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Dynamic Fatigue Does Not Alter Soleus H-Reflexes Conditioned by Homonymous or Heteronymous Pathways

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Abstract

H-reflex depression (diminution of amplitude after a conditioning stimulus) is mediated presynaptically and therefore can help distinguish central versus peripheral mechanisms of fatigue. We examined the effects of a dynamic exercise protocol on H-reflex depression using two conditioning methods: homonymous conditioning (paired-pulse tibial nerve stimulation); and heteronymous conditioning (common peroneal nerve stimulation). Ten subjects performed dynamic contractions of the soleus muscle through 30° ankle range of motion. The concentric phase required a target force of 10% of maximum voluntary isometric contraction (MVIC) and the eccentric phase force target was 80% MVIC. Fatigue persisted for >20 min after cessation of the exercise. Compared with pre-fatigue values, the dynamic fatigue protocol did not increase presynaptic inhibition after either homonymous or heteronymous conditioning. Peak to peak amplitude of unconditioned H-reflexes was likewise unchanged despite a long term depression of muscle force (long duration fatigue). These results suggest that persistent fatigue after dynamic exercise is attributed to muscle changes and not altered spinal mechanisms.

Keywords

presynaptic inhibition; post-activation depression; eccentric; concentric

Among the many factors which contribute to muscle fatigue, the type of muscular activity is key (for reviews see Enoka and Stuart 1992, Gandevia 2001). Static (isometric) contractions involve negligible changes in muscle length and yield occlusion of blood flow. The resulting ischemic conditions cause the accumulation of metabolic by-products that trigger group III and IV afferent input to nociceptive centers (Garland 1991). Group III and IV afferents instigate presynaptic inhibition of motor neurons (Garland and McComas 1990), thereby reducing motor neuron excitability (Bigland-Ritchie, Dawson, Johansson and Lippold 1986, Loscher, Cresswell and Thorstensson 1996, Cresswell and Loscher 2000, Walton, Kuchinad, Ivanova and Garland 2002) and causing a decrease in muscular force. Fatigue during static

exercise is therefore strongly influenced by metabolic factors brought about by ischemia. On the other hand, dynamic muscle contractions involve changes in muscle length and do not occlude blood flow, yielding less accumulation of metabolic byproducts. While presynaptically-mediated reductions in motor neuron excitability still occur, they are believed to reflect input by a different set of peripheral (Ia afferent) or central (descending drive) influences (Avela, Kyrolainen and Komi 2001, Klass, Guissard and Duchateau 2004). Dynamic fatigue is therefore not principally due to metabolite accumulation in muscle cells.

While presynaptic inhibition is implicated in both static and dynamic fatigue, it has only been widely studied with static tasks (Duchateau and Hainaut 1993, Nordlund, Thorstensson and Cresswell 2004, Oya and Cresswell 2008, Iguchi and Shields 2012). Soleus H-reflexes obtained during dynamic plantar flexion contractions did not vary across soleus torque levels, a finding that contrasts with previous isometric protocols (Nordlund, Thorstensson and Cresswell 2002, Oya et al. 2008). This difference in soleus H-reflex behavior may indicate that important neurophysiologic differences exist between dynamic and static contractions.

Accordingly, we proposed to study the effects of a dynamic exercise protocol involving both concentric and eccentric contractions on the excitability of the soleus motor neuronal pool before and after a fatiguing task. We employed two different tests to examine motor neuronal excitability; in the first, soleus H-reflexes were elicited by pairs of tibial nerve stimuli (homonymous conditioning). The depression of the second H-reflex amplitude indicates the magnitude of post activation depression (PAD) of the soleus motor neuronal pool by the homonymous conditioning stimulus (Hultborn et al. 1996). Secondly, we applied conditioning stimulus to the common peroneal nerve before eliciting a soleus H-reflex (heteronymous conditioning). The depression of the H-reflex amplitude indicates the magnitude of “classical” presynaptic inhibition (PSI) of the soleus motor neuronal pool by the heteronymous conditioning stimulus (Hultborn, Meunier, Morin and Pierrot-Deseilligny 1987).

It should be noted that both methods employed in this study represent a form of “pre-synaptic” inhibition; albeit the mechanisms for each are distinct. Specifically, homonymous conditioning is thought to be triggered by a presynaptic reduction in transmitter release from the same afferent fibers activated. Conversely, heteronymous conditioning is triggered by a presynaptic inhibition to the activated afferent fibers by way of an inhibitory interneuron (activated from another nerve; peroneal nerve in this study). Because there are different underlying mechanisms for each test, we employed conditioning-test intervals of 100 ms and 500 ms in both test protocols. The 500 ms condition was long enough to assure that any force produced in the muscle was back to baseline and offering a stable environment for recording reflex signals. This study provided us a unique opportunity to study the differential effects of fatigue on reflex excitability of soleus motor neurons conditioned by stimuli from a heteronymous (tibialis anterior) and homonymous source (soleus). To our knowledge, no previous studies have compared these two neural pathways of presynaptic inhibition before and after dynamic fatigue in the lower extremity.

Methods

Subjects

Seventeen subjects (11 women; height = 168.19 ± 3.5 cm; weight = 67.21 ± 4.03 kg; age = 28 years) participated in the study. The subjects were free from any musculoskeletal, neurological, vascular or metabolic disease. They had no lower limb surgeries or significant injuries to the lower extremities. They had no existing contraindications to exercise and were not taking any medications that could affect muscle function. The protocol was approved by the University of Iowa human subjects institutional review board. Subjects provided written informed consent before participating. The subjects were asked to refrain from vigorous exercise for at least 48 hr before each session. All testing was performed on the right leg and all subjects were right leg dominant as established by the leg used to kick a ball.

Mechanical Recordings

Subjects performed alternating concentric and eccentric soleus contractions with the LIDO isokinetic testing and measurement system (Loredan Biomedical Inc., CA). Subjects sat on the LIDO chair with a stabilizing belt at the hips. The left (untested) leg was stabilized with a strap at the thigh. The right knee was flexed to ninety degrees and the foot was attached to a foot plate. The knee and the ankle joints were aligned with the axes of the LIDO actuator. The right limb was then secured by straps to prevent hip or knee movement.

A torque transducer in the LIDO actuator measured soleus torque. The subject received visual feedback of torque via an oscilloscope. The fatigue task consisted of alternating eccentric and concentric contractions of the soleus muscle through a 30° ankle range of motion. The concentric component required a target force of 10% of maximum voluntary isometric contraction (MVIC) and the eccentric component required a target force of 80% MVIC. Pilot studies with a 60% MVIC eccentric workload indicated that the soleus muscle required > 45 min to reach fatigue (task failure). We used 10% during the concentric phase to assure that adequate blood flow was perfusing the muscle; otherwise, it would merely represent an isometric condition. Because a lengthy experimental procedure could compromise the validity of H-reflex results, we opted for a higher eccentric workload (80% vs 60%) and a shorter experiment duration.

Electrical Recordings

Soleus M-waves and H-reflexes were elicited from the tibial nerve in the popliteal fossa via a two-pronged stimulating electrode. A constant current stimulator (Digit-mer model DS7A) with a current range of 50 μ A–1A and a total output of 400V was used to deliver square waves with a pulse width of 1000 μ s. The stimulator was triggered by a digital pulse delivered by a computer via custom software. Tibialis anterior stimulation was delivered via a second Digitimer stimulator controlled by the same computer software. The stimulating electrode was affixed over the common peroneal nerve at the fibular head. Peroneal nerve stimulation intensity was sufficient to produce a visible twitch of the tibialis anterior (TA) muscle without producing ankle movement.

Electromyographic (EMG) activity of the soleus and tibialis anterior muscles was recorded via bipolar recording electrodes consisting of two Ag-AgCl disc recording surfaces (8-mm diameter; 20-mm distance between the electrodes) and a high impedance miniature differential preamplifier ($\times 35$) (Therapeutics Unlimited, Iowa City, IA). The EMG pickup electrode was placed at the junction of upper 2/3rd and lower 1/3rd of the soleus, distal to the insertion of the gastrocnemius muscle in the Achilles tendon, medial to the midline of the leg, in parallel with the direction of the muscle fibers. The tibialis anterior EMG electrode was placed 1/3rd of the distance between the fibular head and medial malleolus, in parallel to the direction of the muscle fibers. The EMG reference electrode was positioned on the anterior surface of the distal tibia. EMG signals were amplified 500–2000 $\times$ (Therapeutics Unlimited, Iowa City, IA), digitized, and recorded using Data Pac II software (Run Technologies, Mission Viejo, CA).

Study 1: Validation of Dynamic Fatigue Protocol

Before the H-reflex investigation, seven pilot subjects performed the concentric-eccentric task so that we could characterize the extent and duration of soleus fatigue. The subjects began by performing three maximum voluntary isometric contractions (MVIC) with strong verbal encouragement. The fatigue protocol consisted of cyclic concentric and eccentric contractions at specified %MVIC force targets. Subjects viewed force targets on an oscilloscope during fatigue testing. During the concentric phase, subjects produced a 10% MVC concentric force at a velocity of 15°/sec through a 30 degree range of ankle motion. Subjects then immediately produced an eccentric force of 80% MVC at 7°/sec. The endurance limit was defined as a drop in force to 60% MVC during the eccentric phase and inability to increase the force with strong verbal cues for three successive trials.

Before fatigue, subjects received a single-pulse stimulus (S) and double-pulse stimulus (D) (interpulse interval = 6ms) to the tibial nerve at the maximum tolerated stimulus intensity. Fifteen D–S pairs were recorded prefatigue. The D/S ratio was used as an estimate of low frequency fatigue (LFF). After fatigue, three MVIC and ten D–S pairs were recorded immediately after, 5 min post, 10 min post, 15 min post and 20 min post fatigue.

Study 2: Conditioned H-Reflexes

Ten subjects underwent testing of conditioned soleus H-reflexes before and after the dynamic fatigue protocol. Table 1 depicts the order of experimental procedures. Subjects performed three MVICs with strong verbal encouragement. To obtain an estimate of the maximum M-wave (MaxM), tibial nerve stimulation was given at 150% of the stimulus intensity required to elicit a maximal M. Next, five unconditioned H-reflexes were obtained at 15-s intervals to avoid inhibitory effects of the previous activation (Crone and Nielsen 1989). The stimulator output was adjusted to elicit unconditioned H-reflexes of $25 \pm 5\%$ MaxM. The small m-waves accompanying these H-reflexes were closely monitored. The stimulus intensity was adjusted as needed during the experiment to maintain m-waves of this amplitude. For the homonymous conditioning phase of the experiment, paired-pulse H-reflexes were elicited via doublet stimuli to the tibial nerve (Crone et al. 1989). Five paired H-reflexes were obtained with an interpulse interval (IPI) of 100 ms, followed by five paired H-reflexes with an IPI of 500 ms. The peak to peak amplitude of the second H-reflex (H2)

was compared with the first H-reflex (H1) to determine H-reflex suppression. Next, five more unconditioned H-reflexes (Hu) were obtained at 15-s intervals. For the heteronymous conditioning phase of the experiment, stimulation was applied to the common peroneal nerve, followed by tibial nerve stimulation at two conditioning-test (C–T) intervals: 100 ms and 500 ms. The amplitude of the resulting conditioned H-reflex (Hc) was compared with the mean amplitude of five unconditioned H-reflexes (Hu) collected to conclude the experiment.

The set of 7 measurements shown in Table 1 were conducted before the dynamic fatigue protocol and were repeated after cessation of exercise. However, we inserted a 5 min rest between step 1 (MVIC) and step 2 (MaxM) to allow normalization of soleus fatigue metabolites, as we wished to limit our investigation to fatigue of nonmetabolic causes. After completion of the 7 experimental steps, the subjects rested for 15 min, after which the 7 steps were repeated (“15-post” condition).

Control Experiment

Subjects returned after one week to participate in a control experiment. The prefatigue H-reflex test protocol was administered, followed by passive ankle joint rotation through the same 30 degree range of motion previously used in the fatigue protocol. Subjects received passive ankle movement for the average fatigue time obtained for the cohort. The passive movement was then followed by the postfatigue test protocol and the 15-post protocol. The fatigue experiment was conducted before the control experiment for all subjects. Administering the fatigue session first was necessary to determine the duration of passive movement subjects would receive in the control experiment (12.8 min). We opted to use the average time rather than the exact time for convenience because pilot work supported that there were no changes over 30 min during the control condition.

Data Analysis

Data were digitized at 5000 samples/sec using Datapac 2000 (RUN technologies, Mission Viejo, CA). Peak to peak amplitudes of all H-reflexes and M-waves were manually selected. The H-reflexes and their preceding small m-waves were normalized to the amplitude of the corresponding MaxM; for example, postfatigue H-reflexes were normalized to postfatigue MaxM. The means were then computed for each latency (100ms, 500ms), for each conditioning method (heteronymous, homonymous), across time (pre, post and 15-post), and for each session (fatigue, control).

Statistical Analysis

Data were analyzed with a mixed model design via repeated measures analysis of variance (ANOVA) with four-way cross over of subjects across IPI/C–T interval, time (pre, post, 15-post), session (fatigue vs control) and conditioning method (homonymous vs. heteronymous). For homonymous conditioning, a two-factor (Time $\times$ Session) repeated-measures ANOVA was used to evaluate H2 within each session and between the two sessions (control vs. fatigue). Another two-factor (time $\times$ session) repeated-measures ANOVA was used to evaluate Hc during the heteronymous conditioning test.

Two-factor (Time $\times$ Session) repeated-measures ANOVAs were used to analyze the target m-waves and accompanying unconditioned H-reflexes (Hu) for steps 3, 5, and 7 in Table 1. These analyses provided information about the consistency of stimulation across sessions (fatigue, control) and across each level of time (pre, post, 15-post).

In the fatigue session, the soleus MVIC was analyzed across each level of time (pre, post, 15-post) with a one-factor repeated-measures ANOVA. A two-factor repeated-measures ANOVA was used to assess for systematic differences in soleus MaxM across session (fatigue, control) and time (pre, post, 15-post). A second two-factor repeated-measures ANOVA was used to test for systematic differences according to session and time for TA M-wave amplitude during the heteronymous conditioning test.

Since the data were collected on two separate days, we also used an analysis of covariance (ANCOVA) to equate the two groups on the prefatigue data and assess the postfatigue data accordingly.

Any significant main effects in these analyses were further examined using Tukey's post hoc multiple comparisons procedures. Alpha was set at $p < .05$. Data are presented as mean $\pm$ SE.

Results

Study 1: Characterization of Dynamic Fatigue

Figure 1 depicts soleus MVIC torque before and at several times after the dynamic fatigue protocol. The average fatigue time for the seven pilot subjects was 9.42 min. MVIC torque decreased by 31.5% after fatigue ($p < .001$) and remained depressed for > 20 min (all $p < .02$). Single and doublet twitch torques likewise declined after fatigue and remained depressed for > 20 min (Figure 2). The double/single twitch torque ratio, an index of low-frequency fatigue, increased by 37.1% at 5 min postfatigue and remained elevated at least 34.6% above the prefatigue ratio for 20 min. This enhanced ability of doublet stimulation to elicit torque after the fatigue bout supports that the soleus persisted in a state of low-frequency fatigue throughout the 20-min protocol.

Study 2: Conditioned H-Reflex Testing

Figure 3 depicts representative examples of soleus H-reflex depression during heteronymous and homonymous conditioning. During homonymous conditioning, a significant main effect of interpulse interval (IPI) existed for both the fatigue and control sessions ($p < .05$). For both sessions, H2 at 100ms was significantly smaller than H2 at 500ms for all time intervals of the experiment ($p < .05$) (Figure 4, top row). No differences in H2 existed according to experiment time (Pre, 5 min, 15 min) ($p > .05$) or between the fatigue and control sessions ($p > .05$). ANCOVA with prefatigue H-reflex as a covariate for between-day comparisons did not yield any significant difference with or without the fatigue protocol.

During heteronymous conditioning, no differences in the conditioned H-reflex (Hc) existed between sessions (fatigue vs. control) or according to time (pre, 5 min, 15 min) ($p > .05$) (Figure 4, bottom row). Hc did not differ between the C-T intervals (100ms vs 50ms) ($p >$

0.05) in fatigue or control sessions ($p > .05$). Thus neither the fatigue protocol nor passive mobilization influenced heteronymous H-reflex depression.

Stimulus Consistency

Soleus MaxM did not differ before and after fatigue or between the fatigue and control sessions ($p > .05$), confirming that changes in neuromuscular transmission did not occur during the study.

During the heteronymous conditioning procedure, no difference existed in tibialis anterior M-wave within or between sessions ($p > .05$). Thus a consistent conditioning stimulus to the common peroneal nerve was maintained throughout the experiment.

During the homonymous conditioning procedure, no systematic differences emerged in the tibialis anterior M-wave according to session (fatigue, control) or time (pre, 5 min, 15 min), supporting that the heteronymous conditioning stimulus remained stable. In general, the soleus m-wave recruited <10% of the soleus motor neuron pool while the H-reflexes were recorded. No difference was found in the amplitude of the small soleus m-waves accompanying the unconditioned H-reflexes at any time for the fatigue or control sessions ($p > .05$).

Discussion

This study was conducted to examine the effects of a dynamic fatigue protocol on H-reflex depression under two different conditioning methods: from a homonymous source (paired-pulse stimulation) and a heteronymous source (peroneal nerve stimulation). The results indicated that, unlike static muscle fatigue protocols (isometric), the dynamic fatigue protocol did not increase presynaptic inhibition; verified by both a homonymous and heteronymous conditioning.

Dynamic Fatigue and H-reflex Depression

During static (isometric) contractions, H-reflex peak to peak amplitude varies according to the strength of the muscular contraction (Loscher et al. 1996, Oya et al. 2008). During sustained maximal contractions, H-reflex amplitude declines gradually, most likely due to group III and IV afferent input (Duchateau et al. 1993). H-reflex paired-pulse suppression, on the other hand, demonstrates a biphasic pattern, with facilitation of the conditioned H early during fatigue and a return of H-reflex suppression as exhaustion approaches (Baudry, Maerz, Gould and Enoka 2011). During intermittent contractions, interposed rest cycles limit metabolite accumulation and H-reflex amplitude increases with fatigue (Loscher et al. 1996, Nordlund et al. 2004). Less is known about the effects of intermittent fatigue upon H reflex suppression. In an intermittent fatigue task, Nordlund and coauthors observed enhancement of H2 compared with H1, indicating facilitation rather than depression of conditioned H-reflexes (Nordlund et al. 2004). This may suggest a diminution of postactivation depression/presynaptic inhibition of the motor neuronal pool during intermittent fatigue tasks.

In the previous studies that examined H-reflex behavior during a dynamic task, as soleus torque (and thus central drive) increased, H-reflex amplitude did not vary (Nordlund et al. 2002, Oya et al. 2008). Likewise, the magnitude of homosynaptic H-reflex depression did not vary according to contraction strength, suggesting that post activation depression was uniform despite variations in central drive (Oya et al. 2008). These results implicate an enhancement of presynaptic inhibition during short dynamic tasks. However, during longer fatigue-inducing dynamic contractions, the modulation of presynaptic inhibition had not previously been studied. Our results suggest that a dynamic fatigue protocol had little impact on H-reflex depression, examined either via homonymous or heteronymous conditioning methods (Figure 3). Likewise, the dynamic fatigue protocol did not affect the peak-to-peak amplitude of the unconditioned H-reflex. (Though unconditioned H-reflex amplitude was higher in the control session, this difference was already evident at the Prefatigue time point and is not a fatigue-related difference.) The stability of H-reflex amplitude and suppression after dynamic fatigue suggests that as was previously observed in short dynamic contractions (Nordlund et al. 2002, 2004, Oya et al. 2008), an enhancement of presynaptic inhibition may offset activity-related increases in descending drive and motor unit excitability.

No previous study has examined changes in H-reflex depression via heteronymous and homonymous stimulation after a dynamic fatigue task in the lower extremity; both considered a form of presynaptic inhibition, but through distinct pathways (see intro). One previous report used these two conditioning methods in the upper extremity during a pair of static (isometric) fatigue tasks (Baudry et al. 2011). Unlike our dynamic fatigue task, these authors observed pre/post fatigue changes in H-reflex depression. However, like us they did not observe an effect of conditioning method on fatigue-related behavior of H-reflex depression. These observations potentially implicate a similar neural mechanism for heteronymous and homonymous conditioning (e.g., presynaptic inhibition). However, other sources of modulation cannot be ruled out (such as Ib afferent input (Baudry et al. 2011)) and we could not methodologically ensure that equal amounts of conditioning stimuli reached the soleus motor neuron pool with the two conditioning methods. Quantification of afferent input in the two methods would require direct recording of potentials at the presynaptic terminal.

Mechanisms of Fatigue

In this protocol, strenuous work during the eccentric phase (80% MVC) ensured that the soleus muscle fatigued, while less resistance during the concentric phase (10% MVC) ensured that the muscle was protected from an ischemic state. Subjects did not report any delayed onset muscle soreness, suggesting that muscle damage was minimal with the fatigue protocol. Eccentric muscle damage is most prevalent in fast fatigable fibers (Newham, Mills, Quigley and Edwards 1983, Jones, Newham, Round and Tolfree 1986, Jones 1996), so its absence is to be expected in the slow soleus muscle. The capacity to produce force was thus not affected by muscle damage. Stable MaxM throughout the experiment likewise supported that fatigue was not attributable to neuromuscular propagation failure.

A key contributor to fatigue during dynamic contractions could be either a decrease or a fluctuation in descending drive to the motor neuronal pool. fMRI studies have shown that strenuous exercise triggers alterations in descending drive (Liu, Dai, Sahgal, Brown and Yue 2002, Liu, Zhang, Yao, Sahgal and Yue 2005). It has also been suggested that separate areas/cells in the motor cortex control static and dynamic force production (Ashe 1997). Thus the differences in H-reflex behavior between this dynamic fatigue task and previous static fatigue investigations (Baudry et al. 2011) may reflect different neuronal substrates.

Fluctuations in central drive may also be attributed to sensory feedback from the periphery. Such a fluctuation may represent a motor control strategy to recruit different motor neurons at different times to maintain the muscle force. In addition, as fatigue progresses, higher centers may resort to fluctuating drive to protect the muscle fibers from excessive activation, yielding a slow decrease in the muscle force. Our results may suggest that higher centers have an inbuilt predilection to sustain motor neuron excitability or to protect the required excitability of motor neurons during dynamic tasks, a regular feature of daily activities. This strategy could potentially resist changes in motor neuron excitability by peripheral mechanisms, especially early during the fatigue process.

A decrease in the muscle force production has been reported with eccentric fatigue without any changes in EMG (Deschenes et al. 2000). After examining biochemical, immunological and functional consequences, the authors concluded that this discrepancy can be attributed to excitation-contraction coupling dysfunction. Other studies have also shown that there is no change in the electrical activity of muscles following eccentric exercise (Howell, Chila, Ford, David and Gates 1985, Jones, Newham and Clarkson 1987). Thus the fatigue elicited in the current study (Figures 1 and 2) may have been triggered by excitation-contraction uncoupling; supported by the increase (35%) in the double-single twitch torque ratio for >20 min.

Conclusion

Soleus H-reflex amplitude and depression (homonymous and heteronymous mechanisms) are not changed after a dynamic fatigue task, despite evidence for prolonged (>20 min) low frequency fatigue of the muscle. The uncoupling of muscle torque and spinal reflex responses and the similar behavior of heteronymous and homonymous conditioning helps to illuminate underlying mechanisms for these phenomena. In the context of fatigue studies, task-specificity (e.g., static vs. dynamic, intermittent vs. constant) appears to play a key role in H-reflex depression.

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Figure 1.

Mean (*SE*) soleus maximum voluntary isometric contraction (MVIC) torque before and after the dynamic fatigue protocol. * = Significantly different from all times postfatigue (all $p < .02$).

Figure 2. Mean (*SE*) soleus twitch torque before and after the dynamic fatigue protocol. Twitches were elicited via single pulses (black) and via a doublet (interpulse interval 6 ms).

Figure 3.

Representative example of soleus EMG responses. During homonymous conditioning (top), paired tibial nerve stimulation at an interpulse interval of 100 ms yields two soleus H-reflexes (H1, H2). During heteronymous conditioning (bottom), peroneal nerve stimulation (arrow) is followed at 100 ms by tibial nerve stimulation, which elicits a conditioned H-reflex (Hc). Suppression of Hc is determined via comparison with unconditioned h-reflexes elsewhere in the experimental protocol (see Table 1).

Figure 4.

H-reflex depression tested via two conditioning methods (homonymous: top row; heteronymous: bottom row) at two different conditioning intervals (100 ms: left column; 500 ms: right column) on two test days (fatigue session and control session). IPI = interpulse interval. C-T = conditioning-test interval. H1 and H2 were elicited via paired-pulse stimulation of the tibial nerve. He is the conditioned H-reflex elicited after common peroneal nerve stimulation. It is shown in comparison with unconditioned H-reflexes (Hu) collected elsewhere in the protocol (see Table 1). All values are mean (*SE*), normalized to the maximal M-wave. * = Significantly different from the corresponding IPI 500 condition ($p < .05$).

Table 1

Experimental Protocol

Steps	Test Procedure	Outcome Variables
Step 1	MVC $\times$ 3	MVC
Step 2	Mmax	Mmax
Step 3	Unconditioned H $\times$ 5	Hu
Step 4	Homonymous testing	
	100ms IPI $\times$ 5	H1, H2
	500 ms IPI $\times$ 5	H1, H2
Step 5	Unconditioned H $\times$ 5	Hu
Step 6	Heteronymous testing	
	100ms C-T $\times$ 5	Hc
	500 ms C-T $\times$ 5	Hc