Bimodal recovery of quadriceps muscle force within 24 hours after sprint cycling for 30 seconds

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Key words: *bimodal recovery; electrical stimulation; low-frequency fatigue; potentiation; sprint cycling.*

Summary. *The aim of the study was to investigate the manifestation of potentiation and fatigue as well as the coexistence of these phenomena at different muscle lengths during a 24-hour period after a sprint cycling for 30 s.*

Material and methods. Twelve healthy untrained men (mean age 23.6±1.7 years) took part in the experiment. The contractility of quadriceps muscle was studied before (Initial) and 2, 5, 30, 60 min and 24 h after exercise via the electrically evoked contractions at 1, 15, 50 Hz and maximal voluntary contractions at short and long muscle length.

Results. 1) In early, fast-recovery phase (within the first 5 min), muscle force evoked by electrical stimulation of 1, 15, 50 Hz was restored at short muscle length, conversely at long length (Initial vs. 5 min: 15 Hz and 50 Hz, both $P<0.05$), whereas maximal voluntary contraction force was still suppressed at both muscle lengths; 2) in the second phase (from 5 min to 30–60 min), muscle force decreased at low- and high-frequency stimulations and was more expressed at low-frequency stimulation and at short muscle length than that at long length, but the maximum voluntary contraction force recovered to initial; 3) in long-lasting phase (within 24 hours), 15 Hz force was still suppressed at both muscle lengths.

Conclusion. A bimodal recovery of contractility of the quadriceps following sprint cycling for 30 s is determined by the concomitant complex interaction of mechanisms enhancing (potentiation) and suppressing (fatigue) contractile potential of the muscle.

Introduction

Changes in muscle force during and after physical activity depend on metabolic fatigue (MF) (1, 2), non-metabolic fatigue (NMF) (3–5), and potentiation (PT) (6–9). Most often contractility is determined by the coexistence of these factors; however, it is difficult to discriminate among their influences. For instance, after an intense exercise inducing MF, effect of PT is hidden (10), whereas following exercise that causes NMF, expression of low-frequency fatigue (LFF), the best characterized type of NMF, can be compensated by PT (6–9).

The dominance of MF, NMF, or PT depends on the type, intensity, and duration of exercise and duration of the recovery before contractility is tested. It has been shown that intense and very short (about 5–10 s) isometric contractions induce PT, *i.e.*, an increase in contractile force evoked by a single twitch and/or low-frequency stimulation, lasting for 5–10 min (6). Temporal characteristics of contraction are also affected in a way that force development and relaxation occur at a faster rate (11). Phosphorylation of myosin regulatory light chains has been implicated as the underlying mechanism of PT in human skeletal muscles (6).

A prolonged duration of intense contraction (10–60 s) induces a substantial disturbance of metabolic profile causing the MF (12). An increase in ADP (13) and P_i (14) concentrations occurs with a concomitant decrease in the concentrations of ATP and PCr (6). The consequence of these metabolic alterations is a reduction of free Ca^{2+} concentration in response to action potential (15) and impaired function at the level of cross-bridges (16), which in turn results in a decrease of contraction force (6, 14). Restoration of metabolic homeostasis following the exercise occurs within the range of minutes and is concomitant with rapid recovery of contractility (6, 14). Studies on isolated single fibers demonstrated that association between increase in ADP (1) and P_i (17) concentrations

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and impaired contractility is causative.

Various modes of exercise were shown to induce long-lasting NMF. A type of NMF that manifests itself by a reduced force ratio at low- and high-frequency stimulations is referred to as LFF. A selective reduction of force at low-frequency stimulation might be due to a reduction in Ca^{2+} release and a right-ward shift of force-frequency relationship (15, 18). Although the underlying mechanism is unknown, an impaired link between T-tubule and sarcoplasmic reticulum was proposed to be the cause of reduced calcium release (15).

It has been established that muscle PT is retained 3–10 min after the exercise (6), whereas MF disappears very soon after the exercise, and within 10 to 20 min after the high-intensity exercise it may have fully recovered (12, 14). LFF, however, can even increase within 15–30 min after exercise (10, 19, 20), and recovery may take as long as several days (5).

Besides, it has been found out that the manifestation of PT and LFF depends on the muscle length at which PT and LFF have been established. Several studies with human muscles have shown that the degree of potentiation is greater when the contractile response is measured at a short muscle length (21). The same holds true for LFF (22). However, up to now, the coexistence of PT and fatigue in skeletal muscle has been studied mainly after isometric contractions (8, 11). There have been no studies, to our knowledge, that document PT, moreover the coexistence of PT and fatigue following short-term maximal dynamic exercise in humans. Therefore, the main objective of this study was to investigate the manifestation of PT and fatigue as well as coexistence of these phenomena at different muscle lengths during a 24-hour period after a sprint cycling for 30 s.

We hypothesize that contractility of quadriceps muscle during a 24-hour period after a sprint cycling for 30 s is determined by the coexistence of potentiation, metabolic fatigue, and nonmetabolic fatigue.

Methods

Subjects

Twelve healthy untrained men (mean age 23.6±1.7 years, body weight 74.5±5.6 kg) gave their informed consent to participate in this study. The subjects were physically active but did not take part in any formal physical exercise or sport program. Each subject read and signed written informed consent form consistent with the principles outlined in the Declaration of Helsinki.

Sprint cycling

A 30-s Wingate test was performed according to procedures described earlier (23). The Wingate test was administered for 30 s, and resistance was set at 7.5% of body weight. Subjects were seated on the Monark ergometer (Monark 834E), and appropriate adjustments were made to ensure an optimal riding position. Rapid adjustment of flywheel tension was performed by one of the investigators so that the required tension was achieved at the start of the 30-s test. Subjects were encouraged to pedal as fast as they could prior to the application of resistance. Following the application of resistance, the subjects attempted to pedal at maximal speed throughout 30 s. Verbal encouragement was provided by the investigators. Software calculated the power (W) averaged every 5 s of the exercise task.

Force measurements

The equipment and technique used for measuring force were the same as used in the previous studies (3, 7, 19). Subjects sat upright in the experimental chair with a vertical back support. A strap secured the hips and thighs to minimize uncontrolled movements. The right leg was clamped in a force-measuring device (full knee extension being 180°) with the knee kept at an angle of 90° ("long" muscle length, LL) or 135° ("short" muscle length, SL). A 6-cm-wide plastic cuff, placed around the right leg just proximal to the malleoli, was tightly attached to a linear variable differential transducer. The output of the transducer, proportional to isometric knee extension force, was amplified and digitized at a sampling rate of 1 kHz by a 12-bit analogue-to-digital converter incorporated in a personal computer. The digitized signal was stored on a hard disk for subsequent analysis. The output from the force transducer was also displayed on a voltmeter in front of the subject.

Electrical stimulation

Equipment and procedure for electrical stimulation were essentially the same as it has been described previously (3, 10, 19). A high-voltage stimulator (MG 440, Medicor, Budapest, Hungary) was used to deliver electrical stimuli to the quadriceps muscle through surface electrodes (9×18 cm) padded with cotton cloth and soaked in saline solution. One stimulation electrode was placed just above the patella, while another one covered a large portion of the muscle belly in the proximal third of the thigh. The electrical stimulation was always delivered in trains of square-wave pulses of 1-ms duration. To maximize recruitment of fibres, the highest possible stimulation

voltage was employed. Voltage was set at 120–150 V, which induces approximately 60% of maximal voluntary contraction (MVC) force. The subjects were familiarized with electrical stimulation procedure during the introductory visit before the onset of experiments (the muscle was stimulated 2–3 times by a single stimulus at 70–90 V).

We measured the contractile force of the quadriceps muscle, evoked by electrical stimulation at 1 Hz (P1), 15 Hz (P15), and 50 Hz (P50) (the duration of each electrical stimulation series was 1 s). The MVC was also determined (the peak MVC was reached and maintained for about 2 s before relaxation). The rest interval between muscle electrical stimulation was 3 s. The ratio of P15/P50 kinetics after exercise was used for the evaluation of LFF (22, 25). The contractile force was tested in a random order at knee-joint angles of 135° (SL) and 90° (LL).

Muscle soreness

Muscle soreness was reported subjectively using a visual analogue scale of 0 to 10, where 0 represented "no pain" and 10 represented "intolerably intense pain." The participants were required to indicate the severity of soreness in their quadriceps in response to muscle compression as well as when standing up and walking. Muscle soreness was evaluated at 24 h after the sprint cycling for 30 s. These methods for the evaluation of muscle soreness have also been used in our previous researches (3, 4, 10).

Experimental protocol

The experiment was designed to assess the time-course of recovery of muscle contraction properties after maximal sprint cycling for 30 s. Before the Wingate test, a resting blood sample was taken to determine resting lactate levels (26). After the initial blood sample was taken, the participants performed a warm up of at least 5 min that would prepare them for maximal effort. Right afterwards, the subject was seated in the experimental chair, and after 5 min, the initial contractile properties of the muscle (Ini) were recorded in the following sequence: P1, P15, P50, and MVC (MVC was reached twice with 1-min rest between). During the period of recovery, at each time point MVC was evaluated but once. Two min, 5 min, 30 min, 60 min, and 24 h after the exercise, the contractile properties of skeletal muscle were tested at SL and LL. In addition, during the next day, the subjects were subjectively evaluated for their muscle pain (during walking) using a 10-point scale. A post-exercise blood lactate sample was taken at 2 and 20 min. Subjects were instructed not to perform any exercise before their visit to the laboratory and 24 hours between measurements.

Statistics

Values are expressed as the mean ± standard deviation (SD). The one-way ANOVA for repeated measurements was used to test the statistical differences within the group (pre- *vs.* post-exercise). When the ANOVA was significant, Tukey's post-hoc test was applied to locate the difference. P values of the post-hoc analysis were adjusted for multiple comparisons and presented at two different levels: <0.05 or <0.01.

Results

After sprint cycling for 30 s, there was a significant decrease in the cycling peak power expressed in relation to body weight 9.97±0.84 $W \cdot kg^{-1}$ from start to 6.34±0.49 $W \cdot kg^{-1}$ at the end of exercise. Following 2 min after the exercise, lactate concentration was 6.67±0.70 mmol/L, but after 30 min and 60 min it had recovered to 3.69±0.60 mmol/L and 2.3±0.50 mmol/L, respectively. This indicates significant ($P<0.01$) differences in all the cases, comparing with the lactate concentration before the exercise (0.83±0.20 mmol/L).

Initial values of contractile properties are presented in Table. We found that the force evoked by electrical stimulation was muscle length dependent. The force-frequency curve was steeper at SL compared to LL, since P15/P50 was significantly smaller ($P<0.01$).

The time course of changes in P1, P15, P50, and MVC is shown in Fig. 1 a–d. The results show that there is a bimodal recovery in muscle force during the 24 hours after sprint cycling for 30 s: 1) fast recovery during the first 5 min while muscle force rapidly recovered at SL, (post-hoc test Ini *vs.* 5 min: P1, P15, P50, all N.S.) except MVC (Ini *vs.* 5 min: MVC, $P<0.05$), whereas at LL the P15, P50 and MVC did not recover (Ini *vs.* 5 min: P15, P50, MVC all $P<0.05$); 2) secondary decrease in muscle force at low- and high-frequency stimulations from 5 min to 30–60 min was more marked at low-frequency stimulation and at SL (Ini *vs.* 30, 60 min: P1, P15, all $P<0.01$; P50, $P<0.05$), also at LL (Ini *vs.* 30, 60 min: P1, $P<0.05$; P15, $P<0.01$; P50, $P<0.05$), but the MVC force recovered to initial within 30 minutes (Ini *vs.* 30 min, N.S.) 3) long-lasting recovery to 24 hours where fatigue after sprint cycling for 30 s still present at P15 (Ini *vs.* 24 h: P15, $P<0.05$) and the recovery in this case was not dependent on muscle length. The following three recovery phases specific to the stimu-

Table. **Initial values (mean±SD) for parameters of electrical stimulation-induced contractions of the quadriceps muscle and MVC, (n=12)**

Muscle length	P1, N	P15, N	P50, N	MVC, N	P15/P50
LL	44.1±11.4	164.4±38.2	233.8±41.2	577.8±89.5	0.71±0.11
SL	29.2±9.1	134.7±48.2	346.8±63.1	543.1±94.5	0.38±0.11
P	<0.01	N.S.	<0.01	N.S.	<0.01

P1, P15, and P50 – muscle contraction force evoked by stimulating the quadriceps muscle at a rate of 1 Hz, 15 Hz, and 50 Hz, respectively. MVC – maximal voluntary contraction force; N – newton; SL – short muscle length (knee-joint angle at 135°); LL – long muscle length (knee-joint angle at 90°); N.S. – not significant.

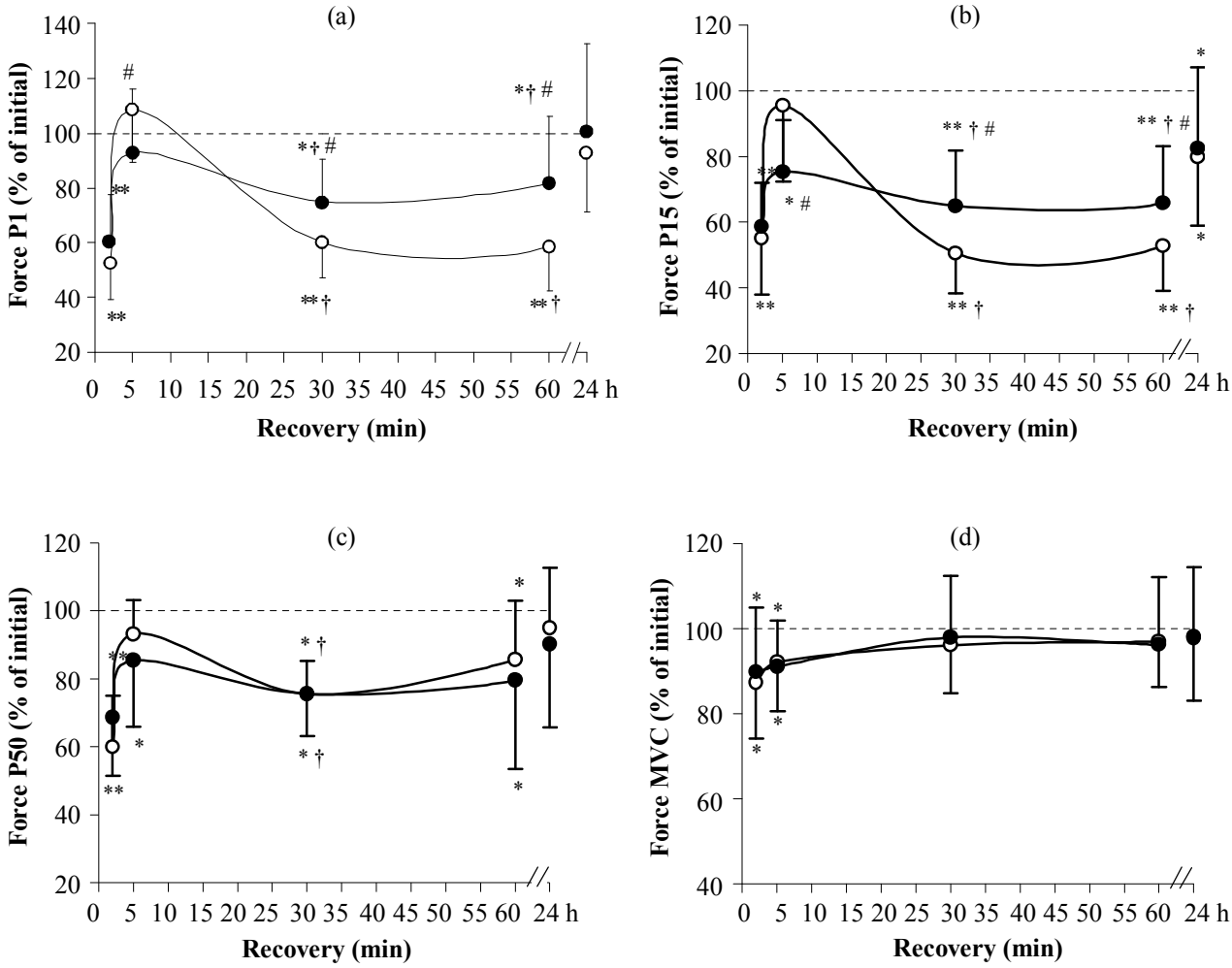

Fig. 1. **The time-course of changes in P1 (a), P15 (b), P50 (c), and MVC (d) after sprint cycling for 30 s at short (○) and long (●) muscle length (mean±SD)**

The dashed line indicates the mean initial level (100%). P1, P15, and P50 – muscle contraction force at a stimulation rate of 1 Hz, 15 Hz, and 50 Hz, respectively. MVC – maximal voluntary contraction force. Statistically significant differences as compared to initial are indicated as:
* P<0.05, ** P<0.01; † – significant difference with respect to the value at 5 min of recovery;
# – significant difference between short and long muscle length.

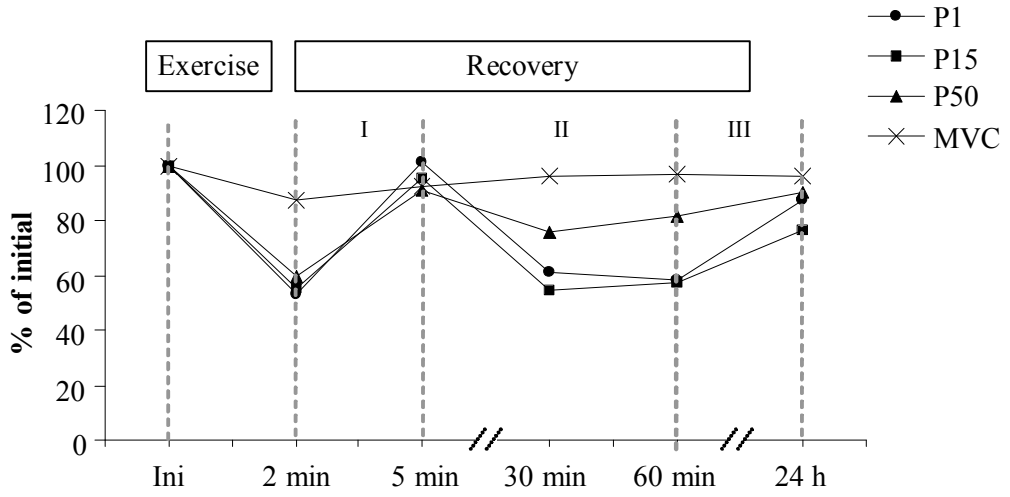

Fig. 2. **The time-course of changes in muscle force evoked by electrical stimulation at frequencies of 1 Hz (P1), 15 Hz (P15), 50 Hz (P50) and maximal voluntary force (MVC) at short muscle length after sprint cycling for 30 s**

Records show that the following three phases are observed: 1) fast recovery phase (I) within 5 min after the exercise; 2) secondary phase of force decrease (II) from 5 min to 30–60 min (not in the case of MVC); and 3) slow recovery phase (III) within 24 h.

lated contractions and recorded at SL are indicated in Fig. 2.

The recovery of force induced by low-frequency stimulation (1–15 Hz) was muscle length dependent because P1 and P15 recorded at SL during recovery period from 5 min to 30–60 min decreased significantly more than recorded at LL (Fig. 1a, b).

A comparison of MVC before, 2, and 5 min after sprint cycling has shown that there was a significant decrease in MVC force (post-hoc test Ini *vs.* 2, 5 min: MVC, $P<0.05$) at both SL and LL (Fig. 1d). Within 30 min after exercise and later, MVC force had recovered to 96–98% of its initial level.

Time-course of P15/P50 during muscle recovery period was dependent on muscle length (Fig. 3). The ratio of P15/P50 recorded at LL, however, decreased significantly at 2 min and remained stable to 60 min (Ini *vs.* 2, 5, 30, 60 min, all $P<0.05$), while the ratio of P15/P50 recorded at SL decreased 30 min after the exercise and remained unchanged up to 60 min (Ini *vs.* 30, 60 min: $P<0.01$). Data analysis showed that the ratio of P15/P50 recorded at SL was significantly lower at 30 min and 60 min than that at LL (SL<LL, $P<0.05$). No significant changes in the ratio of P15/

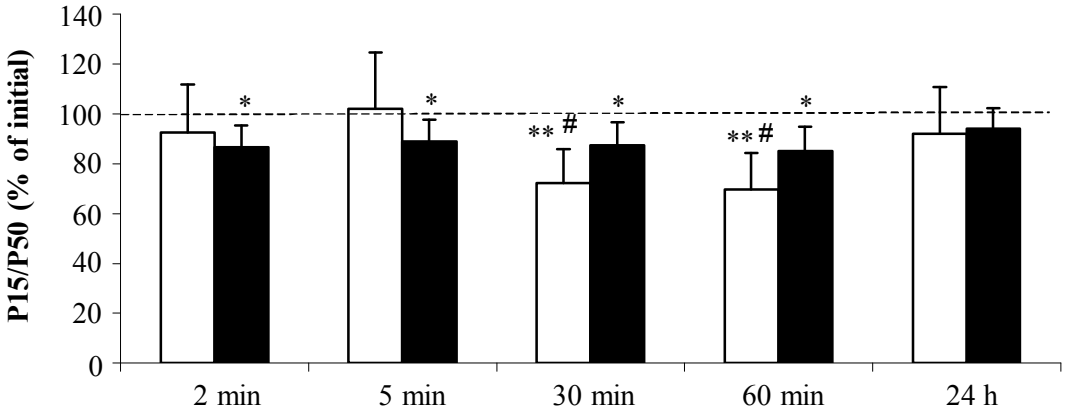

Fig. 3. **The changes in P15/P50 ratio after sprint cycling for 30 s at short (open bars) and long (solid bars) muscle length (means±SD)**

The dashed line indicates the mean initial level (100%). P15 and P50 – muscle contraction force evoked by stimulating the quadriceps muscle at a rate of 15 Hz and 50 Hz, respectively. Statistically significant differences as compared to initial are indicated as: * $P<0.05$, ** $P<0.01$; # – significant difference between short and long muscle length.

P50 recorded at SL and LL were present 24 h after the exercise (Ini *vs.* 24 h: SL and LL, N.S.).

Within 24 h after the exercise, the subjects experienced low muscle pain of 0.34±0.21 points.

Discussion

The main finding of the present study is a bimodal recovery of the stimulated contractions force within 24 h after sprint cycling for 30 s: 1) in early, fast-recovery phase (within the first 5 min) muscle force evoked by electrical stimulation was restored after performing exercise at SL, conversely at LL (Ini *vs.* 5 min: P15 and P50, both $P<0.05$), whereas MVC was still suppressed at both muscle lengths; 2) in the second phase (from 5 min to 30–60 min) muscle force decreased at low- and high- frequency stimulations and was more expressed at low-frequency stimulation and at SL than that at LL, but the MVC recovered to initial by the 30 minute; 3) in long-lasting phase (within 24 h), P15 force was still suppressed at both muscle lengths.

Potentiation

A prominent feature of the PT phenomenon is an increased twitch and low-frequency force accompanied by a shortening of contraction and relaxation time (6, 9). It has been suggested that PT is caused by phosphorylation of myosin regulatory light chains (27). J. M. Metzger *et al.* proposed that phosphorylation of myosin regulatory light chains induces retraction of myosin head from the backbone of thick filament and thus shortens the distance to the actin filament which in turn could enhance cross-bridge attachment rate (28). The importance of spatial proximity between actin and myosin is supported by the observation that PT is more pronounced at a short muscle length when distance between filaments is plausibly greater. A significant increase in twitch force was observed in the present study (Fig. 1a). The finding that 5 min after the end of sprint cycling for 30 s, P1 was completely recovered indicates that one has to do not only with the fatigue caused by metabolic changes in the muscle, but also with muscular potentiation favoring the process of recovery. The faster recovery of P15 (Fig. 1b) (during the first 5 min after the exercise) at SL compared with LL might also be associated with potentiation since it has been established that potentiation is more pronounced at SL than at LL (8).

Yet, we hypothesize that mechanisms underlying PT were engaged causing slightly greater low-frequency forces and a greater P15/P50 ratio (especially at SL) at 5 min after exercise, but complete expression of the PT was counteracted by the MF and NMF.

Metabolic fatigue

The muscle fatigue arising after sprint cycling for 30 s is dependent on metabolic factor because lactate concentration increased markedly. It has been shown that after sprint cycling for 30 s a considerable reduction in ATP (50%), phosphocreatine (83%), and glycogen (35%) levels was found in type II muscle fibers (24). Therefore, the rapid recovery of muscle contraction force within 5 min after the exercise

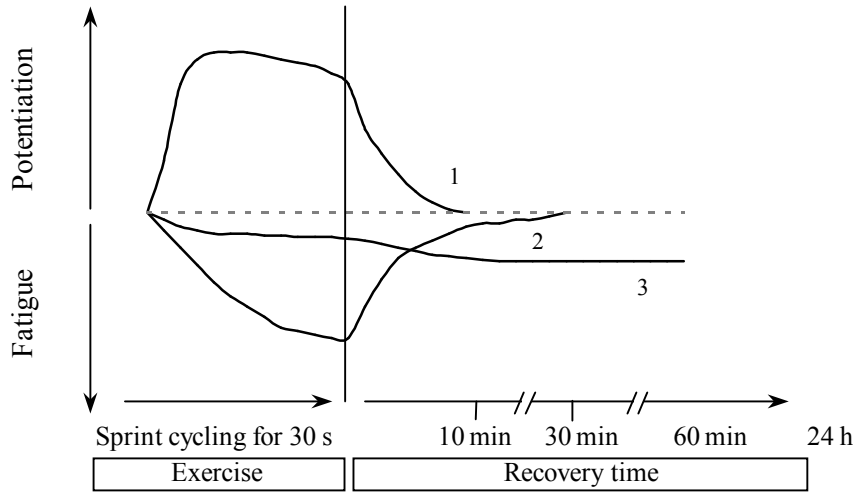

Fig. 4. **Hypothetical model of muscle performance during and after short-term maximal cycling for 30 s**

1 – muscle potentiation-dependent component; 2 – metabolite-dependent component; and 3 – non–metabolite-dependent component.

observed in our study could also be dependent on fast recovery of ATP and phosphocreatine levels.

A prolonged intense exercise increases the concentration of hydrogen ions (29) suggesting their possible association with muscular fatigue. A number of studies support the suppressive influence of hydrogen ions on muscle contractility (12, 16, 30). Doubts regarding their importance were raised by the fact that the influence on contractility diminished as the temperature of experimental conditions was approaching the physiological temperatures (31). The most recent findings indicate that in fact intracellular acidosis preserves excitability of muscle fibers, thus acting as a fatigue-resisting rather that causing mechanism (2).

Nonmetabolic fatigue

The concentration of metabolites, *i.e.*, ADP, P_i, Cr, recovers within 5–10 min after the sprint cycling for 30 s (32); therefore, decrease in force at 30 min after exercise has to be attributed to NMF. To explain long-lasting NMF: reduced Ca^{2+} release from sarcoplasmic reticulum is due to impaired excitation-contraction coupling (ECC) (15, 18). In support of the impaired ECC theory, the *in vivo* experiments indicated that ingestion of caffeine ameliorated symptoms of LFF in humans (33). The studies on single fibers also showed that fatigue caused by reduced free Ca^{2+} concentration can be overcome by application of caffeine (15), which facilitates release of Ca^{2+} from SR.

The decrease in P15/P50 was an indication of the LFF following sprint cycling for 30 s (Fig. 3). Although the effect of NMF became obvious only in the later phase of recovery, a decrease in P15/P50 at LL following the sprint cycling for 30 s indicates that the onset of it occurs as early as 2 min after the exercise.

Previous researches have typically examined LFF at 10, 30, and/or 60 min of recovery (15, 18), and the changes in the responses to low-frequency stimulation between 2 and 30–60 min have not been detected as it is seen in our study (Fig. 3), although a similar decrease and subsequent increase in LFF have been previously reported (19). Within 7 to 15 min after the exercise, the metabolic component of muscle fatigue may have fully recovered (14), but the $[Ca^{2+}]_i$-time-integral-dependent component may be active enough to counteract recovery of the metabolism-dependent component (18). This Ca^{2+}-dependent long-lasting component of LFF may have a longer onset time than the metabolic component, which would explain the rapid initial decrease at 5 min of recovery and a subsequent increase at 30–60 min of recovery in LFF, especially at SL (Fig. 3).

Coexistence of MF, PT, and NMF

We hypothesize that time-course of muscle recovery within 24 h after sprint cycling for 30 s is dependent on PT, MF, and NMF (Fig. 4). Our findings are in accordance with the data of previous researches (7, 9, 19), where it has been noted the coexistence of PT and fatigue and that the interaction of PT and fatigue during voluntary activity is complex. The bimodal recovery of muscle function might be explained by a coexistence of PT, MF, and NMF as found in the present study (Fig. 2). Depression of force 2 min after sprint cycling for 30 s is caused by MF and NMF, the former being a major factor. Rapid recovery of contractile properties during the first 5 min is brought about by fading MF and still present traces of the PT, which compensated for the effect of NMF. The subsequent (5 to 30–60 min) decline in low- and high-frequency stimulations is an outcome of diminishing influence of PT on the background of persistent NMF.

Conclusion

A bimodal recovery of contractility of the quadriceps following sprint cycling for 30 s is determined by the concomitant complex interaction of mechanisms enhancing (potentiation) and suppressing (metabolic and non metabolic fatigue) contractile potential of the muscle, also depends on muscle length and activation mode. This has to be taken into account when the function of skeletal muscle is being assessed after dynamic exercise performed with maximal intensity.

Keturgalvio šlaunies raumens jėgos bimodalinis atsigavimas per 24 valandas po 30 sekundžių sprinto veloergometru

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Raktažodžiai: bimodalinis atsigavimas, elektrostimuliacija, mažų dažnių nuovargis, potenciacija, veloergometrija.

Santrauka. *Tyrimo tikslas.* Nustatyti ir paaiškinti potenciacijos ir nuovargio sąveiką atsigavimo metu po 30 sekundžių veloergometrinio krūvio esant skirtingam raumens ilgiui.

Metodai. Tirta 12 aktyviai nesportuojančių sveikų vyrų (23,6±1,7 metų). Keturgalvio šlaunies raumens jėga registruota prieš tyrimą ir praėjus 2, 5, 30, 60 min. bei 24 val. po 30 sekundžių sprinto veloergometru. Raumens susitraukimo jėga sukelta elektros stimulais (1, 15, 50 Hz dažniu). Maksimali valingoji jėga buvo registruota esant mažam ir dideliam raumens ilgiui.

Rezultatai. 1. Greitosios raumens atsigavimo fazės metu (per pirmąsias 5 min.) atsigavo tik elektros stimuliacijos sukelta jėga esant mažam raumens ilgiui (išskyrus 1 Hz jėgą, kuri atsigavo nepriklausomai nuo raumens ilgio). 2. Antrinės elektros stimuliacijos sukeltos jėgos sumažėjimo metu (nuo 5 iki 30–60 min.) labiau sumažėjo mažų (1–15 Hz) nei didelių (50 Hz) stimuliavimo dažnių jėga, ypač esant mažam raumens ilgiui, o maksimali valingoji jėga visiškai atsigavo. 3. Lėtosios raumens atsigavimo fazės metu (iki 24 val.) mažų (15 Hz) stimuliavimo dažnių sukelta jėga išliko sumažėjusi nepriklausomai nuo raumens ilgio.

Išvada. Keturgalvio šlaunies raumens jėgos bimodalinis atsigavimas po 30 sekundžių maksimalaus dinaminio krūvio yra sąlygotas vienu metu vykstančių procesų, t. y. aktyvinančių (potenciacija) ir slopinančių (metabolinis ir nemetabolinis nuovargis) raumens susitraukimo jėgą.

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