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RESEARCH ARTICLE

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Central fatigue during a long-lasting submaximal contraction of the triceps surae

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Abstract Our purpose was to study central fatigue and its dependence on peripheral reflex inhibition during a sustained submaximal contraction of the triceps surae. In 11 healthy subjects, superimposed twitches, surface electromyograms (EMG) from the medial head of the gastrocnemius (MG) and soleus (SOL) muscles, maximal compound motor action potentials (M_{max}), tracking error and tremor were recorded during sustained fatiguing contractions at a torque level corresponding to 30% of maximal voluntary contraction (MVC). When the endurance limit (401±91 s) of the voluntary contraction (VC-I) was reached, the triceps surae could be electrically stimulated to the same torque level for an additional 1 min in 10 of the 11 subjects. These subjects were then able to continue the contraction voluntarily (voluntary contraction II, VC-II) for another 85±48 s. At the endurance limit of VC-I, the superimposed twitch was larger than during the unfatigued MVC, while there was no significant difference between the twitch at the endurance limit of VC-II and MVC. The EMG amplitude of both MG and SOL at the endurance limit of VC-I was significantly less than that during the MVC. While the EMG amplitude of MG increased further during VC-II, SOL EMG remained unchanged, neither muscle reaching their unfatigued MVC values. This difference was diminished for SOL by taking into account its decrease in M_{max} found during VC-II, and relative EMG levels approached their MVC values. These results clearly indicate that a higher voluntary muscle activation was achievable after 1 min of electrical muscle stimulation, which continued metabolic stress and contractile fatigue processes but allowed for supraspinal, muscle spindle and/or motoneuronal recovery. It is concluded that peripheral reflex inhibition of α-motoneurons via small-diameter muscle afferents is of minor significance for the development of the central fatigue that was found to occur during the first voluntary contraction.

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Introduction

Central fatigue has been defined as a reduced ability to fully activate a fatigued muscle during voluntary contractions (Gandevia 1992) and has been found to develop in several muscles and during various types of contractions (Bigland-Ritchie et al. 1986c; Lloyd et al. 1991; McKenzie et al. 1992; Löscher et al. 1995a).

The maximality of voluntary muscle activation can be assessed by electromyography (EMG; Lippold 1952) and the twitch occlusion technique (Merton 1954). The EMG amplitude is known to increase with the degree of voluntary muscle activation (e.g. Lippold 1952) and this relationship was found to be quasilinear for the human triceps surae muscles (Cresswell et al. 1995). During sustained submaximal contractions, it is expected that at endurance limit the EMG amplitude would increase to a value corresponding to that during an unfatigued maximal voluntary contraction (MVC). However, previous studies on the triceps surae have shown that sustained 30% MVC contractions were terminated at an EMG amplitude corresponding to only about 50% of that of an unfatigued MVC (Löscher et al. 1994, 1995a). Similarly, Fuglevand et al. (1993) reported an inability of the EMG amplitude to reach unfatigued MVC values during submaximal fatiguing contractions of the first dorsal interosseous muscle.

The amount of muscle activation can be further assessed by the superimposition of a supramaximal stimulus onto a voluntary contraction, i.e. the twitch occlusion technique (Merton 1954). Such a stimulus excites all motor axons, including those not voluntarily recruited or not driven at high enough frequencies to achieve fusion, and thereby adds additional force to the contraction. It has been shown that the superimposed twitch declines in proportion to the voluntary torque exerted (Merton 1954;

Belanger and McComas 1981). Thus, the amplitude of the superimposed twitch, compared with that elicited from the relaxed muscle, provides a means of assessing the relative degree of muscle activation. Previously, we have shown that during sustained 30% MVC plantar flexions the twitch occlusion at endurance limit is not complete, but corresponds to an occlusion found during an unfatigued 60% MVC contraction (Löscher et al. 1995a).

Both the findings of an inability of the EMG to reach unfatigued MVC values and an incomplete twitch occlusion at endurance limit are indications of central fatigue. Such central fatigue can be due to: (1) supraspinal factors, such as a decreased efficiency in the generation of the central motor command due to neurotransmitter depletion (Brasil-Neto et al. 1993); (2) reflex inhibition of the α-motoneuron pool by muscle afferents (Garland and McComas 1990); (3) a progressive disfacilitation of muscle spindle support to the α-motoneuron pool (Bongiovanni and Hagbarth 1990; Macefield et al. 1991); (4) late adaptation of α-motoneurons (Kernell and Monster 1982) and/or neuromodulation of synaptic efficiency (Binder et al. 1987).

The present study was designed to investigate central fatigue during a submaximal fatiguing plantar flexion at 30% of MVC. Based on previous results (Löscher et al. 1995a), we hypothesize that central fatigue is not caused by reflex inhibition of α-motoneurons. To test this, the triceps surae was activated involuntarily by transcutaneous electrical stimulation to the same torque level for 1 min after the termination of the fatiguing voluntary contraction. This stimulation protocol was thought to allow supraspinal, muscle spindle and/or motoneuronal recovery while continuing peripheral fatiguing processes. If the contraction time could be voluntarily extended and higher levels of muscle activation achieved after this stimulation, it would indicate that peripheral fatigue and inhibitory input from small-diameter muscle afferents are of minor significance for the termination of the first voluntary contraction.

Materials and methods

Subjects

Eleven habitually active, healthy male subjects, between 22 and 37 years of age, participated in the study. Subjects were instructed about the experimental protocol and gave their informed consent. The experimental procedure was approved by the Ethics Committee of the Karolinska Institute.

Experimental design

Subjects lay prone on a solid chassis with their knees extended and their left foot tightly secured to a non-compliant foot-plate to ensure isometric conditions. A constant ankle angle of 85° (including the angle between the shank and the sole of the foot) was used.

The tibial nerve was stimulated in the popliteal fossa using a bipolar, surface stimulating electrode (Grass E10S2; Grass, USA), with an electrode diameter of 4 mm and an inter-electrode distance of 30 mm, to elicit triceps surae twitches and the maximal compound motor action potential (M_{max}). A square pulse of 500 μs duration was delivered from a digital stimulator (Grass S8800) in series with a stimulus-isolation (Grass SIU5) and constant-current unit (Grass CCU1). Supramaximal stimulus intensity was determined at rest by step-wise increases of current, until the triceps surae twitch and M_{max} of the medial head of the gastrocnemius (MG) and soleus (SOL) muscles showed no further increase. Stimulus intensity was then increased by an additional 50% and thereafter kept constant throughout the experiment.

A series of three plantar flexion MVCs, each lasting 3–5 s, was performed and tibial nerve stimulation applied during each contraction. A 1-s period of stable plantar flexion torque was taken from the trial that gave the highest torque output and used to compute 30% of MVC.

For the transcutaneous muscle stimulation, a pair of 4×6-cm pad electrodes were used. The cathode was placed over the proximal third of the gastrocnemius muscle and the anode over the point where the gastrocnemius muscle joins the Achilles tendon. Square pulses of 200 μs duration were delivered at a frequency of 30 Hz from a somatosensory stimulator (S10DSCM; Grass, USA) in series with a stimulus isolation unit (Grass SIU5). Prior to the fatigue protocol, the triceps surae was stimulated via these electrodes for a brief period of 5–10 s to determine the voltage and acquaint the subject with the stimulation necessary to produce the desired plantar flexion torque of 30% of MVC. During electrical stimulation, contraction of both MG and SOL muscles was verified visually and by palpation.

After a 5-min rest period, resting twitches and M_{max} were evoked. Following the resting stimulation, a torque level corresponding to 30% of plantar flexion MVC was displayed as a beam on an oscilloscope, along with a second beam corresponding to the plantar flexion torque. The subjects were instructed to align the two beams as precisely as possible. Fatigue was induced by maintaining this isometric contraction until the limit of endurance (voluntary contraction I, VC-I), which was defined as the point when subjects could no longer keep the desired torque level. Subjects were well motivated and strong verbal reinforcement was given throughout the contraction. The triceps surae was then stimulated transcutaneously for 1 min and the stimulation voltage adjusted to keep the torque output constant at 30% of MVC. Immediately after this stimulation, subjects were required to maintain a second voluntary contraction at 30% of MVC (VC-II). Strong verbal reinforcement was given and the endurance limit defined as for VC-I. The time interval between the end of VC-I and the time when the electrical muscle stimulation reached the desired torque level was 3.18 ± 1.71 s. Furthermore, the time interval between the end of the electrical stimulation and the time when VC-II reached the appropriate torque level was 1.82 ± 0.69 s.

Once the subject maintained a constant torque output (after 2–3 s), supramaximal stimulation was superimposed upon both VC-I and VC-II and thereafter repeated every 30 s to elicit the superimposed twitch and the M_{max} of MG and SOL.

Torque recordings

Force was measured from beneath the ball of the foot by a load cell (unloaded frequency range 0–2.6 kHz, maximum force 2 kN; Bofors KRG-4; Nobel Electronic, Sweden). The axes of the ankle and foot-plate were aligned as closely as possible. The perpendicular distance between the measurement direction of the load cell and axis of the foot-plate was used to convert the force to plantar flexion torque. The torque signal was low-pass filtered at 50 Hz (Neurolog, NL125 filters; Digitimer, UK), analog-digital (A/D) converted (12 bits) at a sample rate of 1000 Hz, using the 1401plus and Spike2 system (Cambridge Electronics Design, UK) and stored on hard disk.

EMG recordings

Bipolar surface EMG was recorded from the muscle belly of MG, just distal of its motor point, and from SOL along the mid-dorsal

line of the leg, 4 cm distal from where the two heads of the gastrocnemius muscle join the Achilles tendon. The skin was shaved, abraded with sandpaper and cleaned with 95% ethanol prior to electrode application. Surface electrodes, Ag/AgCl, each with a recording diameter of 2.5 mm (SensorMedics, USA), were placed with an inter-electrode distance of 20 mm. EMG signals were amplified ×1000 (Myosystem 2134; Noraxon, Finland), bandpass filtered between 10 and 500 Hz (Neurolog, NL125 filters; Digitimer, UK), A/D converted (12 bits) at a sample rate of 2 kHz, using the 1401 plus and Spike2 data collection system, and stored on hard disk.

Signal processing

The amplitudes of the resting and superimposed twitches were computed. The precision of tracking the target force, the tracking error, was determined by detrending the torque signal, i.e. removing its mean (Gallasch et al. 1994). Furthermore, the torque signal was bandpass filtered using a fourth-order, zero-lag Butterworth filter between 5 and 30 Hz to obtain the tremor signal (Gallasch and Löscher 1992; Löscher et al. 1994). Both the tracking error and the tremor were analysed in 10-s intervals and their root mean squares (RMS) within these periods were calculated and normalised to the actual static torque level.

Raw EMG signals were taken from the same time periods as the tracking error and tremor. The EMG was detrended and low-pass filtered using a fourth-order, zero-lag Butterworth filter with a cut-off frequency of 300 Hz . The RMS of the EMG was calculated within these 10-s periods and normalised to the EMG RMS computed from the MVC recordings. Maximum peak-to-peak amplitudes of M_{max} were measured and normalised to the first elicited during VC-I.

Data were analysed at rest, at the beginning, middle and end of VC-I and at the beginning and end of VC-II.

Statistics

One subject was excluded from the statistical analysis, as it was impossible to sustain a 30% MVC torque with the electrical stimulation (see Discussion). Normality of data distribution was tested using Shapiro-Wilks's *W*-test. Anova for repeated measurements or Friedman Anova were accordingly used to compare data, and post hoc tests were applied using the Scheffé test or Wilcoxon signed-ranked test with Bonferroni-corrected probabilities. Mean values in the text are given with ±1 SD and significance is expressed at $P<0.05$.

Results

MVC torque, twitch occlusion at MVC and endurance time

The mean MVC torque was 174.50±38.77 Nm and the mean amplitude of the twitches superimposed on the MVCs was 1.45±1.27 Nm, corresponding to 9±7% of the resting twitch. VC-I lasted for 401±91 s (range 307–605 s) and VC-II was sustained for another 85±48 s

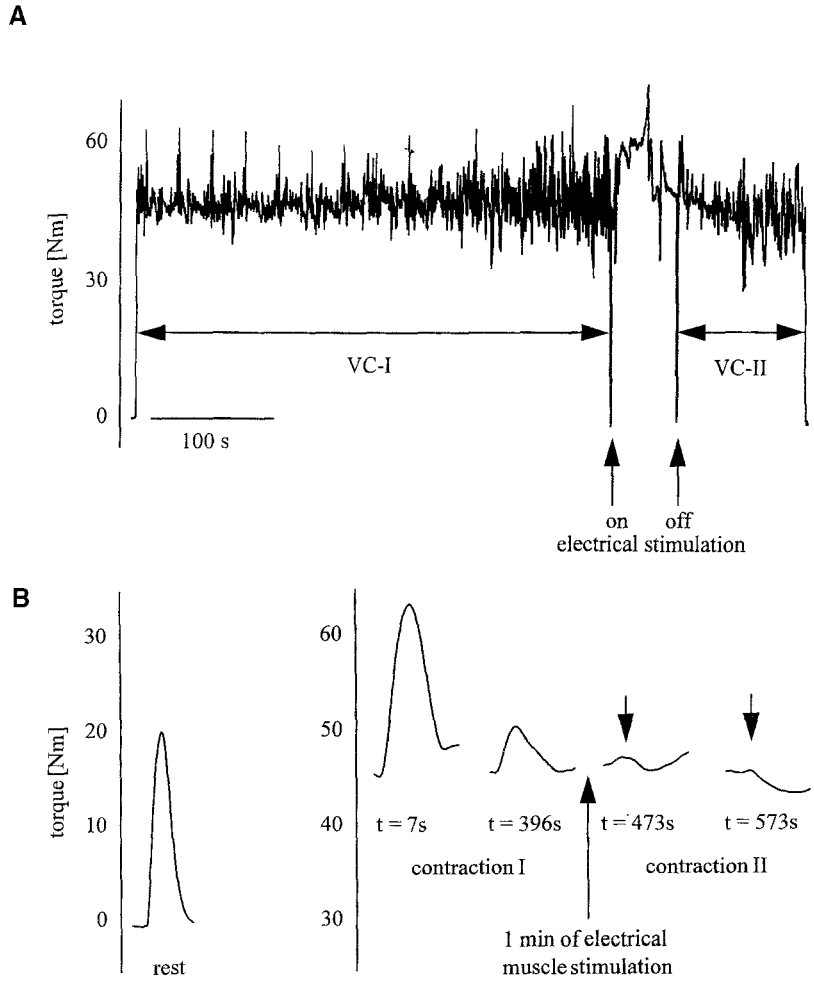

Fig. 1 **A** An example of a torque recording during the contraction sequence of the triceps surae at 30% of MVC is shown for one subject. After the end of the first voluntary contraction (410 s, *VC-I*), the triceps surae was electrically stimulated to the same torque level for 1 min and then the subject maintained this torque level voluntarily for an additional 109 s (*VC-II*). **B** Examples of the triceps surae twitch are shown for the same subject as in **A**. Twitches were elicited at rest and at the beginning and end of the first and second voluntary contraction, which were separated by 1 min of electrical stimulation of the triceps surae. Note that the relative scaling for the resting and the superimposed twitches is the same. *Downward-pointing arrows*, the superimposed twitch during VC-II

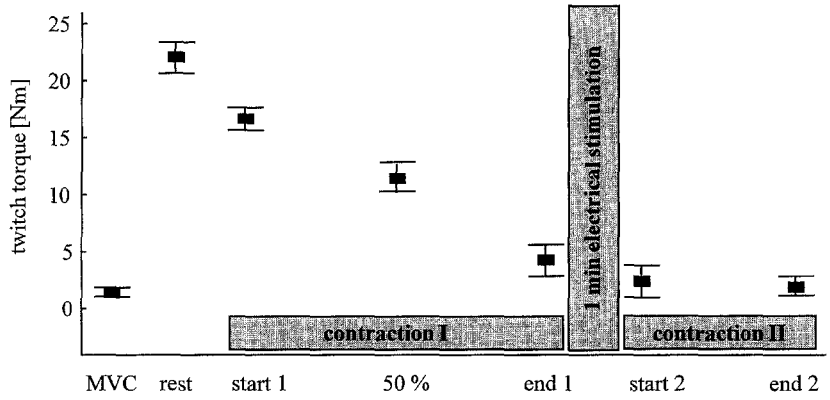

Fig. 2 Amplitudes (means ±1 SEM) of twitches elicited during the maximal voluntary contraction (*MVC*), resting twitches and twitches superimposed on sustained plantar flexions at 30% of MVC, separated by 1 min of electrical stimulation of the triceps surae

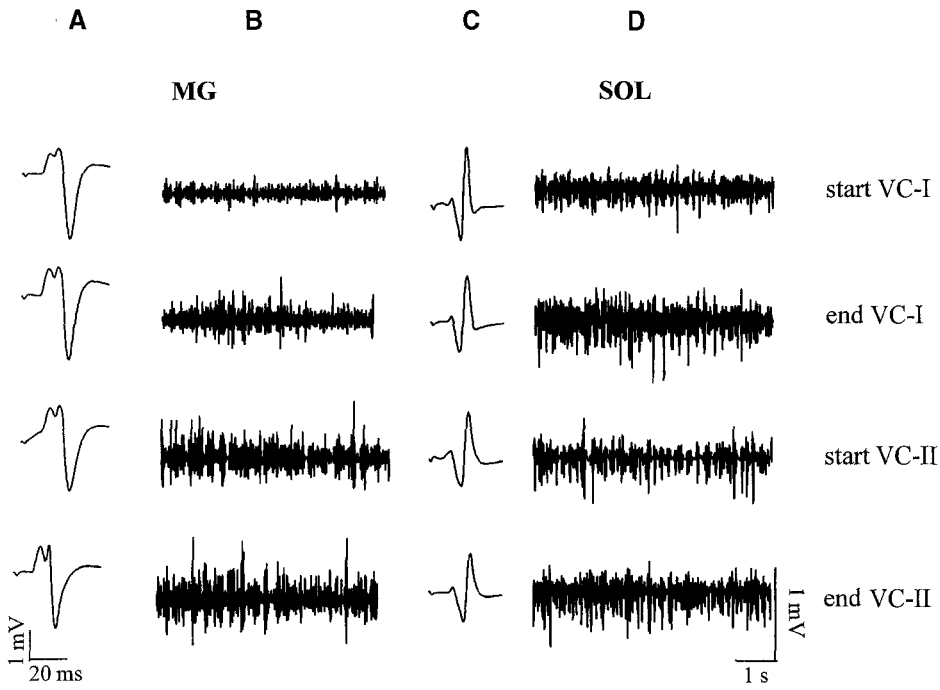

Fig. 3 Examples are shown of the maximal compound motor action potentials (M_{max}) of **A** medial head of the gastrocnemius (*MG*) and **C** soleus (*SOL*) and EMG recordings of **B** MG and **D** SOL from the same subject as in Fig. 1. M_{max} and EMG were recorded at the beginning and end of the first and second voluntary contraction, which were separated by 1 min of electrical stimulation of the triceps surae. The scales in **A** and **C**, and **B** and **D**, respectively, are the same

(range 33–189 s). A torque recording during the entire contraction sequence is depicted in Fig. 1A.

Twitch recordings

Single twitch recordings from one subject at rest, during VC-I and VC-II, are shown in Fig. 1B. The amplitude of the superimposed twitch at the beginning of VC-I was significantly reduced to 76±7% of the resting twitch. The changes of the superimposed twitch amplitude with endurance time were statistically significant (Anova χ^2=17.76, Fig. 2). The twitch amplitude decreased significantly during VC-I to 24±17% of the resting twitch. During VC-II the twitch decreased further from 15±16% to 10±10% of the resting twitch, respectively, and the twitch at the end of VC-II was significantly less than that at the end of VC-I. There was no significant difference between the twitch at the end of VC-I and at the beginning of VC-II.

While the twitch amplitude at the end of VC-I was significantly greater than that recorded during the MVC, the twitch at the beginning and end of VC-II did not significantly differ from the MVC twitch (ANOVA χ^2=13.41). This indicates that central fatigue was present at the end of VC-I, but not at the endurance limit of VC-II.

Electromyography

EMG recordings of MG and SOL of the same subject and during corresponding time periods, as in Fig. 1B, are shown in Fig. 3B and D. The changes of MG and SOL EMG RMS with endurance time were statistically significant (F=17.92 and F=29.52, respectively; Fig. 4A, B). During VC-I, MG EMG RMS increased significantly during the second part of the contraction up to 61±18% of MVC values. During VC-II, MG EMG RMS increased significantly from 63±29% to 76±25%, which was significantly greater than at the end of VC-I, but did not reach unfatigued MVC values. SOL EMG RMS at the end of VC-I (56±17% of MVC values) was significantly greater than that at the beginning and the middle

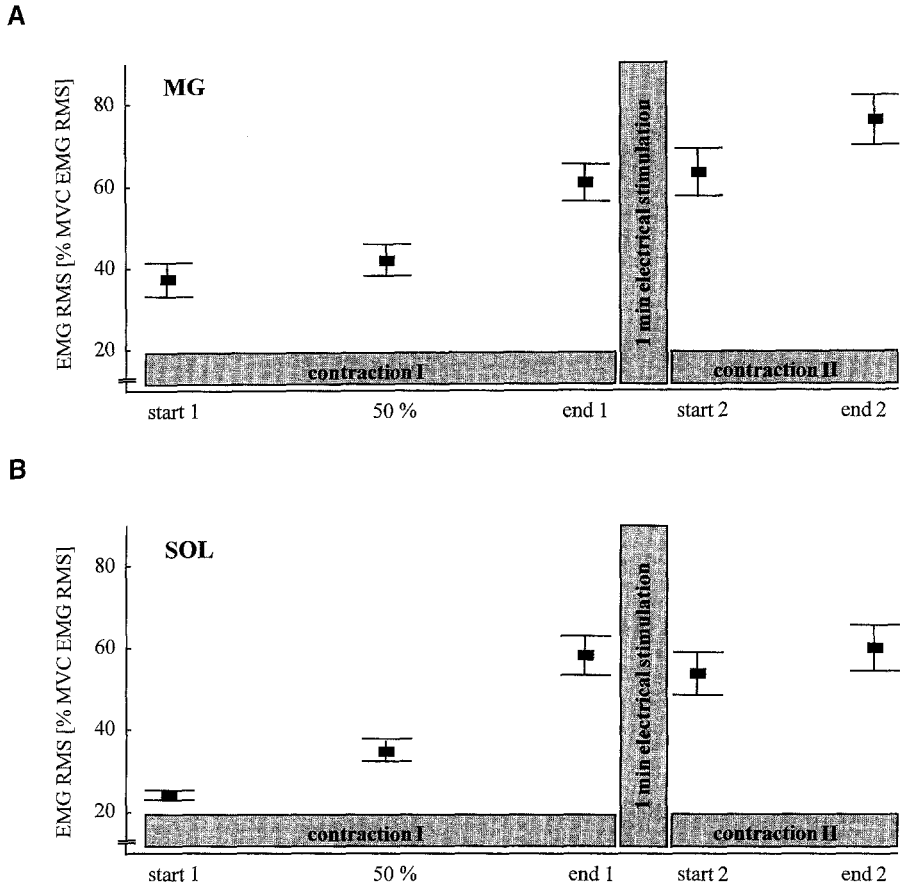

Fig. 4 The root mean square (*RMS*) of EMG recordings (means±1 SEM) for **A** MG and **B** SOL during sustained plantar flexions at 30% of MVC, separated by 1 min of electrical stimulation of the triceps surae. The EMG RMS is normalised to that during the unfatigued MVC

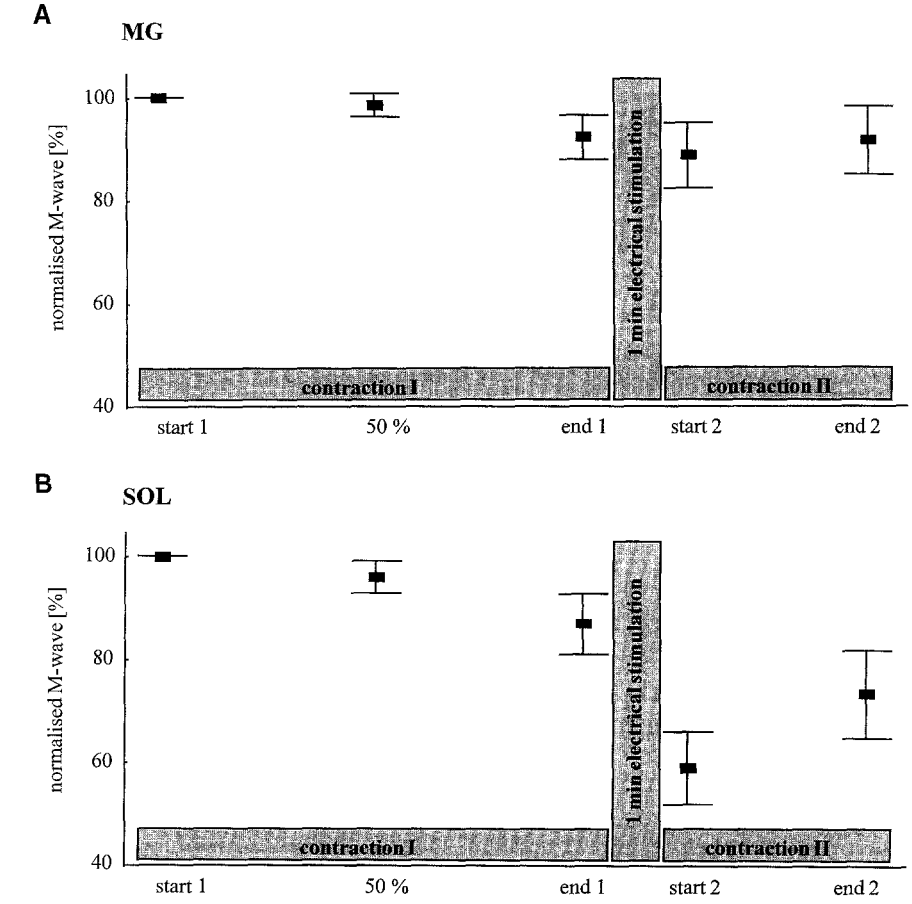

Fig. 5 Amplitudes of M_{max} (means±1 SEM) for **A** MG and **B** SOL during sustained plantar flexions at 30% of MVC, separated by 1 min of electrical stimulation of the triceps surae. The M_{max} is normalised to the first M_{max} recorded during contraction I

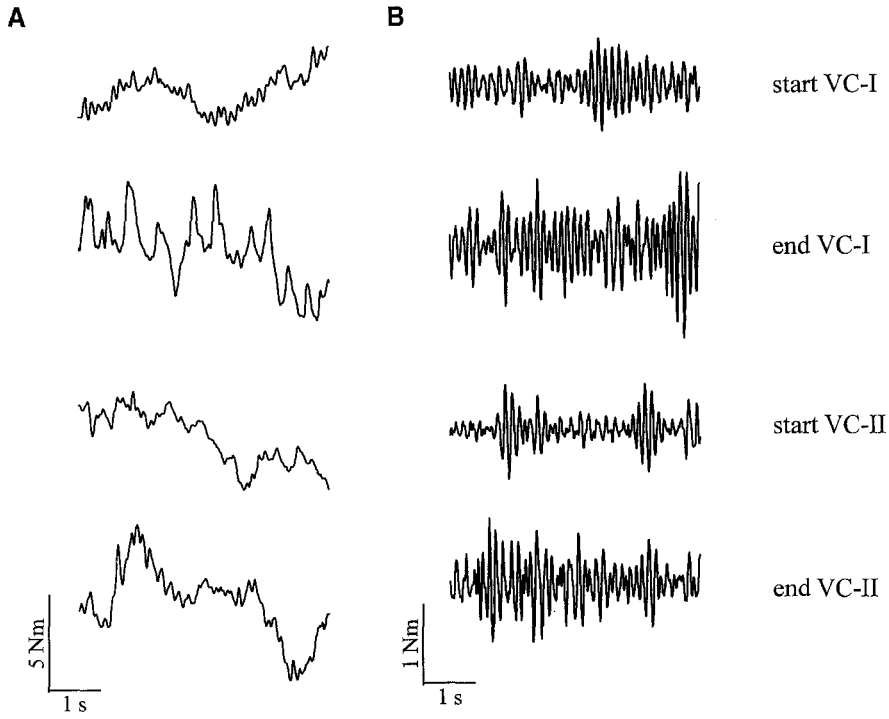

Fig. 6 Examples are shown of recordings of **A** tracking error and **B** tremor for the same subject and corresponding time periods as in Figs. 1B and 3. Tracking error and tremor were recorded at the beginning and end of the first and second voluntary contraction, which were separated by 1 min of electrical stimulation of the triceps surae

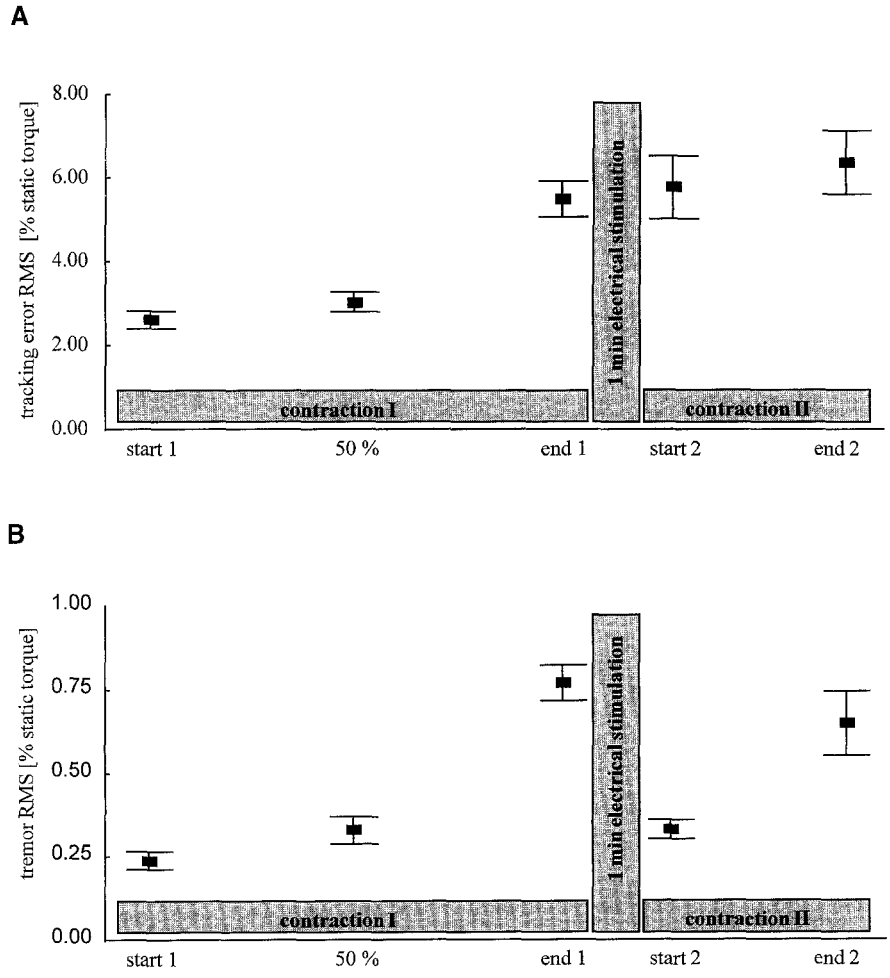

Fig. 7 The RMS (means±1 SEM) of **A** tracking error and **B** tremor during sustained plantar flexions at 30% of MVC, which were separated by 1 min of electrical stimulation of the triceps surae. Tracking error and tremor values are normalised to the actual static torque level during corresponding time periods

of VC-I and did not increase further during VC-II. At the end of VC-II, SOL EMG RMS was 59±20% of unfatigued MVC values.

M_{max} recordings of MG and SOL of the same subject and during corresponding time periods, as in Fig. 1B, are shown in Fig. 3A and C. MG M_{max} did not change significantly with endurance time (F=1.713; Fig. 5A) and was 91±19% at the end of VC-II. In contrast, SOL M_{max} changes with endurance time were statistically significant (F=12.04; Fig. 5B). Post hoc comparisons of SOL M_{max} during VC-I did not reach significance level, while SOL M_{max} at the beginning (58±24%) and end (73±26%) of VC-II were significantly less than that at the beginning of VC-I. SOL M_{max} at the beginning of VC-II was significantly less than that at the end of VC-I (87±16%), while no significant difference was found between the end of VC-II and VC-I.

Tracking error and tremor

Examples of tracking error signals during 5-s periods from the beginning and end of VC-I and VC-II are shown in Fig. 6A. Changes of tracking error RMS with endurance time were statistically significant (Anova χ^2 =25.23; Fig. 7A). Tracking error RMS increased significantly during the second part of VC-I to a mean of 210% of that at the beginning of VC-I. During VC-II, tracking error RMS remained unchanged and did not differ significantly from that at the end of VC-I. At the endurance limit of VC-II, the mean tracking error was 243% of that at the beginning of VC-I.

Examples of tremor signals during VC-I and VC-II are shown in Fig. 6B for the same subject and corresponding time periods as the tracking error. Changes of tremor RMS with endurance time were statistically significant (Anova χ^2=16.56; Fig. 7B). During VC-I, the tremor RMS increased significantly during the second half of the contraction. Tremor RMS at the beginning of VC-II (0.32±0.12% of the actual torque) was significantly less than that at the end of VC-I and VC-II (0.78±0.23% and 0.65±0.42%, respectively), while no significant difference was found between the latter two.

Discussion

The present study shows that the central nervous system is not capable of achieving full voluntary activation of the triceps surae towards the endurance limit of a sustained submaximal plantar flexion at 30% of MVC. First, an electrical stimulation of the triceps surae to previous torque levels was achievable immediately after subjects were unable to voluntarily maintain the contraction. Second, after this electrical stimulation, the subjects were capable of voluntarily continuing the plantar flexion at the same torque level for an additional amount of time. Finally, greater twitch occlusions and higher relative EMG levels were achieved at the endurance limit of the second voluntary contraction. As the temporary removal of central drive via electrical stimulation allowed for a continuation of the voluntary contraction, despite continued metabolic stress and contractile fatigue, it appears that central fatigue is not caused by peripheral inhibitory reflex mechanisms from small-diameter muscle afferents.

The VC-I was impossible to maintain for longer than an average of 401 s, however, after 60 s of electrical stimulation at the same torque level, the contraction could be maintained for another 85 s. As the muscle itself was capable of producing a constant torque output for an additional 36% of its initial endurance time, endurance limit was not caused by contractile failure, but by central fatigue. Several factors indicate that the endurance limit of VC-I was the true maximal time the contraction could be sustained under physiological conditions. The subjects of the present study were highly motivated physical education students, and strong verbal encouragement was given throughout the experiments. Furthermore, four subjects took part in a previous study (Löscher et al. 1995a) in which the same set-up and contraction level were used. The comparison of these previous data with those in the present study revealed no significant differences between endurance times, twitch occlusion values or relative EMG levels. Additionally, previous studies have also reported similar twitch occlusions and EMG levels at the endurance limit of a sustained 30% MVC plantar flexion (Löscher et al. 1994, 1995a).

In one subject, however, the required torque level dropped gradually after 20 s of the electrical stimulation to approximately 15% of his MVC, even with maximal stimulation intensity. His superimposed twitch at endurance limit of VC-I (304 s) was 15%, and during the MVC 2% of the resting twitch. After electrical stimulation of the triceps surae, his maximum achievable voluntary torque corresponded to approximately 15% of MVC, and the twitch superimposed onto the 13-s VC-II was 0.4% of the resting twitch. It appears from the high torque value of this subject (MVC torque: 211 Nm) that his triceps surae consisted of a high proportion of fast, fatiguable motor units. Furthermore, his twitch occlusion at the end of VC-I indicates a relatively small degree of central fatigue as compared to the other subjects. This could explain the high degree of contractile fatigue that developed in this subject during electrical muscle stimulation. Although the required torque level could not be achieved during VC-II, the greater twitch occlusion than at endurance limit of VC-I evidences that central fatigue developed during VC-I, and that a complete voluntary muscle activation was achievable after 1 min of central rest.

To assess the amount of voluntary activation, twitches and EMG levels at endurance limits were compared with those obtained during the unfatigued MVCs. During VC-I the twitch decreased and EMG levels increased without reaching their respective unfatigued MVC values. Immediately after the endurance limit, the muscle could be

made to maintain the same torque level for 1 min by electrical stimulation, indicating that central fatigue developed during VC-I and subjects ended the contraction when muscular capacities for further torque production were still available. This central fatigue can result from several factors. Transcranial magnetic stimulation studies (Brasil-Neto et al. 1993, 1994) have suggested that fatigue occurs at levels upstream of corticospinal neurons, presumably due to neurotransmitter depletion, which could result in an impaired efficiency in generating the central command. Fatigue at a spinal level could result from peripheral reflex inhibition of the α-motoneuron pool (Garland and McComas 1990) and/or disfacilitation of the α-motoneuron pool by muscle spindle afferents (Bongiovanni and Hagbarth 1990). In addition, the late adaptation of α-motoneurons (Kernell and Monster 1982) and/or neuromodulation of motoneuron properties could contribute to the central fatigue found at the end of VC-I. Peripheral inhibitory input has been postulated to be increased after MVCs, and is thought to be conveyed from metabo-receptors via group III/IV afferents (Bigland-Ritchie et al. 1986b; Garland and McComas 1990; Garland 1991; Duchateau and Hainaut 1993). Furthermore, increased inhibition from nociceptors seems to have a detrimental effect on voluntary muscle activation (Rutherford et al. 1986; Gandevia and McKenzie 1988). During electrical stimulation, peripheral inhibitory input was perpetuated, as metabolic turnover, contractile fatigue and contraction induced-pain were maintained, while central drive was removed. Since this period of supraspinal and/or motoneuronal rest allowed for a continuation of the voluntary contraction and central drive could be increased further, it is concluded that peripheral reflex inhibition of the α-motoneurons did not contribute to the central fatigue that developed during VC-I.

It is additionally feasible that, during the period of electrical stimulation, recovery from muscle spindle fatigue could occur. It has been shown that muscle spindle support to α-motoneurons declines, at least during the 1st min of a sustained contraction, which has been proposed to result in a reduced excitatory drive to the α-motoneuron pool (Macefield et al. 1991). Recovery from such muscle spindle fatigue should result in higher spindle support at the beginning of VC-II as compared to that the end of VC-I, and thereby allow for continuation of the voluntary contraction. However, results from experiments with long-lasting, continuous tendon vibrations, reducing Ia-afferent input to the α-motoneuron pool, failed to show any difference in contraction time, EMG amplitudes and amplitudes of the superimposed twitches as compared to a control experiment (Löscher et al. 1995b). Although intrafusal muscle fibre fatigue seems to be an important mechanism adjusting motor unit firing rates (Bongiovanni and Hagbarth 1990, Macefield et al. 1991), it appears to be of minor significance for the central fatigue found during VC-I.

We cannot exclude that the electrical muscle stimulation may have provided facilitation to the α-motoneuron pool via activation of cutaneous afferent pathways, as cutaneous stimulation can facilitate recruitment of high-threshold motoneurons, while inhibiting recruitment of low-threshold ones (Kanda et al. 1977). In a study on human subjects, this recruitment reversal took 2–4 min of cutaneous stimulation to stabilise, and after the end of stimulation, normal recruitment was quickly re-established within 15–30 s (Garnett and Stephens 1981). However, as VC-II was maintained for an average of 85 s, it seems unlikely that α-motoneuron facilitation via cutaneous afferent pathways was an important mechanism in allowing for the continuation of VC-II.

Neuromodulation of synaptic efficacy and the late adaptation of α-motoneurons could contribute to the central fatigue found at the end of VC-I. Neuromodulation has been shown to alter the frequency-current relationship and the threshold for repetitive firing of motoneurons (Binder et al. 1993). Similarly, the late adaptation of motoneurons (Kernell and Monster 1982) has been discussed as an intrinsic mechanism of motoneurons, capable of reducing their firing rates. However, the results from the present study cannot elucidate the contribution of these adaptations to the central fatigue found.

The minor role played by peripheral reflex inhibition was further evidenced by the twitch occlusion and EMG results. During VC-II, the superimposed twitch decreased further and finally did not differ from the twitch during unfatigued MVCs, signifying a maximum achievable voluntary muscle activation of the triceps surae. Interestingly, the twitch occlusion was not complete during the unfatigued MVC, showing that, despite maximal efforts in well-motivated subjects, the voluntary "drive" to the triceps surae during the MVCs was not completely maximal, a finding similar to those reported previously (Belanger and McComas 1981; Cresswell et al. 1995). The EMG levels of MG increased further during VC-II, up to a mean of 76% of the unfatigued MVC. The EMG amplitude increase during sustained submaximal contraction is viewed as an indication of additional recruitment of new, unfatigued motor units and increasing motor unit firing rates (Edwards and Lippold 1956; Bigland-Ritchie et al. 1986a; Löscher et al. 1994) brought about by an enhancement of central drive (DeLuca and Erim 1994; Löscher et al. 1995a). Theoretically, without central fatigue, the EMG levels at endurance limit should reach levels corresponding to approximately MVC values. However, during sustained MVCs, EMG levels have been shown to decrease despite maximum effort, which has been attributed to a slowing of motor unit firing rates (Bigland-Ritchie et al. 1986b). Such a slowing of motor unit firing rates, although being minimal, has recently been reported to occur during submaximal contractions (Garland et al. 1994). As this seems to reduce the surface EMG amplitude recorded during a fatiguing contraction, unfatigued MVC levels might be impossible to reach, even with maximum voluntary drive to the α-motoneuron pool. The EMG levels found in MG at the endurance limit of VC-II could therefore approximate a maximum α-motoneuron pool activation. In SOL, the EMG RMS did not increase further during VC-II. However, during a

sustained contraction, the myoelectric activity measured is not only a result of motor unit recruitment and motor unit firing rates, but can additionally be influenced by an impairment of neuromuscular transmission and/or propagation. The latter two changes have been shown to occur during fatiguing contractions (Sandercock et al. 1985; Fuglevand et al. 1993) and would reduce the myoelectric activity measured.

A possible reduction of the myoelectric activity was assessed by recording M_{max} from MG and SOL throughout the contraction. The significant decrease in SOL M_{max} after electrical stimulation is likely to be a result of the high stimulation frequency used, the mean firing rates of single SOL motor units being only 10.7 ± 2.9 Hz during MVCs (Bellemare et al. 1983). However, this decrease in M_{max} in SOL may be within the safety margin where force production remains unchanged despite a reduction in action potential amplitude (Lännergren and Westerblad 1986). At the endurance limit of VC-II, a significant reduction of SOL M_{max} to 73% occurred, while no significant changes were seen at the end of VC-I. This reduction of M_{max} indicates that the relative SOL EMG levels at the endurance limit of VC-II were closer to MVC values than those actually measured (cf. Fuglevand et al. 1993), although an exact quantitative comparison between M_{max} and EMG RMS is impossible at the present time. Thus, the increase in MG EMG and the relative increase in SOL EMG, taking into account the reduction of its M_{max}, indicate that the voluntary activation of the triceps surae α-motoneuron pool was increased after 1 min of electrical stimulation.

Both tremor and tracking error increased during VC-I. Interestingly, while the tracking error during VC-II was not different from that at the end of VC-I, the tremor was significantly less at the beginning of VC-II. This supports the notion that tracking error is not a result of the involuntary tremor, but an additional effect of fatigue. While the tracking error involves supraspinal factors such as visual feedback, the tremor accompanying isometric muscle contractions has been thought to originate from unfused force ripples of active motor units (Allum et al. 1978) and/or oscillations in the stretch reflex arc (Lippold 1970; Hagbarth and Young 1979). The latter has been proposed to bring about the pronounced tremor increase during fatiguing submaximal contractions (Gottlieb and Lippold 1983; Loggian et al. 1988; Löscher et al. 1994, 1995a). As the net excitatory drive to the α-motoneuron pool increased during the sustained contraction, the gain of the stretch reflex arc could have increased, which would have increased both the amplitude of oscillations and their likelihood to occur (for a detailed discussion see Löscher et al. 1994). Although this theory explains the increase in tremor during VC-I and VC-II, it seem unclear why the tremor at the beginning of VC-II was significantly less. Three mechanisms might be responsible for this observation. It seems reasonable that oscillations do not occur immediately in a system with high gain, but take some time to develop. On the other hand, intrinsic motoneuron properties due to neuromodulation (Binder et al. 1993) or late adaptation (Kernell and Monster 1982) might have recovered during the 1 min of electrical stimulation and thus altered the input-output relationship of the stretch reflex arc. And finally, recovery from muscle spindle fatigue (Bongiovanni and Hagbarth 1990) could have occurred during the period of electrical muscle stimulation, which would result in higher Ia-afferent discharge rates at the beginning of VC-II. It seems unlikely, however, that an increased Ia-afferent input to the α-motoneuron pool results in less tremor. This notion is supported by results from fatigue experiments with reduced Ia-afferent input (Löscher et al. 1995b), which showed a significant reduction of tremor when compared with a control experiment

In conclusion, the results from the present study show that central fatigue developed during a submaximal fatiguing contraction of the triceps surae. After an electrical muscle stimulation for 1 min at the same torque level, the contraction could be prolonged voluntarily for a considerable amount of time, and superimposed twitches and relative EMG levels approached MVC values. As the electrical muscle stimulation removed central drive, while it continued metabolic turnover and contractile fatigue, it is concluded that the central fatigue found at the endurance limit of VC-I was independent of inhibitory input from small-diameter muscle afferents. However, whether this central fatigue occurs at a supraspinal level, at motoneuronal level and/or is caused by spindle fatigue cannot be decided on the basis of the results of the present experiments.

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