Exercise-induced ST-segment Elevation

Correlation of Thallium-201 Myocardial Perfusion Scanning and Coronary Arteriography

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SUMMARY Exercise-induced ST-segment elevation was correlated with myocardial perfusion abnormalities and coronary artery obstruction in 35 patients. Ten patients (group 1) developed exercise ST elevation in leads without Q waves on the resting ECG. The site of ST elevation corresponded to both a reversible perfusion defect and a severely obstructed coronary artery. Associated ST-segment depression in other leads occurred in seven patients, but only one had a second perfusion defect at the site of ST depression. In three of the 10 patients, abnormal left ventricular wall motion at the site of exercise-induced ST elevation was demonstrated by ventriculography. Twenty-five patients (group 2) developed exercise ST elevation in leads with Q waves on the resting ECG. The site of ST elevation corresponded to severe coronary artery stenosis and a thallium perfusion defect that persisted on the 4-hour scan (constant in 12 patients, decreased in 13). Associated ST depression in other leads occurred in 11 patients and eight (73%) had a second perfusion defect at the site of ST depression. In all 25 patients with previous transmural infarction, abnormal left ventricular wall motion at the site of the Q waves was shown by ventriculography.

In patients without previous myocardial infarction, the site of exercise-induced ST-segment elevation indicates the site of severe transient myocardial ischemia, and associated ST depression is usually reciprocal. In patients with Q waves on the resting ECG, exercise ST elevation may be due to peri-infarctional ischemia, abnormal ventricular wall motion or both. Exercise ST-segment depression may be due to a second area of myocardial ischemia rather than being reciprocal to ST elevation.

TRANSIENT ST-segment elevation during stress electrocardiography, although infrequent, has been observed in patients with severe and unstable ischemic heart disease, variant angina and left ventricular aneurysm. The ST-segment elevation is thought to be due to myocardial ischemia or abnormal ventricular wall motion. A clear distinction has not been made between the significance of exercise-induced ST-segment elevation in the presence and the absence of Q waves on the resting ECG. The purpose of this study was to correlate exercise-induced ST-segment elevation in patients with and without previous myocardial infarction with both the perfusion abnormalities on thallium-201 myocardial scanning and coronary artery anatomy.

Patients

Thirty-five patients (34 males, one female) developed exercise-induced ST-segment elevation during routine exercise testing between January and December 1978. The mean age of the patients was 50.1 years (range 34–64 years). Thirty of the 35 patients complained of angina pectoris and 28 had a history of previous myocardial infarction. The relevant clinical data for each patient are presented in table 1. Exercise thallium-201 myocardial perfusion scanning, coronary arteriography and ventriculography were performed in all patients.

Exercise Testing

Patients exercised on an electrically braked, upright bicycle ergometer using a multistage, intermittent protocol. After a 1-minute warm-up period of cycling without work load, the load was set at 150 kilopond-meters (kpm) for men and 100 kpm for women and increased every 2 minutes (by 150 kpm for men and 100 kpm for women) until chest pain, breathlessness or fatigue occurred. Twelve-lead ECG monitoring was used during each minute of exercise and recovery. Thallium-201 was injected at peak exercise through an intravenous cannula previously inserted. Patients continued exercising for 60 more seconds to enable adequate blood clearance and myocardial uptake of thallium during conditions of stress. Antianginal medications, including vasodilators and β-adrenergic blocking drugs, were not modified before testing and patients were not exercised within 3 months of myocardial infarction.

ECG Interpretation

The ECGs were interpreted by two independent observers according to the criteria of the American Heart Association. Previous myocardial infarction was considered to be present if there were abnormal Q waves on the resting ECG. Exercise-induced ST-segment elevation was considered to be present if in
Table 1. Clinical Data

<table>
<thead>
<tr>
<th>Pt</th>
<th>Age (years)</th>
<th>Sex</th>
<th>History</th>
<th>Resting ECG</th>
<th>Exercise testing</th>
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<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Abnormal Q waves</td>
<td>Abnormal ST-T waves</td>
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<tr>
<td>1</td>
<td>47</td>
<td>M</td>
<td>SE, AP</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>55</td>
<td>M</td>
<td>AP</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>51</td>
<td>M</td>
<td>AP</td>
<td>L</td>
<td>1, L, V1,4</td>
</tr>
<tr>
<td>4</td>
<td>54</td>
<td>M</td>
<td>AP</td>
<td></td>
<td></td>
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<td>5</td>
<td>51</td>
<td>M</td>
<td>AP</td>
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<tr>
<td>6</td>
<td>38</td>
<td>M</td>
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<td>51</td>
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<td>9</td>
<td>54</td>
<td>M</td>
<td>SE, AP</td>
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<tr>
<td>10</td>
<td>42</td>
<td>M</td>
<td>MI</td>
<td>2, F</td>
<td>2, F, L, V4,6</td>
</tr>
</tbody>
</table>

1. Group 1. ST-segment elevation without Q waves

2. Group 2. ST-segment elevation over Q waves

*Ventricular aneurysm.

Abbreviations: Max HR = maximum heart rate (beats/min); ECG ST↑ = leads with ST-segment elevation during exercise; ECG ST↓ = leads with ischemic ST-segment depression during exercise; exercise defect ST↑ = exercise perfusion defect at a site corresponding to the ECG site of ST-segment elevation; exercise defect 2nd site = exercise perfusion defect at a site unrelated to the ECG site of ST-segment elevation; 4-hour defect ST↑ = perfusion defect on 4-hour redistribution scan corresponding to ECG site of ST-segment elevation; 4-hour defect 2nd site = perfusion defect on 4-hour redistribution scan unrelated to ECG ST-segment elevation; LMCA = left main coronary artery; LAD = left anterior descending coronary artery; LCX = left circumflex coronary artery; RCA = right coronary artery; LMCA = left main coronary artery; LAD = left anterior descending coronary artery; LCX = left circumflex coronary artery; RCA = right coronary artery; LV WMA = left ventricular wall motion abnormality (akinetic or severely hypokinetic areas) on ventriculography; SE = subendocardial infarction; AP = angina pectoris; MI = transmural myocardial infarction; + = abnormality present; — = no abnormality present; b = ingestion of β-adrenergic blocking drugs within 48 hours of exercise testing; d = ingestion of digitalis preparation within 2 weeks of exercise testing; P = partial redistribution on 4-hour thallium scan; T = total or complete redistribution on 4-hour thallium scan; C = constant defect when exercise and 4-hour thallium scans were compared; +e = obstruction of LAD before first perforator with collaterals; +nc = obstruction of LAD before first perforator with no collaterals; —e = obstruction of LAD after first perforator with collaterals; —nc = no collaterals; c = collateral; AntAp = anteroseptal; Ant = anterior; I = inferior; IAp = inferoseptal.

Myocardial Perfusion Scanning

Scanning was begun in the exercise laboratory less than 10 minutes after the intravenous administration of 1.5–2 mCi of thallium-201 (exercise scan). With the patient supine, anterior, 40° left anterior oblique, 60° left anterior oblique and left lateral views were taken using an Ohio Nuclear Sigma 420 mobile camera, as previously described.10 Scans were repeated 4 hours later at rest in the same four views without further ad-
ministration of thallium (4-hour redistribution scan).

The scans were interpreted from the original Polaroid scintiphotos without computer enhancement or background subtraction by three independent and blinded observers. (Computer enhancement was used in the illustrations.) A thallium defect was considered to be present if there was a discrete reduction of tracer activity (visually estimated at greater than 50%) involving more than 15% of the left ventricular circumference (visually estimated). Exercise and 4-hour redistribution scans were compared for the presence or absence of a thallium defect. A consensus of the three observers was taken. A thallium defect on the exercise scan was classified as either reversible if it decreased, or constant if it did not change on the 4-hour redistribution scan.

### Coronary Arteriography and Ventriculography

Selective coronary arteriography was performed using the Judkins or Sones technique. Each study was interpreted by an independent observer. Coronary artery narrowings of 70% or greater of the luminal diameter were considered significant. Left ventriculography was performed in the right anterior oblique projection and myocardial segments were defined according to the criteria of the American Heart Association. The presence or absence of a contraction abnormality was visually assessed in each segment on the cineangiogram. A left ventricular wall motion abnormality was considered to be present if there were one or more segments with dyskinesis (paradoxical systolic expansion), akinesis (no systolic wall motion) or severe hypokinesis (minimal systolic wall motion). An aneurysm was defined as a circumscribed dilatation of the ventricle with dyskinesis.

Informed consent was obtained from all patients. There were no complications resulting from any of the procedures.

The data were analyzed using Fisher's exact test for 2 x 2 contingency tables.

## Results

The 35 patients were divided into two groups. Group 1 consisted of 10 patients with exercise-induced ST-segment elevation in leads without Q waves; group

### Table 1. (Continued)

<table>
<thead>
<tr>
<th>Thallium-201 perfusion scanning</th>
<th>Stenosis at cardiac catheterization (%)</th>
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<tr>
<td><em>ST</em></td>
<td>2nd Site</td>
</tr>
<tr>
<td>+</td>
<td>-</td>
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<td>+</td>
<td>-</td>
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Table 2. Summary of Results

<table>
<thead>
<tr>
<th></th>
<th>Group 1 (n = 10)</th>
<th>Group 2 (n = 25)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reversible thallium defect corresponding to ST elevation</td>
<td>10</td>
<td>13</td>
<td>&lt; 0.02</td>
</tr>
<tr>
<td>ST-segment depression in reciprocal leads</td>
<td>7</td>
<td>11</td>
<td>NS</td>
</tr>
<tr>
<td>Thallium defect corresponding to site of ST depression</td>
<td>1</td>
<td>8</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td>Left ventricular wall motion abnormality</td>
<td>3</td>
<td>25</td>
<td>&lt; 0.0005</td>
</tr>
</tbody>
</table>

2 consisted of 25 patients with ST elevation over resting Q waves.

Group 1

The results are summarized in table 2. Stable angina pectoris was present in all 10 patients. The ECG showed no evidence of myocardial infarction in nine and inferior Q waves in one. On exercise, all 10 patients developed angina and ST-segment elevation in leads without Q waves on the resting ECG. In the nine patients with anterior ST elevation, the exercise thallium scan showed an anteroseptal myocardial perfusion abnormality that decreased in size on the 4-hour scan (fig. 1). The redistribution was complete in five patients and partial in five. The ECG and the thallium scan of a patient with exercise-induced anterior ST-segment elevation are shown in figure 2. At coronary arteriography the left anterior descending coronary artery was severely obstructed in all nine patients (≥ 90% in eight and 75% in one). The location of obstruction was before the first perforator in eight patients and associated collaterals were present in only two. The ST segments were elevated inferiorly in one patient who showed an inferior exercise myocardial perfusion abnormality with complete redistribution at 4 hours. At arteriography, a 90% obstruction of the right coronary artery with no collaterals was seen.

Seven patients had exercise-induced ST-segment depression in leads reciprocal to those showing ST elevations (fig. 2A). On the thallium scan, a perfusion abnormality corresponding to the site of ST-segment depression was present in only one of these patients (fig. 3). In three patients, left ventricular wall motion abnormalities were present on ventriculography.

Group 2

The results are summarized in table 2. On exercise, 19 patients developed angina and all 25 developed ST-segment elevation in leads with Q waves on the resting ECG. The exercise thallium-201 myocardial perfusion scan showed an extensive perfusion defect corresponding to the site of Q waves and ST-segment elevation in all 25 patients. The defect persisted in all patients on the 4-hour redistribution scan, but in 13 patients (52%), the defect was smaller on the 4-hour scan than on the exercise scan. Significantly fewer patients with Q waves showed reversible thallium defects at the site of ST elevation than patients without Q waves (52% vs 100%, p < 0.02) (fig. 1). Both the ECG site of Q wave/ST elevation and the myocardial perfusion defect on scanning corresponded to the site of coronary artery obstruction in all 25 patients.

The ECG and the thallium scan of a patient with exercise-induced ST elevation in leads with resting Q waves are shown in figure 4.

Associated exercise-induced ST-segment depression in other leads (fig. 4A) was found in 11 of the 25 patients with exercise-induced ST-segment elevation over Q waves (table 2). A second perfusion defect at the site of ST-segment depression was present in eight of these 11 patients (73%), compared with one of the seven patients (14%) with ST depression in group 1 (p < 0.05) (fig. 3).

In all 25 patients, left ventricular wall motion abnormalities corresponded to the ECG site of Q waves and exercise-induced ST elevation. Two patients were found to have a left ventricular aneurysm and 23 akinetic or severely hypokinetic segments.

Discussion

This study suggests that in patients who have not suffered a transmural myocardial infarction the site of
exercise-induced ST-segment elevation rather than the site of associated ST-segment depression indicates the site of transient myocardial ischemia. ST-segment elevation on exercise corresponded to both the site of perfusion defect on thallium scan and severe obstruction on arteriography. Previous studies suggest that exercise-induced ST-segment depression poorly localizes the site of myocardial ischemia. The site of transient ST-segment elevation both at rest and on exercise predicts the site of coronary artery obstruction more accurately and is confirmed in our patients. The appearance of associated ST-segment depression has usually been disregarded and assumed not to predict myocardial ischemia at that site. In group 1 patients with both exercise-induced ST-segment elevation and associated ST depression, a perfusion abnormality at the site of ST-segment depression was uncommon on the thallium scan, and suggests that the ST depression was reciprocal to ST elevation in other leads instead of being associated with transient myocardial ischemia at that site.

Previous studies suggest that ST-segment elevation may represent a more severe degree of myocardial ischemia than ST-segment depression. Prinzmetal et al. showed in dogs that severe myocardial ischemia produced ST-segment elevation rather than depression, but when both the extent and degree of the myocardial ischemia were decreased, ST-segment depression rather than elevation usually occurred. Guyton et al. showed that ST-segment elevation represents severe transmural myocardial ischemia. During exercise electrocardiography, ST-segment elevation has been associated with severe obstruction of the corresponding coronary artery in patients both with and without previous myocardial infarction.

Our data suggest that the exercise-induced ST-segment elevation in leads without Q waves on the resting ECG is associated with severe myocardial ischemia. In nine of the 10 patients, the coronary artery was at least 90% obstructed, and when the left anterior descending coronary vessel was involved, the obstruction was usually before the first perforator and collaterals were uncommon. In all these patients a large perfusion defect was found, which returned to normal at 4 hours in only 50% of the patients. This incomplete redistribution of thallium at 4 hours in patients without clinical evidence of previous myocardial ischemia was also seen in patients with previous myocardial infarction.
Exercise-induced ST-segment depression may be either reciprocal to ST-segment elevation occurring in other leads\(^{21}\) or primary and due to underlying myocardial ischemia.\(^{16},^{17}\) After myocardial infarction, the combination of ST-segment elevation over Q waves and associated ST-segment depression is thought to predict multivessel disease.\(^{19},^{20}\) A second thallium defect corresponding to the site of ST-segment depression was found in 73% of our patients with both ST-segment elevation over Q waves and ST-segment depression in other leads, suggesting that the ST-segment depression in these patients is a primary change rather than reciprocal to ST elevation and that it represents another area of myocardial ischemia.

References

Exercise-induced ST-segment elevation. Correlation of thallium-201 myocardial perfusion scanning and coronary arteriography.
R F Dunn, I K Bailey, R Uren and D T Kelly

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