Effect of Rhythmic Arm Movement on Reflexes in the Legs: Modulation of Soleus H-Reflexes and Somatosensory Conditioning

Alain Frigon, David F. Collins, and E. Paul Zehr

1Neural Control of Human Movement Laboratory, Department of Integrative Physiology, University of Colorado, Boulder, Colorado 80309; 2Human Neurophysiology Laboratory, Faculty of Physical Education and Recreation, University of Alberta, Edmonton, Alberta T6G 2H9, Canada; and 3Rehabilitation Neuroscience Laboratory, School of Physical Education, University of Victoria, Victoria, British Columbia V8W 3P1, Canada.

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INTRODUCTION

Typically, motor tasks such as walking, running, and swimming demand that the upper limbs move rhythmically with the movement of the lower limbs. The movement of the arms during walking is due to muscular activity and is not simply a passive pendular movement (Jackson 1983; Jackson et al. 1983a,b). Furthermore, when the upper limbs are restrained, the arm musculature still contracts rhythmically (Fernandez-Ballesteros et al. 1965). Interlimb coordination is thought to stem from neural linkages connecting cervical and lumbosacral networks (Dietz 2002). Several recent papers have examined cutaneous and H-reflex modulation and have suggested that the neural control of rhythmic arm movement (Zehr and Haridas 2003; Zehr and Kido 2001; Zehr et al. 2003) is very similar to the control of rhythmic leg movement. It is thought that this control of reflex gain may rely on the output of central pattern generating (CPG) networks (Duyens and Van de Crommert 1998). To produce coordinated movement, some form of linkage between the control of arm and leg movement would be anticipated. However, very little is known about the extent to which the interactions between the arms and legs can influence reflex excitability. Neural pathways linking the upper and lower limbs have been well established in other species and have recently been implicated for the control of human bipedal locomotion (Dietz 2002; Haridas and Zehr 2003). To date, these connections have been demonstrated in neurologically intact human subjects during static motor tasks by stimulating nerves in the arm and recording from muscles in the leg or vice versa (Gassel and Ott 1973; Meineck and Piesiur-Strehlow 1981; Piesiur-Strehlow and Meineck 1980; Zehr et al. 2001a). Calancie and colleagues (Calancie 1991; Calancie et al. 1996, 2002) reported similar responses in arm muscles in cervical spinal cord injured subjects following stimulation of mixed nerves in the leg. Despite the important role for spinal pathways in coordinating movements of all four limbs in quadrupeds (Miller et al. 1973; Schomburg and Behrends 1978), little is know about interactions between arm movement and reflex excitability in the legs of humans. A few studies have examined the effects of arm position on soleus H-reflex amplitude. Soleus H-reflexes in the right leg were facilitated when the right shoulder was in flexion (45–90° in front of the mid-axillary line) and inhibited when the left shoulder was positioned similarly (Delwaide et al. 1973, 1977; Eke-Okoro 1994). Passive movement of the elbow joint (flexion/extension) was shown to facilitate (Hiraoka and Nagata 1999), while arm swing inhibited soleus H-reflexes (Hiraoka 2001). However, these studies did not control several important factors such as stimulus constancy...
(M-wave size) and activation level of the soleus, and thus the results should be interpreted with caution.

The first objective of this study was to test the hypothesis that rhythmic movement of the arms alters reflex transmission in the soleus H-reflex pathway. It is generally accepted that presynaptic inhibition (PSI) is one of the major control mechanisms associated with rhythmic movement (for a review see Stein 1995) and is known to strongly modulate this reflex pathway (Brooke et al. 1997). The second objective was to test the hypothesis that changes in PSI were responsible for the modulation of soleus H-reflexes. Stimulation of the sural nerve facilitates soleus H-reflexes by reducing PSI (Demaire et al. 1989; Iles 1996), while CP nerve stimulation reduces soleus H-reflexes by increasing PSI (Capaday et al. 1995; Iles 1996; Zehr and Stein 1999). Thus using conditioning stimuli known to decrease (cutaneous input from the sural nerve) or increase (group I input from the antagonist muscle via common peroneal nerve) the PSI of the soleus H-reflex pathway was assessed.

METHODS

Subjects

Ten subjects, 22–43 yr of age, with no known history of neurological disorders, participated in this study. The subjects gave written consent to participate under the sanction of the Health Research Ethics Board at the University of Alberta. The experiments were conducted in accordance with the Declaration of Helsinki.

Protocol

The experimental set-up is illustrated in Fig. 1. Subjects were seated in a chair with their backs supported. Hip, knee, and ankle angles were set at approximately 90, 110, and 90°, respectively. Both feet were placed securely on metallic blocks. Rhythmic arm cycling was performed in a clockwise direction (viewed from the right side of the body) at a comfortable pace (approximately 60 rpm) using a custom-made hydraulic arm ergometer (described in Zehr et al. 2003) positioned directly in front of the subjects. Restraints were placed around the trunk, right thigh, and right foot to minimize unwanted movement. For each trial, subjects maintained a consistent low-level tonic contraction (≈10% maximum voluntary contraction (MVC)) of their right soleus muscle using visual feedback of the rectified and filtered EMG signal.

Nerve stimulation (see Nerve stimulation) was delivered during cycling and static trials at two arm positions (see Fig. 1). 1) Right elbow extended and shoulder flexed (~70° in front of the mid-axillary line; hereafter referred to as shoulder flexion). In this position the right arm was halfway through the downstroke of the right arm corresponding to the 3 o’clock position described in previous studies (Zehr and Kido 2001; Zehr et al. 2003). 2) Right elbow flexed and shoulder extended (~10° behind the mid-axillary line; hereafter referred to as shoulder extension). In this position the right arm is halfway through the upstroke corresponding to the 9 o’clock position described previously. At all times, the left arm was 180° out of phase with the right arm. These positions were chosen because previous studies demonstrated significant effects of similar shoulder positions on reflex excitability in the legs (Delwaide et al. 1973, 1977; Eke-Okoro 1994).

Nerve stimulation

All nerves were stimulated with bipolar surface electrodes using a Grass S88 (Grass Instruments, AstroMed) stimulator connected in series with SIU5 isolator and CCI1 constant current units. Stimulation was delivered approximately once every two to three cycles during cycling and pseudo-randomly between 1 and 3 s during static trials.

SOLEUS H-REFLEX. The tibial nerve was stimulated with single 1 ms square-wave pulses delivered over the right popliteal fossa. At the start of each experiment, M-wave/H-reflex recruitment curves were constructed from responses to 75 stimuli delivered at intensities ranging from below response threshold to >1.5 times that required to produce a maximal M wave. From these data the maximal M-wave (M max) was calculated as the mean of the three largest M-wave values. These data were also used to determine the M-wave amplitude that occurred simultaneously with an H-reflex on the ascending limb. For the remainder of the experiment, stimulation intensity was set to evoke an M-wave of approximately this size (~10% M max). M-waves were monitored on-line, and stimulation intensity was adjusted occasionally to maintain consistent amplitude and stimulation current was measured (mA-2000 Noncontact Milliammeter, Bell Technologies, Orlando, FL).

SURAL NERVE AND COMMON PERONEAL NERVE. The sural nerve was stimulated at the ankle just below the lateral malleolus with a train of five 1-ms pulses delivered at 300 Hz at two times the radiating threshold. Radiating threshold was defined as the lowest stimulation intensity required to evoke a clear paraphasia radiating throughout the whole innervation territory of the nerve (Zehr et al. 1998). The interval between the sural nerve conditioning and the test H-reflex (C-T interval) was 80 ms.

The CP nerve was stimulated with single 1-ms square-wave pulses just distal to the head of the fibula at 1.5 times the motor threshold (Capaday et al. 1995; Zehr and Stein 1999). Motor threshold was defined as the weakest stimulation that produced a muscle twitch in tibialis anterior. The C-T interval was 100 ms.

During cycling and static trials, reflexes were evoked by delivering nerve stimulation in three ways: 1) Tibial nerve to evoke an H-reflex in soleus; 2) sural nerve + tibial nerve to reduce PSI of the H-reflex; and 3) CP + tibial to increase PSI of the H-reflex. Additionally, to evaluate the postsynaptic effects of the conditioning stimulus on the soleus, sural and CP nerve stimulations were delivered alone. Each type of nerve stimulation was delivered in a separate set of trials, and the order of the trials was randomized across subjects.

EMG

EMG was recorded with surface recording electrodes (Vermed, Bellows Falls, VT). EMG signals were preamplified and band-pass filtered at 30–3,000 Hz (P511 Grass Instruments, AstroMed). EMG signals were full-wave rectified except for the ipsilateral soleus. The anterior deltoid (AD), soleus (SOL), and tibialis anterior (TA) were recorded bilaterally. The vastus medialis (VM), biceps femoris (BF), biceps brachii (BB), and flexor carpi radialis (FCR) were recorded on the right side only. BF was only recorded in five subjects; BB and FCR were recorded in nine subjects.
from all 20 sweeps in each condition for M-waves, H-reflexes from the soleus. Met al. 2001b). Responses were normalized to responses at early (50 ms) subtracted EMG traces from the soleus by analyzing for peak reflexes, either the sural or CP nerve were quantified for each subject from subtracted EMG traces from the soleus by analyzing for peak responses at early (50–80 ms) and middle (80–120 ms) latencies (Zehr et al. 2001b). Responses were normalized to $M_{\text{max}}$ values obtained from the soleus.

**EMG analysis**

**EMG ACTIVITY DURING MOVEMENT.** EMG activity from each muscle was recorded during all trials to determine the level of muscle activation during the different tasks and conditions. The prestimulus (50 ms) EMG was rectified, averaged, and used as a measure of muscle activity at the time of nerve stimulation.

**H-REFLEXES.** Peak-to-peak amplitudes of M-waves and H-reflexes were determined off-line (custom-written software, MatLab, Nantick) from the single unrectified sweeps of ipsilateral soleus EMG. For each subject, M-waves and H-reflexes were normalized to the corresponding $M_{\text{max}}$ to reduce inter-subject variability. Averages were calculated from all 20 sweeps in each condition for M-waves, H-reflexes, and prestimulus EMG in the right soleus.

**RESPONSES EVOCKED BY SURAL AND CP NERVE STIMULATION.** To assess whether changes in H-reflex amplitude induced by the conditioning methods were due to changes in motoneuronal excitability (change in EMG of soleus), responses in soleus evoked by stimulating either the sural or CP nerve were quantified for each subject from subtracted EMG traces from the soleus by analyzing for peak responses at early (50–80 ms) and middle (80–120 ms) latencies (Zehr et al. 2001b). Responses were normalized to $M_{\text{max}}$ values obtained from the soleus.

**Statistics**

Using STATISTICA software (StatSoft), a three-factor (3 conditions $\times$ 2 tasks $\times$ 2 positions) with repeated measures ANOVA was used to identify significant effects of conditioning, task, and position (independent variables) on the amplitude of soleus H-reflexes, M-waves, prestimulus EMG levels, and responses evoked by sural and CP nerve stimulation (dependent variables). Planned comparisons were used by using the $t$-ratio (described as the difference between the pair of means of interest and the SE of the comparison using the error mean square from the ANOVA) to evaluate specific differences between conditions. Descriptive statistics are given as means $\pm$ SE. An $\alpha$ level of 0.05 was used for statistical significance.

**RESULTS**

The experiments were designed to investigate the effect of arm cycling on the amplitude of soleus H-reflexes. The mean amplitudes of the unconditioned H-reflex during static trials at each position are used for the control values. The percentages below are expressed relative to the corresponding control values recorded at the shoulder flexion and extension positions.

**Soleus H-reflexes during arm cycling**

The effect of arm cycling on the unconditioned soleus H-reflex is shown for a single subject (at shoulder flexion) in Fig. 2. Despite similar M-waves and EMG level (data not shown), H-reflex amplitude in this subject was reduced during arm cycling. For the group ($n = 10$), soleus H-reflexes were significantly smaller (main effect for task: $P = 0.0025$) during arm cycling compared with static controls. The bars in Figs. 4 and 5 show the amplitudes of the unconditioned and conditioned H-reflexes ($A$, M-waves ($B$), and prestimulus background EMG ($C$) at the shoulder flexion and extension positions, respectively. All data are expressed as a percent of the static unconditioned value (left bars). Black and gray bars represent amplitudes during static positions and arm cycling, respectively. Asterisks indicate a significant difference between static and arm cycling, and crosses indicate significant difference compared with unconditioned static controls. On average, unconditioned soleus H-reflexes during arm cycling were 78.3% ($P = 0.0005$; Fig. 4A, left bars) and 91.2% ($P = 0.0178$; Fig. 5A, left bars) of controls at shoulder flexion and extension, respectively.

**Effects of conditioning the soleus H-reflex**

**STATIC TRIALS.** The effects of conditioning stimuli on soleus H-reflexes with the upper limbs held stationary at shoulder extension are shown for a single subject in Fig. 3. Facilitation induced by sural nerve (dotted line) and inhibition (gray line) evoked by CP nerve stimulation can be seen. Averaged across all subjects, soleus H-reflexes conditioned with sural nerve stimulation were 112.3% at shoulder flexion and 114.5% at shoulder extension. This effect was significant at shoulder extension ($P = 0.0069$; Fig. 5A, middle bars) but not at shoulder flexion ($P = 0.1143$, Fig. 4A, middle bars). CP conditioning significantly reduced the amplitude of soleus H-reflexes compared with control amplitudes at the two positions. Reflexes were on average 70.3% at shoulder flexion ($P = 0.0019$; Fig. 4A, right bars) and 84.3% at shoulder extension ($P = 0.0069$; Fig. 5A, right bars) relative to control amplitudes.
tudes. The reduction was significant at shoulder flexion and extension.

ARM CYCLING. During arm cycling, soleus H-reflexes with sural nerve conditioning as a percentage of control values were 80.4% at shoulder flexion (P = 0.0262; Fig. 4A, middle bars) and 101.4% at shoulder extension (P = 0.5940; Fig. 5B, middle bars). For CP conditioning during arm cycling, H-reflexes were on average 61.2% and 75.2% of control values at shoulder flexion (P = 0.0007; Fig. 4A, right bars) and extension (P = 0.0055; Fig. 5A, right bars), respectively.

Effect of arm position on the amplitude of soleus H-reflexes

To determine whether arm position influenced H-reflex amplitude, data between the shoulder flexion and extension positions were compared during static and arm cycling trials. There was no significant difference between H-reflexes evoked at shoulder flexion and extension during static trials (main effect, P = 0.3442). During arm cycling, H-reflexes evoked at shoulder extension were significantly larger than at shoulder flexion (main effect, P = 0.0091). Soleus H-reflexes conditioned with CP nerve stimulation during cycling trials were significantly larger at shoulder extension compared with shoulder flexion (P = 0.0070).

Responses to sural and CP nerve stimulation

Across the group early and middle latencies (data not shown) reflexes evoked by stimulation of the sural and CP nerves were not significantly different between static and cycling trials. Therefore changes in rectified and averaged EMG (reflecting motoneuron pool excitability) evoked by stimulating sural and CP nerves is not significantly altered by rhythmic movement of the arms.

EMG and M-wave amplitudes during H-reflex trials

There were no significant differences in prestimulus background EMG amplitudes for lower limb muscles (SOL, TA, BF, VM) between trials irrespective of conditioning or arm cycling. For all trials, the amplitude of M-waves and background prestimulus EMG of the soleus were not significantly different (see Figs. 4, B and C, and 5, B and C). Arm cycling, however, resulted in significant differences in arm muscle EMG amplitudes (P < 0.05). EMG activity of muscles of the ipsilateral limb (iAD, iBB, iFCR) were significantly higher when the ipsilateral arm was moving through the shoulder extension position, while EMG amplitude of contralateral AD was significantly higher when the ipsilateral was moving through the shoulder flexion position during arm cycling.

DISCUSSION

There are two main findings to this study. First, rhythmic cyclical movement of the upper limbs significantly reduces the amplitude of soleus H-reflexes compared with stationary controls. Second, this modulation arises prior to the motoneuronal membrane, probably by presynaptic inhibition of IA afferent terminals.

FIG. 4. H-reflex (A), M-wave (B), and soleus EMG (C) data averaged across all subjects (n = 10) recorded at the shoulder flexion position. Values are expressed as a percent of the control value (static, unconditioned at shoulder flexion). *Significant differences between static and cycling trials. †Significant differences compared with the unconditioned static control value. Shown are means ± SE.

FIG. 5. H-reflex (A), M-wave (B), and soleus EMG (C) data averaged across all subjects (n = 10) recorded at the shoulder extension position. Values are expressed as a percent of the control value (static, unconditioned at shoulder extension). *Significant differences between static and cycling trials. †Significant differences compared with the unconditioned static control value. Shown are means ± SE.
Methodological considerations

In this study, the amplitude of the direct motor response (M-wave) was used as indication of the constancy of the afferent test volley. By maintaining a similar sized M-wave for all comparisons, it is assumed that the afferent volley also remains constant (Brooke et al. 1997; Dietz et al. 1990; Mayer and Mawdsley 1965; Zehr et al. 2003). Furthermore, H-reflex and M-wave amplitudes are typically normalized to $M_{\text{max}}$ to reduce inter-subject variability (Zehr 2002). However, $M_{\text{max}}$ can change over the course of an experiment (Crone et al. 1999) and at different limb positions (Gerilovsky et al. 1989).

To minimize the impact of these effects in this study, the order of the different trials was randomized across subjects and the position of the ankle (thus soleus muscle length) was the same for all trials. Also, the activation level of the motoneuronal pool profoundly influences H-reflex amplitude (Capaday and Stein 1987a,b). To ensure a consistent level of soleus motoneuronal activation in this study, subjects maintained a similar plantar flexion contraction for all trials. Heteronymous sources such as reciprocal or recurrent inhibition (Crone et al. 1987; Morita et al. 2001; Petersen et al. 1999; Rossi et al. 1994; Tanaka 1974) can affect H-reflex amplitude. However, there were no significant differences in EMG activity of leg muscles between tasks; thus it is improbable that the modulation was the result of heteronymous effects from leg muscles, at least arising from the muscles that we recorded (VM, BF, TA).

Moreover, small and large H-reflexes are affected differently by excitatory and inhibitory conditioning signals due to the involvement of different motor unit populations (Crone et al. 1990). However, Zehr et al. (2001b, 2003) showed that reflexes on three different parts of the M-H recruitment curves were modulated similarly during movement, and thus in this study, H-reflexes were only sampled on the ascending limb of the M-H curve in each condition.

Modulation of reflexes in leg muscles induced by arm movement

There is some evidence that passive flexion/extension movement at the elbow joint (Hiraoka and Nagata 1999), rhythmic arm swinging (Hiraoka 2001; Hiraoka and Nagata 1999), and static positioning (Delaide et al. 1973, 1977; Eke-Okoro 1994) of the arms influences the amplitude of the human soleus H- and stretch reflexes. However, due to several methodological concerns, the source of the modulation is not clear.

Hiraoka (2001) found that soleus H-reflexes were reduced (~10%) during arm swing but only when the shoulder was extended beyond the mid-axillary line; it was proposed that this modulation was due to lengthening of the anterior deltoid (AD) during the backward swing and the onset of the forward swing. With arm cycling, we also found a reduction (~9%) of H-reflexes during shoulder extension, but H-reflexes were also significantly depressed (~22%) during shoulder flexion (see Fig. 1 for arm positions). Therefore AD stretch alone cannot explain our findings since the reduction in H-reflex amplitude was also observed when AD was in a shortened position. In this study, although unconditioned soleus H-reflexes were significantly reduced at two positions during arm cycling compared with static controls (see Figs. 4A and 5A), there was no significant difference in reflex amplitude between shoulder flexion and extension during static trials. Previous studies (Delaide et al. 1973, 1977; Eke-Okoro 1994) have shown that H- and stretch reflexes of the soleus are facilitated (~10%) during static shoulder flexion (i.e., arm in front 45–90°) and inhibited (~7%) during static shoulder extension (i.e., arm behind 30°). We did not find an effect of arm position on unconditioned soleus H-reflex amplitude during static trials. This discrepancy from previous findings may be due to slightly different arm positions or lack of adequate controls in previous studies. Our results suggest that the reduction of soleus H-reflexes is the result of arm cycling (task-dependence) and not the position of the arm in the cycle (phase-dependence). We acknowledge, however, that we only sampled at two positions in the cycle, and a more robust phase-dependent effect may have been present if more positions were recorded. Furthermore, since the H-reflex modulation occurred at similar levels of soleus EMG and responses from sural and CP nerves evoked alone were unaffected by arm cycling, we suggest that arm cycling acts via presynaptic inhibition (see Possible pathways below).

Interaction between arm movement and somatosensory conditioning of the soleus H-reflex pathway

The modulation of H-reflexes recorded at similar contraction levels is due predominantly to PSI of the afferent volley (Brooke et al. 1997; Rudomin and Schmidt 1999; Stein 1995). One way to assess changes in PSI is to use methods to condition the H-reflex. Stimulation of the sural nerve at C-T intervals of 70–90 ms decreases PSI of the soleus H-reflex pathway (Demaire et al. 1989; Iles 1996), while CP nerve stimulation at C-T intervals of 100–120 ms increases PSI of this pathway (Capaday et al. 1995; Iles 1996; Zehr and Stein 1999). In these experiments, we tested whether arm movement influenced the effect of sural and CP conditioning (i.e., the segmental level of IA PSI) on the soleus H-reflex. Sural nerve stimulation facilitated the amplitude of the H-reflex during static contraction (Delaide and Crenna 1984; Demaire and Ciancia 2000; Delwaide et al. 1989; Honore et al. 1983; Lebizec et al. 1983). However, cutaneous reflexes evoked by sural nerve stimulation were not significantly different between static and cycling trials. However, soleus H-reflexes conditioned with sural nerve were significantly reduced during arm cycling compared with static trials with sural nerve conditioning (see Figs. 4A and 5A, middle bars). Therefore despite the facilitation induced by sural nerve stimulation, the reduction in soleus H-reflexes resulting from the arm cycling was still present. Furthermore, at the shoulder extension position (see Fig. 5A, middle bars), sural nerve stimulation removed the inhibition resulting from arm cycling. H-reflex facilitation during arm cycling, in effect, returned the sural conditioned value to unconditioned static values. In other words, H-reflex amplitude during arm cycling combined with sural nerve stimulation is equivalent to that observed in static unconditioned trials. This effect is not clearly identified at the shoulder flexion position since significant facilitation of soleus H-reflexes with sural nerve conditioning was not achieved (see Fig. 4A, middle black bar). This is evidence that arm cycling and sural nerve conditioning share a common presynaptic pathway.

Responses evoked with CP nerve stimulation were not significantly different from static to arm cycling. However, soleus
H-reflex amplitude with CP nerve conditioning was reduced with arm cycling compared with CP conditioned static trials at shoulder flexion (Fig. 4) but not at shoulder extension (Fig. 5). The interaction between arm cycling and CP nerve conditioning resulting in a larger decrease in H-reflex amplitude suggests that they both affect PSI of the soleus H-reflex pathway.

**Possible pathways of reflex modulation**

Several pathways could be responsible for the modulation of soleus H-reflexes during arm cycling. As illustrated in Fig. 6, supraspinal and propriospinal inputs and CPG activity may all be involved in modulating PSI of the afferent pathway to the soleus motoneuronal (MN) pool. 

Supraspinal and propriospinal pathways influence the excitability of the PSI pathway and the motoneuronal pool (see Fig. 6, top, thick solid black lines). Studies using transcranial magnetic stimulation (TMS) and voluntary contraction of the homonymous muscle have shown that descending input from supraspinal centers can influence the PSI on this pathway during static contractions (Iles 1996; Iles and Roberts 1987; Valls-Sole et al. 1994). The cancellation of the CP induced PSI of soleus H-reflexes by the Jendrassik maneuver is thought to be due to converging input from supraspinal centers and CP nerve on the same presynaptic pathway (Zehr and Stein 1999). Furthermore, soleus H-reflexes are facilitated by stimulating the ulnar nerve (Meinck and Piesiur-Strehlow 1981) and the median nerve (Kagamihara et al. 2003) at the elbow. Kagamihara et al. (2003) attributed this facilitation to reducing PSI through a long-loop pathway reaching levels at least as rostral as thepons. Along with supraspinal influences, propriospinal pathways may also be involved. Based on short latency (<45 ms) interlimb responses reported by several authors (Gassel and Ott 1973; Meinck and Piesiur-Strehlow 1981; Piesiur-Strehlow and Meinck 1980; Zehr et al. 2001a) and the presence of interlimb responses in spinal cord injured subjects (Calancie 1991; Calancie et al. 1996, 2002), it is likely that propriospinal pathways coupling the cervical and lumbosacral enlargements of the spinal cord contribute to interlimb coordination. Interlimb reflex activity from 50 to 60 ms is likely confined to the spinal cord, while latencies of approximately 110 ms probably includes both spinal and supraspinal pathways (Calancie et al. 2002; Christensen et al. 2000). Propriospinal connections have been found in several species including humans (Nathan et al. 1996), and direct coupling of the cervical and lumbosacral enlargements of the human spinal cord could convey afferent feedback from several upper limb muscles during arm movement.

Input from antagonist muscles (Capaday et al. 1995; Iles 1996; Iles and Roberts 1987; Zehr and Stein 1999) and from cutaneous afferents of the leg (Demaire et al. 1989; Iles 1996) can alter the level of PSI at the IA terminal to soleus motoneurons. Figure 6 shows the CP nerve with excitatory connections to the PSI interneuron and the sural (SU) nerve with inhibitory connections. Iles (1996) showed that activation of cutaneous afferents from the sural nerve reduced the PSI of soleus IA afferents induced by stimulating the CP nerve, suggesting convergence on a common presynaptic pathway. Joint, cutaneous, and muscle receptors from the arms (Fig. 6, dotted box and lines) might send descending signal via supraspinal or propriospinal pathways on the presynaptic machinery of the lower limbs. The pattern of activation of several arm muscles during arm cycling is rhythmically modulated according to the position in the movement cycle (Zehr and Chua 2000; Zehr and Kido 2001; Zehr et al. 2003). Muscles that exhibit the largest modulation are the shoulder extensors (iAD, cAD), the elbow flexors (BB), and extensors (triceps) (Zehr and Kido 2001). In this study, however, the reduction in soleus H-reflexes was observed at two positions in the cycle where activation of arm muscles is markedly different; it is unlikely that activation of any one muscle is responsible for the modulation.

Perhaps a CPG activated during arm cycling is responsible for changing the PSI at the IA afferent terminal (Fig. 6, gray box and lines). Recent studies have shown that reflex modulation in the arms and legs is similar, suggesting similar control mechanisms (Zehr and Haridas 2003; Zehr and Kido 2001; Zehr et al. 2003). The finding that the modulation is present during active, but not passive, movements suggests that CPG networks may be involved (Zehr and Haridas 2003; Zehr et al. 2003). It is likely that CPGs at the cervical and lumbosacral level are coupled during locomotion and that they produce synchronous outputs for interlimb coordination and interlimb reflex modulation (Guadagnoli et al. 2000). Outflow from one or more CPGs could gate the activity of reflexes in the arms and legs during coordinated movements of both pairs of limbs. Other possibilities include descending voluntary motor com-

![FIG. 6. Possible pathways that influence presynaptic inhibition (PSI) and the excitability of the soleus motoneuronal (MN) pool. Excitatory and inhibitory connections are shown with an open triangle and a filled black circle, respectively. At the bottom is a simplified H-reflex pathway from primary muscle spindles (group IA) synapsing with α-motoneurons of the soleus. PSI changes the excitability of this pathway. PSI and the MN are controlled by supraspinal and propriospinal (thick black lines), and CPG pathways (gray lines). Input from the common peroneal (CP) and sural (SU) nerves have excitatory and inhibitory connections to PSI, respectively.](attachment://image.png)
mands or anticipatory postural adjustments associated with planned movements.

**Functional implications**

In the cat, the function of interlimb pathways coupling cervical and lumbar enlargements in the spinal cord has been ascribed to coordinating fore- and hindlimbs during locomotor activities (Miller and van der Meche 1976). In humans, locomotor tasks such as walking, running, and swimming also require the coordinated movement of our arms and legs. It has recently been shown that stimulating peripheral nerves in the arms changes the excitability of motoneuronal pools in the legs during both static motor tasks (Kagamihara et al. 2003; Zehr et al. 2001a) and walking (Haridas and Zehr 2003) by activating interlimb pathways that couple cervical and lumbar sacral regions of the spinal cords. The present demonstration that rhythmic arm cycling can change the gain of H-reflex pathways in the leg provides evidence that movement of the arms also activates interlimb pathways. Functional connections between cervical and lumbar sacral regions of the spinal cord in humans are influenced by movement and it is likely that they are involved in interlimb coordination (Dietz 2002; Dietz et al. 2001; Haridas and Zehr 2003). It is suggested that rhythmic arm swing contributes to the pattern of H-reflex modulation reported during human walking and running (Capaday and Stein 1986, 1987b; Simonsen and Dyhre-Poulsen 1999). We speculate that this may form a “background” of general bias regulation in reflex excitability that is fine tuned at the segmental level according to the appropriate requirements for gait. This speculation remains to be confirmed or rejected by future experimentation.

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