Effects of Physical Training
on Exertional S-T-Segment Depression
in Coronary Heart Disease

By Jean-Marie Detry, M.D., and Robert A. Bruce, M.D.

SUMMARY
Symptom-limited maximal oxygen intake ($V_{O_2 \text{SL}}$) and electrocardiographic responses
to multistage treadmill test were determined before and after 3 months of physical
training in 14 patients with coronary heart disease. Six patients also had hemodynamic
studies done at several submaximal work loads in the upright position before and
after training. S-T-segment responses were measured by computer averaging of 100-
beat samples.

After physical training, the heart rate ($P < 0.01$) and the product of heart rate
and systolic blood pressure ($P < 0.02$) at submaximal exercise were lower and at-
tended by significantly less S-T-segment depression. At maximal exercise, however,
the heart rate ($P < 0.01$) and the product of heart rate and systolic blood pressure
($P < 0.02$) were higher after training, and the S-T-segment depression was more
pronounced ($P < 0.005$). The quantitative relationships of S-T-segment depression to
either exercise heart rate, product of heart rate and systolic blood pressure, or to
pressure-rate product (product of heart rate and mean blood pressure) were unaffect-
bly by physical training. The $V_{O_2 \text{SL}}$ increased by 20.8% ($P < 0.001$); the product of
heart rate and systolic blood pressure at symptom-limited exercise increased by 10%
($P < 0.005$) in the angina patients, which may indicate a higher angina threshold
after physical training.

It is concluded that physical training of coronary patients changes S-T-segment
responses to submaximal and maximal exercise: these modifications presumably result
from changes in heart-rate and blood-pressure responses to exercise, rather than from
an improved coronary circulation.

Additional Indexing Words:
Hemodynamic studies Multistage treadmill test

A given level of submaximal exercise,
physical training of patients with coro-
nary heart disease (CHD) decreases heart
rate, blood pressure, pressure-rate product, tension-time index, and presumably myocar-
dial oxygen requirements ($MV_{O_2}$). After
physical training, the magnitude of the S-T-
segment depression at a given submaximal
level of exercise is also diminished, which
may be related to a lower heart rate.

A diminution of the S-T-segment depression
at the same level of submaximal exercise with
physical training may indicate either an
improvement of the coronary circulation or a
change in the hemodynamic determinants of

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the S-T response. Only a significant modification of the relationship between the amount of S-T-segment depression during exercise and appropriate indices of MV\textsubscript{O2} might permit inferences about the coronary circulation from electrocardiographic data. This assessment requires quantitative data collected at two or more exercise levels before and after training, in contrast to previous studies based on comparisons at only one submaximal level of exercise.

The purpose of this study was to determine whether the relationship of the magnitude of S-T-segment depression during exercise to its hemodynamic determinants was altered with physical training in patients with coronary heart disease.

### Material and Methods

Of 14 male patients (34 to 68 years of age with a mean age of 50 years) with coronary heart disease, nine had exertional angina pectoris, while five were free of angina at all levels of exercise (table 1). Patients with hypertensive disease, ventricular aneurysm, clinically manifest heart failure, or digitalis therapy were excluded from this study.

Six of these patients also had hemodynamic studies at rest and at selected submaximal exercise levels which were reported elsewhere; two other patients from this previous study were excluded since one had a right bundle-branch block and the other low-voltage QRS complexes in the bipolar lead which prevented satisfactory triggering of the computer averaging circuit.

Signed informed consent was obtained from each patient before initiating the study. After data were obtained from history, physical examination, 12-lead supine electrocardiogram, and chest X-ray for measurement of heart volume, each patient performed a multistage treadmill test of maximal exertion. Speed and grade of the treadmill were increased every 3 min until a self-determined limit of maximally tolerated fatigue, dyspnea, and/or chest pain was reached. A continuous magnetic tape recording of a bipolar (V\textsubscript{5} to inferior right scapula) precordial lead was obtained for 3 min when the patient was sitting at rest before the test, throughout the entire exercise period, and for the first 6 min of recovery in the sitting position. Blood pressure was measured by sphygmomanometer before the test, after 2½ min of exercise (end of stage 1), and during the first seconds of recovery.

The symptom-limited maximal oxygen intake (MV\textsubscript{O2,1}) was determined from consecutive 1-min samples of expired air collected through a low-resistance open circuit during the last 3 or 4 min of exercise. S-T responses were measured by computer averaging of 100-beat samples obtained at rest, at the end of each exercise stage, and at maximal exercise, at initial recovery, and at each minute of 6 min of recovery.

The protocol and the methods for the hemodynamic study were reported previously. Briefly, the patients had cardiac output by direct Fick principle with intravascular pressure measurements at rest and at selected submaximal exercise levels. A bipolar electrocardiographic lead was

### Table 1

<table>
<thead>
<tr>
<th>No.</th>
<th>Name</th>
<th>Age (yr)</th>
<th>Heart volume (ml)</th>
<th>Clinical history</th>
</tr>
</thead>
<tbody>
<tr>
<td>1*</td>
<td>A.D.</td>
<td>34</td>
<td>1277</td>
<td>MI May 1969, Nov 1969; angina pectoris</td>
</tr>
<tr>
<td>2*</td>
<td>B.W.</td>
<td>50</td>
<td>727</td>
<td>MI Oct 1969; angina pectoris</td>
</tr>
<tr>
<td>3*</td>
<td>B.C.</td>
<td>42</td>
<td>737</td>
<td>MI Dec 1969; angina pectoris</td>
</tr>
<tr>
<td>4</td>
<td>B.H.</td>
<td>54</td>
<td>831</td>
<td>MI July 1968; angina pectoris</td>
</tr>
<tr>
<td>5</td>
<td>C.K.</td>
<td>61</td>
<td>1037</td>
<td>MI Oct 1969</td>
</tr>
<tr>
<td>6</td>
<td>D.P.</td>
<td>53</td>
<td>631</td>
<td>Angina pectoris since 1967</td>
</tr>
<tr>
<td>7</td>
<td>D.J.</td>
<td>43</td>
<td>-</td>
<td>MI Nov 1969</td>
</tr>
<tr>
<td>8</td>
<td>H.D.</td>
<td>61</td>
<td>1015</td>
<td>Angina pectoris since May 1969</td>
</tr>
<tr>
<td>9*</td>
<td>K.A.</td>
<td>46</td>
<td>759</td>
<td>MI Sept 1969</td>
</tr>
<tr>
<td>10*</td>
<td>K.W.</td>
<td>38</td>
<td>855</td>
<td>MI Sept 1969</td>
</tr>
<tr>
<td>11</td>
<td>O.F.</td>
<td>51</td>
<td>844</td>
<td>Angina pectoris since Nov 1969</td>
</tr>
<tr>
<td>12</td>
<td>S.W.</td>
<td>50</td>
<td>616</td>
<td>MI July 1969; angina pectoris</td>
</tr>
<tr>
<td>13</td>
<td>St.W.</td>
<td>50</td>
<td>1264</td>
<td>MI March 1968, Sept 1969</td>
</tr>
<tr>
<td>14*</td>
<td>S.G.</td>
<td>68</td>
<td>797</td>
<td>MI 1963, May 1969; angina pectoris</td>
</tr>
</tbody>
</table>

Abbreviation: MI = myocardial infarction.
*These cases had hemodynamic studies.
Table 2

Multistage Treadmill Test Data in 14 Patients

<table>
<thead>
<tr>
<th>State</th>
<th>Study done</th>
<th>Before training</th>
<th>After training</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rest</td>
<td>HR</td>
<td>70</td>
<td>68</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>SBP</td>
<td>118</td>
<td>115</td>
<td>NS</td>
</tr>
<tr>
<td>HR × SBP</td>
<td>100</td>
<td>83</td>
<td>78</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>S-Tb</td>
<td>+0.035</td>
<td>+0.061</td>
<td>NS</td>
</tr>
<tr>
<td>Submaximal exercise (stage I)*</td>
<td>HR</td>
<td>117</td>
<td>107</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td></td>
<td>SBP</td>
<td>150</td>
<td>144</td>
<td>NS</td>
</tr>
<tr>
<td>HR × SBP</td>
<td>100</td>
<td>179</td>
<td>155</td>
<td>&lt;0.02</td>
</tr>
<tr>
<td></td>
<td>S-Tb</td>
<td>−0.095</td>
<td>−0.027</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Maximal exercise</td>
<td>HR</td>
<td>141</td>
<td>147</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td></td>
<td>SBP</td>
<td>159</td>
<td>163</td>
<td>NS</td>
</tr>
<tr>
<td>HR × SBP</td>
<td>100</td>
<td>226†</td>
<td>241†</td>
<td>&lt;0.02</td>
</tr>
<tr>
<td></td>
<td>S-Tb</td>
<td>−0.202</td>
<td>−0.262</td>
<td>&lt;0.005</td>
</tr>
<tr>
<td></td>
<td>VO2</td>
<td>22.05</td>
<td>26.63</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Early recovery</td>
<td>HR</td>
<td>121</td>
<td>129</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>S-Tb</td>
<td>−0.098</td>
<td>−0.129</td>
<td>NS</td>
</tr>
</tbody>
</table>

Abbreviations: HR = heart rate; SBP = systolic blood pressure; S-Tb = mean voltage difference; VO2 = oxygen intake at symptom-limited maximal exercise.

*Only 13 patients.
†In the nine patients with exercise angina pectoris, these values were 211 before training and 232 after training (P < 0.005).

recorded on magnetic tape during the last 2 to 3 min of each exercise level. The mean arterial blood pressure and the heart rate were recorded at the same time as the electrocardiographic data. The product of the mean peripheral blood pressure and the heart rate (pressure-rate product) was used as an index of myocardial oxygen consumption (MV02).6,7

S-Tb designates the mean voltage difference from the S-T segment 50 to 69 msec after the nadir of S wave relative to that of the P-R segment 20 to 39 msec prior to the nadir of Q in 100 consecutive QRS-T complexes for each period of observation.17

The physical training program involved individually graded exercise (walking, jogging, calisthenics) for 45 min, three times a week for 3 months.1 The same experimental protocol was utilized after 3 months of physical training. Significances of the differences were analyzed by the paired t-test.

Results

The symptom-limited V02s,L increased by 20.8% (P < 0.001) after physical training (table 2). At maximal exercise, the heart rate (P < 0.01) and the product of heart rate and systolic pressure (P < 0.02) were higher after physical training and were attended by a greater depression of the S-T segment (P < 0.005). At submaximal exercise levels (third minute of stage I) however, the heart rate (P < 0.01), the systolic blood pressure (NS), and their product (P < 0.02) were lower after physical training and the S-T segment was less depressed (P < 0.05). During the recovery from more strenuous exertion, after physical training, the S-T-segment depression was greater, but these modifications were not significant (fig. 1).

The relationship of the magnitude of S-T-segment depression to the heart rate (fig. 2) or to the product of heart rate and systolic blood pressure during the multistage treadmill test was not altered by physical training (fig. 3). Neither was the relationship between the magnitude of the S-T-segment depression and the pressure-rate product modified by physical training in the patients who had hemodynamic studies during bicycle exercise at several

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submaximal levels (fig. 4). Only one patient (A.D.) failed to exhibit any relationship between the pressure-rate product and the magnitude of exercise S-T-segment depression, either before or after training; this patient had the largest heart volume.

Discussion

The major finding of this study is that physical training did not alter the relationship between the magnitude of the S-T-segment depression during exercise and the pressure-rate product used as an index of MV_{O_2}. The relationship of the magnitude of the S-T-segment depression during the multistage treadmill test to the heart rate or to the product of heart rate and systolic blood pressure was not affected either. Accordingly, the changes in the S-T-segment response to submaximal and maximal exercise after training appear to result from changes in the hemodynamic response to exercise rather than from alterations in myocardial oxygen supply.

The pressure-rate product is an incomplete index of the MV_{O_2} since it does not take into account other important determinants of MV_{O_2}, namely heart volume, myocardial contractility, and systolic ejection time. The relative importance of the time during which tension is maintained in the determination of MV_{O_2} is unclear. A prolongation of the systolic ejection time after physical training appears unlikely since physically active patients with CHD have shorter ejection-time index than sedentary cardiac patients, and in middle-aged healthy subjects the ejection-time index was decreased after physical training. Although heart volume at rest is unaffected by physical training of patients with coronary

**Figure 1**

Effect of physical training on S-T-segment depression during multistage treadmill test and ensuing recovery in 14 patients with coronary heart disease. Stage 1 refers to data collected during the third minute of the first stage of the test (grade of 10% and speed of 1.7 mph). ST_B is the mean voltage from 50 to 69 msec after the nadir of S wave, with the P-R interval as zero reference voltage.

**Figure 2**

Relationship of S-T-segment depression to heart rate during multistage treadmill test before (left) and after (center) physical training in 14 patients with coronary heart disease. Data collected during exercise (third minute of stage 1 and maximal exercise) before and after training are shown on the right; thin lines indicate the standard error of the mean.
Influence of physical training on the relationship between the magnitude of S-T-segment depression and the product of heart rate (HR) and systolic blood pressure (SBP) during multistage treadmill test in 14 patients. Submaximal exercise refers to data collected during the third minute of the first stage of the test; thin lines indicate the standard error of the mean.

After training, higher heart rates and brachial systolic blood pressures at the symptom-limited maximal exercise in the angina patients suggest a modification in the angina threshold. The latter should however be confirmed by measurements of central blood pressure and ejection time at the anginal level before and after training to appraise the adequacy of this alteration. The importance of this possible change in the angina threshold appears however to be small since the increased $V_{O2\,s.t}$ with training in angina patients results mainly from their lower pressure-rate product at submaximal exercise.

The effects of physical training on the relationship between the magnitude of the exercise S-T-segment depression and the product of systolic blood pressure and heart rate are different from those produced by sublingual nitroglycerin. Nitroglycerin indeed modifies acutely this relationship so that the magnitude of the S-T-segment depression is decreased at all levels of exercise despite no change or even an increase in the product of systolic blood pressure and heart rate. These effects of nitroglycerin are probably related to...
a decreased heart volume secondary to the peripheral effects of this drug.24, 25

It is concluded that physical training of CHD patients does not modify the relationship of the magnitude of S-T-segment depression during exercise to its hemodynamic determinants. Quantitative modifications of S-T-segment response to exercise after physical training result therefore presumably from changes in heart-rate and blood-pressure responses, rather than from an improved coronary circulation. This interpretation of the data is tentative since it assumes that neither myocardial contractility nor left ventricular volumes during exercise are changed by physical training.

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