Comparison of Cardiovascular Response to Combined Static-Dynamic Effort, Postprandial Dynamic Effort and Dynamic Effort Alone In Patients with Chronic Ischemic Heart Disease

JOSEPH HUNG, M.B., JAMES McKILLIP, M.B., PH.D., WILLIAM SAVIN, M.A., SHELDON MAGDER, M.D., ROBERT KRAUS, M.D., NANCY HOUSTON, R.N., MICHAEL GORIS, M.D., PH.D., WILLIAM HASKELL, PH.D., and ROBERT DEBUSK, M.D.

SUMMARY The cardiovascular responses to combined static-dynamic effort, postprandial dynamic effort and dynamic effort alone were evaluated by upright bicycle ergometry during equilibrium-gated blood pool scintigraphy in 24 men, mean age 59 ± 8 years, with chronic ischemic heart disease. Combined static-dynamic effort and the postprandial state elicited a peak cardiovascular response similar to that of dynamic effort alone: work load 643 ± 156 and 638 ± 161 vs 650 ± 153 kg-m/min, respectively; heart rate 147 ± 14 and 145 ± 14 vs 143 ± 17 beats/min; systolic pressure 195 ± 26 and 200 ± 25 vs 197 ± 25 mm Hg; and rate-pressure product 286 ± 48 and 292 ± 55 vs 282 ± 52. Heart rate, intraaortic systolic and diastolic pressures, rate-pressure product and ejection fraction were similar for the three test conditions at the onset of ischemia and at peak effort. The prevalence and extent of exercise-induced ischemic left ventricular dysfunction, ST-segment depression, angina pectoris and ventricular ectopic activity were also similar during the three test conditions. Direct and indirect measurements of systolic and diastolic blood pressure were highly correlated. The onset of ischemic ST-segment depression and angina pectoris correlated as strongly with heart rate alone as with the rate-pressure product during all three test conditions. The cardiovascular response to combined static-dynamic effort and to postprandial dynamic effort becomes more similar to that of dynamic effort alone as dynamic effort reaches a symptom limit. If significant ischemic and arrhythmia abnormalities are absent during symptom-limited dynamic exercise testing, they are unlikely to appear during combined static-dynamic or postprandial dynamic effort. This simplifies the task of formulating guidelines for physical effort in patients with chronic ischemic heart disease, especially in providing “clearance” to perform avocational and vocational tasks involving combined static-dynamic and postprandial dynamic effort.

EXERCISE TESTING for the detection of ischemic ST-segment depression or angina pectoris is usually performed in the fasting state, with care to avoid the static effort involved in gripping of handrails or of handlebars. Although such testing accurately reflects the cardiovascular response to isolated dynamic effort, it does not simulate the superimposition of static on dynamic effort or of dynamic effort in the postprandial state — circumstances to which patients with ischemic heart disease are exposed during their customary activities.

Patients with ischemic heart disease are usually advised to avoid dynamic activities in which there is a significant static component, but we and others have noted a lower incidence of exercise-induced ischemic ST-segment depression or angina pectoris when patients performed combined static-dynamic effort than when they performed dynamic effort alone.

Haislly et al. measured a higher diastolic blood pressure during combined static-dynamic effort than during dynamic effort alone and attributed the lower incidence of ischemic abnormalities during combined static-dynamic effort to the tendency of augmented diastolic pressure to improve coronary perfusion. In a previous study, we did not directly measure diastolic pressure, but the higher rate-pressure product at the onset of ischemic abnormalities in our patients during combined static-dynamic effort than during dynamic effort alone is consistent with this hypothesis. The ingestion of food is also thought to hasten the onset of angina pectoris in some coronary patients. Goldstein et al. ascribed the earlier onset of angina pectoris during postprandial exercise to an augmentation of myocardial oxygen consumption.

We sought to evaluate the effectiveness of a simple laboratory test — symptom-limited bicycle ergometry — in detecting cardiovascular abnormalities likely to occur during conditions outside the laboratory. Our specific objectives were to compare the extent to which the incidence of ischemic ST-segment depression, angina pectoris and ventricular ectopic activity during dynamic effort is altered by static effort and by prior food ingestion; to compare the hemodynamic response, including directly measured blood pressure, and the left ventricular functional response to these three test conditions during submaximal and maximal effort; and to determine
what is responsible for differences in the incidence of ischemic and arrhythmic abnormalities during these three conditions.

**Methods**

Twenty-four men, mean age 59 ± 8 years, with chronic ischemic heart disease who were free of clinical congestive heart failure, unstable angina pectoris or other limiting medical conditions were selected for study. Twenty-three of the patients had had a myocardial infarction 4–48 months before the study and the other patient had arteriographically demonstrated three-vessel coronary artery disease. All patients had demonstrated treadmill-induced ischemic ST-segment depression with or without accompanying angina pectoris within 2 years of the study. At the time of study, six patients were taking propranolol for angina pectoris, mean dose 80 mg/day, and one patient was taking encainide, 150 mg/day, for ventricular ectopic activity. No patient was taking digitalis. Six patients were taking diuretics for the control of mild essential hypertension. Cardioactive medications except diuretics and sublingual nitroglycerin were withheld for at least 24 hours before the study. Each patient gave written informed consent before the study.

**Patient Testing Protocol** (fig. 1)

**Visit 1 — Pretest**

A cardiovascular history and examination were performed. A 12-lead ECG was recorded at rest and during symptom-limited bicycle ergometry, performed on a Monark or Collins bicycle ergometer, beginning with a work load of 150 kg-m/min and increasing 150 kg-m/min every 3 minutes until the onset of limiting symptoms of angina, dyspnea or fatigue. Other potential end points were a decrease in systolic blood pressure of 10 mm Hg or more from the peak value during the earlier stage of exercise or the appearance of ventricular tachycardia (three or more consecutive premature ventricular complexes), but these end points were not noted. Neither the extent of ST-segment depression nor the attainment of a target heart rate was used as an end point. Maximal voluntary contraction (MVC) for handgrip was determined in the right hand using a Jaymar hand dynamometer (Preston). MVC was taken to be the highest of three maximal handgrip contractions. Fifteen minutes after recovery from bicycle ergometry, all patients could sustain handgrip at 33% of MVC for at least 5 minutes.

**Visit 2**

Within 3 weeks after pretesting, patients returned to the laboratory at 12:30 p.m. They had not taken solid foods or fluids for 3 hours. A 12-lead ECG was attached, and under local anesthesia a 20-gauge Teflon catheter (Quik-cath) was inserted into the left radial artery. A mimitransducer (Ailtech MS 20) and a physiological pressure monitor (Gould Statham SP 1405) were used for continuous monitoring of arterial pressures. The mimitransducer was calibrated with a mercury manometer and maintained at the level of the fourth intercostal space when the patient was upright. Preparatory to equilibrium ECG-gated blood pool scintigraphy, in vivo red blood cell labeling was accomplished using the method of Pavel et al.® Fifteen milligrams of cold stannous pyrophosphate were injected intravenously, followed 30 minutes later by an intravenous injection of 15–20 mCi of technetium-99m pertechnetate. Because of radioisotope decay, 15 mCi of technetium-99m labeled to human serum albumin was injected 2 hours later.

The first bout of exercise started 45–60 minutes after the patient arrived in the laboratory. Patients performed three upright symptom-limited exercise tests on a Schwinn electrically braked bicycle ergometer during equilibrium-gated blood pool scanning. The three exercise tests, performed in random order, consisted of dynamic effort alone, combined static-dynamic effort (dynamic effort combined with handgrip contraction), and postprandial dynamic effort (dynamic effort after eating a 1000-calorie meal). Protocols for exercise and for collection of radionuclide data were identical for each test condition.

While the patient was seated erect at a comfortable height on a Schwinn bicycle ergometer, the gamma camera (Ohio Nuclear Series 120) was positioned against the left chest wall so as to achieve an ideal separation of right and left ventricles in the left anterior oblique projection. A Plexiglas brace attached to the camera supported the patient's back and a stiff
foam wedge and canvas restraint were used to restrict chest movement relative to the camera head during exercise. The gamma camera was fitted with a parallel-hole, high-sensitivity collimator. Radionuclide data were collected with the patient sitting at rest on the bicycle for 4 minutes, during 12 or fewer minutes of graded bicycle ergometry and for 4 minutes of recovery — a total duration of 20 minutes or less, depending on the duration of exercise. Initial ergometer work loads of 150 or 300 kg-m/min were increased every 3 minutes by 150–300 kg-m/min, based on each patient’s exercise capacity in the pretest, to achieve a symptom limit within the four 3-minute exercise stages available for radionuclide imaging. For combined static-dynamic testing, handgrip contraction at 33% of MVC commenced at the start of the work load preceding the expected peak exercise work load and was sustained for as long as 6 minutes during the remainder of the test. For postprandial dynamic testing, each subject was given a 1000-calorie formula meal of ¼ cup Meritene powder in 24 ounces of cold milk and 5 ounces of Sustacal pudding. The composition of this meal was approximately 20% protein, 40% carbohydrate and 40% fat. Exercise started 30 minutes after ingestion of the meal, which was consumed in 10 minutes or less. At least 40 minutes elapsed between bouts of exercise. To characterize the response to static effort alone, patients also sustained 3 or more minutes of handgrip at 33% of MVC while sitting quietly in a chair strapped to the gamma camera. This was performed 20 minutes after the last bout of exercise. One of the 24 patients could not perform this static effort because of general fatigue after the bicycle test.

During each test, analog blood pressure was recorded continuously and a 12-lead ECG was recorded during the last 10 seconds of each minute of exercise and during the 4 minutes of recovery from each bout of exercise. Peak bicycle work load was defined as the highest work level that could be sustained for at least 2 minutes. Flat or downsloping ST segments that, 0.05 second after the J point, were depressed 0.1 mV or more below a line drawn through the PQ segment were defined as ischemic. During dynamic bicycle ergometry alone and after food ingestion, indirect arterial pressures were obtained at the end of each 3-minute work load and at peak effort by means of a cuff sphygmomanometer attached to the right arm. Handgrip involving the right hand precluded indirect measurement of blood pressure during combined static-dynamic effort.

For count data acquisition, the gamma camera was interfaced with an Informatek Simis IV computer. Counts gated to the patient’s ECG were collected in 16 equal time frames within each cardiac cycle and summed to produce a single composite cycle of 16 frames for each minute. For each successive cycle of 16 frames, a normalization program was used to compensate for variations in the number of counts in each frame due to alterations of cycle length.

All studies were analyzed automatically, that is, without operator intervention, using a series of algorithms developed in the Stanford nuclear medicine department. This method of analysis yields values of left ventricular ejection fraction that correlate well with those obtained from contrast ventriculography. The first step in processing the ECG-gated radionuclide angiographic study was to combine the data from individual minutes at rest or at the same exercise level. Data from exercise stages lasting less than 2 minutes were discarded. Subtraction of noncardiac background activity was achieved by a threshold technique. A threshold activity level was identified as that at which no structure significantly decreased its activity during systole. Structures below this threshold were assumed to be noncardiac and this level of activity was subtracted as background. Motion analysis and edge detection algorithms were then used for automatic identification of the true left ventricular region of interest on the background-subtracted images, and the time-activity curve was obtained from this region. End-diastolic and end-systolic count rates were obtained from the time-activity curve and left ventricular ejection fraction was calculated from the standard formula: end-diastolic counts - end-systolic counts/end-diastolic counts. Observer variability in determining ejection fraction was essentially removed, since the studies were analyzed without operator intervention. The left ventricular end-diastolic and end-systolic counts used to measure ejection fraction were also the count correlates of the left ventricular end-diastolic and end-systolic volumes, respectively, and thus were used to measure relative changes in left ventricular volumes from rest to exercise during each study.

Analysis of variance was used to test for differences in continuous variables measured during the three test conditions, and when such differences were found, matched-pairs t tests were used to test for differences between individual conditions. Cochran’s Q test was used to detect differences in the frequency of discrete variables such as ischemic ST-segment depression, angina pectoris and ventricular ectopic activity measured during each test condition.

Results

Exercise Performance

The peak work load and heart rate during dynamic bicycle effort at visit 2 were not significantly different from those during visit 1 (pretest). 650 ± 150 kg-m/min and 143 ± 17 beats/min vs 662 ± 176 kg-m/min and 135 ± 15 beats/min, respectively. Seventeen of the 24 patients completed four bicycle work loads before reaching a symptom limit. The other seven patients reached their limit after three bicycle work loads. Twenty-one patients completed the same peak work load during dynamic, combined dynamic-static and postprandial dynamic effort. Cardiovascular variables during each of the three test conditions were compared for the peak work load achieved (max) and the two preceding work loads (max − 2 and max − 1).

Peak bicycle work loads were not significantly different between dynamic, combined static-dynamic...
and postprandial dynamic effort, 650 ± 153, 643 ± 156 and 638 ± 161 kg-m/min, respectively. Submaximal bicycle work loads for dynamic effort alone were 286 ± 108 (max – 2) and 486 ± 148 (max – 1) kg-m/min — also not significantly different among the three test conditions. The mean durations of dynamic, combined static-dynamic and postprandial dynamic effort were also similar, 10.6 ± 1.3, 10.2 ± 1.3 and 10.2 ± 1.5 minutes, respectively.

The frequency of the various test end points was similar among dynamic, combined static-dynamic and postprandial dynamic effort. Almost two-thirds of all tests were terminated because of leg fatigue or dyspnea and one-third by angina pectoris. Systolic pressure dropped at peak dynamic effort in two patients, at peak combined static-dynamic effort in three and during peak postprandial dynamic effort in three.

**Cardiovascular Responses (fig. 2)**

The order in which the patients performed dynamic, combined static-dynamic and postprandial dynamic effort did not significantly influence the rest and exercise heart rate, blood pressure, rate-pressure product and left ventricular ejection fraction. Mean resting values for heart rate, systolic, diastolic and mean arterial pressures were not significantly different among the three test conditions. Heart rate and arterial pressures increased significantly (p < 0.001) above resting values during both submaximal bicycle work loads and at peak effort during all three exercise conditions. Addition of sustained handgrip at 33% of MVC during the last two bicycle work loads did not significantly augment heart rate, systolic pressure or rate-pressure product response to dynamic effort, except for a slight increase in the peak exercise heart rate (147 ± 14 vs 143 ± 17 beats/min, p < 0.05). Submaximal and peak exercise heart rate, systolic pressure and rate-pressure product were slightly higher during postprandial dynamic effort than during dynamic effort alone, but these differences were significant only for rate-pressure product at max – 2 (196 ± 47 vs 183 ± 42, p < 0.01). Diastolic pressures at peak effort rose significantly above resting values, by 17 ± 11, 21 ± 10 and 18 ± 13 mm Hg during dynamic, combined static-dynamic and postprandial dynamic effort, respectively (all p < 0.01), but the differences were not significant among the three test conditions.

**Ischemic Responses**

The prevalence of angina pectoris and ischemic ST-segment depression was similar during dynamic, combined static-dynamic and postprandial dynamic effort (table 1). At visit 1 (pretest), 17 of the 24 patients manifested an ischemic response, that is, angina pectoris or ischemic ST-segment depression during bicycle effort. Only one of these 17 patients failed to show an ischemic response during at least one of the three bicycle exercise conditions on visit 2. Patients without an ischemic response on visit 1 did not demonstrate
an ischemic response during testing on visit 2. The prevalence of angina pectoris or ischemic ST-segment depression, alone or in combination, was not significantly different among dynamic, combined static-dynamic and postprandial dynamic effort. Ischemic ST-segment depression was manifest during exercise; in no case was it present only after exercise. The average peak magnitude of ST-segment depression was not significantly different among dynamic, combined static-dynamic and postprandial dynamic effort, 

\[ -0.09 \pm 0.13, -0.11 \pm 0.13 \text{ and } -0.11 \pm 0.14 \text{ mV} \], respectively. Exercise-induced angina pectoris or ischemic ST-segment depression was noted in 16 patients (67%) during at least one of the three exercise conditions and in 12 patients (50%) during all three exercise conditions. Patient 1 showed an ischemic response with both combined static-dynamic and postprandial dynamic testing, but not with dynamic testing alone; patient 15 had an ischemic response during dynamic effort alone; and patients 9 and 19 had an ischemic response during postprandial dynamic effort alone. The other eight patients (33%) demonstrated no ischemic abnormality during any of the three exercise conditions.

The prevalence, frequency and complexity of ventricular ectopic activity during or after exercise were not significantly different among the three test conditions. Ventricular ectopic activity was associated with dynamic effort in eight patients and with combined static-dynamic and postprandial dynamic effort in 10 patients each. Ventricular ectopic activity was isolated except in one patient, who manifested ventricular bigeminy and ventricular couplets during recovery from both combined dynamic-static and postprandial dynamic effort.

Table 1. Ischemic Responses to Dynamic, Combined Static-Dynamic and Postprandial Dynamic Effort

<table>
<thead>
<tr>
<th>Pt</th>
<th>Angina*</th>
<th>ST depression†</th>
<th>Angina and/or ST depression‡</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>D S-D PP-D</td>
<td>D S-D PP-D</td>
<td>D S-D PP-D</td>
</tr>
<tr>
<td>1</td>
<td>0 0 0</td>
<td>-0.35 -0.30 -0.30</td>
<td>0 1 1</td>
</tr>
<tr>
<td>2</td>
<td>1 1 0</td>
<td>0 -0.35 -0.35</td>
<td>1 1 1</td>
</tr>
<tr>
<td>3</td>
<td>1 1 1</td>
<td>0 0 0</td>
<td>1 1 1</td>
</tr>
<tr>
<td>4</td>
<td>0 0 0</td>
<td>0 0 0</td>
<td>0 0 0</td>
</tr>
<tr>
<td>5</td>
<td>0 0 0</td>
<td>0 0 0</td>
<td>0 0 0</td>
</tr>
<tr>
<td>6</td>
<td>0 0 0</td>
<td>0 0 0</td>
<td>0 0 0</td>
</tr>
<tr>
<td>7</td>
<td>0 0 0</td>
<td>0 0 0</td>
<td>0 0 0</td>
</tr>
<tr>
<td>8</td>
<td>0 0 0</td>
<td>0 0 0</td>
<td>0 0 0</td>
</tr>
<tr>
<td>9</td>
<td>0 0 0</td>
<td>0 0 0</td>
<td>0 0 0</td>
</tr>
<tr>
<td>10</td>
<td>0 0 1</td>
<td>0 0 0</td>
<td>0 0 1</td>
</tr>
<tr>
<td>11</td>
<td>0 0 0</td>
<td>0 0 0</td>
<td>0 0 0</td>
</tr>
<tr>
<td>12</td>
<td>0 0 0</td>
<td>-0.15 -0.20 -0.25</td>
<td>1 1 1</td>
</tr>
<tr>
<td>13</td>
<td>0 0 0</td>
<td>-0.25 -0.25 -0.30</td>
<td>1 1 1</td>
</tr>
<tr>
<td>14</td>
<td>0 0 0</td>
<td>0 0 0</td>
<td>0 0 0</td>
</tr>
<tr>
<td>15</td>
<td>1 0 0</td>
<td>0 0 0</td>
<td>1 0 0</td>
</tr>
<tr>
<td>16</td>
<td>0 0 0</td>
<td>-0.20 -0.20 -0.20</td>
<td>1 1 1</td>
</tr>
<tr>
<td>17</td>
<td>1 1 1</td>
<td>-0.40 -0.35 -0.40</td>
<td>1 1 1</td>
</tr>
<tr>
<td>18</td>
<td>1 1 1</td>
<td>-0.10 -0.10 -0.10</td>
<td>1 1 1</td>
</tr>
<tr>
<td>19</td>
<td>1 1 1</td>
<td>-0.10 -0.15 -0.15</td>
<td>1 1 1</td>
</tr>
<tr>
<td>20</td>
<td>0 0 1</td>
<td>0 0 0</td>
<td>0 0 1</td>
</tr>
<tr>
<td>21</td>
<td>0 0 0</td>
<td>-0.20 -0.20 -0.20</td>
<td>1 1 1</td>
</tr>
<tr>
<td>22</td>
<td>1 1 0</td>
<td>-0.15 -0.15 -0.10</td>
<td>1 1 1</td>
</tr>
<tr>
<td>23</td>
<td>1 1 1</td>
<td>0 -0.10 -0.10</td>
<td>1 1 1</td>
</tr>
<tr>
<td>24</td>
<td>1 1 1</td>
<td>-0.30 -0.25 -0.30</td>
<td>1 1 1</td>
</tr>
<tr>
<td>25</td>
<td>1 1 1</td>
<td>-0.09 -0.11 -0.11</td>
<td>13 13 15</td>
</tr>
</tbody>
</table>

*Exercise-induced angina pectoris. 1 = present; 0 = absent.
†Peak exercise or recovery ST-segment depression. Less than 0.1 mV of horizontal or downsloping ST-segment depression is coded as 0. Numbers at bottom of columns represent the average peak magnitude of ST-segment depression.
‡Exercise-induced angina or ischemic ST-segment depression (0.10 mV of horizontal or downsloping ST-segment depression). 1 = present; 0 = absent.

Abbreviations: D = dynamic effort alone; S-D = combined static-dynamic effort; PP-D = postprandial dynamic effort.

Left Ventricular Function (fig. 3)

The resting left ventricular ejection fraction was 0.45 or greater in 16 patients, 0.35-0.45 in four patients and less than 0.35 in four patients. The mean
postprandial resting left ventricular ejection fraction was slightly higher \((p < 0.05)\) than before dynamic or combined static-dynamic effort \((0.49 \pm 0.14 vs 0.47 \pm 0.15 and 0.46 \pm 0.12, respectively)\). Overall, left ventricular ejection fraction increased significantly from rest during both submaximal exercise levels during the three test conditions \((p < 0.01)\). After an initial increase during submaximal effort, left ventricular ejection fraction decreased significantly \((p < 0.01)\) at peak effort in all three exercise conditions, without significant differences in peak exercise ejection fraction values between conditions. The decrease in ejection fraction at peak effort compared with resting values was not significantly different among dynamic, combined static-dynamic and postprandial dynamic effort, \(-0.02 \pm 0.07, -0.05 \pm 0.09, \text{ and } -0.05 \pm 0.11 \text{ units, respectively.}\) An absolute drop in ejection fraction of 0.05 units or greater was noted in 29\%, 46\% and 50\% of patients, respectively, also not statistically significant between conditions. The average fall in peak exercise ejection fraction compared with resting values was also similar among the 16 patients with exercise-induced angina pectoris or ischemic ST-segment depression: \(-0.03 \pm 0.06, -0.07 \pm 0.08 \text{ and } -0.07 \pm 0.10 \text{ units during dynamic, combined static-dynamic and postprandial dynamic testing, respectively (NS).}\) These decreases were larger, although not significantly, than in the eight patients without an ischemic response during any of the three test conditions; in the latter group, ejection fraction decreased \(-0.01 \pm 0.07, -0.01 \pm 0.10 \text{ and } -0.01 \pm 0.11, \text{ respectively.}\)

The decrease in ejection fraction at peak effort was accompanied by an increase in left ventricular end-systolic and end-diastolic volumes. End-systolic volume increased to 137 \(\pm 35\%, 136 \pm 50\% \text{ and } 134 \pm 63\% \text{ of resting values at peak dynamic, combined static-dynamic and postprandial dynamic effort, respectively (}p < 0.01\), without significant differences among the three test conditions. End-diastolic volume at peak effort was 119 \(\pm 19\%, 123 \pm 26\% \text{ and } 118 \pm 25\% \text{ of resting values, respectively, (NS among the three test conditions).}\)

### Ischemic Threshold

The values of peak heart rate, systolic pressure and rate-pressure product attained by the 16 patients who had an ischemic response during one or more of the three test conditions were not significantly different from those in the eight patients without an ischemic response (table 2). Values of heart rate, systolic pressure and rate-pressure product at the onset of 0.1 mV of ischemic ST-segment depression or angina pectoris (if ST-segment depression was absent) are shown in table 2. During test conditions in which an ischemic response did not occur, the peak heart rate and systolic pressures were used to define the ischemic threshold. The ischemic threshold of heart rate, systolic pressure and rate-pressure product were similar during dynamic, combined static-dynamic and postprandial dynamic effort. The ischemic threshold of heart rate was only six or seven beats below the peak heart rate during all three test conditions. Similarly, the duration of exercise after the onset of ischemia was brief, 1.1 \(\pm 1.5, 1.2 \pm 1.5 \text{ and } 1.4 \pm 1.5 \text{ minutes during dynamic, combined static-dynamic and postprandial dynamic effort, respectively.}\) The values of systolic pressure and rate-pressure product at the onset of ischemia were slightly but significantly higher during postprandial dynamic effort than during dynamic effort alone \((196 \pm 24 vs 190 \pm 23 \text{ mm Hg and 276} \pm 57 vs 260 \pm 52, \text{ respectively (both }p < 0.05\).\)

Point biserial correlation coefficients were used to express the relationship between a dichotomy — the presence or absence of exercise-induced abnormalities — and the continuous variables of heart rate and

---

**Table 2. Ischemic Threshold During Dynamic, Combined Static-Dynamic and Postprandial Dynamic Effort**

<table>
<thead>
<tr>
<th>At onset of ischemia</th>
<th>At peak effort</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate (beats/min)</td>
<td>SBP (mm Hg)</td>
</tr>
<tr>
<td>Dynamic</td>
<td>137 (\pm 22)</td>
</tr>
<tr>
<td>Combined static-dynamic</td>
<td>140 (\pm 20)</td>
</tr>
<tr>
<td>Postprandial dynamic</td>
<td>140 (\pm 22)</td>
</tr>
</tbody>
</table>

Values are mean \(\pm\) SD.

*Eight of the 24 patients without any exercise-induced angina pectoris or ischemic ST-segment depression during the three test conditions were excluded.

\(\dagger\) \(p < 0.05\) vs dynamic.

Abbreviations: SBP = systolic blood pressure; RPP = rate-pressure product.
rate-pressure product measured during exercise. The presence or absence of exercise-induced ischemic ST-segment depression and angina pectoris was as negatively correlated with peak heart rate alone \( (r = -0.28, -0.22, \text{ and } -0.36) \) as with peak rate-pressure product \( (r = -0.26, -0.22 \text{ and } -0.37) \) during dynamic, static-dynamic and postprandial dynamic effort, respectively.

Direct and indirect measurements of arterial blood pressure were highly correlated at rest and during peak exercise \( (r = 0.99 \text{ for systolic pressure both at rest and peak exercise; see } = 10.8 \text{ and } 14.4 \text{ mm Hg, respectively). Corresponding correlation coefficients for diastolic pressure were both 0.97 \text{ (see } = 8.0 \text{ and } 9.7 \text{ mm Hg at rest and peak exercise, respectively).}

Cardiovascular Responses to Static Effort

Performed at Rest (fig. 2)

Sustained handgrip at 33% of MVC was terminated by arm fatigue in the 23 patients who performed static effort during visit 2. During peak static effort, heart rate increased by 7 ± 11 beats/min, systolic blood pressure by 49 ± 19 mm Hg, diastolic blood pressure by 22 ± 13 mm Hg and mean arterial pressure by 32 ± 16 mm Hg compared with resting values \( (p < 0.001) \). The rate-pressure product at peak static effort was 171 ± 48, significantly less \( (p < 0.001) \) than the value of 282 ± 52 during maximal dynamic effort. Left ventricular ejection fraction decreased from 0.47 ± 0.14 at baseline to 0.44 ± 0.13 at the peak of static effort \( (p < 0.01) \). Left ventricular end-systolic and end-diastolic volumes both increased to 120 ± 17% and 113 ± 11% of resting values at peak static effort \( (p < 0.01) \).

During or after sustained, static effort, no patient had angina pectoris, one patient demonstrated ischemic ST-segment depression and two patients had isolated ventricular ectopic activity.

Discussion

Although the cardiovascular effects of static effort or the postprandial state may be additive to those of dynamic effort at low dynamic work loads — a finding we and others have noted\(^\text{14-15} \) — their effects are largely attenuated as peak dynamic effort is reached. In the present study, we found that the myocardial oxygen demand reflected in the rate-pressure product, left ventricular volume and ejection fraction and the myocardial oxygen delivery reflected in the diastolic arterial pressure were not substantially different among the three test conditions at the onset of ischemia and at peak effort. Further, the prevalence and severity of exercise-induced ischemic left ventricular dysfunction, angina pectoris, ischemic ST-segment abnormality and ventricular ectopic activity were similar during dynamic, combined static-dynamic and postprandial dynamic effort. The failure of added static effort or the postprandial state to alter significantly the peak cardiovascular response to dynamic effort reflects the fact that vigorous, large-muscle-group, dynamic effort alone taxes the functional limits of the cardiovascular system.

The widely held belief that patients with chronic ischemic heart disease should avoid static effort or combined static-dynamic effort is largely based on extrapolation from findings in normal persons that static effort alone or in combination with low-level dynamic effort substantially augments heart rate and systolic blood pressure.\(^\text{10, 11} \) In the present study, static effort performed at rest augmented mean arterial pressure to an extent similar to that observed during peak dynamic effort \( (32 \text{ vs } 35 \text{ mm Hg, respectively), but peak heart rate \( (98 \text{ vs } 143 \text{ beats/min}) \text{ and the peak rate-pressure product (171 vs 282) differed substantially. The rate-pressure product correlates closely with the myocardial oxygen consumption and myocardial blood flow during static, dynamic and combined static-dynamic effort,\(^\text{12-16} \) so it is not surprising that static testing is far less effective than dynamic testing in producing angina or ischemic ST-segment abnormalities.\(^\text{1-3, 12, 14} \) The increased pressure load on the myocardium induced by static effort was reflected by a small decrease in left ventricular ejection fraction and an increase in end-systolic and end-diastolic volumes, similar to that reported by other investigators in coronary patients performing static effect.\(^\text{16, 17} \) However, these hemodynamic changes are seldom adequate to induce symptoms and are limited by the relatively brief duration that static effort can be sustained. Frequent premature ventricular complexes are also not usually induced by static effort.\(^\text{1-3, 12} \)

Whereas we studied the effect of static effort added to symptom-limited maximal dynamic effort, both Haissley et al.\(^\text{9} \) and Kerber et al.\(^\text{8} \) studied the effects of combined static-dynamic effort during submaximal dynamic effort only. Although the addition of a static load to low-level dynamic effort augmented heart rate and particularly systolic pressure.\(^\text{10-12} \) in the present study, combined static-dynamic effort did not elicit greater left ventricular dysfunction or ischemic responses than dynamic effort alone. Because handgrip effort could be sustained for a relatively brief period, it was added at a relatively high level of dynamic effort, approximating 75% of the peak cardiovascular response to dynamic effort alone. Studies in normal subjects have indicated that heart rate and systolic blood pressure responses to handgrip during dynamic effort are also increasingly attenuated as maximal oxygen consumption is approached.\(^\text{10, 18} \) The intense vasodilation produced by large muscle groups engaged in near-maximal dynamic effort tends to override vasoconstrictor effects of static effort. As the cardiovascular system approaches the limits of its capacity, its response to combined static-dynamic effort therefore resembles that of symptom-limited dynamic effort alone. Hence, in the present study, peak rate-pressure product was not significantly augmented by the addition of static effort, and whereas peak heart rate was significantly higher than during dynamic effort alone, the difference was small \( (147 \text{ vs } 143 \text{ beats/min}) \).

The substantial increases in diastolic pressure in our patients during dynamic effort alone, that is, 17 mm Hg above baseline, obscured further increases in
diastolic pressure due to combined static-dynamic effort, that is, 22 mm Hg above baseline. The increasing
degree of resistive force associated with bicycle
ergometry as pedal impedance is increased may result in
an augmentation of diastolic pressure, whereas diastolic pressure is usually unaffected by
symptom-limited treadmill exercise. Our data do not discount the role of augmented diastolic pressure in
enhancing myocardial perfusion during combined static-dynamic effort, and therefore, the lower in-
cidence of exercise-induced ischemic ST-segment
depression and angina pectoris noted by us and others
when static load was added to treadmill exercise. On the contrary, the effects of combined static-
dynamic effort on the ischemic threshold may simply
be more readily demonstrable during treadmill exer-
cise than during bicycle exercise.

Patients with exertional angina pectoris may ex-
perience an earlier onset of angina pectoris and
ischemic ST-segment depression and a diminished ex-
ercise capacity after eating.6-8 While earlier observa-
tions in normal subjects22, 23 and in patients24, 26 have
led some investigators to postulate a postprandial im-
pairment of myocardial oxygen delivery, Goldstein et
al.4 demonstrated that prior food ingestion augmented
heart rate and blood pressure during submaximal
(bicycle) dynamic effort. These effects of food diges-
tion, which increase myocardial oxygen consumption,
begin immediately and lasted for at least 1 hour.
Thus, an augmentation of myocardial oxygen require-
ments appeared to be responsible for the earlier
development of angina pectoris and for the decreased
exercise capacity in these patients. However, the rate-
pressure product at the onset of angina pectoris and
the peak double product were not significantly altered
by prior food ingestion, a finding similar to that of the
present study and inconsistent with any postprandial
impairment of myocardial oxygen delivery.
The formula meal given to our patients was similar
in size and composition to that used by Goldstein et
al.8 Although the rate-pressure product at the onset of ischemia was slightly but significantly higher during
postprandial effort, this was of doubtful clinical significance because the prevalence and severity of
exercise-induced angina pectoris or ischemic ST-seg-
ment depression was similar to that during dynamic
effort alone. Also, apart from a slight increase in rest-
ing left ventricular ejection fraction, consistent with
a postabsorptive increase in myocardial inotropic state,22 we found no significant difference in left ven-
tricular functional response to dynamic effort after
eating. Despite a slightly higher double product during
dynamic effort 30 minutes after food ingestion, exer-
cise capacity in our patients did not deteriorate
significantly after eating. Similar to the cardiovascular effects of added static effort, the effects of diges-
tion are apparently largely attenuated at higher
dynamic work loads, as postprandial mesenteric vas-
odilation is offset by the visceral vasoconstrictor effects
of vigorous exercise.28 Hence, cardiac output and car-
diac work may not be increased above preprandial
levels with vigorous dynamic effort after eating.6, 27
The results of the present study should be applied
with caution to patients who are more severely limited
by angina pectoris or left ventricular dysfunction. Our
patients were selected on the basis of their ability to
complete at least 9 minutes of a graded exercise protocol. Previous studies of combined static-dynamic
effort2-8 and of postprandial effort6 have usually
selected patients who developed ischemic symptoms at
low work loads. For example, the peak heart rate dur-
ing dynamic effort was 143 beats/min in the present
study, compared with 113, 129 and 109 beats/min in
the studies of Haissly et al., Kerber et al.6 and Gold-
stein et al.4 Since the added cardiovascular effects of
static effort or the postprandial state are relatively
greater during low-level dynamic effort, these may
cause an earlier onset of limiting symptoms.
The results of the present study indicate that there is
no unique cardiovascular hazard associated with com-
bined static-dynamic effort or with postprandial
dynamic effort compared to dynamic effort alone. In
fact, the ischemic threshold appears to be stable dur-
ing a variety of environmental conditions. This was
demonstrated recently by Lassvik and Areskog28 in
coronary patients who performed exercise in warm
ambient temperature, in cold ambient temperature
and during cold air inhalation. If the incidence of left
ventricular dysfunction, ischemic ST-segment depres-
sion, angina pectoris and ventricular ectopic activity is
also similar in these diverse circumstances — as was
true for all three test conditions of the present study —
the clinician can use the simple and readily available
method of symptom-limited bicycle ergometry or
postprandial treadmill exercise testing in lieu of more cumbersome
testing methods. This simplifies the task of providing
"cleansing" to perform vocational and avocational tasks involving combined static-dynamic and post-
prandial effort.

It is also important to know whether measurement
of arterial pressures is required for the evaluation of
vocational and avocational tasks in patients with
chronic ischemic heart disease. Noninvasive measure-
ment of systolic pressure during customary activity
outside the laboratory is technically demanding and
direct measurement of arterial pressure by ambu-
latory recording devices is not without hazard.
While previous studies by Nelson et al.18 in young nor-
mal persons demonstrated that myocardial oxygen
consumption correlated slightly more with rate-pres-
sure product than with heart rate alone (r = 0.88 vs
0.80), our results indicate that this advantage is small
in patients with ischemic heart disease. In fact, we
found the negative correlation between the onset of
ischemic abnormality to be as strong for heart rate
alone as for rate-pressure product. The fact that heart
rate reliably "tracked" the onset of ischemic abnor-
malities during all three conditions of the present
study may obviate the need to measure blood pres-
sure noninvasively or invasively during avocational
and vocational activities.

Acknowledgment

The authors thank J. Kent Garman, M.D., for assistance with
arterial cannulations, Lynda Fisher and Cay Fotopolos for technical
assistance, Betsy Bingham for data analysis, Helena Kraemer,
References


16. Ludbrook PA, Byrne JD, Reed FR, McKnight RC: Modification of left ventricular diastolic behavior by isometric handgrip exercise. Circulation 62: 357, 1980


Comparison of cardiovascular response to combined static-dynamic effort, postprandial dynamic effort and dynamic effort alone in patients with chronic ischemic heart disease.

J Hung, J McKillip, W Savin, S Magder, R Kraus, N Houston, M Goris, W Haskell and R DeBusk

_Circulation_. 1982;65:1411-1419
doi: 10.1161/01.CIR.65.7.1411

_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1982 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/65/7/1411

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in _Circulation_ can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to _Circulation_ is online at:
http://circ.ahajournals.org/subscriptions/