Reduced reflex sensitivity persists several days after long-lasting stretch-shortening cycle exercise

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Avela, Janne, Heikki Kyröläinen, Paavo V. Komi, and Daniel Rama. Reduced reflex sensitivity persists several days after long-lasting stretch-shortening cycle exercise. *J. Appl. Physiol.* 86(4): 1292–1300, 1999.—The mechanisms related to the acute and delayed secondary impairment of the stretch reflex function were investigated after long-lasting stretch-shortening cycle exercise. The results demonstrated a clear deterioration in muscle function immediately after fatigue, which was accompanied by a clear reduction in active and passive reflex sensitivity. For active and passive stretch reflexes, this reduction was biphasic ($P < 0.05$ to $P < 0.001$). However, for the ratio of the electrically induced maximal Hoffmann reflex to the maximal mass compound action potential, only one significant reduction was seen immediately after fatigue (71.2%, $P < 0.01$). A similar significant ($P < 0.01$) decrease in the stretch-resisting force of the muscle was also detected. Clear increases were found in the indirect markers of muscle damage (serum creatine kinese activity and skeletal troponin I), which could imply the occurrence of ultrastructural muscle damage. It is suggested that the acute reduction in reflex sensitivity is of reflex origin and due to two active mechanisms, disfacilitation and presynaptic inhibition. However, the delayed second decline in the sensitivity of some reflex parameters may be attributable to the secondary injury, because of some inflammatory response to the muscle damage. This might emphasize the role of presynaptic inhibition via group III and IV muscle afferents.

neuromuscular fatigue; central fatigue; stretch reflex; electromyography; marathon running

SEVERAL STUDIES have demonstrated that prolonged stretch-shortening cycle (SSC) exercise results in acute reduction in performance, with an associated decrease in the neural input to the muscle (21, 30). These changes may occur concomitantly with reduced stretch reflex sensitivity (2). This is in line with the suggestion by Asmussen and Mazin (1) that the origin of the decline in the neural input to the muscle is the fatiguing muscle itself, through certain reflex pathways. As emphasized by Bigland-Ritchie et al. (3), this decline can be also advantageous because it helps to protect peripheral neuromuscular structures from excessive exhaustion by preventing the impulse frequencies from exceeding those needed for full tetanic activation of the fatiguing muscle fibers.

Two major hypotheses have been presented to explain the mechanism that might be responsible for the acute and also the delayed secondary (e.g., see Ref. 11) impairment of reflex sensitivity. The first of these impairments, promoted by Bigland-Ritchie et al. (3) and supported by Garland (13), relies on an increased inhibitory drive to the α-motoneuron pool, probably provided by metabolically induced activity in small myelinated and unmyelinated muscle afferents, such as those belonging to groups III and IV. These afferents are mostly polymodal and are thus sensitive to several parameters associated with either metabolic fatigue or muscle damage (19, 32). The other hypothesis depends on decreased facilitation of the α-motoneuron pool, in which a progressive withdrawal of spindle-mediated fusimotor support may also play a role (4, 25). In addition, Bongiovanni and Hagbarth (4) have suggested the possibility of direct fatigue effects on the intrafusal fibers.

We have also demonstrated a reduction in stretch reflex sensitivity with passively repeated ankle dorsiflexions (2a), the intensity and frequency of which simulated that of the marathon run. Because of these stretches, maximal voluntary contraction (MVC) of the triceps surae muscle declined by 22.9%, the ratio of the electrically induced maximal Hoffmann (H) reflex to the maximal mass compound action potential (M wave; H/M ratio) by 43.8%, and the stretch reflex peak-to-peak amplitude by 74.4%. These changes were associated with a reduction in the stretch-resisting force (16.5%) of the muscle. Thus, in the passive fatigue condition, the reduced reflex function could be a result of a weakened mechanical response of the muscle spindle to stretch, leading to disfacilitation of the α-motoneuron pool.

The purpose of the present study was to investigate further the mechanisms related to the acute and delayed secondary impairment of the stretch reflex function. In addition, special emphasis was placed on the testing of the possible interactions between reflex sensitivity and the compliance characteristics of the muscle-tendon complex.

METHODS

Subjects. A group of seven experienced triathlonists (6 men and 1 woman) volunteered for the experiment. The range of their best times in the marathon run varied from 2 h 45 min to 3 h 15 min. Averages for the subjects were as follows: age 28.7 (25–39) yr, height 181.9 (168–192) cm, and mass 82.0 (61–99) kg. All the subjects were fully informed of the procedures and the risks involved in this study and gave their informed consent. They were also allowed to withdraw from the measurements at will. The project was accepted by the ethics committee of the University of Jyväskylä.

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Experimental design. The experiment was designed as a before-and-after-marathon follow-up study (Fig. 1). The marathon run was selected as a way of inducing fatigue because it demands repeated, long-term SSC muscle actions (~10,000). The before-marathon tests took place 1 h before the actual marathon, and the after-marathon follow-up tests were performed immediately after, 2 h after, and 2, 4, and 6 days after the run. The marathon was run individually, and the target times were set according to each participant's best times to confirm high levels of motivation, performance, and, consequently, fatigue. The race was controlled by a cyclist who kept the predetermined speed as constant as possible.

Measurements. The testing protocol included measurements of isometric MVCs, maximal M waves and H reflexes, and stretch reflexes. In all the experimental conditions, the subject sat on an ergometer (29) chair, and both legs were measured separately. Depending on the testing condition, the thigh of the right or left leg was fixed and the foot was mounted on the rotation platform so that the rotation axes of the ankle joint and motor drive coincided. The initial ankle position was 90°, and the knee angle was set at 120°. In the MVC and stretch reflex tests, the torque around the rotational axis of the ergometer motor was measured by a piezoelectric crystal transducer (Kistler). In the final results the torque was divided by the moment arm, and, therefore, was expressed as force. The stretch reflexes were measured passively, and the stretching was applied by a motor torque device (Geisinger, 150 Nm) controlled by a digital feedback system. The skin temperature of the legs was measured during every testing unit. At the end of the marathon, a blood pressure cuff, which was wrapped around the middle portion of the right thigh of the subject, was inflated to at least 200 mmHg. The pressure was kept on until measurements were completed immediately after the marathon (~3 min). This procedure served to retain the possible metabolic fatigue substances in the fatigued muscles.

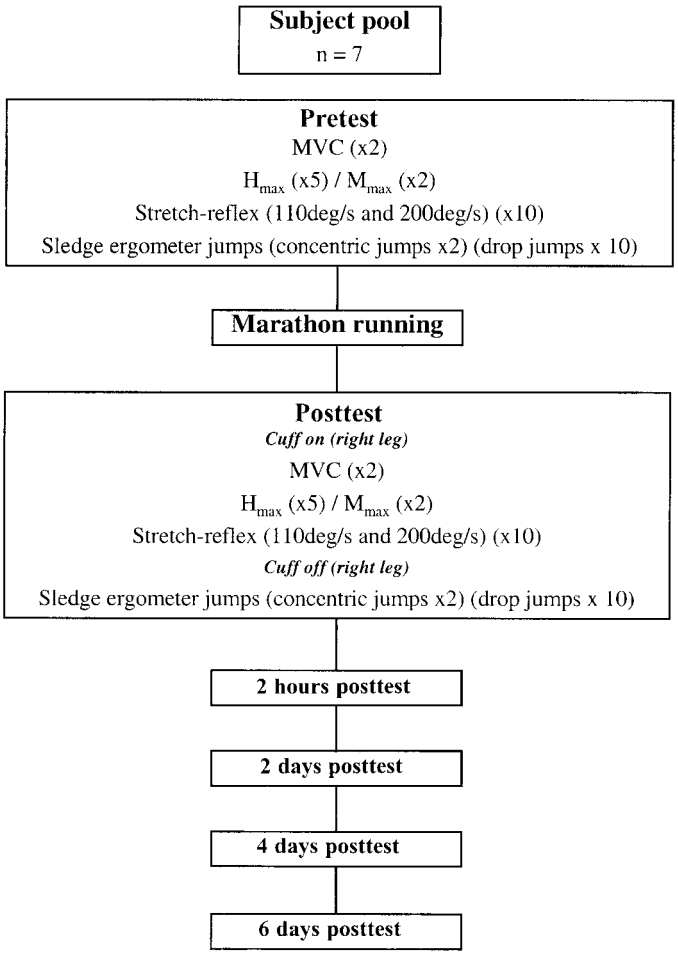

Fig. 1. Experimental protocol. *n*, No. of subjects. MVC, maximal voluntary contraction; H_{max}, maximum H reflex; M_{max}, maximum M wave.

The stretch reflexes of the plantar flexors were measured as a bout of 10 consecutive stretches at 2 different stretching velocities. The amplitude of the ankle joint dorsiflexions was 10° in both cases; the mean stretching velocities of the stretches were 110 and 200°/s. The frequency of all the stretches was 0.5 Hz, and their order was randomized.

In the recording of the H reflex, a standard methodology was adopted. After the skin was prepared, the stimulation electrodes (pregelified Ag-AgCl electrodes, Niko) were positioned bilaterally for H-reflex and M-wave testing. The H reflex was evoked by the electrical stimulation of the Ia afferent fibers of the tibial nerve. The M wave (muscle compound action potential) was evoked by the supramaximal stimulation of the motoneurons of the same nerve. For each leg, the cathode (1.5 × 1.5 cm) was placed over the tibial nerve in the popliteal fossa and the anode (5 × 8 cm) was placed superior to the patella. For H-reflex and M-wave testing, single rectangular pulses of 1-ms duration were delivered with random time delay (average 2 s) from an evoked-potential-measuring system (MEB-5304K, Nihon Kohden). The H reflex was also measured in the left leg, for which the ischemia was not induced during the immediate after-fatigue measurements. The reasoning for this protocol was to compare the changes in the H reflex between legs, to test the effect of the ischemia itself and the possible recovery during that time on the reflex sensitivity.

After the ankle ergometer measurements were made, the subjects moved to a sledge ergometer (21, 29), where they performed 10 maximal drop jumps from a predetermined optimal dropping height. In the sledge ergometer, the subjects sat on a sliding chair on rails inclined 21° to the floor. A force platform, placed perpendicularly to the sliding surface, served as a jumping stand. The optimal dropping height (best rebound height) of each subject was determined 1 wk before the actual marathon run on the sledge ergometer by using a short series of maximal single SSC repetitions, in which the subject was dropped from progressively increasing heights and instructed to rebound as high as possible. In the sledge ergometer, all the joints of the lower extremities are able to move freely, with the exception of the hip joint, the movement of which is restricted by the sitting position.

The recording electrodes for H reflexes, M waves, stretch reflexes, and the electromyograms (EMGs) associated with the MVC and sledge ergometer jumps were bipolar surface electrodes (Beckman 650437 miniature skin electrodes) fixed at a constant interelectrode distance of 20 mm. The electrodes were placed on both legs, ~6 cm above the superior aspect of the calcaneous on the soleus (Sol) muscle and between the center of the innervation zone and distal end of the lateral head of the gastrocnemius (GA) and vastus lateralis muscles. The ground electrodes (textile band dipped in a physiological saline solution) were placed around the shank between the stimulating and recording electrodes. The position of the recording electrodes was marked carefully on the skin. For this reason, EMG activity could be compared intraindividually (20, 35) between the pre- and postmarathon follow-up tests. On the basis of our earlier study (2a), the extent of EMG

cross talk between the measured muscles was considered negligible.

Analysis. All the EMG activity associated with voluntary contractions and stretch reflexes was transferred telemetrically, amplified by an FM μV amplifier (Glonner Electronic, Munich, Germany; 3- to 360-Hz bandwidth, 1-kHz sampling frequency), and finally transferred through an analog-to-digital converter to a microcomputer. From each stretch reflex test, 10 consecutive reflex responses were averaged and analyzed for peak-to-peak amplitudes and latencies in synchrony with resisting torques and angular displacements of the ankle joint. The passive stretch-resisting force of the muscle was measured from the stretch reflex tests and analyzed as an average plantar flexion force for the first 40 ms of the stretch, before the possible contribution of the stretch reflex responses.

MVC with corresponding force-time curves and EMGs was analyzed trial by trial as an average for a 500-ms window. The setting of the window was determined so that the maximal force onset occurred in the middle of this time interval. The maximal rate of force production was taken from the MVC force-time curve as the steepest rise of the curve.

The H-reflex and M-wave recording signals were amplified (10 Hz-1 kHz), stored and analyzed trial by trial, and averaged when necessary by the Nihon Kohden measuring system. Maximal H-reflex peak-to-peak amplitudes were expressed in relation to the maximal M-wave peak-to-peak amplitudes. Theoretically, it is expected that possible changes in peripheral excitability should affect both the H-reflex and M-wave amplitudes in the same way. Therefore, changes in the H/M ratio, so determined, should imply only the changes in α-motoneuron pool excitability.

In the sledge jumps, the full-wave-rectified EMG was recorded simultaneously with the corresponding force signal (1-kHz sampling frequency) and was phase integrated to preactivation, impact, and push-off EMG. The integrated EMG was then divided by the integration time and was taken to represent the average EMG (aEMG). To analyze the postlanding, short-latency reflex function (M_1) (7) from the SSC jumps, the rectified EMG signal was low-pass filtered at 75 Hz (Butterworth-type, 4th-order, 0-lag digital filter; see also Ref. 16). The M_1 reflex component was then identified according to the original definition of Lee and Tatton (24). The first rapid EMG burst usually appeared with ~30-ms latency after ground contact. Therefore, M_1 was quantified by analyzing the area under the curve from 30 to 50 ms after the onset of ground contact and was then expressed as M_1 aEMG. From the reaction force-time curve, it was easy to read off the impact peak force. This peak force falls rapidly and later turns into push-off force. The analysis included a parameter called peak force reduction (PFR), which was determined as the force difference between the impact peak force and the immediate lowest force level (14). M_1 and PFR are illustrated in Fig. 6.

Blood samples were drawn from the ulnar vein before each testing unit to determine possible markers of muscle damage [serum creatine kinase (CK) and skeletal troponin I (TnI)]. Furthermore, capillary blood samples from the fingertip were taken for blood lactate determination 5 min after the marathon run. Serum CK was analyzed by using a CK ultraviolet test kit (Boehringer Mannheim). Skeletal TnI was analyzed by using two immunoenzymometric assays. The first determines the specific human cardiac troponin concentration in serum. The second recognizes all troponin isoforms. Then, by exclusion of the cardiac troponin concentration, the skeletal TnI concentration is determined (31). Blood lactate were analyzed enzymatically by using a commercial kit (Biochemica Boeringer).

Statistical analysis. Descriptive statistical methods were implied to calculate mean and SD values for the various parameters in all the tests. Statistical significances for the different parameters between tests and between legs were determined by using double multivariate analysis of variance. When a significant *F*-ratio occurred for the main effects, profile analysis was carried out by multivariate analysis of variance to locate the source of the difference. Correlation coefficients were calculated to determine the relationships between selected parameters.

RESULTS

All the changes in the measured metabolic parameters are summarized in Table 1. As expected, blood lactate did not show any significant changes immediately after the marathon run. Serum CK activity at rest was 251 ± 115 U/l. Thus all the postmarathon CK activity follow-up values were significantly ($P < 0.05$–0.001) higher, peaking at 2 days after (1,458 ± 551 U/l), whereas skeletal TnI peaked earlier (2 h after). In addition, skeletal TnI showed almost complete recovery by 4 days after.

MVC and reflex responses. Multivariate analysis of variance revealed that the left and right legs showed identical changes. Consequently, in this section, only the results of the right leg are referred to. The only exception is the maximal H/M ratio, which also demonstrated that the left and right legs behaved similarly.

Changes in MVC are shown in Fig. 2. These results demonstrated a clear deterioration in muscle function immediately after the marathon. Maximal isometric force, maximal rate of force production, and aEMG of the Sol and GA muscles fell by 29.8 ± 10.9, 29.5 ± 27.1, 38.3 ± 19.9, and 28.3 ± 23.1%, respectively. Maximal isometric force and the simultaneously recorded EMGs in the Sol and GA muscles behaved very similarly during the marathon follow-up. The 2 h after values showed a significant recovery ($P < 0.05$), which continued until a full recovery in these parameters was already reached by 2 days after. However, the maximal rate of force production was still significantly low ($P < 0.05$) at 2 days after.

Table 1. *Changes in capillary blood lactate, serum creatine kinese activity, and skeletal troponin I*

Variable	Before	Immediately After	2 h After	2 Days After	4 Days After	6 Days After
Blood lactate, mmol/l	2.23 ± 0.31	2.66 ± 0.61		1.97 ± 0.43	1.57 ± 0.25†	1.69 ± 0.29*
CK activity, U/l	251 ± 115	848 ± 245‡	1,147 ± 520†	1,458 ± 551‡	718 ± 364†	440 ± 148*
Skeletal TnI, ng/ml	2.2 ± 3.5	138.3 ± 55.2‡	164.3 ± 74.8†	36.2 ± 19.5†	20.9 ± 19.7	9.4 ± 7.5

Values are means ± SD. CK, creatine kinase; TnI, troponin I. * Significantly different from before marathon: * $P < 0.05$, † $P < 0.01$, and ‡ $P < 0.001$.

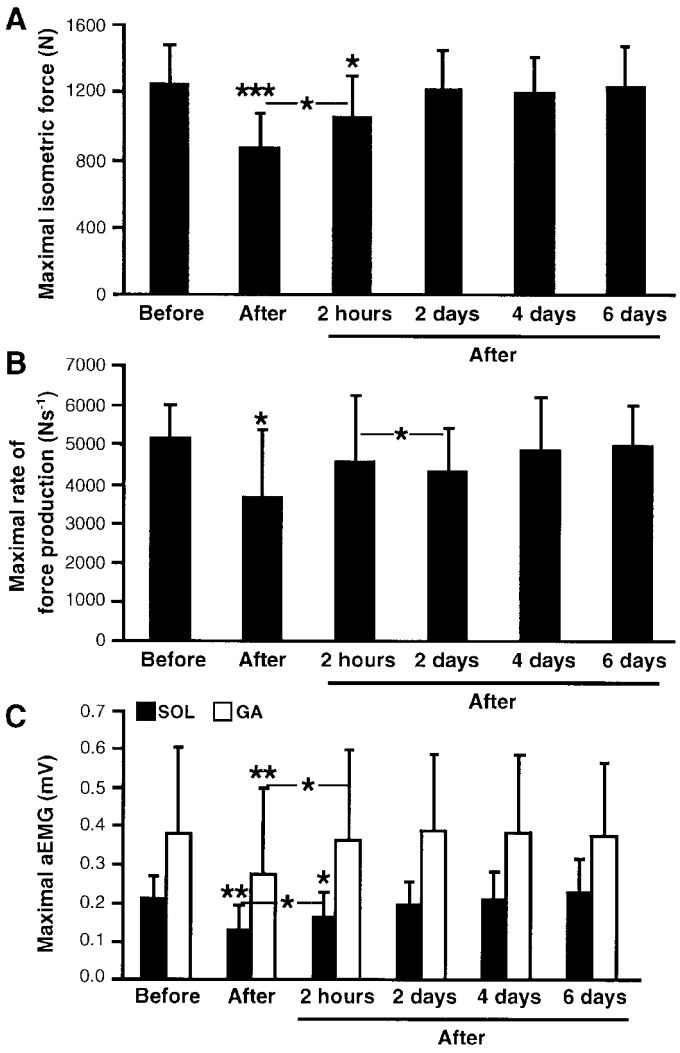

Fig. 2. Mean values (±SD) for all 7 subjects in MVC of plantar flexors, measured in ankle ergometer. *A*: maximal isometric force. *B*: maximal rate of force production. *C*: maximal averaged EMG (aEMG) of soleus (Sol) and gastrocnemius (GA) muscles. $*P < 0.05$, $**P < 0.01$, and $***P < 0.001$: statistically significant compared with before-marathon condition or another condition.

The recordings of the stretch reflexes showed a dramatic reduction in peak-to-peak amplitude ($P < 0.01$) immediately after the marathon run at both stretching velocities (Fig. 3). This reduction had recovered almost to the premarathon value at 2 h after. However, a second depression in reflex sensitivity could be seen at 2 days after. Surprisingly, a significant immediate postmarathon decrease in the stretch-resisting force took place in both stretch conditions: 12.4 ± 7.6 ($P < 0.01$) and 8.7 ± 6.3% ($P < 0.01$) for 110 and 200°/s, respectively. However, during the recovery period the stretch-resisting force exceeded the premarathon value. Immediately after the marathon run, the change in the stretch-resisting force correlated negatively ($P < 0.05$) with the change in serum CK activity (Fig. 4*A*). Interestingly, this correlation had turned positive (nonsignificant) by 2 h after the run (Fig. 4*B*).

Maximal H-reflex peak-to-peak amplitude declined immediately after the marathon run by 74.5 ± 16.3%

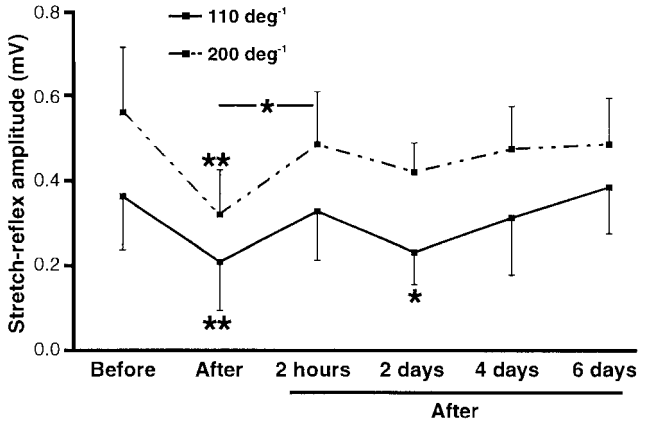

Fig. 3. Mean values (±SD) for 7 subjects of stretch reflex peak-to-peak amplitude for both stretching velocities, measured during stretch reflex tests. $*P < 0.05$ and $**P < 0.01$: statistically significant compared with the before-marathon condition or another condition.

($P < 0.01$). This reduction was associated with only a moderate decrement (from 7.9 ± 3.3 to 7.7 ± 2.3 mV, nonsignificant) in the maximal M-wave peak-to-peak amplitude. Therefore, changes in the maximal H reflex resulted in a depressed maximal H/M ratio (Fig. 5; mean decline 71.2 ± 7.3%). The follow-up measurements revealed almost a linear recovery pattern up to 4 days after, when the H/M ratio reached its premarathon value. The H/M ratio of the nonischemic left leg decreased immediately after fatigue by 68.2 ± 16.5%. The differences between the changes in the left and

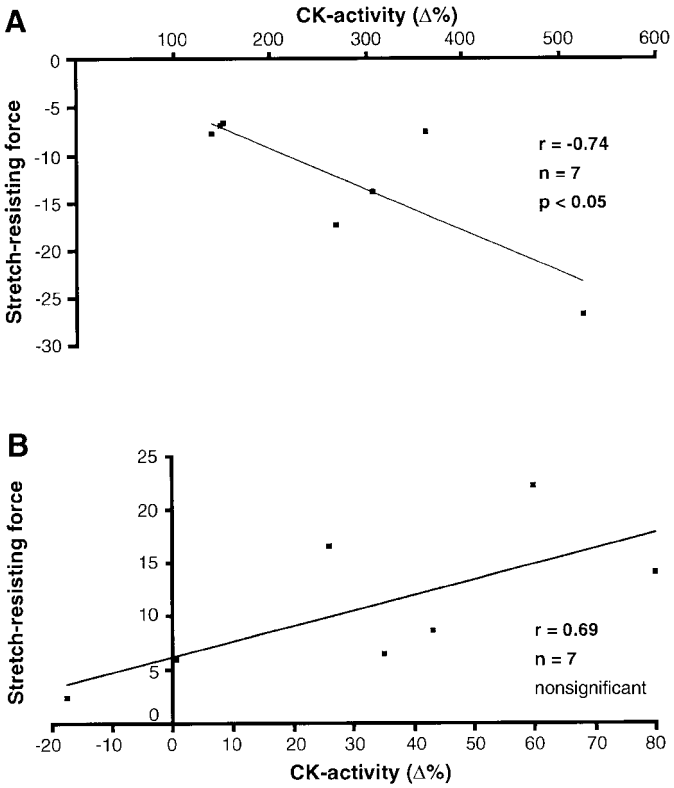

Fig. 4. Correlation between changes (Δ) in stretch-resisting force and serum creatine kinase (CK) activity immediately after (*A*) and 2 h after (*B*) marathon. *n*, No. of subjects. Stretching velocity was 110°/s.

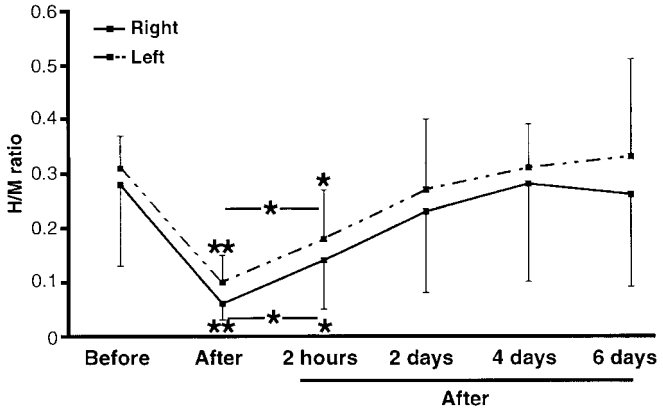

Fig. 5. Maximal ratio of electrically induced maximal Hoffmann reflex to maximal mass compound action potential (H/M ratio) for both legs. Values are means ± SD; $n = 7$ subjects. *$P < 0.05$ and **$P < 0.01$: statistically significant compared with before-marathon condition or another condition.

right leg were nonsignificant. Therefore, it can be suggested that no recovery occurred during this 3-min time period (ischemia time). In addition, this nonsignificant difference in changes between the legs suggests that the 3-min ischemia in the right leg itself during the immediate after-marathon measurements did not affect the results of reflex sensitivity.

SSC postlanding responses. The results of the parameters, which are closely related to the SSC performance capability, are summarized in Table 2 and clearly show a deterioration in muscle function immediately after the marathon run. In general, the most significant changes can be seen in the impact phase (impact time and average impact force), which did not seem to have recovered even after 6 days. On the other hand, the push-off phase showed significant reduction immediately after the marathon. In this situation, the reduced average push-off force ($P < 0.01$) and push-off time ($P < 0.05$) resulted in decreased take-off velocity ($P < 0.05$).

Figure 6 was constructed to reveal possible changes in the EMG patterns of the Sol and vastus lateralis muscles and the reaction force in the SSC sledge jumps before and immediately after the marathon. The shaded area represents the short-latency M_1 reflex component, which decreased significantly ($P < 0.05$–0.01) in both muscles immediately after the marathon run. It should be mentioned, however, that there was a similar reduction ($P < 0.01$) in the aEMG of the same muscles throughout the impact phase. In addition, as Fig. 6 clearly demonstrates, there was a dramatic increase ($P < 0.01$) in PFR immediately after the marathon run. The recovery of the above-mentioned parameters (Fig. 7, *A* and *B*) demonstrates once again the biphasic pattern, a clear recovery by 2 h after and a second reduction by 2 days after. Recovery seems to be complete by 6 days after.

DISCUSSION

The marathon run induced a dramatic deterioration in maximal muscle function, as was to be expected from previous studies (2, 30). This was seen as a reduction in maximal isometric force and in the maximal rate of force production. These fatigue effects showed some similarities to that of low-frequency fatigue (LFF), which is known to be more pronounced in muscles that have been stretched when active than in those fatigued by comparable shortening contractions (28). LFF is thought to signify an impairment of excitation-contraction coupling, a process linking the action potential in the surface membrane with the activation of actomyosin by calcium (37). This type of fatigue persists even when muscle metabolite levels have returned to normal (10). Therefore, it has been suggested that LFF is not simply a consequence of the metabolic cost of the exercise but is due to some form of muscle damage caused by the exercise (18). The postmarathon increased CK activity and skeletal TnI in the present study, when taken as indirect markers of muscle damage (34), support this idea. This is also in line with the findings by Warhol et al. (36). Therefore, on the basis of these findings, part of the deterioration in muscle function after marathon running could be due to some impairment in the contractile properties of the muscle. Our nonsignificant changes in the maximal M waves suggest that neuromuscular transmission failure did not take place.

The marathon run also altered the neural drive into the muscle, as shown by the reduced maximal aEMG values for the GA and Sol muscles. Corresponding reductions in maximal EMG have been reported earlier for the marathon run (30). These results make it tempting to assume that the reduced neural input to the muscle is partly responsible for the weakened force output.

During fatigue the neural inflow to the muscle can be altered through changes in central motoneuron excitability from either supraspinal (5) or from peripheral sources through certain reflex pathways (1). Unfortunately, the present study lacked appropriate methods for testing supraspinal fatigue. However, the reduction in reflex sensitivity, which could be seen as a decline in

Table 2. *Changes in sledge jump performance: takeoff velocity, impact time, push-off time, average impact force, and average push-off force*

Variable	Before	Immediately After	2 h After	2 Days After	4 Days After	6 Days After
Takeoff velocity, m/s	2.24 ± 0.14	2.06 ± 0.13*	2.18 ± 0.15	2.17 ± 0.09	2.19 ± 0.15	2.24 ± 0.16
Impact time, ms	255 ± 30	354 ± 67‡	315 ± 42*	322 ± 55*	296 ± 37†	291 ± 25*
Push-off time, ms	335 ± 44	430 ± 85*	359 ± 38	373 ± 77	358 ± 45	348 ± 52
Impact force, N	1,686 ± 248	1,297 ± 122‡	1,418 ± 153†	1,419 ± 137*	1,479 ± 121†	1,498 ± 154*
Push-off force, N	1,311 ± 224	954 ± 173†	1,175 ± 64	1,151 ± 159	1,190 ± 104	1,256 ± 126

For definition of symbols, see Table 1.

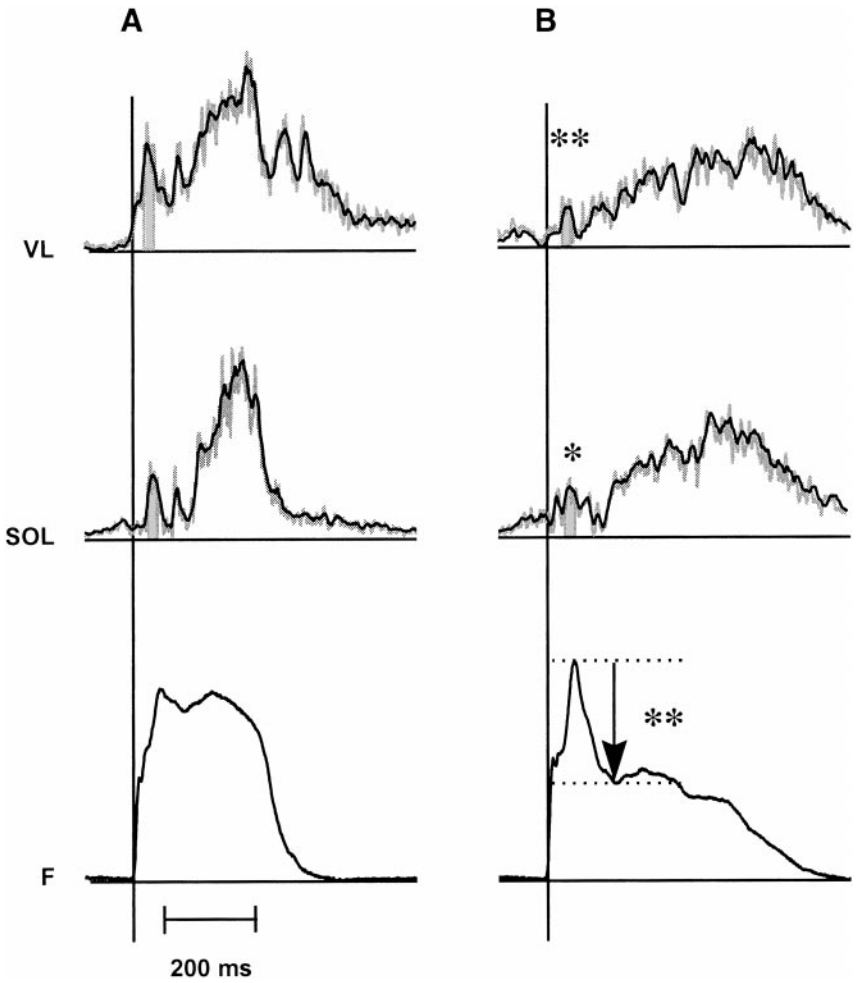

Fig. 6. Rectified and aEMG pattern of vasus lateralis (VL; *top*) and Sol (*middle*) muscles and reaction force (F; *bottom*; $n = 7$ subjects; 10 successive jumps) in stretch-shortening cycle (SSC) exercise sledge jumps before (*A*) and after (*B*) marathon running. Vertical lines, onset of ground contact; shaded areas, short-latency reflex component (M_1) areas. $*P < 0.05$ and $**P < 0.01$: statistically significant compared with before-marathon condition.

the immediate postmarathon values of the H/M ratio, stretch reflexes, and the M_1 reflex component, strengthens the possible role of the peripheral reflex pathways as a possible origin of the reduction in central excitability. The literature has presented two major mechanisms that could be responsible for this impairment.

Bigland-Ritchie et al. (3) demonstrated a decline in motoneuron discharge rates with sustained maximal voluntary contraction. Almost full recovery took place 3 min after the exercise under normal blood supply conditions. However, they did not find any recovery in the firing rates when the fatigued muscle was kept ischemic. Therefore, they hypothesized that during fatigue the motoneuron firing rates may be regulated by a peripheral reflex originating from the fatigued muscle. Furthermore, they suggested that the muscle afferent for such a reflex could be group III and IV free nerve endings. This suggestion has since been supported by Garland (13). These small myelinated and unmyelinated muscle afferents are polymodal and known to be sensitive to several parameters associated with either metabolic fatigue or muscle damage (bradykinin and prostaglandins) (19, 32). It is known that these muscle afferents have a powerful input to inhibitory interneurons (6), stimulation of which could lead to presynaptic inhibition of the Ia terminals and/or inhibition of interneurons in the oligosynaptic pathways (8). In both cases, this could result in a reduced neural drive to the muscle. Nicol et al. (29) suggested that this mechanism could have relevance to the reduction in immediate postexercise reflex sensitivity and have a strong metabolic origin. However, they used short-duration high-intensity SSC exercise, which induced relatively high blood lactate levels. In our case, blood lactate did not show any significant changes. In addition, glycogen depletion, which is likely to take place in marathon running (36), is known to reduce muscle metaboreceptor-mediated responses (33). This mechanism could still be a tenable explanation in the present study, because the lowered reflex sensitivity, as demonstrated by measurements of the H reflex and stretch reflex, was associated with increases in immediate CK activity and skeletal TnI postexercise values. However, the recovery patterns of these parameters did not follow each other, implying possible activity on the part of other additional mechanisms.

Bongiovanni and Hagbarth (4) suggested that reduced γ-loop support to the α-motoneurons (disfacilitation) can also result in reduced α-motoneuron pool excitability. Furthermore, they hypothesized, along with Macefield et al. (25), that the origin of the impaired γ-loop activity could be modified fusimotor support to

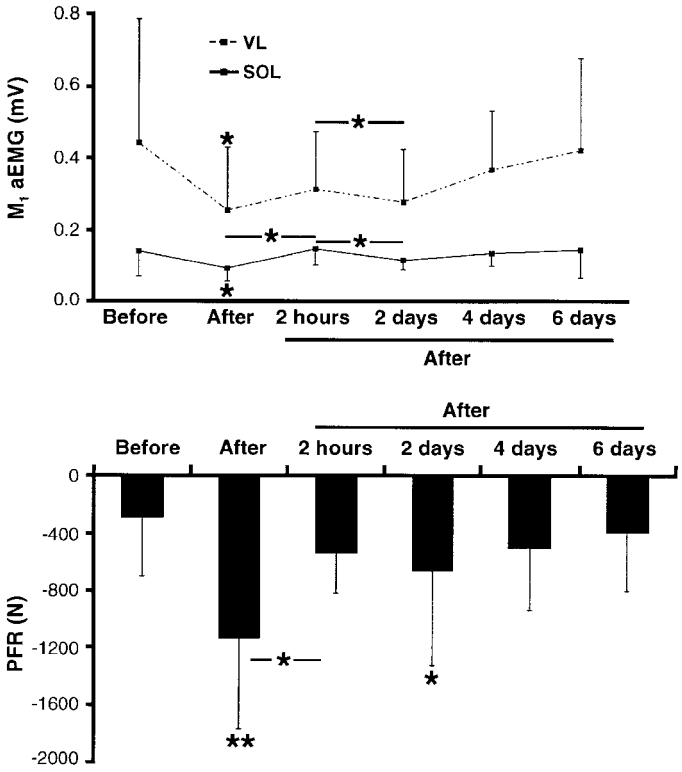

Fig. 7. Mean values (±SD) for 7 subjects of average area of short-latency stretch reflex component (M_1 aEMG) for Sol and VL (*top*) and peak force reduction (PFR; *bottom*). Both parameters were measured from same SSC sledge jumps. $*P < 0.05$ and $**P < 0.01$: statistically significant compared with before-marathon condition or another condition.

the muscle spindles. The present results could support the role of the γ-loop system in reducing reflex sensitivity during long-lasting SSC fatigue. However, the explanation of the withdrawal of fusimotor support to the muscle spindle may not be the only one possible because some of the reflex measurements were performed under passive muscle conditions. A more attractive explanation might be to attribute at least part of the impaired γ-activity to other more direct fatigue effects on the muscle spindles themselves. This effect could be either metabolic or mechanical in nature. Fukami (12) observed in mammalian muscle that spindle activity can be reduced and even suppressed at low intracellular pH. This possibility is eliminated in our case by the nonsignificant changes in blood lactate after the marathon run. However, the possibility that, e.g., glycogen depletion in intrafusal fibers (38) or the direct release of chemical agents due to muscle damage could reduce muscle spindle sensitivity (27) cannot be disregarded.

With regard to the view that group III and IV afferents can also excite fusimotoneurons (17), the observed reduction in reflex sensitivity is of considerable amplitude. This possible increased γ-motoneuron excitation seems to be, therefore, insufficient to compensate for the loss of reflex activity.

The intrafusal fatigue theory relies on the fact that, in such a case, the internal force of the intrafusal fibers decreases, resulting in impaired sensitivity of the spindle. This leads to a decreased inflow of excitatory impulses mediated to α-motoneurons via the Ia afferents. It might, therefore, be of interest to see whether such a reduction in the internal force could be obtained by changes of a more mechanical nature.

The measurement of the stretch-resisting force from the stretch reflex tests was an attempt to clarify the total effect of long-lasting SSC exercise on the passive stiffness properties of the whole tendomuscular complex. Edman and Tsuchiya (9) suggested that, in frogs, the origin of the elastic element affected during stretch is a longitudinal filament that links together the Z and M lines. This filament has been named titin (also known as connectin) (23). Higuchi et al. (15) also suggested that this is the major filament responsible for generating passive muscle tension in the sarcomere. If this is true and because the compliance properties of the tendon are smaller than that of titin (9), it could be suggested that the stretch-resisting force represents the passive stiffness properties of the elastic element, which is parallel to the contractile one. These properties could then take place in both the extrafusal and intrafusal fibers. We have earlier demonstrated (2a) that under the passive stretch condition the repeated stretching of the muscle could modify the muscle tissue so that its compliance increases. It was hypothesized that this would lead to a reduced internal force response from the muscle spindle, resulting in disfacilitation of the α-motoneuron pool. Interestingly, stretch-resisting force behavior was similar after marathon running compared with that under the passive stretch condition. It seems reasonable to suggest, therefore, that at least part of the reduced Ia afferent activity after long-lasting low-intensity SSC exercise could originate from the reduced internal force response from the muscle spindle after the increased compliance of the extrafusal and/or intrafusal fibers.

The stretch-resisting force and stretch reflexes were measured under passive conditions in the present study. Consequently, it could be argued that the mechanical modification hypothesis does not necessarily hold in active muscle function. However, the stretch reflexes were also measured during sledge ergometer jumps. These results showed that the M_1 reflex component declined significantly in both measured muscles immediately after the marathon. This was also the case for PFR, which may indirectly have reflected the changes in the active stiffness properties of the muscle. It seems apparent, therefore, that the theory regarding the mechanical modification of the muscle tissue and/or muscle spindle could also have relevance to active muscle function.

If it can be agreed that the increased compliance of the muscle acts, at least partly, as an operative mechanism in the reduction of immediate postexercise reflex sensitivity, then it could be suggested that the opposite direction of the correlation between the changes in the stretch-resisting force and CK activity immediately after and 2 days after the exercise implies the involvement of other forms of mechanisms during the recovery phase. In the present study, the recovery pattern of the

maximal rate of force production, stretch reflex peak-to-peak amplitude, M_1 aEMG, and the peak force difference showed a bimodal trend that involves the initial decline followed by the early recovery and a secondary decline. This same trend has been reported earlier by Faulkner et al. (11) in animal studies and MacIntyre et al. (26) and Nicol et al. (29) in human studies. Faulkner et al. suggested that the bimodal recovery pattern could be due to muscle damage, which can be divided into initial injury and secondary injury. They implied that the initial injury is more mechanical in nature and the secondary injury is related to the phagocytic activity after the acute inflammatory response at the site of the initial damage. The delayed increase in the stretch-resisting force, which could be due to muscle swelling caused by various inflammation processes, together with the delayed increase in CK activity, might support the existence of secondary injury in the present study. Hence, in addition to the impaired contractile properties of the muscle, such secondary damage could also activate the group III and IV free nerve endings and result in presynaptic inhibition at the site of the Ia afferent terminal (29). The reduced stretch reflex peak-to-peak amplitude 2 days after the marathon run supports this suggestion. However, the maximal isometric force, corresponding maximal aEMG, and the H/M ratio did not demonstrate the bimodal pattern. This could imply that the inhibitory agent is operative during active or passive lengthening or shortening action of the muscle. If this is the case, the most likely possibility is that the group III and IV mechanoreceptors are more heavily involved than has been earlier thought.

In conclusion, long-lasting low-intensity SSC exercise induces an initial decline in muscle function and reflex sensitivity and a delayed second decline in the reflex parameters associated with the lengthening action of the muscle. It is suggested that the initial decline in the neural input to the muscle is of reflex origin in the fatigued muscle. Thus it seems that the regulation of γ-loop activity is of importance. The results of the passive reflex measurements imply the possibility of fatigue, whether metabolic or mechanical in nature, in the intrafusal fibers themselves. The second decline in some of the reflex parameters may be attributable to secondary injury, which might well point to the involvement of the group III and IV mechanoreceptors.

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