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DEVELOPMENT OF ACCUMLATED AND TEMPORARY FATIGUE DURING REPEATED CYCLING SPRINTS

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Abstract

The purpose of this study was to determine whether accumulated and temporary fatigue could be separately observed in repeated cycling sprints (RCS) with combined short and long recovery periods. Eight male performed three RCS with 35-sec (RCS_{35}), 350-sec (RCS_{350}) and combined 35-sec and 350-sec recovery periods (RCS_{comb}). RCS_{comb} consisted of ten 10-sec cycling sprints (CSs) with 35-sec and 350-sec recovery periods before the 5th and 9th CS. In RCS_{comb}, peak power output (PPO) was restored in the 5th and not in 9th CS. Blood lactate concentration ([La]) progressively increased, but there were no significant differences among conditions despite the difference in PPO. In RCS_{comb}, mean power frequency determined on the vastus lateralis was correlated with PPO and oxygen uptake before CSs ($preV\dot{O}_2$) showed high in short recovery periods and low values in long recovery periods. Accumulated and temporary fatigue cannot be explained by effects of $preV\dot{O}_2$ and [La].

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key word : creatine phosphate, blood lactate concentration, surface electromyogram

Introduction

Muscle fatigue is generally defined as a decrease in force production[4] or an inability to regenerate the original force[1] despite increased perception of effort. Recent studies[2,3] have focused on muscle fatigue in repeated cycling sprints (RCS) with large muscle groups as a simulation of actual sports or actions.

Gaitanos et al.[3] reported that peak power output (PPO) progressively decreased when ten 10-sec cycling sprints with 30-sec recovery periods were performed. Since it takes 6 min for PCr resynthesis following high-intensity exercise[6], it is thought that the impairment of performance observed during RCS in the study of Gaitanos et al.[3] is dependent on the degree of PCr resynthesis. Furthermore, since it is thought that the degree of PCr resynthesis is constant when recovery periods during RCS are constant, the impairment induced by incomplete PCr resynthesis is 'temporary fatigue.' However, progressive increase in muscle lactate level with increase in blood lactate would also be observed during RCS. This progressive increase could induce decrease in intramuscular pH, resulting in inhibition of PCr resynthesis and impairment of excitation-contraction coupling. Since muscle lactate would accumulate, 'accumulated fatigue' might also occur during RCS.

Thus, if a 6-min recovery period, which is sufficient for PCr resynthesis, is inserted into the RCS, the effect of PCr resynthesis on muscle contractility could be eliminated and only the effect of accumulated lactate on muscle contractility would be observed after the 6-min recovery period. The purpose of this study was to determine whether accumulated and temporary fatigue could be separately observed in RCS with combined short and long recovery periods.

Methods

Eight healthy male undergraduate students participated in this study. Each subject performed three RCS tests with different recovery periods. The tests consisted of ten 10-sec cycling sprints with 35-sec recovery periods (25 sec of low-intensity cycling exercise with 0 kp at 60 rpm and 10 sec of passive recovery on the bicycle seat : RCS_{35}), ten 10-sec cycling sprints with 350-sec recovery periods (340 sec

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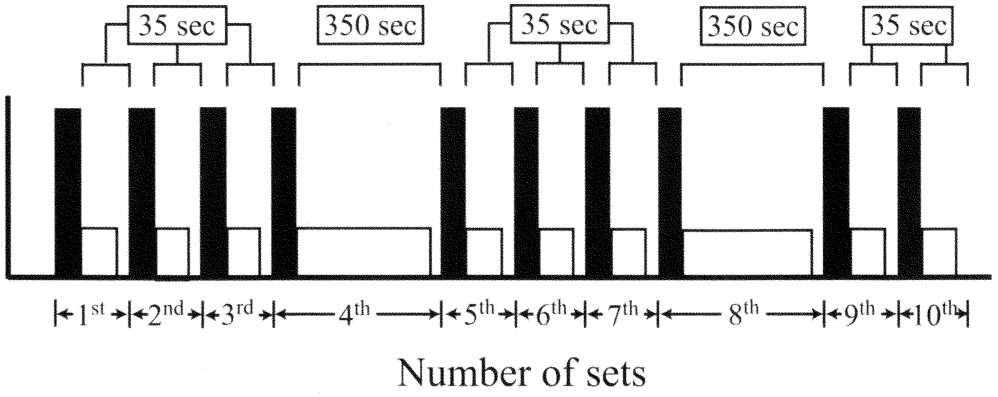

Fig. 1. RCS_{comb} consisted of ten 10-sec cycling sprints with 35-sec and 350-sec recovery periods before the 5^{th} and 9^{th} cycling sprints.

of low-intensity cycling exercise with 0 kp at 60 rpm and 10 sec of passive recovery on the bicycle seat : RCS_{350}), and a combination of RCS_{35} and RCS_{350} (RCScomb) (Fig. 1). All cycling sprints were performed with a load [kp] of 0.075·body weight(BW). PPO during cycling sprints was calculated by a built-in computer of the bicycle ergometer (POWERMAX-V_{II}, Combi, Tokyo, Japan). Blood sampled at rest and immediately after the 5^{th} and 10^{th} cycling sprints were analyzed using a lactate analyzer (YSI-1500 sport, YSI, Tokyo, Japan) to measure the blood lactate concentration ([La]). 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). $\dot{V}O_2$ was measured continuously during rest, exercise, and recovery periods. For each 15-sec interval, the averages of $\dot{V}O_2$ were calculated. To examine oxygen debt im-

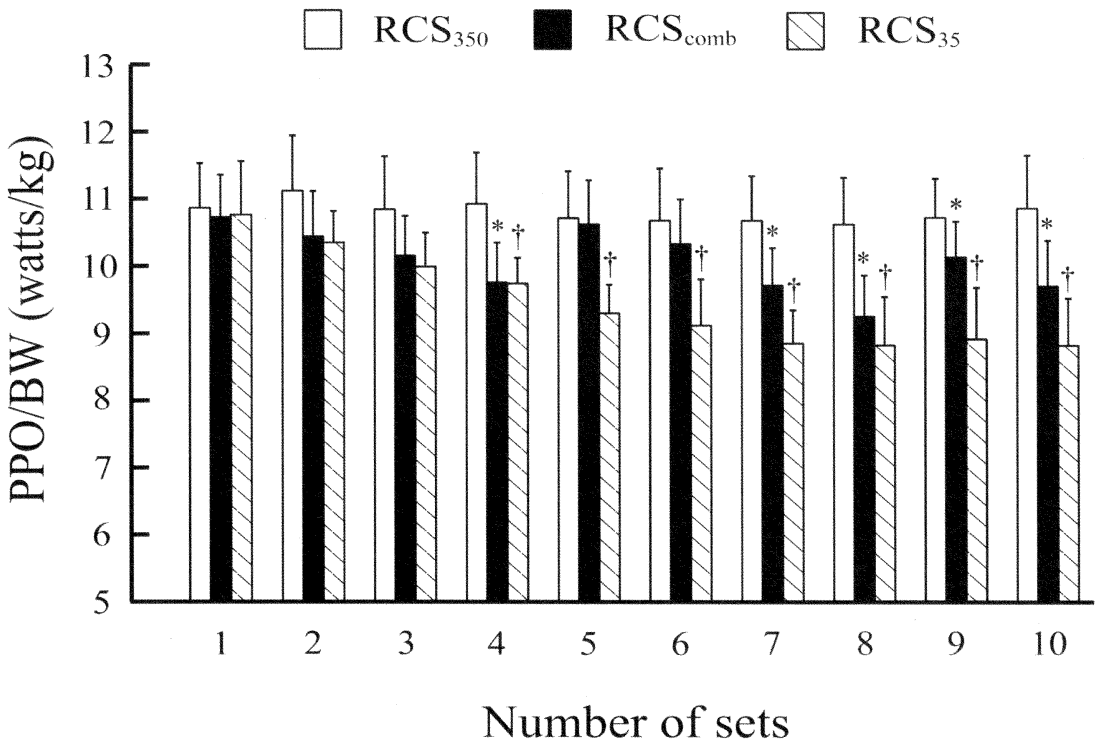

Fig. 2. Development of peak power output divided by BW (PPO/BW) during each of the ten 10-sec cycling sprints in RCS_{35} (hatched bar), RCS_{350} (blank bar), and RCS_{comb} (black bar). *: significantly different ($P<0.05$) from the value in the 1^{st} set in RCS_{comb}. †: significantly different ($P<0.05$) from the value in the 1^{st} set in RCS_{35}.

mediately before each of the ten cycling sprints, $\dot{V}O_2$ for 15 sec immediately before each of the ten cycling sprints ($pre\dot{V}O_2$) was analyzed. A surface electromyogram (sEMG) was recorded from the vastus lateralis (VL) at a rate of 1000 Hz. A bipolar surface EMG sensor (SX230, Biometrics Ltd, United Kingdom ; inter-electrode distance of 20 mm) was placed on the belly of the left VL. Mean power frequency (MPF) and integrated EMG (iEMG) during each of the ten cycling sprints were calculated using analysis software (Acknowledge, BIOPAC Systems, United States). The iEMG values were divided by mean rpm (Rpm_{mean}) exerted during each of cycling sprints to give averaged rectified value (ARV). Both ARV and MPF were normalized as a percentage of the 1st set value. A value of $P<0.05$ was regarded as statistically significant.

Results

In RCS_{comb}, PPO/BW values were significantly lower in the 4th, 7th, 8th, 9th and 10th sets than in the 1st set. PPO/BW values after 350-sec recovery periods did not decrease in the 5th set but significantly decreased in the 9th set (Fig. 2). No significant effect of the recovery period on [La] was found, and progressive increase in [La] was observed. In RCS_{35}, $pre\dot{V}O_2$ was significantly lower in the 1st set than in the 5th set, and no significant differences were found between the value of $pre\dot{V}O_2$ in the 5th set and values in sets other than the 1st set. In RCS_{comb}, no significant differences were found between $pre\dot{V}O_2$ values in the 5th and 9th sets. In RCS_{350}, MPF significantly increased in the 6th-10th sets compared to that in the 1st set and ARV significantly declined in the 5th-10th sets compared to that in the 1st set. In both RCS_{35} and RCS_{comb}, there was a positive correlation between MPF values and PPO/BW values.

Discussion

Rossiter et al.[7] have reported that there was a negative correlation between the degree of PCr resynthesis and $\dot{V}O_2$ after high-intensity exercise. In RCS_{35}, since $pre\dot{V}O_2$ showed higher values from the 2nd set and remained high throughout the RCS, PPO should have significantly declined in the 2nd set and the degree of this decline should have been maintained. However, PPO in fact significantly declined in the 4th set, suggesting that the degree of PCr resynthesis is not the only factor governing temporary fatigue.

Since [La] progressively increased, muscle lactate level would increase under all conditions, suggesting that intramuscular pH progressively decreased. Therefore, PPO/BW should have decreased at least in RCS_{350}. Thus, it is unlikely that accumulated fatigue is associated only with progressive increase in [La].

In RCS_{350}, decrease in ARV and increase in MPF were observed despite maximal effort. Matsuura et al.[5] have pointed out the possibility that impairment of muscle contractility associated with progressive increase in [La] and with increase in $pre\dot{V}O_2$ due to incomplete PCr resynthesis is compensated by changes in ARV and MPF. Therefore, it seems that impairment of muscle contractility associated with $pre\dot{V}O_2$ and [La] was compensated by change in sEMG. On the other hand, PPO decreased in RCS_{35}. This decrease may be an indication of decompensation of the sEMG. Since $pre\dot{V}O_2$ was significantly high in the initial stage in RCS_{35}, impairment of muscle contractility associated with incomplete PCr resynthesis may occur. However, a significant decrease in PPO was not observed in the initial stage. Accordingly, change in muscle recruitment indicated by the change in sEMG may firstly compensate this impairment associated with PCr resynthesis. Furthermore, since [La] progressively increased in RCS, the degree of impairment of muscle contractility would gradually increase. In RCS_{comb}, PPO/BW was restored after the first 350-sec recovery period. Therefore, the impairment of contractility due to the increase in [La] during RCS is mainly compensated by change in muscle recruitment, but PPO/BW after the second 350-sec recovery period could not be compensated due to the change in the

recruitment.

It is concluded that the first restoration and the second reduction in PPO/BW after the long recovery period in RCS_{comb} indicated accumulated and temporary fatigue. It seems that accumulated and temporary fatigue cannot be explained by effects of pre $\dot{V}O_2$ and [La] on muscle contractility since these fatigues are affected by muscle recruitment.

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