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Effect of Blood Lactate Concentration and the Level of Oxygen Uptake Immediately before a Cycling Sprint on Neuromuscular Activation during Repeated Cycling Sprints

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Abstract The purpose of this study was to determine whether neuromuscular activation is affected by blood lactate concentration (La) and the level of oxygen uptake immediately before a cycling sprint (pre$\dot{V}O_2$). The tests consisted of ten repeated cycling sprints for 10 sec with 35-sec (RCS_{35}) and 350-sec recovery periods (RCS_{350}). Peak power output (PPO) was not significantly changed despite an increase in La concentration up to 12 mmol/L in RCS_{350}. Mean power frequency (MPF) of the power spectrum calculated from a surface electromyogram on the vastus lateralis showed a significantly higher level in RCS_{350}. In RCS_{35}, pre$\dot{V}O_2$ level and La were higher than those in RCS_{350} in the initial stage of the RCS and in the last half of the RCS, respectively. Thus, neuromuscular activation during exercise with maximal effort is affected by blood lactate concentration and the level of oxygen uptake immediately before exercise, suggesting a cyclic system between muscle recruitment pattern and muscle metabolites. *J Physiol Anthropol 25(4): 267–273, 2006* http://www.jstage.jst.go.jp/browse/jpa2
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Keywords: phosphocreatine, metabolic acidosis, surface electromyogram, fatigue

Introduction

Muscle fatigue has been physiologically examined in relation to energy metabolism in exercise with large muscle groups (Bonen and Belcastro, 1976; Choi et al., 1994; McCann et al., 1995; Sahlin et al., 1976; Sahlin and Ren, 1989; Weltman et al., 1977). It has been shown that two metabolites, lactate and phosphocreatine (PCr), have a key role in metabolic muscle fatigue during brief exercise with maximal effort. PCr breakdown provides an immediate energy source for resynthesis of ATP, but due to the limited amount of PCr in humans, the level of PCr is insufficient for the resynthesis of ATP required during brief exercise with maximal effort. This lack of PCr is a factor causing muscle fatigue. Recently, Westerblad et al. (2002) proposed that accumulation of inorganic phosphate (Pi) due to PCr breakdown induces muscle fatigue. Glycolysis, which results in lactate production, also contributes to the energy supply during brief exercise with maximal effort such as a 6-10-sec cycling sprint (Bogdanis et al., 1998; Gaitanos et al., 1993). The lactate production induces a decrease in intramuscular pH. This decrease in intramuscular pH results in metabolic acidosis, which indirectly affects performance during brief exercise with maximal effort through the inhibition of ATP generation derived from glycolysis (Sahlin et al., 1998).

Recently, Matsuura et al. (2005) reported that peak power output (PPO) in ten repeated cycling sprints (RCS) for 10 sec with 350-sec recovery periods was maintained despite a dramatic increase in blood lactate concentration (La). They also found that mean power frequency (MPF) of the power spectrum calculated from a surface electromyogram (SEMG) on the vastus lateralis (VL) progressively shifted to higher frequencies with increases in La. Therefore, they concluded that the maintenance of PPO during RCS with 350-sec recovery periods resulted from counteraction between the detrimental effect by La increase on PPO and the beneficial effect of high MPF on PPO.

Based on this conclusion by Matsuura et al. (2005), we modified the traditional fatigue concept as follows (see Fig. 1): Firstly, in intermittent exercise with maximal effort, the conscious division in the central nervous system (CNS) gives a signal to perform exercise with maximal effort to the subconscious division in the CNS (I: 1). In response to this signal, the subconscious division in the CNS sends an efferent signal, muscle recruitment pattern, to a muscle (I: 2). In consequence, the muscle produces performance (e.g., PPO). Metabolites produced and those consumed in previous exercise

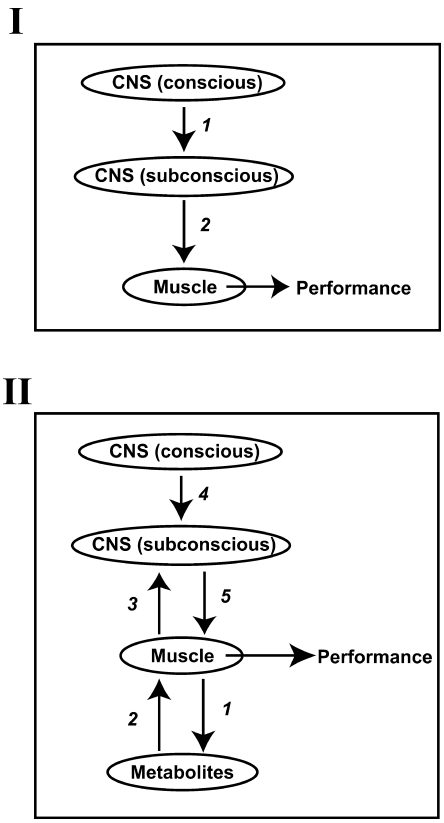

Fig. 1 Hypothesized mechanism of fatigue. I: Performance in the first sprint during RCS (top). II: Performance from the second sprint during RCS (bottom). See text for details.

(II: 1) decrease the energy supply to muscles. This is the so-called metabolic muscle fatigue (II: 2). Before the following exercise, information about the state of metabolites in a muscle is signaled to the subconscious division in the CNS by afferent nerves (II: 3). Accordingly, even though the conscious division subsequently gives a signal to perform exercise with maximal effort to the subconscious division (II: 4), the subconscious division sends an efferent signal to the muscle based on the information signaled by afferent nerves (II: 5).

Therefore, in this new fatigue concept, performance is regulated by both functions of metabolic muscle fatigue and muscle recruitment pattern. Thus, it is hypothesized that there is an interaction in this system between muscle recruitment pattern and muscle metabolites (a cyclic system).

The purpose of the present study was, therefore, to determine whether neuromuscular activation is affected by La and/or the level of oxygen uptake immediately before a cycling sprint during repeated cycling sprints.

Methods

Subjects

Eight healthy male undergraduate students participated in this study. The subjects' mean age, height and body weight were 20.4±1.4 (SD) years, 171.2±5.9 cm and 66.7±10.6 kg, respectively. They had no neuromuscular disorders and were participating in regular swimming training programs. Each subject signed a statement of informed consent following a full explanation regarding the nature of the experiment. The Ethics Committee of Hokkaido University Graduate School of Education approved the present study.

Design

Each subject attended our laboratory for three tests. The time interval between two consecutive tests was at least 3 days, and all tests were completed within 2 weeks. On the first test day, the subject's body characteristics were measured and each subject performed one of the two experimental protocols described below to become familiarized with an RCS. Body weight (BW) was used to determine the loads of the cycling sprint. Each subject was instructed to refrain from intense physical exercise, drinking, and taking caffeine for 24 h prior to each visit. None of the subjects had a smoking habit.

Experimental protocol

Each subject came to the laboratory 30 min before the start of the test. Then experimental instruments were fitted to each subject. After resting for 3 min on the bicycle seat, each subject performed two RCS tests with different recovery periods in randomized order on separate days. The tests consisted of ten 10-sec cycling sprints with 35-sec recovery periods (25 sec of low-intensity cycling exercise with 0 W (60 rpm) and 10 sec of passive recovery on the bicycle seat: RCS_{35}) and ten 10-sec cycling sprints with 350-sec recovery periods (340 sec of low-intensity cycling exercise with 0 W (60 rpm) and 10 sec of passive recovery on the bicycle seat: RCS_{350}). All cycling sprints were performed with a load (F) [N] of $0.075 \cdot BW \cdot 9.81^{-1}$ (Ayalon et al., 1974) from a standing start. Subjects were instructed to pedal as many revolutions as possible during cycling sprints. The number of sets in the RCS was not announced in advance to prevent the subjects from making an unconscious plan for whole power output.

Peak power output during 10-sec cycling sprints

All exercise tests were carried out on a bicycle ergometer (POWERMAX-V_{II}, Combi, Tokyo, Japan). The duration and load were adjusted by a built-in computer. The computer also calculated mean power output and peak rpm (Rpm_{peak}) in a given exercise and displayed the results. Since the screen on the bicycle ergometer displayed Rpm_{peak}, the screen was covered. Each subject's feet were strapped to the pedals to prevent them from slipping. The seat height was adjusted so that there was a slight bend in the knee joint when the foot pedal was at its lowest position. PPO was calculated by the following equation:

$$\text{PPO [watts]} = Rpm_{peak} \cdot 6 \cdot F \cdot 0.624^{-1},$$

where 6 is the distance calculated by the built-in computer as the flywheel went into a 360-degree roll [m], and 0.624 is the

value for transforming Nm units to watt units [$Nm \cdot min^{-1} \cdot watt^{-1}$]. To reduce variation due to the difference in body characteristics of subjects, PPO divided by BW ($PPO \cdot BW^{-1}$) was used.

Blood lactate concentration

Blood samples (25 μL) were collected from fingertips using capillary tubes and analyzed using a lactate analyzer (YSI-1500 sport, YSI, Tokyo, Japan) to measure La. The lactate analyzer was calibrated by a standard lactate solution of 5 $mmol \cdot L^{-1}$ before each test. Blood was sampled at rest and immediately after the 5th and 10th cycling sprints.

Oxygen uptake

Data on oxygen uptake ($\dot{V}O_2$) were obtained breath-by-breath using a respiratory gas analyzer (AE-280S, Minato Medical Science, Osaka, Japan). Ventilation ($\dot{V}E$) was measured by a hot-wire flow meter, and the flow meter was calibrated with a syringe of known volume (2.0 L). O_2 and CO_2 concentrations were measured by 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%). $\dot{V}O_2$ was measured continuously during rest, exercise, and recovery periods. For each 15-sec interval, the averages of $\dot{V}O_2$ were calculated. To reduce variation due to the difference in body characteristics of subjects, $\dot{V}O_2$ divided by BW ($\dot{V}O_2 \cdot BW^{-1}$) was used.

Surface electromyogram

An SEMG was recorded from the left vastus lateralis (VL) and the left rectus femoris (RF) at a rate of 1000 Hz during each of the ten cycling sprints. Before attachment of the surface electrodes, the skin was shaved, abraded, and cleaned with alcohol in order to reduce source impedance. A bipolar SEMG sensor (SX230, Biometrics Ltd, United Kingdom; inter-electrode distance of 20 mm) was placed on the lateral side of the crural area five-fingers proximal from the patella of the belly of the VL and on the lower limb midway between the superior surface of the patella and the anterior superior iliac spine of the belly of the RF. The ground electrode was placed over the right lateral malleolus. The SEMG signals were amplified using an amplifier imbedded in the EMG sensor (bandwidth=20–450 Hz; common mode rejection ratio, CMRR>96 dB; input impedance>10 TΩ; gain=1000) and converted into digital signals using an analog-digital converter (MacLab/8s, ADInstruments, Australia). Then SEMG data were processed offline by using analysis software (Acknowledge, BIOPAC Systems, United States). Raw data were filtered using a band pass Finite Impulse Response filter with a cut-off frequency of 10 to 500 Hz. Since a previous study in our laboratory (Matsuura et al., 2005) showed that power output during a 10-sec cycling sprint reached a peak at 6.2 sec on average, four seconds (4–8 sec) of raw data were used for subsequent analysis. The four seconds of raw data were full-wave-rectified and then integrated (IEMG). Furthermore, the raw data were processed with fast Fourier transform to obtain a frequency power spectrum. The frequency spectrum analysis was restricted to frequencies in the range 5–500 Hz (St Clair Gibson et al., 2001). MPF was defined as the ratio between spectral moments of orders one and zero (Moritani et al., 1982). IEMG and MPF calculated from raw data of an SEMG in a given time period during dynamic exercise such as a cycling sprint are used to determine neuromuscular activation (Hunter et al., 2003; Kay et al., 2001; St Clair Gibson et al., 2001). Analysis was performed using the technique described by Lowery et al. (1998), as a modification of the work of Lo Conte and Merletti (1996) and Merletti and Lo Conte (1997). The position of the electrodes for SEMG detection was similar in the two conditions because reference points were marked on the skin. The IEMG and MPF were normalized as a percentage of the 1st set value.

Statistical analysis

Results are presented as means±standard deviations (SD). A paired *t*-test was used to examine differences between conditions. One-way ANOVA (for $PPO \cdot BW^{-1}$, La, and $\dot{V}O_2 \cdot BW^{-1}$) for repeated measures was used to examine differences over sprint number of sets. Two-way ANOVA (for IEMG and MPF) for repeated measures on both factors (sprint number and condition) was used. When significant *F* ratios were found, the means were compared by using Tukey-Kramer's post hoc test. If significant interaction was indicated, a paired t-test was used to examine the difference between conditions. A value of $p<0.05$ was regarded as statistically significant.

Results

Peak power output

$PPO \cdot BW^{-1}$ values in the 1st set in the two RCS tests were similar (RCS_{35}: 10.8±0.8 $W \cdot kg^{-1}$, RCS_{350}: 10.9±0.7 $W \cdot kg^{-1}$; $P=0.40$). In the RCS_{35} test, $PPO \cdot BW^{-1}$ values were significantly lower in the 4th–10th sets than in the 1st set but, in the RCS_{350} test, $PPO \cdot BW^{-1}$ values did not change (Fig. 2).

Blood lactate concentration

La recorded immediately after the 5th and 10th cycling sprints are expressed as 5th and 10th set values, respectively (Fig. 3). La in the 1st set in the two RCS tests was similar (RCS_{35}: 1.1±0.3 $mmol \cdot L^{-1}$, RCS_{350}: 1.0±0.3 $mmol \cdot L^{-1}$; P=0.84). In both RCS_{35} and RCS_{350} tests, La was significantly higher in the 5th and 10th sets than at rest. Furthermore, in the RCS_{35} test, La in the 10th set was significantly higher than that in the 5th set. La in the 10th set in RCS_{35} and RCS_{350} were 13.9±3.1 $mmol \cdot L^{-1}$ and 11.6±3.1 $mmol \cdot L^{-1}$, respectively.

Oxygen uptake immediately before cycling sprint

Changes in $\dot{V}O_2 \cdot BW^{-1}$ for 15 sec immediately before each of the ten cycling sprints ($pre\dot{V}O_2$) in the two RCS tests are

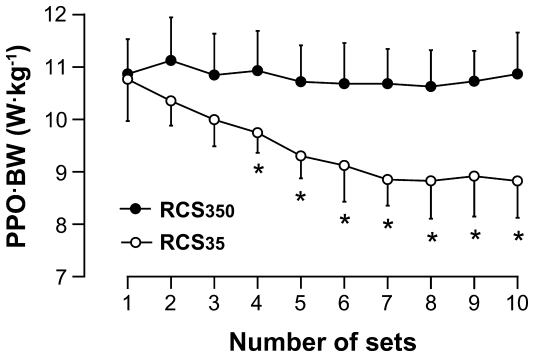

Fig. 2 Changes in peak power output divided by BW in each of the subjects ($PPO \cdot BW^{-1}$) in RCS_{35} (○) and RCS_{350} (●). *: significantly different ($p<0.05$) from the value in the 1st set in RCS_{35}.

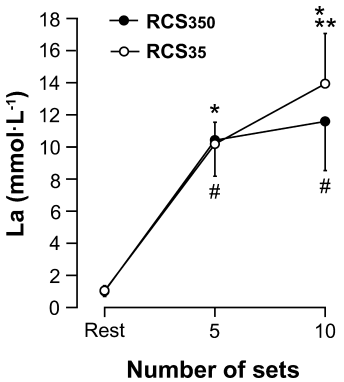

Fig. 3 Changes in blood lactate concentration (La) during RCS_{35} (○) and RCS_{350} (●). *: significantly different ($p<0.05$) from the value at rest in RCS_{35}. #: significantly different ($p<0.05$) from the value at rest in RCS_{350}. ##: significantly different ($p<0.05$) from the value in the 5th set in RCS_{350}.

shown in Fig. 4. The value of $preV\dot{O}_2$ recorded immediately before the 1st cycling sprint is expressed as the 1st set value. The 5th set value was used as a baseline value since active recovery immediately before the 1st cycling sprint was not performed. In the RCS_{35} test, $preV\dot{O}_2$ was significantly lower in the 1st set than in the 5th set, and no significant differences were found from the 2nd set to the 10th set. In the RCS_{350} test, a pattern similar to that in RCS_{35} was found. The value of $preV\dot{O}_2$ in the 5th set during RCS_{35} ($28.9 \pm 3.2\,mL \cdot min^{-1} \cdot kg^{-1}$) was significantly higher than that in the 5th set during RCS_{350} ($8.8 \pm 1.3\,mL \cdot min^{-1} \cdot kg^{-1}$). Because of technical difficulties, the proportion of low-intensity exercise/passive recovery time intervals contributing to $preV\dot{O}_2$ was not strictly constant.

Surface electromyogram

There were no differences between the two RCS tests for absolute IEMG value (VL: $p=0.54$, RF: $p=0.63$; data not shown) and absolute MPF value (VL: $p=0.36$, RF: $p=0.31$; data not shown) in the 1st set. Changes in IEMG and MPF in the two RCS tests are shown in Fig. 5 and Fig. 6, respectively.

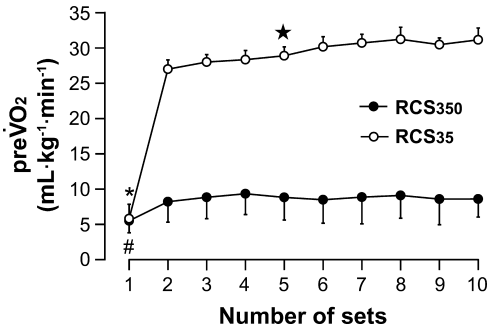

Fig. 4 Changes in $\dot{V}O_2$ divided by BW in each of the subjects for 15 sec immediately before each of the ten cycling sprints ($pre\dot{V}O_2$) during RCS_{35} (○) and RCS_{350} (●). #: significantly different ($p<0.05$) from the value in the 5th set in RCS_{350}. *: significantly different ($p<0.05$) from the value in the 5th set in RCS_{35}. #: significantly different ($p<0.05$) from the value in the 5th set in RCS_{350}. ★: significant difference ($p<0.05$) between the value in RCS_{35} and that in RCS_{350}.

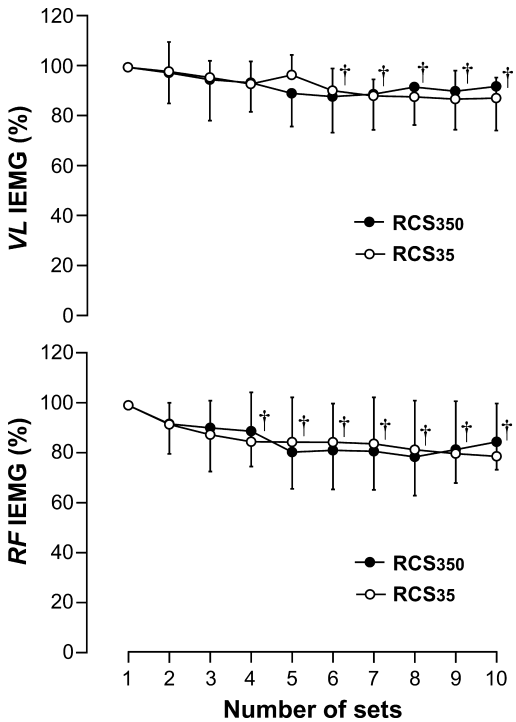

Fig. 5 Changes in integrated EMG (IEMG) from the left vastus lateralis (VL: top) and from the left rectus femoris (RF: bottom) normalized by the 1st set value during RCS_{35} (○) and RCS_{350} (●). †: significantly different ($p<0.05$) from the value in the 1st set.

When two-way ANOVA for repeated measures was used, no significant interaction was found in both IEMG from the VL and that from the RF. No significant effects of the condition on both IEMG from the VL and that from the RF were found. In both RCS_{35} and RCS_{350}, IEMG from the VL significantly decreased from the 6th set compared to that in the 1st set, and IEMG from the RF significantly decreased from the 4th set compared to that in the 1st set. When two-way ANOVA for repeated measures was used, no significant interaction was

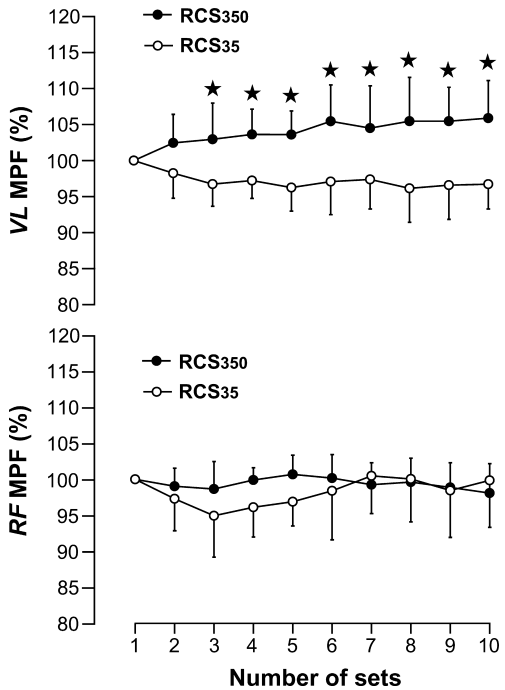

Fig. 6 Changes in mean power frequency (MPF) from the left vastus lateralis (VL: top) and from the left rectus femoris (RF: bottom) normalized by the 1st set value during RCS_{35} (○) and RCS_{350} (●). ★: significant difference ($p<0.05$) between the values in RCS_{35} and those in RCS_{350}.

found in MPF from the RF, but a significant interaction was found in MPF from the VL ($p<0.0001$). No significant effects of the condition on MPF from the RF were found. In both RCS_{35} and RCS_{350}, MPF from the RF did not change. In the RCS_{350} test, MPF values from the VL in the 3rd–10th sets were significantly higher than those in the same sets in the RCS_{35} test.

Discussion

Relationship among physiological parameters in RCS_{350}

Since Rossiter et al. (2002) reported that restoration of PCr after high-intensity exercise corresponds to decrease in $\dot{V}O_2$ during the recovery period, the degree of PCr level can indirectly be estimated by $\dot{V}O_2$ kinetics. Although the 15-sec data of pre$\dot{V}O_2$ significantly increased from the 2nd set due to active recovery, pre$\dot{V}O_2$ values did not change from the 2nd set to the 10th set. Additionally, it has been reported that the PCr consumed by high-intensity exercise recovers to almost the resting level in 6 min (Arsac et al., 2004; Harris et al., 1976; McCann et al., 1995). Therefore, it is likely that PCr level immediately before each cycling sprint was similar over the 2nd set. In contrast to the PCr level, La increased up to approximately 12 mmol/L, suggesting that metabolic acidosis was induced in the RCS_{350} test. Under these conditions, IEMG and MPF were progressively changed.

Although some authors have reported that there is no linear relation between the characteristic frequencies of an EMG power spectrum and muscle fiber action potential conduction velocity (MFCV) (Linssen et al., 1990; Naejie and Zorn, 1982), many authors have reported that a slowdown of MFCV prolongs the muscle fiber action potential wave form (Lindström et al., 1970), which in turn brings about a decrease in MPF (Moritani et al., 1986; Stulen and De Luca, 1981). The slowdown of MFCV, which is responsible for the shift of the EMG power spectrum to lower frequencies, has been reported to result from the effect of a decrease in intramuscular pH on muscle contractility, i.e., E-C coupling (Brody et al., 1991; Mortimer et al., 1970; Tesch et al., 1983). *In vitro* studies of skinned muscle fibers have shown that muscle contractility is impaired during acidotic conditions (Cooke et al., 1988; Godt and Nosek, 1989).

However, it has recently been shown in studies using mammalian muscle that these direct effects of metabolic acidosis on muscle contractility were absent at more physiological temperatures (Pate et al., 1995; Westerblad et al., 1997). Furthermore, in the present study, MPF in the VL and the RF did not decrease despite a considerable increase in La. This result agrees with the finding that a regular relationship did not exist between MPF and La in human quadriceps muscle (Gamet et al., 1993). Therefore, it is likely that the slowdown of MFCV is due to a recruitment of muscle fibers with a lower MFCV rather than the effect of a decrease in intramuscular pH on muscle contractility. Consequently, the MPF shift to high frequencies is indicative of a recruitment of type IIb fibers with higher MFCV (Moritani and Muro, 1987).

IEMG increases in proportion to the increase in the number of motor units (MUs) recruited and/or the rate of discharge of the recruited MUs in exercising muscles (Lind and Petrofsky, 1979; Moritani et al., 1982). Since the rate of discharge of type IIb muscle fibers is higher, IEMG should have increased during the RCS_{350}, in which MPF increased. Therefore, it is possible that the decline in IEMG during RCS_{350} was associated with a decrease in the number of MUs recruited.

Thus, the decrease in IEMG and the increase in MPF during RCS_{350} are attributable to selective recruitment of type IIb muscle fibers (Moritani and Mimasa, 1990). These results of the RCS_{350} test suggest that maximal effort does not result in the same muscle recruitment pattern.

Relationship among physiological parameters in RCS_{35}

Since the active and passive recovery periods in the present study were the same immediately before each cycling sprint during RCS tests, although the ratio between active recovery period and passive recovery period might not be constant in the 15-sec data, it is possible to compare pre$\dot{V}O_2$ values among sets or between the two RCS tests. The value of pre$\dot{V}O_2$ in RCS_{35} was higher from the 2nd set to the 10th set than that in RCS_{350}. This higher pre$\dot{V}O_2$ level could induce a lower PPO by a higher Pi level since a high level of Pi causes muscle fatigue (Westerblad et al., 2002). Furthermore, in the 10th set, La in RCS_{35} was higher than that in RCS_{350}, suggesting that, in the last half of the RCS, the degree of metabolic acidosis in RCS_{35}

was higher than that in RCS_{350}. Under those conditions, a lower MPF in the VL was observed in RCS_{35}.

In the RCS_{35} test, the decline in IEMG was similar to that in RCS_{350} but the MPF level was lower. These results for SEMG are attributed to the selective recruitment of muscle fibers with a lower MFCV, i.e., type IIa or type I muscle fibers. Since muscle fibers with a lower MFCV also have a lower rate of discharge of MUs, this lower rate of discharge might affect the decline in IEMG.

Thus, these results of the RCS_{35} test suggest that a higher state of metabolic fatigue induces recruitment of non-fatigable muscle fibers.

Alternative interpretation of SEMG

It is possible that metabolic acidosis affects sarcolemma Na^+-K^+ ATPase pumps through inhibition of ATP generation, which induces a decrease in amplitude and prolongation of duration of the sarcolemma action potential (Balog et al., 1994). These effects of acidosis on sarcolemma action potential prolongs the compound action potential wave form detected by SEMG (Dimitrova and Dimitrov, 2003), resulting in a decrease in MPF. Therefore, it is thought that the degree of selective recruitment of type IIb muscle fibers in the RCS_{350} test was underestimated. Furthermore, in the RCS_{35} test, some type IIb muscle fibers might have been recruited, but this recruitment might have been underestimated.

Additionally, surface-detected EMG amplitude characteristics can increase even when intramuscular action potential amplitude decreases (Dimitrova and Dimitrov, 2003). Therefore, it is likely that the number of motor MUs recruited and/or the rate of discharge of the recruited MUs in the two RCS tests were overestimated.

However, these alternative interpretations do not alter the conclusion in the present study.

Cyclic system

Since changes in La in the two RCS tests differed in the latter period, the difference in MPF levels in the latter period might be induced by this difference in La. Although pre$\dot{V}O_2$ was different in the two RCS tests over the 2nd set, a difference between MPF levels in the two RCS tests was found from the 3rd set. Therefore, the difference in MPF levels in the former period can not be explained by only PCr level or Pi level. This discrepancy between changes in La and/or pre$\dot{V}O_2$ and changes in MPF may be due to the fact that intramuscular metabolites were not directly measured in the present study.

Lambert et al. (2005) proposed an integrative model in which muscle metabolites provide signals to the brain rather than cause fatigue. In this model, the brain perceives the state of all metabolites, reserve of energy resources, and external environment and then selects an exercise pace for maintaining homeostasis after taking into account training history. Furthermore, it is assumed in this model that there is an interaction between conscious and subconscious components in the brain. Since the results of the present study suggest that a difference in metabolic state of the muscle affects neuromuscular activation, the present study partly supports this hypothetical model of Lambert et al. (2005).

Conclusion

Blood lactate concentration and the level of oxygen uptake immediately before exercise alter neuromuscular activation during exercise with maximal effort, but evidence that the level of pre$\dot{V}O_2$ alone affects neuromuscular activation was not obtained in the present study. The results of the present study suggest a cyclic system between muscle recruitment pattern and muscle metabolites.

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