RELATIONSHIP BETWEEN OXYGEN UPTAKE AND OXYGEN SUPPLY SYSTEM DURING CONSTANT-LOAD SUPINE EXERCISE

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ABSTRACT: The purpose of this study was to determine the relationship between oxygen uptake (VO2) kinetics and oxygen supply system during constant-load exercise in the supine position. The main exercises which were carried in supine position were moderate at an intensity corresponding to 80% of ventilatory threshold (VT) and heavy at an intensity corresponding to 20% of the difference between VT and peak VO2. Oxygenation level was obtained from inactive muscle by near-infrared spectroscopy (NIRS). This index is used to express the distribution of oxygen supply to inactive muscle during exercise [19]. After an exponential rise in VO2 (primary component), VO2 during moderate exercise reached a steady state, while during heavy exercise it continued to increase gradually (slow component). The HR kinetics which reflected systemic O2 supply in the supine position was similar to that of VO2 in main exercise tests. However, time constants of primary and slow components in VO2 were not significantly related to those in HR in each exercise mode. Furthermore, oxygenation level decreased after about 0.5 min from the onset of exercise and showed a minimum value at about 2 min and then recovered to the initial level during moderate and heavy exercises. Since there were no significant correlation coefficients in the time constant between VO2 and HR in each component in each exercise mode and since O2 supply to active muscle is affected by systemic O2 supply and distribution of O2 supply to inactive muscle, it is unlikely that VO2 is related to O2 supply to active muscle in supine position.

KEY WORDS: primary component, slow component, inactive muscle, oxygen supply, oxygenation level

INTRODUCTION

Oxygen uptake (VO2) has been analyzed by the application of a mathematical equation for VO2 kinetics in constant load exercise [1, 28]. In moderate exercise intensity (below the ventilatory threshold, VT), VO2 rises mono-exponentially (primary component) after a time delay until a steady state of VO2 is achieved. In heavy exercise (above the VT), VO2 is additionally increased after the primary component until the end of exercise [2-4, 13, 14]. This additional increase in VO2 starting from 2-3 min after the onset of exercise is defined as the slow component [1, 3-4, 20, 22, 26]. Poole et al. [22] simultaneously measured pulmonary VO2 and leg VO2 during exercise using a cycle ergometer, and they demonstrated that ~86% of the increment in pulmonary VO2 beyond the third minute of exercise (i.e., slow component) could be accounted for by the increase in leg VO2. However, by this experiment, it is not proved whether VO2 kinetics is determined by a substance within active muscle or the oxygen supply to active muscle.

The possible factor that limits VO2 kinetics has been discussed [23]. One theory is oxygen delivery to the exercising muscle. Results of some previous studies, for example, studies using inspired hypoxia [9], supine exercise [12] and prior load exercise [11], have supported the oxygen delivery theory. Another theory is oxygen utilization in the exercising muscle such as oxygen delivery to oxygen requirement mismatch or oxidative enzyme inertia. According to this theory, 1) the kinetics of oxygen supply to the muscle is appreciably faster than pulmonary VO2 kinetics, 2) increased arterial O2 content and muscle O2 availability do not speed VO2 kinetics during moderate or heavy exercise [17], and 3) there is a temporal correspondence between phosphor creatine kinetics and VO2 kinetics [24]. Thus, it is still unclear whether VO2 kinetics is determined by peripheral oxygen consumption such as oxygen consumption in active muscle or by oxygen delivery. Recently, near-infrared spectroscopy (NIRS) has been used to determine oxygenation levels in tissues. The oxygenation level
determined by NIRS varies depending on the balance of oxygen supply and oxygen consumption [7], but oxygen consumption in an inactive muscle is considered to be constant [19]. Therefore, oxygenation level in an inactive muscle can reflect the oxygen supply [19, 29]. Furthermore, it is known that the majority of cardiac output (Q) is distributed to active muscle with an increase in exercise intensity during exercise, though the majority of Q at rest is distributed to internal organs such as the brain and kidney. Therefore, if VO₂ kinetics is affected by oxygen supply, VO₂ kinetics should be affected by not only oxygen supply to the whole body but also by the attenuating degree of oxygen supply in inactive muscle. However, to our knowledge, there has been no study to determine whether the distribution of systemic oxygen supply affects VO₂ kinetics. Therefore, the purpose of this study was to determine the relationship between VO₂ kinetics and oxygen supply system in supine-leg exercise.

MATERIALS AND METHODS

Subjects: Nine healthy males participated in this study (Table 1). After the objective and procedure of the experiment and the risks associated with the experiment had been explained, written consent to participate in the study was obtained from each subject. This study was approved by the local ethics committee at our university.

Experimental protocol: Each subject attended our laboratory for seven tests. The time interval between two consecutive tests was at least 2 days. On the first test day, the subjects’ body characteristics were measured. Each subject was instructed to refrain from intense physical exercise, drinking, and taking caffeine for 24 h prior to each visit.

Incremental-load exercise. Incremental-load exercise was performed using a bicycle ergometer in which the work load can be adjusted by a computer (232CXL, Combi, Tokyo, Japan) in the supine position to determine the ventilatory threshold (VT). Each subject rested for 3 min in the supine position. After 5-min warming-up at 10 watts, the work load was increased by 20 watts every minute until exhaustion, i.e., until the subject could no longer maintain a rotation rate of 60 rpm. Peak oxygen uptake (VO₂peak) obtained during exercise was defined as the peak value.

Constant-load exercise. Constant-load exercise was performed using a bicycle ergometer in the supine position. Each subject was instructed not to gear up for the upper limbs and not to move the upper limbs during exercise. Each subject rested for 3 min in the supine position. After 5 min warming-up at 10 watts, each subject performed 6-min constant-load exercises. One exercise was moderate exercise performed at an exercise intensity of 80% of the VT (Moderate) and the other was heavy exercise performed at VT + (VO₂peak - VT) × 0.2 (Heavy). All exercises were performed at 60 rpm. Each test was performed three times to reduce the breath-by-breath noise.

Measurements: Respiratory gas exchanges and heart rate. Data on oxygen uptake (VO₂) and carbon dioxide output (VCO₂) were obtained breath-by-breath using a respiratory gas analyzer (AE-280s, Minato Medical Science, Osaka, Japan). The flow volumes of inspiration and expiration were determined using a hot-wire respiratory flow meter. The respiratory flow meter was calibrated using a 2-L syringe. The O₂ and CO₂ concentrations were analyzed using a zirconium sensor and infrared absorption analyzer, respectively. The gas analyzer was calibrated by known standard gas (O₂: 15.17%, CO₂: 4.92%). Heart rate (HR) was recorded using a heart rate monitor installed in the respiratory gas analyzer. These data were measured continuously during rest, warming-up, exercise, and recovery periods.

Muscle oxygenation level. Changes in muscle oxygenation were estimated using near-infrared spectroscopy (NIRS) (HEO-200N, Omron, Kyoto, Japan). The device used for measurements consisted of a probe and a computerized control system. The NIRS probe consisted of a light source and an optical detector, with a distance of 3.0 cm between the light source and the detector. The dual-wavelength light (760 and 850 nm) emitted from the light source penetrates tissue, where it is either absorbed or scattered, and some of the scattered light returns to the optical detector. The depth of penetration of the radiation is about 1.5 cm [16].

The NIRS probe and a pneumatic cuff (MT-720, Mizuho, Japan) were fixed to the biceps brachii muscle of the left upper arm of each subject. NIRS signals were measured during rest, warming-up, exercise, and recovery periods with a sampling time of 5 s. After a recovery period, arterial occlusion by inflation of the cuff to 300 mmHg was carried out for more than 10 min. During arterial occlusion, oxygenation level decreased abruptly and then showed a plateau in all subjects.

Data analysis: VT was determined using the following criteria: (1) an increase in VCO₂ related to VO₂, (2) an increase in ventilation (VE) related to VO₂, (3) an increase in VE · VO₂ without a decrease in VE · CO₂, (3) an increase in the fractional concentration of oxygen in end-tidal gas (FETO₂) without a decrease in the fractional concentration of carbon dioxide output in end-tidal gas (FETCO₂) [5, 25, 27]. During constant-load exercise, VO₂ measured breath-by-breath was plotted against time. The VO₂ data were time-interpolated second-by-second and then VO₂ kinetics was approximated by the following equations (Moderate: eq. (1), Heavy: eq. (2)):
TABLE 1. CHARACTERISTICS OF SUBJECTS AND RESULTS OF INCREMENTAL-LOAD EXERCISE IN THE SUPINE POSITIONS

<table>
<thead>
<tr>
<th>Subject</th>
<th>Age (yr)</th>
<th>Height (cm)</th>
<th>Weight (kg)</th>
<th>Peak VO₂ (ml min⁻¹)</th>
<th>WR (W)</th>
<th>VT (W)</th>
<th>%peak</th>
<th>Exercise Intensity</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>31</td>
<td>165.5</td>
<td>64.3</td>
<td>2303</td>
<td>225</td>
<td>109</td>
<td>48</td>
<td>83</td>
</tr>
<tr>
<td>B</td>
<td>21</td>
<td>169.0</td>
<td>63.1</td>
<td>2322</td>
<td>235</td>
<td>106</td>
<td>45</td>
<td>78</td>
</tr>
<tr>
<td>C</td>
<td>21</td>
<td>177.0</td>
<td>59.5</td>
<td>2166</td>
<td>200</td>
<td>108</td>
<td>54</td>
<td>76</td>
</tr>
<tr>
<td>D</td>
<td>19</td>
<td>165.0</td>
<td>55.6</td>
<td>2066</td>
<td>220</td>
<td>102</td>
<td>46</td>
<td>74</td>
</tr>
<tr>
<td>E</td>
<td>25</td>
<td>170.4</td>
<td>66.5</td>
<td>2574</td>
<td>240</td>
<td>118</td>
<td>49</td>
<td>87</td>
</tr>
<tr>
<td>F</td>
<td>23</td>
<td>177.0</td>
<td>73.3</td>
<td>2750</td>
<td>270</td>
<td>129</td>
<td>48</td>
<td>94</td>
</tr>
<tr>
<td>G</td>
<td>22</td>
<td>166.0</td>
<td>60.0</td>
<td>2842</td>
<td>275</td>
<td>134</td>
<td>49</td>
<td>100</td>
</tr>
<tr>
<td>H</td>
<td>23</td>
<td>168.0</td>
<td>64.3</td>
<td>1973</td>
<td>205</td>
<td>109</td>
<td>53</td>
<td>79</td>
</tr>
<tr>
<td>I</td>
<td>27</td>
<td>176.0</td>
<td>72.9</td>
<td>3278</td>
<td>263</td>
<td>109</td>
<td>41</td>
<td>86</td>
</tr>
<tr>
<td>Mean</td>
<td>23.6</td>
<td>170.4</td>
<td>64.4</td>
<td>2474.9</td>
<td>237.0</td>
<td>113.9</td>
<td>48.3</td>
<td>84.0</td>
</tr>
<tr>
<td>SD</td>
<td>3.6</td>
<td>5.0</td>
<td>5.9</td>
<td>423.2</td>
<td>27.5</td>
<td>11.0</td>
<td>3.9</td>
<td>8.8</td>
</tr>
</tbody>
</table>

RESULTS

The subject’s results are presented in Table 1. Figure 1 shows the kinetics of VO₂ during moderate (●) and heavy (●) constant-load exercises in supine position. Both VO₂ kinetics in moderate exercise and that in heavy exercise increased rapidly. Thereafter, VO₂ kinetics becomes constant in moderate exercise but continued to increase slightly in heavy exercise until the end of exercise. Results of two-way ANOVA for repeated measures showed that both VO₂ kinetics for moderate exercise and that for heavy exercise had significant interactive effects (p<0.05). Thus, the results of one-way ANOVA for repeated measures showed that both VO₂ kinetics for moderate and heavy exercises at 0.5 min from the onset of exercise significantly increased compared to the baseline level (p<0.05).

Figure 2 shows the kinetics of HR during moderate (●) and heavy (●) constant-load exercises in supine position. Both HR kinetics in moderate exercise and that in heavy exercise increased rapidly. Thereafter, HR kinetics become constant in moderate exercise but continued to increase slightly in heavy exercise until the end of exercise. Results of two-way ANOVA for repeated measures showed that both VO₂ kinetics for moderate and heavy exercises at 0.5 min from the onset of exercise significantly increased compared to the baseline level (p<0.05).

Figure 3 shows oxygenation level in inactive muscle during moderate (●) and heavy (●) constant-load exercises in supine position. Both oxygenation level in moderate exercise and that in heavy exercise showed significant interactive effects (p<0.05). Oxygenation level in moderate exercise and that in heavy exercise were significantly decreased at about 2 min after onset of exercise compared to the baseline level (p<0.05).

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Figure 2 shows the kinetics of HR during moderate (●) and heavy (●) constant-load exercises in supine position. Both HR kinetics in moderate exercise and that in heavy exercise increased rapidly. Thereafter, HR kinetics become constant in moderate exercise but continued to increase slightly in heavy exercise until the end of exercise. Results of two-way ANOVA for repeated measures showed that both HR kinetics for moderate exercise and that for heavy exercise had significant interactive effects (p<0.05). Thus, the results of one-way ANOVA for repeated measures showed that both HR kinetics for moderate and heavy exercises at 0.5 min from the onset of exercise significantly increased compared to the baseline level (p<0.05).

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...
level increased in both heavy and moderate exercises until the end of exercise.

Table 2 shows the parameters of VO₂ and HR predicted by model fitting in moderate and heavy constant-load exercises in supine position. VO₂\textsubscript{base}, HR\textsubscript{base}, time delays (TD\textsubscript{p}) of the primary component, and time constants (τ\textsubscript{p}) of the primary component were not significantly different between moderate and heavy exercise, but amplitudes (A\textsubscript{p}) of the primary component in moderate exercise showed significant differences (p<0.05) from those in heavy exercise. There was no significant difference in time constants of the primary component between VO₂ kinetics and HR kinetics. There was also no significant difference in TD\textsubscript{s} of VO₂ kinetics and HR kinetics. There was no significant difference in time constants of the slow component between VO₂ kinetics and HR kinetics. There was also no significant difference in TD\textsubscript{s} of VO₂ kinetics and HR kinetics. The kinetics of VO₂ and that of HR increased at similar times (p<0.05).

However, there was no significant correlation coefficient in τ\textsubscript{p} in moderate (r=0.609) and heavy exercises (r=0.300) and there was also no correlation coefficient in τ\textsubscript{s} (r=0.137) between VO₂ kinetics and HR kinetics.

The major results of this study performed in supine position were as follows. The time constants of the primary component (τ\textsubscript{p}) in VO₂ kinetics were not significantly different from those of the HR kinetics in moderate and heavy exercise. Time delays (TD\textsubscript{s}) and time constants of the slow component (τ\textsubscript{s}) in VO₂ kinetics were not significantly different from those in HR kinetics. However, there were no significant correlation coefficients in τ\textsubscript{p} and τ\textsubscript{s} between VO₂ kinetics and HR kinetics. On the other hand, the kinetics of oxygenation in inactive muscle started to decrease from about 0.5 min after the onset of exercise. The magnitude of decrease in oxygenation level was greater in heavy exercise than in moderate exercise. Furthermore, oxygenation level showed a minimum (89.0±4.4% in moderate exercise and 80.0±6.9% in heavy exercise) at about 2 min after the onset of exercise and then recovered to the baseline level in both moderate and heavy exercises.

In the supine leg exercise, unlike in upright leg exercise, hydrostatic pressure in the foot is decreased and blood flow shifts to the heart. As a result, since preload for the heart increases, SV is higher than that at the sitting position [21]. In fact, SV slightly increases from rest to light exercise intensity and then does not change even if exercise intensity is increased [15]. Since exercise was started from pre-exercise to moderate or heavy exercise in this study, SV should have been constant. Therefore, the kinetics of Q was estimated by HR response in the supine exercise. The value of τ\textsubscript{p} in HR kinetics during moderate exercise was not significantly different from that in VO₂ kinetics and the correlation coefficient between τ\textsubscript{p} in VO₂ and that in HR was not significant, suggesting that systemic oxygen supply is not the determining factor of VO₂ kinetics at the onset of exercise in supine position. Furthermore, we indirectly estimated oxygen supply to inactive
Relationship between oxygen uptake and oxygen supply system during constant-load supine exercise

<table>
<thead>
<tr>
<th>Parameters</th>
<th>VO₂ (ml min⁻¹, beats min⁻¹)</th>
<th>HR (beats min⁻¹)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Heavy</td>
<td>Moderate</td>
</tr>
<tr>
<td></td>
<td>521.0 ± 34.6</td>
<td>509.7 ± 35.8</td>
</tr>
<tr>
<td></td>
<td>75.6 ± 10.3</td>
<td>74.2 ± 12.6</td>
</tr>
<tr>
<td><strong>Primary component</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>1263.9 ± 165.7</td>
<td>702.1 ± 126.8</td>
</tr>
<tr>
<td>A₀ (ml min⁻¹, beats min⁻¹)</td>
<td>14.0 ± 3.6</td>
<td>17.4 ± 3.5</td>
</tr>
<tr>
<td>TD₀ (s)</td>
<td>25.4 ± 4.6</td>
<td>23.5 ± 6.7</td>
</tr>
<tr>
<td>τ₀ (s)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>48.0 ± 10.2</td>
<td>27.4 ± 5.4</td>
</tr>
<tr>
<td><strong>Slow component</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>148.5 ± 66.3</td>
<td>14.1 ± 7.2</td>
</tr>
<tr>
<td>Aₚ (ml min⁻¹, beats min⁻¹)</td>
<td>113.9 ± 11.6</td>
<td>116.8 ± 41.5</td>
</tr>
<tr>
<td>TDₚ (s)</td>
<td>142.5 ± 84.0</td>
<td>152.6 ± 99.0</td>
</tr>
<tr>
<td>τₚ (s)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note: Values are means±SD; n=9 subjects. A₀ and Aₚ, amplitudes of response; τ₀ and τₚ, time constants; TD₀ and TDₚ, time delay.

* Significantly different from Heavy (p<0.05).
# Significantly different from VO₂ (p<0.05).

In this study, oxygenation level in inactive muscle started to decrease at about 0.5 min after the onset of exercise, suggesting inhibition of oxygen supply to inactive muscle. This means that the oxygen supply to active muscle was greater and faster than that predicted by HR kinetics.

With regard to heavy exercise, τ₀ in HR kinetics was not significantly different from that in VO₂ kinetics and the correlation coefficient between τ₀ in VO₂ and that in HR were not significant. Additionally, oxygenation level started to decrease at about 0.5 min after the onset of exercise. These results suggest that oxygen supply to active muscle is unlikely to determine the oxygen uptake as suggested in moderate exercise. On the other hand, in the slow component, TDₚ and τₚ in VO₂ kinetics were not significantly different from those in HR kinetics and the correlation coefficient between τₚ in VO₂ and that in HR was also not significant. Therefore, it is unlikely that VO₂ kinetics in slow component is also determined by oxygen supply to active muscle in supine position.

Buono et al. [6] and Davis et al. [8] reported that oxygenation level measured by NIRS is influenced by skin blood flow. If this is the case, oxygenation level should be increased. Thus, the actual oxygenation level must be much lower than the measured level. Therefore, it is thought that oxygen supply to inactive muscle is more inhibited and in consequently reduced during exercise. However, after 2 min, oxygenation levels in moderate and heavy exercises returned to baseline level after the decrease. This might be due to the effect of skin blood flow. After the onset of exercise, body temperature is increased and then skin blood flow could be increased especially in heavy exercise compared to moderate exercise. But the kinetics of recovery in oxygenation level in moderate exercise where skin blood flow could not be increased so much was the same as that in heavy exercise. This suggests that the effect of skin blood flow on oxygenation might be minimal. The results of present study support the result of study by Mancini et al. [18] showing that the effect of skin blood flow is minimal.

CONCLUSIONS

In summary, we evaluated oxygen supply to the whole body (Q) estimated by HR kinetics during supine constant-load exercise and peripheral oxygen supply to active muscle estimated by oxygenation level in the inactive biceps brachii muscle. Consequently, there were no significant correlation coefficients in the time constant between VO₂ and HR in primary component in moderate and heavy exercises as well as slow component in heavy exercise. Furthermore, VO₂ supply to active muscle is not affected by systemic O₂ supply and its distribution of O₂ supply to inactive muscle. Therefore, it is unlikely that VO₂ is related to O₂ supply to active muscle in supine position. And VO₂ may be determined in peripheral portion.

REFERENCES


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