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ORIGINAL ARTICLE

Fatiguing handgrip exercise alters maximal force-generating capacity of plantar-flexors

Ashleigh Kennedy · François Hug · Heidi Sveistrup · Arnaud Guével

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Abstract Exercise-induced fatigue causes changes within the central nervous system that decrease force production capacity in fatigued muscles. The impact on unrelated, non-exercised muscle performance is still unclear. The primary aim of this study was to examine the impact of a bilateral forearm muscle contraction on the motor function of the distal and unrelated ankle plantar-flexor muscles. The secondary aim was to compare the impact of maximal and submaximal forearm contractions on the non-fatigued ankle plantar-flexor muscles. Maximal voluntary contractions (MVC) of the forearm and ankle plantar-flexor muscles as well as voluntary activation (VA) and twitch torque of the ankle plantar-flexor muscles were assessed pre-fatigue and throughout a 10-min recovery period. Maximal (100 % MVC) and submaximal (30 % MVC) sustained isometric handgrip contractions caused a decreased handgrip MVC (to 49.3 ± 15.4 and 45.4 ± 11.4 % of the initial MVC for maximal and submaximal contraction, respectively) that remained throughout the 10-min recovery period. The fatigue protocols also caused a decreased ankle plantar-flexor MVC (to 77 ± 8.3 and 92.4 ± 6.2 % of pre-fatigue MVC for maximal and submaximal contraction, respectively) and VA (to 84.3 ± 15.7 and 97.7 ± 16.1 % of pre-fatigue VA for maximal and submaximal contraction, respectively). These results suggest central fatigue created by the fatiguing handgrip contraction translated to the performance of the non-exercised ankle muscles. Our results also show that the maximal fatigue protocol affected ankle plantar-flexor MVC and VA more severely than the submaximal protocol, highlighting the task-specificity of neuromuscular fatigue.

Keywords Central fatigue · Peripheral fatigue · Recovery · Voluntary activation

Abbreviations

MVC	Maximal voluntary contraction
CNS	Central nervous system
VA	Voluntary activation
TT	Twitch torque
EMG	Electromyography
FDS	Flexor digitorum superficialis
BB	Biceps brachii
GM	Medial gastrocnemius
GL	Lateral gastrocnemius
SOL	Soleus
R0	Trial occurring immediately post-fatigue
R1	Trial occurring 1 min post-fatigue
R2	Trial occurring 2 min post-fatigue
R5	Trial occurring 5 min post-fatigue
R7	Trial occurring 7 min post-fatigue
R10	Trial occurring 10 min post-fatigue
ANOVA	Analysis of variance
SD	Standard deviation

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A. Kennedy · H. Sveistrup
School Human Kinetics, Faculty of Health Sciences, University of Ottawa, Ottawa, ON, Canada

A. Kennedy · F. Hug · A. Guével (✉)
Laboratory "Motricité, Interactions, Performance" (EA 4334), University of Nantes, 25 bis Boulevard Guy Mollet, 44322 Nantes Cedex 3, France
e-mail: arnaud.guevel@univ-nantes.fr

H. Sveistrup
School of Rehabilitation Sciences, Faculty of Health Sciences, University of Ottawa, Ottawa, ON, Canada

Introduction

Neuromuscular fatigue is defined as a progressive reduction in the ability of a muscle to produce power or force regardless of whether the task can be sustained (Bigland-Ritchie and Woods 1984; Gandevia 2001). Fatigue affects the entire neuromuscular system from the muscle fibres and neuromuscular junction, i.e., peripheral fatigue, to the higher planning centres in the brain, i.e., central fatigue. Although it is difficult to quantify directly, central fatigue is defined as the decrease in central drive to the motoneurons (Gandevia 2001). Central fatigue contributes significantly to the decreased force production capacity of an exercised muscle (Gandevia et al. 1996; Smith et al. 2007; Søgaard et al. 2006) and is hypothesised to modulate the planning and execution of motor tasks beyond those involving the fatigued muscle group (Amann and Dempsey 2008; Forestier et al. 2002).

The manifestations of central fatigue are highly task specific (Enoka 2008), making it important to characterise the type of fatigue produced by the exercise before assessing how this fatigue alters motor performance. Although both maximal and submaximal contractions elicit central and peripheral changes (Bilodeau 2006; Søgaard et al. 2006; Taylor and Gandevia 2008), low-intensity, continuous exercise is often associated with central failure while maximal contractions are more associated with peripheral failure (Place et al. 2008). Eichelberger and Bilodeau (2007) proposed that these differences may not only be caused by the intensity of the contractions but that disparate stopping criteria and contraction duration may also contribute to the differences found post-fatigue (Eichelberger and Bilodeau 2007).

Researchers have been able to examine central fatigue more closely by examining the impact of exercise-induced fatigue on the motor performance of a muscle group that is not involved in the exercise (i.e., "non-exercised muscle"). Using this approach McLean and Samorezov (2009) found that the central fatigue created during single leg squats crossed over to the non-exercised leg resulting in a less efficient strategy when landing on that leg. They postulated that central fatigue, possibly caused by the inhibitory action of the fatigued leg muscles, decreased the proprioceptive abilities and altered the decision-making and movement execution of the central nervous system (CNS) (Mclean and Samorezov 2009). Unfortunately, it is not clear if the fatiguing exercise performed in this study also created fatigue within the muscles surrounding the hip joint and lower back. If these muscles were fatigued they could have been responsible for the post-exercise changes in postural control. Furthermore, the amount of central fatigue and the duration of the central changes were not quantified in this study making it difficult to assess the relationship between central fatigue and the execution of the motor task immediately after the exercise and as fatigue subsided.

The influence of central fatigue can be more clearly assessed during a simple motor task where the level of voluntary muscle activation (VA) and/or cortical excitability is examined. The VA is a measure of central fatigue that is quantified by measuring the additional force produced by neuromuscular stimulation performed during a maximal voluntary contraction (MVC) (Belanger and McComas 1981). This basic measure has been used to explore the cross-over effect that central fatigue created in one limb has on the contralateral, homogeneous muscle group (Benwell et al. 2006; Humphry et al. 2004; Post et al. 2008; Todd et al. 2003). One such study found that the central fatigue created during an unilateral upper arm task was powerful enough to decreased the force production of the non-exercised arm (Post et al. 2008). Another study examined the impact of unilateral leg fatigued on the contralateral limb (Rattey et al. 2006). Although the force production and the peripheral measures of fatigue, i.e., the twitch torque (TT) and M-wave properties, of the non-exercised leg were unchanged, the level of VA was significantly decreased in this leg after the unilateral fatigue protocol. The authors concluded that central fatigue crossed over to the non-exercised limb but that it did not affect their basic force production capacity.

Presently, it is unclear whether the central fatigue created by an upper limb exercise would also cross-over to and/or affect the motor performance of distal and unrelated lower limb muscles. An investigation into this relationship may increase our understanding of the impact that central fatigue has on the motor control of unrelated, non-fatigued muscle groups. Therefore, the aim of the present study was to determine whether the neuromuscular fatigue created during an upper limb fatigue protocol would be transferred to an unrelated muscle group at the ankle. More specifically, we investigated how fatigue induced by a sustained isometric handgrip exercise affected the ability to voluntarily activate and produce force in ankle plantar-flexor muscles immediately post-fatigue and throughout a 10 min recovery period. Since neuromuscular fatigue is highly dependent on the type of exercise (Enoka and Duchateau 2008), the secondary aim was to determine if the effects of neuromuscular fatigue differed between a maximal and submaximal fatigue task. It was hypothesised that the central fatigue created by a sustained submaximal fatigue protocol would affect the motor performance of an unrelated, non-fatigued muscle group more severely than that produced by a sustained maximal contraction.

Methods

Participants

Fourteen young healthy, habitually active participants (6 women, 8 men; ranged from 20 to 30 years; female height 170.5 ± 3.9 cm; weight 60.5 ± 4.5 kg, male height 176.8 ± 4.9 cm; weight 71.8 ± 6.7 kg) volunteered to participate in two separate testing sessions; one testing a maximal fatigue protocol and the other a submaximal fatigue protocol. Participants were informed as to the nature of the experiment and the study was approved by the local ethics committee and was conducted according to the Helsinki declaration.

Experimental setup

As shown in Fig. 1a, participants sat on an isokinetic dynamometer (Biodex System 3 Research, Biodex Medical, Shirley, USA) with their torso reclined by 15° from upright and both legs extended in front of them. Their right foot was strapped into an ankle attachment that held the foot at 10° plantar-flexion. The torso, waist and right thigh were strapped to the dynamometer chair to ensure that the participant's body position did not change throughout the experiment. The participant's forearms were placed on a table in front of them in a supinated position. Their hands were strapped to the table and an instrumented handgrip (Pinch/Grip Digital Analyzer; MIE Medical Research Ltd, Leeds, UK) was placed in each hand. The width of the handgrip was adjusted to each participant's hand size so that the proximal inter-phalangeal joints of the 4 fingers rested on one side of the handgrip and that of the thumb rested on the other side. The fingers of the right hand were taped to the handgrip to ensure that the finger placement did not change between trials, that the basal grip force remained stable throughout the testing period and that the grip was relatively constant between participants. The torque signals from the ankle attachment on the isokinetic dynamometer (Biodex) and each of the handgrips were digitized at a sampling rate of 4 kHz (Bagnoli 16, Delsys Inc., Boston, USA), low-pass filtered at 50 Hz and displayed on a monitor in front of the participant.

EMG recording

Surface electromyography (EMG) was recorded from the, *flexor digitorum superficialis* (FDS) and *biceps brachii* (BB) of the right arm. The BB EMG was recorded to ensure that its recruitment did not change during the fatigue protocol and that the putative changes observed at the level of PF were only due to handgrip muscles. EMG was also recorded from the *medial* (GM) and *lateral* (GL) *gastrocnemius*, and *soleus* (SOL) of the right leg. For each muscle, a dry-surface electrode (Delsys DE 2.1, Delsys Inc, Boston, USA; 1 cm interelectrode distance) was attached to the skin. Prior to electrode application, the skin was shaved and cleaned with a mixture of alcohol and ether to minimise impedance. Each electrode was placed in

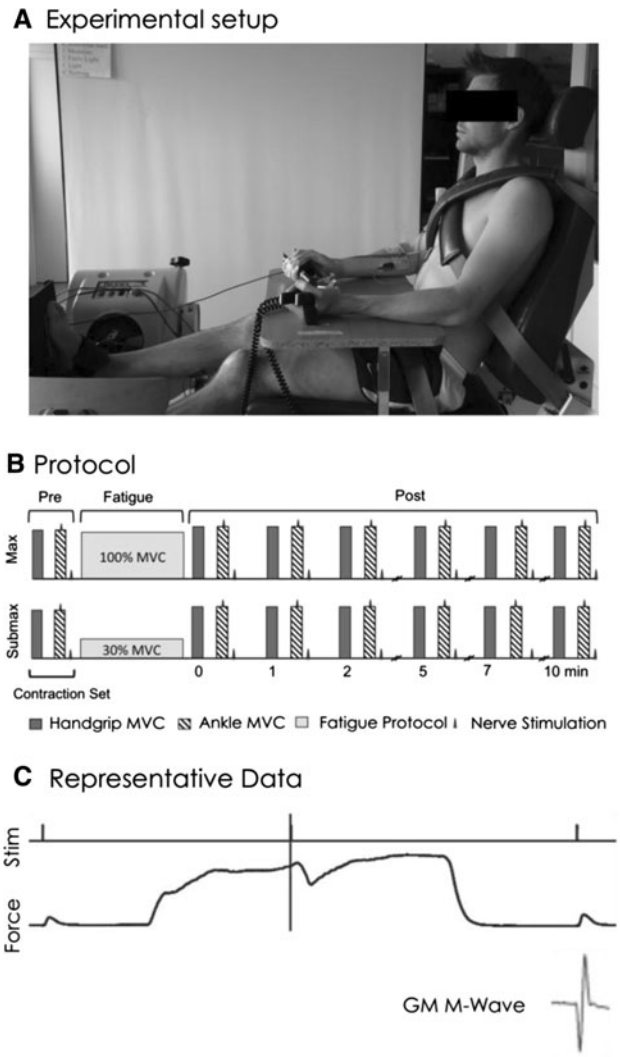

Fig. 1 Submaximal and maximal experimental protocols performed on separate testing days. **a** Experimental setup. Participants sat on a dynamometer with their shoulders and waist strapped to the chair, their hands strapped to the table in front of them and their right ankle and foot strapped to an ankle attachment. **b** Submaximal and maximal fatigue protocol. The participants performed pre-fatigue maximal voluntary contractions (MVCs) with the right hand and ankle, a fatiguing contraction with both hands simultaneously, and a series of post-fatigue contractions performed with the right hand and ankle. During and immediately after each ankle plantar-flexor MVC performed pre and post-fatigue, electrical stimuli were delivered to the tibial nerve. **c** Representative data from the neuromuscular stimulation. The *top trace* represents the timing of the stimulation (stim). The *bottom trace* represents the ankle PF force from which the VA and TT were calculated. The *inset* image is a close up of the GM M-wave

accordance with the SENIAM recommendations (Hermens et al. 2000). The FDS electrode was placed on the muscle belly located in the lateral component of the forearm approximately half way between the medial epicondyle and the base of the 4th finger. Placement of the FDS was verified by an intense handgrip contraction. A ground electrode was placed on the pisiform bone of the right hand. The EMG signals were amplified (1,000×) and digitized (bandwidth of 6–400 Hz) at a sampling rate of 4 kHz (Bagnoli 16, Delsys Inc., Boston, USA).

Electrical stimulation

Twitch contractions of the right ankle plantar-flexors were elicited by electrical stimulation of the tibial nerve found in the popliteal fossa (Scaglioni and Martin 2009). The cathodes were placed 10 cm distal to the popliteal fossa. A conductive probe was used to determine the precise stimulation location that provided the largest mechanical response in the ankle. A 2 × 2 cm anode was placed on this location and a constant current stimulator (Digitimer DS7A, Digitimer Ltd., Letchworth Garden City, UK) delivered single electric pulses to the tibial nerve (pulse duration = 200 μs). To determine the appropriate stimulation intensity, stimuli began at low level and were increased in incremental steps of 20 mA until the force produced by the stimulation no longer increased (mean maximum current = 173.0 ± 30.5 mA). To ensure the reliability of our stimulation technique we checked that the force produced by the stimulation performed at rest was equal to or greater than 25 % of the participant's MVC (Bulow et al. 1993).

Procedure

Each participant performed two separate testing sessions. One session examined the impact of handgrip muscle fatigue created by a bilateral maximal isometric handgrip contraction while the other examined that created by a bilateral submaximal handgrip contraction. The order of the sessions was randomly assigned and there were 48–72 h separating the two sessions. Each session consisted of a warm-up, pre-fatigue MVCs, a bilateral handgrip fatigue protocol, consisting of either a sustained maximal or a submaximal contraction, and a series of post-fatigue MVCs (Fig. 1b). During the pre and post-fatigue MVCs the handgrip and ankle plantar-flexor contractions were performed in sequence. For clarity this sequence was called a 'contraction set'. Each 'contraction set' consisted of a 4 s long isometric handgrip contraction followed 15 s later by a 4 s plantar-flexion of the right ankle. The warm-up and fatiguing contractions were performed bilaterally in order to fatigue as much forearm muscle mass as possible. The MVCs performed pre and post-fatigue were performed unilaterally with the participants right arm.

The warm-up was performed with both handgrips simultaneously and consisted of 4 contraction sets performed at 25 %, 4 at 50 % and 4 at 75 % of the participants perceived maximal handgrip and ankle MVC. Fifteen seconds of rest were given between the contraction sets and 1 min was afforded between the 3 different levels.

Pre-fatigue

Maximal contraction sets were repeated twice for both hands and the right ankle. The largest initial handgrip force produced (MVC) was used to calculate the stopping criterion (20 % MVC) used in the submaximal and maximal fatigue protocols, as well as the level of force to be maintained in the both hands during the submaximal fatigue protocol (30 % MVC).

Next, the participants performed two more contraction sets in which the right hand and right ankle plantar-flexor were maximally contracted. The larger of these two contractions was used as the pre-fatigue value in the statistical analysis. During these contraction sets, the ankle plantar-flexors were superimposed with electrical stimulations. The stimulations were performed during the ankle MVCs as soon as the individual reached a stable force plateau and immediately after each of the contractions. Two minutes of recovery was given between each contraction set and 15 s was afforded between the handgrip and stimulated ankle plantar-flexor contractions.

Fatigue protocol

After 3 min of rest the participants began either the maximal or submaximal handgrip fatigue protocol. These fatiguing contractions were performed bilaterally to increase the amount fatigue created within the forearm muscles in order to maximise the central changes occurring in reaction to the peripheral changes (Noakes et al. 2005). The force produced was visualised separately for both hands throughout the fatigue protocols. During the maximal isometric handgrip protocol, the participants were verbally encouraged to produce the maximal amount of force possible with both hands. During the submaximal isometric handgrip protocol, the participants were verbally encouraged to maintain their hand force production at 30 % MVC, which was visually indicated on each of the graphs, for as long as possible with both hands. The fatigue protocol was stopped in both the maximal and submaximal conditions when the force production was reduced to 20 % of MVC previously determined for the right hand. The participants were unaware of the stopping criteria.

A 10-point Borg Scale was used to determine the perceived exertion of participants throughout the maximal and submaximal handgrip fatigue protocols (Adkin et al. 2002). Every 30 s the participants were asked to assess their perceived effort on a scale of 0–10 where 0 represented the resting state and 10 represented the strongest contraction that fingers could perform.

Post-fatigue

Immediately after the fatigue protocol (R0) the handgrip was removed from the left hand and the participants were asked to perform a set of MVCs with their right hand followed by one with their right ankle plantar-flexors. Electrical stimuli were delivered during and after each of the ankle contractions. This process was repeated after 1, 2, 5, 7 and 10 min of rest.

Data analysis

Data processing was performed using Matlab® scripts (The Mathworks, Natick, USA). Before data analysis was performed the EMG data was band-pass filtered (20–450 Hz). The forearm and ankle MVCs, as well as the ankle TT and level of VA were calculated for the largest of the two contractions performed pre-fatigue and for the contractions occurring immediately post-fatigue (R0), and throughout the recovery period (R1, R2, R5, R7, R10). The VA level was estimated by Eq. 1:

$$\mathrm{VA} = [1 - (\text{extra torque}/\text{control twitch torque})] \quad (1)$$

The M-wave peak-to-peak amplitude was calculated for the GM and SOL using Spike2 (v5.06, Cambridge Electronic Design, UK) to investigate the possibility of peripheral fatigue in the ankle PF muscles (Fig. 1c).

Statistics

A *t*-test assessed the difference between the duration of the maximal and submaximal fatigue protocols. A two-way repeated measure ANOVA [factors = trial type (submaximal and maximal) and fatigue (pre- and post-fatigue)] was performed to determine the effect of fatigue on the handgrip MVCs, ankle PF MVC, VA, TT and the Sol and GM M-wave amplitudes. Next, a two-way repeated measure ANOVA [factors = trial type (submaximal and maximal) and recovery time (R0, R1, R2, R5, R7 and R10)] was used to assess the duration of the fatigue effects throughout the 10-min post-fatigue testing session. Post hoc analyses were performed when appropriate using Bonferroni corrected paired *t*-tests. Bivariate Pearson correlations compared the reduction of the handgrip and PF MVC to the changes in PF VA caused by the maximal and submaximal fatigue protocols.

A p value below 0.05 was considered significant. Values are reported as mean ± standard deviation (SD) throughout the text and the figures.

Results

Fatiguing contraction

The maximal fatigue protocol was significantly shorter than the submaximal fatigue protocol (t (14) = 10.08, $p = 0.0001$; 134 ± 31 vs. 234 ± 36 s). While the perception of effort increased from 2.2 ± 0.9 (fairly light) at the start of the submaximal fatigue protocol to 6.1 ± 1.2 (hard) halfway through the protocol and 10 ± 0 ("very very hard") at the end of the protocol, it remained at 10 throughout the maximal protocol.

Handgrip

There was a main effect of the fatigue protocols on the handgrip MVC (F (1, 13) = 32.78, $p = 0.001$) but no main effect of trial type (i.e., maximal or submaximal) (F (1, 13) = 0.25, $p = 0.627$) or interaction (F (1, 13) = 0.61, $p = 0.448$) indicating that the MVC was similarly affected by the two exercises immediately post-fatigue (R0). The maximal fatigue protocol decreased the MVC to 49.3 ± 15.4 % MVC while the submaximal protocol decreased the value to 45.4 ± 11.4 % MVC. However, post-fatigue there was a main effect of recovery time (F (6, 72) = 27.91, $p = 0.001$), trial type (F (1, 12) = 5.18, $p = 0.040$) and interaction (F (6, 72) = 2.78, $p = 0.017$), indicating that MVC did not recover in the same way after the maximal and submaximal forearm contractions. Post hoc analysis revealed that the post-fatigue values were significantly lower than the pre-fatigue values throughout the entire post-fatigue testing session ($p < 0.05$) and that the handgrip MVC values were lower after the submaximal fatigue protocols relative to the maximal values at R5 ($p = 0.018$), R7 ($p = 0.01$) and R10 ($p = 0.034$) (Fig. 2).

Ankle plantar-flexors

There was a main effect of the handgrip fatigue protocols on the ankle force production (F (1, 13) = 9.32 $p = 0.009$), trial type (F (1, 13) = 92.15 $p = 0.001$) and interaction of fatigue × trial type (F (1, 13) = 9.32 $p = 0.009$) indicating that the two fatigue protocols affected the ankle PF MVC differently. The maximal fatigue protocol decreased the ankle PF MVC to 77 ± 8.3 % of pre-fatigue MVC while the submaximal fatigue protocol decreased the value to 92.4 ± 6.2 % MVC. Post-fatigue there was a main effect of recovery

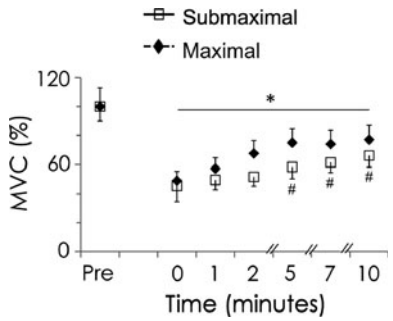

Fig. 2 Handgrip force production. Maximal voluntary contractions (MVC) of the handgrip are depicted pre and post-fatigue for the submaximal (*solid diamonds*) and maximal (*open squares*) fatigue protocols. The data are shown as mean ± SD pre-fatigue (pre), immediately post-fatigue (0) and throughout the 10 min recovery period (1–10). *Significant difference from the pre-fatigue value and #difference between the maximal and submaximal data ($p < 0.05$)

time (F (6, 72) = 4.34, $p < 0.05$) and trial type (F (1, 12) = 6.49, $p < 0.05$). Post hoc analysis revealed that the ankle PF MVC was reduced for the first two post-fatigue trials performed at R0 ($p = 0.001$) and R1 ($p = 0.019$) before it returned to the pre-fatigue values. However, there was no main effect for the interaction (F (6, 72) = 2.17, $p > 0.05$).

There was a main effect of both fatigue protocols (F (1, 13) = 6.33, $p = 0.026$) and trial type (F (1, 13) = 7.07, $p = 0.02$) on the ankle PF VA. The interaction fatigue protocols × trial type was significant (F (1, 13) = 6.33, $p = 0.026$) indicating that the maximal and submaximal fatigue protocols affected the PF VA differently immediately post-fatigue (R0). More precisely, the maximal fatigue protocol caused the ankle PF VA to be decreased to 84.3 ± 15.7 % and the submaximal protocol to be decreased to 97.7 ± 16.1 %. Two-way repeated measure ANOVAs performed on the post-fatigue data showed that there was a main effect of recovery time (F (6, 72) = 3.05, $p = 0.010$) but not trial type (F (1, 12) = 0.15, $p = 0.70$) or interaction (F (6, 72) = 0.94, $p = 0.47$). The ankle VA was significantly lower at R0 ($p = 0.007$) than it was before the fatigue protocols. After this point the ankle VA returned to the pre-fatigue values (i.e., R1, $p = 0.072$).

There was no main effect of fatigue (F (1, 13) = 0.029, $p = 0.87$), trial type (F (1, 13) = 2.30, $p = 0.153$) or interaction (F (1, 13) = 0.29, $p = 0.601$) on the ankle PF TT. Furthermore, no main effect of recovery time (F (6, 72) = 1.39, $p = 0.231$), trial type (F (1, 12) = 0.58, $p = 0.46$) or interaction (F (6, 72) = 0.49, $p = 0.81$) was found. There was no main effect of fatigue on the peak-to-peak M-wave amplitude recorded from the GM (F (1, 13) = 0.050 $p = 0.828$) or SOL (F (1, 13) = 0.17, $p = 0.69$) muscles. Similarly there was no main effect of trial type (F (1, 13) = 0.84, $p = 0.377$, F (1, 13) = 0.97, $p = 0.76$) or interaction (F (1, 13) = 1.72, $p = 0.22$, F (1, 13) = 3.0, $p = 0.11$) on either muscle. The repeated measure two-way ANOVA revealed that there was no main effect of the recovery time (F (6, 72) = 0.28, $p = 0.95$, (F (6, 72) = 0.083, $p = 0.99$) trial type (F (1, 12) = 0.35, $p = 0.57$), (F (1,12) = 0.008, $p = 0.93$) or interaction (F (6, 72) = 0.21, $p = 0.98$, (F (6, 72) = 0.24, $p = 0.97$) for either the GM or the SOL muscles. Overall, these results indicate that no peripheral fatigue occurred in PF.

No significant correlation was found between the handgrip MVC and the PF VA after both the maximal (r (12) = 0.138, $p = 0.638$) and the submaximal fatigue protocol (r (12) = 0.004, $p = 0.988$). Similarly, no significant correlation was found between the ankle PF MVC and PF VA for the maximal (r (12) = −0.297, $p = 0.303$) or submaximal (r (12) = 0.144, $p = 0.623$) fatigue protocol.

Discussion

The present study investigated whether the neuromuscular fatigue induced by a sustained maximal and submaximal isometric handgrip contraction would result in decreased voluntary muscle activity and force production capacity of non-exercised ankle plantar-flexor muscles. The results indicate that the forearm fatigue protocols caused a temporary decrease in the MVC and level of voluntary muscle activation of the non-fatigued ankle plantar-flexor muscles. Moreover, the ankle PF MVC and VA were more affected by the maximal fatigue protocol relative to the submaximal protocol.

Twitch interpolation was used to assess the central and peripheral changes that occurred in the non-exercising ankle PF muscles. While the number of stimuli best suited for twitch interpolation has been disputed, single pulse stimuli were used to quantify the fatigue found in this study because it is the least painful method and has been shown to give a reliable measure of the participant's maximal voluntary activation (Behm et al. 1996; Merton 1954; Scaglioni and Martin 2009; Taylor 2009). Using this methodology we found that the MVC and VA of the non-exercising ankle plantar-flexor muscles were significantly decreased by the sustained handgrip contractions (Fig. 3). Since there was no peripheral fatigue found within the plantar-flexor muscles (i.e., no change in TT or in GM or SOL M-wave amplitudes), we argue that fatigue created by the handgrip exercise was responsible for the impaired motor performance at the ankle. More precisely, the decreased VA and MVC as well as the substantial increase in perceived effort demonstrated that changes within the CNS temporarily prevented full activation of the muscles involved in the ankle plantar-flexion.

Recovery of the ankle MVC and VA and handgrip MVC were assessed throughout a 10-min post-fatigue recovery

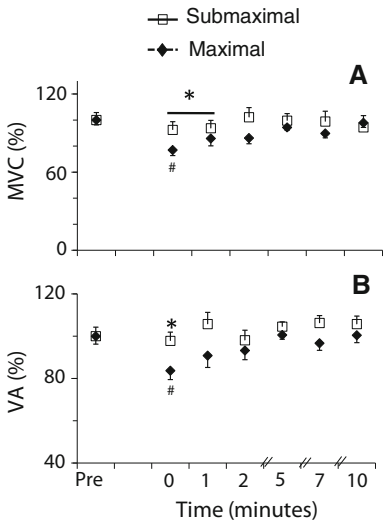

Fig. 3 Ankle plantar-flexor muscle fatigue. The maximal voluntary contraction (MVC) (**a**) and level of voluntary activation (VA) (**b**) are depicted for the ankle plantar-flexors pre and post-fatigue for the submaximal (*solid diamonds*) and maximal fatigue (*open squares*) protocols. All data are shown as mean ± SD pre-fatigue (pre), immediately post-fatigue (0) and throughout the 10 min recovery period (1–10). *Significant difference from the pre-fatigue value and #indicates a difference between the maximal and submaximal data ($p < 0.05$)

period. The data demonstrated that the ankle PF VA recovered after the first test performed post-fatigue (R0) and that the ankle PF MVC recovered after a minute of rest (R1). These changes occurred despite the lingering impairment of the handgrip MVC found throughout the 10-min post-fatigue testing period. The disparity in recovery time between the ankle MVC and the handgrip MVC can be explained by the type of fatigue causing the impairment. The decreased handgrip MVC was caused by both peripheral and central changes while the ankle MVC was only affected by systemic central fatigue. Recovery of the central changes, which occurs more quickly than recovery of peripheral fatigue (Søgaard et al. 2006), allowed the ankle MVC to return to the pre-fatigue levels rapidly post-fatigue. We postulate that slower recovering peripheral changes within the forearm muscles were responsible for a large proportion of the decreased forearm MVC found throughout the 10-min post-fatigue testing period.

The second aim of this study was to compare the effect of a maximal and a submaximal (30 % MVC) handgrip contraction on the muscle performance of the non-fatigued ankle plantar-flexors. The maximal and submaximal contractions were both performed until the force production capacity was reduced to 20 % MVC to ensure that the exhaustion/stopping criteria did not influence the data (Eichelberger and Bilodeau 2007). The results indicated that even when the stopping criteria were controlled, the maximal and submaximal forearm contractions did not affect ankle performance in the same way. More specifically, the ankle PF MVC and VA were both decreased further immediately after the maximal fatigue protocol relative to the submaximal protocol. Furthermore, the PF MVC recovered more slowly after the maximal protocol than after the submaximal protocol. These differences did not support our initial hypothesis, which predicted that the submaximal fatigue protocol would create more central fatigue than the maximal protocol. It is possible that the energy required to drive the forearm muscles maximally created more severe central changes than the submaximal contraction; however, it is difficult to support this theory without a precise quantification of central/peripheral fatigue created at the level of handgrip muscles.

In conclusion, this study was the first to demonstrate that fatigue created by a forearm muscle contraction resulted in a decreased motor performance of the functionally unrelated and distally located ankle plantar-flexor muscles. Specifically, we reported that isometric handgrip contractions briefly decreased the ankle plantar-flexor MVC and VA. Since peripheral fatigue was not found in the ankle plantar-flexors, we suggest that the changes found in the ankle PF were caused by the central fatigue developed during the handgrip exercise. Further analysis revealed that the maximal handgrip fatigue protocol caused a larger decrease in the ankle MVC and VA than the submaximal protocol. The results from this study increase our understanding of how central fatigue affects simple motor performance of a non-exercised and functionally unrelated muscle group. Additional research is required to explore how this systemic change affects more complex motor tasks that involve the lower limbs such as postural stability and locomotion.

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