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Research article

Surround inhibition in motor execution and motor imagery

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HIGHLIGHTS

- We examined the extent of surround inhibition (SI) during motor execution (ME) and motor imagery (MI).
- There was a moderate correlation between the extent of SI during ME and MI.
- The extent of SI during MI was depended on the vividness of MI.
- A common neural substrate related to SI would be recruited during ME and MI.

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ABSTRACT

Surround inhibition (SI) is a neural mechanism to focus neuronal activity and facilitate selective motor execution (ME). The aim of the present study was to investigate whether SI is also generated during motor imagery (MI). Furthermore, we investigated whether the extent of SI during MI depends on the strength of SI during ME and/or vividness of MI. The extent of SI was examined during MI and ME of index finger flexion. Transcranial magnetic stimulation was applied at rest, during initiation of the movement (phasic phase) and during tonic muscle contraction of the index finger flexors. Motor evoked potentials (MEPs) were recorded from a surround muscle, abductor digiti minimi (ADM) and a synergistic muscle, the first dorsal interosseous muscle. The amplitude of ADM MEP was reduced during the phasic phase, which indicates that SI occurred during ME. In seven of 14 subjects, SI was also observed during MI, although this effect was not significant. There was a moderate correlation between the extent of SI during ME and MI. Furthermore, good imagers who experienced vivid MI during the MI task showed stronger SI than poor imagers. These results indicate that common neural substrates involved in SI during ME are at least in part recruited during MI. In clinical situations, the therapeutic use of MI to generate vivid MI may be one of effective tool to develop the strength of SI, which facilitate selective execution of desired movements

1. Introduction

Motor imagery (MI) is the mental simulation of a given movement without any overt movement [1–4]. Functional imaging studies [5–7] have revealed that motor execution (ME) and MI share many common neural substrates, such as the primary motor cortex, supplementary motor area, premotor cortex, parietal cortex and cerebellum [3]. Some electrophysiological studies using transcranial magnetic stimulation (TMS) have shown increased corticospinal excitability of the prime mover of the imagined movement during MI [8–13]. However, there have been conflicting reports regarding the corticospinal excitability of surround muscles not involved in the imagined movement. Some researchers have reported that corticospinal excitability of surround muscles increases during MI [8,13], whereas others have reported no change in surround muscles [9–12,14].

There are two reasons related to methodology that could explain these inconsistent results. First, most MI studies did not define the stimulation site as a 'motor hot spot' of surround muscles [9–12,14,15]. This may have resulted in an underestimation of the change in corticospinal excitability of surround muscles [16].

Abbreviation: ADM, abductor digiti minimi; ANOVA, analysis of variance; EMG, electromyography; FDI, first dorsal interosseous; GABA, γ-amino butyric acid; MVC, maximum voluntary contraction; MEP, smotor evoked potentials; ME, motor execution; MI, motor imagery; SI, surround inhibition.

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Second, different stimulus timing may contribute to inconsistent results. During ME, changes in the corticospinal excitability of surround muscles depend on the timing of the movement. At the initiation of the movement, corticospinal excitability of surround muscles decreases compared with the resting state, but this effect disappears during the following tonic muscle contraction phase [17,18]. As described above, if MI shares common neural substrates with ME, similar temporal modulation of corticospinal excitability would be expected. However, most previous studies have not considered stimulus timing. Therefore, it is possible that an inhibitory effect at the initiation of MI has been overlooked.

The inhibition of corticospinal excitability at the initiation of movement during ME is generated by γ-amino butyric acid (GABA)ergic inhibition in a cortical area surrounding activated neurons, referred to as surround inhibition (SI) [18,19]. In the motor system, SI is a neural mechanism to focus neuronal activity and facilitate selective execution of desired movements [20]. Previous studies showed that the strength of SI during ME changes with repetitive motor training [21,22]. Assuming that ME and MI share common neural substrates, the strength of SI during MI should correlate with that during ME. Furthermore, although it had been demonstrated that the corticospinal excitability change during MI depends on MI ability [23,24], it is unclear whether the strength of SI is affected.

In the present study, therefore, we determined whether SI occurs during MI as well as ME. In addition, we investigated whether the extent of SI during MI depends on SI during ME and/or vividness of MI. We used TMS to examine the corticospinal excitability of a surround muscle (abductor digiti minimi; ADM) during ME and MI of index finger flexion at rest, at initiation of the movement (phasic) and during tonic muscle contraction (tonic). We hypothesized that SI would also be observed during MI, and the strength of SI during MI would be correlated with the strength of SI during ME and vividness of MI.

2. Material and methods

2.1. Subjects

Fourteen healthy adults (mean ± SD, 21.2 ± 1.5 years, range 20–25 years, 7 males) participated in this experiment. They were all right handed according to the Edinburgh handedness inventory [25]. All subjects provided written informed consent before taking part in this experiment. The experimental protocol was approved by the ethics committee of the Ibaraki Prefectural University of Health Sciences. All procedures conformed to the standards set out in the World Medical Association Declaration of Helsinki, 2013.

2.2. Motor execution and motor imagery tasks

The participants were seated comfortably in a chair with their right hand lying relaxed on a side table. Their right index finger was placed on a force transducer and the participants were instructed to perform a ME or MI task. The ME task was flexion of the metacarpophalangeal joint of the right index finger. At the beginning of the experiment, the maximum voluntary contraction (MVC) of the index finger flexors was measured three times using the force transducer. A target force level of 10% MVC was selected because previous research has shown that this force produces the highest level of SI [26]. The target force level was displayed on a monitor in front of the subjects and they were instructed to flex the index finger selectively with other hand muscles relaxed. They performed index finger flexion of 10% MVC for 3 s as quickly as possible after an auditory signal (200 ms, 250 Hz). Prior to recording, the subjects practiced the task until they achieved a consistent motor performance (reaction time and force level).

For the MI task, the subjects were instructed to imagine the kinaesthetic sensation generated by the actual index finger flexion of 10% MVC for 3 s, which they accomplished during the ME task [15,27]. In advance, the auditory reaction time of actual index finger flexion was measured. The timing of the onset of MI was defined as the average reaction time of 25 measurements (334.6 ± 99.4 ms). The subjects were instructed to practice this MI task without any overt muscle contraction using electromyographic feedback from finger muscles (see below). To verify the vividness of MI achieved, after the experiment, the participants completed a five-point Likert scale, based on the kinesthetic subscales of the Kinaesthetic and Visual Imagery Questionnaire (5: As intense as executing the action, 1: No sensation) [28].

2.3. Electromyography

Before electrode attachment, the skin was rubbed with alcohol and abraded with abrasive skin prepping gel. Surface Ag-AgCl electrodes were placed over the first dorsal interosseous (FDI) muscle and the ADM in a belly-tendon montage. The FDI is a synergistic muscle of the index finger flexion rather than a prime mover, and the ADM is not involved in index finger flexion (surround muscle). Previous studies have demonstrated that, at the initiation of index finger flexion, ME induces corticospinal excitation in the FDI muscle and inhibition in the ADM [18,29]. In the present study, electromyography (EMG) signals were amplified (Neuropack MEB2300; Nihon Kohden, Japan) at an appropriate level and band-pass filtered at 5 Hz–5 kHz. All signals were sampled at 10 kHz and stored on a laboratory computer for offline analysis.

2.4. Transcranial magnetic stimulation

A Magstim 200^2 stimulator (Magstim Co., Whitland, UK) connected to a figure-of-eight coil (diameter of each loop = 70 mm) was used to elicit motor evoked potentials (MEPs) from the right FDI and ADM muscles. The handle of the coil was positioned pointing backward and laterally at a 45° angle from the midline to induce anteromedial current direction in the left brain, and to activate the corticospinal tract *trans*-synaptically [30,31]. TMS was applied over the hot spot of the right ADM, which is the optimal position to produce the largest and most consistent MEPs amplitude with stimulus intensity slightly above the threshold. The resting motor threshold was defined as the lowest intensity that produced an MEP amplitude of >50 μV in at least five out of 10 trials. In the test trials, a stimulus intensity of 140% was used. Only the hot spot of ADM was tested in the present experiment, but stable MEPs in the FDI muscle were also recorded. The time course of the experimental tasks is shown in Fig. 1. The Lab VIEW program (National Instruments, Japan) was used to apply TMS during the rest, phasic and tonic phases in a randomized order. The phasic phase of the ME task was defined as the time for onset of FDI EMG activity (EMG signal amplitude >100 μV). The phasic phase of the MI task was defined as the individual latency (determined based on individual reaction time during the ME task) after the onset of the auditory signal. The tonic phase started 1.5 s after the phasic phase (representing tonic contraction phase of the FDI muscle or its MI). The rest phase started 8 s after the phasic phase (5 s after the end of FDI muscle contraction or its MI). This timing was considered valid to investigate the resting state because corticospinal excitability returns to baseline approximately 1 s after muscle contraction [18]. At least 16 MEPs were recorded during each phase.