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RESEARCH ARTICLE

Spinal reciprocal inhibition in the co-contraction of the lower leg depends on muscle activity ratio

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Abstract
The spinal reciprocal inhibition during co-contraction remains unclear. Reports on the reciprocal Ia and D1 inhibitions in the co-contraction are lacking, and a point about the muscle activity amount during co-contraction is unclear. This study aimed to clarify the influence of changes in the ratio of soleus (Sol) and tibialis anterior (TA) muscle activities in co-contraction on reciprocal Ia and D1 inhibitions. Twenty healthy adults were subjected to four stimulatory conditions: a conditioning stimulus–test stimulation interval (CTI) of − 2, 2, or 20 ms or a test stimulus without a conditioning stimulus (single). Co-contraction [change in (Sol)/(TA) activity] was examined at task A, 0%/0% maximal voluntary contraction (MVC); task B, 5%/5% MVC; task C, 15%/15% MVC; task D, 5%/15% MVC; and task E, 15%/5% MVC. At 2-ms CTI, the H-reflex amplitude value was significantly lower in tasks A, B, C, and D than in the single condition. Among the tasks, the H-reflex amplitude values were lower for A, B, C, and D than for E. At 20-ms CTI, the H-reflex amplitude was significantly lower in tasks A, B, C, D, and E. Among the tasks, the H-reflex amplitude was significantly lower from task A and B to task E. The change in the muscle activity ratio during co-contraction could modulate reciprocal Ia inhibition depending on the Sol/TA muscle activity ratio. D1 inhibition at rest did not differ significantly when the Sol/TA ratio was equal or when TA muscle activity was high. During co-contraction with high Sol muscle activity, D1 inhibition decreased from rest.

Keywords Spinal reciprocal inhibition · Contraction intensity · Co-contraction · H-reflex amplitude

Introduction

Muscle co-contraction or co-activation refers to the simultaneous activation of agonist and antagonist muscles (Baratta et al. 1988; Kellis 1998; Aagaard et al. 2000). Co-contraction is defined as a voluntary activity and co-activation as involuntary. These strategies are considered important motion control mechanisms for improving joint stability. In various upper motor neuron disorders including spastic diseases, cerebellar ataxia, Parkinson's disease, and spinal cord injury, when movement of only an agonist muscle is required (e.g., during reciprocal inhibition exercise), collapse of the spinal reciprocal inhibition mechanism against the antagonist may cause excessive simultaneous activation. In other words, spinal reciprocal inhibition fails to affect the antagonist, thereby impairing smooth joint movement (Hayashi et al. 1988; Kagamihara et al. 1993; Kagamihara and Tanaka 1996). In addition to pathological states, the amount of such simultaneous activation of muscle activity is known to increase with age (Morita et al. 1995, 2000; Hortobagyi and Devita 2006; Hortobagyi et al. 2009; Baudry et al. 2010; Nagai et al. 2011). In athletes, excessive simultaneous activation may interfere with joint movement and degrade agility (Blackwell and Cole 1994).

The cause of excessive simultaneous activation has been identified as reduced presynaptic inhibition (Milanov 1992; Kagamihara and Masakado 2005) at the terminal ends of Ia fibers, which are afferent fibers from muscle spindles, and enhanced stretch reflex due to decreased post-activation depression (Nielsen et al. 1995). During joint movement, decreased reciprocal Ia inhibition from agonist Ia fibers (Mizuno et al. 1971; Nielsen and Kagamihara 1992; Crone et al. 1994; Okuma et al. 2002) can decrease presynaptic inhibition (D1 inhibition) (Mizuno et al. 1971; Tanaka 1974;

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Crone and Nielsen 1989; Faist et al. 1994; Nielsen et al. 1995) and antagonist-prompting input (Crone et al. 2000). Taken together, these findings suggest that many factors contribute to excessive simultaneous activation.

Previous studies have reported inhibition of inhibitory interneurons against antagonist muscles from primary acting muscles during co-contraction, which is voluntary, and inhibition of smooth joint movement (Nielsen and Kagamihara 1992; Crone and Nielsen 1994). A highly cited study of co-contraction and spinal reciprocal inhibition by Nielsen and Kagamihara (1992) reported that spinal reciprocal inhibition did not depend on contraction intensity during co-contraction of the tibialis anterior muscle (TA) and soleus muscle (Sol). However, many previous studies, including that by (Nielsen and Kagamihara 1992), used joint torque as an index of intensity of co-contraction (Nielsen and Kagamihara 1992; Nielsen et al. 1992, 1994; Morita et al. 2001; Oya and Cresswell 2008; Magalhaes et al. 2015). Given that several muscles are involved in plantar- and dorsiflexion during co-contraction, the amounts of TA and Sol muscle activity differ in these studies. Since the H-reflex and amount of reciprocal Ia inhibition may vary (Nielsen et al. 1994; Morita et al. 2001), muscle activity should be determined during co-contraction. In consideration of the proportion of exercise units recruited as the output of each muscle, the present study focused on the amount of muscle activity during co-contraction and aimed to clarify whether spinal reciprocal inhibition is dependent on muscle activity or joint torque. In other words, if we can clarify the factor modulating spinal reciprocal inhibition during co-contraction, this will serve as basic knowledge for elucidating remedial measures against excessive co-contraction and moderate co-contraction as pre-contraction before joint movement.

In our previous study, we examined the suppression of spinal reciprocal inhibition in co-contraction with the same amount of muscle activity of TA and Sol. From the results of the study, in the co-contraction with the same amount of muscle activity, reciprocal Ia inhibition worked at $\leq 15\%$ maximum voluntary contraction (MVC), and reciprocal Ia inhibition did not work at 30% MVC (Hirabayashi et al. 2018).

There are four considerations when evaluating spinal reciprocal inhibition during co-contraction. (1) Studies of the relationship between co-contraction and spinal reciprocal inhibition were only reported prior to 2000 (Nielsen and Kagamihara 1992). Because there is no research focusing on muscle activity, we believe it is necessary to re-examine reciprocal Ia inhibition and the conditioning stimulus–test stimulation interval (CTI) of D1 inhibition to clarify the influence of spinal reciprocal inhibition during co-contraction. (2) In previous studies, examination of the CTI only lasted up to 10 ms (Nielsen and Kagamihara 1992), and thus only allowed for review of reciprocal Ia inhibition, which has a short latency. To investigate spinal reciprocal inhibition, it is also necessary to consider D1 inhibition, which is presynaptic inhibition with a longer latency. (3) Because previous studies used joint torque as an index (Nielsen and Kagamihara 1992; Nielsen et al. 1992, 1994; Morita et al. 2001; Oya and Cresswell 2008; Magalhaes et al. 2015), it is necessary to examine joint torque using muscle activity. (4) To examine whether activity of TA and Sol is the same during co-contraction, it is necessary to clarify whether spinal reciprocal inhibition changes the degree of inhibition depending on muscle activity and joint torque.

The function of spinal reciprocal inhibition in co-contraction of Sol and TA to the same extent is unknown. We hypothesize that the Co-contraction also occurs when joint torques are not balanced. Therefore, the spinal reciprocal inhibition should be modulated by the muscle activity ratio, not joint torque.

Patients and methods

Study participants

In total, 20 healthy adults (10 males; 10 females; age, 20.0 ± 0.7 years; height, 167.2 ± 8.2 cm; body weight, 56.8 ± 7.9 kg) provided written informed consent to participate in this study. The study was approved by the Ethics Committee at Niigata University of Health and Welfare (18089—181004). All experiments were proceeded in accordance with the ethical standards of Niigata University of Health and Welfare and with the 1964 Helsinki Declaration and its later amendments.

Measurement of limb position

Right lower limb position was measured at the hip (100°), knee (120°), and ankle (110°) joints. The ankle was immobilized using a foot plate equipped with a joint torque sensor (TAKEI SCIENTIFIC INSTRUMENTS, Niigata, Japan) (Fig. 1). Prior to starting the experiment, participants practiced contracting the TA and Sol muscles without moving their ankle.

Electromyography

The distance between the Ag/AgCl electrodes (Blue Sensor, METS, Tokyo, Japan) of the surface electromyogram was set at 20 mm. Electrodes were placed on the TA and Sol muscles according to SENIAM (Hermens et al. 2000). A ground electrode was placed between the electrical stimulation electrode and the surface electromyogram electrode. Electromyographic activity was filtered at a band-pass filter of 10–1,000 Hz and amplified 100x (FA-DL-720-140;

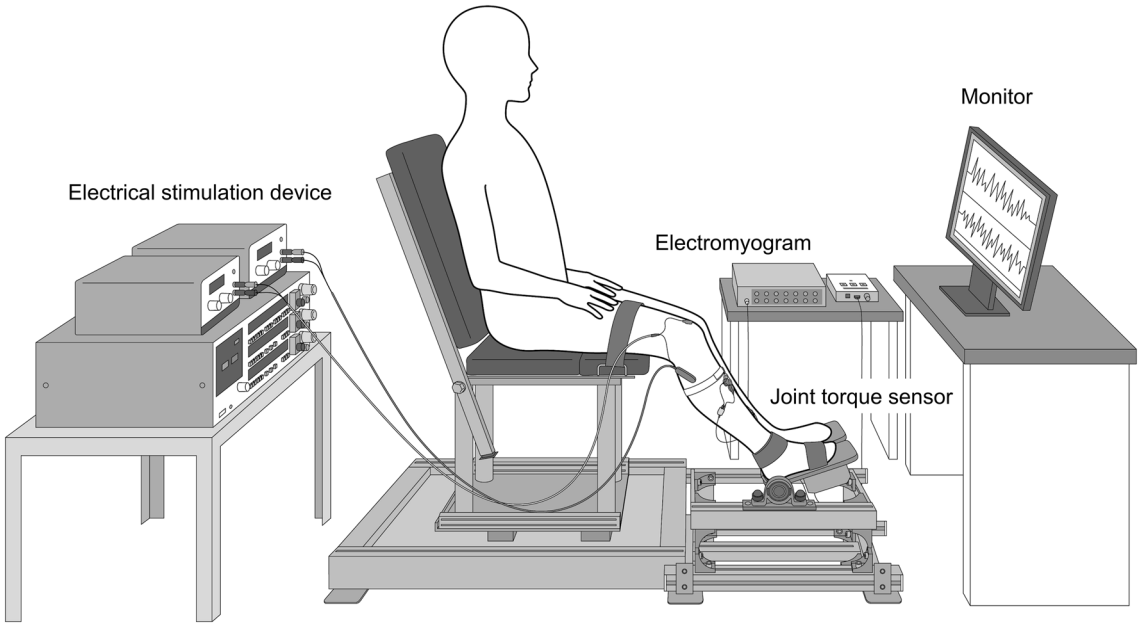

Fig. 1 Limb position for measurements. The right hip flexion (100°), right knee joint (120°), and ankle joint (110°) were measured. We stimulated the dominant deep peroneal nerve of the tibialis anterior muscle (TA). The test stimulus was applied to the dominant tibial nerve of the soleus muscle (Sol). Electrodes were placed on the TA and Sol muscles. The ground electrode was placed between the electrical stimulation electrode and the surface electromyogram electrode

4Assist, Tokyo, Japan) before being digitally stored (10 kHz sampling rate) on a personal computer for offline analysis. Analysis was performed using PowerLab 8/30 (AD Instruments, Colorado Springs, CO, USA) and LabChart 7 (AD Instruments).

Joint torque

Joint torque was measured with a joint torque sensor (TAKEI SCIENTIFIC INSTRUMENTS) attached to the shaft of the foot plate used for ankle joint fixation. Joint torque was filtered at a low-pass of 190 Hz (T-K-K-1268b; TAKEI SCIENTIFIC INSTRUMENTS) before being digitally stored (500 Hz sampling rate) on a personal computer for offline analysis using PowerLab 8/30 (AD Instruments). Data were analyzed using LabChart 7 (AD Instruments).

Electrical stimulation

Nerves were stimulated for 1 ms (rectangular wave) using a SEN-8203 electrical stimulation device (Nihon Kohden, Tokyo, Japan) via a SS-104J isolator (Nihon Kohden). The tibial nerve was selectively stimulated in a monopolar fashion to induce the Sol H-reflex and M waves. The anode and cathode were located on the upper patella and popliteal area, respectively, for the test stimulus. M waves were induced in the TA muscle via bipolar stimulation and the conditioning stimulus was applied along the deep peroneal nerve below the fibula head (Mizuno et al. 1971; Crone et al. 1987; Nielsen and Kagamihara 1992).

Measurement of spinal reciprocal inhibition

Spinal reciprocal inhibition was measured as previously described (Nielsen and Kagamihara 1992). A test stimulus was applied to the dominant (tibial) nerve of the Sol after a conditioning stimulus was delivered to the dominant (deep peroneal) nerve of the TA, and the Sol H-reflex amplitude was then recorded. Condition stimulation preceding the deep peroneal nerve inhibits the excitability of Sol's spinal cord anterior horn cells via inhibitory interneurons. Therefore, when the test stimulus is applied to the tibial nerve later, the Sol H-reflex amplitude value decreases. The intensity of the conditioning stimulus was set to the M wave threshold of the TA (Mizuno et al. 1971). The conditioning stimulus was carefully positioned to avoid activation of the peroneus muscles, ensuring selective stimulation of the deep peroneal nerve (Yamaguchi et al. 2018). Because the amount of spinal reciprocal inhibition varies with the size of the H-reflex (Crone et al. 1990), the intensity of the test stimulus was set to elicit H-reflex reaching 15–25% of the maximum amplitude of the Sol M wave (Mmax). The four stimulation conditions comprised a conditioning stimulus–test stimulation interval (CTI) of − 2, 2, or 20 ms plus a test stimulus without a conditioning stimulus (single). CTI of − 2 ms shows whether there is any influence of the condition

stimulus, whereas 2 ms is the stimulation condition with the most reciprocal Ia inhibition amount (Mizuno et al. 1971; Nielsen and Kagamihara 1992), and 20 ms is the stimulation condition with the most D1 inhibition amount (Mizuno et al. 1971). The number of stimuli was randomly determined for the four stimulation conditions. There were a total of 60 stimulations (4 stimulation conditions × 5 times × 3 sets) and delivered at a frequency of 0.3 Hz. When the stimulation frequency was 0.3 Hz, because H-reflex stabilizes after the third time (Floeter and Kohn 1997), there were at least three stimulations before measurement. There was a 1-min. period of rest between sets.

Co-contraction task

The contraction intensities of TA and Sol were determined by measuring the maximal voluntary contraction (MVC). MVC performed plantar and dorsiflexion with the thighs and feet fixed as shown in Fig. 1. Based on the previous study (Hirabayashi et al. 2018), co-contraction task recognized reciprocal Ia inhibition with co-contraction of ≤ 15% MVC; therefore, it was set as ≤ 15% MVC. The co-contraction task comprised a set of five tasks (Sol vs. TA; task A, 0% MVC vs. 0% MVC; task B, 5% MVC vs. 5% MVC; task C, 15% MVC vs. 15% MVC; task D, 5% MVC vs. 15% MVC; task E, 15% MVC vs. 5% MVC) (Fig. 2). After executing task A, the four tasks were performed in a random order with a rest period of 3 min between tasks. A monitor positioned in front of the participant displayed the amount of muscle activity. For the electromyogram waveform, the raw waveform was processed to full-wave rectification and then set as a 501 point smoothing waveform. During co-contraction, the foot was immobilized to prevent any change in the angle of the ankle. Prior to the experiment, participants practiced achieving constant Sol and TA muscles without moving the ankle. To guide the subjects, we increased the rigidity of only the ankle joint, fed back electromyogram (EMG) from the monitor installed in front of the subject, and adjusted the muscle activity. The background EMG level of each co-contraction task is the averaged EMG 30–50 ms before the test stimulus (Table 1).

Experimental protocol

The experimental procedure is shown in Fig. 3. The MVC of TA and Sol was measured and then 5% and 15% MVC was calculated for each muscle. Subjects rested for 1 min. between sets. Spinal reciprocal inhibition was measured for each stimulation condition during each co-contraction task. Between tasks, subjects rested for at least 3 min.

Statistical analysis

The sol H-reflex amplitude and M wave amplitude values were calculated as the mean ± standard error of the peak-to-peak values of the amplitude of each waveform. Spinal reciprocal inhibition for each co-contraction task was calculated as a percentage (%) by dividing the Sol H-reflex amplitude by the Mmax amplitude [(Sol H-reflex amplitude/Mmax amplitude) × 100]. For the comparisons of the single condition of each co-contraction task and the single condition vs. the other three conditions for each task, iterative values were generated using repeated measures two-way analysis of variance (ANOVA) involving the factors, co-contraction task, and stimulation conditions. For post hoc analysis, the Tukey–Kramer multiple comparison test was

Table 1 EMG level

	Sol	TA	Sol/TA ratio
Task B	4.53 ± 0.17	4.95 ± 0.21	0.95 ± 0.05
Task C	15.30 ± 0.38	15.13 ± 0.52	1.03 ± 0.03
Task D	4.75 ± 0.21	16.03 ± 0.49	0.30 ± 0.02
Task E	15.40 ± 0.45	5.11 ± 0.38	3.27 ± 0.23

(% MVC) (mean ± standard error)

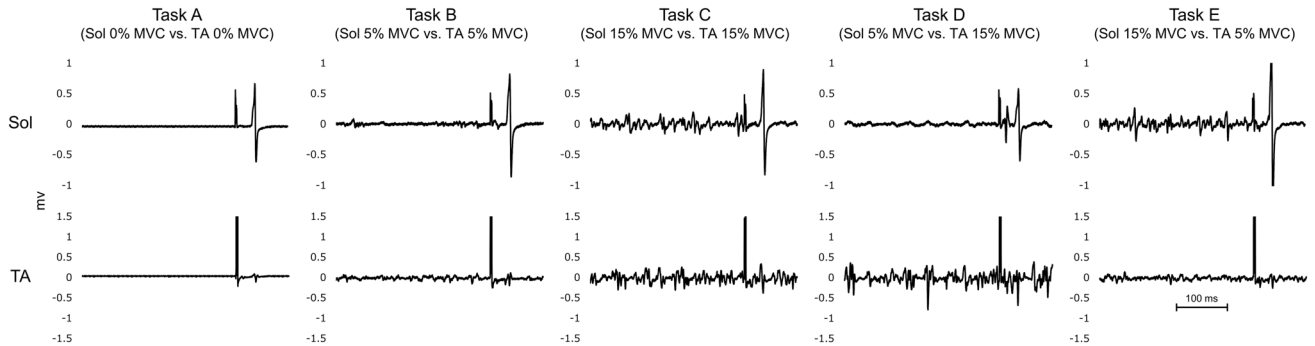

Fig. 2 Co-contraction task. This figure shows muscle activity during co-contraction in one subject. The electromyogram of the Sol, M wave, and H wave appear after the artifacts of electrical stimulation. *Sol* soleus muscle, *TA* tibialis anterior muscle, *MVC* maximal voluntary contraction

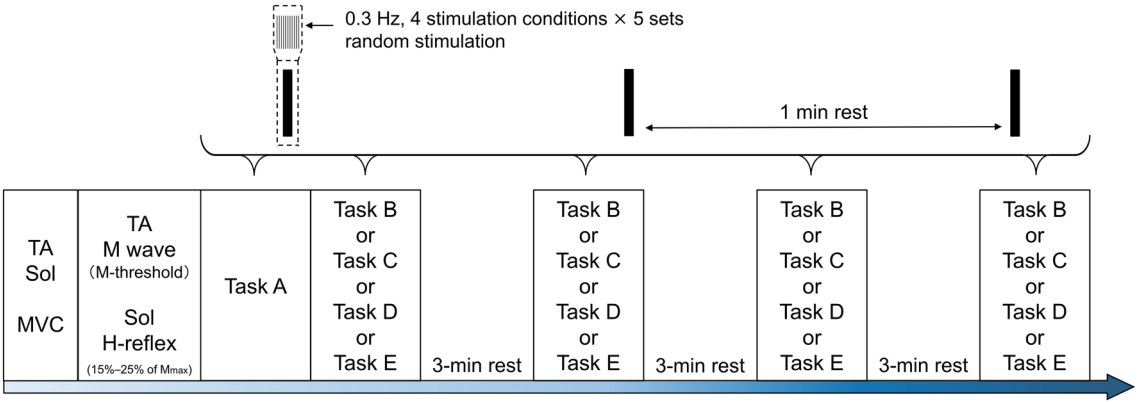

Fig. 3 Experimental protocol. The co-contraction tasks (Sol vs. TA) are task A (0% MVC vs. 0% MVC), task B (5% MVC vs. 5% MVC), task C (15% MVC vs. 15% MVC), task D (5% MVC vs. 15% MVC), and task E (15% MVC vs. 5% MVC). *Mmax* maximum motor response, *MVC* maximal voluntary contraction, *Sol* soleus, *TA* tibialis anterior muscle

used to compare the single conditions of each co-contraction task and the three stimulation conditions were compared with the single condition using paired *t* tests with Bonferroni correction for multiple comparisons.

To compare the amount of spinal reciprocal inhibition between simultaneous contraction tasks under the three stimulation conditions (excluding the single condition), the H-reflex amplitude of the conditioning stimulus was divided by the H-reflex amplitude of the test stimulus only and expressed as a percentage (%) [(conditioned H-reflex amplitude/test H-reflex amplitude) × 100]. Comparison between co-contraction tasks under each stimulus condition was performed using repeated measures two-way ANOVA with the co-contraction task and stimulation condition. The Tukey–Kramer test was used for additional post hoc analysis.

For the analysis of ankle joint torque, the joint torque 30–50 ms before each test stimulus was measured and averaged. We also performed repeated measures one-way ANOVA on the co-contraction task. The Tukey–Kramer test for multiple comparisons was used for post hoc analysis. The level of statistical significance was set at $p < 0.05$.

Results

H-reflex amplitude between stimulation conditions

In the ANOVA of co-contraction task and stimulation conditions, there was a main effect of the CTI [$F(3, 57) = 74.237$, $p < 0.001$, partial $\eta^2 = 0.796$] but not of the co-contraction task [$F(4, 76) = 1.286$, $p = 0.283$, partial $\eta^2 = 0.063$]. In addition, the CTI and co-contraction task significantly interacted [$F(12, 228) = 6.800$, $p < 0.001$, partial $\eta^2 = 0.264$]. There were no significant differences in the single Sol H-reflex amplitude values between the five co-contraction tasks after correction for multiple comparisons (Table 2). These findings confirmed that changes in the Sol H-reflex amplitude in response to conditioning stimuli are not dependent on the test stimulus intensity.

The three stimulation conditions were then compared with the single condition using paired *t* tests (Fig. 4). Compared with the single condition, the H-reflex amplitude was significantly reduced for tasks A, B, C, and D at CTI 2 ($p < 0.001$) and 20 ms ($p < 0.001$). For task E, the H-reflex amplitude was significantly reduced at CTI 20 ms ($p < 0.001$) relative to the single condition.

H-reflex amplitude between co-contraction tasks (Fig. 5)

In the comparison between the co-contraction tasks for each CTI, there was a main effect [$F(2, 38) = 71.388$, $p < 0.001$, partial $\eta^2 = 0.790$] of each CTI and the co-contraction task [$F(4, 76) = 7.548$, $p < 0.001$, partial $\eta^2 = 0.284$]. In addition, the CTI and co-contraction task significantly interacted [$F(8, 152) = 7.135$, $p < 0.001$, partial $\eta^2 = 0.273$]. There were no significant differences between co-contraction tasks at CTI − 2 ms after correction for multiple comparisons. At

Table 2 Test H-reflex amplitudes for co-contraction tasks

	Task A	Task B	Task C	Task D	Task E
Test H-reflection amplitude value (% of Mmax)	19.6 ± 2.6	21.1 ± 2.3	20.0 ± 3.0	19.8 ± 2.4	19.4 ± 2.8

Mean ± standard error

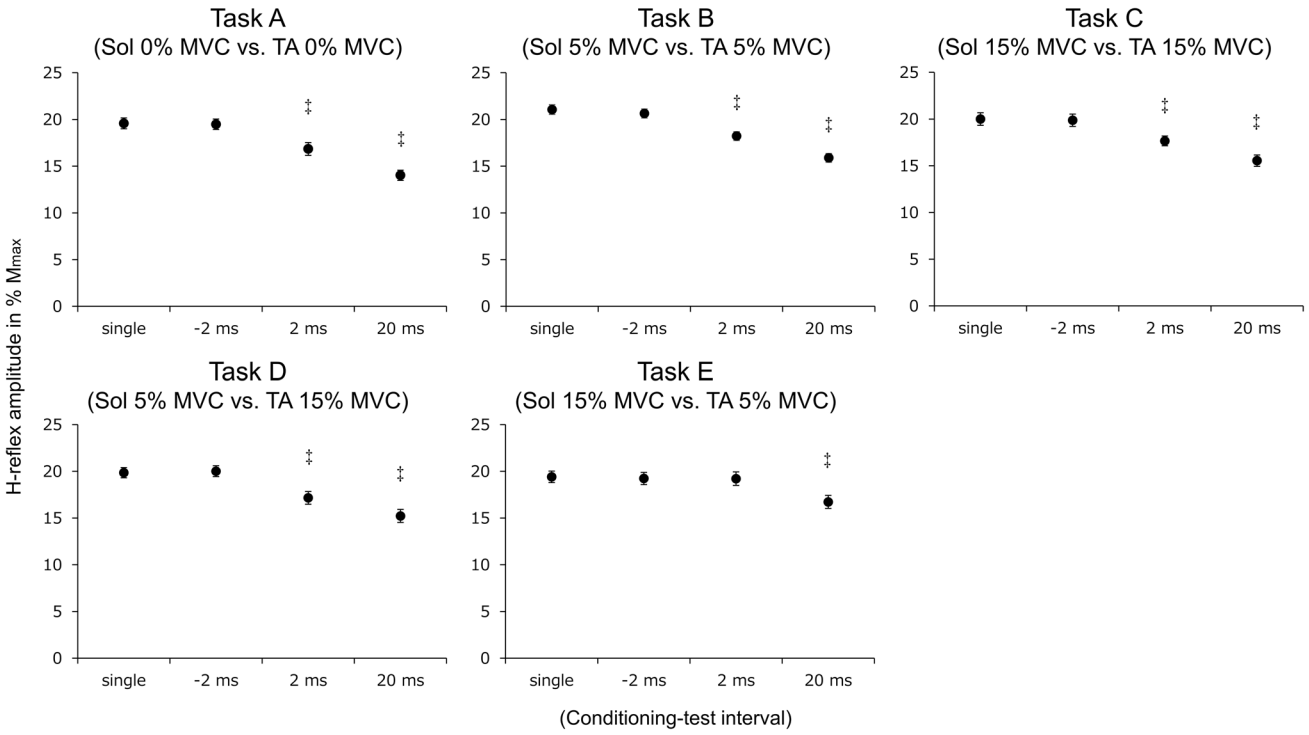

Fig. 4 H-reflex amplitude values between stimulation conditions. The sole H-reflex and M wave amplitude values were calculated as the mean ± standard error of the peak-to-peak values of the amplitude of each waveform. The vertical axis represents H-reflex/Mmax × 100 for the five co-contraction tasks. Data were analyzed by comparing the H-reflex amplitude value of the single condition (divided by Mmax) vs. the H-reflex amplitude value (divided by Mmax) for each of the three conditions (− 2, 2, and 20 ms). This figure shows the data for 20 subjects. MVC, maximal voluntary contraction; $^{\ddagger}p < 0.001$

CTI 2 ms, the H-reflex amplitude decreased significantly for tasks A ($p < 0.001$), B ($p < 0.001$), C ($p = 0.007$), and D ($p < 0.001$) relative to task E. At CTI 20 ms, the H-reflex amplitude decreased significantly for tasks A ($p < 0.001$) and B ($p = 0.032$) relative to task E.

Ankle joint torque (Table 3)

In the comparison of ankle joint torque for each co-contraction task, there was a main effect between the tasks [$F(4, 76) = 51.371$, $p < 0.001$, partial $\eta^2 = 0.730$]. Relative to task A, task C significantly increased ankle joint plantar flexion torque ($p < 0.001$), task D significantly increased ankle dorsiflexion torque ($p = 0.003$), and task E significantly increased ankle joint plantarflexion torque ($p < 0.001$). Relative to task B, task C significantly increased ankle joint plantarflexion torque ($p < 0.001$), task D significantly increased ankle dorsiflexion torque ($p = 0.002$), and task E significantly increased ankle joint plantarflexion torque ($p < 0.001$). Relative to task C, task D significantly increased ankle dorsiflexion torque ($p < 0.001$), whereas task E significantly increased ankle joint plantarflexion torque ($p < 0.001$). Relative to task D, task E significantly increased ankle joint plantarflexion torque ($p < 0.001$).

Discussion

The main findings of this study are that reciprocal Ia inhibition against TA to Sol showed inhibition to the same degree as the Sol/TA muscle activity ratio or co-contraction with high TA muscle activity and there was no inhibition during Sol muscle activity high co-contraction. In addition, the reciprocal Ia inhibition is similar at rest and when the Sol / TA muscle activity ration is equal and when TA activity is high. D1 inhibition from TA to Sol was inhibited during co-contraction when the Sol/TA ratio was comparable or TA muscle activity was high. During co-contraction when the Sol muscle activity ratio is high, the amount of D1 inhibition decreased, but inhibition functions remained.

The results of task A in the present study were comparable to those in previous work (Mizuno et al. 1971; Nielsen and Kagamihara 1992; Okuma et al. 2002), and the Sol H-reflex amplitude was significantly reduced relative to the single condition at CTI 2 ms and 20 ms. The presence of inhibition at CTI 2 ms is consistent with findings from animal experiments (Baldissera et al. 1981) and is thought to be disynaptic reciprocal Ia inhibition (Okuma and Lee 1996). Because inhibition was large at CTI 2 ms, we believe that the excitability of Sol alpha motoneurons was attenuated

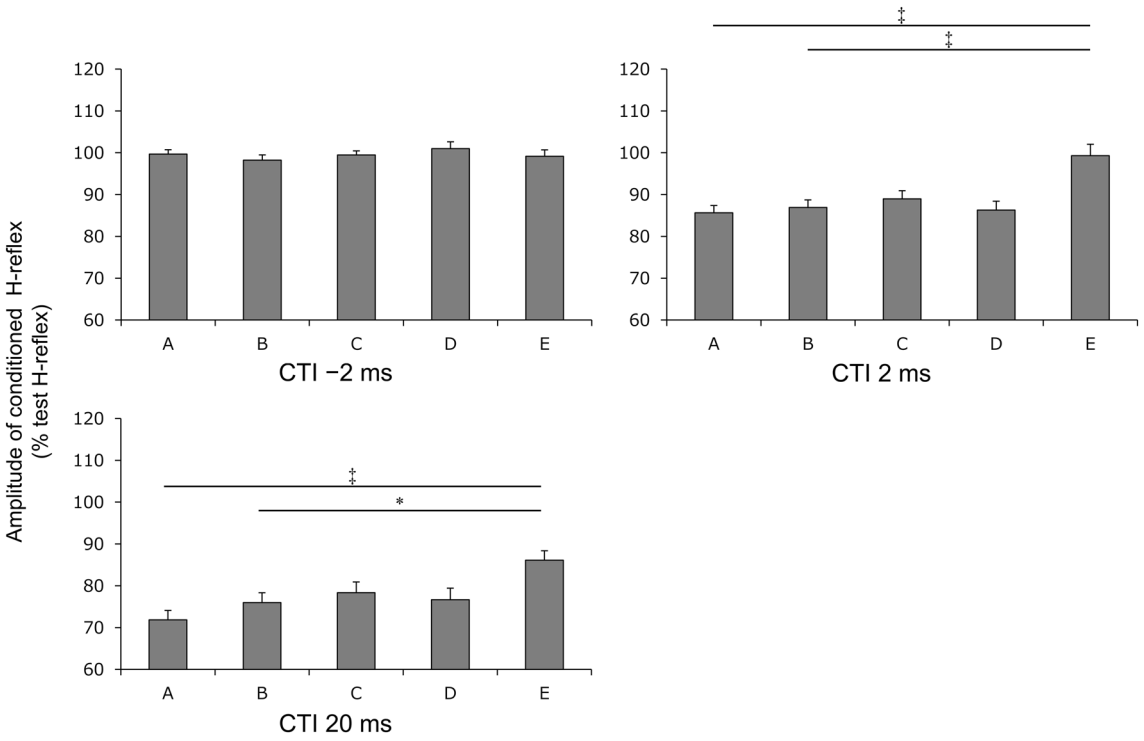

Fig. 5 H-reflex amplitude between co-contraction tasks. The vertical axis indicates the amplitude of the conditioning H-reflex/amplitude of the test H-reflex × 100 of the five co-contraction tasks. The values represent the mean ± standard error. Two factors comprising the co-contraction task and stimulation condition were iteratively measured using two-way repeated measures analysis of variance and the Tukey–Kramer post hoc test for multiple comparisons. MVC, maximal voluntary contraction; $^{*}p < 0.05$, $^{\dagger}p < 0.01$, $^{\ddagger}p < 0.001$

Table 3 Ankle joint torque

Task A	Task B	Task C	Task D	Task E
0.01 ± 0.02	0.10 ± 0.16	3.31 ± 0.83*,†	−2.81 ± 0.47*,†,‡	6.80 ± 0.68*,†,‡,§

(Nm) (mean ± standard error) (+: plantarflexion, −: dorsiflexion)

$p < 0.05$

*vs Task A

†vs Task B

‡vs Task C

§vs Task D

by disynaptic reciprocal Ia inhibition as in previous studies (Nielsen and Kagamihara 1992; Okuma et al. 2002). The presence of inhibition at CTI 20 ms is called D1 inhibition (Mizuno et al. 1971) and is thought to inhibit the excitability of soleus α motor neurons by presynaptic inhibition at the terminal of afferent Ia fibers (Tanaka 1974; El-Tohamy and Sedgwick 1983; Morita et al. 2001).

Due to reciprocal Ia inhibition at CTI 2 ms, the H-reflex amplitude decreased significantly in tasks A, B, C, and D relative to the single condition. Since the H-reflex amplitudes also decreased in tasks B and C, it was revealed that reciprocal Ia inhibition works during co-contraction when Sol and TA are less than 15% MVC of the same muscle activity amount. In a previous study (Nielsen and Kagamihara 1992), there was no reciprocal Ia inhibition during co-contraction and it was believed that reciprocal Ia inhibition was modulated depending on the ankle joint plantarflexion torque. However, the H-reflex amplitude value during co-contraction seems to decrease when it is observed around 2 ms. Co-contraction is not only when the joint torque is 0 Nm. Therefore, using co-contraction, which is an indicator of muscle activity, we revealed reciprocal Ia inhibition in tasks B and C. These findings are in line with the results of our previous work (Hirabayashi et al. 2018). In task D, the muscle activity of TA was high and the dorsiflexion torque of the ankle joint torque was 2.81 Nm. Previous studies have reported that reciprocal Ia inhibition is not affected by the amount of contraction intensity during ankle dorsiflexion

(Nielsen and Kagamihara 1992). Reciprocal Ia inhibition was equivalent between tasks A and D in the present study. During co-contraction when the Sol/TA ratio is equal or TA activity is high (tasks B, C, and D), reciprocal Ia inhibition may work.

Task E showed no significant change in H-reflex amplitude relative to the single condition. In the comparison between tasks, the H-reflex amplitude of task E was significantly increased relative to tasks A, B, C, and D and reciprocal Ia inhibition was not observed. Previous studies (Nielsen and Kagamihara 1992) have reported that ankle joint plantarflexion torque is 4 Nm or greater and reciprocal Ia inhibition decreases; when ankle joint plantarflexion torque is 8 Nm or more, reciprocal Ia inhibition has not been confirmed. Task E showed that ankle joint plantarflexion torque was 6.80 Nm and reciprocal Ia inhibition suppressed, consistent with previous reports. As a mechanism to suppress reciprocal Ia inhibition from TA during Sol contraction, Ib inhibition and repetitive inhibition (Renshaw cells) from Golgi tendon organs without reciprocal inhibition function suppress inhibitory interneurons from TA (Jankowska and McCrea 1983). Recurrent inhibition mediated by Renshaw cells inhibits Ia inhibitory interneurons from antagonist muscle (Hultborn et al. 1971; Katz et al. 1991; Baret et al. 2003) and inhibits spinal reciprocal inhibition (McIntire et al. 1997). Thus, it is conceivable that reciprocal Ia inhibition disappeared due to the high muscle activity of Sol relative to TA during co-contraction.

As a result of D1 inhibition at CTI 20 ms, D1 inhibition was observed in all tasks where the amount of muscle activity changed. This revealed that D1 inhibition works even in co-contraction when the ratio of contraction intensity between antagonistic muscles was modulated. D1 inhibition is the inhibition of long latency spinal reciprocal inhibition functions. The mechanism of D1 inhibition is thought to be multi-synaptic inhibition with group II fibers as centripetal inputs (El-Tohamy and Sedgwick 1983), whereas the condition stimulation that induces D1 inhibition is thought to be presynaptic inhibition at terminal Ia because it does not inhibit the exercise evoked potential due to cerebral magnetic stimulation (Faist et al. 1996). In the literature to date, there has been no report on the effect of contraction intensity change and D1 inhibition during co-contraction; however, D1 inhibition is reported to work during ankle joint dorsiflexion, the amount of D1 inhibition did not change significantly in response to changes in contraction intensity, and plantarflexion has the same D1 inhibition as at dorsiflexion (El-Tohamy and Sedgwick 1983; Morita et al. 2001). Focusing on change in contraction intensity, it has been reported that even if plantarflexion torque increases, the inhibited amount remains unchanged at rest (Morita et al. 2001). In the present study, the comparison between contraction intensities showed that task E had significantly reduced D1 inhibition relative to tasks A and B. From this, it was found that during co-contraction when the Sol/TA ratio has a high proportion of Sol muscle activity, the amount of D1 inhibition from TA to Sol decreases. However, D1 inhibition differed from reciprocal Ia inhibition, and the amount of D1 inhibition decreased in task E but did not disappear. In a previous study (El-Tohamy and Sedgwick 1983) where ankle joint plantarflexion was performed with isometric contraction, D1 inhibition tended to decrease even when plantarflexion torque increased during isometric contraction of ankle joint plantarflexion, although D1 inhibition worked. The reason for this is that reciprocal Ia inhibition and D1 inhibition have different inhibition pathways. Reciprocal Ia inhibition inhibits Sol spinal anterior horn cells directly via inhibitory interneurons and the inhibition time is on the scale of several ms (Mizuno et al. 1971; Nielsen and Kagamihara 1992). In addition, many inhibitory receptors are glycinergic synapses (Aprison and Werman 1965; Davidoff et al. 1967a, b). In contrast, D1 inhibition inhibits excitation of Sol spinal anterior horn cells by presynaptic inhibition at the terminal Ia and inhibition time is several tens of ms (Mizuno et al. 1971; El-Tohamy and Sedgwick 1983; Morita et al. 2001). Many inhibitory receptors are GABAergic synapses (Rudomin et al. 1990), which are known to have a higher filling efficiency (Christensen et al. 1991; McIntire et al. 1997) and longer inhibition time relative to glycinergic synapses (Jonas et al. 1998; Gao et al. 2001). Therefore, D1 inhibition may be due to the long inhibition time, effects of repression on Ia afferent fiber terminals of Sol, and differences in inhibitory sites and inhibitory receptors.

The present study revealed that reciprocal Ia inhibition and D1 inhibition, which are functions of spinal reciprocal inhibition, work during low-intensity co-contraction. Although it is considered that spinal reciprocal inhibition does not occur during the co-contraction process to increase the rigidity of the joints, the function of inhibitory interneurons against antagonistic muscles may still be present during low co-contraction. However, the present study examined spinal reciprocal inhibition targeting the lower leg. In daily life, the lower leg is a weight-bearing body part. Since this study did not apply a load to the foot, it is necessary to consider the influence on standing balance separately from cooperation between agonist and antagonist muscles while walking. The findings of this study help clarify the relationship between co-contraction and spinal reciprocal inhibition in the ankle joint. We believe that these findings can be applied in future studies of spinal reciprocal inhibition, such as in standing balance and walking where excessive co-contraction occurs.

This study is subject to two main limitations. The first is specifying the CTI. We set the CTI as the maximum value of inhibition of reciprocal Ia inhibition and D1 inhibition (El-Tohamy and Sedgwick 1983; Nielsen and Kagamihara

1992). Since we did not measure the Sol H-reflex amplitude for other CTIs, the inhibition amount for such conditions is unknown. The second is that we only studied contraction intensities of 5% and 15% MVC. To examine the influence of Sol/TA ratio in greater detail, investigation of other contraction intensities is needed in future work. Another limitation of this investigation is that it is unclear whether the muscle activity reached the peak at the time of MVC measurement, especially Sol. We fixed the thighs and feet tightly and took maximum care to make MVC possible. Furthermore, because it is Sol, the knee joint was performed in flexion position to eliminate the influence of the gastrocnemius muscle.

By focusing on contraction intensity (muscle activity) of Sol and TA, this study clarified the influence of spinal reciprocal inhibition during co-contraction. It was revealed that reciprocal Ia inhibition and D1 inhibition worked at 15% MVC or less during co-contraction when Sol and TA muscle activity was the same. This finding suggests that the Sol/TA ratio may play a role in reciprocal Ia inhibition by changing the proportion of contraction intensity of Sol and TA. D1 inhibition does depend on the Sol/TA ratio; however, during co-contraction when Sol activity is high, the inhibitory amount decreased while D1 inhibition function was remained.

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Compliance with ethical standards

Conflict of interest No conflicts of interest, financial or otherwise are declared by the authors.

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