Precordial ST-segment Depression During Acute Inferior Myocardial Infarction: Clinical, Scintigraphic and Angiographic Correlations

ROBERT S. GIBSON, M.D., RICHARD S. CRAMPTON, M.D., DENNY D. WATSON, PH.D., GEORGE J. TAYLOR, M.D., BLASE A. CARABELLO, M.D., NINA D. HOLT, B.S., R.N., and GEORGE A. BELLER, M.D.

SUMMARY: The cause and associated pathophysiology of precardial ST-segment depression (ST↓) during acute inferior myocardial infarction (IMI) are controversial. To investigate this problem, electrocardiographic findings in 48 consecutive patients with acute IMI were prospectively compared with results of coronary angiography, submaximal exercise thallium-201 (201TI) scintigraphy and multigated blood pool imaging, all obtained 2 weeks after IMI, and with clinical follow-up at 3 months. Patients were classified according to the admission ECG obtained 3.3 ± 3.1 hours after the onset of chest pain. Twenty-one patients (group A) had no or < 1.0 mm ST↓, and 27 (group B) had ≥ 1.0 mm ST↓ in two or more precardial (V1-6) leads. Patients in group B had more prolonged chest pain after admission to the coronary care unit than those in group A (2.8 ± 3.0 vs 1.2 ± 1.1 hours, p < 0.03), greater summed ST-segment elevation in leads II, III, aVf (6.7 ± 4.7 vs 3.3 ± 4.5 mm, p < 0.02), higher plasma peak creatine kinase levels (1133 ± 781 vs 653 ± 482 U/L, p < 0.01), a higher prevalence of “true posterior” infarction by ECG criteria (26% vs 5%, p < 0.05), a lower radionuclide ejection fraction (46 ± 9% vs 54 ± 6%, p < 0.001), more extensive infarct-related asynergy (p < 0.001) and 201TI perfusion abnormalities (p < 0.01), more complications during hospitalization (p < 0.03), and more cardiac events at 3 months (p < 0.02). There were no significant differences between group A and group B in the extent of underlying coronary disease, prevalence of left anterior descending coronary artery disease, exercise-induced ST↓ or angina, and 201TI defects or wall motion abnormalities in anterior or septal segments.

Thus, patients with acute IMI who have associated precardial ST↓ have greater global and regional left ventricular dysfunction due to more extensive inferior or inferoposterior wall infarction, rather than concomitant anteroseptal ischemic injury.

THE CLINICAL IMPLICATIONS of precardial ST-segment depression during acute transmural inferior myocardial infarction (IMI) are controversial. Such electrocardiographic abnormalities have been attributed to benign reciprocal alteration, extensive inferior or additional posterior wall infarction and concomitant anterior ischemia or nontransmural infarction.

We prospectively studied 48 consecutive patients with acute IMI who underwent submaximal exercise thallium-201 (201TI) scintigraphy, multigated blood pool imaging and coronary angiography before hospital discharge. The presence of significant precardial ST-segment depression was determined at the time of admission and compared with exercise test results, regional myocardial perfusion patterns, global and regional left ventricular contractile function, coronary anatomic findings and clinical outcome.

Methods

Patient Selection Criteria

The study population consisted of patients admitted consecutively to the coronary care unit (CCU) at the University of Virginia Hospital who were age 65 years or younger and who satisfied the following criteria: (1) acute transmural IMI diagnosed by typical history of chest pain, serial ECG changes, and a rise and fall of the creatine kinase isoenzyme (MB-CK); (2) no historical or ECG evidence of prior MI; (3) absence of valvular, congenital or cardiomyopathic heart disease or history of coronary bypass surgery; (4) absence of cardiogenic shock, ventricular septal defect or papillary muscle rupture; (5) absence of serious noncoronary disease that might limit long-term follow-up; (6) absence of bundle branch block or left ventricular hypertrophy by standard ECG criteria; and (7) willingness to give informed consent to undergo submaximal exercise thallium-201 (201TI) scintigraphy, multigated blood pool imaging at rest and coronary angiography before hospital discharge.

Between August 1979 and July 1981, 410 patients were admitted to the CCU with ECG- and enzyme-confirmed acute myocardial infarction. Of these, 219 were age-eligible and 68 had a first transmural IMI. Of 56 patients who met selection criteria 1–6, 48 (86%) agreed to undergo complete predischarge testing and return to the Post–Myocardial Infarction Clinic for longitudinal follow-up. The eight patients who refused study were similar to the 48 who consented with respect to age, peak CK, admission Killip class and prevalence of precardial ST-segment depression. The study population included 42 men and six women, ages 38–65 years (mean 52 years).
Electrocardiographic Evaluation

In all patients, the first standard 12-lead ECG was recorded 0.5–9 hours (mean 3.3 ± 3.1 hours) after onset of chest pain. All ECGs were recorded at 25 mm/sec and at 1 cm = 1 mV. ST-segment depression in leads V1–V6 was quantified by one investigator without knowledge of the patient’s identity or clinical data. The maximum ST-segment depression below the TP interval was measured by calipers and expressed in millimeters. The patients were then divided into two groups. Patients in group A had no or insignificant (< 1.0 mm) precordial ST-segment depression; those in group B had 1.0 mm or more ST-segment depression in two or more of the six precordial leads.

Electrocardiographic criteria for acute transmural MI included the development of new Q waves ≥ 40 msec in duration in two or three leads (II, III and aV1).2 “True posterior” MI was diagnosed if a tall R wave appeared in leads V1–2 with an R/S ratio ≥ 1.0.9

Clinical Evaluation

A detailed clinical history was obtained from each patient upon admission. Patients were assigned by clinical criteria to Killip classes I–III.10 To characterize further the population under study, the Norris Coronary Prognostic Index,11 which combines age, history of ischemia, heart size and signs of congestive failure on the admission chest roentgenogram, was calculated for each patient. CK levels were measured upon admission, every 4 hours for the first 24 hours, and then daily until a normal value was measured.

Patients were evaluated daily by at least one staff cardiologist and a research nurse during the course of hospitalization. Complications included pulmonary edema; angina pectoris more than 2 days after onset of infarction and after at least a 24-hour pain-free interval; ventricular tachycardia or ventricular fibrillation requiring countershock; supraventricular tachycardia, including atrial fibrillation, atrial flutter and atrioventricular junctional tachycardia; right ventricular infarction by physical examination;12 atrioventricular block requiring insertion of a temporary pacemaker; pericarditis, indicated by a pericardial friction rub or typical pain responsive to anti-inflammatory agents and no reappearance of MB-CK in the plasma; and infarct extension, indicated by recurrent pain, typical ECG changes and a secondary increase in plasma MB-CK.

Exercise Myocardial Scintigraphy

Patients were exercised on a treadmill using the Naughton protocol13 a mean of 11 ± 2 days (range 7–14 days) after the onset of infarction. Patients were exercised to an end point that consisted of a maximum heart rate of 120 beats/min or the onset of limiting symptoms. A 12-lead ECG was recorded at 1-minute intervals during the exercise period and at 1, 2, 3 and 5 minutes during the recovery period. An i.v. dose of 1.5 mCi of thallium-201 (New England Nuclear Corporation) was administered, followed by a 10-mI saline flush as the patient approached either target heart rate or limiting symptoms. Exercise was continued for an additional 60 seconds if symptoms, ECG changes and blood pressure were stable. Significant ST-segment depression was defined as 1 mm or more of horizontal or downsloping ST depression that extended at least 80 msec after the J point in three consecutive beats.

Thallium imaging commenced 10 minutes after injection with the patient supine in the anterior projection, followed sequentially by the 45° left anterior oblique (LAO) and 70° LAO projection, respectively. The anterior and 45° LAO images were repeated 1 hour and 2–3 hours after 201TI administration. All images were recorded for 10 minutes on an Ohio Nuclear 420 portable gamma camera using an all-purpose (GAP) medium-sensitivity collimator and a 25% window centered on the 80-keV x-ray peak. All studies were stored in a computer (MDS-MUGA Cart of A3) for standardized image formation and quantification of relative 201TI activity in six standard myocardial scan segments by methods described previously.14–16 Initial 201TI uptake and washout were determined for each myocardial segment by two independent observers without knowledge of ECG, other radionuclide or angiographic findings. In cases of disagreement, differences in interpretation were resolved by consensus with a third observer. Criteria for designating a scan segment as abnormal were identical to those previously described.16

Multigated Blood Pool Imaging

After the delayed 201TI images were obtained, equilibrium-gated blood pool imaging was performed at rest to determine left ventricular ejection fraction and segmental wall motion patterns. Patients were injected with unlabeled stannous pyrophosphate (Mallinckrodt, Inc.), followed 30 minutes later by 20 mCi of technetium-99m pertechnetate to complete the in vivo labeling of red blood cells. After equilibration of the blood pool tracer, imaging commenced in the anterior projection, followed sequentially by the 45° and 70° LAO views. A General Electric Maxi-II 15-inch gamma camera equipped with a standard, low-energy, high-resolution, parallel-hole collimator was used. The camera was set at the 140-keV photo peak of technetium-99m with a 20% energy window. Images were collected over 8 minutes using a 2 × image magnification with a mobile computer coupled to an electrocardiographic gating device (American Optical Synchronizer). The cardiac cycle was divided into 14–16 equal segments, and at least 350,000 counts were collected in each frame.

Left ventricular ejection fraction was calculated from the straight 45° LAO projection without caudal angulation using a standard count-volume method. For analysis of regional left ventricular wall motion, scintigraphic data were displayed in a flicker-free endless-loop movie format. Wall motion was assessed qualitatively by dividing the left ventricle into anterolateral, apical and inferior walls in the anterior view and septal, inferoapical and posterolateral walls on the 45°
LAO view, based on expected coronary distribution patterns (fig. 1). Each segment was evaluated for the presence and degree of asynergy according to the terminology used by Herman and Gorlin. Wall motion was graded by three observers without knowledge of ECG. **20**TI or angiographic findings, and a consensus interpretation was obtained based on a four-point scale: 1 = normal; 2 = hypokinetic; 3 = akinetic; and 4 = dyskinetic. A regional wall motion score was derived for both infarct-related segments and remote, noninfarcted segments by summing the scores of individual segments (fig. 1).

For purposes of analysis, both the anterolateral and anteroseptal segments were considered as remote, noninfarcted segments. Infarct-related segments were identified on the radionuclide ventriculogram after comparison with the coronary angiogram. In all 48 patients, the inferior and inferoapical segments were considered. Also, in four patients (two from each group), the mid-high posterolateral wall was designated an infarct-related segment because angiography revealed that the circumflex artery was the infarct vessel. Finally, since the apex represents a confluence of coronary supply areas, it was considered an infarct segment only when an apical abnormality was seen adjacent to an akinetic inferior or inferoapical segment and either one-vessel disease of the right coronary or circumflex artery was demonstrated by angiography, the posterior descending artery extended well into the apex, or the anterior descending artery was normal.

**Coronary Angiography**

All patients underwent selective coronary angiography a mean of 12 days (range 9–15 days) after onset of infarction, using the percutaneous femoral (Judkins) or brachial (Sones) technique. The three major coronary arteries and their branches were independently examined by two experienced angiographers without knowledge of ECG or radionuclide findings using multiple oblique projections, according to a 15-segment model. **20**Maximal luminal diameter narrowing for each major coronary artery was estimated visually and differences of interpretation were resolved by consensus with a third angiographer present. Narrowings in diagonal or marginal branches were considered lesions of the left anterior descending (LAD) and circumflex coronary arteries, respectively.

**Clinical Follow-up**

After hospital discharge, each patient was followed by his private physician who had access to all study results and who regulated therapy. No attempt was made to standardize medical therapy. The clinical status of each patient was reevaluated 3 months after discharge through direct outpatient interview. Recurrent cardiac events were recorded and included: sudden death, reinfarction, angina pectoris and congestive heart failure. Sudden death was diagnosed if the patient died within 1 hour of documented ventricular fibrillation in the absence of cardiogenic shock or pulmonary edema. Congestive heart failure was diagnosed if new exertional dyspnea developed after only minimal or moderate effort.

**Statistical Analysis**

Continuous data are recorded as mean ± SD. The t test was used to determine differences between means of independent observations. A chi-square analysis (with Yates correction) was used to determine differences between proportions.

**Results**

Of the 48 patients with acute transmural IMI, 27 (56%) had significant precordial ST-segment depression on admission. Representative electrocardiographic patterns from the two groups are shown in figure 2.

**Clinical Data**

Table 1 depicts pertinent clinical and ECG findings at the time of admission. The groups were similar with respect to age, sex, admission Killip class, rate-pressure product and the Norris Coronary Prognostic Index. Although the duration of chest pain before admission was similar in the two groups, patients with precordial ST-segment depression had more prolonged chest pain after admission to the CCU (2.8 ± 3.0 vs 1.2 ± 1.1 hours, p < 0.03). The magnitude of summed (Σ) ST segment elevation in leads II, III and aVf was significantly greater in group B than in group A (6.7 ± 4.7 vs 3.3 ± 4.5 mm, p < 0.02). When the
 maximal amount of inferior ST-segment elevation was compared with maximal anterior ST-segment depression (all patients, single-lead comparison), a moderate but highly significant correlation was found ($r = 0.65$, $p < 0.001$).

Based on analysis of serial ECGs, seven patients (26%) in group B demonstrated evidence of “true posterior” MIs (R/S in $V_1$ and $V_2 > 1.0$), compared with only one (5%) in group A ($p < 0.05$).

Figure 3 demonstrates the peak CK levels in the two groups. The mean peak concentration was significantly higher, 1133 ± 781 IU/l (range 154–3081), in patients with precordial ST-segment depression than in patients without precordial depression, 653 ± 482 IU/l (range 116–1682 IU/l) ($p < 0.01$).

Table 2 shows the incidence of in-hospital complications. During hospitalization, only one patient (from group B) died. Death occurred 12 hours after coronary bypass surgery undertaken for recurrent refractory angina and complicated by an intraoperative infarction. Eight patients in group B had pericarditis, compared with only two in group A ($p = 0.07$). Although there were no significant differences between the two groups in the number of patients with complications ($p = 0.08$), including the frequency of pulmonary edema, postinfarction angina, ventricular or supraventricular arrhythmias, right ventricular infarction, atrioventricular block or infarct extension, more complications occurred in group B than group A (32 vs 13, $p < 0.03$).

**Angiographic Findings**

Figures 4 and 5 summarize the angiographic findings in patients with and without precordial ST-segment depression. If 70% or greater luminal diameter reduction is used as a criterion for a significant coronary stenosis, the prevalence of one-, two- and three-vessel disease was similar in the two groups (fig. 4). Fifteen of 27 patients (55%) with precordial ST-segment depression had multivessel disease, compared with 10 of 21 (48%) without precordial depression (NS). Figure 5 demonstrates the prevalence of LAD disease in the two groups. When lesions of 50% or greater were considered, the prevalence of LAD dis-
expression
than
Figure 3. Peak plasma creatine kinase levels are significantly higher in the group with precordial ST-segment depression (ST ↓).

ease in patients with and without precordial ST-segment depression was similar, 59% and 52%, respectively (NS). When lesions of 70% or greater were considered, the prevalence was 37% and 38%, respectively (NS). Thus, the prevalence of multivessel disease or LAD stenosis in IMI patients with precordial ST depression was no higher than that among those without precordial ST-segment depression.

Global and Regional Left Ventricular Function

The differences in left ventricular ejection fraction at the time of discharge in the two groups are shown in figure 6. A value of 55% was taken as the lower limit of normal. In the group with precordial ST-segment depression, 24 of 27 patients (89%) had left ventricular ejection fractions of less than 55%. In contrast, only seven of 21 patients (33%) without precordial depression had subnormal ejection fractions (p < 0.01). The mean ejection fraction in patients with precordial ST depression was 46 ± 9%, significantly lower than the value of 54 ± 6% in the group without precordial depression (p < 0.001).

Figure 7 shows the regional wall motion scores for infarct-related segments at the time of hospital discharge. The mean score of infarct-related segments was higher in patients with precordial ST-segment depression than in those without precordial ST depression (6.7 ± 1.9 vs 4.2 ± 1.4, p < 0.001). Twenty-

| Table 2. In-hospital Complications in Patients Without (Group A) and With (Group B) Precordial ST-segment Depression |
|---------------------------------|-----------------|-----------------|---|
| No. of pts with complications   | Group A (n = 21) | Group B (n = 27) | p   |
|                                 |                 |                 |    |
| Death                           | 0 (0%)          | 1 (4%)          | NS  |
| Pulmonary edema                 | 0 (0%)          | 2 (7%)          | NS  |
| Postinfarction angina           | 5 (24%)         | 7 (26%)         | NS  |
| Ventricular arrhythmias*        | 1 (5%)          | 3 (11%)         | NS  |
| Supraventricular arrhythmias†   | 2 (9%)          | 5 (18%)         | NS  |
| RV infarction                   | 1 (5%)          | 1 (4%)          | NS  |
| AV block                        | 1 (5%)          | 3 (11%)         | NS  |
| Pericarditis                    | 2 (9%)          | 8 (30%)         | NS  |
| Infarct extension‡              | 1 (5%)          | 2 (7%)          | NS  |

*Includes ventricular tachycardia or fibrillation requiring countershock.
†Includes atrial fibrillation or flutter and atrioventricular junctional tachycardia.
‡All three patients had new lateral lead (I, aV L, V 5,6) ECG changes.

Abbreviation: RV = right ventricular; AV = atrioventricular.

four of 27 patients (89%) with precordial ST-segment depression, but only eight of 21 (38%) of those without precordial depression, had akinetic or dyskinetic wall motion in at least one infarct-related segment (p < 0.001). Figure 8 shows the prevalence of asynergy involving remote, noninfarcted left ventricular segments. Overall, 44 of the 48 patients (92%) demonstrated normal wall motion in the anterolateral and anteroseptal segments. Of the four patients judged to have anterior asynergy, two had one-vessel right coronary artery disease.

Hence, IMI patients with precordial ST-segment depression demonstrated more severe depression of global left ventricular function and more severe wall motion abnormalities in infarct regions than those without
precordial ST depression on admission. On the other hand, the group with precordial ST depression did not have more anterior wall abnormalities at the time of discharge.

Exercise Myocardial Perfusion Scintigraphy

Table 3 is a summary of the exercise test results in patients with and without precordial ST-segment depression. There were no significant differences between the two groups with respect to exercise duration, peak heart rate or systolic blood pressure, exercise-induced ST-segment depression or provoked angina. Thus, the prevalence of residual myocardial ischemia by exercise ECG was similar in the two groups.

Figure 6. Comparison of left ventricular (LV) ejection fraction in the two groups. Precordial ST-segment depression (ST ↓) correlated with lower ejection fraction. The lower limit of normal is indicated by the broken horizontal line.

Figure 7. Regional wall motion scores of infarct-related segments. Patients with no left anterior descending coronary artery disease are indicated by triangles and those with 50% and 70% (or greater) stenoses by open and closed circles, respectively. Only one patient had normal wall motion (score of 2) in the infarct region. ST ↓ = ST-segment depression.

Figure 9 shows the distribution of 201Tl perfusion abnormalities detected after submaximal exercise stress before hospital discharge. Only one patient had a normal myocardial scan; all others had at least one defect in the infarct region. Patients with precordial ST-segment depression had significantly more inferior (p = 0.001) and inferoapical (p < 0.03) 201Tl defects than patients without precordial depression. As ex-

Figure 8. Regional wall motion scores of noninfarcted segments. Only four patients had abnormal wall motion in anterolateral or anteroseptal segments at the time of hospital discharge. Symbols are as in figure 7. ST ↓ = ST-segment depression.
expected, the prevalence of mid-high posterolateral wall defects was similar in the two groups, since this myocardial region is generally supplied by the circumflex artery. The prevalence of apical, posterolateral and septal defects was similar in the two groups. This finding was consistent with the lack of anterior or septal wall motion abnormalities in the patients with precordial ST-segment depression and the similar prevalence rate of LAD disease in the two groups.

Table 4 is a comparison of the type of 201TI perfusion patterns corresponding to the inferior and inferoapical scan segments in the two groups. The percent reduction in 201TI uptake was quantified by computer-assisted analysis. Although 47 of the 48 patients had either an inferior or inferoapical defect, group A had more normal scan segments than group B: 13 of 42 (31%) vs four of 54 (8%) (p < 0.01). As expected, the majority of inferior and inferoapical scan segments in both groups were associated with persistent 201TI defects. However, patients in group B had a significantly higher incidence of inferior or inferoapical defects, characterized by a greater than 50% reduction in myocardial 201TI activity above background compared with the normal areas (17 of 54 [31%] scan segments in group B vs two of 42 [5%] in group A, p < 0.01) (fig. 10). Thus, precordial ST-segment depression during acute IMI was associated with more frequent and severe 201TI perfusion abnormalities in the inferior and
Although group B events were larger mean size, significantly more frequent in patients with precordial ST-segment depression. Despite this statistical association, the relatively low prevalence of posterior wall involvement by ECG criteria deserves further comment. We observed that the majority of patients with a first IMI have posterior wall asynergy by two-dimensional echocardiography, yet the ECG rarely detects posterior MI when the criteria of Perloff are used. This suggests that the sensitivity for diagnosing posterior infarction by standard ECG criteria is low and may account for the low prevalence in our patients and in those of Shah et al.

Our observation that the magnitude of precordial ST-segment depression is proportional to the magnitude of inferior ST-segment elevation (r = 0.65, p < 0.001) confirms previous clinical and experimental work. Earlier work in a canine model showed that ECG leads overlying nonischemic myocardium opposite the region affected by coronary ligation invariably demonstrates ST-segment depression during coronary occlusion. Moreover, in every study in which a total occlusion of one vessel was performed in the absence of a stenosis of a remote vessel, the magnitude of ST-segment depression was proportional to the magnitude of ST-segment elevation from the region of infarction. These findings are consistent with classic electrocardiographic theory and tend to support the concept that precordial ST depression during acute IMI may result in cross-cavitary vectoral changes, without presuming anterior ischemia.

### Scintigraphic Correlations

Our results with radionuclide ventriculography are in agreement with previous studies that indicate that precordial ST-segment depression is accompanied by greater depression of left ventricular ejection fraction due to more extensive myocardial damage. The major issue of controversy that our study addresses is the location of this damage. Our data indicate that more extensive inferior or inferoposterior infarction accounts for the greater depression in left ventricular function, rather than concomitant anteroseptal injury. Not only did patients with precordial ST-segment depression show more frequent and severe wall motion abnormalities within the infarct zone, but they also

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**Table 5. Three-month Clinical Outcome in Patients Without (Group A) and with (Group B) Precordial ST-segment Depression**

<table>
<thead>
<tr>
<th></th>
<th>Group A (21 patients)</th>
<th>Group B (26 patients)*</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of pts with event</td>
<td>8 (38%)</td>
<td>13 (50%)</td>
<td>NS</td>
</tr>
<tr>
<td>No. of events</td>
<td>8</td>
<td>17</td>
<td>&lt; 0.02</td>
</tr>
<tr>
<td>Angina pectoris</td>
<td>7 (33%)</td>
<td>11 (42%)</td>
<td>NS</td>
</tr>
<tr>
<td>Congestive heart failure</td>
<td>0</td>
<td>4 (15%)</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td>Reinfarction</td>
<td>1 (5%)†</td>
<td>1 (4%)‡</td>
<td>NS</td>
</tr>
<tr>
<td>Sudden death</td>
<td>0</td>
<td>1 (4%)§</td>
<td>NS</td>
</tr>
</tbody>
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*One patient who died during hospitalization not included.
†Anterior nontransmural infarction.
‡Anterior transmural infarction.
§Out-of-hospital ventricular fibrillation.
demonstrated more extensive $^{201}$TI perfusion defects in the inferior and inferoapical scan segments. Of particular interest was the difference between the two groups with respect to the prevalence of severe, persistent defects characterized by a greater than 50% reduction in $^{201}$TI uptake (table 5, fig. 10). Of 19 such persistent defects, 17 (89%) were in patients with precordial ST-segment depression, all of which were associated with akinetic or dyskinetic wall motion abnormalities on the gated blood pool scan.

Since only two of 27 patients (7%) with precordial ST depression and LAD disease demonstrated anterior wall motion abnormalities (hypokinesis in both), one cannot explain the higher plasma CK levels and lower ejection fractions in this group on the basis of nontransmural anterior infarction. This conclusion is supported by finding a similar prevalence of apical, anterolateral and septal $^{201}$TI perfusion defects in IMI patients with and without precordial ST depression.

Prevalence of LAD Disease and Anterior Wall Ischemia

Over the past few years, much attention has focused on the concept of "ischemia at a distance." In 1940, Blumgart and co-workers postulated that, due to collateral circulation, an acute occlusion of one coronary artery could lead to infarction in the distribution of another coronary artery. Extending this hypothesis, ECG changes outside the zone of infarction as a manifestation of distant ischemia might arise in the distribution of a second coronary stenoses if collateral channels were interrupted by occlusion of the feeding vessel (infarct vessel).

Alternatively, since the ECGs in our study were recorded during the very early phase of infarction, anterior ST-segment depression might be attributed to increased oxygen demands in noninfarcted myocardium perfused by a second critically narrowed vessel. This increased oxygen demand could arise from the increased catecholamine stimulation that usually accompanies acute infarction or alterations in preload and afterload. We found no significant differences between the two groups in the extent of underlying coronary artery disease, prevalence of LAD disease or heart rate and systolic blood pressure upon admission. Thus, our findings appear to refute the concept that precordial ST-segment depression during acute IMI usually represents anterior wall ischemia.

Mere delineation of the number and location of coronary artery stenoses may not provide sufficient information to exclude concomitant anterior wall ischemia in patients with precordial ST-segment depression; thus, the functional consequences of these apparently similar anatomic derangements were further investigated with exercise electrocardiography combined with $^{201}$TI scintigraphy at the time of hospital discharge. Several studies have shown that patients with ischemic ST-segment responses during submaximal exercise testing before hospital discharge have a significantly higher mortality and nonfatal cardiac event rate over the next 12 months than do patients without these changes. Moreover, the amount of residual hypoperfused myocardium may be better delineated by combining $^{201}$TI scintigraphy with submaximal exercise stress electrocardiography. The two groups in our study were similar with respect to exercise-induced ST-segment depression, provoked angina and $^{201}$TI perfusion abnormalities in anterolateral or anteroseptal left ventricular regions. Thus, these findings support our conclusion that precordial ST-segment depression during acute IMI cannot be entirely explained on the basis of anterior wall ischemia due to significant stenoses of the LAD. Indeed, if such ECG changes in the early phase of IMI indicate predominantly anterior ischemia, the specificity and predictive value of precordial ST-segment depression for such ischemia is quite low, since many patients with one-vessel disease of either the right or left circumflex artery or two-vessel disease without LAD involvement demonstrated such electrocardiographic changes.

Our coronary anatomic findings differ with those reported by Salcedo et al., who found a 96% prevalence of LAD disease in patients with precordial ST-segment depression. Although the prevalence of LAD disease in their cohort was similar to that reported by Miller et al. and Turner et al., the patient groups in each of these studies differ substantially from our study population. All of the patients in our study underwent coronary angiography as part of a prospective investigation after acute IMI. When angiographic findings are compared with those of Taylor et al., in post-MI patients in which selection criteria were similar to ours, the prevalence of LAD disease is similar. Of 35 patients with transmural IMI, Taylor et al. found that 18 (51%) had stenoses of 50% or greater involving the LAD; we found 56%.

Clinical Implications

Our data indicate that patients with acute IMI and associated precordial ST-segment depression have sustained more extensive myocardial damage due to larger inferior or inferoposterior infarction. This observation confirms the earlier work by Goldberg et al. who found that precordial ST-segment depression was invariably associated with severe asynnergy of both the inferoapical-low posterolateral wall (93%) as well as the inferior wall. Patients with no or minor precordial ST-segment changes demonstrated only inferior asynnergy with normal wall motion of the low posterolateral wall in that study. Moreover, our results indicate that precordial ST segment depression during acute IMI is predictive of prognosis during and after hospitalization. This finding is also in agreement with studies that have identified the extent of myocardial damage as an important prognostic factor in short-term morbidity and mortality after acute infarction. Since precordial ST-segment depression during early acute IMI appears to identify a subset of patients who have larger infarcts as determined by subsequent quantitative plasma CK analysis, assessment of such anterior ECG changes upon admission may be useful in selecting patients for
pharmacologic or surgical interventions aimed at limiting infarct size. The value of such therapy aimed at myocardial preservation must be evaluated.

Acknowledgment

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