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Original Article

Tonic suppression of the soleus H-reflex during rhythmic movement of the contralateral ankle

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Abstract. [Purpose] We investigated the effect of rhythmic ankle movement on the contralateral soleus H-reflex. The H-reflex was evoked from the right soleus muscle. [Subjects and Methods] Healthy humans rhythmically moved the left ankle (movement condition) or held the left ankle stationary (stationary condition) at one of three positions corresponding to the ankle positions at which the H-reflex was evoked in the movement condition. The background electromyographic amplitude in the right soleus muscle was maintained at 10% of the maximum voluntary contraction level, and that in the right tibialis anterior muscle was matched between the stationary and movement conditions. [Results] The soleus H-reflex was suppressed throughout all phases of contralateral rhythmic ankle movement. [Conclusion] Rhythmic movement of the contralateral joint suppresses the H-reflex in the muscle that is the prime mover of the joint homologous to the rhythmically moving joint. This inhibitory mechanism may be activated during unilateral rhythmic movement to isolate the motor control of the moving ankle from that of the contralateral stationary ankle.

Key words: Bilateral coordination, Interlimb coordination, H-reflex

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INTRODUCTION

It has been well established that rhythmic movement is produced by central pattern generators in the spinal neural network in both cats[1)] and humans[2)]. Gait in humans is defined as a typical rhythmic movement of the legs[3, 4)]. Time-locked bilateral interlimb coordination of leg movement occurs during gait[5)]. To produce phase-locked bilateral rhythmic movement, neural interaction between the central pattern generator controlling one limb and that controlling the contralateral limb is needed[6, 7)].

Suppression of the H-reflex induced by contralateral rhythmic movement provides evidence in favor of the existence of a neural mechanism mediating the bilateral interaction of rhythmic movement. Rhythmic movement of the wrist suppresses the contralateral H-reflex in the flexor carpi radialis muscle[8)]. Such suppression is also present in the soleus (SOL) muscle. Active unilateral leg pedaling suppresses the SOL H-reflex amplitude in the contralateral stationary leg[9)]. Furthermore, passive pedaling of the contralateral leg or passive contralateral hip or knee movement also suppresses the SOL H-reflex[9–12)]. In previous studies on the SOL H-reflex, the hip and/or knee joints contralateral to the tested side rhythmically moved even though the muscle being tested was one of the prime movers of the ankle joint movement. That is, these previous findings uncovered an effect of contralateral movement of the joint non-homologous on the tested joint. The purpose of the present study was to elucidate the effect of contralateral rhythmic movement on the H-reflex in the SOL muscle, which is the prime mover of the joint homologous to the conditioned joint.

SUBJECTS AND METHODS

Subjects

Ten healthy humans aged 28.9 ± 1.8 years participated in the present study. The subjects had no history of neurological disease. All experimental procedures were conducted in accordance with the ethical standards of the Declaration of Helsinki (1975, revised 1983). All subjects provided written informed consent prior to the experiment, which was approved by the Ethics Committee of Osaka Prefecture University.

Methods

Each subject laid on his or her back on a rigid table with his or her arms positioned along the trunk and the head in the midline position. The hips and knees were extended. The left foot was placed off the surface of the table to allow the subject to freely move the left ankle (conditioned ankle). An electrogoniometer measuring the position of the conditioned ankle on the sagittal plane was placed on the dorsal side of

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the left foot, and the signals from the electrogoniometer were amplified via a strain amplifier (DPM-601A; Kyowa Dengyo, Tokyo, Japan). The right foot was firmly fixed by a metal frame at the 0-degree ankle flexion position (tested ankle). Ag/AgCl surface electrodes recording electromyographic (EMG) activity were placed 2 cm apart on the bellies of the tibialis anterior (TA) and SOL muscles in both legs. EMG signals were amplified via an EMG amplifier (MEG-2100; Nihon Kohden, Tokyo, Japan) with a bandpass filter from 15 Hz to 3 kHz. The EMG and electrogoniometer signals were converted to digital signals at a sampling rate of 10 kHz using an A/D converter (Unique Acquisition; Unique Medical, Tokyo, Japan) and stored on a personal computer. The background EMG (BEMG) amplitude in the SOL muscle of the tested ankle was displayed on a monitor in front of the subject.

The subject watched the indicator of the BEMG amplitude in the SOL muscle of the tested ankle and maintained the amplitude at 10% of the maximal voluntary contraction level while simultaneously producing rhythmic movement of the conditioned ankle at a 1-s cycle duration paced by a metronome with a 1-Hz beeping sound under the movement condition. Before beginning the experiment, each subject practiced the relevant motor tasks until he or she could perform the movements precisely. The maximal dorsiflexion position of the ankle (DFmax position), maximal plantarflexion position of the ankle (PFmax position), and midpoint between these two positions (MP position) in the conditioned ankle were estimated from the recordings of ankle movement during rhythmic movement. The conditioned ankle was passively immobilized with a splint at the DFmax, PFmax, or MP position under the stationary condition.

Electrodes stimulating the right tibial nerve were placed over the right popliteal fossa 2 cm apart. The duration of the stimulus was 1 ms. At the beginning and end of the experiment, the intensity of the stimulation was gradually increased with each successive trial until the supramaximal size of the M-wave in the SOL muscle of the tested ankle was evoked at rest, and we estimated the maximal M-wave amplitude (Mmax), defined as the average of the three largest M-wave amplitudes, to ensure that the Mmax values obtained at the beginning and end of the experiment were similar. Tibial nerve stimulation was delivered in the PFmax, DF, DFmax, or PF phase of the conditioned ankle in each trial under the movement condition. The PFmax and DFmax phases were defined as the phases in which the ankle passed the PFmax or DFmax position, respectively. The DF phase was defined the phase in which the ankle passed the MP position moving from the PFmax to the DFmax position, and likewise, the PF phase was defined as the phase in which the ankle passed the MP position moving from the DFmax to the PFmax position.

The stimulation was triggered by a pulse generator (EN-611 J; Nihon Kohden). The tibial nerve stimulation was manually triggered at the DFmax, PFmax, or MP position under the stationary condition. To monitor the M-wave amplitudes and the tested ankle positions in which the SOL H-reflex was evoked, the signals of the tested ankle positions and the EMG activity in the tested SOL muscle were converted to digital signals at a sampling rate of 10 kHz using an A/D converter (PowerLab800s; ADInstruments, Colorado Springs, CO, USA), and a sweep of the signals in the time window from 50 ms before to 150 ms after tibial nerve stimulation was displayed on a monitor. An experimenter monitored the amplitude of the M-wave and the ankle position, adjusted the intensity of the tibial nerve stimulation so that an M-wave of 10% Mmax was constantly evoked, and adjusted the timing of the tibial nerve stimulation so that the H-reflex was evoked at the intended movement phase. The experimenter also counted the number of successful trials that accompanied an M-wave of 10% Mmax and that in which an H-reflex was evoked in one of the movement phases. The experiment lasted until 15 successful trials were recorded for each phase. It was confirmed that the SOL H-reflex size at rest was similar at the beginning and end of the experiment.

Table 1. Position of the conditioned ankle

	Stationary (degrees)		Movement (degrees)	
PF max	−40.2	(3.1)	−40.3	(3.1)
DF	−24.2	(2.0)	−23.8	(2.1)
DF max	−7.3	(2.2)	−7.6	(2.4)
PF	−24.2	(2.0)	−23.8	(2.1)

Mean (SE)
PFmax: maximal plantarflexion position of the ankle; DF: dorsiflexion; DFmax, maximal dorsiflexion position of the ankle, PF: plantarflexion; TA: tibialis anterior muscle

The dorsiflexion position beyond the neutral position of the ankle was expressed as a positive value. The H-reflex and M-wave amplitudes were estimated on a peak-to-peak basis and were expressed as a percentage of Mmax. EMG traces were rectified, and the BEMG amplitude was estimated from the rectified EMG traces in the time window from 0 to 50 ms before tibial nerve stimulation. Trials not accompanied by an M-wave of 10% Mmax, trials in which the H-reflex was not evoked in either of the movement phases, and trials in which the BEMG amplitude in the TA muscle in the tested leg did not match between the stationary and movement conditions were excluded from data analysis. One- or two-way factorial analysis of variance (ANOVA) was conducted, and a post-hoc test was conducted when ANOVA revealed a significant difference without a significant interaction between factors. When the interaction between the factors was significant, a test of simple main effect was conducted. Data are presented as the mean and standard error of the mean.

RESULTS

The cycle duration of conditioned ankle movement was 998 ± 3 ms under the movement condition. The ankle position was significantly different between the phases [$F(3,72) = 61.30$, $p < 0.01$], but it was not significantly different between the conditions [$F(1,72) = 0.00$, $p = 0.95$] (Table 1). There was no significant interaction between the factors [$F(3,72) = 0.01$, $p = 1.00$].

The BEMG amplitudes in the TA and SOL muscles of the tested ankle were not significantly different between the conditions or between the phases without significant interac-

tion (Table 2). The BEMG amplitudes in the SOL muscle of the conditioned ankle under the movement condition were significantly larger than those under the stationary condition [$F(1,72) = 37.29$, $p < 0.01$], but they were not significantly different between the phases [$F(3,72) = 1.08$, $p = 0.86$]. The interaction between the two factors was not significant [$F(3,72) = 1.14$, $p = 0.34$]. The BEMG amplitudes in the TA muscle of the conditioned ankle under the movement condition were significantly larger than those under the stationary condition [$F(1,72) = 47.15$, $p < 0.01$] and were significantly different between the phases [$F(3,72) = 10.50$, $p < 0.01$] with significant interaction [$F(3,72) = 10.44$, $p < 0.01$]. A test of simple main effect revealed that the difference in the amplitudes was significantly different between the phases under the movement condition [$F(3,72) = 20.95$, $p < 0.01$]. The post-hoc test revealed significant differences in the amplitudes between the PF max and DF phases, between the PFmax and DFmax phases, between the PF and DF phases, and between the PF and DFmax phases under the movement condition ($p < 0.05$). Furthermore, additional tests of simple main effect revealed that the amplitude under the movement condition was significantly larger than that under the stationary condition in the DF [$F(1,72) = 39.60$, $p < 0.01$] and DFmax phases [$F(1,72) = 38.07$, $p < 0.01$].

The overall average of the M-wave amplitude from all trials was 9.9 ± 0.1% of Mmax (Table 3). The amplitude was not significantly different between the phases [$F(3,72) = 0.53$, $p = 0.67$] or conditions [$F(1,72) = 0.00$, $p = 0.97$] without significant interaction [$F(3,72) = 0.53$, $p = 0.66$]. The overall average of the H-reflex amplitude from all trials under the stationary condition was 52.0 ± 1.5% of Mmax. The amplitude under the stationary condition was not significantly different between the phases [$F(3,72) = 0.00$, $p = 1.00$]. The amplitude under the movement condition was significantly smaller than that under the stationary condition [$F(1,72) = 4.38$, $p = 0.04$], but it was not significantly different between the phases [$F(3,72) = 0.05$, $p = 0.99$]. There was no significant interaction between the factors [$F(3,72) = 0.11$, $p = 0.96$].

Table 2. BEMG amplitude

	BEMG amplitude on the conditioned side (μV)				BEMG amplitude on the tested side (μV)			
	Stationary		Movement		Stationary		Movement	
SOL								
PFmax	1.9	(0.5)	7.6	(1.9)	16.0	(2.4)	15.4	(3.0)
DF	1.8	(0.5)	12.9	(3.1)	16.0	(2.5)	15.2	(3.0)
DFmax	2.1	(0.4)	9.1	(1.9)	15.8	(2.7)	15.2	(3.1)
PF	1.8	(0.5)	7.5	(2.3)	16.0	(2.5)	14.3	(2.5)
TA								
PFmax	2.0	(0.4)	9.7	(3.3)	8.9	(3.8)	4.8	(1.6)
DF	1.8	(0.3)	74.0	(12.5)	4.9	(2.0)	3.5	(0.8)
DFmax	2.2	(0.4)	72.9	(18.7)	6.1	(2.2)	4.5	(1.1)
PF	1.8	(0.3)	8.6	(2.7)	4.9	(2.0)	5.0	(2.0)

Mean (SE)
BEMG: background electromyographic; SOL: soleus muscle; PFmax: maximal plantarflexion position of the ankle; DF: dorsiflexion; DFmax: maximal dorsiflexion position of the ankle; PF: plantarflexion; TA: tibialis anterior muscle

Table 3. M-wave and H-reflex amplitudes

	M-wave amplitude (% of Mmax)				H-reflex amplitude (% of Mmax)			
	Stationary		Movement		Stationary		Movement	
PFmax	10.2	(0.1)	9.9	(0.2)	54.9	(4.1)	49.4	(4.9)
DF	9.7	(0.2)	9.9	(0.3)	55.1	(4.4)	50.4	(4.8)
DFmax	9.9	(0.2)	9.9	(0.2)	55.8	(4.6)	46.4	(4.2)
PF	9.7	(0.2)	9.9	(0.3)	55.1	(4.4)	48.5	(3.6)

Mean (SE)
Mmax: maximal M-wave amplitude; PFmax: maximal plantarflexion position of the ankle; DF: dorsiflexion; DFmax: maximal dorsiflexion position of the ankle, PF: plantarflexion; TA: tibialis anterior muscle

DISCUSSION

The SOL H-reflex amplitude was suppressed during contralateral ankle movement throughout all phases of ankle movement. H-reflex amplitude depends on the BEMG activity of the tested muscle[13, 14]. In addition, voluntary contraction of the TA muscle reciprocally suppresses the SOL H-reflex amplitude[15]. However, the activation level of the muscles on the tested side is not related to rhythmic movement-induced suppression of the contralateral SOL H-reflex amplitude because the BEMG amplitudes in the TA and SOL muscles of the tested ankle under the stationary condition matched those under the movement condition. Suppression of the SOL H-reflex without changes in the BEMG activity level in the tested SOL muscle indicates that the suppression is caused predominantly by presynaptic inhibition[16]. M-wave amplitude is a good indicator of the consistency of stimulation of the group Ia afferent pathway[17]. In the present study, only trials accompanied by an M-wave of 10% Mmax were included in the data analysis. Thus, the group Ia afferent pathway should have been stimulated equally under all experimental conditions. The sensitivity of the H-reflex to the facilitatory or inhibitory input depends on the control H-reflex size[18]. However, the sensitivity of the H-reflex to the facilitatory or inhibitory input was similar across all movement phases because the H-reflex amplitude under the stationary condition was not significantly different between the phases.

Neural pathways mediating crossed afferent feedback between the legs probably exist based on previous findings that passive leg pedaling or rhythmic hip or knee movement suppresses the contralateral SOL H-reflex amplitude[9–12], and electrical stimulation of the tibial nerve induces short-latency crossed inhibition of the EMG or H-reflex amplitude in the contralateral leg[19, 20]. Active movement of the joint apparently produces proprioceptive afferent feedback. Nevertheless, it is inconclusive to state that suppression of the SOL H-reflex during contralateral rhythmic ankle movement is caused by crossed afferent feedback because descending motor commands must be accompanied by afferent feedback when active movement of the ankle is executed.

Tonic suppression of the SOL H-reflex during rhythmic movement of the contralateral ankle was consistent with a previous finding that the H-reflex in the flexor carpi radialis muscle was suppressed throughout all phases of contralateral rhythmic movement of the wrist[8]. By contrast, anti-phase bilateral ankle movement slightly suppressed the SOL H-reflex only in the maximum plantarflexion phase of the tested ankle[21]. The amount of suppression during anti-phase bilateral ankle movement observed in the previous study was much less than that of suppression during unilateral movement of the contralateral ankle or wrist. This difference may be explained by a switch mechanism through which an inhibitory mechanism is active during unilateral rhythmic movement but is inactive during bilateral rhythmic movement. Indeed, voluntary contraction of the contralateral hand muscle suppresses corticospinal excitability in the hand muscle at rest through interhemispheric inhibition[22]. This mechanism is believed to suppress the production of unwanted movement associated with the intended contralateral movement[22]. Such an inhibitory mechanism may exist even during rhythmic movement to isolate the motor control of one limb segment from that of the contralateral limb segment for both the wrists and ankles.

REFERENCES

1) Brown TG: The intinsic factors in the act of progression in the mammal. Proc R Soc Lond, 1911, B84: 308–319. [CrossRef]
2) Dimitrijevic MR, Gerasimenko Y, Pinter MM: Evidence for a spinal central pattern generator in humans. Ann N Y Acad Sci, 1998, 860: 360–376. [Medline] [CrossRef]
3) Asaka T, Saito H, Takamori Y, et al.: Gait control in young adults trained on a low friction floor. J Phys Ther Sci, 2004, 16: 151–158. [CrossRef]
4) Henmi O, Shiba Y, Saito T, et al.: Spectral analysis of gait variability of stride interval time series: comparison of young, elderly and parkinson's disease patients. J Phys Ther Sci, 2009, 21: 105–111. [CrossRef]
5) Perry J: Gait Analysis: Normal and Pathological Function. New Jersey: Slack Inc, 1992.
6) Prokop T, Berger W, Zijlstra W, et al.: Adaptational and learning processes during human split-belt locomotion: interaction between central mechanisms and afferent input. Exp Brain Res, 1995, 106: 449–456. [Medline] [CrossRef]
7) Ting LH, Raasch CC, Brown DA, et al.: Sensorimotor state of the contralateral leg affects ipsilateral muscle coordination of pedaling. J Neurophysiol, 1998, 80: 1341–1351. [Medline]
8) Carson RG, Riek S, Mackey DC, et al.: Excitability changes in human forearm corticospinal projections and spinal reflex pathways during rhythmic voluntary movement of the opposite limb. J Physiol, 2004, 560: 929–940. [Medline] [CrossRef]
9) McIlroy WE, Collins DF, Brooke JD: Movement features and H-reflex modulation. II. Passive rotation, movement velocity and single leg movement. Brain Res, 1992, 582: 85–93. [Medline] [CrossRef]
10) Collins DF, McIlroy WE, Brooke JD: Contralateral inhibition of soleus H reflexes with different velocities of passive movement of the opposite leg. Brain Res, 1993, 603: 96–101. [Medline] [CrossRef]
11) Cheng J, Brooke JD, Misiaszek JE, et al.: Crossed inhibition of the soleus H reflex during passive pedalling movement. Brain Res, 1998, 779: 280–284. [Medline] [CrossRef]
12) Misiaszek JE, Cheng J, Brooke JD, et al.: Movement-induced modulation of soleus H reflexes with altered length of biarticular muscles. Brain Res, 1998, 795: 25–36. [Medline] [CrossRef]
13) Verrier MC: Alterations in H reflex magnitude by variations in baseline EMG excitability. Electroencephalogr Clin Neurophysiol, 1985, 60: 492–499. [Medline] [CrossRef]
14) Crenna P, Frigo C: Excitability of the soleus H-reflex arc during walking and stepping in man. Exp Brain Res, 1987, 66: 49–60. [Medline] [CrossRef]
15) Crone C, Nielsen J: Spinal mechanisms in man contributing to reciprocal inhibition during voluntary dorsiflexion of the foot. J Physiol, 1989, 416: 255–272. [Medline] [CrossRef]
16) Stein RB: Presynaptic inhibition in humans. Prog Neurobiol, 1995, 47: 533–544. [Medline] [CrossRef]
17) Boorman GI, Hoffer JA, Kallesoe K, et al.: A measure of peripheral nerve stimulation efficacy applicable to H-reflex studies. Can J Neurol Sci, 1996, 23: 264–270. [Medline]
18) Crone C, Hultborn H, Mazières L, et al.: Sensitivity of monosynaptic test reflexes to facilitation and inhibition as a function of the test reflex size: a study in man and the cat. Exp Brain Res, 1990, 81: 35–45. [Medline] [CrossRef]
19) Stubbs PW, Mrachacz-Kersting N: Short-latency crossed inhibitory responses in the human soleus muscle. J Neurophysiol, 2009, 102: 3596–3605. [Medline] [CrossRef]
20) Stubbs PW, Nielsen JF, Sinkjaer T, et al.: Crossed spinal soleus muscle communication demonstrated by H-reflex conditioning. Muscle Nerve, 2011, 43: 845–850. [Medline] [CrossRef]
21) Hiraoka K, Chujyo Y, Hatano S, et al.: Effects of contralateral movement on the soleus H-reflex during in-phase and antiphase movements of the ankles. Mot Contr, 2014, 18: 88–100. [Medline] [CrossRef]
22) Vercauteren K, Pleysier T, Van Belle L, et al.: Unimanual muscle activation increases interhemispheric inhibition from the active to the resting hemisphere. Neurosci Lett, 2008, 445: 209–213. [Medline] [CrossRef]