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Mechanisms of Postural Control in Older Adults Based on Surface Electromyography Data

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ABSTRACT

Objectives: The present study aimed to clarify the mechanisms of postural control during standing in older adults and document the mechanisms of age-related motor control based on changes in muscle activities.

Methods: A total of 26 healthy male adults (older adult group, ≥65–78 years: $n = 16$; younger adult group, 20–23 years: $n = 10$) participated in this study. Ground reaction force and kinematic data of the lower limbs (hip, knee, and ankle), and electromyographic data from 6 postural muscles on the right side were recorded and quantified for each motor phase during rapid voluntary center of pressure (COP) shift.

Results: Although hip strategy was more frequently observed in older adults than in young adults (56.3% vs. 20.0%), no muscle activity of hip agonists was observed in some (31.3%) older adults. Furthermore, older adults had a statistically significant delay in the inhibition of postural muscles during anticipatory postural adjustments ($p < 0.05$). After the onset of COP motion, the co-contraction time between agonists and antagonists was significantly prolonged in the older adults than in the younger adults ($p < 0.05$), and the reciprocal muscle pattern was unclear in the older adults. Prior to the termination of movement, agonist activity continued longer in the older adult group than in the younger adult group; that is, inhibition was insufficient in the older adult group.

Conclusion: A series of postural strategies during the voluntary movement task were altered

in older adults, and this was significantly related not only with the activation but also the inhibition of postural muscles.

Keywords: postural control, aging, center of pressure, co-contraction, voluntary movement

1. Introduction

In daily activities, humans need to instantaneously control equilibrium, which includes both static and dynamic elements, in real time and in response to changes to the body and environment. These abilities decline with advancing age, and the risk of falls is consequently higher among the elderly population (Okada, et al. 2001; Perry, et al. 2001). Thus, investigating age-related postural control is essential for understanding the mechanism of falls in older adults (Smith & Fisher, 2018).

Several strategies for postural control during standing have been proposed from kinematic (i.e., joint movements) and electromyographic (EMG; i.e., muscle synergy) data recorded in the lower limbs (Horak, 2006; Horak, et al., 1997; Winter, 1995). Based on the inverted pendulum model, the ankle strategy corresponds to small perturbations and predictable situations, while the hip strategy is recruited in unexpected or more perturbed situations that exceed the ability of the ankle strategy (Winter, 1995). Both the ankle and hip strategies participate primarily in postural control in the anteroposterior and lateral directions. Moreover, in the vertical direction, the suspensory strategy acts to stabilize standing posture by flexing the joints of the lower limbs, including the knee joint, and lowering the center of mass (COM) (Kasahara et al., 2015; Nashner & McCollum, 1985). Furthermore, when external perturbation increases, either of the two dynamic strategies can be recruited: the load-unloading strategy or the step strategy (Hof, 2007; Horak & Nashner, 1986).

Young, healthy adults can select the necessary strategy from these postural strategies depending on conditions, and they can perform it adequately; however, older adults often cannot adopt the optimal strategy. In general, older adults use the hip strategy more often than young adults (Nashner & McCollum, 1985). The hip strategy is thought to compensate for the decline of postural control that occurs in the ankle strategy (Sturnieks, et al., 2008; Alghwiri,

2012). The excessive movement at the hip or knee joint often observed among older adults, and in patients with motor disorders, is termed “buckling” and is considered to be the behavioral outcome of uncoordinated movements (Horak et al., 1997). Although frequent hip movement can be a good marker for age-related changes in postural control, there is still discussion about the meaning of the hip movement that is observed in older adults, and whether hip movements are produced actively (i.e., compensation for the deficit of the ankle strategy) or passively (i.e., dysfunction of the hip strategy).

Many previous studies (Amiridis et al., 2003; Horak, 2006; Horak et al., 1997; Kasahara et al., 2015) have demonstrated this difference in postural strategy between older and young adults. Each postural strategy is detected based on observed joint movements, and muscle activities reflect each joint movement. Coordinated movements can be determined from surface electromyography (sEMG) data of the trunk and lower limb muscles. The sequence of muscle activation in young adults is from distal to proximal (Winter, 1995; Woollacott, et al., 1986) under perturbation with platform movement. This order is reversed among older persons (Horak & Nashner, 1986; Woollacott et al., 1986). Another distinctive aspect of sEMG data in older adults is the co-contraction between agonist and antagonist muscles during posture control. This effect of co-contraction is debatable, depending on the case, and may be positive or negative (Craig, et al. 2016). The coordination of the initial movement in the series of postural control has a strong link with anticipatory postural adjustments (APAs) in voluntary movement. Recently, several studies (Baldissera, & Tesio, 2017; Barlaam, et al., 2016; Bolzoni et al., 2018) have focused not only on the excitation but also the inhibition of postural muscle activities in the APA phase. To the best of our knowledge, however, information on the relationship between inhibitory APAs and subsequent postural control is lacking.

The excitatory and inhibitory states of postural muscles have been partly assessed

using EMG; nevertheless, to comprehend motor control in its entirety, it is important to understand premovement, initiation, execution, and termination. Likewise, interrelations among kinematic, kinetic, and EMG data are unclear because most of such data has often been investigated separately. The present study aimed to clarify the mechanism of postural control (i.e., hip strategy) in older adults during standing. While in healthy older adults falls occur most frequently as a result of trips and slips, in residents of long-term care facilities, falls frequently occur during the shift from the static to dynamic state (Robinovitch et al., 2013). Primarily, we attempted to clarify the association between the preferred postural strategy and changes in the sequence of muscle activities. Additionally, because the difficulty in motor control among older adults appears not only during initiation but also during termination, we focused on motor control in the terminal phase through joint movements and muscle activities, as we had in a previous study (Kasahara & Saito, 2019). We hypothesized that there is a difference in the pattern of muscle activities, particularly in the modulation (i.e., inhibitions and facilitations) of the agonist and antagonist muscles at each joint, between young and older adults during the APA phase. Therefore, after confirming the hip strategy in older adults during the COP shift task, we analyzed the activity and inactivity of muscles to understand the mechanisms of co-contraction in older adults (i.e., the inability to release the co-contraction).

2. Methods

2.1. Participants

A total of 26 healthy adults (young adult group, 20–23 years: $n = 10$; older adult group, ≥65–78 years; $n = 16$) participated in this study. This prospective experimental study was conducted at the college laboratory. The demographic data of both groups are

summarized in Table 1. There were no statistically significant differences between the groups, except for age. The young adult participants were college students, and the older adult participants were randomly selected from community-dwelling older adults registered at an employment agency. All participants were physically active, lived independently in their community, and had no neurological, vestibular, orthopedic, or cognitive disorders or injuries that could interfere with balance. Because we used visually guided motor tasks, participants with visual acuity <1.0 in the Landolt ring chart were excluded, based on our previous study (Kasahara et al., 2015). The older adults had no falls in the 6 months prior to their participation in this study. All participants provided written informed consent for their participation, and the procedures were approved by the ethics committee of Hokkaido University School of Medicine (no. 11-03).

2.2. Procedures

All COP forward shift tasks during standing were performed on a force plate (Kistler type 9286A; Kistler Instrumente AG, Winterthur, Switzerland). Participants stood with their bare feet apart, with the foot and arm position as previously described (Kasahara & Saito, 2019; Kasahara et al., 2015). Participants were instructed to maintain their gaze at the computer monitor (~1 m at their eye level). The upward direction in the monitor corresponded to the forward direction on the force plate. The positions of the target and the COP were displayed simultaneously in the monitor, and they could also be observed by the examiners through a second monitor. The motion of the target was controlled by a program

158 customized using LabView 2009 (National Instruments, Austin, TX, USA). After the
159 examiner checked the steady state of the COP within 1 cm of the start position, the target
160 movement was started at random intervals between 10 and 30 s to avoid the prediction of
161 target start, and was shifted 5 cm (~20% of the foot length) upward from the center of the
162 monitor (Kasahara & Saito, 2019). This constant amplitude was selected to produce
163 equivalent amounts of postural sway in both groups, to eliminate effects of aged-related
164 changes in voluntary movement performance on postural and motor control (Craig et al.,
165 2016; Kasahara & Saito, 2019). In response to the target motion, the participants were
166 instructed to move their COP immediately and to match the target as fast and/or accurately as
167 possible, without heel-up, toe-up, and/or stepping. Moreover, the participants were asked to
168 remain still, in the same place, until the examiner instructed otherwise. Failed trials were
169 excluded from the following data analysis. To avoid postural strategy bias, no instructions on
170 the use of body parts were provided. Each participant performed 8–12 trials, with a few
171 minutes of rest between the trials to minimize fatigue.

172

173 *2.3 Measurements*

174 *2.3.1 Kinetic measurements*

175 This study used the velocity data of the COP to estimate the motor control ability of
176 the participants, as velocity is considered the most reliable parameter for postural and motor
177 control (Jeka, et al., 2004; Kasahara & Saito, 2019). Therefore, the mean COP velocity of

each participant was used in the following analysis to clarify some key points of postural and motor control, including premovement, initiation, execution, and termination. When the COP moves forward, it must shift backward first; this is called the reversal phenomenon (Cau et al., 2014; Kasahara & Saito, 2019; Klous, et al., 2012). From these findings, the onset of COP was defined as the first point where the COP velocity increased by 2 standard deviations (SDs) from the baseline in the backward direction, which was calculated 1 s before the target onset, and continued for 200 ms (Kasahara & Saito, 2019). Reaction time was calculated as the interval from the onset of the target to the onset of the COP. The offset of the shift of the COP was defined as the first point where the COP velocity decreased within the range of the mean ± 2 SD of the baseline and continued for 1 s. The total movement time was calculated as the interval from the onset to the offset of COP movement.

2.3.2 Kinematic measurements

A motion analysis system with six cameras was used at a sampling rate of 100 Hz to capture the joint motion of the hip, knee, and ankle (Motion Analysis Corporation, Santa Rosa, CA, USA). Reflective markers were attached based on anatomical landmarks according to both Winter (1990) and our previous studies (Kasahara & Saito, 2019; Kasahara et al., 2015). Three-dimensional marker data with COP data were digitally low-pass filtered, using a zero-lag, second-order Butterworth filter with a cutoff frequency of 10 Hz (Kasahara & Saito, 2019; Kasahara et al., 2015; Saito, et al., 2014). Similar to the definition of the COP movement, the onset and offset of the joints of the lower limbs, including the hip, knee, and ankle joints, were detected and calculated in the sagittal plane. This was done because the movement direction in the voluntary COP shift task was in the anteroposterior direction (Fig. 1A and B). If the onset was not detected or the amplitude of the joint angle was <1.0°, the joint movement was considered absent, as described in previous reports (Boisgontier &

Nougier, 2013; Dickstein, et al., 1996). The angular displacement and velocity of each joint were calculated using a customized MATLAB program (MathWorks, Natick, MA, USA). These data from the left and right joints were summarized (Kasahara et al., 2015; Kasahara et al., 2015; Tokuno, et al., 2010), and the angular displacement and velocity of each trial were averaged as the representative data for each participant.

2.3.3 Electromyographic measurements.

sEMG data were collected from postural muscles at a sampling rate of 1 kHz using the Bagnoli-2 EMG System (Delsys, Boston, MA, USA). Muscle activity was recorded for the following 6 postural muscles on the right side in accordance with our previous study (Kasahara et al., 2015; Nashner & McCollum, 1985): rectus abdominis (RA), erector spinae (ES), rectus femoris (RF), biceps femoris (BF), tibialis anterior (TA), and gastrocnemius (GA). Reference electrodes were attached to the iliac crest, head of the fibula, and lateral malleolus. All EMG data were amplified 1000 times, rectified, and bandpass filtered from 10 to 500 Hz using a fourth-order Butterworth filter (Kasahara et al., 2015). Based on the agonist–antagonist muscle pairs (TA–GA, RF–BF, and RA–ES) used in previous studies (Kasahara et al., 2015; Li & Aruin, 2009), the current study examined the muscle activities by adding inhibition of antagonists for each phase. The muscle onset, which is the beginning of the activation/inhibition of muscle, occurred in the self-initiated movement (Crenna & Frigo, 1991; Kanekar & Aruin, 2014), and in the initiation of rapid movement; both muscle activation of agonists and muscle inhibition of antagonists occurred (Gottlieb, Agarwal, & Stark, 1970; Hallett, et al., 1975; Hufschmidt & Hufschmidt, 1954). Thus, this study attempted to investigate both the activation and inhibition of muscles. The onset of antagonist inhibition was defined as the first point where the averaged EMG decreased by 2 SD from the mean, which was calculated from the 1-s period of the baseline period, and continued for 30

ms, as reported previously (Kanekar & Aruin, 2014; Tokuno et al., 2010). The reaction time of the antagonist inhibition was defined as the time period between the target onset and the initiation onset of its muscle. The activation onset of both agonists and antagonists were defined as the point where the level of the average EMG increased more than 100 ms, and more than double the SD from the average value within the baseline (Kanekar & Aruin, 2014; Klous et al., 2012). The reaction time of the agonist activation was defined as the period between the target onset and the activation onset of its muscle. As reported in previous studies (Kanekar & Aruin, 2014, 2015), the criteria of inhibition offset, and activation onset of antagonists, were equivalent. Following activation onset, the activation offset in each muscle was defined as the first point wherein the SD of EMG for a 25-ms time window (Hodges & Bui, 1996; Mickelborough, et al., 2004) decreased <1 SD from the baseline and continued for 250 ms. The inhibition duration of antagonists was defined as the period between its inhibition onset and activation onset (Fig. 2A and B). The activation duration of each muscle was defined as the period between the activation onset and offset. As the co-contraction index (CCI), the co-contraction duration of agonist–antagonist was defined as the period where the activation of agonist and antagonist overlapped after COP onset. The detections and calculations at all point were performed by a customized MATLAB program (Kasahara & Saito, 2019; Kasahara et al., 2015) and were reconfirmed by visual inspection (Kanekar & Aruin, 2015; Klous et al., 2012; Tokuno et al., 2010). If the change in EMG activity did not adhere to any of these criteria, the activation or inhibition was considered absent.

2.4 Statistical analysis

The adequacy of the sample size and significance level was confirmed by G*Power,

253 with the effect size set at 0.4, the alpha at 0.05, and the power at 0.8 (Faul et al., 2007),
254 according to Cohen's criteria (Cohen, 1988). Statistical analyses were performed using SPSS
255 Statistics version 18.0 (IBM Corp., Armonk, NY, USA). All data are presented as mean ± SD.
256 Independent sample *t*-tests were conducted first to assess group differences in demographic
257 data. To determine which joint of the lower limbs was activated in the motor task, the
258 occurrence rate of joint movements at each joint was determined according to the number of
259 subjects who used it, divided by the total number of subjects in each group, and multiplied by
260 100. This occurrence rate was also used to assess muscle synergy for the inhibition of
261 antagonists and activation of all muscles. Chi-square analyses were used to determine age
262 group differences in all occurrence rates in the current task. Continuous variables were
263 compared using unpaired or paired t-tests for normally distributed data, and the Mann–
264 Whitney U test was used for non-normally distributed data. Analysis of variance (ANOVA)
265 was used for age or muscle groups. Further, if the occurrence of each event was low, the
266 Friedman Chi-square test, which adapts to small numbers, was conducted to compare the
267 relative time difference among joints or muscles within groups, and the Wilcoxon signed rank
268 test was used for post hoc comparisons between each mean value. Lastly, Spearman's rank
269 method (*R*) was used to investigate the relationship between the reaction time of the COP and
270 inhibition onset of the antagonists, and between the total movement times of the COP and
271 CCI. For effect sizes of the Chi-square tests, we used Cramér's phi (φ) (Cohen, 1992). Effect
272 sizes of t-tests was calculated using Cohen *d*, and those of Mann–Whitney *U* tests and
273 Wilcoxon signed rank tests were calculated using *r* values with the *Z* value ($r = Z$ value/
274 square root of (sample size)). The effect sizes used in ANOVA are expressed as partial eta
275 square (η^2_p) values and the effect sizes for differences in means were based on Cohen's report
276 (Cohen, 1988). All statistical significance levels were set at $p < 0.05$.
277

3. Results

3.1 Occurrence rate of joint movement

Although the occurrence rate of joint movement at the hip in the older adult group (56.3%) was higher than that in the young adult group (20.0%), the Chi-square analyses showed no statistically significant difference between the groups ($\chi^2 = 3.31$, $df = 1$, $p = 0.069$, $\varphi = 0.36$). No statistically significant differences in the occurrence rate of joint movement at the knee (older adult group, 68.8%; young adult group, 60.0%, $\chi^2 = 0.21$, $df = 1$, $p = 0.648$, $\varphi = 0.05$) and ankle (older adult group, 93.8%; young adult group, 100%, $\chi^2 = 0.65$, $df = 1$, $p = 0.420$, $\varphi = 0.09$) were noted between the groups. Because the occurrence rate of hip movements in the young adult group was very low (i.e., <50%), the subsequent statistical analyses conducted for age difference did not include the hip joint.

3.2 Time and sequence of joint movements in lower limbs

No statistically significant difference in the reaction time of the ankle joint was observed between the groups ($t_{(23)} = 1.22$, $p = 0.235$, $r = 0.50$), but the increase in the reaction time of the knee joint was significantly longer in the older adult group than in the young adult group ($t_{(14)} = 2.72$, $p = 0.017$, $r = 1.42$) (Table 2). In the young adult group, there were no statistically significant differences in the reaction times between the knee and ankle joints; therefore, both joints acted at the same time (Fig. 3A). Conversely, in the older adult group, there were statistically significant differences in the reaction times among all joints (Wilcoxon test: hip vs. knee, $z = -2.37$, $p = 0.018$, $r = -0.54$; knee vs. ankle, $z = -2.13$, $p = 0.033$, $r = -0.43$; hip vs. ankle, $z = -2.38$, $p = 0.017$, $r = -0.49$) (Fig. 3B). These results demonstrate that, in the older adult group, joint movement was performed from the bottom to

the top in the following sequence: ankle, knee, and hip joint. Movement times of the knee and ankle joints were longer in the older adult group than in the young adult group (knee: $t_{(14)} = 2.72$, $p = 0.015$, $r = 1.43$; ankle: $t_{(21)} = 2.69$, $p = 0.027$, $r = 0.96$) (Table 2). In the young adult group, the movement time of the knee joint was significantly longer than that of the ankle joint (Wilcoxon test: $z = -2.20$, $p = 0.028$, $r = -0.55$) (Fig. 3C). In contrast, in the older adult group, no statistically significant differences in the movement times were found between the knee and ankle joints (Wilcoxon test: $z = -1.72$, $p = 0.086$, $r = -0.34$); however, there was a statistically significant difference in the movement time between the hip and ankle joints (Wilcoxon test: $z = -2.10$, $p = 0.038$, $r = -0.43$) (Table 2 and Fig. 3D). There were no statistically significant differences between groups in the amplitude of all joint movements.

3.3 Occurrence rate of activations and inhibitions in postural muscles

According to our definitions of EMG events, the occurrence rate of BF inhibition was significantly lower in the older adult group (62.5 %) than in the young adult group (both 100%) ($\chi^2 = 3.87$, $df = 1$, $p = 0.049$, $\varphi = 0.39$). Further, the occurrence rate of GA inhibition was similarly different between groups (older group: 62.5%, young group: 100%, $\chi^2 = 3.87$, $df = 1$, $p = 0.049$, $\varphi = 0.39$). There were no statistically significant between-group differences in the occurrence rates of inhibition and activation in other muscles. Because the occurrence rate of RA activation in the older adult group (31.3%) was very low (i.e., <50%), the subsequent statistical analysis conducted for age difference did not include the RA.

3.4 Time and sequence of postural muscles

Table 3 shows the reaction time and the duration of muscle activity. Based on the

repeated-measures two-way ANOVA, there was a statistically significant interaction for reaction time between age (young and older adult groups) and muscle type (agonists and antagonists) ($F_{(1,35)} = 6.31$, $p = 0.017$, $\eta^2_p = 0.15$) and a statistically significant main effect of age ($F_{(1,35)} = 4.30$, $p = 0.045$, $\eta^2_p = 0.11$). Post hoc testing revealed that the onset of antagonist inhibition was significantly more delayed in the older adult group than in the young adult group ($p = 0.004$, $\eta^2_p = 0.22$). Although there was no statistically significant difference in reaction time between antagonist inhibition and agonist activation in the young adult group, the onset of antagonist inhibition was significantly later than that of agonist activation in the older adult group ($p = 0.049$, $\eta^2_p = 0.11$).

Hence, we statistically analyzed the inhibition of antagonists first, and subsequently analyzed the activation of agonists and antagonists. The repeated-measures two-way ANOVA showed no interaction for the inhibitory reaction time between age and antagonists and a significant main effect of age ($F_{(1,14)} = 12.83$, $p = 0.003$, $\eta^2_p = 0.48$). Post hoc testing revealed that the inhibitory reaction time of the ES and BF in the older adult group was significantly longer than that in the young adult group (ES: $p = 0.027$, $\eta^2_p = 0.30$, BF: $p = 0.021$, $\eta^2_p = 0.33$) (Fig. 4A). For the inhibition duration of antagonists, the two-way ANOVA revealed no interaction between age groups and antagonists and a statistically significant main effect of age ($F_{(1,56)} = 16.34$, $p = 0.001$, $\eta^2_p = 0.27$). In addition, the inhibition duration of the antagonist was significantly shorter in the older adult group than that in the young adult group. Post hoc testing revealed that the inhibition durations of the ES and BF were significantly shorter in the older adult group than those in the young adult group (ES: $p = 0.010$, $\eta^2_p = 0.11$, BF: $p < 0.005$ $\eta^2_p = 0.13$) (Fig. 4D). There were no statistically significant differences in the inhibition duration among antagonists in either age group.

The repeated-measures two-way ANOVA showed no interaction for the excitatory reaction times between age groups and agonists (RF and TA, but not the RA) or statistically

significant main effects. Post hoc testing revealed that the excitatory reaction time of the RF in the older adult group was significantly longer than that in the young adult group (p = 0.026, η^2_p =0.21) (Fig. 4B). For the activation duration of the agonist, there was no interaction between age and muscles. There was a statistically significant main effect of age ($F_{(1,45)}$ = 8.496, p = 0.006, η^2_p =0.16), and the activation durations of agonists were significantly longer in the older adult group than those in the young adult group. Post hoc tests also showed that the activation durations of the RF and TA in the older adult group were significantly longer than those in the young adult group (RF: p = 0.045, η^2_p =0.09, TA: p = 0.044, η^2_p =0.09) (Fig. 4E).

For the activation reaction time of antagonists following the activation of agonists, the repeated-measures two-way ANOVAs showed no interaction between age and muscles or main effects. Post hoc testing showed that the reaction time of the GA was significantly later than that of the BF in the older adult group (p = 0.010, η^2_p =0.40) (Fig. 4C). The two-way ANOVA for the activation duration of antagonists showed no interaction between age and antagonists and a statistically significant main effect of age ($F_{(1,63)}$ = 18.584, p = 0.001, η^2_p =0.23). Post hoc testing revealed that all antagonist durations were significantly longer in the older adult group than those in the young adult group (ES: p = 0.019, η^2_p =0.08; BF: p = 0.037, η^2_p =0.07; GA: p = 0.005, η^2_p =0.12) (Fig. 4F).

3.5 Relationship between co-contraction and COP performance

The co-contraction duration between the RF and the BF in the older adult group (724.6 ± 622.6 ms) was significantly longer than that in the young adult group (216.4 ± 155.2 ms) ($t_{(16)}$ = −2.90, p = 0.011, d =1.00), and the co-contraction duration between the TA and the GA in the older adult group (837.2 ± 766.7 ms) was also significantly longer than that in the young adult group (231.2 ± 174.9 ms) ($t_{(14)}$ = −2.75, p = 0.016, d =1.00) (Fig. 5).

Fig. 6 shows the correlation between reaction times of the COP and inhibition onsets

of antagonists, and between movement times of the COP and CCIs for all participants. The relationship between movement times of the COP and CCIs of the RA–ES was not analyzed because the occurrence rate of the RA activation in the older adult group was very low. There were statistically significant positive correlations between reaction times of the COP and inhibition onsets of the BF ($R = 0.63$, $p = 0.003$) and GA ($R = 0.48$, $p = 0.032$), but not of the ES (Fig. 6A–C). There was a statistically significant positive correlation between movement times of the COP and CCIs of the RF–BF ($R = 0.46$, $p = 0.030$), and there was no correlation between movement times of the COP and CCIs of the TA–GA ($R = 0.41$, $p = 0.067$) (Fig. 6D and E).

4. Discussion

4.1 Postural strategy in older adults during dynamic balance

Regarding joint movement in the lower limbs, our results showed that during dynamic balance, the young adult group performed the ankle strategy, whereas the older adult group performed the hip strategy in addition to the ankle strategy, similar to findings of previous studies (Amiridis et al., 2003; Horak, 2006; Kasahara et al., 2015). In this study, although the occurrence of hip movement in the older adult group (56.3%) was more than two times that of the young adult group (20%), it was not as high as expected. To actively shift from a static posture to a new posture, it is necessary to interrupt the static posture, and this disruption is provided by internal perturbation. Most previous studies on postural control have investigated the involuntary or responsive postural strategy with external perturbations. These include unexpected anteroposterior motion of the support surface (Nashner & McCollum, 1985) and a narrow base of support (Amiridis et al., 2003). Thus, the difference we observed in the incidence of hip motion may depend on task properties (i.e., whether the task is passive or

active).

In this study, when performing the voluntary motor task, the participants were required to provide an internal perturbation to produce the motion from the static condition. When postural perturbation is applied, excessive knee and hip movements (i.e., excessive flexion) are caused by ankle torque produced during the ankle strategy (Horak et al., 1997). Therefore, it is necessary to control the hips and knees to prevent falling and accomplish the task immediately after an internal perturbation. Although only few studies have investigated the contribution of knee movement to postural control (Frey-Law & Avin, 2013; Smith & Fisher, 2018), knee movement is thought to be a possible trigger for internal perturbation (Cheron, et al., 1997). Our previous study suggested that knee flexion was involved in the suspensory strategy (i.e., “mixed strategy”), and its role was to maintain equilibrium by lowering the COM (Kasahara et al., 2015). In fact, in the present study, knee movement was observed in both groups, and it played a role in simultaneously disrupting and stabilizing the static standing posture. Our results indicate that the knee movement in the young adult group was approximately 20 ms faster and 1 s longer than the ankle movement (Table 2). These findings suggest that the static state of standing posture was first disrupted by knee flexion and the dynamic stabilization of standing posture subsequently occurred through the lowering of the COM by successive knee flexion movements.

In the young adult group, ankle movement occurred concurrently and/or subsequently, and the COP and COM were stably and smoothly shifted forward. The knee movement in the older adult group was approximately 40 ms later than the ankle movement, and the movement time of the COP in the older adult group was significantly longer than that in the young adult group. The knee movement in the older adult group was speculated to provide balance stabilization (Horak et al., 1997) to avoid falling due to the first perturbation induced by the ankle movement, rather than the internal perturbation (Kasahara & Saito, 2019).

Another interesting finding was that the hip movement in the older adult group was delayed the most among the lower limb joints. This results in questions about whether the older adult group actually used the hip movement aggressively for postural control, and suggests that the hip buckling that occurred in the older adult group resulted from behavioral outcomes of uncoordinated movement (Horak et al., 1997).

4.2 Effects of age on the inhibition and activation of postural muscles during the APA phase

Some changes in measurements associated with postural control (e.g., COP, EMG), preceding voluntary movement, are anticipatory in nature (Friedli, Hallett, & Simon, 1984; Kanekar & Aruin, 2014). Prior to voluntary initiation, forward predictive models of the internal model are used to predict adverse consequences of an upcoming action before it takes place (Barlaam et al., 2016; Frey-Law & Avin, 2013), and then the APAs are set and performed to stabilize the subsequent changes in posture. Both excitation and inhibition of postural muscles occur during the APA phase in predictable (Frey-Law & Avin, 2013) and external perturbations (Kanekar & Aruin, 2015). However, some postural muscles remain in a certain level of active state to stabilize posture. These include the antigravity muscles (Kasahara et al., 2015) making it necessary to suppress the activation of antagonists (i.e., postural muscles in this case) before agonist activation to initiate movement (Gottlieb et al., 1970; Hallett et al., 1975; Hufschmidt & Hufschmidt, 1954; Kanekar & Aruin, 2015).

Gottlieb et al. (1970) and Morimer, et al., (1987) suggested that the earliest manifestation of rapid movement is not activation, but rather a depression or silencing of EMG activity of the antagonist muscles, and our present study focused not only on muscle activation, but also muscle inhibition in the APA phase (Baldissera, & Tesio, 2017; Barlaam et al., 2016; Bolzoni et al., 2018; Kanekar & Aruin, 2015). We found earlier inhibition of the

BF in the young adult group during the APA phase (−150 to −50 ms before movement onset), similar to findings from previous studies (Cheron et al., 1997; Kanekar & Aruin, 2015). In the measurement of changes in muscle activity during voluntary tasks, the inhibition of muscle activity was sufficiently detectable, and the accurate timing of the onset of muscle inhibition was crucial for the fine adjustments of APAs (Barlaam et al., 2016). Therefore, the mistimed inhibition onset led to inefficient APAs (Barlaam et al., 2016) and, consequently, resulted in the delayed onset of movement in the older adult group.

Conventional evidence of the hip strategy in older adults is demonstrated based on changes in EMG patterns of postural muscles. When using the hip strategy under perturbation with platform movement, the sequence of muscle activation is from distal to proximal (Woollacott et al., 1986, 1988), and this normal pattern of muscle activation in automatic postural responses also appears constantly in voluntary sway (Winter, 1995). Regarding muscle activities of agonists (i.e., RA, RF, and TA), our results showed that, in the young adult group, the sequence of muscle activation had the same timing for the RF and TA, but the RA activity occurred later. However, this sequence of muscle activation has been found to be reversed in older adults (Horak & Nashner, 1986; Woollacott et al., 1986). This change in sEMG was confirmed in our study on only a few subjects. Furthermore, in our older adult group, the activation of the RA was low (~33%); therefore, we could not find firm evidence that the older adult group actively used the hip strategy.

Another muscle pattern that includes both activation and inhibition has been observed in a sequence of voluntary movements from the stable posture to the dynamic state (Crenna & Frigo, 1991; Gottlieb et al., 1970; Hallett et al., 1975; Hufschmidt & Hufschmidt, 1954; Kanekar & Aruin, 2014). The triphasic muscle pattern in the rapid voluntary arm movement consists of the first, strong tonic contraction of antagonists, simultaneous inhibition of tonic antagonist contractions, excitation of the agonist, and re-contraction of antagonists (Crenna &

Frigo, 1991; Gottlieb et al., 1970; Hallett et al., 1975; Hufschmidt & Hufschmidt, 1954; Kanekar & Aruin, 2014). This reciprocity between the agonist and antagonist muscles is accurate as regulated by the neural system, and periods of co-contraction rarely occur (Oddsson & Thorstensson, 1987). In our experiment, the dorsal muscles (including the ES, BF, and GA), as antigravity muscles, always maintained a certain level of muscle tone to support an erect standing posture (Friedli et al., 1984; Kasahara et al., 2015). They also acted as antagonists for forward COP shift task during the baseline phase (i.e., the first phase of the triphasic pattern). Subsequently, the inhibition of the dorsal muscle group occurred faster than the activation of the ventral muscle group in both age groups (i.e., the second phase of the triphasic pattern). Moreover, the ventral muscles (including the RA, RF, and TA) acted as agonists for this motor task. Finally, the dorsal muscle group re-acted as the brake for the forward COP shift (i.e., the third phase of the triphasic pattern). Therefore, we speculated that the first behavior of motion onset is the disruption of stable posture and the release of postural muscle contraction—that is, the "unlocking" of the previous erect posture (subserved by the inhibition of the tonic hip extensor activity) (Cheron et al., 1997) could be considered as another key control in the APA phase (Barlaam et al., 2016). In the current study, the triphasic muscle pattern was relatively clear in the young adult group but was unclear in the older adult group (specifically, the lack of inhibition of antagonists; see section 3). These findings suggest that the onset delay or extended reaction time in the older adult group was caused not only by the delay in agonist activation but also by the delay in, or lack of, antagonist inhibition.

Herein, one contradiction occurred between RA activities and hip joint movement in each group, after the APA phase. Because the iliopsoas muscle was difficult to palpate and detect (Cheron et al., 1997), the RA in this experiment was presumed to be one of the hip flexors, as seen in earlier studies (Horak & Nashner, 1986; Kasahara et al., 2015; Kanekar &

Aruin, 2014; Li & Aruin, 2009; Nashner & McCollum, 1985). In previous studies on postural control using external perturbation (Horak & Nashner, 1986; Nashner & McCollum, 1985), one evidence for the hip strategy was the onset of hip muscle activities that preceded the onset of ankle muscle activities. However, in the current study, hip movement did not occur in the young adult group, despite RA activity, which was the opposite of that observed in the older adult group, in which hip movement occurred without RA activity. A possible explanation for this observation can be found in the other role of the RA. Along with the transverse abdominal muscle and diaphragm, the RA increases the stiffness of the upper trunk, as well as the extension moment, by increasing intra-abdominal pressure (Cholewicki, et al., 2002; Hodges, et al., 2001), thus, suppressing the disturbance in the trunk and hip joint. Based on this fact, the deactivation of the RA in the older adult group was considered to lower the stabilization of the heavy trunk, which resulted in excessive flexion (i.e., buckling) at the hip joint. Furthermore, increased muscle activities of the dorsal muscles—specifically the ES—may be required for buckling. In fact, the latency of ES inhibition was significantly more extended in the older adult group than in the young adult group (see section 3). Although these findings suggest the avoidance or prevention of buckling through tonic and/or eccentric contraction of the ES through the postural muscles, older adults cannot support the heavy trunk due to general muscle weakness (Miyatani, et al., 2003), which consequently leads to the hip motion. Our sEMG data provide evidence that the hip buckling observed in the older adult group was due to the general dysfunction of the hip and trunk, and not because of the effective use of the hip joint for postural stabilization following the dysfunction of the ankle strategy (Horak et al., 1997). Needle EMG of the iliopsoas muscle should be performed to confirm this.

4.3 Effects of age on the inhibition and activation of postural muscles in the terminal phase

Similar to our previous results (Kasahara & Saito, 2019; Nagai, et al., 2011), the results of this study also showed that phasic contractions between agonists and antagonists were unclear in the older adult group and revealed that co-contraction was significantly related to movement time—the duration for stopping the movements. Generally, to rapidly stop ongoing movements, fast suppression of agonists and/or activation of antagonists is needed (Kasahara & Saito, 2019). For the former, the reaction time of the RF in the older adult group was approximately 150 ms later than that seen in the young adult group, and even when this delay was deducted from the total movement time of COP, the duration of the RF in the older adult group was extended for approximately 330 ms than that in the young adult group. Similarly, the duration of the TA in the older adult group was extended for approximately 460 ms than that in the young adult group. However, in their study of external perturbation on a movable platform, Manchester, Woollacott, Zederbauer-Hylton, and Marin (1989), reported that although older adults had increased muscle co-contraction of antagonists, the temporal characteristics of lower extremity muscles did not significantly differ between the age groups. Our current study also found no significant differences in the reactivation of antagonists that produced the braking force between the groups (data not shown). Therefore, based on the results of the muscle sequence, we think it is possible that older adults have difficulty in suppressing ongoing agonist activity to stop the motion.

Researchers have different views regarding the behavior of muscle co-contraction, which may depend on the feature of the task (Nagai et al., 2011). For static balance, co-contraction increases joint stiffness and enhances postural stability (Craig et al., 2016). For dynamic balance (e.g., gait, functional reach), however, co-contraction decreases the coordination between joints and subsequently the motor performance (Nagai et al., 2011). Although age-related increases in co-contraction undoubtedly occur, the contribution of the

co-contraction of agonists and antagonists to motor performance differs among cases.

4.4 Change in muscle inhibition in older adults

Our findings showed an obvious deficit in the inhibition of muscle activation among the older adults. This was observed through the delay of inhibition of postural muscles during the APAs and extended co-contraction, which caused delayed inhibition of agonists during termination in older adults. The initial inhibition (i.e., inhibitory APAs) prior to the motion is the EMG signature of postural predictive control (Barlaam et al., 2016) and is centrally programmed (Hallett et al., 1975). In older adults, shifted representations of the hip musculature in the motor cortex cause altered temporal organization of APA synergies; furthermore, age-related greater overlap between individual muscle representational areas induces greater co-contraction between those muscles during APAs (Frey-Law & Avin, 2013).

After movement onset, sensorimotor systems monitor motions through various afferents (i.e., vision, vestibular sense, somatosensory) for balance and control velocity by tuning effectors (i.e., muscles). In this study, the second inhibition (i.e., the inhibition of the agonist in the terminal phase) was considered to be dependent on online use of feedback information (Barlaam et al., 2016), with the inhibition of the active muscle arising from an online corrective mechanism based on a proprioceptive feedback loop. In older adults, however, these sensory inputs for balance are inaccurate and inadequate (Craig et al., 2016). Although co-contraction may compensate for proprioceptive deficits by increasing proprioceptive information from muscle spindles, another study found that it does not always compensate for age-related proprioceptive deficits (Craig et al., 2016). Co-contraction from weakness of the secondary inhibition of agonists was speculated to work better for postural

stability by increasing joint stiffness through a normal proprioceptive feedback loop, as the older adults in the current study had no sensory system deficit.

The change in modulation of muscle activities, especially inhibition, was deeply involved in postural and motor control in the older adults. The overall temporal delay in the older adult group consisted of reaction time delay due to inadequate inhibition of postural muscles and extended duration of stopping the movement due to insufficient inhibition of ongoing muscle activities. Some hip movements in the older adult group were actually performed without hip muscle activity and, thus, hip buckling could not be completely ruled out. Owing to the preceding postural instability of the upper body caused by hip buckling in older adults, it was speculated that co-contraction could be used to increase stiffness around the ankle joint to minimize the degree of freedom of joint motion, and consequently, the hip strategy, as the remaining postural strategy, was recruited to avoid falls after postural deterioration.

4.5 Limitations

The present study has some limitations. First, the sample size was small, and, as such, our results should be interpreted with caution, as they are based on a small number of data points. Second, despite random selection, only men were included in the study. Hence, female participants should be targeted for enrollment in future studies. Third, postural and motor control was investigated in a limited motor task (i.e., a voluntary task). Therefore, the occurrence rate of each joint movement depended upon the difficulty of the task. As fall risk is also high in older adults during unpredictable external disturbance, more evidence documented in various tasks is required to support our conclusions.

5. Conclusion

This study showed that inhibition of muscle activities influenced postural and motor control in older adults and that hip movement in older adults was not always recruited or executed aggressively. Hip movements observed frequently in the older adults, while maintaining standing balance, included buckling from the changes of the sequence of muscle activity at the hip joint. In addition, this study revealed that the extended co-contraction appeared not only in the ankle but also in the knee. These extended co-contractions were related to the delay of termination (i.e., an extension of movement time) in older adults.

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Declarations of interest

The authors have no conflict of interest to disclose

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780 **Figure legends**

781 **Fig. 1.** (A and B) Representative traces of each joint movement based on one young adult
782 subject (A) and one older adult subject (B) showing onsets (open triangles) and offsets (close
783 triangles). Neither onset nor offset of the hip movement was detected in the young adult
784 subject. The vertical line at 0 is the onset of the visual target.

785

786 **Fig. 2.** (A and B) Representative electromyography (EMG) traces of each muscle based on
787 one young adult subject (A) and one older adult subject (B) showing inhibition (open inverted
788 triangles) and activation onsets (close inverted triangles). The activation onset of the rectus
789 abdominis and the inhibition onset of the gastrocnemius in this older adult subject were not
790 detected. RA: rectus abdominis; ES: erector spinae; RF: rectus femoris; BF: biceps femoris;
791 TA: tibialis anterior; GA: gastrocnemius.

792

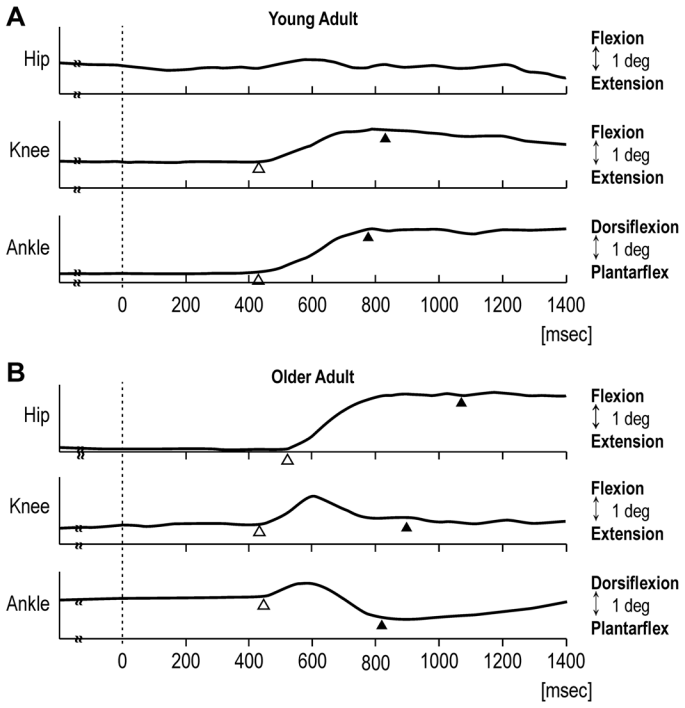
793 **Fig. 3.** Interquartile range boxes and whiskers of the reaction time of each joint in the young
794 adult (A) and older adult groups (B) and the movement time of each joint in the young adult
795 (C) and older adult groups (D). The box plot shows the median values and interquartile range
796 of the entire sample in each joint. The upper and lower whiskers show the maximum and
797 minimum values, respectively. Dots are plotted as each subject's data on the left side of the
798 box. The reaction time and movement time of the hip joint in the young adult group are not
799 shown because of the low occurrence rate of hip movement. *Statistically significant
800 differences between joints ($p < 0.05$).

Fig. 4. Interquartile range (represented by boxes and whiskers) for the reaction time (A–C) and duration (D–F) of each muscle. The white and gray boxes represent the young adult and older adult groups, respectively. Dots are plotted as each subject's data on the left side of the box. Data for the rectus abdominis are not shown for either group because of the low occurrence rate of rectus abdominis activation in the older adult group. *Statistically significant differences between groups ($p < 0.05$). ES: erector spinae; BF: biceps femoris; GA: gastrocnemius; RF: rectus femoris; TA: tibialis anterior.

Fig. 5. Co-contraction index in each pair of agonist and antagonist. The white and gray bars represent the young adult and older adult groups, respectively. Each individual subject's data is plotted on the left side of the box. *Statistically significant differences between groups ($p < 0.05$). CCI: co-contraction index; RF: rectus femoris; BF: biceps femoris; TA: tibialis anterior; GA: gastrocnemius.

Fig. 6. Scatterplot showing the correlation between the reaction time and inhibition onset of antagonists (A–C) and between the movement time and co-contraction index (D, E) for each subject. RT: reaction time; MT: movement time; CCI: co-contraction index; ES: erector spinae; BF: biceps femoris; GA: gastrocnemius; RF: rectus femoris; TA: tibialis anterior.

Fig. 1.


A
Young Adult
Hip
Knee
Ankle
Flexion
1 deg
Extension
Flexion
1 deg
Extension
Dorsiflexion
1 deg
Plantarflex
0 200 400 600 800 1000 1200 1400
[msec]
B
Older Adult
Hip
Knee
Ankle
Flexion
1 deg
Extension
Flexion
1 deg
Extension
Dorsiflexion
1 deg
Plantarflex
0 200 400 600 800 1000 1200 1400
[msec]

833 **Fig. 2.**

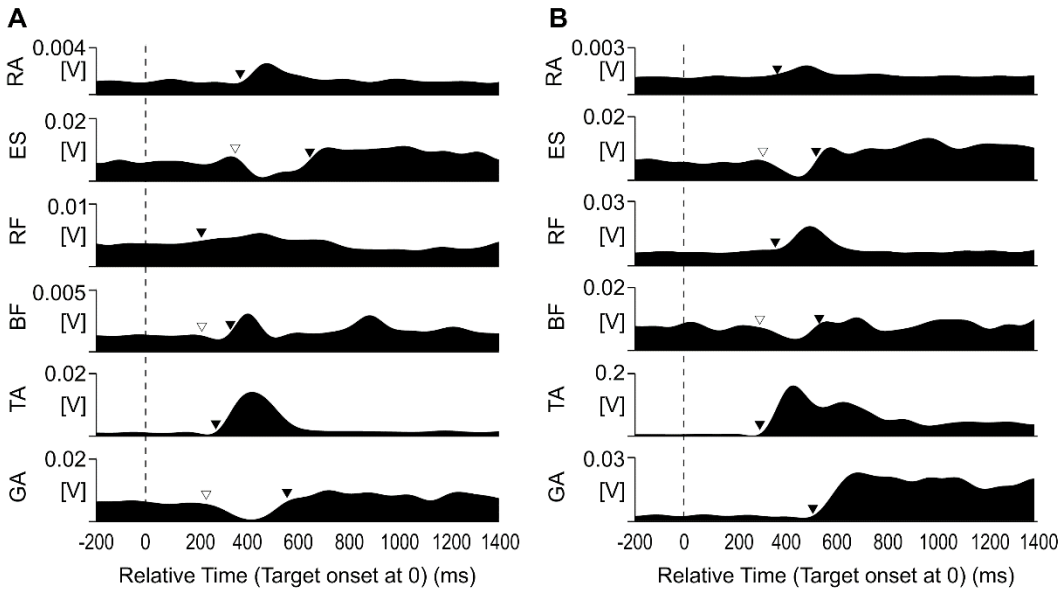


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Fig. 3.

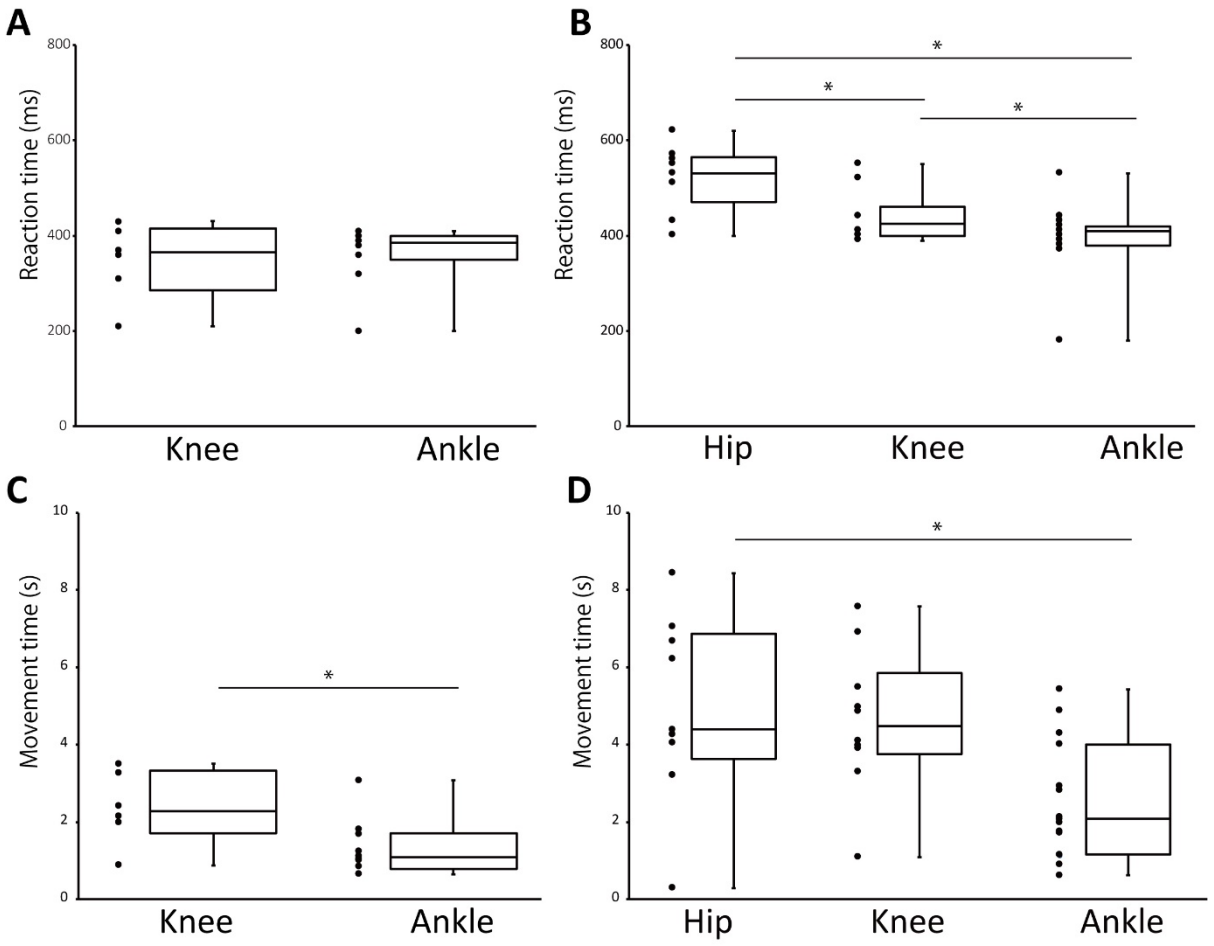
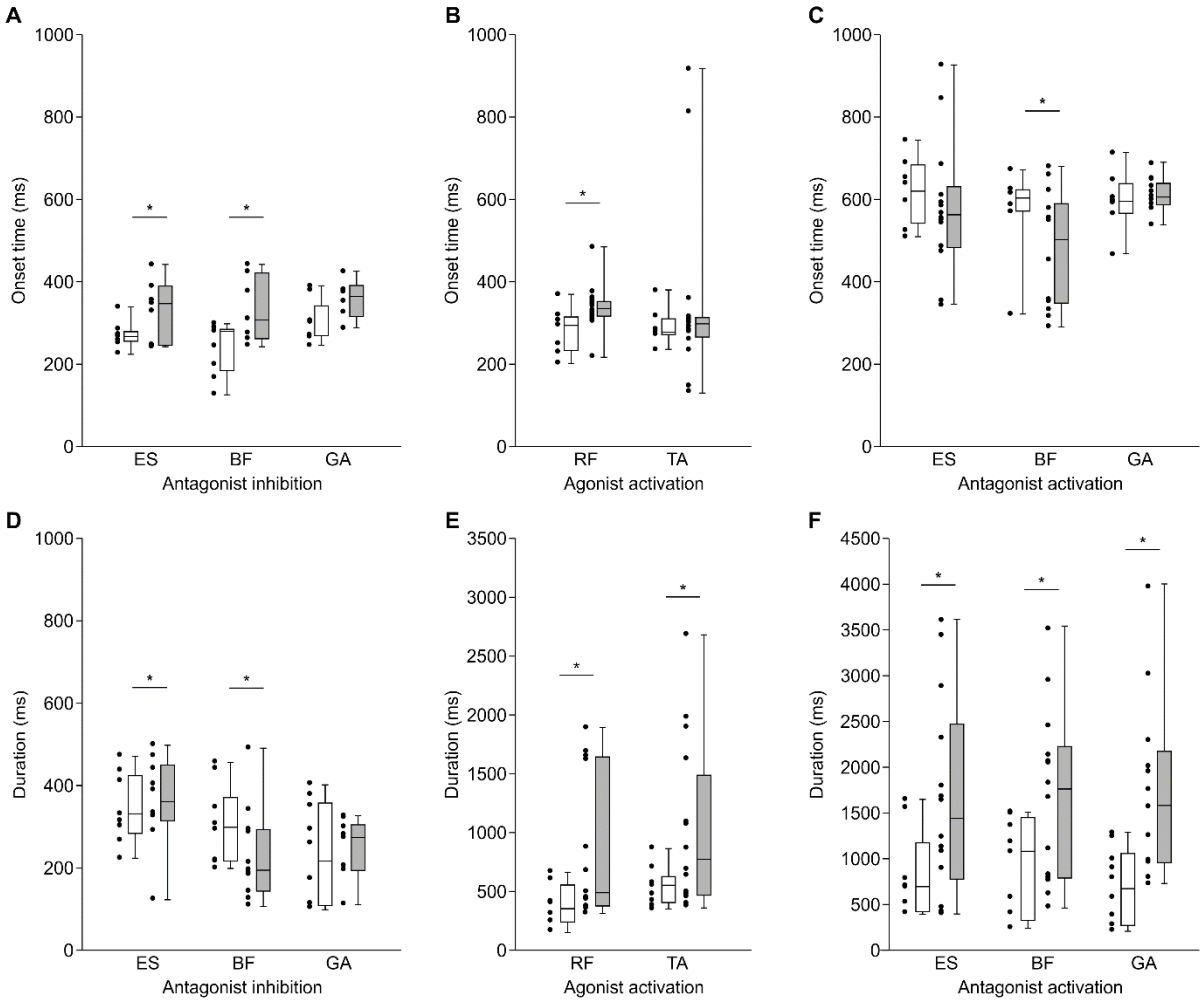

A
Reaction time (ms)
800
600
400
200
0
Knee
Ankle
B
Reaction time (ms)
*
*
*
Hip
Knee
Ankle
C
Movement time (s)
10
8
6
4
2
0
*
Knee
Ankle
D
Movement time (s)
*
Hip
Knee
Ankle

Fig. 4.


A
B
C
D
E
F
Onset time (ms)
Duration (ms)
ES
BF
GA
RF
TA
Antagonist inhibition
Agonist activation
Antagonist activation
*

867 **Fig. 5.**

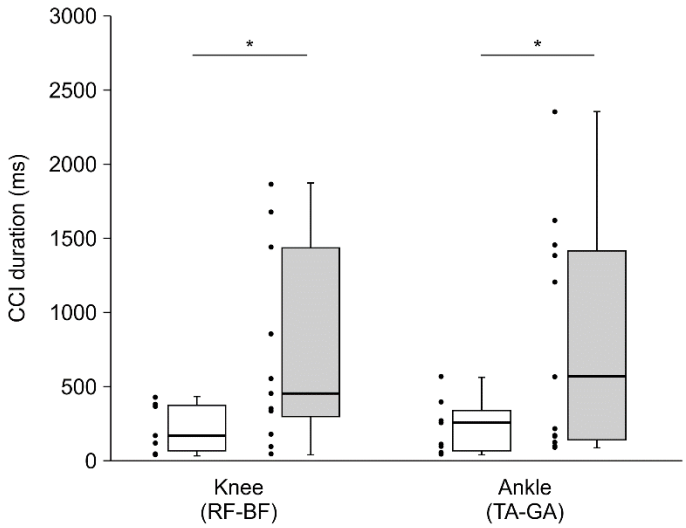


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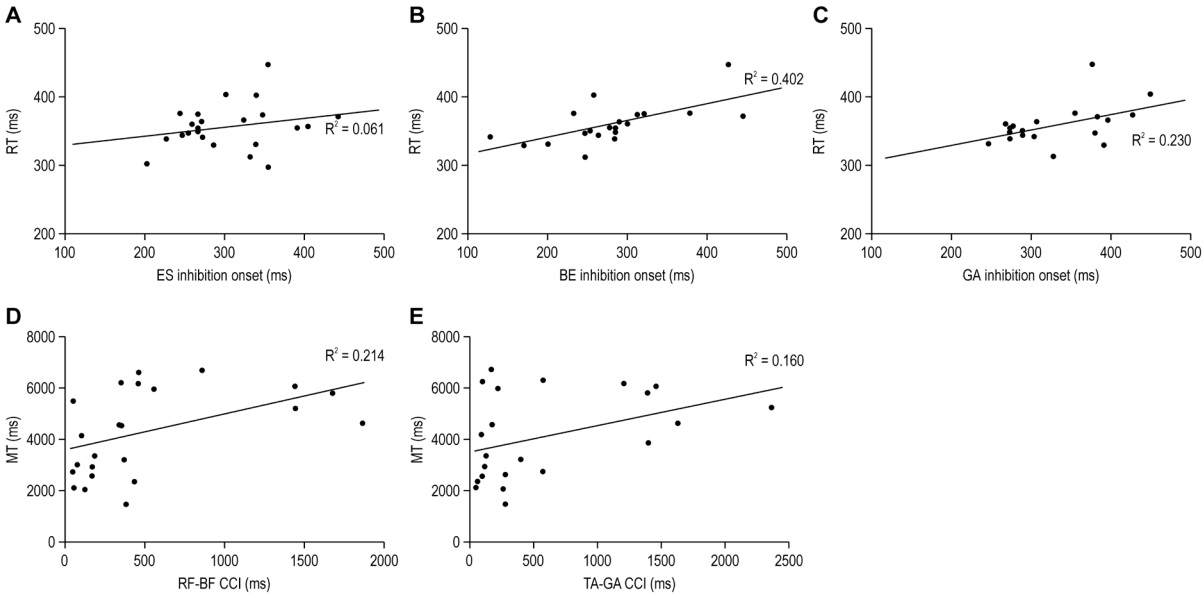
Fig. 6.


A
B
C
D
E
RT (ms)
MT (ms)
ES inhibition onset (ms)
BE inhibition onset (ms)
GA inhibition onset (ms)
RF-BF CCI (ms)
TA-GA CCI (ms)
R² = 0.061
R² = 0.402
R² = 0.230
R² = 0.214
R² = 0.160

Tables

Table 1. Participant characteristics.

	Young adult group	Older adult group
Age (years)	20.7 ± 0.5	70.1 ± 3.4*
Height (cm)	171.8 ± 4.3	164.5 ± 5.3
Weight (kg)	64.7 ± 5.5	62.7 ± 9.7
BMI (kg/m^2)	21.9 ± 1.5	23.1 ± 9.7

Values are presented as mean ± standard deviation.

BMI: body mass index.

*Statistically significant difference between groups ($p < 0.05$).

Table 2. Comparison of reaction time, total movement time, and amplitudes of the hip, knee, and ankle joints.

	Young adult group	**Older adult group**	***p*-value**
Reaction time (ms)			
Hip	-	520.0 ± 68.7	-
Knee	348.3 ± 79.6	439.0 ± 54.5	0.017
Ankle	362.0 ± 63.0	396.0 ± 71.4	0.235
Movement time (ms)			
Hip	-	4951.1 ± 2435.5	-
Knee	2368.3 ± 948.9	4618.0 ± 1835.2	0.015
Ankle	1310.0 ± 729.0	2521.3 ± 1501.7	0.027
Joint amplitude (°)			
Hip	-	3.1 ± 2.8	-
Knee	3.0 ± 2.0	2.6 ± 1.4	0.505
Ankle	2.4 ± 1.0	2.5 ± 1.0	0.966

Values are presented as mean ± standard deviation.

The reaction time, movement time, and amplitude of the hip joint in the young adult group are not shown because of the low occurrence rate of hip movement.

Table 3. Comparison of the reaction time and duration of the rectus abdominis, erector spinae, rectus femoris, biceps femoris, tibialis anterior, and gastrocnemius.

	Young adult group	**Older adult group**	***p*-value**
Hip			
RA activation reaction time	372.8 ± 58.6	-	-
RA activation duration	742.6 ± 239.7	-	-
ES inhibition reaction time	271.6 ± 30.4	325.4 ± 67.2	0.017
ES inhibition duration	344.2 ± 81.6	231.5 ± 107.4	0.015
ES activation reaction time	615.8 ± 76.1	581.3 ± 159.9	0.554
ES activation duration	789.2 ± 484.3	1644.4 ± 1077.2	0.018
Knee			
RF activation reaction time	284.5 ± 53.7	339.4 ± 52.6	0.026
RF activation duration	374.9 ± 180.8	857.8 ± 609.8	0.009
BF inhibition reaction time	243.3 ± 58.8	315.5 ± 76.1	0.029
BF inhibition duration	361.8 ± 108.1	235.3 ± 117.3	0.022
BF activation reaction time	591.2 ± 111.6	468.0 ± 138.6	0.027
BF activation duration	904.2 ± 540.9	1663.2 ± 945.5	0.041
Ankle			
TA activation reaction time	308.3 ± 66.1	347.9 ± 211.3	0.592

TA activation duration	537.2 ± 164.8	1007.8 ± 698.1	0.019
GA inhibition reaction time	300.2 ± 48.3	355.2 ± 62.2	0.040
GA inhibition duration	312.6 ± 89.3	248.8 ± 69.0	0.091
GA activation reaction time	612.8 ± 78.4	625.4 ± 51.5	0.625
GA activation duration	688.6 ± 410.1	1719.5 ± 971.3	0.005

908 Values are presented as mean ± standard deviation.

909 RA: rectus abdominis; ES: erector spinae; RF: rectus femoris; BF: biceps femoris; TA:

910 tibialis anterior; GA: gastrocnemius.

911 The reaction time and amplitude of the RA in the older adult group are not shown because of

912 the low occurrence rate of RA movement.