

The electrocardiographic diagnosis of acute myocardial infarction in the thrombolytic era

Paul Schweitzer MD. *New York, N.Y.*

Early diagnosis of acute myocardial infarction (AMI) is an important requirement for achieving successful coronary reperfusion.¹ Such diagnosis can be made by electrocardiography,^{2,3} echocardiography,⁴ or thallium scintigraphy.⁵ Because the electrocardiogram (ECG) is readily available, simple, and inexpensive, it is best suited to diagnose AMI and remains an important criterion for the initiation of coronary reperfusion.⁶ The purpose of this report is to review the diagnostic accuracy of the ECG in patients with AMI, to examine its ability to predict the infarct-related artery and estimate infarct size, and to evaluate its role in selecting and following patients undergoing thrombolytic therapy.

THE VALUE AND LIMITATIONS OF THE ECG IN THE DIAGNOSIS OF AMI

The diagnostic accuracy of the ECG in AMI, regardless of its localization or type (Q wave or non-Q wave infarction), was reported by Rude et al.,² who evaluated the ECG in 3697 patients with suspected AMI. The ECG criteria for AMI were: new or presumably new Q waves, ST segment elevation or depression, and left bundle branch block (LBBB). An AMI was confirmed by elevation of serum creatine phosphokinase or its MB fraction (CK-MB). In this study, 1888 patients (51%) with chest pain did not have an AMI and in the subset of 1809 patients with documented AMI, the ECG did not show ST segment elevation or pathologic Q waves in 341 subjects (18%). However, in 577 (30%) of 1888 with normal CK-MB, the ECG was suggestive of an AMI. Thus the sensitivity and specificity of the ECG for AMI were 81% and 69%, respectively. As expected, not all ECG findings had the same diagnostic value. For ex-

ample, the combination of new or presumably new Q waves in two leads with ST segment elevation, with or without reciprocal ST segment depression, was diagnostic of AMI in 82% to 100% of cases. The same was true for ST segment elevation alone. However, ST segment depression or LBBB was a poor predictor of AMI, correctly doing so in only 52% to 56% of patients.

Zarling et al.⁷ reported 100 case of AMI confirmed by autopsy with the ECG available in 85 patients. According to these authors, the ECG was diagnostic in 25 patients (29.5%), suggestive in 27 patients (31.8%), nonspecific in 22 cases (25.8%), and normal in 11 patients (12.9%). The low diagnostic yield of the ECG found by Zarling et al.⁷ is in contrast with the 82% to 100% diagnostic accuracy of the ECG in patients with suspected AMI reported by Rude et al.² The most important difference between these two studies is the patient selection. Patients reported by Rude et al.² were hospitalized in the coronary care unit with suspected AMI, while Zarling et al.⁷ analyzed patients with anatomically confirmed AMI in whom only 56% had a history consistent with AMI; in the remaining 44% the history was either atypical or unobtainable because of coma, aphasia, or intubation.

There are several causes of false negative ECGs in AMI, including interobserver variability of ECG interpretation,⁸ delay in evolution of ECG changes, and myocardial infarction (MI) in which the ECG either remains normal or reveals only nonspecific STT wave changes.^{2,7,9-11} Additionally, in some patients the ECG diagnosis of AMI is simply missed, which according to Lee et al.¹² occurs in 4% of AMI cases presenting to the emergency room.

A nondiagnostic ECG in AMI is clinically significant in that patients with either a normal or nonspecific ECG have a low incidence of serious complications (cardiogenic shock, ventricular tachyarrhythmia, high-degree atrioventricular [AV] block, or congestive heart failure).^{2,7,9-11} It also suggests a smaller MI, or an occlusion of the circumflex artery (CFA),¹³ with less overall myocardial damage.

From the Division of Cardiology, Department of Medicine, Bronx Veterans Administration Medical Center, and the Division of Cardiology, Department of Medicine, Mount Sinai School of Medicine.

Received for publication May 5, 1989; accepted Oct. 10, 1989.

Reprint requests: Paul Schweitzer MD, Division of Cardiology, Bronx VA Medical Center, 130 W. Kingsbridge Rd., Bronx, NY 10468.

4/1/18039

THE VALUE OF THE ECG IN THE IDENTIFICATION OF INFARCT-RELATED VESSEL

The identification of the infarct-related artery is relatively easy in patients with single-vessel disease or anterior MI due to left anterior descending artery (LAD) occlusion, but may be difficult in patients with multivessel disease or inferior myocardial infarction (IMI), in whom either the right coronary artery (RCA) or the CFA may be the infarct-related artery.

The diagnostic accuracy of the ECG for predicting the infarct-related artery in AMI has been studied by Blanke et al.¹⁴ and by others.^{13, 15} Blanke et al.¹⁴ performed coronary angiography in 146 patients within a 6.3 ± 6 hours after the onset of chest pain. The most frequent infarct-related artery was the LAD (56.2%), followed by the RCA (26.7%) and CFA (17.1%). Huey et al.¹³ studied 241 patients with CK-MB-confirmed AMI who underwent coronary angiography before discharge from the hospital. The RCA, LAD, and CFA were the infarct-related artery in 44.4%, 39%, and 16.6% of cases, respectively. In the Western Washington trial,¹⁵ the incidence of LAD, RCA, and CFA occlusions was 47%, 45%, and 8%, respectively. The most likely cause of the differences between these reports was patient selection. Blanke et al.¹⁴ studied patients with clinical findings suggestive of AMI regardless of the ECG, while Huey et al.¹³ analyzed patients with documented AMI by enzymes; the Western Washington study¹⁵ included patients only with ST segment elevation on the initial ECG.

The ECG in left anterior descending artery occlusion. The LAD supplies the anterior and anterolateral walls and two thirds of the interventricular septum.¹⁶ Depending on the site of the occlusion and the collateral circulation, the extent of necrosis can vary markedly. Proximal occlusion of the LAD results in a more extensive MI than a mid artery occlusion. According to Stadius et al.,¹⁵ 70% of patients with LAD disease have a proximal occlusion before the first diagonal branch.

In patients with anterior AMI, the ECG changes are seen in the precordial leads and in leads I and aVL. The correlation between LAD occlusion and anterior AMI on the ECG is good. As shown by Blanke et al.,¹⁴ anterior AMI was diagnosed in 90% and 83% of patients with total and subtotal occlusion of the LAD, respectively. Similar results were reported by Huey et al.¹³ The best lead for detecting LAD occlusion was lead V₂, followed by leads V₁ and V₃. This applies not only for the presence of the Q waves but also for ST segment elevation.

The results of Blanke et al.¹⁴ suggest that the ECG is not helpful in localizing the site of occlusion within

the LAD. However, there are some ECG findings that may indicate larger MIs. According to Haraphongse et al.,¹⁷ the degree of ST segment elevation and associated ST segment depression in the inferior leads may suggest a more extensive anterior MI. Lew et al.¹⁸ believe that ST segment elevation in the inferior leads indicates that the LAD also supplies part of the inferior wall and therefore suggests a more extensive necrosis. Fig. 1 shows the ECG from a patient with a mid LAD occlusion. In addition to an extensive anterior MI, there is also an IMI, because the LAD reaches the apical portion of the inferior wall.

The ECG in right coronary artery occlusion. The RCA supplies the right and left ventricles, the posterior third of the interventricular septum, and in some subjects the posterolateral wall of the left ventricle.¹⁶ In RCA occlusion, ST segment elevation and/or Q waves in leads III and aVF are seen in 70% to 90% of patients.^{13, 14, 19}

Occlusion of the RCA can either be proximal (before the right ventricular branches) or distal. The site of the RCA occlusion is almost always before the vessel reaches the left ventricle, which explains why most patients with RCA disease have an IMI. According to Stadius et al.,¹⁵ RCA occlusion was proximal, mid, and at the level of the posterior descending artery in 57.7%, 38.7%, and 3.6% of patients, respectively. Proximal RCA occlusion causes IMI, which is associated with right ventricular ischemia and/or infarction (RVMI). One of the most useful ECG findings in RVMI is ST segment elevation in lead V_{4R}.^{20, 21} According to Braat et al.,²² the ST segment elevation is of short duration, and in 50% of cases becomes isoelectric within 10 hours. The ECG diagnosis of RVMI was recently reviewed in *this Journal* by Robalino et al.²³

Several ECG findings are helpful in differentiating between proximal and distal RCA occlusion. First, in lead V_{4R}, ST segment elevation is seen with proximal occlusions and isoelectric ST segments with upright T waves are seen in distal RCA occlusions.²⁴ Second, in proximal occlusions, the ST segment can be elevated in leads V₁²⁵ or V₁²⁶. This is illustrated in Fig. 2, which shows an IMI with minimal ST segment elevation in leads V₁ and V₂. Without the IMI, the ST elevation in leads V₁ and V₂ could easily be considered a normal variant. However, the combination of a IMI with ST segment elevation in leads V₁ and V₂ should raise the possibility of RVMI. Third, RVMI should be suspected in patients with IMI complicated by second- or third-degree AV block.²⁷ Fourth, according to some authors²⁸ but not others,^{29, 30} reciprocal changes can help to localize the site of occlusion of the RCA. Berland et al.²⁸ suggested that

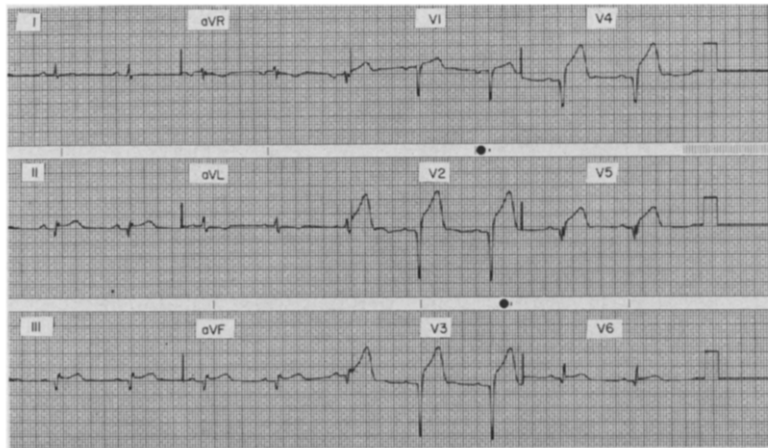


Fig. 1. Twelve-lead ECG showing an extensive acute anterior and inferior myocardial infarction due to occlusion of the mid LAD. The RCA and CFA were normal.

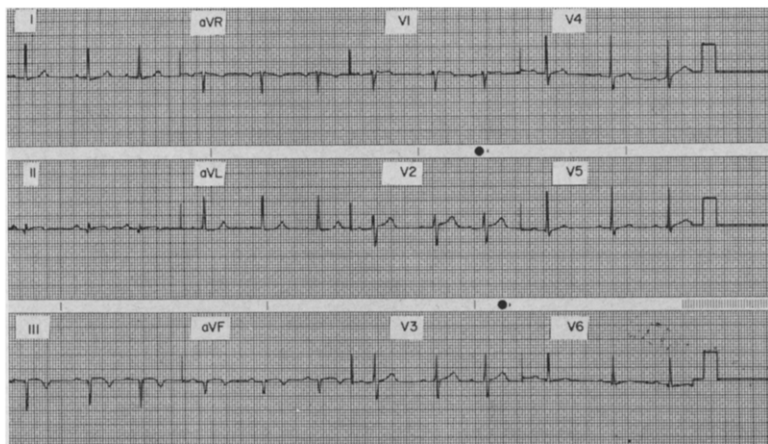


Fig. 2. Twelve-lead ECG showing an acute IMI with ST segment elevation of 0.1 to 0.15 mV in lead V_2 in a patient with proximal RCA occlusion causing RVMI or right ventricular ischemia. The LAD and CFA were normal.

patients with proximal RCA occlusion have a higher incidence of ST segment depression in leads V_2 to V_4 . This view, however, is not supported by the results of Lew et al.,^{29,30} who showed that proximal RCA occlusion with the involvement of the RV attenuates ST segment depression in leads V_1 to V_4 . According to these authors,^{29,30} a ratio of ST segment depression in lead V_2 to ST segment elevation in lead aVF of 50% or less suggests proximal RCA occlusion and RV involvement. Consistent with this assumption is the ECG in Fig. 3, which was recorded from a patient with acute IMI due to proximal RCA occlusion who only had minimal ST segment depression in leads V_2 and V_3 . In contrast, mid RCA occlusion (after the

right ventricular branch) and lateral extension potentiate reciprocal changes in leads V_2 to V_4 .^{29,30} However, as seen in Fig. 4, this is not always the case. This ECG was recorded from a patient with inferolateral MI due to mid RCA occlusion after the right ventricular branch. The RCA also supplied the posterolateral wall of the left ventricle. Despite the presence of two factors that potentiate ST segment depression in leads V_2 to V_4 , the reciprocal changes were minimal.

In patients with RVMI, the ST segment elevation in leads V_1 to V_4 can imitate an anteroseptal AMI.²⁶ The differential diagnosis between these two localizations of MI is usually not difficult. In patients with

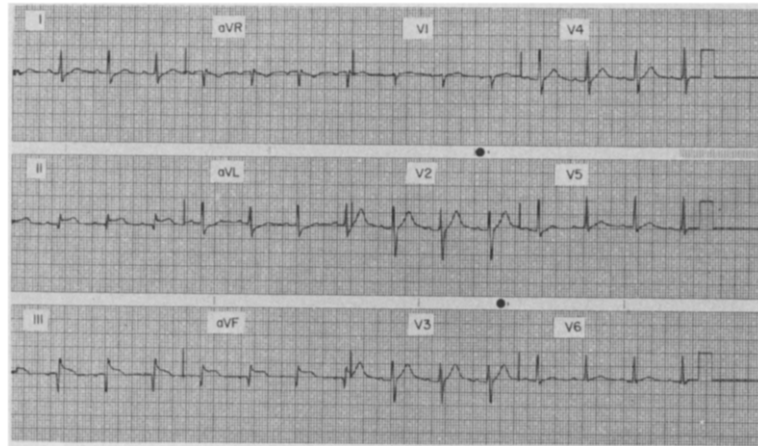


Fig. 3. Twelve-lead ECG of an acute IMI due to occlusion of the RCA before the right ventricular branch. In the first 24 hours the patient developed type 1 second-degree AV block. Note the absence of reciprocal ST segment depression in leads V_2 to V_4 , which supports proximal RCA occlusion. The LAD and CFA were normal.

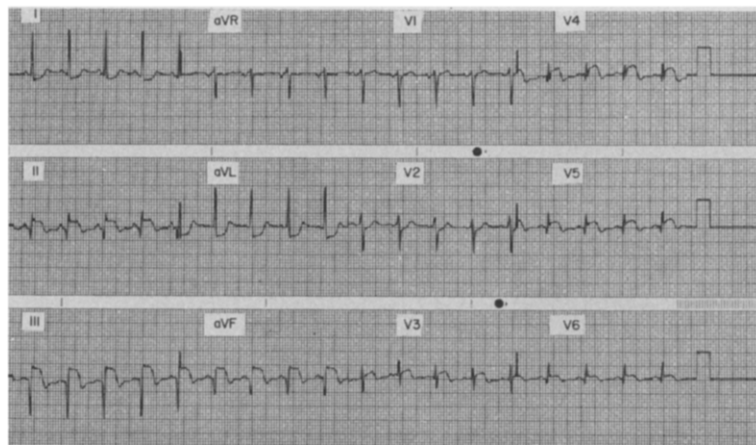


Fig. 4. Twelve-lead ECG of an acute inferolateral MI due to occlusion of the mid RCA. Note the minimal ST segment depression in lead V_2 despite the fact that the site of occlusion was after the right ventricular branch and the lateral extension, both of which potentiate reciprocal changes. The LAD and CFA were normal.

RCA occlusion, the precordial ST segment elevation is associated with upright T waves and Q waves and small or absent ST segment elevation in the inferior leads.²⁶ On the other hand, in anteroseptal AMI there is a loss of R waves and normalization of the precordial ST segment associated with T wave inversion. Furthermore, in RVMI there is a gradual decrease of the ST segment elevation from leads V_1 to V_4 , while in anteroseptal MI the opposite is true. Of note, according to Boden et al.³¹ and others,³² the initial ECG in posterior MI due to RCA or CFA occlusion can show only ST segment depression in leads V_2 to V_4 , thereby imitating anterior non-Q wave infarction.

The ECG in circumflex artery occlusion. The CFA

with its obtuse marginal branches supplies the posterolateral wall of the left ventricle, and in some patients, the inferior wall of the left ventricle as well.¹⁶ In patients with CFA occlusion, ST segment elevation in the lateral leads (I, aVL, V_5 , and V_6) was seen only in 50% of patients^{13, 14} and abnormal Q waves were observed even less frequently (15% to 19%).¹³ This low sensitivity of the ECG may be caused by late depolarization of the posterolateral wall, where necrosis does not cause abnormal Q waves, or it may be due to a small MI or to the absence of leads close to the posterolateral wall. Occlusion of the CFA can cause an IMI, a lateral MI (leads I and aVL, with or without changes in leads V_5 and V_6), or a true pos-

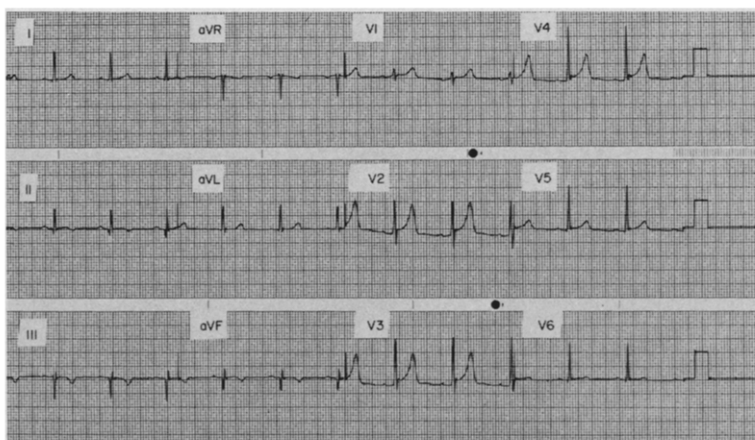


Fig. 5. Twelve-lead ECG showing acute lateral MI due to CFA disease. This patient had a 70% obstruction of both the second obtuse marginal branch and the mid CFA. The LAD and RCA were normal.

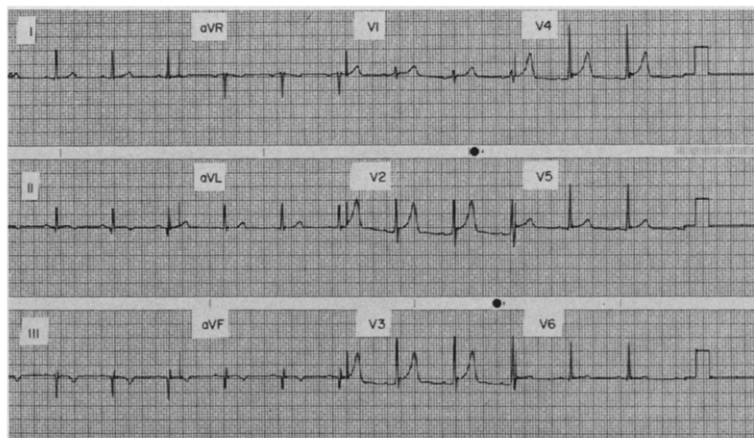


Fig. 6. Twelve-lead ECG 24 hours after chest pain in a patient with inferior and posterior MI. The patient had a total proximal CFA occlusion and 90% narrowing of the proximal RCA. The LAD was without significant stenosis.

terior MI (R duration of 0.04 second or more, and $R > S$ in lead V_1 or V_2). In 80% of patients with CFA disease the occlusion is within the main vessel, and in 20% it is due to obstruction of an obtuse marginal branch.¹⁵

According to Dunn et al.,³³ there is some relationship between the ECG and the site of CFA obstruction. In patients with lateral MI the occlusion is in the proximal CFA, the high lateral branch, or the first obtuse marginal branch. The ECG in Fig. 5 illustrates this, depicting a lateral MI due to 70% obstruction of both the second obtuse marginal branch and the mid CFA. On the other hand, an IMI is usually caused by distal occlusion of the CFA. A tall R wave in leads V_1 or V_2 may be caused by a proximal or distal CFA occlusion. In addition, the presence of a tall R wave in

V_1 suggests a larger MI and multivessel disease,¹³ which may make the identification of the infarct-related artery more difficult. This is illustrated in Fig. 6, which shows an inferoposterior MI. This patient had both proximal CFA occlusion and 90% RCA obstruction.

Differential diagnosis between RCA and CFA occlusion. The differentiation between RCA and CFA occlusion is difficult, or according to some authors, impossible.^{14,19} ECG findings helpful in localizing the infarct-related artery (Table I) include an isoelectric or elevated ST segment in lead I, which suggests CFA occlusion,³⁴ while ST segment depression in the same lead is seen more frequently in RCA occlusion.¹³ ST segment elevation in the lateral leads suggests CFA disease, and a tall R wave in lead V_1 but

not in V_2 is seen more frequently in patients with CFA occlusion than in those with RCA occlusion.¹³ Finally, the T wave in lead V_{4R} is negative in CFA occlusion and positive in distal RCA disease.²⁴

RECIPROCAL ST SEGMENT CHANGES

Reciprocal ST segment changes (RSTCHs), i.e., ST segment depression in the leads opposite the ST segment elevation, can be present in MIs of different locations. RSTCHs are more frequent in patients with IMI than in those with anterior MI. The mechanism of RSTCH, particularly in IMI, remains controversial.³⁵⁻⁵³

Reciprocal changes in inferior myocardial infarction. This topic was recently reviewed in *this Journal* by Mirvis,⁵⁰ and also by Boden and Spodick⁵³ elsewhere, and we will therefore limit our discussion to a few major points (Table II). First, RSTCHs have more than one cause. Besides being an electrophysiologic phenomenon,³⁵⁻³⁸ RSTCH may be due to distant ischemia³⁹⁻⁴² or be a manifestation of larger infarct, with extension to the posterolateral and posterior wall or to the posterior third of the interventricular septum.⁴³⁻⁴⁹

Second, the good correlation between the ST segment depression in lead aVL and the ST segment elevation in the inferior leads suggests that in these leads the ST segment depression is a true electrophysiologic phenomenon.⁴⁴ In contrast, the correlation between ST segment elevation in the inferior leads and ST segment depression in leads V_1 to V_4 is variable.⁴⁴ As mentioned, a RVMI decreases the degree of the ST segment depression in leads V_1 to V_4 , while a lateral MI has the opposite effect.^{29, 30} Yet, as seen in Fig. 4, this is not always the case.

From a practical point of view, the most important question is whether the ST segment depression in the precordial leads is due to more extensive MI or is secondary to anterior ischemia. The ECG in Fig. 7 illustrates the limitation of the coronary anatomy alone in the clarification of this question. The ECG was recorded from a patient with acute IMI and prominent reciprocal changes in leads V_2 to V_5 and in leads I and aVL. Coronary angiography 2 weeks later showed subtotal occlusion of the mid RCA as well as 70% stenosis of the mid LAD and of the CFA after the second obtuse marginal artery. Anterior ischemia secondary to LAD disease was ruled out because of the absence of anterior wall motion abnormalities or ischemia during thallium stress testing 2 month later. The impaired contractility of the diaphragmatic and posterobasal segments of the left ventricle suggests that the cause of the precordial reciprocal ST changes was an extensive MI.

Table I. Differential diagnosis between RCA and CFA occlusion

ECG	RCA	CFA
<i>ST segment</i>		
I	Depression	Elevation or isoelectric
aVL	Depression	Depression or elevation
V_5 or V_6	Isoelectric or elevation	Elevation
<i>T wave</i>		
V_{4R}	Upright	Inverted
$R > S$ in V_1	+	+++

RCA, Right coronary artery; CFA, circumflex artery.

Table II. Reciprocal ST segment changes in inferior myocardial infarction

1. Multiple causes
2. More extensive impairment of left ventricular function
3. Higher complication rate during the acute phase of AMI
4. ? Worse long-term prognosis
5. May help to localize the site of occlusion (proximal versus distal)
6. Thrombolytic therapy may be more beneficial in patients with reciprocal changes

Third, regardless of the mechanism, patients with RSTCHs in leads V_1 to V_4 have more impaired ventricular function, a higher complication rate, and increased mortality. However, the significance of RSTCH for the long-term prognosis is not clear. Gelman et al.⁵⁴ and others^{55, 56} hold that long-term survival is lower in patients with RSTCH, though others have not confirmed these findings.^{57, 58}

Reciprocal changes in anterior myocardial infarction. RSTCHs are less frequent in patients with anterior AMI. Katz et al.⁵⁸ found RSTCH in 72% of patients with acute IMI and in only 37% of patients with anterior AMI. The reason for this lower incidence of RSTCH in anterior AMI is not clear, but may be due to the smaller number of leads in which RSTCH can be recorded. According to Haraphongse et al.,¹⁷ RSTCH in anterior MI suggests a more extensive MI, a higher incidence of multivessel disease, a lower ejection fraction, and more extensive wall motion abnormalities. Notwithstanding, Lew et al.¹⁸ believe that RSTCHs in anterior AMI are related to the length of the LAD, and found ST segment depression only when the LAD artery terminated before or at the apex of the heart. Similar findings were reported by Ferguson et al.³⁸ and by Myers et al.⁵⁹

THE EVOLUTION OF ECG CHANGES IN AMI

Coronary artery occlusion is followed by the well-known evolution of the ECG. The earliest changes are

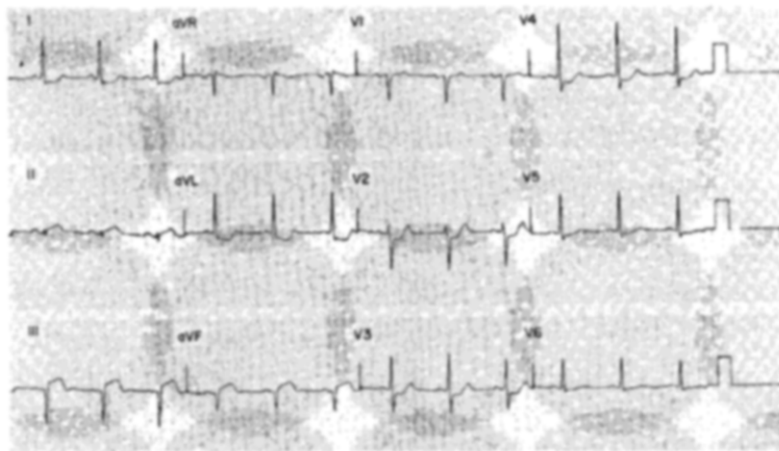


Fig. 7. Twelve-lead ECG in a patient with acute IMI and prominent ST segment depression in the precordial leads and in leads I and aVL due to subtotal mid RCA obstruction. The mid LAD and the CFA after the second obtuse marginal artery had 70% obstruction.

an increase of the amplitude of the R and T waves and ST segment elevation, which are followed by the development of Q waves, loss of R wave amplitude, and T wave inversion.⁶⁰⁻⁷¹ In general, ST segment elevation is a manifestation of ischemic injury, while the QRS changes represent myocardial necrosis. The natural course of these ECG changes was studied by several authors using either 12-lead ECGs or precordial ST segment mapping.⁶⁰⁻⁷¹ Because of differences in the number of leads, the time from the onset of the presumed myocardial necrosis to the recording of the ECG, and the exact point at which the ST segment elevation was measured, it is not surprising that the results of these studies are not uniform. In the majority of patients, there is a rapid fall of the ST segment within the first 12 hours, followed by a plateau and a gradual normalization.^{62-64, 66, 68} Abnormal Q waves are seen as early as 2 hours after the onset of pain, and are usually fully developed within 9 hours (4 to 14 hours).^{60, 62, 65} The loss of R waves follows a similar course.^{62, 64, 65} According to Yusuf et al.,⁶⁸ the evolution of ECG changes in AMI may be delayed for up to 30 hours. In addition, the development of ECG changes is faster in inferior than in anterior MI.⁶⁰ In some patients there is a re-elevation of the ST segment,^{63, 64, 69, 70} which may be a manifestation of infarct extension or expansion,⁷² pericarditis,⁶⁷ or tachycardia.⁷³

RELATIONSHIP BETWEEN INFARCT SIZE AND ECG

Since the introduction of the concept of limitation of infarct size and coronary thrombolysis, the role of the ECG in the estimation of infarct size was extensively studied.⁷⁴⁻⁸² The following questions are of clinical significance.

First, does the extent of ST segment elevation reflect infarct size? Experimental and some but not all clinical studies showed good correlation between the extent of ST segment elevation and infarct size, as measured by enzymes or histologic techniques.^{71, 75-82} However, because the degree of ST segment elevation is influenced not only by the magnitude of myocardial injury but also by other factors such as electrolytes, pericarditis, heart rate, and infarct localization, it is not surprising that the results were not uniform.^{60, 71} The limitations of the ST segment analysis for evaluation of the extent of myocardial injury and necrosis was reviewed by Fozzard and DasGupta.⁸³

Second, can the degree of ST segment elevation predict the development of QRS complex changes that might better reflect the extent of necrosis? According to Muller et al.⁷³ and others,⁷⁴⁻⁷⁸ there is good correlation between the extent of ST segment elevation and loss of R waves or the development of Q waves. On the other hand, Thygesen et al.⁶⁶ found only a weak relationship between ST segment elevation and QRS complex changes. A poor correlation may be expected, since 40% to 60% of patients with ST segment elevation develop non-Q wave infarction⁸⁴⁻⁸⁶ without QRS complex changes.

Third, can infarct size be measured from the QRS complex changes? In the last 10 to 15 years different approaches were used to measure infarct size from the QRS complex.^{80, 81, 87, 88} Probably the best known method is the Selvester QRS scoring system.⁸⁹ According to the results from Duke University,⁹⁰⁻⁹³ the closest correlation is between the QRS complex and the anatomic size of anterior MI, and the worst correlation is in posterolateral MI. Similarly, the corre-

lation between CK-MB fraction and the QRS complex is better for anterior than for inferior MI.⁹⁴ In addition, the QRS scoring system can not be used in patients with left ventricular hypertrophy or conduction abnormalities including left anterior fascicular block⁸⁹ and in the 10% to 20% of patients whose AMI does not show classic ECG changes.^{2, 7, 9-11}

THE ROLE OF THE ECG IN CORONARY THROMBOLYSIS

The role of the ECG in patients undergoing coronary thrombolysis is twofold: first, identification of candidates for coronary reperfusion and second, evaluation of coronary thrombolysis.

According to the Netherlands trial,^{95, 96} coronary thrombolysis is more effective in patients with prominent ST segment elevation than in those with a smaller degree of ST segment elevation. In this study, the end point for efficacy of thrombolytic therapy was early mortality and limitation of enzymatic infarct size. However, because of the rapid decrease of the ST segment elevation within the first 12 hours,^{62-64, 66, 68} this criterion may not be helpful in patients seen later (>6 hours) who still could benefit from coronary reperfusion.^{97, 98} In other words, there is a need for additional criteria to identify patients in whom thrombolysis could be beneficial. According to Mauri et al.,⁹⁹ the number of leads with ST segment elevation correlates with the efficacy of coronary thrombolysis. In patients with ST elevation in two to three leads, the mortality was similar in the streptokinase and placebo group regardless of infarct localization. In patients with ST elevation in four or more leads there was a statistically significant decrease in mortality in patients treated with streptokinase.

According to Mauri et al.,⁹⁹ the majority of patients with IMI had a small MI (92.6%), which explains the failure of coronary reperfusion to improve survival. Thrombolysis was beneficial in the remaining 7.4% who had posterior or lateral extension, supporting the assumption that opening the coronary artery could be effective in larger IMIs. As mentioned, the most frequent cause of ST segment depression in the precordial leads is a more extensive IMI, and therefore patients in this subset should be candidates for coronary reperfusion.^{28, 100}

The ECG can also identify patients who will not benefit from thrombolysis. Two large studies showed that coronary reperfusion is not effective in patients with ST segment depression^{1, 97} or in those with previous MI.¹

What are the limitations of the ECG for selecting patients for thrombolysis? The most important one

is the absence of classic ECG changes in 10% to 20% of patients with AMI.^{2, 7, 9-11} This finding raises the issue whether patients with suspected AMI without diagnostic ECG findings should have the diagnosis of AMI confirmed by echocardiography⁴ or thallium-201 imaging.⁵ Until recently, it was felt that this was not necessary because coronary reperfusion was most effective in patients with ST segment elevation.^{95, 96} In contrast, according to newer studies patients without ST segment elevation, except those with ST depression,^{97, 98} may also benefit from thrombolysis. These findings imply that patients with normal or nonspecific ST-T wave changes should have the diagnosis of AMI substantiated or excluded by other methods. However, because of the possibility of some adverse effects of late reperfusion, particularly increased myocardial rupture,¹⁰¹ further studies are needed to clarify the indication for late reperfusion in AMI. Another limitation of the ECG is that in some patients with prolonged chest pain the ST segment elevation is a manifestation of unstable angina.¹⁰² If in patients with ischemic chest pain unstable angina can not be ruled out, coronary reperfusion should be considered because of some beneficial effect of thrombolysis in these patients.¹⁰³

The ECG's role in evaluating successful coronary reperfusion remains controversial. According to the initial studies of Rentrop et al.¹⁰⁴ and of others,¹⁰⁵⁻¹⁰⁷ and a more recent report of Hackworthy et al.,¹⁰⁸ coronary recanalization is associated with more rapid evolution of the ST segment. In contrast, the usefulness of the ST segment monitoring to assess coronary reperfusion was not confirmed by Kircher et al.¹⁰⁹ or by others^{110, 111}—i.e., a significant decrease or normalization of the ST segment is consistent with coronary reperfusion, while minimal or no change of the ST segment does not rule out opening of an occluded vessel. In all of these studies the ECG was recorded intermittently.

Investigators who used continuous ST segment monitoring for evaluation of coronary reperfusion¹¹²⁻¹¹⁵ achieved better results. Essen et al.¹¹² found that the ST segment normalized rapidly following reperfusion and remained unchanged in the setting of a resistantly occluded vessel. Kruckoff et al.,¹¹³ using the time at which the ST segment reached a steady-state condition and the rate of ST segment recovery (millimeters per hour), showed that in patients with successful coronary reperfusion a steady-state condition was reached earlier and the ST segment recovery was more rapid in those with successful coronary reperfusion in comparison with those with an occluded coronary artery. However, because of an overlap of the ST segment changes in

the two groups, this technique is of limited value in assessing coronary reperfusion in individual patients. According to Hackett et al.,¹¹⁴ there was a good correlation between ST segment elevation and intermittent reocclusion of the coronary artery. Finally, Hogg et al.¹¹⁵ found good correlation between the fractional change of ST segment elevation, i.e., the difference between the ST segment elevation before and after thrombolytic therapy, and successful coronary reperfusion. The main drawback of this study was the small number of patients, especially those without reperfusion, and the use of two different monitoring techniques. The effect of thrombolytic therapy was evaluated by Holter monitoring and a 12-lead ECG within 2 to 3 and at 5 hours, respectively.

Another proposed marker of successful coronary reperfusion is the appearance of reperfusion arrhythmias (accelerated idioventricular rhythm, marked bradycardia), whose value as markers of coronary thrombolysis remains unsettled. Goldberger et al.¹¹⁶ found good correlation between coronary recanalization and the appearance of reperfusion arrhythmias, although Kircher et al.¹⁰⁹ and others^{110, 117} did not.

The effect of coronary thrombolysis on the QRS complex was studied by Ganz et al.¹⁰⁵ and Essen et al.¹¹² In the majority of patients, coronary reperfusion did not prevent the development of Q waves but did cause more rapid evolution of Q waves, a smaller sum of Q waves, and some normalization of the Q waves in the next 4 to 6 months.¹¹² Furthermore, according to Essen et al.¹¹² and others,¹¹⁸ in patients with successful coronary reperfusion the loss of R waves was less extensive and was followed by regrowth of the R waves. Another interesting finding was reported by Mikell et al.,¹¹⁹ who found that after successful coronary reperfusion, despite the development of pathologic Q waves, the left ventricular function was better preserved than in patients in whom the infarct-related artery remained occluded.

Conclusions. The value and limitations of the 12-lead ECG in the diagnosis of AMI are as follows. First, the ECG remains a simple and inexpensive method to diagnose early AMI and, with some limitation, it also helps to differentiate between Q and non-Q wave infarction. Second, 10% to 20% of patients with AMI do not have classic ECG changes. These patients have a low incidence of life-threatening complications and therefore do not require admission into the coronary care unit. However, because of the potential benefit from coronary thrombolysis, the diagnosis of AMI should be confirmed by non-ECG methods. Third, classic ECG changes are present in 90% of patients in whom the infarct-

related artery is the LAD, in 70% to 80% of those with RCA occlusion, and in only 50% of patients with CFA occlusion. These findings suggest that in patients with suspected AMI and a nondiagnostic ECG, CFA is the most likely infarct-related artery. Fourth, the view that ST segment elevation represents the initial phase of Q wave infarction is not completely correct because 40% to 60% of patients with ST segment elevation only develop non-Q wave MI. In contrast, ST depression in leads V₂ to V₄ can be either an anterior non-Q or a posterior MI. Fifth, in the majority of patients with IMI, the reciprocal changes in the precordial leads are a manifestation of larger infarction, i.e., involvement of the lateral and or posterior wall of the left ventricle. These patients may benefit from coronary thrombolysis. Sixth, the value of the ECG in estimating myocardial injury and infarct size remains controversial. However, certain findings (number of involved leads or the degree of ST segment elevation) are related to the extent of myocardial injury or necrosis. Seventh, patients with ST segment elevation are most likely to benefit from coronary thrombolysis; however, the absence of ECG changes does not preclude improvement in mortality except in those with ST segment depression and probably also in patients with a previous MI. The role of the ECG in evaluating the reperfusion status after coronary thrombolysis is not clear. A rapid return to baseline or a normalization of the ST segment suggest opening of the occluded vessel, though a small or negligible change does not exclude successful reperfusion.

SUMMARY

The 12-lead ECG remains a simple and inexpensive technique to diagnose AMI in its early phases. The diagnostic accuracy of the ECG depends upon the extent of myocardial necrosis and its localization. The ECG is most sensitive in patients with occlusion of the LAD artery, followed by the RCA and the left CFA. In 10% to 20% of patients with AMI the initial ECG either shows nonspecific changes or is normal. The correlation between the ECG and infarct-related artery varies according to the involved vessel. Classic ECG changes are seen in 90% of the LAD artery, in 70% to 80% of RCA, and in only 50% of CFA occlusions. A second important issue is the mechanism and clinical significance of reciprocal ST segment changes, which usually indicate larger MI, more impaired ventricular function, worse prognosis, and in some patients, significant disease of a noninfarct-related artery. Furthermore, the value of the ECG in estimating myocardial injury and infarct size remains controversial. The ECG plays an important role in

coronary reperfusion. ST segment elevation is one of the principal criteria for instituting thrombolytic therapy, and helps predict those who will most likely benefit from coronary reperfusion. The role of the ECG in evaluating the reperfusion status after coronary thrombolysis is not clear. Rapid return to baseline or normalization of the ST segment suggests opening of the occluded vessel, though a small or negligible change does not exclude successful reperfusion.

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