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RESEARCH ARTICLE

Effects of repetitive passive movement on ankle joint on spinal reciprocal inhibition

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Abstract

Repetitive passive movement (RPM) activates afferent Ia fibers. The input of afferent Ia fibers from antagonist muscle may modulate the extent of spinal reciprocal inhibition (RI). However, effects of RPM on RI remain unknown. We aimed to clarify these effects in 20 healthy adults. Four RPM tasks (40°/s, 80°/s, 120°/s, and 160°/s), with the range of ankle joint movement set to 40°, ranging from 10° in dorsiflexion to 30° in plantar flexion, were performed for 10 min. For measuring RI, a deep peroneal nerve as a conditioning stimulus, tibial nerve as a test stimulus, and three condition–test stimulus intervals (CTIs; single, 2 ms, and 20 ms) were used. The stimulation frequency was 0.3 Hz for 36 times (3 stimulation conditions × 12 sets). RI was measured before, immediately after, and 5, 10, 15, and 20 min (Pre, Post 5, 10, 15, and 20, respectively) after the task. The extent of reciprocal Ia inhibition (CTI 2 ms) significantly increased in Post 5 and 10 at RPM speed of ≥ 120°/s. The extent of D1 inhibition (CTI 20 ms) significantly increased in Post 5 and 10 at RPM speed of ≥ 80°/s, and continued to increase until Post 15 at RPM speed of 160°/s. The extent of RI was the highest at RPM speed of 160°/s for both Ia and D1. Therefore, high RPM may increase the extent of reciprocal Ia inhibition and D1 inhibition, suggesting that rapid movements affect RI by increasing the firing frequency from the muscle spindle to afferent Ia fibers.

Keywords H-reflex · M wave · Electromyograph · Joint movement · Electrical stimulation

Introduction

In various upper motor neuron disorders, such as spastic diseases, cerebellar ataxia, Parkinson's disease, and spinal cord injury, the collapse of the spinal reciprocal inhibition (RI) mechanism against the antagonist may cause excessive simultaneous muscle activation when movement of only one agonist muscle is required (e.g., during joint movement). In other words, RI fails to affect the antagonist, leading to the impairment of smooth joint movement in these disorders (Hayashi et al. 1988; Kagamihara and Tanaka 1996). In addition to pathological states, simultaneous muscle activation increases with age (Morita et al. 2000; Hortobagyi et al. 2009; Baudry et al. 2010; Nagai et al. 2011). In athletes, excessive simultaneous muscle activation may interfere with joint movement and reduce agility (Blackwell and Cole 1994).

Excessive simultaneous muscle activation is caused by reduced presynaptic inhibition (Milanov 1992; Kagamihara and Masakado 2005) at the terminal ends of Ia fibers, which are afferent fibers from muscle spindles, as well as by enhanced stretch reflex due to decreased post-activation depression (Nielsen et al. 1995). During joint movement, decreased reciprocal Ia inhibition from agonist Ia fibers to antagonist muscles (Mizuno et al. 1971; Nielsen and Kagamihara 1992; Okuma et al. 2002) decreases presynaptic inhibition (D1 inhibition) (Mizuno et al. 1971; Tanaka 1974; Crone and Nielsen 1989; Nielsen et al. 1995) and antagonist-prompting input (Crone et al. 2000). Taken together, these findings suggest that many factors contribute to excessive simultaneous muscle activation.

In recent years, several studies enhancing RI to inhibit excessive simultaneous activity have been reported and are attracting attention (Kubota et al. 2015; Yamaguchi et al. 2016; Ritzmann et al. 2018; Yamaguchi et al. 2018). Previously, we have reported on repetitive passive movement

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(RPM) as a widely used rehabilitation technique (Miyaguchi et al. 2013; Sasaki et al. 2017, 2018). Moreover, the excitability of the primary somatosensory cortex (S1) and primary motor area (M1) was reportedly modulated after RPM. The firing frequency of the muscle spindle was increased with increased RPM speed, while the excitability of M1 was inhibited after RPM (Sasaki et al. 2017). Based on these findings, we assumed that the input of afferent Ia fibers increases, affecting reciprocal Ia inhibition and DI inhibition. However, the association between RPM and RI is a novel topic. Moreover, if RPM can bring about RI, excessive simultaneous muscle activity may be inhibited. Therefore, the purpose of the present study was to clarify the effects of RPM on RI.

The hypothesis of this study is founded on the assumption that faster RPM increases the firing frequency from muscle spindles to afferent Ia fibers, promoting RI and prolonging its duration.

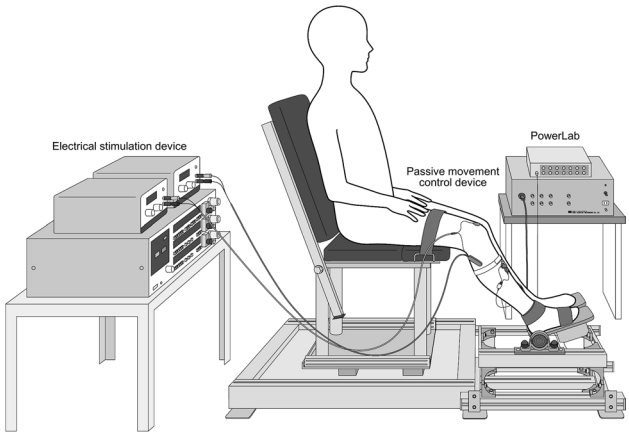

Fig. 1 Limb position during measurements. Right hip (100°), right knee joint (120°), and ankle joint (110°) flexion was measured. The dominant deep peroneal nerve of the tibialis anterior muscle (TA) was stimulated. The test stimulus was applied to the dominant tibial nerve of the soleus muscle (Sol).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

Materials and methods

Study participants

A total of 20 healthy adults (10 males and 10 females; age, 20.4 ± 0.5 years; height, 166.0 ± 8.4 cm; body weight, 56.0 ± 8.2 kg) provided written informed consent to participate in this study. The study was approved by the Ethics Committee of Niigata University of Health and Welfare (18155-190311). All experiments were performed 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

The right lower limb position was measured at the hip (100°), knee (120°), and ankle (110°) joints. To maintain the participant's position during the experiment, the lower leg was immobilized to the seat and the foot was immobilized to the foot plate (Takei Scientific Instruments, Niigata, Japan; Fig. 1).

Experimental protocol

The experimental procedure is illustrated in Fig. 2. RPM tasks at four speeds (40°/s, 80°/s, 120°/s, and 160°/s) were randomly performed for 10 min. RI was assessed before (Pre), immediately after (Post), and 5 (Post 5), 10 (Post 10), 15 (Post 15), and 20 (Post 20) min after the task.

Electromyography

The distance between the electrodes of a surface electromyogram (EMG) was set to 20 mm using an Ag/AgCl electrode (Blue Sensor, METS, Tokyo, Japan). Electrodes were placed on the tibialis anterior (TA) and soleus (Sol) muscles in accordance with 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 10–1000 Hz and amplified 100 × (FA-DL-720-140; 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).

RPM tasks

In this study, RPM was performed with a passive movement control device (movement device) used to set the movement speed and joint angle (Takei Scientific Instruments; Fig. 3). The range ankle joint movement of the device was set to 40° total, ranging from 10° in dorsiflexion to 30° in plantar flexion. This ankle joint angle range is within the normal range of motion (Baumbach et al. 2014), and the participants reported experiencing no pain during passive movement. RPM tasks were performed at a speed of 40°/s, 80°/s, 120°/s, or 160°/s. The interval between

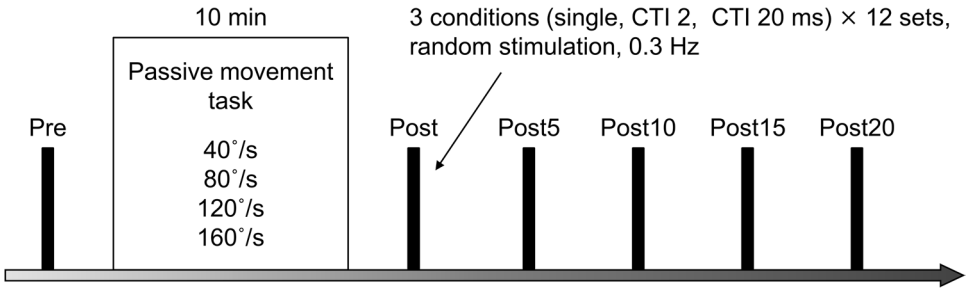

Fig. 2 Experimental protocol. Four RPM tasks were performed (40°/s, 80°/s, 120°/s, and 160°/s). RI was measured under three conditions (single, CTI 2 ms, and CTI 20 ms) before (Pre), immediately after (Post), and at 5 (Post 5), 10 (Post 10), 15 (Post 15), and 20 (Post 20) min after the RPM tasks. All values are in minutes. *CTI* conditioning stim-test–stim interval, *RI* reciprocal inhibition, *RPM* repetitive passive movement

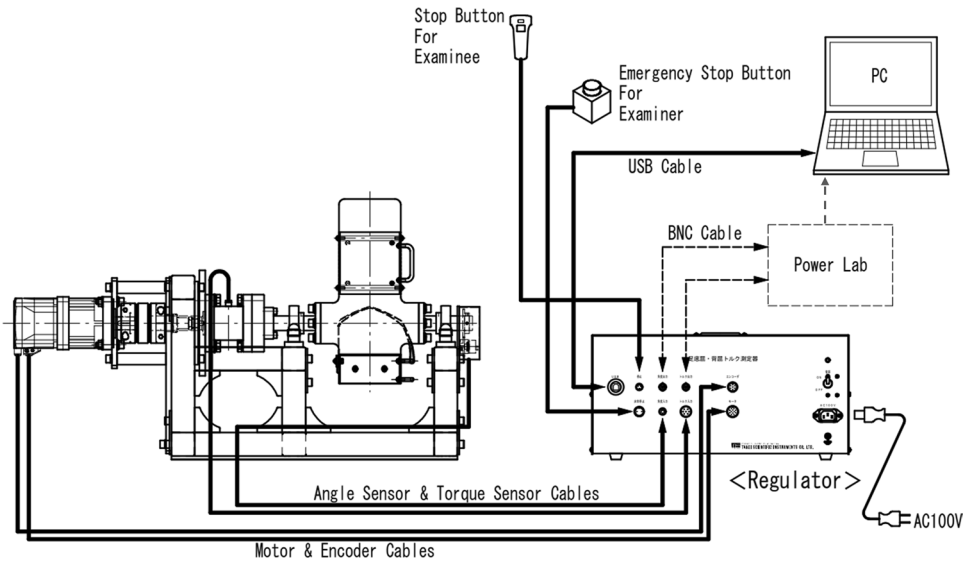

Fig. 3 Passive movement control device. This figure is a simplified diagram of the equipment connected to the passive movement control device

tasks was 200 ms (Fig. 4a–c). The execution time of each task was set to 10 min, as previously described (Miyaguchi et al. 2013; Sasaki et al. 2017, 2018). During RPM tasks, participants looked forward and not toward their lower extremities, and they were instructed to not image the joint movement. During the RPM task, electromyogram was constantly observed to check that no muscle contraction occurred in the TA and Sol. The experimental environment was set up, such that the participant or experimenter could press an emergency stop button to prevent a serious accident caused by malfunction of the movement device.

Electrical stimulation

Nerves were stimulated for 1 ms (rectangular wave) using an SEN-8203 electrical stimulation device (Nihon Kohden, Tokyo, Japan) with an SS-104J isolator (Nihon Kohden). The tibial nerve was selectively stimulated in a monopolar manner to induce a Sol H-reflex and M waves. 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; Hirabayashi et al. 2018; Yamaguchi et al. 2018; Hirabayashi et al. 2019).

Measurement of spinal RI

RI was measured as previously described (Mizuno et al. 1971; Hirabayashi et al. 2018; Yamaguchi et al. 2018; Hirabayashi et al. 2019). A test stimulus was applied to the dominant (tibial) nerve of Sol after a conditioning stimulus was delivered to the dominant (deep peroneal) nerve of TA. The Sol H-reflex amplitude was then recorded. Conditioning stimulation prior to deep peroneal nerve stimulation inhibits the excitability of spinal cord anterior horn cells of Sol via inhibitory interneurons. Therefore, when the test stimulus is later applied to the tibial nerve, the Sol H-reflex amplitude

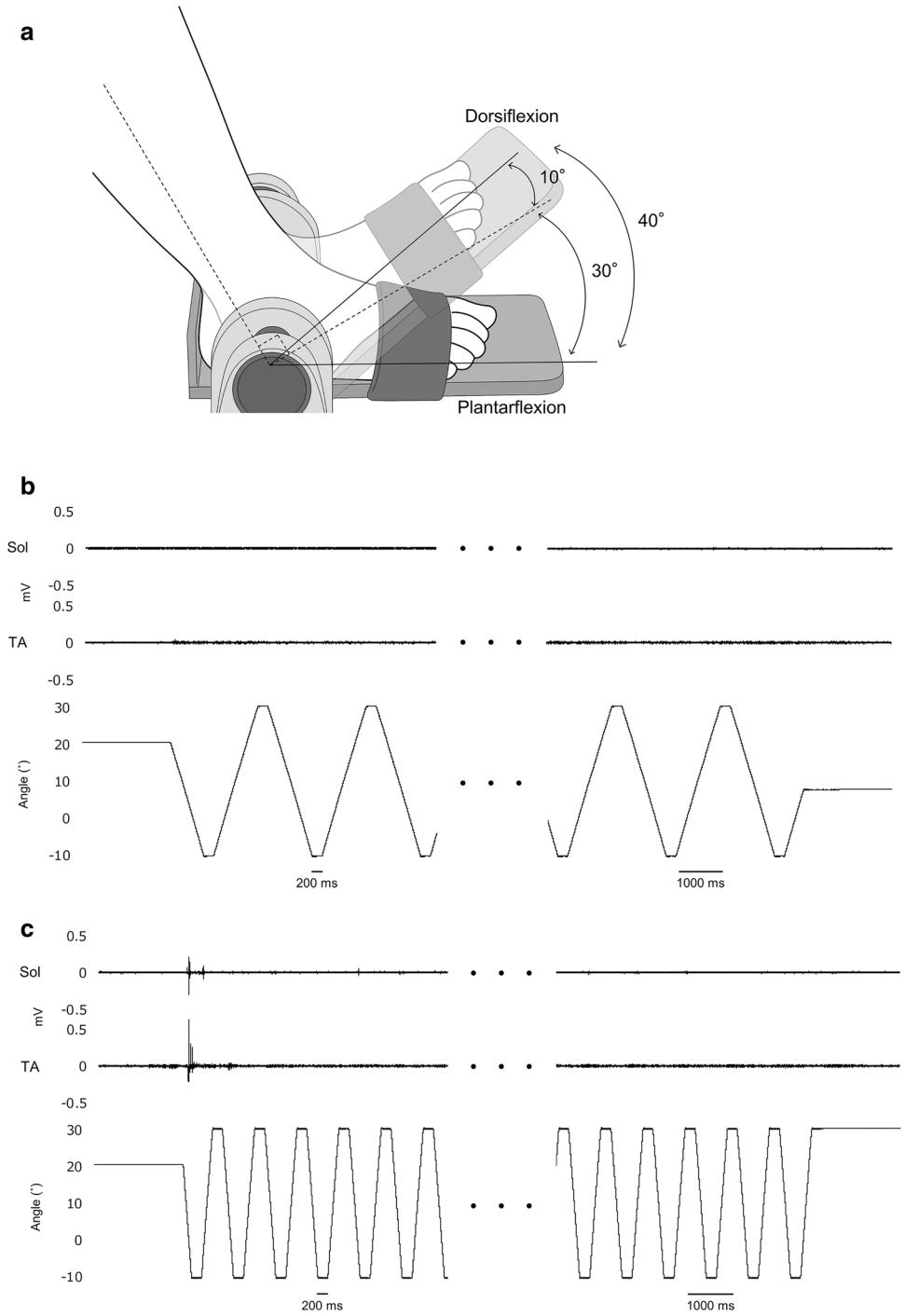

Fig. 4 RPM tasks. **a** A diagram showing the ankle range of movement 40°, ranging from 30° in ankle plantar flexion to 10° in ankle dorsiflexion. RPM tasks at 40°/s (**b**) and 160°/s (**c**) are shown. The interval was 200 ms. The upper electromyogram indicates Sol and the lower electromyogram indicates TA. The bottom panel indicates the joint angle. *RPM* repetitive passive movement, *Sol* soleus, *TA* tibialis anterior muscle

decreases. The intensity of the conditioning stimulus was set to the M-wave threshold of TA (stimulus intensity that induces 100 µV or less) (Mizuno et al. 1971; Yamaguchi et al. 2018). The conditioning stimulus was carefully positioned to avoid the activation of the peroneus muscles, ensuring the selective stimulation of the deep peroneal nerve (Hirabayashi et al. 2018; Yamaguchi et al. 2018; Hirabayashi et al. 2019). Because the extent of spinal RI varies with the size of the H-reflex (Crone et al. 1990), the intensity of the test stimulus was set to elicit an H-reflex of 15–25% of the maximum amplitude of the Sol M wave (Mmax). The three stimulation conditions comprised a conditioning stimulus–test stimulation interval (CTI) of 2 or 20 ms plus a test stimulus without a conditioning stimulus (single). The CTI of 2 ms is the stimulation condition with the highest reciprocal Ia inhibition (Mizuno et al. 1971; Nielsen and Kagamihara 1992), whereas 20 ms is the stimulation condition with the highest D1 inhibition (Mizuno et al. 1971).

Three stimulation conditions were randomly stimulated, and the number of stimulations was 36 times more (3 stimulation conditions × 12 sets). The stimulation frequency was 0.3 Hz. When the stimulation frequency reached 0.3 Hz, at least three stimulations were applied before measurement because the H-reflex stabilizes after the third stimulus (Floeter and Kohn 1997).

Statistical analysis

The Sol H-reflex amplitude and M-wave amplitude were calculated as mean ± standard error of the peak-to-peak values of the amplitude of each waveform. RI with each RPM task was calculated as a percentage (%) by dividing the Sol H-reflex amplitude by the Mmax amplitude (Sol H-reflex amplitude in %Mmax). In addition, when comparing the changes between each task and temporal change, the H-reflex amplitude value of the test stimulus subjected to the conditional stimulus was divided by the H-reflex amplitude value of the test stimulus alone to calculate the % notation [(amplitude of conditioned H-reflex amplitude/test H-reflex amplitude) × 100]. The effect of the RPM task × stimulation condition × measurement time was assessed by a repeated measures three-way analysis of variance (ANOVA). A repeated-measures two-way ANOVA, repeated measures one-way ANOVA, and the Tukey–Kramer multiple comparison test were performed for the post hoc analysis. The stimulation conditions (single and two stimulation conditions) for each RPM task were compared using paired *t* tests with the Bonferroni correction. The level of statistical significance was set at $p < 0.05$.

Results

Interactions of RPM task, stimulation condition, and measurement time

Repeated-measures three-way ANOVA (RPM task × stimulation condition × measurement time) revealed a main effect of RPM task [$F(3, 57) = 53.821$, $p < 0.001$, partial $\eta^2 = 0.739$], stimulation condition [$F(2, 38) = 40.207$, $p < 0.001$, partial $\eta^2 = 0.679$], and measurement time [$F(5, 95) = 177.501$, $p < 0.001$, partial $\eta^2 = 0.903$]. Moreover, there was a significant interaction among the three factors [$F(30, 570) = 185.786$, $p < 0.001$, partial $\eta^2 = 0.907$]. Two-factor repeated-measures ANOVA showed an interaction of RPM task × stimulus condition [$F(6, 114) = 88.008$, $p < 0.001$, partial $\eta^2 = 0.822$], RPM task × measurement time [$F(15, 285) = 204.470$, $p < 0.001$, partial $\eta^2 = 0.915$], and stimulus condition × measurement time [$F(10, 190) = 177.162$, $p < 0.001$, partial $\eta^2 = 0.903$].

H-reflex amplitude of the single condition

There were no significant differences in Sol H-reflex amplitude values obtained under the same condition at each measurement time (Tables 1, 2). This result confirmed that changes in Sol H-reflex amplitude in response to conditioning stimuli are independent of the test stimulus intensity.

Table 1 H-reflex amplitude values under a single stimulation condition

Task	Pre	Post	Post 5	Post 10	Post 15	Post 20
40°/s	20.8 ± 0.3	20.5 ± 0.4	20.4 ± 0.4	20.4 ± 0.3	20.3 ± 0.2	20.4 ± 0.3
80°/s	20.8 ± 0.5	20.9 ± 0.6	20.4 ± 0.5	20.9 ± 0.5	20.2 ± 0.5	20.5 ± 0.5
120°/s	21.5 ± 0.3	21.8 ± 0.4	21.0 ± 0.4	21.2 ± 0.4	20.8 ± 0.4	21.1 ± 0.3
180°/s	21.2 ± 0.5	21.3 ± 0.6	20.3 ± 0.5	20.4 ± 0.5	20.0 ± 0.5	20.1 ± 0.5

Mean ± standard error

The value was calculated as a percentage (%) by dividing the Sol H-reflex amplitude by the Mmax amplitude (Sol H-reflex amplitude in %Mmax)

Table 2 M-wave amplitude values of the TA

Task	Pre	Post	Post 5	Post 10	Post 15	Post 20
40°/s	80 ± 5	85 ± 3	88 ± 3	85 ± 5	88 ± 2	88 ± 2
80°/s	86 ± 4	83 ± 6	83 ± 4	86 ± 2	84 ± 3	87 ± 3
120°/s	84 ± 6	84 ± 5	87 ± 3	90 ± 4	81 ± 4	82 ± 3
180°/s	81 ± 6	81 ± 3	88 ± 5	88 ± 3	84 ± 4	85 ± 3

Mean ± standard error

The value is M-wave amplitude values of the TA (μV)

Comparison between stimulation conditions

Comparisons of the H-reflex amplitude values under the two stimulation conditions (single or two stimulation conditions) revealed that the H-reflex amplitude was significantly decreased at CTI 2 ms and 20 ms compared to that at the single condition at each measurement time ($p < 0.001$, paired t tests with the Bonferroni correction, Fig. 5). According to the previous study, reciprocal Ia inhibition (CTI 2 ms) and D1 inhibition (CTI 20 ms) were observed under all conditions (Mizuno et al. 1971).

Temporal change of H-reflex amplitude value before and after RPM

The results were analyzed as the amplitude of conditioned H-reflex (% test H-reflex). There were significant differences in the H-reflex amplitude values at each measurement time under CTI 2 ms and 20 ms compared with the Pre value, as assessed using paired t tests with the Bonferroni correction (Fig. 5). Under 2 ms CTI, RPM speed of 120°/s showed a significantly reduced H-reflex amplitude value in Post 5 ($p < 0.01$) and 10 ($p < 0.001$) compared with that at Pre. Similarly, RPM speed of 160°/s significantly decreased the H-reflex amplitude value in Post 5 ($p < 0.001$) and 10 ($p < 0.001$) compared with that at Pre. Under CTI 20 ms, RPM speed of 80°/significantly decreased H-reflex amplitude values in Post 5 ($p < 0.01$) and 10 ($p < 0.01$), compared with that at Pre. Similarly, RPM speed of 120°/s significantly decreased the H-reflex amplitude value in Post 5 ($p < 0.05$) and 10 ($p < 0.05$) compared with that at Pre. Finally, RPM speed of 160°/s significantly decreased the H-reflex amplitude value in Post 5 ($p < 0.001$), 10 ($p < 0.01$), and 15 ($p < 0.05$) compared with that at Pre.

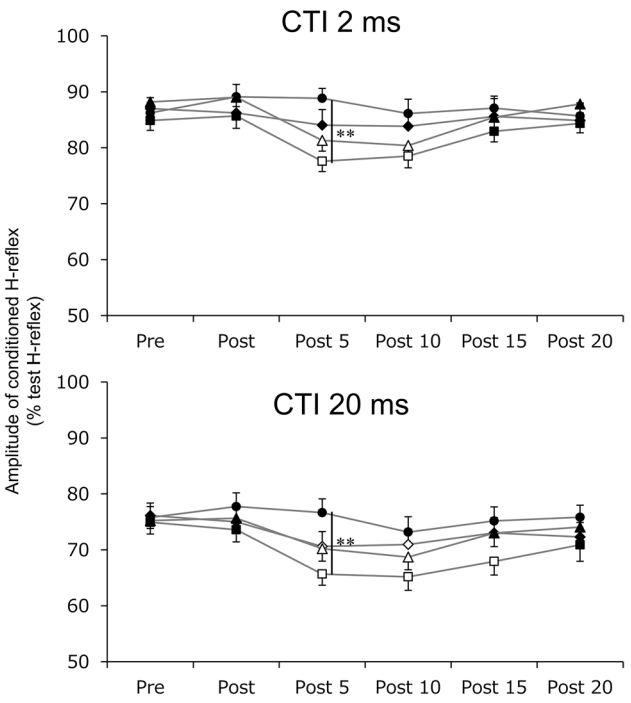

Fig. 5 Comparison between e temporal changes in RI in RPM tasks. Circle indicates 40°/s, rhombus indicates 80°/s, triangle indicates 120°/s, and square indicates 160°/s RPM. The vertical axis indicates the amplitude of the conditioning H-reflex/amplitude of the test H-reflex × 100. The values represent the mean ± standard error. Data were analyzed using paired t tests with a Bonferroni correction to compare Pre vs. other measurement points. Values with filled marks did not show a significant difference compared to Pre. Open marks indicate significant differences compared to Pre ($p < 0.05$). Comparisons among RPM tasks (40°/s, 80°/s, 120°/s, and 160°/s) were performed by multiple comparison tests using Tukey–Kramer. **$p < 0.01$. *RI* reciprocal inhibition, *RPM* repetitive passive movement

Comparison between RPM tasks

The H-reflex amplitude values in Post 5 and 10 were compared between CTI 2 ms and 20 ms using Tukey–Kramer multiple comparison tests. Under CTI 2 ms, the H-reflex amplitude value in Post 5 at RPM speed of 160°/s was significantly reduced compared with that at RPM speed of 40°/s ($p < 0.01$). There were no significant differences in H-reflex amplitude values in Post 10 across RPM tasks under CTI 2 ms. Under CTI 20 ms, the H-reflex amplitude value in Post 5 at RPM speed of 160°/s was significantly decreased compared with that at RPM speed of 40°/s. There were no significant differences in H-reflex amplitude values in Post 10 across RPM tasks under CTI 20 ms.

Discussion

The present study demonstrated that after 10 min of the RPM task, reciprocal Ia inhibition increased from Post 5 to 10 for when RPM speed was ≥ 120°/s. The extent of D1 inhibition increased from Post 5 to 10 at RMP speed of ≥ 80°/s, and it continued to increase up to Post 15 at RPM speed of 160°/s.

The H-reflex amplitude value was significantly reduced under CTI 2 ms and 20 ms compared with that under a single stimulation condition across all RPM speeds (40°/s, 80°/s, 120°/s, and 160°/s) and measurement times (Pre, Post, Post 5, Post 10, Post 15, and Post 20). The presence of inhibition under CTI 2 ms is consistent with previously reported findings of animal experiments (Baldissera et al. 1981) and is thought to be due to disynaptic reciprocal Ia inhibition (Mizuno et al. 1971). Because inhibition was large under CTI 2 ms, we believe that the excitability of Sol α motoneurons was attenuated by disynaptic reciprocal Ia inhibition, which has been shown in the previous studies (Nielsen and Kagamihara 1992; Okuma et al. 2002; Hirabayashi et al. 2018).

The presence of inhibition under CTI 20 ms is termed D1 inhibition (Mizuno et al. 1971) and is thought to inhibit the excitability of Sol α motor neurons via presynaptic inhibition at the terminal ends of afferent Ia fibers (Tanaka 1974; Morita et al. 2001). Thus, this study demonstrated reciprocal Ia inhibition and D1 inhibition at all measurement times and speeds of RPM tasks.

Compared with that in Pre, the extent of reciprocal Ia inhibition was significantly increased in Post 5 and 10 at RPM speeds of 120°/s and 160°/s. Similarly, the extent of D1 inhibition was significantly increased in Post 5 and 10 at RPM speeds of 80°/s and 120°/s and was significantly increased in Post 5, 10, and 15 at RPM speed of 160°/s. Furthermore, the highest reciprocal Ia inhibition and D1 inhibition occurred in Post 5 at RPM speed of 160°/s. However, there have been no previous studies on the association between RPM and RI. Meanwhile, numerous studies have measured brain activity during passive movement using functional magnetic resonance imaging and positron emission tomography, and revealed that RPM without motor commands activates S1 and M1, supplementary motor area (SMA), posterior parietal cortex (PPC), and bilateral secondary somatosensory areas (S2) (Weiller et al. 1996; Alary et al. 1998; Reddy et al. 2001; Radovanovic et al. 2002; Onishi et al. 2013). However, M1 activation in response to passive movement was not observed in patients with severe distal sensory neuropathy, suggesting that peripheral somatosensory afferent activation contributes to M1 activation (Reddy et al. 2001). Given these findings, passive movement increases afferent sensory input and affects many brain areas. In a post-RPM study, Sasaki et al. (2017) have observed the highest decreases in motor-evoked potential amplitude following index finger RPM at a speed of 200°/s, which lasted for up to 15 min after RPM. Simultaneously, they have reported that the excitability of spinal anterior horn cells remained unchanged, because the F wave amplitude value remained unchanged. Regarding the activity of afferent sensory nerves, the firing frequency of muscle spindles reportedly increases with the extension speed and amplitude of muscles (Matthews and Stein 1969). RI activation following RPM at a high speed indicates that the firing frequency of afferent Ia fibers from the muscle spindles of TA may increase and this increase in firing frequency may play a role in the activity of Ia inhibitory interneurons and subsequent Ia presynaptic inhibition.

In reciprocal Ia inhibition, Ia inhibitory interneurons connected to Sol motor neurons receive convergent inputs from the motor cortex and from the Ia afferents of the TA muscle (Nielsen et al. 1993; Masakado et al. 2001). The activity of Ia inhibitory interneurons that synapse directly to Sol anterior horn cells inhibits excitability of Sol motor neurons (Mizuno et al. 1971; Nielsen and Kagamihara 1992). In D1 inhibition, primary afferent depolarization (PAD) interneurons connected to the end of Ia fibers of Sol receive convergent inputs from the motor cortex and from the Ia afferents of the TA muscle. Activity of these PAD interneurons (Jankowska et al. 1981; Meunier and Pierrot-Deseilligny 1998) enhances presynaptic inhibition at Ia fiber terminals connected to Sol motor neurons and inhibits the excitability of Sol motor neurons (Mizuno et al. 1971; Iles and Pisini 1992; Pierrot-Deseilligny and Burke 2012). Patterned electrical stimulation (PES), which specifically stimulates afferent Ia fibers, modulates RI. PES specifically stimulates afferent Ia fibers, and such stimulation enhances reciprocal Ia inhibition via periodic (1.5–2 Hz) stimulation via a pulse train (10 pulses of 100 Hz) (Perez et al. 2003; Fujiwara et al. 2011; Yamaguchi et al. 2016; Takahashi et al. 2017; Yamaguchi et al. 2018). In animals, the PES frequency of primary afferent fibers of ankle flexor muscles that produce short bursts of firing ranged from 100 to 200 Hz at the beginning of the swing phase during stepping (Prochazka and Gorassini 1998). However, Perez et al. (2003) have reported that stimulation with one pulse every 150 ms did not enhance reciprocal Ia inhibition. Thus, periodic stimulation of afferent nerves with high-frequency firing is important to enhance reciprocal Ia inhibition. RPM increases the frequency of regular firings from the muscle spindles to afferent nerves as the muscles stretch. Faster movement speed RPM increased the firing rate of the muscle spindle (Matthews and Stein 1969), suggesting that reciprocal Ia inhibition might be enhanced. In the present study, the extent of D1 inhibition also increased following RPM (80°/s, 120°/s, and 160°/s). In a study using a combination of iTBS and PES, the extent of D1 inhibition was increased (Yamaguchi et al. 2018). A previous study (Yamaguchi et al. 2018) has reported that peripheral stimulation with PES is important during brain area activity. RPM at speeds used in the present study activates M1, S1, SMA, PPC, and S2 (Weiller et al. 1996; Alary et al. 1998; Reddy et al. 2001; Radovanovic et al. 2002; Onishi et al. 2013). Similarly, during RPM, many brain regions are active, and peripheral input from the muscle spindle enters. Therefore, in this study, reciprocal Ia inhibition and D1 inhibition were enhanced by RPM, and the time during which it was enhanced was the same as the inhibition time using iTBS and PES. Therefore, it is a basic knowledge useful for future rehabilitation that simple RPM can produce the same effect without using expensive equipment.

In the present study, both reciprocal Ia inhibition and D1 inhibition showed a delayed after-effect. Delayed after-effects in brain research remain poorly understood, although some studies have reported non-invasive plasticity induction protocols in animal experiments and humans (Bindman et al. 1964; Bi and Poo 1998; Stefan et al. 2000). Furthermore, the findings of this study suggested that the motor cortex is activated

during RPM and that it takes time for the descending input to modulate RI after RPM; thus, the after-effect may be delayed.

Given our findings, RPM evidently affects RI activity at high speeds. RPM is widely used as a rehabilitation method for patients with central nervous system diseases as well as for healthy elderly people and athletes. The present study revealed that RPM can bring about RI. These findings may be applied for the development of effective rehabilitation strategies.

One limitation of this study is its experimental design in which RPM tasks were performed for 10 min with variable number of movements. Based on our results, RI depends on RPM speed. However, if the firing frequency from the muscle spindle is affected, the effect of number of movements should also be considered. Future studies should thus consider unifying the number movements.

The present study revealed that RPM speed contributes RI. The extent of reciprocal Ia increased for up to 10 min following RPM at a speed of ≥ 120°/s. The extent of D1 inhibition increased for up to 10 min following RPM at a speed of 80°/s and continued to increase for up to 15 min at RPM speed of 160°/s. Therefore, RPM activates RI at high speeds.

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Author contributions Study conception and design: RH, ME, and HO; experiments: RH; data interpretation: RH, SM, SK, and HO; statistical analysis: RH, SK, and HO; writing and revising the manuscript: HO, ME, SK, and RH.

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Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

Ethical approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee (include name of committee + reference number) and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed consent Informed consent was obtained from all individual participants included in the study.

Consent for publication The subject signed consent for the use and publication of data obtained in experiments.

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