Usefulness of the postexercise response of systolic blood pressure in the diagnosis of coronary artery disease

K. WRAY AMON, B.S., KENT L. RICHARDS, M.D., AND MICHAEL H. CRAWFORD, M.D.

ABSTRACT The normal decline in systolic blood pressure (SBP) during the recovery phase of treadmill exercise does not occur in some patients with coronary artery disease (CAD). In others the recovery values of SBP exceed the peak exercise values. To examine the diagnostic value of this observation, we studied 31 normal subjects and 56 patients undergoing treadmill exercise before coronary cineangiography. Because of large differences in peak exercise pressures between the two groups, recovery ratios were derived by dividing the SBP at 1, 2, and 3 min after exercise by the peak exercise SBP. The 1, 2, and 3 min ratios in the normal subjects declined steadily from 0.85 ± 0.07 (SD) to 0.79 ± 0.06 and to 0.73 ± 0.06, respectively, while the ratios in the patients with CAD remained elevated at 0.97 ± 0.12 to 0.97 ± 0.11 to 0.93 ± 0.13. With use of the upper limits defined by two SDs of the normal value, recovery ratios were compared with the occurrence of angina and with ST segment depression on the exercise electrocardiogram in the patients with CAD. Abnormal ratios were more frequent in patients with CAD (53/56, 95%) than in those with ST segment depression (33/56, 59%), angina (37/56, 66%), and either ST segment depression or angina (42/56, 75%). Twenty of the patients with CAD who were on no medication underwent an additional treadmill exercise test on a separate day and no significant differences were found in the ratios from the two tests. Ten additional patients with CAD underwent treadmill exercise testing while on placebo and while on a β-blocker. There were no significant differences in the ratios from the two tests. Twenty-eight of the 31 (90%) normal subjects had normal recovery ratios. We conclude that the ratios of early recovery SBP to the peak exercise SBP are more sensitive than exercise electrocardiographic changes and angina for identifying patients with CAD.


SINCE the original descriptions of electrocardiographic (ECG) changes during angina pectoris,1,2 numerous investigations have been conducted to assess the feasibility of increasing the sensitivity and specificity of the ECG for detecting ischemic heart disease. Changes in the ECG ST segment during exercise have received the most attention and a wide range of sensitivities have been demonstrated, depending on the patient population selected and other variables related to the exercise testing procedure.3–4 The diagnostic usefulness of the ST segment is decreased by pharmacologic therapy, the presence of conduction abnormalities, or left ventricular hypertrophy. Therefore, several investigators have raised concern regarding the relative usefulness of exercise-induced ECG ST segment changes when they are applied to the diagnosis of coronary artery disease (CAD).5–7

In an attempt to increase the sensitivity of exercise ECG testing for the detection of ischemic heart disease, some investigators have utilized changes in the R wave9 and the T wave.9,10 Greenberg et al.11 employed a multivariate approach in which 21 parameters were used and found a sensitivity of 85%. Whether or not the sensitivity of exercise ECG testing can be increased consistently above 85% is doubtful.

Routine exercise stress testing usually includes measurements of the patient’s blood pressure before, during, and for several minutes after exercise. The postexercise blood pressure has been recorded primarily to monitor the patient’s recovery from exertion, and thus its diagnostic implications have not been studied extensively. We have observed that some patients have
systolic blood pressures (SBPs) in the recovery period that are higher than the peak-exercise pressures, and that patients who exhibit this phenomenon often have severe CAD, as determined by selective coronary cineangiography. Accordingly, this study was undertaken to compare the postexercise SBP response in normal subjects and in patients with CAD to evaluate its diagnostic value and relationship to the severity of disease.

Methods

Patient population. The study population consisted of 77 symptomatic patients undergoing ECG treadmill exercise testing and cardiac catheterization because of suspected ischemic heart disease and 10 young asymptomatic subjects. Twenty-one of the 77 patients had atypical chest pain and normal coronary arteriograms; the addition of these patients to the 10 young subjects resulted in a normal group of 31 with an age range of 23 to 66 years (mean 44). The 56 patients with CAD had an age range of 34 to 75 years (mean 57). The mean age of the 21 catheterization-proven normal subjects was 51 years with a range of 37 to 66. Table 1 lists the clinical characteristics and medications received in the two groups. Medications were not withheld since treadmill exercise tests were performed as part of the patients’ precardiac catheterization evaluation. Patients with left bundle branch block, left ventricular hypertrophy, or other conduction abnormalities that would preclude evaluating ST wave changes with exercise on their resting ECGs were excluded. Patients with hypertension were not included in the normal or patient groups because of the potential variability in their coronary reserves. However, nine additional patients who had a history of hypertension, whose resting blood pressures were controlled by medications, and who had normal coronary arteriograms were studied as a separate group. Twenty of the patients with CAD who were on no medications underwent a second exercise test on a separate day. An additional group of 10 patients with CAD underwent treadmill exercise testing during a placebo period and while on a β-blocking medication. Cardiac catheterization. Selective coronary cineangiograms were obtained in multiple views of both the left and right coronary systems. The arteriograms were interpreted by two or more experienced angiographers who had no knowledge of other study data. A significant coronary lesion was defined as one causing greater than 50% luminal diameter narrowing of the left main coronary artery or one major epicardial coronary vessel or the proximal portion of one of its main branches. This group was then divided according to the severity of CAD. The results are listed in Table 2. Of the total group, 50 of 56 or 89% had at least one major coronary artery or a proximal branch containing 70% or greater luminal narrowing or left main coronary artery stenosis of 50% or more. Three of the five patients with significant left main disease had associated 50% or more stenosis of one other vessel, while the remaining two had concomitant three-vessel disease.

Treadmill exercise testing. Within 2 weeks of cardiac catheterization patients underwent treadmill exercise testing by a Bruce protocol[15] and with the use of a 12-lead ECG system. Subjects were encouraged to walk as long as possible on the treadmill or until predetermined indications for termination of the test were observed by the responsible physician. Reasons for stopping the test included severe chest pain, dizziness, ventricular tachycardia, fatigue, claudication, or severe dyspnea.

Blood pressures were recorded with the patient standing before exercise, during each minute of exercise, and with the patient seated immediately after exercise, as well as for 3 min during the recovery period. Blood pressures were measured with a conventional cuff sphygmomanometer. The exercise ECG was monitored and recorded at 1 min intervals during and after exercise. The exercise ECG was considered positive if there was 0.1 mV or more of J point depression and the ST segment was flat or downsloping 0.08 sec after the J point. If there were resting ST wave changes present, an additional 0.1 mV of ST segment depression was required to classify the exercise test as positive. The exercise ECGs were evaluated by two or more observers without knowledge of the other clinical data. One patient developed left bundle branch block during exercise and it persisted well into the recovery period.

Data analysis. Sensitivity and specificity were defined as follows[17]:

\[
\text{Sensitivity} = \frac{\text{True positive results}}{\text{Total patients with disease}} \times 100\%
\]

\[
\text{Specificity} = \frac{\text{True negative results}}{\text{Total patients without disease}} \times 100\%
\]

To evaluate changes in blood pressure between individuals with markedly different pressures, postexercise SBP recovery ratios were derived by dividing the pressure at each minute of recovery by that at peak exercise. Recovery heart rate ratios for each subject were determined in a similar fashion.

Statistical analysis of the measured variables between groups was by unpaired and paired Student’s t testing or chi-square analysis, as appropriate. In each case p < .05 was considered significant. All mean values shown are ± 1 SD.

Results

Treadmill exercise findings. Positive ST segment changes were found either with exercise or during the recovery period in 33 of the 56 (59%) CAD patients. In

### Table 1

**Clinical characteristics of the two study groups**

<table>
<thead>
<tr>
<th></th>
<th>Normal subjects (n)</th>
<th>CAD patients (n)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total</td>
<td>31</td>
<td>56</td>
</tr>
<tr>
<td>Male</td>
<td>25</td>
<td>56</td>
</tr>
<tr>
<td>Female</td>
<td>6</td>
<td>0</td>
</tr>
<tr>
<td>Prior MI</td>
<td>-</td>
<td>32</td>
</tr>
<tr>
<td>Propranolol therapy</td>
<td>5</td>
<td>30</td>
</tr>
<tr>
<td>Digitalis therapy</td>
<td>-</td>
<td>8</td>
</tr>
<tr>
<td>Nitrate therapy</td>
<td>2</td>
<td>22</td>
</tr>
</tbody>
</table>

MI = myocardial infarction.
the normal group four of the 31 subjects had unequivocally positive ST segment changes on their ECG both during and after exercise. All four subjects were part of the subgroup with no coronary disease by coronary angiography. The 27 remaining normal subjects had no ECG changes suggestive of ischemia. Chest pain occurred in 38 of the 56 (67%) patients with CAD during and/or after treadmill exercise testing. Two of the normal subjects had chest pain during exercise testing. Test duration ranged from 2 min to 12 min of exercise in patients with CAD and from 3 to 13½ min in the normal subjects.

Heart rate and blood pressure data. The heart rate and blood pressure results are listed in table 3. Resting and maximum exercise heart rates were significantly different between the two groups. Resting SBPs were not significantly different in the two groups, but were significantly different during maximal exercise. In six of the 56 patients with CAD a decrease in SBP of 10 mm Hg or less in the last minute of exercise was observed.

Recovery ratios. Mean SBP recovery ratios are shown in figure 1, left. The values at all 3 min of recovery were significantly different between the patients and the normal subjects. Since the recovery ratios in all normal subjects fell in a linear fashion throughout the first 3 min of recovery, upper limits for each recovery ratio were defined as two SDs above the mean value rounded off to the nearest tenth as follows: 1 min of recovery less than 1.0, 2 min of recovery less than 0.9, and 3 min of recovery less than 0.8. Also, in 32 of the 56 (57%) patients with CAD the SBP between 1 and 2 min after exercise increased or did not change. Since no normal subject exhibited this phenomenon we classified this as an abnormal blood pressure response to exercise. Thirty of the 32 with this response also had abnormal recovery ratios.

Figure 1, right, illustrates the recovery heart rate ratios for the two groups. There was a progressive decline in both groups, but the rate in normal subjects fell slightly faster than that in patients with CAD and therefore the ratios in the two groups at minute 3 of recovery were statistically different.

TABLE 3
Mean heart rate and SBP (± SD) in normal subjects and patients with CAD

<table>
<thead>
<tr>
<th></th>
<th>Resting</th>
<th>Peak</th>
<th>Resting</th>
<th>Peak</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>HR</td>
<td>HR</td>
<td>SBP</td>
<td>SBP</td>
</tr>
<tr>
<td>NL</td>
<td>31</td>
<td>82±14</td>
<td>154±38</td>
<td>113±23</td>
</tr>
<tr>
<td>CAD</td>
<td>56</td>
<td>68±17A</td>
<td>115±32A</td>
<td>119±18</td>
</tr>
</tbody>
</table>

NL = normal subjects; HR = heart rate.
A p < .001 compared with normal.

Twenty-eight of the 31 (90%) normal subjects had normal SBP recovery ratios. In two of the three with abnormal ratios they were abnormal only at 3 min of recovery, while in the third ratios were abnormal during all 3 min of the recovery period. In 53 of the 56 (95%) patients with CAD an abnormal SBP recovery ratio at some time during the postexercise period and/or a failure to decrease or an increase in the recovery ratio between minutes 1 and 2 of the recovery phase was observed. Of the three patients with falsely negative responses according to their SBP recovery ratios, one had two-vessel disease with a 100% obstruction of the right coronary artery and a 70% lesion of the left anterior descending coronary artery, and the remaining two had single-vessel disease with less than 70% luminal narrowing in one coronary artery.

The patients with CAD were grouped according to the severity of their disease and the 2 min SBP recovery ratios were examined as shown in figure 2. The mean values were progressively higher in patients with a greater number of major coronary arteries that were diseased. In addition, at 2 min of recovery no patient with three-vessel or left main disease had an SBP recovery ratio less than 0.9.

Forty-three CAD patients had resting ejection fractions of 50% or greater, with a range of 50% to 82%. The remaining 13 CAD patients had resting ejection fractions of less than 50%, with a range from 38% to 48%. SBP recovery ratios in these two patient groups were not significantly different.

Reproducibility. Twenty patients with CAD who were not taking medications underwent two treadmill
exercise tests to evaluate the reproducibility of the recovery ratios. Patients exercised for a similar amount of time on each test. The mean values for the SBP recovery ratios during test I were 1.00 ± 0.08 at 1 min of recovery, 0.96 ± 0.12 at 2 min, and 0.91 ± 0.14 at 3 min. The respective values for test II were 1.01 ± 0.12, 0.95 ± 0.12, and 0.90 ± 0.13. There were no significant differences in the ratios between test I and II, and results in none of the patients changed from positive to negative or vice versa between tests, even though two of the three patients with CAD and normal SBP recovery ratios were in this group. Also, the mean recovery ratios at each minute in this sample group were not statistically different from those in the CAD group as a whole.

**β-Blocker therapy.** The mean SBP recovery ratios for the additional 10 patients undergoing treadmill exercise testing during a placebo period and while on a β-blocking medication are shown in table 4. During administration of placebo, the SBPs of these 10 patients rose from a resting pressure of 137 ± 18 to 166 ± 25 mm Hg at peak exercise, for a mean increase of 29 mm Hg. During administration of β-blockers their SBPs went from 128 ± 19 mm Hg at rest to 151 ± 23 mm Hg at peak exercise, for a mean increase of 23 mm Hg during exercise. There were no significant differences between the SBP recovery ratios for the two tests at any time during recovery, nor were these results statistically different from those in the CAD group as a whole.

SBP recovery ratios for the 30 of 56 CAD patients taking β-blockers and the 26 of 56 not taking β-blockers are shown in figure 3. Ratios for both groups were statistically higher than those for the normal subjects at each of the first 3 min of recovery. Those on β-blockers had a mean resting SBP of 114 ± 25 mm Hg, which rose to 141 ± 24 mm Hg at peak exercise for a mean difference of 27 mm Hg. The SBP of the patients with CAD who were not on β-blockers rose from 124 ± 18 mm Hg at rest to 168 ± 28 mm Hg at peak exercise (difference of 44 mm Hg).

**Hypertensive subjects.** In the nine patients with a history of hypertension and normal coronary arteriograms, the mean SBP recovery ratio was 1.05 ± 0.19 at 1 min of recovery and decreased to 1.03 ± 0.17 at 2 min and to 0.95 ± 0.19 at 3 min. These values were

<table>
<thead>
<tr>
<th>Table 4</th>
<th>Mean SBP recovery ratios (±SD) at 1, 2, and 3 min after treadmill exercise in 10 patients both while on and off β-blockers</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1 min</td>
<td>2 min</td>
<td>3 min</td>
</tr>
<tr>
<td>Placebo</td>
<td>1.05 ± 0.05</td>
<td>0.99 ± 0.08</td>
</tr>
<tr>
<td>β-Blockers</td>
<td>1.00 ± 0.09</td>
<td>0.97 ± 0.07</td>
</tr>
</tbody>
</table>

**Figure 3.** Comparison of SBP recovery ratios in 30 patients with CAD who were on a β-blocking medication, in 26 patients not taking β-blockers, and in the 31 normal subjects.
not significantly different than those in the CAD group and all hypertensive subjects in the normal group were abnormal by recovery ratio criteria. In addition, the mean peak exercise systolic pressures were not significantly different between the hypertensive normal subjects and the normal subjects without hypertension.

Comparison of diagnostic methods. Exercise ECG ST segment changes identified 33 of the 56 (59%) patients with CAD and angina pectoris alone present in 37 (66%). If those patients with evidence on the resting ECG of a prior myocardial infarction or angina are added to the group of patients with ECG changes during exercise, 42 of the 56 (75%) patients with CAD are identified, and when all three findings are considered 46 of 56 (82%) are detected. SBP recovery ratios correctly identified 53 of 56 (95%) of the patients with CAD.

Discussion

It has been suggested that systemic arterial pressure during exercise is controlled almost totally by reflex mechanisms of the sympathetic nervous system rather than by changes in cardiac output. In patients with CAD, however, postexercise cardiac output may play a much greater role in the control of blood pressure because of improvement in myocardial asynergy during the recovery period. Rozanski et al., using upright bicycle exercise equilibrium radionuclide ventriculography in patients with CAD, found that left ventricular ejection fraction during the postexercise period was increased when compared with both peak exercise and resting levels. Even though in this study upright bicycle rather than treadmill exercise was used, it is reasonable to hypothesize that without a dramatic decrease in peripheral vascular resistance there would be an increase in blood pressure in the early postexercise period. Other investigators have shown that increases in the postexercise ejection fraction of the left ventricle may be an indicator of myocardial viability because of improvement in the wall motion of segments abnormal at rest. Thus, an increase in postexercise SBP in patients with CAD may indicate ischemic left ventricular dysfunction during exercise. Profound decreases in SBP during exercise have been observed in some patients with CAD who presumably develop severely impaired left ventricular function during exercise-induced myocardial ischemia. None of our patients with CAD had decreases of greater than 10 mm Hg. Therefore, the increases in SBP recovery ratios during the postexercise period in our patients were not dependent on marked decreases in blood pressure during exercise.

Ellestad noted that in normal subjects the SBP from 1 min after treadmill exercise throughout the recovery period declined steadily. This uniform decline throughout the postexercise period is consistent with our findings in normal subjects. Using the ratios of recovery SBP to peak exercise SBP (recovery ratios) greater than 1.0, 0.9, and 0.8 at 1, 2, and 3 min of recovery, respectively, enabled us to identify patients with CAD with an excellent degree of sensitivity (95%). In addition to this high sensitivity, the specificity of recovery ratios was 90%. Furthermore, an increase in recovery ratio from 1 to 2 min after exercise was more prevalent in patients with severe multivessel disease. Substitution of the maximum SBP achieved during exercise for the peak exercise systolic pressure in the derivation of recovery ratios did not significantly change the data. The fact that 10 patients with CAD underwent exercise testing while both off and on B-blocking medications and had abnormal recovery ratios in both cases suggests that recovery ratios are a sensitive method of diagnosing CAD even when patients are taking B-blockers. The usefulness of recovery ratios for diagnosis also seemed independent of the duration of the exercise test since several patients exercised for only a few minutes, but still had positive test results.

The heart rate recovery ratios declined slightly faster in the normal subjects and were significantly larger in the patients with CAD at 3 min after exercise. This may be explained by the fact that the peak exercise heart rates in the normal subjects were much higher than in the patients. If the values for both groups decreased to resting levels in the same period of time, the larger change in the values for the normal subjects would mean that their heart rate recovery ratios would decrease faster than those of the patients. This concept was also illustrated by the SBP response in patients with CAD. The patients who were not on B-blockers had higher SBPs during exercise than those on B-blockers. This was presumably because of the fact that the patients not on B-blockers had lesser degrees of CAD and could exercise longer as well as because of B-blockade. Resting levels were reached at approximately the same time, however, and therefore the SBP recovery ratios decreased at a faster rate in the group not on B-blockers. Despite these differences in the rate of change in the SBP recovery ratios, the values in the CAD patients on and off B-blockers were markedly different from those in the normal subjects (figure 3).

Patients with hypertension while at rest are usually not exercised because of safety considerations and difficulty in interpreting the data. Patients with hyperten-
sion controlled by medications appear to represent a unique group. All nine of our patients in whom hypertension was controlled and who had normal coronary arteriograms had abnormal recovery ratios. Although we eliminated patients with a history of hypertension from our CAD group because it would have been difficult to separate the effects of the two diseases, we cannot exclude the possibility that some of our patients with CAD also had hypertension that was controlled by their antianginal medications.

SBP recovery ratios appear to be a useful clinical tool. The blood pressure data required for their derivation is readily available in most exercise laboratories and the calculation of the ratios is easy to perform. It is usually not necessary that patients exercise maximally and the diagnostic value of the ratios is not affected by medications the patient may be taking at the time of the study. Also, in our study subjects, many of whom were receiving medications, postexercise SBP recovery ratios were more sensitive than ECG changes during exercise or angina for identifying patients with CAD. In addition, the ratios may be useful in identifying a subgroup of patients with more severe CAD. Further investigation concerning the diagnostic value of this measurement in other populations therefore seems warranted.

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Usefulness of the postexercise response of systolic blood pressure in the diagnosis of coronary artery disease.
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