Body Surface Distribution of Exercise-Induced ST Depression in Patients with Angina Pectoris

Isao Kubota, M.D., Yoshihiko Watanabe, M.D.,
Kai Tsuiki, M.D., and Shoji Yasui, M.D.

SUMMARY

In 38 patients with angina pectoris, 87 unipolar lead electrocardiograms were recorded from different sites over the entire thoracic surface both before and after submaximal treadmill exercise. The site of exercise-induced ST depression on the body surface was correlated with the findings of the coronary arteriography. The number of leads which showed exercise-induced ST depression was 7.2 ± 1.8 in one vessel disease, 15.6 ± 2.1 in two vessel disease, and 23.6 ± 2.0 in three vessel disease (values are mean ± SEM). Thus, the number of leads showing ST depression was proportional to the severity of the coronary artery disease. Exercise-induced ST depression was most often seen in left anterior chest leads, especially in V5 of the standard 12-ECG. However, it was not possible to identify the obstructed coronary artery from the body surface distribution of ST depression.

Additional Indexing Words:
Coronary arteriography Treadmill exercise test Body surface mapping

IT is generally believed that exercise-induced ST depression in patients with angina pectoris is a reflection of exercise-induced myocardial ischemia.1) Although exercise-induced ST elevation has been reported to identify the anatomical site of myocardial ischemia,2)-4) it is controversial whether exercise-induced ST depression can identify an ischemic region.5)-11) This study examines the body surface distribution of exercise-induced ST depression in patients with angina pectoris by means of body surface mapping and compares these results with findings from coronary arteriography.

From the First Department of Internal Medicine, Yamagata University School of Medicine, Zao-Iida, Yamagata City, Yamagata 990-23, Japan.
Presented in part at the 9th International Congress on Electrocardiology, Tokyo, June, 1982.
Received for publication October 9, 1982.
Manuscript revised January 21, 1983.

853
MATERIALS AND METHODS

Thirty-eight patients (male 36, female 2) with angina pectoris (ages 45–68 years; mean=56 years) were studied. Patients with electrocardiographic evidence of previous transmural infarction (Q waves in the resting standard 12-ECG) and patients with intraventricular conduction disturbances were excluded from the study. Those with variant angina pectoris were also excluded. All patients showed a horizontal or downsloping ST depression (greater than 0.05 mV) during the treadmill test (using the standard 12-ECG and the lead CM-5) performed before this study. The diagnosis of coronary artery disease was established by selective coronary arteriography. Coronary narrowing of Grade 3 or more of Pujadas’ criteria\textsuperscript{12,13} (Table I) was considered significant stenosis. Left main disease was identified as a two vessel disease of the left anterior descending artery (LAD) and the left circumflex artery (LCX). Table II shows the results of coronary arteriography in the 38 patients.

Body surface mapping was performed using a HPM-5100 system (Chu-

Table I. Coronary Arteriographic Scoring System

<table>
<thead>
<tr>
<th>Grade</th>
<th>Criteria for judging</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Normal</td>
</tr>
<tr>
<td>1</td>
<td>Irregular arterial contour without a definite degree of stenosis</td>
</tr>
<tr>
<td>2</td>
<td>Non-significant stenosis: (&lt;40% \text{ short}^{<em>}, &lt;20% \text{ long}^{</em>}, &lt;10% \text{ tubular}^{*})</td>
</tr>
<tr>
<td>3</td>
<td>Significant stenosis: 40–85% short, 20–60% long, 10–50% tubular</td>
</tr>
<tr>
<td>4</td>
<td>Subocclusion: (&gt;85% \text{ short}, &gt;60% \text{ long}, &gt;50% \text{ tubular} )</td>
</tr>
<tr>
<td>5</td>
<td>Occlusion</td>
</tr>
</tbody>
</table>

\* \text{ short}=shorter than 5 mm, \text{ long}=5–10 mm, \text{ tubular}=longer than 10 mm, \%=% reduction in diameter.

Table II. Results of Coronary Arteriography in 38 Patients

<table>
<thead>
<tr>
<th>One vessel disease</th>
<th>Patients (N)</th>
</tr>
</thead>
<tbody>
<tr>
<td>RCA</td>
<td>9</td>
</tr>
<tr>
<td>LAD</td>
<td>10</td>
</tr>
<tr>
<td>LCX</td>
<td>3</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Two vessel disease</th>
<th>Patients (N)</th>
</tr>
</thead>
<tbody>
<tr>
<td>RCA+LAD</td>
<td>3</td>
</tr>
<tr>
<td>LAD+LCX</td>
<td>3</td>
</tr>
<tr>
<td>LCX+RCA</td>
<td>2</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Three vessel disease</th>
<th>Patients (N)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>8</td>
</tr>
</tbody>
</table>

| Total                | 38           |
nichi Denshi Company, Nagoya). The localization of the lead points and the procedure for the data sampling and processing have been described.\textsuperscript{14,15} By means of this system, 87 unipolar electrocardiograms distributed over the entire thoracic surface (anterior 59, back 28) were recorded simultaneously before and after submaximal treadmill exercise.\textsuperscript{13,16} Pre-exercise data were recorded immediately before the exercise. The post-exercise data were recorded 1.5 min after the cessation of the exercise. Data sampling was always done at the resting expiratory level in the supine position.

Several criteria were used to identify exercise-induced ST depression in any of the 87 leads. A horizontal or downsloping ST depression of 0.05 mV or more from the baseline (the flat portion of the PQ segment), lasting at least 0.08 sec, was considered significant. If ST depression was present before exercise, additional 0.05 mV was required. Its amplitude was measured 0.04 sec after the J point.

Statistical comparisons were done by the unpaired t-test; \( p < 0.05 \) was considered significant.

**Results**

Of the 38 patients studied, 22 had one vessel disease, 8 had two, and 8 had three vessel disease (Table II). The number of leads which showed exercise-induced ST depression was 7.2±1.8 in one vessel disease, 15.6±2.1 in two vessel disease, and 23.6±2.0 in three vessel disease (values are mean±SEM, Fig. 1). The number was significantly larger in three vessel disease than in one (\( p < 0.001 \)) or two vessel disease (\( p < 0.02 \)), and in two vessel disease than in one vessel disease (\( p < 0.02 \)).

All patients with two or three vessel disease (\( n = 16 \)) had exercise-induced ST depression in at least one of the 87 leads, while 8 out of the 22 patients with one vessel disease did not display ST depression. Therefore, 30 out of the 38 patients showed exercise-induced ST depression on the body surface. Fig. 2 shows the body surface distribution of ST depression in these 30 patients. The left side of the square shows the anterior chest and the right side the back. The vertical line from lead point I1 to I5 represents the left midaxillary line, and the vertical line from A1 to A5 represents the right midaxillary line. G4, H4, and I4 correspond to the V4, V5, and V6 of the standard 12-ECG, respectively. The numerals in the format represent the number of patients who developed ST depression after exercise at each lead point. ST depression occurred most frequently in the left anterior chest leads, especially in V5 of the standard 12-ECG (26/30, 87\%). Conversely, none of the patients developed ST depression in leads on the right back or right anterior chest.
The area which develops ST depression is triangular in shape; the apex is the left upper border of the anterior chest. We have tentatively decided to call this triangular area the "ST depression region".

Of 22 patients with one vessel disease, 9 had right coronary artery (RCA) disease, 10 had LAD disease, and 3 had LCX disease. ST depression was found in 6 of 9 patients with RCA lesions, in 6 of 10 with LAD lesions, and in 2 of 3 with LCX lesions. Fig. 3 shows the body surface distribution of ST depression in each group with one vessel disease. Each figure shows a similar body surface distribution of ST depression, and these areas of ST depression occupy the central part of the "ST depression region". There seem to be no specific regions that predict which coronary artery is obstructed.

Fig. 4 shows the site of the lead which showed maximal degree of ST depression in patients with one vessel disease. No definite correlation was found between the site of maximal ST depression and the obstructed coronary...
Out of the 30 patients with one or two vessel disease, 16 had no RCA disease (LAD 10, LCX 3, LAD+LCX 3, Table II). Eleven out of the 16 patients with LAD and/or LCX disease but no RCA disease developed ST depression on the body surface. Similarly, 10 out of the 14 patients without LAD disease but with RCA and/or LCX disease and 15 out of the 22 patients without LCX disease but with RCA and/or LAD disease had exercise-induced ST depression. Fig. 5 shows the body surface distribution of ST depression in these 3 groups. Each group had a similar spatial distribution of ST depression. Furthermore, the triangular area of ST depression in each group was nearly identical to the “ST depression region”. There seem to be no specific regions that predict which coronary artery is free from disease.

**DISCUSSION**

In this study, patients with three vessel disease had significantly larger number of leads which showed ischemic (horizontal or downsloping) ST depression than those with one or two vessel disease. Similarly, more leads from patients with two vessel disease showed ST depression than in one vessel
Fig. 3. Body surface distribution of exercise-induced ST depression in one vessel disease. The format is as in Fig. 2. There seem to be no specific regions that predict which coronary artery is obstructed. RCA = right coronary artery; LAD = left anterior descending artery; LCX = left circumflex artery.

disease (Fig. 1). Fox et al\textsuperscript{10} reported similar results using 16 lead precordial mapping after exercise. Therefore, the severity of the coronary artery disease in patients with angina pectoris could be evaluated non-invasively from the area of ST depression on the body surface.

All patients displayed exercise-induced ST depression in a treadmill test (using standard 12-ECG and lead CM-5) performed before this study. However, we could not detect significant ST depression in 8 patients with one vessel disease in the present study. This probably reflects the fact that the post-exercise electrocardiographic data were recorded 1.5 min rather than immediately after the exercise. Thus ST depression in patients who did not show ST depression in our study may have disappeared by the time of the post-exercise recording.

By means of total body surface mapping, we established the body surface
distribution of the exercise-induced ST depression in patients with angina pectoris (Fig. 2). ST depression was most often seen in lead V5 of the standard 12-ECG. Twenty-six out of the 30 patients (87%) who developed ischemic ST depression in our study were detected from V5 recordings. This result supports the fact that the most popular lead system in exercise electrocardiography is V5 or modified bipolar lead V3, for example CM5 or CC5, when a single-channel recorder is available.

Fig. 2 also shows the extent of exercise-induced ST depression in patients with angina pectoris. Note that the ST depression frequently occurred in leads on the left anterior chest and did not appear in leads on the right back or right upper anterior chest. We decided to call the area which had developed ST depression the “ST depression region”. These data are necessary to elucidate the clinical significance of ST depression in exercise testing.

Our results indicate that the body surface distribution of ST depression is not simply determined by the anatomical site of coronary stenosis. It was difficult to identify the obstructed coronary artery (Fig. 3) or the intact coronary artery (Fig. 5) from the body surface distribution of ST depression. Using 12 lead exercise electrocardiography, Dunn et al11) also reported that the site of ST segment depression on exercise did not identify the anatomic site of coronary artery obstruction. By contrast, Fox et al10) using 16 lead
precordial mapping after exercise, concluded that typical precordial projections of ST segment changes were found in 74% (14/19) of patients with one vessel disease. They postulated that isolated disease of LAD, LCX, and RCA was present when ST changes were confined to the anterior, lateral and inferior region of the precordium, respectively. However, their study may have included patients with previous transmural myocardial infarction or variant angina pectoris, since they only utilized results of coronary arteriography and did not mention whether these patients were excluded. In addition, they used both ST depression and ST elevation in interpreting the exercise-induced ST changes. Thus, it is difficult to compare their results with ours. As far as our study population is concerned, we could not obtain any definite correlation between the site of maximal ST depression and the obstructed coronary artery (Fig. 4). However the sample size should be increased to see if the site of the lead showing maximal ST depression is cor-
related with the site of coronary stenosis.

One limitation of the present study must be considered. The results of this study were obtained from the data recorded 1.5 min after the cessation of exercise, compared with those just before exercise. Thus, we lack continuous data about the body surface distribution of ST depression both during and after exercise. We are preparing to clarify the body surface distribution of ST depression both during and after, especially immediately at the end, of exercise.

The lack of a clear correlation between the location of exercise-induced ST depression and the site of coronary narrowing may be related to various factors. These include (a) the presence of collateral circulation, (b) differences of coronary artery anatomy, for example right predominant pattern or left predominant pattern, and (c) individual differences in heart position in relation to the electrode positions. One plausible explanation is that exercise-induced ischemia extends through the greater part of the sub-endocardial region of the left ventricle. Accordingly, the left ventricular apex may be most vulnerable to myocardial ischemia. This is in agreement with the fact that exercise-induced ST depression is most often seen in leads near V5 of the standard 12-ECG, in both the total study group (Fig. 2), and in patients with one vessel disease (Fig. 3) or with no obstruction of one major coronary artery (Fig. 5).

Acknowledgments

The authors wish to express their gratitude to Professor Masahiko Washio, Professor Kozui Miyazawa, and Professor Shozo Tuboi for their valuable comments on the manuscript.

References


