

# Factors that Determine the Direction and Magnitude of Precordial ST-Segment Deviations During Inferior Wall Acute Myocardial Infarction

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Sixty-one patients with inferior acute myocardial infarction (AMI) and no evidence of prior AMI were studied to determine which factors influence the magnitude of precordial ST-segment depression. In the total study group, there was a significant but weak correlation between the magnitude of precordial ST-segment depression and the magnitude of inferior ST-segment elevation ( $r = -0.46$ ,  $p < 0.001$ ). In the 29 patients with evidence of concomitant right ventricular (RV) involvement, precordial ST-segment depression was significantly smaller both in absolute terms ( $-1.3 \pm 1.8$  vs  $-2.8 \pm 1.9$  mm,  $p < 0.01$ ) and relative to the magnitude of inferior ST-segment elevation (ratio of  $-0.2 \pm 1.0$  vs  $-1.1 \pm 0.5$ ,  $p < 0.01$ ), whereas in the 15 patients with lateral ST-segment elevation ( $\geq 1$  mm in lead  $V_6$ ), precordial ST-segment depression was signif-

icantly greater both in absolute terms ( $-3.5 \pm 2.3$  vs  $-1.6 \pm 1.7$  mm,  $p < 0.01$ ) and relative to the magnitude of inferior ST-segment elevation (ratio of  $-1.1 \pm 0.8$  vs  $-0.5 \pm 0.9$ ,  $p < 0.02$ ). Consistent with these findings, the correlation between the magnitudes of precordial and inferior ST-segment deviations was considerably improved when only the 24 patients with neither evidence of RV involvement nor lateral ST-segment elevation were analyzed ( $r = -0.89$ ,  $p < 0.001$ ,  $n = 24$ ). These data suggest that in patients with inferior AMI, there is a reciprocal relation between precordial and inferior ST-segment deviations, which is distorted by concomitant RV involvement and by concomitant lateral left ventricular wall involvement.

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The pathogenesis of precordial ST-segment depression accompanying inferior wall acute myocardial infarction (AMI) is controversial. Precordial ST-segment depression has been attributed to reciprocal effects of inferior or posterior ST-segment elevation,<sup>1-7</sup> ischemic involvement of the posterolateral or inferoseptal myocardial segments adjacent to the left ventricular (LV) inferior wall<sup>8-13</sup> and remote ischemia of the anterior wall as a result of disease of the left anterior descending coronary artery.<sup>14-17</sup> In this study, we reexamine the genesis of precordial ST-segment depression during inferior AMI by investigating the hypothesis that precordial ST-segment depression is primarily a reciprocal

manifestation of inferior ischemia that may be modified by factors that independently alter the position of the precordial ST segment. We postulated that right ventricular (RV) ischemic involvement would be such a modifier of precordial ST-segment depression, because a recent report showed that RV AMI can cause precordial ST-segment elevation.<sup>18</sup> We also postulated that lateral LV wall involvement would modify precordial ST-segment depression, consistent with reports that isolated lateral LV wall infarction causes precordial ST-segment depression.<sup>19,20</sup>

## Methods

**Study population:** The study population consisted of 61 patients (50 men, 11 women), mean age  $57 \pm 12$  years, who were admitted within 3 hours of the onset of an inferior wall AMI characterized by persistent typical ischemic chest pain, ST-segment elevation in lead III and lead aVF, and failure of both chest pain and ST-segment elevation to resolve after administration of sublingual nitroglycerin. The diagnosis of AMI was confirmed by a rise in serum levels of total and MB-creatinine kinase in all patients. Coronary angiography was

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**TABLE I Correlations of Inferior ST-Segment Elevation with Precordial ST-Segment Depression and with ST-Segment Depression in Lead aVL**

	ST ↑ in lead III	ST ↑ in lead aVF
ST deviation in lead V <sub>2</sub>	r = -0.42 <sup>‡</sup>	r = -0.46 <sup>‡</sup>
Maximum precordial ST depression	r = -0.41 <sup>†</sup>	r = -0.44 <sup>‡</sup>
Sum of ST deviations in leads V <sub>1</sub> -V <sub>4</sub>	r = -0.28*	r = -0.33 <sup>†</sup>
ST depression in lead aVL	r = -0.95 <sup>‡</sup>	r = -0.94 <sup>‡</sup>

\* p &lt; 0.05; † p &lt; 0.01; ‡ p &lt; 0.001.

performed within 7 days of admission. Exclusion criteria were a history or electrocardiographic evidence of prior AMI, prior coronary artery bypass surgery and a QRS width of  $\geq 110$  ms. All patients in this study received streptokinase as part of an ongoing research project, 32 by selective intracoronary infusion and 29 by intravenous administration.

**Electrocardiography:** Standard 12-lead electrocardiograms were recorded at a speed of 25 mm/s (10 mm = 1 mV) on a Marquette series 5000 electrocardiograph within 4 hours of the onset of symptoms and before the administration of streptokinase. The electrocardiograms were quantitatively analyzed by an experienced cardiologist who was unaware of the results of any other investigation. ST-segment elevation (positive value) or depression (negative value) was measured to the nearest 0.5 mm as follows. A QRS complex with no or minimal baseline drift was selected. A horizontal baseline connecting consecutive TP segments was drawn. ST-segment deviation was referred to this baseline and measured at 80 ms after the J point. The magnitude of inferior ST-segment elevation was defined in 2 ways: ST-segment elevation in lead III and ST-segment elevation in lead aVF. The magnitude of precordial ST-segment depression was defined in 3 ways: ST-segment deviation in lead V<sub>2</sub>, maximal ST-segment depression in leads V<sub>1</sub> to V<sub>4</sub>, and the arithmetic sum of ST-segment deviations in leads V<sub>1</sub> to V<sub>4</sub>.

**Lateral wall involvement:** The 15 patients who had at least 1 mm of ST-segment elevation in lead V<sub>6</sub> were considered to have electrocardiographic evidence of concomitant lateral LV wall involvement.

**Technetium-99m radionuclide ventriculography:** Multiple-gated equilibrium radionuclide ventriculography

was performed in 60 of the 61 patients within 18 hours of the onset of chest pain using in vitro technetium-99m-labeled autologous red blood cells. LV and RV ejection fractions were determined by methods previously reported.<sup>21,22</sup> The regional contraction pattern of the right ventricle was visually assessed by consensus of 2 experienced nuclear cardiologists who were unaware of the results of any other investigation.

**Thallium-201 scintigraphy:** In 40 patients, standard 3-view resting myocardial scintigraphy was performed after a 2-mCi dose of thallium-201, administered intravenously before streptokinase. Imaging was performed before reperfusion in 15 patients and an average  $52 \pm 36$  minutes (range 5 to 120) after reperfusion in 25. Thallium-201 uptake of the anterior LV wall was visually scored in the anterior and steep left anterior oblique views using a 4-point scoring system (0 = normal, 1 = mildly reduced, 2 = moderately reduced, and 3 = severely reduced) by consensus of 2 nuclear cardiologists, who were unaware of the results of any other investigation.

**Coronary angiography:** All 61 patients underwent coronary angiography and contrast left ventriculography. These procedures were performed within 2 hours of admission in the 32 patients who received intracoronary streptokinase and within 7 days of admission in the 29 who received intravenous streptokinase. All angiograms were analyzed by consensus of 2 experienced angiographers who were unaware of the results of the other investigation. Significant stenosis was defined as a reduction of more than 50% in luminal diameter at the site of maximal luminal narrowing relative to an apparently normal adjacent arterial segment. The artery of infarction and the site of occlusion were obvious in 52 patients (all 32 patients who received intracoronary streptokinase and 20 of 29 who received intravenous streptokinase and did not have coexistent right and circumflex coronary artery disease). In 9 patients the artery of infarction and the site of occlusion were identified by angiographic criteria of an ulcerated atheromatous plaque, i.e., indistinct luminal margins or subintimal ulceration of the coronary artery after thrombolysis,<sup>23</sup> and by the regional pattern of LV dysfunction and perfusion.

**Right ventricular involvement:** The 29 patients in whom the site of right coronary artery occlusion was proximal to the origin of the branch to the right ventricle were considered to have RV involvement. All 29 of these patients also had evidence of RV dysfunction by radionuclide ventriculography; specifically, 25 had an RV ejection fraction  $\leq 39\%$  and 4 had RV dilation and hypokinesia.

**TABLE II Effects of Concomitant Right Ventricular Involvement**

Dependent Variable	RV Involvement Present (n = 29)	RV Involvement Absent (n = 32)	p Value
Age (yr)	54 $\pm$ 12	59 $\pm$ 13	NS
Male sex (% of pts)	83	81	NS
Multiple-vessel CAD (% of pts)	48	78	<0.05
LAD disease (% of pts)	38	59	NS
LV ejection fraction (%)	51 $\pm$ 8	53 $\pm$ 9	NS
RV ejection fraction (%)	30 $\pm$ 10	42 $\pm$ 7	<0.001
ST ↑ in lead III (mm)	4.1 $\pm$ 2.1	3.3 $\pm$ 2.0	NS
ST ↑ in lead aVF (mm)	3.4 $\pm$ 1.9	2.7 $\pm$ 1.6	NS
ST ↓ in lead V <sub>2</sub> (mm)	-1.3 $\pm$ 1.8	-2.8 $\pm$ 1.9	<0.01
Maximal precordial ST ↓ (mm)	-1.8 $\pm$ 1.8	-3.1 $\pm$ 2.0	<0.01
Sum ST ↓ in leads V <sub>1</sub> -V <sub>4</sub> (mm)	-3.1 $\pm$ 5.8	-8.2 $\pm$ 6.4	<0.01
Ratio ST deviations leads V <sub>2</sub> /aVF	-0.2 $\pm$ 1.0	-1.1 $\pm$ 0.5	<0.001
Ratio ST deviations leads V <sub>2</sub> /III	-0.1 $\pm$ 0.9	-1.0 $\pm$ 0.7	<0.001
Ratio ST deviations PRE <sub>max</sub> /aVF	-0.4 $\pm$ 0.8	-1.2 $\pm$ 0.6	<0.001
Ratio ST deviations PRE <sub>max</sub> /III	-0.3 $\pm$ 0.7	-1.1 $\pm$ 0.7	<0.001

CAD = coronary artery disease; LAD = left anterior descending coronary artery; LV = left ventricular; NS = not significant; PRE<sub>max</sub> = lead with maximum precordial ST depression; RV = right ventricular.

**Statistical methods:** Continuous gaussian variables are described by their mean and standard deviation. The unpaired Student *t* test and the 1-way analysis of variance were used for comparison of the distribution of gaussian variables between 2 or more subgroups. Proportional differences between subgroups were compared using Fisher's exact test. Correlations between the magnitudes of inferior ST-segment elevation and precordial ST-segment depression were calculated by the unweighted linear least-squares method and differences between subgroups were compared by an analysis of variance that tested deviations of individual regression lines from the overall regression line. All statistical analyses were performed on a VAX 11-750 computer using BMDP biostatistical programs. A 2-tailed *p* value <0.05 was considered statistically significant.

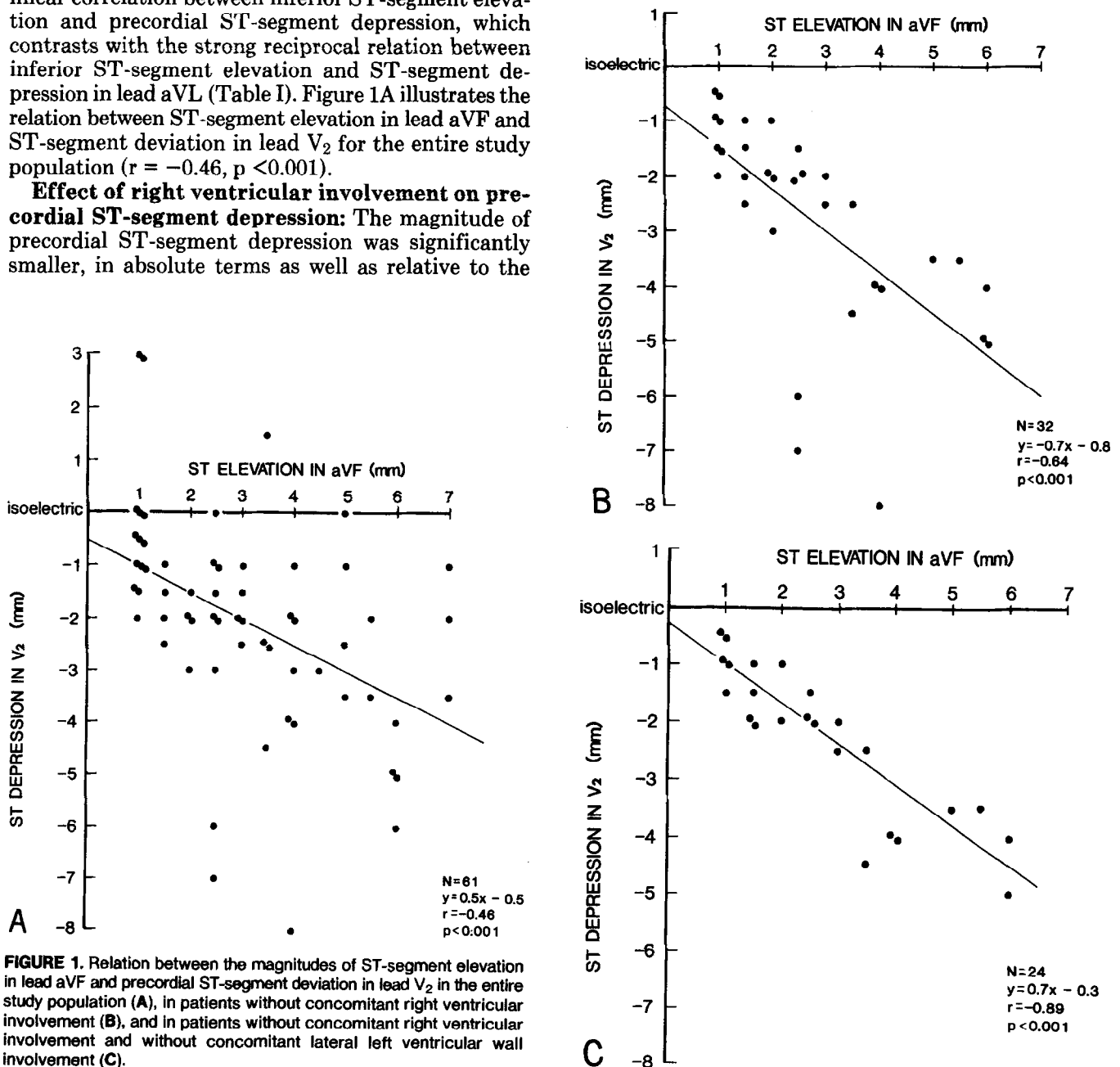
## Results

**Correlation between inferior and precordial ST-segment deviations:** There was a weak inverse linear correlation between inferior ST-segment elevation and precordial ST-segment depression, which contrasts with the strong reciprocal relation between inferior ST-segment elevation and ST-segment depression in lead aVL (Table I). Figure 1A illustrates the relation between ST-segment elevation in lead aVF and ST-segment deviation in lead V<sub>2</sub> for the entire study population ( $r = -0.46$ ,  $p < 0.001$ ).

**Effect of right ventricular involvement on precordial ST-segment depression:** The magnitude of precordial ST-segment depression was significantly smaller, in absolute terms as well as relative to the

magnitude of inferior ST-segment elevation, in the patients with concomitant RV involvement than in patients without evidence of RV involvement, irrespective of which measure of precordial ST-segment deviation was used (Table II). When 29 patients with evidence of concomitant RV involvement were excluded from analysis, the correlation between the magnitudes of inferior and precordial ST-segment deviations was strengthened to  $r = -0.64$ ,  $p < 0.001$  (Fig. 1B).

**Effect of lateral wall involvement on precordial ST-segment depression:** The magnitude of precordial ST-segment depression was significantly greater, in absolute terms as well as relative to the magnitude of inferior ST-segment elevation, in patients with concomitant lateral LV wall involvement than in those without such involvement (Table III). When the pa-



**FIGURE 1.** Relation between the magnitudes of ST-segment elevation in lead aVF and precordial ST-segment deviation in lead V<sub>2</sub> in the entire study population (A), in patients without concomitant right ventricular involvement (B), and in patients without concomitant right ventricular involvement and without concomitant lateral left ventricular wall involvement (C).

**TABLE III** Effects of Concomitant Lateral Left Ventricular Wall Involvement

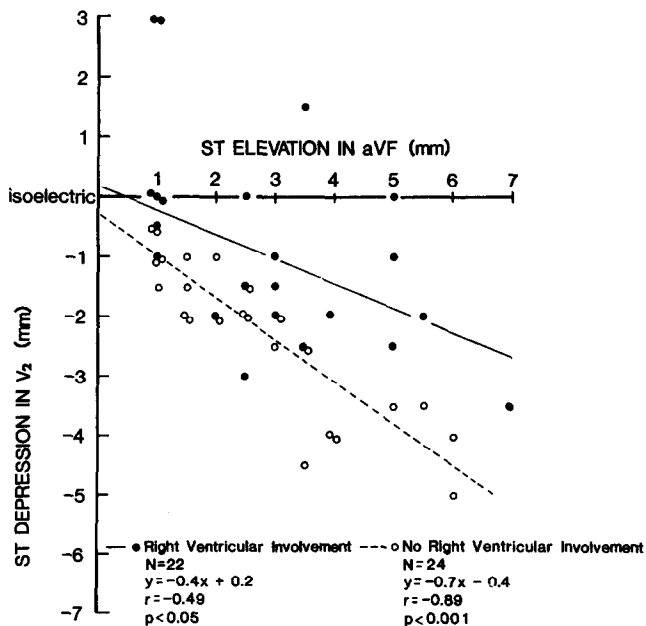
Dependent Variable	LAT Involvement Present (n = 15)	LAT Involvement Absent (n = 46)	p Value
Age (yr)	56 ± 14	57 ± 12	NS
Male sex (% of pts)	80	80	NS
Multiple-vessel CAD (% of pts)	53	67	NS
LAD disease (% of pts)	33	54	NS
LV ejection fraction (%)	49 ± 8	52 ± 9	NS
RV ejection fraction (%)	38 ± 10	36 ± 11	NS
ST ↑ in lead III (mm)	4.1 ± 2.1	3.6 ± 2.1	NS
ST ↑ in lead aVF (mm)	3.7 ± 1.8	2.9 ± 1.7	NS
ST ↓ in lead V <sub>2</sub> (mm)	-3.5 ± 2.3	-1.6 ± 1.7	<0.01
Maximum precordial ST ↓ (mm)	-3.6 ± 2.4	-2.1 ± 1.8	<0.05
Sum ST ↓ in leads V <sub>1</sub> -V <sub>4</sub> (mm)	-8.5 ± 7.8	-4.9 ± 6.0	=0.07
Ratio ST deviations leads V <sub>2</sub> /aVF	-1.1 ± 0.8	-0.5 ± 0.9	<0.02
Ratio ST deviations leads V <sub>2</sub> /III	-1.1 ± 0.9	-0.4 ± 0.8	<0.01
Ratio ST deviations PRE <sub>max</sub> /aVF	-1.2 ± 0.9	-0.7 ± 0.8	=0.08
Ratio ST deviations PRE <sub>max</sub> /III	-1.2 ± 1.0	-0.6 ± 0.7	<0.05

LAT = lateral left ventricular wall; other abbreviations as in Table II.

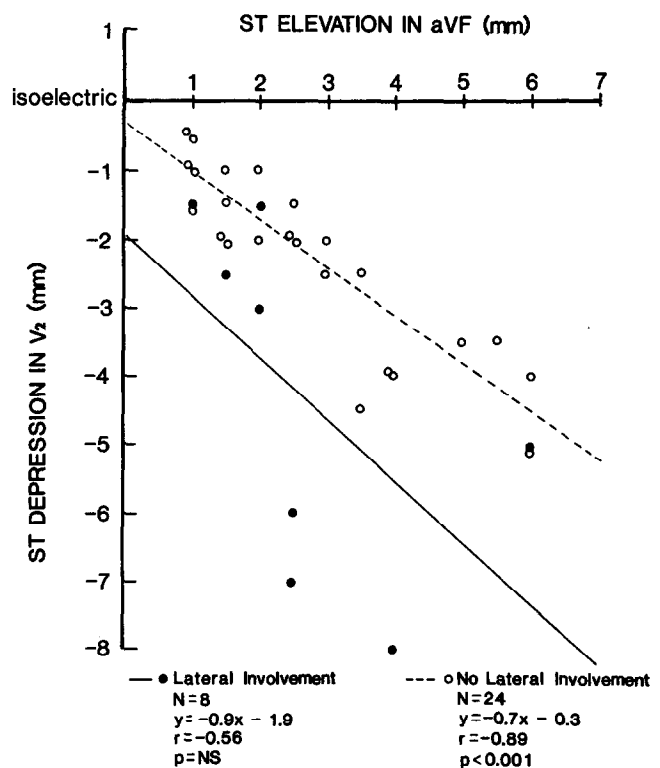
tients with concomitant lateral LV wall involvement were also excluded from analysis, the correlation between the magnitudes of inferior and precordial ST-segment deviations was further strengthened to  $r = -0.89$ ,  $p < 0.001$  (Fig. 1C).

To separate the effects of concomitant RV and lateral wall involvement on precordial ST-segment depression, the study population was divided into 4 subgroups as follows: (1) 22 patients with RV involvement but no lateral wall involvement, (2) 7 patients with both RV and lateral wall involvement, 8 patients with lateral wall involvement but no RV involvement, and 24 patients with neither RV nor lateral wall involvement. The separate effects of concomitant RV and lateral LV wall involvement on the magnitude of precordial ST-seg-

ment depression are presented in Table IV. The separate effects of these 2 factors on the relation between inferior and precordial ST-segment deviations are illustrated in Figures 2 and 3, respectively. The data indicate that RV involvement and lateral LV wall involvement have independent and opposite effects on the magnitude of precordial ST-segment depression, irrespective of which measure of inferior ST-segment elevation or precordial ST-segment depression is used. In the 7 patients with both RV and lateral LV wall in-



**FIGURE 2.** Relation between the magnitudes of ST-segment elevation in lead aVF and ST-segment deviation in lead V<sub>2</sub> in the presence and in the absence of concomitant right ventricular involvement after exclusion of patients with concomitant lateral left ventricular wall involvement. There was a significant difference between the lines of regression for these 2 groups ( $p < 0.01$ ).



**FIGURE 3.** The relation between the magnitudes of ST-segment elevation in lead aVF and ST-segment depression in lead V<sub>2</sub> in the presence and in the absence of concomitant lateral left ventricular wall involvement, after exclusion of patients with concomitant right ventricular involvement. There was a significant difference between the lines of regression for these 2 groups ( $p < 0.05$ ).

**TABLE IV** Electrocardiographic Effects of Concomitant Right Ventricular or Lateral Wall Involvement During Inferior Infarction

Dependent Variable	RV Involvement (n = 22)	RV and LAT Involvement (n = 7)	LAT Involvement (n = 8)	No RV or LAT Involvement (n = 24)
ST-segment elevation in lead III (mm)	3.7 ± 2.1	5.6 ± 1.2*	2.7 ± 1.6	3.5 ± 2.1
ST-segment elevation in lead aVF (mm)	3.0 ± 1.9	4.9 ± 1.3†	2.7 ± 1.6	2.8 ± 1.6
ST-segment deviation in lead V <sub>2</sub> (mm)	-0.9 ± 1.7†	-2.5 ± 1.8	-4.3 ± 2.5†	-2.3 ± 1.3
Maximum precordial ST-segment depression (mm)	-1.5 ± 1.8*	-2.5 ± 1.8	-4.5 ± 2.7*	-2.7 ± 1.6
Sum of ST-segment deviations leads V <sub>1</sub> -V <sub>4</sub> (mm)	-2.7 ± 5.8*	-4.2 ± 5.9	-12.3 ± 7.5*	-6.9 ± 5.6
Ratio ST deviations lead V <sub>2</sub> /lead III	-0.0 ± 1.0†	-0.4 ± 0.3	-1.7 ± 0.9†	-0.7 ± 0.3
Ratio ST deviations lead V <sub>2</sub> /lead aVF	-0.1 ± 1.0†	-0.5 ± 0.3	-1.7 ± 0.7†	-0.9 ± 0.3
Ratio ST deviations PRE <sub>max</sub> /lead III	-0.3 ± 0.8†	-0.4 ± 0.3	-1.8 ± 1.0†	-0.8 ± 0.4
Ratio ST deviations PRE <sub>max</sub> /lead aVF	-0.4 ± 0.9†	-0.5 ± 0.3	-1.8 ± 0.8*	-1.1 ± 0.4

\* p < 0.05; † p < 0.01 vs inferior infarction with no right ventricular or lateral involvement.

PRE<sub>max</sub> = lead with maximum precordial ST segment depression; RV = right ventricular.

involvement, these effects tend to cancel each other (Table IV).

**Anterior wall involvement:** No patient had an occluded left anterior descending coronary artery and in no patient were collateral vessels from the artery of infarction to the anterior wall apparent at angiography. Consistent with this finding, thallium-201 uptake in the proximal anterior wall was normal in all 40 patients who underwent scintigraphy. A mild reduction of thallium-201 uptake, which was limited to a small distal segment of the anterior wall and contiguous with inferoapical involvement of the inferior wall, was noted in 13 patients.

### Discussion

This study indicates that precordial ST-segment deviation during inferior AMI is linearly related to the magnitude of inferior ST-segment elevation and is influenced by concomitant RV involvement and concomitant lateral LV wall involvement. The strong correlation between the magnitudes of inferior ST-segment elevation and precordial ST-segment depression in the 24 patients with neither concomitant RV nor lateral LV wall involvement supports the concept that precordial ST-segment depression during inferior AMI reflects a "precordial view" of the inferior ST-segment vector and is therefore a reciprocal manifestation of inferior ST-segment elevation.<sup>1-7</sup> The similarity of this correlation to the close correlation between the magnitudes of ST-segment deviations in leads aVF and aVL suggests that in the precordial leads, but not in lead aVL, this reciprocal relation is distorted by both concomitant RV involvement and by concomitant LV lateral wall involvement. This implies that these influences affect the ST-segment vector in planes that are substantially perpendicular to the plane of the standard limb leads.

The modifying effect of concomitant RV involvement on the precordial ST segment has not been considered previously. The possibility of such an effect was raised by the finding that proximal right coronary artery occlusion and acute RV ischemia can cause ST-segment elevation in leads V<sub>1</sub> to V<sub>5</sub>.<sup>18</sup> Our criterion of RV involvement was based on the anatomic consideration of whether the right ventricle would have sustained an ischemic injury as a result of occlusion of the proximal right coronary artery and was corroborated by early functional assessment. Demonstration of RV necrosis

was not considered necessary because necrosis is not a prerequisite for ischemic ST-segment deviations<sup>24</sup> and RV ischemia often does not progress to necrosis, especially after early reperfusion.<sup>25</sup>

Consistent with our earlier finding that RV AMI tends to elevate the precordial ST segments<sup>18</sup> and with a recent report that RV infarction causes a loss of anterior forces on the vectorcardiogram,<sup>26</sup> there was significantly less precordial ST-segment depression in patients with inferior AMI and concomitant RV involvement than in patients without RV involvement. In 5 patients with RV involvement, the precordial ST segments were not depressed but were isoelectric, and in 3 patients they were elevated, implying that the direction and magnitude of precordial ST-segment deviations depend on the relative effects of 2 opposite influences—LV inferior wall ischemia and RV ischemia—on the ST-segment vector.

The effect of lateral ST-segment elevation on the precordial ST-segment during inferior AMI has also not been considered in previous studies. Our finding of greater precordial ST-segment depression in patients with lateral ST-segment elevation is consistent with the reported electrocardiographic effects of lateral wall infarction<sup>19,20</sup> and is also consistent with reports of an increased incidence of posterolateral involvement in patients with precordial ST-segment depression.<sup>9,13</sup>

Although no patient in this study had anterior LV wall ischemia in the territory of the left anterior descending coronary artery, one can anticipate that concomitant subendocardial ischemia of the anterior LV wall tends to depress the precordial ST segments and thereby increase precordial ST-segment depression. In contrast, concomitant transmural ischemia of the anterior LV wall, which could occur if collateral vessels from the artery of infarction supplied an occluded left anterior descending coronary artery, theoretically tends to elevate the precordial ST segments and thereby attenuate, or even reverse, precordial ST-segment depression.

In several studies, more extensive inferior infarction has been observed in patients with at least 1 or 2 mm of precordial ST-segment depression. These reports are not inconsistent with our finding of a reciprocal relation between inferior and precordial ST-segment deviations during inferior AMI, because the magnitude of inferior ST-segment elevation is generally proportional to the

severity of inferior wall ischemia, and therefore, by reciprocity, patients with more extensive inferior AMI will tend to have greater precordial ST-segment depression. Patients with lateral ST-segment elevation also tend to have greater precordial ST-segment depression as well as more extensive ischemia as a result of concomitant lateral LV wall involvement.

When our study population is dichotomized according to the criteria of Shah et al<sup>12</sup> into a group with precordial ST-segment depression and a group without precordial ST-segment depression, our findings are consistent with those of Shah et al. In the 42 patients with precordial ST-segment depression, the peak serum level of creatine kinase-MB was 55% higher than in the 19 patients without precordial ST-segment depression; however, the magnitude of inferior ST-segment elevation was also 46% higher in that group. Therefore, in patients with precordial ST-segment depression, information in the precordial leads often does not add to the information contained in inferior and lateral leads.

On the other hand, concomitant RV involvement may attenuate precordial ST-segment depression, and in such patients, smaller precordial ST-segment depression may reflect biventricular and, possibly, more extensive, rather than less extensive, total myocardial involvement. Theoretically, in the presence of concomitant transmural ischemia of the anterior LV wall, smaller precordial ST-segment depression may also reflect more extensive, rather than less extensive, total myocardial involvement.

In conclusion, our results suggest that there is a complex quantitative relation between inferior and precordial ST-segment deviation during inferior AMI. The variability of this relation is caused by factors that independently alter the magnitude and direction of the ST-segment vector, particularly concomitant RV involvement and lateral LV wall involvement. Individual differences in cardiac rotation, in the position of the heart within the chest and in body geometry also contribute to the variability of this relation.<sup>27</sup> In some patients, the relation may be distorted by precordial ST-segment deviations that were present before the occurrence of AMI, that may be due to early repolarization, LV hypertrophy or abnormalities of serum electrolyte concentration.<sup>28</sup>

**Clinical implications:** Precordial ST-segment depression during inferior AMI is primarily a reciprocal manifestation of inferior ST-segment elevation. Greater precordial ST-segment depression may reflect more extensive inferior AMI or concomitant LV lateral wall involvement, which is generally also manifested by greater inferior lead ST-segment elevation or by lateral ST-segment elevation. In contrast, concomitant RV involvement attenuates precordial ST-segment depression, and in these patients smaller precordial ST-segment depression may reflect more extensive, rather than less extensive, total myocardial involvement. The factors that may influence the direction and magnitude of precordial ST-segment deviations must be considered when interpreting the significance of precordial ST-segment deviation in patients with inferior AMI.

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