Exp Brain Res (2006) 169: 226–231
DOI 10.1007/s00221-005-0140-9

RESEARCH ARTICLE

**Bernhard Voller · Alan St Clair Gibson
James Dambrosia · Sarah Pirio Richardson
Mikhail Lomarev · Nguyet Dang · Mark Hallett**

Short-latency afferent inhibition during selective finger movement

Received: 29 December 2004 / Accepted: 16 July 2005 / Published online: 12 November 2005

Abstract During individual finger movement, two opposite phenomena occur at the level of the central nervous system that could affect other intrinsic hand muscle representations, unintentional co-activation, and surround inhibition (SI). At rest, excitability in the motor cortex (M1) is inhibited at about 20 ms after electric stimulation of a peripheral nerve [short-latency afferent inhibition (SAI)]. We sought to determine whether SAI changes during selective index finger movement. Effects were measured by the response to transcranial magnetic stimulation in two functionally distinct target muscles of the hand [abductor digiti minimi muscle (ADM), first dorsal interosseus muscle (FDI)]. An increase in SAI in the ADM during index finger movement compared to at rest could help explain the genesis of SI. Electrical stimulation was applied to either the little finger (homotopic for ADM, heterotopic for FDI) or the index finger (heterotopic for ADM, homotopic for FDI). During index finger movement, homotopic SAI was present only in the ADM, and the effect of peripheral stimulation was greater when there was less co-activation. Heterotopic SAI found at rest disappeared with movement. We conclude that during movement, homotopic SAI on the muscle in the surround of the intended movement may contribute to SI.

B. Voller · J. Dambrosia · S. Pirio Richardson
M. Lomarev · N. Dang · M. Hallett (✉)
Human Motor Control Section, NINDS/NIH, Bldg. 10/5N226,
10 Center Dr., Bethesda, MD, 20892 USA
E-mail: hallettm@ninds.nih.gov
Tel.: +1-301-4969526
Fax: +1-301-4802286

B. Voller
Department of Neurology, Medical University of Vienna,
Vienna, Austria

A. St Clair Gibson
Research Unit of Exercise Science and Sports Medicine,
Department of Human Biology, University of Cape Town,
Cape Town, South Africa

Keywords Motor cortex · Afferent inhibition · Surround inhibition · Transcranial magnetic stimulation · Sensorimotor integration

Introduction

Motor cortex (M1) excitability can be modulated by applying a conditioning electrical stimulus to a peripheral nerve followed by transcranial magnetic stimulation (TMS) over the contralateral M1 at different interstimulus intervals. In general, inhibition of the motor evoked potential (MEP) appears to be most consistent with interstimulus interval (ISI) at approximately 20 ms, called "short-latency afferent inhibition" (SAI) and 200 ms called, "long-latency afferent inhibition" (LAI) (Classen et al. 2000; Tokimura et al. 2000; Tamburin et al. 2001; Sailer et al. 2002; Chen 2004). Modulation of M1 excitability also depends on the location of the peripheral stimulus (Classen et al. 2000; Tamburin et al. 2001; Kobayashi et al. 2003). In these studies, MEPs elicited by TMS were recorded from two different hand muscles. Electrical stimulation was applied to a digital nerve near (homotopic) or distant from (heterotopic) each target muscle. SAI at rest seems to be stronger with homotopic stimulation (Classen et al. 2000; Tamburin et al. 2001). What happens to SAI during selective finger movement is unknown.

In studying selective finger movement using TMS, at least two other related central phenomena should be considered, co-activation and its opposite, surround inhibition (SI). Although it is generally assumed that fingers move independently, it has been shown that humans hardly ever move one finger alone (Fish and Soechting 1992; Soechting and Flanders 1992; Engel et al. 1997). Co-activation in muscles not related to the movement has, in part, a central cortical origin (Hager-Ross and Schieber 2000; Slobounov et al. 2002), which we refer to as unintentional co-activation. The concept of SI in the human motor system is that unwanted movements in surrounding muscles during voluntary

actions are suppressed (Hallett 2003). In two studies, the effect of a moving finger on intracortical inhibition (ICI) was explored (Stinear and Byblow 2003; Sohn and Hallett 2004a). In both studies, MEP reduction in the muscle in the surround depended on the amount of unintentional co-activation which is associated with less effective SI. From the results, it can be suggested that short ICI contributes to SI. In a recent study, we proposed that LAI also may contribute to SI and that the effect depends on co-activation (Voller et al. 2005).

In this study, we wanted to explore if SAI also exists during movement and if there is a difference between stimulating near or distant to the target muscle in the surround of the intended movement in healthy persons. Moreover, the effects of stimulation were calculated in relation to the background electromyography (EMG) found in the unintentionally co-activated target muscle. If SAI increases in the target muscle in the surround of the movement, it may possibly contribute to SI or be greater as a consequence of more effective SI.

Methods

We studied 20 healthy volunteers (11 men, 9 women; mean age 32.5 years, range 22–50 years). All except one, were right-handed; all gave written informed consent. The protocol was approved by the NINDS IRB.

Recording

During the whole experiment, subjects were seated comfortably with both hands lying prone on their lap which was covered by a soft pillow. Surface EMG activity was recorded from the abductor digiti minimi (ADM), the flexor digitorum superficialis muscle (FDS), the first dorsal interosseus (FDI), and the extensor indicis prorius (EIP) muscles of the dominant hand (Sohn and Hallett 2004a; Voller et al. 2005), using silver-silver chloride surface EMG electrodes placed over the muscles in a belly-tendon montage. EMG signals were amplified using a Nicolet Viking electromyograph (Skovlunde, Denmark) and bandpass filtered between 10 and 2,000 Hz. Signals were digitized at a frequency of 5 kHz and fed into a laboratory computer for further off-line analysis.

Transcranial magnetic stimulation

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). The coil was positioned on the scalp over M1 at the optimal site for evoking maximal amplitude MEPs from the ADM (hot spot). TMS was always performed with the ADM at rest except for the unintentional co-activation during movement of digit 2. As measures of cortical excitability, resting motor threshold (RMT) and MEP amplitude were determined. Individual RMT was defined as the minimal stimulus intensity required producing MEPs of >50 μV in at least five of ten consecutive trials. MEP size at rest was determined by averaging peak-to-peak amplitudes over 25 single trials for each session. TMS over the ADM hot spot was used to simultaneously measure corticospinal output to the ADM and FDI (with and without movement of digit 2), based on the assumption that the RMT for the FDI is generally lower than for the ADM and the FDI hot spot is anatomically close to the ADM hot spot. TMS stimulus intensity was set at 140% of the individual RMT of the ADM.

Peripheral cutaneous stimulation

Peripheral cutaneous stimulation was performed using ring electrodes around the interphalangeal joints of digits 2 and 5. The stimuli were applied at 200% of perception threshold (Classen et al. 2000). The ring electrodes were connected to a Nicolet Viking EMG machine (Skovlunde, Denmark). A stimulus was defined as being homotopic if it was applied to the finger related to the target muscle (digit 5 for ADM; digit 2 for FDI) and heterotopic if the peripheral stimulation was applied to the finger distant from the target muscle (digit 2 for ADM; digit 5 for FDI) regardless whether digit 2 was moved or not.

Experiment 1: effect of homotopic and heterotopic stimulation at rest and during movement on corticospinal output

The experiment consisted of six trial blocks. Each block consisted of 25 MEPs. Sixteen subjects (10 men and 6 women, aged 22–50 years) participated. The first three trial blocks were performed at rest in random order (no peripheral stimulation = control, digit 2 stimulation, and digit 5 stimulation). In the conditioning trials at rest, stimuli were applied 20 ms prior to the onset of TMS. During the movement trials the subjects were asked to flex their second finger about 2 cm down into the pillow. Before testing, during active movement, the stimulator output was adjusted in each subject to produce the same MEP size compared to the MEP size at rest. The rationale for this adjustment was that inhibition tested by TMS also depends on the magnitude of the test stimulus. Therefore, with the change of circumstance (from rest to movement of digit 2), the test stimulus response was set to the same level as in the (former) rest condition. After obtaining the mean size of the MEP during movement, the stimulator output was adjusted in steps of 2%. The mean of 20 MEPs was calculated after each step. Adjustment was stopped as soon as the mean ADM MEP during movement of digit 2 reached the size of the mean MEP size at rest. After adjustment, the

three movement trials were randomly performed (no peripheral stimulation = control, digit 2 stimulation, and digit 5 stimulation).

During movement, TMS was triggered by the incoming EMG signal of the FDS (self-triggered TMS) by using a LabVIEW program (National Instruments, Austin, TX, USA) and a Schmidt discriminator. The sensitivity of the Schmidt discriminator was set at a level sufficient to detect onset of EMG activity and not to produce triggering while resting (usually 100 μV peak-to-peak EMG amplitude). Subjects were asked to flex the index finger briefly with a self-paced random delay after the 'go' signal which was presented every 5–9 s. There was instruction not to react immediately. The self-triggered TMS was set to activate at an interval of 20 ms between EMG onset and TMS pulse. Electrical finger stimulation was delivered at the onset of movement activity so as to occur 20 ms prior to activation of TMS (Fig. 1a).

Although finger movement is impossible with absolute isolation, subjects were repeatedly asked to keep their fifth digit relaxed during all trials to maintain unintentional co-activation as minimal as possible. To document any unintentional co-activation from the ADM, background EMG activity in the 20 ms period before TMS of each individual stimulated trial was recorded (Fig. 1b).

The primary endpoint measure was the change in size of the MEP evoked by TMS in the ADM compared to the FDI during volitional flexion of the index finger accompanied by electrical stimulation of either the index or the little finger 20 ms prior to TMS.

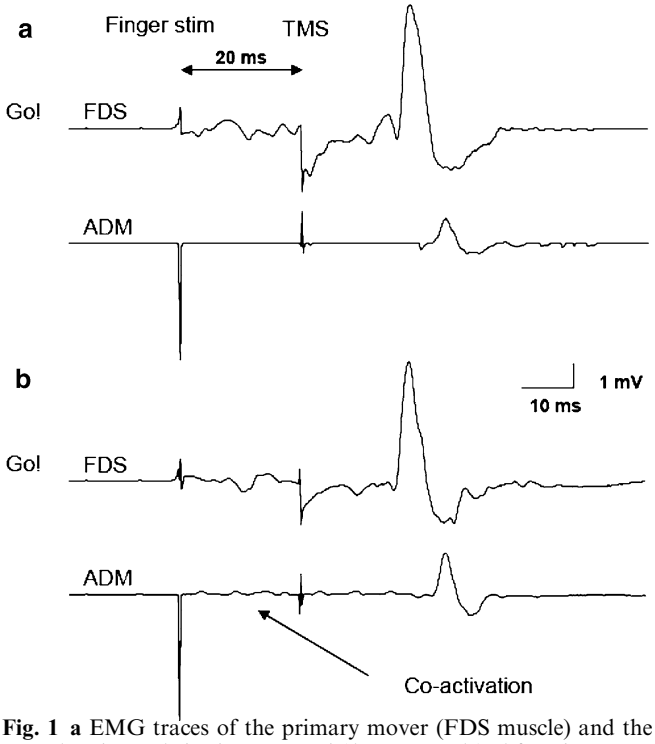

Fig. 1 **a** EMG traces of the primary mover (FDS muscle) and the nonrelated muscle in the surround (ADM muscle). After the "Go!" signal, subjects flexed their index finger and were told to keep the other fingers and in particular, the little finger, as relaxed as possible. The electric stimulus was triggered with movement onset and applied to the finger (here little finger). This peripheral stimulus was followed 20 ms later by a TMS pulse over the scalp. Note in the recordings the stimulus artefacts of both stimuli followed by the MEP. **b** Same setting as in **a**. In this trial, some unintentional co-activation in the nonrelated ADM muscle (*arrow*) was present during movement of digit 2. MEP amplitude of the ADM was higher in this trial than in **a**, as an effect of the (co-)activation during the magnetic stimulation

Experiment 2: effects of homotopic and heterotopic stimulation on *F*-wave amplitude

The effects of homotopic and heterotopic stimulation on spinal excitability at rest and during movement were tested by *F*-waves in seven subjects (four men and three woman, aged 22–50 years), one of whom also participated in experiment 1. Surface EMG was recorded from the ADM muscle of the right hand. *F*-waves were elicited by supramaximal stimulation of the ulnar nerve at the wrist (constant current pulse, 0.2 ms). Six blocks were randomly tested: ulnar nerve stimulation alone or with preceding stimulation of digits 2 or 5 using an ISI of 13 ms (three blocks at rest and three blocks during movement) in order to rule out that the changes of SAI are of spinal origin. Using TMS at an ISI of 20 ms, the conditioning impulse arriving at the cortex would have an effect on the alpha motor neuron output about 6 ms after TMS [central motor conduction time (CMCT)]. In that case the conditioning stimulus would have an effect on the alpha motor neuron 26 ms later. Using supramaximal stimulation of the ulnar nerve at the same ISI of 20 ms the conditioning impulse arriving at the spinal cord would have an effect on the alpha motor neuron output about 13 ms later. In that case the conditioning stimulus would have an effect on the alpha motor neuron 33 ms later (20 ms ISI plus 13 ms for conduction from the periphery to the spinal cord) which is 7 ms later than the effect measured using TMS over the cortex. Therefore, with the intention to get the effect of the conditioning stimulus on the alpha motor neuron with supramaximal stimulation of the ulnar nerve at the same time as with TMS over the cortex, we shortened the ISI from 20 to 13 ms. For the movement trials, subjects were asked to contract the target muscle at about 10–15% of maximal isometric voluntary force by flexing digit 2 against a force transducer wired for feedback into an oscilloscope. The trials were presented in random order, not faster than 0.2 Hz, with 24 trials for each condition.

Data analysis

For comparisons between the effects of movement and stimulated finger on the MEP of the ADM and FDI, we used a repeated measure analysis of variance (rmANOVA)

(MOVE × SITE × TARGET, 2×2×2) with MOVE (rest, movement) as the movement condition, SITE (digit 2, digit 5) as the location of the conditioning stimulus and TARGET as the target muscle on which the MEP was measured (ADM, FDI). Results for MEPs are expressed as mean ± SE. MEPs of the stimulated trials were tested against the control MEP amplitude using the paired *t* test and Bonferroni correction for multiple comparisons.

Unintentional co-activation level was calculated as the proportion of the entire motor neuron pool. Therefore, r.m.s. amplitude of the ADM background EMG activity in the 20-ms period before TMS of every single trial was divided by the individual CMAP amplitude (= percentage of CMAP) to account for variation in co-activation between subjects. To assess the effect of co-activation level on the MEP of the ADM, all conditioned movement trials were categorized (low and high co-activation) and *t* tests were applied.

The effect of homotopic and heterotopic stimulation on ADM *F*-waves at rest and during movement was analyzed using rmANOVA with MOVE (rest, movement) as the movement condition and SITE (digit 2, digit 5) as the location of the conditioning stimulus.

Results

Experiment 1: Homotopic and heterotopic stimulation at rest and during movement

During movement, before adjusting the stimulator output, mean MEPs increased significantly in the movement-related muscles compared to the individual baseline value (FDS 201 ± 20%, FDI 292 ± 40%, EIP 236 ± 34%, all mean ± SE, $P < 0.01$) but not in the ADM (148 ± 25% SE, $P > 0.05$). The mean value for stimulator output adjustment was −1.25% (± 1.91% SE) or from 140% to average 139% of the RMT (in ten subjects up, in six down). The averages of the ADM MEP amplitude in all conditions (including movement before and after adjustment) are shown in Table 1. In the rmANOVA (SITE × TARGET × MOVE), there was a significant effect in SITE ($F_{(1,15)} = 19.2$, $P < 0.01$), TARGET ($F_{(1,15)} = 5.2$, $P < 0.05$) and MOVE ($F_{(1,15)} = 68.3$, $P < 0.01$). Significant interactions were found between SITE × TARGET ($F_{(1,15)} = 6.3$, $P < 0.05$), MOVE × TARGET ($F_{(1,15)} = 4.9$, $P < 0.05$), and SITE × TARGET × MOVE ($F_{(1,15)} = 17.6$, $P < 0.01$) (Fig. 2)

At rest in the stimulated trials compared to the corresponding control trials, paired *t* tests (Bonferroni corrected) revealed significantly reduced MEP amplitudes with homotopic stimulation in the ADM and FDI ($P < 0.001$) (Fig. 2, left graph, black bars). Heterotopic stimulation at rest also reduced MEP, but was significant only in the ADM ($P < 0.001$) (Fig. 2, left graph, black bars). During movement, reduced MEP amplitude was found only in the ADM with homotopic stimulation ($P < 0.001$) (SAI with movement).

MEPs in relation to unintentional co-activation during movement

Off-line analysis of EMG recordings in the 20-ms period before the TMS pulse showed that ADM was not (co-)activated at rest. During movement, background EMG recordings of the ADM showed no or a small and brief co-activation in all subjects in all movement conditions. Figure 1b demonstrates a typical example of unintentional co-activation in the ADM (lower graph) during movement of digit 2. The mean co-activation level (= percentage of CMAP) was 0.31%. In 50% of all movement trials, co-activation level was less than 0.25%. In this group of movement trials, ADM MEP was further inhibited with homotopic stimulation but not with heterotopic stimulation (*t* test, $P < 0.01$) (Fig. 2, right graph).

F-wave measurements

F-wave amplitudes were only measured in the ADM. As expected, *F*-wave amplitudes were higher during movement (+ 26%; paired *t* test, $P < 0.01$). In all stimulated trials (rest and movement), amplitudes tended to be higher than in the control trials (n.s.) (Fig. 3). In the rmANOVA of F-wave amplitudes in the ADM, there was no change in MOVE, SITE, and MOVE x SITE interaction.

Discussion

Short-latency sensory nerve stimulation *at rest* inhibits corticospinal output more consistently with homotopic stimulation than with heterotopic stimulation, a finding similar to previous studies (Classen et al. 2000; Tamburin et al. 2001).

Table 1 Mean MEP amplitudes (mV ± SE) of the unconditioned and conditioned trials

	Rest baseline	Rest conditioned	Movement unadjusted	Movement baseline	Movement conditioned
Homotopic	2.36 ± 0.09	1.94 ± 0.10	3.25 ± 0.16	2.55 ± 0.11	2.24 ± 0.11
Heterotopic		2.22 ± 0.11			2.74 ± 0.12

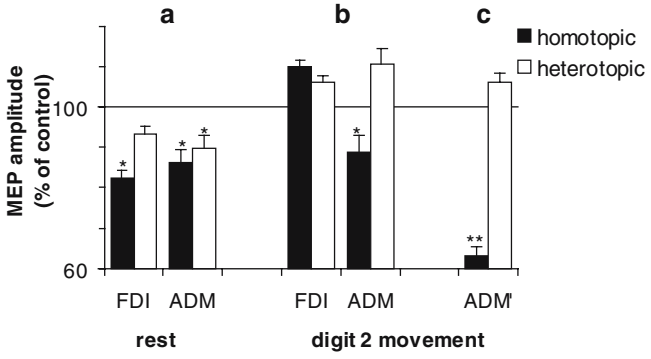

Fig. 2 Effect of homotopic (*black bars*) and heterotopic stimulation (*white bars*) on group mean MEP amplitudes (±SE) of the moving FDI and the ADM muscle in the surround at rest and during movement of digit 2. MEP amplitudes are standardized to the mean MEP of the control trials without stimulation (*dashed line*). **a** At rest there is inhibition with homotopic more than with heterotopic stimulation. (ANOVA, $*P < 0.01$). **b** During movement there is only inhibition in the ADM with homotopic stimulation ($*P < 0.01$). **c** Effects of stimulation related to the level of unintentional co-activation (percentage of CMAP, see Methods). With less co-activation, homotopic stimulation (*black bar*) further decreases MEP amplitudes. Compared to stimulated trials, at rest the difference reaches significance ($**P < 0.01$) suggesting a specific effect of homotopic SAI related to SI

During movement, the homotopic SAI inhibitory network decreases the corticospinal output to the muscle in the surround but not to the moving muscle. The network would therefore act to diminish the unintended activation of the muscle in the surround. Increased inhibition may occur and this appears to depend on the level of co-activation. Stronger movements result in more co-activation (Slobounov et al. 2002) which would oppose SI more strongly. In addition, in previous studies during movement, only nonsignificant changes of group mean MEP amplitude in the nonrelated hand muscle were found together with a corresponding significant

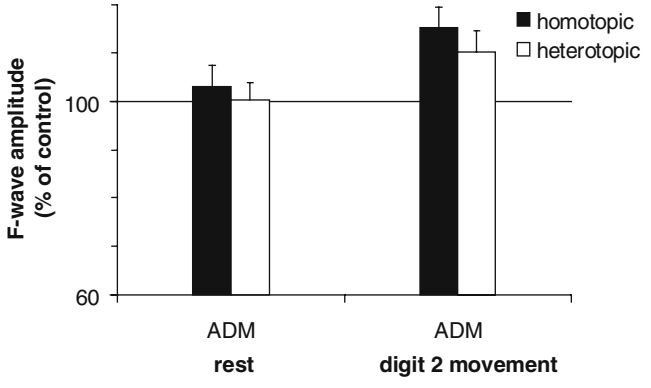

Fig. 3 Effect of homotopic (*black bars*) and heterotopic stimulation (*white bars*) on group mean *F*-wave amplitude (±SE) of the ADM at rest and during movement of digit 2. Amplitudes are standardized to the mean *F*-wave of the control trials without stimulation (*dashed line*). No significant differences were found between the measurements

increase in the movement-related muscles FDS, FDI, EIP (Sohn and Hallett 2004a; Voller et al. 2005). The increased group mean MEP in the ADM in our study was also not significant, although there was an increase of ADM MEP during movement in 6 of our subjects before adjusting the test stimulus. The important point seems to be that MEP amplitude of ADM increased much less than that found in the movement-related muscles FDS, FDI, EIP. This indicates that SI may be rather a relative than an absolute phenomenon. Some of the increased MEP amplitude is due to a generalized increase in spinal excitability. This increase possibly masks processes occurring at the cortical level (Sohn and Hallett 2004a).

Corresponding to previous reports, the amount of co-activation determines the facilitatory effect on motor output of the nonrelated muscle (Stinear and Byblow 2003; Sohn and Hallett 2004a; Voller et al. 2005). In these studies, subjects were instructed not to use their nonrelated target muscle. Our findings corroborate the concept that less co-activation reflects more effective SI. Additionally, we demonstrated that the effect of SAI is enhanced with less co-activation. Therefore, during movement, homotopic SAI may either contribute to SI or is greater as a consequence of more effective SI.

The site of interaction of the digital nerve stimulus and the TMS at ISI 20 ms could be cortical or subcortical or a combination of the two. Different intervals have been used in previous studies. Inhibition was found in healthy volunteers at ISI 18 ms but was absent in patients with Parkinson's disease (Delwaide and Olivier 1990). Inhibition of the TMS response by a preceding digital nerve stimulus was repeatedly reported at ISI 20 ms (Tamburin et al. 2001; Tamburin et al. 2002; Tamburin et al. 2003). On the other hand, in another study, the minimum ISI at which a digital nerve stimulus could suppress MEPs evoked by TMS was found to be 22 ms (Tokimura et al. 2000). In that study, cervical epidural recordings of the TMS response demonstrated the major inhibitory effect to be on the cortical I2 and I3 waves. Consequently, the authors argued that MEPs recorded from a small hand muscle could be reduced even if the TMS is given before the arrival of the sensory volley at the cortex. In our study, we found a significant inhibition at ISI 20 ms. It may be conceivable that we would have found stronger inhibition by adding 2–3 ms for conduction time between fingers and wrist to the ISI resulting in an optimal ISI of 22–23 ms.

At the investigated ISI, no consistent changes in spinal motor neuron excitability have ever been reported. We conducted *F*-wave amplitude measurements at rest and during tonic contraction. Adding the conditioning stimulus only revealed trends toward excitation either at rest or during movement. Hence, spinal excitability, if anything, goes the opposite direction of what we are trying to explain. Thus, we conclude that the enhanced SAI during movement may be in part cortical, in part subcortical, but is likely not spinal. Nevertheless, it also has to be mentioned that interpretation of *F*-wave

measurements in this regard has limitations. The population of motoneurones tested by F-waves is probably not exactly the same as that activated by TMS, and there is even some doubt about the *F*-wave being a good indicator for short-term modulation of spinal motoneuronal excitability (Lin and Floeter 2004).

The heterotopic SAI network during movement leads to enhanced corticospinal output in the representation of the target muscles, suggesting that a different mechanism is involved at rest than with homotopic stimulation. In an earlier study, we found that LICI is also markedly diminished with movement (Sohn and Hallett 2004a). It appears that movement leads to two types of effects with inhibitory mechanisms, either slight increases in inhibition or marked loss of inhibition. The latter effect certainly makes individuated movement more difficult.

These new findings extend the work of others (Classen et al. 2000; Tokimura et al. 2000; Tamburin et al. 2001; Stinear and Byblow 2003; Sohn and Hallett 2004a; Voller et al. 2005) by demonstrating that the neural system mediating SAI appears to differently influence modulation of corticospinal output to the surround during movement compared to at rest. It is also another example of the role that somatosensory integration plays during volitional movement. This may be important for analysis of various movement disorders particularly those in which either loss of somatosensory integration was demonstrated during movement (Sohn and Hallett 2004b) or different effects of peripheral nerve stimulation on M1 excitability have been shown at rest (Delwaide and Olivier 1990; Di Lazzaro et al. 2002; Tamburin et al. 2002; Sailer et al. 2003).

Acknowledgements BV was supported by the Max-Kade-foundation through the Austrian Academy of Sciences. We thank D.G. Schoenberg, M.Sc., for skilful editing.

References

Chen R (2004) Interactions between inhibitory and excitatory circuits in the human motor cortex. Exp Brain Res 154:1–10

Classen J, Steinfelder B, Liepert J, Stefan K, Celnik P, Cohen LG, Hess A, Kunesch E, Chen R, Benecke R, Hallett M (2000) Cutaneomotor integration in humans is somatotopically organized at various levels of the nervous system and is task dependent. Exp Brain Res 130:48–59

Delwaide PJ, Olivier E (1990) Conditioning transcranial cortical stimulation (TCCS) by exteroceptive stimulation in parkinsonian patients. In: Streifler MB, Korczyn AD, Melamed E, Youdim MBH (eds) Advances in neurology, vol 53. Raven, New York, pp 175–181

Di Lazzaro V, Oliviero A, Tonali P, Marra C, Daniele A, Profice P, Saturno E, Pilato F, Masullo C, Rothwell JC (2002) Noninvasive in vivo assessment of cholinergic cortical circuits in AD using transcranial magnetic stimulation. Neurology 59:392–397

Engel KC, Flanders M, Soechting JF (1997) Anticipatory and sequential motor control in piano playing. Exp Brain Res 113:189–199

Fish J, Soechting JF (1992) Synergistic finger movements in a skilled motor task. Exp Brain Res 91:327–334

Hager-Ross C, Schieber MH (2000) Quantifying the independence of human finger movements: comparisons of digits, hands, and movement frequencies. J Neurosci 20:8542–8550

Hallett M (2003) Surround inhibition. Suppl Clin Neurophysiol 56:153–159

Kobayashi M, Ng J, Theoret H, Pascual-Leone A (2003) Modulation of intracortical neuronal circuits in human hand motor area by digit stimulation. Exp Brain Res 149:1–8

Lin JZ, Floeter MK (2004) Do F-wave measurements detect changes in motor neuron excitability? Muscle Nerve 30:289–294

Sailer A, Molnar GF, Cunic DI, Chen R (2002) Effects of peripheral sensory input on cortical inhibition in humans. J Physiol (Lond) 544:617–629

Sailer A, Molnar GF, Paradiso G, Gunraj CA, Lang AE, Chen R (2003) Short and long latency afferent inhibition in Parkinson's disease. Brain 126:1883–1894

Slobounov S, Johnston J, Chiang H, Ray W (2002) The role of submaximal force production in the enslaving phenomenon. Brain Research 954:212–219

Soechting JF, Flanders M (1992) Organization of sequential typing movements. J Neurophysiol 67:1275–1290

Sohn Y, Hallett M (2004a) Surround inhibition in human motor system. Exp Brain Res 158:397–404

Sohn YH, Hallett M (2004b) Disturbed surround inhibition in focal hand dystonia. Ann Neurol 56:595–599

Stinear CM, Byblow WD (2003) Role of intracortical inhibition in selective hand muscle activation. J Neurophysiol 89:2014–2020

Tamburin S, Fiaschi A, Andreoli A, Forgione A, Manganotti P, Zanette G (2003) Abnormal cutaneomotor integration in patients with cerebellar syndromes: a transcranial magnetic stimulation study. Clin Neurophysiol 114:643–651

Tamburin S, Manganotti P, Marzi CA, Fiaschi A, Zanette G (2002) Abnormal somatotopic arrangement of sensorimotor interactions in dystonic patients. Brain 125:2719–2730

Tamburin S, Manganotti P, Zanette G, Fiaschi A (2001) Cutaneomotor integration in human hand motor areas: somatotopic effect and interaction of afferents. Exp Brain Res 141:232–241

Tokimura H, Di Lazzaro V, Tokimura Y, Oliviero A, Profice P, Insola A, Mazzone P, Tonali P, Rothwell JC (2000) Short latency inhibition of human hand motor cortex by somatosensory input from the hand. J Physiol (Lond) 523:503–513

Voller B, St Clair Gibson A, Lomarev M, Kanchana S, Dambrosia J, Dang N, Hallett M (2005) Long-latency afferent inhibition (LAI) during selective finger movement. J Neurophysiol 94:1115–1119