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Temporal modulations of agonist and antagonist muscle activities accompanying improved performance of ballistic movements

by

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Abstract

Although there are many reports relating to the performance improvements of ballistic movement through practice, how performance is improved while maintaining maximum velocity, and what changes in accompanying triphasic electromyographic (EMG) activity occur, are still unclear. The present study focused on changes in the triphasic EMG activity, i.e., the first agonist burst (AG1), the second agonist burst (AG2) and the antagonist burst (ANT), correspondent to the performance improvements of decreased movement time and error. Twelve healthy volunteers with the instruction of ‘‘Maintaining maximum velocity throughout the experiment and stopping the limb at the target as fast and accurately as possible’’, performed 100 ballistic wrist flexion movements in 10 sessions of 10 trials. Kinematic parameters (position and velocity) and triphasic EMG activities from the agonist (flexor carpi radialis) and antagonist (extensor carpi radialis) muscles, were recorded. By comparison with the results obtained from the first and the last 10 trials, although maximum velocity and time to maximum velocity were unchanged, movement time, error, and variability of amplitudes were expectedly reduced through practice. EMG activities showed that durations of AG1 and AG2 were reduced, whereas duration of ANT did not change. Additionally, latencies of ANT and AG2 were reduced. Integrated EMG of AG1 was also significantly reduced. Analysis of the α angle (an index of the rate of recruitment of the motoneurons) showed that there was no change in either AG1 or AG2. Correlation analysis of α angles between these two bursts further reveals that the close relationship of AG1 and AG2 was kept constant through practice. The present evidence leads us to conclude that improvement of performance in ballistic movement is mainly due to the temporal modulations of agonist and antagonist muscle activities when maximum velocity is kept constant. Presumably, a specific strategy is consistently applied during practice.

PsycINFO classification

2343; 2530

Keywords

Ballistic movement; Triphasic EMG pattern; Practice; Motor strategy; Maximum velocity

1. Introduction

In our daily life, we experience numerous movements at various speeds in keeping with the speed-accuracy trade-off (Fitts, 1954). That is, higher accuracy combines with lower velocity and vice versa. Smooth movements are executed more accurately with continuous modification by peripheral feedback (closed-loop). In contrast, ballistic movements are rapid and may be completed before sufficient feedback is received (open-loop), with less accuracy of performance.

Several body parts have been studied in ballistic movements, for example, flexion and extension of the forearm (Brown & Cooke, 1981; Gottlieb et al., 1989a; Corcos et al., 1993; Jaric et al., 1993; Flament et al., 1999; Kempf et al., 2001), flexion and extension of the wrist (MacKinnon & Rothwell, 2000; Kempf et al., 2001), and abduction of the index finger (Mills & Kimiskidis, 1996). When these ballistic target movements were performed, a similar triphasic electromyographic (EMG) activity could be observed in the agonist and antagonist muscles. That is, two bursts in the agonist muscle (AG1 and AG2) separated by a single burst in the antagonist muscle (ANT). AG1, playing an important role in accelerating the limb, is responsible for the initial phase of movement. The duration and amplitude of AG1 vary in different motor tasks, and no one set of rules has been found that is adequate for all types of movement (Berardelli et al., 1996). Previous studies provided two useful models to explain the form of AG1: pulse height and pulse width hypotheses (Gottlieb et al., 1989a; Corcos et al., 1989). The former assumes that the duration of input to the motoneuron pool is constant, and the amount of excitation is only generated by changing the height of the pulse. The latter assumes that a constant excitatory pulse is given to the motoneuron pool with different durations. These opposing models, which correspond to speed-sensitive (SS) and speed-insensitive (SI) strategies respectively, could account for various ballistic movements with different kinematic requirements (Gottlieb et al., 1989b). ANT, which begins near the end of AG1, is considered as halting the movement at the desired position (Brown &

Cooke, 1990). It has been suggested that ANT varies dependent on the task requirements (Waters & Strick, 1981), and the mechanisms responsible for it are somewhat different to those for AG1 (MacKinnon & Rothwell, 2000). AG2 plays a role in reducing the oscillations that occur at the endpoint of the movement (Hallett & Marsden, 1979; Berardelli et al., 1996), although the mechanisms are less clear. These EMG bursts persisted even when somesthetic afferent information was blocked by ischemia (Sanes & Jennings, 1984), and could be observed in patients with pan-sensory neuropathy, cerebellar tremor, essential tremor, motor ataxia after a thalamus hemorrhage, or primary dystonia (Rothwell et al., 1982; Britton et al., 1994; Berardelli et al., 1996; van Blercom et al., 1997; MacKinnon et al., 2004). These data support the view that afferent information is not necessary for the occurrence of the triphasic EMG activity, and that the characteristic pattern is centrally pre-programmed (Cooke et al., 1985; Sanes et al., 1985; Mills & Kimiskidis, 1996; MacKinnon & Rothwell, 2000).

The pre-programmed descending command could be varied dependent on different requirements of the task (Gottlieb et al., 1989b; Berardelli et al., 1996). Moreover, the performance could be improved in a certain circumstance through motor learning (Corcos et al., 1993; Flament et al., 1999; Kempf et al., 2001), and this improvement could then be transferred to the task with different amplitudes (Jaric et al., 1993). EMG profiles always showed larger and steeper EMG activities in AG1, ANT and AG2, and a decrease in latency of ANT, suggesting that the descending command, or motor strategy, might be somewhat modulated for improving performance of movement. In most of the previous studies, the instructions for a targeted ballistic task given to the participants are ‘‘as fast as possible’’ or ‘‘as fast and accurate as possible’’. Regarding these instructions, it is presumed that the participants have different strategies in order to increase the maximum velocity, to decrease movement time and error, or all of these (Engelhorn, 1997). In the previous reports, because these variables were all improved through practice, we may not clearly

identify whether the larger EMG activities are contributory to the increased maximum velocity or to the improved performance. Such larger EMG activities of agonist and antagonist muscles are required for increasing maximum velocity and stopping the movement; it can be observed not only by practice but also when participants intended to increase the maximum velocity. When performing with the maximum velocity from the beginning and maintaining it during practice, how the pre-programmed motor command changes to improve performance, is still unclear.

To address this question, we encouraged subjects to train the ballistic wrist flexions while maintaining maximum velocity throughout practice, and put emphasis on stopping their limb at the target position as fast and accurately as possible. According to the experimental protocol, we assumed that the problems which subjects have to solve are to shorten the movement time and to decrease the error of movement, rather than to increase the maximum velocity. We recorded kinematics of movement and EMG activities of the agonist and antagonist muscles, and focused on the qualitative and quantitative changes in descending motor commands, which are characterized by the triphasic EMG pattern. In particular, we were interested in whether pulse height and pulse width hypotheses, as previously reported, could explain the changes in EMG activities. These investigations may provide further evidence of the mechanisms of motor learning in relation to performance improvement of ballistic movement.

2. Materials and methods

2.1. Subjects

Twelve right-handed healthy volunteers (nine males and three females, mean age: 29.5 years, range: 22-50 years) participated in the present study. All participants gave their informed consent prior to the experiment. The experimental procedures were in accordance with the

Declaration of Helsinki, and were approved by the Local Ethics Committee of Hiroshima University.

2.2. Experimental procedure

Subjects were seated in a comfortable armchair with their shoulders relaxed in a neutral position. Right upper arm rested with the elbow flexed approximately 90°, with the right forearm and wrist joint in a neutral position (0° of pronation/supination, 0° of flexion/extension, respectively). A homemade table which consisted of a manipulandum mounted vertically was set up in front of the subject (Fig. 1A). The manipulandum below the right wrist could be rotated smoothly on the vertical axis, so that flexion at the wrist joint accompanied with rotation of the manipulandum could be made in the horizontal plane. To confirm the maximum velocity for each subject, at the beginning of the experiment a minimum of 10 practice trials were performed with the instruction of ‘‘as fast as possible’’ regardless of target position. Then, subjects were instructed to flex their wrists from the starting position (0°) to the target (30°) after a ‘‘go’’ signal (experimenter’s voice ‘‘go’’), always maintaining maximum velocity, and to make maximum effort to stop their limb at the target position as fast and accurately as possible. They were also required to make their performance smooth and continuous, and to hold the final position for several seconds. Subjects were informed that it was not a reaction task, so they could initiate the movement at anytime after the ‘‘go’’ signal. To obtain information of the accuracy of each movement, in addition to the visual feedback, subjects could receive auditory feedback (a lasting beep) only when they stopped their wrists at the target position exactly (approximately 30±1°). Each subject performed 10 trials per session and 10 sessions were done in the present experiment. Special attention was paid to avoid fatigue-induced changes in the EMG pattern and movement performance (Corcos et al., 2002), by having an inter-trial interval of 20 sec and an inter-session interval of 2-3 min. This issue was

reconfirmed by evaluating median frequency of EMG in the off-line analysis (Stulen & De Luca, 1981; Hägg et al., 2000; Farina & Merletti, 2000).

Insert Figure 1 here

2.3. Performance and EMG recordings

Kinematics of the movement were recorded by a potentiometer. The potentiometer was attached to the axis of rotation of the manipulandum, of which the mechanical load was minimized to enable subjects to perform the movements smoothly, and to keep their movements consistently in the horizontal plane. Potential changes corresponding to the ongoing position changes were obtained by a transmitter system (model 1418 A2 N2627 and model 1968 A2 N3028, SAN-EI, Japan). The data was stored in a computer for later off-line analysis. Surface EMGs were recorded from the right flexor carpi radialis (FCR, agonist) and extensor carpi radialis (ECR, antagonist) muscles using 9 mm diameter Ag-AgCl surface cup electrodes. The electrodes were placed about 5cm apart on the bellies of FCR and ECR muscles, respectively. EMG signals were amplified at a bandwidth of 5Hz to 5kHz, sampled at 5kHz (model AB-621G, Nihonkohden, Tokyo, Japan), and were recorded by the computer for off-line analysis.

2.4. Data analysis

We collected kinematic and EMG data of the first session (Pre-practice) and the last session (Post-practice) for the analysis of practice effects. Regarding position changes, the overt movement onset was determined as the time point at which movement amplitude reached a threshold (averaged baseline+ 2SD). Termination of movement was estimated when the absolute value of velocity was below 0.03deg/ms, and simultaneously the absolute values of position change after the endpoint was less than 1deg. The temporal kinematic parameters included: 1) from the first agonist burst onset to overt movement onset (motor time, MT; position in Fig. 1B), 2) from overt

movement onset to the time when the wrist reached 30° of flexion (T1; position in Fig. 1B), 3) from the time of reaching 30° to the termination of the movement (T2; position in Fig. 1B), and 4) T1+T2 (Movement time). Since the change of MT is attributable not only to the peripheral delay but also to the central processing of organization of movement (Nagasaki et al., 1983), we calculated it to determine if any change occurred in the motor center during the initial phase of ballistic movement. T1 allow us to confirm whether time spent in moving the limb to the target before the terminal oscillation is unchanged through practice. T2, the oscillation time, is the index of adjusting time. The sum of T1 and T2, the movement time, is an index of movement speed. Data of the maximum velocity (MV; velocity in Fig. 1B) and the time to maximum velocity (Time; velocity in Fig. 1B) which is equal to the duration of the initial phase of limb acceleration, were also measured and calculated. The amounts of overshooting and undershooting which were measured and calculated by the angle differences to the target (30°), indicated the error of the movement (Error A and B; position in Fig. 1B). The variability of error was calculated to evaluate the steadiness of the movement.

EMG signals of FCR and ECR muscles were amplified and full-wave rectified (EMG in Fig. 1B). The onset of AG1 (0 ms in the present study) was defined as the time point at which EMG amplitude reached a threshold (averaged background EMG+ 4SD), and after the time point EMG amplitude remained above the level for at least 25ms (Hodges & Bui, 1996; Kudo & Ohtsuki, 1998). The threshold was determined for each subject. All data analyses used this standard reference (0 ms, the onset of AG1). At first, we adopted median frequency contents of EMG to rule out the confounding factor of fatigue. Fast Fourier Transform (FFT) was performed, and median frequency (Hz) of the power spectrum over 500ms was determined in the EMGs of both FCR and ECR muscles (MATLAB 6.1; The MathWorks, Inc.). Since small spontaneous twitches can usually be observed in the agonist and antagonist muscles during ballistic movement, we adopted the root

mean square of EMG to identify the end of AG1, and the onset and end of ANT and AG2 (Hägg et al., 2000; Farina & Merletti, 2000). Then, we calculated the duration of each burst and the integrated EMG (iEMG) of each burst using automated measurement software (Excel 2002; Microsoft Corporation). Also measured and calculated were overlapping iEMG between AG1 and ANT, or AG2 and ANT at Pre- and Post-practice. The excitation pulse of descending motor command, which is delivered to the motoneuron pool of the agonist muscle, is an important aspect in the present study. To investigate qualitative changes of the descending motor command, after applying a low pass filter to EMG of FCR muscle (22.15 Hz) we calculated the α angles of AG1 and AG2 and investigated the relationship between them. The α angle is comprised of the rectified EMG baseline and the line that joins the onset and the peak of the rectified EMG (Possamai et al., 2002). It could be an index of the rate of recruitments in the motoneuron pool. The larger α angle would reflect a faster rate of recruitment in the motoneuron pool, in which the motor units might be discharged more synchronously (Meijers et al., 1976; Ulrich & Wing, 1991). In each burst, the latency between onset and peak (peak latency), peak value, and iEMG between onset and peak were also calculated and these values were compared between Pre- and Post-practice. Data analyses were undertaken randomly and blindly for all sessions.

In kinematics, each parameter at Pre- and Post-practice was compared by a paired *t*-test. The correlation between MV and error of the movement in the Pre- and Post-practice was tested by a linear regression analysis based on Pearson's coefficient analysis, followed by a multiple analysis of variance (MANOVA; correlation between MV and error at Pre- and Post-practice). The comparison of the two correlation coefficients was also tested. In EMG profiles, each parameter at Pre- and Post-practice was compared by a paired *t*-test. The correlations of α angles between AG1 and AG2 at Pre- and Post-practice were tested by linear regression analyses based on Pearson's coefficient analyses, and the comparison of the correlation coefficients was tested. Also tested were

correlations between kinematics and EMG by linear regression analyses (Pearson's coefficient analyses). The level of statistical significance was set at $P<0.05$. The data values are expressed as means± SE.

3. Results

3.1. Changes of kinematic parameters

Fig. 2A shows the typical recordings of position (left traces) and velocity (right traces) changes at Pre- (grey lines) and Post- (black lines) practice. The means and SE of all subjects (N=12) are shown in Fig. 2B, C and D. There were no changes of MT, T1, MV, and time to MV after practice (Fig. 2B and C), although both error and variability of movement were expectedly improved (error; overshooting, $P<0.05$, undershooting, $P<0.05$, variability; overshooting, $P<0.05$, undershooting, $P<0.05$, Fig. 2D). Moreover, movement time (and T2) was significantly shorter at Post-practice compared to Pre-practice ($P<0.001$).

Correlation analysis showed that the close relationship between MV and error after practice had no change in spite of reduction of errors (Pre; $r=0.78$, $P<0.01$, Post; $r=0.88$, $P<0.001$, MANOVA; $F=8.95$, $P<0.01$, Fig. 2E). These results indicated that the relationship between velocity and accuracy of ballistic wrist movement did not change in spite of improvement of accuracy through practice.

Insert Figure 2 here

3.2. Changes of EMG profiles

Median frequencies of EMG activities revealed that there were no changes between Pre- and Post-practice in FCR and ECR muscles, respectively (Table. 1). It means that any changes in EMG profiles that are not caused by muscle fatigue. EMG activities at Pre- and Post-practice are

shown in Fig. 3. Fig. 3A shows the typical EMG recordings of agonist (upper traces; FCR) and antagonist (lower traces; ECR) muscles, corresponding to the representative examples of kinematics in Fig. 2A. EMG recordings of Pre- and Post-practice are expressed in grey and black traces, respectively. The means and SE of all subjects (N=12) are shown in Fig. 3B and C. After practice, duration of AG1 was significantly shorter ($P<0.01$) and iEMG was smaller ($P<0.05$) than those of Pre-practice. With regard to ANT, latency significantly decreased at Post-practice ($P<0.05$), although there were no changes in duration and iEMG. Concerning AG2, both duration and latency at Post-practice significantly decreased compared with Pre-practice ($P<0.01$, $P<0.05$, respectively) in spite of no change in amount of iEMG. It would be worthwhile noting here that intervals of the AG1 end to ANT onset, or to AG2 onset, and interval of the ANT onset to AG2 onset were all similar irrespective of practice. The amounts of overlapping iEMG (or FCR/ECR ratio), namely co-contractions between these bursts, were not significantly different between Pre- and Post-practice (Table. 2). It was suggested that ANT and AG2 following AG1 were both forward shifted through practice.

Insert Table 1 here	Insert Figure 3 here	Insert Table 2 here

Fig. 4 shows the results of α angles (N=12) calculated in FCR muscle at Pre- and Post-practice. The α angles of AG1 and AG2 showed no change between Pre- and Post-practice (Fig. 4A). Correlation analysis of α angles further revealed that relationships of α angles between AG1 and AG2 were not modified and then, both relationships were consistently kept at Pre- and Post-practice (Pre; $r=0.68$, $P<0.05$, Post; $r=0.74$, $P<0.01$, Fig. 4B).

Insert Figure 4 here

Fig. 5 shows the results of peak latency (Fig. 5A), peak value (Fig. 5B), and iEMG between onset and peak (Fig. 5C) of rectified EMG activities at Pre- and Post-practice. Although there were no changes observed in AG2, in AG1 the peak latency was significantly shorter ($P<0.05$)

and the peak value was significantly smaller (P<0.05) at Post-practice compared with those at Pre-practice. Therefore, although the α angle of AG1 showed no change through practice, the iEMG between onset and peak of AG1 became smaller (P<0.05). Regarding ANT, the peak latency was also significantly shorter (P<0.01) and similarly, the iEMG between onset and peak became smaller (P<0.05) through practice, while there was no change in the peak value. Additionally, statistical analyses revealed that peak latency of ANT significantly correlated to that of AG1 (r=0.46, P<0.05), and iEMG between onset and peak of ANT significantly correlated to that of AG1 (r=0.64, P<0.001), while there was no significant correlation of peak value between them. In other words, the duration and iEMG in the early phase of ANT are dependent on those of AG1. It suggested that when AG1 activated efficiently through practice, ANT provided a smaller braking force in the early phase to ensure maintenance of maximum velocity.

Insert Figure 5 here

4. Discussion

The novel finding of the present study was that, the performance of ballistic movement was prominently improved through practice when maintaining maximum velocity. Importantly, accompanied EMG activities showed the temporal, rather than quantitative, modulations correspondent to the performance improvements. The results suggested that through practice, a shorter excitation pulse was given to the motoneuron pool of agonist muscle and an earlier excitation pulse was given to that of antagonist muscle for improving the performance.

4.1. Improvement of performance

Through practice, decreases in movement time (especially the adjusting time), error, and variability of amplitudes were clearly observed in the present study (Fig. 2D). Contrary to previous

studies in which maximum velocity clearly increased with the instruction of ‘‘as fast as possible’’ or ‘‘both fast and accurate’’ (Corcos et al., 1993; Flament et al., 1999; Kempf et al., 2001), the present results showed no change in maximum velocity, or time to maximum velocity through practice (Fig. 2C). This suggests that instruction can influence the performance of ballistic movement (Gottlieb et al., 1989b; Kempf et al., 2001), and the protocol which we applied eventually produced movements that were both fast and accurate while the maximum velocity was constant. There was no change in the relationship between maximum velocity and error through practice, indicating qualitative changes in motor strategies in keeping with the speed-accuracy trade-off (Fig. 2E).

4.2. EMG changes accompanying improved performance

EMG discharges showed clear changes accompanying improved performance through practice, which are demonstrated by the decreases of duration in AG1 and AG2, and the forward shifts of ANT and AG2 (Fig. 3B). Since triphasic EMG bursts would be centrally programmed as previously reported (MacKinnon & Rothwell, 2000), the present results suggest that descending motor commands were improved through practice, particularly in the temporal modulations of these bursts.

AG1 has been studied extensively, and it is well-known that the burst provides an estimate of the force produce by the muscle. As commented in the introduction, SS and SI strategies can modify the form of AG1 in different circumstances with ballistic movement (Gottlieb et al., 1989b). In the present results, we could not find any changes in the kinematics parameters in the initial phase of movement, whereas EMG activity and duration of AG1 decreased through practice. It has been proposed that practiced movements are often characterized by smaller muscle activations compared with unpracticed movements, i.e., improved performance is accompanied by more efficient activities of muscle (Payton & Kelley, 1972; Engelhorn, 1983; Yahagi et al. 2005). If this is

the case, one can consider the smaller AG1 as a result of the superabundant activity being removed after practice. In other words, it is most likely that AG1 was activated more efficiently through practice. How does the central pre-program change, and what kind of strategy was applied? It has been proposed that iEMG would vary by both amplitude and duration of EMG activities (Gottlieb et al., 1989b), i.e., EMG activity would decrease by either reducing the intensity (pulse height hypothesis) or the duration (pulse width hypothesis) of the excitation pulse that is delivered to the motoneuron pool. Taken together with our results of unchanged α angle and decreased duration of AG1, it is most likely that decreased EMG activity of AG1 was caused by a shorter excitation pulse with constant intensity given to the motoneuron pool. Thus, the SI strategy, rather than the SS, might be dominantly applied for improving performance in the present study.

Regarding ANT, it has been shown that performance improvements are accompanied with decreased latency and an increased amount of ANT (Corcos et al., 1993; Flament et al., 1999). It is conceivable that either an earlier or a larger ANT can make a stronger braking force during ballistic movement. In the present study, while the maximum velocity was unchanged through practice, ANT activated earlier in order to stop the limb at the target position quickly and accurately. The duration and EMG activity in the early phase of ANT are respectively dependent on those of AG1, suggesting that when AG1 is activated efficiently through practice, ANT with smaller EMG activity in the early phase ensures the unchanged maximum velocity. Thus, not only the timing but also the activation of the early phase of ANT is important to improve performance when a ballistic movement is initiated with the same velocity. Previous studies have suggested that ANT could be generated independently from AG1 (Crammond & Kalaska, 1996; MacKinnon & Rothwell, 2000), mainly at the subcortical level (Flament & Hore, 1986; Hore et al., 1991; MacKinnon & Rothwell, 2000; Ito, 2001). Therefore, if the onset timing of ANT is modified by the cerebellum as previously reported, the change of ANT observed in the present study might reflect a practice-induced effect on

the cerebellar output drives.

Unlike those for AG1 and ANT, the underlying mechanisms of AG2 are less clear, and no previous report refers to the relationship between AG2 and performance of ballistic movement. In the present study, decreased latency and shortened duration of AG2 were clearly observed through practice. In particular, analyses of the relationship between kinematics and EMG revealed that the latency and end of AG2 significantly correlated to the movement time (r=0.44, P<0.05, r=0.47, P<0.05, respectively). That is, the earlier the AG2, or the shorter the whole burst, the shorter the movement time. As described above, if decreased latency of ANT plays an important role in braking, it is reasonable that an earlier anti-braking produced by AG2 is needed to prevent excessive braking. What are the underlying mechanisms and motor strategies related to the changes of AG2? In the present study, similar to AG1, AG2 had a shorter burst while the α angle was unchanged through practice. In addition, α angles of AG2 and AG1 maintained a close relationship irrespective of practice. These results suggest that the firing patterns of motoneurons corresponding to AG1 and AG2 are similar. In other words, it is most likely that AG2 is generated by cortical excitability in the same way as AG1 (MacKinnon & Rothwell, 2000). Descending excitation pulses delivered to the motoneuron pools of the agonist muscle may therefore have an identical intensity with a shorter duration accompanied with improved performance of ballistic movement.

4.3. Relations between kinematics and EMG

In the present study, although decreases of latency or end time of AG2 directly reduce the movement time as described above, there were no significant correlations between EMG activities and movement errors of kinematics (not illustrated). Regarding movement error, overshooting is likely dependent on both AG1 (size and duration) and ANT (size, duration and latency), and undershooting might depend on ANT and AG2 (size, duration and latency), and possibly even on

AG1. Although EMG activities could show drastic changes in certain rules dependent on motor strategic changes corresponding to movement amplitude, inertial loads, and instructions of the task (Brown & Cooke, 1981; Gottlieb et al., 1989; Berardelli et al., 1996), through practice, it was suggested that the amounts of EMG activities might not be directly reflected in kinematics, especially the magnitude-related ones. Moreover, in the present study several parameters were simultaneously changed through practice. The issues of which parameters of EMG activities are related to each other, and how they interact, are still unclear. These interactions may be reflected in kinematic changes through practice, and it is suggested that not only quantitative but also qualitative changes of EMG activities induced by motor strategic changes might occur. Therefore, one possible explanation for the results is that, although the smaller AG1 and forward shifted ANT and AG2 are responsible for the improved performance, parameters of EMG complexly interacted with one another, and as a result the pattern of muscle activations related to the error of movement is not unique (Corcos et al., 1993).

4.4. Speed-insensitive strategy for improving performance of ballistic movement

The similar behavior of AG1 and AG2 from the present results suggests that at least in the agonist muscle, a SI strategy was dominantly used through practice. That is, EMG activities rose at the same rate (unchanged α angle in the EMG profiles), and initial muscle force was unaffected (unchanged maximum velocity in the kinematics) by practice. The temporal modulations of these bursts are likely the only, or the more efficient way to improve the performance of ballistic movement. Although the underlying mechanisms of temporally-forward-shifted bursts following AG1 are still not clear, the unchanged intervals between these bursts reveal some temporal patterns in the pre-programmed descending excitation pulses during ballistic movement.

5. Conclusion

All findings of the present study lead us to further consider that improvement of ballistic movement through practice is associated with changes in descending motor commands. When participants perform ballistic movements while maintaining maximum velocity, they can successfully decrease the movement time and error through practice. It is most likely that performance improvements are due to the temporal modulations of triphasic EMG bursts rather than the quantitative ones. Through practice of ballistic movements, therefore, increased amounts of EMG bursts indicated by previous studies might be a result of increased maximum velocity. Based on the present results, it is suggested that the improvement in performance is caused by the muscle activating in a more efficient way, and that the speed-insensitive strategy likely plays a dominant role during practice. The present findings may contribute to further understanding of the mechanisms of motor learning in relation to performance improvements of ballistic movement.

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Figure legends

Figure 1

(A) Illustration of experimental setup of wrist flexion in the present motor task. (B) Recordings of kinematics and EMG bursts in wrist flexion. Durations of motor time (MT), movement time (T1+T2) and errors of movement (overshooting; error A, undershooting; error B) are illustrated in

position. Movement velocity (MV) and time to it illustrated in velocity. Recordings of EMG (rectified EMG) show activities in agonist (FCR; AG1 and AG2) and antagonist (ECR; ANT) muscles.

Figure 2

(A) Typical recordings (average of ten trials in one session) of position (*left traces*) and velocity (*right traces*). Grey lines show the recordings for the Pre-practice and black lines show the recordings for the Post-practice. (B) Means and standard errors (N=12) of durations of MT, T1 and T2 at Pre- and Post-practice. (C) Means and standard errors (N=12) of maximum velocity and the time to it at Pre- and Post-practice. (D) Means and standard errors (N=12) of overshooting and undershooting at Pre- and Post-practice. (E) Correlations between maximum velocity and error at Pre- and Post-practice. *P<0.05, **P<0.01, †P<0.001, ††P<0.0001

Figure 3

(A) Typical EMG recordings (average of ten rectified trials in one session) of agonist (FCR; *upper*) and antagonist (ECR; *lower*) muscles. Grey traces show the recordings at Pre-practice and black traces show the recordings at Post-practice. (B) Means and standard errors (N=12) of temporal EMG profiles. Latency (*circles*) and EMG duration (*columns*) are shown at Pre- and Post-practice. (C) Means and standard errors (N=12) of iEMG at Pre- and Post-practice. *P<0.05, **P<0.01

Figure 4

(A) Means and standard errors (N=12) of α angles obtained from EMG recordings of agonist (FCR) muscle. (B) Correlations of α angles between AG1 and AG2 at Pre- and Post-practice. *P<0.05, **P<0.01

Figure 5

Means and standard errors (N=12) of (A) latency between onset and peak, (B) peak value, and (C) iEMG between onset and peak obtained from the rectified EMG recordings at Pre- and Post-practice. *P<0.05, **P<0.01

Table 1
Median frequency of EMG activities of agonist (FCR) and antagonist (ECR) muscles

	FCR		ECR	
	Pre	Post	Pre	Post
Median frequency (Hz)	105.78±7.50	113.95±5.87	135.80±8.05	132.59±9.66

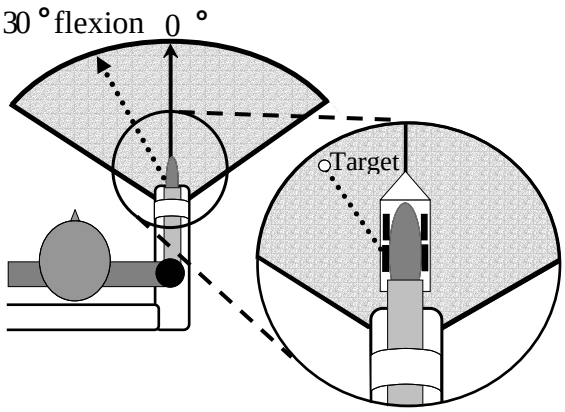
Values are mean±SE
FCR; flexor carpi radialis muscle, ECR; extensor carpi radialis muscle, Pre; pre-practice, Post; post-practice.

Table 2
Amounts of co-contraction between triphasic EMG bursts

	AG1- ANT		AG2- ANT	
	Pre	Post	Pre	Post
FCR (mV.ms)	1.46±0.33	1.82±0.50	8.32±2.27	8.46±2.56
ECR (mV.ms)	1.16±0.25	1.13±0.21	6.14±1.18	5.29±0.95
FCR/ ECR ratio (arb.u.)	1.29±0.16	1.52±0.30	1.31±0.16	1.35±0.23

Values are mean±SE
AG1; first agonist burst, ANT; antagonist burst, AG2; second agonist burst.
Pre; pre-practice, Post; post-practice, FCR; flexor carpi radialis muscle, ECR; extensor carpi radialis muscle.

A


30 ° flexion
0 °
Target

B

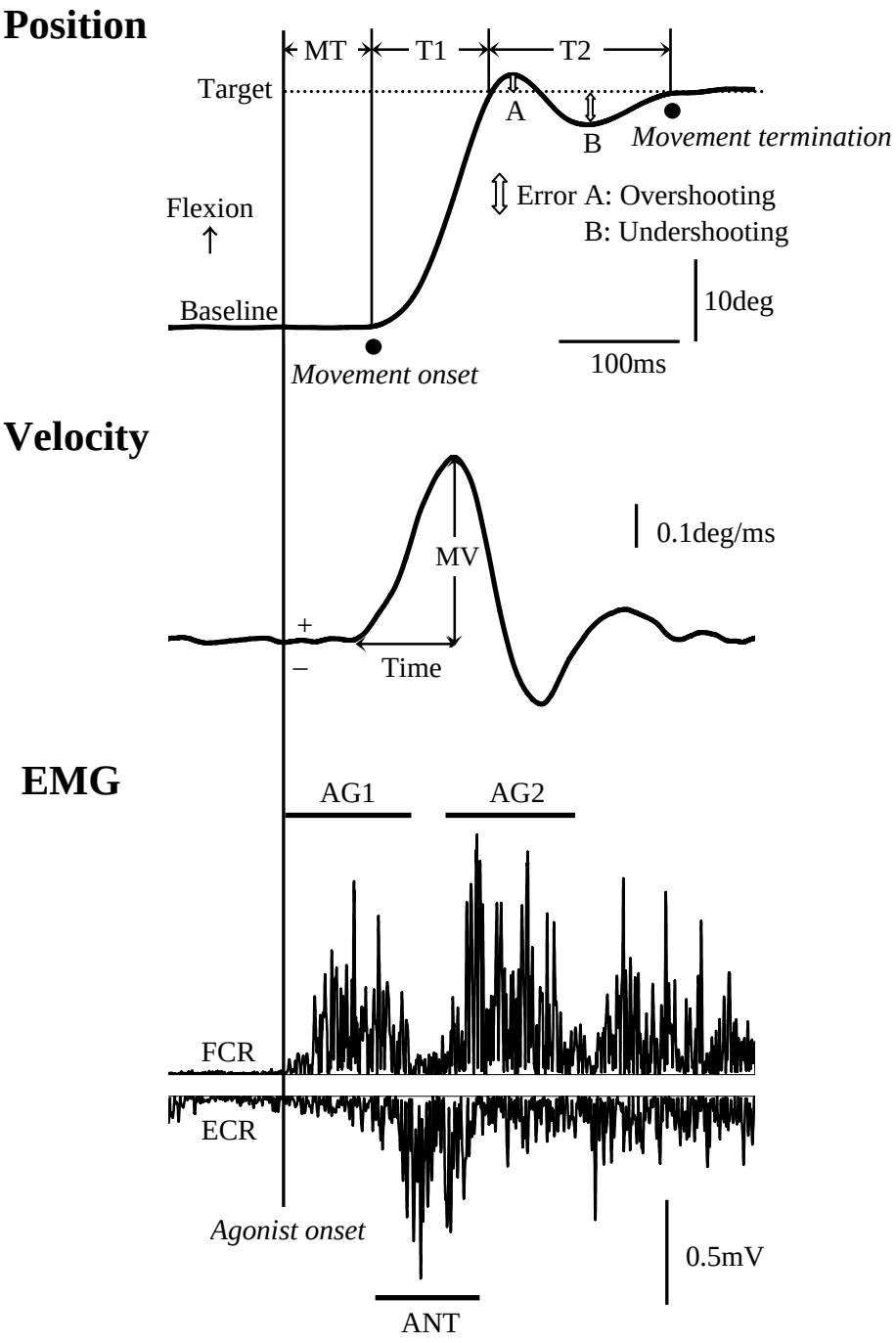

Position
MT
T1
T2
Target
A
B
Movement termination
Flexion
Error A: Overshooting
B: Undershooting
Baseline
10deg
100ms
Movement onset
Velocity
0.1deg/ms
MV
+
–
Time
EMG
AG1
AG2
FCR
ECR
Agonist onset
0.5mV
ANT

Fig 1

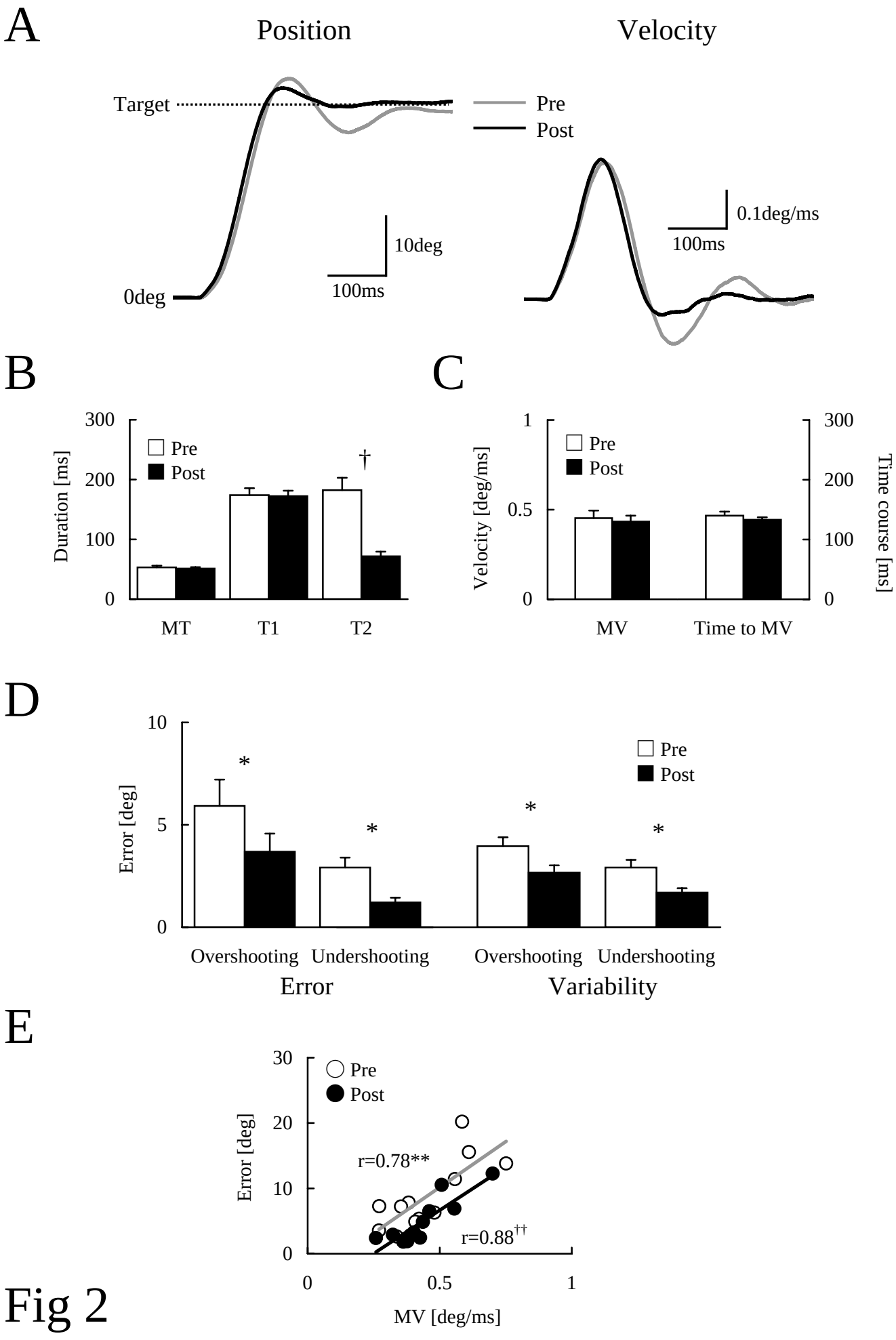
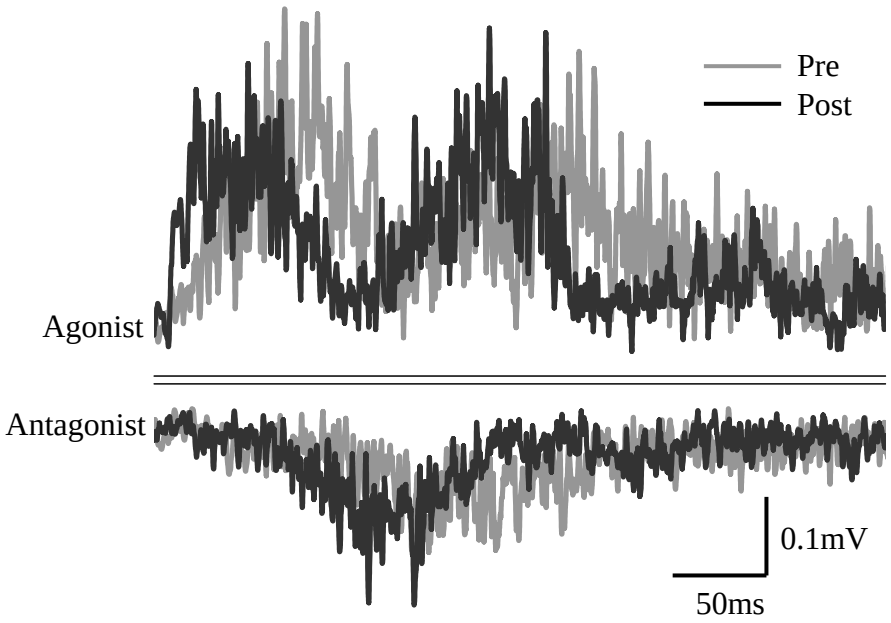
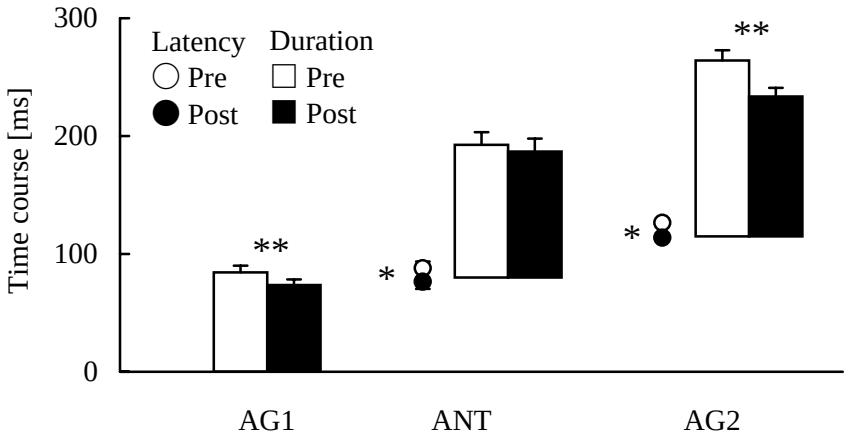

A
Position
Velocity
Target
Pre
Post
10deg
100ms
0deg
0.1deg/ms
100ms
B
C
Duration [ms]
300
200
100
0
Pre
Post
†
MT
T1
T2
Velocity [deg/ms]
1
0.5
0
Time course [ms]
MV
Time to MV
D
Error [deg]
10
5
0
*
Overshooting
Undershooting
Error
Variability
E
Error [deg]
30
20
10
0
r=0.78**
r=0.88††
0
0.5
1
MV [deg/ms]

Fig 2

A


Pre
Post
Agonist
Antagonist
0.1mV
50ms

B


300
200
100
0
Time course [ms]
Latency
Pre
Post
Duration
Pre
Post
**
*
*
**
AG1
ANT
AG2

C

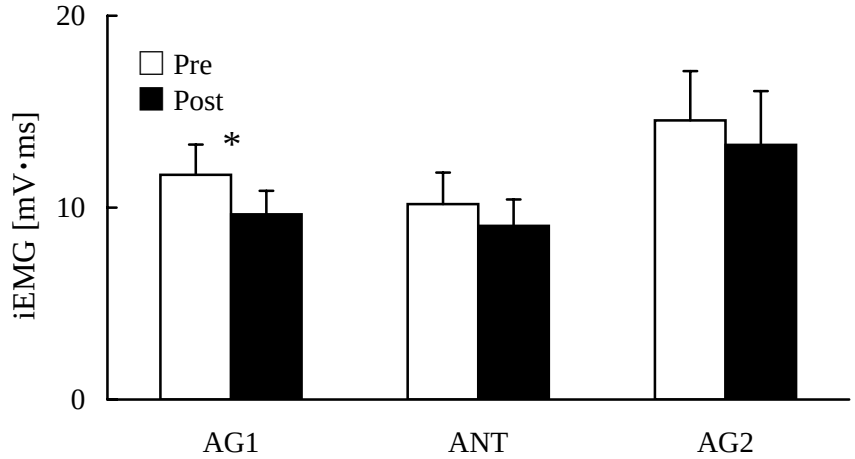
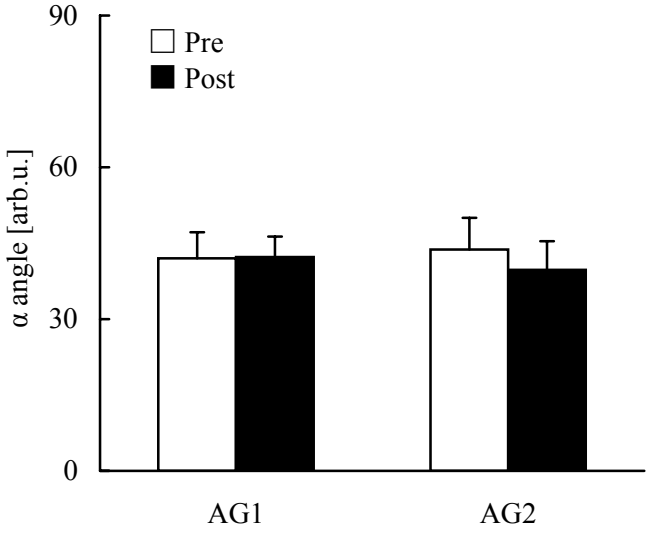

20
10
0
iEMG [mV・ms]
Pre
Post
*
AG1
ANT
AG2

Fig 3

A


90
60
30
0
Pre
Post
α angle [arb.u.]
AG1
AG2

B

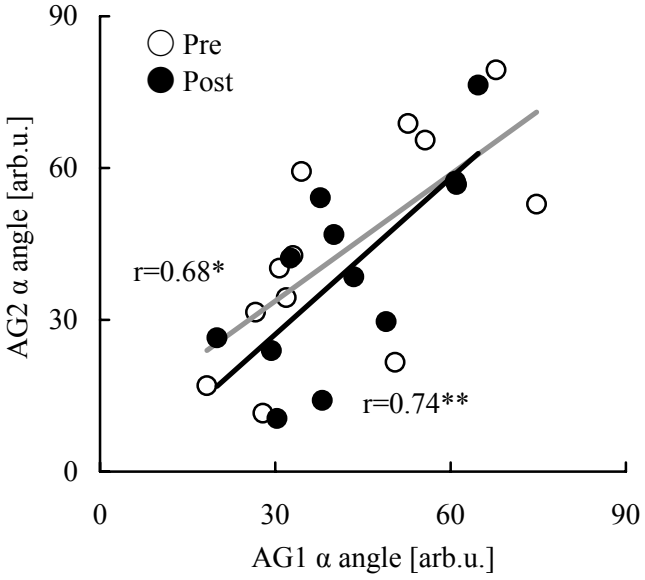

90
60
30
0
Pre
Post
AG2 α angle [arb.u.]
r=0.68*
r=0.74**
0
30
60
90
AG1 α angle [arb.u.]

Fig 4

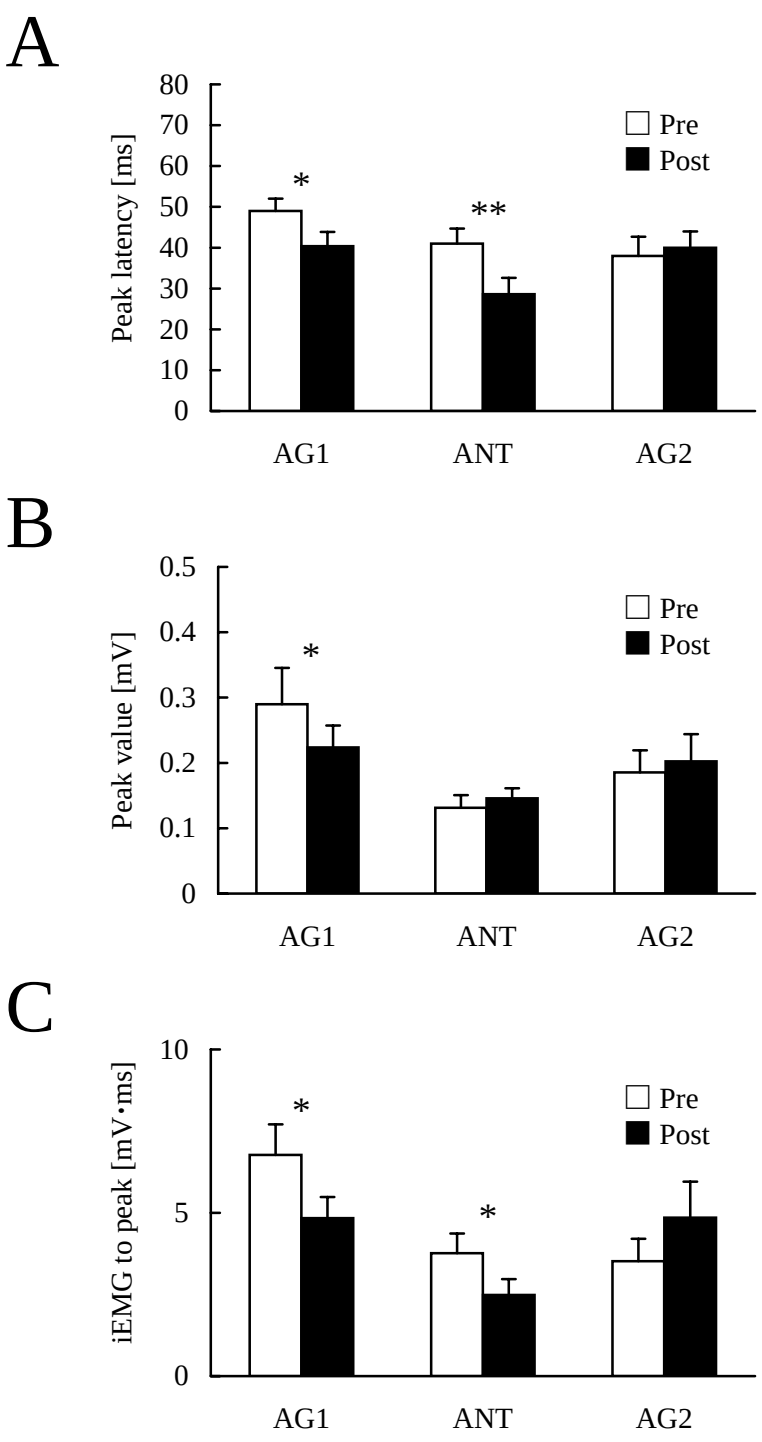

A
Peak latency [ms]
80
70
60
50
40
30
20
10
0
*
**
Pre
Post
AG1
ANT
AG2
B
Peak value [mV]
0.5
0.4
0.3
0.2
0.1
0
*
Pre
Post
AG1
ANT
AG2
C
iEMG to peak [mV･ms]
10
5
0
*
*
Pre
Post
AG1
ANT
AG2

Fig 5