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

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ABSTRACT: The purpose of this study was to determine the relationship between oxygen uptake ($\dot{V}O_2$) and the oxygen supply system during incremental load in the supine position. Each subject (n=6) performed an incremental-load supine exercise test (20 W min^{-1}). $\dot{V}O_2$ and heart rate (HR) were measured breath-by-breath during exercise. Oxygenation level was obtained from the inactive biceps brachii muscle using near-infrared spectroscopy (NIRS). $\dot{V}O_2$ and HR increased linearly in proportion to increase in work rate (WR) after a time delay. The linear relationships between $\dot{V}O_2$ and WR and between HR and WR below the ventilatory threshold (VT, 112.9±11.7 W) were extrapolated to WR above the VT. Estimated HR was not significantly different from that measured above VT. Oxygenation level slightly decreased after the VT and decreased exponentially after the respiratory compensation point (RCP, 175.8±21.3 W). Oxygenation level decreased from 100% at warming-up exercise (10 W) to 63.3±14.0% at exhaustion. In this study, HR and oxygenation level were regarded as oxygen supply to the whole body in the supine position and oxygen supply to inactive muscle, respectively. Therefore, an increase in HR suggests a linear increase in oxygen supply to active muscle through exercise. In addition to the effect of HR, a decrease in oxygenation level in inactive muscle could induce an increase in oxygen supply to active muscle. However, $\dot{V}O_2$ showed a linear increase throughout the exercise in the supine position. Judging from the present indirect indicators of HR and oxygenation level, it is unlikely that $\dot{V}O_2$ kinetics is determined by oxygen supply to active muscle in the supine position.

KEY WORDS: oxygen consumption, oxygen supply, inactive muscle, excess oxygen uptake, oxygenation level

INTRODUCTION

It has been reported that oxygen uptake ($\dot{V}O_2$) kinetics in incremental-load exercise was linearly increased in relation to an increase in exercise intensity [31]. However, it has been suggested that the $\dot{V}O_2$ kinetics shows nonlinearity during both constant- and incremental-load exercises at high power output [1,33]. This nonlinear component in high work rate has been defined as "excess $\dot{V}O_2$". There has been discussion as to whether the linear and/or nonlinear components of $\dot{V}O_2$ kinetics are determined by oxygen utilization or oxygen supply [6,8,17,21,24]. In the oxygen supply hypothesis, it has been shown that the rate of increase in $\dot{V}O_2$ is slowed when muscle O_2 delivery is impaired by 1) inspired hypoxia [6], 2) supine exercise [13], and 3) prior exercise [12]. On the other hand, in the oxygen utilization theory, it has been shown that 1) the kinetics of oxygen supply to the muscle is appreciably faster than pulmonary $\dot{V}O_2$ kinetics, 2) increased arterial O_2 content and muscle O_2 availability do not speed up $\dot{V}O_2$ kinetics during moderate or heavy exercise [21], and 3) there is a temporal correspondence between creatine phosphate kinetics and $\dot{V}O_2$ kinetics [25]. Thus, it is still unclear whether $\dot{V}O_2$ kinetics is determined by the oxygen consumption system subsisting in active muscle or by oxygen supply.

It has been shown that the majority of cardiac output ($\dot{Q}$) is distributed to active muscle with an increase in exercise intensity, while the majority of $\dot{Q}$ at rest is distributed to internal organs such as the brain and kidney [26]. In incremental-load exercise, the work load starts from a light work rate and ends with a high work rate. Therefore, the distributable rate of oxygen supply evidently becomes high for active muscle at a high work rate level. In fact, Yano et al. [32] reported that oxygenation level in an inactive upper arm increased slightly for a short period and then gradually decreased and started to sharply decrease after a certain point of ramp leg exercise, suggesting a decrease in oxygen supply to inactive muscle at a high exercise intensity. During this decreasing period, excess $\dot{V}O_2$ was

also observed. They found that the decrement of oxygenation level in an inactive muscle at exhaustion was related to maximal $\dot{V}O_2$ [32]. These results suggest that oxygen supply to active muscle was increased by a decrease in oxygen supply to inactive muscle, and hence $\dot{V}O_2$ is increased in high-intensity exercise. However, this was not demonstrated except for the maximal level in their study. Since they only measured HR in increment-load exercise in the upright exercise position, oxygen supply to the whole body by cardiac output could not be estimated due to the change in stroke volume.

If it is hypothesized that $\dot{V}O_2$ kinetics is affected by oxygen supply, not only oxygen supply to the whole body but also the attenuation ratio of oxygen supply in an inactive muscle against whole body oxygen supply should be taken into consideration. However, to our knowledge, the effect of oxygen supply on $\dot{V}O_2$ kinetics has not been systemically examined. Therefore, the purpose of this study was to determine the relationship between $\dot{V}O_2$ kinetics and the oxygen supply system in supine leg exercise.

MATERIALS AND METHODS

Subjects. Six healthy males with a mean±SD age of 23.2±4.3 yr, mean body mass of 61.5±3.9 kg, and mean height of 1.69±4.5 m participated in this study. 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. The Ethics Committee of Hokkaido University Graduate School of Education approved the present study.

Experimental protocol

Each subject attended our laboratory for incremental-load supine exercise. 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. Exercise was performed using a bicycle ergometer in the supine position, and under ambient conditions (temperature: 24.1±0.4 °C, atmosphere: 757.1±1.6 hPa, humidity: 50%).

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 (W), the work load was increased by 20 W every minute until exhaustion, i.e., until the subject could no longer maintain a rotation rate of 60 rpm. Peak oxygen uptake ($\dot{V}O_2$peak) obtained during exercise was defined as the peak value. VT was determined using the following criteria: (1) an increase in carbon dioxide output ($\dot{V}CO_2$) related to $\dot{V}O_2$, (2) an increase in ventilation ($\dot{V}_E$) related to $\dot{V}O_2$, (3) an increase in $\dot{V}_E$ $\dot{V}O_2^{-1}$ without a decrease in $\dot{V}_E$ $\dot{V}CO_2^{-1}$, and (4) an increase in fractional concentration of oxygen in end-tidal gas ($FETO_2$) without a decrease in fractional concentration of carbon dioxide output in end-tidal gas ($FETCO_2$) [2,27,30]. Each subject was instructed not to gear up for the upper limbs and not to move the upper limbs during exercise. Incremental-load exercise was performed using a bicycle ergometer in the supine position.

Measurements

Respiratory gas exchanges and heart rate

Data on $\dot{V}O_2$ and $\dot{V}CO_2$ were obtained breath-by-breath using a respiratory gas analyser (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 O_2 and CO_2 concentrations were analysed using a zirconium sensor and infrared absorption analyser, respectively. The gas analyser was calibrated by known standard gas (O_2: 15.17%, CO_2: 4.92%). HR was recorded using a heart rate monitor installed in the respiratory gas analyser. These data were measured continuously during rest, warming-up, exercise, and recovery periods.

Oxygenation level in inactive muscle

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

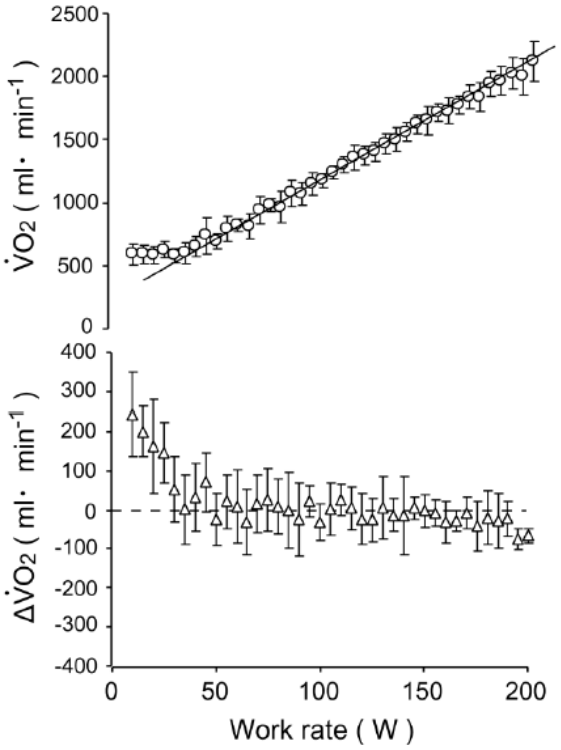

FIG. 1. OXYGEN UPTAKE ($\dot{V}O_2$) KINETICS (UPPER PANEL) AND DIFFERENCE IN OXYGEN UPTAKE ($\Delta\dot{V}O_2$) MEASURED AND THAT ESTIMATED BY THE WORK RATE – $\dot{V}O_2$ RELATIONSHIP OBTAINED BELOW THE VENTILATORY THRESHOLD DURING INCREMENTAL-LOAD EXERCISE IN THE SUPINE POSITION (LOWER PANEL).

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 [20].

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.

The NIRS data were normalized using the following methods. The oxygenation level at warming-up was defined as 100% and the lowest value recorded during arterial occlusion was defined as 0% [11]. NIRS data were obtained continuously from rest periods to recovery periods. A 5-s sampling time was used and recorded. The mean value for each 30-s period was used for analysis.

Statistical analysis

All data are presented as means and standard deviations (SD). Two-way ANOVA for repeated measures on both factors (time × treatments) was used. When main effects were found, the means were compared by using Tukey-Kramer's post hoc test. If a significant interactive effect was indicated, one-way ANOVA for repeated measures was used to examine $\dot{V}O_2$, HR, and oxygenation level. The level of significance was set at $p<0.05$.

RESULTS

The highest values of work rate (WR) and oxygen uptake ($\dot{V}O_2$peak) obtained in incremental-load supine exercise were 232.5±25.0 W and 2.38±0.28 L min^{-1}, respectively. The WR values of VT and respiratory compensation point (RCP) were 112.9±11.7 W and 175.8±21.3 W, respectively. $\dot{V}O_2$ values at VT and RCP were 1.3±0.2 L min^{-1} and 1.9±0.2 L min^{-1}, and the percent values of peak $\dot{V}O_2$ at VT and RCP were 56.1±3.7% and 79.5±2.1%, respectively.

Figure 1 shows the kinetics of $\dot{V}O_2$ during incremental-load supine exercise. $\dot{V}O_2$ kinetics increased linearly during exercise (upper panel). The difference ($\Delta\dot{V}O_2$) between $\dot{V}O_2$ measured and $\dot{V}O_2$ extrapolated from a linear line obtained between work rate and $\dot{V}O_2$ from 50 W to VT is shown in the lower panel in figure 1. Below 50 W and above VT, $\dot{V}O_2$ deviated from a linear line of the relation. $\Delta\dot{V}O_2$ showed a positive value at the onset of exercise and then decreased and showed a constant level of 0 ml min^{-1} after about 50 W. The rate of increment in $\dot{V}O_2$ kinetics was not significantly different between work rates above and below the VT. Excess $\dot{V}O_2$ was not found at a high work rate ($p>0.05$).

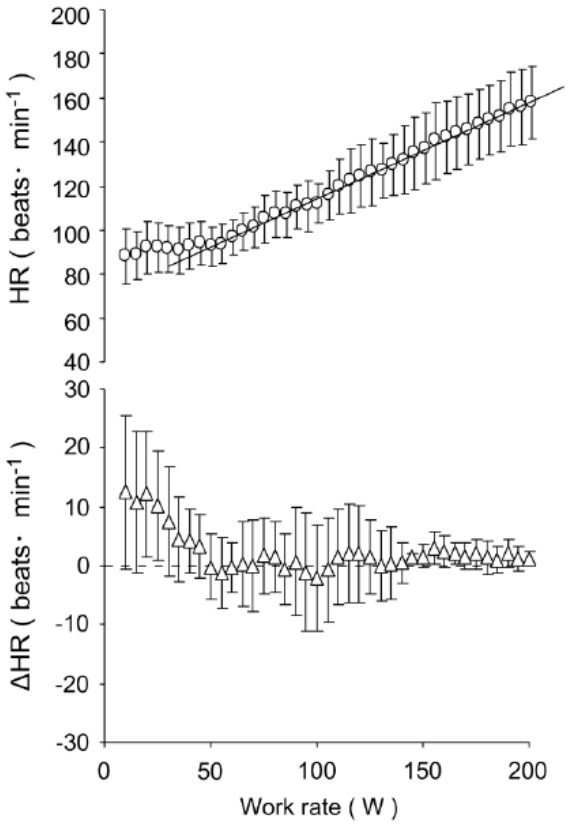

FIG. 2. HEART RATE (HR) KINETICS (UPPER PANEL) AND DIFFERENCE IN HEART RATE (ΔHR) MEASURED AND THAT ESTIMATED BY THE WORK RATE – HR RELATIONSHIP OBTAINED BELOW THE VENTILATORY THRESHOLD DURING INCREMENTAL-LOAD EXERCISE IN THE SUPINE POSITION (LOWER PANEL).

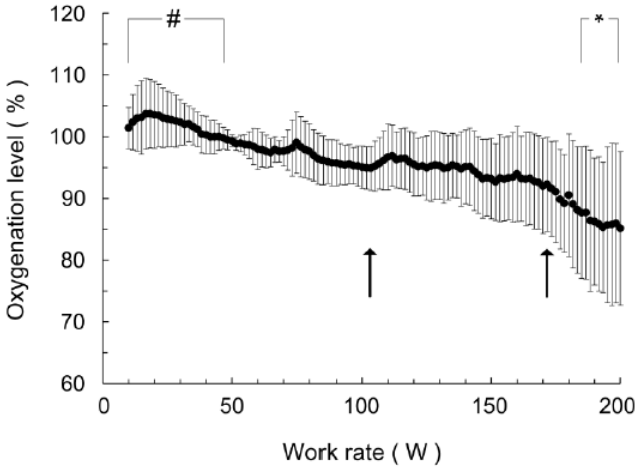

FIG. 3. KINETICS OF OXYGENATION LEVEL IN INACTIVE BICEPS BRACHII MUSCLE DURING INCREMENTAL-LOAD EXERCISE IN THE SUPINE POSITION. ARROWS SHOW VENTILATORY THRESHOLD (VT) AND RESPIRATORY COMPENSATION POINT (RCP).
* shows significant differences between the values at VT and at high work rate ($p<0.05$).
\# shows significant differences between the values at RCP and at low work rate ($p<0.05$).

Figure 2 shows the kinetics of HR during incremental-load supine exercise. HR kinetics increased linearly during exercise (upper panel). The difference (ΔHR) between HR measured and HR extrapolated from a linear line obtained between the work rate and HR from 50 W to VT is shown in the lower panel in figure 2. ΔHR showed a positive

value at the onset of exercise and then decreased and showed a constant level of 0 ml min^{-1} after about 50 W. The rate of increment in HR kinetics was not significantly different between the work rates above and below the VT. Excess HR was not found at high power output ($p>0.05$).

Figure 3 shows the kinetics of oxygenation level during incremental-load supine exercise. Oxygenation level increased slightly at the onset of exercise and was then maintained at the baseline level (warm-up level). Oxygenation level in inactive muscle started to decrease around the VT and then decreased rapidly after the RCP ($p<0.05$).

DISCUSSION

In this study, HR kinetics increased linearly after a time delay during supine exercise. It is thought that the increment of HR kinetics in this study reflects the increment of cardiac output ($\dot{Q}$) as in previous studies [5,29]. Actually, in the supine position or at HR>120 bpm in the sitting position, stroke volume (SV) is higher than SV at rest in the sitting position. It is also known that SV in the supine position slightly increases from resting status to light exercise and then remains unchanged [19]. Thus, HR can reflect $\dot{Q}$ in this situation.

It seems that $\dot{V}O_2$ increases in relation to increment of $\dot{Q}$ as the HR results suggest. However, since exercise on a cycle ergometer is leg exercise rather than whole body exercise, the upper body becomes an inactive part, and since there is a limit to use of the upper body in the supine position, the inactive part in the supine position is more than that in the upright position. In this case, if the influence of blood flow to inactive muscle is not examined, it cannot simply be concluded that oxygen uptake is limited by oxygen supply to active muscle.

Oxygenation level determined by near-infrared spectroscopy (NIRS) in an inactive muscle can reflect oxygen supply [23,32]. The reason for this is as follows. Oxygenation level in an inactive muscle was shown to decrease after a time delay during arm cranking heavy exercise [23]. A similar time delay was found in enhancement of activity of sympathetic nervous tone related to accumulation of metabolites during exercise that leads to vasoconstriction in an inactive muscle [10]. Furthermore, a decrease in oxygenation level in the resting leg during arm cranking exercise was depressed with light leg exercise. This inhibition suggests functional sympatholysis. It has also been reported that oxygenation level in forearm muscle correlates with blood flow during lower body negative pressure leading to enhancement of sympathetic tone [7]. Thus, it is likely that the change in oxygenation level reflects the change in oxygen supply to inactive muscle.

After the onset of exercise, oxygenation level increased slightly and then decreased toward to the baseline level. Moreover, oxygenation level started to decrease slightly from around the VT and then rapidly decreased from around the RCP. It has been reported that oxygenation level in an inactive muscle was affected by skin blood flow [3,4]. The increment of skin blood flow is derived from increased body temperature when exercise is performed. It has also been reported that skin blood flow during incremental-load exercise increased in proportion to the increase in exercise intensity [28]. If skin blood flow affects the oxygenation level, it must be raised since skin blood flow has a high oxygen concentration. However, at low intensity, oxygenation level did not increase over the baseline level but decreased in the present study. At high intensity, oxygenation level started to decrease. Thus, our results support the results of a study by Mancini et al. [22] showing that the effect of skin blood flow is minimal. Thus, in normal environmental conditions and ordinary exercise, skin blood flow might not have a great effect on oxygenation. Although supine cycle leg exercise was used in the present study, it is likely that static exercise is unconsciously carried out in the upper body, and this exercise could affect blood flow and oxygenation level. Indeed, it has been reported that blood flow in the quadriceps muscle decreased during plantar flexion exercise plus static exercise (handgrip until exhaustion) [14,15]. In contrast, Kilbom and Persson, [16] reported that blood flow and arterio-venous oxygen difference in the lower body and upper body at 30%MVC did not change significantly compared to the rest value during static arm exercise. Therefore, we thought that the effect of the upper body on blood flow and oxygenation level was small because the intensity of upper body exercise in the present study was the resting level, which does not exceed 30% MVC. However, the real activity conditions of the upper body in the present study were not measured by surface electromyography, and the effect of static exercise can therefore not be disregarded.

Recently, computer simulations have been performed to examine the $\dot{V}O_2$ kinetics during incremental-load exercise, and it was assumed that excess $\dot{V}O_2$ was responsible for fatigue [18]. However, excess $\dot{V}O_2$ was not found in this study. This result was attributed to application of an exercise protocol in which the rate of increase in exercise intensity was fast. In such an exercise protocol, the exercise might be completed before the effect of fatigue on $\dot{V}O_2$ kinetics appears. In fact, it has been reported that when the rate of increase in exercise intensity is slow, the rate of increment in $\dot{V}O_2$ in relation to power output at high power output is higher than that at low power output [9].

CONCLUSIONS

In summary, in this study, $\dot{V}O_2$ kinetics increased linearly and excess $\dot{V}O_2$ did not appear at high power output. HR kinetics, which reflects oxygen supply to the whole body, increased linearly. Oxygenation level started to decrease from around the VT and then decreased rapidly from the RCP. Therefore, judging from the present indirect indicators of HR and oxygenation level, it is unlikely that $\dot{V}O_2$ kinetics is affected by oxygen supply during exercise in the supine position.

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