

ORIGINAL ARTICLE

The electrocardiographic differential diagnosis of ST segment depression

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The importance of the electrocardiographic differential diagnosis of ST segment depression in patients presenting with acute chest pain is discussed.

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Patients presenting to the emergency department (ED) with acute chest pain potentially of ischaemic origin are evaluated with three principal tools: the history of the event, the 12-lead electrocardiogram (ECG), and cardiac enzymes and other serum markers of myocardial injury. The ECG is a powerful clinical tool in the evaluation of such patients and assists the physician in the selection of the proper treatment, in particular the application of treatment aimed at coronary reperfusion. Considerable electrocardiographic discussion has focused on the interpretation of ST segment elevation^{1,2}; comparatively little emphasis has been placed on the differential diagnosis of ST segment depression (STD). In many instances, STD is associated with acute coronary syndromes (ACS)—both acute ischaemia and acute infarction; this electrocardiographic pattern, however, may also be found in patients with non-ischaemic events, such as left bundle branch block (LBBB), left ventricular hypertrophy (LVH), and those with therapeutic digitalis levels (fig 1). Proper interpretation of the ECG in these patients will assist the clinician in arriving at the correct diagnosis—in effect, separating acute coronary syndrome from the non-ischaemic, more “benign” causes of STD. Correct interpretation of the ECG will then permit appropriate diagnostic and therapeutic decisions to follow. The following cases illustrate the use of the ECG in patients presenting with chest pain and electrocardiographic STD attributable to ACS, LVH, LBBB or digitalis.

CASE PRESENTATIONS

Case 1

A 58 year old woman with a past history of angina and diabetes mellitus presented to the ED with dyspnea and substernal chest pain. Examination revealed diaphoresis and was otherwise unremarkable. A 12-lead ECG (fig 2) demonstrated NSR with ST segment depression in the anterolateral leads (V2 to V5). The treating physician felt that the patient was experiencing myocardial ischaemia; nitrates, morphine, and aspirin were administered with resolution of the discomfort and normalisation of the electrocardiographic abnormalities. The patient was admitted to the hospital where serial cardiac enzymes did not show evidence of infarction.

Case 2

A 49 year old man with a history of diabetes mellitus presented to accident and emergency with chest pain of two hours duration, which was associated with diaphoresis and nausea. The examination was significant only for diaphoresis and rales in the lung bases. The 12-lead ECG (fig 3) demonstrated ST segment elevation (STE) in the anterolateral leads with ST segment depression in the inferior leads, consistent with an acute anterolateral wall myocardial infarction (AMI) with inferior reciprocal change. The patient received thrombolytic therapy with resolution of both his pain and the STE. Creatinine phosphokinase increase (with positive MB fraction) confirmed the diagnosis of AMI; echocardiographic examination revealed hypokinesia of the anterior wall with marked reduction in the left ventricular ejection fraction.

Case 3

A 43 year old man with a history of hypertension and diabetes mellitus presented to the ED with chest pain associated with diaphoresis and nausea. The examination was unremarkable. The 12-lead ECG (fig 4A) demonstrated pronounced STD in leads V1 to V3 with prominent R waves; these findings were felt to be consistent with posterior wall AMI versus anterior wall ischaemia; posterior electrocardiographic leads V8 and V9 (fig 4B) revealed ST segment elevation, confirming the diagnosis of acute, isolated posterior wall myocardial infarction. The patient received streptokinase with resolution of both his pain and normalisation of the electrocardiographic changes. Creatinine phosphokinase increase (with positive MB fraction) confirmed the diagnosis of AMI; echocardiographic examination revealed hypokinesia of the inferior and posterior walls of the left ventricle.

Case 4

A 69 year old woman with a past history of myocardial infarction, angina, and diabetes mellitus presented via ambulance to the ED with chest pain. Examination revealed partially reproducible chest discomfort. A 12-lead ECG (fig 5) demonstrated NSR with a LBBB; no evidence of inappropriate ST segment or T wave morphologies were seen. The LBBB pattern had been noted in the past on a previous ECG. The patient was admitted to the hospital where serial cardiac

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Abbreviations: ECG, electrocardiogram; ACS, acute coronary syndrome; LBBB, left bundle branch block; LVH, left ventricular hypertrophy; STD, ST segment depression

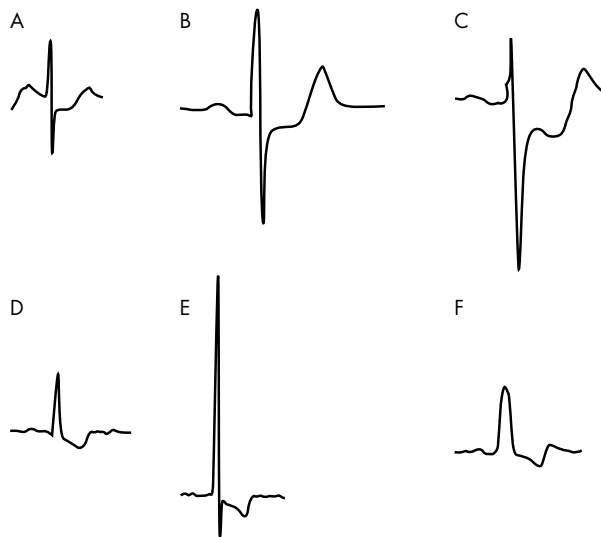


Figure 1 The various causes of electrocardiographic ST segment depression. (A) ST segment depression related to non-infarction ischaemia, horizontal in morphology. (B) Reciprocal ST segment depression in lead III in a patient with acute anterior wall AMI. (C) Lead V2 STD attributable to posterior wall AMI. (D) Digoxin effect. (E) Left ventricular hypertrophy. (F) Left bundle branch block.

serum markers were not increased, excluding the diagnosis of AMI. The history and examination did not suggest an acute coronary ischaemic event; the patient was discharged from the ED with musculoskeletal chest pain.

Case 5

A 49 year old woman with a past history of chronic renal insufficiency, congestive heart failure, and hypertension presented to the ED with dyspnea. The examination was remarkable for pulmonary congestion. A 12-lead ECG (fig 6) demonstrated NSR with LVH; the chest radiograph confirmed pulmonary oedema with cardiomegaly. The patient received oxygen, furosemide, topical nitrates, morphine, and aspirin; she slowly improved and was admitted to the hospital. No evidence of myocardial infarction was discovered.

Case 6

A 61 year old woman with a past history of congestive heart failure and atrial fibrillation presented via ambulance to the

ED with dyspnea. The patient used digoxin. The examination revealed rales bilaterally to the mid-lung zones. A 12-lead ECG (fig 7) demonstrated NSR with diffuse STD. The patient received aspirin, intravenous diuretics, and topical nitrates with resolution of the discomfort. Repeat ECG revealed no further change in the STD. The ST segment depression on the ECG was felt to result from the digoxin effect. The patient was discharged from the accident and emergency department with unremarkable follow up in days.

DISCUSSION

ST segment depression may be the initial abnormality on the ECG of patients with acute coronary syndromes. Electrocardiographic ST segment depression in this population may indicate one of four diagnoses: myocardial ischaemia (without infarction), acute posterior wall AMI, reciprocal ST segment change in the setting of AMI, and non-ST segment elevation AMI (formerly the non-Q wave AMI).

Acute coronary ischaemic syndromes

Acute coronary ischaemia (non-infarction)

STD attributable to ischaemia is often diffuse and can be located in both anterior and inferior leads, and is not necessarily localising. Extensive experience from cardiac stress testing shows that the actual configuration of the ST segment depression influences the specificity of this finding, with a downsloping segment more specific for the diagnosis of ischaemia than horizontal depression.^{3,7} A depressed but upsloping ST segment lacks adequate specificity for ischaemia. A combination of two diagnostic criteria have typically been required in at least one electrocardiographic lead to diagnose subendocardial injury—that is, myocardial ischaemia: at least 1.0 mm (0.10 mV) depression at the J point and either horizontal or downward sloping ST segment depression.^{3,4} Some investigators have postulated that patients with a “low probability” of coronary artery disease (that is—minimal cardiac risk factors) may need a stricter criteria of STD—such as greater than 1.5 mm or even greater than 2.0 mm of depression—to be regarded as significant.^{5,6} However, these criteria are based on several small studies of otherwise healthy, asymptomatic patients; the applicability of these criteria to patients presenting with acute cardiopulmonary symptoms is unclear. Until proved otherwise, STD in the patient with a clinical history consistent with an acute coronary event should be considered an ominous finding, requiring aggressive therapy and inpatient disposition. See figures 2, 8, and 9 for examples of STD related to acute ischaemia.

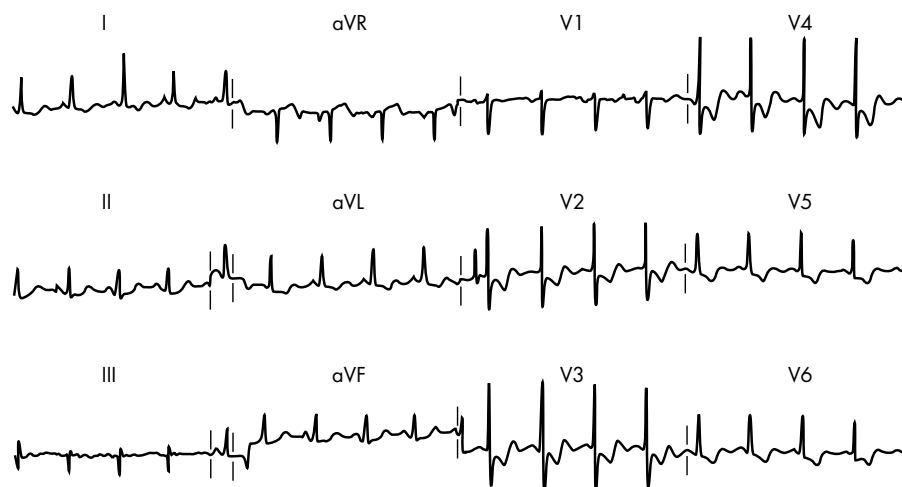


Figure 2 (Case 1) ST segment depression attributable to myocardial ischaemia (non-infarction)—ECG demonstrated NSR with ST segment depression in the anterolateral leads (V2 to V6) consistent with a non-infarction acute coronary ischaemic syndrome. The STD is downsloping in morphology, suggestive of ischaemia.

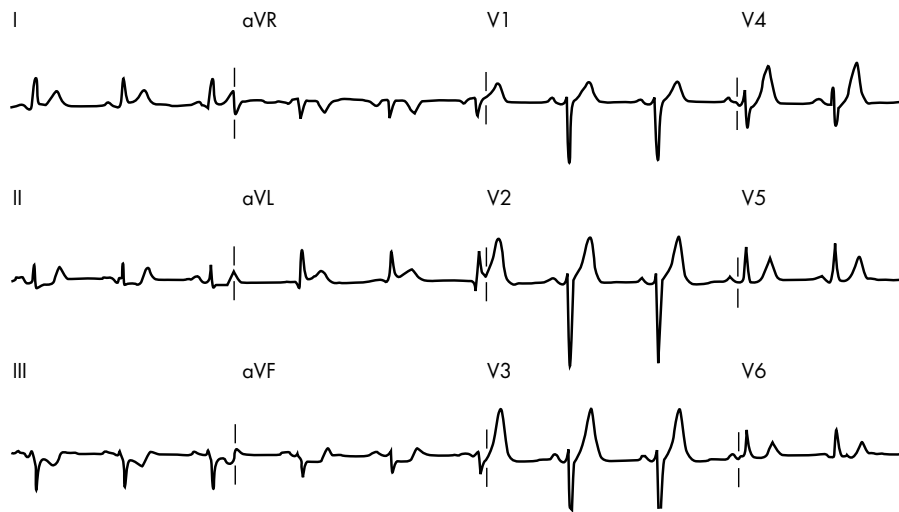


Figure 3 (Case 2) Reciprocal ST segment depression—the ECG demonstrated early anterolateral AMI with reciprocal ST segment depression in the inferior leads. Reciprocal ST segment depression in the setting of STE on the ECG strongly suggests an ST segment elevation AMI; furthermore, the presence of reciprocal change is also associated with an increased chance of poor outcome.

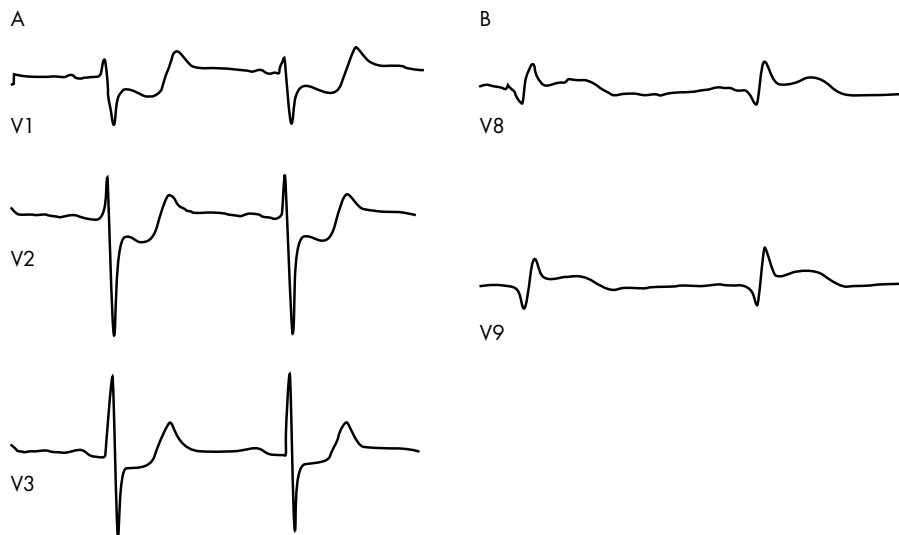


Figure 4 (Case 3) Acute, isolated posterior wall myocardial infarction. (A) The ECG demonstrated pronounced ST segment depression in leads V1 to V3 with prominent R waves; these findings are consistent with posterior wall AMI versus anterior wall ischaemia. (B) Posterior electrocardiographic leads V8 and V9 revealed ST segment elevation, confirming the diagnosis of acute, isolated posterior wall myocardial infarction.

Acute posterior wall myocardial infarction

Most cases of posterior AMIs are attributable to lesions in a dominant right coronary or left circumflex coronary artery and thus mainly affect the dorsal—or posterior—area of the heart. Acute posterior wall myocardial infarction occurs in up to 20% of AMIs, with the vast majority occurring along with inferior or lateral AMI.^{10–11} Isolated posterior wall AMIs, however, do occur.¹² Electrocardiographic abnormalities suggestive of an posterior wall AMI include the following (in leads V1, V2, or V3) (fig 4A): (1) horizontal STD with tall, upright T waves; (2) a tall, wide R wave; and (3) an R/S wave ratio greater than 1.0 in lead V2.^{3–11} STD in the right precordial leads may therefore represent either reciprocal change secondary to an inferior or lateral AMI or may indicate a posterior AMI. Boden *et al* noted that in patients presenting with angina or an anginal equivalent and an ECG demonstrating STD in leads V1–V3, about one half of the patients were found to have had a posterior wall AMI.¹³ Perhaps the best way to help the clinician determine the aetiology of right precordial horizontal STD is to use additional electrocardiographic leads that imag-

ine the posterior wall of the left ventricular directly—namely, leads V8 and V9 (fig 4B). ST segment elevation greater than 1 mm in the posterior leads V8 and V9 confirms the presence of posterior wall AMI.^{10–11–14}

An additional lead ECG uses the standard 12 leads in addition to other leads, including the posterior leads V8 and V9, placed under the left mid-scapular line and the left paraspinal border. Refer to fig 4B for STE seen in the patient with posterior AMI. Numerous studies have shown using additional lead ECGs in selective cases (that is, suspected posterior AMI) increases the sensitivity without sacrificing the specificity of detecting acute posterior wall AMI.^{10–11–14}

Reciprocal ST segment depression

Reciprocal STD—also known as reciprocal change—is defined as horizontal or downsloping STD in leads that are separate and distinct from leads manifesting STE. The causes of reciprocal change are thought to be secondary to coexisting distant ischaemia, a manifestation of infarct extension, or an electrophysiological phenomenon caused by displacement of

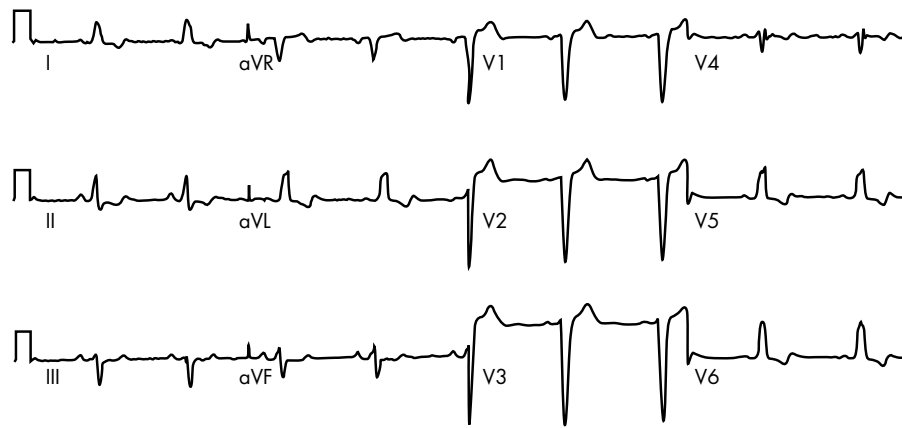


Figure 5 (Case 4) Left bundle branch block—a 12-lead ECG demonstrated NSR with a LBBB. The ST segment depression in the lateral leads is consistent with the altered conduction pattern in the LBBB patient; no evidence of inappropriate ST segment or T wave morphologies were seen. The LBBB pattern had been noted in the past on a previous ECG.

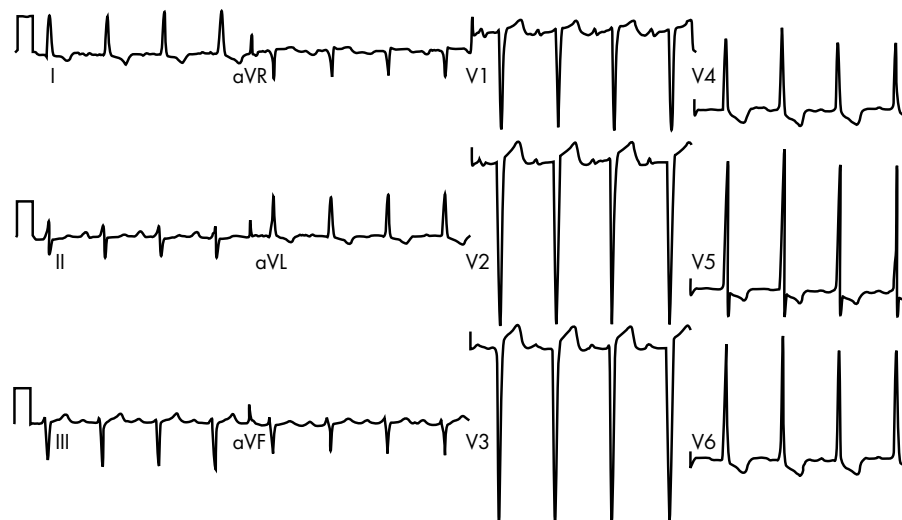


Figure 6 (Case 5) Left ventricular hypertrophy—12-lead ECG demonstrating NSR with LVH. The ST segment depression in the lateral leads (I, aVL, V5, and V6) is seen in approximately 70% of patients with electrocardiographic LVH; such a finding has been termed the “strain pattern.” While this finding may represent altered repolarisation attributable to the presence of LVH, it may also be a manifestation of myocardial ischaemia. The clinical history as well as the results of other investigations will guide the doctor in determining the diagnosis and appropriate disposition.

the injury current vector away from the non-infarcted myocardium.³ Reciprocal change can be identified in about one third of patients with anterior wall AMIs and up to 80% of patients with inferior AMIs will demonstrate anterior ST segment depression in leads V1, V2, or V3. The presence of reciprocal change increases the positive predictive value for a diagnosis of AMI to greater than 90%.⁸ Perhaps the greatest utility of reciprocal change is in patients with acute cardiac symptoms and ST segment elevation of uncertain aetiology; such is the case in approximately 5% to 10% of AMI patients in the ED.⁸ Although an STE AMI may be obvious in many instances, the presence of reciprocal change identifies a subset of patients with more extensive disease, and thus may benefit from more aggressive treatments. In the setting of an inferior AMI without obvious changes indicative of acute transmural ischaemia (that is, no STE), the presence of reciprocal, significant STD in lead aVL—especially if disproportionate to the size of the QRS complex—may herald early cardiac ischaemia. This lead is perhaps not as closely scrutinised as other leads, but recognition of this STD in aVL may give the clinician an “early warning” of an impending inferior AMI.⁹ Refer to figure 3 for an example of reciprocal change.

Non-ST segment elevation AMI

Patients with non-ST segment elevation AMI—formerly known as the non-Q wave AMI—may have transient and non-specific findings, such as ST segment depression (figs 8 and 9) or T wave abnormalities (fig 10) in any of the anatomic leads of the 12-lead ECG. Symmetric convex downward ST segment depression or inverted or biphasic T waves are characteristically seen. Differentiating non-ST segment elevation anterior myocardial infarction from posterior AMI can be difficult and can be done with the use of the additional lead ECG.^{10–14}

Non-acute coronary ischaemic syndrome causes of ST segment depression

Intraventricular conduction disorders

Intraventricular conduction delays such as LBBB and the associated ST segment-T wave abnormalities can mimic both acute and chronic ischaemic changes. Much has been written about the evaluation of the ST segment elevation in the presence of LBBB¹⁵; considering chest pain patients in the ED, LBBB is responsible for 15% of STE syndromes and is the second most frequently encountered electrocardiographic pattern responsible for non-ischaemic STE.^{17,18} LBBB, however, can also cause significant ST segment depression, and it is

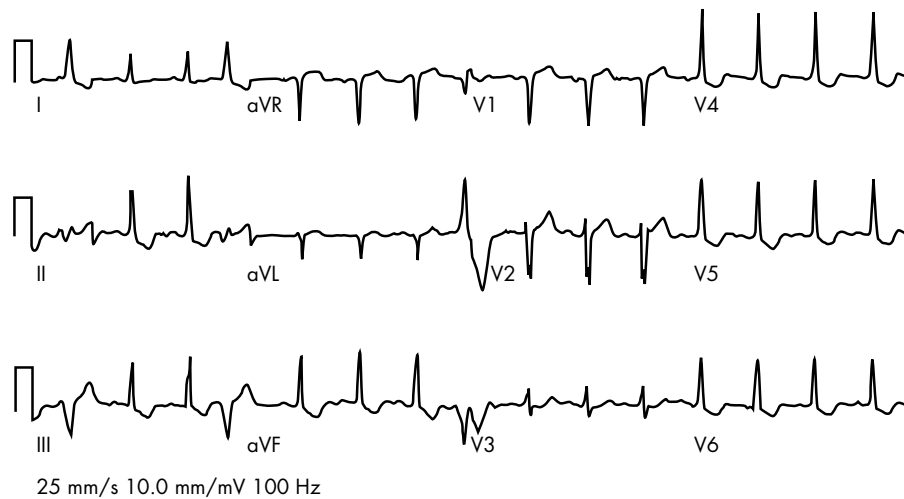


Figure 7 (Case 6) Digoxin effect— 12-lead ECG demonstrated NSR with diffuse ST segment depression. Repeat ECG revealed no change in the STD; a previous ECG was similar. The ST segment depression on the ECG was felt to result from the digoxin effect. The morphology of the ST segment depression is highly characteristic of the digoxin effect. In this example, the ST segment is depressed in a downsloping manner with a gradually increasing depression and more rapid return of the depressed segment to the baseline.

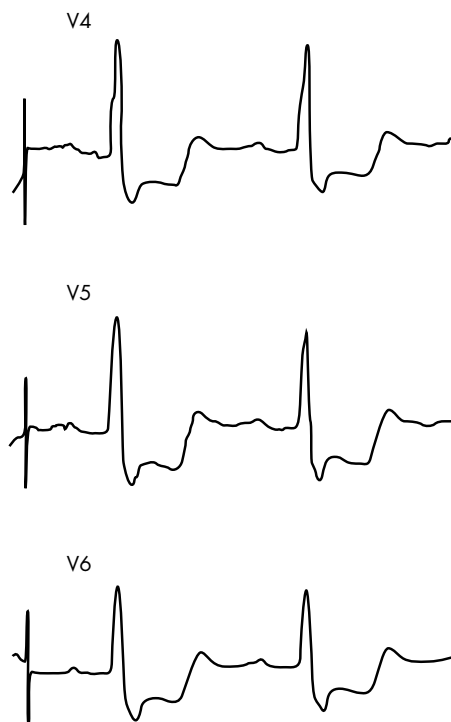


Figure 8 Non-ST segment elevation AMI—AMI characteristically presents with STE in approximately 50% of myocardial infarction patients; in the remainder of the AMI population, electrocardiographic ST segment depression and T wave inversion are frequent presentations. In this example, STD is seen in leads V3 to V6. Cardiac serum markers were increased, confirming the diagnosis of non-STE AMI.

imperative that these electrocardiographic changes be distinguished from those that occur in the presence of ACS.³ The “rule of appropriate discordance” states that in LBBB, ST segment-T wave configurations are directed opposite from the major, terminal portion of the QRS complex. As such, leads with either QS or rS complexes should have significantly elevated ST segments mimicking an AMI while leads with a large monophasic R wave demonstrate ST segment depression. T waves in leads with monophasic R waves are frequently inverted. Loss of this normal QRS complex-T wave discord-

ance may imply acute ischaemia in patients with LBBB.¹⁸ Refer to figure 5 for an example of LBBB.

Using data from the GUSTO-1 trial, Sgarbossa *et al* reported three specific electrocardiographic criteria that are independent predictors of infarction in the setting of LBBB.¹⁹ These criteria were ranked by a scoring system based on the probability of AMI: (1) ST segment elevation >1 mm concordant with the QRS complex (score of 5); (2) ST segment depression >1 mm in leads V1, V2, or V3 (score of 3); and (3) ST segment elevation >5 mm discordant with the QRS complex—“too much” discordance (score of 2). A score of 3 or more suggests that the patient is having an AMI based on the electrocardiographic criteria. With a score less than three, the electrocardiographic diagnosis is less assured and further non-electrocardiographic studies are indicated. Several, more recent studies have questioned the usefulness of these criteria and found the sensitivity to detect an AMI as low as 10%.^{20, 21} Despite this, many clinicians find these criteria useful and dispel the notion that it is not possible to diagnose AMI in the face of LBBB.

Left ventricular hypertrophy

There have been several electrocardiographic criteria and scoring systems proposed to diagnose LVH, with the Estes²² and Scott²³ criteria being most widely used; however, despite adequate specificity, the sensitivity to detect LVH using a wide variety of electrocardiographic criteria has ranged from only 12% to 29%.^{4, 24} This poor sensitivity and the inability to consistently appreciate the ST-segment—T wave changes created by LVH has confounded the ability to distinguish between ischaemic and LVH related STD.⁴ The LVH related repolarisation abnormalities are referred to as a “strain pattern”, and can be encountered in approximately 70% of LVH cases.¹ This strain pattern by itself has a 52% sensitivity in the recognition of LVH with a specificity nearing 95%.²⁵ This strain pattern is characterised by downsloping STD with abnormal T waves in leads with prominent R waves (I, aVL, V5, and V6). The downsloping STD—usually without J point depression—is greater than 1 mm and is followed by an inverted T wave. This T wave inversion is asymmetrical, with gradual downsloping and a rapid return to baseline, often with the terminal portion of the T wave becoming positive (so called “overshoot”).²⁶ Additionally, T wave inversion is greater in lead V6 than in V4, with greater than 3 mm of depression in V6.¹ Identifying this strain pattern is consistent with LVH repolarisation changes and could easily be confused with acute ischaemia. The strain pattern, however, has a relative permanence and should not

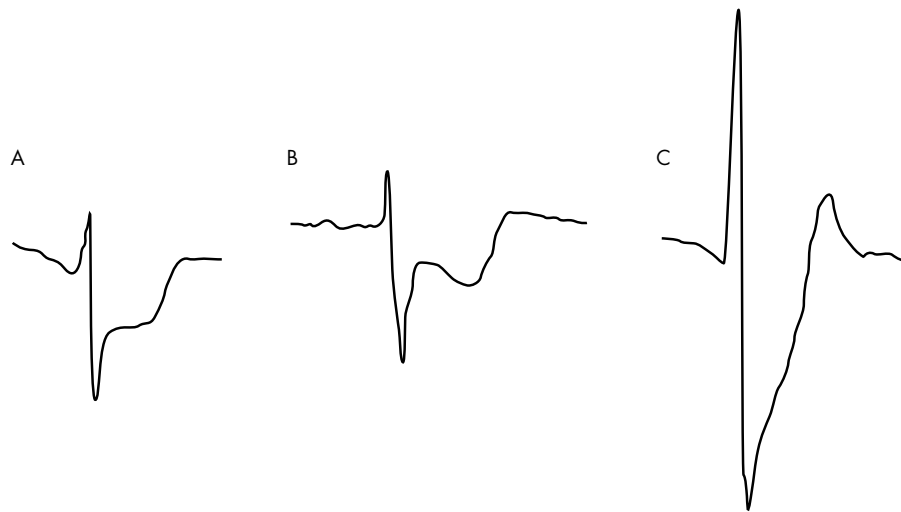


Figure 9 The types of ST segment depression in the patient with acute coronary ischaemic syndrome and no evidence of infarction. (A) Horizontal. (B) Horizontal. (C) Downsloping. (D) Upsloping.

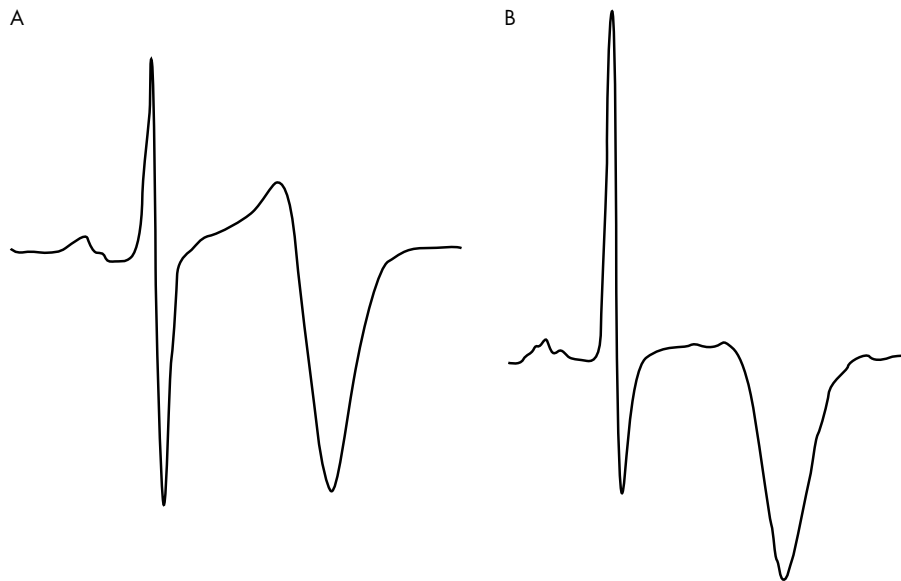


Figure 10 (A) The right precordial leads V1 to V3 in a chest pain patient ultimately diagnosed with AMI. (B) Additional posterior leads V8 and V9 reveal STE, confirming the diagnosis of isolated posterior wall AMI.

change over the short-term as compared with the dynamic changes seen in ACS. Refer to figure 6 for an example of LVH.

Digoxin effect

At therapeutic levels, digitalis produces characteristic electrocardiographic changes, including PR interval prolongation (vagal effect), STD, T wave inversion, and shortening of the QT interval. These changes are referred to as the digitalis effect, which must be distinguished from digitalis toxicity, which manifests primarily as cardiac arrhythmia. The electrocardiographic manifestations of digitalis—the digoxin effect—are as follows: “scooped” ST segment depression, most prominent in the inferolateral precordial leads and usually absent in the rightward leads; flattened T waves; increased U wave; and shortening of the QT interval.^{3 4 26} Occasionally, the J point is depressed, mimicking acute ischaemia. This extreme example of digitalis effect usually occurs only in those leads with tall R waves.⁴ Patients with baseline STD or T wave inversion will have an accentuation of these repolarisation abnormalities when treated with digitalis, while patients with normally upright T waves will experience T wave inversion.³ Often it

may be impossible to differentiate the STD created by digitalis and those occurring with ischaemia. In general, however, digitalis will create a “sagging” ST segment while ischaemia creates the typical horizontal or downsloping depression. See figure 7 for an example of digoxin effect on the ECG.

CONCLUSION

There are several strategies to assist the clinician in differentiating among the various causes of electrocardiographic ST segment depression. The most time sensitive concern is determining whether the STD is attributable to an ACS or attributable to less acute causes such as LBBB, LVH or digitalis. With regard to ACS, determining if the STD represents reciprocal change or a posterior AMI also has significant implications. Using the rule of appropriate discordance, using additional ECG leads in select cases, and performing waveform shape analysis all can be of great benefit when faced with the patient with cardiopulmonary complaints and STD.

Obtaining serial ECGs is perhaps the most powerful tool available to helping distinguishing from among the causes of

ST segment changes. The dynamic ECG changes seen with ACS are absent from the relative short-term permanence seen with LVH, LBBB, and the digitalis effect. A comparison with a prior ECG tracing is, of course, invaluable. However, with the often unavailability of old ECGs and without the luxury of time to obtain serial ECGs, a thorough knowledge of confounding electrocardiographic patterns in patients with STD will assist the physician in making timely and important clinical decisions.

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