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

Accepted for publication 05.07.2010

AUTHORS: Arimitsu T.[1], Matsuura R.[2,3], Yunoki T.[2], Yamanaka R.[1], Kimura T.[1], Lian C.S.[1], Afroundeh R.[1], Yano T.[2]

[1] Laboratory of Exercise Physiology, Health and Sport Education, Graduate School of Education, Hokkaido University
[2] Department of Human Developmental Sciences, Faculty of Education, Hokkaido University
[3] Department of Sports Science, Faculty of Sports Science, Kyushu Kyoritsu University

Reprint request to:
Takuma Arimitsu,
Laboratory of Exercise Physiology,
Health and Sport Education,
Graduate School of Education,
Hokkaido University, Kita-11,
Nishi-7, Kita-ku, Sapporo
060-0811, Japan
Phone: +81-11-706-3422
Fax: +81-11-706-4943
E-mail: arimitsu@edu.hokudai.ac.jp

ABSTRACT: The purpose of this study was to determine the relationship between oxygen uptake ($\dot{V}O_2$) 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 $\dot{V}O_2$. 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 $\dot{V}O_2$ (primary component), $\dot{V}O_2$ during moderate exercise reached a steady state, while during heavy exercise it continued to increase gradually (slow component). The HR kinetics which reflected systemic O_2 supply in the supine position was similar to that of $\dot{V}O_2$ in main exercise tests. However, time constants of primary and slow components in $\dot{V}O_2$ 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 $\dot{V}O_2$ and HR in each component in each exercise mode and since O_2 supply to active muscle is affected by systemic O_2 supply and distribution of O_2 supply to inactive muscle, it is unlikely that $\dot{V}O_2$ is related to O_2 supply to active muscle in supine position.

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

INTRODUCTION

Oxygen uptake ($\dot{V}O_2$) has been analyzed by the application of a mathematical equation for $\dot{V}O_2$ kinetics in constant load exercise [1, 28]. In moderate exercise intensity (below the ventilatory threshold, VT), $\dot{V}O_2$ rises mono-exponentially (primary component) after a time delay until a steady state of $\dot{V}O_2$ is achieved. In heavy exercise (above the VT), $\dot{V}O_2$ is additionally increased after the primary component until the end of exercise [2-4, 13, 14]. This additional increase in $\dot{V}O_2$ 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 $\dot{V}O_2$ and leg $\dot{V}O_2$ during exercise using a cycle ergometer, and they demonstrated that ~86% of the increment in pulmonary $\dot{V}O_2$ beyond the third minute of exercise (i.e., slow component) could be accounted for by the increase in leg $\dot{V}O_2$. However, by this experiment, it is not proved whether $\dot{V}O_2$kinetics is determined by a substance within active muscle or the oxygen supply to active muscle.

The possible factor that limits $\dot{V}O_2$ 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 $\dot{V}O_2$ kinetics, 2) increased arterial O_2 content and muscle O_2 availability do not speed $\dot{V}O_2$ kinetics during moderate or heavy exercise [17], and 3) there is a temporal correspondence between phosphor creatine kinetics and $\dot{V}O_2$ kinetics [24]. Thus, it is still unclear whether $\dot{V}O_2$ 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 $\dot{V}O_2$ kinetics is affected by oxygen supply, $\dot{V}O_2$ 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 $\dot{V}O_2$ kinetics. Therefore, the purpose of this study was to determine the relationship between $\dot{V}O_2$ 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 ($\dot{V}O_2$peak) 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 + ($\dot{V}O_2$peak - 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 ($\dot{V}O_2$) and carbon dioxide output ($\dot{V}CO_2$) 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 O_2 and CO_2 concentrations were analyzed using a zirconium sensor and infrared absorption analyzer, respectively. The gas analyzer was calibrated by known standard gas (O_2: 15.17%, CO_2: 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 $\dot{V}CO_2$ related to $\dot{V}O_2$, (2) an increase in ventilation ($\dot{V}_E$) related to $\dot{V}O_2$, (3) an increase in $\dot{V}_E \cdot \dot{V}O_2^{-1}$ without a decrease in $\dot{V}_E \cdot \dot{V}CO_2^{-1}$, and (4) an increase in the fractional concentration of oxygen in end-tidal gas ($FETO_2$) without a decrease in the fractional concentration of carbon dioxide output in end-tidal gas ($FETCO_2$) [5, 25, 27]. During constant-load exercise, $\dot{V}O_2$ measured breath-by-breath was plotted against time. The $\dot{V}O_2$ data were time-interpolated second-by-second and then $\dot{V}O_2$ kinetics was approximated by the following equations (Moderate: eq. (1), Heavy: eq. (2)):

$$\dot{V}O_2(t) = \dot{V}O_{2,base} + A_p \times (1 - \exp(-(t - TD_p)/\tau_p)), \quad (1)$$

$$\dot{V}O_2(t) = \dot{V}O_{2,base} + A_p \times (1 - \exp(-(t - TD_p)/\tau_p)) + As \times (1 - \exp(-(t - TD_s)/\tau_s)), \quad (2)$$

where $\dot{V}O_2$ (t) is $\dot{V}O_2$ at time t, $\dot{V}O_{2,base}$ is baseline $\dot{V}O_2$, Ap and As are $\dot{V}O_2$ amplitudes for the primary and slow components, respectively, TD_p and TD_s are time delays for the primary and slow components, respectively, and τ_p and τ_s are the time constants for the primary and slow components, respectively. In the same way, during constant-load exercise, HR was measured and plotted against time. The HR parameters were time-interpolated second-by-second and

TABLE I. CHARACTERISTICS OF SUBJECTS AND RESULTS OF INCREMENTAL-LOAD EXERCISE IN THE SUPINE POSITIONS

				Peak Value		VT		Exercise Intensity	
Subject	Age	Height	Weight	$\dot{V}O_2$	WR	WR	%peak	Moderate	Heavy
	(yr)	(cm)	(kg)	(ml min^{-1})	(W)	(W)	(%)	(W)	(W)
A	31	165.5	64.3	2303	225	109	48	83	132
B	21	169.0	63.1	2322	235	106	45	78	131
C	21	177.0	59.5	2166	200	108	54	76	127
D	19	165.0	55.6	2066	220	102	46	74	124
E	25	170.4	66.5	2574	240	118	49	87	144
F	23	177.0	73.3	2750	270	129	48	94	160
G	22	166.0	60.0	2842	275	134	49	100	161
H	23	168.0	64.3	1973	205	109	53	79	129
I	27	176.0	72.9	3278	263	109	41	86	148
Mean	23.6	170.4	64.4	2474.9	237.0	113.9	48.3	84.0	139.5
SD	3.6	5.0	5.9	423.2	27.5	11.0	3.9	8.8	14.3

then HR kinetics was approximated by the following equations (Moderate: eq. (3), Heavy: eq. (4)):

$$HR(t)=HR_{,base} + A_p\times(1-\exp(-(t-TD_p)/\tau_p)), \quad (3)$$

$$HR(t)=HR_{,base} + A_p\times(1-\exp(-(t-TD_p)/\tau_p)) + As\times (1-\exp(-(t-TD_s)/\tau_s)), \quad (4)$$

where HR (t) is HR at time t, $HR_{,base}$ is baseline HR, Ap and As are HR amplitudes for the primary and slow components, respectively, TD_p and TD_s are time delays for the primary and slow components, respectively, and τ_p and τ_s are the time constants for the primary and slow components, respectively. The time constant (τ) was defined as the time required to change from the baseline value to 63.2% of the new steady-state value obtained during exercise periods. $\dot{V}O_2$ data obtained during a period of 20 s after the onset of exercise were excluded from all model fittings.

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% [10]. 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±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 parameters predicted by model fitting were compared by using the paired t-test. The level of significance was set at $p<0.05$.

RESULTS

The subject's results are presented in Table 1. Figure 1 shows the kinetics of $\dot{V}O_2$ during moderate (o) and heavy (●) constant-load exercises in supine position. Both $\dot{V}O_2$ kinetics in moderate exercise and that in heavy exercise increased rapidly. Thereafter, $\dot{V}O_2$ 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 $\dot{V}O_2$ 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 $\dot{V}O_2$ 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 (o) 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 0.25 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 (o) and heavy (●) constant-load exercises in supine position. Both oxygenation level in heavy exercise and that in moderate 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 value ($p<0.05$). Thereafter, oxygenation

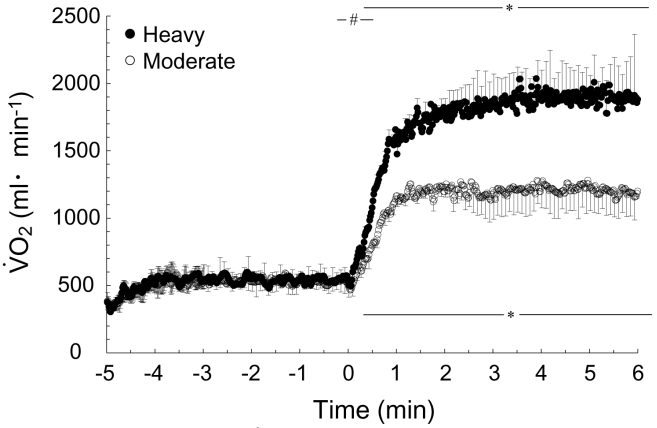

FIG. 1. OXYGEN UPTAKE ($\dot{V}O_2$) KINETICS FOLLOWING STEPWISE INCREASE IN WORK RATE FROM 10 W TO MODERATE (o) AND HEAVY (•) CONSTANT-LOAD EXERCISES IN THE SUPINE POSITION.
* SIGNIFICANT DIFFERENCE (P<0.05) COMPARED TO THE BASELINE VALUE.
# SIGNIFICANT DIFFERENCE (P<0.05) COMPARED TO THE VALUE AT 2 MIN.

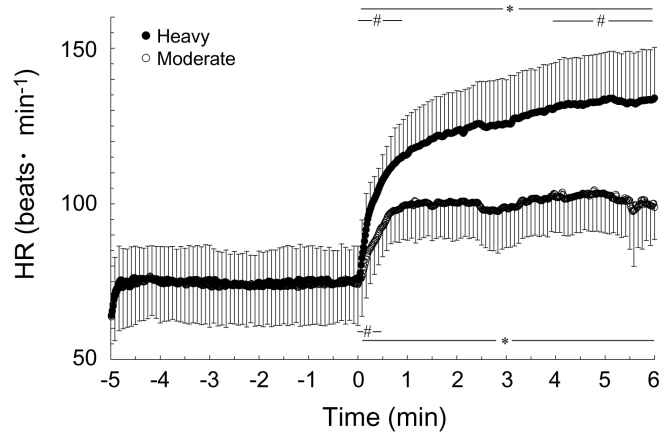

FIG. 2. HEART RATE (HR) KINETICS FOLLOWING STEPWISE INCREASE IN WORK RATE FROM 10 W TO MODERATE (o) AND HEAVY (•) CONSTANT-LOAD EXERCISES IN THE SUPINE POSITION.
* SIGNIFICANT DIFFERENCE (P<0.05) COMPARED TO THE BASELINE VALUE.
# SIGNIFICANT DIFFERENCE (P<0.05) COMPARED TO THE VALUE AT 2 MIN.

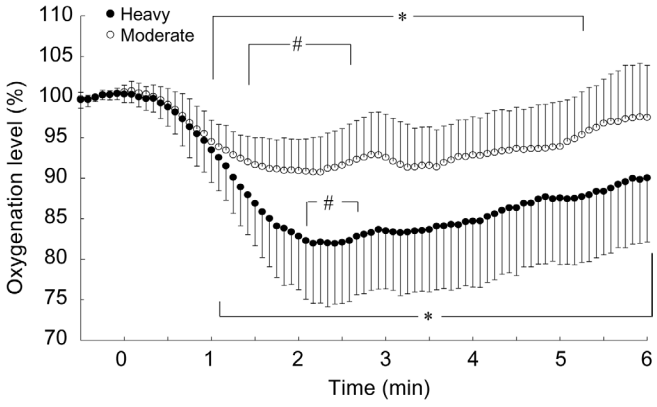

FIG. 3. KINETICS OF OXYGENATION LEVEL IN INACTIVE BICEPS BRACHII MUSCLE DURING MODERATE (o) AND HEAVY (•) CONSTANT-LOAD EXERCISES IN THE SUPINE POSITION.
* SIGNIFICANT DIFFERENCE (P<0.05) COMPARED TO THE BASELINE VALUE.
# SIGNIFICANT DIFFERENCE (P<0.05) COMPARED TO THE VALUE AT 2 MIN.

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

Table 2 shows the parameters of $\dot{V}O_2$ and HR predicted by model fitting in moderate and heavy constant-load exercises in supine position. $\dot{V}O_{2,base}$, $HR_{,base}$, time delays (TD_p) of the primary component, and time constants (τ_p) of the primary component were not significantly different between moderate and heavy exercise, but amplitudes (A_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 $\dot{V}O_2$ kinetics and HR kinetics. There was no significant difference in time constants of the slow component between $\dot{V}O_2$ kinetics and HR kinetics. There was also no significant difference in TD_s of $\dot{V}O_2$ kinetics and HR kinetics. The data of TD_s showed that the kinetics of $\dot{V}O_2$ and that of HR increased at similar times ($p<0.05$). However, there was no significant correlation coefficient in τ_p in moderate ($r=0.609$) and heavy exercises ($r=0.300$) and there was also no correlation coefficient in τ_s ($r=0.137$) between $\dot{V}O_2$ kinetics and HR kinetics.

DISCUSSION

The major results of this study performed in supine position were as follows. The time constants of the primary component (τ_p) in $\dot{V}O_2$ kinetics were not significantly different from those of the HR kinetics in moderate and heavy exercise. Time delays (TD_s) and time constants of the slow component (τ_s) in $\dot{V}O_2$ kinetics were not significantly different from those in HR kinetics. However, there were no significant correlation coefficients in τ_p and τ_s between $\dot{V}O_2$ 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_p in HR kinetics during moderate exercise was not significantly different from that in $\dot{V}O_2$ kinetics and the correlation coefficient between τ_p in $\dot{V}O_2$ and that in HR was not significant, suggesting that systemic oxygen supply is not the determining factor of $\dot{V}O_2$ kinetics at the onset of exercise in supine position. Furthermore, we indirectly estimated oxygen supply to inactive

TABLE 2. $\dot{V}O_2$ AND HR KINETICS PARAMETERS FOR HEAVY (Δ20+VT) AND MODERATE (80%VT) CONSTANT-LOAD SUPINE EXERCISE

Parameters	$\dot{V}O_2$		HR	
	Heavy	Moderate	Heavy	Moderate
$\dot{V}O_2$base and HR base (ml min^{-1}, beats min^{-1})	521.0 ± 34.6	509.7 ± 35.8	75.6 ± 10.3	74.2 ± 12.6
Primary component				
A_p (ml min^{-1}, beats min^{-1})	1263.9 ± 165.7	702.1 ± 126.8 *	48.0 ± 10.2	27.4 ± 5.4 *
TD_p (s)	14.0 ± 3.6	17.4 ± 3.5	-1.1 ± 5.4 #	3.1 ± 7.3
τ_p (s)	25.4 ± 4.6	23.5 ± 6.7	28.5 ± 8.5	24.5 ± 12.5
Slow component				
A_p (ml min^{-1}, beats min^{-1})	148.5 ± 66.3		14.1 ± 7.2	
TD_s (s)	113.9 ± 11.6		116.8 ± 41.3	
τ_s (s)	142.5 ± 84.0		152.6 ± 99.0	

Note: Values are means±SD; n=9 subjects. A_p and A_s, amplitudes of response; τ_p and τ_s, time constants; TD_p and TD_s, time delay.
* Significantly different from Heavy ($p<0.05$).
# Significantly different from $\dot{V}O_2$ ($p<0.05$).

muscle by using the method developed by Ogata et al. [19]. 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, τ_p in HR kinetics was not significantly different from that in $\dot{V}O_2$ kinetics and the correlation coefficient between τ_p in $\dot{V}O_2$ 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, TD_s and τ_s in $\dot{V}O_2$ kinetics were not significantly different from those in HR kinetics and the correlation coefficient between τ_s in $\dot{V}O_2$ and that in HR was also not significant. Therefore, it is unlikely that $\dot{V}O_2$ 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 in active muscle estimated by oxygenation level in the inactive biceps brachii muscle. Consequently, there were no significant correlation coefficients in the time constant between $\dot{V}O_2$ and HR in primary component in moderate and heavy exercises as well as slow component in heavy exercise. Furthermore, O_2 supply to active muscle is not affected by systemic O_2 supply and its distribution of O_2 supply to inactive muscle. Therefore, it is unlikely that $\dot{V}O_2$ is related to O_2 supply to active muscle in supine position. And $\dot{V}O_2$ may be determined in peripheral portion.

REFERENCES

1. Barstow T.J., Molé P.A . Linear and nonlinear characteristics of oxygen uptake kinetics during heavy exercise. J. Appl. Physiol. 1991;71: 2099-2106.
2. Barstow T.J., Casaburi R., Wasserman K. O_2 uptake kinetics and the O_2 deficit as related to exercise intensity and blood lactate. J. Appl. Physiol. 1993;75: 755-762.
3. Barstow T.J., Jones A.M., Nguyen P.H., Casaburi R. Influence of muscle fiber type and pedal frequency on oxygen uptake kinetics of heavy exercise. J. Appl. Physiol. 1996;81: 1642-1650.
4. Bearden S.E., Moffatt R.J. VO_2 and heart rate kinetics in cycling: transitions from an elevated baseline. J. Appl. Physiol. 2001;90: 2081-2087.

5. Beaver W.L., Wasserman K, Whipp B.J. A new method for detecting anaerobic threshold by gas exchange. J. Appl. Physiol. 1986;60: 2020-2027.
6. Buono M.J., Miller P.W., Hom C., Pozos R.S., Kolkhorst F.W. Skin blood flow affects in vivo near-infrared spectroscopy measurements in human skeletal muscle. Jpn. J. Physiol. 2005;55: 241-244.
7. Chance B, Dait M.T., Zhang C, Hamaoka T., Hagerman F. Recovery from exercise-induced desaturation in the quadriceps muscles of elite competitive rowers. Am. J. Physiol. 1992;262: C766-C775.
8. Davis S.L., Fadel P.J., Cui J, Thomas G.D., Crandall C.G. Skin blood flow influences near-infrared spectroscopy-derived measurements of tissue oxygenation during heat stress. J. Appl. Physiol. 2006;100: 221-224.
9. Engelen M., Porszasz J., Riley M., Wasserman K., Maehara K., Barstow T.J. Effects of hypoxic hypoxia on O_2 uptake and heart rate kinetics during heavy exercise. J. Appl. Physiol. 1996;81: 2500-2508.
10. Higuchi H., Hamaoka T., Sako T., Nishio S., Kime R., Murakami M., Katsumura T. Oxygenation in vastus lateralis and lateral head of gastrocnemius during treadmill walking and running in humans. Eur. J. Appl. Physiol. 2002;87: 343-349.
11. Hughson R.L., Morrissey M.A. Delayed kinetics of respiratory gas exchange in the transition from prior exercise. J. Appl. Physiol. 1982;52: 921-929.
12. Hughson R.L., Cochrane J.E., Butler G.C. Faster O_2 uptake kinetics at onset of supine exercise with than without lower negative pressure. J. Appl. Physiol. 1993;75: 1962-1967.
13. Koga S., Shiojiri T., Shibasaki M., Kondo N., Fukuba Y., Barstow T.J. Kinetics of oxygen uptake during supine and upright heavy exercise. J. Appl. Physiol. 1999;87: 253-260.
14. Koga S., Barstow T.J., Shiojiri T., Takaishi T., Fukuba Y., Kondo N., Shibasaki M., Poole D.C. Effects of muscle mass on VO_2 kinetics at the onset of work. J. Appl. Physiol. 2001;90: 461-468.
15. Leyk D., Eßfeld D., Hoffmann U., Wunderlich H.G., Baum K., Stegemann J. Postural effect on cardiac output, oxygen uptake and lactate during cycle exercise of varying intensity. Eur. J. Appl. Physiol. 1994;68: 30-35.
16. MacCully K.K., Hamaoka T. Near-infrared spectroscopy: what can it tell us about oxygen saturation in skeletal muscle? Exerc. Sports. Sci. Rev. 2000;28: 123-127.
17. MacDonald M.J., Pedersen P.K., Hughson R.L. Acceleration of VO_2 kinetics in heavy submaximal exercise by hyperoxia and prior high-intensity exercise. J. Appl. Physiol. 1997;83: 1318-1325.
18. Mancini D.M., Bolinger L., Li H., Kendrich K., Chance B., Wilson J.R. Validation of near-infrared spectroscopy in humans. J. Appl. Physiol. 1994;77: 2740-2747.
19. Ogata H., Yunoki T., Yano T. Effects of arm cranking on the NIRS-determined blood volume and oxygenation of human in active and exercising vastus lateralis muscle. Eur. J. Appl. Physiol. 2002;86: 191-195.
20. Özyener F., Rossiter H.B., Ward S.A., Whipp B.J. Influence of exercise intensity on the on and off-transient kinetics of pulmonary oxygen uptake in humans. J. Physiol. (Lond). 2001;533: 891-902.
21. Poliner L.R., Dehmer G.J., Lewis S.E., Parkey R.W., Blomqvist C.G., Willerson J.T. Left ventricular performance in normal subjects: a comparison of the responses to exercise in the upright and supine positions. Circulation. 1980;62: 528-534.
22. Poole D.C., Schaffartzik W., Knight D.R., Derion T., Kennedy B., Guy H.J., Prediletto R., Wagner P.D. Contribution of excising legs to the slow component of oxygen uptake kinetics in humans. J. Appl. Physiol. 1991;71: 1245–1260.
23. Poole D.C., Barstow T.J., McDonough P., Jones A.M. Control of oxygen uptake during exercise. Med. Sci. Sports. Exerc. 2008;40(3): 462-474.
24. Rossiter H.B., Ward A., Doyle V.L., Howe F.A., Griffiths J.R., Whipp B.J. Inferences from pulmonary O_2 uptake with respect to intramuscular [phosphocreatine] kinetics during moderate exercise in humans. J. Physiol. 1999;518: 921-932.
25. Scheuermann B.W., Kowalchuk J.M. Attenuated respiratory compensation during rapidly incremented ramp exercise. Respir. Physiol. 1998;114: 227-238.
26. Scheuermann B.W., Hoelting B.D., Noble M.L., Barstow T.J. The slow component of O_2 uptake is not accompanied by changes in muscle EMG during repeated bouts of heavy exercise in humans. J. Physiol. 2001;531: 245-256.
27. Wassermann K., Wahipp B.J., Koyal S.N., Beaver W.L. Anaerobic threshold and respiratory gas exchange during exercise. J. Appl. Physiol. 1973;35: 236-243.
28. Whipp B.J., Ward S.A., Lamarra N., Davis J.A., Wasserman K. Parameters of ventilatory and gas exchange dynamics during exercise. J. Appl. Physiol. 1982;52: 1506-1513.
29. Yano T., Horiuchi M., Yunoki T., Matsuura R., Ogata H. Relationship between maximal oxygen uptake and oxygenation level in inactive muscle at exhaustion in incremental exercise in humans. Physiol. Res. 2005;54: 679-685.