Electrocardiographic Diagnosis of ST-elevation Myocardial Infarction

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The ECG is the most useful and feasible diagnostic tool for the initial evaluation, early risk stratification, triage, and guidance of therapy in patients who have chest pain. There is currently a growing trend for 12-lead ECGs to be recorded in the field by paramedics and transmitted by cellular telephone or fax to the target emergency department. It is conceivable that emergency department physicians will be involved in triaging patients in the prehospital phase to hospitals offering primary percutaneous coronary intervention (PCI), which is now recognized to be a superior reperfusion strategy than thrombolytic therapy [1].

Patients who have ST-segment elevation or new left bundle-branch block are usually referred for immediate reperfusion therapy, whereas those who do not have ST-segment elevation are being treated initially with medications [2,3]. Patients are diagnosed as having anterior, inferior-posterior, or lateral myocardial infarction based on the patterns of ST deviation, and assessment of risk is usually based on simple crude measurements of the absolute magnitude of ST-segment deviation or the width of the QRS complexes [4]. Much more information concerning the exact site of the infarct related lesion, prediction of final infarct size, and estimation of prognosis can be obtained from the admission ECG without extra cost or time. Although some clinicians believe that with the increased use of primary PCI in patients who have ST-elevation myocardial infarction (STEMI) this information is no longer needed, there are many instances in which, even with immediate coronary angiography, identification of the infarct-related site and estimation of the myocardial area supplied by each of the branches distal to the coronary artery occlusion is difficult. In some patients, more than one occlusive lesion may be found, and identification of the acutely thrombosed lesion may not always be apparent. In other cases, total occlusion of side branches at bifurcation of coronary arteries may be missed during coronary angiography.

It is important to appreciate that the ECG provides information about a totally different aspect of pathophysiology in STEMI than does the coronary angiogram. Coronary angiography identifies vessel lumen anatomy, whereas the ECG reflects the physiology of the myocardium during acute ischemia. For this reason, it is possible to observe severe coronary stenoses on angiography without ECG evidence of acute ischemia. On the other hand, it is possible to observe restored vessel patency while the ECG continues to show signs of ongoing “ischemia” or “injury pattern” caused by the no-reflow phenomenon, reperfusion injury, or myocardial damage that has already developed before reperfusion occurs. Thus, although coronary angiography remains the reference standard for identifying the infarct-related artery, the ECG remains the reference standard for identifying the presence, location, and extent of acute myocardial ischemia and injury. Moreover, with current imaging techniques, including contrast ventriculography, echocardiography, and radionuclide
perfusion imaging, differentiation of ischemic but still viable myocardium from necrotic myocardium during the acute phase of STEMI is not feasible, but such differentiation may be possible with a correct interpretation of the ECG.

Nevertheless, several conditions other than STEMI may present with ST elevation and need immediate recognition to avoid false treatment [5]. These conditions include left ventricular hypertrophy with secondary repolarization abnormalities, early repolarization pattern, Prinzmetal’s angina, chronic left ventricular aneurysm, acute pericarditis, pulmonary embolism, and the Brugada syndrome. Moreover, studies have pointed out that in a healthy population, more than 90% of men between the ages of 16 and 58 years have ST-segment elevation of 1 to 3 mm in one or more of the precordial leads, mainly in lead V<sub>2</sub> [6]. The prevalence of these changes declined with age, reaching 30% in men over the age of 76 years, whereas in women the prevalence is only 20% and is constant throughout the ages [7].

Some of the patients who have acute chest pain and ST elevation may subsequently have an increase in cardiac markers without any further ECG changes (no ST resolution, no new Q-wave development, and no T-wave inversion). These patients may have non-STEMI with baseline ST elevation, or pseudo STEMI, as shown in Fig. 1. On the other hand, there are patients who have transient ST elevation who have ST resolution without an increase in the cardiac markers. Although some of them may have acute pericarditis, Prinzmetal’s angina, or aborted myocardial infarction [8], many simply have transient early repolarization or pseudo-pseudo STEMI. This last entity has not been well characterized. The clinician encountering a patient who has suggestive symptoms and ST elevation must make rapid therapeutic decisions concerning urgent revascularization without waiting for the results of cardiac markers. It is currently uncertain how many patients who had pseudo or pseudo-pseudo STEMI have been included in randomized trials of STEMI. For example, in a recent analysis of the Hirulog and Early Reperfusion or Occlusion (HERO)-2 trial, 11.3% of the patients who had ST-segment elevation who received reperfusion therapy did not have enzymatically confirmed myocardial infarction [9]. Increasing the threshold for ST elevation (ie, 2 mm in the precordial leads) may decrease the occurrence of false-positive cases but may result in reduced sensitivity and underuse of reperfusion therapy. Comparison with previous ECG recordings or repeating ECG recordings for evolution of subtle changes, along with selective use of echocardiography, may enable the clinician to increase the accuracy of diagnosing true

Fig. 1. ECG of a 57-year-old diabetic and hypertensive man admitted with chest pain of 3 hours’ duration. ECG shows left ventricular hypertrophy and ST elevation in the anterolateral leads with reciprocal changes in the inferior leads. Subsequently his creatine kinase muscle-brain increased to 28.9 pg/mL, and his troponin I was positive. His ECG 3 years before admission showed the same pattern of ST elevation. Repeat ECGs during current admission did not show resolution of ST elevation, T-wave inversion, or development of new Q waves. The diagnosis is pseudo STEMI.
STEMI that may benefit from urgent reperfusion therapy.

This article concentrates on the information that can be obtained from the admission ECG in patients who have STEMI. In particular, it discusses the association of various ECG patterns of the acute phase of STEMI with estimation of infarct size and prognosis and the correlation of various ECG patterns with the underlying coronary anatomy.

Factors that determine prognosis in ST-elevation myocardial infarction

The immediate prognosis in patients who have STEMI is inversely related to the amount of myocardial reserves (total myocardial mass less the myocardium involved in the acute STEMI [ischemic area at risk], scarred territories caused by previous myocardial infarction or fibrosis, and remote ischemic myocardial segments supplied by critically narrowed coronary arteries). Among patients who do not have prior myocardial infarction or major pre-existing stenotic lesions in the coronary arteries, prognosis is directly related to the size of the ischemic myocardium supplied by the culprit coronary artery distal to the occlusion. In patients who have low myocardial reserves because of previous myocardial infarctions or diffuse fibrosis, however, even a relatively small infarction may be detrimental. Moreover, in patients who have diffuse severe coronary artery disease, a small myocardial infarction may interfere with the delicate balance and induce remote ischemia by obliterating collateral flow or creating a need for (compensatory) augmentation of contractility in the remote noninfarcted segments supplied by stenosed coronary arteries. Therefore, in addition to accurate diagnosis, there is a need for early estimation of the size of the ischemic myocardium at risk and myocardial reserves.

The ECG may help in assessing the size of the ischemic myocardial area at risk, may help in differentiation between subendocardial (nontransmural) and transmural ischemia, and may assist in identifying the presence of previous infarctions (abnormal Q waves in leads not involved in the present infarction; for example, abnormal Q waves in the precordial leads in a patient who has inferior ST elevation) [10]. Furthermore, some ECG patterns may indicate the presence of diffuse coronary artery disease and remote ischemia [11–13].

On admission, part of the myocardial area at risk (supplied by the culprit coronary artery) might have already undergone irreversible damage. The proportion of the ischemic area at risk that has undergone irreversible necrosis depends on the total ischemic time and on the rate of progression of the wave front of necrosis. The rate of progression of necrosis is highly variable and is dependent on the presence of residual perfusion through collateral circulation [14] or incomplete or intermittent occlusion of the infarct related artery [15], as well as on metabolic factors including ischemic preconditioning [16].

ECG changes during the acute phase of ST-elevation myocardial infarction

Shortly after occlusion of a coronary artery, serial ECG changes are detected by leads facing the ischemic zone, as shown in Fig. 2. First, the T waves become tall, symmetrical, and peaked (grade 1 ischemia); second, there is ST elevation (grade 2 ischemia) without distortion of the terminal portion of the QRS; and third, changes in the terminal portion of the QRS complex may appear (grade 3 ischemia) [17–19]. The changes in the terminal portion of the QRS are explained by prolongation of the electrical conduction in the Purkinje fibers in the ischemic region. The delayed conduction decreases the degree of cancellation,
resulting in an increase in R-wave amplitude in leads with terminal R wave and a decrease in the S-wave amplitude in leads with terminal S wave on the surface ECG [20,21]. The Purkinje fibers are less sensitive to ischemia than the contracting myocytes [22]. Hence, for an alteration in the terminal portion of the QRS to occur, there probably should be a severe and prolonged ischemia that would affect the Purkinje fibers [23]. In patients who have collateral circulation, no changes are detected in the QRS complex during balloon angioplasty [20]. Thus, absence of distortion of the terminal portion of the QRS, despite prolonged ischemia, may be a sign for myocardial protection (probably by persistent myocardial flow caused by subtotal occlusion or collateral circulation or by myocardial preconditioning). The disappearance of the S waves in leads with terminal S (RS configuration), mainly leads V1 to V3, can be recognized easily (Fig. 3). In contrast, the absolute R-wave height is influenced by many other variables. Therefore, the absolute R-wave amplitude is not helpful in determining the severity of ischemia. Changes in the R-wave amplitude can be detected reliably only by continuous ECG monitoring; comparison of the admission ECG with previous ECG recordings often is difficult because of differences in ECG instruments and different placement of the precordial electrodes. Therefore, a second empiric criterion for leads with terminal R configuration was developed. This criterion relates the J point of the ST to the R-wave amplitude in leads with terminal R waves (qR configuration). As shown in Fig. 4, a ratio of 0.5 or greater indicates grade 3 ischemia [17,19]. Although the transition between the grades of ischemia is gradual and continuous, for practical clinical purposes it is convenient to define grade 2 ischemia as ST elevation greater than 0.1 mV without distortion of the terminal portion of the QRS and grade 3 as ST elevation with distortion of the terminal portion of the QRS (emergence

Fig. 3. (A) Grade 2 ischemia in a patient who has acute anterior STEMI. There are deep S waves in leads V1 to V3. (B) Grade 3 ischemia in a patient who has acute anterior STEMI. There is loss of S waves in leads V1 to V3, and the J point/R wave ratio is greater than 0.5 in leads V4 and V5.
of the J point > 50% of the R wave in leads with qR configuration, or disappearance of the S wave in leads with an Rs configuration (see Figs. 3, 4) [17,24–28]. Only later, the T waves become negative, the amplitude of the R waves decreases, and Q waves may appear. Only a minority of patients who have STEMI presents with grade 3 ischemia upon admission. Although the underlying mechanism for this difference is still unclear, grade 3 ischemia has large implications regarding prognosis, as discussed later.

Diagnosis of acute ST-elevation myocardial infarction

In a patient who has typical symptoms, the presence of ST-segment elevation, especially when accompanied with reciprocal changes, is highly predictive of evolving STEMI. Several investigators, however, reported that the sensitivity of the ECG for acute myocardial infarction may be as low as 50% [29–31]. In most of these studies only one admission ECG was analyzed. Hedges and colleagues [32] used the admission and a second ECG performed 3 to 4 hours after admission and found serial ECG changes in 15% of the patients. Continuous ECG monitoring or multiple ECG recordings over time or during fluctuations in the intensity of symptoms were not performed, however. Repeated ECG recordings may improve the ability to detect subtle ischemic changes. Furthermore, as determined by independent reviewers, 49% of the missed acute myocardial infarctions could have been diagnosed through improved ECG-reading skills or by comparing the current ECG with a previous one [30]. It should be remembered that acute myocardial infarction detected by elevated creatine kinase muscle-brain (MB) or troponin levels without ST elevation is not an indication for urgent
reperfusion therapy. The only exception is new left bundle-branch block in a patient who has acute chest pain. Menown and colleagues [33] studied the sensitivity and specificity of the admission ECG for diagnosing acute myocardial infarction by studying patients who had \( n = 1041 \) or did not have \( n = 149 \) chest pain. Acute myocardial infarction was defined by the presence of chest pain of 20 minutes’ duration or longer, elevation of creatine kinase two times or more the upper laboratory normal reference level (creatine kinase-MB activity ≥ 7% if the etiology of the total creatine kinase was equivocal), or elevation of creatine kinase less than two times the upper laboratory normal reference level accompanied by serial ECG changes consistent with new myocardial infarction (new Q waves ≥ 0.03 seconds’ duration or new persistent T-wave inversion in two or more contiguous leads). The best ECG variables for the diagnosis of acute myocardial infarction were ST elevation greater than 0.1 mV in more than one lateral or inferior lead or ST elevation greater than 0.2 mV in more than one anteroseptal precordial lead. These criteria correctly classified 83% of subjects, with a sensitivity of 56% and a specificity of 94%. Changing the degree of ST elevation significantly modified both the sensitivity (45%–69%) and the specificity (81%–98%). The addition of multiple QRST variables (Q waves, ST depression, T-wave inversion, bundle-branch block, axes deviations, and left ventricular hypertrophy) increased specificity but only marginally improved overall classification [33].

**Estimation of the size of ischemic myocardium at risk**

There is no currently defined reference standard to measure the ischemic area at risk in the acute setting. The extent of regional wall motion abnormalities can be appreciated easily soon after admission by two-dimensional echocardiography or left ventriculography. With both methods, however, a differentiation between old scars and the acutely ischemic but viable zones is not always possible. Because of the effect of stunning, regional wall motion may persist for long periods of time after reperfusion has occurred [34]. Moreover, differentiation of transmural from subendocardial ischemia/infarction is not always possible, because akinesis may occur when only the inner myocardial layers are ischemic [35].

Several studies have tried to estimate the ischemic area at risk or final infarct size by the admission ECG. In these studies, either the number of leads with ST deviation (elevation or depression) [36–39] or the absolute amplitude of ST deviation [4,36,39–41] was used. The results were conflicting, however. Arnold and Simoons [4] have evaluated the “expected infarct size without thrombolysis” by multivariate regression analysis in 885 patients in the rt-PA/placebo and rt-PA/PTCA trial conducted by the European Cooperative Study Group and validated the findings in 533 patients from the Intracoronary Streptokinase trial of the Interuniversity Cardiology Institute of The Netherlands and in 1741 patients from the Intravenous Streptokinase in Acute Myocardial Infarction study; both trials contained a nonthrombolized control group. They defined an infarct size score that included the absolute sum of the amplitude of ST deviation, the QRS width exceeding 0.12 seconds, anterior STEMI location, Killip class 3 or 4 on admission, and delay from symptom onset to treatment allocation and found that the expected infarct size correlated well with the actual enzymatic infarct size in the nonthrombolized patients of the latter two studies. Limitation of infarct size by thrombolytic therapy was greatest in patients who had a large expected infarct size and was absent in patients who had a small expected infarct size. Similarly, 1-year mortality reduction was greatest in patients who had a large expected infarct size without thrombolysis.

Aldrich and colleagues [36] studied 144 patients who had STEMI who did not receive thrombolytic therapy. The best correlation between the final ECG Selvester QRS scoring system (an estimation of infarct size) and the admission ECG was found using the magnitude of ST elevation in leads II, III, and aVF in inferior STEMI and the number of leads with ST elevation in anterior STEMI. Another study in patients who received reperfusion therapy showed only a weak correlation between the Aldrich score and either the ischemic area at risk or final infarct size, as measured by pretreatment and predischarge techneutium-99m (Tc-99m) sestamibi scans, respectively [38]. The Aldrich formula was related more to the collateral score than to the ischemic area at risk or final infarct size [38]. Clemmensen and colleagues [37] reported a good correlation between the final Selvester score and the number of leads with ST elevation \( r = 0.70 \) in anterior STEMI. Neither the magnitude of ST segment elevation in all leads nor the number of leads with ST elevation correlated with the final Selvester
score in inferior STEMI. Clements and colleagues [39] also reported only a weak correlation between myocardial area at risk (as assessed by Tc-99m sestamibi scan) and the number of leads with ST deviation, total ST deviation, total ST elevation, or total ST depression. The myocardial area at risk correlated modestly \((r = 0.58)\) with total ST deviation in anterior STEMI and with total ST depression normalized to the R wave \((r = 0.70)\) in inferior STEMI. Because of large standard errors (9%-15% of the left ventricle), however, these formulas for estimation of the myocardial area at risk cannot be used in the clinical setting for estimation of infarct size [39]. Birnbaum and colleagues [42] showed that among patients with first anterior STEMI, the correlation between either the number of leads with ST elevation or the sum of ST elevation and the extent and severity of regional left ventricular dysfunction (both at 90 minutes after initiation of thrombolytic therapy and at predischarge) was poor.

These ECG studies were based on the assumption that each lead represents the same amount of myocardium and that a similar size of ischemic area in different locations of the left ventricle will result in similar magnitude of ST deviation in the same number of leads. The 12-lead ECG does not represent all myocardial regions equally, however. The inferior and anterior walls of the left ventricle are well represented, but the lateral, posterior, septal, and apical regions are relatively ECG silent [43,44]. Moreover, ischemia in opposed regions may attenuate or augment ST deviation. For example, in patients who have ischemia of the high anterolateral and inferior regions caused by proximal occlusion of a dominant left circumflex coronary artery (LCX), attenuation of ST deviation in leads I, aVL, and the inferior leads may occur, whereas subendocardial high anterolateral ischemia may augment ST-segment elevation in the inferior leads. Posterior myocardial infarction is commonly associated with ST depression in the precordial leads V1 to V3 [45], whereas right ventricular infarction may cause ST elevation in leads V1 and V2 [46]. In concomitant right ventricle and posterior myocardial infarction, the opposing forces may neutralize each other, and therefore, no ST deviation may occur in these leads. Because different leads represent different areas of the myocardium, a different coefficient probably should be used for each lead and even for each type of infarction. To overcome the unequal representation of the myocardium by the different leads, another technique has been suggested [47,48]. In this technique the maximal points of the Selvester QRS score are given to each lead with ST elevation greater than 0.1 mV. The sum of these initial scores is considered to represent the percentage of the left ventricle that is ischemic. This method was compared with thallium-201 perfusion scans in 28 patients (10 patients on admission and 18 patients on day 5 after reperfusion therapy) [47]. A good correlation was found between this potential Selvester score and the extent of thallium-201 perfusion defect \((r = 0.79; P < .005)\). Birnbaum and colleagues [42] found only a weak correlation between the maximal potential Selvester score and the extent or severity of left ventricular dysfunction among patients who had first anterior STEMI and underwent left ventriculography at 90 minutes after initiation of thrombolytic therapy and at predischarge. On the other hand, in patients receiving streptokinase for STEMI, Wong and colleagues [49] found that the initial Selvester QRS score and T-wave inversion grade were the only predictors of myocardial salvage \((P < .001)\), with no difference between anterior and nonanterior STEMI [49].

Another qualitative approach for predicting final infarct size by the admission ECG based on the grades of ischemia has been reported by Birnbaum and colleagues [26,27,42,50]. In the Thrombolysis in Myocardial Infarction–4 (TIMI-4) trial, patients presenting with grade 3 ischemia \((n = 85)\) on admission had a larger infarct as assessed by creatine kinase release over 24 hours \((P = .023)\), and a larger predischarge Tc-99m sestamibi defect size \((P = .001)\) [26]. In a comparison of patients with first anterior STEMI who were assigned randomly to thrombolytic therapy or conservative treatment, the final QRS Selvester score was lowered by thrombolytic therapy only in patients who had grade 2 ischemia on enrollment, but not grade 3 [27]. Overall, final QRS Selvester score was higher for patients who had grade 3 than grade 2 ischemia on enrollment, both in those who received and in those who did not receive thrombolytic therapy. Among patients with a first anterior STEMI who participated in the Global Use of Streptokinase and t-PA for Occluded Coronary Arteries (GUSTO)-I angiographic substudy and underwent angiography both at 90 minutes after initiation of thrombolytic therapy and at predischarge, patients who had grade 2 ischemia on enrollment had higher left ventricular ejection fraction at 90 minutes than patients who had grade 3 ischemia [42]. The difference in global left ventricular function was related...
mainly to the severity of regional dysfunction in the involved segments and less to the extent of involvement (size of the area at risk) [42]. On predischarge evaluation, the grade 3 group tended to have lower left ventricular ejection fraction and had significantly more chords with dysfunction and more severe regional dysfunction than the grade 2 group. The number of dysfunctional chords tended to decrease from 90 minutes to predischarge in the grade 2 group, whereas it tended to increase in the grade 3 group. This finding may reflect partial recovery from stunning at the predischarge ventriculography in the grade 2 group [42]. There was no difference in the time to therapy or the success of thrombolysis between the grade 2 and 3 groups. Thus, it seems that the difference in infarct size between the grade 2 and grade 3 groups is explained by more severe ischemia and not by larger ischemic area at risk, longer ischemia, or lower rates of successful reperfusion [42]. Findings from the Acute Myocardial Infarction Study of Adenosine trial confirmed this hypothesis [50]. In this study, patients who received thrombolytic therapy were assigned randomly to pretreatment with intravenous adenosine or placebo. For placebo-treated patients, the median pretreatment Tc-99m sestamibi single-photon-emission CT (SPECT) perfusion defect (ischemic area at risk) did not differ significantly between grade 2 and grade 3 patients; however, the median infarction index (infarct size/area at risk) was smaller in patients who had grade 2 ischemia (66% versus 90%; P = .006). Overall, infarct size was related to baseline ischemia grade (P = .0121) and was reduced by adenosine treatment (P = .045). In a recently published study, Billgren and colleagues [51] have assessed the relation of baseline ECG ischemia grades to area at risk and myocardial salvage [100(area at risk – infarct size)/area at risk] in 79 patients who underwent primary PCI for first STEMI and had technetium Tc-99m sestamibi SPECT before PCI and predischarge final infarct size. Patients were classified as having grade 2 ischemia (ST elevation without terminal QRS distortion in any of the leads; n = 48), grade 2.5 ischemia (ST elevation with terminal QRS distortion in 1 lead, n = 16), or grade 3 ischemia (ST elevation with terminal QRS distortion in ≥ two adjacent leads; n = 15). Time to treatment and area at risk were comparable among groups. Myocardial salvage, as a percentage of the area at risk, tended to be smaller for the grade 3 ischemia group than in the other two groups (65%, 65%, and 45%, for grade 2, 2.5, and 3, respectively; P = .16). Salvage was dependent on time only in the grade 3 group. The authors conclude that patients who have grade 3 ischemia have rapid progression of necrosis over time and less myocardial salvage, and that grade 3 ischemia is a predictor of smaller myocardial salvage by primary PCI.

Most, but not all, studies have shown that, as a group, patients who have inferior STEMI and ST depression in leads V1–V3 have larger infarcts than their counterparts who do not have ST depression, as evidenced by higher peak creatine kinase release; more extensive wall motion abnormalities; larger defect size by thallium-201, Tc-99m, and positron emission tomography; and higher QRS scores of infarct size [13,52–57]. In addition, ST elevation in lead V6 in patients who have inferior STEMI also is associated with larger myocardial infarction [58]. Others have reported that ST elevation in the posterior leads (V7–V9) in patients who have inferior STEMI is associated with larger infarct size [59]. It is unclear whether there is additive value for ST elevation in leads V7 to V9 over V6 alone.

Many variables, such as the width of the chest wall, the distance of the electrode from the ischemic zone, the myocardial mass, and the presence of ischemic preconditioning and collateral circulation, have a major influence on the absolute magnitude of ST deviation. Therefore, currently, there is no accurate method for estimating the area at risk by the admission ECG that can be used in the individual patient, although, in general, patients who have ST deviation (elevation plus depression) in large number of leads or high absolute sum of ST deviation have a larger myocardial infarction than patients who have ST deviation in a small number of leads or low sum of ST deviation [4,40]. There are patients who have a relatively large infarction who have only minor absolute ST deviation. Infarct size underestimation in these patients may lead to underuse of reperfusion therapy. The grades of ischemia predict final infarct size but not the size of the ischemic area at risk.

**Differentiation between viable and necrotic myocardium at the ischemic area at risk**

Although with echocardiography old myocardial scars with thinning of the ventricular wall and dense echo reflections can be identified, none of the direct imaging modalities (contrast ventriculography and echocardiography) can differentiate
between ischemic but viable myocardium and myocardium that has already undergone irreversible necrosis in the acute stage of infarction. Q waves were traditionally considered as a sign of myocardial necrosis [60]. The mechanism and significance of Q waves that appear very early in the course of STEMI in leads with ST elevation are probably different, however [60–63]. Fifty-three percent of the patients who had STEMI admitted within 1 hour of onset of symptoms had abnormal Q waves on presentation, even before reperfusion therapy had been initiated [61]. It has been suggested that Q waves that appear within 6 hours from onset of symptoms do not signify irreversible damage and do not preclude myocardial salvage by thrombolytic therapy [64]. Furthermore, Q waves that appear early in the course of acute ischemia may be transient and disappear later [64,65]. Several authors have found early Q waves to be associated with larger ischemic zone and ultimate infarct size [61,66]. Such Q waves have been explained by a transient loss of electrophysiologic function caused by intense ischemia [60,64]. In contrast, some investigators found that Q waves develop rapidly only after reperfusion [62,63,67]. It has been suggested that the presence of Q waves may be masked by the injury current during ischemia [63], and they frequently can be seen only after resolution of the injury current. These changes, however, may reflect reperfusion injury, interstitial edema, or hemorrhage that later may resolve partially [67]. Ninety minutes after thrombolytic therapy, however, TIMI flow grade 3 is achieved less often in patients with than without abnormal Q waves on presentation [68]. Further studies are needed to find other ECG markers that will assist in differentiation between acutely ischemic but viable from irreversible necrotic myocardium. Regardless of the underlying mechanism, the presence of abnormal Q waves in the leads with ST elevation on the admission ECG is associated with larger final infarct size and increased in-hospital mortality [69].

Patients who have grade 3 ischemia on enrollment have larger final infarct size [26,50] but not larger initial ischemic area at risk [42,50]. In addition, patients who have grade 3 ischemia are less likely to benefit from thrombolytic therapy than patients who have grade 2 ischemia [27]. Distortion of the terminal portion of the QRS complex in leads with ST elevation is not a sign that irreversible damage had already occurred upon presentation, however, because the same ECG pattern frequently is detected in patients who have Prinzmetal’s angina during ischemic episodes that are not associated with significant myocardial damage.

Some patients who have STEMI present with negative T waves (Fig. 5). Early inversion of the T waves, along with resolution of ST elevation, is a sign of reperfusion [70]; however, the significance of negative T waves in leads with ST elevation before reperfusion therapy is initiated is currently unclear. Wong and colleagues [68] reported that 90 minutes after thrombolytic therapy, TIMI flow grade 3 was seen less often in patients who presented with ST elevation and negative T waves than in those who had positive T waves. In a retrospective analysis of the HERO-1 study [49], the same authors found on multivariate analysis that the T-wave inversion grade (graded according to the depth and location of T-wave inversion) on admission in patients with first STEMI was the strongest predictor of less
myocardial salvage ($r = 0.57; P < .001$). Herz and colleagues [71] reported that among patients treated 2 to 6 hours after onset of symptoms, those who presented with inverted T waves in leads with ST elevation had higher in-hospital mortality than patients with positive T waves. In contrast, among patients treated within the first 2 hours of onset of symptoms, patients who had negative T waves had no hospital mortality (0/52 patients), as compared with a 5.0% mortality rate in patients who had positive T waves (36/726 patients; $P = .19$) [71]. Therefore, ST elevation with negative T waves, especially if it occurs in patients presenting more than 2 hours of onset of symptoms, may be used as an ECG sign of a more advanced stage of infarction or presence of irreversible damage, associated with lesser chance of achieving successful reperfusion, and subsequently leading to higher mortality.

**Expected rate of progression of myocardial infarction**

It currently is impossible to assess the severity of myocardial ischemia or the expected rate of progression of myocardial necrosis by direct myocardial imaging. The magnitude of ST elevation reflects mainly the severity of the subepicardial ischemia. The standard surface 12-lead ECG is less sensitive to subendocardial ischemia. Subendocardial ischemia may cause either ST depression or no change in the ST segment. ST depression, however, also may result from reciprocal changes in leads oriented away from the ischemic zone [72]. Augmentation of collateral flow ameliorates the magnitude of ST deviation during coronary balloon occlusion [73]. Moreover, ischemic preconditioning by preceding brief ischemic episodes attenuates the magnitude of ST deviation [73,74]. Data regarding the effects of myocardial preconditioning or the presence of sufficient collateral circulation on the 12-lead ECG during acute myocardial infarction are sparse, however. Collateral circulation reduces the severity of the subepicardial ischemia and hence attenuates ST elevation [38]. Indeed, Sagie and colleagues [75] showed that in patients who had acute anterior STEMI and who had good collateral circulation, only T-wave changes, without ST elevation (grade 1 ischemia) were observed. Other cardiac and noncardiac variables, such as presence of myocardial hypertrophy, the distance of the heart from the chest wall, or the width of the chest wall, may also affect the magnitude of ST deviation. Therefore, the absolute magnitude of ST deviation can give only a rough estimation of the magnitude of myocardial protection or the severity of ischemia.

**Identifying ischemia at a distance**

Patients who have ST elevation in one territory often have ST depression in other territories. The additional ST deviation may represent ischemia in a myocardial region other than the area of infarction or may represent pure reciprocal changes. There is abundant literature on the significance of different types of ST depression during STEMI [72]. Most of the common patterns of remote ST depression probably represent reciprocal changes and not ischemia at a distance. In anterior STEMI, ST depression in the inferior leads is reciprocal to involvement of the basal anterolateral region, supplied by the first diagonal branch and represented by ST elevation in leads I and aVL [76,77]. In patients who have inferior STEMI, ST depression in lead aVL is a pure reciprocal change and is found in almost all patients [78], and ST depression in leads V1 to V3 probably does not represent ischemia at a distance but rather reciprocal changes caused by more posterior, inferoseptal, apical, or lateral left ventricular involvement [53,55,56]. In contrast, among patients who have inferior STEMI, ST depression in leads V4 to V6 is associated with concomitant left anterior descending (LAD) coronary artery stenosis or three-vessel disease [11,13,79]. Thus, presence of an atypical pattern of ST depression and especially ST depression in leads V4 to V6 in inferior STEMI may signify ischemia at a distance.

In special circumstances both types of ST depression may be present. In STEMI caused by occlusion of the first diagonal branch, in addition to ST elevation with positive T waves in leads aVL and V2, there usually is reciprocal ST depression with negative T waves in the inferior leads (pure mirror image) and a different pattern of ST depression with tall, peaked T waves (subendocardial ischemia) in V4 and V5; the ST segment in lead V3 is either isoelectric or depressed [77].

Boden and colleagues [80] reported that in patients who have non-STEMI, isolated ST-segment depression in leads V1 to V4 was more likely to caused by posterior wall STEMI (reciprocal changes) when it was associated with upright T waves; it was caused by anterior subendocardial ischemia when the T waves were negative., Porter
and colleagues [81], however, found that the polarity of the T waves in the precordial leads with ST depression cannot be used to differentiate between the two etiologies of ST depression. In many patients who have inferior STEMI and ST depression in leads V1 to V3, the associated T waves in these leads are negative initially (a reciprocal image of ST elevation with positive T waves in leads facing the infarct zone), and only later do the T waves become positive and the R-wave amplitude increase (a reciprocal image of inversion of the T waves and development of Q waves in leads facing the infarction).

Identification of the exact site of the infarct-related artery

The admission ECG, by suggesting the location of the ischemic area at risk, may assist in identifying the exact site of coronary artery occlusion. Because of variability in the coronary anatomy, in some instances there may be more than one possible explanation for a specific ECG pattern. Moreover, because the size and exact location of the vascular bed supplied by the occluded artery varies considerably, occlusion in the same site of a coronary artery in different patients may result in a different size and location of the ischemic area at risk and hence different ECG changes. In addition, presence of severe pre-existing narrowing in a nonculprit coronary artery may cause ischemia at a distance that may alter the classic ECG picture. Much of the work that studied the correlation between various ECG patterns and the site of the culprit lesion included only patients who had single-vessel disease and a first myocardial infarction; thus the applicability of these criteria to the general population, and especially to the patients who had prior STEMI or coronary artery bypass graft, is unclear.

Acute anterior ST-elevation myocardial infarction

Classic ECG patterns

LAD obstruction usually causes ST elevation in the precordial leads V1 to V4 [82]. Aldrich and colleagues [83] reported similar findings showing the frequency of ST elevation in patients who have acute STEMI caused by LAD occlusion to be, in descending order: V2, V3, V4, V5, aVL, V1, and V6 [83]. Uncommonly, ST elevation in leads V1 to V4 may be caused by proximal right coronary artery (RCA) occlusion with concomitant right ventricular infarction [46,84,85]. In Blanke and co-workers’ [82] analysis of patients who have acute STEMI caused by RCA occlusion, the frequency of ST elevation in these four precordial leads was as follows: V1, 5%; V2 and V3, 15%; and V4, 8%. These investigators found no instances of ST elevation in leads V1 to V4 in the patients who had acute myocardial infarction caused by LCX occlusion. Right ventricular infarction that produces ST elevation in leads V1 to V4 may be distinguished from anterior STEMI by observing an ST elevation in lead V1 greater than in lead V2. ST elevation in the right precordial leads V3R and V4R, ST depression in lead V6, and ST elevation in the inferior leads II, III, and aVF [84,86]. The magnitude of ST elevation in lead V1 correlates better with the magnitude of ST elevation in lead V2 and V3R than with lead V2, suggesting that ST elevation in lead V1 reflects the right ventricle more than the left ventricle [86]. The typical ECG findings in acute anterior STEMI are presented in Box 1.

Diagnosis of anterior infarction extending to contiguous myocardial zones

Anterosuperior myocardial zone

The high anterolateral wall at the base of the left ventricle receives its coronary blood flow from the first diagonal branch of the LAD, the first obtuse marginal branch of the LCX, or, occasionally, from the ramus intermedius artery [87]. The ECG lead that most directly faces this anterosuperior myocardial zone is lead aVL [87,88]. In acute anterior STEMI, ST elevation in lead I, and particularly in lead aVL, signifies an LAD occlusion proximal to the first diagonal branch [88,89]. In contrast, ST depression in lead aVL during acute anterior STEMI signifies LAD occlusion distal to the first diagonal branch [90]. Although ST elevation in lead aVL is a very specific sign of proximal LAD occlusion, it has a relatively low sensitivity for this diagnosis. Sasaki and colleagues [91] noted that patients who have a proximal occlusion of long LAD artery that wraps around the cardiac apex have concomitant injury to the inferorapical and anterosuperior walls of the left ventricle. When this happens, no ST elevation may be seen in either anterosuperior leads (ie, I, aVL) or inferior leads (ie, II, III, aVF) because the opposing forces cancel each other. Isolated occlusion of the first diagonal branch also may result in ST elevation in lead aVL [77,88]. The ECG can be useful in distinguishing isolated diagonal branch occlusion from LAD occlusion proximal...
Box 1. ECG findings in acute anterior STEMI

**Precordial leads**
- ST elevation is usually present in V2 to V4.
- ST elevation in V4 to V6 without ST elevation in V1 to V3 usually is caused by LCX or distal diagonal occlusion.
- ST elevation in leads V2 to V6 may represent LAD occlusion proximal to the first diagonal branch.

**Leads I and aVL**
- ST elevation in lead I and aVL signifies
  1. Occlusion of a short LAD coronary artery before the first diagonal branch (if there is ST elevation in V2 to V4)
  2. Occlusion of first diagonal branch (if associated with ST elevation in V2 and isoelectric ST or ST depression in V3 to V6)
  3. Occlusion of the first marginal branch of the LCX (if there is ST depression in V2)
- ST depression in aVL signifies LAD artery occlusion distal to the first diagonal branch.

**Leads II, III, and aVF**
- ST depression in the inferior leads signifies
  1. Occlusion of a short LAD coronary artery before the first diagonal branch (if there is ST elevation in V2 to V4)
  2. Occlusion of the first diagonal branch (if associated with ST elevation in V2 and isoelectric ST or ST depression in V3 to V6)
- ST elevation in the inferior leads signifies occlusion of a long LAD artery (that wraps the cardiac apex) distal to the first diagonal branch.

**Lead aVR**
- ST elevation in aVR signifies LAD artery occlusion proximal to the first septal branch.

**Right bundle-branch block (new)**
- Right bundle-branch block signifies LAD artery occlusion proximal to the first septal branch.

to the first diagonal branch, however. Occlusion of the diagonal branch typically results in ST elevation in leads I, aVL, and V2 with ST segments in leads V3 and V4 either isoelectric or depressed [77,87]. In contrast, LAD occlusion proximal to the first diagonal branch results in ST elevation extending beyond lead V2 or V3 and occasionally, to V4 or V6 [77,87]. In addition, when ST elevation in leads I and aVL are caused by occlusion of the LCX, reciprocal ST depression is usually observed in lead V2 because the vascular bed supplied by the LCX extends more posteriorly [25].

Several ECG criteria have been reported by Engelen and colleagues [90] to indicate an LAD artery occlusion proximal to the first septal perforator branch: (1) ST elevation in lead aVR (sensitivity 43%; specificity 95%; \( P = .000 \)); (2) right bundle branch block (sensitivity 14%; specificity 100%; \( P = .004 \)); (3) ST depression in lead V5 (sensitivity 17%; specificity 100%; \( P = .009 \)); and (4) ST elevation in lead V1 greater than 2.5 mm (sensitivity 12%; specificity 100%; \( P = .011 \)). These findings were also supported by a recent study done by Vasudevan and colleagues [92] on 50 patients who had acute anterior STEMI and underwent angiography within 3 days of the infarct. Criteria reported to indicate LAD artery occlusion distal to the first diagonal perforator branch include abnormal Q waves in leads V4 to V6 [90]. On the other hand, Birnbaum and colleagues [93] did not find an association between ST elevation in lead V1 and LAD artery occlusion proximal to the first septal branch. Ben-Gal and colleagues [94] suggested that because the right paraseptal area is supplied by the septal
branches of the LAD, alone or together with the conal branch originating from the RCA, ST elevation in lead V₁ in anterior STEMI is caused by right paraseptal ischemia in patients who have an anatomically small and nonprotective conal branch of the RCA.

**Lateral and apical myocardial zones**

Most patients (93%) who have an acute anterior STEMI caused by LAD occlusion have an anteroseptal pattern (ST elevation in leads V₁ to V₃) [82,83]. In contrast, isolated ST elevation in leads V₄ to V₆, without ST elevation in leads V₁ to V₃, usually is caused by an occlusion of the LCX or distal diagonal branch. Many assume that in patients who have extensive anterior myocardial infarction (ST elevation in leads V₁ to V₆), the injury extends to the distal anterolateral wall and cardiac apex caused by a long LAD artery or prominent diagonal branches, whereas patients who have an anteroseptal pattern (ST elevation confined to leads V₁ to V₃) have a short LAD or large obtuse marginal branches or ramus intermediate branch supplying these anterolateral and apical zones. A study by Shalev and colleagues [95] investigated the correlation of the ECG pattern of anteroseptal myocardial infarction with the echocardiographic and angiographic findings. They found that 48 of 52 patients (92%) who presented with ST elevation in leads V₁ to V₃ had an anteroapical infarct and a normal septal motion. The culprit narrowing was found more frequently in the mid to distal LAD (in 85% of patients). They conclude that the ECG pattern traditionally termed “anteroseptal STEMI” should be called an “anteroapical myocardial infarction”; the term “extensive anterior STEMI” should be used when associated with diffuse ST changes involving the anterior, lateral, and occasionally, inferior leads. Recently, using transthoracic echocardiography, Porter and coworkers [96] found no difference in regional wall motion abnormalities in the lateral and apical segments in patients presenting with first acute anterior STEMI, with or without ST elevation in leads V₅ and V₆.

**Inferior myocardial zone**

During acute anterior STEMI, injury may extend to the inferior wall, as evidenced by ST elevation in leads II, III, and aVF, if the LAD artery wraps around the cardiac apex [82,97,98]. As previously mentioned, however, anterior STEMI that is caused by a wrapping LAD occlusion proximal to the first diagonal branch does not manifest as an anterior and inferior injury pattern because of cancellation of opposing vectors [91,98]. In theory, occlusion of an LAD artery that supplies collateral blood flow to an obstructed RCA or LCX may produce a similar anterior and inferior pattern; however, Tamura and colleagues [98] did not observe such a pattern in the 12 patients they studied with this type of ECG.

ST depression in the inferior leads II, III, and aVF during acute anterior STEMI indicates injury to the high anterolateral wall and does not signify inferior wall ischemia [99,100]. Several investigators found such reciprocal ST depression in the inferior leads to indicate LAD artery occlusion proximal to the first diagonal branch [76,90]. In patients who have a long LAD artery that wraps around the cardiac apex, however, proximal LAD artery occlusion may not produce reciprocal ST depression in the inferior leads because of extension of the infarction to the inferoapical wall [91].

**Acute inferior ST-elevation myocardial infarction**

**Classic ECG patterns**

In inferior STEMI, the leads showing the greatest magnitude of ST elevation, in descending order, are leads III, aVF, and II. Most patients who have ST elevation in these inferior leads (80%–90%) have an occlusion of the RCA; however, an occlusion of the LCX can produce a similar ECG pattern [101]. In addition to ST elevation in the inferior leads II, III, and aVF, reciprocal ST depression in lead aVL is seen in almost all patients who have acute inferior STEMI [78]. The ECG distinction between RCA- and LCX-related inferior STEMI is presented in Table 1 [101–107]. ECG confirmation of the infarct-related artery during acute inferior STEMI may be particularly valuable when coronary angiography indicates lesions in both the RCA and LCX.

**Criteria in the precordial leads**

Because the right ventricular branch originates from the RCA, criteria for right ventricular infarction, especially ST elevation in leads V₃R and V₄R, provide compelling evidence that the infarct-related artery in acute inferior STEMI is the proximal RCA. Kontos and colleagues [102] reported that LCX-related inferior STEMI was
suggested by reciprocal ST depression in leads V1 and V2. When Birnbaum and colleagues [13] compared patients who had inferior STEMI caused by mid or distal RCA versus LCX occlusion, however, they found no difference in the frequency of ST depression in leads V1 to V3. ST depression is absent in leads V1 to V3 only during proximal RCA occlusion, because the resultant right ventricular injury pattern cancels out such reciprocal ST depression [13]. Some investigators have reported a higher frequency of ST elevation in leads V4 to V6 in patients who had LCX-related acute inferior STEMI [102,103]. Hasdai and colleagues [105], however, found little difference in the frequency of ST elevation in leads V4 to V6 between patients who have LCX- versus RCA-related inferior STEMI. The authors state that because most cases of acute inferior STEMI are caused by RCA occlusions, the positive predictive value (PPV) of ST elevation in leads V5 or V6 for LCX-related infarction is only 59% [105]. Kosuge and colleagues [106] reported that the magnitude of ST depression in lead V3 relative to the ST elevation in lead III (V3:III ratio) was useful in distinguishing the culprit artery in inferior STEMI. They found that a V3:III ratio of less than 0.5 indicated a proximal RCA occlusion; a V3:III ratio of 0.5 to 1.2 indicated a distal RCA occlusion; and a V3:III ratio of more than 1.2 indicated LCX occlusion [106].

Again, these criteria are based on the fact that with RCA occlusion the vector that causes ST depression in lead V3 is masked by the vector of right ventricular injury.

**Criteria in the limb leads**

ECG criteria in the limb leads also have been found to be useful in distinguishing RCA and LCX occlusion in acute inferior STEMI. For example, greater ST elevation in lead III than in lead II has been shown to indicate RCA infarction [104,108]. Additional limb lead criteria involve careful analysis of leads I and aVL. Patients who have LCX-related STEMI less frequently show reciprocal ST depression in lead aVL and more often show an isoelectric or a raised ST segment in leads I and aVL compared with patients who have RCA-related inferior infarction [102,104]. Hasdai and colleagues [105] reported that such absence of reciprocal ST depression in lead aVL indicates injury of the anterosuperior base of the heart typically caused by LCX occlusion proximal to the first obtuse marginal branch. These investigators found absence of reciprocal ST depression in leads V4 to V6 in patients who had LCX-related acute inferior STEMI [102,103].

<table>
<thead>
<tr>
<th>Left Circumflex Artery Occlusion</th>
<th>Sensitivity (%)</th>
<th>Specificity (%)</th>
<th>Right Coronary Artery Occlusion</th>
<th>Sensitivity (%)</th>
<th>Specificity (%)</th>
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<tbody>
<tr>
<td></td>
<td>80</td>
<td>84</td>
<td>ST V3 / III &gt; 1.2</td>
<td>84</td>
<td>93</td>
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<tr>
<td></td>
<td>88</td>
<td>94</td>
<td>ST II &gt; ST III</td>
<td>86</td>
<td>94</td>
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**Table 1**

| Common ECG features distinguishing the culprit artery in acute inferior wall myocardial infarction |
|----------------------------------|----------------|
| Right Coronary Artery Occlusion  | Sensitivity (%) | Specificity (%) |
| V3:II > 1 | 91 | 91 |
| V3:II < 0.5 | 91 | 91 |
| V3:II = 0.5-1.2 | 91 | 91 |
| V3:II > 1.2 | 91 | 91 |
| Abbreviation: RCA, right coronary artery. |
ST depression in lead aVL in 86% of patients who had proximal LCX-related inferior STEMI but in none of the patients who had RCA- or distal LCX-related infarctions (P = .0001). An additional criterion for identifying the culprit artery in inferior STEMI is the magnitude of ST depression in lead aVL compared with lead I [104]. Greater reciprocal ST depression in lead aVL than in lead I suggests an RCA-related inferior STEMI [104]. A likely explanation for this phenomenon is that injury of the high posterolateral region caused by LCX occlusion attenuates ST depression in lead aVL more than in lead I, which has a less superior orientation. A final criterion in lead aVL to distinguish the culprit artery in inferior STEMI relates to the amplitude of the respective R and S waves in this lead [107]. In the initial stages, leads facing an infarcted wall with ST elevation tend to show QRS changes as well, including an increase in R-wave amplitude and a decrease in S-wave amplitude [109]. Therefore, in inferior STEMI, the opposite pattern (that is, decrease in R wave and increase in S wave) would be expected in lead aVL, if there is no involvement of the high posterolateral region (RCA infarction). In contrast, if there is concomitant involvement of the high posterolateral segments (as expected especially in proximal LCX infarction), these reciprocal changes in the QRS may not be apparent. Indeed, Assali and colleagues [107] found that a decrease in R-wave amplitude and an increase in S-wave amplitude with a S:R ratio greater than 3 predicted RCA occlusion, whereas a S:R ratio less than 3 predicted LCX occlusion. A summary of the criteria to distinguish the culprit artery in inferior STEMI is provided in Table 1, and an example of the criteria is shown in Fig. 6. From these criteria it is clear that the ability to differentiate RCA from LCX occlusion is greater for proximal occlusion. Because occlusion occurs more distally in the culprit artery, the distinctive characteristics are lost, and the ECG cannot be expected to distinguish between right and left posterior descending artery occlusion.

A recent study by Fiol and colleagues [110] has suggested using the following ECG criteria in a three-step algorithm to differentiate RCA from LCX occlusion as the culprit artery: (1) ST changes in lead I, (2) the ratio of ST elevation in lead III to that in lead II, and (3) the ratio of the sum ST depression in leads V1 to V3 divided by the sum ST elevation in leads II, III, and aVF. ST elevation in lead I has a 100% PPV for LCX occlusion, whereas ST depression of 0.5 mm or more has a 94% PPV for RCA occlusion. A ratio of ST elevation in lead III to lead II greater than 1 has a 92% PPV for RCA occlusion, and the ratio of the sum of ST depression in leads V1 to V3/ST elevation in II, III, aVF of 1 or less has a 90% PPV for RCA occlusion. Application of this sensitive algorithm suggested the location of the culprit coronary artery (RCA versus LCX) in 60 of 63 patients (>95%).

Diagnosis of inferior infarction extending to contiguous myocardial zones

Right ventricular myocardial zone

Right ventricular infarction occurs almost exclusively in the setting of inferior STEMI. Although isolated right ventricular infarction has been reported, it is rare and occurs most often in patients who have right ventricular hypertrophy [111]. Several investigators have found that ST elevation in lead V4R is diagnostic of right ventricular infarction with a sensitivity and specificity well over 90% [101,112,113]. It is important to point out that ST elevation in the right precordial leads (eg, V4R) is most prominent in the early hours of inferior STEMI and dissipates rapidly thereafter. Hence, the window of opportunity to diagnose right ventricular infarction by ECG is limited, and right precordial leads should be recorded immediately as a patient who has ST elevation in the inferior leads presents to the emergency room. As mentioned previously, ST elevation in leads V1 to V3 in a patient who has inferior STEMI is a manifestation of associated right ventricular infarction caused by a proximal RCA occlusion [46,84,85]. Lopez-Sendon and colleagues [114] reported that the criterion of ST elevation in lead V4R greater than ST elevation in any of leads V1 to V3 is a very specific sign of right ventricular infarction (specificity, 100%). This criterion was less sensitive (sensitivity 78.6%) than ST elevation in V4R alone, however [114].

A recent analysis of the GUSTO-I angiographic substudy by Sadanandan and colleagues [115], in patients who had concomitant ST elevation in the anterior (V1-V4) and inferior (II, III, aVF) leads, revealed that the infarct-related artery was the RCA in 59% of the cases and was the LAD in 36%. More patients who had RCA occlusion had ST elevation in lead V1 equal to or greater than the ST elevation in lead V3 compared with those with LAD occlusion (35% versus 12%; P = .001). Furthermore, the progression of ST
elevation from lead V₁ to lead V₃ was significantly greater in patients who LAD occlusion than in those who had RCA occlusion. Thus, larger ST elevation in leads II, III, and aVF and absence of progression of ST elevation from V₁ to V₃ differentiate RCA from LAD occlusion in patients who have combined anterior and inferior ST elevation. Only 35% of the patients who had RCA occlusion had ST elevation in lead V₁ equal to or greater than the ST elevation in V₃, however. Therefore, the absence of this criterion does not exclude RCA occlusion.

Lateral apical myocardial zone

In patients who have inferior STEMI, ST elevation in leads V₅ and V₆ is considered to indicate extension of the infarct to the lateral aspect of the cardiac apex; however, there is as yet no direct evidence for this extension [116]. The cause of such an extension may be occlusion of either the LCX or RCA with a posterior descending or posterolateral branch that extends to the lateral apical zone [116]. Tsuka and coworkers [58] found that ST elevation in lead V₆ during inferior STEMI was associated with a larger infarct size, a greater frequency of major cardiac arrhythmias, and a higher incidence of pericarditis during the patient’s hospital course. A study by Golovchiner and colleagues [117] assessed the correlation between ST deviation in each of the six precordial leads and the presence of regional wall motion abnormalities by transthoracic echocardiography in 109 patients who had first inferior STEMI. ST elevation in lead V₅ was associated with more frequent involvement of the apical portion of the

Fig. 6. (A) Admission ECG of a patient who has acute inferior STEMI and total occlusion of the proximal right coronary artery on immediate angiography. There is ST elevation in leads II, III, and aVF. ST elevation is greater in lead III than in lead II. There is reciprocal ST depression and deep S waves in lead aVL. The magnitude of ST depression in aVL is greater than in lead I. In addition, there is no ST depression in leads V₁ to V₃. (B) Admission ECG of a patient who has acute inferior STEMI caused by occlusion of the proximal LCX on immediate angiography. There is ST elevation in leads II, III, and aVF and ST depression in leads V₁ to V₃. The magnitude of ST elevation in lead III is smaller than in lead II. Of note, there is no reciprocal ST depression in lead aVL.
inferior wall \((P < .02)\), with a specificity of 88% and a sensitivity of 33%. Global regional wall motion abnormality score was significantly worse for patients who had ST elevation than for patients who had isoelectric ST in lead V3 \((P = .024)\). ST elevation in lead V6 was associated with regional wall motion abnormality in the mid-posterior segment \((P < .006)\), with a specificity of 91% and a sensitivity of 33%, and worse global regional wall motion abnormality score \((P = .022)\).

**Posterior myocardial zone**

In patients who have inferior STEMI, ST depression in leads V1 to V3 has been shown to indicate a larger infarction with extension of the injury to the posterolateral or the inferoseptal wall [45,53–56,118–122]. Such ST depression in these anterior leads during inferior STEMI is a reciprocal change and does not indicate concomitant LAD coronary artery disease [11,79]. It may be seen in both RCA and LCX inferior infarctions [44,123]. In inferior STEMI caused by proximal RCA occlusion with concomitant right ventricular infarction, however, posterior wall injury may be masked because the two opposed electrical vectors may cancel each other (that is, ST elevation in leads V1 to V3 with right ventricular infarction and reciprocal ST depression in these same leads with concurrent posterior infarction) [122]. A more direct sign of posterior wall injury is ST elevation in leads V7 to V9 [59,124–126]. Waveform amplitudes in these posterior leads are smaller than in standard precordial leads, however. There is preliminary evidence that ST elevation of 0.5 mm should be considered a sign of injury when analyzing the posterior leads [127]. Isolated ST elevation in leads V7 to V9 without ST elevation in the inferior leads occurs in only 4% of patients who have acute myocardial infarction [126] and usually is caused by LCX occlusion [124]. In patients who have acute inferior STEMI, ST elevation in leads V7 to V9 is associated with a higher incidence of reinfarction, heart failure, and death [59].

**Ischemia at a distance in acute inferior ST-elevation myocardial infarction**

Most of the ST depression patterns seen during STEMI represent reciprocal changes rather than ischemia at a distance [72]. One ECG pattern, ST depression in leads V5 and V6 in inferior STEMI, signifies concomitant disease of the LAD with acute ischemia in a myocardial zone remote from the infarct zone [11,13,79,128]. Patients who have maximal ST depression in leads V4 to V6 during inferior STEMI have higher morbidity and mortality than patients without precordial ST depression or with maximal ST depression in leads V1 to V3 [25]. Likewise, patients who have maximal ST depression in leads V4 to V6 undergo multivessel revascularization (multivessel PCI or coronary artery bypass surgery) more often than do patients who do not have such an ECG pattern [128].

ST segment elevation in lead aVR has been shown to be a marker of severe diffuse coronary disease in patients who have unstable angina or non-STEMI. ST elevation in lead aVR also can be a marker of acute left main coronary (LMCA) occlusion. Yamaji and colleagues [129] studied the admission 12-lead ECG in 16 consecutive patients who had acute LMCA obstruction, 46 patients who had acute LAD obstruction, and 24 patients who had acute RCA obstruction. Lead aVR ST-segment elevation \((>0.05 \text{ mV})\) occurred with a significantly higher incidence \((P < .01)\) in the LMCA group (88%) than in the LAD (43%) or RCA (8%) groups. Lead aVR ST-segment elevation was significantly higher in the LMCA group \((0.16 \pm 0.13 \text{ mV})\) than in the LAD group \((0.04 \pm 0.10 \text{ mV})\). Lead V1 ST-segment elevation was lower in the LMCA group \((0.00 \pm 0.21 \text{ mV})\) than in the LAD group \((0.14 \pm 0.11 \text{ mV})\). The finding of lead aVR ST-segment elevation greater than or equal to lead V1 ST elevation distinguished the LMCA group from the LAD group with 81% sensitivity, 80% specificity, and 81% accuracy. ST segment shift in lead aVR and the inferior leads distinguished the LMCA group from the RCA group. The authors conclude that lead aVR ST-segment elevation with less ST-segment elevation in lead V1 is an important predictor of acute LMCA obstruction [129].

**Discussion**

The admission ECG pattern is the most informative noninvasive tool for the diagnosis, triage, and risk stratification in patients who have STEMI. Three ECG patterns presented in this article are especially relevant: (1) right ventricular infarction accompanying acute inferior STEMI, (2) a very proximal LAD occlusion in anterior STEMI, and (3) patients at higher risk (ie, those who have grade 3 ischemia, ST depression in V4–V6 concomitant with inferior STEMI, and ST elevation in lead aVR).
Moreover, it is crucial to recognize cases in which opposing ECG vectors cancel each other and result in attenuation of the ischemic changes, such as occlusion of a proximal LAD that wraps the cardiac apex or a proximal dominant LCX. In terms of the first assessment, the opportunity to diagnose right ventricular infarction using the ECG is greatest in the emergency department because ST elevation in the right precordial leads resolves quickly. The admitting physician should make certain that all patients who have acute inferior STEMI have a second ECG recorded with right ventricular leads. If ST-segment elevation of 1 mm is observed in lead VdR, the diagnosis of right ventricular infarction can be made.

Another ECG assessment of importance to the admitting physician is the identification of a very proximal LAD occlusion in acute anterior STEMI. If the occlusion site is proximal to the first diagonal branch of the LAD, a large portion of the left ventricle is at risk of irreversible damage, including the anteroseptal, anteroseptal, anterolateral, and apical regions. Such high-risk patients should be transferred urgently to a cardiac catheterization laboratory for primary PCI.

Patients who have grade 3 ischemia on the admission ECG have higher mortality [24–26,130,131] and reinfarction rates [130,132]. Retrospective analysis of the GUSTO IIB trial patients revealed that grade 3 ischemia was associated with higher mortality both in the primary PCI group and in the thrombolysis group [130]. In the grade 2 group, in-hospital mortality was similar in the thrombolysis and angioplasty subgroups (3.2% and 3.3%; \( P = .941 \)). In patients who had grade 3 ischemia, in-hospital mortality was 6.4% and 7.3%, respectively (\( P = .762 \)). These findings were confirmed in an analysis of the Danish multicenter randomized study on fibrinolytic therapy versus acute coronary angioplasty in acute myocardial infarction, DANAMI–2 [133]. The study showed that mortality increased significantly with symptom duration (> 3 hours) in both grade 2 and grade 3 ischemia regardless of treatment strategy. For patients presenting early (< 3 hours of symptom onset) who had grade 3 ischemia, however, those in the primary PCI group had a 5.5% 30-day mortality reduction (1.4% versus 6.9%) compared with the thrombolysis group.

Summary

The admission ECG in patients who have STEMI is valuable for selecting patients who are candidates for early reperfusion treatment and also for providing information regarding the location and extent of acute myocardial injury. By reflecting the pathophysiology of the myocardium during acute ischemia, the ECG conveys information not provided by coronary angiography and provides important information to guide clinical decision making.

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