Effects of Leg Pedaling on Early Latency Cutaneous Reflexes in Upper Limb Muscles

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1United Graduate School of Education, Tokyo Gakugei University, Tokyo, Japan; 2Department of Health and Sports Sciences, Faculty of Education, Chiba University, Chiba, Japan; 3Department of Rehabilitation for Movement Functions, Research Institute, National Rehabilitation Center for Persons with Disabilities, Saitama, Japan; and 4Rehabilitation Neuroscience Laboratory, University of Victoria, Victoria, British Columbia, Canada

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Sasada S, Tazoe T, Nakajima T, Zehr EP, Komiyama T. Effects of leg pedaling on early latency cutaneous reflexes in upper limb muscles. J Neurophysiol 104: 210–217, 2010. First published May 5, 2010; doi:10.1152/jn.00774.2009. The functional coupling of neural circuits between the upper and lower limbs involving rhythmic movements is of interest to both motor control research and rehabilitation science. This coupling can be detected by examining the effect of remote rhythmic limb movement on the modulation of reflex amplitude in stationary limbs. The present study investigated the extent to which rhythmic leg pedaling modulates the amplitude of an early latency (peak 30–70 ms) cutaneous reflex (ELCR) in the upper limb muscles. Thirteen neurologically intact volunteers performed leg pedaling (60 or 90 rpm) while simultaneously contracting their arm muscles isotonically. Control experiments included isolated isometric contractions and discrete movements of the leg. ELCRs were evoked by stimulation of the superficial radial nerve with a train of rectangular pulses (three pulses at 333 Hz, intensity 2.0- to 2.5-fold perceptual threshold). Reflex amplitudes were significantly increased in the flexor carpi radialis and posterior deltoid and significantly decreased in the biceps brachii muscles during leg pedaling compared with that during stationary isometric contraction of the lower leg muscles. This effect was also sensitive to cadence. No significant modulation was seen during the isometric contractions or discrete movements of the leg. Additionally, there was no phase-dependent modulation of the ELCR. These findings suggest that activation of the rhythm generating system of the limbs affects the excitability of the early latency cutaneous reflex pathways in the upper limbs.

INTRODUCTION

Many rhythmic movements in humans and quadruped animals are thought to be regulated by neural systems that are distinct from those for voluntary movements (Alstermark et al. 2007; Dietz 2002; Komiyama et al. 2000; Orlovsky et al. 1999; Zehr et al. 2004). For the regulatory systems of human rhythmic movements, one of the most intriguing issues is how rhythm generating systems for the upper and lower limbs, which would encompass spinal and supraspinal neural mechanisms, interact with each other. Although diagonal out-of-phase rhythmic movements of the arms and legs are rudimentary for human walking, we can also walk while holding items in our hands. Therefore it is likely that the interlimb neural coupling between the upper and lower limbs can be changed, depending on the motor task or biomechanical constraints (Klimstra et al. 2009). Previous studies demonstrated the existence of interlimb cutaneous reflexes even when the subject was sitting still (Zehr et al. 2001). In addition, neural coupling during rhythmic movement has been shown, although it appears to depend strongly on the motor task (Balter and Zehr 2007; Wannier et al. 2001; Zehr et al. 2007). These findings suggest the existence of intrinsic neural systems for formulating interlimb coordination for the persistence of locomotion across species (Zehr et al. 2009).

During human locomotion, such as walking or simultaneous arm and leg pedaling, the amplitude of the cutaneous reflexes in the arm muscles is strongly modulated and this modulation depends on the functional locomotor phase of the legs (Balter and Zehr 2007; Carroll et al. 2005; Sakamoto et al. 2006; Zehr et al. 2007). Importantly, amplitude modulation (AM) related to electromyographic (EMG) background activity is quite different when contracting rhythmically compared with static activation (Carroll et al. 2005; Zehr et al. 2007). The separation of the reflex amplitude from the background EMG amplitude has previously been ascribed to the activity of a rhythm generating system subserving the premotoneuronal gating of cutaneous afferent feedback (see Zehr et al. 2007 for an extensive discussion). Therefore findings of independence between cutaneous reflex amplitudes and background EMG levels further suggest the contribution of a rhythm generating system to the coupling between the upper and lower limbs. However, the AM of the interlimb cutaneous reflexes is smaller than that of the intralimb reflexes and could easily be masked by effects arising when the tested limb is rhythmically moving. Thus it is uncertain whether the reflex circuitry responsible for the cutaneous reflexes in the rhythm generating systems of the upper limbs can be driven when the upper limb is stationary and the lower limb moves rhythmically. This question has not been tested so far by using the AM of cutaneous reflexes, which have been demonstrated to be useful probes for investigating the function of rhythm generating systems in humans (Burke 1999; Zehr and Duysens 2004; but see Dragert and Zehr 2009; Sakamoto et al. 2006).

To investigate coupling between the upper and lower rhythm generating systems, we analyzed the AM induced by movement on the cutaneous reflexes in the arm muscles following the application of low-threshold, nonnoxious, electrical stimulation of a nerve in the hand. Previous studies have focused on the modulation of the middle latency cutaneous reflex (MLCR) (onset latency: ~70–120 ms; Balter and Zehr 2007; Haridas and Zehr 2003; Sakamoto et al. 2006; Zehr et al. 2007). The early latency (<70 ms) cutaneous reflex (ELCR) can also be
evoked in the upper arm muscles and may be mediated differentially from the MLCR (Zehr and Chua 2000; Zehr and Kido 2001; Zehr et al. 2001). Zehr and colleagues demonstrated that the ELCR and the MLCR were modulated in a phase-dependent manner and their amplitudes were not proportional to the background EMG activity during rhythmic locomotor movement (for review, see Zehr and Duyssens 2004). There is thus a strong suggestion that circuits that influence the amplitude of the ELCR and the MLCR are under the control of rhythm generating systems. However, modulation of the ELCR in stationary arm muscles during rhythmic leg pedaling has not been tested in detail. We hypothesized that if the ELCR in the arm was under the control of a rhythm generating system and if there was an interaction between the rhythm generating systems of the upper and lower limbs, then the reflex amplitude in the stationary arm muscles would be modulated by the rhythmic movement of the legs. Therefore the purpose of the present study was to investigate to what extent the ELCR in the upper limb is modulated during rhythmic movement of the lower leg.

METHODS

Subjects and experimental setting

Thirteen subjects aged 21–49 yr participated in the present study. All subjects gave their informed consent according to the Declaration of Helsinki before participating in the experimental procedures. No subject had a neurological deficit nor had been involved in any resistance training programs. The procedures of the present study were approved by the local ethics committee, Faculty of Education, Chiba University.

Nerve stimulation

ELCRs were evoked by applying a burst (3 × 1.0 ms pulses at 333 Hz) of rectangular electrical stimuli to the right superficial radial (SR) nerve at the wrist. Electrical pulses were delivered from a constant-current stimulator (SS-100; Nihon Kohden, Tokyo) controlled by a pulse generating system (SEM7201; Nihon Kohden) Ag/AgCl disk electrodes (ϕ: 1 cm, Ne-101; Nihon Kohden) were placed on the dorsal surface of the right forearm just proximal to the radial head. The stimulation intensities were set at 0.7 kpm for all experiments. The stimulation intensities were adjusted so that the knee was semiflexed at phase 3.

Electromyography

Electromyographic (EMG) signals were recorded from the right flexor carpi radialis (FCR), extensor carpi radialis (ECR), biceps brachii (BB), triceps brachii (TB), anterior deltoid (AD), and posterior deltoid (PD) muscles. EMG was also recorded from the right vastus lateralis muscle (VL) to monitor incidental activation level. EMG electrodes were placed longitudinally, 3 cm apart, over each muscle belly and fixed with surgical tape. EMG signals were amplified (×1,000, Model 1206; NEC San-ai Instruments, Tokyo) and band-pass filtered at 32–1,000 Hz. EMG data were full-wave rectified and smoothed (moving average, 5 ms interval) with built-in software (CED 1401 interface with Spike2 software; Cambridge Electronic Design, Cambridge, UK). All signals were converted into digital data via an A/D converter system at a sampling rate of 3 kHz for later off-line analysis. Visual feedback for the activation level of different target muscles during isometric contraction (see following text) was given with an analog voltmeter. The maximum EMG activity (EMGmax) was determined for each muscle during a nearly 3 s maximum voluntary contraction (MVC) performed before the start of the main experiments.

Ergometer settings

Each subject sat in an armchair with the forearm supinated and the elbow and shoulder positioned at 120 and 90°, respectively. The feet were fixed to the pedal of the leg cycling ergometer (COMBI Power Max V) that was placed in front of the subject. The length of the ergometer pedal crank was 15 cm. The crank position during pedaling was detected by a photocell (PS-102; COCO Research, Tokyo) placed on the gear wheel. The cadence was displayed through a digital meter (TDP-3301A-E; COCO Research) connected to the photocell, so that subjects were able to monitor the cadence. The load of the ergometer was set at 0.7 kpm for all experiments.

For analysis, the pedaling cycle was divided into eight phases with the top dead center representing phase 1 with reference to the side of stimulation (see Fig. 1). The position of the chair was carefully adjusted so that the knee was semiflexed at phase 3.

Protocol

To explore the effect of lower limb movement on the upper limb reflex amplitude, subjects participated in five separate motor tasks.
**TASK 1.** We first investigated the effect of leg pedaling on the amplitude of the ELCR in the upper limb muscles. In separate trials, subjects performed isometric contractions of the upper limb muscles at three different levels while keeping the legs stationary or performing leg pedaling. The contraction level of each target muscle was maintained at 5, 10, or 15% of EMGmax and the pedaling cadence was set at 60 rpm. This resulted in a total of 36 trials (2 leg conditions × 3 contraction levels × 6 upper muscles). Electrical stimulation was given in total 50 times to the SR nerve in a random manner (0.8–1.5 s interval) to evoke the ELCR. Therefore stimulation was delivered across the phases of cycling. Additionally, 11 of these subjects performed leg pedaling with a cadence of 90 rpm during upper limb muscle contraction at 10% EMGmax.

**TASK 2.** Second, we investigated phase-dependent modulation of the ELCR during leg pedaling. Pedaling cadence was 60 rpm and the contraction level of the upper muscles was specified as 10% EMGmax. The electrical stimulation was delivered 50 times at each of the eight phases every one or two cycles (Fig. 1). Therefore the ELCR in the target muscle was obtained from eight positions for each subject.

**TASK 3.** In this series, the effect of tonic isometric contraction of the knee extensors on the amplitude of ELCR in the upper limb muscles was investigated. The position of the ipsilateral leg was held at phase 1 during leg pedaling while two different levels of isometric contractions of the right knee extensors were performed. In a separate trial, we measured beforehand the levels of the background EMG activity in the VL at phase 1 during leg pedaling at 60 and 90 rpm and found that they were about 17% (EMGmax) and 28%, respectively. Accordingly, the levels of isometric contraction of the VL were set to approximate these values (Iso60 and Iso90). The level of isometric contraction of the upper limb muscles was set at roughly 10% EMGmax. Additionally, subjects also performed rhythmic and isometric contraction of the right VL at 1 Hz. Again the leg position was set at phase 1 during leg pedaling (Rhythm) and the contraction level was set to the same as that for Iso60. Electrical stimulation was delivered 50 times at 1 Hz.

**TASK 4.** To determine whether modulation of reflexes in the arms was related to the activity state of the legs, the subjects performed bilateral (Bi), ipsilateral (Ipsi; with respect to side of stimulation), or contralateral (Cont) leg pedaling. The contraction level of the upper limb muscles was set at 5% EMGmax so that facilitations could be easily observed. Electrical stimulation was applied 50 times every one to two cycles when the leg position was at phase 1. The pedaling cadence was set at 60 rpm in all trials.

**TASK 5.** To determine whether there was a difference between automatic and targeted leg muscle activation, the effect of discrete leg extension movement on the ELCR was investigated. Subjects were asked to extend their legs and push the right pedal from phase 8 but to stop at phase 4 (Discrete). Next, the subjects performed ipsilateral (right; stimulated side) leg pedaling (Ipsi). As a control, the ELCR in the upper limb muscle was recorded while the legs were stationary. The intertrial interval of the Discrete test was set at ±3 s. The level of isometric contraction of the upper limb muscles was set at 5% EMGmax. Electrical stimulation was delivered 50 times at phase 1 for all trials.

**Data analysis**

EMG was analyzed for a 400 ms time window (100 ms prestimulus and 300 ms poststimulus). Onset latency was identified when the EMG continuously rose above or fell below 2SDs of the mean background EMG for ≥5 ms. The amplitude and peak latency of the ELCR were determined by measuring the base-to-peak amplitude within the preset time window from 30 to 70 ms after the electrical stimulation. The amplitude of the ELCR was normalized by EMGmax for each subject.

**Statistics**

To test for statistical differences in the modulation of the ELCR in the upper limb muscles between the different leg conditions (pedaling vs. stationary) and contraction level (5, 10, and 15% EMGmax), two-way repeated measures ANOVA was performed. To determine whether the reflex amplitude was significantly increased or decreased from the baseline, multiple comparisons were conducted with the Bonferroni post hoc test. Differences in latency and amplitude of the ELCR at different cadences were examined with the Student’s t-test. One-way ANOVA with repeated measures was performed to determine the significance of the phase modulation between the control values and the values for Iso60, Iso90, Rhythm, Discrete, and Ipsi. Post hoc multiple comparisons were conducted using the Bonferroni test. The significance of the F values was obtained after the Greenhouse–Geisser correction, when appropriate, and then a correction coefficient epsilon was determined. Statistical significance level was set at P < 0.05.

**RESULTS**

Across subjects and in all muscles tested, the ELCR (peak latency: ~30–70 ms) could be clearly detected following stimulation of the SR nerve. The mean onset latency of the ELCR during stationary isometric contraction (5–15% EMGmax) ranged between 27 ± 5 and 47 ± 12 ms (TB and PD, respectively; see Table 1). In the same condition, the mean peak latency of the ELCR was distributed between 44 ± 10 and 57 ± 10 ms (BB and FCR, respectively; see Table 1). There were no significant differences in the onset and peak latencies across tasks, except for the onset latency of the PD [F(1,12) = 19.38, P = 0.0008]. However, the sign of the reflex showed a strong muscle dependence. Reflexes in the FCR and PD were facilitatory, whereas suppressive responses were found in the ECR, BB, TB, and AD muscles.

**Modulation of the ELCR in the upper limb muscles during leg pedaling**

Figure 2A shows typical recordings of the cutaneous reflexes in all muscles tested following stimulation of the SR nerve during stationary isometric contractions and 60 rpm leg pedaling for a single subject. Amplitudes of the ELCR in the FCR and PD (despite consistent background EMG levels) were larger during leg pedaling than those during stationary contraction. The suppressive response in the BB was decreased during leg pedaling compared with stationary control. In contrast, there were no significant changes in the amplitude of the ELCR.

**TABLE 1. Summary of averaged onset and peak latencies (ms) for early latency reflexes in all muscles during each task**

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Stationary</th>
<th>Pedaling</th>
</tr>
</thead>
<tbody>
<tr>
<td>FCR</td>
<td>42 ± 13</td>
<td>57 ± 10</td>
</tr>
<tr>
<td>ECR</td>
<td>30 ± 4</td>
<td>48 ± 5</td>
</tr>
<tr>
<td>BB</td>
<td>33 ± 9</td>
<td>46 ± 9</td>
</tr>
<tr>
<td>TB</td>
<td>27 ± 5</td>
<td>46 ± 6</td>
</tr>
<tr>
<td>AD</td>
<td>41 ± 5</td>
<td>51 ± 5</td>
</tr>
<tr>
<td>PD</td>
<td>47 ± 12</td>
<td>54 ± 7</td>
</tr>
</tbody>
</table>

Values are grand means ± SD (n = 13) of the onset and peak latencies for the ELCR during both the leg stationary and pedaling phases in all muscles. Each averaged latency included the data collected at three contraction levels of 5, 10, and 15% EMGmax.
in the ECR, TB, and AD muscles. Figure 2B illustrates the group means (and SD) of the amplitude of the ELCR in all muscles during leg pedaling (black bars) and when stationary (gray bars) while exerting different levels of isometric contraction (5, 10, 15% EMGmax). ANOVA showed that there were significant main effects for task in the FCR, BB, and PD (leg pedaling × stationary: FCR [F(1,12) = 6.84, P = 0.022], BB [F(1,12) = 7.80, P = 0.016], and PD [F(1,12) = 13.75, P = 0.002]). For the ECR, TB, and AD, there were no significant main effects between tasks (all P > 0.05). There was a significant main effect for the EMG level (FCR [F(2,24) = 27.47, P = 0.000], ECR [F(2,24) = 83.37, P = 0.000], BB [F(2,24) = 23.64, P = 0.000], TB [F(2,24) = 35.36, P = 0.000], AD [F(2,24) = 31.64, P = 0.000], and PD [F(2,24) = 8.33, P = 0.001]). Because the amplitudes of the ELCR in the FCR, BB, and PD muscles were very sensitive to the motor task, we specifically focused on these muscles to test in the other tasks.

Phase dependence of the ELCR in arm muscles during leg pedaling

Figure 3 shows the changes in the amplitude of the ELCR at the eight different crank positions (grand means, n = 8) for the FCR and PD. In some subjects, a small increase in ELCR amplitude was found in the PD at the phase 4 crank position. However, there were no significant main effects (e.g., effect of crank position) in either muscle for the amplitude of the ELCR [FCR, F(7,49) = 0.88, P = 0.572; PD, F(7,49) = 1.47, P = 0.199].

Modulation of ELCR amplitude during graded leg pedaling

Figure 4A illustrates typical recordings of the ELCR in the FCR and PD muscles while stationary (dotted lines) and whole leg pedaling at 60 (thin lines) and 90 (thick lines) rpm from a single subject. For both muscles, the amplitude of the ELCR was larger during cycling at 90 rpm than that at 60 rpm. However, there was no significant change for the BB [not illustrated; F(2,20) = 0.08, P = 0.921]. Figure 4B illustrates the grand means (n = 11) of the ELCR while stationary (white bars) and cycling at 60 (gray bars) and 90 rpm (black bars). ANOVA showed that the amplitude of the ELCR was significantly larger during cycling for the FCR [F(2,20) = 10.90, P = 0.0006] and PD [F(2,20) = 4.77, P = 0.020]. There were no significant differences in the background EMG activity or the latency of the ELCR (background EMG activity, FCR [F(2,20) = 1.57, P = 0.231] and PD [F(2,20) = 0.32, P = 0.729]; Latency, FCR [F(2,20) = 1.70, P = 0.206], and PD [F(2,20) = 0.95, P = 0.400]).

Effect of isometric knee extension on ELCR amplitude

Figure 4C shows the grand mean (n = 8) amplitudes of the ELCR in the FCR and PD during stationary control and isometric contractions. The amplitude of the ELCR did not change between the two levels of isometric contraction (Iso60 and Iso90) (FCR [F(2,14) = 1.41, P = 0.276] and PD [F(2,14) = 2.11, P = 0.157]). In addition, as shown in Fig. 5, the amplitude of the ELCR did not change during the rhythmic isometric contractions (n = 9). However, it was interesting to note that when the subjects performed ipsilateral pedaling the amplitude of the ELCR in the FCR and PD was significantly
stationary task for both muscles (FCR; three pedaling tasks were significantly larger than those in the bilateral cycling at 60 rpm. The ELCR amplitudes in these while performing ipsilateral one-leg, contralateral one-leg, and amplitude of the ELCR, we investigated changes in the ELCR among these three tasks in the FCR and PD (FCR \( F_{(2,16)} = 7.56, P = 0.004 \)). The modulation strongly depended on the level of isometric contraction of the test muscles and the cadence of leg pedaling. However, we did not observe phase-dependent modulation of the ELCR in arm muscles. In addition, we found that rhythmic continuous pedaling is a key factor for AM of the ELCR in the upper limb and that a simple discrete leg movement is not enough to modulate the ELCR. Therefore it appears to be more than the simple level of activity of the leg muscles and that the activity state (i.e., involving rhythmic movement) is crucial to induce the interlimb modulation.

Interlimb neural coupling

Neural coupling between the upper and lower limbs in humans has been suggested in many studies (Balter and Zehr 2007; Dietz et al. 2001; Haridas and Zehr 2003; Sakamoto et al. 2006). During walking, interlimb cutaneous reflexes in the upper (or lower) limb muscles were demonstrated to be modulated depending on the phase of the lower (or upper) limbs, suggesting the existence of neural coupling between the upper and lower limbs (Dietz et al. 2001; Haridas and Zehr 2003). In addition, phase-dependent reflex modulation during rhythmic movement that is uncoupled from background EMG has been consistently attributed to the rhythm generating system (Dietz 2003; Zehr and Duysens 2004). Taking these previous studies into consideration, one can expect that the activation of the neural system in the lower limb affects the amplitude of the ELCR in the upper limb even if the corresponding limb is stationary. We therefore tested the effect of leg pedaling on the ELCR in the upper limb under stationary conditions. We found no evidence of phase-dependent modulation in the ELCR in the PD, BB, and FCR during leg pedaling. However, this result does not necessarily deny possible neural coupling between the limbs.

The significant increase in the ELCR in the upper limb muscles during leg pedaling may signify offset regulation, which may be detectable when the tested limb is stationary, and can be easily masked by phase modulation when the corresponding limb moves rhythmically. Rhythmic arm or leg pedaling is known to modulate the amplitude of cutaneous reflexes and the H-reflex. In particular, the reflexes in the muscles of the moving limb are strongly modulated in a phase-dependent manner (Capaday and Stein 1986; Yang and Stein 1990). Carroll et al. (2005) also demonstrated that contralateral arm cycling was insufficient to generate phase modulation of cutaneous reflexes in the stationary arm. Our results here are consistent with the suggestion that the activity state of the limb in which a reflex is evoked has the largest effect on reflex amplitude. It may be that the rhythmic movement of the corresponding limb is integral for generating phase modulation of the cutaneous reflexes when the tested limb is stationary.

Muscle specificity of the ELCR in the upper limb during leg pedaling

The ELCR was evoked in a variety of muscles in the upper limb and the sign of the responses was dependent on the elicited muscles as described in previous reports (Zehr and Kido 2001; Zehr et al. 2001). Importantly, the AM of the ELCR during lower leg pedaling was observed in only some muscles (PD, BB, and FCR) and was dependent on the level of their background EMG. These results partly corresponded with

Laterality-dependent modulation of the ELCR

To determine in greater detail the factors affecting the amplitude of the ELCR, we investigated changes in the ELCR while performing ipsilateral one-leg, contralateral one-leg, and bilateral cycling at 60 rpm. The ELCR amplitudes in these three pedaling tasks were significantly larger than those in the stationary task for both muscles [FCR; \( F_{(3,24)} = 4.412, P = 0.013 \); PD; \( F_{(3,24)} = 6.512, P = 0.002 \)]. However, there were no significant differences in the amplitudes of the ELCR among these three tasks in the FCR and PD [FCR \( F_{(2,16)} = 0.67, P = 0.521 \) ] and PD [FCR \( F_{(2,16)} = 0.419, P = 0.620 \); data not shown].

In six subjects, we tested the effect of discrete leg movement on the ELCR (Fig. 6). Note that the amplitude of the ELCR was larger during ipsilateral pedaling than that during the discrete and stationary conditions for the FCR \( F_{(2,12)} = 7.46, P = 0.008 \) and PD \( F_{(2,10)} = 7.80, P = 0.007 \) (shown in Fig. 6B).
previous reports in which phase-dependent modulation was found in some muscles in the upper limb during rhythmic arm movement (Zehr and Kido 2001; Zehr et al. 2007). Our findings also indicate that the rhythm generating systems for leg movements are able to differentially modulate motor reflex responses in different upper limb muscles.

To simply consider the complex pattern of modulation of the ELCR for arm muscles (the increase in both the excitatory PD and FCR response and the suppression of the inhibitory BB response) from a functional view, it could be interpreted as promoting a parachute reaction that effectively evades from a disturbance to the hand area. In addition, this complex reflex pattern would be modulated depending on the level of muscle activation, the cadence change of leg pedaling. It would be beneficial for rehabilitation training to seek the optimal position or posture for increasing or decreasing spinal cord excitability using the amplitude of the cutaneous reflexes in a given muscle as an outcome measure (Klimstra et al. 2009). Conversely, we cannot deny the possibility that the muscle-specific modulation found in the present study could be dependent on task constraints, posture, or arm position (Misiaszek and Krauss 2005; Zehr et al. 2007). These issues are, of course, to be determined in future experiments.

**Short-latency cutaneous reflexes in the upper arm muscles**

Based on the mean onset latency of the ELCR (FCR, 40 ms; BB, 30 ms; and PD, 39 ms), spinal reflex circuitry must be, at least partly, involved with the ELCR (Jenner and Stephens 1982). However, short-latency cutaneous reflexes in the first dorsal interosseous muscle (E1, latency ~35 ms and I1, ~45 ms; Evans et al. 1989; Jenner and Stephens 1982) were slightly shorter than the ELCR observed in the present study. Also, it was notable that the latency of the PD was longer than that of the FCR and BB. Although these results were consistent with previous reports (Zehr and Kido 2001; Zehr et al. 2001), the reasons for the latency difference among the arm muscles remain unknown. In contrast, the E2 (~50 ms) response, in which the motor cortex plays an important role in its generation, was easily altered depending on the task (Evans et al. 1989; Gibbs et al. 1995). Our findings for the ELCR are clearly different from the behaviors of the E1 and I1, but have some similarities with E2. Therefore the activity of the spinal and supraspinal neural circuitry would contribute to the modulation of the ELCR during leg pedaling in the present study (Baken et al. 2005; Burke et al. 1991; Zehr and Chua 2000). Furthermore, it is still unknown whether an interaction between the rhythm generating system for the lower leg and the neural circuitry mediating the ELCR within the spinal cord can give rise to the modulation of the ELCR in the upper limb muscles during leg pedaling. It is possible that supraspinal mechanisms could play a role in integrating these reflex interactions during rhythmic movements.

We demonstrated that the AM of the ELCR strongly depended on the task performed. As such, the amplitude of the ELCR during leg pedaling was significantly larger than that of static contraction, independent from equivalent background EMG activity (Figs. 4C and 5). In addition, the amplitude of the ELCR was graded with the cadence of leg pedaling (Fig. 4, A and B). Furthermore, the AM of the ELCR was not seen during discrete leg movements (Fig. 6). Thus it is likely that the generation of an interlimb effect on the ELCR from leg to arm muscles requires the sustained rhythmic movement of the leg, although it remains to be determined for how long and how fast the cyclical leg movement is needed to generate the interlimb effect on the ELCR. This argument was previously...
explored in some detail by Hundza and Zehr (2007) and our results concur with the conclusion that rhythmic activation is a necessary condition for cutaneous reflex modulation. In previous studies, the cutaneous reflexes were modulated in a task-dependent manner during cycling and static contraction (Brown and Kukulka 1993; Zehr and Kido 2001). As argued elsewhere, this task dependence suggests a contribution of a rhythm generating system. Therefore it seems likely that the AM of the cutaneous reflex in the upper limb reflects a function of the rhythm generating system associated with leg pedaling. With a higher cadence of leg pedaling, stabilizing the body by activating postural muscles would be required, which would lead to the modification of the ELCR amplitude. Ensuring postural stability during different locomotor tasks would be a key factor for the modulation of the cutaneous reflexes (Hari-das et al. 2006).

Our findings further support the contribution of an inter-limb interaction between the presumed lower limb neural system and the neural circuit of the cutaneous reflex in the upper limb. This would result in an increase of the excitability of the circuitry for the cutaneous reflex (offset regulation).

FIG. 5. A: typical EMG recordings of the VL during pedaling of the right leg (ipsilateral side with respect to the electrical stimulation, top trace) and rhythmic isometric contraction of the right knee extensors (bottom trace). B: grand means and SDs (n = 9) of the ELCR for the FCR (left) and PD (right) while performing ipsilateral pedaling (black boxes) and while performing rhythmic isometric contraction of the right knee extensors (gray boxes). The white box shows the data obtained while the leg was stationary. Contraction level of the tested muscles was set at 5% EMGmax. *P < 0.05, **P < 0.01, ***P < 0.001.

FIG. 6. A: schematic illustrations of the changes in crank position (top), typical EMG recordings of the VL (middle), and knee angle position (bottom) while performing a one-shot discrete leg movement. B: grand means and SDs (n = 7) of the ELCR for the FCR (left) and PD (right) while performing ipsilateral pedaling (black boxes) and while performing a discrete movement of the right knee (gray boxes). The white box shows the data obtained while the leg was stationary. Contraction level of the tested muscles was set at 5% EMGmax. *P < 0.05, **P < 0.01, ***P < 0.001.
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