

Alterations of neuromuscular function after an ultramarathon

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Millet, G. Y., R. Lepers, N. A. Maffiuletti, N. Babaut, V. Martin, and G. Lattier. Alterations of neuromuscular function after an ultramarathon. *J Appl Physiol* 92: 486–492, 2002; 10.1152/jappphysiol.00122.2001.—Neuromuscular fatigue of the knee extensor (KE) and plantar flexor (PF) muscles was characterized after a 65-km ultramarathon race in nine well-trained runners by stimulating the femoral and tibial nerves, respectively. One week before and immediately after the ultramarathon, maximal twitches were elicited from the relaxed KE and PF. Electrically evoked superimposed twitches of the KE were also elicited during maximal voluntary contractions (MVCs) to determine maximal voluntary activation. MVC and maximal voluntary activation decreased significantly after the ultramarathon ($-30.2 \pm 18.0\%$ and $-27.7 \pm 13.0\%$, respectively; $P < 0.001$). Surprisingly, peak twitch increased after the ultramarathon from 15.8 ± 6.3 to 19.7 ± 3.3 N·m for PF ($P < 0.01$) and from 131.9 ± 21.2 to 157.1 ± 35.9 N for KE ($P < 0.05$). Also, shorter contraction and half-relaxation times were observed for both muscles. The compound muscle action potentials (M wave) were not significantly altered by the ultramarathon with the exception of the soleus, which showed a slightly higher M-wave amplitude after the running. From these results, it can be concluded that 65 km of running 1) severely depressed the maximal voluntary force capacity mainly because of a decrease in maximal voluntary activation, 2) potentiated the twitch mechanical response, and 3) did not change significantly the M-wave characteristics.

electrically evoked twitch; potentiation; M wave; central activation; electromyography

FATIGUE IS OFTEN CHARACTERIZED by an impaired performance involving both motor and sensory processes (16). Many protocols manipulating the type of activation (voluntary vs. electrically induced, continuous vs. intermittent), the type of contraction, as well as the stimulus duration and intensity have been designed to study muscle fatigue. Nevertheless, very few studies have focused on alterations of neuromuscular function after an ultra-long-duration exercise. It has been argued that central fatigue could occur after long-duration exercise (14). For example, Viitasalo et al. (45) found a 30–35% lower vastus lateralis (VL) and vastus medialis (VM) integrated electromyography (iEMG) activity during maximal isometric contraction after a

85-km ski race. Similar results were found by Nicol et al. (35) after a marathon running. However, a decrease of iEMG activity during maximal contractions does not necessarily imply a decrease of maximal voluntary activation because the sarcolemma excitability can be modified as well (38). In fact, it has been shown that the amplitude of the M wave was depressed after a 1- or 2-h duration of exercise (5, 26). It is not known whether decreases of iEMG are entirely explained by changes of M-wave amplitude after such long-term exercises or whether central fatigue really occurs. Another experiment was designed to help understand the decrease of the force-producing capacity after a running exercise longer than 2 h (12), but neither muscular activity nor activation was measured so that data on neural modification, such as central command or sarcolemma action potential, are missing for this type of fatigue. In fact, the rare experiments in which fatigue was induced by an ultramarathon, a highly strenuous stretch-shortening cycle exercise for lower limb extensor muscles lasting several hours, have only studied structural alteration (10, 23), endocrine effects (e.g., Ref. 17), or energetic aspects (36). Therefore, the aim of this experiment was to examine the alterations of neuromuscular function after an ultralong running exercise. For this purpose, we tested changes in voluntary and electrically evoked force of lower limbs extensor muscles, i.e., knee extensors (KE) and plantar flexors (PF), that occur between before and after an ultramarathon.

METHODS

Subjects. Nine healthy male subjects (age, 41.6 ± 5.9 yr; mass, 69.0 ± 7.0 kg; height, 177.3 ± 10.5 cm; body fat, $10.6 \pm 2.5\%$) completed the study. All subjects regularly participated in competitions either in running or triathlon and were specially trained for the race supporting the study (65 km, altitude difference of 2,500 m). Written informed consent was obtained from the subjects, and the study was conducted according to the Declaration of Helsinki. Approval for the project was obtained from the local Committee on Human Research.

Two testing sessions were organized. The first one, in the nonfatigued state, was performed during the week preceding

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the race and started with a 10-min warm-up on a bicycle ergometer (Excalibur, Lode, Groningen, The Netherlands) at a self-selected power output. The second session, in the fatigued state, was performed immediately after the race, within 2 min of completion, and lasted ~20 min in duration. Because the fatiguing exercise was a race, each subject was well motivated to perform maximally over the distance.

Measurements of twitch contractile properties. All muscle contractile measurements were conducted on both KE and PF. Electrical stimulations were given by using a high-voltage stimulator (model DS-7, Digitimer Stimulator, Hertfordshire, UK). During the tests performed in the nonfatigued condition, the intensity of the stimulus was increased until there was no further increase in the height of the twitch or the amplitude of the M wave (81.1 ± 16.9 mA for KE and 96.3 ± 27.2 mA for PF). This same stimulus intensity was used for stimulation in the fatigued condition. A previous study in our laboratory (4) using the same method for determination of maximal intensity showed that no electromyographic (EMG) activity was detected in the biceps femoris. Thus the antagonist contribution to the twitch can be excluded. Whatever the muscle and the condition, a train of three stimuli was delivered over a 15-s period and averaged.

For stimulations designed to study contractile properties of KE, the subject lay prone on a strength-training device adapted from a leg-curl machine with the hip and knee angles fixed at 90° of flexion. The force was recorded by a strain gauge (model SBB, 200 kg, Tempo Technologies, Taipei, Taiwan) securely strapped around the ankle. The femoral nerve was stimulated by using a monopolar cathodal electrode (0.5-cm diameter) pressed in the femoral triangle. The anode was a 10×5 -cm rectangular electrode (Medicom-pex, Ecublens, Switzerland) located in the gluteal fold opposite the cathode. For measurement of contractile properties of PF, the subject was seated with the knee and ankle angles fixed at 90° of flexion. The posterior tibial nerve was stimulated with the cathodal electrode pressed in the poplitea fossa and the anode located on the anterior surface of the knee. A pedal equipped with strain gauges and specifically developed for the study by the local engineer school recorded the mechanical response.

The following parameters were measured from the mechanical response of the evoked contraction: 1) peak twitch tension (P_t), i.e., the highest value of twitch tension production; 2) twitch contraction time (CT), i.e., the time from the origin of the mechanical response to P_t ; 3) maximal rate of twitch tension development (MRFD), i.e., maximal value of the first derivative of the force signal; 4) average rate of twitch tension development ($RFD = P_t/CT$); 5) half-relaxation time, i.e., the time to obtain half of the decline in twitch maximal force; and 6) maximal rate of twitch tension relaxation, i.e., lowest value of the first derivative of the force signal.

Measurements of maximal voluntary isometric strength and activation. Force during maximal voluntary isometric contraction (MVC) was determined for KE. This involved two maximal contractions with a 1-min rest between trials in the same position as for the KE-evoked twitch. The subjects were strongly encouraged, and the best result was used for further analysis. KE maximal voluntary activation was estimated by using the twitch interpolation technique (e.g., Ref. 1). An electrically evoked twitch was superimposed to the plateau reached during each MVC. The ratio of the amplitude of the superimposed twitch over the size of the twitch at rest (recorded before the MVC determination) was then used to calculate the level of voluntary activation as follows

Voluntary activation (%)

$$= [1 - (\text{superimposed twitch} / \text{mean control twitch})] \cdot 100$$

EMG recording. Two EMG signals per muscular group studied were recorded bipolarly by silver chloride surface electrodes during percutaneous electrical stimulation, i.e., VL and VM for KE as well as soleus and gastrocnemius medialis for PF. The recording circular electrodes (Controle Graphique Medical, Brie-Comte-Robert, France) were fixed lengthwise over the middle of the muscle belly (15) with an interelectrode distance of 20 mm. The reference electrode was attached to the wrist of the opposite arm. Low impedance at the skin-electrode surface was obtained (<2 k Ω) by abrading the skin with emery paper and cleaning with alcohol. Myoelectrical signals were amplified with a bandwidth frequency ranging from 1.5 Hz to 2 kHz and simultaneously digitized on-line (sampling frequency 1 kHz). The root mean square (RMS) value was analyzed during the MVC determination for both VM and VL muscles over a 0.5-s period after the torque had reached a plateau. Peak-to-peak amplitude and peak-to-peak duration of electrically evoked compound action potentials (M wave) were determined for the four muscles during the maximal twitches. All mechanical and EMG data were stored with commercially available software (Tida, Heka Elektronik, Lambrecht/Pfalz, Germany). For each study variable, the percent changes were calculated as the ratio (after – before)/before. KE cutaneous temperature was also measured on the VL after the warm-up for the nonfatigued testing session and at the beginning of the session in the fatigued state (model SM 785, Physiogard, Odam, Wissembourg, France).

Statistical analysis. Each study variable was compared between the nonfatigued and the fatigued state with a one-tailed Student's paired *t*-test. Correlation coefficients were calculated to determine the relationships between selected parameters. For all statistical analyses, a *P* value of 0.05 was accepted as the level of significance.

RESULTS

The time of the winner of the race supporting the study was 355.4 min, and the average time of the subjects participating to the study was 511.0 ± 92.2 (SD) min, i.e., $143.8 \pm 26.0\%$ of the winning time.

Twitch contractile properties. Figure 1 shows a typical change for the twitch mechanical and M-wave response.

Table 1 displays the main changes in the mechanical response after the electrically evoked twitch. A greater P_t , faster rate of force development, and shorter CT were observed for both muscular groups studied.

The peak-to-peak duration and amplitude of the M wave recorded after the evoked twitch are presented in Table 2. The only significant modification induced by the ultramarathon was the increase in soleus M-wave amplitude. No correlations were found between the changes in M-wave amplitude and the changes in P_t for both muscles studied.

MVC and maximal voluntary activation. Figure 2 shows typical force trace during the knee extensor MVC and maximal activation determination before (A) and after (B) the ultramarathon.

MVC and maximal voluntary activation decreased significantly after the run ($P < 0.005$ and $P < 0.001$,

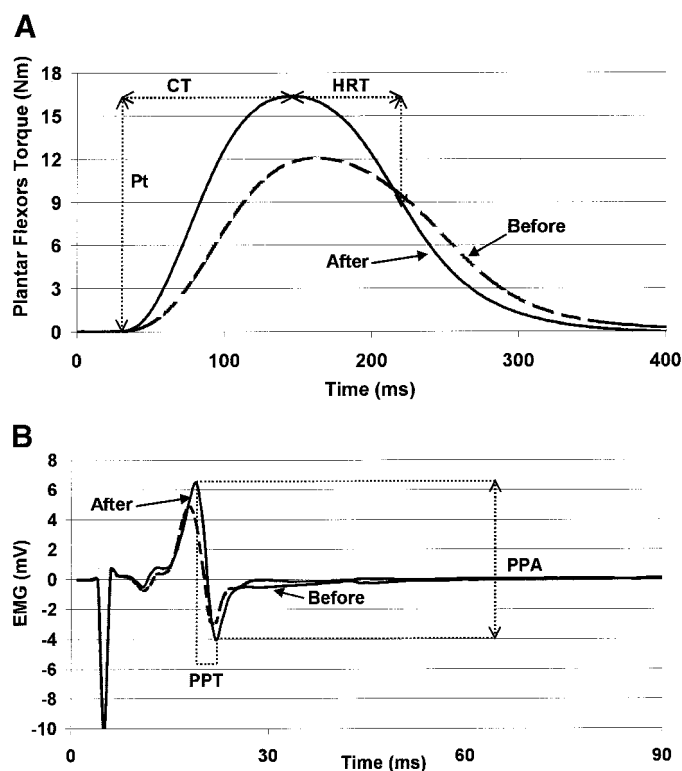


Fig. 1. Typical change for the evoked twitch of the plantar flexors (A) and the associated soleus compound action potential (B). M-wave duration (PPT), M-wave amplitude (PPA), peak twitch (P_t), contraction time (CT), and half-relaxation time (HRT) are noted for the traces recorded after the run. EMG, electromyograph.

respectively; Fig. 3). Table 3 shows the individual activation changes after the ultramarathon.

The electrophysiological data obtained during MVC are consistent with the decrease of maximal activation because VM RMS decreased from 0.32 ± 0.05 to 0.27 ± 0.09 mV ($P < 0.05$), and a trend toward a decrease of VL RMS from 0.34 ± 0.15 to 0.25 ± 0.08 mV was observed ($P = 0.06$). Similarly, the changes of the ratios between RMS and the M-wave amplitude (RMS/M) were not significant but tended to decrease for both VL and VM. Cutaneous temperature did not change significantly between the nonfatigued and the fatigued state (31.2 ± 1.3 vs. $30.7 \pm 1.0^\circ\text{C}$).

DISCUSSION

The main findings of the present study are that a 65-km ultramarathon race severely depressed the maximal voluntary force capacity of KE muscles mainly because of a decrease in maximal voluntary activation. Also, despite the lack of changes in the M-wave characteristics, the twitch mechanical responses of KE and PF muscles were potentiated after an ultramarathon. Because the magnitude of the twitch response is dependent on potentiation- and fatigue-associated effects, the higher twitch mechanical responses found after the ultramarathon do not imply that contractile function was improved by such long-term exercise but suggest that fatigue due to an ultramarathon differs compared with shorter term exercise (i.e., several minutes to 1–2 h).

Twitch contractile properties. Koller et al. (23) have clearly observed skeletal muscle fiber damages after a race similar to the competition, supporting the present study in terms of distance and altitude difference. A decrease of P_t with fatigue was then expected. Interestingly, a 22–35% raise was observed for this parameter. Up to now, all studies conducted on fatigue induced by exercises lasting several minutes to 1–2 h (7, 26) have demonstrated a decrease in twitch mechanical response. Among the explaining factors, the coupling effects of accumulation of H^+ and inorganic phosphate, known to limit Ca^{2+} release, to reduce myofibrillar Ca^{2+} sensitivity and to decrease the number of strong binding cross bridges, have probably the most dramatic influence (2, 30). To our knowledge, this experiment is the first one designed to study the changes of neuromuscular properties after ultra-long-term fatigue. The main results were that P_t , RFD, and MRFD increased after a 65-km running race for both muscular groups, suggesting that twitch mechanical response cannot always be used as a good indicator of peripheral fatigue, as previously suggested (8).

Several hypotheses can be evoked to explain the higher P_t in fatigued state for KE and PF. First, it is worth considering the potential increase in the stiffness of the muscle-tendon complex, which is known to affect twitch mechanical response (9). Howell and co-workers (22) observed an increase in the passive stiffness after a series of near-maximal eccentric contrac-

Table 1. Main mechanical characteristics of the evoked twitch before and after the fatiguing exercise for the two muscular groups studied

	Plantar Flexor Muscles		Knee Extensor Muscles	
	Before	After	Before	After
P_t , N·m or N	15.8 ± 6.3	$19.7 \pm 3.3^\dagger$	131.9 ± 21.2	$157.1 \pm 35.9^*$
CT, ms	138.1 ± 15.5	$129.4 \pm 11.8^\ddagger$	74.4 ± 4.5	$67.9 \pm 4.5^\ddagger$
RFD, $\text{N} \cdot \text{m} \cdot \text{s}^{-1}$ or N/s	113.5 ± 13.8	$152.5 \pm 20.9^\ddagger$	$1,780 \pm 328$	$2,338 \pm 607^*$
MRFD, $\text{N} \cdot \text{m} \cdot \text{s}^{-1}$ or N/s	223.6 ± 82.9	$304.1 \pm 42.3^\ddagger$	$3,703 \pm 886$	$4,715 \pm 1,387^*$
HRT, ms	93.8 ± 19.5	$89.6 \pm 13.6^\S$	68.9 ± 13.5	$57.8 \pm 12.7^\ddagger$
MRFR, $\text{N} \cdot \text{m} \cdot \text{s}^{-1}$ or N/s	143.5 ± 54.2	$183.0 \pm 47.1^\ddagger$	$1,365 \pm 325$	$2,004 \pm 634^*$

Values are means \pm SD. P_t , peak twitch; CT, contraction time; RFD, average rate of force development; MRFD, peak rate of force development; HRT, half-relaxation time; MRFR, peak rate of force relaxation. Significant difference between the nonfatigued (before) and the fatigued (after) condition. * $P < 0.05$; $^\dagger P < 0.01$; $^\ddagger P < 0.001$. § Not significant.

Table 2. *M*-wave duration and amplitude after the evoked twitch before and after the fatiguing exercise for the four muscles studied

	Vastus Lateralis		Vastus Medialis		Gastrocnemius Medialis		Soleus	
	Before	After	Before	After	Before	After	Before	After
Duration, ms	8.0 ± 2.5	9.3 ± 3.0	6.8 ± 2.4	8.1 ± 2.7	1.8 ± 0.4	2.7 ± 1.2	3.9 ± 1.9	3.1 ± 0.7
Amplitude, mV	11.9 ± 3.9	11.4 ± 3.8	8.2 ± 4.5	7.9 ± 5.3	7.1 ± 2.3	9.7 ± 3.9	7.7 ± 3.7	9.8 ± 2.6*

Values are means ± SD. *Significant difference between before and after, $P < 0.05$.

tions. However, Morgan (33) observed that eccentric work induced a disruption of myofilament interdigitation, leading to an increase in the series compliance of the muscle. Compliance was also raised after 1 h of passive muscle stretching (3). Moreover, even if changes in stiffness cannot be completely ruled out as a mechanism for the increase in P_t , RFD, and MRFD, the CT decreased significantly for both PF and KE. This suggests that stiffness had no influence, or a weak influence, on the RFD and P_t modifications.

Muscle temperature may also affect twitch properties. In the present study, cutaneous temperature did not change between the nonfatigued and the fatigued state, although we did not measure intramuscular temperature. Although it is well known that an increase in muscle temperature affects twitch force development (e.g., Ref. 39), it should be emphasized that experiments studying those modifications are often con-

ducted with ranges of temperature out of physiological range. A recent study has showed that 10 min of warm-up increased muscle temperature from 35 to 38°C and that a subsequent 30 min of running only increased muscle temperature to 38.8°C (29). Furthermore, Davies et al. (11) have shown that heating enhanced twitch tension at submaximal stimulation voltage but had no effect on supramaximal twitch force. Thus a change in muscle temperature probably did not entirely explain the changes in twitch parameters found in the present study.

It is well known that twitch force can be potentiated after a tetanic contraction and repetitive low-frequency stimulation. In fact, potentiation- and fatigue-associated effects are probably simultaneously present, and the net result depends on their sum. During low-frequency stimulation, the potentiation phenomenon called staircase (40) is due to phosphate incorporation into regulatory myosin light chain. Phosphorylation of myosin light chain could induce higher Ca^{2+} sensitivity (44) and ATPase activity (32). Even if phosphate incorporation is depressed in fatigued muscles, it has been shown that this has little effect on tension potentiation (27, 43). Interestingly, potentiation was still evident 20 min after the end of a fatiguing exercise (27, 37). Thus, although no direct evidence of phosphate incorporation is present in our study, this phenomenon could represent a valid explanation for the potentiation of twitch tension and rate of force development. In this context, it is worthy to note that postactivation potentiation has been found to be higher in endurance athletes compared with sedentary subjects (19).

One could argue that modifications in motoneuronal excitability, leading to higher neural input, could explain the higher P_t . At submaximal levels, it has been shown that 1–2 h of running enhanced twitch response (13). However, the stimulation was maximal in the present study so that all motor units were spatially recruited in the resting condition. Furthermore, because 1) *M*-wave peak-to-peak amplitude did not change in VL and VM muscles, whereas P_t tended to increase in KE, and 2) there was no relationship between the changes in soleus *M*-wave amplitude and the changes in P_t for PF, the hypothesis linked to sarcolemma and neuromuscular junction effect can be excluded. The lack of modification in *M*-wave duration and amplitude (with the exception of soleus) is also a novel and interesting finding, which reinforces the specificity of the type of fatigue investigated here. In fact, it has been a general finding that compound

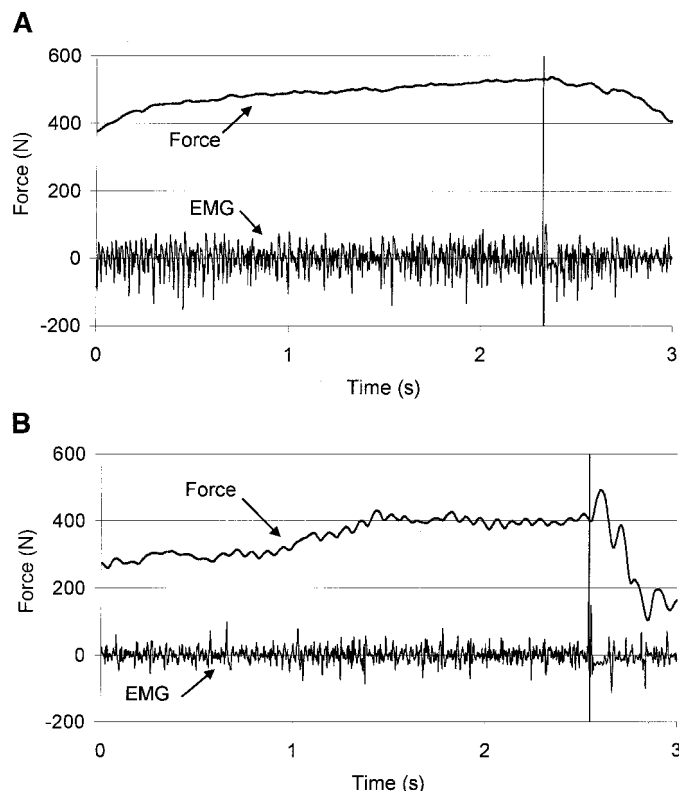
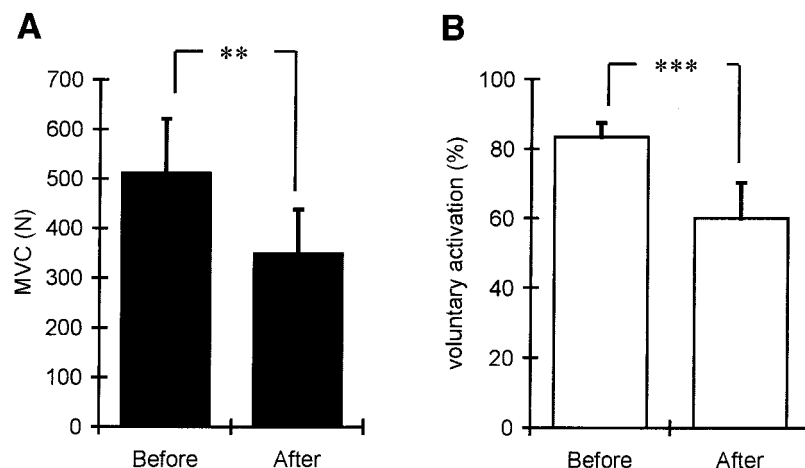


Fig. 2. Typical force trace during the knee extensor maximal voluntary contraction (MVC) and maximal activation determination before (A) and after (B) the ultramarathon. EMG recordings of the vastus medialis are represented to visualize the time of the superimposed twitch.

Fig. 3. Knee extensor MVC (A) and maximal voluntary activation (B) before and after the ultramarathon. Values are means \pm SD. Significant difference between the nonfatigued (before) and the fatigued (after) condition: ** $P < 0.01$; *** $P < 0.001$.



action potential of skeletal muscle, i.e., neuromuscular propagation, changes after repetitive stimulations (21). For instance, 20 min of an intermittent submaximal fatigue protocol induced an $\sim 15\%$ decrease in M-wave amplitude, whereas a shorter fatigue duration had opposite effects (5). Thus alteration of M wave depends on the type of fatigue. In this study, a longer M-wave duration was anticipated because such fatigue is known to induce K^+ and ammonia accumulation (28), which alters fiber excitability and increases M-wave duration (41). Surprisingly, even if a tendency was present for VM, M-wave duration was not significantly affected by the ultramarathon.

The faster rate of force development in fatigued conditions was accompanied by a faster rate of force relaxation for both the KE and PF. This result is also in opposition with literature published on shorter term fatigue because slowing of muscle relaxation with fatigue has long been recognized (28). Rate of force relaxation has been described as a function of Ca^{2+} reuptake by the sarcoplasmic reticulum, which could be affected with long-term fatigue. In fact, the Ca^{2+} -sensitive protease and phospholipase, having a deleterious effect on the ryanodine receptors, could be activated in sustained contractions (41). However, as stated by Booth et al. (7), because there have been no studies investigating Ca^{2+} transients during a pro-

longed (i.e., >1 h) stimulation period, discussion on the effects of very prolonged contraction on cytosolic Ca^{2+} concentration during exercise can only be speculative. Also, these authors found that the measured decrease in Ca^{2+} uptake after 72 min of cycling was not associated with a slowing of relaxation (7).

It must, however, be emphasized that the higher P_t found after the ultramarathon does not imply that contractile function was improved by such long-term fatigue. In fact, twitch could be enhanced with depressed high-frequency contractile response (38). Thus our study is not in contradiction with previous studies showing muscular protein degradation after an ultramarathon (23). Again, the net twitch tension depends on potentiation- and fatigue-associated effects. The most interesting result of this study is that twitch response differs when fatigue due to an ultramarathon is compared with fatigue due to shorter term exercise (i.e., several minutes to 1–2 h; Refs. 7, 26, 42).

MVC and maximal voluntary activation. In the present study, maximal activation of KE in the nonfatigued state was found to be $83.4 \pm 4.2\%$. This value may seem low in comparison to some previous data recorded on the quadriceps muscle in isometric condition, showing that subjects were able to fully activate their motor unit pool (6, 34). However, other studies have failed to measure fully quadriceps activation for all subjects (e.g., Ref. 5). Also, similarly to the value found in the present study, maximal voluntary activation of 79–83% has been reported in other experiments (8, 20). One purpose of the present study was to measure the changes in maximal voluntary activation after an ultramarathon. Because multiple measurements of maximal voluntary activation were not possible after the race, it was decided to only allow two trials before the race. Variability that exists in the determination of this parameter (1) may explain the relatively low degree of maximal activation at rest.

This is the first study to examine changes in MVC force and activation after an ultramarathon run. We found a 30% reduction in MVC force and a 28% reduction in activation after the race. After 5–9 h of skiing, Viitasalo et al. (45) observed a 10% loss of MVC force.

Table 3. Individual values of maximal activation of the knee extensor muscle before and after the ultramarathon

Subject No.	Activation, %	
	Before	After
1	80.7	50.6
2	80.2	58.6
3	81.0	67.1
4	93.1	48.5
5	82.0	55.3
6	82.7	62.3
7	82.5	57.0
8	84.7	80.9
Mean	83.4	60.0
SD	4.2	10.3

Whereas cross-country skiing is similar to running in the way that it uses stretch-shortening cycle for the lower limbs extensor muscles (25), skiing does not induce the same muscular damage as running. In the experiment of Viitasalo et al. as well as in the study of Nicol et al. (35), a large decrease of iEMG activity during maximal contractions were observed with fatigue. However, this can be due to a lower sarcolemma excitability in fatigued state that was not measured in these experiments. In the present study, a lower maximal muscle activity was found. Also, although not statistically significant for VL, a lower RMS/M was observed. More importantly, the present findings show a $27.7 \pm 13.0\%$ decrease in maximal voluntary activation. This emphasizes the role of central fatigue in force loss after an ultramarathon. Such high activation failure has been observed for much shorter but more intense exercise (e.g., Ref. 34). The large decrease in neural inputs to the muscle caused by the ultramarathon is probably multifactorial. It has been argued that, for prolonged exercise, there is a reduction in the corticospinal impulses reaching the motoneurons (14). We showed that the respiratory exchange ratio of subjects running the ultramarathon supporting this study decreased from 0.92 ± 0.06 to 0.79 ± 0.04 at a given submaximal intensity, suggesting a higher oxidation of free fatty acids (31). This finding and those of Davies and Thompson (12), which showed that glycogen stores were 37–53% of the resting values after 4 h of running, suggest that brain free tryptophan may have contributed to the reduction in activation (see Ref. 14 for details). Because 1) free tryptophan is a precursor of serotonin and 2) increased serotonergic activity may induce fatigue through inhibition of the dopaminergic system, the above-described modifications in the substrate oxidation may have led to a decrease in the corticospinal impulses.

Changes in cortical excitability per se are not necessarily the only cause of central fatigue. It has been suggested that neurally mediated afferent feedback from the muscle (i.e., presynaptic inhibition and stretch-reflex disfacilitation) play a part in the inhibition of motoneuron excitability (24). This factor may explain part of the large loss in maximal activation found in the present study. Briefly, it has been demonstrated that exhaustive stretch-shortening cycle exercise reduces stretch-reflex sensitivity because of the modifications of afferent feedbacks from muscle spindles. These afferent feedbacks may be affected by both fatigue of the intrafusal fibers and changes in the viscous and elastic properties of the muscle (3). It also appears that type III and IV afferent activation, also inducing motoneuronal inhibition, occurs in response to local inflammation. The actions of large- (fusimotor system) and small-diameter (type III and IV fibers) afferents may not be mutually exclusive, but the two effects could rather occur together (18). On the contrary, the effects of muscle fatigue on Golgi tendinous organs responsiveness, and thus their influence on alpha motoneuron pool inhibition, is not clear (46).

In summary, the large loss in MVC induced by a 65-km ultramarathon was mostly due to central fatigue. In contrast, the twitch mechanical responses of the KE and PF muscles were potentiated and the characteristics of the M-wave amplitude were mostly not altered by 6–9 h of running. Also, with the exception of the soleus M-wave amplitude and the half-relaxation time, neurophysiological and mechanical modifications measured in the plantar flexor muscle were comparable to those observed for the knee extensors. Taken together, these results demonstrate that fatigue is task dependent such that an ultramarathon has very specific effects on neuromuscular properties compared with a shorter duration of fatiguing exercise.

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