Neuromuscular fatigue after maximal stretch-shortening cycle exercise

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Strojnič, V., and P. V. Komi. Neuromuscular fatigue after maximal stretch-shortening cycle exercise. J. Appl. Physiol. 84(1): 344–350, 1998.—To examine some possible sites of fatigue during short-lasting maximally intensive stretch-shortening cycle exercise, drop jumps on an inclined sledge apparatus were analyzed. Twelve healthy volunteers performed jumps until they were unable to maintain jumping height >90% of their maximum. After the workout, the increases in the blood lactate concentration and serum creatine kinase activation were statistically significant (P < 0.001 and P < 0.05, respectively) but rather small in physiological terms. The major changes after the workout were as follows: the single twitch was characterized by smaller peak torque (P < 0.05) and shorter time to peak (P < 0.05) and half-relaxation time (P < 0.01). The double-twitch torque remained at the same level (P > 0.05), but with a steeper maximal slope of torque rise (P < 0.05); during 20- and 100-Hz stimulation the torque declined (both P < 0.01) but with a smaller maximal slope of torque rise (P < 0.01) and a higher activation level (P < 0.05), accompanied by an increased electromyogram amplitude. These findings indicate that the muscle response after the short-lasting consecutive maximum jumps on the sledge apparatus may involve two distinct mechanisms acting in opposite directions: 1) The contractile mechanism seems to be potentiated through a transient and faster cross-bridge cycling, as implied by twitch changes. 2) High-frequency action potential propagation shows an impairment, which is suggested as the possible dominant reason for fatigue in exercise of this type. Fatigue sites; maximal intensity; short duration; electrical stimulation.

Fatigue is a very complex phenomenon, and it may be described as a loss of force-generating capability (4) or an inability to sustain further exercise at the required level (7). During sustained isometric maximum voluntary contraction (MVC), force declines progressively. Fatigue processes can develop in many places during the activation-contraction chain (2). They can be found in an insufficient central neural drive, i.e., central fatigue, or in events beyond the neuromuscular junction, i.e., peripheral fatigue. After the activation-contraction chain, peripheral fatigue can be further divided into high- and low-frequency fatigue (7). High-frequency fatigue occurs as a result of an impairment in action potential propagation over the sarcolemma; low-frequency fatigue denotes an impairment in excitation-contraction coupling.

Stretch-shortening cycle (SSC) is defined as a stretching of an active muscle immediately followed by concentric contraction (17). Fatigue during submaximal SSC is characterized by reduced movement efficiency (11) as well as a dramatic decline in maximal isometric force (15). High peak lactate values also appear after this type of exercise. The peak of serum creatine kinase (CK) accumulation is delayed, occurring on the 2nd day after exercise (22). The fatigue during an SSC exercise may be even more complex, since neural control, besides central activation, depends on reflexively induced activation. Reduced muscle activation in the eccentric phase (10) and a smaller monosynaptic response to a sudden stretch of the relaxed soleus (Sol) muscle (23) have been obtained in submaximal SSC fatigue exercise.

Most fatigue studies in SSC exercise have concentrated on submaximal workout intensity levels (17, 18, 22). However, the fatigue mechanisms in maximum SSC exercise may differ from the mechanisms at the submaximal level, since exercise at higher intensity involves greater reaction forces, greater muscle activation, and shorter exercise duration. Because of these factors, more muscle stress and lower blood lactate may be expected at maximal-intensity exercise. According to our previous comparison of maximal and submaximal concentric workouts (25), a shift from low- to high-frequency fatigue may be expected as well. The aim of the present study was to examine some possible fatigue events during short-lasting maximally intensive SSC exercise. Contractile characteristics of the muscle, i.e., force during low- and high-frequency stimulation, activation level, and blood analysis, were measured and used in an attempt to clarify further the possible fatigue mechanisms.

Methods

Subjects. Twelve healthy men (physical education students) volunteered for the study (age = 28.1 ± 5.8 yr; height = 179.8 ± 5.3 cm, body mass = 78.9 ± 12.3 kg). They were not involved in intensive sport training. Subjects were well informed about possible risks associated with the experiment and gave their informed consent before the experiment. The study was approved by the University Ethical Committee.

Experimental design. Subjects performed a warm-up, which consisted of 6 min of stepping on a 20-cm-high bench with a frequency of 0.5 Hz, with a leg exchange each minute. After the warm-up, tests for the assessment of the initial status were performed in the following sequence: blood sample, response of the relaxed vastus lateralis (VL) muscle to single electrical impulse (twitch), response of the relaxed quadriceps femoris muscle to double electrical impulse (double twitch), response of the VL muscle to electrical stimulation (ES) at 20 and 100 Hz, explosive isometric maximum voluntary knee extension, and steady maximum voluntary extension with superimposed double twitch. All these measurements were performed at predefined times after the end of the warm-up. Then, the maximum jumping height was determined on the sledge ergometer, which was used for the fatigue exercise. The same test sequence was used after fatigue; the last test ended 4 min after the workout. Additionally, blood was collected 4 min after the workout. After the experiment, the subjects were allowed to rest for 2 days before the measurements were repeated.
collected 5 min, 2 h, and 2 days (at the same time of day and after the same warm-up) after fatigue was induced. Surface electromyogram (EMG) activity during some pre- and post-fatigue tests was recorded from the tibialis anterior (TA), Sol, gastrocnemius medialis, VL, and vastus medialis (VM) muscles by bipolar silver chloride miniature skin electrodes (Beckman) with an interelectrode distance of 20 mm and transmitted telemetrically (model 2000, Glonner Biomes). The electrodes were placed longitudinally on the belly of the observed muscles. Care was taken to ensure that the interelectrode resistance did not exceed 5 kΩ. All signals were digitized with a sampling frequency of 1 kHz and fed to a computer for further processing. They were also stored simultaneously on a magnetic tape recorder (Racal).

Fatigue workout. The fatigue workout was performed on a special sledge gliding apparatus (Fig. 1), which consisted of a 33-kg sledge gliding on a track inclined at 23° from the horizontal. The apparatus has been described in more detail elsewhere (18). The maximum jumping height was measured by releasing the sledge from 80 cm above the position in which subjects’ legs were maximally extended (zero position). Once the maximum jumping height was established, 90% of the distance between the zero position and the maximum height was marked as the limit for the fatigue workout termination. Subjects were asked to perform consecutive maximum jumps (in an “all-out” manner) without pause, until told to stop. When they were no longer able to jump to 90% of their preset maximum height, the workout was terminated. During jumping, the subjects maintained a 90° knee angle in the lowest sledge position. Oral feedback was provided to secure correct angular position. To prevent trunk movements, the subjects were secured to the sledge. Their arms were placed on the seat sides next to the thighs and were not moved during jumping. Verbal encouragement was used to motivate subjects for maximum output throughout the workout. The mean duration of the fatigue workout was 62.4 ± 24.8 s, during which the subjects were able to execute 36.5 ± 16 rebound jumps.

Electrical stimulation. In all measurements with ES and during explosive voluntary knee extension, the subjects sat in an isometric knee extension-measuring device (adapted model 200, David) and were fixed to the apparatus at the pelvis and over the distal part of the thigh to prevent trunk and thigh movements. The distal part of the shank was fixed to the force transducer, which had a constant lever arm to the knee joint axis. The knee joint angle was fixed at 45°. Self-adhering neurostimulation electrodes (5 × 5 cm; Axelgaard, Fallbrook, CA) were placed over the VL, VM, and rectus femoris muscles. Distal electrodes were placed over the distal part of the muscle belly, and proximal electrodes were placed over the middle part of the muscle belly. Each pair of electrodes was galvanically separated from the other pairs. On all occasions, constant-current square biphasic impulses of 0.3-ms duration were employed, and a custom-made computer-controlled electrical stimulator was used.

Stretch reflex test. The stretch reflex was measured with a special ankle dynamometer (23), which could induce different angular displacements at constant angular velocities around the ankle joint. The subject sat relaxed in the chair; his right foot was placed and fixed to the footplate with the ankle angle at 90°. Thus the rotational axis of the platform and the ankle joint were aligned. The knee was fixed at 60° to prevent knee and shank movements. Eight consecutive dorsiflexions (amplitude = 6°, velocity = 160°/s) were applied. The interval between two flexions was 5 s. EMG activity from the Sol, gastrocnemius medialis, and TA muscles and the positional data from the ankle ergometer were collected. Only those EMG records without TA activation and no EMG signal in the Sol before the stretch were analyzed. EMG records were high-pass filtered (cutoff frequency = 10 Hz), rectified, and averaged. The latency, duration, and area of EMG responses were examined. EMG cross talk was minimal. A method described earlier (23) was employed for evaluation.

Single twitch test. Three supramaximal stimuli were delivered consecutively with 1-s delay to the relaxed VL muscle. The torque signals from the twitch responses were smoothed (moving average, n = 5) and averaged. Maximum twitch torque (T_TW), electromechanical delay (EMD), contraction time (CT), half relaxation time (RTH), and maximum rate of torque development (MTR_TW) were obtained.

Double twitch test. Two consecutive supramaximal stimuli with 10-ms delay were delivered to the relaxed quadriceps femoris muscle via all three electrode pairs simultaneously. The torque signal of the double-twitch response was smoothed (moving average, n = 5) and analyzed for maximum torque (T_DTW) and maximum torque development (MTR DTW). Low- and high-frequency torque tests. The relaxed VL muscle was stimulated with two consecutive trains of impulses with a frequency of 20 Hz (1-s duration) and 100 Hz (0.8-s duration; Fig. 2). Stimulation amplitude was set to three times that of the motor threshold amplitude (30–54 mA) and was kept the same for both frequencies. The mean torque during the last 50 ms of stimulation for each frequency (F20 and F100) was obtained.

Voluntary explosive knee extension test. Each subject was placed in turn on the same chair under the same conditions as
for the ES measurements. On command, the subjects tried to achieve their maximum isometric knee extension torque as quickly as possible. They started from a relaxed position, without preparatory movements, and maintained the maximum activation for 2 s. The force signal was smoothed (moving average, n = 5) and analyzed for maximum torque (TMAXC) and maximum rate of torque development (MTRMAXC).

Activation level. The activation level (AL) was assessed by a superimposed double-twitch technique derived from the twitch interpolation method described by Merton (19) (Fig. 3). TDTW was compared with the torque rise during superimposed double twitch over maximum voluntary knee extension. At the command signal, the subjects started to exert force. Two seconds after the start of voluntary contraction, when the maximum torque (Tmax) was attained, the subjects received two stimuli in the same way as in the double-twitch test. The difference (D) between the torque level just before the double twitch (Tb) and the maximum torque during the double twitch was compared with TDTW as follows

\[ AL = 100 - D \times (T_b/T_{max})/T_{DTW} \times 100 \]  

A correction of D was included in the original equation, since the double twitch was not always applied during maximum torque level, but also later when the torque was already slightly declining. Because D tends to grow at torques lower than maximal and because this relationship was shown to be linear (19), the expression T_b/T_{max} can be used to correct D.

EMG activity during isometric MVC. Both EMG signals were rectified and integrated. Time was normalized (EMG1VM and EMG2VM) for the steady part of the maximum knee extension at the activation level measurement, appearing before the superimposed double twitch during a 0.5-s interval. The EMG signal was also normalized to a mean torque established for the same interval (nEMG1VM and nEMG2VM), respectively.

Blood analysis. Blood samples for blood lactate concentration analysis (20 µl) were drawn from the fingertip, whereas the blood samples used to determine serum CK activation were taken from the antecubital vein of the right arm. Both samples were analyzed with a spectrophotometer (model U-2000, Hitachi) and reagents from Boehringer (Mannheim, Germany).

Statistics. The t-test for paired samples was used to calculate the statistical significance of differences between the initial measurement and the state after workout. Additionally, Pearson correlation coefficients were calculated between relative changes in parameters after workout according to the initial state. Statistical significance was accepted at P < 0.05 (2-tailed).

RESULTS

Responses to single and double supramaximal stimulus had some common features (Fig. 4). In the single-twitch test the mean maximum torque dropped from an initial 12.7 ± 4.4 to 11.6 ± 4.6 (SD) N·m after the fatigue test (P < 0.05). EMD showed no statistically significant change, whereas CT as well as RTs became shorter: from 68.7 ± 10.5 to 60.1 ± 8.4 ms (P < 0.05) and from 67.8 ± 7.0 to 56.1 ± 12.9 ms (P < 0.01), respectively. In contrast, the mean double-twitch maximum torque did not change significantly (from 55.5 ± 10.6 to 57.5 ± 13.2 N·m), although the mean peak rate of torque development was greater after the exercise (from 1,470 ± 300 to 1,672 ± 412 N·m·s⁻¹, P < 0.05). The peak rate of torque development increased also in the single twitch (6.7 ± 10.8%), although not significantly. When it was normalized to TTW, it became significantly greater (19.5 ± 12.1%, P < 0.001).

In the low- to high-frequency stimulation test, both torques (F20 and F100) were significantly reduced: from 15.3 ± 6.3 to 12.8 ± 6.4 N·m (P < 0.01) and from 26.0 ± 12.3 to 19.9 ± 9.6 N·m (P < 0.01), respectively. The correlation analysis between the blood lactate change and the F20 change revealed a significant positive correlation (r = 0.75, P < 0.01); the subjects with the smallest lactate change exhibited a greater F20 reduction after exercise.

In the isometric explosive MVC knee extension, the subjects demonstrated a significantly lower mean rate of torque development (from 2,163 ± 724 to 1,735 ± 576 N·m·s⁻¹, P < 0.01) but no significant changes in the
maximal torque level. Comparison in maximal torque and rate of torque development between the double twitch and the explosive MVC knee extension revealed no significant change in the mean ratio of the maximal torque levels, whereas the mean ratio of MTR_{MVC} to MTR_{DTW} decreased by 8.5 ± 5.4% (P < 0.001). Relative changes in other MVC and ES parameters are presented in Fig. 5.

During the sustained isometric MVC knee extension, the mean EMG amplitudes in VL and VM muscles demonstrated no significant change (Fig. 6). The changes in the mean MVC torque during sustained contraction were related to the changes in EMG amplitude of the VL muscle (r = 0.72, P < 0.01) but not to changes in EMG amplitude of the VM muscle. When the EMG amplitudes were normalized to torque, EMG appeared to be significantly greater (P < 0.001) in the VL muscle after exercise.

In the stretch reflex test, no significant mean change was observed after the workout in any of the analyzed parameters.

The blood sample analysis revealed a statistically significant increase in blood lactate concentration and CK (P < 0.001 and P < 0.05, respectively), with mean peak values after the workout of 5.4 ± 1.4 mmol/l and 359.4 ± 287.4 U/l, respectively, compared with initial values of 1.93 ± 0.3 mmol/l and 259.3 ± 206.0 U/l, respectively. The mean peak lactate level was observed 5 min after the workout and the mean peak CK level 2 h after the workout. These changes were much smaller than those obtained in our earlier studies (35) with longer duration of submaximal SSC exercises.

**DISCUSSION**

The main findings in this study were 1) a shorter single twitch with a smaller peak torque, 2) preserved peak torque and an even faster rate of torque development in the double twitch, 3) smaller low- and high-
frequency stimulation torque, 4) preserved maximal MVC torque with a slower rate of torque development, 5) no changes in the mean stretch reflex response, and 6) a significant but small increase in blood lactate concentration.

In fatigue the twitch tension decreased in human (3, 13) and frog muscles (9, 27). Recovery from twitch tension falls into two phases, the first of which is very rapid and takes ~2 min (27). In the present study the twitch was measured 2.5 min after the end of the fatigue protocol, which means that the twitch tension at the end of the workout might be lower than the twitch tension measured. Hainaut and Duchateau (13) reported 67% of the initial level after a protocol with ES, whereas after 60 s of MVC the twitch tension may decline almost to 70% (3). If a time delay is assumed, the twitch tension after the workout might well fall into that range in the present study.

Twitch times, on the contrary, showed completely different behavior. A normal response to fatigue is a prolongation of CT and RT of which may increase up to 300% and 800%, respectively (26). These changes seem to be smaller in human muscles (12-17% and 36-41%, respectively) (13). Shorter CT and RT obtained in the present study do not seem to agree well with the fatigue response, especially when it is considered that the maximum rate of torque development increased as well. Again, the recovery effect must be considered when our data are examined, even though possible changes would probably be quantitative rather than qualitative.

An impairment in the contractile mechanism seems to be the most direct reason for fatigue. An isometric twitch has frequently been used as an indicator of muscle fatigue, providing information about the number of active cross bridges. In the present study the peak torque response to a single stimulus declined after exercise, which is in accordance with other fatigue studies (9, 27). The reduced torque could be associated with a reduced Ca²⁺ release from the sarcoplasmatic reticulum and/or reduced capability of cross bridges to form strong binding (20). Shorter CT and RT suggest that the reduced T_TW may have resulted from a shorter Ca²⁺ transient. This view can be further supported by T_DTW, which remained unchanged after the exercise. Because of two consecutive stimuli, the Ca²⁺ transient was longer, thus making T_DTW less sensitive to changes in the Ca²⁺ transient. The small change in blood lactate concentration may indicate that the decline in T_TW was not accompanied by significant changes in H⁺ and/or PO4³⁻. Such an occurrence was also observed by Vøllestad et al. (28), although after different exercise. In addition, the peak rate in torque development in single- and double-twitch conditions increased, suggesting a higher rate of cross-bridge binding, as observed by Metzger and Moss (20). If a low pH inhibits the rate of cross-bridge binding (21), the increased slope of torque rise would suggest no physiologically significant change in pH. However, shorter CT and RT may also be due to increased muscle temperature because of exercise. The temperature effect on muscle contractile characteris-

![Fig. 7. Relationship between relative changes in lactate and torque during 20-Hz ES. For subjects with a very small change in lactate, decrease in F_20 was most pronounced. It is thus most unlikely that decrease in F_20 was due to acidosis.](http://jap.physiology.org/)

In the present study the torque during 20-Hz stimulation was lower after the exercise (rightward shift). Normally, a leftward shift would be expected (8). The torque decrease may be explained by a shorter Ca²⁺ transient and faster contraction, causing decreased fusion of tension of twitches at subtetanic stimulation frequency. The small change in CK, measured 2 h after exercise, does not support mechanical damage as a reason for the lower 20-Hz force (16) in the present study.

![Fig. 8. Responses of 1 subject to double stimuli, superimposed over MVC, before and after workout. Torques at instant of stimulus delivery were aligned (difference was 2% of MVC torque) and set to zero to enable better comparison. Torque rise, due to double stimuli, was much smaller after workout, denoting that less voluntarily unused muscle contractile capacity was left to be activated with ES. Mean activation level before workout was 75% and increased to 80% after workout.](http://jap.physiology.org/)
study. Despite the generally low lactate levels, it was possible to observe that the relative lactate change significantly discriminates the response in F20 (Fig. 7). The relationship is in the direction opposite that expected, since low-frequency fatigue normally develops during submaximal exercise. Such a differential response would additionally support the suggestion that a shorter Ca²⁺ transient and faster contraction may be the cause for a smaller F20, because the lowest F20 was exhibited by subjects with the smallest lactate response. Thus it does not seem very likely that the exercise-induced impairment in activation-contraction coupling could have occurred to a larger extent.

Decline in 100-Hz torque after the workout in the present study suggests impaired high-frequency action potential propagation over the sarcolemma. It has been suggested that high-intensity exercise may cause shifts of Na⁺ and K⁺ concentration in intracellular and extracellular fluids, resulting in membrane depolarization (24). Because of the concentration change, a depolarization block may occur, which may reduce action potential frequency considerably during high-frequency stimulation (1), finally resulting in lower muscle force.

Decline in maximum voluntary force has been considered one of the most important signs of fatigue (4). It has been demonstrated that the discharge frequency of motoneurons declines progressively during sustained MVC (3). Simultaneously, the relaxation rate of the muscle declines so that the lower firing frequency maintains the tetanus and thus optimizes maximum force production. Because of faster contraction and relaxation, as observed in single and double twitch, it would be difficult to maintain the same force with a lower firing frequency. No significant change in EMD additionally supports the idea that conduction velocity might not have been significantly reduced. That activation of additional motor units is what very likely happened in the present study may be supported by the integrated EMG (6) of VL and VM muscles and increased activation ratio, where the dominant factor for the higher activation level was the decreased superimposed double-twitch force rise over MVC force (Fig. 8). If new motor units were recruited voluntarily and a part of the motor units was inactive due to a contractile failure, then a smaller portion of the muscle contractile potential would remain to be activated by the superimposed double twitch, causing the activation level to increase.

Even though the force level in MVC was preserved, the rate of maximum torque development was significantly reduced. In an unfatigued condition, the firing frequencies are higher during force rise than during force maintenance (12). During fatigue, such a frequency can be efficiently reduced by a depolarization block (24). Inability to deliver action potentials with high frequency, together with faster contraction, may thus depress the rate of force development because of reduced fusion of tension of twitches.

It is possible to conclude that the short-lasting consecutive maximum jumps on the sledge apparatus induced a potentiation of the contractile mechanism, which could be characterized by a shorter Ca²⁺ transient and faster cross-bridge cycling as well as an impairment of high-frequency action potential propagation, which could be the dominant reason for fatigue.

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