CHAPTER SIX

Acute Coronary Ischemia and Infarction

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Emergency physicians encounter patients on a daily basis with symptoms suggestive of ACS. Among these symptoms, chest pain is by far the most common. According to the National Hospital Ambulatory Medical Care Survey, conducted by the Centers for Disease Control and Prevention, an estimated 110 million visits were made to hospital emergency departments in 2004; chest pain was the chief complaint for 5,637,000 (5.4%) of them.[1] Not all patients with chest pain have ACS. The American Heart Association calculated that 879,000 people with ACS were discharged from hospitals in 2003 (considered a conservative estimate). This number was derived by adding the number of hospital discharges for myocardial infarction (MI) (767,000) and for unstable angina (112,000).[2] Given this patient volume, an understanding of the ECG changes indicative of ACS is paramount to emergency physicians and extremely important for patients, as it allows immediate risk stratification and therapeutic decisions (eg, administration of a β-blocker, thrombolysis, or activation of a catheterization team for primary percutaneous intervention [PPCI]).

The Prehospital ECG

The prehospital ECG is an underutilized component in modern ACS care. Prehospital ECGs can be obtained by advanced EMS personnel and transmitted while en route to a hospital in advanced EMS systems in the United States. If transmission is a problem, the computer read is highly accurate and can be called into the receiving emergency department. This will allow emergency department personnel to be ready to initiate fibrinolysis or alert cardiology for PPCI. The use of prehospital ECGs reduces door-to-needle time for in-hospital fibrinolysis by a mean of 10 minutes and, according to data from the National Registry of Myocardial Infarction 2, reduces the door-to-balloon-time for PPCI by a mean of 23 minutes.[3] The use of prehospital ECGs can also help EMS systems triage more efficiently. The patient with MI with ST-segment elevation (STEMI) who is in cardiogenic shock benefits from being transferred to a center with PPCI capability and emergent revascularization.[4] If an ECG is not obtained during prehospital transport or if the patient arrives by private vehicle, a door-to-ECG time of 10 minutes is encouraged.[5]

Normal and Nondiagnostic ECGs

Patients with ACS include those whose clinical presentations cover the following range of diagnoses: unstable angina, MI without ST-segment elevation (NSTEMI), and STEMI.[5] The standard 12-lead ECG and use of additional ECG techniques, including right-sided leads (V_3R through V_6R), posterior leads (V_7, V_8, and V_9), and continuous ST-segment monitoring, allow detection of changes suggestive of ischemia. However, among patients who have chest pain and are subsequently diagnosed with AMI by cardiac isoenzyme concentration (CK-MB), 6% to 8% have normal ECGs and 22% to 35% have nonspecific ECGs.[6] In fact, malpractice cases often focus on the performance and use of electrocardiography (Table 6-1).[7,8] Patients with AMI who are mistakenly discharged from the emergency department have short-term mortality rates of about 25%, at least twice what would be expected if they were admitted.[9] The legal costs that can result from such cases constitute the largest category of losses from emergency department malpractice litigation.[10]

ECG Changes of ACS

Abnormalities in the ST segment, QRS complex, and T waves indicate the extent of ischemia, injury, and necrosis.

TABLE 6-1.

Most common causes of malpractice losses related to acute chest pain and patients at higher risk of being discharged from the emergency department with AMI[7,8]

Most common causes of malpractice losses related to chest pain:

- failure to obtain an ECG
- misinterpretation of an ECG
- failure to record data from the clinical evaluation

Characteristics of patients at risk of being discharged from the emergency department with AMI:

- women younger than 55 years of age
- nonwhite patients
- shortness of breath as chief symptom
- normal or nondiagnostic ECG

Patterns of Ischemia

In the presence of normal conduction, the T wave is usually upright in I, II, and V_3 to V_6; inverted in lead aVR; and variable in leads III, aVF, aVL, and V_1, with rare normal inversion in V_2. In ischemia, T waves are inverted, symmetric, and mostly transient (the aberrations occur while the patient is symptomatic) (Figure 6-1). Therefore, any T-wave inversions in V_2 to V_6 are considered pathologic. In such a case and in the presence of chest pain or its equivalent, there is usually no myocardial damage, as measured by CK-MB or troponin, and the diagnosis is unstable angina (UA) (Figure 6-1). If the T-wave inversion is persistent, there is nearly always some minimal troponin elevation; this pattern frequently is termed *non–Q wave MI*. A more contemporary term denotes no ST-segment elevation recorded, appropriately termed *non-STEMI* (NSTEMI). UA and NSTEMI result from a nonocclusive thrombus, small risk area, brief occlusion (spontaneously reperfused), or an occlusion that maintains good collateral circulation. In many such cases, ST-segment elevation or other ST-segment or T-wave abnormalities would have been noted if an ECG had been recorded at the appropriate time.[6]

Patterns of Injury

In the early stages of an evolving STEMI, prominent T waves are termed *hyperacute* and are defined as larger than 6 mm in the limb leads and larger than 10 mm in the precordial leads. Unfortunately, these prominent T waves are not specific for ischemia. Table 6-2 describes the

TABLE 6-2.

Differential diagnosis of prominent T waves

AMI (usually bulky, wide waves associated with chest pain and other associated cardiovascular symptoms)

Normal variant (in mid precordial leads of young patients)

Hyperkalemia (usually not associated with chest pain)

Intracranial hemorrhage (associated with prolonged QT and presence of U waves)

Left ventricular hypertrophy

LBBB

differential diagnosis of prominent T waves. Table 6-3 describes the evolution of ischemic changes in relation to the onset of patient symptoms. Bulky, wide T waves are highly suggestive of early STEMI and might be seen within the first 30 minutes after the onset of symptoms. In fact, the height of these T waves generally correlates well with the acuteness of the injury. At this early phase, there is no cellular death. With prolonged and significant occlusion (90% of the coronary artery), the prominent T waves remain as ST deviation develops (Figures 6-2 and 6-3). ST-segment elevation represents a myocardial region at risk for (irreversible) MI and usually leads to at least some myocardial cell death (measured by troponin elevation). The ST-segment elevation seen with AMI is called a *current of injury*, indicating that damage has occurred to the epicardial layer of the heart. Normally, the ST segment is isoelectric because no net current flow is occurring at this time. MI alters the electrical charge on the myocardial cell membranes, resulting in abnormal current flow and, in turn, ST-segment deviations.

Disagreement exists regarding whether the ST-segment elevation or depression should be measured from the upper edge of the PR segment or from the TP segment to the upper edge of the ST segment at the J point. It should be noted, however, that the PR segment can be altered by atrial infarction, abnormal atrial repolarization, or pericarditis; thus, the TP segment is often preferred as the baseline. STEMI is defined as ST-segment elevation of at least 0.1 mV (1 mm) in two or more contiguous leads[11] (Figures 6-3, 6-4, and 6-5). Leads I and aVL are considered contiguous leads, reflecting the "high lateral" portion of the left ventricle, even though they are not proximate to each other on the 12-lead ECG. Measurement of ST deviation at other levels (60 or 80 msec after the J point) is not advised. Some studies have suggested that a well-informed subjective interpretation of the appearance of the ST segment is more accurate than measured criteria.[12,13] The morphology of the ST segment is equally important, because it evolves from a normal minimally upward concave to one that is straight and then convex. (See discussion in Chapter 9, ACS Mimics: Non–AMI Causes of ST-Segment Elevation.)

After prolonged, non-reperfused coronary occlusion, as regional ST segments resolve toward the isoelectric level, T waves invert (resulting in a biphasic appearance) in the same region (Figure 6-6). The ST segment usually stabilizes within 12 hours, with full ST-segment resolution over the ensuing 72 hours. ST-segment elevation completely resolves within 2 weeks after 95% of inferior and 40% of anterior MIs; persistence for more than 2 weeks is associated with greater morbidity.[14] T waves can normalize over days, weeks, or months (Figure 6-2).[15] In the presence of previous T-wave inversion, re-

TABLE 6-3.

Evolution of ischemic changes in relation to onset of symptoms

T waves: Peak within 30 minutes but can last several hours. T waves invert with reperfusion (spontaneous or therapeutic); frequently normalize in days, weeks, or months; and, less commonly, persist inverted indefinitely.
ST segment: Elevates within minutes to hours. Without early therapeutic reperfusion, usually stabilizes within 12 hours but might remain elevated for days. Usually resolves within 2 or 3 weeks. Persistence after 3 or 4 weeks is highly suggestive of a ventricular aneurysm.
Pathologic Q waves: Evolve within hours. With early reperfusion, might disappear completely. Without early reperfusion, persist indefinitely in 70% of cases.

TABLE 6-4.

Prognostic features of ST-segment elevation (in decreasing order of importance) associated with larger MI, higher mortality rate, and greater benefit from reperfusion therapy*

Anterior location, compared with inferior or lateral[16-20]
Presence of ST-segment elevation and ST-segment depression or total ST deviation (absolute sum of ST-segment elevation and ST-segment depression)[21-23]
ST score (the sum of all ST-segment elevations) greater than 1.2 mV (12 mm)[19]
Distortion of the terminal portion of the QRS (as evidenced by loss of S wave in leads with RS configuration), or J point more than 50% of the height of the R wave[24]

*Some patients without these features might also have a large AMI.

occlusion of the coronary artery manifests as ST-segment re-elevation and normalization of terminal T-wave inversion, called *T-wave pseudo-normalization* because the T wave flips upright. With upright T waves, pseudo-normalization *should not* be assumed if the previous ECG showing T-wave inversion was recorded more than 1 month earlier. Table 6-4 lists the prognostic features of ST-segment elevation.

ST-Segment Depression

Primary ST-segment depression, if not caused by posterior STEMI or reciprocal changes to ST-segment elevation, is an ECG sign of subendocardial ischemia (Figure 6-7). In the context of ACS, it indicates UA/NSTEMI. ST-segment depression of even 0.5 mm from baseline is associated with increased mortality, but it is particularly significant when it is more than 1 mm (0.1 mV) in two or more contiguous leads.[25] This adverse prognostic association is independent of an elevated troponin level.[26] Although ST-segment depression, especially up-sloping ST-segment depression, might be baseline and stable, the depression associated with UA/NSTEMI is transient and dynamic with a morphology that is usually flat or down-sloping. Even 1 mm of ST-segment depression following an R of more than 20 mm is very specific for ischemia; R less than 10 mm is sensitive but not specific (Figures 6-7 and 6-8).

TABLE 6-5.

Reciprocal ST-segment depression: changes according to area of infarction

Anterior STEMI: Reciprocal ST-segment depression in at least one of leads II, III, and aVF in 40%-70% of cases (Figures 6-3 and 6-10) suggestive of a proximal LAD artery occlusion.
Inferior STEMI: Reciprocal ST-segment depression usually is present in leads I and aVL, and often in the precordial leads, especially V_1 through V_3 in 56% of cases (Figure 6-9).
Posterior STEMI: Reciprocal ST-segment depression in V_1 through V_4, with or without ST-segment elevation in leads V_5 and V_6 or leads II, III, and aVF (truly reciprocal to what would be ST-segment elevation on posterior leads) (Figure 6-15). Upright T waves and posterior lead ST-segment elevation help to differentiate this entity from inferior STEMI reciprocal ST-segment depression.

ST-segment depression of more than 2 mm in three or more leads carries a high probability that cardiac enzymes will be elevated. If PPCI is not performed, the 30-day mortality rate is 35%.[27]

Reciprocal ST-segment depression improves the likelihood of STEMI. It represents the electrical mirroring phenomenon observed on the ventricular wall opposite the transmural injury and therefore does not reflect ischemia in the territory of the ST-segment depression. Table 6-5 describes the reciprocal changes associated with anterior, inferior, and posterior STEMI.

Patterns of Necrosis

When necrosis occurs, the electrical voltages produced by this portion of the myocardium disappear. Instead of positive (R) waves over the infarcted area, Q waves are recorded (either a QR or QS complex). Q-wave formation begins within 1 hour and can be completed in 8 to 12 hours. Before the reperfusion era, MI was classified based on its clinical pathology either as Q-wave or non–Q-wave MI, or as transmural versus subendocardial MI. These terms were later discovered to be clinically and pathologically unrelated. Q waves were considered markers of irreversible infarction. Today it is well known that Q waves eventually disappear in up to 30% of patients with AMI who receive no reperfusion therapy and that, with early reperfusion therapy, the Q waves disappear earlier (within a few days to weeks). Hence, AMI is now classified as STEMI or NSTEMI. The Q-wave/non–Q-wave distinction remains somewhat useful, because Q waves are associated with a lower ejection fraction and a larger MI.

Normal septal q waves must be differentiated from the pathologic Q waves of infarction.

TABLE 6-6.

Q-wave equivalents in the precordial leads

R-wave diminution or poor R-wave progression
Reverse R-wave progression, in which R waves increase then decrease in amplitude across the precordial leads (although this must be distinguished from precordial electrode misconnection)
Tall R waves in leads V_1 and V_2, representing "Q waves" of posterior infarction

Normal septal q waves are characteristically narrow and of low amplitude (as a rule, <0.04 sec in duration). A Q wave is abnormal if its duration is 0.04 second or more in lead I, all three inferior leads (II, III, aVF), or leads V_3 through V_6 (Figure 6-10). A large QS complex can be a normal variant in leads aVL, V_1, and, rarely, in V_2, making it difficult to distinguish from an old anterior septal MI. Q waves in V_3 will hint toward infarct. In general, in the acute setting, pathologic ischemic Q waves (whether in V_1 to V_2 or aVL) without ST changes are relevant only from a patient's historical perspective. Risk stratification and decision to treat are based primarily on clinical presentation, risk factors, and ST- and T-wave abnormalities. There are Q-wave equivalents suggestive of ischemia (Table 6-6) and Q waves that are pathologic but nonischemic (Table 6-7).

Reperfusion

A combination of angiographic evidence of microvascular perfusion and resolution of ST-segment elevation are the best predictors of outcome from STEMI.[19] In fact, coronary occlusion can be transient or dynamic with cyclic reperfusion and reocclusion and is represented as transient ST-segment elevation (occurs in up to 20% of STEMI).[28] On continuous ST-segment monitoring after reperfusion therapy, recovery of the ST segment to less than 50% of its maximal height by 60 minutes is associated strongly with Thrombolysis in Myocardial Infarction category 3 (TIMI-3) reperfusion and even more strongly associated with good microvascular perfusion. A less sensitive but highly specific predictor of reperfusion is terminal T-wave inversion identical to Wellens T waves. In patients who have STEMI, the presence of negative T waves very early after presentation or very soon after therapy is associated with a good prognosis (Figure 6-6).

Wellens Syndrome

Wellens syndrome (Figure 6-11) refers to angina with T-wave inversion or biphasic T-waves in the left anterior descending (LAD) artery distribution, particularly V_2 through V_4, in the presence of persistent R waves and critical stenosis of the LAD artery.[29–31] ST-segment elevation or depression is not a necessary component of this syndrome. The ECG pattern is also present in a pain-free state. Wellens' group noted that patients who were managed medically without angioplasty fared poorly; 75% developed an anterior wall AMI, usually within a matter of days. Identical T-wave morphology is recorded in approximately 60% of patients who have undergone successful reperfusion therapy for anterior STEMI,[32,33] suggesting that Wellens syndrome is a clinical condition created by spontaneous reperfusion of a previously occluded critical stenosis. Similar patterns occur in other coronary distributions, but the syndrome was described originally in the LAD artery. Wellens syndrome is distinguished from benign T-wave inversion by 1) longer QT interval (>425 msec as opposed to 400 to 425 msec) and 2) location V_2 through V_4 (as opposed to V_3 through V_5).

Area of Infarction

The cardiac blood supply is delivered by the three main coronary arteries (Figure 6-12). The ECG changes seen in patients with ACS can also be described in terms of the location of the infarct. The anatomic location of the infarct determines the leads in which the typical patterns appear. For example, with an acute

TABLE 6-7.
Differential diagnosis of pathologic Q waves

Ischemic Q waves
LBBB
Left ventricular hypertrophy
Chronic lung disease
Hypertrophic cardiomyopathy
Dilated cardiomyopathy

TABLE 6-8.
Indications to obtain a 15-lead ECG

ST-segment depression in leads V_1 through V_3
Borderline ST-segment elevation in leads V_5 and V_6 or borderline ST-segment elevation in leads II, III, and aVF
All ST-segment elevation, inferior wall AMIs (ST-segment elevation in leads II, III, and aVF)
Isolated ST-segment elevation in lead V_1 or ST-segment elevation in leads V_1 and V_2

anterior wall MI, ST-segment elevations and tall hyperacute T waves appear in one or more of the anterior leads (chest leads V_1 through V_6 and extremity leads I and aVL) (Figures 6-3, 6-4, 6-13, and 6-14). With an inferior wall MI, the ST-segment elevations and tall hyperacute T waves are seen in inferior leads II, III, and aVF (Figure 6-9). Table 6-9 describes the relationship between the area of occlusion and the distribution of the ST-segment elevation.

Several distinct observations regarding anatomic distribution patterns will enhance the understanding of the ECG findings of ACS. The characteristic feature of an anterior wall infarct is the loss of normal R-wave progression in the chest leads. Normally, the height of the R wave increases progressively from lead V_1 to lead V_6. An anterior infarct interrupts this progression; the result is pathologic Q waves in one or more of the chest leads. Anteroseptal refers to these changes in V_1 and V_2. Changes in V_3 and V_4 are present in isolated anterior infarcts and V_5 and V_6 for anteroapical infarcts (Figure 6-13). Because distribution patterns often overlap, a simplified approach describes leads I, aVL, and V_1 to V_6 as anterior and then specifies the leads that show Q waves and ST/T changes.

Posterior Infarctions

Posterior infarctions occur on the posterior surface of the *left* ventricle as a result of occlusion of either the left circumflex or the right coronary artery. Isolated posterior infarction is uncommon (occurring in 3% to 8% of all AMIs). STEMI in this distribution is difficult to diagnose, as it does not appear as ST-segment elevation in the conventional 12-lead ECG. Fortunately, pure posterior STEMI is uncommon and most cases extend either to the lateral wall, with changes in V_6, or to the inferior wall, with changes in II, III, and aVF .

In pure posterior AMI, V_1 through V_3 offer several diagnostic clues[39] (Figure 6-15). The sensitivity and positive predictive value for the diagnosis of posterior STEMI increase with the addition of posterior leads V_7, V_8, and V_9.

Rapid recognition of acute posterior MI is of clinical importance for several reasons. First, patients with acute inferior or lateral wall MI who also have posterior involvement are experiencing a large infarct. With increasing infarct size, the risk of dysrhythmia, left ventricular dysfunction, and death increases proportionally. Second, approximately 5% of patients initially diagnosed with ST-segment depression representing subendocardial ischemia in the septal leads actually have experienced posterior STEMI, with a subsequent unfortunate delay in reperfusion therapy. The easiest way to distinguish between subendocardial ischemia of the LAD artery represented as ST-segment depression and reciprocal ST-segment depression

TABLE 6-9.
ST-segment elevation, location of STEMI, and corresponding coronary artery

AMI location	ST-segment elevation
Inferior	II, III, and aVF
Inferior and right ventricular	II, III, and aVF plus V_1
	Right-sided ECG: V_4R
Inferior and posterior	II, III, and aVF plus ST-segment depression V_1 through V_4
Inferior and lateral	II, III, and aVF plus I, aVL, and/or V_5 and V_6
Anterior	V_2 through V_4
Lateral	I, aVL, V_5, and/or V_6
Right ventricular	V_1 through V_3
	Also, V_1R through V_6R, especially V_4R
Anteroseptal	V_1 through V_4
Posterior	ST-segment depression in V_1 through V_4
	ST-segment elevation in posterior leads V_7, V_8, and V_9

from a posterior STEMI is to obtain a 15-lead ECG that includes posterior leads (Table 6-8).

Body surface mapping, an ECG technique that uses numerous leads on a patient's anterior and posterior chest, allows more complete visualization of cardiac electrical activity. It is an extension of an additional-lead ECG. Output from body surface mapping is displayed in a 12-lead ECG format, an 80-lead ECG format, and on color contour maps. Posterior AMI is the most common AMI detected by body surface mapping not detected by 12-lead ECG.[40] Body surface mapping is discussed in greater detail in Chapter 8, New ECG Technologies for Detection of Acute Myocardial Ischemia and Infarction in the Emergency Department.

Right Ventricular Infarctions

Right ventricular infarction is present in 25% (range, 20%–60%) of patients with inferior infarction.[41] The ECG demonstrates ST-segment elevation of greatest magnitude in lead III (inferior/right ventricular infarction compared with leads II and aVF) and ST-segment elevation in the right chest leads (V_1R through V_6R); in addition, it is important not to miss the subtle ST-segment elevation in lead V_1 (Figure 6-16). Clinically, the presentation of a patient with chest pain, distended jugular veins, and hypotension should prompt an immediate ECG with the addition of right-sided leads, as a large right ventricular infarction might cause cardiogenic shock. Patients with inferior wall STEMI with right ventricular infarction have a markedly worse prognosis (both acute cardiovascular complications and death) than do patients with isolated inferior wall STEMI.

Ventricular Aneurysm

After a large anterior MI (less commonly after an inferior MI), a ventricular aneurysm might develop. It is critical to distinguish ST-segment elevation from an aneurysm and ST-segment elevation from an MI. The persistence of ST-segment elevation for 4 weeks or more suggests a ventricular aneurysm. When no previous ECG is available, the presence of a QS wave in the setting of ST-segment elevation without T-wave inversion is highly suggestive of a left ventricular aneurysm (Figure 6-17). If left ventricular aneurysm is suspected, echocardiography should be performed to rule out the diagnosis prior to administration of a thrombolytic. If echocardiography is not possible and the suspicion is high, the patient should be referred for a diagnostic angiogram with possible PPCI where a ventriculogram can also be performed. Ventricular aneurysms can lead to congestive heart failure and ventricular arrhythmias, and a thrombus could form in an aneurysm and break off, causing a stroke or other embolic complication.

Right Bundle-Branch Block with Myocardial Infarction

MI can be diagnosed relatively easily in the presence of right bundle-branch block (RBBB). MI affects the initial phase of ventricular depolarization, producing abnormal Q waves. When RBBB and an infarct occur together, a combination of patterns is seen: the QRS complex is abnormally wide (0.12 sec or more) as a result of the bundle-branch block, lead V_1 shows a terminal positive deflection, and lead V_6 shows a wide S wave (Figure 6-18).

TABLE 6-10.
Sgarbossa criteria and index score for diagnosis of AMI* in the setting of LBBB[42]

Criterion	Sensitivity	Specificity	Points
ST-segment elevation of 1 mm or more in the same direction as the QRS complex	73%	92%	5
ST-segment depression of 1 mm or more in lead V_1, V_2, or V_3	25%	96%	3
ST-segment elevation greater than 5 mm opposite the direction of the QRS complex	31%	92%	2

*An accurate diagnosis requires a minimum total score of 3.

Left Bundle-Branch Block with Myocardial Infarction

Currently, no single or combination diagnostic approach will *reliably* reveal AMI in a timely fashion in the presence of left bundle-branch block (LBBB). The best strategy for detection of AMI encompasses a sound understanding of the anticipated ST-segment changes resulting from LBBB, identification of predefined abnormal findings suggestive of ischemia, a comparison with old ECGs, and an examination of serial ECGs. As a general rule, LBBB typically shows poor R-wave progression in the septal leads. Consequently, in LBBB, the normal septal R waves are lost, simulating the pattern seen with an anterior wall infarct. LBBB also interrupts the late phases of ventricular stimulation and therefore produces secondary ST/T changes.

During the past 5 decades, several ECG signs have been proposed to aid in the diagnosis of infarction, but, because of methodologic and applicability limitations, none has gained widespread acceptance. The best validated criteria are those of Sgarbossa and colleagues,[42] who developed and validated a clinical prediction rule based on 10 ECG criteria tested in 26,003 patients; in this study population, 131 (0.5%) patients with AMI had LBBB. Table 6-10 lists the three ECG criteria with highest specificity (90%) for diagnosing AMI in this setting. Figure 6-19 demonstrates an ECG with an uncomplicated LBBB as well as an evolving AMI in the presence of the LBBB.

Other groups have applied the Sgarbossa criteria to patients with chest pain and LBBB in the emergency department and found much less promising results—very low sensitivity coupled with poor interobserver reliability. Gula et al[43] published the results of a community-based cohort study in which the Sgarbossa algorithm had been applied, followed by a decision analysis regarding thrombolysis made with or without use of the algorithm in an emergency department between 1994 and 1997. The ECG algorithm indicated positive findings in only 3% of presentations and had a sensitivity of 10% (95% confidence interval, 2%–26%). Despite the low prevalence and poor sensitivity of the Sgarbossa criteria for AMI in the setting of LBBB, the high specificity of these criteria is reason for acute care physicians to know and understand them.

The ECG diagnosis of MI and myocardial

KEY FACTS

- STEMI is defined as at least 1 mm of ST-segment elevation in two or more contiguous leads.
- During STEMI, the morphology of the initial portion of the ST segment evolves from a normal minimally upward concave shape to one that is large and straight ("hyperacute") and then convex.
- A persistent concave ST morphology is more common in nonischemic pathologic states.
- T-wave pseudo-normalization *should not* be assumed if the previous ECG showing T-wave inversion was recorded more than 1 month earlier.
- ST-segment depression is associated with an adverse prognosis in the ACS patient, regardless of troponin elevation.
- Reciprocal ST-segment depression strongly favors STEMI over other causes of ST-segment elevation on the ECG (eg, pericarditis, benign early repolarization) and portends a worse prognosis than for patients with STEMI without reciprocal changes.
- Wellens syndrome is associated with a high risk of anterior MI if medical therapy alone is employed. Invasive therapy (angioplasty, stent placement, or revascularization) is associated with much better outcomes.
- Sgarbossa's criteria for AMI in the setting of LBBB have very high specificity, although poor sensitivity. Their presence, therefore, warrants aggressive anti-ischemia therapy, but their absence does not rule out ACS.

ischemia in patients with a paced rhythm is challenging. However, the diagnosis of AMI in the setting of a paced rhythm can be made in a limited number of cases. Sgarbossa and colleagues[44,45] recently applied their criteria (see section on diagnosis of AMI in the setting of LBBB) and reported the value of ST-segment abnormalities in the diagnosis of AMI during ventricular pacing. ST-segment elevation of 5 mm or more in predominantly negative QRS complexes is the best marker, with a sensitivity of 53% and specificity of 88%, and was the only criterion of statistical significance (Figure 6-20). The other criteria were not statistically significant, were less specific, and had a high interobserver variability. Beyond the criteria, the presence of new ST-segment abnormalities strongly suggests the diagnosis of AMI or severe ischemia and the need for possible emergency revascularization. Most frequently, emergency physicians encounter patients with a history of chest pain and a nondiagnostic paced ECG. If the history is concerning, similar to a new LBBB, one should consider fibrinolytics or a diagnostic coronary angiography with possible percutaneous intervention. If uncertain, consider obtaining an emergency cardiology consultation for help in defining the next steps, which likely will depend on the clinical pretest probability and the resources available at the time of consultation.

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FIGURES

FIGURE 6-1.

T waves from ischemia are inverted and symmetric, mostly transient, and present while the patient is symptomatic. T-wave inversions in V_2 through V_6 are always considered pathologic.

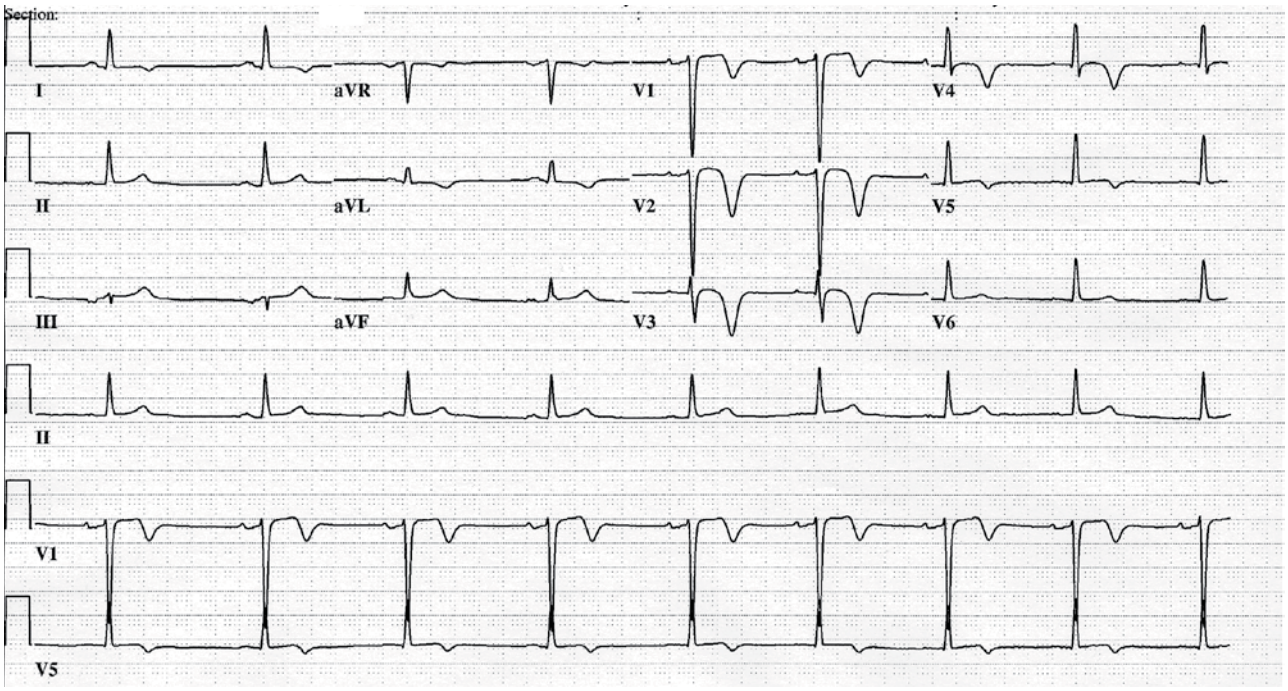
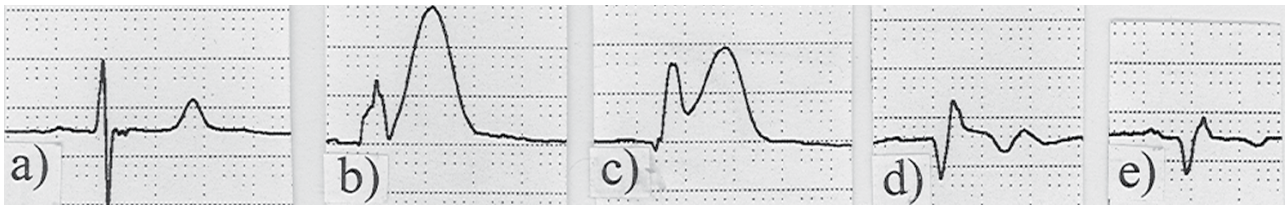


FIGURE 6-2.

Evolution of anterior STEMI, lead V_2. A: Arrival at emergency department after 30 minutes of chest pain. B: 45 minutes after onset of symptoms, with hyperacute T waves. C: 70 minutes after onset of symptoms, with ST-segment elevation, referred for emergent PPCI with stent to proximal LAD (symptoms-to-balloon time of 105 minutes). D: 3 hours post procedure, Q is present, suggesting transmural infarct. ST segments have decreased and T waves have inverted (markers of reperfusion). E: 5 days after PPCI. ST segment is back to baseline. T-wave inversion is near baseline and eventually might normalize.



FIGURES

FIGURE 6-3.

In the early stages of evolving ischemia, a pattern of injury presents as hyperacute T waves (bulky and wide T waves). This ECG has peaked T waves in V_3 through V_6. (Note that the J point is at baseline.) ST-segment elevations are present in V_6, I, and aVL. A bifascicular block and ST-segment depression represent reciprocal changes in II, III, and aVF.

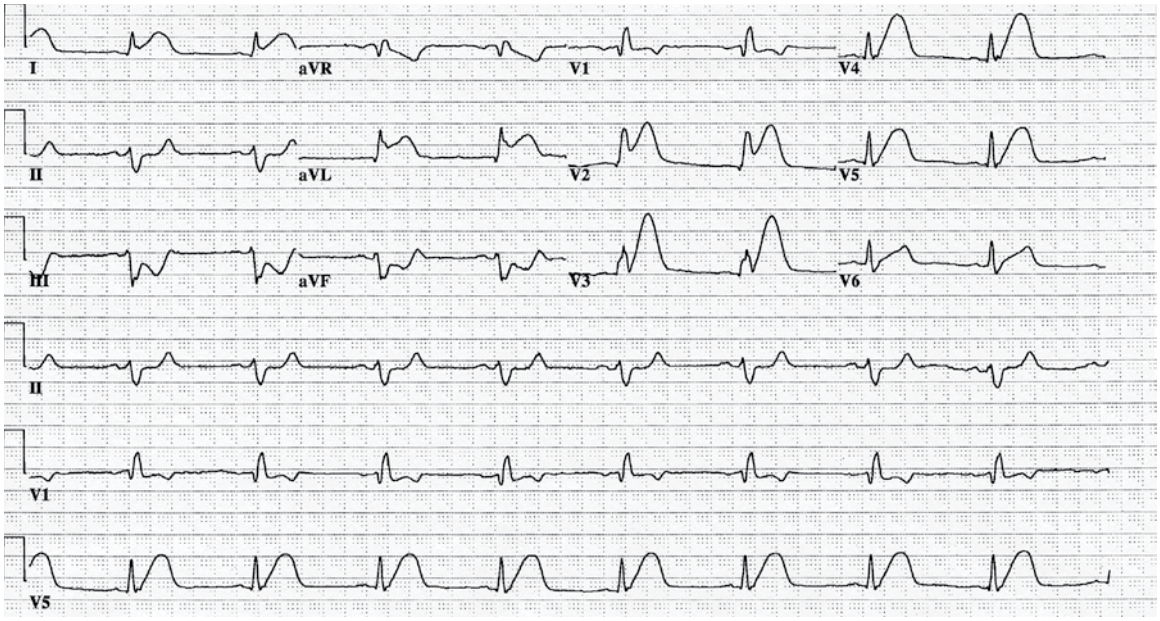
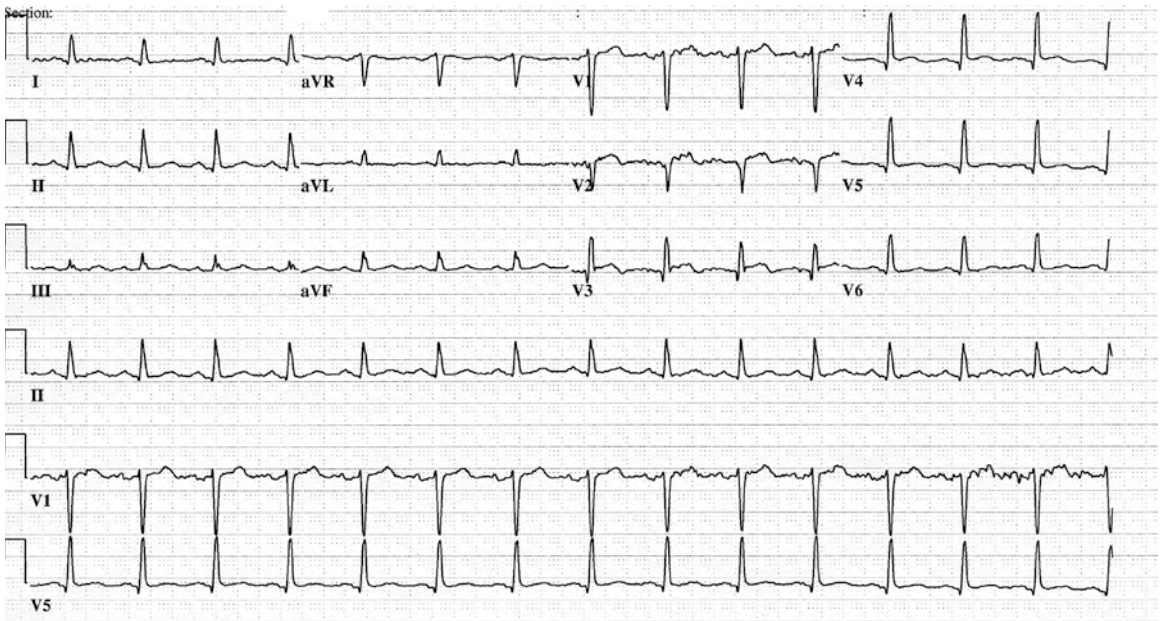


FIGURE 6-4.

ST-segment elevation in the anterior leads, as evidenced by J-point elevation in V_1 through V_3. ST morphology is straight with elevation greater than 1 mm.



FIGURES

FIGURE 6-5.

ST-segment elevation in the anterior and lateral leads, as evidenced by a J-point elevation in V_1 through V_5, I, and aVL. There is loss of R-wave progression and early Q formation in V_1 through V_4.

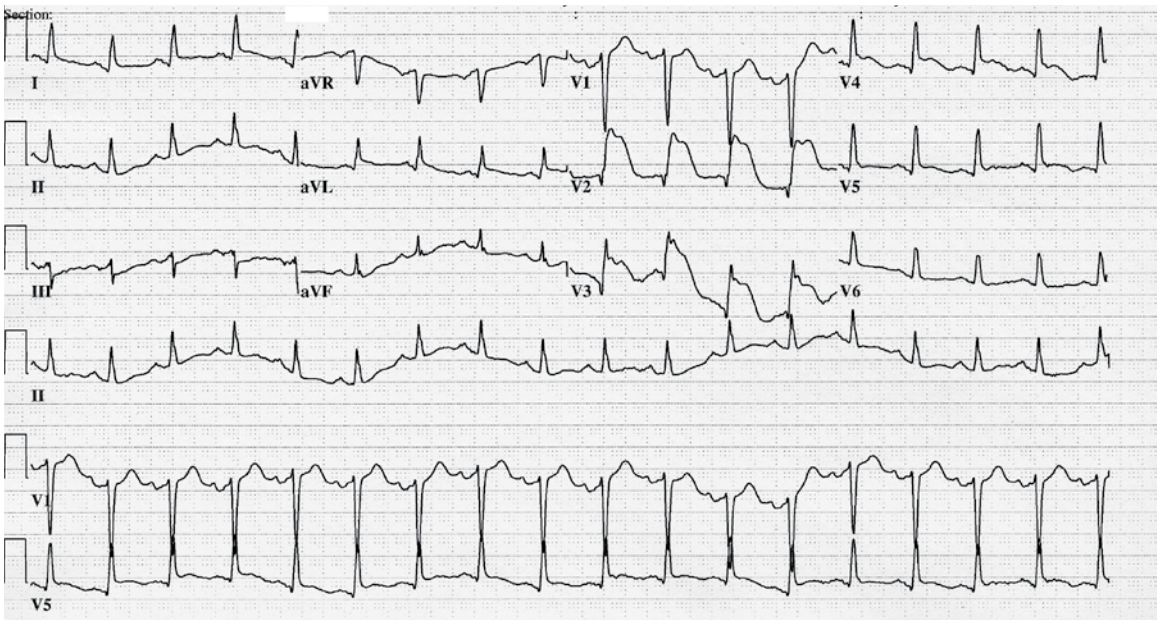
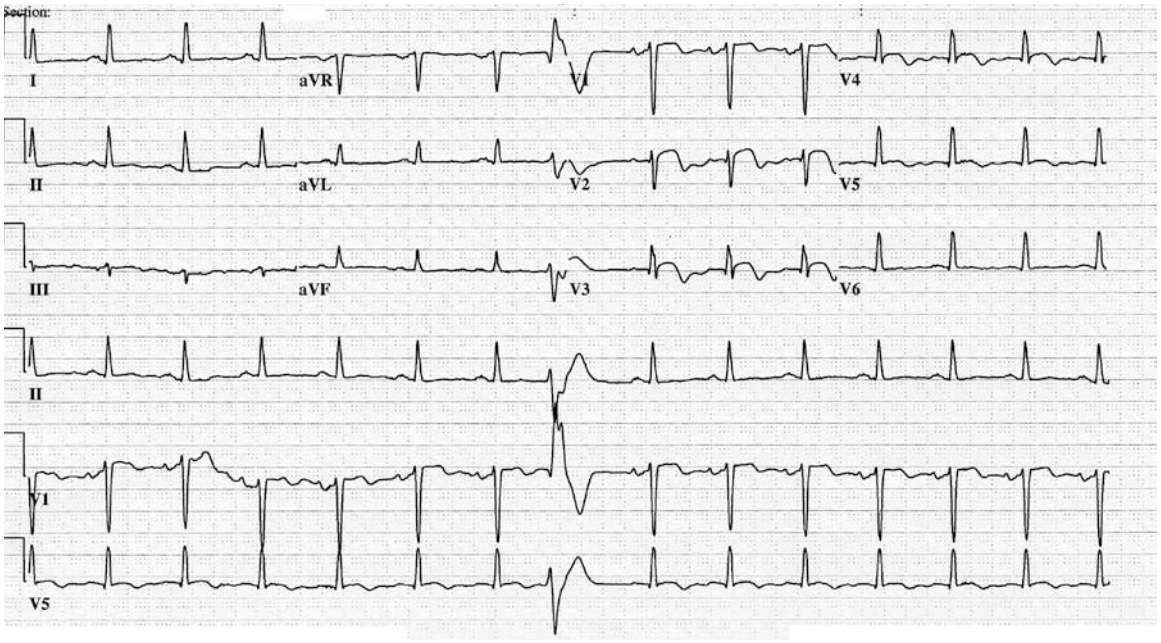


FIGURE 6-6.

After prolonged, nonreperfused coronary occlusion, as regional ST segments resolve toward the isoelectric level, T waves invert up to 3 mm in the same region.



FIGURES

FIGURE 6-7.

Primary ST-segment depression not caused by posterior STEMI or reciprocal changes to ST-segment elevation is a sign of subendocardial ischemia and, in the context of ACS, indicates UA/NSTEMI. ST-segment depression of even 0.5 mm from baseline is associated with increased mortality, but it is particularly significant when it is more than 1 mm (0.1 mV) in two or more contiguous leads. This ECG demonstrates the ST-segment depression best in the inferior leads (I, II and aVF). There is also a lateral component. One millimeter of ST-segment depression following an R of more than 20 mm is very specific for ischemia; R less than 10 mm is sensitive but not specific.

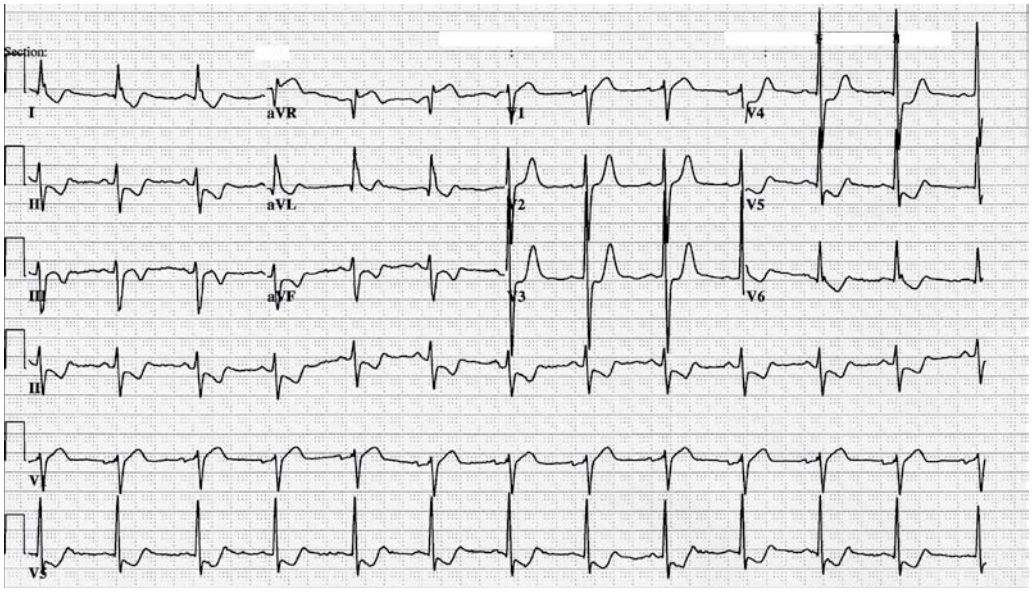
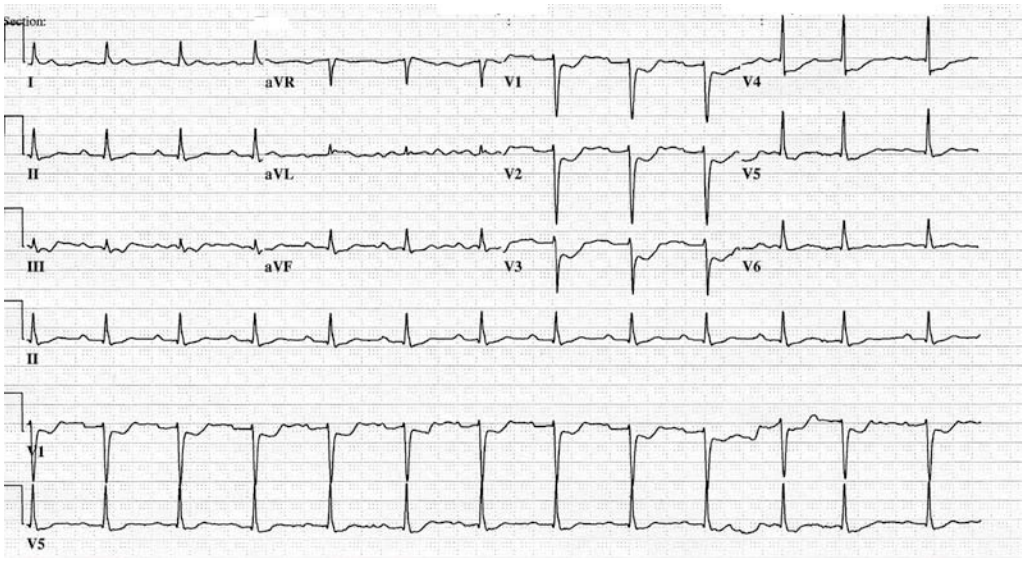


FIGURE 6-8.

The ST-segment depression associated with UA/NSTEMI is transient and dynamic. ST-segment depression morphology is usually flat or downsloping. This ECG demonstrates the ST-segment depression in the anteroseptal leads V_1 through V_4. Posterior leads were normal.



FIGURES

FIGURE 6-9.

Acute phase of an inferior wall STEMI. Note the reciprocal ST-segment depression in leads V_1 through V_3, I, and aVL. Posterior leads (not shown) were normal.

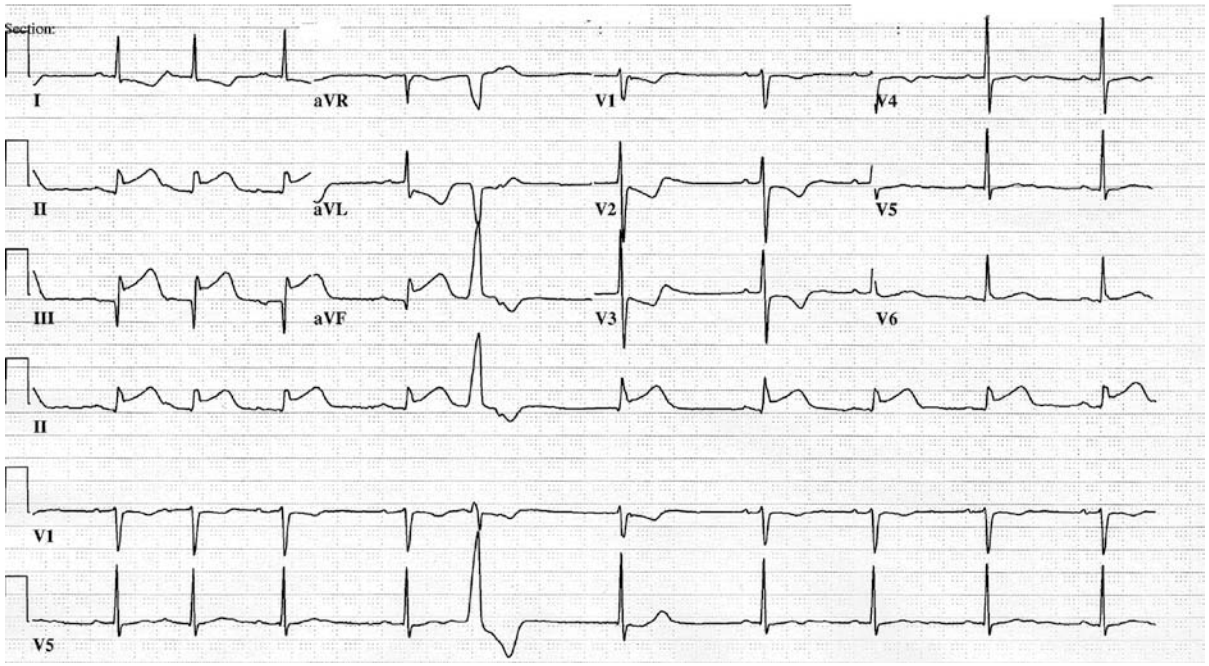
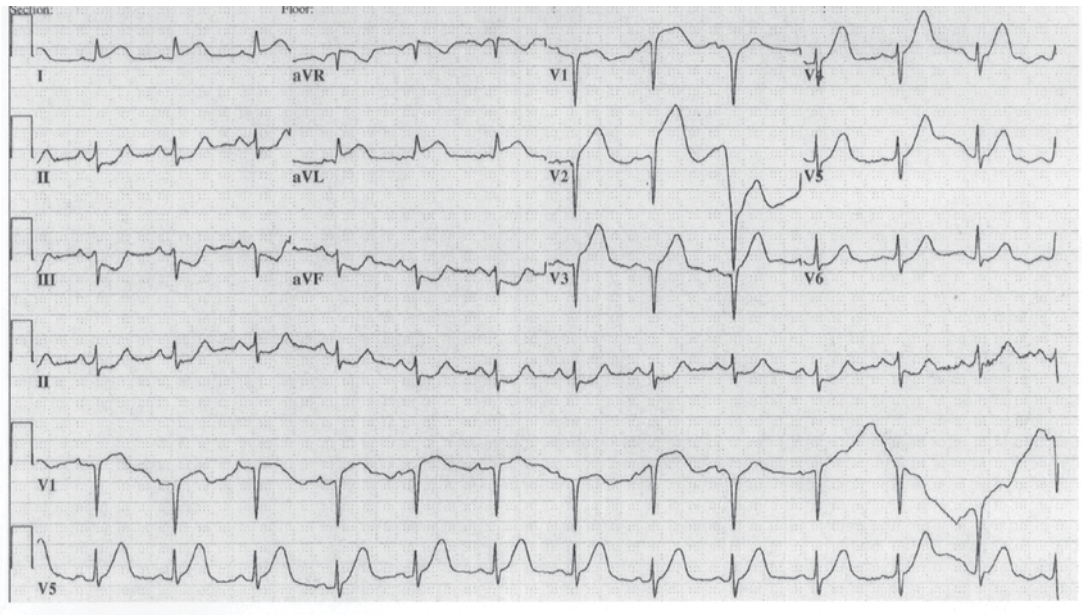


FIGURE 6-10.

Acute phase of an anterolateral STEMI. As a rule, normal septal Q waves are less than 0.04 sec in duration. A Q wave is abnormal if its duration is 0.04 sec or more in lead I, all three inferior leads (II, III, aVF), or leads V_3 through V_6. In this ECG, Q waves are present in V_1 through V_3 of 0.08-sec duration as well as ST-segment elevation. Notice the ST-segment elevation in I and aVL and reciprocal ST/T changes in the inferior leads II, III, and aVF.



FIGURES

FIGURE 6-11.

Wellens syndrome refers to angina with T-wave inversion in the LAD distribution, particularly V_2 through V_4. In this case, the patient was symptom free at arrival. Based on the T-wave changes on V_2 through V_4 and a history of chest pain, he was taken for an emergent angiogram, which revealed 100% occlusion of the middle LAD artery.

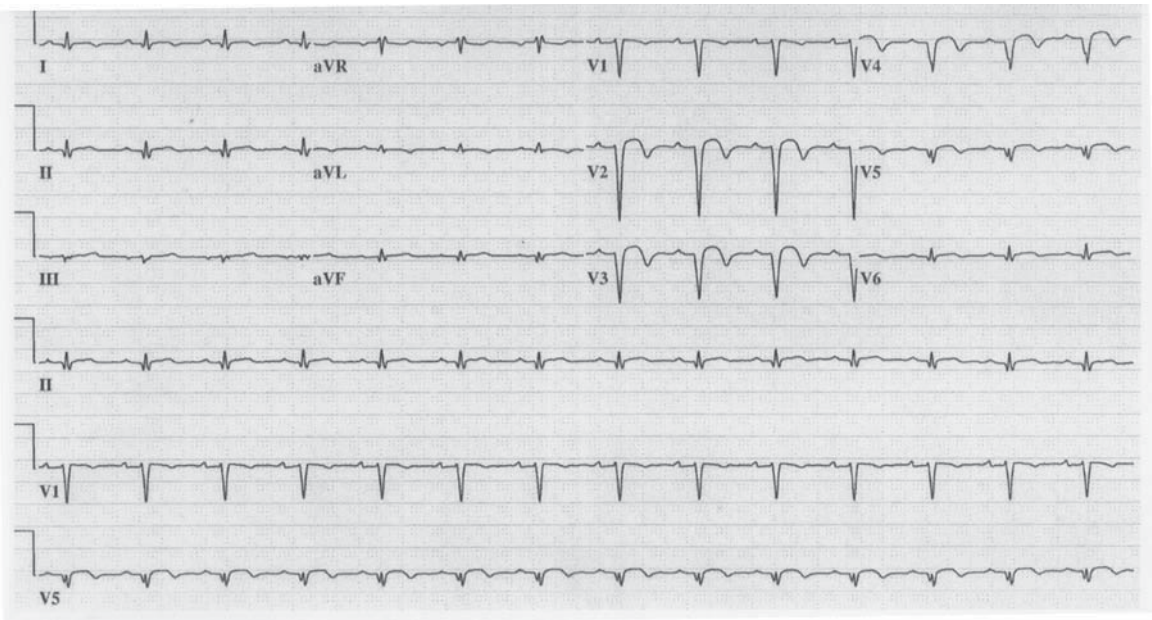
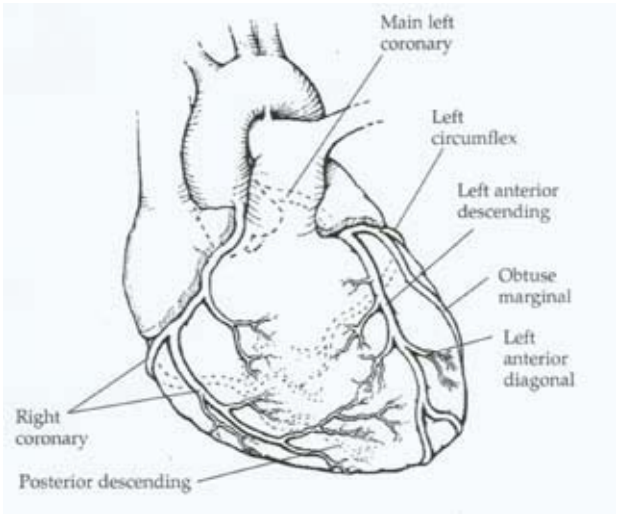


FIGURE 6-12.

Myocardial blood supply. The right coronary artery supplies both the inferior (diaphragmatic) portion of the heart and the right ventricle. The left anterior descending coronary artery generally supplies the ventricular septum and a large part of the left ventricular free wall. The left circumflex coronary artery supplies the lateral wall of the left ventricle. This circulation pattern can be variable. Sometimes, for example, the left circumflex artery also supplies the inferior portion of the left ventricle. Occlusion of the left circumflex or first diagonal artery (a branch of the left circumflex) can present with minimal or no ST-segment elevation despite a large myocardial risk area, because the lateral wall is more electrocardiographically silent.[34-38]



FIGURES

FIGURE 6-13.
Anterolateral STEMI. Note the loss of R-wave progression, replaced with pathologic Q waves in leads V_1 through V_4. Early biphasic T-wave inversion suggests that some spontaneous reperfusion has taken place.

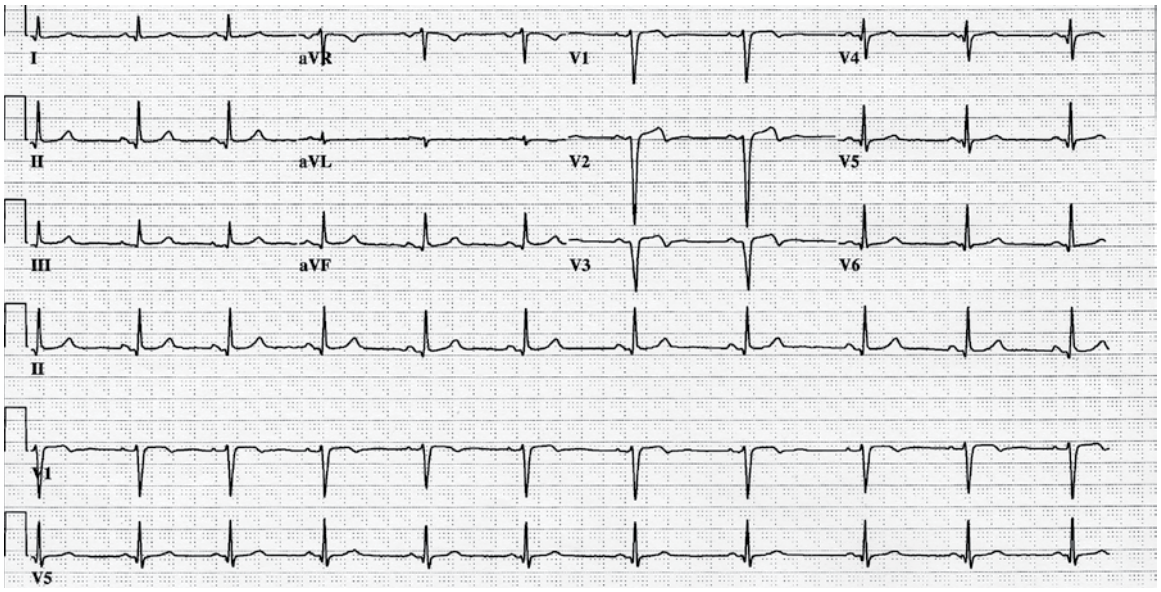
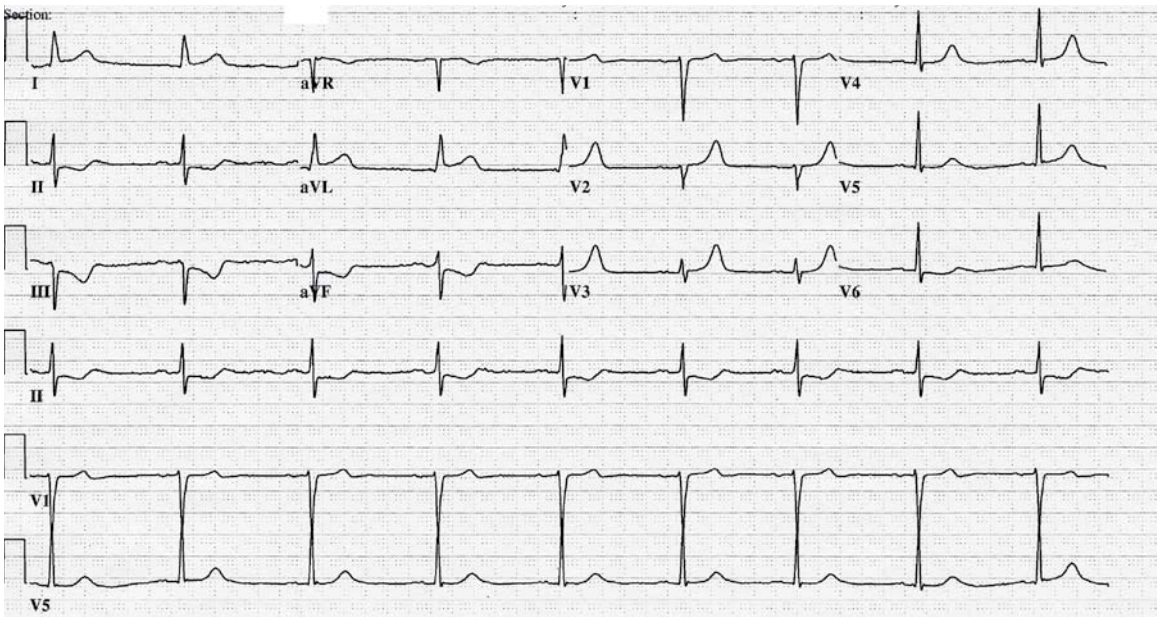


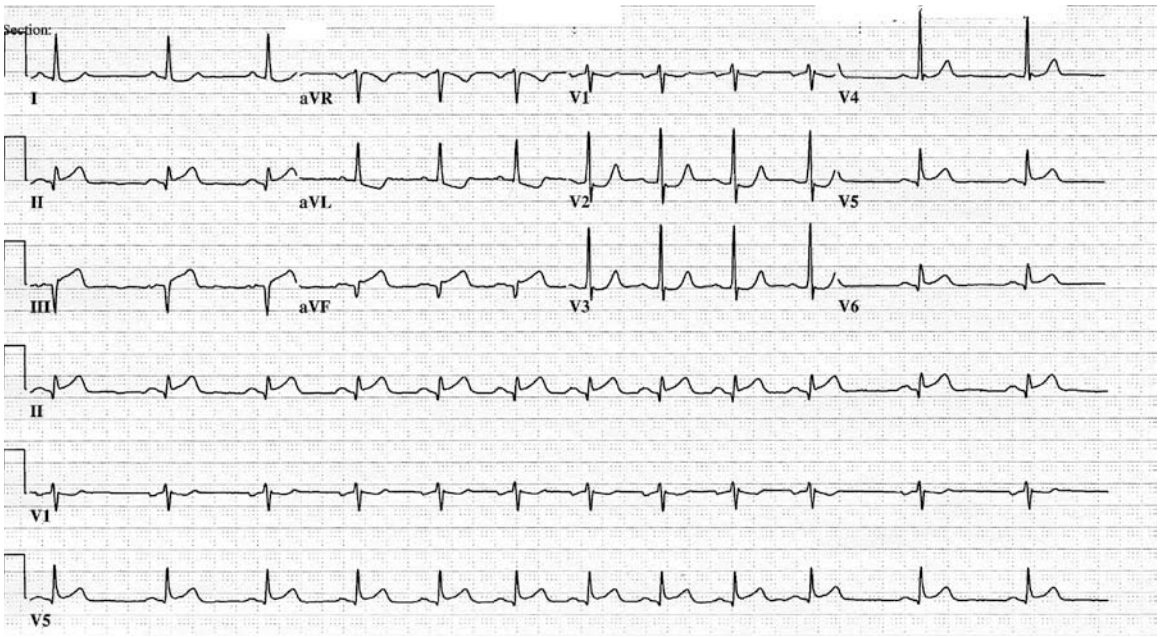
FIGURE 6-14.
Lateral STEMI. Ischemic changes in I and aVL with reciprocal changes in inferior leads II, III, and aVF.



FIGURES

FIGURE 6-15.

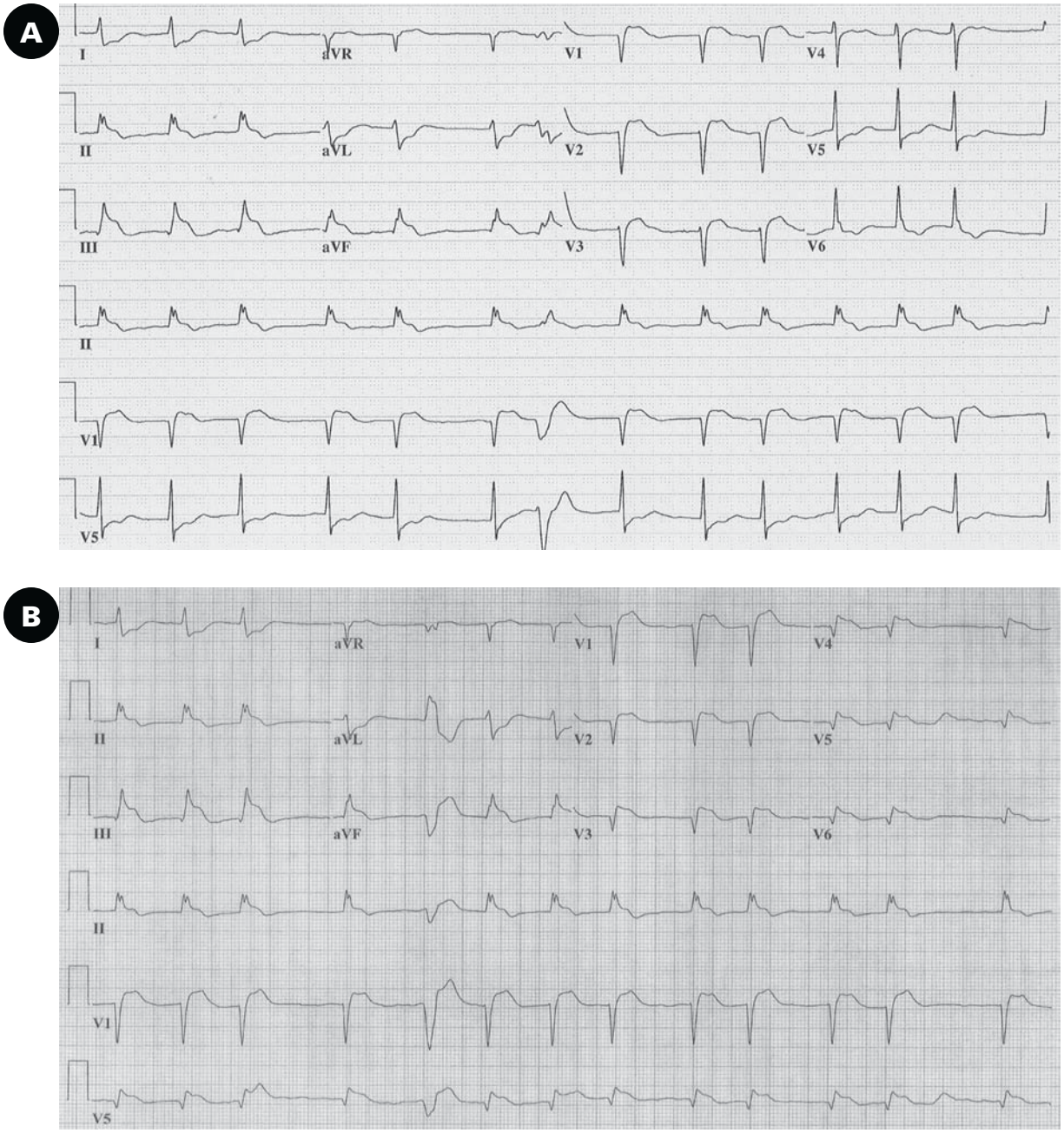
Posterior infarction. The tall R waves and ST-segment depressions are reciprocal to the Q waves and ST-segment elevations that would be recorded at the back of the heart. The positive taller and wider T-wave changes represent the T-wave inversion that would be recorded in the back of the heart. The R:S wave ratio is more than 1 in lead V_2. These findings are representative of a posterior infarction. This patient also had an inferior STEMI (leads II, III, aVF).



FIGURES

FIGURE 6-16.

Right ventricular ischemia with inferior wall infarction. A: ST-segment elevations in leads II, III, and aVF are accompanied by ST-segment elevations in V_1. B: The right-sided ECG confirms ST-segment elevation in the right ventricle (V_4R).



FIGURES

FIGURE 6-17.

Anterior wall aneurysm. This patient presented after 2 hours of chest pain. He had a history of myocardial infarction, but no previous ECG was available. The prominent QS waves in leads V_1 through V_5 and the ST-segment elevations in these leads without significant T-wave abnormalities suggest aneurysm. Reciprocal changes in the inferior leads are absent. Emergent angiogram for presumed STEMI revealed an apical aneurysm with impending rupture and thrombus. No significant LAD lesions were detected. The persistence of ST-segment elevations more than 2 to 3 weeks after infarction suggests a ventricular aneurysm. A followup ECG was not available in this case.

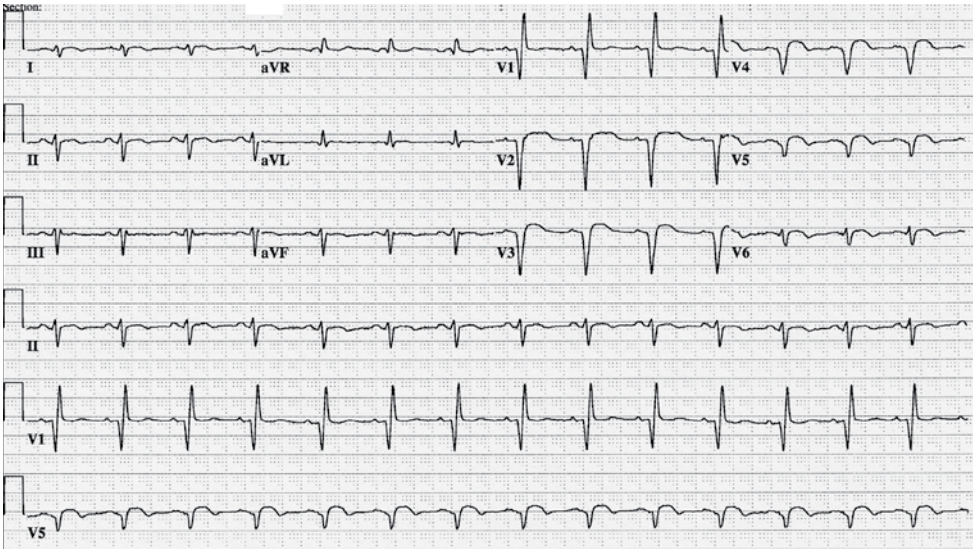
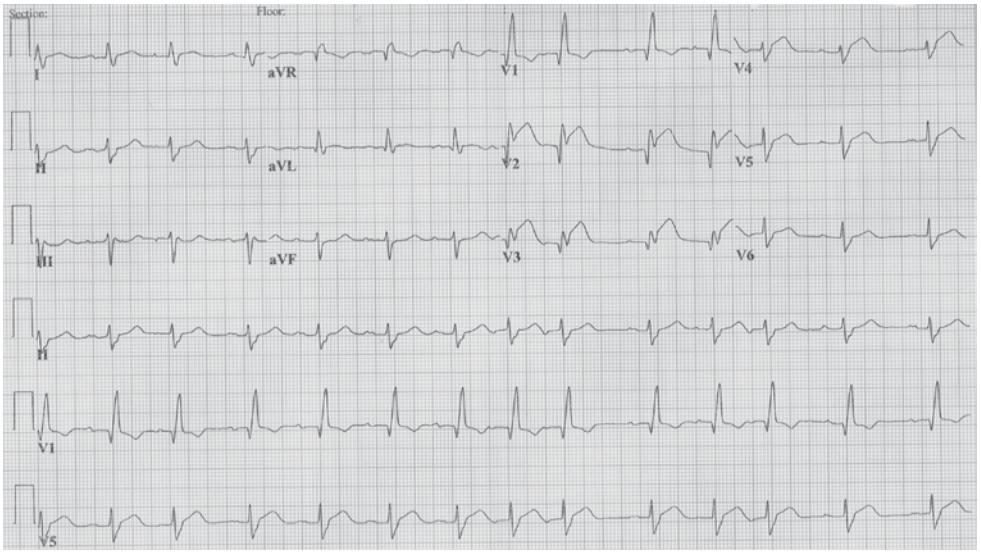


FIGURE 6-18.

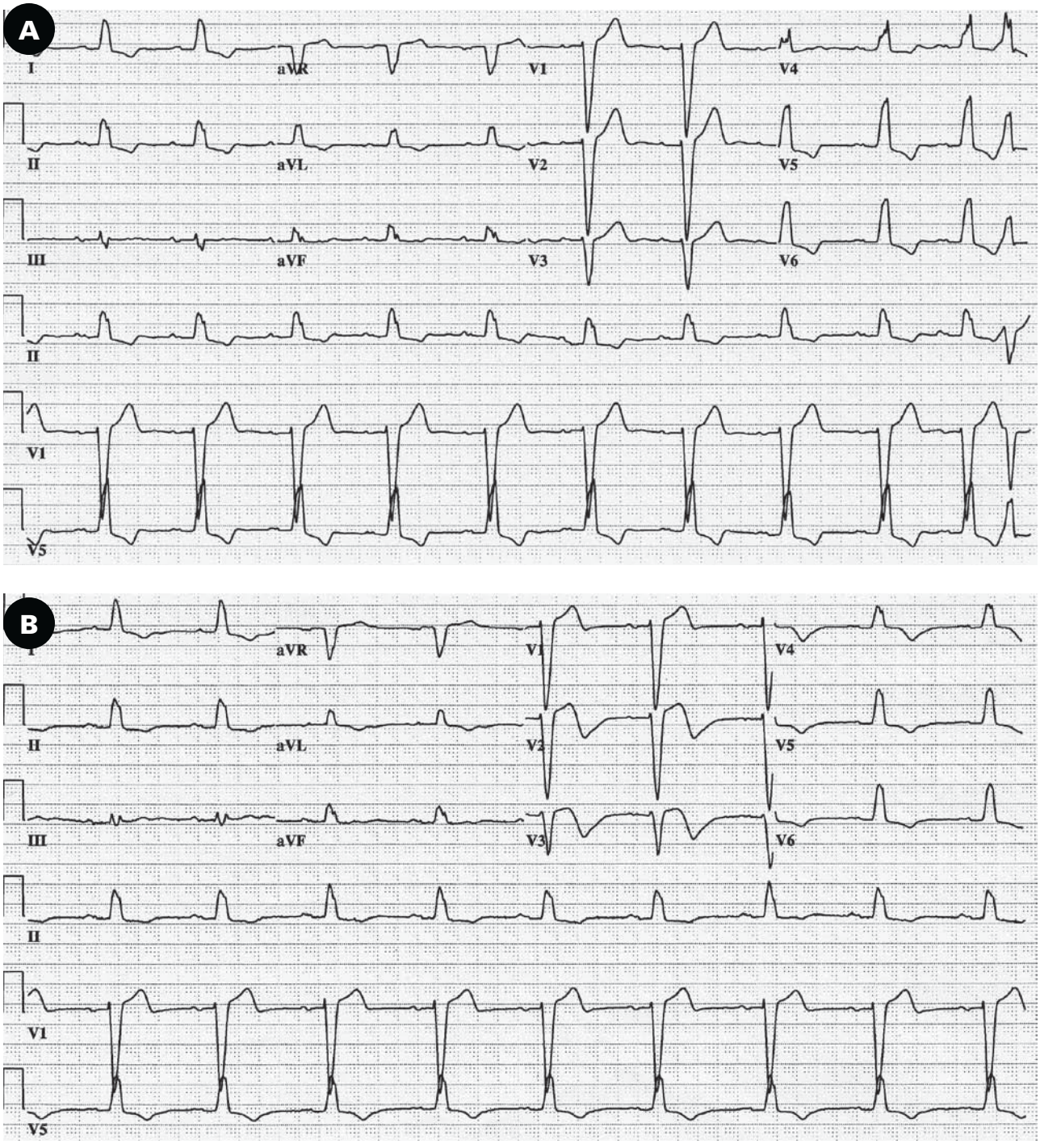
Anterolateral STEMI and the RBBB pattern. RBBB primarily affects the terminal phase of ventricular depolarization, producing a wide R wave in the right chest leads (V_1 through V_2) and a wide S wave in the left chest leads (V_3 through V_6). A pattern of acute anterior lateral wall infarction is indicated by the ST-segment elevations in leads V_1 through V_6.



FIGURES

FIGURE 6-19.

A: Typical LBBB pattern. Note the poor R-wave progression in the right precordial leads and the discordance of QRS and ST/T vectors reflected by ST-segment elevations in the right precordial leads and ST-segment depressions with T-wave inversions in the left precordial leads. B: A subsequent ECG from this patient showed the development of ST-segment elevation of more than 1 mm in lead V_3 (5 points in the Sgarbossa criteria). There are also terminal T-wave inversions in leads V_2 and V_3 caused by anterior ischemia. An angiogram showed 100% occlusion of the proximal LAD coronary artery.



FIGURES

FIGURE 6-20.

AMI in the setting of a paced rhythm. This ECG has ST-segment depression of 1 mm or more in lead V_1, V_2, or V_3 concordant to (in same direction as) the QRS complex and ST-segment elevation of 5 mm or more that was discordant with (in the opposite direction from) the QRS complex. ST-segment elevation or 5 mm or more in predominantly negative QRS complexes is the best marker, with a sensitivity of 53% and specificity of 88%.