Are the Clinical and Hemodynamic Events During Exercise Stress Testing in Invasive Studies in Patients with Angina Pectoris Reproducible?

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SUMMARY  The effect of exercise on resting hemodynamics and the reproducibility of clinical and hemodynamic events during two successive exercise periods 25 minutes apart were evaluated in 20 patients with stable angina pectoris.

Comparison of the resting data during the first and second control periods (C1 and C2) separated by a period of exercise showed that the values for heart rate (HR) were higher (76 ± 11 vs 79 ± 11 beats/min [mean ± SD]; p < 0.05), while brachial arterial systolic pressure (BASP) (142 ± 13 vs 137 ± 13 mm Hg; p < 0.05), brachial arterial mean pressure (BAMP) (103 ± 10 vs 99 ± 9 mm Hg; p < 0.05), pulmonary arterial mean pressure (PAMP) (22 ± 5 vs 18 ± 5 mm Hg; p < 0.01), and left ventricular end-diastolic pressure (LVEDP) (18 ± 4 vs 15 ± 4 mm Hg; p < 0.001) were lower during C2.

Angina was experienced by all 20 patients during both exercise studies (Ex1 and Ex2) and the group mean values for the duration of exercise to angina, ST-segment depression, HR, and rate-pressure product at the onset of angina were similar during Ex1 and Ex2. However, in five of the 20 patients, exercise duration to angina varied by 60 seconds or more during the two exercise studies and two of these patients had to be exercised at higher work loads to induce angina during Ex2. Comparison of hemodynamic data at the onset of angina induced by Ex1 and Ex2 showed that the group values for LVEDP (29 ± 6 vs 25 ± 6 mm Hg), PAMP (33 ± 8 vs 29 ± 10 mm Hg), BASP (167 ± 15 vs 162 ± 16 mm Hg) and BAMP (120 ± 10 vs 115 ± 10 mm Hg) were lower (p < 0.02) during Ex2.

Clinical and electrocardiographic events and HR, rate-pressure product and cardiac output during two successive exercise periods were reproducible, but LVEDP and PAMP were consistently lower during the Ex2. These results should be considered when the effects of therapeutic interventions are being studied during invasive exercise testing in angina pectoris.

DURING CARDIAC CATHETERIZATION, supine bicycle exercise stress testing is widely used to study cardiac performance. In patients with coronary artery disease, such exercise testing is used to assess the hemodynamic consequences of myocardial ischemia and to study the therapeutic efficacy of antianginal drugs and surgical interventions.1-29 The reproducibility of the anginal threshold and the hemodynamic patterns have been well documented during noninvasive treadmill and bicycle exercise stress testing.20-33 There is little information regarding such reproducibility with supine leg exercise during invasive studies. Investigators have assumed, however, that the anginal threshold and hemodynamic profiles of repeated exercise in a given patient are reproducible, and that the data obtained during a control period of exercise have been compared with those during a similar period of exercise after a therapeutic intervention.1, 7-9, 13, 15, 20-25, 28, 29 The present investigation was designed to determine whether the clinical and hemodynamic events induced by supine exercise stress testing during invasive studies are reproducible in patients with angina pectoris.

Methods

Patients

Hemodynamic investigations with subsequent selective coronary cineangiography and left ventriculography were performed in 20 patients whose average age was 50 years (range 38-63 years). All patients were in sinus rhythm and had stable exertional angina pectoris that could be reproduced during treadmill exercise. Six of the 20 patients had sustained a myocardial infarction at least 6 months before the study. Nineteen of the 20 patients were normotensive, and one had a systolic blood pressure of 165 mm Hg and normal diastolic pressure. All patients had a normal cardiac examination, and none had clinical or radiologic evidence of cardiomegaly or cardiac failure or was receiving digoxin, diuretics or antihypertensive agents. Some had been treated with propranolol and long-acting nitrates, but none had received these for at least 7 days before the investigation. The only medication taken during the 48 hours before the study was sublingual nitroglycerin, but none took this drug on the morning of the study. The study was explained to each patient and written, informed consent obtained.
Exercise Technique

During supine exercise, the bicycle ergometer was positioned over the catheterization table, with the axle 12 inches (30 cm) above table level. Exercise was commenced with both legs at a work load of 200 or 300 kilopond-meters (kpm) per minute and this was increased by 100 kpm/min every 3 minutes until the onset of angina or fatigue. Once the patient experienced angina pectoris, exercise was continued for 2-3 minutes to allow cardiac output (CO) and final hemodynamic measurements. Thereafter, exercise was discontinued. For a given patient, the exercise was commenced at the same initial work load during the two exercise studies.

Study Protocol

Patients were studied in a fasting state without premedication. Under local anesthesia, the brachial artery and a vein were isolated in the right antecubital fossa. A #8 Cournand catheter was passed into the right side of the heart and advanced to the main pulmonary artery. A #8 Sones catheter was inserted into the left ventricle from the right brachial artery, and the left brachial artery was cannulated with a Teflon needle using the Seldinger technique. After a 15-minute rest period, control measurements were made with the patient’s feet in position on the bicycle ergometer. Modified lead V₅ of the ECG and intracardiac and intravascular pressures were recorded at 5-minute intervals during a 10-minute control period (C₁). During the final 2 minutes of C₁, CO was measured by the dye-dilution technique using indocyanine green and the blood withdrawn for this measurement was reinforced. CO was performed in duplicate or in triplicate with a variation of less than 10%. Exercise was then commenced as described above under exercise technique. During exercise (Ex₁), the ECG and pressures were monitored continuously and recorded at 1-minute intervals. Further records of the ECG and pressures were made at the onset of angina, but exercise was continued to permit the duplicate determination of CO. Thereafter, final pressures were recorded and exercise was discontinued. Pressures and the ECG were recorded immediately after exercise and after 3 and 5 minutes. The legs were then lowered from the bicycle and after a 15-minute recovery period, the protocol was repeated and observations were made during a 10-minute control period (C₂) with the patient’s feet in position on the bicycle ergometer, during exercise (Ex₂), and after exercise.

Pressures were recorded with P23Db Statham strain gauges from a zero reference level 5 cm below the level of the angle of Louis. Recordings were made on a photographic recorder. Pressures were measured over at least two respiratory cycles, and the mean pressures in the brachial artery and pulmonary artery were obtained electronically. The recording speed was normally 25 mm/sec, but for the left ventricular end-diastolic pressure (LVEDP), a speed of 100 mm/sec at a high sensitivity was used. The diagnostic criteria of ischemic response was a flat or downward depression of the ST segment of at least 1 mm (1 mv) persisting for 0.08 second or longer during or immediately after exercise. ST-segment measurements were averaged over 10 beats. Left ventricular stroke work index (LVSWI) in g-m/m² was calculated using the formula:

\[ LVSWI = \frac{SI \times (BAM - LVEDP) \times 13.6}{1000} \]

where \( SI \) = stroke index in ml/beat/m² and \( BAM = \) brachial arterial mean pressure in mm Hg. The rate-pressure product (RPP) was calculated as the product of systolic arterial pressure and heart rate (HR) \( \times 10^2 \).

After these hemodynamic studies were completed, selective cine coronary angiography and left ventriculography were carried out in all patients. The paired \( t \) test was used for statistical analysis.

Results

The hemodynamic and angiographic studies were completed without complications. All patients had angiographic evidence of at least 75% cross-sectional luminal area narrowing of one or more coronary arteries. Sixteen patients had triple-vessel disease, two had double-vessel disease and two had single-vessel disease. The left ventricular wall motion was normal in 14, there was evidence of localized hypokinesis in four, and generalized hypokinesis in one, and the remaining patient had an apical aneurysm.

Table 1 shows the group mean data for the various hemodynamic parameters and ST-segment depression at rest during \( C₁ \) and \( C₂ \) studies and at the onset of angina during Ex₁ and Ex₂. Table 2 shows the average difference and the range of differences in hemodynamics between \( C₁ \) and \( C₂ \) and between Ex₁ and Ex₂.

Studies at Rest

Comparison of data during \( C₁ \) and \( C₂ \) studies showed that the group mean values during \( C₂ \) were slightly higher for HR (\( p < 0.05 \)) (fig. 1), while brachial arterial systolic (fig. 1) and mean pressures (\( p < 0.05 \)), pulmonary arterial, systolic (\( p < 0.05 \)), diastolic (\( p < 0.001 \)), and mean (\( p < 0.001 \)) pressures (table 1) and LVEDP (\( p < 0.001 \)) (fig. 2) were lower. RPP, CO, stroke volume (SV), and LVSWI were similar during \( C₁ \) and \( C₂ \) (table 1). Examination of individual data during \( C₁ \) and \( C₂ \) showed that HR varied 10 beats/min or more in three patients (fig. 1), brachial arterial systolic pressure by 10 mm Hg or more in six (fig. 1), RPP by 200 mm Hg/min or more in 16, LVEDP by 5 mm Hg or more in five (fig. 2), CO by 10% or more in 11 (fig. 3) and SV by 10 ml or more in 10.

Studies During Exercise

During both exercise periods, the initial work loads were 200 kpm in 12 patients and 300 kpm in the remaining eight patients. All 20 patients had angina during both exercise periods. Figure 4 shows the group
TABLE 1. Summary of Hemodynamic Data at Rest and During Exercise-induced Angina

<table>
<thead>
<tr>
<th></th>
<th>Rest</th>
<th>Exercise</th>
<th>Exercise</th>
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<tr>
<td></td>
<td>C₁</td>
<td>C₂</td>
<td>Ex₁</td>
</tr>
<tr>
<td>HR (beats/min)</td>
<td>76 ± 11</td>
<td>79 ± 11</td>
<td>114 ± 12</td>
</tr>
<tr>
<td>BAP (mm Hg)</td>
<td></td>
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<tr>
<td>Systolic</td>
<td>142 ± 13</td>
<td>137 ± 13</td>
<td>167 ± 15</td>
</tr>
<tr>
<td>Diastolic</td>
<td>77 ± 8</td>
<td>76 ± 9</td>
<td>88 ± 8</td>
</tr>
<tr>
<td>Mean</td>
<td>103 ± 10</td>
<td>99 ± 9</td>
<td>120 ± 10</td>
</tr>
<tr>
<td>RPP (mm Hg/min × 10³)</td>
<td>108 ± 23</td>
<td>108 ± 21</td>
<td>190 ± 28</td>
</tr>
<tr>
<td>PAP* (mm Hg)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic</td>
<td>23 ± 9</td>
<td>20 ± 9</td>
<td>43 ± 8</td>
</tr>
<tr>
<td>Diastolic</td>
<td>15 ± 4</td>
<td>13 ± 4</td>
<td>23 ± 4</td>
</tr>
<tr>
<td>Mean</td>
<td>22 ± 5</td>
<td>18 ± 5</td>
<td>33 ± 8</td>
</tr>
<tr>
<td>LVEDP (mm Hg)</td>
<td>18 ± 4</td>
<td>15 ± 4</td>
<td>29 ± 6</td>
</tr>
<tr>
<td>CO (l/min)</td>
<td>7.5 ± 2.1</td>
<td>7.8 ± 1.8</td>
<td>11.4 ± 3.0</td>
</tr>
<tr>
<td>CI (l/min/m²)</td>
<td>3.7 ± 0.8</td>
<td>3.8 ± 0.7</td>
<td>5.9 ± 1.2</td>
</tr>
<tr>
<td>SV (ml/beat)</td>
<td>99 ± 20</td>
<td>100 ± 19</td>
<td>103 ± 21</td>
</tr>
<tr>
<td>LVSWI (g-m/beat/m²)</td>
<td>58 ± 14</td>
<td>58 ± 11</td>
<td>64 ± 19</td>
</tr>
</tbody>
</table>

Values are mean ± sd.

*Data available for 16 patients only.

Abbreviations: C₁ = first control study; C₂ = second control study; Ex₁ = first exercise study; Ex₂ = second exercise study; HR = heart rate; BAP = brachial arterial pressure; RPP = rate-pressure product; PAP = pulmonary arterial pressure; LVEDP = left ventricular end-diastolic pressure; CO = cardiac output; CI = cardiac index; SV = stroke volume; LVSWI = left ventricular stroke work index.

and individual values for the duration of exercise that induced angina during Ex₁ and Ex₂ studies. For the whole group, exercise duration to angina was similar during Ex₁ and Ex₂ (fig. 4). However, the duration of exercise to angina varied by 60 seconds or more in five patients, two of whom experienced angina at higher work loads during Ex₂ (fig. 4). Comparison of hemodynamic data at the onset of angina during Ex₁ and Ex₂ showed that the group mean values for brachial arterial systolic (fig. 1) and mean pressures (p < 0.02), pulmonary arterial mean pressure (p < 0.02) (table 1) and LVEDP (p < 0.02) (fig. 2) were significantly lower during Ex₂. The group mean values for HR (fig. 1), RPP, CO (fig. 3), SV and LVSWI were similar during Ex₁ and Ex₂ (table 1). Significant ST-segment depression at the onset of angina was observed in 16 of the 20 patients during both Ex₁ and Ex₂, and the mean values for ST-segment depression were similar during both Ex₁ and Ex₂ (fig. 4). Four patients did not develop ST depression during either exercise period (fig. 4). During Ex₁ and Ex₂, HR varied by 10 beats/min or more in two patients (fig. 1), brachial arterial systolic pressure by 10 mm Hg or more in seven (fig 1), RPP by 200 mm Hg or more in five, LVEDP by 5 mm Hg or more in eight (fig. 2), CO by 10% or more in five (fig. 3) and SV by 10 ml or more in six.

FIGURE 1. Individual and group values (mean ± sd) for the heart rate and systolic blood pressure during the first control (C₁) and second control (C₂) studies and at the onset of angina during the first exercise (Ex₁) and second exercise (Ex₂) studies. The group mean values for heart rate during C₂ were higher than those during C₁ (p < 0.05), but the values were similar during Ex₁ and Ex₂. The group values for systolic blood pressure were lower during C₁ and Ex₁ (p < 0.02) than the corresponding values during C₂ and Ex₂.
Because the exercise times to angina were not always identical in many patients during Ex1 and Ex2, we examined the hemodynamic data at the same time of exercise before the onset of angina during the two exercise studies. Comparison of data during Ex1 and Ex2 showed lower values for pulmonary arterial mean pressure (29 ± 8 vs 24 ± 7 mm Hg; \( p < 0.01 \)), LVEDP (24 ± 6 vs 20 ± 7 mm Hg; \( p < 0.01 \)), brachial arterial mean pressure (114 ± 8 vs 108 ± 10 mm Hg; \( p < 0.02 \)) during Ex2, while the values for HR (98 ± 12 vs 101 ± 11 beats/min) were similar during the two exercise periods. During Ex1 and Ex2, HR varied by 10 beats/min or more in six patients, brachial arterial mean pressure by 10 mm Hg or more in six and LVEDP by 5 mm Hg or more in 10 patients.

The results therefore consistently show variations in the measured hemodynamic variables during Ex1 and Ex2 at the onset of angina and before the onset of angina at the same exercise time during the two periods. Finally, we also examined the hemodynamic data just before the termination of Ex1 and Ex2, while the patients had already experienced angina for 1½-2 minutes and found lower values for LVEDP (31 ± 6 vs

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**Figure 2.** Individual and group values (mean ± sd) for left ventricular end-diastolic pressure (LVEDP) during the first control (C1) and second control (C2) periods, and at the onset of angina during the first exercise (Ex1) and second exercise (Ex2) studies. The group values for LVEDP were lower during C2 (\( p < 0.001 \)) and Ex2 (\( p < 0.02 \)) than the corresponding values during C1 and Ex1.

**Figure 3.** Individual and group values (mean ± sd) for cardiac output during the first control (C1) and second control (C2) studies and at the onset of angina induced by the first exercise (Ex1) and second exercise (Ex2) studies. The group values were similar during C1 and C2 and during Ex1 and Ex2.

**Figure 4.** Individual and group values (mean ± sd) for exercise duration to angina and ST-segment depression at the onset of angina during the first exercise (Ex1) and second exercise (Ex2) studies. The group values for both variables were similar during Ex1 and Ex2.
26 ± 9 mm Hg; \( p < 0.05 \) and pulmonary arterial mean pressure (37 ± 9 vs 29 ± 1 mm Hg; \( p < 0.02 \)) during Ex₂, while values for HR (114 ± 14 vs 117 ± 15 beats/min) were similar during the two exercise periods.

**Discussion**

Results of the present study indicate that a period of exercise modified the subsequent resting hemodynamics and that many, but not all, of the hemodynamic variables during two successive exercise periods were reproducible.

The patients in the present study were carefully selected, all had stable, exertional angina due to coronary artery disease and none were taking medications or received premedications that could have modified hemodynamics. Strict selection criteria were necessary to evaluate the exercise protocol used in the present study.

After the initial exercise period, many of the circulatory variables at rest were significantly different from the pre-exercise resting values. Thus, after exercise, the values for HR were higher, while brachial arterial systolic and mean pressures, pulmonary arterial systolic, diastolic and mean pressures and LVEDP were significantly lower than the corresponding pre-exercise values. These differences were small but statistically significant. In a previous study of nine patients with ischemic heart disease, Malmborg⁵ reported small reductions in systemic pressure, CO and SV that were not significant. However, Sweatman and colleagues²⁴ restudied eight patients 1 hour after exercise and found that the pulmonary arterial wedge pressure had decreased significantly compared with the pre-exercise values. In 10 patients with cardiac murmurs and no evidence of coronary artery disease, Burkart and colleagues⁴⁴ reported significant changes in resting HR, blood pressure, and SV after exercise. These changes were apparent even after a 30-minute rest after upright bicycle exercise. A reduction in SV after exercise was also reported by Granath and coworkers.⁴⁵ Widimsky and colleagues⁴⁶ found no significant changes in any of the measured variables after exercise.

In the present study, the hemodynamic and clinical variables during two successive periods of exercise-induced angina were not regularly reproducible. Group values for brachial arterial, systolic, and mean pressures, pulmonary arterial mean pressure and LVEDP were significantly lower during Ex₂. The exercise duration to angina, HR, RPP at the onset of angina and CO during exercise-induced anginapectoris were reproducible. The time to angina varied by 60 seconds or more in five of the 20 patients, and two of these five patients had to be exercised at a higher work load to induce angina during Ex₂. Similarly, during the two exercise studies HR varied by 10 beats/min or more in two patients, systolic blood pressure by 10 mm Hg or more in seven, RPP by 200 mm Hg/min or more in five, LVEDP by 5 mm Hg or more in eight, CO by 10% or more in five and SV by 10 ml or more in six.

Our studies have also shown that the hemodynamic variables were not regularly reproducible at the same exercise duration during the two studies before the onset of angina. Thus, the group values for brachial arterial mean pressure, pulmonary arterial mean pressure, and LVEDP were significantly lower during Ex₂.

In similar studies in the supine position, Malmborg⁵ found reduced pulmonary arterial pressure and oxygen uptake, Widimsky and colleagues⁴⁶ found significantly lower pulmonary arterial pressures and pulmonary vascular resistance, and Sweatman and coworkers reported significantly lower pulmonary arterial wedge pressure during Ex₂. Our results agree with those reported by Burkart and colleagues,⁴⁴ who found differences in the hemodynamics during two
successive periods of upright exercise in patients who did not have coronary artery disease. These workers found that at similar work loads (300–900 kpm/min), pulmonary arterial and aortic mean pressures and tension-time index were significantly lower during the Ex₂. Changes in CO were not significant, but SV and LVSW were lower during Ex₂. However, Epstein and co-workers²⁷ found no significant differences in pressures, CO, oxygen uptake, HR, arteriogenous oxygen difference, and left ventricular work in four patients during upright treadmill exercise.

It is difficult to be certain of the mechanism responsible for the differences in the hemodynamic variables during two successive exercise periods. It is possible that the patients were more nervous during the first study, but the HRs at angina during the two exercise periods were similar and the resting HRs during C₂ were consistently higher than during C₁. Catecholamine release during Ex₁ could be responsible for some of the changes between the two rest periods.

Burkart and co-workers²⁸ suggested that the differences in hemodynamic variables during the two successive exercise periods may be due to a shift of blood from the central to the peripheral circulation during Ex₁ that persists throughout the remainder of the investigation. That hypothesis could explain the significantly lower left ventricular filling pressure immediately before and during Ex₂ in the present study.

Sweating induced by exercise could have produced fluid loss and, therefore, volume depletion. However, in the present study, the intensity of exercise was relatively low (all patients experienced angina at work loads of 200–400 kpm/min) and the duration of exercise was less than 6 minutes in all patients. The blood withdrawn during cardiac output determination was reinfused into the patient, thus minimizing any volume depletion due to the blood loss.

A period of ischemia induced by exercise could have increased coronary blood flow due to opening of collaterals, which could have modified the subsequent hemodynamics. We have no information to assess this possibility.

The lack of reproducibility of clinical and hemodynamic events is not unique during invasive exercise stress testing, and similar observations have also been made during pacing-induced angina²⁹,³⁰

Whatever the mechanisms responsible for the differences in hemodynamic variables during the two exercise periods, the results of the present study have important implications. In studies in which exercise stress testing is used to assess the efficacy of therapeutic interventions, it is important to recognize that some hemodynamic variables may not be reproducible.

Thus, in the present study, the group values for exercise time to angina, HR and RPP at the onset of angina were reproducible, but the group values for pulmonary artery pressure and LVEDP were significantly lower during Ex₂. Further, in five of the 20 patients, the exercise duration to angina varied by 60 seconds or more during the two exercise studies. In studies where exercise stress testing is used to evaluate the efficacy of therapeutic interventions, we recommend that the reproducibility of clinical and hemodynamic variables be shown in a given patient, especially when only a small number of patients is being studied. Without the demonstration of reproducibility we do not feel that it is valid to subdivide patients into smaller subgroups of responders and nonresponders to therapy on the basis of changes in clinical and hemodynamic variables. Even when a larger number of patients is studied before and after an intervention, hemodynamic alterations induced by the intervention should be interpreted with caution in view of the findings of the present study. To avoid misinterpretation of the effects of therapy, we strongly recommend that the effects of therapy on hemodynamics should be compared with double-blind placebo therapy, which is regarded as a prerequisite in noninvasive studies.

**References**

2. Messer JV, Levine HJ, Wagman RJ, Gorlin R: Effect of exer-

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**Table 2. (Continued)**

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<th>Pulmonary arterial pressure (mm Hg) Mean</th>
<th>LVEDP (mm Hg)</th>
<th>CO (l/min)</th>
<th>SV (ml/beat)</th>
<th>LVSWI (g-m/beat/m²)</th>
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