Metaboreceptor-mediated Muscle Oxygen Saturation during Recovery
Following Isometric Handgrip Exercise

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Abstract  The aim of the present study was to determine whether oxygen supply to non-exercised muscle during recovery following fatiguing exercise is influenced by accumulated metabolites within exercised muscle. Twelve healthy male subjects performed 2-min isometric handgrip exercise at 40% maximal voluntary contraction with their right hand and the exercise was followed by a 3-min recovery period. Muscle oxygen saturation (SmO₂) determined by near-infrared spatially resolved spectroscopy was used as an index of oxygen supply to non-exercised muscle and was measured in biceps brachii and tibialis anterior muscles on the left side. Compared to the pre-exercise baseline level, SmO₂ in the biceps brachii muscle (SmO₂BB) increased significantly from 30 sec to 1 min after the start of exercise, while SmO₂ in the tibialis anterior muscle (SmO₂TA) remained stable during the initial 1 min of exercise. Both SmO₂BB and SmO₂TA began to decrease at about 1 min and continued to decrease thereafter. Due to the initial increase in SmO₂BB only SmO₂TA showed a significant decrease during exercise. During recovery, SmO₂BB did not differ significantly from the pre-exercise baseline level, whereas SmO₂TA remained significantly lower until about 1.5 min of recovery and then it did not differ significantly from the baseline level. In another bout, subjects performed handgrip exercise of the same intensity, but post-exercise arterial occlusion (PEAO) of the exercised muscle was imposed for 2 min immediately after the end of exercise. During PEAO, SmO₂BB decreased significantly compared to the baseline level, whereas SmO₂TA remained significantly lower until the end of PEAO. The significant decrease in SmO₂BB and the prolongation of decrease in SmO₂TA by PEAO suggests that the recovery of SmO₂ in the non-exercised arm and leg is mediated by muscle metaboreceptors. J Physiol Anthropol 27(2): 83–91, 2008 http://www.jstage.jst.go.jp/browse/jpa2 [DOI: 10.2214/jpa2.27.83]

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Introduction

Since oxygen delivery to exercised muscle during recovery can have a profound effect on the recuperation of muscle metabolism to the pre-exercise level (Haseler et al., 1999), considerable interest has been shown in the mechanisms responsible for changes in oxygen supply (i.e., blood flow) during recovery (Ferreira et al., 2005; Kilborn and Wennmalm, 1976; Kille and Klabunde, 1984; Radegran and Saltin, 1999). However, to our knowledge, all previous studies have focused on the factors that increase oxygen supply (e.g., local metabolic vasodilation and muscle pump). Since oxygen supply is determined by the balance of the effect of factors that increase oxygen supply and the effect of factors that decrease oxygen supply (e.g., sympathetic vasoconstriction) (Buckwalter and Clifford, 2001; Delp and Laughlin, 1998; Delp and O’Leary, 2004; Rowell, 2004; Thomas and Segal, 2004), the latter factors will also have an effect on changes during recovery. This effect can be elucidated by investigation of responses in “non-exercised muscles”, because the contribution of local metabolic vasodilation and muscle pump is assumed to be absent in these muscles.

Previous findings have suggested that oxygen supply to non-exercised muscle decreases during high-intensity dynamic exercise and during static exercise at 30% of maximal voluntary contraction and then returns to the pre-exercise level after exercise (Bevegard and Shepherd, 1966; Ogata et al., 2002; Saito et al., 1990) and that the decrease in oxygen supply to non-exercised muscle during exercise is closely related to activation of muscle sympathetic nerve activity (MSNA) (Bevegard and Shepherd, 1966; Saito et al., 1990). MSNA during exercise is triggered by a buildup of metabolites within exercised muscle (metaboreflex) (Costa et al., 2001; Cui et al., 2007; Fadel et al., 2003; MacLean et al., 2000; Mark et al., 1985; Pryor et al., 1990; Victor et al., 1988). Several metabolites have been identified as possible candidates that cause metaboreflex during exercise in humans, e.g., protons (Fadel et al., 2003; Pryor et al., 1990; Victor et al., 1988), potassium (MacLean et al., 2000), phosphate (MacLean et al.,
2000), adenosine (Costa et al., 2001) and prostaglandin (Cui et al., 2007). During recovery following exercise, metabolites that have built up during exercise are expected to remain within exercised muscle for a while. Therefore, we hypothesized that the recovery of oxygen supply to non-exercised muscle is influenced also by remaining metabolites remaining within exercised muscle. The validity of this hypothesis would be confirmed by demonstrating that application of post-exercise arterial occlusion of the exercised muscle (PEAO), which traps metabolites in the occluded muscle, delays the recovery of oxygen supply to non-exercised muscle.

When changes in oxygen supply to non-exercised muscle are examined, electromyographic activity in the non-exercised muscle must be measured to check the presence of involuntary muscle contraction because muscle contraction causes metabolic vasodilation, and involuntary muscle contraction is thought to affect oxygen supply (Cotzias and Marshall, 1993; Jacobsen et al., 1994). Metabolic vasodilation has been suggested to be localized in muscles at which electromyographic activity is recorded within a limb (Ogata and Yano, 2005). Therefore, if electromyographic activities in certain muscles are compared with total limb blood flow measured using a plethysmography method or a Xenon-washout method, the effect of metabolic vasodilation cannot be assessed accurately. To avoid this problem, changes in oxygen supply in local muscles in which electromyographic activities are recorded should be determined. Near-infrared spatially resolved spectroscopy (SRS) provides measurements of local changes in muscle oxygen saturation. Oxygen saturation is determined by a dynamic balance between oxygen supply and oxygen utilization. If there is little or no electromyographic activity, the contribution of change in oxygen utilization to change in oxygen saturation as well as the role of metabolic vasodilation is assumed to be minimal or absent. Thus, change in oxygen saturation reflects change in oxygen supply.

The aim of the present study was to determine whether PEAO delays the recovery of SRS-determined oxygen saturation in non-exercised muscle following isometric handgrip exercise. We chose the biceps brachii and tibialis anterior muscles for measurement sites because previous studies have demonstrated different patterns of change in MSNA (Anderson et al., 1987; Wallin et al., 1989) and α-adrenergic vascular responsiveness (Pawelezyk and Levine, 2002) in the arm and leg and therefore because the pattern of change in oxygen saturation during recovery is expected to be different in the arm and leg.

Methods

Subjects

Twelve healthy male subjects with a mean age of 29±4 (standard deviation: SD) years, a mean weight of 66±13 (SD) kg, and a mean height of 1.72±0.07 (SD) m participated in this study. Voluntary consent for participation in this study was obtained from all subjects after they were informed of the purpose of the experiment, the procedure and possible risks. The study was conducted in accordance with the Helsinki Declaration and was approved by the Ethics Committee of the National Rehabilitation Center for Persons with Disabilities in Tokorozawa, Japan.

Experimental protocols

At least 2 hours before the test, the subjects refrained from eating and from taking caffeine. During measurements, the temperature in the experimental room was set at a temperature comfortable for the subject within the range of 20–26°C.

The subjects performed isometric handgrip exercise on the right side in a supine position using a hand dynamometer (G-B, Takei, Tokyo, Japan). The two measurement sites (i.e., biceps brachii and tibialis anterior muscles) were positioned at the same height as the heart throughout the protocol. First, the subjects performed 3-sec maximal voluntary contractions (MVCs) twice with a 1-min interval between contractions, and the higher force was considered as MVC. After a rest period of more than 20 min, data recording was started. First, subjects rested for 3 min and then went on to perform 2-min submaximal voluntary contraction at 40% MVC. After the exercise, the subjects rested for 3 min. Subjects performed handgrip exercise of the same intensity again. In this bout, post-exercise arterial occlusion (PEAO) of the exercised muscle at 250 mmHg was imposed for 2 min immediately after the end of exercise. After the release of PEAO, the subject rested for 3 min. There was a recovery period of at least 20 minutes between tests to recover from muscle fatigue. The two tests were performed in random order. During exercise, the target force was displayed on an oscilloscope to provide visual feedback.

After completion of the two bouts, the subjects exerted their maximal voluntary contraction of the muscles in question (i.e., the biceps brachii and tibialis anterior muscles) for 3 sec to measure maximal electromyographic activity.

Physiological measurements

Muscle oxygenation was measured using a near-infrared spatially resolved spectroscopy device (NIRO 300, Hamamatsu Photonics, Hamamatsu, Japan). The device consisted of two optical probes and a computerized control system. Each of the optical probes consisted of one emitter and one detector (comprising three separate sensors). Four different wavelength laser diodes (775, 810, 850, and 910 nm) in the emitter provided the light source. The emitted light penetrated the tissue, where it was either absorbed or scattered, and some of the scattered light was detected by the detector. The magnitude of light attenuation by the tissue absorption was used, according to the Beer-Lambert Law, for estimation of concentration changes from baseline values of oxygenated, deoxygenated, and total hemoglobin/myoglobin (\(\Delta M \cdot 1^{-1}\)). Muscle oxygen saturation (SmO\(_2\), %) was determined as the ratio of oxygenated hemoglobin/myoglobin concentration to total hemoglobin/myoglobin concentration and therefore is...
measured as an absolute value (Cardinale et al., 2007). SmO₂ measured by SRS has been used elsewhere for determination of intramuscular oxygenation status (Cardinale et al., 2007; Kawaguchi et al., 2001; Komiyama et al., 2001).

Muscle oxygenation (oxygenated hemoglobin/myoglobin concentration and SmO₂) is influenced by oxygen supply and oxygen utilization. Thus, in non-exercised muscle, in which the rate of oxygen utilization is almost constant, change in oxygenation reflects change in oxygen supply. However, oxygenated hemoglobin/myoglobin concentration is known to be influenced also by change in blood volume (total hemoglobin/myoglobin) (McCully and Hamaoka, 2000). Thus, we used SmO₂ as an indicator of oxygen supply, which is free from the influence of blood volume, rather than the oxygenated hemoglobin/myoglobin concentration in use elsewhere (Fadel et al., 2004; Hansen et al., 1996; Mizuno et al., 2006; Ogata et al., 2002).

The optodes were housed in an optically dense plastic holder, thus ensuring that the relative positions of the optodes would be fixed and invariant. The interoptode spacing was 5 cm. The mean depth of penetration into the tissue was approximately equal to half the interoptode spacing (Chance et al., 1992).

Continuous surface electromyograms (EMGs, μV) were recorded using bipolar electrodes (DE-2.1, Delsys, Boston, USA). The electrode contacts were made from two silver bars each 10 mm in length and 1 mm in diameter and spaced 10 mm apart. The detected signals were amplified before being sent along a shielded cable to the rest of the EMG system. The EMG signals were then amplified using a main amplifier (Bagnoli-8, Delsys, Boston, USA) with a gain of 1000 fold. Prior to EMG electrode application, the measurement site was prepared by the removal of dead skin by gentle abrasion with abrasive paper.

Systolic and diastolic blood pressures (mmHg) were determined using an electro-sphygmomanometer (APM-2050, Nihon Kohden, Tokyo, Japan). This device consisted of a pressure sensor and its control unit. It provided one data point per 15 heartbeats. The sensor was placed on the skin just above the radial artery at the wrist. The mean blood pressure (MBP) was calculated as diastolic pressure plus one third of pulse pressure.

Heart rate (HR, bpm) was recorded using a wireless heart rate monitor (Lifescope 8/Two; Nihon Kohden, Tokyo, Japan).

SmO₂, EMG, blood pressure, and HR were recorded continuously at rest and during and after exercise. SRS optodes were placed on the surface of the skin over the belly of the biceps brachii muscle (BB) as a representative muscle of the upper limb and over the belly of the tibialis anterior muscle (TA) as a representative muscle of the lower limb. Both optodes were attached to left limbs. The EMG electrodes were attached with double-sided adhesive tape to the skin over the BB and TA adjacent to the SRS probes in a direction parallel to the muscle fiber orientation. The reference electrode for each of the EMGs was placed over the medial epicondyle of the humerus or patella. The blood pressure sensor was attached to left side. The wrist from which blood pressure recordings were made was always held at the level of the heart. Measurement of blood pressure was conducted in eleven of the twelve subjects, because the shape of the sensor fastener did not fit the wrist in one subject.

Analog signals of contraction force, SmO₂, and EMG were sampled at 1000 Hz by a dedicated computerized data acquisition system (Powerlab; AD Instruments, Castle Hill, Australia) and stored on hard disk for later analysis. Data on blood pressure and HR were recorded online and stored on a personal computer.

Data analysis

Integrated EMGs (iEMGs) during and after exercise were calculated using data obtained every 2 sec. iEMG during maximal voluntary contraction was also calculated using data over the middle 2-sec period in 3-sec exercise. The iEMGs during and after exercise were expressed as a percentage of that during maximal voluntary contraction.

Averages of data on SmO₂, iEMG, HR, and MBP recorded during and after exercise were calculated for each 30-s interval. We carried out a two-way ANOVA between measurement sites for SmO₂, and we examined the main effects of exercise time and positions as well as interaction between changes in the arm and leg. For all data, one-way analysis of variance for repeated measures was used. When the F-ratio revealed a p<0.05 level of significance, post hoc analysis was conducted using the Tukey-Kramer test to detect values during and after exercise that were different from baseline values. We used the following three values as baseline values: averages over 3-min intervals during the pre-exercise period, the value during the last 30 sec of exercise, and the value during the last 30 sec of PEAO. The paired Student's t-test was used for comparison of pre-exercise baseline values between the 1st and 2nd bouts and between bouts with and without arterial occlusion (PEAO_w and PEAO_w/o, respectively). A value of p<0.05 was regarded as statistically significant. All data are presented as means±S.E.M.

Results

Pre-exercise baseline values

Table 1 shows average pre-exercise baseline values of SmO₂, MBP, and HR. There were no significant differences in any of the baseline values, except that for MBP between the 1st and 2nd bouts. In addition, there were no significant differences in the baseline values between PEAO_w and PEAO_w/o bouts. The baseline value of SmO₂ for the 1st bout for TA was significantly lower than that for BB.

Electromyogram

Figure 1 shows average changes in iEMG. There were no significant increases in the iEMG of arm and leg muscles in both PEAO_w and PEAO_w/o bouts except for the iEMG of the
leg muscle during the last 30 sec of exercise in the PEOA_w,bout. However, the magnitude of increases in the iEMG was only 1.6% of the maximal iEMG value.

**Muscle oxygen saturation**

Figure 2 shows average changes in SmO,
2 from the pre-exercise baseline value in PEOA_w,b and PEOA_w,o bouts. The results of two-way ANOVA indicate a statistically significant main effect of time and interaction between changes in SmO,
2 of BB (SmO2_BB) and SmO,
2 of TA (SmO2_TA) in both bouts. Change in each of SmO2_BB and SmO2_TA during exercise was similar in PEOA_w,b and PEOA_w,o bouts. SmO2_BB increased significantly from 30 sec to 1 min after the start of exercise. Then SmO2_BB decreased to the baseline level. SmO2_TA on the other hand, remained unchanged during the first min of exercise and then began to decrease. SmO2_TA during the last 30 sec of exercise was significantly lower than the pre-exercise baseline value.

With regard to changes in SmO,
2 during recovery in the PEOA_w,o bout, SmO2_BB continued to decrease for about 1.5 min and then began to increase. The value of SmO2_BB from 1 min to 1.5 min in the recovery period was significantly lower than that during the last 30 sec of exercise but was not significantly different from the pre-exercise baseline value. On the other hand, SmO2_TA began to increase at about 30 sec of recovery but remained significantly lower than the pre-exercise baseline value until about 1.5 min of recovery.

In the PEOA_w,o bout, neither SmO2_BB nor SmO2_TA increased during PEOA. SmO2_BB during PEOA continued to decrease

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**Table 1** Baseline values of muscle oxygen saturation in biceps brachii and tibialis anterior (SmO2_BB and SmO2_TA, respectively), heart rate (HR) and mean blood pressure (MBP) before the onset of isometric handgrip exercises

<table>
<thead>
<tr>
<th></th>
<th>SmO2_BB</th>
<th>SmO2_TA</th>
<th>HR</th>
<th>MBP</th>
</tr>
</thead>
<tbody>
<tr>
<td>1st</td>
<td>70±1</td>
<td>60±1</td>
<td>62±2</td>
<td>86±2</td>
</tr>
<tr>
<td>2nd</td>
<td>69±1</td>
<td>58±1</td>
<td>61±2</td>
<td>90±2*</td>
</tr>
<tr>
<td>PEOA_w,o</td>
<td>69±1</td>
<td>59±1</td>
<td>61±2</td>
<td>89±2</td>
</tr>
<tr>
<td>PEOA_w,o</td>
<td>69±1</td>
<td>59±1</td>
<td>62±2</td>
<td>87±2</td>
</tr>
</tbody>
</table>

The subjects performed two types of isometric handgrip exercise. In one bout, post-exercise arterial occlusion (PEAO) of the exercised muscle at 250 mmHg was imposed for 2 min (PEAO_w), and in the other bout, PEAO was not imposed after exercise (PEAO_w,o). There was a recovery period of at least 20 minutes between exercise bouts. The order of the two types of tests was randomized across subjects.

The baseline values were defined as the averages over 3-min intervals before the onset of exercise.

*: significant difference from the value in the first exercise or the PEOA_w,o bout.

![Fig. 1](image-url)  
**Fig. 1** Average changes (n=12) in integrated electromyogram activities of left biceps brachii and left tibialis anterior muscles (left and right panels, respectively) during and after 2 min of right-hand isometric handgrip exercise at 40% maximal voluntary contraction (MVC). The subjects performed two exercise bouts, one with 2-min arterial occlusion of the exercised muscle immediately after the end of exercise (lower panels) and the other without arterial occlusion (upper panels). The integrated electromyogram activity was normalized to that recorded during maximum isometric contractions for each muscle.

**: significant difference from the value during the pre-exercise period and the value during the last 30 sec of exercise, respectively.
Fig. 2 Average changes (n=12) in muscle oxygen saturation in left biceps brachii (closed circles) and left tibialis anterior (open circles) from pre-exercise baseline value during and after 2 min of right-hand isometric handgrip exercise performed at 40% maximal voluntary contraction (MVC).

The subjects performed two exercise bouts, one with 2-min arterial occlusion of the exercised muscle immediately after the end of exercise (lower panels) and the other without arterial occlusion (upper panels).

*, #: significant difference in muscle oxygen saturation in biceps brachii and tibialis anterior from the pre-exercise baseline value, respectively.

**, ##: significant difference in muscle oxygen saturation in biceps brachii and tibialis anterior from the value during the last 30 sec of exercise, respectively.

***, ###: significant difference in muscle oxygen saturation in biceps brachii and tibialis anterior from the value during the last 30 sec of arterial occlusion, respectively.

with time until the end of PEAO. The values of SmO2BB after 1 min of PEAO were significantly lower than the pre-exercise baseline value, and the values of SmO2BB after the 30 sec of PEAO were significantly lower than the value during the last 30 sec of exercise. On the other hand, SmO2TA throughout PEAO remained significantly lower than the pre-exercise baseline value and was not significantly different from that during the last 30 sec of exercise.

During recovery after the end of PEAO, both SmO2BB and SmO2TA began to increase toward the pre-exercise level. Compared to the value during the last 30 sec of PEAO, significantly larger values of SmO2BB were recorded after 2 min of recovery, while significantly larger values of SmO2TA were recorded after 30 sec of recovery.

Mean blood pressure

Figure 3 shows average changes in MBP from the pre-exercise baseline value. During exercise, MBP increased with time in both PEAOw and PEAOw/o bouts. Significantly larger values than the pre-exercise baseline value were recorded after 30 sec of exercise in both bouts. After the end of exercise in the PEAOw/o bout, MBP reached the baseline level within the initial 30 sec. In the PEAOw bout, on the other hand, after a transient decrease in MBP during the initial 30 sec of PEAO, MBP began to increase and showed significantly larger values than the pre-exercise baseline value during the rest of the PEAO period. After the cessation of PEAO, MBP decreased to the pre-exercise baseline level within the initial 30 sec.

Heart rate

Figure 4 shows average changes in HR from the pre-exercise baseline value. HR increased with time during exercise in both PEAOw and PEAOw/o bouts. Significantly larger values than the pre-exercise baseline value were recorded from the initial 30 sec of exercise in both bouts. After the end of exercise, HR reached the pre-exercise baseline level within the initial 30 sec in both PEAOw and PEAOw/o bouts.

Discussion

The main findings of the present study were as follows: 1) although during recovery without PEAO, SmO2BB did not differ significantly from the pre-exercise baseline value, and SmO2TA, which had decreased during exercise, remained significantly lower only until about 1.5 min of recovery,
SmO$_2$ decreased significantly during 2-min PEAO and SmO$_2$TA remained significantly lower until the end of PEAO; and 2) the patterns of change in SmO$_2$BA and SmO$_2$TA were different not only during exercise but also during the post-exercise period. We discuss below the possible physiological mechanisms responsible for changes in SmO$_2$.

**Baseline**

In the major part of this section, we discuss the patterns of change in SmO$_2$ rather than absolute values of SmO$_2$ because we were not sure that absolute values of SmO$_2$ have any physiological meaning. However, we found that the pre-exercise baseline value of SmO$_2$TA was significantly lower than that of SmO$_2$BA. Saltin et al. (1977) showed that the percentage of slow twitch fibers in humans tends to be higher in the tibialis anterior muscle (60–80%) than in the biceps brachii muscle (40–77%). Slow twitch fibers generate their energy largely through oxidative phosphorylation of pyruvate and fatty acids. Thus, it is possible that the greater oxygen utilization in muscles with relatively large percentages of slow twitch fibers leads to lower values of SmO$_2$.

**SmO$_2$ during exercise**

SmO$_2$ is determined by the dynamic balance between oxygen supply and oxygen utilization. In the present study, changes in SmO$_2$ were accompanied by little or no electromyographic activity, indicating that the rate of oxygen utilization was almost constant and that the effect of metabolic vasoconstriction was minimal. Therefore, changes in SmO$_2$ will reflect oxygen supply that is unrelated to metabolic vasoconstriction.

The patterns of change in SmO$_2$ during exercise observed in the present study were very similar to those in blood flow during isometric handgrip exercise observed in previous studies (Jacobson et al., 1994); i.e., in the arm, blood flow increased markedly for the 1st min of exercise and then began to decrease toward the baseline level, while in the leg, blood flow remained unchanged during the 1st min of exercise and then began to decrease. Controversy has existed regarding the mechanism underlying the initial increase in arm blood flow (Blair et al., 1961; Eklund and Kaijser, 1978; Sanders et al., 1989). It has been suggested that the increase is closely coupled with exercise-related emotional stress (Blair et al., 1961) and/or somatomotor activity (central command) (Eklund and Kaijser, 1978; Sanders et al., 1989), because an increase in forearm blood flow during exercise was not seen in subjects oriented to the exercise protocol (Blair et al., 1961) and because an increase in blood flow occurred abruptly with the onset of exercise (Eklund and Kaijser, 1978; Sanders et al., 1989). In the present study, SmO$_2$BA was found to increase rapidly after the onset of exercise. Therefore, the initial increase in SmO$_2$ in the arm would have been related to exercise-related emotional stress and/or central command.

The decreases in SmO$_2$ in the arm and leg in the latter part of exercise were accompanied by an increase in MBP, suggesting that vasoconstriction caused the decrease in SmO$_2$. Furthermore, the time points at which SmO$_2$ began to decrease correspond to those at which MSNA begins to increase, as observed by Wallin et al. (1989). They compared changes in MSNA in radial and peroneal nerves during 2-min isometric handgrip exercise at 30% MVC and demonstrated that MSNA in both sites began to increase 1 min after the onset of exercise and then continued to increase until the end of exercise. This finding suggests a close relationship between decreases in SmO$_2$ and increases in MSNA.

**SmO$_2$ after exercise**

In the PEAO$_{W_2}$ bout, SmO$_2$TA remained below the pre-exercise level during the initial 1.5 min of recovery, although MBP during this period was the same as the pre-exercise level. Furthermore, in the PEAO$_{W_2}$ bout, SmO$_2$BA after 1 min of PEAO and SmO$_2$TA throughout PEAO were significantly lower than the pre-exercise baseline value, although MBP during this period was the same as the pre-exercise baseline value or higher than the baseline value. These findings suggest that the lowered SmO$_2$ during the post-exercise period was also caused by vasoconstriction.

Sinoway et al. (1989) found that isometric handgrip exercise at 30% MVC and subsequent PEAO induced calf vasoconstriction and that the vasoconstriction was associated with muscle acidosis of the exercised forearm muscle. It is well known that PEAO continuously stimulates muscle metaboreceptors, resulting in a reflexive increase in blood pressure. Indeed, an increase in MBP during PEAO was found in the present study. PEAO was also found to diminish recovery of SmO$_2$ in the arm and leg. Therefore, the diminished recovery of SmO$_2$ in the arm and leg during PEAO would have been mediated by metaboreceptors in the exercised muscle.

To our knowledge, there has been no study in which circulatory differences in non-exercised arm and leg muscles after isometric handgrip exercise were compared. Rusch et al. (1981) measured calf and forearm blood flow by venous...
occlusion plethysmography during recovery with and without PEAO after isometric handgrip exercise at 33%MVC. Unfortunately, there was little in their report about the blood flow responses in the arm and leg.

There were two obvious differences in the patterns of change in SmO2_Hb and SmO2_Tra during the post-exercise period. One difference is that SmO2_Hb continued to decrease for about 1.5 min, whereas SmO2_Tra tended to begin increasing at about 30 sec of recovery in the PEAO_WO bout. Another difference is that SmO2_Hb continued to decrease until the end of PEAO, whereas SmO2_Tra remained unchanged during PEAO from the value during the last 30 sec of exercise in the PEAO_WO bout. With regard to the latter difference, Wallin et al. (1989) also compared changes in MSNA in radial and peroneal nerves during 2-min PEAO following isometric handgrip exercise at 30%MVC, and they demonstrated that peroneal MSNA during post-exercise ischemia remained at the same level as that at the 2nd min of exercise but that there was a further increase in radial MSNA. These patterns of change in MSNA during PEAO are comparable to those in SmO2 during PEAO observed in the present study. Thus, it is possible that the different patterns of change in SmO2 during recovery in the arm and leg are caused by the different patterns of change in MSNA.

Another possibility is as follows. In both PEAO_WO and PEAO_WO bouts, HR returned to the baseline level within 30 sec after the end of exercise, indicating that the effect of central command was withdrawn immediately during the post-exercise period (Mark et al., 1985; Mitchell, 1990). Thus, it is possible that the increase in SmO2_Hb, which had presumably been related to central command, was stopped immediately after the end of exercise and therefore that SmO2_Hb began to decrease. This decrease might have been reflected by the decrease in SmO2 during recovery observed only in the arm. In addition to the factors mentioned above, differences in α-adrenergic vascular responsiveness might have had some effects on the patterns of change in SmO2 in the arm and leg (Pawelczyk and Levine, 2002). Although the mechanisms underlying the different responses in SmO2 in the arm and leg could not be elucidated in this study, the findings of the present study clearly indicate that there are different patterns of change in SmO2 in the arm and leg during recovery.

**Limitation**

The effect of skin blood flow (SBF) on oxygenation measured by near-infrared spectroscopy (NIRS) has been examined in previous studies. Davis et al. (2006) demonstrated that local and whole body heating causes increases in oxygenated hemoglobin/myoglobin concentration and that these increases were closely related to increases in SBF during both local heating and whole body heating. In addition, Buono et al. (2005) demonstrated that when SBF was increased and decreased following local heating and intradermal epinephrine injection, muscle oxygen saturation was also increased and decreased. In contrast, Mancini et al. (1994) concluded that the effect of SBF on the NIRS signal is minimal based on the finding that oxygenation remained unchanged in spite of an increase in SBF during hot water immersion. Buono et al. (2005) suggested that the effect on the NIRS signal is related to the magnitude of change in SBF; since SBF was increased by only 2 to 4 fold in the study by Mancini et al. (1994), whereas it was increased by approximately 9 fold over resting values in the study by Buono et al. (2005). In the present study, since subjects performed brief exercise of small muscle masses, the absolute level of increase in SBF would have been low and therefore the effect of SBF on SmO2 would have been minimal.

**Conclusions**

Our findings suggest that oxygen supply during recovery in non-exercised arm and leg muscles is mediated by metaboreceptors in the exercised muscle but that the patterns of change during recovery are different in these two muscles. It is expected that these two factors, i.e., metaboreceptor-mediated control and different patterns of change in the arm and leg, also affect the recovery kinetics of oxygen supply in “exercised arm and leg muscles”.

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**References**


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