivalents from vague atypical chest pain to severe chest pain with acute heart failure and malignant dysrhythmia.

The spectrum of ECG findings is likewise varied, from minimal abnormality to profound anterior ST

segment elevation (**Fig. 10**). In its most common apical form, the ECG changes are limited to the anterior precordial leads, reflecting apical location of injury. The inferior leads can also be involved, reflecting inferoapical injury. Because the history and ECG and even echocardiogram can be indistinguishable from true AMI, the diagnosis of ABS is made in the heart catheterization laboratory following diagnostic angiography.

Brugada syndrome and idiopathic ventricular fibrillation

The Brugada pattern includes right bundle branch block (RBBB) (both incomplete and complete) with ST segment elevation in leads V1 to V3; the natural history of this syndrome is sudden cardiac death. Brugada syndrome was first described as a clinical entity in 1992 to explain the observation that a particular cohort of patients prone to ventricular fibrillation despite structurally normal hearts had a distinct ECG pattern.¹² Since its first description,

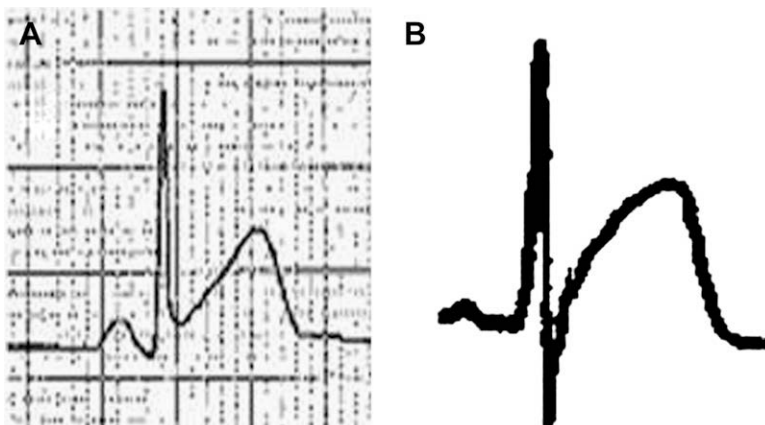


Fig. 9. ST segment elevation in coronary vasospasm (A) versus STEMI (B).

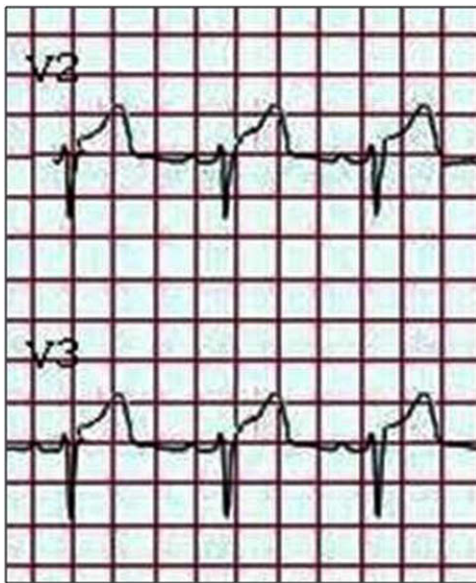


Fig. 10. ST segment elevation in Takotsubo cardiomyopathy.

a great deal has been learned about this relatively rare inherited disease. It occurs in approximately 5 out of 100 000 people across all nationalities, has an autosomal-dominant inheritance pattern, and a male predominance (~70%).¹³ Three subtypes have been described, with subtle variations in the shape of the ST segment. The underlying mechanism, resulting from a gene mutation, is a sodium channel loss of function.¹⁴

Clinically, the disease can present at any age, but the mean age of sudden death is 41 years. The characteristic ECG findings (**Fig. 11**) are dynamic and may vary from one ECG to the next. Chest pain is not part of the Brugada syndrome; however, syncope is a significant poor prognostic marker. The natural history includes cardiac arrest resulting from polymorphic ventricular tachycardia and/or ventricular fibrillation. The disease and ECG findings can be exacerbated by multiple medications as well as conditions that alter myocardial membrane electrical stability (eg, fever, hypokalemia, ischemia, bradycardia, and increased autonomic tone). Vaughan Williams class Ic antiarrhythmic medications (eg, flecainide and procainamide) are potent sodium channel blockers and are clinically used to elicit the Brugada pattern in suspected cases. A large number of medications in addition to sodium channel blockers have been reported to elicit a Brugada-like pattern on ECG. The significance of these findings to a particular patient remains unclear.

Hyperkalemia

As the serum potassium concentration increases, changes occur in ECG that can mimic ACS,

including STEMI.¹⁵ The elevation of the ST segment occurs following peaking of the T wave and widening of the QRS complex, suggesting high serum potassium concentrations. Although the accompanying ST segment elevation may be most impressive in a single region, the ECG changes of hyperkalemia are present throughout the limb and precordial leads.

Postelectrical cardioversion/defibrillation

Following transthoracic electrical cardioversion or defibrillation, the ECG can manifest ST segment elevation, among other abnormalities. ST segment elevation has been noted following both cardioversion for atrial as well as ventricular tachyarrhythmias in up to 20% of patients. Additionally, ST segment deviation occurs following countershock by internal cardioverter-defibrillator in 25% of patients.¹⁶ The ST segment elevation is transient and resolves within minutes but can be profound, as much as 5 mm.¹⁷ Although impressive and alarming, this finding has not been associated with evidence of ongoing myocardial injury or additional adverse sequelae.¹⁸

Hypothermia and Osborn waves

Prominent J-point elevations, also known as Osborn waves, are a common finding in patients who are hypothermic.¹⁹ When profound, the ST segment can be elevated, mimicking STEMI. Other findings include bradycardia and motion artifact. The mechanism and clinical implications of the Osborn wave remain unclear, but it is a transient finding that resolves with normothermia. Osborn waves can occur with either therapeutic hypothermia or accidental hypothermia.²⁰

STEMI Confounding Patterns

The confounder electrocardiographic patterns are those ECG entities that markedly reduce the electrocardiogram's ability to detect changes related to ACS, not because the findings mimic STEMI but because the condition obscures the detection of ST segment elevation. The commonly confounding patterns include left bundle branch block (LBBB), ventricular paced rhythms, and LV hypertrophy.

LBBB

LBBB is the most classic example of a confounder and is exceptionally common in the evaluation of patients with chest pain. This pattern is linked very closely with ischemic heart disease in that the development of LBBB is associated with significant heart disease; it also identifies a population at an increased risk for STEMI, acute cardiovascular complication, and poor outcome.²¹ The LBBB pattern on the ECG includes an elevated

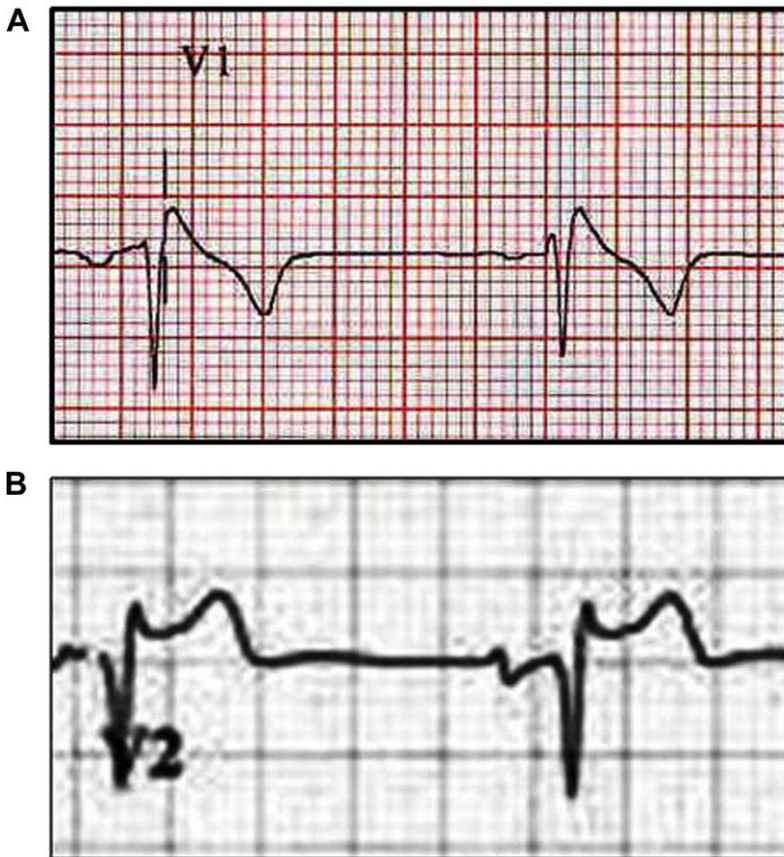


Fig. 11. Two forms of ST segment elevation as seen in the Brugada syndrome. (A) Convex form. (B) Concave (saddle-type) form.

ST segment at baseline making it impossible to use the standard STEMI criteria for ST segment elevation. The issue is further complicated by the fact that LBBB itself is a risk factor for cardiac death in that a new LBBB can be the presenting ECG pattern in a proximal left anterior descending coronary artery (LAD) occlusion, thus a new LBBB pattern ECG in patients with a compatible clinical scenario is considered a *STEMI equivalent*. Unfortunately, the rate of patients with AMI who present with new LBBB remains so small the mere presence of new or presumed new LBBB does not itself confer an increased risk of AMI.²² Moreover, although LBBB is associated with a poor prognosis for patients with chest pain, the diagnostic utility of its presence alone has been shown to be weak with respect to the diagnosis of AMI.²³

Two recurring scenarios illustrate the difficulty of correctly identifying AMI in patients with LBBB. The first is patients with an intermediate risk story and a previously undiagnosed or unrecognized LBBB. The question, then, is this: should this new LBBB be considered a STEMI? In considering the answer to this important question, it is instructive to recall

the mechanism through which AMI causes a new LBBB infarction of that portion of the conduction system related to LAD occlusion proximal to the first septal perforator branch. Given the proximal location of the occlusion in the LAD, these AMIs involve a larger area of ischemic heart muscle and tend to have more severe presentations and hemodynamic compromise. It is likewise less common for the new LBBB to be the ECG finding of AMI in patients with an otherwise nonthreatening clinical presentation; in other words, these patients with AMI tend to be rather ill on presentation, with a toxic appearance, pulmonary congestion, and compromised perfusion.

The second clinical scenario occurs when patients with known LBBB present with acute chest pain, potentially suggestive of AMI; the clinical challenge is focusing on the possible presence of AMI. This situation is the most appropriate application of the criteria Sgarbossa and colleagues²⁴ derived from the Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries (GUSTO) trial. These criteria are based on the finding that STEMI, when presenting

in the setting of LBBB, often seems different than the LBBB at baseline. Of course, an understanding of the ECG in the LBBB presentation is important. Such an understanding allows the clinician to recognize the expected findings of LBBB and, thus, the inappropriate findings that are associated with AMI, not only those described by Sgarbossa and colleagues but also other abnormalities. The 12-lead ECG (Figs. 12 and 13) in patients with LBBB records the abnormal ventricular activation as it moves from right to left, producing a broad, mainly negative QS or rS complex in lead V1. In lead V6, late intrinsicoid deflection is noted, resulting in a positive, monophasic R wave; similar structures are frequently found in leads I and aVL. Poor R wave progression or QS complexes are noted in the right to mid precordial leads, rarely extending beyond leads V4 or V5. QS complexes may also be encountered in leads III and aVF. The anticipated or expected ST segment–T-wave configurations are discordant, directed opposite from the terminal portion of the QRS complex, and called QRS complex–T-wave axes discordance. As such, leads with either QS or rS complexes may have markedly elevated ST segments, mimicking AMI. Leads with a large monophasic R wave demonstrate ST segment depression. The T wave, especially in the right to midprecordial leads, has a convex upward shape or a tall, vaulting appearance, similar to the hyperacute T wave of early myocardial infarction. The T waves in leads with the monophasic R wave are frequently inverted. Loss of this normal QRS complex–T-wave axes discordance in patients with LBBB may imply an acute process, such as AMI. An inspection of the ECG in patients with LBBB must be performed,

looking for a loss of this QRS complex–T-wave axes discordance.

Patients with known LBBB presenting with a clinical presentation potentially suggestive of AMI is the most appropriate application of the criteria Sgarbossa and colleagues²⁴ derived from the GUSTO trial. These criteria are based on the finding that STEMI, when presenting in the setting of LBBB, often seems different than the LBBB at baseline. Unfortunately, although the most accepted and widely used clinical criteria for diagnosis of AMI in LBBB, the Sgarbossa criteria suffer from low sensitivity of 36% while retaining a relatively robust specificity of 96% in the validation cohort. The most appropriate course in these cases, then, is to proceed most briskly in patients meeting the Sgarbossa criteria (with a score of 3 or more) and, in those who do not, relying on the pretest probability based on history and clinical risk to determine the overall risk of AMI. This rule states that 3 specific ECG criteria (Fig. 14, Table 1) are independent predictors of AMI in patients with LBBB. The ECG criteria suggesting a diagnosis of AMI, ranked with a scoring system based on the probability of such a diagnosis, include (1) ST segment elevation greater than 1 mm, which was concordant with the QRS complex (score of 5); (2) ST segment depression greater than 1 mm in leads V1, V2, or V3 (score of 3); and (3) ST segment elevation greater than 5 mm, which is discordant with the QRS complex (score of 2). A total score of 3 or more suggests that patients are likely experiencing an AMI based on the ECG criteria. With a score less than 3, the electrocardiographic diagnosis is less assured, requiring

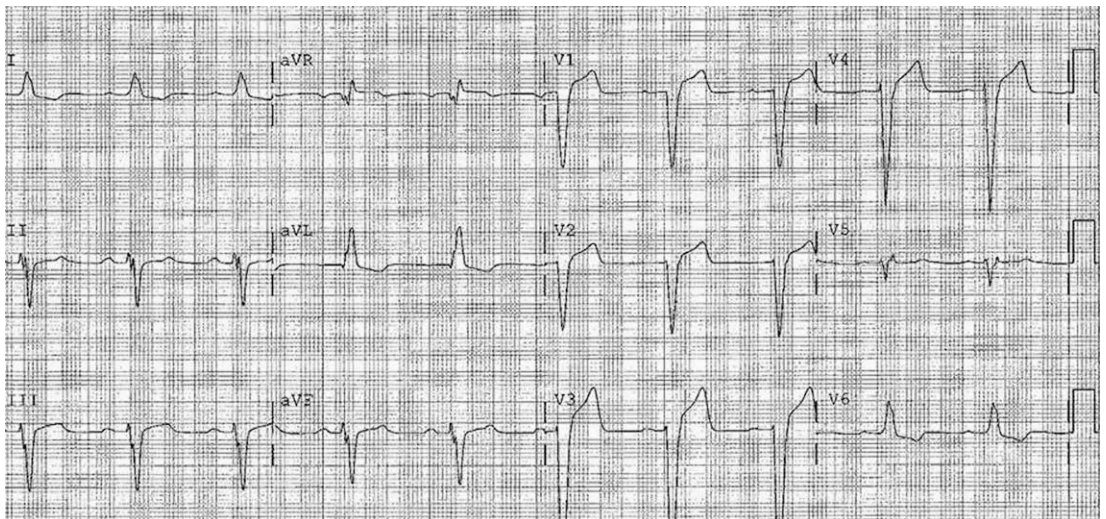


Fig. 12. ST segment and T-wave changes seen in the normal appearing left bundle branch block.



Fig. 13. ST segment elevation as seen in LBBB (A) versus STEMI (B).

additional evaluation. This clinical prediction instrument supports the contention that a detailed knowledge of the associated, or anticipated, ST segment–T-wave changes resulting from the abnormal ventricular conduction of the LBBB is a must. Such an understanding of the ECG in LBBB consequently allows the clinician to

recognize the unanticipated morphologies that may be suspicious for AMI.

LV Hypertrophy

LV hypertrophy (LVH) pattern is one of the most common clinical pathologic findings on the

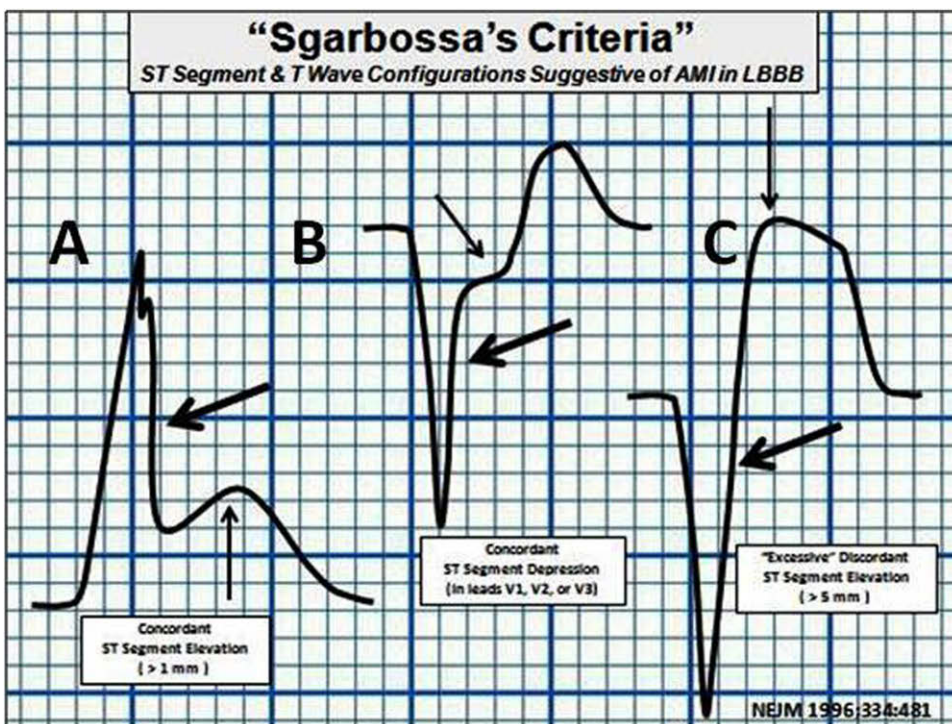


Fig. 14. The Sgarbossa criteria. (A) Concordant ST segment elevation. (B) Concordant ST segment depression limited to leads V1, V2, and V3. (C) Excessive discordant ST segment greater than 5 mm. Note that the relationship of the major, terminal portion of the QRS complex (*thick arrow*) and the initial portion of the ST segment (*thin arrow*) is key determinant in the consideration of the Sgarbossa criteria.

Table 1
The Sgarbossa criteria

Odds Ratios Supporting AMI and Prediction Scores for Independent Electrocardiographic Criteria

| Criterion | Odds Ratio (95% CI) | Score |
|--|---------------------|-------|
| ST segment elevation ≥ 1 mm concordant with QRS complex | 25.2 (11.6–54.7) | 5 |
| ST segment depression ≥ 1 mm in lead V1, V2, or V3 | 6.0 (1.9–19.3) | 3 |
| ST segment elevation ≥ 5 mm & discordant with QRS complex | 4.3 (1.8–10.6) | 2 |

Abbreviation: CI, confidence interval.

electrocardiogram. Although 50% of the adult population diagnosed with hypertension will have LVH by echocardiogram, only 20% of those will have characteristic ECG findings of LVH. Indeed, the presence of LVH does carry a worse prognosis for a given patient, and the presence of concomitant downsloping ST segment depression, the strain appearance further degrades prognosis.²⁵

There are many criteria for determining LVH by ECG, all of which rely on the increase in voltage reflected by the increased myocardial mass along with either leftward axis or left atrial abnormality. Unfortunately, all ECG criteria suffer from relatively poor performance characteristics (sensitivity approximately 50% and specificity approximately 80%) when validated against LV mass calculated by echocardiography or cardiac computed tomography (CT).²⁶ Although the detection of underlying, anatomic LVH is important with respect to patient management, in this particular instance, the authors are only addressing the secondary repolarization abnormalities caused by LVH, in essence, the ST segment deviations (both elevation and depression) as well as T-wave inversions.

Approximately 80% of patients with the LVH by voltage (Figs. 15 and 16) pattern demonstrate the strain pattern; the strain pattern includes significant ST segment changes (elevation and depression) and T-wave abnormalities (prominent T waves and T-wave inversion). ST segment elevation in the setting of LVH is almost exclusively seen in the anterior distribution (leads V1 to V4). ST segment elevation is encountered in this distribution along with prominent T waves; the ST segment elevation can reach up to 5 mm in height in the

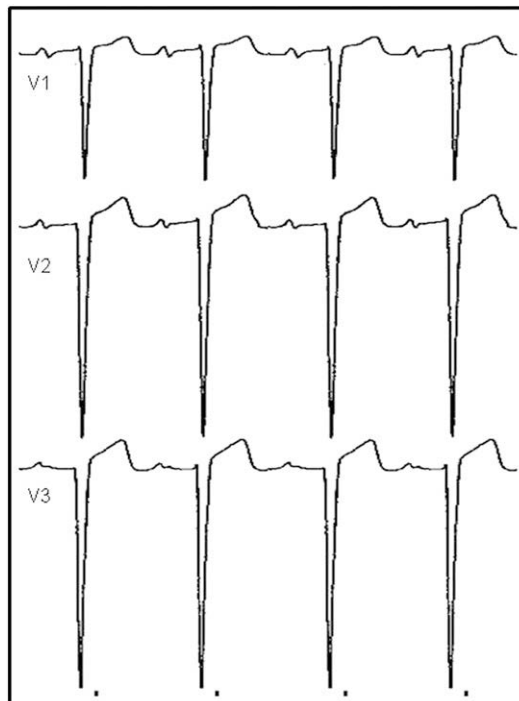


Fig. 15. ST segment elevation in leads V1 to V3 as seen in the LVH with strain pattern.

anterior leads. The lateral leads (leads I, aVL, V5, and V6) demonstrate large, prominent, positively oriented QRS complexes with marked ST segment depression and T-wave inversion. Although the strain pattern can also be seen inferiorly, inferior ST segment elevation should not quickly be ascribed to LVH. The LVH with pattern is associated with poor R-wave progression, most commonly producing a QS pattern; these complexes are located in leads V1, V2, and V3; furthermore, a leftward axis and left atrial abnormality add credence to ST segment elevation stemming from LVH. The ST segment elevation associated with LVH is generally unchanging over time, making a previous ECG for comparison particularly useful. Serial ECGs are similarly useful because there should be no evidence of evolving changes unless there is active ischemia.

Ventricular Paced Pattern from Implanted Device

More than 2 million people in the United States have an implanted pacemaker or internal cardioverter-defibrillator. Because a paced ventricular rhythm causes bundle-branch block morphology, it produces many of the same diagnostic limitations as LBBB described earlier. Moreover, patients with implanted cardiac devices

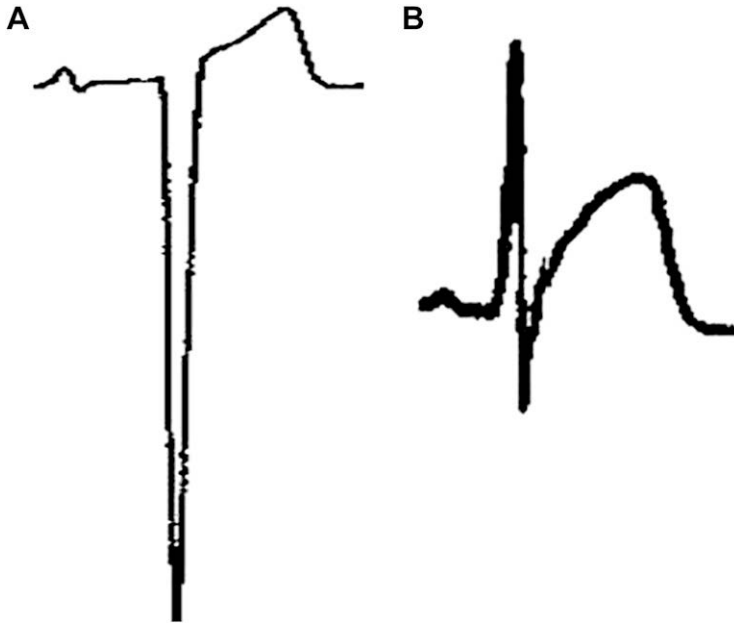


Fig. 16. ST segment elevation in LVH with strain (A) versus STEMI (B).

tend to be older and are more likely to have underlying heart disease.

Ventricular pacing leads can be placed in the apex of the right ventricle causing a left bundle pattern or on the surface of the LV either in a coronary vein or in an epicardial location causing more of a RBBB type of pattern. Patients with LV pacing leads frequently have such units placed for cardiac resynchronization therapy as part of the treatment

of heart failure. Patients presenting with a paced rhythm may or may not be dependent on their pacemaker to maintain a perfusing ventricular rate. It can be tempting to pause pacing and analyze the morphology of the underlying rhythm, but this is unreliable because of T-wave memory wherein the myocyte repolarization vector remains despite the change in depolarization vector.²⁷ Sgarbossa and colleagues²⁸ have investigated

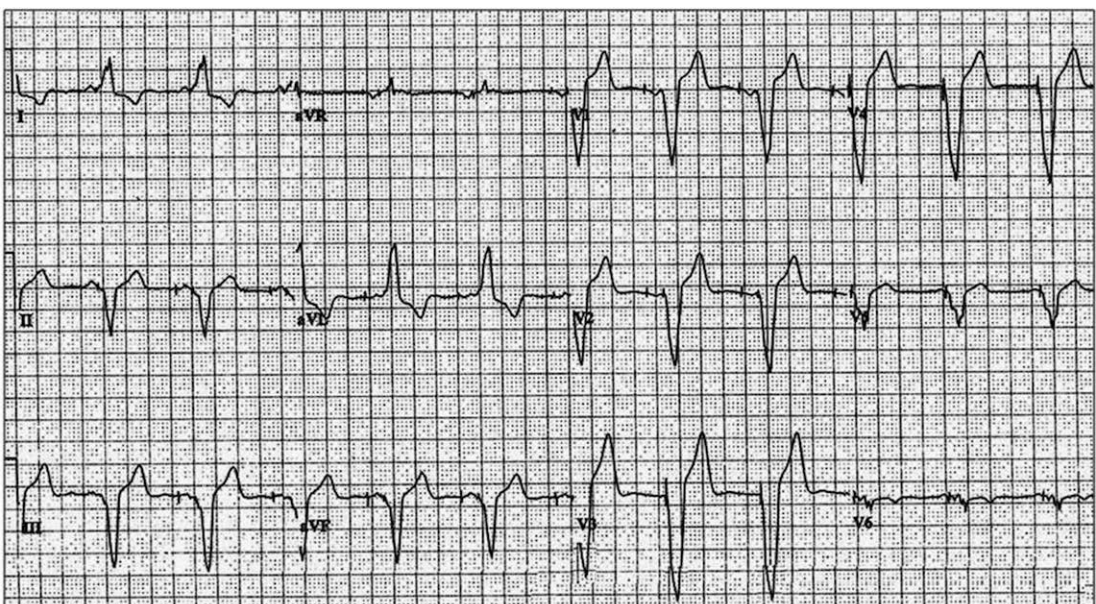


Fig. 17. ST segment elevation in the ventricular paced pattern.

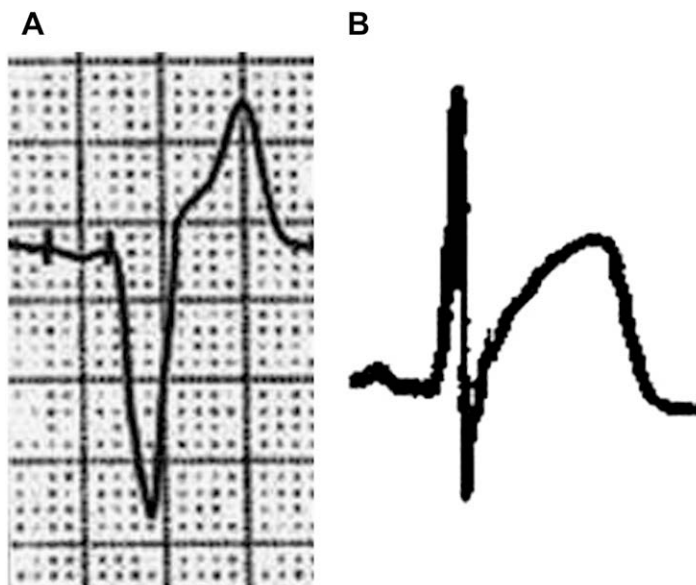


Fig. 18. ST segment elevation in the ventricular paced pattern (A) versus STEMI (B).

the utility of applying their criteria to patients with an LBBB caused by paced rhythm and found very poor sensitivity and modest specificity; in a very basic sense, they found similar findings as noted in the ECG diagnosis of AMI in the setting of LBBB. Refer to **Figs. 17** and **18** for a depiction of the ST segment changes seen in the ventricular paced pattern.

The RBBB has long been recognized as a high-risk feature in the presentation of AMI; in fact, it has been noted that the presence of a new RBBB on presentation with AMI significantly increases 30-day mortality.²⁹ Current guidelines require ST segment elevation be present with RBBB for the diagnosis of STEMI; further, the longstanding maxim has been the presence of RBBB does not alter the detection of ST elevation. However, this notion has been challenged by those who point out T-wave inversions in the anterior leads of patients with RBBB can impair the detection of more subtle ST segment changes; also, severe proximal coronary occlusions can be associated with new RBBB with hemiblock and, thus, do not demonstrate typical ST segment elevation.^{30–35} Patients with a clinically worrisome presentation and new bundle branch block (both left and right) should be carefully considered for AMI because the benefit of early reperfusion therapy is quite high in these patients.

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