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Research report

Modification of motor cortex excitability during muscle relaxation in motor learning

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HIGHLIGHTS

- Controlling muscle release by motor learning induces changes in the motor cortex.
- After training, controlled muscle relaxation increased the SICI in related muscles.
- Such findings may be important for rehabilitating patients with motor disturbances.

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ABSTRACT

We postulated that gradual muscle relaxation during motor learning would dynamically change activity in the primary motor cortex (M1) and modify short-interval intracortical inhibition (SICI). Thus, we compared changes in M1 excitability both pre and post motor learning during gradual muscle relaxation.

Thirteen healthy participants were asked to gradually relax their muscles from an isometric right wrist extension (30% maximum voluntary contraction; MVC) using a tracking task for motor learning. Single or paired transcranial magnetic stimulation (TMS) was applied at either 20% or 80% of the downward force output during muscle release from 30% MVC, and we compared the effects of motor learning immediately after the 1st and 10th blocks. Motor-evoked potentials (MEPs) from the extensor and flexor carpi radialis (ECR and FCR) were then measured and compared to evaluate their relationship before and after motor learning. In both muscles and each downward force output, motor cortex excitability during muscle relaxation was significantly increased following motor learning. In the ECR, the SICI in the 10th block was significantly increased during the 80% waveform decline compared to the SICI in the 1st block. In the FCR, the SICI also exhibited a greater inhibitory effect when muscle relaxation was terminated following motor learning. During motor training, acquisition of the ability to control muscle relaxation increased the SICI in both the ECR and FCR during motor termination. This finding aids in our understanding of the cortical mechanisms that underlie muscle relaxation during motor learning.

1. Introduction

The ability to control muscle relaxation allows for skillful and dynamic movements in activities of daily living and in activities requiring precise temporal modulation and force output, such as reaching [1,2]. If control over the timing of motor onset and release of muscle contraction is disrupted, skillful movements will be difficult or impossible [3,4]. It is important to consider such imprecise controls when assessing the clinical motor dysfunctions of patients with stroke [5,6], Parkinson's disease [7], and dystonia [8,9]. In disorders of the central nervous system (CNS), deficits in skillful movements may be caused by spasticity and abnormal muscle tone. For example, in patients with spastic hemiplegia, the initiation of muscle relaxation is more difficult due to increased muscle tone (spasticity), which can produce unwanted activation of the antagonist muscle (co-contraction) [10]. Elucidating the mechanisms underlying the control of muscle relaxation could improve our ability to treat patients with CNS disorders.

The CNS has been shown to play an active role in the process of relaxing a muscle from a contracted state. In particular, imaging studies have revealed that voluntary muscle relaxation is preceded and accompanied by activation of the primary (M1) and supple-

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mentary motor areas [11–13]. However, how these areas control the termination of spinal motoneuron activity and mediate muscle relaxation remains unclear.

The cortical mechanisms of voluntary relaxation have been investigated using transcranial magnetic stimulation (TMS) [14,15]. These studies indicate that before and at the onset of relaxation, M1 activity declines and short interval intracortical inhibition (SICI) increases [16]. On the other hand, Begum et al. [14] reported a decrease in SICI before muscle relaxation. The contradictory results of these two reports might be due to the employment of different relaxation tasks. Moreover, Motawar et al. [15] indicated that these two studies used different paired-pulse TMS techniques and suggested that SICI gradually increased along with the progression of muscle relaxation (i.e., not prior to muscle relaxation). It should be noted that these time course studies analyzed transitions from muscle contraction to relaxation. In other words, these studies mainly explored mechanisms of motor control needed to generate muscle release. Therefore, the neurophysiological mechanisms underlying the gradual onset of muscle contraction may differ from those involved in the release of muscle contraction after motor learning.

Motor learning has been used in the rehabilitation of various disorders, as it leads to improved performance and results in characteristic excitability changes in the motor cortex (M1) [17,18]. Previous studies using TMS have demonstrated that improvements in performance are associated with the cortical reorganization of specific muscles (or movements) involved in skilled motor tasks [19–23]. However, rehabilitation strategies aimed at patients with muscle relaxation difficulties need to be improved to help them smoothly control their muscles through appropriate motor learning.

After CNS damage, motor skill training is important for successful rehabilitation. Thus, a greater understanding of the relationship between neural function and the acquisition of motor skills may have clinical relevance. Since muscle relaxation must be a type of motor skill, both healthy and disabled individuals should be able to learn muscle relaxation strategies. Moreover, using motor learning techniques to improve the control of muscle relaxation may aid in the successful performance of skillful movements. As previous studies did not evaluate changes in M1 excitability related to gradual muscle relaxation during skill training, we sought to assess changes in motor cortex excitability with the progression of motor learning during muscle relaxation. We previously reported that evaluating gradual relaxation with a tracking task results in increased corticospinal excitability in the antagonist muscle [24]. However, we did not analyze any changes that could be accompanied by the acquisition of motor learning.

From this viewpoint, we hypothesized that during progressive motor learning for muscle relaxation M1 activity would not only gradually decrease to attenuate muscle contraction, but would also dynamically change with the force output of muscle contraction. Furthermore, we postulated that the motor inhibition circuit (i.e., SICI) would also be modified during motor skill learning and gradual muscle relaxation. Therefore, this study analyzed changes in M1 excitability both pre and post motor learning at high and low levels of muscle output during gradual muscle relaxation.

2. Methods

2.1. Participants

Thirteen healthy right-handed volunteers (seven men and six women, aged 20–37 years old) participated in the present experiments after providing informed consent. Handedness was confirmed using the oldfield handedness inventory [25]. None of the participants had any history of impairments in neuromuscular or physical function that may have affected task performance. The experiment was performed in accordance with the Declaration of Helsinki and with the approval of the Local Ethics Committee of Kanagawa University of Human Services.

2.2. Electromyography recording

Electromyography (EMG) activities of the extensor carpi radialis muscle (ECR) and flexor carpi radialis muscle (FCR) on the right side of the body were recorded using pairs of surface Ag/AgCl electrodes placed over these muscles in a belly-tendon montage. The raw signal was amplified and filtered (band-pass 5–3000 Hz) using a bioelectric amplifier (Neuropack MEB-2200; Nihon Kohden Corp., Tokyo, Japan), digitized at 4000 Hz, and stored for offline analysis on a laboratory computer (Power Lab system; AD Instruments Pty Ltd., New South Wales, Australia). The background EMG (B.EMG) activity for each muscle was calculated using the root mean square (RMS) in a 100-ms window before TMS.

2.3. Experimental paradigm

Subjects sat comfortably on a chair with their right forearms positioned horizontally over a table. The elbows of the participants were at a 45° angle in a pronated position. Before initiating the experiment, we measured the force of the wrist extension produced by each subject's maximal voluntary contraction (MVC) of the ECR against a plate. A strain gauge (Kyowa Electronic Instruments Co., Tokyo, Japan) was mounted on the vertically bent portion of this plate. The analog signal was amplified (SA-250 STRAIN AMPLIFIER; TEAC, Tokyo, Japan), filtered, and converted to digital data (NI USB-6229 BNC; National Instruments Corp., Austin, TX, USA).

A custom-written computer program (LabVIEW software, ver. 7.1; National Instruments Corp., USA) was used for the muscle relaxation training, as well as for the test block. The motor task consisted of two phases: isometric contraction and gradual muscle release. First, the subjects pushed the abovementioned plate with the distal metacarpal palm while maintaining 30% of the MVC for 3 s by extending the right wrist joint. Then, the subjects were asked to perform controlled, gradual muscle release for 2 s (Fig. 1). The contractions and muscle releases were performed in accordance with a moving dot (measured by the force transducer), and subjects were required to track the dot as it moved down a waveform that was presented on the computer screen. The dot, which moved automatically from left to right, could be adjusted up or down through the subjects' output force (Fig. 1). Subjects were able to control the force output of the cursor by performing a wrist extension without contracting the wrist flexor muscle. During wrist extension, the cursor moved to the top of the screen. Conversely, the cursor moved to the bottom of the screen when the force was released. The training consisted of 10 blocks, and each block was composed of 10 trials. To measure motor performance in the test phase, the target waveform suddenly disappeared 2 s after the presentation of a go-signal, and the subject was asked to continue the tracking task by memory. To prevent participants from experiencing excessive fatigue, each trial lasted for only 5 s. The next trial began after an intertrial interval of 5 s. Moreover, participants were given breaks of ~2 min between blocks. Errors were measured as the RMS of the difference between the target and the actual force output. The RMS was used to characterize the acquired skill level of the learned task, and to compare the averages among three blocks (1st, 5th, and 10th). Signals were recorded by LabVIEW and stored for later analysis.