Hemodynamic Differences Between Supine and Upright Exercise in Patients with Congestive Heart Failure

BARRY KRAMER, M.D., BARRY MASSIE, M.D., AND NINA TOPC, R.N.

SUMMARY Although the differences in hemodynamic responses to supine and upright exercise have been studied in normal subjects and in patients with angina pectoris, no such comparison has been made in patients with congestive heart failure. Many investigators measure exercise hemodynamics in heart failure patients to assess the effect of vasodilator and inotropic drugs. Both modes of exercise have been used and have often yielded differing results.

We compared the hemodynamic response to supine and upright exercise in 14 patients with stable, New York Heart Association class III chronic heart failure. During upright exercise, peak heart rate was higher (124 ± 15 vs 115 ± 18 beats/min, p < 0.025) and peak mean arterial pressure was lower (95 ± 17 mm Hg, p < 0.25), yielding similar double products. Although the peak left ventricular filling pressure was slightly lower during upright exercise (40 ± 7 vs 35 ± 10 mm Hg, p < 0.05), the maximum cardiac and stroke indexes were not significantly different (3.6 ± 0.8 vs 3.4 ± 0.8 l/min/m² and 30 ± 8 vs 30 ± 6 ml/m², upright vs supine exercise). In contrast to these relatively similar hemodynamic responses, exercise capacity was significantly greater during upright exercise (peak work load 336 ± 84 vs 293 ± 73 kpm/min, p < 0.1; maximum oxygen consumption 12.1 ± 2.4 vs 9.8 ± 1.9 ml/min/kg, p < 0.001). We conclude that either exercise method may be used to assess the hemodynamic effects of drugs, but that exercise capacity should be measured in the upright position.

THE HEMODYNAMIC RESPONSES to supine and upright exercise have been compared in normal subjects and in patients with coronary disease who were not in heart failure, and important differences have been noted. 1-4 Recently, there has been considerable interest in hemodynamic measurements during exercise in chronic congestive heart failure, especially in patients given vasodilator and inotropic drugs. 5-24 Although both supine and upright bicycle exercise have been used, no information is available comparing these different forms of stress in patients with congestive heart failure.

It is particularly important to compare the hemodynamic differences between these two forms of exercise in this setting. Because of their increased end-diastolic volumes, they might be affected less by positional variations in venous return. In addition, alterations in autonomic nervous system responsiveness sometimes present in chronic heart failure might affect the reflex responses to change in posture.25,26 We designed the present study to perform such a comparison in a cohort of patients with stable, congestive heart failure in order to choose a method for evaluating their exercise capacity and their hemodynamic responses to drug interventions.

Methods

Patients
We studied 14 male patients, ages 48–72 years (mean 60 years) with New York Heart Association class III congestive heart failure who were undergoing right-heart catheterization for monitoring the initiation of vasodilator therapy. The etiology of cardiac decompensation was coronary artery disease in nine patients, coronary disease with a history of hypertension in two, primary cardiomyopathy in two and rheumatic heart disease after mitral valve replacement in one patient. Symptomatic heart failure had been present for 3 months to 8 years (mean 3.1 years). Left ventricular ejection fraction determined by radionuclide angiocardiography ranged from 10% to 31% (mean 19 ± 6%) (±SD).

All patients were clinically stable on constant doses of digoxin and diuretics for at least 2 weeks before the study. None of the patients were receiving vasodilator therapy during this period, although four had been treated previously with hydralazine, nitrates or prazosin. Clinical stability was further documented by treadmill exercise testing. In each subject, two treadmill tests performed at least 1 week apart during this observation period had shown less than a 2-minute difference in exercise tolerance, which varied from 7 to 12 minutes on a modified Naughton protocol in which work load was increased in 2-minute stages.27 The end point during exercise testing was dyspnea or fatigue in each patient. None experienced chest pain during exercise. Dyspnea was felt to be of cardiac origin in each subject, since none had clinically significant pulmonary disease on pulmonary function testing (as judged by FEV₁, FVC, or FEV₁/FVC below 70% of predicted).

Study Design
This study was conducted under a protocol approved by the Institutional Review Board, and all patients gave informed consent before participating. One to 4 days before the actual measurements, patients practiced on both the supine and upright bicycle ergometers. Each patient performed supine and sitting bicy-
cicle exercise to exhaustion using identical inertial ergometers, beginning at a work load of 200 kpm/min and increasing by 100 kpm/min every 3 minutes. This permitted a preliminary determination of their maximal work load and exercise time. During the subsequent hemodynamic studies, measurements were taken during maximal exercise in the final 2 minutes of the highest work load completed during the preliminary tests.

All patients were hospitalized 24 hours before catheterization to ensure medication and dietary compliance. On the day after admission, diuretics were withheld for at least 8 hours before the first study. Right-heart catheterization was performed with a balloon-tipped thermodilution catheter, and a small cannula was inserted into a radial artery. Hemodynamic values were measured at least 1 hour after instrumentation to ensure accurate resting baseline measurements. In both positions, the transducers were set 5 cm below the angle of Louis. Baseline rest heart rate, arterial pressure, pulmonary arterial pressure, pulmonary capillary wedge pressure, right atrial pressure, and cardiac output with the patient supine and then after sitting on the bicycle ergometer for 3–5 minutes were measured. Then, sitting bicycle exercise was performed. During exercise, heart rate and arterial and pulmonary arterial pressures were monitored continuously, and pulmonary capillary wedge and right atrial pressures were measured every minute. The pulmonary capillary wedge pressure at end expiration was used as an index of left ventricular filling pressure, except in four patients in whom pulmonary arterial diastolic pressure was substituted because of inability to obtain a satisfactory wedge tracing. In each of these patients, the comparability of the two pressure readings at rest had previously been established. Cardiac output was measured in triplicate by the thermodilution technique in the final 2 minutes of the highest completed exercise stage. When a greater than 10% variability was encountered, an additional value was obtained and the outlying measurement excluded. Arterial and pulmonary arterial blood gases were drawn at rest and at peak exercise for calculation of arterial venous oxygen difference and oxygen consumption.

Patients were allowed to recover for at least 12 hours before supine exercise, with both the pulmonary arterial and arterial catheters remaining in place. Although diuretics were administered after the initial exercise test, they were again withheld for a comparable period before the second exercise run. Supine resting and supine exercise hemodynamic measurements were performed in the same manner described for the upright tests. Supine exercise was performed on the day after upright exercise in all patients, since it took place in conjunction with the first of several scintigraphic studies necessarily scheduled in the morning because of laboratory time constraints.

Calculations and Analysis

The following indexes were derived from the hemodynamic measurements:

\[
\text{Cardiac index} = \frac{\text{cardiac output}}{\text{body surface area}}
\]

\[
\text{Stroke volume index} = \frac{\text{cardiac index}}{\text{heart rate}}
\]

\[
\text{Systemic vascular resistance} = \frac{\text{mean arterial pressure} - \text{right atrial pressure}}{\text{cardiac output} \times 80}
\]

\[
\text{O}_2 \text{ consumption} = (\text{arterial O}_2 \text{ content} - \text{mixed venous O}_2 \text{ content}) \times \text{cardiac output} \times 1.34
\]

The supine resting, maximum supine exercise, upright resting, and maximum upright exercise measurements were submitted to a two-way analysis of variance to determine the effects of change in both posture and exercise. If this test revealed a significant \((p < 0.05)\) difference, the significance level of various comparisons between these states was determined by the Newman-Keuls multiple-range test, with a \(p\) value \(< 0.05\) considered significant. All values are expressed as mean \(\pm\) SD.

Results

Comparability of Patients at Time of the Two Exercise Tests

The clinical status of the patients did not change between the two tests. Weight did not fluctuate by more than 1 kg, and there was no trend toward either an increase or a decrease. Supine resting hemodynamic measurements before each exercise run were comparable, further confirming that the patients were in a comparable state at the time of the two exercise measurements. Each patient exercised to the same maximum work load achieved in his preliminary test.

Comparison of Supine and Upright Measurements at Rest

There were few significant differences between the hemodynamic values obtained in the supine and sitting positions at rest (table 1). Heart rate was similar \((80 \pm 12\) vs \(85 \pm 10\) beats/min). Mean arterial pressure rose slightly, but insignificantly, in the sitting position \((87 \pm 11\) vs \(91 \pm 12\) mm Hg), but the increase in systemic vascular resistance was statistically significant \((1540 \pm 320\) vs \(1830 \pm 380\) dyn-sec-cm\(^{-5}\), \(p < 0.001)\). Both ventricular filling pressures were slightly lower in the upright position \((23 \pm 7\) vs \(21 \pm 12\) mm Hg for the left ventricular filling pressure; \(8 \pm 5\) vs \(6 \pm 5\) for the right atrial pressure), but only the latter difference achieved statistical significance. While cardiac index was not significantly different in the two positions, stroke volume was modestly lower in the sitting position \((28 \pm 9\) vs \(24 \pm 5\) ml/m\(^2\), \(p < 0.01)\).

Hemodynamic Effects of Exercise

Significant changes in the majority of indexes occurred during both supine and upright exercise. During supine exercise, heart rate, mean arterial, pulmonary arterial, left ventricular filling, and right atrial pressures all rose significantly, as did cardiac index. Sys-
Table 1. Supine and Upright Hemodynamic Measurements

<table>
<thead>
<tr>
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<th>Supine</th>
<th>Upright</th>
<th></th>
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<tbody>
<tr>
<td></td>
<td>Rest I</td>
<td>Exercise II</td>
<td>Rest III</td>
<td>Exercise IV</td>
<td></td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>80 ± 12</td>
<td>115 ± 18</td>
<td>85 ± 10</td>
<td>124 ± 15</td>
<td>0.001</td>
</tr>
<tr>
<td>Mean arterial pressure (mm Hg)</td>
<td>87 ± 11</td>
<td>102 ± 15</td>
<td>91 ± 12</td>
<td>95 ± 17</td>
<td>0.001</td>
</tr>
<tr>
<td>Mean pulmonary artery pressure (mm Hg)</td>
<td>34 ± 11</td>
<td>55 ± 10</td>
<td>33 ± 15</td>
<td>50 ± 15</td>
<td>0.001</td>
</tr>
<tr>
<td>Left ventricular filling pressure (mm Hg)</td>
<td>23 ± 7</td>
<td>40 ± 7</td>
<td>21 ± 12</td>
<td>35 ± 10</td>
<td>0.001</td>
</tr>
<tr>
<td>Right atrial pressure (mm Hg)</td>
<td>8 ± 5</td>
<td>18 ± 6</td>
<td>6 ± 5</td>
<td>14 ± 5</td>
<td>0.001</td>
</tr>
<tr>
<td>Cardiac index (l/min/m²)</td>
<td>2.2 ± 0.7</td>
<td>3.4 ± 0.8</td>
<td>2.0 ± 0.5</td>
<td>3.6 ± 0.8</td>
<td>0.001</td>
</tr>
<tr>
<td>Stroke index (ml/m²)</td>
<td>28 ± 9</td>
<td>30 ± 8</td>
<td>24 ± 5</td>
<td>30 ± 6</td>
<td>NS</td>
</tr>
<tr>
<td>Systemic vascular resistance (dyn·sec·cm⁻⁵)</td>
<td>1540 ± 320</td>
<td>1040 ± 250</td>
<td>1830 ± 380</td>
<td>940 ± 170</td>
<td>0.001</td>
</tr>
<tr>
<td>Pulmonary vascular resistance (dyn·sec·cm⁻⁵)</td>
<td>240 ± 100</td>
<td>180 ± 90</td>
<td>250 ± 120</td>
<td>170 ± 80</td>
<td>0.025</td>
</tr>
<tr>
<td>Double product (× 10³)</td>
<td>7.0 ± 2.0</td>
<td>11.8 ± 3.0</td>
<td>7.8 ± 1.9</td>
<td>11.7 ± 2.8</td>
<td>0.001</td>
</tr>
<tr>
<td>Arteriovenous oxygen difference (ml/dl)</td>
<td>6.2 ± 1.2</td>
<td>11.7 ± 1.8</td>
<td>6.9 ± 1.4</td>
<td>12.6 ± 1.7</td>
<td>0.001</td>
</tr>
<tr>
<td>Oxygen consumption (ml/min/kg)</td>
<td>3.6 ± 0.5</td>
<td>9.8 ± 1.9</td>
<td>3.7 ± 0.6</td>
<td>12.1 ± 2.4</td>
<td>0.001</td>
</tr>
<tr>
<td>Maximum work load (kpm/min)</td>
<td>—</td>
<td>293 ± 73</td>
<td>—</td>
<td>336 ± 84</td>
<td>—</td>
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</table>

The slight increase in stroke volume (from 28 ± 9 to 30 ± 8 ml/m²) during supine exercise was not statistically significant.

Similar changes occurred with upright exercise. There were significant increases in heart rate and pulmonary arterial, left ventricular filling and right atrial pressures, and in cardiac and stroke volume indexes. Systemic and pulmonary vascular resistances decreased. The modest increase in mean arterial pressure (from 91 ± 12 to 95 ± 17 mm Hg) was not statistically significant.

Comparison of Hemodynamic Responses to Upright and Supine Exercise (figs. 1–3)

While heart rate rose with both forms of exercise, the peak heart rate was higher in the sitting position (124 ± 15 vs 115 ± 18 beats/min, p < 0.025). Conversely, mean arterial pressure was higher during maximum supine exercise (102 ± 15 vs 95 ± 17 mm Hg, p < 0.025), as a result of a significantly greater increase during exercise (15 vs 4 mm Hg). However, the rate-pressure products at maximal exercise were nearly identical in the two positions (11.8 × 10³ vs 11.7 × 10³).

Both ventricular filling pressures were lower during maximum upright exercise (35 ± 10 vs 40 ± 7 mm Hg for the left ventricle, p < 0.05; 14 ± 5 vs 18 ± 6 mm Hg for the right atrial pressure, p < 0.001). These differences in maximum exercise resulted from a combination of lower resting values together with slightly smaller increases during exercise. There were no significant differences between cardiac and stroke volume indexes during maximal supine and upright exercise. However, the increase in both of these measurements was significantly greater in the upright position (cardiac index 1.6 ± 0.6 vs 1.2 ± 0.8 l/min/m², p < 0.05; stroke volume index 6 ± 4 vs 2 ± 8 ml/m², p < 0.05). Systemic vascular resistance fell to comparable levels at maximal exercise in both positions, but since it was higher in the upright position at rest, this reflected a significantly greater decrease during upright exercise (889 ± 358 vs 507 ± 313 dyn·sec·cm⁻⁵, p < 0.005).

Patients exercised longer (8.8 ± 2.0 vs 7.3 ± 2.1 min, p < 0.001) and completed a higher work load (336 ± 84 vs 293 ± 73 kpm/min, p < 0.01) in the upright position. Similarly, maximum oxygen consumption was significantly higher with upright exercise (12.1 ± 2.4 vs 9.8 ± 1.9 ml/min/kg, p < 0.001).

Discussion

Background

The availability of new modalities of treatment for patients with refractory congestive heart failure has resulted in many hemodynamic studies in these patients. Since most of these patients are symptomatic predominantly with activity, a number of investigators have evaluated the effects of drug therapy on their hemodynamic response to exercise. These studies have produced variable results. In some, little change in exercise hemodynamic measurements has been noted. In others, exercise hemodynamics have im-
proved, but exercise capacity has not. A combination of hemodynamic improvement and an increase in exercise tolerance has only occasionally been reported.

These data are difficult to evaluate because investigators have used a variety of drugs and exercise protocols. Some have used supine bicycle exercise and others have used upright exercise. Although the differing responses to these two forms of exercise have been evaluated in normal subjects and in patients with coronary artery disease, no such comparison has been made in patients with congestive heart failure. The increased ventricular preload and the more marked effect of changes in afterload in this group might be expected to produce responses different from those in normal subjects. In addition, the alterations in autonomic responses that often characterize chronic heart failure might affect the hemodynamic response to change in posture at rest and, possibly, during exercise as well.

The hemodynamic differences at rest and during exercise between the supine and upright positions in normal subjects, as reported by Thadani and Parker, are summarized in Table 2. At rest, they noted higher heart rates and lower left ventricular filling pressures, stroke indexes, and cardiac indexes in the sitting position. Mean arterial and pulmonary arterial pressures were similar. At maximal exercise, heart rate and rate-pressure product were higher in the sitting position, while left ventricular filling pressure and stroke index were significantly lower. No differences in mean arterial pressure, cardiac index, or peak work load were noted between the two methods of exercise.

Thadani et al. compared the effect of position on rest and exercise hemodynamic measurements in 20 patients with coronary artery disease. As in normal subjects, cardiac and stroke indexes were lower at rest in the sitting position. Unlike normal subjects at rest, they exhibited no appreciable change in left ventricular filling pressure, mean pulmonary arterial pressure, or heart rate with a change in posture, and mean arterial pressure was higher in the upright position. During exercise, which in this group was limited by angina pectoris, heart rate, mean arterial pressure, cardiac index, and rate-pressure product were higher in the sitting position, while left ventricular filling pressure was significantly lower than during supine exercise. The authors concluded that left ventricular performance during exercise was better in the sitting position in patients with coronary disease.

Little information is available concerning differences in hemodynamic measurements in the supine and upright positions in patients with congestive heart failure.
failure. We reported the effect of change in posture on resting hemodynamics before and during vasodilator therapy in a different group of heart failure patients. Our findings before vasodilators were similar to the present results, but we noted a much greater effect from changing posture during combined hydralazine and nitrate therapy.

Findings of the Present Study

Our findings in congestive heart failure are compared to those in normal and coronary disease in Table 2. As in normal subjects and patients with coronary disease, there are differences between supine and upright positions in the hemodynamics both at rest and during exercise. In addition, congestive heart failure patients differ from these other groups in several important ways.

At rest, the only significant differences between the upright and supine measurements were the slightly lower right atrial pressure and stroke volume index and a somewhat higher systemic vascular resistance while sitting. Cardiac index and left ventricular filling pressure did not fall and heart rate did not increase in the upright position, as in normal subjects. These differences from normal subjects probably reflect the elevated left ventricular preload that characterizes heart failure patients and its relative independence from postural changes in venous return.

Not surprisingly, our congestive heart failure patients performed poorly during exercise. They attained only low maximum heart rates and cardiac indexes, while developing very high left ventricular filling pressures. However, as in normal subjects, maximum heart rate was higher in the upright position, left ventricular filling pressure was somewhat lower, and cardiac indexes were comparable in the two positions. Unlike normal subjects and coronary patients, congestive heart failure patients had a higher mean arterial pressure in the supine position.

Peak double products during the two forms of exercise were similar in the congestive heart failure patients. In contrast, this index of cardiac work load was significantly higher with upright exercise in both normal subjects and patients with coronary disease. Exercise duration, maximum attained external work load, and maximal oxygen consumption were both higher with upright exercise in congestive heart failure patients. In contrast, Thadani and Parker reported that normal subjects could complete comparable work loads with the two forms of exercise.

This disparity between the supine and upright exercise capacities in congestive heart failure patients is of interest. Exercise capacity in normal subjects is determined by oxygen delivery to exercising muscle, and this mainly reflects maximal cardiac output. Since in normal persons maximum upright and supine cardiac indexes with the two methods are comparable, the finding that their exercise capacities with the two methods are equal is not surprising. Maximum supine and upright cardiac measurements were also similar in our heart failure group, but as noted, upright exercise capacity was significantly higher. This discordance indicates that at least in the supine position, exercise is limited by factors other than the cardiac output. Possi-

Table 2. Positional Differences in Hemodynamic Measurements in Heart Failure Patients Compared with Normal Subjects and Coronary Disease Patients

<table>
<thead>
<tr>
<th></th>
<th>Normal</th>
<th>CHF</th>
<th>CAD</th>
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<tbody>
<tr>
<td></td>
<td>R</td>
<td>EX</td>
<td>R</td>
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<tr>
<td>Heart rate</td>
<td>↑</td>
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<tr>
<td>Mean arterial pressure</td>
<td>↔</td>
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<tr>
<td>Left ventricular filling pressure</td>
<td>↓</td>
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<td>↔</td>
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<tr>
<td>Cardiac index</td>
<td>↓</td>
<td>↔</td>
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<tr>
<td>Stroke index</td>
<td>↓</td>
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<td>↓</td>
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<tr>
<td>Double product</td>
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<tr>
<td>Work load</td>
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</table>

Directional changes in normal subjects and coronary patients are those reported by Thadani et al. Abbreviations: ↑ = significantly higher upright vs supine; ↓ = significantly lower upright vs supine; ↔ = no significant difference upright vs supine; CHF = congestive heart failure; CAD = coronary artery disease; R = rest; EX = exercise.
bly, the cardiac output is less efficiently delivered to the exercising muscle groups in the supine position. A second possibility is that these muscles, because of severe deconditioning, are less able to utilize the delivered oxygen. The significantly different arteriovenous oxygen differences noted with the two forms of exercise is consistent with both of these explanations. Another factor may be the somewhat higher left ventricular filling pressures during supine exercise, which could explain the earlier appearance of limiting dyspnea in some patients. Finally, the identical double products at peak exercise in both positions in the heart failure patients suggest that myocardial oxygen demand might be a limiting factor in them, although not in normal persons.

Implications

Thus, patients with congestive heart failure are in some ways similar but in other ways different from normal subjects in their hemodynamic performance in the supine and upright positions. At rest, they show less of an effect of the decreased venous return in the upright position. Directional changes in all variables during exercise were similar to those in normal subjects. Since cardiac output rises to similar levels with the two forms of exercise, and since the exercise-induced increases in left ventricular filling pressure are also similar, both forms of exercise offer the opportunity to evaluate the hemodynamic effects of interventions, such as vasodilator or inotropic drugs. However, exercise capacity is clearly greater in the upright position in patients with congestive heart failure. Thus, if one’s objective is to evaluate exercise capacity or to assess the effect of drug therapy on such measurements, upright exercise appears to be a more suitable method of stress.

References

Hemodynamic differences between supine and upright exercise in patients with congestive heart failure.
B Kramer, B Massie and N Topic

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