The effect of simultaneous repetition of unrelated finger movements on surround inhibition in the motor system

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The effect of simultaneous repetition of unrelated finger movements on surround inhibition in the motor system

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<ABSTRACT>

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Surround inhibition is a neural mechanism by which desired neural processes can be adjusted by suppression of unwanted, neighboring neural activity for the selection of the proper neuronal response. It is an essential mechanism of the motor system to aid the selective execution of desired movements. Disturbed surround inhibition in the motor system could account for various movement disorders because this impairment prevents the ability to perform desired movements. Dystonia, which is characterized by co-contraction of agonists and surrounding muscles,

may be more pertinent clinically to impaired surround inhibition than other movement disorders. Accordingly, disturbed surround inhibition has been demonstrated in patients with focal hand dystonia (FHD). FHD is often task-specific, and is associated with overuse of the affected muscles. FHD usually develops following a period of highly repetitive movements that typically require extreme motor precision. In this study, I hypothesize that repetitive exercise of unrelated finger movements can lead to disturbed surround inhibition, which could provide physiological evidence to support the potential contribution of muscle overuse to the development of FHD. To explore the effect of muscle overuse on surround inhibition, we measured changes in surround inhibition after short-term exercise with repetition of two different types of finger movements. As reported previously, motor evoked potentials (MEPs) were suppressed in the little finger muscle (abductor digiti mini; ADM) during flexion of the index finger muscle (flexor digitorum superficialis; FDS). After short-term exercise consisting of repetitive finger movements, MEPs of the ADM were no longer suppressed but instead were enhanced during the flexion of the FDS in non-musician healthy volunteers. Exercise comprising simultaneous ADM and FDS movements produced significantly larger and longer-lasting

enhancements than those of the ADM movement alone. In professional pianists, on the contrary, alterations in surround inhibition after both types of exercise were similar. The results of this study provide neurophysiological evidence that repetitive practice of unrelated finger movements can disturb surround inhibition and support the idea that muscle overuse may contribute to the development of task-specific dystonia. The finding that the effect of exercise types on surround inhibition are different between non-musicians and professional musicians supports the hypothesis that neurophysiological mechanisms regulating surround inhibition in professional musicians may be different from those in non-musicians, probably due to previous long-term practice of unrelated finger movements.

Key words: surround inhibition; transcranial magnetic stimulation; motor evoked potentials; motor cortex; musicians; dystonia

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I. INTRODUCTION

Surround inhibition is an important physiological mechanism by which individuals focus neuronal activity and select appropriate neuronal responses, which is achieved by suppression of excitability in an area surrounding an activated neural network.[1] It is also called 'lateral inhibition' and is a common mechanism of the sensory systems of many species, which was first discovered in the visual system of the horseshoe crab, Limulus.[2] The central portion of the receptive field is excited by sensory input, whereas the peripheral portion of the receptive field is inhibited. In the sensory system, surround inhibition plays a role in spatiotemporal discrimination of various sensory inputs.[3] This enables the sensory system to deliver sensory information effectively via gradients and augments the acuity of the sensory system.[2] For example, if a person sees several objects on the table, he can perceive the exact location of each object with clear demarcation of the boundaries. It has been suggested that surround inhibition also may be present in the motor system and help the selective activation of wanted movements in humans. Recently, using transcranial magnetic stimulation (TMS), the functional existence of surround inhibition in the human motor system was demonstrated.[1, 4, 5] For example, during voluntary flexion of the index finger (wanted movement), motor evoked potentials (MEPs) of the little finger muscle (unwanted movements of neighboring, unrelated muscles) are significantly suppressed in healthy people.[1]

Dystonia is characterized by abnormal involuntary muscle contractions resulting from the co-contraction of agonist and antagonist muscles. Focal hand dystonia (FHD) is one of the common forms of acquired dystonia that typically is task-specific, which means it occurs exclusively or mainly when patients perform a specific task. There are several examples of task-specific dystonias such as writer's cramp and musician's cramp. A previous TMS study demonstrated that the operation of surround inhibition was impaired in patients with FHD,[6] supporting the idea that disturbed surround inhibition contributes to the development of dystonia. FHD usually develops following a long-term period of excessive repetitive activity while performing a fine motor skill.[7] Up to 1% of professional musicians develops focal dystonia, called musician's cramp.[10] The prevalence of FHD in professional musicians is much higher than in other professions (i.e., watch makers) demanding skilled hand movements, but not requiring excessive and repetitive practice.[8, 9] Owl monkeys who were over-trained to make a highly specific hand movement developed difficulties in moving their hands, similar to FHD in humans. These animals showed significant dedifferentiation of cortical sensory representations, suggesting the contribution of alterations in cortical connections to the development of overuse dystonia.[10] However, the mechanism of muscle overuse in development of dystonia in humans remains to be clarified. TMS studies showed abnormal sensorimotor integration in patients with FHD, suggesting that maladaptive cortical plasticity could be induced by repetitive skilled practice.[11, 12] Therefore, repetitive unrelated finger movements could lead to changes in cortical connections, resulting in reduced surround inhibition between them. Alteration of surround inhibition may produce co-contraction of unrelated finger muscles and cause dystonia.

In this study, whether repetitive exercise of unrelated finger movements could induce reduction in surround inhibition was investigated in order to explore the contribution of muscle overuse to the development of dystonia. In addition, the effect of repetitive exercise on surround inhibition was assessed in healthy professional pianists to investigate whether the pattern of surround inhibition alteration induced by hand exercise is different in people who already may have chronic adaptive cortical plasticity. Professional musicians are a good model in which to investigate the adaptive plasticity of the sensorimotor integration in humans as a result of long-term regular practice.[13, 14] The hypothesis is that muscle overuse of unrelated finger movements could lead to disturbed surround inhibition, which would provide physiological evidence to support a potential relationship between muscle overuse and FHD.

II. MATERIALS AND METHODS

1. Participants

Thirty-three healthy non-musicians and 23 professional pianists were enrolled after screening. Eighteen of the 33 healthy non-musicians completed the full protocol (nine men and nine women; range 19-34 years, mean age 23.3 ± 3.1 years). Reasons for dropout were: technical problems at initial set-up (eight participants) and no surround inhibition (seven participants). Thirteen of 23 professional pianists completed the full protocol (1man and 12 women; range 19-25 years, mean age 21.9 ± 1.8 years). The remaining could not because of a lack of surround inhibition (five participants); extraordinarily high resting motor threshold (two participants, 88% and 90%, respectively); lack of an appropriate cortical stimulation site (one individual); and refusal to participate further (two participants).

All participants were right-handed according to the criteria of the Edinburgh Handedness Inventory.[15] Non-musicians were defined as having a major outside the field of music, no hobby for playing musical instruments, and no experience with piano lessons before formal school education. Professional pianists were defined as at least undergraduate students in a college of music, whose major was piano or organ performance and who had taken piano lessons from very early childhood. No participant had any history of neurological disease, and all individuals gave written informed consent to participate in this study. The study was approved by the local ethical committee.

2. Transcranial magnetic stimulation

TMS was performed as described previously.[1, 16] Surface electromyography (EMG) activity was recorded (bandpass, 10-2000Hz) from the abductor digiti mini (ADM) and flexor digitorum superficialis (FDS) muscles of the right arm using a conventional EMG machine (Viking IV, Nicolet Biomedical, Madison, WA, USA). The signal was digitized at a frequency of 5 kHz and fed into a laboratory computer for further off-line analysis. A figure-eight-shaped coil (each loop measured 70 mm in diameter) connected to a Magstim 200 magnetic stimulator (Magstim, Dyfed, UK) was placed flat on the scalp over the left motor cortex at the optimal site for eliciting maximal amplitude MEPs in the ADM. TMS triggering and data acquisition were controlled using the LabVIEW program (National Instrument, Austin, TX, USA),[17] with which TMS was set to elicit stimuli only when the EMG activity of ADM was silent (less than 50 μV). The individual resting motor threshold (RMT) was determined to the nearest 1% of the maximum stimulator output and was defined as the minimum stimulus intensity required to produce MEPs of >50 μV in at least 5 of 10 consecutive trials. Using the LabVIEW program and a Schmidt discriminator, TMS was set to be triggered by EMG activity characteristic of FDS (self-triggered TMS). The sensitivity of the Schmidt discriminator was set at a level sufficient to correctly detect the onset of EMG activity and not to produce triggering while resting (usually 100 μV peak-to-peak EMG amplitude). ‘Go’ signals were given at random intervals between five and nine seconds. Participants were asked to flex their right index finger after the ‘go’ signal with a self-paced delay (they were instructed not to react immediately). Before the experiment, participants practiced by making a brief (duration ~100 ms) and selective movement while monitoring their own EMG activity. The interval between the EMG onset and the TMS trigger was set at 3 ms, which produced maximal inhibition as shown in the previous study.[1] MEP size was determined by averaging peak-to-peak amplitudes over 18 trials for each session at the stimulus intensity of 140% of the individual RMT. MEPs of ADM were obtained with and without self-triggered TMS before and after exercise (0, 10, 20, and 30 minutes). Average MEP amplitudes obtained with self-triggered TMS (control TMS) were normalized to the average MEPs during rest (control TMS). Normalized MEP amplitudes were compared between the two exercise sessions.

3. Peripheral nerve stimulation

To assess spinal and peripheral motor excitability, peak-to-peak amplitude and persistence of F waves (average, 20 trials) of ADM were determined with supramaximal electrical stimulation of the ulnar nerve at the wrist. Peak-to-peak amplitude above 20 μV was defined as the presence of the F-wave.[18] Compound muscle action potentials (CMAP; maximum, three trials) of ADM also were also determined before and after exercise. Average peak-to-peak amplitudes, persistence of the F wave, and average CMAP amplitudes after exercise were normalized to the values before exercise. The normalized values were compared between the two exercise sessions.

4. Exercise of repetitive finger movements

After baseline measurements, each volunteer was assigned randomly to perform either little finger abduction alone (single exercise) or the simultaneous repetition of index finger flexion and little finger abduction (simultaneous exercise). Each individual practiced the assigned exercise for 2-3 minutes to become familiar with the experimental setup. Then, repetition of each movement set at 0.5 Hz with a metronome was performed for 30 minutes. Control and self-triggered TMS and peripheral nerve stimulation were performed immediately after the exercise and repeated 10, 20, and 30 minutes thereafter. Intervals between the two experimental sessions (the single and the simultaneous exercise sessions) were at least one week or longer. During the exercise, participants were asked to make a brisk movement of short duration after each beat of the metronome and then completely relax their right hand until the next beat. Since the different EMG activities of ADM during 30 minutes between single and simultaneous exercise could potentially affect control and self-triggered MEPs, we observed the EMG activity with online monitoring. Continuous visual EMG feedback was obtained to ensure each

EMG burst of less than 300 ms. For off-line measurements, EMG activities of ADM during 30 minutes of exercise were recorded. The average rectified area of each EMG activity was measured using the LabVIEW program (Figure 1). At the end of each experiment, each participant's feeling of the degree of attention, difficulty, and fatigability while performing each exercise was assessed with a questionnaire that had a five-point rating scale from 0 to 100 (0, the least and 100, the most; Figure 2).

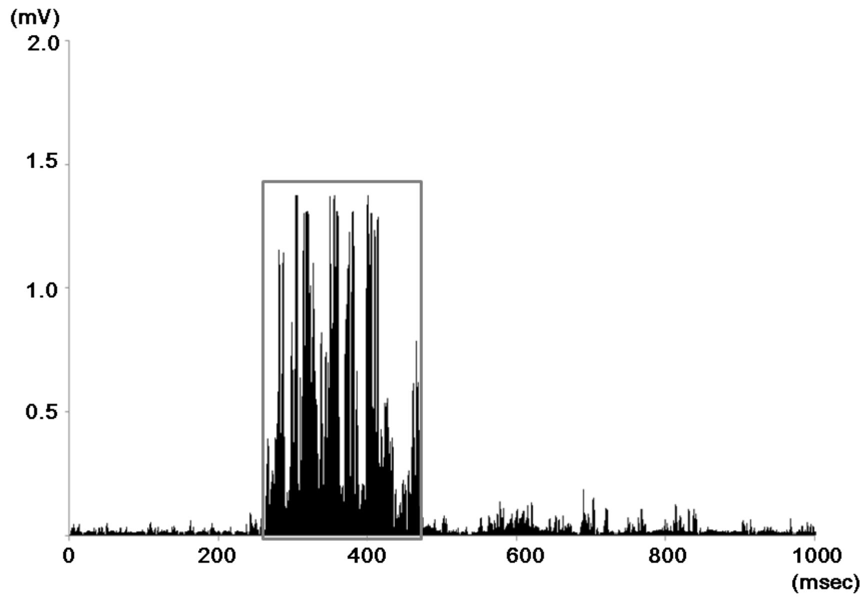


Figure 1. An example of rectified EMG activity of ADM during exercise. Area of EMG activity (black color in the box) was measured.

5. Statistical analysis

Data were expressed as means ± SEMs. Repeated-measures analysis of variance (ANOVA) was used to determine the effect of time intervals after exercise (0, 10, 20, and 30 minutes), exercise types (single and simultaneous), and participant groups (non-musicians and professional pianists) on normalized MEP amplitude of ADM.

A paired t-test was conducted to compare the normalized MEPs at each interval between the two exercise sessions. I also performed a repeated-measures ANOVA to determine the effect of exercise on normalized values of F waves (amplitudes and persistence) and CMAP amplitude, with the time and the exercise type as within-subject factors. The degree of attention, difficulty, and fatigability related to each exercise were compared between the two exercises using a paired t-test. *P*-values of <0.05 were regarded as significant.

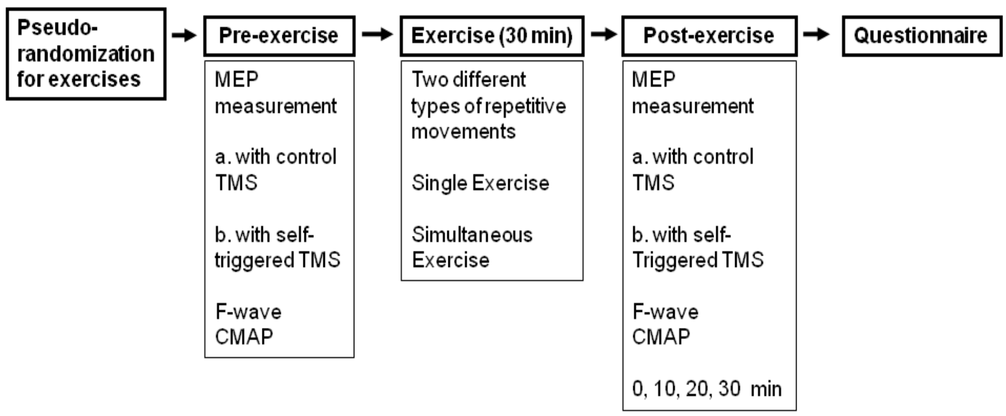


Figure 2. Overview of study protocol. Volunteers participated in two sets of exercises after pseudo-randomization with more than 1-week intervals between the two experiments.

III. RESULTS

1. Experiments with healthy non-musicians

Data from 13 (seven men and six women, age 19-26 years, mean age 22.8±1.8 years) healthy non-musicians were analyzed. The remaining five non-musicians were excluded because their RMTs (three participants) or 140% RMTs (two participants) were different between the two experiments, which could bias the results.

A. Transcranial magnetic stimulation

Before exercise, mean RMT, control MEP, and self-triggered MEP amplitudes were comparable between the single and simultaneous exercise session (RMT, 62.8±1.9% and 63.8±2.1%; control MEP, 1.6±0.3 mV and 1.5±0.2 mV; self-triggered MEP, 1.3±0.2mV and 1.2±0.2 mV, respectively). As previously reported, MEPs were suppressed during index finger flexion at 3-

ms intervals between EMG onset of FDS and TMS for both exercise sessions ($p<0.05$, respectively). The degree of suppression prior to the exercise was also comparable between single and simultaneous exercise sessions (82.9±3.3% and 85.8±6.3%, respectively, $p=0.69$)._

Control MEPs of ADM were slightly increased immediately after exercise in both exercise sessions, compared to those before exercise (Figure 3A). However, a repeated measures ANOVA showed that time (d.f. = 4, F=1.56, $p>0.10$), exercise type (d.f. =1, F=0.70, $p>0.40$) and their interaction (d.f. =4, F=0.60, $p>0.60$) did not have significant effects. In contrast, self-triggered MEPs of ADM were significantly enhanced after exercise in both exercise sessions, compared to those before exercise, but the amount of MEP enhancement was significantly greater and lasted longer after simultaneous exercise than single exercise (Figure 3B). Repeated measures ANOVA revealed that time (d.f . =4, F=4.66, $p<0.005$), exercise type (d.f. =1, F=5.10, $p<0.05$), and their interaction (d.f.=4, F=3.42, $p<0.02$) had significant effects. Individual analysis using a paired t-test revealed a significant difference in self-triggered MEPs between two exercise sessions at 0 and 20 minutes after exercise ($p<0.05$) and also showed a trend of difference at 30 minutes after exercise ($p<0.1$).

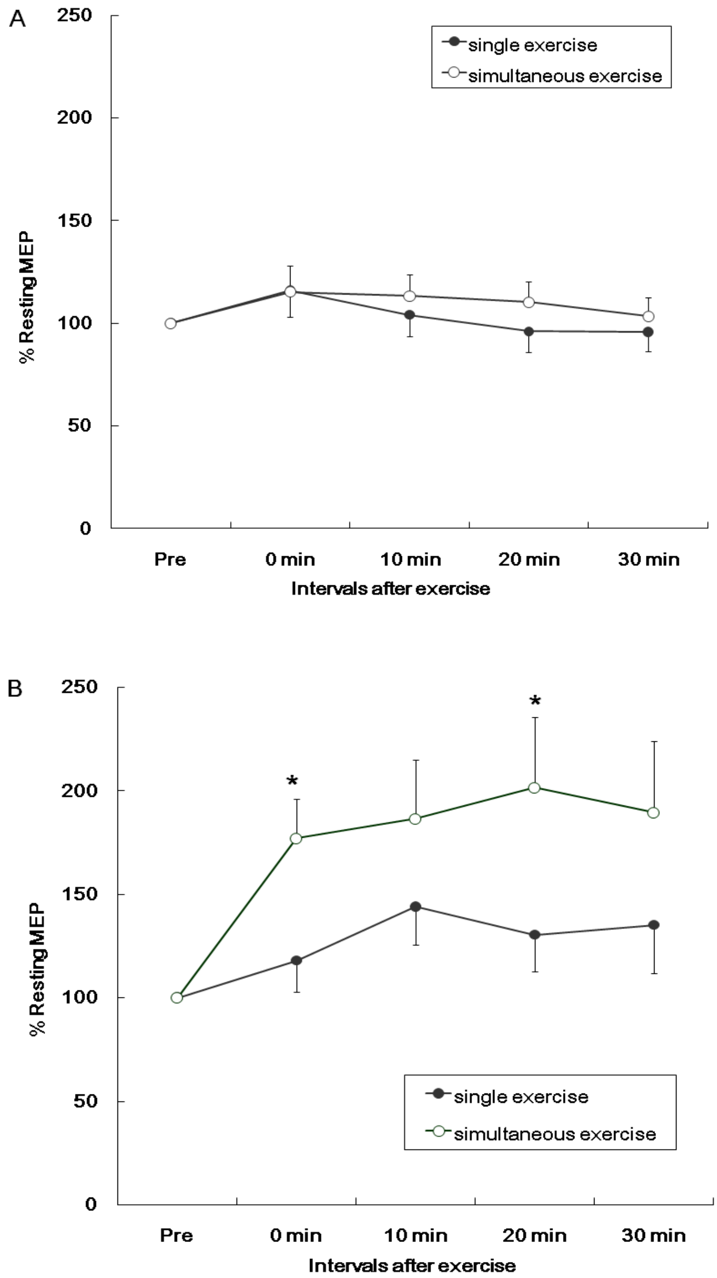

A
B
% Resting MEP
250
200
150
100
50
0
Pre
0 min
10 min
20 min
30 min
Intervals after exercise
single exercise
simultaneous exercise
*
*

Figure 3. Transcranial magnetic stimulation in non-musicians. A. Normalized MEP amplitudes of the abductor digiti minimi (ADM) in a control TMS session were compared between the two exercise sessions, before and after short-term exercise. The normalized control MEPs of ADM were slightly increased just after exercise in both the simultaneous and single exercise sessions. B. Normalized MEP amplitudes of the ADM in the self-triggered TMS session were compared between the two exercise sessions. Self-triggered MEPs of ADM at each time normalized to self-triggered MEPs before exercise were enhanced after exercise in both the simultaneous and single exercise session, but the enhancement was significantly stronger and lasted longer after the simultaneous exercise session. * indicates statistical significance (paired t-test, $p<0.02$).

B. Peripheral nerve stimulation

Of 13 participants, one was excluded as an outlier for F-wave analysis because the value of the amplitude was beyond the 3-SD of the mean. Spinal and peripheral excitability, measured by the amplitude and persistence of normalized F waves and normalized CMAP were not different between the two exercise sessions (Figure 4). Repeated-measures ANOVA did not reveal any significant effect for time (amplitude, d.f. =4, F=2.03, p=0.11; persistence, d.f. =1.68, F=1.15, p=0.33), for exercise type (amplitude, d.f.=1, F=2.30, p=0.16; persistence, d.f.=1, F=0.87, p=0.37), or for their interaction (amplitude, d.f. =4, F=0.69, p=0.61; persistence, d.f. =2.02, F=0.96, p=0.40) in the F wave measurements. Repeated-measures ANOVA showed no significant effect for time (d.f. =4, F=1.66, p>0.10), for exercise type (d.f. =1, F=0.007, p>0.90), or for their interaction (d.f. =4, F=1.26, p>0.90) in the CMAP measurements.

C. EMG activities and the participants' feeling during exercise

The rectified areas of ADM EMG activities during the 30-minute exercise were comparable between single and simultaneous exercise sessions (56.7±10.3 mV.msec, and 75.8±22.94 mV.msec, p=0.47). In addition, all participants described that their feelings of the degree of attention, difficulty, and fatigability between the two exercise sessions were comparable (Figure 5, all p>0.3)

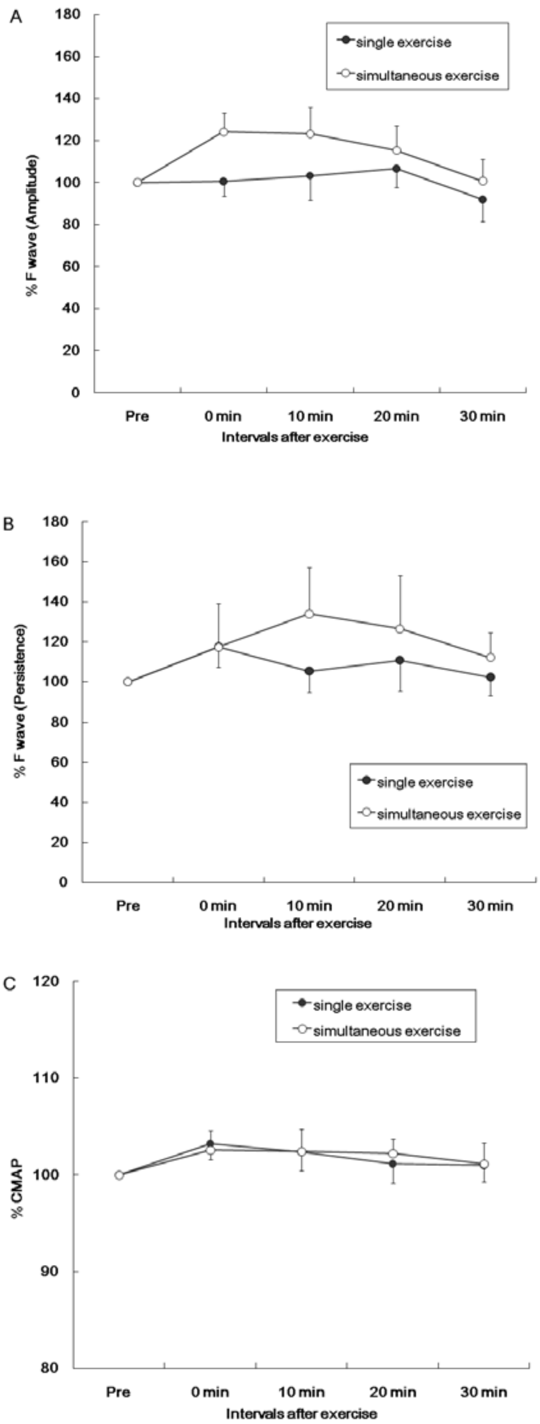

A
B
C
% F wave (Amplitude)
% F wave (Persistence)
% CMAP
single exercise
simultaneous exercise
Pre
0 min
10 min
20 min
30 min
Intervals after exercise

Figure 4. Peripheral nerve stimulation in non-musicians. Average peak-to-peak amplitude and persistence of F wave and average CMAP amplitude were normalized to the values before exercise. The normalized values were not significantly different for time, for exercise type, and for their interaction between the two exercise sessions. A. Normalized F-wave amplitudes of the abductor digiti minimi (ADM) between two exercise sessions. B. Normalized F-wave persistence. C. Normalized CMAPs.

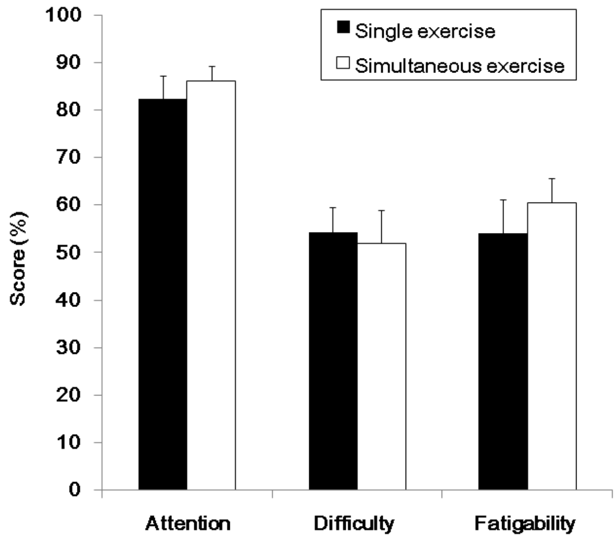


Figure 5. The responses of non-musicians to the questionnaire about the two exercises. There was no significant difference in degree of attention, difficulty, and fatigability between the two exercise sessions (paired t-test, all p>0.3).

2. Experiments with professional pianists

The same experiments were conducted in professional pianists to see the effect of the repetitive exercise of different finger movements on surround inhibition in these people. I analyzed the data of 13 (1 man and 12 women, age 19-25 years, mean age 21.9±1.8 years) healthy professional pianists. The participants began piano playing at a mean age of 5.4±1.5 years (age 3-8 years) and had played piano for a mean period of 16.8±3.0 years. All participants have practiced piano playing regularly for longer than 2 hours per day, with an average of 3.4±1.3 hours per day (range 2-6 hours per day).

A. Transcranial magnetic stimulation

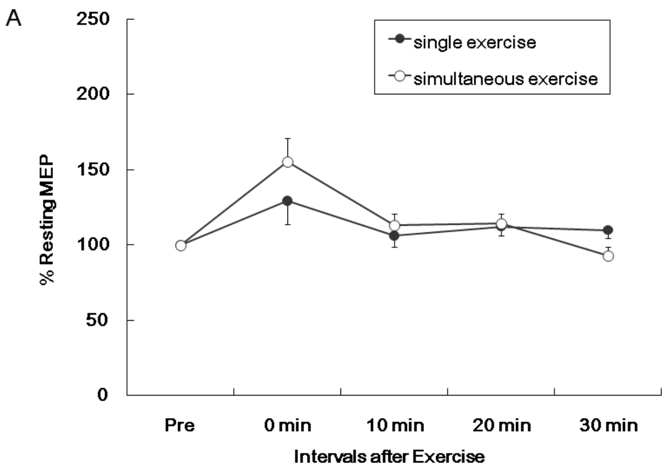
Before exercise, mean RMT, control MEP, and self-triggered MEP amplitudes were comparable between the single and simultaneous exercise sessions (RMT, 54.8±1.7% and 54.4±2.0%; control MEP, 2.1±0.3 mV and 2.0±0.4 mV; self-triggered MEP, 1.7±0.3 mV and 1.6±0.3 mV, respectively). As shown in healthy non-musicians, MEPs of ADM were suppressed during index finger flexion at 3-ms intervals between EMG onset of FDS and TMS for both exercise sessions ($p<0.01$, respectively). The degree of suppression was comparable between single and simultaneous exercise sessions (78.8±5.3% and

80.9±3.1%, respectively, p=0.58).

Control MEPs of ADM were increased immediately after exercise and returned to the baseline 10 minutes after exercise in both exercise sessions, compared to those before exercise (Figure 6A). A repeated-measures ANOVA revealed a significant difference for time (d.f. = 4, F=10.63, p=0.001), but neither for exercise type (d.f. =1, F=0.21, p>0.65) nor for their interaction (d.f. = 4, F=2.8, p>0.08). Self-triggered MEPs of ADM were significantly enhanced after exercise in both exercise sessions (Figure 6B). Similar to control MEPs, a repeated-measures ANOVA revealed only a significant effect for time (d.f. =4, F=15.68, p<0.001), but neither for exercise type (d.f. =1, F=0.33, p>0.58), nor for their interaction (d.f. =4, F=1.08, p>0.38).

B. Peripheral nerve stimulation

Of 13 pianists, we excluded two participants as outliers for F-wave analysis because either the value of the amplitude or the persistence was beyond 3 SD of the mean values. Like the healthy non-musicians, spinal and peripheral excitability measurements were comparable between two exercise sessions (Figure 7). A repeated-measures ANOVA did not reveal any significant effect for time (amplitude, d.f.=4, F=1.52, p>0.21; persistence, d.f.=4, F=2.04, p=0.11), for exercise type (amplitude, d.f.=1, F=2.82, p>0.12; persistence, d.f.=1, F=0.01, p=0.94), and for their interaction (amplitude, d.f. =4, F=1.54, p=0.21; persistence, d.f.=4, F=0.99, p>0.42) in the F wave measurements. In CMAP measurements, a repeated-measures ANOVA also showed no significant effect for time (d.f. =4, F=0.83, p>0.50), for exercise type (d.f. =1, F=1.30, p>0.28), and for their interaction (d.f. =4, F=1.80, p>0.10).



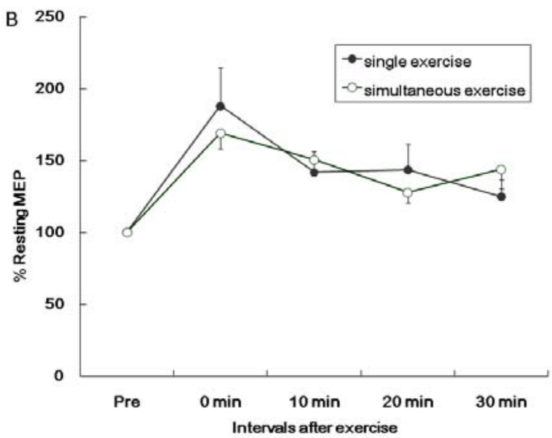


Figure 6. Transcranial magnetic stimulation in pianists. A. Normalized MEP amplitudes of the abductor digiti minimi (ADM) in the control TMS session were compared between the two exercise sessions, before and after short-term exercise. B. Normalized MEP amplitudes of the ADM in the self-triggered TMS session were compared between the two exercise sessions. The normalized control MEPs of ADM and self-triggered MEPs, respectively, were increased after exercise with a similar pattern in both the simultaneous and single exercise sessions.

C. EMG activities and the participants' feeling during exercise

The rectified areas of ADM EMG activities during the 30-minute exercise were comparable between single and simultaneous exercise sessions (52.4±5.4 mV.msec and 49.6±5.4 mV.msec, respectively, $p>0.5$). All pianists described no difference in the degree of attention, but they felt greater difficulty and fatigue during the simultaneous exercise session, compared to the single exercise ($p<0.05$, $p<0.01$, respectively, Figure 8).

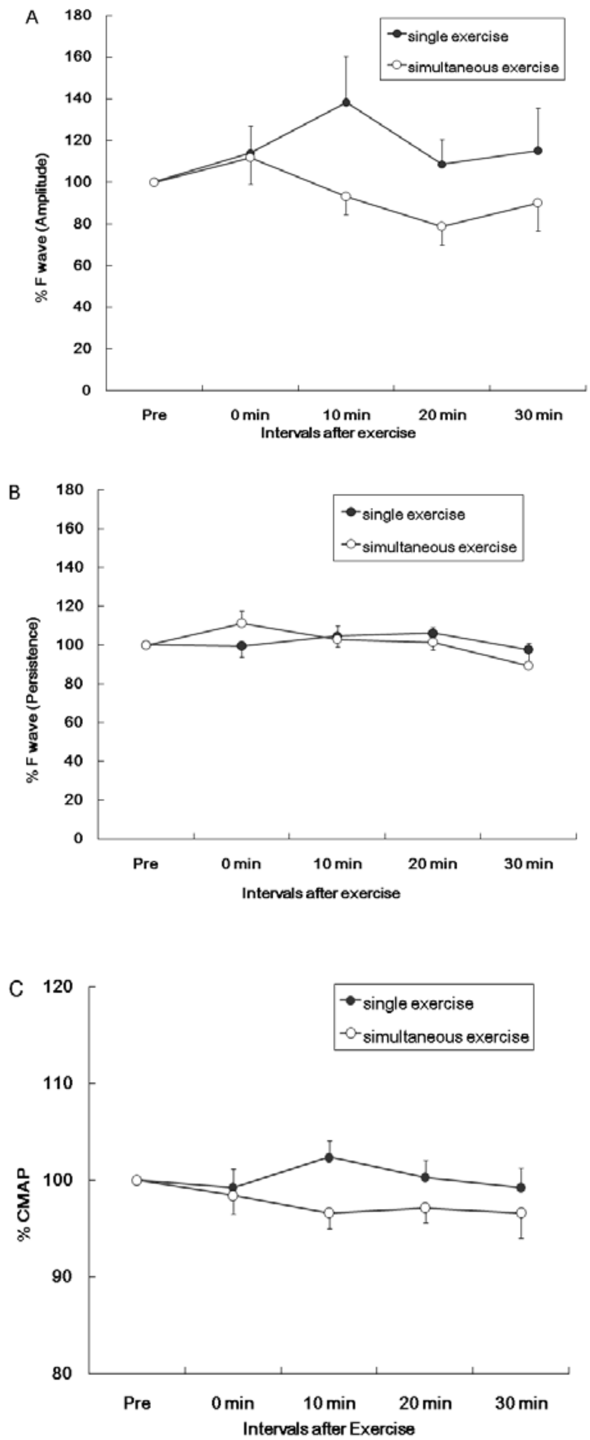

A
180
160
140
120
100
80
60
40
20
0
% F wave (Amplitude)
single exercise
simultaneous exercise
Pre
0 min
10 min
20 min
30 min
Intervals after exercise
B
180
160
140
120
100
80
60
40
20
0
% F wave (Persistence)
single exercise
simultaneous exercise
Pre
0 min
10 min
20 min
30 min
Intervals after exercise
C
120
110
100
90
80
% CMAP
single exercise
simultaneous exercise
Pre
0 min
10 min
20 min
30 min
Intervals after Exercise

Figure 7. Peripheral nerve stimulations in pianists. Average peak-to-peak amplitude and persistence of F wave and average CMAP amplitude were normalized to the values before exercise. The normalized values were not significantly different for time, for exercise type, or for their interaction between the two exercise sessions. A. Normalized F-wave amplitudes of the abductor digiti minimi (ADM) between two exercise sessions. B. Normalized F-wave persistence. C. Normalized CMAPs.

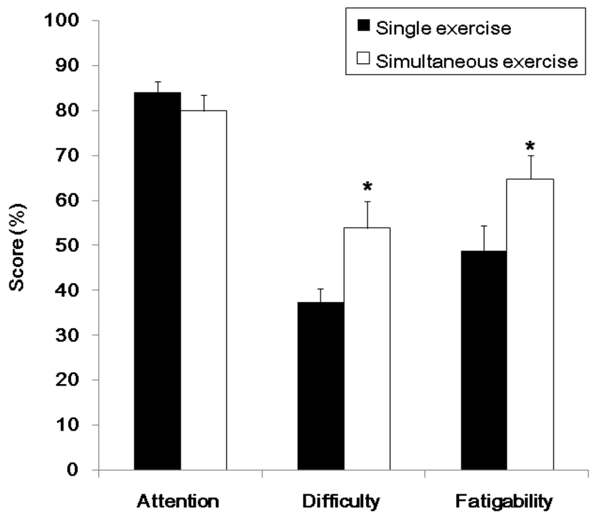
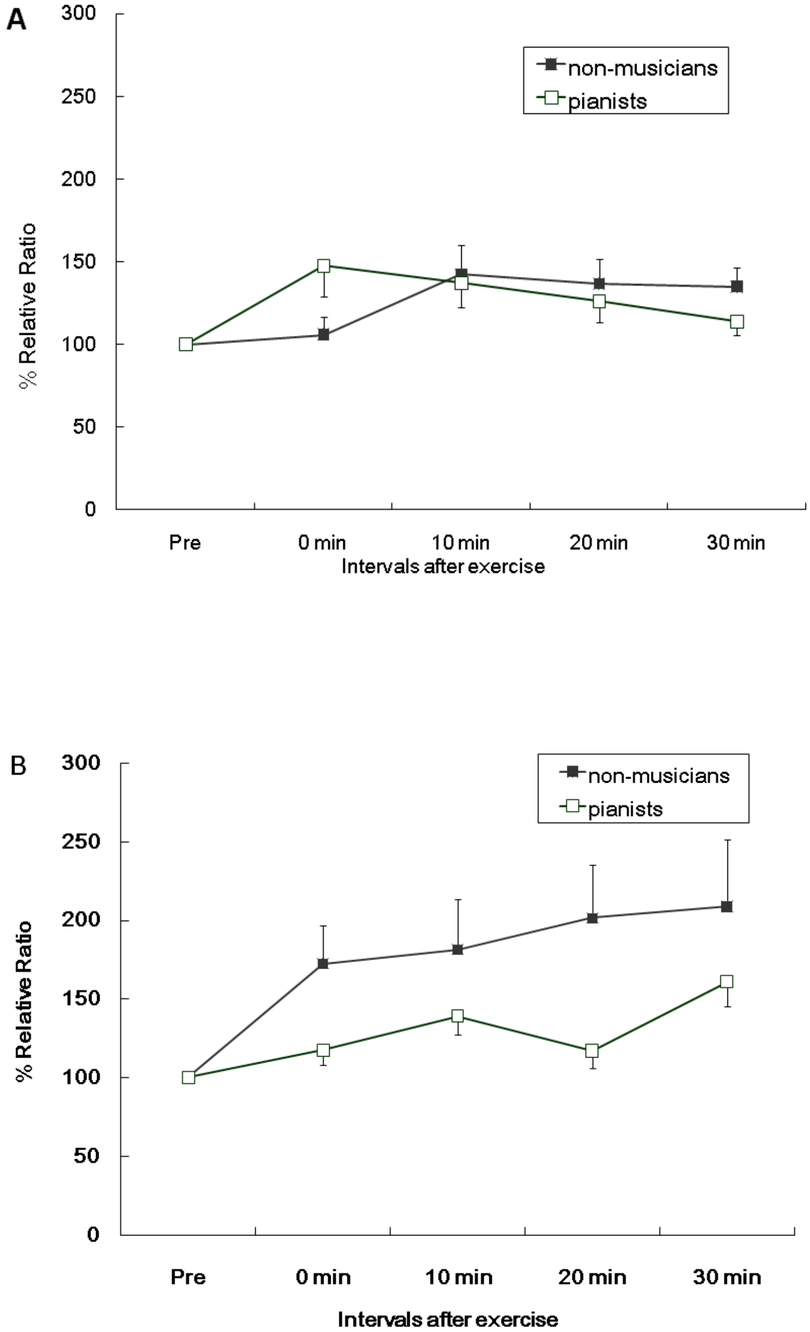


Figure 8. The responses of pianists to the questionnaire about the two exercises. All pianists described that there was no difference of the degree of attention, but they felt more difficulty and fatigue in simultaneous exercise session. *, ** indicates statistical significance (paired t-test, *p<0.05,

**p<0.01, respectively).

3. Comparison of the results between healthy non-musicians and professional pianists

For comparison of the results between healthy non-musicians and professional pianists, we used relative ratio (expressed as the percent) of ADM MEPs in self-triggered sessions normalized to those in control sessions. For single exercise sessions, a repeated-measures ANOVA revealed a significant effect for time (d.f. = 4, F=3.80, p=0.007) and for their interaction (d.f. = 4, F=2.59, p=0.042), but not for group (non-musician vs professional pianists; d.f= 1, F= 0.04, p= 0.85; Figure 9A). An individual analysis showed a significant tendency in the relative ratio between non-musicians and professional pianists at 0 minute after exercise (p=0.67), but did not show any significant difference at 10, 20, and 30 minutes (all p>0.2). For simultaneous exercise sessions, a repeated-measures ANOVA showed a significant effect for time (d.f. = 4, F=8.93, p<0.001), and tended to show an effect for group (d.f. = 1, F=3.10, p=0.09), but not for their interaction (d.f. = 4, F=2.10, p>0.10; Figure 9B).


A
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pianists
Pre
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non-musicians
pianists
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0 min
10 min
20 min
30 min
Intervals after exercise

Figure 9. Comparison of the change of surround inhibition between non-musicians and professional pianists. A. The change in relative ratio in the single exercise. The relative ratio of normalized MEP amplitude was calculated as [(normalized MEP amplitude in self-triggered TMS)/ (normalized MEP amplitude in control TMS) ×100] for each group. There was a main effect of time and a significant interaction between time and group. B. The change of relative ratio in the simultaneous exercise. There was a main effect of time, but no significant interaction between time and group. The mean value of the relative ratio was higher in the non-musician group after exercise, but showed a tendency for the main effect of group (p=0.09).

IV. DISCUSSION

This study demonstrated that surround inhibition in the human motor system could be altered by repetitive exercise of finger movements. This alteration was greater in the exercise of simultantous, unrelated finger movements, compared to that of single finger movements. Considering the previous observation that surround inhibition is impaired in patients with FHD,[6] this finding supports the idea that muscle overuse can lead to disturbed surround inhibition, which then could induce the development of dystonia. In contrast to healthy non-musicians, alterations in surround inhibition after both types of exercises were comparable in professional musicians, suggesting that the neurophysiological mechanisms regulating surround inhibition in professional musicians may be different from those in non-musicians, presumably due to prolonged practice of unrelated finger movements prior to this study.

Surround inhibition is proposed to be an essential mechanism to perform precise voluntary motor control. Surround inhibition is known to be related with individual phasic, fine finger movements that contribute to the generation of short intracortical inhibition (SICI),[19] which is $GABA_A$-mediated.[20, 21] This implies that surround inhibition may have an important role in movement initiation.[19] Surround inhibition was more obvious in fine finger movements with lower power levels,[22] suggesting that surround inhibition may be involved in spatial and temporal motor control with selective inhibition like spatiotemporal modulation for sensory input.[23] Thus, disturbed surround inhibition may make induce difficulties in voluntary movement control, and produce dystonia as shown in previous studies.[4, 6] The present results provide evidences to support the relationship between repetitive finger movements (i.e., muscle overuse) and disturbances in surround inhibition.

It is well-known that repetitive exercises may produce alterations in synaptic plasticity[24] by inducing long-term potentiation/long-term depression (LTP/LTD).[25] The dynamic change of synaptic plasticity usually accompanies the improvement of motor performance over time.[26] Cortical plasticity can even be develop by a short-term exercise, which is called rapid plasticity[27, 28] and that is known to be related to a GABAergic mechanism.[29] The change of intracortical inhibition can occur even during very short-term practice of around 5-minute period and the degree of the change is variable with different tasks.[30] Neurons in the cortical zone for motor control have vertical and horizontal connections,[31] which makes it possible to control movement precisely through intracortical inhibition and facilitation.[32] In addition to intracortical inhibition, facilitation is also associated with motor control, induction of LTP/LTD, and synaptic plasticity. Intracortical inhibition and facilitation may play unique and independent roles in these physiological mechanisms. For example, excitatory plasticity affects neuronal input-output function, whereas inhibitory plasticity modulates the adjustment of gain and threshold in the hippocampus.[33] Although previous studies mainly dealt with SICI, surround inhibition also may have a role in cortical plasticity. It is likely that there are common features shared with other intracortical inhibitory mechanisms, because of the association with SICI and surround inhibition.[1, 19, 34] Previous studies have shown that surround inhibition is also involved in cortical plasticity. In animal experiments, sensory or motor cortical reorganization relied on loss of surround inhibition.[35, 36] In patients with stroke, the favored direction of motor cortical reorganization occurred towards the less inhibited area before rehabilitation.[37] New tasks make a functional linkage through synaptogenesis within the primary motor cortex. This functional network enables more effective synergistic muscle recruitment. These muscle recruitments lead to improved behavioral performance after several days of practice.[38] As normal plasticity induces normal voluntary movements, abnormal plasticity could cause abnormal involuntary movements. Long-term perturbation of surround inhibition could induce abnormal plastic organization in the motor cortex, subsequently leading to the development of disordered motor control, such as dystonia.

No differences were observed regarding the effect of the two exercise regimens on surround inhibition in professional pianists. The results of pianists were expected initially to be similar to those of non-musicians, i.e., simultaneous exercise could reduce surround inhibition more effectively than a single exercise. However, the changes in ADM MEPs in the self-triggered TMS session were similar between the two exercises sessions in professional pianists, unlike to non-musicians. It is well known that the brains of professional musicians are anatomically and physiologically different from those of non-musicians brain.[14, 39-43] Magnetoencephalography or functional magnetic resonance imaging (fMRI) showed increased somatosensory cortical area[39] and more focused, increased activation of the motor cortex in musicians.[42] Voxel-based morphometry in MRI revealed increased gray matter volume of the left precentral gyrus, left Heschl's gyrus, and right superior parietal cortex.[40] Furthermore, pianists had enhanced perceptual abilities concurrent with the duration of practice that was not training-specific, which suggests that pianists have a greater capacity for plastic reorganization, implying improved learning abilities.[43, 44] These differences might explain the differences in exercise-induced changes of surround inhibition between professional pianists and non-musicians as observed in this study.

Neurophysiological mechanisms regulating surround inhibition in professional musicians might be different from those in non-musicians, presumably because the professional musicians already have sufficient plastic changes in their motor cortex as a result of prolonged practice of complex finger movements since early childhood prior to this study. In learning new complex movements, as muscles became synchronized, functional connectivity rapidly increases.[45] Extensive daily exercise might increase the integration of cortical representation for muscles, making it possible to learn new movements faster.[45] It seems that musicians may have the ability to learn new tasks faster and there may be a susceptible period of motor learning in childhood.[46] Musicians in the present study showed that they learned new task quickly and performed better, compared to non-musicians. In addition, musicians who started to learn musical instruments before 7 years of age showed better performance than those who started after 7 years of age, although they had similar levels of training and the same duration of formal training.[46] From these previous studies, it was postulated that the alteration of surround inhibition might be sufficiently present in musicians prior to this experiment. The studies showing that prior motor learning reduces subsequent induction of motor cortical plasticity support this assumption.[25, 47] In addition, other possibility could be that the amount of practice was too small to cause any alteration of surround inhibition in professional musicians. Musicians are considered to control individual finger movements very well with great accuracy and independence.[22, 48] Musicians showed increased slopes of both the corticospinal (input-output curve) and intracortical (SICI) input-output relationships, compared to non-musicians, meaning that any small modulation in TMS intensity (either a single or conditioning pulse) would lead to a greater proportional change. SICI, associated with surround inhibition, showed quick recruitment with only a small change in conditioning stimuli and a stronger effect in musicians.[43] These observations suggest that at higher stimulus, greater inhibition may operate to effectively block unwanted expansion of activation.[43] These findings could also be consistent with more focused motor cortical activation during movement in professional musicians than non-musicians.[42, 43, 49-51] Since surround inhibition is closely related to these selective movements, surround inhibition in musicians may be less vulnerable to be altered by short-term exercises performed in this study, compared to non-musicians. Much longer periods of practice may be needed to disturb surround inhibition in professional musicians than non-musicians.

From the results with healthy non-musicians and healthy professional musicians in our study, it is possible that surround inhibition might return to the pre-practice state or even become strengthened if the healthy non-musicians would practice simultaneous exercise over a very long time period. Cortical excitability and plasticity showed dynamic changes in different phases of motor learning, and parameters for LTP/LTD-like plasticity returned to the pre-practice level in healthy participants.[26] Dystonia might occur only in the case that the altered surround inhibition would persist over long time periods, because the altered surround inhibition could cause maladaptive changes in the sensorimotor cortex (Figure 10). However, these assumptions require further investigation.

The degree of attention was not different between the two exercise sessions in both participant groups, suggesting that attention did not bias our results. Degree of difficulty and fatigability were not different in non-musicians, but were different in pianists. Pianists described that single exercise was less difficult and produced less fatigue than simultaneous exercise. The degree of difficulty and fatigability for simultaneous exercise were comparable between non-musicians and pianists, respectively, but those for single exercise were lower in pianists than in non-musicians. We assume that the difference in the degree of difficulty (or fatigability) did not affect the results in pianists, because there was no difference of control MEP and self-triggered MEP, respectively, between single and simultaneous exercise in pianists.

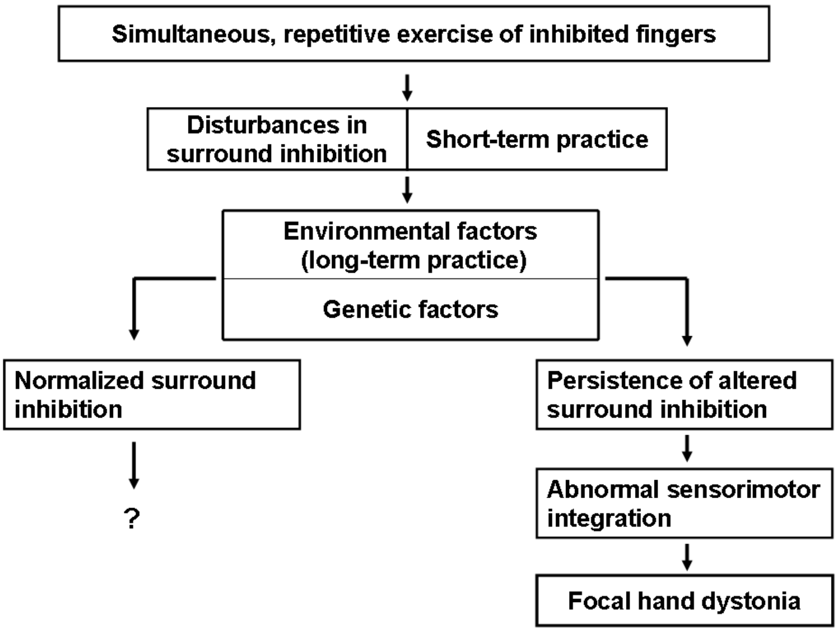

Figure 10. Schematic diagram of a possible pathogenesis of focal hand dystonia

Usually the difficulty of the task is related to its complexity. It seems that a complex task may increase or decrease cortical excitability in non-musicians,[52, 53] which was postulated to be due to the duration and task type,[53] whereas a complex task may have no influence on cortical excitability in musicians.[51] In one fMRI study, musicians did not show any differential activation between simple and complex motor tasks, whereas non-musicians showed additional activation (pre-supplementary motor area and the dorsal part of the dorsal premotor cortex) in complex motor tasks, suggesting the re-organization of motor system in musicians.[51] Fatigue is correlated with the reduction of cortical excitability showing decreased control MEP[54-56] and reduced SICI.[54, 55]; but our study did not show this trend. Pianists reported that they felt more fatigue in simultaneous exercise, but the normalized control MEP after exercise was higher than in the single exercise and the normalized self-triggered MEP immediately after exercise was lower than in the single exercise.

Some limitations should be considered when interpreting our results. We investigated the effect of short-term exercise, but focal hand dystonia usually develops following a long-term period of excessive repetitive activity in professionals. The effect of long-term exercise should be investigated in the future. We selected only participants who showed a sufficient surround inhibition before performing exercises, which might produce a subject selection bias. We did not consider the degree of surround inhibition for the selection of participants. All but one pianists were female. Because musician's cramp is more common in men,[57] there is a possibility that the sex difference in the study population might affect the results.

V. CONCLUSION

The present results demonstrate that surround inhibition in the motor system is reduced after repetitive exercise of simultaneous, unrelated finger movements, compared to that of single finger movements. This finding provides neurophysiological evidence to support the contribution of muscle overuse to the development of task-specific dystonia, considering the previous observation that surround inhibition is impaired in patients with FHD.[6] This study also shows that the effect of exercise types on surround inhibition is different between non-musicians and professional musicians, and supports the hypothesis that neurophysiological mechanisms regulating surround inhibition in professional musicians might be different from those in non-musicians, presumably due to the existence of chronic adaptive cortical plasticity in musicians induced by prolonged practice of complex finger movements prior to this study.

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< ABSTRACT>

반복운동 훈련에 의한 운동신경계내의 주위억제체계의 변화

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강 석윤

주위억제기전(surround inhibition)은 원하는 신경 활성을 적절하게 유도시키고 원하지 않는 신경 활성을 억제하는 생리학적 기전으로 운동 및 감각신경계의 활성조절에 매우 중요한 기전으로 알려져 있다. 자발적인 움직임을 정확히 수행하기 위해서는 주위억제기전이 필수적이기 때문에 불수의적인 움직임이 주된 문제인 이상운동질환에서는 질환의 병태 생리를 파악하기 위해서 주위억제기전의 특징을 이해해야만 한다. 특히, 근육긴장이상(dystonia)은 관련 근육의 불수의적인 수축이 지속되어 일어나는 이상운동질환으로

주위억제기전의 손상되어 있음이 보고되었다. 이러한 보고는 움직이지 않고자 하는 근육의 과도한 수축을 보이는 임상적 소견과 잘 일치한다. 근육긴장이상은 현재까지는 자세한 병리기전이나 뚜렷한 치료방침이 확립되어 있지 않아 대부분의 환자들이 이로 인한 일상생활을 제대로 수행하지 못하여 많은 고통을 받고 있다. 국소근육긴장이상(focal dystonia)은 근육긴장이상의 한 형태로 신체의 일정부위를 침범하며 얼굴부위나 손을 침범하는 경우가 흔한데, 특히, 국소손근육긴장이상(focal hand dystonia)은 주로 손에 증상이 나타나는 경우를 말한다. 국소손근육긴장이상은 아주 오랜 기간 동안 매우 과도하게 관련 근육의 반복적인 훈련과 관련이 있으며 흔히 과도하게 손가락 훈련을 오랫동안 해야 하는 음악가 등에서 발생빈도가 높다고 알려져 있다. 하지만, 정확한 발생기전은 제대로 알려져 있지 않다. 주위억제기전이 선택적인 움직임과 밀접한 관련이 있기 때문에 과도한 훈련이 주위억제기전 손상을 유발시키고 결국은 근육긴장이상이 발생할 것으로 추정하였다. 본 연구의 목적은 악기를 다뤄본 적이 없는 정상인과 어려서부터 피아노 훈련을 전문적으로 해 왔던 피아니스트를 대상으로 주위억제기전을 연구함으로써 이 기전에 대한 식견을 넓히고 자 함이다. 본 연구의 가설은 서로 관련이

없는 두 손가락(한쪽 손가락의 움직일 때 다른 손가락은 억제되어야 하는)의 과도한 반복적인 움직임이 주위억제기전 손상을 유발시킬 것이라고 가정하였다. 반복적인 움직임이 주위억제기전에 미치는 영향을 연구하기 위해서 서로 다른 두 종류 훈련 후에 나타나는 주위억제기전의 변화를 비교하였다. 이미 이전 연구에서 정상인의 경우 집게손가락의 단순굴곡운동 시, 연관이 없고 움직임이 없어야 하는 새끼 손가락에서 주위억제기전이 관찰되었다. 본 연구에서도 이전에 사용되었던 집게손가락의 얕은손가락굽힘근(flexor digitorum superficialis)과 새끼손가락의 짧은새끼외전근(abductor digiti mini)과 경두개자기자극술을 이용하였다. 집게손가락만 외전을 시키는 훈련과 집게손가락 굴곡 시 새끼손가락을 동시에 외전 시키는 두 종류의 훈련을 하고 이 훈련 전후의 주위억제기전의 변화를 비교하였다. 음악가가 아닌 정상인의 경우에는 단기간 동시운동훈련 뒤에는 짧은새끼외전근의 운동유발전위는 훈련 전처럼 감소하지 않고 오히려 증가하였다. 이 효과는 새끼손가락만 외전 시킨 훈련보다 유의하게 증가되었고 또한 오랫동안 지속되었다. 이와 반대로 전문적인 피아니스트의 경우에는 두 훈련이 주위억제기전에 미치는 효과는 차이가 없었다. 본 연구는 근육의 과도한 사용이 주위억제기전을

손상시킨다는 것을 명백하게 보여 주었으며 이러한 결과는 주위억제기전변화가 국소근육긴장이상발생에 기여할 수 있다는 가설을 지지한다. 동시반복훈련이 음악가가 아닌 정상인과 전문적인 피아니스트간에 차이가 있었던 이유는 피아니스트의 경우에는 유년기부터 매우 오랜 기간 동안 손가락의 반복훈련을 해왔기 때문에 이로 인해서 주위억제기전을 조절하는 신경생리학적 기전이 악기를 다룬 적이 없는 정상인과는 다를 것으로 추정된다.

핵심 되는 말: 주위억제기전, 경두개자기자극술, 운동유발전위, 운동피질, 인간, 반복 운동, 비음악가, 음악가, 근육긴장이상