Published in final edited form as:

Clin Neurophysiol. 2016 June ; 127(6): 2343–2349. doi:10.1016/j.clinph.2016.02.020.

Dynamic Modulation of Corticospinal Excitability and Short-Latency Afferent Inhibition during Onset and Maintenance Phase of Selective Finger Movement

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Abstract

Objective—During highly selective finger movement, corticospinal excitability is reduced in surrounding muscles at the onset of movement but this phenomenon has not been demonstrated during maintenance of movement. Sensorimotor integration may play an important role in selective movement. We sought to investigate how corticospinal excitability and short-latency afferent inhibition changes in active and surrounding muscles during onset and maintenance of selective finger movement.

Methods—Using transcranial magnetic stimulation (TMS) and paired peripheral stimulation, input-output recruitment curve and short-latency afferent inhibition (SAI) were measured in the first dorsal interosseus and abductor digiti minimi muscles during selective index finger flexion. Results: Motor surround inhibition was present only at the onset phase, but not at the maintenance phase of movement. SAI was reduced at onset but not at the maintenance phase of movement in both active and surrounding muscles.

Conclusions—Our study showed dynamic changes in corticospinal excitability and sensorimotor modulation for active and surrounding muscles in different movement states. SAI does not appear to contribute to motor surround inhibition at the movement onset phase. Also, there seems to be different inhibitory circuit(s) other than SAI for the movement maintenance phase in order to delineate the motor output selectively when corticospinal excitability is increased in both active and surrounding muscles.

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Conflict of interest

None of the authors have potential conflicts of interest to be disclosed.

Significance—This study enhances our knowledge of dynamic changes in corticospinal excitability and sensorimotor interaction in different movement states to understand normal and disordered movements.

Keywords

Surround inhibition; short latency afferent inhibition (SAI); transcranial magnetic stimulation; tonic movement; motor evoked potential

1. Introduction

Ability to make highly selective finger movements is a unique feature of human motor control. It is believed that the human motor system has a physiological mechanism to suppress unwanted movements and release only desired movements: this phenomenon is called “motor surround inhibition (mSI)” (Sohn et al., 2004). Several studies demonstrated mSI at the onset of movement or phasic movement, but not during maintenance of muscle contraction or tonic movement. One study showed that there was surround facilitation rather than inhibition for the maintenance phase of movement (Beck et al., 2008).

The exact mechanism of mSI is still unknown. It is possible that sensorimotor interaction also plays a role in selective movement. For example, the somatosensory evoked potential is selectively attenuated for the particular body part that is engaged in the movement at the onset as well as during maintenance of contraction (Rushton et al., 1981, Tapia et al., 1987). Sensorimotor integration can be also assessed by transcranial magnetic stimulation (TMS). The motor evoked potential (MEP) amplitude is substantially reduced when preceded by peripheral nerve stimulation at a short latency (~20 ms), an effect known as short-latency afferent inhibition (SAI) (Tokimura et al., 2000). A previous study showed that SAI was reduced in the active hand muscle during both the onset and maintenance phases of movement (Asmussen et al., 2013), but SAI in surrounding muscle at the onset phase showed contradictory results (Voller et al., 2006, Richardson et al., 2008). SAI in the surrounding muscle during the maintenance movement phase has never been tested.

It is crucial to learn the whole scope of dynamic changes in corticospinal excitability and sensorimotor interaction in the different movement states to understand normal and disordered movements. Therefore, we have addressed two questions in this study: How does corticospinal excitability change in active and surrounding muscles for different movement states? Based on results from previous studies (Beck et al., 2008), we expected that there would be surround inhibition only for onset phase and not for maintenance phase. Our second question was how SAI was modulated in active and surrounding muscles for different movement states. We speculated that SAI would be enhanced in the surrounding muscle for the maintenance phase compared to active muscle to counteract increased corticospinal excitability.

2. Methods

2.1. Experiment

2.1.1. Subjects—Thirteen healthy right-handed individuals (7 males, 6 females, age 33.92 ± 8.67) participated in this study. All subjects were at least 18 years of age, right-handed, with no history of neurological or psychiatric disorders and were not taking any medications. They all were normal on neurological examination done within the past year. All participants provided written, informed consent before the experiments. The protocol was approved by the Combined Neurosciences - Institutional Review Board of the National Institutes of Health, USA.

2.1.2. Recording—The subjects were seated in a comfortable chair and their right hand was placed on the table, which was adjusted to their comfort level. Disposable surface Ag-AgCl electrodes were placed on the right abductor digiti minimi (ADM) and first dorsal interosseus (FDI) muscles in a belly-tendon montage. The EMG signal was amplified and band-pass filtered (10–2000 Hz) using a conventional EMG machine (Nihon Kohden). The signal was digitized at 5 kHz with Signal software version 5.09 (Cambridge Electronic Design, Cambridge, UK) and stored in a computer for off-line analysis. Individual MEPs were measured during three phases of movement (rest, onset, and maintenance).

2.1.3. Motor task—All tests were performed at rest or during different phases of a selective movement of the right index finger activating FDI as a synergist while keeping other muscles relaxed. With their right palm flat on a table in front of them, subjects were instructed to push down on a small force transducer (Strain Measurement Devices; model S215 load cell) to produce 10% of their maximum force (10% F_{max}). EMG activity corresponding to 10% F_{max} was marked on the EMG screen for visual feedback. Then, the force transducer was removed and they were instructed to press on the table to match the EMG activity of 10% F_{max} at the tone and maintain the same force for the duration of the tone (3s) (figure 1). The tone was repeated every 7 seconds with 15% variation. The reason for not using the force transducer for the main experiment was to keep the subject's hand relaxed as much as possible in the same position while recording different movement states. Muscle activities of FDI and ADM were monitored on a continuous EMG screen to ensure that only the target muscle was activated.

2.1.4. MS—TMS was performed with a figure-of-eight-shaped coil (7-cm diameter for each half) connected to a Magstim 200 magnetic stimulator (Magstim, Whitland, Dyfed, UK) that delivers monophasic pulses. The coil was positioned tangentially on the scalp over M1 at an angle of 45° to the midline with the handle pointing backwards to induce a current in the postero-anterior direction in the brain. The optimal site for evoking maximal amplitude MEPs from the ADM was identified as the hot spot. TMS over the ADM hot spot was used to simultaneously measure corticospinal output to the ADM and FDI.

2.1.5. Paired pulse experiment with peripheral stimulation—Ring electrodes were put in the right 2nd and 5th fingers around proximal and distal interphalangeal joints and electrical stimulation was given through Digitimer (Stimulator model DS7A, Hertfordshire,

England) using a pulse duration of 200 μ s. Sensory threshold was measured for the 2nd and 5th fingers and 300% of the perceptual threshold of each finger was used for digital stimulation for paired stimulation (Tokimura et al., 2000). The peripheral stimulation was given 25ms prior to the TMS pulse for each movement state to elicit SAI. The fixed interval of 25ms was frequently used in previous studies for SAI (Asmussen et al., 2013).

2.1.6. Experimental design—MEPs were measured at three different phases of the movement: rest, onset (at the onset of EMG > 100 μ V in FDI), and maintenance (2 seconds after the onset of the movement) (Figure 1). For each movement state, input-output recruitment curve (IOC) was obtained and the data were fitted to the Boltzmann sigmoidal function (see below, outcome measures). For IOC, 60 single pulses were given; 3 pulses for each 5 % increment from 5% to 100% maximum stimulation output. Then the corresponding S50 (stimulation intensity required to obtain a response of 50% of the maximum) for FDI and ADM were used as test stimulus (TS) intensities for SAI measurements for the muscle and movement state that was tested. Six blocks of SAI were recorded for each movement state (rest, onset and maintenance) and muscle (ADM and FDI). Thirty pulses were given in one SAI block; TMS with 2nd digit stimulation, TMS with 5th digit stimulation and TMS only. The subject did not know which muscles were being recorded for SAI. The order of the 6 blocks was randomized.

2.2. Outcome measures

MEP was measured as peak-to-peak amplitude. Motor threshold (MT) and S50 were obtained from IOC after fitting the data to the Boltzmann sigmoid function. The Boltzmann function was given by the equation of ‘MEP amplitude = offset + MEP_{max} / (1 + exp ((S50 – X)/k))’ where MEP_{max} is the MEP amplitude at plateau, S50 is the stimulation intensity required to obtain a response of 50% of the maximum, X is the stimulation intensity (independent variable), k is Boltzmann’s slope parameter, the inverse of which is directly proportional to the maximum steepness function at S50 (Devanne et al., 1997). MT was taken to be equal to the x-intercept of the line drawn tangential to the midpoint of the curve. In order to compare individual subjects’ IOC, MEP amplitudes for stimulation intensities ranging from 80% - 200% MT were calculated from the individual curve equations and normalized to the individual subject’s MEP_{max}. The normalized IOCs were used for statistical analysis. The recruitment curves that did not reach the plateau even at 100% maximum stimulator output were discarded from further analysis since the parameters of the Boltzmann sigmoid function could not be estimated reliably. The number of discarded curves was: 2 for ADM at rest, 3 for ADM at onset phase, and 1 for FDI at rest. SAI was expressed as the ratio of the mean peak-to-peak amplitudes of the conditioned MEPs to that of the unconditioned MEPs.

2.3. Statistical Analysis

Since a repeated measures design with two within-subject factors (muscle and movement state) was applied in this study, a mixed model was used to evaluate the effect of the two factors on the outcome measures. Tukey’s method was used for multiple comparison tests and the Shapiro-Wilk test assessed normality of the model residuals. A significance level of

0.01 was used to adjust for multiplicity. Statistical analyses were performed using SAS version 9.3.

For outcome measures of S50, MT, k and SAI, the mixed model included movement state, muscle and their interaction as fixed effects and subject as the random effect. A covariance structure of direct product compound symmetry (UN@CS) was used, where the unstructured (UN) matrix was for muscle and a compound symmetry (CS) matrix was used for movement state (Moser, 2004).

For IOC analysis, the MEP amplitude was log-transformed. The analysis for FDI and ADM muscles was performed separately. The mixed model included movement state, intensity and their interaction as fixed effects and subject as random effect. Considering the large number of repeated measurements ($13 \times 3 = 36$) relative to the small number of subjects (13), a covariance structure of CS for movement state with intensity as repeated factor, instead of (UN@CS), was used.

3. Results

3.1. Stimulation intensities and MEP amplitudes

The group-averaged unconditioned MEP amplitudes for the active (FDI) and surround (ADM) muscles for the different movement states are presented in Table 1.

3.2. S50, MT and k

For S 50, the interaction between muscle and movement state was significant ($F_{2,219}=49.2$, $p<0.0001$) (Figure 2A), indicating that the effect of movement on S50 was different for active and surrounding muscles. In ADM, there was no significant effect of movement state on S50. In FDI, S50 was decreased at movement onset and maintenance phase compared to rest ($p<0.0001$ for both states), but the difference between onset and maintenance phases was not significant ($p=0.18$). When S50 was compared between the two muscles, the difference was significant at both onset and the maintenance phase ($p<0.0001$ for both conditions), but not at rest.

For MT, the interaction between muscle and movement state was significant ($F_{2,20}=19.6$, $p<0.0001$) (Figure 2B), indicating that the effect of movement on MT was different for active and surrounding muscles. In ADM, there was no significant effect of movement state on MT.

In FDI, MT was lower at onset phase compared to rest ($p<0.0001$) but not to the maintenance phase ($p=0.0282$). When MT was compared between muscles, the difference was significant at onset and the maintenance phase ($p<0.0001$ and 0.0037 , respectively) but not at rest.

For the slope of IOC represented by the k value, there was a significant effect of muscle x movement state interaction ($F_{2,25.7}=4.62$, $p=0.0193$), suggesting that the slope change in ADM differed from that in FDI. However, no significance was found in the multiple comparisons in *post-hoc* analysis.

3.3. MEP amplitudes of IOCs of surround (ADM) and active (FDI) muscle for different movement states

The effect of movement state on the normalized MEP amplitudes was significant for both surrounding (ADM) and active (FDI) muscles. (Figure 3)

For normalized MEP amplitudes with stimulation at suprathreshold intensities ($MT > 100\%$), there was significant main effect of intensity and movement condition, but no interaction between intensity and movement state ($p=0.08$ for ADM and $p=0.828$ for FDI). Thus, the effect of movement state on normalized MEP amplitude was consistent across the different intensities and MEP amplitudes across all suprathreshold intensities were compared between different movement states. Normalized MEP amplitudes with subthreshold stimulation were not included in the analysis because they are close to zero with less than 50% chance to have meaningful MEP ($>50 \mu V$) by definition; therefore, it is not relevant to modulation of corticospinal excitability in different movement states.

For ADM, the mixed model analysis showed that the effects of stimulation intensity ($F_{9,91}=135.6$, $p<0.0001$) and movement state ($F_{2,206}=12.6$, $p<0.0001$) on normalized MEP amplitudes were significant. Pair-wise multiple comparisons revealed that normalized MEP size at onset phase was significantly lower than that at rest ($p<0.0001$) and at the maintenance phase ($p=0.0007$). There was no significant difference in normalized MEP size between rest and the maintenance state.

For FDI, the mixed model analysis revealed that the effects of intensity ($F_{9,99}=193.14$, $p<0.0001$) and movement condition ($F_{2,220}=20.1$, $p<0.0001$) on normalized MEP amplitudes were significant. Normalized MEP amplitudes at onset and maintenance phase were significantly higher compared to that at rest ($p<0.0001$ for both states).

3.4. SAI modulation in active and surrounding muscle for different movement states

The effect of interaction between muscle and movement condition on SAI was not significant ($p=0.3824$). SAI significantly differed between movement states ($p=0.0034$) but not between muscles ($p=0.4666$) (Figure 4). Multiple comparisons showed that SAI was significantly reduced at the onset phase compared to rest for FDI ($p=0.0003$). In ADM, there was a strong trend for reduced SAI at the onset phase compared to rest ($p=0.0418$). SAI was not significantly different at the maintenance phase compared to rest in both ADM and FDI ($p=0.9821$ and $p=0.103$ respectively).

4. Discussion

Our study investigated the dynamic changes of corticospinal excitability in active and surrounding muscles during selective finger movement by examining IOC at rest, movement onset, and maintenance phases. We used SAI to test how corticospinal excitability is modulated differently to sensory afferent inputs for different movement states. We found that corticospinal excitability decreased in surrounding muscle only at movement onset but not at the maintenance phase. SAI was differently modulated by movement states but there was no difference between active and surrounding muscles.

4.1. Corticospinal excitability reduces only at movement onset but not in the maintenance phase in surrounding muscle

Our finding proves that surround inhibition exists only for the onset phase but not the maintenance phase. However, we did not find surround facilitation that was reported in previous study (Beck et al., 2008). The reason why the surround inhibition is reversed during maintenance is unclear. Surround inhibition is believed to be an intracortical mechanism to suppress increased spinal excitability during movement initiation (Thirugnanasambandam et al., 2015). A previous study showed that spinal excitability examined by H-reflex in surround muscle is only increased during the movement onset phase (Beck et al., 2008). These results suggest that there are different mechanisms for movement initiation and maintenance. The motor output is still sculpted selectively even though MEPs are increased in both active and surrounding muscles during movement maintenance. Hence, there should be another mechanism other than surround inhibition to make the contrast between active and surrounding muscles. It is possible that a subcortical area such as the basal ganglia or cerebellum could play an important role for maintaining selective movement. It was found that the discharge of pallidal neurons was related more to the build-up of EMG activity in monkeys (Anderson et al., 1985). Therefore, it can be assumed that the basal ganglia is heavily involved during movement maintenance. Onset phase of movement may rely more on a feedforward mechanism while maintaining movement might use more of a feedback mechanism. Therefore, it is conceivable that feedforward control of movement utilizes more cortical mechanism such as surround inhibition for initiation of movement whereas the feedback nature of the basal ganglia and cerebellum is more important during movement maintenance (Manto et al., 2012, Hwang, 2013). Cerebellum may be involved in this feedback loop from the beginning of the movement through the fast spinocerebellar tract, whereas basal ganglia will contribute after the movement already has begun through indirect sensory feedback through thalamocorticopallidal connections (Bosch-Bouju et al., 2013). However, it is still unclear how the basal ganglia and cerebellum can shape selective movement maintenance. This issue could not be addressed in the current TMS study.

4.2. SAI modulated differently by movement states

SAI was reduced for onset, but not for the maintenance phase of movement in both active and surrounding muscles. This result is somewhat discordant from what was previously reported about SAI in active muscle (Asmussen et al., 2013). Here, SAI was reduced in all movement states compared to rest either with mixed nerve or digital nerve stimulation. Furthermore, SAI was even lower at movement onset compared to maintenance phase with mixed nerve stimulation (Asmussen et al., 2013). This inconsistency could be due to a different methodology in the experiments. For example, subjects who did not show SAI at rest were excluded in some previous studies; however, we included every subject because our interest was the relative changes of SAI in an individual subject across different movement states. Nonetheless, the trend of SAI modulation is similar in current study; SAI is reduced at movement onset more than at maintenance phase.

There are several mechanisms that could explain the reduced SAI during movement. The mechanism of SAI was reported to be involved in changes in S1 excitability by GABAergic, cholinergic neurons, or both (Di Lazzaro et al., 2000, Di Lazzaro et al., 2005). Previous

tDCS studies suggest that less activity in S1 is related to decreased SAI (Scelzo et al., 2011, Kojima et al., 2015). It is well known that somatosensory evoked potentials are selectively attenuated for the particular body part that is engaged in the movement at the onset as well as during maintenance of contraction (Rushton et al., 1981, Tapia et al., 1987, Jones et al., 1989). Therefore, less S1 activity during movement could lead to decreased SAI. Another consideration is increased spinal excitability during movement onset. It seems clear that SAI modulation is largely cortical in origin for the pre-movement state (Ferreri et al., 2012, Asmussen et al., 2013), but spinal excitability may contribute during the movement onset phase.

SAI may not play a role in surround inhibition during selective finger movements

Our study demonstrated that SAI does not contribute to SI during onset phase of selective finger movements. SI and SAI might use different neuronal elements for its mechanism. A recent study suggested that intracortical interneurons are primarily involved in surround inhibition (Thirugnanasambandam et al., 2015). In contrast, SAI is due to reduction of later I waves, especially I3 (Tokimura et al., 2000, Ni et al., 2011) believed to arise from cortico-cortical interaction (Di Lazzaro et al., 2012).

Also, SAI did not contribute to providing a contrast between active and surrounding muscles during movement maintenance. SAI is thought to represent relatively direct cortico-cortical connection within a sensorimotor area that may start up to 5 ms after arrival of sensory input at the cortex (Tokimura et al., 2000). Thus, the lack of difference in SAI for active and surrounding muscles might be that the feedback loop required for selective maintenance of movement may involve a more widespread area other than the sensorimotor cortex that may take longer than 5 ms. In that sense, it would be interesting to investigate the role of long-latency afferent inhibition (LAI) because it involves more widespread subcortical structures for sensorimotor integration.

4.3.Limitations

This was an exploratory study in small number of healthy subjects. Moreover, we demonstrated changes of corticospinal excitability only from the IOCs. Since the differences in MEP amplitudes were not the same across all intensities, it would also be useful to study a single suprathreshold intensity to quantify the difference. However, we believe that IOC can give us more options to examine corticospinal excitability by measuring MT, S50, and the slope. Also, we did not examine spinal excitability during different the movement states. However, it was already proven in previous studies that SI and SAI occur at the supraspinal level (Beck et al., 2008, Ferreri et al., 2012).

5. Conclusions

Our study finding showed dynamic changes in corticospinal excitability and sensorimotor modulation for active and surrounding muscles in different movement states. Corticospinal excitability changed differently for active and surrounding muscles but SAI did not differ between the two muscles. SAI does not seem to contribute to motor surround inhibition at the movement onset phase. Also, there might be a different inhibitory circuit other than SAI

for the movement maintenance phase in order to counteract enhanced corticospinal excitability in surrounding muscles.

Acknowledgments

Funding source

NINDS Intramural Program. Hyun Joo Cho is supported by a research grant from the Dystonia Medical Research Foundation.

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Highlights

- Motor surround inhibition was present only at the onset phase, but not at the maintenance phase of movement.
- Short-latency afferent inhibition (SAI) was decreased at onset, but not for the maintenance phase of selective movement in both active and surrounding muscles.
- SAI does not contribute either to initiation phase or to maintenance phase of selective movement.

Figure 1. Experimental design. IO and SAI were measured in three movement states (rest, onset and maintenance) in ADM and FDI.

Figure 2. Changes of S50 (A) and MT (B) in surround (ADM) and active muscle (FDI) in different movement states (Mean $\pm$ SEM).

Figure 3. Normalized IOCs of the surround (ADM) (A) and active muscle (FDI) (B) in different movement states. (Mean $\pm$ SEM).

Figure 4. Modulation of SAI in surround (ADM) and active (FDI) muscle in different movement states.

Table 1

Unconditioned MEP amplitudes (Mean $\pm$ SEM mV) in ADM and FDI in three movement states (rest, onset and maintenance) with stimulation at S50 of each movement state.

	Rest	Onset	Maintenance
ADM	2.08 $\pm$ 0.43	1.74 $\pm$ 0.36	1.83 $\pm$ 0.46
FDI	2.09 $\pm$ 0.49	3.34 $\pm$ 0.25	3.07 $\pm$ 0.33