Effect of afferent feedback and central motor commands on soleus H-reflex suppression during arm cycling

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Submitted 31 May 2012; accepted in final form 4 September 2012

Hundza SR, de Ruiter GC, Klimstra M, Zehr EP. Effect of afferent feedback and central motor commands on soleus H-reflex suppression during arm cycling. J Neurophysiol 108: 3049–3058, 2012. First published September 5, 2012; doi:10.1152/jn.00485.2011.—Suppression of soleus H-reflex amplitude in stationary legs is seen during rhythmic arm cycling. We examined the influence of various arm-cycling parameters on this interlimb reflex modulation to determine the origin of the effect. We previously showed the suppression to be graded with the frequency of arm cycling but not largely influenced by changes in peripheral input associated with crank length. Here, we more explicitly explored the contribution of afferent feedback related to arm movement on the soleus H-reflex suppression. We explored the influence of load and rate of muscle stretch by manipulating crank-load and arm-muscle vibration during arm cycling. Furthermore, internally driven (“Active”) and externally driven (“Passive”) arm cycling was compared. Soleus H-reflexes were evoked with tibial nerve stimulation during stationary control and rhythmic arm-cycling conditions, including: 1) six different loads; 2) with and without vibration to arm muscles; and 3) Active and Passive conditions. No significant differences were seen in the level of suppression between the different crank loads or between conditions with and without arm-muscle vibration. Furthermore, in contrast to the clear effect seen during active cycling, passive arm cycling did not significantly suppress the soleus H-reflex amplitude. Current results, in conjunction with previous findings, suggest that the afferent feedback examined in these studies is not the primary source responsible for soleus H-reflex suppression. Instead, it appears that central motor commands (supraspinal or spinal in origin) associated with frequency of arm cycling are relatively more dominant sources.

arm cycling; H-reflex; interlimb; motor control IN REDUCED ANIMAL PREPARATIONS, there is direct and conclusive evidence of neural interlimb communication coordinating movement between the fore- and hindlimbs (Ballion et al. 2001; Juvin et al. 2005; Miller et al. 1973; Schomburg et al. 1978; Zaporozhets et al. 2006). In comparison, less extensive and more indirect evidence of interlimb communication between arms and legs is available for humans (Balter and Zehr 2007; Dietz 2002; Dietz et al. 2001; Zehr and Duysens 2004; Zehr and Haridas 2003). One experimental paradigm used to explore the role of neural interlimb communication in humans is to perform rhythmic arm movement and measure changes in electromyography (EMG) activity or reflex modulation in stationary legs (Balter and Zehr 2007; de Ruiter et al. 2010; Frigon et al. 2004; Hundza and Zehr 2009; Zehr et al. 2007). This allows the specific influence of arm movement on leg neuromuscular activity to be isolated from interactions of arms and legs moving together. With the use of this approach, Frigon et al. (2004) demonstrated that rhythmic arm cycling significantly suppressed the H-reflex amplitude in stationary legs. Furthermore, they showed that this H-reflex suppression occurs at a premotorneuronal level by demonstrating an interaction between arm-cycling and somatosensory conditioning, known to presynaptically inhibit soleus (SOL) Ia afferents.

In further experiments, we examined specific arm-cycling parameters in attempts to determine possible contributions to the signal responsible for this suppression. The suppression was graded with the frequency of arm cycling (Hundza and Zehr 2009) but was not largely influenced by changes in peripheral inputs associated with alterations of crank length and arm range of movement (Loadman and Zehr 2007). With increased arm-cycling frequency, there are increases in force (Kao and Ferris 2005), level of muscle activation, as well as the rate of muscle stretch. Afferent feedback plays a strong role in modulating H-reflex amplitude within a spinal segment [see review in Brooke et al. (1997)]; however, the role of afferent feedback related to frequency of arm movement in the remote modulation of SOL H-reflex amplitude remains to be determined. In the current study, we comprehensively explored the role of these afferent inputs to clarify their contribution to the signal mediating the SOL H-reflex suppression.

Load has been shown to significantly influence interlimb neural communication. Inertial loading of the foot during rhythmic foot movements induced a significant phase shift of the flexor carpi radialis (FCR) H-reflex in the stationary arm (Borroni et al. 2004). Increased resistance to active upper-limb movement increased neuromuscular activation of the lower limbs during recumbent arm and leg stepping (Huang and Ferris 2004). Based on the significant role that loading has been shown to play in this interlimb communication, we hypothesized that applying a series of arm-cycling loads would significantly affect the H-reflex amplitudes in the stationary legs.

The activation of muscle spindles has been shown to play an important role in the direct and indirect modulation of excitability in reflex pathways [for review, see Brooke et al. (1997); Pearson (2004)]. However, Mazzaro and colleagues (2005) proposed that
decimals in SOL EMG amplitude during gait are not likely mediated by Ia afferents. Tendon vibration is a powerful stimulus for muscle spindle Ia afferents (Brown et al. 1967; Burke et al. 1976) and has been shown to increase the firing rate of Ia afferents (Verschueren et al. 1998). In the current study, vibration was applied to the arm muscles during arm cycling to further clarify the role of muscle spindle feedback on SOL H-reflex modulation. Our previous work showed that altered joint range of motion during arm cycling did not influence SOL H-reflex suppression, suggesting a limited influence of muscle spindle feedback from the arms (Loadman and Zehr 2007). Given this, we hypothesized that vibration to arm extensors during arm cycling would not significantly affect the H-reflex amplitudes in the stationary legs.

Similar patterns of movement-dependent reflex modulation in both passive and active cycling are purported to demonstrate that afferent input, arising as a consequence of the movement, lays down the inhibitory foundation for the movement-dependent reflex modulation (Brooke et al. 1995, 1997; Carroll et al. 2005; Kamibayashi et al. 2010). In the current study, the influence of voluntary, internally driven (“Active”) and involuntary, externally driven (“Passive”) arm cycling on SOL H-reflex suppression in stationary legs was contrasted. We hypothesized that if afferent feedback related to kinematics was the primary source of the signal-mediating SOL H-reflex suppression, then passive and active cycling would similarly suppress the SOL H-reflex (Brooke et al. 1995). However, if central motor commands regulating arm cycling or afferent feedback related to muscle activation rather than afferent feedback related to the movement (i.e., joint kinematics) were the primary source of the signal mediating the suppression of the SOL H-reflex, then active arm cycling would significantly suppress the SOL H-reflex amplitude from the stationary condition, whereas passive arm cycling would not. Operationally defined central motor commands as spinal or supraspinal in origin and as regulating movement parameters related to the act of rhythmic arm cycling (e.g., movement frequency or pattern and level of muscle activation).

**METHODS**

**Participants**

Twenty healthy men and women between 20 and 44 years of age participated in the study. Each participant gave written and informed consent and reported no known history of neurological or metabolic disorder. Experimental procedures were approved by the Human Ethics Board at the University of Victoria and were conducted in accordance with the Declaration of Helsinki.

**Protocol**

The experimental methodology is similar to previous studies (Frigon et al. 2004; Hundza and Zehr 2009), and thus methodology described here is abbreviated with differences highlighted. Participants were seated in a custom-designed chair that constrained movement of the trunk and legs, while feet were secured into metallic frames. This maintained the hips, knees, and ankles at ~90°, ~110°, and ~90°, respectively. Participants performed stationary trials and rhythmic arm-cycling trials for Experiment I, II, or III (outlined below) in the forward direction using a custom-built, hydraulically resisted cycle ergometer. All cycling trials were performed at 1 Hz for Experiments I, II, and III. Participants used online feedback from an oscilloscope to maintain this cycling frequency, which was also monitored by an experimenter. The ergometer was positioned relative to the participant such that when the elbow joints were at their most extended position, they were at ~10° of flexion. The cycle positions were named consistent with the clockface viewed from the right-hand side of the ergometer [i.e., left elbow maximal extension occurs at the 3-o’clock position; see Frigon et al. (2004)]. With the use of online visual feedback of the rectified and filtered EMG signal for SOL and tibialis anterior (TA) muscles, produced by a custom-written (Dr. T. Carroll, University of New South Wales, Australia) LabVIEW (National Instruments, Austin, TX) program, participants maintained a consistent, low-level contraction (~10% of maximum voluntary contraction (MVC)) in their SOL muscle. Participants were also instructed to keep all of the other leg muscles quiescent. Experiments I, II, and III were conducted on different days.

**SOL H-reflex stimulation.** The left posterior tibial nerve at the popliteal fossa was stimulated pseudorandomly (within a fixed time range of 3-5 s apart) with 1-ms square wave pulses using bipolar self-adhesive Ag-AgCl surface electrodes (Thought Technologies, Canada) and a Grass S88 (Grass Technologies, Astro-Med, West Warwick, RI), connected in series with a SIU5 isolation unit and CCU1 constant current unit (both Grass Technologies, Astro-Med) to evoke SOL H-reflexes. Nerve stimulation was delivered when the left arm was in the 3-o’clock position (i.e., with the left elbow in the most extended position) (Frigon et al. 2004; Hundza and Zehr 2009). Stimulation intensity was set to evoke an H-reflex size at ~70% of H-max on the ascending limb. The corresponding direct motor response (M-wave) was monitored online and was maintained consistently across the cycling and stationary trials. Current was measured using a mA-2000 Non-Contact Milliammeter (Bell Technologies, Orlando, FL).

Additionally, at the beginning and end of each experiment, H-reflex and M-wave recruitment curves were constructed during stationary trials. Recruitment curve data provided maximum M-wave (M-max) and H-reflex (H-max) amplitudes, which were used to calculate H-max/M-max ratios.

**Experiment I—effect of arm-cycling load.** Thirteen participants completed the Load protocol. Female participants performed six arm-cycling trials in a random order against one of six loads (27, 793, 1,559, 2,325, 3,091, and 3,857 kPa), whereas male participants performed eight arm-cycling trials in a random order against the same loads (4,623 and 5,389 kPa). Loads ranged from the lowest resistance offered from the hydraulic system to the highest load that could be maintained without excessive fatigue by all participants for the duration of the trial. The range of loads was established as equal increments (n = 6 for females; n = 8 for males) from the baseline lowest resistance to the highest. For three participants, the forces measured at one hand grip across the eight loads were 10 ± 1, 17 ± 1, 22 ± 2, 25 ± 4, 31 ± 2, 34 ± 1, 42 ± 2, and 44 ± 2 N, respectively. For reference, forces generated at 1, 1.25, 1.5, 1.75, and 2 Hz cycling frequencies were 10 ± 1, 15 ± 1, 14 ± 2, 21 ± 5, and 29 ± 3 N, respectively. Control data were obtained from stationary trials performed at three different times throughout the load protocol: 1) prior to any cycling trials (pre); 2) after all of the cycling trials (post); and 3) midway through the cycling trials (mid). Pressure (kPa) corresponding to each cycling trial produced by the custom-built hydraulic ergometer was measured.

Heart rate was measured using a Polar A3 electro-heart rate monitor (Polar Electro Oy, Finland), and peak heart rate during each trial of the protocol was recorded. Heart rate from maximal extension occurred as percentages of estimated maximum heart rate (maximum heart rate = 220 – age). There was no significant difference in the heart rates across the stationary trials (pre, mid, post), allowing heart rate data from the stationary trials to be averaged and collapsed into one stationary control value used for comparisons with the cycling trials. Increases in heart rate from stationary control and each loaded cycling trial were calculated and averaged across males and females separately. These heart rate “increase” values were used to align the six
cycling loads for the females with the six of the eight “best-matched” loaded cycling trials for the males to accommodate for gender differences. This resulted in the female loads of 27, 793, 1,559, 2,325, 3,091, and 3,857 kPa being matched to the male loads of 793, 1,559, and 3,091 and 3,857, 4,623, and 5,389 kPa, respectively. With data aligned based on best-matched heart rate increases, heart rate and SOL background EMG (bEMG) levels, as well as M-wave and H-reflex amplitudes, were compared between genders for all stationary and cycling conditions. No significant differences between genders were found for any of these measures, allowing aligned gender data to be combined for further comparisons. The six “matched” loaded cycling trials were named in ascending order of resistance as Control Load, Load 1 (L1), Load 2 (L2), Load 3 (L3), Load 4 (L3), and Load 5 (L5).

In addition, heart rate and EMG levels were used to ensure that the increments in load were not trivial and were associated with significant changes in physiological cost.

**Experiment II—effect of arm-muscle vibration.** Ten participants completed the Vibration protocol. Participants performed two cycling trials in random order: one with vibration applied to triceps brachii (TB) and one with no vibration. A 3-V eccentric motor-driven vibrator delivered mechanical vibration at 85 Hz with an amplitude of 0.5 mm to the distal tendon of TB ipsilateral to SOL H-reflex stimulation. Similar vibration parameters have been shown to successfully activate Ia afferents of the vibrated muscle tendon (Roll et al. 1989). Data gathered in vibration trials were compared with cycling trials with no vibration. Control data were obtained from stationary trials performed prior to and after the cycling trials.

**Vibration validation.** To be confident that the conditioning vibration applied in the current study was sufficient to modulate the neural control of movement, the influence of the conditioning TB muscle vibration on ipsilateral FCR H-reflex amplitude was evaluated in five participants. The conditioning vibration was applied to the TB tendon in both stationary and arm-cycling trials, and modulation of FCR H-reflex amplitude was compared with trials without vibration. H-reflex and M-wave recruitment curves were constructed, and the FCR H-reflex was elicited in an identical fashion to the SOL H-reflex. The left median nerve was stimulated just proximal to the left medial epicondyle of the humerus near the cubital fossa when the left arm was in the 3-o’clock position, and the reflex response was measured in the FCR muscle. The left wrist and hand were immobilized with a brace throughout the cycling and stationary trials.

**Experiment III—comparison of active and passive arm cycling.** Six participants completed the Passive movement protocol. Participants performed two separate cycling trials (Passive and Active) in random order. In the Passive trial, the participants’ arm ipsilateral to the stimulation was passively rotated by an experimenter, thus making the limb movement involuntary. For each participant, the hand and forearm ipsilateral to the stimulation were secured within a brace affixed to the ergometer crank arm that immobilized the wrist and hand and allowed the arm to cycle without gripping. The contralateral limb remained stationary in the participants’ lap. In a separate Active trial, participants actively cycled with their ipsilateral arm while their contralateral arm remained stationary in their lap. Control data were obtained from stationary trials performed prior to and following movement trials. We are confident that unilateral passive arm cycling represents a valid comparative paradigm since similar significant H-reflex suppression was observed previously with both active unilateral arm cycling and active bilateral arm cycling compared with static (post hoc analysis $P = 0.0004$, and $P = 0.0002$, respectively) (de Ruiter et al. 2010). Also, previously, no significant difference in reflex amplitude was found when comparing bilateral with ipsilateral, unilateral active arm cycling across the cycle path ($P = 0.35$) (de Ruiter et al. 2010) nor when comparing bilateral with ipsilateral, unilateral active arm cycling specifically at the 3-o’clock position (Loadman and Zehr 2007).

**EMG**

With the use of Ag–AgCl self-adhesive electrodes (Thought Technologies), bipolar surface EMG recordings were made bilaterally from the posterior deltoid (PD) and biceps brachii (BB), as well as unilaterally from the left SOL, TA, medial gastrocnemius (MG), vastus lateralis (VL), biceps femoris (BF), and TB for the Load and Vibration experiments. In the Passive cycling experiment, EMG was also recorded from ipsilateral FCR and ipsilateral anterior deltoid (iAD) but not from contralateral BB (cBB).

**Kinematics**

Lightweight electrogoniometers (Biometrics, Cwmfelinfach, Gwent, UK) were used to record kinematic information from the left elbow joint of participants (Load $n = 10$, Vibration $n = 10$, Passive $n = 5$).

**Data Acquisition and Analysis**

With the use of custom-written LabVIEW (Dr. T. Carroll, University of New South Wales) computer programs (National Instruments), data were sampled at 5,000 Hz with a 12-bit analog-to-digital converter. EMG signals were filtered at 100–300 Hz and rectified with the exception of SOL, which was filtered at 100–1,000 Hz and remained unrectified. Twenty sweeps were collected for the trials when a stable M-wave was maintained, whereas 40 sweeps were collected to construct the H-reflex and M-wave recruitment curves. For all sweeps (20 ms prestimulus and 50–60 ms poststimulus), peak-to-peak amplitudes of M-waves and H-reflexes were determined offline using custom-written software (MATLAB, MathWorks, Natick, MA). The rectified prestimulus EMG (20 ms) was used as a measure of muscle activity bEMG at the time of nerve stimulation for all muscles. For each trial, averages of M-waves, H-reflexes, and SOL prestimulus EMG were calculated from the sweeps and normalized to the M-max values obtained for each subject.

**Statistics**

STATISTICA software (StatSoft, Tulsa, OK) was used to conduct separate repeated-measures (RM) ANOVA for SOL M-wave and H-reflex amplitude, heart rate, kinematics, frequency, and bEMG in all muscles to determine the conditioning effects of arm-cycling load, arm-muscle vibration, and passive cycling. Tukey’s honestly significant difference (HSD) test was used for the post hoc analysis of main effects. Planned comparisons were used to evaluate an apriori-determined comparison of H-reflex amplitudes between Control Load and other Load cycling trials, between trials with and without Vibration, and among Active, Passive, and Stationary trials. Student’s $t$-tests were conducted on pre and post M-max, H-max, and M–H ratios from stationary trials. Additionally, to allow for a more direct comparison of the conditioning effects of load and frequency during arm cycling, select SOL H-reflex data from a previous experiment evaluating frequency [1–2.0 Hz cycling trials; frequency experiment, see Hundza and Zehr (2009)] were reanalyzed with RM ANOVA; planned comparisons were used to identify cycling trials, which differed significantly from a 1-Hz cycling trial. Descriptive statistics included means ± SE. Statistical significance was set at $P ≤ 0.05$.

**RESULTS**

**Experiment I—Effect of Arm-Cycling Load**

**Heart rate.** Average heart rate across participants increased from 62 beats/min in the stationary trials to 104 beats/min in the L5 cycling trial. Heart rates were significantly different across loads [F(6,12) = 51.32; $P < 0.00001$]. Post hoc analysis showed that heart rates in the Control Load trial were signifi-
from Experiment I

Fig. 1. Reflex traces from 2 single subjects

SOL H-reflex amplitudes during stationary and loaded arm-cycling conditions. Based on recruitment curve data from stationary trials, there were no significant differences in M-max, H-max, or M-max/H-max ratio over the course of Experiment I. Additionally, there were no significant differences in the SOL M-wave, H-reflex, or bEMG amplitudes across the stationary trials (pre, mid, post), allowing these stationary trials to be averaged and collapsed into one stationary control value used for comparison with the cycling trials.

Arm cycling suppressed SOL H-reflex amplitudes relative to stationary control; however, increases in crank load applied during the cycling trials had little effect on SOL H-reflex amplitude, as can be seen in in Fig. 1A, where the single-subject reflex traces from the cycling trials are displayed. Here, both the H-reflex and M-wave amplitude remained consistent across all of the cycling trials, despite the increasing load. The effect of load on arm cycling is similar between the single-participant data (Fig. 1A) and the group data (Fig. 2A). H-reflex amplitudes for the L1–L5 cycling trials were expressed as percentages of the Control Load cycling trial and were averaged across participants (Fig. 2A). For reference, the H-reflex amplitude for the Control Load is indicated at 100% in Fig. 2A. Averaged H-reflex amplitudes showed a main effect [F(6,10) = 8.05; P < 0.00001; not shown in Fig. 2A] across all trials (i.e., stationary and cycling). The H-reflex amplitudes in all cycling trials were significantly suppressed from stationary control (P < 0.003; Tukey’s HSD). However, planned comparisons showed no significant difference in H-reflex amplitudes between the Control Load cycling trial and any of the L1–L5 cycling trials. That is, SOL H-reflex amplitude was independent from arm-cycling crank load. SOL M-wave and bEMG were well controlled and not significantly different across the trials. The stimulation intensity for all trials was sufficient to evoke a stable M-wave for each participant (average of \( \sim 4.0\% \) of M-max ± 0.65 SE), and the H-reflex was elicited on the ascending limb of the H-I curve (average of 82.1% of H-max ± 3.81).

To allow for a direct comparison between the conditioning effects of load and frequency during arm cycling on the SOL H-reflex in stationary legs, data from a previous experiment evaluating frequency (frequency experiment, see Hundza and Zehr (2009)) were reanalyzed, contrasting H-reflex amplitudes from 1-Hz trials (Control Frequency) to seven cycling trials ranging in frequency from 1.1- to 2.0-Hz trials. Thus the arm-cycling load and the frequency are identical for the Control Load in the current experiment and the Control Frequency (Hundza and Zehr 2009). In Fig. 1B, single-subject reflex traces from the frequency experiment demonstrate that increases in cycling frequency progressively suppressed the H-reflex amplitude. H-reflex amplitudes from 1.1- to 2.0-Hz cycling trials, expressed as percentages of Control Frequency (1.0 Hz), were averaged across participants and plotted in Fig. 2B. The average of H-reflex amplitudes for the Control Frequency trials was therefore 100% and plotted; thus the cycling parameters were identical in the Control Load plot in Fig. 2A and Control Frequency plot in Fig. 2B. There was a main effect for frequency [F(7,10) = 8.74; P < 0.0001], and planned comparisons showed that 1.0 Hz was significantly different from 1.2 (P = 0.05), 1.4 (P = 0.02), 1.5 (P = 0.008), 1.75 (P = 0.01), and 2.0 (P = 0.006)-Hz trials, as indicated in Fig. 2B.

bEMG in leg and arm muscles. Background muscle activity for ipsilateral SOL (iSOL), ipsilateral TA (iTA), ipsilateral VL (iVL), ipsilateral BF (iBF), and ipsilateral MG (iMG) averaged across participants for stationary and cycling trials is displayed in Fig. 3A. RM ANOVAs were performed separately on each muscle across all conditions (i.e., cycling and stationary); a main effect for condition was found for every muscle except SOL (noted above). bEMG activity was maintained at \( \sim 12\% \) of MVC across all trials. No significant differences were found with post hoc analyses (Tukey’s HSD) in bEMG between the Control Load cycling trial and other cycling trials in any muscle. The stationary condition was significantly different from the following arm-cycling trials: L3–L5 in BF (P ≤ 0.04); L5 in MG (P = 0.05); L4 in VL (P = 0.02); and L3 and L4 in TA (P = 0.04).

bEMG levels for ipsilateral PD (iPD), ipsilateral BB (iBB), ipsilateral TB (iTB), contralateral PD (cPD), and cBB muscles averaged across all participants are displayed in Fig. 3B. Each muscle was analyzed independently, and a main effect for condition was found in all arm muscles. Post hoc analysis (Tukey’s HSD) found significant differences in bEMG between the Control Load cycling trial and the following loaded cycling trials: iTB L3 (P = 0.02), L4 (P = 0.03), L5 (P = 0.006); cBB L4 (P = 0.02), L5 (P = 0.0001); iPD L5 (P = 0.01); cPD L4 (P = 0.02), L5 (P = 0.0001); and cBB L5 (P = 0.02; displayed in Fig. 3B). Significant differences were also found in bEMG between the stationary condition and the following loaded cycling trials: Control Load–L5 in cPD, L2–L5 in cBB, and L3–L5 in iPD.

Fig. 1. Reflex traces from 2 single subjects from Experiment I displaying the soleus (SOL) H-reflex modulation across 6 arm-crank loads and 8 arm-cycling frequencies. A: resisted arm-cycling trials include Control Load (L), L1, L2, L3, L4, and L5. B: arm-cycling frequencies include Control Frequency, 1.1, 1.2, 1.3, 1.4, 1.5, 1.75, and 2.0 Hz. Data in B have been redrawn from a previous study (Hundza and Zehr 2009) and reanalyzed for the current comparison. Both the load applied and the arm-cycling frequency are identical between the Control Load and Control Frequency trials. Reflex traces represent averages of 10–15 sweeps recorded during each trial. Stimulus artefact, M-wave, and H-reflex are indicated.

A  Constant H-reflex amplitude across loads

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B  Modulation of H-reflex amplitude across frequencies

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J Neurophysiol • doi:10.1152/jn.00485.2011 • www.jn.org
and iBB \((P \leq 0.05; \text{not indicated in Fig. 3})\). No significant linear correlations were found between SOL H-reflex amplitude and bEMG levels for any arm muscle. [For comparison with arm muscle bEMG activity across the different arm-cycling frequencies, see Hundza and Zehr (2009); Fig. 4B].

Experiment II—Effect of Arm-Muscle Vibration

FCR H-reflex amplitudes with and without vibration of arm muscles. Trials with and without vibration showed that vibration significantly suppressed the maximum FCR H-reflex amplitude (normalized to M-max) with a main effect for vibration \([F(1,4) = 10.09; P = 0.03]\) seen in both the stationary \((P = 0.05)\) and cycling \((P = 0.05)\) conditions in all five participants. The range of FCR H-reflex suppression induced by vibration was 11–71\% during static task and 19–52\% during cycling. In the limb ipsilateral to stimulation, no differences were found between trials with and without vibration across participants for bEMG in FCR, extensor carpi radialis, AD, or PD or for kinematics and cycling frequency. Significant differences were seen between trials with and without vibration for BB \([F(1,4) = 16.25; P = 0.03]\) and TB \([F(1,4) = 18.74; P = 0.02]\). This modulation of FCR H-reflex amplitude generated by applied vibration demonstrated that sufficiency of the stimulus significantly influenced segmental excitability in both stationary and cycling trials.
**SOL H-reflex amplitudes with and without vibration of arm muscles.** SOL recruitment curve data from stationary trials conducted at the beginning and the end of the Vibration experiments showed no significant difference in M-max \((P = 0.41)\), H-max \((P = 0.66)\), or M-max/H-max ratio \((P = 0.34)\). Additionally, there were no significant differences in the SOL M-wave, H-reflex, or bEMG amplitudes across the stationary constant M-wave trials.

Vibration applied during the stationary and cycling trials did not affect the SOL H-reflex amplitude, as can be seen in the single-subject reflex traces displayed in Fig. 4, A and B, respectively. The M-wave amplitude remained consistent across all trials. The H-reflex amplitude is suppressed in cycling compared with stationary trials; however, the H-reflex amplitudes in trials with and without vibration are not different. The lack of suppression of vibration is similar between the single-participant data (Fig. 4, A and B) and the group data (Fig. 5A). H-reflex amplitudes for all trials were expressed as percentages of M-max and averaged across participants (see Fig. 5A). No main effect was found for Vibration but was seen for task (Cycling vs. Stationary) \([F(1,9) = 6.51; P = 0.03]\) and iBB \([F(1,9) = 6.08; P = 0.04]\). SOL bEMG was maintained, on average, at 10.9% ± 0.86 of MVC.

**Experiment III—Passive Arm Cycling**

**SOL H-reflex amplitudes during passive arm cycling.** Based on recruitment curve data of stationary trials pre and post Experiment III, there was no significant difference in M-max,

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**Fig. 4.** Reflex traces from single subjects displaying the SOL H-reflex modulation across Experiments II [Vibration (vib)] and III (Passive vs. Active) trials. A: arm-cycling and stationary trials with and without vibration. B: ipsilateral active and passive arm-cycling and stationary trial. Reflex traces represent averages of 10 sweeps recorded during each trial. Stimulus artefact, M-wave, and H-reflex are indicated.

**Fig. 5.** SOL H-reflex peak-to-peak amplitude for all participants for Experiment II (A, Vibration) and III (B, Passive Cycling) trials. Reflex amplitudes are expressed as a percentage of the M-max. Values are means ± SE across all participants. ME, main effect. Statistically significant differences (\(P < 0.05\)) are indicated by bars.
H-max, or M-max/H-max ratio over the course of Experiment III.

Single-subject data displayed in Fig. 4C show that Passive arm cycling is less suppressed from Stationary compared with Active cycling. H-reflex amplitudes for all trials were expressed as percentages of M-max and averaged across participants (see Fig. 5B), and a main effect for task was found \([F(2,5) = 7.44; P = 0.01]\). H-reflex amplitudes were not different between Stationary and Passive conditions, whereas Active was significantly different from both Stationary (\(P = 0.01\); planned comparison) and Passive (\(P = 0.04\)). When expressed as percentages of Active cycling, Passive cycling was 150% ± 20 SE, and Stationary was 176% ± 35 SE. The M-wave amplitude remained consistent across all trials.

**bEMG in leg and arm muscles**. bEMG amplitudes were not significantly different among Stationary, Active, and Passive trials across participants for iSOL, iTA, iBF, iVL, iTB, iAD, and iDP. A main effect for task was seen in two arm muscles. A significant main effect was found for FCR \([F(2,5) = 19.98; P = 0.001]\) with significant differences between Stationary and Active (\(P = 0.01\)) and Stationary and Passive (\(P = 0.01\)), whereas Active vs. Passive was not different. In addition, a main effect was found for BB \([F(2,5) = 10.86; P = 0.005]\) with significant differences between Stationary and Active (\(P = 0.004\)) and Stationary and Passive (\(P = 0.04\)), whereas Active vs. Passive was not different. SOL bEMG was, on average, 17.1% ± 4.4% of MVC.

**Kinematics and Cycling Frequency for Experiments I, II, and III**

In Experiment I (Load), no significant differences were found across the loaded cycling trials for the elbow-joint position at 3 o’clock or cycling frequency. In Experiment II (Vibration), no significant difference for the elbow-joint position at 3 o’clock or in cycling frequency was found between trials with vibration and those without. The cycling frequency for trials without vibration was 1.18 Hz and 1.22 Hz for trials with vibration. In Experiment III (Passive), stationary, active, and passive trials were not significantly different in kinematics or frequency.

**DISCUSSION**

There are three novel observations in the current study. First, differences in crank load during arm cycling did not significantly affect SOL H-reflex amplitude. Second, arm-muscle vibration during arm cycling also did not significantly affect SOL H-reflex amplitude. Thus in this study, specific modulation of afferent feedback from load and muscle-length receptors in arm muscles had little direct effect on the interlimb modulation of SOL H-reflex amplitude. Lastly, the SOL H-reflex amplitude was not significantly different between stationary and passive arm-cycling conditions, whereas the reflex amplitude was significantly suppressed during active arm cycling compared with passive arm cycling and stationary. Current results, in conjunction with previous findings, suggest that the afferent feedback examined in these studies is not the primary source responsible for SOL H-reflex suppression.

Instead, it appears that central motor commands regulating rhythmic arm cycling—supraspinal or spinal in origin—may be a more dominant source.

**Methodological Considerations**

Consistent low-level background activity (12% ± 0.14 SE, 10.9% ± 0.86 SE, 17.1% ± 4.4 SE of MVC for Experiments I, II, and III, respectively) was maintained in the SOL muscle across all trials to control for motoneuronal pool activation levels and thereby, decrease variability in H-reflex latency and amplitude (Burke et al. 1989; Funase and Miles 1999). M-wave amplitudes (as a percentage of M-max) compared within each experiment were not significantly different across the trials, confirming stimulus constancy throughout the experiment (Brooke et al. 1997; Zehr 2002). Additionally, because there was no significant difference among M-max, H-max, and M-H ratio determined in recruitment curves at the beginning and end of each experiment or between the H-reflex amplitudes evoked at the stable M-wave amplitude in the pre-, mid-, and poststationary trials, stimulus-response consistency across the experiment can be assumed. In the current study, comparisons of activity levels in the heteronymous leg muscles showed no significant differences among: 1) Control Load and other loaded cycling trials; 2) trials with and without vibration; or 3) Stationary, Active, and Passive trials, thereby precluding their potential influence on the SOL H-reflex amplitude (Crone and Nielsen 1994; Hultborn et al. 1987; Morita et al. 1998; Pierrot-Deseilligny and Mazevet 2000) (for a comprehensive review, see Pierrot-Deseilligny and Burke (2005)]. Given the above methodological control, we do not believe the current results to be confounded by methodological issues.

**Independence of SOL H-Reflex Modulation and Arm Crank Load**

The significant changes in heart rate, bEMG in arm muscles, and hand grip forces demonstrate meaningful physiological differences across the loads. The increase in force at the hand grip was 25 N between the highest and lowest load used for both the men and women, whereas the increase between Control Frequency and 2 Hz was 19 N. If afferent feedback related to load were the main source contributing to the H-reflex modulation with increased cycling frequency, then the loads tested here should be adequate to induce H-reflex suppression. Furthermore, H-reflex amplitudes at 1.2 Hz were significantly suppressed from 1 Hz cycling. Therefore, the H-reflex suppression is sensitive to small changes in frequency. The associated difference in the forces between these frequencies was also incremental (only 5 N). This further confirms that if load were in fact the main source contributing to the graded suppression of H-reflex amplitude, the increase in forces at the hand between highest and lowest loads should be more than adequate to detect the influence related to load.

Results from our laboratory have consistently shown significant suppression of the SOL H-reflex amplitude during rhythmic arm cycling compared with stationary trials, which affirms the presence of interlimb neural connections between arms and legs (de Ruiter et al. 2010; Frigon et al. 2004; Loadman and Zehr 2007). In contrast to our hypothesis, the neuromuscular activation associated with arm cycling against different loads did not significantly influence the excitability of this interlimb reflex pathway. Therefore, afferent feedback from load receptors (including type Ia afferents) activated by the increased crank-load and increased arm-muscle activity is not the major source mediating the SOL H-reflex suppression (Frigon et al. 2010).
Further, the increased motor drive required to increase the EMG levels in arm muscles also did not affect the suppression of SOL H-reflex amplitude. Similar to the current results, FCR H-reflex amplitude was not influenced by loading the foot during rhythmic ankle plantarflexion and dorsiflexion (Borroni et al. 2004). In contrast to the current results, SOL H-reflex amplitude evoked during leg cycling was influenced by crank load (Larsen and Voigt 2004; Sakamoto et al. 2004; Zehr et al. 2001). However, comparisons to research, which investigates the effect of crank load applied during leg cycling on SOL H-reflex amplitudes, must be made cautiously. It remains uncertain whether load-related afferent feedback plays the same role in the neural regulation of rhythmic arm vs. leg tasks given the different role load-related feedback plays in neural control of legs vs. arms during typical rhythmic tasks such as walking. In addition, the influence of load-related feedback may differ depending on whether the H-reflex is evoked in the moving limb rather than a stationary limb remote from the movement.

Independence of Arm-Muscle Vibration and SOL H-Reflex Modulation

Consistent with our hypothesis, vibration of the TB tendon did not influence the modulation of SOL H-reflex amplitude. Thus types Ia and II afferent feedback associated with arm cycling does not appear to be the dominant source of the signal mediating the suppression of the H-reflex seen during arm cycling. This is consistent with our previous work, which found no change in H-reflex amplitude when arm cycling at different crank lengths. Afferent feedback would have varied greatly when cycling with different crank lengths due to significant changes in joint range of motion, suggesting that afferent feedback related to arm-muscle length (types Ia and II afferents), rate of change of muscle length (type Ia afferents), or joint position (types II and III afferents) plays a minor role in remote modulation of SOL H-reflex amplitude (Loadman and Zehr 2007).

Limited SOL H-Reflex Suppression with Passive Arm Cycling

During passive movement in the current experiment, the intent was that the movement would be produced by forces external to the participant and was therefore involuntary. This type of passive movement has been suggested to remove or at a minimum, reduce the central motor commands producing the movement (Brooke et al. 1995, 1997), whereas peripheral feedback related to the movement (i.e., joint kinematics) is suggested to be similar between passive and active arm cycling (Brooke et al. 1995; Carroll et al. 2005; Kamibayashi et al. 2010). As seen in the current study, with passive arm movement, some EMG activity remained in arm muscles despite participants’ lack of volitional contribution and attempts to remain relaxed. In fact, the level of muscle activation was not significantly different between the active and passive tasks. Furthermore, differences in muscle activation do not appear to influence the H-reflex amplitude, as demonstrated by similar H-reflex amplitudes across different arm-cycling loads (Experiment 1).

SOL H-reflex suppression differed between passive and active conditions, even though afferent feedback related to movement kinematics would have been similar between the conditions. This limited interlimb effect may be restricted to neural linkages between arms and legs, given that previous work has shown that SOL H-reflex amplitude in the contralateral stationary leg was influenced similarly by both active and passive stepping (Brooke et al. 1995). It is important to note, however, that the H-reflex amplitude in the passive condition was reduced from stationary, although not significantly. Thus it is likely that the movement-related afferent feedback induced a degree of H-reflex suppression during passive arm cycling, and this afferent feedback-related modulation is encompassed within the larger suppression seen during active arm cycling.

Possible Sources of H-Reflex Suppression

Possible sources responsible for the SOL H-reflex suppression seen during arm cycling include afferent feedback and/or central motor commands, either spinal or supraspinal. In combination with the current results, the graded relationship seen between arm-cycling frequency and SOL H-reflex amplitude suggests that the central motor commands required to modify the frequency of arm cycling likely provide a dominant role in the suppression of the SOL H-reflex (Hundza and Zehr 2009; Loadman and Zehr 2007). Furthermore, given that EMG activity is increased in both the load and frequency paradigms (Hundza and Zehr 2009), but suppression of reflex amplitudes only occurs with increased frequency, it can be reasoned that the suppression is related to the central motor commands associated with arm-cycling frequency rather than increased motor drive required to increase the EMG levels. A significant difference between active and passive conditions as well as a lack of differences between stationary and passive conditions provide further support for this line of reasoning.

When investigating the influence of rhythmic ankle movement on reflex modulation in arm muscles, Baldissera and colleagues (Cerri et al. 2003) similarly concluded that the origin for the interlimb reflex modulation was central in nature (i.e., related to the central motor commands for rhythmic movement) rather than kinesthetic. In their further work, they demonstrated that when muscle-to-movement phase-lag was increased by loading the foot during rhythmic movement, timing of H-reflex modulations remained phase linked to the muscular contractions, not to movement. It was concluded that the H-reflex modulation is related to the cortical motor command rather than afferent feedback related to the movement (Borroni et al. 2004).

Alternatively, one could speculate that the observed frequency-dependent SOL H-reflex suppression in this experiment might be explained by phase dependence of H-reflex amplitude modulation (de Ruiter et al. 2010). That is, increases in cycling frequency could shift the position of the hand further along in the movement relative to the 3-o’clock stimulation position enough that it was in a different phase of the movement path when the reflex was elicited. However, this was not the case. When comparing 1 Hz with 2 Hz, the time to travel 1/12 of the cycle path (i.e., one clock position) would be 83 ms and 42 ms, respectively. Based on a reflex latency of 35–40 ms and an estimated afferent arrival time to the spinal synapse of 15–25 ms, even at 2 Hz, the arm would remain between the 3- and 4-o’clock position. Furthermore, based on previous data (de Ruiter et al. 2010), the effect of phase on H-reflex amplitude was only significant when separated by two or more phases. In
addition, the H-reflex suppression is linearly graded with all cycling frequencies (0.03–2 Hz) (Hundza and Zehr 2009), and at the lower frequencies, the hand would have moved less than a few degrees during the duration of the reflex latency across multiple consecutive lower frequencies.

Although the current results suggest that an important contribution to the signal mediating this interlimb communication is central in origin and related to arm-cycling frequency, they do not isolate the loci of the source (i.e., supraspinal or spinal). The H-reflex suppression is likely not related to increased activation of α-motor neurons from direct corticospinal projections, because the EMG activity is similarly increased in both the load and frequency paradigms, but suppression of reflex amplitudes only occurs with an increase in frequency. Furthermore, the neural signal regulating frequency of cycling (i.e., rhythm generation) would, by definition, change across cycling frequencies, whereas it would remain constant across loads. One could, therefore, reason that the difference in the rhythm generation signal between the frequency and load paradigms is a potential source of their different effect on the SOL H-reflex suppression. The neural communication between arms and legs during rhythmic movement has previously been shown to be influenced by frequency of limb movement. In decerebrate cats, the rate of stepping in the front limbs entrained the stepping frequency of the hind limbs to maintain a 1:1 ratio (Akay et al. 2006). Changes in leg cadence influenced arm cadence during simultaneous arm and leg cycling (Sakamoto et al. 2007). Donker and colleagues (2001, 2005) showed that both the stability of the individual limb movements and interlimb coordination increased with increasing velocity. In contrast, manipulation of load affected the individual limb movements but not the interlimb coordination (Donker et al. 2005).

Given that both leg and arm cycling is suggested to be regulated by locomotor central pattern generators (CPGs; for review see de Ruiter et al. (2010); Zehr and Duyssens (2004); Zehr et al. (2007)), and rhythm generation has been ascribed to components of the CPG networks (McCrea and Rybak 2008), one plausible source of the signal mediating the SOL H-reflex suppression could be CPG mechanisms. This argument is also supported by the supposition that the SOL H-reflex suppression seen during arm cycling is mediated by presynaptic inhibition (PSI) of Ia afferents (Frigon et al. 2004) and that PSI is accepted to be a key control mechanism involved in the regulation of rhythmic movement [for review, see Stein (1995)].

GRANTS

Support for this work was provided by a discovery grant to E. P. Zehr from the Natural Sciences and Engineering Research Council of Canada (NSERC).

DISCLOSURES

The authors declare no potential conflicts of interest, financial or otherwise.

AUTHOR CONTRIBUTIONS


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J Neurophysiol • doi:10.1152/jn.00485.2011 • www.jn.org


