Motor imagery (MI) is the mental rehearsal of a motor act without overt movement. Using transcranial magnetic stimulation (TMS), we tested the effect of MI on corticospinal excitability in patients with writer’s cramp. In 10 patients with writer’s cramp and 10 healthy controls, we applied focal TMS over each primary motor area and recorded motor evoked potentials (MEPs) from contralateral hand and arm muscles while participants imagined a tonic abduction of the index finger contralateral to the stimulated hemisphere. In healthy controls and patients, the MEP amplitude in the relaxed first dorsal interosseus muscle (FDI) showed a muscle-specific increase during MI; however, the increase was less pronounced in patients than in healthy controls. In addition, in patients but not in controls, the MEP amplitude also increased in hand and forearm muscles not involved in the imagined movement. This abnormal spread of facilitation was observed in the affected and unaffected upper limb. MI of simple hand movements is less efficient and less focussed in patients with writer’s cramp than it is in normal subjects. © 2005 Movement Disorder Society

Key words: writer’s cramp; motor imagery; transcranial magnetic stimulation (TMS); dystonia

Writer’s cramp is a task-specific focal dystonia that usually affects only writing1 and is characterized by a pattern of co-contraction of agonist and antagonist muscles and by recruitment of inappropriate muscles for writing.2 Several studies on patients with writer’s cramp have demonstrated reduced inhibition at various motor system levels,2,3 particularly in the hand primary motor area.4,5 Reduced inhibition at cortical level may be responsible for reduced specificity of the muscle activation during movement. Patients with focal hand dystonia also show abnormalities in movement preparation.6 The motor cortical abnormalities reported in patients with writer’s cramp include a reduction in the amplitude of the negative slope (NS’) component of the movement-related cortical potentials7 and a deficit of event-related desynchronization in the beta band before movement onset.8 There is also an abnormality in the electroencephalography (EEG) activity during the waiting period for a motor paradigm “go-signal” after a “warning signal.” The EEG activity associated with this paradigm is called contingent negative variation (CNV) and is specifically abnormal for the affected body part in dystonia.9,10

Motor imagery (MI) has been defined as the mental rehearsal of a motor act without overt movement.11 MI shares features in common with motor preparation and motor execution12 and functional magnetic resonance imaging (fMRI) studies suggest that MI recruits nearly the same set of brain structures as does voluntary movement.13,14 MI has been studied in normal subjects with magnetic stimulation techniques. MI facilitates corticospinal excitability and does so by decreasing motor threshold, enhancing the motor evoked potential (MEP)
size and reducing intracortical inhibition. These effects are topographically selective for the motor cortex representation of the muscles involved in the MI task. No data are available on MI changes in corticospinal excitability during MI in patients with writer’s cramp. MI has been used recently as a therapeutic tool in musician’s cramps.

To gain further insight into the pathophysiological mechanisms of writer’s cramp, we investigated whether motor strategy in patients with writer’s cramp is altered in the absence of movement. Using transcranial magnetic stimulation (TMS), we tested in patients with writer’s cramp corticospinal excitability during MI. To do so, we studied the effects of MI on the size and threshold of MEPs evoked by TMS in upper limb muscles.

**SUBJECTS AND METHODS**

**Subjects**

We studied 10 patients with simple writer’s cramp (8 men, 2 women; age range 24–72 years; mean age, 43.8 ± 15 years) and 10 age-matched controls (7 men, 3 women; age range, 27–68 years; mean age, 44 ± 9 years), all right-handed. All patients had writer’s cramp on the right side. None of them was under treatment with anticholinergic or antidepressant drugs. The clinical data of dystonic patients are summarized in Table 1. All participants gave written informed consent according to the declaration of Helsinki. The experimental protocol was approved by the local ethics committee.

**Study Design**

We applied focal TMS over each primary motor area and recorded MEPs from contralateral hand and arm muscles while participants imagined a tonic abduction of the index finger contralateral to the stimulated hemisphere. The MI task was carried out after a verbal command and was maintained until the magnetic stimulus was delivered (within 3 seconds). Patients and controls were instructed explicitly to perform kinesthetic MI from an “internal” or first person perspective, which relies on an egocentric representation of action.

To test spinal cord excitability, 15 F waves were recorded from the right APB muscle at rest and during MI.

**TMS and Electromyography Recordings**

Subjects were seated on a reclining armchair with both hands pronated on a pillow. Focal TMS was applied through a figure-of-eight coil with an outer diameter of each wing of 9 cm placed over the left and right motor cortical representational field of the target muscles (first dorsal interosseus [FDI], abductor pollicis brevis [APB], abductor digitii minimi [ADM], extensor carpi radialis [ECR], and biceps [BIC]). The coil was connected to a Magstim 200 monophasic stimulator (Magstim 200; Whitland, Dyfed, UK) and was placed tangentially to the scalp with the handle pointing backwards and laterally at a 45-degree angle to the sagittal plane, thus generating a posterior–anterior current in the brain. Stimulus intensity was set at a stimulator output that induced MEPs of about 0.5 mV in the target muscles. The scalp position

**TABLE 1. Demographic and clinical characteristics of the 10 patients with writer’s cramp**

<table>
<thead>
<tr>
<th>Patient no.</th>
<th>Age (yr)</th>
<th>Gender</th>
<th>Disease duration (yr)</th>
<th>Dystonic pattern</th>
<th>Last treatment with botulinum toxin</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>24</td>
<td>F</td>
<td>1</td>
<td>Thumb adduction, wrist extension</td>
<td>No treatment</td>
</tr>
<tr>
<td>2</td>
<td>30</td>
<td>M</td>
<td>4</td>
<td>Ulnar deviation and index extension</td>
<td>No treatment</td>
</tr>
<tr>
<td>3</td>
<td>37</td>
<td>M</td>
<td>3</td>
<td>Ulnar deviation, wrist extension, elbow lifting</td>
<td>No treatment</td>
</tr>
<tr>
<td>4</td>
<td>72</td>
<td>M</td>
<td>30</td>
<td>Ulnar deviation, 4th and 5th finger flexion, thumb extension</td>
<td>6 months</td>
</tr>
<tr>
<td>5</td>
<td>53</td>
<td>M</td>
<td>7</td>
<td>Ulnar deviation, wrist extension, 4th and 5th finger flexion</td>
<td>No treatment</td>
</tr>
<tr>
<td>6</td>
<td>49</td>
<td>M</td>
<td>15</td>
<td>Ulnar deviation, arm abduction</td>
<td>No treatment</td>
</tr>
<tr>
<td>7</td>
<td>60</td>
<td>M</td>
<td>20</td>
<td>Ulnar deviation, wrist extension, elbow lifting</td>
<td>No treatment</td>
</tr>
<tr>
<td>8</td>
<td>38</td>
<td>M</td>
<td>0.5</td>
<td>Thumb adduction, wrist extension, elbow lifting</td>
<td>No treatment</td>
</tr>
<tr>
<td>9</td>
<td>30</td>
<td>M</td>
<td>3</td>
<td>Ulnar deviation and index extension</td>
<td>No treatment</td>
</tr>
<tr>
<td>10</td>
<td>45</td>
<td>F</td>
<td>8</td>
<td>Ulnar deviation, wrist extension</td>
<td>1 yr</td>
</tr>
</tbody>
</table>

 Movement Disorders, Vol. 20, No. 11, 2005
for eliciting stable MEPs in each target muscle was found, using constant stimulus intensity, by moving the coil over the head in steps of 0.5 cm. The optimal coil position for each muscle studied was marked on the scalp with a red pen. Resting motor threshold (RMT) was defined as the minimum intensity that could evoke a peak-to-peak MEP of 50 µV in at least 5 of 10 consecutive trials in the relaxed muscle. MEPs were recorded from the FDI, APB, ADM, ECR, and BIC muscles of the left and right upper limb. High gain audiovisual electromyography (EMG) monitoring was used to ensure complete muscular relaxation. The trigger frequency for the condition MEP at rest was 0.25 Hz. During the MI condition, the timing of TMS was triggered manually and roughly matched to the onset of the MI task. MI and rest conditions were intermingled pseudorandomly in separate blocks of measurements.

MEPs were recorded using a pair of Ag–AgCl surface electrodes placed over the target muscles, using a belly-tendon montage. Raw signals were amplified and filtered using a time constant of 3 msec and a high-pass filter set at 3 kHz (Neurolog System; Digitimer Ltd., Welwyn Garden City, Herts, UK) and digitalized using a CED 1401 laboratory interface (Cambridge Electronic Design Ltd., Cambridge, UK). Data were collected on a personal computer (Signal 2.0; Cambridge Electronic Design Ltd.) and analyzed offline.

F waves were recorded from APB muscle after supra-maximal stimulation (duration 0.2 msec; frequency 1 Hz) of the right median nerve, applied to the wrist (cathode proximal), using a Digitimer D 180 stimulator.

Data Measurements and Statistical Analysis

Fifteen MEPs were recorded at rest and during MI from the FDI, APB, ADM, ECR, and BIC muscles after stimulation of the left and right hemisphere. For each muscle, peak-to-peak amplitude (mV) of each MEP recorded at rest and during MI were measured offline and the mean amplitudes were calculated (NuCursor software; Sobell Dept. of Motor Neurosciences and Movement Disorders, Institute of Neurology, University College of London, UK).

Data obtained with the different measures of motor excitability and spinal excitability were submitted to repeated-measures analysis of variance (ANOVA). The ANOVA model included the factor condition (before and during imagery, two levels) and the factor muscle (FDI vs. APB vs. ADM vs. ECR vs. BIC; five levels) as within-subject factor and the factor group as between-subject factor. Conditional on a significant F value, posthoc paired-sample t tests were used to explore the strength of main effects and the patterns of interaction between experimental factors. A P value less than 0.05 was considered significant.

RESULTS

All subjects were able to carry out the task and reported that they could maintain intense imagination always using an internal mental strategy.

MI of a Tonic Movement Carried Out With the Right Index Finger in Patients and Healthy Controls

In patients and in healthy controls, MI decreased the RMT (Table 2) and increased the MEP size in patients and controls. Repeated-measures ANOVA showed a significant main effect of condition \( F_{1,18} = 35.1; P < 0.001 \). This effect was caused by an overall increase in the mean peak-to-peak MEP amplitudes during imagery in both groups. Moreover, a three-factorial ANOVA disclosed a significant difference between groups in the topographic specificity of the MEP facilitation because there was a condition-by-group-by-muscle interaction \( F_{4,72} = 9.2; P = 0.001 \).

Separate two-factorial ANOVA for each group with condition (before and during imagery) and muscle (FDI vs. APB vs. ADM vs. ECR vs. BIC) as within-subject factors, conducted to explore within group effects, disclosed in patients a prominent main effect for the factor condition \( F_{1,9} = 16.4; P = 0.002 \) and a significant condition-by-muscle interaction \( F_{4,36} = 6.6; P < 0.001 \). Also in healthy controls, ANOVA demonstrated a main effect for the factor condition \( F_{1,9} = 18.5; P = 0.002 \) and a condition-by-muscle interaction \( F_{4,36} = 15.6; P < 0.001 \).

A two-way ANOVA computed for each muscle to test muscle-specific differences in the MEP facilitation during MI between patients and controls, using the factors group and condition, disclosed a group-by-condition interaction for the FDI muscle \( F_{1,18} = 8.6; P = 0.008 \) due to a less significant increase in MEP amplitude during MI in the FDI muscle in patients than in controls (Fig. 1A,B). The two-way ANOVA also identified a group-by-condition interaction for the ADM muscle \( F_{1,18} = 6.4; P = 0.02 \) and ECR muscle \( F_{1,18} = 5.7; P = 0.02 \) indicating a more widespread MEP amplitude facilitation during MI in patients than in controls. In the remaining muscle, ANOVA identified no significant difference in the MEP amplitude facilitation, during MI in either group (APB muscle: \( F_{1,18} = 0.04; P = 0.8 \); BIC muscle: \( F_{1,18} = 0.002; P = 0.9 \)). In patients and controls, F-wave amplitude remained unchanged \( F_{1,18} = 0.6; P = 0.4 \); raw data, Table 2).
Posthoc t tests showed that, in healthy controls, MI led to a marked increase in mean MEP amplitude in the FDI muscle ($t_{1,9} = -5.3; P < 0.001$). MI had no effect on the other muscles (APB muscle: $t_{1,9} = -2.1, P = 0.07$; ADM muscle: $t_{1,9} = -1.2, P = 0.2$; BIC muscle: $t_{1,9} = -1.01, P = 0.3$; Fig. 1A). Conversely, in patients with writer’s cramp MI had a facilitatory effect on the mean MEP amplitude in FDI muscle ($t_{1,9} = -3.84; P = 0.004$) that also spread over the other hand and forearm muscles (APB muscle: $t_{1,9} = -3.88, P = 0.004$; ADM muscle: $t_{1,9} = -3.04, P = 0.001$; ECR muscle: $t_{1,9} = -3.2, P = 0.001$; BIC muscle: $t_{1,9} = -1.4, P = 0.18$; Fig. 1B).

MI of a Tonic Movement Carried Out With the Left Index Finger in Patients and Healthy Controls

The MI task of left index-finger abduction decreased the RMT in left upper-limb muscles in patients and in healthy controls (Table 2). As observed for the affected side, MI increased the MEP size in patients and controls. Repeated-measures ANOVA showed a significant main effect of condition ($F_{1,18} = 35.1; P < 0.001$). This effect was caused by an overall increase in the mean peak-to-peak MEP amplitudes during imagery in both groups. In addition, a three-factorial ANOVA disclosed a significant difference between groups in the topographic specificity of the MEP facilitation and a condition-by-group-by-muscle interaction ($F_{4,72} = 7.2; P < 0.001$).

Separate two-factorial ANOVA for each group with condition (before and during imagery) and muscle (FDI vs. APB vs. ADM vs. ECR vs. BIC) as within-subject factors, conducted to explore within group effects, disclosed in patients a prominent main effect for the factor condition ($F_{1,9} = 16.4; P = 0.02$) and a significant condition-by-muscle interaction ($F_{4,36} = 6.6; P < 0.001$). Also in healthy controls, ANOVA demonstrated a main effect for the factor condition ($F_{1,9} = 21.9; P = 0.01$) and a condition-by-muscle interaction ($F_{4,36} = 15.6; P < 0.001$).

A two-way ANOVA conducted to identify muscle-specific differences in the MEP facilitation during MI between patients and controls for each muscle, using the factor group and condition, revealed a group-by-condition interaction for the FDI muscle ($F_{1,18} = 5.3; P = 0.03$) due to a less significant increase in MEP amplitude during MI in the FDI muscle in patients than in controls (Fig. 2A,B). ANOVA also disclosed a group-by-condition interaction for the ADM muscle ($F_{1,18} = 5.3; P = 0.02$) and ECR muscle ($F_{1,18} = 4.1; P = 0.04$) indicating a more widespread MEP amplitude facilitation during MI in patients than in controls. In the remaining muscle, ANOVA identified no significant difference in the MEP amplitude facilitation during MI in either group (APB muscle: $F_{1,18} = 0.6; P = 0.4$; BIC muscle: $F_{1,18} = 0.07, P = 0.7$).

### TABLE 2. Patients with writer’s cramp

<table>
<thead>
<tr>
<th>Parameter</th>
<th>FDI</th>
<th>APB</th>
<th>ADM</th>
<th>ECR</th>
<th>BIC</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Patients Resting motor threshold</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Affected side</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>At rest (%)</td>
<td>46.4 ± 5.8</td>
<td>44.3 ± 4.4</td>
<td>47.3 ± 2.7</td>
<td>41.4 ± 4.4</td>
<td>46.3 ± 2.3</td>
</tr>
<tr>
<td>During imagery (%)</td>
<td>40.3 ± 4.1</td>
<td>40.3 ± 4</td>
<td>41.7 ± 3.8</td>
<td>37.5 ± 3.6</td>
<td>45.7 ± 2.6</td>
</tr>
<tr>
<td>Unaffected side</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>At rest (%)</td>
<td>48 ± 8.4</td>
<td>44.4 ± 3.6</td>
<td>48 ± 3.4</td>
<td>43.4 ± 5.8</td>
<td>47.7 ± 3.3</td>
</tr>
<tr>
<td>During imagery (%)</td>
<td>41.5 ± 7.1</td>
<td>39.9 ± 3</td>
<td>42 ± 2.2</td>
<td>38 ± 4</td>
<td>46.5 ± 1.7</td>
</tr>
<tr>
<td>F-wave amplitude (mV)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>At rest (affected side)</td>
<td>0.7 ± 0.3</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>During imagery (affected side)</td>
<td>0.6 ± 0.2</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td><strong>Controls Resting motor threshold</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Right side</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>At rest (%)</td>
<td>45 ± 6</td>
<td>42.9 ± 4.2</td>
<td>45.9 ± 3.4</td>
<td>40 ± 4.9</td>
<td>44.9 ± 2.4</td>
</tr>
<tr>
<td>During imagery (%)</td>
<td>38.9 ± 4.7</td>
<td>42.2 ± 4.4</td>
<td>44.8 ± 4.7</td>
<td>39.2 ± 5.9</td>
<td>44.3 ± 3</td>
</tr>
<tr>
<td>Left side</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>At rest (%)</td>
<td>43.4 ± 7.6</td>
<td>44.7 ± 5.6</td>
<td>46 ± 5.5</td>
<td>42.7 ± 6.8</td>
<td>45.2 ± 2</td>
</tr>
<tr>
<td>During imagery (%)</td>
<td>37.4 ± 6</td>
<td>43.9 ± 6.2</td>
<td>46.8 ± 5.4</td>
<td>43.6 ± 8</td>
<td>45.3 ± 4.6</td>
</tr>
<tr>
<td>F-wave amplitude (mV)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>At rest (right side)</td>
<td>0.6 ± 0.3</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>During imagery (right side)</td>
<td>0.7 ± 0.3</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
</tbody>
</table>

Mean group data (± SD) of resting motor threshold (RMT) and F-wave amplitudes at rest and during motor imagery. Motor thresholds are given as percentage of maximum stimulator output.

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Posthoc \(t\) tests showed that in healthy controls, MI led to a marked increase in mean MEP amplitude in the FDI muscle (\(t_{1.9} = -5.3, P < 0.001\)). MI had no effect in the other muscles (APB muscle: \(t_{1.9} = -2.1, P = 0.06\); ADM muscle: \(t_{1.9} = -0.6, P = 0.5\); ECR muscle: \(t_{1.9} = -1.6, P = 0.1\); BIC muscle: \(t_{1.9} = -1.7, P = 0.1\); Fig. 2A). In patients with writer’s cramp, MI significantly increased the mean MEP amplitude in FDI muscle (\(t_{1.9} = -3.8, P = 0.003\)) and produced a spread over the other hand and forearm muscles (APB muscle: \(t_{1.9} = -3.9, P = 0.003\); ADM muscle: \(t_{1.9} = -3, P = 0.01\); ECR muscle: \(t_{1.9} = -3.2, P = 0.01\); BIC muscle: \(t_{1.9} = -1.4, P = 0.1\); Fig. 2B).

**DISCUSSION**

We found that as already observed in normal subjects and in patients with writer’s cramp, MI increases the MEP amplitude and decreases the RMT. The pattern of these MI-induced changes in TMS variables, however, differed in the two groups. The FDI muscle MEP increased less prominently in patients than it did in controls but the facilitation spread also to nontarget muscles in the hand and forearm. MI therefore induced a more attenuated although more widespread increase in corticospinal excitability in patients than it did in healthy controls.

The MI task we tested using TMS relates to movement preparation and execution and activates motor areas anterior to the primary motor area without directly activating the descending motor neurons. The altered responsiveness of corticospinal output neurons to MI observed in this study suggests that motor strategy in patients with writer’s cramp is altered even in the absence of involuntary movement.

A recent article showed that MI results in a stronger MEP facilitation during complex movements than that during simple movements. This difference raises the question whether patients found MI of the “simple” tonic contraction more difficult (or more complex) than controls did. If they...
did, then this difference, rather than a difficulty in focusing central motor commands, might partly explain our results. We nevertheless consider this explanation unlikely because the index finger abduction task is a relatively simple motor task that can be carried out easily even by patients with writer’s cramp who have, after all, a task-specific dystonia.

The widespread increase in MEP amplitude during MI might otherwise depend on the known difficulty that dystonic patients have in focusing cortical activity. Several lines of evidence suggest that in the human motor system mechanisms of surrounding inhibition operate. These mechanisms are well developed in sensory systems. They serve to focus neuronal activity by creating an inhibitory zone around a central core of activation. In the motor system, surrounding inhibition could help to ensure the selective muscle activation needed for executing fine motor movements. Evidence that bicuculline (a γ-aminobutyric acid [GABA]A antagonist) injected into the primate motor cortex produces co-contraction of various muscles shows that surrounding inhibition is mediated through GABA_A inhibitory intracortical mechanisms. Several observations suggest the presence of reduced levels of GABA in both the basal ganglia and the sensory motor cortex in dystonic patients. Several findings therefore support abnormal surrounding inhibition in patients with focal hand dystonia even in the absence of movement.

We have recently shown an alteration of Hebbian-like associative plasticity in writer’s cramp patients. The model we used was “paired associative stimulation” (PAS) described by Stefan and colleagues. According to the Hebbian rule, the mechanism underlying PAS-induced cortical plasticity involves long-term potentiation (LTP) phenomena within the stimulated motor cortex. The PAS-induced facilitation was greater and more widespread in patients with dystonia than in normal subjects, so that MEPs increased also in muscles innervated by the ulnar and median nerves. Because the mechanisms underlying PAS also seem to operate in some forms of motor learning, the abnormal associa-

![FIG. 2. Changes in mean motor evoked potential (MEP) size during motor imagery on the left side of healthy controls and on the unaffected side of patients. TMS was always given to the motor cortex contralateral to the imaged task. MEP size during motor imagery (MI) compared with rest condition recorded from different target muscles of the left upper limb after stimulation of the right hemisphere in controls (A) and patients with writer’s cramp (B). The bar chart shows the mean peak-to-peak amplitude (mV) of MEPs recorded at rest (open columns) and during MI (black columns). Each error bar equals standard error of the mean (SEM). MI elicited an attenuated and less focal increase in MEP amplitude in dystonic patients than in controls. FDI, first dorsal interosseous; APB, abductor pollicis brevis; ADM, abductor digiti minimi; ECR, extensor carpi radialis; BIC, biceps. *P < 0.05.](image-url)
tive LTP in patients with writer’s cramp may promote the consolidation of aberrant motor engrams during repetitive skilled hand movements, thus contributing to the development and maintenance of dystonic postures.26

In keeping with the hypothesis of aberrant plasticity, a positron emission tomography (PET) study showed that patients with focal hand dystonia have a more widespread metabolic after-effect in response to 1-Hz rTMS delivered over the dorsal premotor cortex.31 The hypothesis we favor is that a deficient surround inhibition may predispose patients to a more widespread and maladaptive reorganization within the motor cortex. The deficient focusing of motor programs observed during MI in dystonic patients could also be related to faulty sensory processing.6 In a study of patients with writer’s cramp, Murase and colleagues32 reported that the frontal component N30 of somatosensory evoked potentials is not reduced in amplitude (gated) in the premovement period whereas normal gating occurs during movement. Even if the subjects were only thinking about moving their hands, the dystonic patients suppressed their N30 less than normal subjects did. A motor act is initiated usually by a predetermined program that fixes the strategy and is assisted by sensory feedback control mechanisms. The role of sensory gating may be to filter out the expected sensory re-afference so that resources can be focused on novel sensory inputs.33 The abnormality of sensory gating in the short period just before the imagined or executed movement may contribute to the incorrect choice of motor commands that characterizes dystonic movements. The basal ganglia may play an important role in gating sensory inputs for guiding movements33,34 and can probably control or select automatic movements once they are learned.35,36 If the flexibility in associating sensory input and motor output is lost, recall and execution of motor programming may become incorrect. Even if in our experimental setting, unlike previous studies, patients did not perform voluntary movements after MI, our findings confirm abnormal focusing of motor programs in focal dystonia. Patients may be doubly affected: they may have abnormal motor engrams21 and be unable to recall motor engrams properly.

The reduced increase in MEP amplitude during MI in our patients agrees with a recent study in parkinsonian patients reporting motor cortical hyperreactivity to MI.37 In both conditions (dystonia and Parkinson’s disease), as a consequence of motor cortex hyperexcitability, the neural structures involved in the MEP facilitation during MI were probably unable to increase their activity further (ceiling effect). The abnormal modulation of corticospinal excitability during MI also agrees with the recent findings of a reduced modulation of cortical excitability before the execution of voluntary movements in patients with focal hand dystonia.38

In patients doing the MI task in this study, the abnormal corticospinal excitability increased to a similar extent in the affected and unaffected left upper limb. Others have already reported bilateral physiological abnormalities in clinically unilateral writer’s cramp. For example, after stimulation of the left and right hemispheres in patients with writer’s cramp, short-latency inhibition decreases significantly.4 Regional cerebral blood flow responses to vibrotactile stimulation studied with PET revealed reduced activation of the contralateral primary sensorimotor area and supplementary motor area on stimulation of the unaffected as well as the affected hand.39 These findings of bilateral brain dysfunction could account for the development of focal dystonia, in the substituted hand (mirror dystonia), in patients who switch hands for writing.40

The abnormalities we observed during MI are in keeping with the clinical observation that dystonic movements can be worsened by the attempt to perform a voluntary action. For instance, in patients with writer’s cramp the typical dystonic posture develops just from holding a pen, even before writing.1 In some patients, the mere intention of writing can occasionally trigger dystonic spasms. Altered motor strategy during the premovement stage receives support from several neurophysiological observations. Premovement cortical potentials such as contingent negative variation (CNV) and movement-related cortical potentials (MRCP) are impaired in patients with writer’s cramp.7,10,41

In conclusion, MI is associated with an attenuated and more widespread increase in corticospinal excitability in patients with focal hand dystonia than in healthy subjects. Our findings during MI suggest that even simple motor programs are poorly focused in writer’s cramp. This change is in good agreement with the notion of a deficient center surround inhibition in writer’s cramp.

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MOTOR IMAGERY IN WRITER’S CRAMP 1495