Exercise and the Heart

Pulmonary Artery Pressure and Oxygen Consumption Measurement during Supine Bicycle Exercise*

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Previous studies of hemodynamic changes in patients with angina have documented a rise in left ventricular filling pressure that causes an increase in pulmonary artery wedge pressure and pulmonary artery diastolic pressure. Recently, ambulatory pulmonary artery pressure monitoring was used in patients with documented coronary artery disease. However, there are few data on normal values of pulmonary artery diastolic pressure during dynamic exercise, especially in different age groups and states of fitness. There have been conflicting claims that there is no increase, a significant increase, a consistent increase, or an occasional decrease in the pulmonary artery pressure of healthy man in response to exercise. Moreover, comparison of published data is difficult because of variations in the exercise posture, the duration of exercise and the workloads employed. Measuring ventilatory oxygen consumption is costly, especially in terms of laboratory equipment. Thus, it seems desirable to investigate the possibility of assessing aerobic work capacity by estimating the maximal oxygen uptake based upon a calculation using normal values. The purpose of this study was to determine these normal values and the correlation between the workload increment and ventilatory oxygen consumption.

Methods and Materials

Three groups of individuals were studied. Group A included nine male water-polo players (mean age, 18±3 years) and group B included 11 (eight men, three women) medical students (mean age, 22±1 year). These individuals were examined by a cardiologist and were found to be healthy. Group C consisted of 13 patients (eight men, five women; mean age, 41±7 years), hospitalized for evaluation of atypical chest pain and selected for participation in the study only after clinical evaluation determined that the chest pain was extracardiac in origin. During clinical evaluation, all of the patients had physical examinations, ECG, exercise ECG, x-ray films, echocardiography, lung function tests, and laboratory examinations. In seven patients, coronary angiography was also indicated by the attending clinician. Results of all tests and examinations were negative. Pulmonary artery pressure monitoring was performed during exercise in ten patients from group C; one patient had elevated resting pulmonary artery pressure (45/32 mm Hg), and in two cases, the catheter could not be introduced into the pulmonary artery. The study was approved by the ethics committee and informed written consent was obtained from all patients.

Exercise Test

Patients performed a continuous bicycle exercise test in the supine position, using a Jaeger bicycle ergometer, to individual maximal effort. Each patient was given a one-minute warm-up. The first stage consisted of six minutes of exercise at a workload of 25 watts and a rate of 40 rpm. The workload was then progressively increased 25 watts every six minutes. A two-lead (CM-3) electrocardiogram and pulmonary artery pressure curve were recorded on paper at one minute intervals using a three-channel Hellige EK-26 electrocardiogram.

Pulmonary Artery Pressure Monitoring

A polyurethane Pulmoflex Vygon catheter was introduced via the cephalic vein to a pulmonary artery without fluoroscopy in a special laboratory for floating catheterization. A Statham strain gauge coupled to a Hellige amplifier was used for the pressure recordings. The reference point was taken at the mid-thoracic level in the supine position. No complication occurred during or after the catheterization and exercise testing.

Oxygen Consumption Measurement

Oxygen consumption was measured in 30 patients during dynamic exercise. To determine oxygen uptake, expired air was collected in plastic bags for three minutes at rest, and during the last minute of each progressive workload. The volume of expired air was measured at a constant flow using a Wright respirometer. A Jaeger paramagnetic analyzer was used for oxygen analysis. Corrections were made for oxygen uptake at STPD (0°C, 760 mm Hg, and moisture-free).

Results

The mean working capacity and changes in pulmonary artery pressure are presented during maximal supine exercise (Table 1). The working capacity of group A and B was significantly higher than in group C. The pulmonary artery systolic and diastolic pressures of different study groups were very similar and were within the normal limits. The pulmonary artery systolic pressure increased significantly in all groups, but these values remained "in the normal range" after.

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exercise testing as well (≤38 mm Hg). We observed a slight, but significant increase of diastolic pulmonary artery pressure in groups A and B only. At the same time, these higher values were also within the normal limits (≤22 mm Hg). Using the data of group B, a linear regression was calculated to demonstrate the changes of pulmonary artery pressure during exercise testing (Fig 1). A strong correlation (r = 0.935-0.87) was found between the rise of pulmonary artery systolic, diastolic and mean pressure and the load step.

The values of oxygen consumption are presented in Table 2. We found a normal basal metabolic state in group B, showing an oxygen consumption corresponding to 1 MET. In groups A and C the basal oxygen requirement was about 40 and 30 percent higher, respectively. The voluntary maximal oxygen uptake capacity was between 36.2-24.2 ml O₂/kg/min, and was significantly higher in group A compared to groups B and C. The Vo₂ increment during exercise testing was very similar in the different groups. We found a strong correlation (r = 0.98) between the workload and Vo₂ increments (Table 2).

**DISCUSSION**

Hemodynamic monitoring of patients with acute myocardial infarction is an established method for determining therapeutic measures. Pulmonary artery pressure recording and long-term monitoring during exercise testing or during normal daily activity also is important in patients with coronary artery disease. To define the limit of normal changes, we selected three groups of individuals who presented without any disease that could influence pulmonary artery pressure during exercise testing. Pulmonary artery systolic pressure increased during exercise testing and had a strong correlation with incremental workload. Pulmonary artery systolic pressure values (including the highest measurement of 38 mm Hg) remained within normal limits. Thadani and Parker and Levy et al made similar observations previously. Ekelund and Holmgren also observed a linear increment of right

**PULMONARY ARTERY PRESSURE DURING SUPINE BICYCLE EXERCISE TEST IN MEDICAL STUDENTS (n=11)**

![Graph](image1.png)

**Figure 1.** Linear regression correlation of pulmonary artery pressures with different load steps: + = mean values, dotted lines = confidence intervals.

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**Table 1—Work Capacity and Pulmonary Artery Pressure**

<table>
<thead>
<tr>
<th>Working Capacity (Watts)</th>
<th>Pulmonary Artery Systolic Pressure</th>
<th>Pulmonary Artery Diastolic Pressure</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Rest (range)</td>
<td>Exercise (range)</td>
</tr>
<tr>
<td>Group A</td>
<td></td>
<td></td>
</tr>
<tr>
<td>n = 9</td>
<td>200 ± 43*</td>
<td>19.3 ± 4.4 (15-25)</td>
</tr>
<tr>
<td>Group B</td>
<td></td>
<td></td>
</tr>
<tr>
<td>n = 11</td>
<td>173 ± 34‡</td>
<td>19.4 ± 4.4 (15-25)</td>
</tr>
<tr>
<td>Group C</td>
<td></td>
<td></td>
</tr>
<tr>
<td>n = 10</td>
<td>123 ± 36</td>
<td>22.2 ± 4.5 (15-25)</td>
</tr>
</tbody>
</table>

* = highly significant difference (p<0.001) compared to group C; † = highly significant difference (p<0.001) compared to resting values; ‡ = significant difference (p<0.05) compared to resting values; § = significant difference (p<0.05) compared to Group C.

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Table 2—Measured Oxygen Consumption at Rest and During Supine Bicycle Exercise Test in Different Study Groups

<table>
<thead>
<tr>
<th>Group</th>
<th>n</th>
<th>VO₂ Max (mL/kg/min)</th>
<th>VO₂ Increment (mL/10 Watts)</th>
<th>VO₂ Increment Correlation Coefficient</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group A</td>
<td>9</td>
<td>36.2 ± 8.6*</td>
<td>113</td>
<td>0.98</td>
</tr>
<tr>
<td>Group B</td>
<td>11</td>
<td>27 ± 4.9</td>
<td>122</td>
<td>0.97</td>
</tr>
<tr>
<td>Group C</td>
<td>10</td>
<td>24.2 ± 8.3</td>
<td>107</td>
<td>0.99</td>
</tr>
<tr>
<td>All Groups</td>
<td></td>
<td>29.1</td>
<td>114</td>
<td></td>
</tr>
</tbody>
</table>

* = significant (p<0.005) difference comparing groups B and C.

Epstein et al. observed only a slight increase in mean pulmonary artery pressure during maximal exercise testing in a small group of normal individuals who were considered as having no hemodynamically significant heart disease after thorough investigation (including cardiac catheterization). Pulmonary artery diastolic pressure stayed below the 20 mm Hg value at maximal exercise in all but one case in group B where it was 22 mm Hg at 200 watts. Earlier reports give similar findings.

Regarding the oxygen demand of bicycle exercise, there is a wide scatter (between 96 and 143 ml) of published values, but the generally accepted value is 120-125 ml for each 10 watts. Our result (114 ml O₂/10 watts) is near this accepted value.

In group B, where the oxygen uptake at rest corresponded to the expected 3.5 ml O₂/kg/min, the VO₂ increment per 10 watt increase was 122 ml, almost identical with the conventional value, in spite of the fact that the 40 rpm pedal rate in our study was below the recommended, optimal frequency.

Conclusions

Pulmonary artery systolic and diastolic pressure increased linearly with the external power in a supine, progressive bicycle ergometer test in all three groups. The increased pressure values did not exceed "normal" limits. Ventilatory oxygen demand at a workload of 50 watts and above corresponded to the usually accepted 3.5 ml O₂/kg/min plus 120 ml for each 10 watts.

References

1. Levy RO, Shapiro LM, Wright CC, Mockus L, Fox KM. Hemodynamic response to myocardial ischemia during unrestricted activity, exercise testing and atrial pacing assessed by ambulatory pulmonary artery pressure monitoring. Br Heart J 1986; 56:12-3
15. Davies CTM. Limitations to the prediction of maximum oxygen intake from cardiac frequency measurements. J Appl Physiol 1968; 24:700-06