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Inferior ST Segment Changes During Acute Anterior Myocardial Infarction: A Marker of the Presence or Absence of Concomitant Inferior Wall Ischemia

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The significance of inferior ST segment changes during acute anterior myocardial infarction was studied in 60 patients with acute anterior infarction who had angiographic visualization of the entire distribution of the left anterior descending artery after thrombolytic therapy with streptokinase. In 34 patients (Group 1) this artery supplied the anterior wall of the left ventricle up to or including the apex but did not reach the inferior wall; in 16 patients (Group 2) it continued beyond the apex onto the inferior wall of the left ventricle; and in 10 patients with prior inferior infarction (Group 3) it partially supplied the inferior wall of the left ventricle through collateral channels to an occluded right or dominant circumflex coronary artery.

Consistent with this anatomy, evidence of inferior wall ischemia was significantly more frequent in Groups 2 and 3 than in Group 1 by thallium-201 scintigraphy (91 versus 7%) and by contrast left ventriculography (91 versus 13%). There was no difference in the magnitude of precordial ST segment elevation among the three groups but the inferior ST segment depression was significantly smaller in Groups 2 and 3 with concomitant inferior wall ischemia than in Group 1 (aVF: -0.5 ± 0.7; -0.5 ± 1.0; -1.8 ± 0.8 mm, respectively; $p < 0.001$) with 10 of the 26 patients in Groups 2 and 3 having an elevated or isoelectric ST segment in aVF compared with none of the 34 patients in Group 1 ($p < 0.001$). In patients with inferior ST segment depression, a ratio of ST depression in lead aVF to ST elevation in lead V_2 that was less negative than -0.2 was a reliable marker of concomitant inferior wall ischemia.

The data suggest that the electrocardiogram can identify patients with anterior infarction who have concomitant inferior wall ischemia due to occlusion of a left anterior descending artery that either also supplies the inferior wall or is the source of collateral flow to a previously occluded posterior descending artery in patients with prior inferior infarction.

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During the acute phase of a myocardial infarction, there is frequently "reciprocal" ST segment depression in the electrocardiographic (ECG) leads opposite those presenting the classic pattern of ST segment elevation (1–6). Our studies (7) in patients with acute inferior wall myocardial infarction demonstrated a reciprocal quantitative relation between inferior ST segment elevation and precordial ST segment depression that, in some patients, was distorted by concomitant ischemia of the right ventricle or the lateral wall of the left ventricle. On the basis of these findings, we hypothesized that a quantitative "reciprocal" relation may also exist between anterior ST segment elevation and inferior ST segment depression in acute anterior wall infarction and that this relation may be distorted by concomitant ischemia of the inferior wall of the left ventricle produced by 1) occlusion of a long left anterior descending artery that, in addition to supplying the anterior wall, also partially supplies the inferior wall of the left ventricle, or 2) by occlusion of a left anterior descending artery that contributes collateral flow to the vascular supply of the inferior wall in patients with a prior inferior infarction.

Methods

Study patients. The study group consisted of 60 patients, 45 men and 15 women with a mean age of 59 ± 12

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years (range 31 to 80), with acute anterior myocardial infarction defined as <3 hours of nitroglycerin-resistant chest pain and ST segment elevation in two or more precordial leads from V_1 to V_4 in the absence of bundle branch block. All patients included in this study had undergone successful thrombolytic therapy with intracoronary (n = 16) or intravenous (n = 44) streptokinase with subsequent angiographic visualization of the entire distribution of the left anterior descending artery. The study group comprised two separate cohorts of patients. One cohort included 50 patients without evidence of a prior myocardial infarction and the other cohort included 10 patients with ECG evidence of a prior inferior myocardial infarction who had collateral flow from the left anterior descending artery to an occluded left circumflex (n = 1) or right (n = 9) coronary artery at angiography. Acute myocardial infarction was confirmed in all patients by a diagnostic elevation of the serum creatine kinase, MB fraction (MB CK).

Electrocardiography. One or more standard 12 lead ECGs were recorded at 25 mm/s (1 mV = 10 mm) before administration of streptokinase and the one with the highest anterior precordial ST segment elevations was used for analysis. The magnitude of ST segment elevation or depression was obtained by consensus of two of the investigators who measured it to the nearest 0.5 mm at 80 ms after the J point using the TP segment as the baseline. The anterior (precordial) ST segment elevations were analyzed in three ways: 1) ST segment elevation in lead V_2; 2) the maximal ST segment elevation in leads V_1 to V_4; and 3) the sum of ST segment elevations in leads V_1 to V_4. Inferior ST segment deviations were also analyzed in three ways: 1) ST segment deviation in lead aVF; 2) the maximal ST segment depression in leads II, III or aVF; and 3) the sum of ST segment deviations in leads II, III and aVF.

Coronary angiography and left ventriculography. Multiple view coronary angiography was performed in every patient and contrast left ventriculography in the 30° right anterior oblique projection was performed in 54 of the 60 patients. These studies were obtained within 2 hours of admission in the 16 patients treated with intracoronary streptokinase and within 7 days of admission in the 44 patients who received intravenous streptokinase. All 60 patients had a residual stenosis of the left anterior descending artery ranging from 50 to >90% of the luminal diameter and all 55 patients who underwent ventriculography had an anterior regional wall motion abnormality. The following determinations were made by consensus of two angiographers who were unaware of the results of other investigations: 1) the distribution of the left anterior descending artery from a postreperfusion coronary angiogram, in the right anterior oblique projection; 2) the relation of the site of occlusion or residual stenosis of the left anterior descending artery to the origins of its first septal perforator and first diagonal branches; and 3) the presence or absence of inferior wall regional dysfunction.

Thallium-201 scintigraphy. Rest and 4 hour redistribution thallium-201 scintigraphy in the anterior, 45° left anterior oblique and steep left anterior oblique projections was performed in 51 patients. Thallium was injected intravenously before administration of streptokinase in 40 patients and 16 ± 8 hours after streptokinase in 11 patients. The scintigrams were assessed by consensus of two experienced nuclear cardiologists who were unaware of the results of other investigations.

Definition of groups (Table 1). The 50 patients without prior infarction were classified into two groups according to the pattern of distribution of the left anterior descending artery. *Group 1 comprised 34 patients* whose left anterior descending artery supplied the anterior wall of the left ventricle as far as the apex but did not supply the inferior wall of the left ventricle (Fig. 1A). The absence of inferior wall ischemia was corroborated by the finding of normal inferior wall motion (Fig. 2A) in 28 of the 32 patients who underwent contrast left ventriculography and by normal thallium-201 uptake by the inferior wall (Fig. 3) in 26 of the 28 patients who underwent thallium scintigraphy.

Group 2 comprised 16 patients whose left anterior descending artery continued beyond the apex onto the diaphragmatic surface of the heart to supply ≥25% of the inferior wall of the left ventricle (Fig. 1B). In contrast to Group 1, in Group 2 there was concomitant ischemia of the inferior wall as evidenced by regional dysfunction of the inferior wall (Fig. 2B) in 10 of the 12 Group 2 patients who underwent contrast left ventriculography and the presence of an inferior wall perfusion defect (Fig. 3) in 13 of the 15 Group 2 patients who underwent thallium-201 scintigraphy.

Group 3 comprised the 10 patients with a prior inferior wall myocardial infarction whose left anterior descending artery supplied the inferior wall by visible collateral flow to the posterior descending artery of an occluded left cir-

Table 1. Evidence of Ischemic Involvement of the Inferior Wall of the Left Ventricle in 60 Patients With Anterior Wall Infarction

	Group 1 (n = 34)	Group 2 (n = 16)	Group 3 (n = 10)
Thallium-201 scintigraphy	2 of 28	13 of 15	8 of 8*
Contrast left ventriculography	4 of 32	10 of 12	10 of 10*

*$p < 0.001$ for Group 1 versus Group 2 or Group 3.

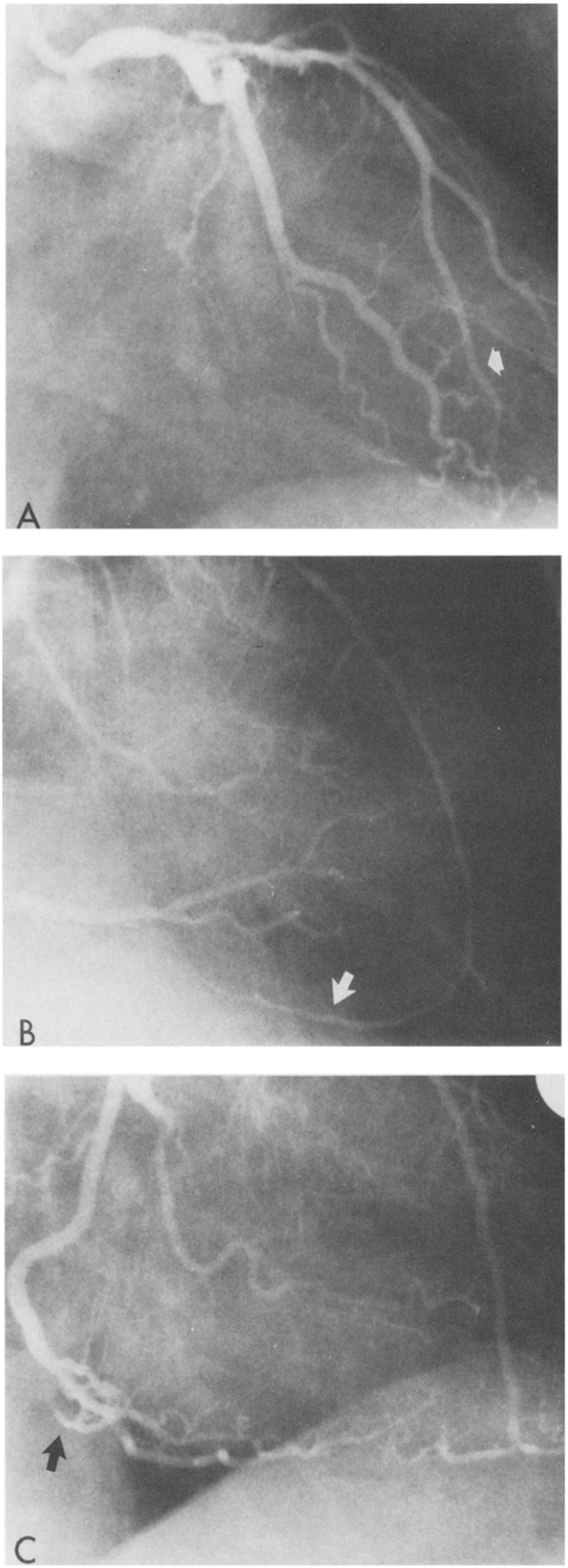

Figure 1. Illustrative angiograms of the left anterior descending artery in the right anterior oblique projection from: (**A**) a Group 1 patient with a short left anterior descending artery (**arrow**) that terminates proximal to the apex of the left ventricle and does not supply the inferior wall, (**B**) a Group 2 patient with a long left anterior descending artery that continues around the apex onto the inferior wall (**arrow**) and (**C**) a Group 3 patient with a left anterior descending artery that supplies the inferior wall through collateral vessels to the posterior descending branch of the previously occluded right coronary artery (**arrow**).

cumflex (n = 1) or right (n = 9) (Fig. 3A) coronary artery. Table 1 indicates that all 10 patients had inferior wall dysfunction (Fig. 3) and all 8 patients who underwent thallium-201 scintigraphy had an inferior wall perfusion defect (Fig. 3C) that in 6 patients was at least partially reversible, indicating an element of inferior wall ischemia in the distribution of the previous inferior infarction.

Hence, in each of the three groups in this study, the distribution of supply of the left anterior descending artery correlated closely with the presence or absence of ischemic involvement of the inferior wall of the left ventricle.

Statistical analysis. Analysis of variance was used for comparing continuous variables in the three groups and pairs of groups were compared using the Bonferroni test. Fisher's exact test was used to assess proportional differences. Correlations between the magnitudes of precordial and inferior ST segment deviations were calculated by the unweighted linear least squares method. Analyses were performed using BMDP biostatistical software with a two-tailed probability (p) value of <0.05 considered to represent statistical significance.

Results

Clinical and angiographic features (Table 2). There was no difference among the three groups with respect to age, sex, interval between onset of symptoms and evidence of reperfusion, peak creatine kinase, MB fraction (MB CK) or relation of the site of the residual stenosis of the left anterior descending artery to the origins of its first septal perforator or first diagonal branch. There was no difference in the extent of coronary artery disease between Groups 1 and 2, but multivessel coronary artery disease and disease of the right coronary artery were universal in Group 3.

Electrocardiographic Findings (Table 3)

Relation to distribution of left anterior descending artery. There was no significant difference among the three groups in precordial ST segment elevation, but inferior ST segment depression was significantly smaller in Groups 2 and 3 than in Group 1, irrespective of which of the three measures of inferior ST segment deviation was used. The ST segment in lead aVF was depressed in all Group 1

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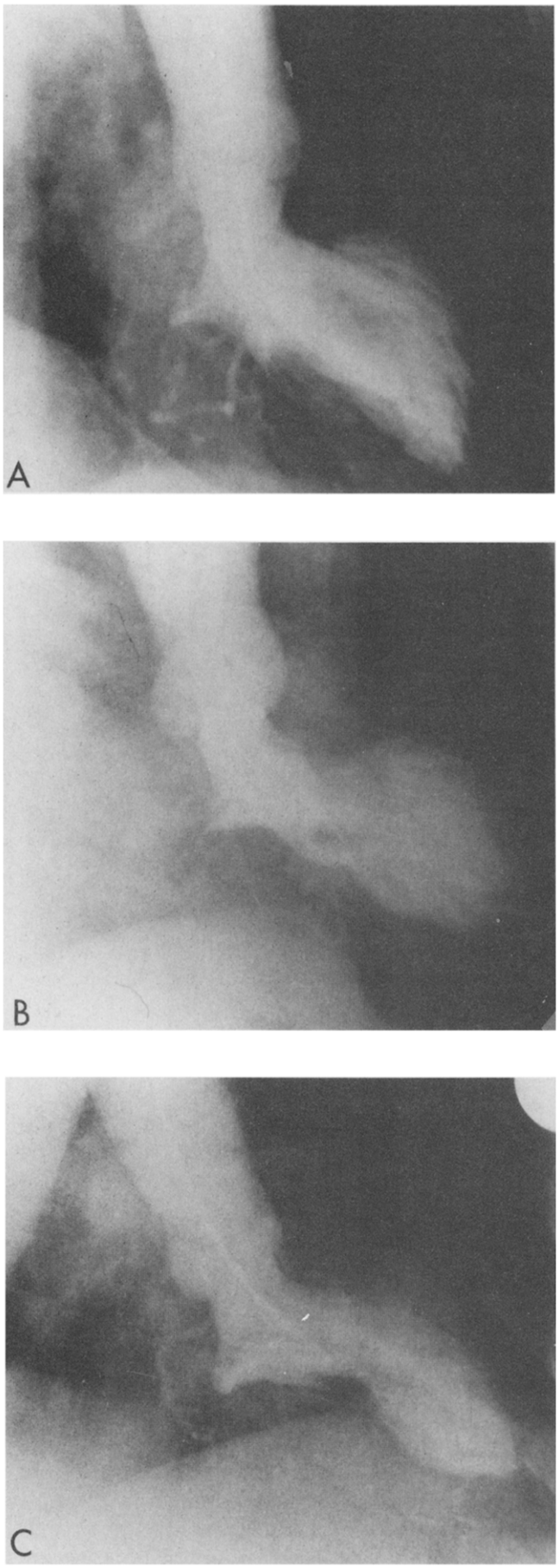


Figure 2. Illustrative end-systolic frame from the contrast left ventriculogram in the right anterior oblique projection from: (**A**) a Group 1 patient, showing normal wall motion of the inferior wall, and (**B**) a Group 2 patient and (**C**) a Group 3 patient, each showing abnormal regional wall motion of both the anterior and the inferior walls.

patients (Fig. 4A), whereas it was isoelectric or elevated in 4 of the 16 Group 2 patients (Fig. 4B) and 6 of the 10 Group 3 patients (Fig. 4C) ($p < 0.001$ for Group 1 versus Group 2 or 3).

Consistent with these findings, the ratio of inferior to precordial ST segment deviations was also significantly less negative in Groups 2 and 3 than in Group 1. The ratio of ST segment deviation in lead aVF to ST segment elevation in lead V_2 was found to be the best ECG discriminator between Group 1 and Groups 2 and 3. This ratio was more negative than -0.2 in 31 of the 34 Group 1 patients compared with 3 of the 16 patients in Group 2 and 1 of the 10 patients in Group 3 (Fig. 5).

For the study group as a whole, there was no significant correlation between the magnitudes of ST segment elevation in lead V_2 and ST segment deviation in lead aVF ($r = -0.20$; $p = 0.13$). However, after exclusion of the 26 Group 2 and Group 3 patients with inferior lead involvement, a separate analysis of the 34 patients in Group 1 who did not have inferior wall involvement revealed a significant inverse or "reciprocal" correlation ($r = -0.47$; $p = 0.005$) between the magnitudes of precordial and inferior lead ST segment changes.

Relation to site of left anterior descending artery occlusion. For each of the three groups, occlusion of the left anterior descending artery proximal to its first diagonal branch or its first septal perforator branch, or both, was associated with greater inferior ST segment depression than that associated with occlusion distal to these two branches, but the site of occlusion did not significantly influence the ratio of inferior to precordial ST segment changes. For the entire study group, ST segment depression in lead aVF was -1.4 ± 1.0 mm in the 48 patients with proximal occlusion of the left anterior descending artery compared with -0.7 ± 0.8 mm in the 12 patients with distal occlusion ($p < 0.05$). Importantly, the site of occlusion did not influence the relation between the distribution of the left anterior descending artery and the ECG findings.

Discussion

Reciprocal inferior ST segment depression during anterior infarction. Our data indicate that elevation of the precordial ST segments in acute anterior myocardial infarction is usually associated with inferior ST segment depression analogous to the reciprocal precordial ST segment depression of acute inferior myocardial infarction

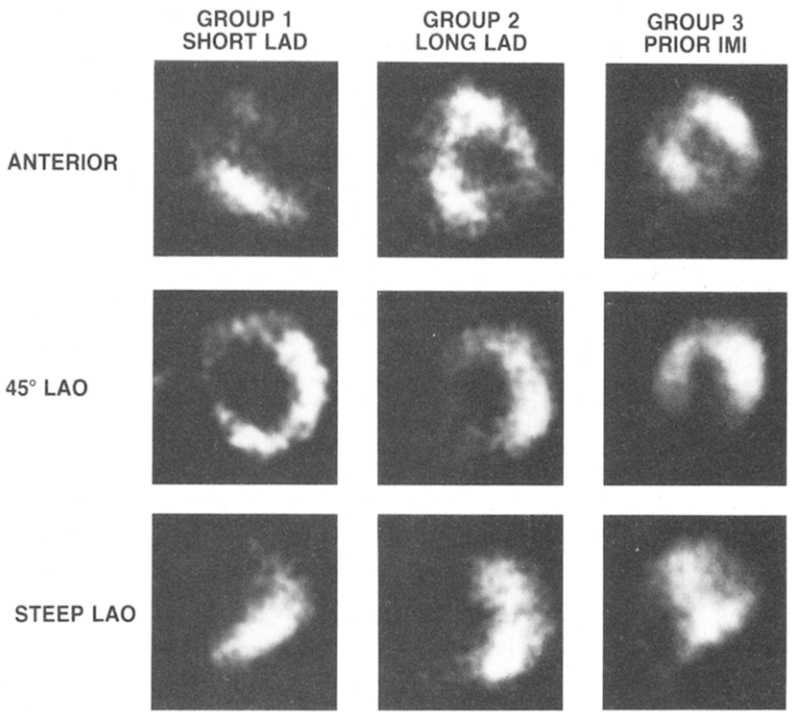


Figure 3. Illustrative "prereperfusion" three-view thallium-201 scintigram from a patient from each of the three groups. The Group 1 patient has an anterior perfusion defect that does not involve the inferior wall, whereas the Group 2 and Group 3 patients each have a perfusion defect involving both the anterior and inferior walls. IMI = inferior myocardial infarction; LAD = left anterior descending coronary artery; LAO = left anterior oblique.

Table 2. Clinical and Angiographic Findings in 60 Patients With Anterior Wall Infarction

	Group 1 (n = 34)	Group 2 (n = 16)	Group 3 (n = 10)
Age (yr)	56 ± 10	61 ± 12	62 ± 15
Male sex (%)	79	69	70
Pain to reperfusion (h)	3.4 ± 1.2	3.5 ± 1.2	3.2 ± 1.3
Peak MB CK (IU/liter)	169 ± 121	167 ± 175	158 ± 71
LAD stenosis proximal to			
First septal perforator branch (%)	65	50	60
First diagonal branch (%)	65	63	70
Three vessel CAD (%)	32	50	100*
RCA stenosis >50% (%)	41	38	100*

*$p < 0.01$ for Group 3 versus Group 1 or Group 2. CAD = coronary artery disease; LAD = left anterior descending coronary artery; RCA = right coronary artery.

Table 3. Electrocardiographic Findings in 60 Patients With Anterior Wall Infarction

	Group 1 (n = 34)	Group 2 (n = 16)	Group 3 (n = 10)
Precordial ST segment elevation (mm)			
ST elevation in lead V_2	4.7 ± 2.1	5.8 ± 3.3	5.8 ± 3.9
Maximal precordial ST elevation	6.7 ± 3.2	6.7 ± 3.2	7.0 ± 4.2
Sum of ST elevations in V_1 to V_4	17.7 ± 8.1	18.3 ± 7.6	18.6 ± 10.7
Inferior ST segment deviations (mm)			
ST deviation in lead aVF	−1.8 ± 0.8	−0.5 ± 0.7	−0.5 ± 1.0*
Maximal inferior ST depression	−2.1 ± 1.1	−0.8 ± 1.0	−0.6 ± 1.0*
Sum of ST deviations in II, III and aVF	−5.2 ± 2.5	1.6 ± 2.6	−1.2 ± 2.7*
Ratio inferior/precordial ST deviations			
Lead aVF/lead V_2	−0.5 ± 0.3	0.1 ± 0.2	0.0 ± 0.2*
Maximal inferior/maximal precordial	−0.4 ± 0.3	−0.1 ± 0.2	0.0 ± 0.1*
Sum of II, III and aVF/sum of V_1 to V_4	−0.3 ± 0.2	0.1 ± 0.1	0.0 ± 0.1*

*$p < 0.001$ for Group 1 versus Group 2 or Group 3. There were no significant differences between Group 2 and Group 3.

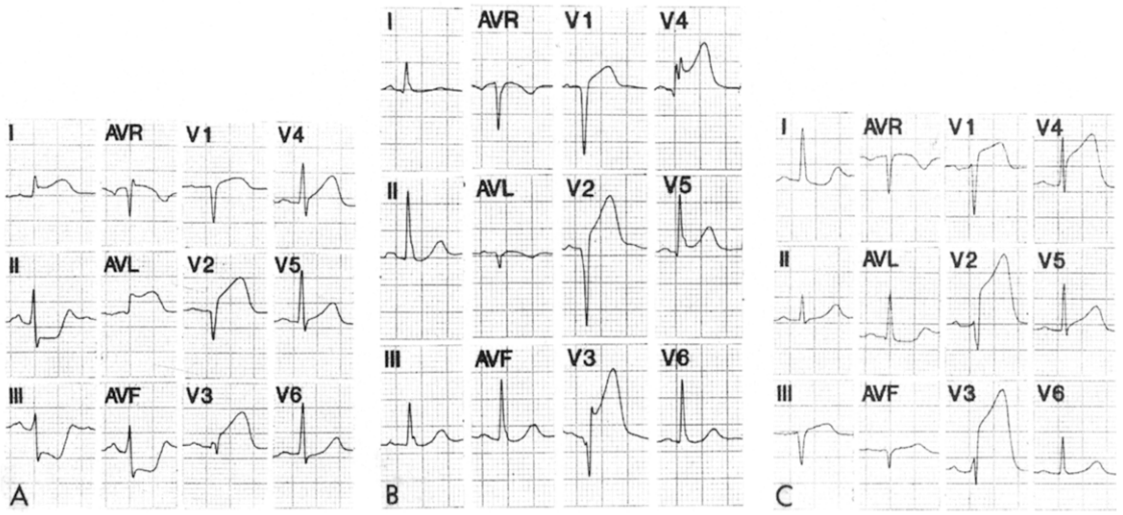
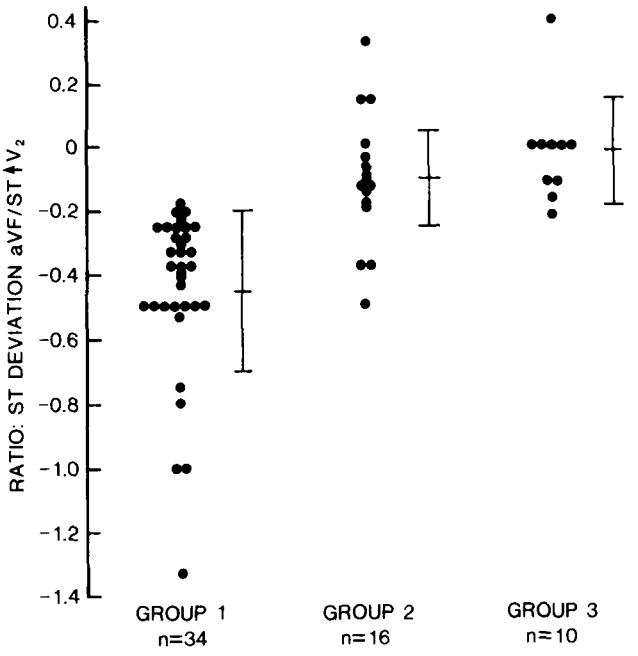


Figure 4. Illustrative electrocardiogram (ECG) from: (**A**) a Group 1 patient in whom the reciprocal inferior ST segment depression is of a similar magnitude to the precordial ST segment elevation, (**B**) a Group 2 patient with precordial ST segment elevation with isoelectric inferior ST segments and (**C**) a Group 3 patient with abnormal inferior Q waves due to a prior inferior infarction and ST segment elevation in the inferior and precordial leads.

(2,5–7). Because greater anterior ST segment elevation tends to cause greater inferior ST segment depression, inferior ST depression during acute anterior infarction is generally indicative of more severe anterior wall ischemia (8). The finding that inferior ST depression was most evident in patients whose left anterior descending artery supplied only the anterior wall of the left ventricle up to or including the apex but not the inferior wall, together with the absence of evidence of inferior wall ischemia or dysfunction in this group of patients, supports the conclusion that inferior ST depression during anterior infarction is usually due to a reciprocal effect and not to ischemic involvement of an additional area of the left ventricle.

Influence of concomitant inferior ischemia on the ECG. Patients whose distribution of the occluded left anterior descending artery included part of the inferior wall had inferior lead ST segments that were elevated or isoelectric or had ST segment depressions that were small relative to the magnitude of the precordial ST segment elevation. We attribute the attenuation or reversal of the reciprocal inferior ST segment depression in these patients to concomitant transmural ischemia of part of the inferior wall that tended to elevate the inferior ST segments and thereby partially or completely neutralize or reverse the reciprocal effect of the precordial ST segment elevation. Such attenuation was also observed in patients with a prior inferior infarction whose left anterior descending artery was the source of collateral flow to a previously occluded left circumflex or right coronary artery (Group 3). The contention that, in these patients, occlusion of the left anterior descending artery resulted in acute ischemia of the inferior wall was supported by the partial reversibility of the thallium-201 perfusion defect in six of the eight patients studied in this group. Because the subendocardium undergoes necrosis first and the subepicardium last (9), it is plausible that in these patients with a prior inferior

Figure 5. Graphic representation comparing the ratio of ST segment deviation in lead aVF to ST segment elevation in lead V_2 in the three groups of patients. A ratio of −0.2 provides a good separation of Group 1 from Groups 2 and 3 but the latter two groups have a similar distribution of values.



myocardial infarction, the remaining viable myocardium supplied by the collateral flow was subject to ischemia predominantly in the subepicardium. This would explain the tendency of inferior ischemic involvement to attenuate the reciprocal ST segment depression because acute subepicardial ischemia tends to elevate the ST segment (10), whereas subendocardial inferior ischemia would have tended to further depress the inferior ST segment.

Our findings are consistent with those of Myers et al. (11) who, in their classic report, correlated the ECG with the pathologic findings in patients with anterior myocardial infarction. They found that the reciprocal inferior ST segment depression in lead aVF that was usually present during an acute transmural anterior infarction was absent when the acute anterior infarction continued around the apex to involve the posterior (inferior) wall of the left ventricle or when there was a prior infarction of the posterior wall.

Therefore, in contrast to the reciprocal influence that tends to depress the inferior ST segments in anterior wall infarction, concomitant ischemia of part of the inferior wall appears to produce an inferiorly directed component to the current of injury and thereby tends to elevate the inferior ST segments. This influence alters the relation between the anterior and the inferior ST segment deviations such that an elevated or isoelectric inferior ST segment or ST segment depression that is small relative to the magnitude of anterior ST segment elevation is indicative of concomitant inferior ischemia. In this study, an ST segment in lead aVF that was elevated, isoelectric or depressed <20% of the ST segment elevation in lead V_2 was a good ECG marker of the presence of concomitant inferior wall ischemia and, hence, was suggestive of an additional area of jeopardized left ventricle. It is not clear why the group with concomitant inferior wall ischemia did not evolve a higher peak MB CK than that of patients without inferior wall involvement, but this may reflect the influence of early reperfusion in this study. Because the inferior wall received perfusion from both the left and right coronary arteries, it may have undergone slower and more limited necrosis than the anterior wall and thereby benefited more from early reperfusion such that its contribution to the final infarct size was diminished. However, it is conceivable that in the absence of reperfusion the inferior wall involvement may have contributed to the infarct size.

Influence of site of left anterior descending artery occlusion. Occlusion of the proximal left anterior descending artery also tended to increase the inferior ST segment depression. Although the size of the study group precluded discrimination between the influences of the diagonal and the septal perforator branches, the augmented inferior ST segment depression in this group was presumably due to more extensive lateral wall ischemia in the distribution of the diagonal branches (11). Of interest, proximal left anterior descending artery occlusion was also associated with a higher peak MB CK than was distal occlusion (184 ± 134 versus 94 ± 77 IU/liter; $p < 0.05$).

Potential study limitations. As discussed previously, all the patients in this study underwent coronary reperfusion by early thrombolytic therapy and, therefore, the data cannot be related to the natural history with respect to infarct size or prognosis. In addition, it is theoretically possible that reperfusion interfered with the assessment of left ventricular function and the interpretation of the 11 postreperfusion thallium scintigrams. However, the excellent concordance between the distribution of the left anterior descending artery and the assessment of inferior involvement suggests that, at least qualitatively, the data reliably reflect the physiology at the time of occlusion. Furthermore, exclusion of the 11 patients with postreperfusion thallium data would not alter the conclusions because nine of the studies were from Group 1 patients, of whom 7 had no inferior wall perfusion defect, and in each of the single studies from Group 2 and Group 3, respectively, the patient had an inferior wall defect.

Conclusion. This study indicates that the inferior ST segment changes that occur during an acute anterior myocardial infarction provide an insight into the presence or absence of concomitant ischemia of the inferior wall of the left ventricle and the distribution of the left anterior descending artery.

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