Comparison of hemodynamic responses to static and dynamic exercise

G. R. BEZUCHA, M. C. LENSER, P. G. HANSON, AND F. J. NAGLE
Biodynamics Laboratory, University of Wisconsin Madison, Madison, Wisconsin 53706

Bezucha, G. R., M. C. Lenser, P. G. Hanson, and F. J. Nagle. Comparison of hemodynamic responses to static and dynamic exercise. J. Appl. Physiol.: Respirat. Environ. Exercise Physiol. 53(6): 1589–1593, 1982.—Eight healthy male adults (25–34 yr) were studied to compare hemodynamic responses to static exercise (30% MVC in leg extension), static–dynamic exercise (one-arm cranking, 66 and 79% \( VO_2 \max \)-arm), and dynamic exercise (two-leg cycling, 58 and 82% \( VO_2 \max \)-legs). Leg extension (LE) strength was measured by a spring scale. Cranking and cycling were performed on a Quinton bicycle ergometer. \( VO_2 \) was measured using an automated open-circuit system. Heart rate (HR) was monitored from a CM-5 ECG lead, and arterial pressure (Pa) was measured from an indwelling brachial artery catheter. Cardiac output (Q) was measured using a CO2 rebreathing procedure. Total peripheral resistance (TPR) was calculated using the mean arterial pressure (Pa) as the systemic pressure gradient. In 30% LE, a significant (P < 0.05) Pa increase occurred (pressor response) mediated primarily by an increase in Q. One-arm cranking and two-leg cycling at similar relative \( VO_2 \) demands resulted in nearly identical increases in Pa due to different contributions of Q and TPR. Q and the arteriovenous O2 difference varied as a function of \( VO_2 \) regardless of the mode of exercise (static or dynamic). On the other hand, the HR response, which accounted for increased Q in the exercises containing a static component, and Pa varied with mode of exercise. Any generalized scheme of cardiovascular control during exercise must account for the potential influence of dynamic and static components of the exercise.

**Methods**

Eight normal healthy adult males, 25–34 yr, served as subjects. All were volunteers, and informed consent was obtained prior to the study. At least 24 h before each experiment subjects completed a series of preliminary tests and measurements.

Resting arterial pressure was determined by auscultation in each arm to verify that there was no discrepancy in blood pressure between right and left arms. An Allen test was also performed to verify a patent palmar arch.

Maximal leg extension strength was measured with the dominant leg. The subject was seated and positioned so that the lower leg extended vertically downward from a table edge. A leather strap was fastened around the ankle. This, in turn, was attached to a cable and a fixed standard spring scale against which subjects exerted knee extension force with the quadriceps muscles.

The one-arm and two-leg cycling maximal work loads and maximal one-arm and two-leg \( VO_2 \) uptake (\( VO_2 \max \)) values were determined with a continuous incremental test to subjective exhaustion. Work loads were increased every 2 min during the respective one-arm cranking and two-leg cycling tests. An electrically braked bicycle ergometer (Quinton, Seattle, WA) was used for this purpose.

On the day of an experiment, the subject was first positioned supine on a table. The antecubital surface of the left arm was then prepared with povidone iodine (Betadine) and alcohol, and 1% lidocaine was infiltrated over the brachial artery. A 20-gauge 1.25-in. Jellico catheter was inserted percutaneously into the brachial artery, 2–3 cm distal to the antecubital fossa. The catheter was connected by 15-cm extension tubing to a Statham
A P23D6 pressure transducer and kept patent with heparinized saline. The transducer calibration was frequently checked against a mercury column.

The subject was then seated on a bicycle ergometer with the seat adjusted to ensure full leg extension on the downward thrust. The pressure transducer was positioned at the level of the fourth intercostal space, and brachial artery systolic, diastolic, and mean pressures were recorded on a Gilson polygraph recorder. Heart rate (HR) was determined from an electrocardiogram (ECG) (CM-5) tracing using the Gilson recorder.

Q was determined by the CO₂-rebreathing technique described by Jones et al. (8). Gas exchange of O₂ and CO₂ at the lung was measured using the Beckman Metabolic Cart. The O₂ analyzer (OM-11) and CO₂ analyzer (LB-2) were calibrated with known gas mixtures analyzed on the Scholander apparatus. End-tidal CO₂ was sampled continuously at the mouthpiece and measured on-line with a Beckman LB-2 analyzer. This output was recorded on a Gilson recorder to estimate arterial PCO₂ and mixed venous PCO₂ for CO₂ rebreathing.

Initially, rest measurements of HR, arterial pressure, Q, and VO₂ were made. Stroke volume (SV) was calculated from HR and Q data and arteriovenous O₂ differences from Q and VO₂ data. TPR was calculated using Pa as the systemic circulation gradient assuming right atrial pressure was near zero or at least not changing from rest to exercise (2). Subjects were coached in maintaining a normal rhythmic breathing pattern to minimize a respiratory influence on right atrial pressure. In all procedures the catheterized arm was supported and immobile and could not contribute to the mechanical work done.

**Dynamic leg exercise.** The subjects cycled at a load to demand 25% VO₂ max-legs. This was continued for 3 min. During steady state (3rd min) all measurements previously described were repeated. After rest intervals of 5 min and the return of the blood pressure to rest levels, the procedure was repeated at loads estimated to demand 50 and 75% VO₂ max-legs. Again in the 3rd min at these loads the measurements were repeated.

**Static leg exercise.** After a 10-min rest and the return of blood pressure to rest levels, the subject was seated on a table for the leg extension maneuver. The pressure transducer was adjusted to the level of the fourth interspace. The subject was asked to hold a 30% MVC for 3 min with the noncatheterized arm resting on his lap. The spring scale, previously calibrated with known weights, was observed by the subject and an attendant to ensure that the required tension was maintained. In the 3rd min, all measurements were completed.

**One-arm cycling.** The Quinton ergometer, adapted for arm work, was positioned in front of the seated subject for one-arm cranking. Following a 10-min rest, when CV functions had returned to rest level, the subject commenced right-arm cranking at a load estimated to demand 50% of VO₂ max-arm. Cardiovascular measurements were again made in the 3rd min. The procedure was repeated at loads estimated to demand 50% of VO₂ max-arm.

**Analysis of data.** Means, standard deviations, and standard errors were calculated for all measurements. An analysis of variance and Scheffe multiple comparison procedure were used to test for differences. For this purpose, the 0.05 level of significance was used.

### RESULTS

Table 1 presents descriptive physical and physiological data on the subjects. The mean VO₂ max of 46.4 ml·kg⁻¹·min⁻¹ for two-leg cycling is somewhat higher than normal values reported for other populations in this age range (18). The VO₂ max-arm was 41% of that attained in two-leg cycling.

Figure 1 shows arterial pressure responses and VO₂ for rest, 30% LE, two-leg cycling, and one-arm cranking. Leg cycling and one-arm cranking loads are expressed as mean percentages of VO₂ max, the VO₂ max values being 3.43 ± 0.22 and 1.40 ± 0.22 l/min for legs and one arm, respectively (Table 1). While the study was designed to achieve identical relative leg cycling and one arm cranking energy expenditures of 50 and 75% VO₂ max, the actual results were somewhat variable. Nonetheless, the means of 58% VO₂ max-legs and 66% VO₂ max-arm and 82% VO₂ max-legs and 79% VO₂ max-arm are similar enough to justify comparison of hemodynamic responses.

Leg extension at 30% MVC resulted in a significant increase in Pa. Both systolic and diastolic pressure increased to effect a pressor response. TPR was increased but not significantly (P > 0.05) in LE.

Leg cycling (Fig. 1) was marked by significant increases (P < 0.05) in systolic and mean pressure as work intensity increased from 38 to 58 to 82% VO₂ max-legs. The increase in diastolic pressure from rest was minimal and nonsignificant (P > 0.05) over these loads. However, one-arm cranking resulted in significant increases in systolic, diastolic, and mean pressures (P < 0.05) as energy expenditure increased from rest to 66 and 79% VO₂ max-arm. Figure 1 and Table 2 show that the mean arterial pressure response at similar relative demands, 58% VO₂ max-legs and 66% VO₂ max-arm, are nearly equal, 113 ± 3 and 115 ± 3 Torr, respectively. A similar pattern occurred at the higher loads, 82% VO₂ max-legs and 79% VO₂ max-arm, where mean pressures were 124 ± 4 and 127 ± 5 Torr, respectively. Pa showed no consistent relationship to absolute VO₂ (Fig. 1, Table 2).

Table 2 shows the responses of VO₂, Q, arteriovenous O₂ difference, HR, SV, Pa, and TPR to the various exercise modes and intensities. In 30% LE, Q increased by 19% from rest, and TPR by 8%. Neither of these changes was statistically significant (P > 0.05). Clearly, the change in Q contributed more to the pressor response observed in 30% LE. The elevation in Q in 30% LE was due to an increase in HR, since SV decreased significantly (P > 0.05) from rest.

### Table 1. Physical data

<table>
<thead>
<tr>
<th>Age, yr</th>
<th>Height, cm</th>
<th>Wt, kg</th>
<th>VO₂ max-legs</th>
<th>VO₂ max-arm</th>
</tr>
</thead>
<tbody>
<tr>
<td>29.6</td>
<td>180.0</td>
<td>74.3</td>
<td>3.43 ± 0.22</td>
<td>4.64 ± 0.22</td>
</tr>
<tr>
<td>±1.0</td>
<td>±2.4</td>
<td>±3.1</td>
<td>±0.22</td>
<td>±0.29</td>
</tr>
</tbody>
</table>

Values are means ± SE; n = 8.
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**FIG. 1.** Systolic, diastolic, and mean arterial pressures (horizontal line within bars) for rest, 30% MVC in leg extension, 66 and 79% VO₂ max-leg, and 38, 58, and 82% VO₂ max-leg. Absolute VO₂ for rest and each exercise mode and intensity is also shown. SE bands are indicated.

![Blood Pressure Graph](image)

### TABLE 2. Hemodynamic and metabolic responses to static and dynamic exercise

<table>
<thead>
<tr>
<th>Condition</th>
<th>VO₂ (_{\text{ml/min}})</th>
<th>Cardiac Output, (\text{l/min})</th>
<th>a-vO₂ Diff, (\text{ml/100 ml})</th>
<th>Heart Rate, beats/min</th>
<th>Stroke Volume, ml</th>
<th>MAP (Pa), mmHg</th>
<th>TPR, dynes-cm⁻¹</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rest</td>
<td>324 ± 33</td>
<td>5.7 ± 0.3</td>
<td>5.6 ± 0.6</td>
<td>70 ± 7</td>
<td>85 ± 7</td>
<td>94 ± 3</td>
<td>1,352 ± 103</td>
</tr>
<tr>
<td>30% LE</td>
<td>356 ± 42</td>
<td>6.8 ± 0.7</td>
<td>8.3 ± 0.6</td>
<td>110 ± 6</td>
<td>62 ± 5</td>
<td>118 ± 6</td>
<td>1,496 ± 131</td>
</tr>
<tr>
<td>Leg 38 ± 1%</td>
<td>1,298 ± 76</td>
<td>13.2 ± 1.3</td>
<td>10.0 ± 0.6</td>
<td>102 ± 4</td>
<td>129 ± 12</td>
<td>103 ± 3</td>
<td>661 ± 56</td>
</tr>
<tr>
<td>VO₂ max-legs</td>
<td>1,969 ± 103</td>
<td>18.9 ± 1.5</td>
<td>10.5 ± 0.6</td>
<td>132 ± 4</td>
<td>144 ± 11</td>
<td>113 ± 3</td>
<td>496 ± 38</td>
</tr>
<tr>
<td>Leg 58 ± 4%</td>
<td>911 ± 27</td>
<td>9.8 ± 0.7</td>
<td>9.4 ± 0.6</td>
<td>112 ± 4</td>
<td>88 ± 7</td>
<td>115 ± 3</td>
<td>971 ± 64</td>
</tr>
<tr>
<td>VO₂ max-legs</td>
<td>2,758 ± 172</td>
<td>21.9 ± 1.0</td>
<td>12.7 ± 0.8</td>
<td>164 ± 4</td>
<td>131 ± 5</td>
<td>124 ± 4</td>
<td>461 ± 56</td>
</tr>
<tr>
<td>Arm 60 ± 4%</td>
<td>1,084 ± 25</td>
<td>10.8 ± 0.7</td>
<td>10.1 ± 0.7</td>
<td>130 ± 6</td>
<td>85 ± 7</td>
<td>127 ± 4</td>
<td>954 ± 70</td>
</tr>
</tbody>
</table>

Values are means ± SE; \(n = 8\). MAPB, mean arterial blood pressure; TPR, total peripheral resistance; LE, leg extension.

In all leg cycling and one-arm cranking efforts, Q was significantly increased from rest \((P < 0.05)\). Both HR and SV contributed to this change in leg cycling. In one-arm cranking, SV changed minimally from rest levels \((P > 0.05)\), so that increased HR was responsible for the increase in Q. TPR decreased significantly \((P < 0.05)\) from rest in leg cycling and one-arm cranking. The magnitude of this decrease in TPR increased with the absolute energy expenditure. The TPR at the higher two loads in leg cycling was approximately one-half that observed in one-arm cranking.

There was a progressive increase in mean arteriovenous O₂ difference as VO₂ increased (Table 2 and Fig. 2). This was also true for Q as Fig. 2 shows. However, when HR was plotted as a function of VO₂, this linear relationship was not observed (Fig. 2). Where a pressor response was observed (30% LE and arm cranking), the mean HR was higher than that at 38% VO₂ max-legs, in which the metabolic cost of the exercise was greater. The mean HR response at 79% VO₂ max-arm approximated that at 58% VO₂ max-legs, despite the fact that the absolute VO₂ in the arm cranking averaged nearly a liter less than that in leg cycling.

**DISCUSSION**

Numerous investigators \((6, 9, 12, 13)\) have shown that the normal CV response to static handgrip exercise is an elevated mean arterial blood pressure mediated primarily by an increased Q with minimal, if any, contribution by TPR. Our observations for static contraction of the quadriceps muscles \((30\% LE)\) are consistent with this finding. Despite the larger muscle mass involved in 30% MVC in LE, the pattern of response was the same. While neither Q nor TPR increased significantly, the magnitude of the increase in Q, 19%, compared with 8% for TPR suggests a primary role for Q in effecting the significant pressor response. As others have shown for handgrip exercise \((12, 13)\), the increased Q in 30% LE was due to increased HR. In our study SV was observed to decrease significantly.

The hemodynamic responses to leg cycling were typical for dynamic exercise of a large muscle mass. This was marked by a widened pulse pressure (increased systolic and minimally changed diastolic), an increased Q with HR and SV contributing to the effect, and a decrease in TPR \((63-66\%)\) at the two higher loads. On the other hand, one-arm cranking, having both dynamic (cycling...
rhythm) and static (pedal gripping) components elicited a predominantly static exercise response (pressor effect). An increased systolic and diastolic pressure was associated with an elevated $Q$ affected solely by a HR increase. TPR decreased but to a far more limited extent than in leg cycling.

Bevegard et al. (2), Secher et al. (16), and Clausen et al. (4) reported a similar pattern of blood pressure responses in progressive exercise in leg cycling and two-arm cranking. However, a unique feature of our study is that where equivalent relative $V_{O2}$ requirements were imposed in cycling and cranking, the mean arterial pressures were nearly identical. This occurred despite marked differences in the relation between $Q$ and TPR during the various exercise modes.

Lind and McNichol (11) reported similar $P_a$ responses for static exercise of different muscle groups during contraction at the same relative percentages of maximal tension. In their experiments, a pressor response was a consistent finding. We also observed $P_a$ to be a function of relative exercise intensity ($\%V_{O2\ max}$), and this occurred whether the exercise was static or dynamic and with and without a pressor response.

We have confirmed this apparent relationship between $P_a$ and relative exercise intensity ($\%V_{O2\ max}$) on seven additional healthy male subjects (mean age 28 yr). This cohort performed one-arm cranking and leg cycling, with each subject completing three to four exercise loads between 20 and 77$\%V_{O2\ max}$ for both modes in continuous tests. Average linear regression lines for $\%V_{O2\ max}$ vs. directly measured mean arterial pressure were determined (Fig. 3) from individual data. Neither the slope nor the intercept mean differences for cranking vs. cycling comparisons approached statistical significance ($P > 0.40$).

In each exercise mode that produced a pressor response, i.e., in 30$\%$ LE and 66 and 79$\%V_{O2\ max-arm}$ TPR remained higher than in the leg exercise mode and $Q$ was increased as a function of HR. The relatively higher TPR may be explained as follows. First, compared with leg cycling, 30$\%$ LE and arm cranking does not involve as large a metabolic demand (Table 2), so that the vascular dilation in active muscle would be limited and probably masked, to some degree, by reflex vasoconstriction in other nonactive vascular beds (4). Second, both leg extension and one-arm cranking are activities with a significant isometric component (gripping the pedals in one-arm cranking), which would result in added mechanical compression of the muscle vasculature. Although maintenance of a higher level of TPR was a consistent response to the static exercise mode compared with the dynamic mode, the TPR response only accounted for a portion of the pressor effect observed.

Increased cardiac output mediated by heart rate had to be a major determinant of the pressor response. However, the mechanism for the heart rate response appeared to differ between exercise modes. In our experiments the mean heart rates in 30$\%$ LE and one-arm cranking (Table 2) exceeded those at 38$\%V_{O2\ max}$ where the total metabolic response was greater. This would argue against HR increasing exclusively as a function of $O_2$ demand, which might be monitored by active muscle receptors sensitive to local metabolic demands (5, 14). The linear responses of $Q$ and arteriovenous $O_2$ difference to increasing absolute $V_{O2}$ (Fig. 2) also argues against active muscle $O_2$ deficiency as a cause of the exaggerated HR response to static and static-dynamic exercise. This relationship of $Q$ and arteriovenous $O_2$ difference to absolute $V_{O2}$ also indicates that these responses are independent of the mode of exercise. Blomqvist et al. (3) have made similar observations. In contrast to the observations of Blomqvist et al. (3), we did not find a consistent relationship between HR and active muscle mass as measured by absolute $V_{O2}$ (Fig. 2). We speculate that the exaggerated HR response to exercise with a high static component may be more related to perception of effort and added cortical input to cardiovascular control centers (7, 10) than to afferent reflexes from peripheral muscle receptors (5, 14).

The close coupling of $Q$ to $V_{O2}$ independent of exercise mode preserves the $O_2$ transport function to active muscle. However, the mode of exercise and corresponding mixture of static and dynamic components act to produce variable influences on HR and $P_a$ which respectively
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Fig. 3. Average linear regression lines and equations for one-arm cranking and leg cycling. \( \% \text{VO}_2\text{max} \) vs. mean arterial pressure (MAP). \( n = 7 \).

assure \( Q \) and peripheral blood flow. Any generalized scheme of CV control during exercise must account for the potential influence of dynamic and static components of the exercise.

REFERENCES


