

# Implications of Inferior ST-Segment Elevation Accompanying Anterior Wall Acute Myocardial Infarction for the Angiographic Morphology of the Left Anterior Descending Coronary Artery Morphology and Site of Occlusion

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**Inferior ST-segment elevation during anterior wall acute myocardial infarction (AMI) due to left anterior descending (LAD) coronary artery occlusion is unusual and was not previously investigated. This study tested the hypothesis that inferior ST-segment elevation during anterior AMI predicts a specific angiographic morphology that satisfies 2 necessary conditions: (1) mass of ischemic anterior wall myocardium is relatively small, resulting in a weaker anterior injury current and less reciprocal inferior ST-segment depression; and (2) there is concomitant inferior wall transmural ischemia that further shifts the inferior ST segments upward.**

**The study group consisted of 42 consecutive patients with anterior AMI undergoing angiography at 4.1 days (range 0 to 14). Coronary angiograms were examined for 3 features: (1) site of LAD artery occlusion (a distal obstruction implying a smaller mass of ischemic anterior wall myocardium), (2) LAD artery extension onto inferior wall of left ventricle (termed a "wrap around" vessel), and (3) collateral flow from LAD artery to inferior wall. The latter 2 features would be expected to contribute to inferior wall transmural ischemia.**

**Acute inferior ST-segment elevation (sum of ST-segment deviation in leads II, III and aVF  $\geq 3.0$  mm) was seen in 7 patients (16%). A greater number of LAD artery branches proximal to the site of occlusion was significantly correlated with less inferior ST-segment depression ( $r = 0.59$ ,  $p < 0.01$ ). The 33 patients with an LAD artery that "wrapped around" the cardiac apex to the inferior wall had less total inferior ST-segment depression than that of the 9 with a shorter artery ( $0.2 \pm 4.0$  vs  $-3.0 \pm 2.8$  mm;  $p < 0.05$ ). Of the 7 patients with inferior ST-segment elevation, 5 had a wrap around artery with the culprit lesion distal to 3 major branches. This combination of angiographic findings occurred in only 2 of the other 35 pa-**

**tients (odds ratio 44, 95% confidence interval 4 to 632;  $p < 0.001$ ). These results support the hypothesis that inferior ST-segment elevation during anterior AMI results from a smaller mass of ischemic anterior myocardium combined with simultaneous inferior transmural ischemia (i.e., a distal occlusion in a wrap around LAD coronary artery). (Am J Cardiol 1992;69:860-865)**

The hallmark of transmural acute myocardial infarction (AMI) is ST-segment elevation on the body surface electrocardiogram. This is often accompanied by reciprocal ST-segment depression in leads oriented opposite to those displaying ST-segment elevation.<sup>1</sup> Reciprocal ST-segment depression is believed to occur because of the orientation of the 2 groups of electrocardiographic leads to the injury current dipole and may be further influenced by the presence of myocardial ischemia in areas remote from that undergoing AMI.<sup>2</sup> Whereas reciprocal inferior ST-segment depression has been extensively studied, little information exists concerning the incidence, cause and clinical significance of simultaneous anterior and inferior ST-segment elevation in the setting of anterior AMI (termed "anterior-inferior" AMI).<sup>3-6</sup> In one study it was shown that when  $\geq 25\%$  of the inferior wall of the left ventricle was supplied by the left anterior descending (LAD) coronary artery (either directly or by collateral flow) there was significantly less acute inferior ST-segment depression.<sup>3</sup> It was reasoned that this anatomic feature caused simultaneous inferior wall transmural ischemia, producing an injury current directed toward leads II, III and aVF, which attenuated the reciprocal ST-segment depression caused by the anterior wall injury current.

To further explain the cause of the anterior-inferior AMI pattern, we propose that a second condition is necessary in addition to the occurrence of transmural ischemia in part of the inferior wall. This condition is that the occlusion of the LAD coronary artery be located in the distal vessel. This results in a smaller mass of ischemic anterior wall myocardium that in turn generates a weaker anteriorly directed injury current and less reciprocal ST-segment depression, allowing the inferior ST segments to move further upward. It is the combination

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of these 2 circumstances that permits frank inferior ST-segment elevation to become manifest. Therefore, the purpose of this study was to: (1) determine the incidence of the anterior-inferior AMI electrocardiographic pattern, and (2) test the hypothesis that this distinctive electrocardiogram predicts a specific angiographic morphology—distal occlusion of an LAD coronary artery that extends around the apex to supply part of the inferior wall.

## METHODS

**Patient selection:** From a review of the cardiac catheterization laboratory records at the University of North Carolina Hospital between November 1988 and November 1990 we identified 70 consecutive patients undergoing cardiac catheterization within 14 days of an anterior AMI or an episode of prolonged transmural anterior myocardial ischemia. Anterior AMI was defined by (1) ischemic chest pain of  $\geq 2$ -hour duration, (2) ST-segment elevation  $\geq 2$  mm (1 mm = 0.1 mv) in  $\geq 3$  of leads V<sub>2</sub>–V<sub>5</sub> of a standard 12-lead electrocardiogram, and (3) subsequent elevation in the creatine phosphokinase-MB isoenzyme. Patients (n = 3) meeting the first 2 criteria, but without subsequent enzyme elevations were considered to have had prolonged transmural ischemia without AMI. They were included in the study group, because it was thought that their acute electrocardiograms reflected the same pathophysiology as those of subjects who had subsequent enzyme elevations.

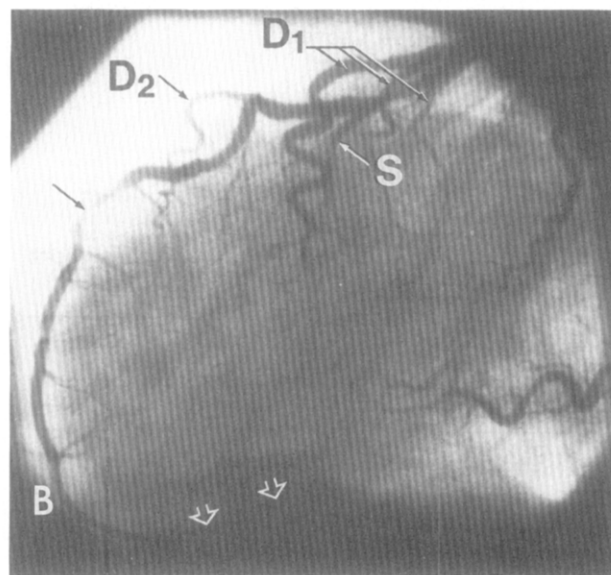
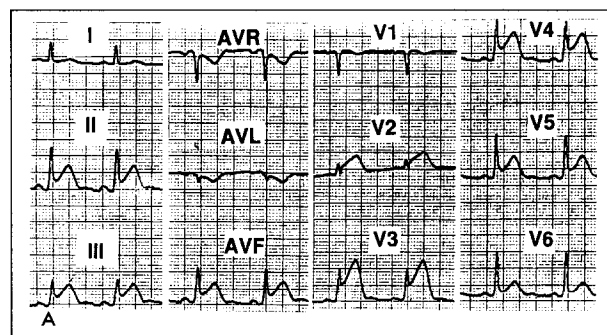
All medical records were reviewed to obtain an electrocardiogram performed within 6 hours of the onset of chest pain and within 2 hours of the initiation of thrombolytic therapy or before percutaneous transluminal coronary angioplasty. In 20 patients who had been transferred from other hospitals, an appropriately timed electrocardiogram was not available, and they were excluded. Other reasons for exclusion included bundle branch block (n = 3), inability to fully visualize the terminal LAD coronary artery (n = 3), and a non-LAD artery infarct (occlusion of diagonal or ramus intermedius branches; n = 2). The remaining 42 patients constituted the study group.

**Analysis of electrocardiograms:** In patients with  $>1$  available tracing, the one with the greatest magnitude of precordial ST-segment elevation was analyzed. The magnitude of ST-segment deviation relative to the TP segment in each of the 12 standard leads was measured to the nearest 0.5 mm at 80 ms after the J point. Measurements obtained by 2 independent observers unaware of all clinical and angiographic information were averaged for each lead. Inferior lead ST-segment deviation was expressed as the sum of ST-segment deviation in leads II, III and aVF, with a positive value representing net ST elevation, and a negative one, net ST-segment depression.

The anterior-inferior AMI pattern was defined as predominantly anterior precordial lead ST-segment elevation accompanied by summed ST-segment elevation in leads II, III and aVF of  $\geq 3$  mm. This definition was chosen because the mean of 1 mm of ST-segment elevation in each inferior lead is consistent with generally

accepted electrocardiographic criteria signifying inferior wall transmural ischemia (Figure 1A).

**Analysis of coronary angiograms:** Coronary angiograms were read in a consensus fashion by 2 investigators unaware of the electrocardiographic findings. Multiple projections of the LAD coronary artery were examined for 3 features. First, the LAD artery was classified as terminating at the cardiac apex or as extending around the apex to supply part of the inferior wall. The cardiac apex was defined in the lateral view by the point at which the artery assumed a course perpendicular to the midportion of the vessel. A vessel extending beyond the apex to an arbitrarily determined point on the inferior apex was termed a wrap around artery (Figure 1B). This dichotomizing point was assessed visually, but represents a true distance of approximately 1.7 cm, determined by comparison with angiographic catheter diameter in the same frame. In 6 patients with totally occluded LAD arteries, this feature was assessed by collateral flow to the vessel. Second,



**FIGURE 1. A, electrocardiogram of patient with acute anterior myocardial infarction showing characteristic precordial lead ST-segment changes, as well as inferior lead ST-segment elevation ("anterior-inferior" myocardial infarction). B, lateral projection of left anterior descending coronary artery in same patient 3 days after infarction. Unlabeled solid arrow indicates distal subtotal occlusion. Double open arrows indicate terminal portion of vessel on inferior wall of left ventricle. In this case, angiographic score (see text) is 4. D<sub>1</sub> = branching first diagonal vessel; D<sub>2</sub> = second diagonal vessel; S = first septal perforating artery.**

the number of patent branches of the LAD artery, which originated proximal to the lesion responsible for the infarct, was enumerated. There were 4 important branches considered: (1) ramus intermedius (if present), (2) first large septal perforating vessel, (3) first diagonal vessel and (4) second diagonal vessel. The site of the infarct-related lesion was determined by the location of a total occlusion or by the stenosis with the greatest reduction in luminal diameter by visual estimate. Finally, collateral flow from the LAD coronary artery to the inferior wall, which originated distal to the culprit lesion, was classified as present or absent.

**Angiographic score:** An angiographic scoring system was devised for the purpose of evaluating the 3 angiographic features proposed to cause the anterior-inferior AMI pattern. The scoring system was designed so

that a low score was associated with the angiographic features predicted to cause more inferior ST-segment depression, and a higher score with those predicted to cause inferior ST-segment elevation.

**MASS OF ISCHEMIC ANTERIOR WALL MYOCARDIUM:** It was reasoned that if the site of occlusion in the LAD artery was located distally there would be more proximal branch vessels patent to perfuse the anterior wall, and thus a lesser volume of ischemic anterior wall myocardium. This would generate a smaller anterior wall injury current and less reciprocal inferior ST-segment depression. Therefore, 1 point was allowed for each important branch of the LAD artery proximal to the site of occlusion, to a maximum of 3 points. A score assigning only  $\frac{1}{2}$  point per vessel was also tested.

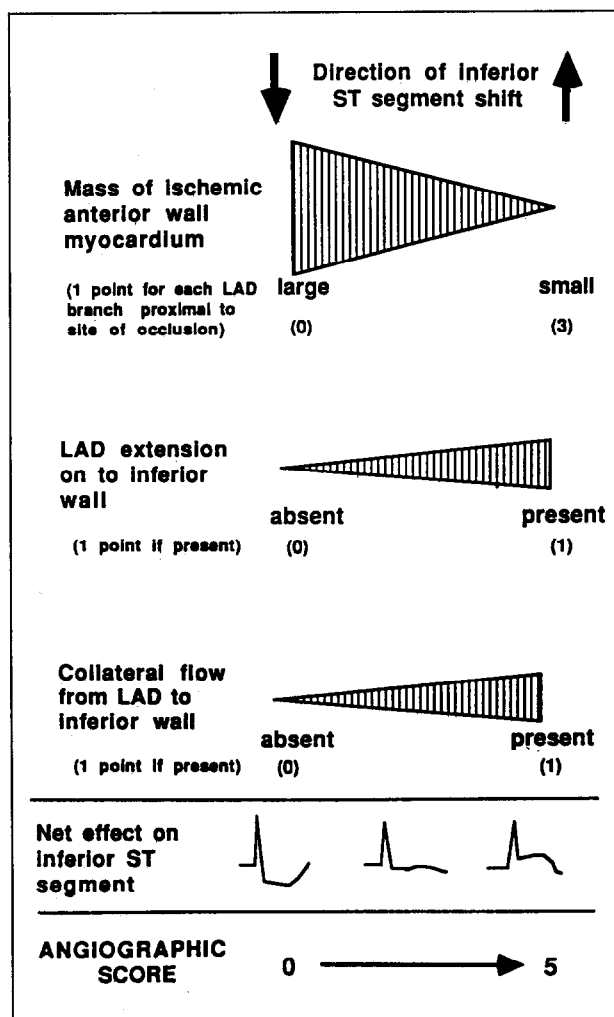
**LEFT ANTERIOR DESCENDING CORONARY ARTERY EXTENDING ONTO THE INFERIOR WALL:** Occlusion of a wrap around LAD artery could result in some inferior wall transmural ischemia and attenuate the reciprocal ST-segment depression expected to result from anterior wall transmural ischemia. Therefore, 1 point was given for a wrap around LAD coronary artery that supplied part of the inferior wall.

**COLLATERAL FLOW FROM THE LEFT ANTERIOR DESCENDING CORONARY ARTERY TO THE INFERIOR WALL:** If the inferior wall was supplied by collaterals from the LAD coronary artery, 1 point was given. In this case, an LAD coronary artery occlusion would be more likely to produce inferior wall transmural ischemia, thereby shifting the inferior lead ST segment upward. The scoring system is depicted graphically in Figure 2.

**Statistical analysis:** Data are expressed as mean  $\pm$  1 standard deviation. The relation between continuous and noncontinuous variables was assessed by the Spearman rank-order correlation method. Linear regression was used to compare 2 continuous variables, and the differences between 2 groups of continuous variables were assessed by a 2-tailed Student's *t* test. The independent contribution of LAD artery extension to inferior ST-segment deviation was assessed by converting the number of vessels proximal to the site of occlusion to a categorical variable and then performing multiple variable regression. The occurrence of the anterior-inferior AMI pattern and its relation to angiographic morphology were evaluated by a chi-square analysis and Fisher exact test. A *p* value  $<0.05$  was considered significant.

## RESULTS

**Patient characteristics:** The study group consisted of 33 men and 9 women (mean age  $57 \pm 12$  years). There was history of AMI in 7 patients (inferior, *n* = 2; anterior, *n* = 1; non-Q-wave, *n* = 1; and indeterminate, *n* = 3). Patients underwent cardiac catheterization and coronary angiography a mean of  $4.1 \pm 3.7$  days after AMI. In all but 1 patient, the residual stenosis of the lesion thought responsible for the AMI was  $\geq 50\%$ . This patient was included in the study because the stenosis (30%) was the only coronary artery lesion identified.<sup>7</sup> Mean left ventricular ejection fraction was  $0.49 \pm 0.13$  (range 0.23 to 0.72), with anterior wall segmental dys-



**FIGURE 2.** Derivation of angiographic score. Each of 3 angiographic features postulated to influence inferior ST segment are shown, with magnitude of effect on inferior ST segment represented graphically. Mass of ischemic myocardium was postulated to be inversely related to number of branches proximal to site of left anterior descending (LAD) occlusion, and 1 point was given for each proximal branch (maximum 3 points). One point was given for LAD that supplied part of inferior wall, and 1 point for LAD that supplied collateral flow to inferior wall.

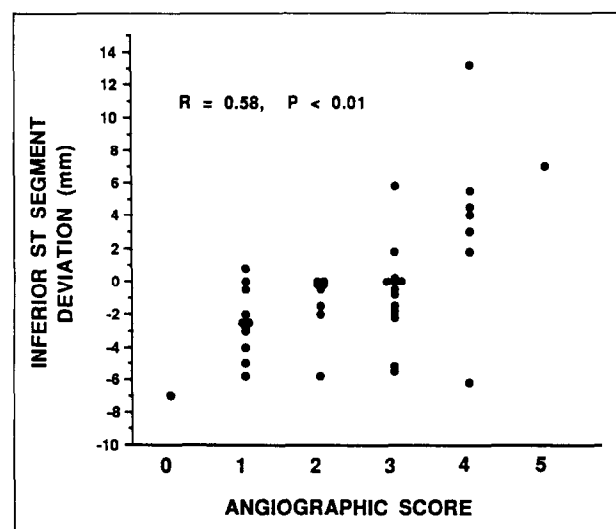
function noted in 33 of 35 patients undergoing left ventriculography.

**"Anterior-inferior" myocardial infarction:** An anterior-inferior AMI pattern was present in 7 of 42 patients (16%), and in these there was a significantly greater incidence of a specific LAD coronary artery angiographic morphology. The combination of an LAD artery occlusion located beyond 3 major branches in a vessel that "wrapped around" the cardiac apex to supply part of the inferior wall of the left ventricle, was seen in 5 of 7 patients with the electrocardiographic pattern of anterior-inferior AMI. In contrast, only 2 of 35 patients without this electrocardiographic pattern had both angiographic features (odds ratio 44, 95% confidence interval 4 to 632;  $p < 0.001$ ). The other 2 patients with anterior-inferior AMI electrocardiograms had wrap around LAD arteries with 2 patent vessels proximal to the occlusion. Patients with and without the anterior-inferior AMI electrocardiogram had similar values for the sum of ST-segment elevation in leads  $V_2$ - $V_6$ , I and aVL ( $19 \pm 5$  vs  $20 \pm 13$  mm).

#### Factors influencing inferior ST-segment deviation:

There was a significant relation ( $r = 0.58$ ;  $p < 0.01$ ) between angiographic score and sum of ST-segment deviation in leads II, III and aVF, such that a lower score was accompanied by more inferior ST depression, and a higher one by inferior ST-segment elevation (Figure 3). Similar results ( $r = 0.52$ ) were obtained when the contribution to the angiographic score of vessels proximal to the occlusion was decreased by 50%.

Each element of the angiographic score was analyzed separately. Inferior ST-segment depression was significantly less in patients ( $n = 33$ ) whose LAD coronary artery extended around the apex to supply part of



**FIGURE 3.** Inferior ST-segment changes in relation to angiographic score in each patient. Ordinate indicates sum of ST-segment deviation in leads II, III and aVF, with positive values representing net ST-segment elevation, and negative values net ST-segment depression. Relation persists ( $r = 0.52$ ,  $p < 0.001$ ) even when 2 highest and lowest values are excluded or when each patent proximal vessel contributes  $\frac{1}{2}$  point (rather than 1) to angiographic score ( $r = 0.52$ ).

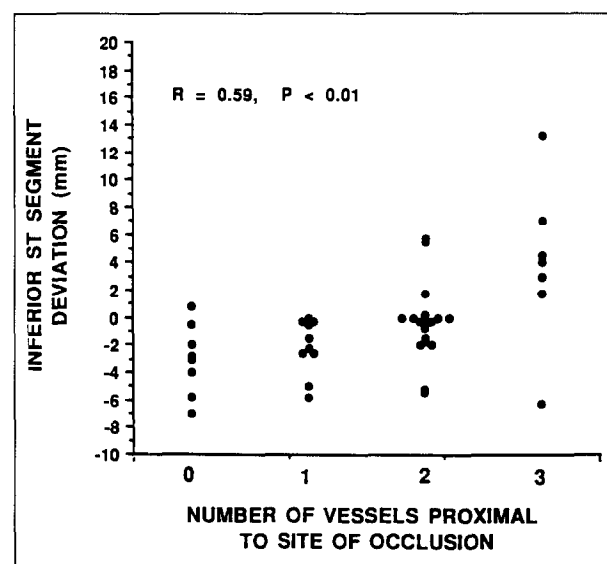
the inferior wall compared with that of those ( $n = 9$ ) in whom the vessel terminated at or before the cardiac apex ( $0.2 \pm 4.0$  vs  $-3.0 \pm 2.8$  mm;  $p < 0.05$ ). There was a significant correlation ( $r = 0.59$ ;  $p < 0.01$ ) between the number of branch vessels proximal to the site of the LAD coronary artery occlusion and the net inferior ST-segment deviation (Figure 4). There were only 5 patients with collateral flow from the LAD coronary artery to the inferior wall of the left ventricle. Although the mean inferior ST-segment depression was less in these patients than in the 37 without such collateral flow ( $-0.2 \pm 6.7$  vs  $-0.6 \pm 3.7$  mm), this difference was not statistically significant. All results were similar when patients with history of AMI were excluded from the analysis.

Because the number of patent vessels proximal to the site of the LAD artery occlusion appeared to be a powerful predictor of inferior ST-segment deviation, multiple regression analysis was performed to determine if the anatomic extent of the LAD artery had an independent influence on the inferior ST segment. In this analysis, the independent effect of a wrap around LAD artery was of borderline statistical significance ( $p = 0.07$ ) when the site of occlusion was taken into account.

#### Creatine phosphokinase-MB isoenzyme levels:

In the 38 patients with  $\geq 1$  value above the normal range (no measurement was obtained in 1 patient), a greater number of perfused LAD artery branches proximal to the occlusion was associated with lower peak creatine phosphokinase-MB isoenzyme levels ( $r = -0.39$ ;  $p < 0.05$ ) (Figure 5).

There were 9 patients whose LAD coronary artery did not wrap around the cardiac apex. In this group, one would expect only the anterior wall, and not a portion of the inferior wall, to have ischemia and AMI. These pa-



**FIGURE 4.** Inferior ST-segment changes in each patient in relation to location of the left anterior descending artery occlusion. Ordinate is as in Figure 2. Abscissa indicates number of major branch vessels (see text) proximal to occlusion.

tients displayed a significant positive correlation between creatine phosphokinase-MB levels and the magnitude of inferior ST-segment depression ( $r = 0.69$ ;  $p < 0.05$ ) (Figure 6).

## DISCUSSION

**Prior studies relating to inferior ST-segment elevation in anterior myocardial infarction:** Studies of the occurrence and clinical significance of reciprocal ST-segment depression in AMI have supported 2 concepts.<sup>2</sup> First, reciprocal ST-segment depression is often associated with more ischemic or damaged myocardium in the infarct region, and thus represents a purely electrical phenomenon determined by the presence and magnitude of the injury current dipole. Second, many patients with reciprocal ST-segment depression have critical coronary artery stenoses and segmental dysfunction in myocardial regions remote from that of the AMI, suggesting that "ischemia at a distance" is also an important contributor to reciprocal ST-segment depression. Subendocardial rather than transmural ischemia is assumed to exist in the remote zone, because the former would be expected to produce ST-segment depression.<sup>8</sup>

In contrast, transmural myocardial ischemia could coexist in both the primary infarct zone and remote areas. In the case of anterior AMI, concomitant inferior transmural ischemia should cause the inferior ST segments to shift upward rather than downward. It was previously shown that patients with an anterior AMI and wrap around LAD coronary artery have less inferior ST-segment depression,<sup>3</sup> and a few apparently had inferior ST-segment elevation. There is little additional information concerning the anterior-inferior AMI electrocardiographic pattern. One recent case report described a patient with simultaneous acute anterior and inferior ST-segment elevations who had occlusion of an LAD coronary artery that supplied one third of the in-

ferior wall.<sup>4</sup> Another study described several patients with 1-vessel disease involving a wrap around LAD artery who displayed both anterior and inferior Q waves.<sup>5</sup> Finally, an older autopsy study reported that when anterior AMI was accompanied by a histologically simultaneous "posterior" AMI, the reciprocal ST-segment depression "commonly" seen in lead aVF was not found in any of 19 cases.<sup>6</sup>

**Further insight into the "anterior-inferior" infarction electrocardiogram:** Pathoanatomic reports suggest that most patients have a wrap around LAD coronary artery,<sup>9</sup> and in our study, 33 of 42 patients had this anatomy. Our study attempts to explain why only 7 of the 33 patients demonstrated the anterior-inferior AMI electrocardiographic pattern. Patients with an LAD coronary artery that was occluded proximally and did not extend to the inferior wall had the most inferior ST-segment depression. Presumably these patients had a larger mass of ischemic anterior wall myocardium and were least likely to have inferior wall transmural ischemia. The 7 patients with the anterior-inferior AMI electrocardiographic pattern were distinguished by the distal location of the LAD coronary artery occlusion (beyond 2 major branches in 2 patients, and beyond 3 branches in the other 5). We postulate that the inferior wall injury current must be combined with the weak reciprocal of a smaller anterior injury current to generate the unusual acute anterior-inferior AMI pattern on the 12-lead electrocardiogram.

An important angiographic feature of the LAD artery related to the magnitude and direction of inferior ST-segment deviation was the number of branches proximal to the occlusion. That this feature is related to infarct size is supported by observations in an animal model,<sup>10</sup> postmortem human hearts<sup>11</sup> and patients with AMI.<sup>3</sup> The relation of infarct size to the magnitude of the anteriorly directed injury current and its reciprocal is also seen in experimental data<sup>12</sup> and clinical stud-

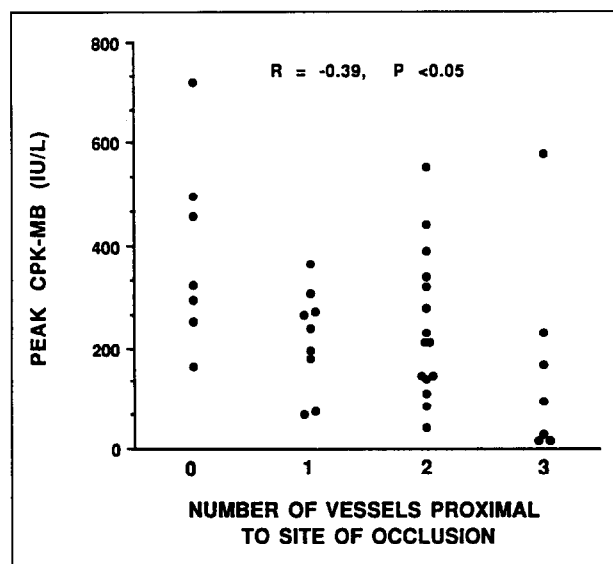


FIGURE 5. Peak creatine phosphokinase (CPK)-MB level for each patient with abnormal elevation after prolonged chest pain in relation to location of left anterior descending artery occlusion (*abscissa* as in Figure 4).

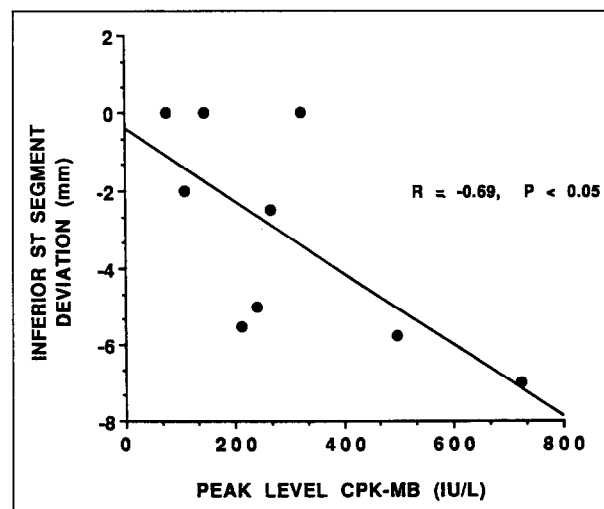


FIGURE 6. Relation between reciprocal inferior ST-segment changes (*ordinate* as in Figure 2) and peak creatine phosphokinase (CPK)-MB (*abscissa* as in Figure 5) in patients with left anterior descending artery that did not extend beyond cardiac apex.

ies.<sup>13-20</sup> Simultaneous anterior and inferior ST-segment elevation can also be seen in the setting of right coronary artery occlusion.<sup>21</sup> However, in this case, leads V<sub>1</sub> and V<sub>2</sub> have greater ST-segment elevation than do V<sub>3</sub> and V<sub>4</sub>, a pattern not seen in any patient in this study.

**Study limitations:** Several methodologic considerations could have affected the results of this study. First, angiographic recognition of the lesion responsible for AMI can be difficult when angiography is performed several days after AMI. In some patients there can be a rapid, marked decrease in the degree of coronary narrowing after occlusion and reperfusion, leaving a residual stenosis of <50%.<sup>7</sup> Second, the use of angiography to estimate the volume of myocardium affected by a coronary artery occlusion is not precise, and the use of cardiac isoenzyme levels to estimate infarct size is hampered by variations in sampling times and therapeutic interventions such as thrombolytic agents, percutaneous transluminal coronary angioplasty and other medical therapies. Other methods of assessing infarct size may have yielded a better correlation between infarct size and reciprocal ST-segment depression. Third, we did not confirm the presence of inferior wall ischemia in patients with a wrap around LAD artery. However, an earlier study did show similar coronary anatomy to be associated with an inferior wall thallium defect in 13 of 15 patients.<sup>3</sup> Finally, this study does not take into account other factors known to influence the magnitude of ST-segment deviation during coronary occlusion. These factors include infarct shape and boundary location, presence of subendocardial as opposed to transmural myocardial ischemia, prior tissue infarction, local metabolic factors and time course of cellular electrical uncoupling.<sup>8,22</sup> However, there is no reason to assume that any of these methodologic considerations biased the results of the study.

**Clinical implications:** In some situations therapy may be influenced by the identification of a lesion in the proximal versus the distal LAD coronary artery. Studies of patients with stable coronary disease have suggested that proximal LAD coronary artery disease may cause greater ventricular dysfunction during exercise,<sup>23</sup> a lower ejection fraction after AMI,<sup>24-25</sup> and a more adverse long-term prognosis<sup>25-26</sup> than may distal disease. Our study shows that despite the electrocardiographic appearance of more widespread ischemia patients with the anterior-inferior AMI pattern frequently had relatively distal LAD artery lesions. Although the mean ejection fraction in the 7 patients with this electrocardiographic pattern was greater than that of the other 35 (0.55 vs 0.48) and the 16 with the most proximal lesions (0.42), the differences were not significant. Because our sample size is relatively small, further study is warranted to determine if this electrocardiographic pattern identifies a clinically distinct subset with improved left ventricular function after AMI.

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