The Clinical Significance of Exercise-induced ST-segment Elevation

ROBERT A. CHAHINE, M.D., ALBERT E. RAIZNER, M.D., TETSUO ISHIMORI, M.D.

SUMMARY The significance of exercise-induced ST-segment elevation remains unsettled. We reviewed the treadmill tests of 840 consecutive patients and exercise-induced ST-segment elevation was noted in 29 (3.5%). Only eight of these (28%) stopped because of angina. Anterior myocardial infarction (AMI) was found on the resting electrocardiogram in 25 (85%). Angiographic studies performed on 21 showed critical lesions of the left anterior descending (LAD) in 19 (90%) and left ventricular aneurysm in 18 (86%). When all the patients who had AMI or critical LAD obstruction during the study period were reviewed, only 22% and 18% respectively showed exercise-induced ST-segment elevation, while 64% of the cases with left ventricular aneurysm displayed this phenomenon. Thus, exercise-induced ST elevation seems to reflect the presence of severe coronary artery disease most commonly with an associated left ventricular aneurysm and may relate more to the abnormal wall motion than to the myocardial ischemia per se.

INVESTIGATORS have yet to agree on the significance of exercise-induced ST-segment elevation. This phenomenon has been described in association with: 1) severe ischemic heart disease and usually during an unstable phase, 2) Prinzmetal's variant angina, 3) left ventricular aneurysm, 4) abnormal cardiac hemodynamics with elevated left ventricular end-diastolic pressure and poor ejection fraction, 5) asymptomatic healthy individuals studied in the Seattle Heart Watch. There seems to be no readily available information as to the relative frequency of these associations. The present study was undertaken in order to determine the incidence of exercise-induced ST-segment elevation in a population of male patients being evaluated for cardiovascular disease and to assess the diagnostic value of this phenomenon with regard to the various possible underlying conditions.

Methods

The experience in our exercise laboratory during 18 consecutive months was reviewed. The exercise tests were performed on a motor driven treadmill according to the Bruce protocol. 

From the Department of Medicine, Baylor College of Medicine and the Section of Cardiology at the Veterans Administration Hospital, Houston, Texas. Supported in part by VA Research Grant 580-103-0455. Presented in part at the 47th Annual Scientific Sessions of the American Heart Association, Dallas, Texas. For reprints: Robert A. Chahine, M.D., Chief, Section of Cardiology, Veterans Administration Hospital, 200 Holcombe Boulevard, Houston, Texas 77211.

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A 12-lead electrocardiogram was obtained within an hour before the exercise test on all patients. Two electrocardiographic leads consisting of modified bipolar leads V5 and V6 of the standard electrocardiogram were continuously monitored. Resting tracings of these two leads were recorded both in the sitting and the standing positions before starting the exercise test. During exercise, strips were obtained at one-minute intervals, at stopping and subsequently at three-minute intervals until return to baseline. The blood pressure was determined every three minutes before increasing the exercise load from one stage to the next, using a standard clinical sphygmomanometer. The patients were exercised to symptomatic end points: moderately severe angina, dyspnea, or exhaustion. Informed consent was obtained to all the studies performed.

We have selected for this study all treadmill tests where ST elevation of 1 mm or more occurred at any phase of the exercise test in either or both of the monitored leads. We included all tests where ST elevation occurred bringing the J point 1 mm or more above the isoelectric line in cases starting with normal or depressed ST segment (fig. 1) or 1 mm or more above baseline in cases starting with initial ST elevation (fig. 2).

Results

There were 1,050 consecutive treadmill tests performed on 840 patients during the study period. Of these, 29 patients (3.5%) showed exercise-induced ST elevation satisfying our criteria. The mean age of these patients was 51 years with a range of 29 to 60 years.

Resting Electrocardiographic Data

Of the 29 patients with exercise-induced ST elevation, 25 (85%) had evidence of old transmural anterior myocardial infarction. Of these, 13 were read as anterolateral and 12 as anteroseptal myocardial infarcts. Of the 25 patients with anterior myocardial infarction, 9 (36%) had definite persistent ST-segment elevation suggestive of left ventricular aneurysm.\(^4\) A representative example is shown in figure 3. Five patients (20%) had no ST elevation at all on the resting 12-lead electrocardiogram. A representative example is shown in figure 4. The remaining 11 patients (44%) had questionable resting ST elevation compatible with aneurysm. In these 11 patients, the ST-segment elevation was suble and the J point was less than 1 mm above baseline. A representative example is shown in figure 5.

Treadmill Exercise Data

The mean exercise duration ± the standard error for the 29 patients with ST elevation was 4 minutes 58 seconds ± 28 seconds. The mean resting heart rate was 95 ± 4 beats/minute and peak exercise heart rate was 149 ± 4 beats/minute, which constitutes more than 85% of the maximal heart rate predicted for the mean age of these patients. The mean resting systolic pressure was 118 ± 4 mm Hg, the mean maximal systolic pressure attained was 143 ± 5 mm Hg and the mean maximal pressure rate product was 213 ± 12 × 10³. Using the Bruce formulas,\(^5\) the left ventricular impairment adjusted for age was 36% and heart rate impairment was 15% for the group as a whole, indicating moderately severe inotropic and mild to moderate chronotropic impairment.

An analysis of the end points that resulted in the stopping of the treadmill in these 29 patients showed that chest pain was the end point in only eight patients (28%). These patients were compared to a series of 75 consecutive patients tested during the same period who manifested unequivocal ischemic ST depression. Of these, 45 (60%) developed an-
FIGURE 3. Representative example of a 12-lead electrocardiogram showing old anterior myocardial infarction with definite persistent ST-segment elevation in the precordial leads suggestive of left ventricular aneurysm.

FIGURE 4. Representative example of a 12-lead electrocardiogram showing old anterolateral myocardial infarction with question persistent ST-segment elevation.

FIGURE 5. Representative example of a 12-lead electrocardiogram showing old anterolateral myocardial infarction with question persistent ST-segment elevation.

ginal chest pain as the end point. The difference between these two groups was statistically significant ($P < 0.01$). The remaining 21 patients with exercise-induced ST elevation had no chest pain during exercise even though 20 (95%) achieved 85% of their predicted maximal heart rate. Those patients stopped because of dyspnea and/or fatigue.

Catheterization and Angiographic Data

Catheterization and angiographic data were available in 21 of the 29 patients and are summarized in Table 1. A coronary artery lesion was considered critical if it resulted in a decrease in lumen diameter of 75% or more. A ventricular aneurysm was defined as a clearly dyskinetic myocardial segment or a well demarcated area of akinesis adjacent to normally moving myocardium and resulting in a bulge. The ventricular angiograms were obtained routinely in the right anterior oblique projection. An additional left anterior

<table>
<thead>
<tr>
<th>Table 1. Catheterization and Angiography Data in 21 Patients</th>
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<tr>
<td><strong>Hemodynamics</strong></td>
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<tr>
<td>Elevated LVEDP (rest &amp; stress)</td>
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<tr>
<td>Elevated LVEDP (stress only)</td>
</tr>
<tr>
<td><strong>Coronary angiography</strong></td>
</tr>
<tr>
<td>Main left coronary: borderline lesions</td>
</tr>
<tr>
<td>Left anterior descending: critical proximal lesion</td>
</tr>
<tr>
<td>Left circumflex: major, critical lesions</td>
</tr>
<tr>
<td>Right coronary: major, critical lesions</td>
</tr>
<tr>
<td>Collaterals: good in</td>
</tr>
<tr>
<td>Three vessel disease</td>
</tr>
<tr>
<td>Two vessel disease</td>
</tr>
<tr>
<td><strong>Left ventricular angiography</strong></td>
</tr>
<tr>
<td>Apical aneurysm</td>
</tr>
<tr>
<td>Apical hypokinesis</td>
</tr>
<tr>
<td>Normal</td>
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oblique view of the left ventricle was obtained only when the possibility of aneurysmectomy was contemplated.

Summary

The above data show that the correlates of exercise-induced ST-segment elevation in the leads monitored in our laboratory are the following: 1) anterior myocardial infarction (85%), 2) critical proximal left anterior descending obstruction (90%), and 3) left ventricular apical aneurysm (86%).

In order to assess the relative importance of each of these correlates, we reviewed all the cases that had evidence of anterior myocardial infarction on the resting pre-treadmill electrocardiogram during the study period (N = 113), those which had critical proximal left anterior descending obstruction (N = 104) and those which had left ventricular apical aneurysm (N = 28). The occurrence of exercise-induced ST elevation in each of these groups is presented in table 2. Thus, exercise-induced ST elevation is not a very sensitive indicator of anterior myocardial infarction or critical left anterior descending disease, but is a reasonably sensitive detector of left ventricular apical aneurysm.

A review of the treadmills of the ten patients with left ventricular aneurysm, who did not manifest exercise-induced ST elevation satisfying our criteria, showed four developing subtle ST elevation of less than 1 mm with exercise; these had a mean maximal heart rate of 136 beats per minute. Another four had normalization or flattening of initially negative T waves. Only two had exercise-induced ST depression in at least one of the monitored leads.

Discussion

Exercise-induced ST-segment elevation is an abnormal response that has been associated with various forms of ischemic heart disease. Since ST-segment depression is the classical response to exercise in ischemic heart disease,10,11 additional implications have been ascribed to the occurrence of ST-segment elevation. Both instability and severity of the ischemia have been invoked as possible causative factors.1 The phenomenon has also been observed in patients with left ventricular aneurysm,6,13 and was described and stressed in the diagnosis of Prinzmetal’s variant angina.5,14

While the occurrence of exercise-induced ST elevation in normal individuals is admittedly rare,4,18 there is no available information regarding the relative incidence of the three previously mentioned associations. Our data suggest that the most commonly observed condition that is associated with exercise-induced ST elevation in a clinical exercise laboratory is left ventricular aneurysm. This finding during a noninvasive test could therefore be helpful in the diagnosis of left ventricular aneurysm which usually necessitates invasive angiography for final documentation. In our study, exercise-induced ST elevation appears to be a relatively sensitive indicator of left ventricular aneurysm since about two-thirds of the patients found to have such aneurysms upon angiography demonstrated this electrocardiographic abnormality on exercise testing. The fact that our data seem to indicate a higher sensitivity than previously reported may be partly explained by the fact that we monitored two apical leads. In nine of the 29 patients with exercise-induced ST elevation, the phenomenon was observed in only one of the monitored leads. In either case, exercise testing appears to be more sensitive than the 12-lead resting electrocardiogram where only 36% of left ventricular aneurysms were identifiable by definite persistent ST elevation suggestive of the diagnosis. On the other hand, the exercise-induced ST elevation is also more specific for the diagnosis of aneurysm (86%) than the resting electrocardiogram where a bulge was found on angiography in only one patient of three who had persistent ST elevation.14 Although not all the patients with exercise-induced ST elevation and left ventricular aneurysm are surgical candidates for aneurysmectomy, this information may be of value in identifying a subgroup of patients with coronary artery disease who are disabled by dyspnea without angina, in whom cardiac surgery is only indicated if an aneurysm is present.

Our data show an impressive association of anterior myocardial infarction, critical proximal left anterior descending obstruction and left ventricular aneurysm with exercise-induced ST-segment elevation. However, exercise-induced ST elevation in the population of patients with anterior myocardial infarction or left anterior descending disease occurs too infrequently (22% and 18%, respectively) to permit speculation on a cause and effect relationship with these lesions. The presence of a left ventricular aneurysm seems to be a more important factor that could be implicated in the causation of this electrocardiographic phenomenon. Further, the less frequent occurrence of anginal chest pain in patients manifesting exercise-induced ST elevation compared to those with exercise-induced ischemic ST depression, though unexplained, suggests the possibility that the ST elevation may relate more to the presence of abnormal wall motion than to myocardial ischemia per se.

Of the three patients who developed ST elevation with exercise in the absence of left ventricular aneurysm, one had a history of resting angina but a diagnosis of Prinzmetal’s variant could not be attained because of unavailability of documentation of ST elevation with spontaneous chest pain. All three patients had ST elevation during one treadmill, but failed to demonstrate this phenomenon on subsequent treadmills performed between one week and three months later. In contradistinction, the patients with left ventricular aneurysm, who had repeat treadmill tests, demonstrated ST elevation repeatedly as long as they attained 85% of their predicted maximal heart rate.

We therefore conclude that the exercise-induced ST elevation most commonly reflects severe coronary artery disease with associated left ventricular aneurysm. This phenomenon probably relates more to the presence of abnormal wall motion than to the myocardial ischemia per se. Finally, treadmill testing may be a helpful noninvasive technique in the diagnosis of left ventricular aneurysm.

<table>
<thead>
<tr>
<th>Number of patients</th>
<th>Exercise-induced ST elevation</th>
<th>Percent</th>
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<tbody>
<tr>
<td>Anterior myocardial infarction</td>
<td>113</td>
<td>25</td>
</tr>
<tr>
<td>Critical proximal LAD obstruction</td>
<td>104</td>
<td>19</td>
</tr>
<tr>
<td>Left ventricular apical aneurysm</td>
<td>28</td>
<td>18</td>
</tr>
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Correlation of Pathologic Q Waves on the Standard Electrocardiogram and the Epicardial Electrogram of the Human Heart

MONTY M. BODENHEIMER, M.D., VIDYA S. BANKA, M.D., ROBERT G. TROUT, M.D., HOMAYOON PASDAR, M.D., AND RICHARD H. HELFANT, M.D.

SUMMARY To evaluate the relationship between abnormal Q waves on the standard ECG and localized ventricular excitation, unipolar epicardial electrograms were recorded over the left ventricle during aortocoronary bypass surgery in 36 patients. Of 20 without standard ECG Q waves, six had abnormal epicardial Q waves, three anteriorly and three inferiorly. Of 16 patients with standard ECG Q waves, four had both precordial and anterior epicardial Q waves while seven had Q waves in leads III and aVF and inferior epicardial Q waves. Three of the 14 had Q waves in both precordial and inferior leads of the ECG but epicardial Q waves only from the antero-apical region in two and only from the inferior wall in one. Two patients with Q waves in both III and aVF had no epicardial Q waves. Thus, the standard electrocardiogram underestimates epicardial Q waves. If Q waves are present in the standard ECG, they correlate with the presence, although not invariably the location of Q waves on the epicardial electrogram.

ALTHOUGH PATHOLOGIC STUDIES HAVE INDICATED that a correlation exists between pathologic Q waves on the standard ECG and myocardial fibrosis, these fibrotic areas frequently do not correspond either to presence of Q waves or to the predicted site. Recently, ventriculographic studies in living man have indicated that the presence of standard electrocardiographic Q waves correlate closely with the presence of ventricular asynergy. However, although quite specific, standard electrocardiogram Q waves were insensitive predictors since they were absent in approximately half of the patients with asynergic zones demonstrated ventriculographically. These findings appeared related primarily to severity since more marked degrees of asynergy were usually associated with Q waves. In addition, the location of asynergy was an important factor in that inferior wall Q waves were less sensitive and less specific than anterior wall Q waves.

Kaiser et al. have demonstrated that intraoperative epicardial electrograms are useful in delineating zones of myocardial fibrosis. To better define the relationship between the presence or absence of Q waves on the standard electrocardiogram and the underlying localized ventricular excitation characteristics of the left ventricular myocardium, intraoperative epicardial electrograms were obtained in 36 patients undergoing surgery for coronary artery disease and these recordings correlated with the standard ECG Q waves.
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R A Chahine, A E Raizner and T Ishimori

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