Transient ST-Segment Elevation in Unstable Angina
Clinical and Hemodynamic Significance

By GARY D. PLOTNICK, M.D., AND C. RICHARD CONTI, M.D.

SUMMARY

The significance of the direction of the ST-segment shifts on the 12-lead electrocardiogram was evaluated in 82 consecutive patients with unstable angina. Eighteen patients with ST-segment elevation (group I) were compared with 64 patients with ST-segment depression (group II).

There was no difference between group I and group II with regard to age, sex, or history of previous myocardial infarction. There also was no difference in the angiographic extent, location or severity of the coronary artery disease, collaterals, or resting hemodynamics.

A larger proportion of patients in group I presented with recent onset angina. Life-threatening arrhythmias were more frequent in group I but were uncommon in both groups.

A normal resting electrocardiogram was associated with normal ventricular function in both group I and group II but was associated with single vessel disease only in group I. An abnormal resting electrocardiogram was associated with multiple coronary vessel disease and abnormal ventricular function in both groups.

Single vessel disease was encountered twice as frequently in group I but this difference was not statistically significant. Left main coronary artery disease was found only in group II.

Additional Indexing Words:
Coronary artery disease
Coronary arteriography
Ischemic heart disease

AN IDEAL LESION for coronary artery bypass graft surgery is a severe proximal narrowing of a major coronary artery with a distal vessel free of severe disease. It would be helpful if it were possible to identify those patients likely to have such ideal "surgical" lesions prior to arteriography by means of the electrocardiogram.

It has been proposed that the presence of transient ST-segment elevation (as opposed to ST-segment depression) in association with ischemic cardiac pain suggests that a severe proximal narrowing of a major coronary artery exists.1,4 This report is designed to test that hypothesis by evaluating the significance of the direction of the ST-segment shift during chest pain in 82 consecutive patients with ischemic heart disease presenting with the clinical syndrome of unstable angina. We have attempted to answer the following question: Does transient ST-segment elevation with ischemic cardiac pain define a subset of patients whose coronary disease is different from that of patients with ST-segment depression?

Methods and Materials

Eighty-two patients presented with the syndrome of unstable angina fulfilling previously reported criteria.5 Twenty-seven of these patients presented with rest pain presumably of ischemic origin with no prior history of exertional angina (recent onset rest angina). The remainder presented with rest pain presumably of ischemic origin with a history of previous stable exertional angina of at least three month's duration (crescendo rest angina). All patients were admitted to a coronary care unit because of a suspected impending myocardial infarction and manifested transient electrocardiographic changes during at least one episode of pain.

Patients with new Q wave formation on the electrocardiogram or rise in serum enzymes (creatine phosphokinase, glutamic oxaloacetic transaminase, and lactate dehydrogenase) over a 24-hour period suggestive of myocardial damage were not included in this study.

Of the 82 patients, 18 had transient ST-segment elevation and will be referred to as group I; 64 had transient ST-segment depression with pain episodes and comprise group II.

The electrocardiographic changes necessary for inclusion in this study were transient ST-segment deviations of 1 mm

From the Cardiovascular Division, Johns Hopkins University, Baltimore, Maryland.

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Dr. Conti's present address is Division of Cardiology, University of Florida, Gainesville, Florida.

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Address for reprints: Gary D. Plotnick, M.D., Veterans Administration Hospital, 3900 Loch Raven Blvd., Baltimore, Maryland 21218.

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or greater from baseline, using the T-P segment as the isoelectric line on the standard 12-lead electrocardiogram. Patients with 1 mm or greater flat or upsloping ST-segment elevation were classified in the group with ST-segment elevation even though there was reciprocal ST depression in opposing leads. Patients were continuously monitored in the coronary care unit for episodes of arrhythmia and/or conduction disturbances during at least the initial five days of hospitalization.

Hemodynamic Evaluation

All patients underwent hemodynamic and angiographic evaluation within 72 hours of admission to the coronary care unit. Measurements of right and left heart pressures and cardiac output were made. Left ventricular volume, regional wall motion and ejection fraction were determined by single plane ventriculography in the right anterior oblique projection. Selective coronary arteriography using the Judkins technique was performed, usually requiring a minimum of eight injections in the left anterior oblique, right anterior oblique and left lateral projections.

Each of the three major coronary arteries (right, left anterior descending and circumflex) was evaluated individually by three separate parameters. First, the extent of coronary artery disease, e.g., single, double, or triple vessel; second, the severity of coronary artery disease, e.g., less than 70% narrowing or greater than 70% narrowing; third, location of coronary artery disease, e.g., proximal or distal. In addition, each of the three major coronary arteries was graded individually according to the scoring system shown in table 1. The score of the individual vessels was then combined for a total angiographic score. The presence or absence of collaterals was noted.

Statistical Analysis

Differences were analyzed by Chi square test using Yates correction for continuity or by two-tailed student's t-test. Differences were considered significant when \( P < 0.05 \).

Results

Clinical Characteristics and Presentation

The clinical characteristics of the patients with ST-segment elevation (group I) are compared with those of the patients with ST-segment depression (group II) in table 2. There was no statistically significant difference in age, sex or incidence of previous myocardial infarction. The majority of patients in group I presented with recent onset rest angina and the majority of patients in group II presented with crescendo rest angina \( (P < 0.05) \).

Five of group I and nine of group II were on digitalis at the time of admission to the hospital.

Life-threatening arrhythmias or conduction disturbances were not common in either group. Of the patients in group I, one developed complete heart block, one developed PVCs with bigeminy and two developed ventricular tachycardia. Contrasted with these four patients, only one of the 64 patients in group II developed PVCs requiring therapy \( (P < 0.01) \).

Resting Electrocardiogram

Eight of the 18 patients in group I and five of the 64 patients in group II had normal resting electrocardiograms between episodes of chest pain \( (P < 0.005; \text{table 3}) \).

<table>
<thead>
<tr>
<th>Clinical Characteristics and Clinical Presentation</th>
<th>Group I</th>
<th>Group II</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Mean age (yr) ± SD</strong></td>
<td>57 ± 10.4</td>
<td>53.2 ± 7.9</td>
</tr>
<tr>
<td><strong>(Range)</strong></td>
<td>(36-73)</td>
<td>(37-71)</td>
</tr>
<tr>
<td><strong>Males</strong></td>
<td>67%</td>
<td>78%</td>
</tr>
<tr>
<td><strong>Previous MI</strong></td>
<td>44%</td>
<td>45%</td>
</tr>
<tr>
<td><strong>Type of pain:</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rest pain</td>
<td>10</td>
<td>17</td>
</tr>
<tr>
<td>Recent onset</td>
<td>8</td>
<td>47</td>
</tr>
<tr>
<td>Crescendo</td>
<td>8</td>
<td>47</td>
</tr>
</tbody>
</table>

Table 2

<table>
<thead>
<tr>
<th>Relationship of Electrocardiogram to Angiographic Findings</th>
<th>Coronary arteriogram</th>
<th>Ventriculogram</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Normal Single Double Tripple</td>
<td>Normal Abnormal</td>
</tr>
<tr>
<td><strong>Group I</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal ECG</td>
<td>N = 8</td>
<td>1 7 0 0 6 2</td>
</tr>
<tr>
<td>Abnormal ECG</td>
<td>N = 10</td>
<td>0 0 1 9 1 9</td>
</tr>
<tr>
<td><strong>Group II</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal ECG</td>
<td>N = 5</td>
<td>0 1 2 2 4 1</td>
</tr>
<tr>
<td>Abnormal ECG</td>
<td>N = 59*</td>
<td>2 11 10 28 4 55</td>
</tr>
</tbody>
</table>

*Includes 8 patients with left main coronary artery disease.

Table 3
Table 4

Hemodynamics

<table>
<thead>
<tr>
<th></th>
<th>Group I</th>
<th></th>
<th>Group II</th>
<th></th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N = 18</td>
<td>N = 64</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LVEDP (mm Hg)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean ± sd (Range)</td>
<td>13.1 ± 6.7 (5-30)</td>
<td>13.5 ± 7.4 (4-40)</td>
<td>N.S.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ventricular volume (cc)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean ± sd (Range)</td>
<td>160.1 ± 90.0 (54-382)</td>
<td>157.7 ± 50.0 (82-290)</td>
<td>N.S.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ejection fraction (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean ± sd (Range)</td>
<td>59.2 ± 16.6 (25-84)</td>
<td>59.0 ± 15.0 (13-86)</td>
<td>N.S.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cardiac index (L/min/m²)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean ± sd (Range)</td>
<td>2.9 ± 1.0 (1.5-5.4)</td>
<td>3.4 ± 1.1 (1.4-6.0)</td>
<td>N.S.</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

was compared to group II, and there was wide variation in each group.

Coronary Arteriography

Extent of Coronary Arterial Disease

For purposes of this report, coronary artery disease is defined as arteriographic narrowings greater than 50% of the intraluminal diameter. Of the 18 patients in group I, one had angiographically normal coronary arteries, seven had single, one had double and nine had triple vessel disease. Of the 64 patients in group II, two had angiographically near normal coronary arteries (minimal irregularities), 12 had single vessel, 12 had double vessel and 30 had triple vessel disease. Eight additional group II patients had left main coronary artery disease. Six of these patients had associated triple vessel disease, one had double vessel and one had single vessel disease. None of the patients in group I had angiographic disease of the left main coronary artery.

Severity of Coronary Arterial Disease

The severity of the narrowings was evaluated in the coronary artery assumed to be perfusing that portion of the myocardium responsible for the ST-segment shift. For example, transient ECG changes occurring in leads I, aVL, and/or the precordial leads were considered to be "anterior" changes and were assumed to reflect myocardial ischemia in the distribution of left anterior descending and/or left circumflex marginal coronary arteries. ECG changes in leads II, III, and aVF were considered as "inferior" changes and were assumed to reflect myocardial ischemia in the distribution of the right coronary artery and/or left posterior circumflex coronary artery. Figure 1 compares the severity of narrowings in the coronary arteries in both groups of patients. Forty-four percent of patients in group I had 90-99% narrowing of the int-

traluminal diameter compared with 53% of patients in group II. The majority of patients in both groups had coronary artery narrowings greater than 70% occluded. There was no significant difference in the severity of coronary artery narrowings between patients with ST-segment elevation and ST-segment depression.

Location of Coronary Artery Narrowing

The exact location of the most severe narrowing in the coronary artery considered to be responsible for the ST-segment shift was evaluated. For this purpose, only coronary artery narrowings greater than 70% were considered hemodynamically significant. A narrowing was considered proximal if it was located either in the right main coronary artery proximal to the acute margin, left main coronary artery, left anterior descending proximal to the first septal perforator and left circumflex artery proximal to the first circumflex obtuse marginal branch. Ten of the patients in group I had proximal narrowing (56%) compared with 41 of the patients in group II (64%).

Table 5

Cumulative Arteriographic Score

<table>
<thead>
<tr>
<th>Score</th>
<th>Group I</th>
<th></th>
<th>Group II</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N = 18</td>
<td></td>
<td>N = 64</td>
<td></td>
</tr>
<tr>
<td>0–3</td>
<td>3</td>
<td></td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>4–6</td>
<td>5</td>
<td></td>
<td>9</td>
<td></td>
</tr>
<tr>
<td>7–9</td>
<td>2</td>
<td></td>
<td>15</td>
<td></td>
</tr>
<tr>
<td>10–12</td>
<td>4</td>
<td></td>
<td>26</td>
<td></td>
</tr>
<tr>
<td>13–15</td>
<td>4</td>
<td></td>
<td>9</td>
<td></td>
</tr>
</tbody>
</table>
Except for the absence of left main coronary artery disease in group I patients, there were no significant differences in location of the coronary artery narrowings.

**Angiographic Score**

Eight of the 18 patients in group I (44%) had angiographic scores above ten compared with 35 of the 64 patients in group II (55%). This difference was not statistically significant (table 5).

**Collateral Circulation**

Collateral circulation was present in six of the 18 patients in group I and 33 of 64 patients in group II. This difference was not statistically significant.

**Discussion**

This study was designed to evaluate the ways in which patients with transient ST-segment elevation differed from patients with transient ST-segment depression. The question is important because it has been asserted that ST-segment elevation signifies a poorer prognosis and a greater likelihood of myocardial infarction and death. It is also held that patients with ST-segment elevation are more likely to have disease in the proximal portions of the coronary arteries with minimal distal disease, and therefore, are more likely to be excellent candidates for surgical revascularization. The belief that the prognosis is poor and the likelihood of surgical disease high has led to a sense of urgency in the management of these patients.

We have compared the coronary anatomy of two groups of patients with similar clinical presentations, i.e., unstable angina presenting with rest pain, who were divided solely on the basis of the direction of the ST-segment shift on the electrocardiogram. We found no difference in the extent, location, or severity of the coronary artery disease as determined by coronary arteriography between the patients with transient ST-segment depression.

If there is no obvious difference in the angiographic extent, location or severity of the coronary artery disease, why do some patients develop transient ST-segment elevation while others develop ST-segment depression during episodes of chest pain? Experimental work suggests that transient ST-segment elevation is associated with "more severe ischemia" than transient ST-segment depression. Ekmekci demonstrated in the dog that total ligation of a major coronary artery produced the greatest ST-segment elevation in the central part of the ischemic area. Also, when the central area was rendered less ischemic, ST-segment depression appeared. One explanation for the production of ST-segment elevation in some patients may relate to the absence of collateral blood flow, e.g., absence of collateral results in more severe ischemia, and would be associated with ST-segment elevation. However, in our patients this was not the case, since there was no significant difference in collateral circulation seen at arteriography in either group. A second explanation might relate to the location and severity of the coronary artery disease.

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