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RESEARCH ARTICLE

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Delay of the execution of rapid finger movement by magnetic stimulation of the ipsilateral hand-associated motor cortex

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Abstract We investigated the influence of focal transcranial magnetic stimulation (TMS) of the hand-associated motor cortex on the execution of ipsilateral finger-lifting movements in six humans. In a simple reaction time paradigm, suprathreshold TMS (1.6- to 2.1-fold of the response threshold determined at rest) was performed at intervals of 40, 70, 80, 90, and 100 ms after the auditory "go" signal. Movement onset was measured with an accelerometer. TMS delayed the execution of ipsilateral finger movement when the cortex stimulus preceded the onset of the intended movement by about 25–65 ms. Taking the corticomuscular conduction times to the activated muscles into account, TMS suppressed the output from the motor cortex in a period 6–45 ms after the contralateral motor cortex was stimulated. Such timing would be compatible with an interhemispheric inhibition similar to the previously described ipsilateral inhibition of ongoing tonic motor activity. The delay of the movement was 40 ms. The function of the neuronal structures mediating interhemispheric inhibition might be to suppress the coactivation of the other hand during unilateral finger movements within bimanual motor tasks.

Key words Motor cortex · Corpus callosum · Interhemispheric inhibition · Ballistic finger movement · Transcranial magnetic cortex stimulation

Introduction

Transcranial magnetic stimulation (TMS) of the human motor cortex has been shown to delay the onset of fast voluntary flexion or extension movements of the contralateral wrist without affecting the pattern of agonist and antagonist EMG bursts. The underlying inhibitory process takes place close to corticospinal output neurons and has been suggested to result from inhibition of a group of strategically placed neurons that prevent motor programs from reaching the corticospinal neurons (Day et al. 1989; Rothwell et al. 1989; Ziemann et al. 1997). The question arose whether TMS of the hand-associated motor cortex also influences the execution of movements of the ipsilateral hand, similar to short-term suppression of tonic EMG activity in ipsilateral hand muscles. The latter phenomenon has been attributed to interhemispheric inhibitory interactions of the motor cortices, based on studies with stimulation mapping of their cortical origin and of their impairment in patients with focal brain lesions (Boroojerdi et al. 1996; Ferbert et al. 1992; Meyer and Röricht 1996; Meyer et al. 1998). We observed that TMS of the right motor cortex interfered with the lifting of the right index finger in a simple reaction time task and attributed this to interhemispheric motor inhibition.

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Materials and methods

With ethics committee approval and informed consent, the study was carried out on eight healthy, right-handed volunteers (two male; age range 23–27 years).

Reaction times

The subjects sat in a chair with both forearms resting on supports and with the hands pronated. They were trained to lift the index finger of the right hand by about 25° in reaction to an auditory stimulus (1-kHz tone, lasting 50 ms) which occurred at randomized intervals of 5–7 s.

Movement onset was measured with a precision piezo AC-coupled resistive accelerometer (model 3021, IC sensor, London, UK) fixed on the nail of the right index finger. EMG activity was recorded with bipolar surface electrodes (area 26 mm^2) from the first dorsal interosseous (FDI) and extensor indicis proprius (EIP) muscles. Data were collected with a personal computer using a CED 1401 interface and a data collection program (Spike 2, sampling frequency of 5000 s^{-1} per channel).

Reaction times were determined from the accelerometer signal since the EIP was the prime mover muscle (Tomberg et al. 1991) in only 66% of the subjects. The relationship between movement

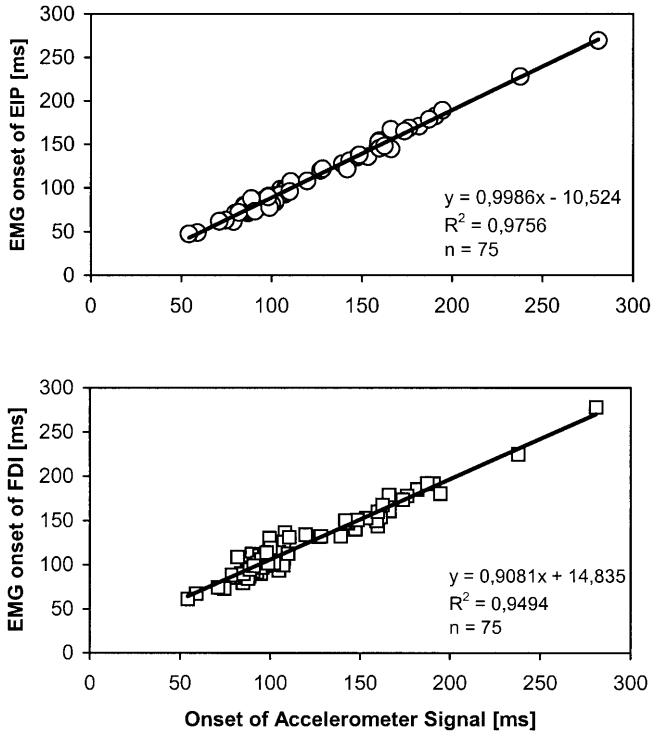

Fig. 1 Onset of lifting the index finger as determined from an accelerometer signal in relation to the onset of EMG activity in the extensor indicis proprius (EIP, *upper box*) and first dorsal interosseus muscle (FDI, *lower box*). In this exemplary case, EMG activity in the EIP preceded movement onset and identified the EIP as prime mover muscle

onset as determined from this signal and from the onset of EMG activity in the FDI and EIP was always linear as shown for one subject in Fig. 1. In this case EMG onset in the EIP preceded the accelerometer signal by 11 ms on average (106±42 ms vs 117±42 ms), while the EMG onset in the FDI (121±39 ms) occurred on average 4 ms after movement onset (Fig. 1).

Subjects were trained for the reaction time task in about 100 trials. Then in 50% of the following 500 reactions TMS of the right-hand-associated motor cortex was performed with different intervals between auditory tone and cortex stimulus (tone-stimulus intervals, TSIs: 40, 70, 80, 90, 100 ms). Within blocks of 100 trials each, the order of 50 control trials and 50 test trials was randomized. Furthermore the TSIs were randomized between the different blocks in the different subjects. Throughout the experiment, a pause of about 5 min was taken after blocks of 100 reactions.

Influence of stimulus intensity on reaction times of finger lifts ipsilateral to cortex stimulation was investigated in two subjects for intensities related to the individual motor threshold. In this experiment the TSI was fixed at 90 ms.

To exclude the possibility that changes in reaction time were due to a non-specific influence of TMS on brain function, stimulation was also performed in three subjects with the coil centered 4 cm right and 10 cm posterior to the vertex. We were aware that stimulating at such a position would not reflect a pure control condition since stimulation above the posterior parietal cortex could interfere with the processes of perception, planning and execution of reaction time movements. However, stimulating over more posterior or more lateral positions or over the forehead would have been painful and might have interfered with the performance of the reaction time task.

Reaction time was measured as the interval between auditory stimulus and first deflection of the accelerometer signal above 0.04 ms^{-2} (Fig. 2). For each experimental condition histograms of the frequency of reaction times were constructed with a bin width of 5 ms.

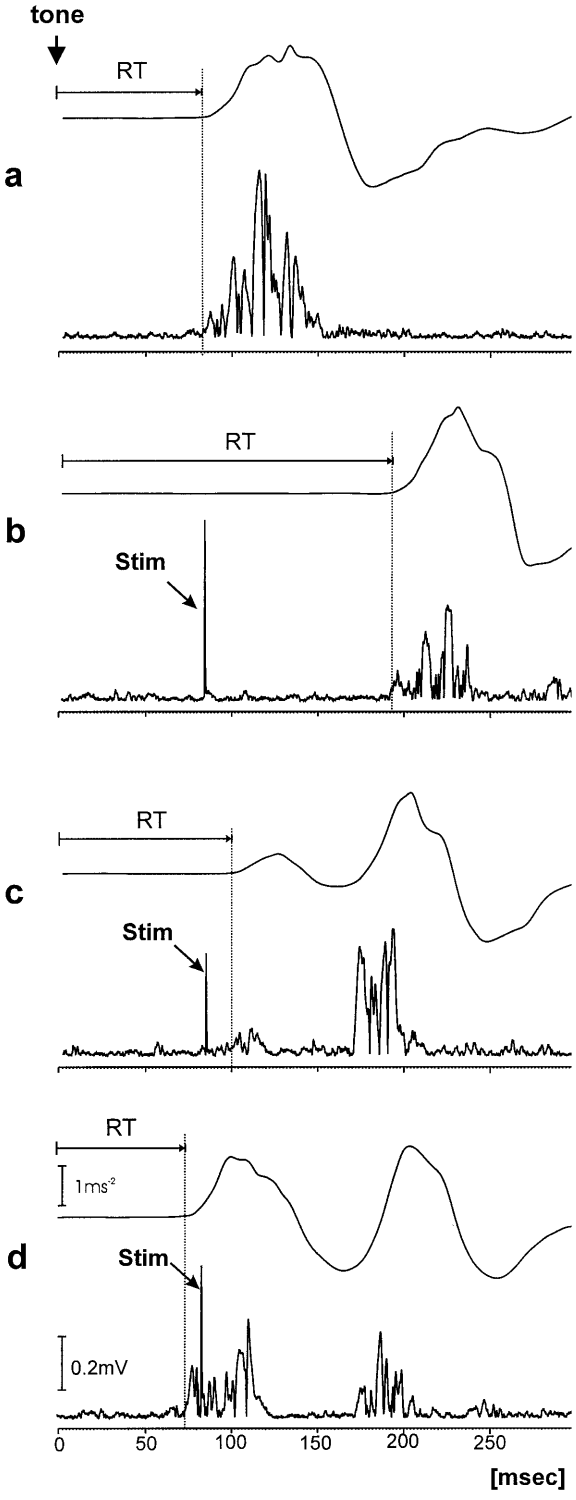

Fig. 2a–d Index finger lifting movement in reaction to a tone as described by the accelerometer signal (*upper trace*) and the rectified EMG activity in the FDI (*lower trace*). Examples of an undisturbed movement **(a)**, a movement delayed by TMS (*Stim*) of the ipsilateral motor cortex **(b)**, and ongoing movements interrupted by TMS **(c,d)** (*RT* reaction time)

Transcranial cortex stimulation

Single-pulse TMS of the hand-associated motor cortex was performed with an eight-shaped coil (outside diameter of one half-coil, 8.5 cm) of the Magstim 200 stimulator (monophasic pulses, 2-Tesla version; Magstim Company, Dyfed, UK). Coil currents were directed anteroposteriorly. Motor cortex stimulation took place at the individually determined site at which the largest corticospinally mediated responses could be elicited in the contralateral FDI. In experiments in which the influence of different TSIs was studied, stimulus intensity was set to 80% of the maximum stimulator output (i.e., 1.6- to 2.1-fold of the individual response thresholds determined at rest), because at this intensity transcallosal inhibition of tonic EMG activity regularly occurs in normal subjects (Meyer et al. 1998).

When studying the influence of stimulus strength on the reaction times of ipsilateral movements, the stimulus intensity was related to the individual motor threshold (T) for eliciting electromyographic hand motor responses at rest (1.2, 1.3, 1.5, 1.8 T).

Results

The distribution of the reaction times of finger lifts to an auditory "go" signal was changed by TMS of the ipsilateral hand-associated motor cortex but not by stimuli applied to the ipsilateral parieto-occipital cortex. This is illustrated in Fig. 3, in which the results as derived from the total number of trials across all subjects are displayed in the form of histograms. The distributions of the reaction time movements were not significantly different for the control condition paralleling different TSIs in the different subjects ($P>0.2$, ANOVA). The changes of the occurrence of movement onsets in distinct bins can also be visualized by subtracting the number of reactions under control conditions from those occurring under stimulation conditions (Fig. 4). Such a display also shows that the period with reduced movement initiations is followed by a period of a similar length in which the number of movement onsets is increased (Fig. 4). This indicates that the execution of the movement was delayed by TMS rather than that the command to move was lost.

Reaction times did not significantly differ between both control conditions (movement without TMS; movement with TMS over the ipsilateral parieto-occipital region with a TSI of 90 ms) and were 141±41 ms and 148±50 ms, respectively ($P>0.2$, *t*-test) (Fig. 3).

TMS over the ipsilateral motor cortex with a TSI of 40 ms reduced the occurrence of reaction times in comparison to the control condition between 85 and 114 ms, i.e., the first six bins of the frequency distribution of reaction times. The occurrence was reduced by on average 91% (range 81–100%). Stimulation with a TSI of 70 ms reduced the occurrence of reaction times between 90 and 129 ms by on average 80% (range 59–100%) (Fig. 3).

Stimulation over the ipsilateral motor cortex with TSIs of 80, 90 or 100 ms caused a gap in the frequency distribution of reaction times (Fig. 3). When for these TSIs the occurrence of reaction time movements was aligned to the cortex stimulus and when inaccuracies arising from a bin width of 5 ms were also considered, then the initiation of reaction time movements was reduced within a period lasting 30–39 ms that followed the

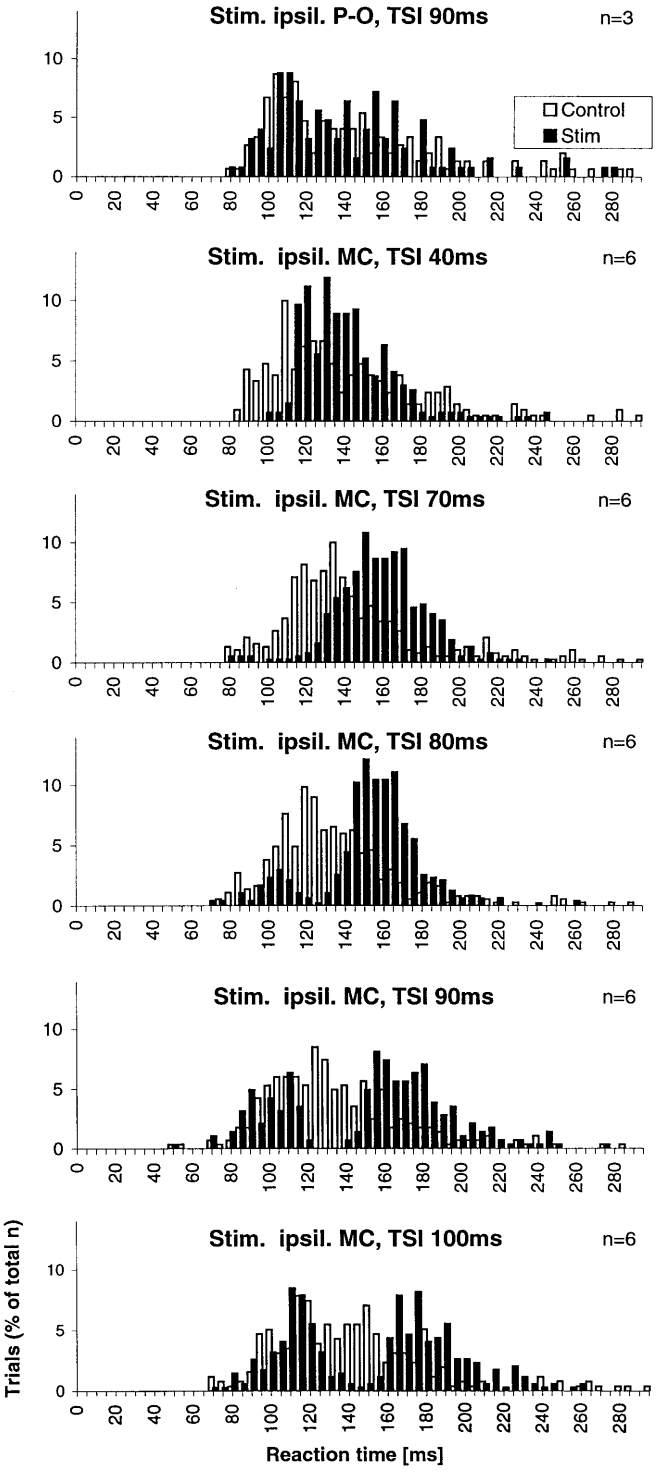

Fig. 3 Distribution of reaction times of lifting the index finger to a tone ("go" signal) when the ipsilateral parieto-occipital (*P-O*) or primary motor cortex (*MC*) was stimulated transcranially at different tone-stimulus intervals (*TSI*) (*black vertical bars*). For comparison also the reaction times of undisturbed movements are given (control, *white vertical bars*). Results as derived from the total number of trials across three (*P-O*) or six (*MC*) subjects. Note that stimulation of the ipsilateral parieto-occipital cortex did not influence reaction times while TMS of the ipsilateral motor cortex caused a reduction of reactions in a period about 25–65 ms after the cortex stimulus (see TSIs of 80–100 ms). The bin width of the histograms is 5 ms

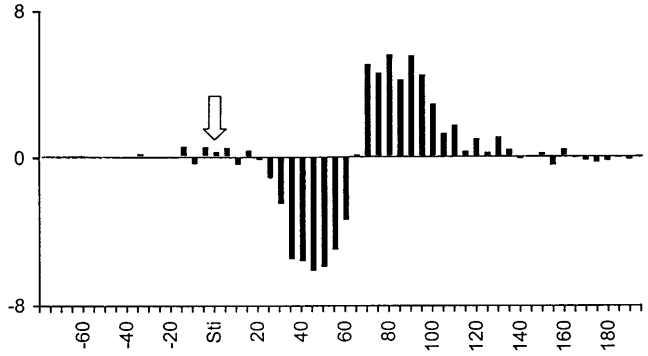

Fig. 4 Changes of the frequency of movement onsets in distinct bins of reaction times of the histograms in Fig. 3 elicited by TMS of the ipsilateral motor cortex (*y*-axis: percentage of total number of reactions in a distinct bin after TMS minus percentage of total reactions in a distinct bin under control conditions). Display of pooled data for TSIs of 80, 90, and 100 ms when the changes were aligned to the cortex stimulus (time "0", *arrow*) (*x*-axis values in milliseconds before and after the cortex stimulus). This type of display visualizes that the period of reduced reactions (values below zero) is followed by a period of similar length in which the number of reactions is increased (values above zero), indicating a constant delay of the movements

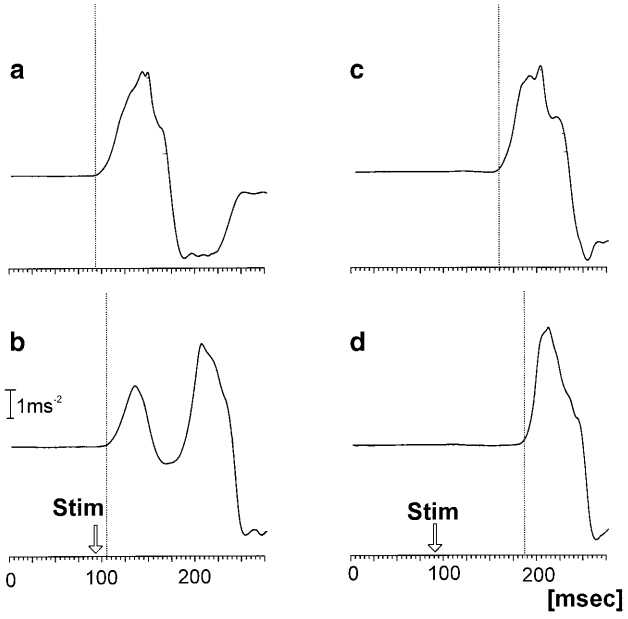

Fig. 5 Averaged accelerometer signals of one subject for four different conditions: short-latency reaction time movement without **(a)** or with **(b)** interfering TMS (*n*=20 each), long-latency reaction time movement without **(c)** or with **(d)** interfering TMS (*n*=10 each). TMS interrupts short-latency reactions and thereby prolongs the overall movement time **(b)** or does not change the duration of the movement in long-latency reactions **(d)**

cortex stimulus by 25–34 ms (Fig. 4). This phase was followed by a phase of similar duration (41–49 ms) in which the previously suppressed movements occurred with a delay.

When stimulating with TMS intensities related to the individual motor threshold (i.e., 1.2, 1.3, 1.5 and 1.8 T, two subjects), the duration of the gap in the distribution of reaction time movements increased by about 10 ms (i.e., two bins) when increasing the stimulus intensity from 1.3 T to 1.8 T. For an intensity of 1.2 T no significant effect of TMS on the distribution of reaction times was observed.

Sometimes TMS interrupted an ongoing finger lift as evidenced by a near complete suppression of EMG activity and disturbance of the movement (Fig. 2c,d). The electromyographic silent period occurred on average 40±3 ms (FDI) or 36±3 ms (EID) after the cortex stimulus and lasted on average 40±7 ms (FDI) or 38±8 ms (EID) (*n*=32 selected trials in two subjects).

To obtain some idea about the question of whether the movement has been delayed or whether the movement has been inhibited for a short period, we averaged the accelerometer signal of one exemplary experiment. Trials were assigned to four different groups (Fig. 5): (a) undisturbed reaction time movement with short reaction time (95±18 ms, *n*=20), (b) cortex stimulation and fast reaction time movement (106±19 ms, *n*=20), (c) undisturbed movement with long reaction time (158±12 ms, *n*=10), and (d) cortex stimulation and movement with late onset (183±21 ms, *n*=10). With all the limitations of analyzing accelerometer signals, short-latency reactions seemed to be mainly interrupted and the whole movement had a longer duration (Fig. 5a,b). A similar duration of the movement was observed when comparing movements with long reaction times for the conditions "without" and "with" cortex stimulation (Fig. 5c,d).

Discussion

Focal TMS of the human hand-associated motor cortex not only inhibits voluntary tonic muscle contraction in ipsilateral hand muscles (Ferbert et al. 1992; Meyer et al. 1998), but also delays or interrupts ipsilateral finger movements in a reaction time task. Delay of movement onset or interruption of EMG activity during the execution of the ballistic movement lasted about 30–40 ms. For intervals of 80–100 ms between the "go" signal and cortex stimulus movement initiation was suppressed when TMS preceded the onset of the intended finger movement by about 25–65 ms. Taking a corticomuscular conduction time of about 20 ms to the first dorsal interosseous or 15 ms to the extensor indicis proprius muscle into account (Kloten et al. 1992), this would mean that the output from the motor cortex generating the movement is suppressed 5–45 ms after the opposite motor cortex has been stimulated. Such timing would be compatible with interhemispheric transfer times between the motor cortices and the duration of transcallosal inhibition that have been estimated from different experimental approaches (Di Lazzaro et al. 1999; Ferbert et al. 1992; Meyer et al. 1998; Thompson et al. 1993). Therefore the observed movement delay might also be caused by transcallosally mediated inhibition. Studies of the cortical origin of the ipsilateral inhibition of tonic EMG activity and the findings in patients with circumscribed lesions of the corpus callosum or corticospinal pathways (Boorojerdi et al. 1996; Meyer and Röricht 1996; Meyer

et al. 1998) have produced a large body of evidence which indicates that this motor inhibition is mediated via the corpus callosum. Such a conclusion is not questioned by the study by Gerloff and colleagues (1998), who found that a conditioning stimulus to the ipsilateral hemisphere reduced the size of EMG responses elicited by direct stimulation of the descending pathways at the brainstem level. As is extensively discussed by Di Lazzaro et al. (1999), ipsilateral cortex stimulation might transcallosally reduce the descending corticospinal drive onto spinal motoneurons and might thereby reduce their susceptibility to stimuli applied to the brainstem. It should be noted that the preceding considerations about latencies and duration of transcallosal motor inhibition are limited by the use of histograms with a bin width of 5 ms. Furthermore movement onset as determined from accelerometer signals was variably related (between –5 ms and +10 ms) to the onset of EMG activity in the FDI and EID, which in different subjects variably served as prime mover muscles in index finger lifting.

The anatomical correlate of transcallosal motor inhibition may be homotopic commissural projections of the hand motor area as such pathways have been identified by electrical stimulation in monkeys (Goldman and Nauta 1977; Jenny 1979). There is also indirect evidence for interconnections of the motor cortices in man. Transcranial stimulation of one motor cortex inhibited the I-wave generation in the opposite cortex (Di Lazzaro et al. 1999) and was followed by blood flow changes, evoked potentials, and electroencephalographic responses in corresponding cortical areas of the other hemisphere (Cracco et al. 1989; Fox et al. 1997; Ilmoniemi et al. 1997). The inhibition of I-wave generation in the other motor cortex was strongest for interstimulus intervals between 7 and 30 ms, and electroencephalographic responses had onset latencies of 8.8–20 ms, both of which would be appropriate for interhemispheric transfers. When assuming a disynaptic pathway – as has been found for commissural projections producing an inhibitory postsynaptic potential (IPSP) in efferent neurons of homologous cat visual cortex (Toyama et al. 1969) – with 2 ms as synaptic transmission time and a distance of 13 cm between the two motor cortices (Cracco et al. 1989) – the conduction velocities of the callosal fibers contributing to the onset of the inhibition would be around 20 m/s. Large-diameter fibers which might conduct with such a velocity have their highest density in the posterior third of the trunk of the human corpus callosum (Tomasch 1954), through which fibers mediating transcallosal inhibition of tonic EMG activity cross the midline (Meyer et al. 1998).

Transcallosal inhibition was found to be most effective in delaying the onset of voluntary movement when it acts in the other motor cortex 55–15 ms before the estimated onset of the intended movement. For comparison, in primates corticomotoneuronal discharges preceded the onset of voluntary EMG activity in target muscles by 70–60 ms (Cheney and Fetz 1980). Similarly, the effectiveness of evoking hand motor response by TMS started to increase about 70 ms before EMG onset within a reaction time movement (Pascual-Leone et al. 1992). During the period of interhemispheric inhibition corticospinal outputs from the motor cortex initiating and executing unidirectional fast finger movements or maintaining a strong tonic pinch grip (Ferbert et al. 1992; Meyer et al. 1998) are blocked by TMS. The complete suppression of EMG activity during this period when using high stimulus intensities suggests that transcallosal inhibition can have access to all the corticomotoneuronal cells that are active during maximal muscle contraction including those that are preferentially fired in other motor tasks. Interestingly, TMS delayed but did not abolish the ability to execute the motor command. This is similar to the findings of a previous study in which TMS delayed contralateral ballistic wrist movements without interfering with the timing of agonist and antagonist EMG bursts (Day et al. 1989). The question of whether transcallosal inhibition interferes with the structure of motor programs was beyond the scope of the present study. However, when analyzing the accelerometer signal recorded during the finger lift, interrupted fast movements appeared to have a longer duration than undisturbed movements of comparable onset latency. In contrast late reaction time movements with and without preceding cortex stimulation had about the same duration and were therefore mainly delayed.

One explanation for the fixed delay of the movement onset of around 40 ms observed for high stimulus intensities could be that there is a minimum time required for rhythmically discharging neurons in rolandic cortex to recover from inhibition. Such activity would roughly be in keeping with the concept of a temporal oscillator controlling the timing of movement (Treisman et al. 1992), which was related to the localized 20-Hz beta activity generated predominantly in the anterior bank of the central sulcus recorded by magnetoencephalography (Salmelin and Hari 1994). This 20-Hz rhythm is blocked by movements and is likely to be related to the conceptual design of the movement to be executed (Niedermeyer 1987; Salenius et al. 1996). To support the hypothesis that TMS blocks rhythmic neuronal activity, the oscillator should not be affected by changes of suprathreshold stimulus intensities. However, this does not seem to be the case in our study since we found that the length of the gap caused by TMS in the reaction time distribution tended to increase by about 10 ms when increasing the stimulus intensity from 1.3 T to 1.8 T. Such a finding may therefore just reflect an inhibition similar to the inhibition of ipsilateral tonic voluntary electromyographic activity, which had a very similar stimulus intensity dependent increase in its duration (Meyer et al. 1995).

Motor inhibition could also be mediated by ipsilateral pathways projecting to the spinal level as they have recently been characterized by TMS in human subjects for connections supplying motoneurons of hand and forearm muscles (Ziemann et al. 1999). One could imagine that activation of the motor cortex might act on the ipsilateral spinal cord and thereby interfere with the execution of

motor commands generated in the contralateral motor cortex without engagement of interhemispheric or other crossing projections. However, one strong argument against a significant contribution of such ipsilateral descending connections to the observed effect is the high thresholds for eliciting ipsilateral excitatory responses in hand and forearm muscles. They were on average 1.8-fold of those for eliciting contralateral motor responses (Ziemann et al. 1999) and thus clearly higher than the intensities (1.3 T) required to produce changes of the distribution of reaction times in our experiments. Inhibition of ipsilateral tonic hand muscle EMG activity also has a lower threshold (1.2±0.2 T, own unpublished observation in nine subjects).

In general it can be concluded that focal TMS is capable of activating interhemispheric connections between the hand motor areas that should be related to hand motor function. This assumption is also supported by impaired bimanual movements in patients with a partial section of the corpus callosum and an absent ipsilateral silent period (Meyer et al. 1998). The neurons producing transcallosal motor inhibition might bring corticospinal neurons into a synchronous refractory state and allow a synchronous return of their excitability afterwards, similar to the previously described phenomenon of premovement inhibition (Conrad et al. 1983). This might support unilateral activation of corticomotoneuronal cells during high-speed unilateral movements in bimanual motor tasks.

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