

Diagnosis of Acute Myocardial Infarction in Angiographically Documented Occluded Infarct Vessel*

Limitations of ST-Segment Elevation in Standard and Extended ECG Leads

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Study objectives: The majority of thrombolysis studies require defined ST-segment elevations as an inclusion criterion for the diagnosis of acute myocardial infarction (AMI). However, depending on the occluded infarct vessel and the criteria applied, the ECG diagnosis of AMI can be difficult to establish. Accordingly, this study was performed to evaluate the sensitivity of ST-segment elevation of standard and extended ECG leads in a cohort of patients with angiographically confirmed diagnosis of AMI.

Patients and methods: In 418 patients (mean $\pm$ SD age, 60 ± 13 years) with AMI (pain onset, 4.8 ± 3.0 h), coronary angiography with percutaneous transluminal coronary angioplasty/stenting of the culprit lesion was performed. The diagnosis of AMI was confirmed by emergency coronary angiography and laboratory analyses. ST-segment elevation (in two contiguous leads) of 1 mm in standard lead I through aVF and ST-segment elevations of 2 mm (or 1 mm, corresponding values presented in parentheses) in V_1 through V_6 were considered significant. In a subset of 102 AMI patients, additional right precordial leads V_3R through V_6R for evaluation of right ventricular infarction and additional chest leads V_7 through V_9 for evaluation of posterior infarction were recorded. ST-segment elevations of 1 mm in the right precordial leads and 1 mm or 0.5 mm in the posterior leads were considered significant.

Results: Standard leads I through V_6 showed ST-segment elevation in 85% (96%) of patients with left anterior descending artery occlusion, in 46% (61%) of patients with left circumflex coronary artery (CX) occlusion, and in 85% (90%) of patients with right coronary artery occlusion. On consideration of additional ECG tracings in the subgroup of 102 patients (V_3R through V_6R and V_7 through V_9), the respective numbers increased by 2 to 8% depending on different criteria for ST-segment elevation; in patients with CX occlusion, the increase amounted to 6 to 14%. There was a trend toward an extended infarct size (maximum creatine kinase [CK] values) with concomitant ST-segment elevation in additional ECG leads as assessed by maximum CK levels.

Conclusions: The sensitivity of the ECG diagnosis of AMI is only marginally increased by extended precordial chest leads. There is a trend toward an extended infarct size in those patients with concomitant ST-segment elevation in additional ECG leads. (CHEST 2001; 120:1540–1546)

Key words: acute myocardial infarction; ECG; left circumflex artery

Abbreviations: AMI = acute myocardial infarction; CK = creatine kinase; CX = left circumflex coronary artery; LAD = left anterior descending coronary artery; NS = not significant; RCA = right coronary artery

Diagnosis of acute myocardial infarction (AMI) is based on a history of acute chest pain in conjunction with ECG criteria and laboratory findings.

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Although new laboratory methods such as measurement of troponin T have emerged, the ECG is still the most readily available and fastest method for the diagnosis of AMI. Thrombolytic trials have shown that reduction in mortality is greatest when reperfusion of the infarct vessel is achieved within 6 h of pain onset and—at least for thrombolytic agents—this benefit may be limited up to 12 h at the most.¹ Rapid intervention in the setting of evolving myocardial infarction has been convincingly shown^{1–19} to be

advantageous with respect to cardiovascular events, short-term as well as long-term survival, infarct size, and ventricular function. However, the risk of major bleeding complications has to be considered, which is generally assumed to be approximately 1% for thrombolytic treatment,^{6,11} and can be as high as 10%,¹² and which can be fatal in the setting of aortic dissections when thrombolysis is performed based on the presence of thoracic pain alone. There is controversy about the optimal management of non-ST-segment elevation myocardial infarction, and lack of ECG changes in conventional leads could possibly lead to an unwarranted denial of thrombolysis in patients with AMI.¹³ Accordingly, it would be desirable to have improved ECG criteria in addition to symptoms and laboratory analyses on which further therapeutic decisions can be based.

In a considerable number of cases, however, the ECG diagnosis of AMI remains questionable.¹⁴ According to older studies,^{14–19} the ECG does not show classical changes such as ST-segment elevation or pathologic Q waves in 10 to 20% of patients with AMI based on laboratory data with elevation of serum creatine phosphokinase or its MB fraction.

The issue is even more troublesome in those situations in which the left circumflex coronary artery (CX) is affected. The CX is the dominant vessel in only about 10% of humans^{20,21} and is the least frequently affected vessel in myocardial infarction.¹⁴ However, posterolateral AMIs due to CX occlusion often elude ECG diagnosis. Another difficulty is encountered with the differentiation from right coronary artery (RCA) involvement in patients with inferior or posterior myocardial infarction.^{14,21–33} In patients with CX infarction, ST-segment elevation has been reported^{14,28,34} in only up to 50% in the lateral leads I, aVL, V₅, and V₆. Other publications reported an improvement in the diagnosis of CX infarction by considering posterior chest leads; however, others observed only a limited value of posterior (and right ventricular) leads in comparison to the standard 12-lead ECG.^{35–38} These data stem primarily from patients with a laboratory diagnosis of AMI in various thrombolysis studies. There are only a few studies with a greater number of patients undergoing exclusively emergency percutaneous transluminal coronary angioplasty/stenting of the infarct vessel; one of the first studies was performed at our institution.³⁹

Accordingly, it was the aim of the present study to evaluate the sensitivity of standard ECG for diagnosis of AMI and whether sensitivity is increased on inclusion of ST-segment elevation in extended leads (right precordial and posterior ECG leads) in patients who underwent percutaneous transluminal coronary angioplasty/stenting and in whom the diagnosis of AMI was confirmed angiographically.

MATERIALS AND METHODS

Patients with an AMI < 12 h from the onset of thoracic pain were enrolled in this prospective study. In all 418 patients, a 12-lead ECG was recorded immediately on hospital admission, in addition to laboratory analysis for confirmation of AMI diagnosis (rise in serum creatine kinase [CK] activity of > 100 IU/L with a concomitant rise in MB isoenzyme to $\geq 8\%$ of the total CK activity); three additional posterior leads (V₇ through V₉) and four right precordial leads (V_{3R} through V_{6R}) were recorded only in a subset of 102 AMI patients. This is due to the fact that these additional leads have been routinely recorded since 1996 in our hospitals, but the study started in 1993. Patients with symptoms suggestive of AMI—with or without ST-segment alterations—underwent emergency coronary arteriography, enabling determination of infarct vessels and area, and subsequent comparison with the hospital admission ECG. The culprit lesion (the thrombotically occluded infarct-related vessel that was subsequently revascularized during the invasive procedure in > 90% of cases) as well as additional stenoses $\geq 50\%$ of cross-sectional area were analyzed. Excluded from the study were patients with left or right bundle-branch block, left or right ventricular hypertrophy, ventricular pre-excitation, onset of thoracic pain > 12 h, valvular or congenital heart disease, and patients receiving digitalis.

The ECGs were quantitatively analyzed for ST-segment changes (*ie*, elevation ≥ 1 mm in surface leads I through aVF, ≥ 2 mm and ≥ 1 mm in V₁ through V₆, ≥ 1 mm in right lateral leads, and ≥ 1 mm and ≥ 0.5 mm in posterior leads V₇ through V₉, respectively; 1 mm = 0.1 mV).⁴⁰ Since ECG criteria are not uniform in all thrombolysis studies with respect to ST-segment elevation in precordial leads, two different modes of ST-segment analysis were applied, namely, ≥ 2 mm in two or more contiguous precordial leads V₁ through V₆, and ≥ 1 mm in two or more contiguous precordial leads V₁ through V₆. Measurements on the ECGs were made independently by two of the investigators using magnifying lenses.

Evaluation of the diagnostic gain when extended leads (V_{3R} through V_{6R} and V₇ through V₉) were considered was done only in the subset of 102 AMI patients in whom extended ECG leads were available. Maximal CK activity was determined to serve as a measure of infarct size.

For statistical analyses, values are expressed as mean $\pm$ SD. To test for homogeneity across all three infarcted vessel-related groups, Pearson's χ^2 was used for categorical variables, and one-way analysis of variance was used for continuous variables (Table 1). All *p* values ≤ 0.05 were considered to indicate statistical significance. Statistical analyses including those for sensitivity were performed with statistical software (SPSS version 8.0; SPSS; Chicago, IL).

RESULTS

Of 1,788 patients presenting with a confirmed diagnosis of AMI, only 418 patients fitted the time frame of < 12 h from the beginning of symptoms and were included in the study. The mean age of all patients was 60 ± 13 years. Three hundred fourteen patients (75%) were men. AMI was confirmed in each of the 418 patients as described above. Previous myocardial infarction had occurred in 19% of patients with an occlusion of the left anterior descending coronary artery (LAD), in 19% of patients with an occlusion of the CX, and 15% of patients with an occlusion of the RCA. Identical reinfarct localization

Table 1—Clinical Parameters of the Subgroup of Patients Presenting With AMI and Standard as Well as Extended ECG Leads*

Parameters	LAD	CX	RCA
Subgroup size, No.	40	36	26
Age, yr	60 ± 12	60 ± 13	58 ± 12
Male:female ratio	3:1	4:1	4:1
Previous myocardial infarction, %	23	22	23
Identical reinfarction localization, %	5	6	8
Time of pain onset to ECG recording, h	4.5 ± 3.2	5.4 ± 3.1	3.6 ± 2.9
Dominant vessel, % RCA/CX/none	59/27/15	57/10/33	70/26/4
Presence of collateral vessels, %	18	17	15
Maximum CK, IU/L	1,399 ± 1,184	928 ± 692	708 ± 570
CK level at hospital admission, IU/L	212 ± 329	294 ± 443	118 ± 149
Arterial hypertension, No. (%)	28 (70)	19 (53)	16 (62)
Diabetes mellitus, No. (%)	6 (15)	4 (11)	1 (4)
Current smokers, No. (%)	19 (48)	18 (50)	12 (46)

*Data are presented as mean ± SD unless otherwise indicated.

was found in 7%, 2%, and 5%, respectively. Average onset of chest pain was 4.8 ± 3.0 h before hospital admission (4.3 ± 2.9 h, 5.5 ± 2.8 h, and 4.8 ± 3.1 h for LAD, CX, and RCA occlusions, respectively). The CK values at the time of the initial ECG were 267 ± 589 IU/L, 293 ± 447 IU/L, and 134 ± 167 IU/L for the groups with LAD, CX, and RCA occlusions, respectively. The corresponding maximum CK values were $1,333 \pm 1,129$ IU/L, 921 ± 714 IU/L, and 734 ± 599 IU/L. Coronary arteriography revealed the culprit obstruction in the LAD in 161 patients (39%), the CX in 120 patients (29%), and the RCA in 137 patients (33%). Patients were subclassified according to availability of standard and extended ECG leads; clinical characteristics of the subgroup with extended leads ($n = 102$) are presented in Table 1. There was no difference for any clinical, ECG, or laboratory parameter between the respective subgroups of patients with and without extended leads, except for time from pain onset to ECG recordings in the RCA group.

ST-Segment Elevation Infarction

LAD Occlusion: The 12-lead standard ECG revealed ST-segment elevations ≥ 1 mm in leads I through aVF plus ≥ 2 mm in V_1 through V_6 in 85% (137 of 161 patients) and ST-segment elevations ≥ 1 mm in leads I through V_6 in 96% (155 of 161 patients) with an occluded LAD. In a subgroup of 40 patients with extended chest leads, the respective numbers for conventional leads I through V_6 were 85% (34 of 40 patients) and 100% (40 of 40 patients). Taking into account all available leads with inclusion of the extended chest leads in this subgroup, the respective numbers are 90% (36 of 40 patients; Fig 1) and 100% (40 of 40 patients); the numbers are equal, regardless if 1 mm or 0.5 mm were considered

as significant ST-segment elevation in V_7 through V_9 . In 28% (11 of 40 patients), we observed ST-segment elevation in V_3R through V_6R , and in 10% (4 of 40 patients), there were ST-segment elevations in V_7 through V_9 ; the majority of these patients had multivessel disease with significant stenoses ($> 50\%$) of the RCA and CX. Two of 40 patients with an isolated ST-segment elevation in V_4R were in the subgroup of patients in whom ≥ 2 mm ST-segment elevation in V_1 through V_6 was considered significant. These patients had multivessel disease with chronic occlusion of the RCA, reinfarction of the LAD, and, in addition, presumably an occlusion of collateral vessels to the RCA. Mean CK level was $1,422 \pm 1,018$ IU/L in the subgroup of 40 patients with ST-segment elevations in the standard leads only as compared to $1,694 \pm 1,589$ IU/L in the subgroup of patients with concomitant ST-segment elevations in the extended ECG leads ($p =$ not significant [NS]).

CX Occlusion: In CX occlusion, the standard 12-lead ECG revealed ST-segment elevation in only 46% (55 of 120 patients) in whom ≥ 2 mm of ST-segment elevation in V_1 through V_6 were considered significant and in 61% (73 of 120 patients) with ST-segment elevation ≥ 1 mm in V_1 through V_6 . In a subgroup of 36 AMI patients with available extended chest leads, the respective numbers for conventional leads I through V_6 were 50% (18 of 36 patients) and 72% (26 of 36 patients). Taking into account these additional chest leads, the respective numbers are 61% (22 of 36 patients; Fig 1) and 75% (27 of 36 patients) when considering ST-segment elevation of 1 mm as significant in V_7 through V_9 . When considering ST-segment elevation of ≥ 0.5 mm as significant in V_7 through V_9 , the numbers are 64% (23 of 36 patients) and 78% (28 of 36 patients). In 6% (2 of 36 patients), we observed ST-segment elevation in

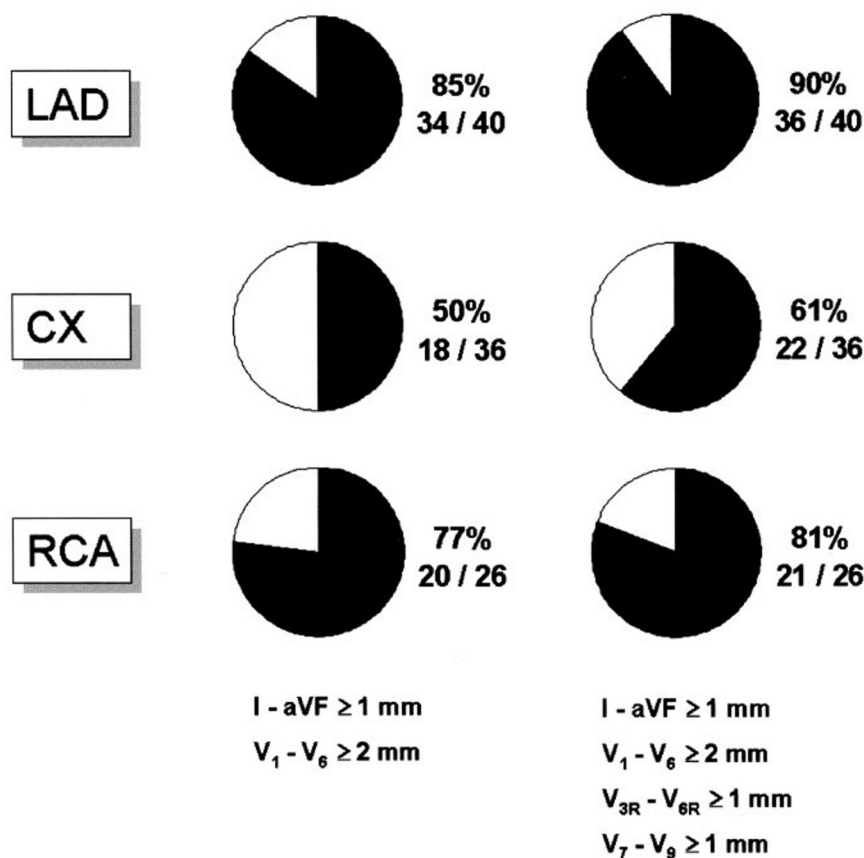


FIGURE 1. Pie charts illustrate sensitivity (in percentage; shaded areas) of contribution for correct diagnosis of AMI of conventional leads I, II, III, aVR, aVL, aVF, and V₁ through V₆ with and without extended leads (V_{3R} through V_{6R} and V₇ through V₉) based on different criteria of ST-segment elevation (≥ 1 mm in at least two contiguous leads in I through aVF plus ≥ 2 mm in V₁ through V₆ and ≥ 1 mm in at least two contiguous leads in V_{3R} through V_{6R} and V₇ through V₉, respectively).

V₃ through V_{6R}. All of these patients had multivessel disease with an occlusion of the RCA. In 36% (13 of 36 patients), we noticed ST-segment elevations in posterior leads. Isolated ST-segment elevation was observed in 3% (1 of 36 patients) in V_{3R} through V_{6R} and in 8% (3 of 36 patients) in V₇ through V₉ in the subgroup of patients in whom ≥ 2 -mm ST-segment elevation in V₁ through V₆ was considered significant. In the subgroup of patients in whom ≥ 1 -mm ST-segment elevation in V₁ through V₆ was considered significant, we observed isolated ST-segment elevation in none in V_{3R} through V_{6R} and in 8% (3 of 36 patients) in V₇ through V₉. Mean maximum CK levels in the subgroup of the 36 patients with ST-segment elevations in the standard leads was 868 ± 601 IU/L, as compared to 942 ± 781 IU/L in the subgroup of patients with additional ST-segment elevation in the extended leads ($p = \text{NS}$).

RCA Occlusion: In RCA occlusion, the standard 12-lead ECG showed relevant ST-segment elevations in 85% (117 of 137 patients) with ≥ 2 mm of ST-segment elevation in leads V₁ through V₆ and in

90% (123 of 137 patients) with ST-segment elevation ≥ 1 mm in V₁ through V₆. In a subgroup of 26 AMI patients with available extended chest leads, the respective numbers are 77% (20 of 26 patients) and 85% (22 of 26 patients). In consideration of these right ventricular and posterior chest leads, the numbers are 81% (21 of 26 patients; Fig 1) and 89% (23 of 26 patients), regardless if 1 mm or 0.5 mm were considered significant in V₇ through V₉. In 62% (16 of 26 patients), we observed significant ST-segment elevation in V_{3R} through V_{6R} and in 31% (8 of 28 patients) of the posterior leads. Isolated ST-segment elevation was noted in 4% (1 of 26 patients) in the right ventricular leads, but in none of the posterior leads. Mean maximum CK levels in the subgroup of 26 patients with ST-segment elevation in the standard leads only was 270 ± 16 IU/L, as compared to 873 ± 581 IU/L in the subgroup of patients with right ventricular infarction ($p = \text{NS}$).

Overall, consideration of the extended ECG leads in the subgroup of 102 patients contributed to the diagnosis of AMI in 2 to 8% of patients, depending

on different criteria of ST-segment elevation. With respect to CX occlusion, the extended chest leads contributed to the diagnosis of AMI in 6 to 14% of patients based on differing ECG criteria of ST-segment elevation in the respective ECG leads.

DISCUSSION

The major finding of the present analysis is that extended precordial ECG leads only marginally enhance the diagnostic sensitivity for AMI in ECG. The ECG diagnosis of CX infarction that had the lowest sensitivity of 50% (72%) in standard ECG leads (depending on the criteria of ST-segment elevation applied) will be improved by 14% (6%). Nevertheless, there was a trend toward an increased infarct size when concomitant ST-segment elevation was present in the extended ECG leads as evaluated by maximum CK levels with infarct involvement of the right ventricle or the posterior wall of the left ventricle. These findings are in keeping with data from previous studies^{14,26,28,32,41,42} reporting comparably low numbers especially for CX infarction. However, the present study is the first to confirm this by determination of the infarct vessel by means of emergency angiography in a larger number of patients. Overall, it should be kept in mind that less rigorous ECG criteria, as they were applied in some of the thrombolysis studies,^{20,25,27,31,41–43} would enhance sensitivity only at the expense of specificity, which was not determined in this study for methodologic reasons.

The value of additional precordial leads for the diagnosis of AMI has been considered differently in various studies.^{41,44,45} The work of Zalenski et al³⁵ observed only a moderate increase in the diagnostic yield of AMI by addition of posterior and right ventricular leads. Most of the studies^{35–38} applied 0.5-mm ST-segment elevation for the posterior leads; however, Chia et al⁴⁰ showed that up to 9% of normal male subjects have 0.5- to 1-mm ST-segment elevation in V₇. Furthermore, for practical reasons, the determination of ST-segment elevation of 0.5 mm seems difficult. Indeed, the most widely used ECG criteria for the conventional precordial leads are 2-mm ST-segment elevation. In the present study, the sensitivity of the conventional ECG in this setting is 50%. Adding the information of the posterior leads (with a minimal ST-segment elevation of 1 mm), the sensitivity will improve by 11%, which represents only 4 patients in our subgroup of 36 patients (Fig 1).

Isolated right ventricular or posterior infarction is a rare finding and is reflected by only 4 to 8% of sole ST-segment elevation in right ventricular and poste-

rior chest leads in the present study, which underscore previous findings^{35–37} with similar numbers. Matetzky et al⁴⁶ pointed to the prognostic implication of concomitant ST-segment changes in the extended posterior chest leads, which stresses the importance of early intervention in this situation. This has already been shown⁴⁷ in the setting of right ventricular involvement in inferior myocardial infarction.

Infarct size, which impacts on prognosis, is reflected by both maximum CK values and ECG alterations, as expressed by the number of leads affected, and the extent of alterations in a given lead.^{27,42,46–49} The former is not available in AMI; consequently, the ECG must serve this purpose. Since there is a relation between infarct size, as expressed in terms of maximum CK values, and number of altered ECG leads during AMI, increasing the number of leads beyond that of a standard ECG should enable estimation of infarct size, on which subsequent therapeutic decisions can be based. However, the difference in maximum CK values in the group with ST-segment elevation in the extended leads as compared to those without ST-segment elevation in each vessel did not reach statistical significance. The remarkably low maximum CK values in the subgroup of patients with RCA occlusion (Table 1) can be explained by a shorter time period between pain onset and the time of ECG recording (3.6 ± 2.9 h vs 5.1 ± 3.1 h).

Nevertheless, the high percentage of electrocardiographically undiagnosed AMI, especially CX occlusion, remains an issue that cannot be satisfactorily addressed by additional posterior ECG. Although extended ECG leads do not contribute substantially to the diagnosis of AMI, in the cohort of patients of the present study the percentage of ECG-undiagnosed CX occlusion was reduced from 50 to 39% (with ST-segment elevations of 2 mm in V₁ through V₆ and 1 mm in V₇ through V₉). Furthermore, additional ST-segment elevations in the extended ECG leads may be of prognostic relevance. As another noninvasive modality of stratifying patients with suspected AMI, detection of wall-motion abnormalities by means of echocardiography may help to minimize the diagnostic hiatus in AMI confirmation, especially in the case of CX occlusion.⁵⁰

In those patients with typical clinical signs of AMI but with nondiagnostic ECG, emergency angiography may be advantageous to confirm the diagnosis and not to postpone therapeutic interventions while awaiting laboratory results. This may help to prevent unwarranted denial or, conversely, potentially harmful administration of thrombolytic therapy.

Study Limitations

Due to the draft of the study that included only patients with angiographically confirmed AMI, calculation of specificity values was precluded. Extended lateral precordial ECG leads were not available in all study participants. The inclusion of patients with reinfarction and of patients with two-vessel and three-vessel disease may have an impact on the ECG diagnosis of AMI. However, it reflects the typical clinical situation. Serial ECG recordings were not obtained, which (in combination with troponin T measurements) may have increased the diagnostic yield in patients with suspected AMI, as recently suggested.⁵¹

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