Effects of Motor Learning on Spinal Plasticity

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Effects of Motor Learning on Spinal Plasticity

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Abstract

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Once a skilled movement is thoroughly learned, it can be performed relatively automatically. The motor cortex is active when learning a new motor skill, but becomes less activated once the skill has become well-learned. Here, I hypothesize that learning a skilled movement is associated with more efficient use of subcortical motor circuits. Subcortical motor circuits can coordinate features of the intended movement such as the timing and patterns of activation of different muscles. The goal of this study is to determine whether learning a motor skill strengthens spinal interneuron circuits that facilitate the movement. Subjects learned to perform a movement consisting of alternating rhythmic wrist movements by hitting the targets that would appear alternatively in the right and left sides on a computer screen. Accuracy was monitored reaching the targets within the specific time window. Motor performance gains were observed significantly after three sessions. The motor performance showed a typical learning curve and was retained a week later. The co-contraction of wrist extensor in flexion period decreased significantly at the seventh session and that of wrist flexor in extension period decreased but not significantly. Reciprocal inhibition was assessed by stimulus triggered averaging of rectified EMG method. The reciprocal inhibition at the transitional period from the extension to the flexion increased significantly and increased at the mid-extension period but not significantly. The present study suggests that our motor task can be learned and training upper extremity to perform alternating movement enhances the strength of short latency inhibition of spinal

interneuron particularly at the phase of movement near transition to alternate phase and this enhancement of reciprocal inhibition can play a role in the facilitation of alternating muscle activity.

Key words : motor learning, reciprocal inhibition, spinal plasticity

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I. INTRODUCTION

1. Background

Human beings acquire new motor skills throughout their lives. Initially, performing a new skilled movement is effortful and clumsy, but with practice, the performance becomes accurate and the movement is carried out fairly automatically. For certain highly practiced, repetitive movements, the ability to perform the movement persists even when it has not been employed for long periods of time. Riding a bicycle is the classic example of such a movement.

Many regions of the central nervous system are known to play a role in producing skilled movements, including motor and pre-motor areas of the cerebral cortex, the basal ganglia, the cerebellum, brainstem motor centers, and spinal interneuron circuits. These areas are all potential candidates for storing or executing the motor programs for highly learned skilled movements. There is some evidence, however, that as movements become over-learned, they require less involvement of the motor cortex. Studies using transcranial magnetic stimulation to map the motor cortical output maps to the muscles involved in learning a sequence of finger movements showed that maps became progressively larger as subjects learned the sequence, but once the movement had been learned, the maps returned to their baseline topography[1]. EEG studies

also found that the overall proportion of cortical neurons required for task performance became smaller as skills developed[2,3]. In addition, some imaging studies with PET[4] and functional MRI[5] showed activity in an increased number of cortical regions during the learning phase which became more focused after acquisition of motor skill.

As repetitive movements become highly learned, it is likely that the motor cortex learns to utilize subcortical motor circuits efficiently to carry out much of the moment-to-moment control of the movement. The motor cortex is known to project via the corticospinal tract onto spinal interneurons that exert effects on motor neurons. In primate studies, recordings from spinal interneurons have shown that they are active with an appropriate timing to contribute to the preparation and execution of normal voluntary movements[6]. In humans, voluntary movements are also associated with changes in the transmission through spinal interneurons. For example, at the onset of voluntary contraction, presynaptic inhibition of Ia afferents projecting to motoneurons of the contracting muscle is decreased, whereas presynaptic inhibition of Ia afferents to motoneurons of muscles not involved in the voluntary contraction is increased[7]. This decrease in presynaptic inhibition of Ia afferents persists unchanged after an ischemic blockade of group I afferents from contracting muscles[8], indicating a central origin for the modulation. Presynaptic inhibition of Ia afferents is mediated by GABAergic interneurons in the spinal cord. Similarly, during voluntary movements, the excitability of antagonistic motor neurons is controlled by central modulation of spinal inhibitory pathways for reciprocal inhibition[9]. Spinal interneurons mediating reciprocal inhibition have rhythmical firing during walking[10], bicycling[11], and cyclical human arm movement[12] in a manner appropriate for assisting these alternating movements. These findings illustrate how networks of interneurons in the spinal cord could be used by higher centers to coordinate patterns of muscle activity in highly-learned movements.

The spinal circuits producing reciprocal inhibition between antagonist motoneurons are excellent candidate circuits that might be strengthened during

learning of repetitive, alternating movements. The glycinergic Ia inhibitory interneurons, which mediate reciprocal inhibition between antagonistic motoneurons, are located in the spinal cord[13]. They receive input from Ia spindle afferents from the agonistic muscle and inhibit the motoneurons of the antagonistic muscle. It has been shown that Ia interneurons are excited by corticospinal, rubrospinal and vestibulospinal tracts[14,15]. Lundberg proposed that the Ia interneurons were activated in parallel with their corresponding motoneurons during voluntary movement. In humans, it seems likely that the corticospinal pathway transmits this parallel activation[9,16]. Corticospinal activation of Ia interneurons has also been reported in humans with transcranial magnetic stimulation (TMS). Thus the Ia inhibitory interneuron is likely to be one of the spinal interneurons capable of being utilized by the motor cortex to shape the intended movement.

Moreover, the strength of reciprocal inhibition can be modified, at least transiently, by sensory stimulation to enhance reciprocal inhibition between ankle flexor and extensor muscles[17]. In humans, it has been suggested that the resting strength of disynaptic reciprocal inhibition between ankle flexor and extensor muscles is influenced by the history of physical activity we perform[9,18]. There is also evidence from animal studies that transmission in the reciprocal inhibitory pathway could also be modified by using an operant conditioning protocol[13] and that the corticospinal tract probably controlled whether this plasticity in the spinal cord occurred[19]. During a repetitive alternating movement, enhancement of reciprocal inhibition at the transition between flexion and extension would be particularly beneficial for performing movement. Furthermore, the mutually inhibitory connections between Ia inhibitory interneurons subserving antagonist motor neuron pools[20] would allow self-sustaining oscillation of reciprocal activity.

Methods for assessing reciprocal inhibition between wrist flexors and extensors are well described and easily tolerated by most patients[10,21,22]. Dynamic modulation of reciprocal inhibition is likely to occur during rhythmic movements and to be phase dependent, as has been shown during locomotion in

studies in animals[23] and in humans[10]. In locomotion, the pattern of modulation produces the strongest reciprocal inhibition of extensor muscles at the beginning of the swing (flexor) phase, remaining somewhat increased throughout the remainder of the swing. Reciprocal inhibition of extensor muscles is depressed during the stance phase of walking, even to a greater extent that occurs with tonic contraction of extensor muscles[10].

Controlled wrist movements have been studied extensively in primates, primarily using unidirectional targeted ramp-and-hold movements. In humans, learning to perform targeted wrist movements produces changes in cortical maps at an early stage of learning[24]. Changes in the pattern of muscle activity occurring while subjects learned to perform targeted wrist flexion-extension task have not been as well described. However, in studies of targeted movements involving other arm muscles, such as those acting across the elbow joint, EMG showed a progressive decline in co-activation of synergistic muscles and in co-contraction of antagonist muscles as the task is learned[25,26]. These studies support the idea that learning leads to a more efficient and focused activation of the muscles needed to perform the task. In our study, we planned to use a task that combined accurate movement to a target with rhythmic alternation between wrist flexion and extension. Studies of rhythmic cycling movements of both arms have shown that H-reflex modulation occurs, similar to the locomotor pattern in the legs[27]. The motor task planned for this study combines elements of learning and rhythm that, in some ways, would be analogous to learning to walk.

2.Objectives

The goals of this study were first, to determine whether co-contraction declined after learning to perform a controlled , rhythmic, alternating wrist movement task, and secondly, to investigate changes in reciprocal inhibition activated by spinal circuits at stages of motor learning.

II. MATERIALS AND METHODS

1. General experimental conditions

Seventeen healthy volunteers (3 men and 14 women, aged 22-45 years) participated in the study. All subjects were right-handed (laterality index was 0.8 to 1.0) as measured by the Edinburgh Inventory[28]. They were free of wrist and neck pain and with no known history of medical problems. The protocol was approved by the Institutional Review Board in National Institute of Health and all subjects gave written, informed consent in accordance with the Declaration of Helsinki. The subjects were seated in a chair, with their forearm supported by a horizontal platform and strapped in a neutral (semi-prone) position. The hand was secured between two horizontal plates of a single-axis manipendulum mounted on a rotating shaft located coaxially with the axis of rotation of the wrists. The output of wrist movements appears as a cursor on the computer screen. The manipendulum was coupled to a brushless servometer (PMA23D, Pacific Scientific, IL, USA) that supplied a constant resistive torque of 0.5 N-m, ensuring a steady and smooth wrist movement. The hand and forearm were initially positioned at a neutral angle that produced no contraction in wrist flexors and extensors. This was designated as the starting position, corresponding to 0˚ of wrist displacement (Figure 1).

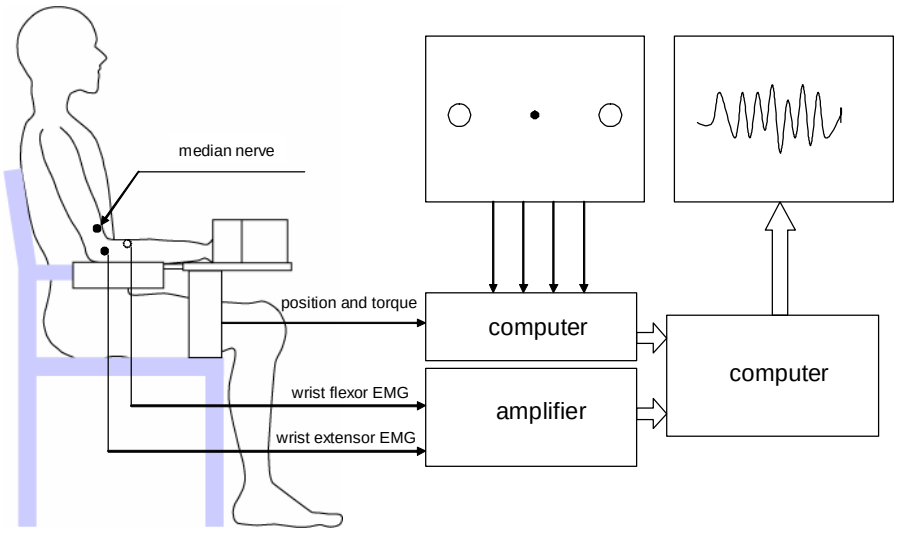


Figure 1. Experimental diagram.
Subjects were seated in a chair and facing the computer screen. The device allowed only wrist movements in the horizontal plane. Wrist movements were measured by transducer and appeared as a cursor on the screen. During the movements, the activities of wrist flexor and extensor were recorded simultaneously through surface electrodes.

2. Motor Task

Subjects were instructed to move their wrists rhythmically and steadily back and forth without overshooting movements to hit targets that would appear alternatively on the right and left sides of a computer screen by LabView computer programming at an interval of 2.5 seconds. There were two alternating sets of targets: large targets at 10 degrees wrist flexion and extension and small targets at 5 degrees wrist flexion and extension. The diameters of the targets were +/- 10 % of the target location angle. A cycle was composed of

small range of flexion and extension and large range of flexion and extension in order. A successful hit was defined as reaching the target without overshoot and staying within 10% of the target diameter. Subjects received visual feedback of the success of each trial by a color change of the target. If the trial is in success the yellow target turns green, but if not the yellow target turns red. The time interval for the cycle was 10 seconds. Each session had 360 trials and lasted for fifteen minutes every weekday until the task was learned. Learning was defined as achieving more than 20% increase in accuracy from the first session in two successive sessions. At this point subjects were asked to return for three more sessions performing the movement. After that, practice ceased for one week. A follow-up session after a week assessed whether the subject had retained the ability to perform the movement accurately.

3. Recording of muscle activity

Muscle activity of flexor and extensor carpi radialis was recorded simultaneously during the session by means of surface EMG with paired 10mm stainless steel disk electrodes using counterpoint EMG machine (Dantec, Allendale, NJ) with filter bandwidth of 10 Hz to 5kHz. The active electrode was placed at the muscle belly and the reference electrode was located at the distal 3cm. Waveforms were digitized for off-line analysis using Spike 2 software program(CED 1401 interface, Cambridge, UK) at a sampling rate 5kHz. EMG signals were amplified and rectified. The area of EMG (μV·ms) was hence obtained. The area of contraction was calculated by subtracting resting area. The ratio of wrist extensor activity in wrist flexion compared to wrist extensor activity in extension and flexion and the ratio of wrist flexor activity in wrist extension compared to total wrist flexor activity in wrist flexion and extension were measured for each cycle and averaged (Figure 2).

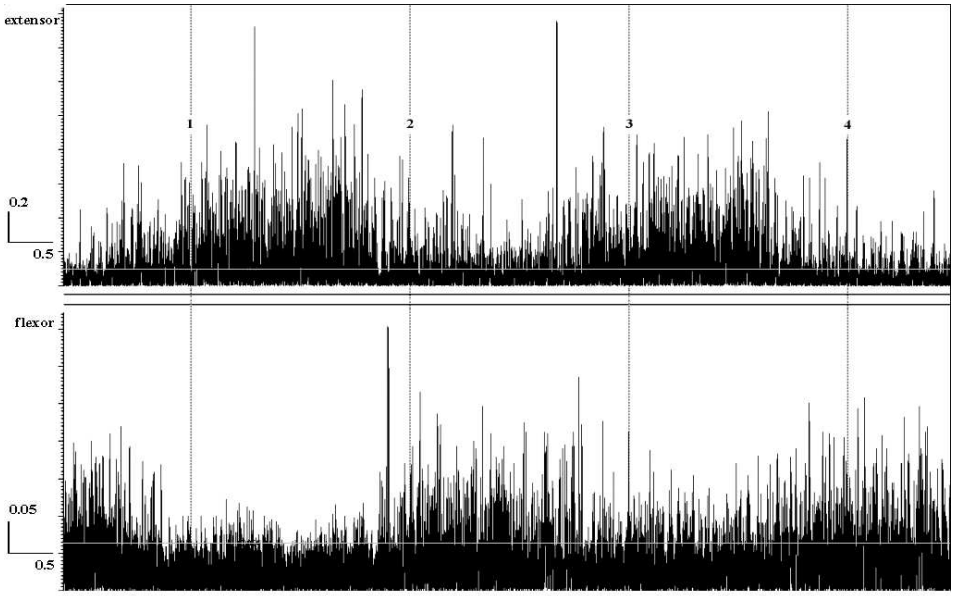
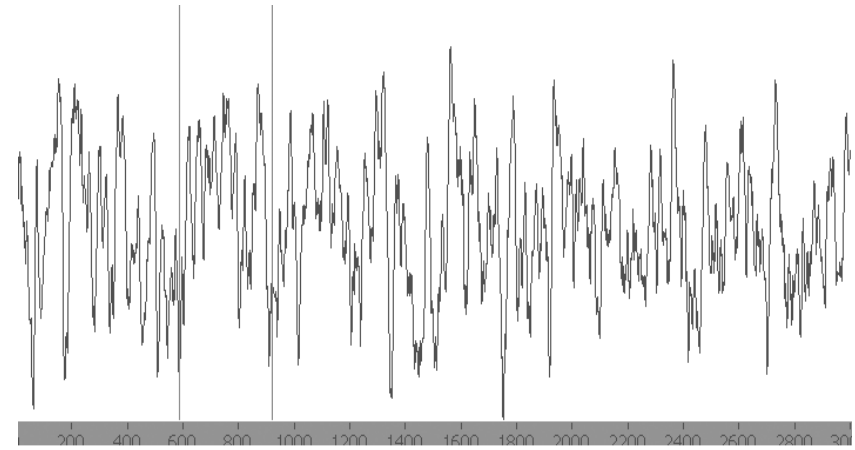


Figure 2. Muscle activity by surface electrode on Spike2 program.
The area of wrist extensor activity in flexion is the area under the curve between vertical line 2 and 3. The total extensor activity is the area under the curve between vertical line 1 and 3. The area of wrist flexor activity in extension is the area under the curve between vertical line 3 and 4. The total flexor activity is the area under the curve between vertical line 2 and 4. Every contracting area is calculated by subtracting resting area ,namely the area under the horizontal line, from each area. (x axis is time unit × seconds, y axis is electrical activity unit × 200μV)

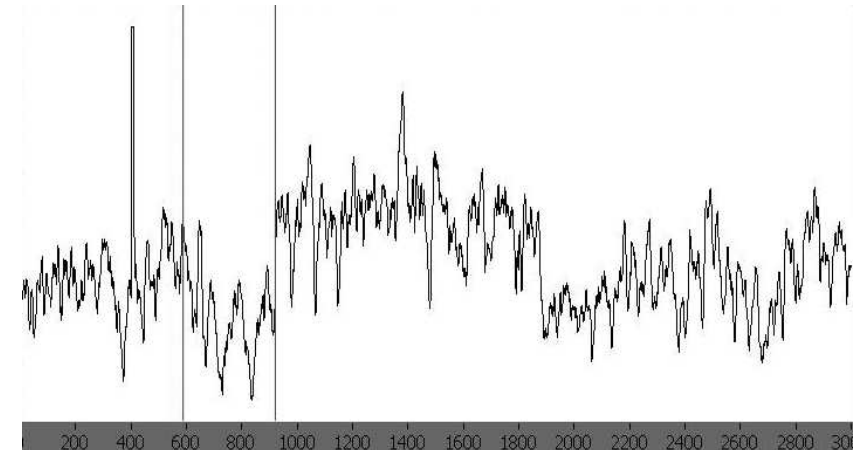
4. Reciprocal inhibition

Reciprocal inhibition was assessed using stimulus-triggered averaging of rectified EMG[10]. Bipolar stimulation of the median nerve at the elbow level, in which cathode was proximal, produces a depression of rectified EMG of wrist extensor carpi radialis. This inhibition is likely to be mediated mostly by Ia inhibitory interneurons projecting from wrist flexors to wrist extensors. For

median nerve stimuli, we used stimuli of less than 1.1 × MT[22]. Motor threshold (MT) was defined as a 100 μV response of the wrist flexor. The pulse duration was 1ms. The stimulus was delivered every other cycle at one of two points during the movement cycle, early flexion and mid-extension. Early flexion was defined as beginning 0.8 seconds before the start of movement in the flexion direction and mid-extension was defined as occurring 1.25 seconds after the start of extension movement. These times were defined in pilot studies to determine the onset of EMG activity prior to mechanical movement. No stimulation was delivered on every other cycle to allow a control recording of wrist extensor EMG activity without stimulation. Traces of wrist extensor EMG activity were extracted from the ongoing recordings by triggering a 300 ms window beginning 40 ms prior to the timing pulse driving the stimulator. 48 traces were averaged for the four conditions: early flexion with and without stimulation, and mid-extension with and without stimulation. The area of inhibition was calculated by subtracting the stimulus-triggered average from the average trace without stimulation with the same timing in the cycle. The time window for reciprocal inhibition was defined visually by cursor placement at the mid-extension with stimulation and this time window was applied all the four conditions(Figure 3). Waveforms were digitized for off-line analysis using custom software (Labview 6.1; National instruments, Austin, TX). The sampling rate was 10,000 per second. Reciprocal inhibition was assessed in this way during the first session of learning the task and after the movement had become well-learned, defined as having met criterion for success on the two previous sessions.



A



B

Figure 3. Measurement of reciprocal inhibition on LabView program.

A. wrist extensor activity without stimulation

B. wrist extensor activity with median nerve stimulation

The area of inhibition is subtracting B from A under the curve between two vertical line. (x axis is time unit × 0.1ms, y axis is electrical activity unit × 20μ V)

5. Statistical analysis

A repeated measure ANOVA was used to analyze the training effect on motor performance and to compare the learning effect on changes of wrist flexor in extension and wrist extensor in flexion at three time points (before learning, at learning, three sessions after learning). Paired t-test was used to compare the changes of reciprocal inhibition in wrist extensor before and after learning at the two time period, early-flexion and mid-extension each and to analyze whether the retention is obtained in motor performance.

III. RESULTS

1. Motor performance

Nine Subjects participated to determine whether the task was retained after learning. They showed a typical learning curve with daily practice sessions over about one week. On the first session, success rates varied among individual subjects (55%-65%). After three sessions, most subjects first achieved a success rate above 80%, and with additional practice sessions variability declined ($p < 0.001$). After a week without practice, the success rate was maintained, without a significant decline in success rate (Figure 4). From these results, we defined session 1 as before learning stage, session 4 as learning stage, and session 7 as after learning stage.

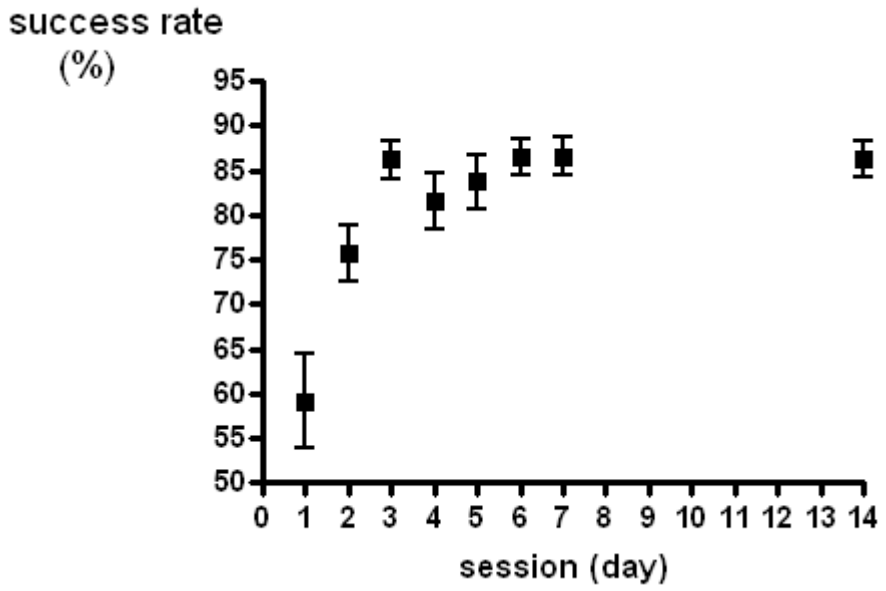


Figure 4. The change of success rates as session goes in motor performance. Success rates in a series of motor task training showed a typical learning curve. After three sessions, success rate reached plateau which was above 20% increase from that of session one. In a week without practice from day seven, learning was retained. Note the decrease of values of standard error as session goes. Values are means ± SE of the means.

2. Co-contraction on Surface EMG activity

Seventeen subjects participated in this experiment. The percents of wrist extensor activity in flexion decreased as subjects learned the task, 42.7 ± 1.1% at session one, 40.6 ± 1.4% at session four, and 39.8 ± 1.0% at session seven (Figure 5). Values at the 7th session was significantly different from those at the 1st session ($p < 0.05$). However, the changes in EMG activity was limited to the wrist extensor, the percents of wrist flexor activity in extension were 39.9 ± 1.2% at session one, 41.0 ± 1.6 at session four and 39.5 ± 1.5% at session seven and there were no significant differences among the stages of

learning. (Figure 6).

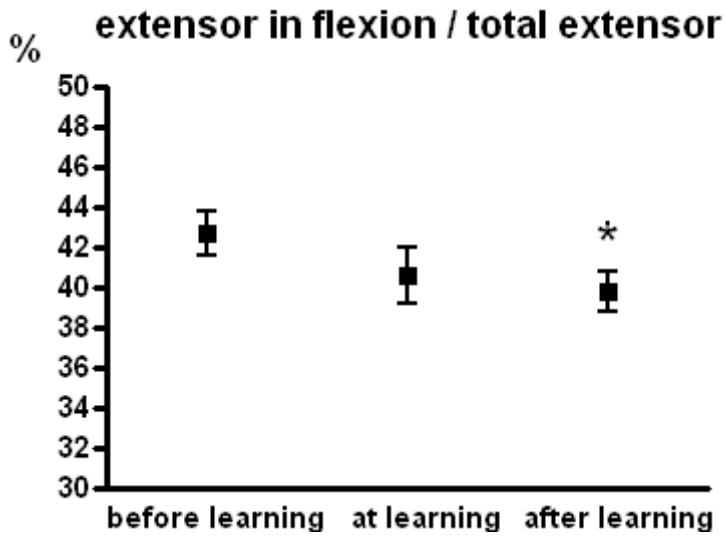


Figure 5. The percent of extensor activity in flexion compared to the extensor activity in a whole cycle.
The percent of wrist extensor activity in flexion decreased as subjects learned the task. The significant decrease was observed at the 7th session compared to the 1st session. Bars indicate standard errors of means (* $p < 0.05$).

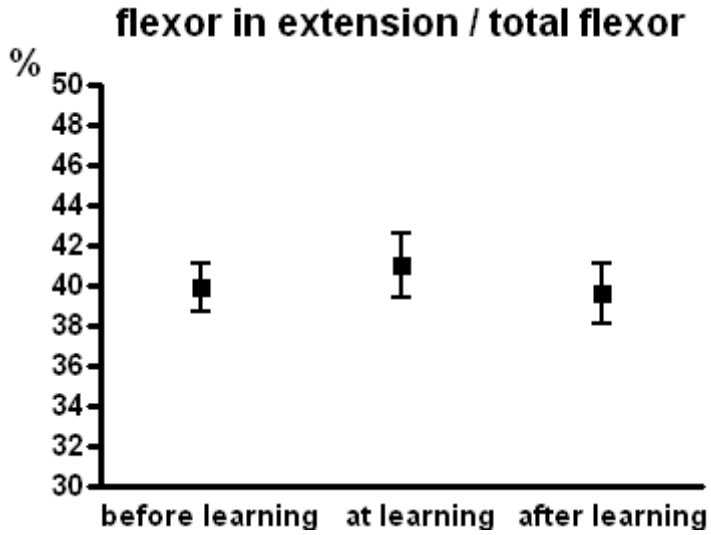


Figure 6. The percent of flexor activity in extension compared to the flexor activity in a whole cycle.
There was no significant difference in the percent of wrist flexor activity in extension among the measurements. Bars indicate standard errors of the means.

3. Reciprocal inhibition

Seven subjects participated in this experiment. The wrist extensor only was tested to determine whether the reciprocal inhibition is strengthened as an underlying mechanism for the decrease of wrist extensor activity in flexion after learning. The onset of inhibition was 22.0 ± 2 ms and the duration of inhibition was 27.6 ± 3 ms. The area of reciprocal inhibition at the early-flexion increased significantly by learning from 40.6 ± 10.9 μV·ms to 85.8 ± 23.7 μV·ms ($p < 0.05$). At the mid-extension of the cycle, the short latency of reciprocal inhibition increased but not significantly by learning from 55.4 ± 16.1 μV·ms to 68.5 ± 23.1 μV·ms (Figure 7). The stimulation intensity was 4.6 ± 2.6 mA at session one, 4.1 ± 1.9 mA at session seven and there was no significant difference .

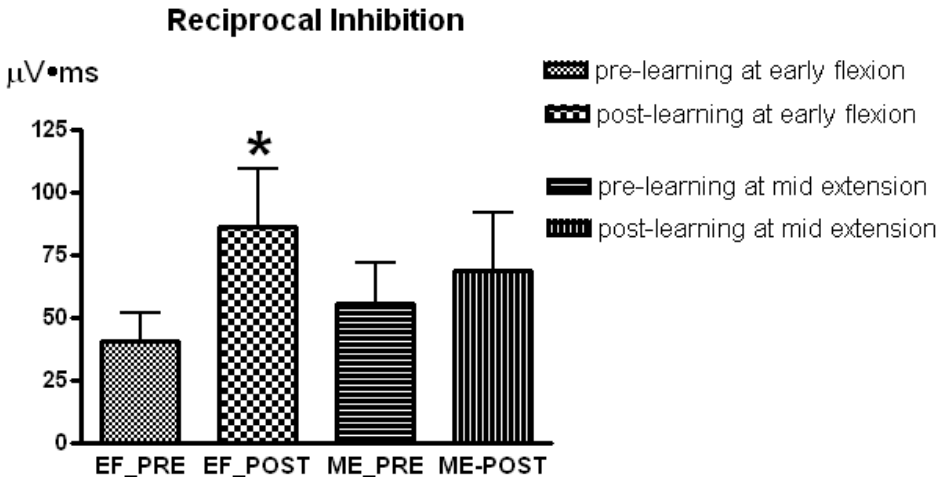


Figure 7. Short latency of reciprocal inhibition before and after learning at the early-flexion and the mid-extension of the dynamic modulation cycle.
The area of reciprocal inhibition at the early-flexion increased significantly.
At the mid-extension, the reciprocal inhibition increased but not significantly.
Bars indicate standard errors of the means (* $p < 0.05$).

IV. DISCUSSION

The present experiments have shown that our motor task can be learned and training upper extremity to perform alternating movement enhances the strength of short latency inhibition of spinal interneuron particularly at the phase of movement near transition to alternate phase and this enhancement of reciprocal inhibition can play a role in the decrease of co-contraction to facilitate alternating muscle activity.

In our motor task, motor performance showed a characteristic learning curve. After three or four sessions, the performance was maintained. One session was composed of ninety cycles therefore performance gains were obtained after around 300 cycles. Interestingly, this repetition is enough to learn the task and corresponds to other study[24]. The motor training elicits measurable performance gains as well as neurophysiological changes. However, there was

time difference between performance gains and neurophysiological changes. That may be different from other motor training paradigm[25,26]. That difference can be explained by decreased sensitivity of our motor task to show changes in surface EMG activity or by time requirement for consolidation of circuit network. In other words, the motor task in our motor task required one wrist joint movement with fixation of other joints in comparison with multi-joint movement of other experiments[25,26]. Therefore, three more sessions after performance gains were required to see the significant decrease of co-contraction in our motor task.

There was difference in changes of muscle activity between wrist flexor in wrist extension period and wrist extensor in wrist flexion period as the training repeated. The continuously decreased activity of wrist extensor in flexion period was observed. On the other hand, in case of wrist flexor in extension period, the activity increased at the 4th session and decreased at the 7th session. This finding seemingly may be contradicted to our assumption. A couple of explanation might be possible. One is that wrist flexor is more coherent than wrist extensor[29] therefore, in case of wrist flexor during acquisition of motor skills, Ia excitatory interneuron can be facilitated. The other is that wrist flexor has higher threshold than wrist extensor for learning a motor skill. In other words the short-latency facilitation of the flexor carpi radialis H reflex had a higher threshold than extensor carpi radialis[30]. After well-learned, decrease of co-contraction between the antagonist muscles was observed which agrees with the other study[31]. The wrist extensor in flexion was chosen to see changes of reciprocal inhibition before and after learning in view of sensitivity.

The modulation of short-latency reciprocal inhibition between wrist flexor and extensor during rhythmic movements was documented[12]. The magnitude of reciprocal inhibition of H-reflex of flexor carpi radialis was more at the transition. Therefore, two time points during wrist extension, early-flexion (actually at the end of extension) and mid-extension were measured for reciprocal inhibition. In the study of reciprocal inhibition, the

significant depression in muscle activity of wrist extensor after learning compared with before learning was observed especially at the phase of transition from wrist extension to flexion. This finding would explain that dynamic modulation of reciprocal inhibition is likely to occur and to be phase dependent in rhythmic alternating wrist movement similar to walking and cyclic arm movement[10,12]. However, this depression can be observed following sustained and fatiquing contraction and it may thus reflect the inhibitory effect of increased group III/IV afferent discharge following development of muscle fatique[31]. It was therefore important in our study to make sure that the this depression was related to the visuomotor skill training and not just to fatigue-related changes. But in the present study an influence of fatigue is unlikely, since all movements were dynamic and submaximal, only 5% of maximal voluntary contraction, and motor performance was improved following the visuo-motor skill training.

The enhanced spinal circuits producing short latency reciprocal inhibition between antagonist motoneurons during learning of repetitive alternating movements would be caused by spinal reflex activation itself , or by central modulation of spinal interneuron. The spinal circuits activated by Ia afferents are transmitted in two pathways, disynaptic Ia inhibition and presynaptic inhibition. Both pathways are modulated prior to the onset of movement, suggesting that central descending fibers converge with sensory afferents on the interneurons in the pathways and modulate their activity in preparation of the movement[32,33]. There is good evidence from other studies that changes in presynaptic inhibition of the synapses between sensory afferents and motoneurones is fundamental in the adaptation of the reflex circuitry during motor learning. Habituation of the monosynaptic gill-withdrawal reflex in the Aplysia has been shown to be caused by a depression of synaptic transmission between the sensory afferents and motoneurons through changes in presynaptic inhibition[34]. In rats and monkeys, a change in motoneuron firing threshold seems to be the main mechanism associated with long term down regulation of the H-reflex during operant conditioning[35], whereas short term down regulation

of the H-reflex is likely to be related to changes in presynaptic inhibition[36]. But there is limitation to say underlying mechanism of this spinal plasticity from our data, since we found changes occur in the strenth of spinal interneuron circuits especially mediated by Ia afferents. Also, spinal interneuron has descending control, like corticospinal tract, propriospinal tract and extrapyramidal tract such as vestibulospinal tract and rubrospinal tract. This influence on the spinal interneuron should be considered in the future.

In this study, we found that rhythmic, alternating movement training can strengthen spinal interneuron circuits, which might decrease co-contraction of agonist and antagonist pairs especially during active in movement. This finding can provide a direction in managing spasticity, a form of co-contraction in some sense, which is encountered in the clinical setting.

Further studies in the future will be necessary to elucidate the underlying mechanism of this spinal plasticity and the influence of descending tract on spinal interneuron.

V. CONCLUSION

The goal of this study was to determine whether learning a motor skill strengthens spinal interneuron circuits that facilitate the movements. The results were as follows,

1. Subjects learned to perform a rhythmic, alternating wrist movements.
2. Co-contraction of wrist extensor in flexion period was declined after learning.
3. Reciprocal inhibition mediated by spinal circuit that is associated with the timing and patterns of activation of agonist and antagonist was enhanced especially in transitional period after learning.

In conclusion, spinal circuit can be modified by learning. This spinal plasticity after learning can facilitate performance of alternating muscle activity.

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국문요약

운동학습이 척수가소성에 미치는 영향

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운동기술의 학습이 완성되면, 그 운동기술은 상대적으로 자동적으로 수행될 수 있다. 새로운 운동기술을 습득할 때는 뇌의 운동영역이 활성화되어 있지만, 학습이 이루어진 후에는 활성도가 감소된다. 이것은 뇌피질이 운동기술의 습득 후에는 하위 회로들을 보다 효율적으로 사용할 것으로 생각된다. 뇌피질 아래의 운동회로는 각기 다른 근육의 활성시기와 활성패턴과 같은 계획된 움직임의 특성을 조절한다. 이 연구의 목적은 운동기술의 학습이 원활한 움직임을 조절하는 척수간신경원회로(spinal interneuron circuit)를 강화시키는지 알아보려 한다. 연구에 참여하는 대상군은 컴퓨터 스크린의 좌측과 우측에 번갈아 나타나는 목표물에 손목의 굴곡과 신전에 의해 나타나는 커서를 움직여 맞춤으로써 손목의 주기적인 리듬감 있는 움직임을 수행하는 것을 배웠다. 운동수행의 정확도는 일정한 시간 내에 목표물에 도달하는 것으로 측정되었다. 의미 있는 운동수행능력의 향상이 세 번째 세션 후에 관찰되었다. 운동수행은 전형적인 학습곡선을 보였고 일주일 연습을 쉰 후에도 향상된 운동수행능력의 유지되는 것이 관찰되었다. 손목 근육의 활성도를 표면전극을 이용하여 기록하였는데, 일곱 번째 세션 때에, 초기에 비해서 의미있게 손목굴곡 시기에 손목신근의 동시수축이 감소하는 것이 관찰되었다. 손목신전 시기에 손목굴근의 동시수축은 감소하였으나 의미가 있지는 않았다. 상호억제는 정중신경의 자극에 의해 유발되는 손목신근의 활성도 감소 영역을 측정함으로써 평가되었다. 손목의 주기적인 움직임 중에서 신전에서 굴곡으로 변환시기에 상호억제가 의미 있게 증가되었다. 이 연구는 손목의 굴곡과 신근이 주기적으로 번갈아 움직이는 운동이 학습 가능하며, 이러한 움직임의 주기적인 훈련이 특별히 움직임의 변환시기에 척수간신경원의 상호억제의 강도를 증가시킬 수 있고, 이러한 상호억제의 증가가 원활한 움직임에 역할을 할 수 있을 것이다.

핵심되는 말 : 운동학습, 상호억제, 척수가소성