

Original articles

# Motor excitability during movement imagination and movement observation in psychogenic lower limb paresis

Joachim Liepert<sup>a,b,\*</sup>, Thomas Hassa<sup>a</sup>, Oliver Tüscher<sup>b</sup>, Roger Schmidt<sup>c,d</sup>

<sup>a</sup>Department of Neurorehabilitation, Kliniken Schmieder, Allensbach

<sup>b</sup>Department of Neurology, University of Freiburg, Germany

<sup>c</sup>Department of Psychotherapeutic Neurology, Kliniken Schmieder, Konstanz

<sup>d</sup>Department of Psychotherapeutic Neurology, Kliniken Schmieder, Gailingen

Received 8 February 2010; received in revised form 8 June 2010; accepted 9 June 2010

## Abstract

**Background:** Patients with a psychogenic paresis have difficulties performing voluntary movements. Typically, diagnostic interventions are normal. We tested whether patients with a psychogenic lower limb paresis exhibit abnormal motor excitability during motor imagery or movement observation. **Methods:** Transcranial magnetic stimulation (TMS) with single and paired pulses was used to explore motor excitability at rest, during imagination of ankle dorsiflexions and during watching another person perform ankle dorsiflexions. Results obtained in ten patients with a flaccid psychogenic leg paresis were compared with a healthy age-matched control group. In addition, results of two patients with a psychogenic fixed dystonia of the leg are presented. **Results:** During rest, motor excitability evaluated by motor thresholds, size of motor-evoked potentials (MEP) by single pulse TMS, intracortical inhibition and intracortical facilitation tested by

paired-pulse TMS were similar in patients and healthy subjects. MEPs recorded in five patients during movement observation were also comparable across the two groups. During motor imagery, patient MEPs were significantly smaller than in the control group and smaller than during rest, indicating an inhibition. **Conclusion:** In patients with motor conversion disorder, the imagination of own body movements induces a reduction of corticospinal motor excitability whereas it induces an excitability increase in healthy subjects. This discrepancy might be the electrophysiological substrate of the inability to move voluntarily. Watching another person perform movements induces a normal excitability increase, indicating a crucial role of the perspective and suggesting that focusing the patient's attention on a different person might become a therapeutic approach.

© 2011 Published by Elsevier Inc.

**Keywords:** Motor conversion disorder; Psychogenic paresis; Motor excitability; Transcranial magnetic stimulation; Motor imagery; Action observation

## Introduction

Patients with a motor conversion disorder may present a broad variety of motor symptoms, ranging from flaccid plegia to spasticity, dystonia, tremor, and jerks. The diagnosis is based on the presence of symptoms affecting voluntary motor or sensory function that suggest a neurological or other medical condition and follows the criteria of the *International Classification of Diseases, 10th*

*Revision (ICD-10; F 44.4)*. A retrospective analysis indicated that a physical injury as a triggering event can be found in 37% of the patients [1]. An underlying psychological reason is assumed but cannot always be detected [2]. Typically, all somatic diagnostic investigations exhibit normal results or do at least not explain the symptoms. In several studies, transcranial magnetic stimulation (TMS) has been used to test the integrity of corticospinal tract [3–7]. In these studies, amplitudes of motor evoked potentials (MEP) and latencies were normal, indicating that weakness was not due to impaired transmission. In functional brain imaging studies, several abnormalities have been reported. Hyperactivity of prefrontal areas (orbitofrontal cortex and anterior cingulate cortex) during attempts to activate the affected limb was

\* Corresponding author. Kliniken Schmieder, Department of Neurorehabilitation, Zum Tafelholz 8, D-78476 Allensbach, Germany. Tel.: +49 7533 8081236; fax: +49 7533 8081441.

E-mail address: [j.liepert@kliniken-schmieder.de](mailto:j.liepert@kliniken-schmieder.de) (J. Liepert).

interpreted as an indicator of increased inhibition [8–12]. A suppressed activation of motor cortex during attempted movements has been described [e.g., 13–15]. An involvement of striatothalamocortical circuits and dorsolateral prefrontal cortex resulting in impaired volition and dysfunctional motor programs is also discussed [16]. A recent publication emphasized that conversion symptoms are associated with activations in brain areas that are related to emotion regulation and self-related representations [14]. A disconnected crosstalk between the dorsal, “cognitive” and ventral, “emotional” subdivisions of the anterior cingulate and the prefrontal cortex has already been hypothesized some years ago [17].

In our study with TMS, we were interested if patients with a psychogenic lower limb paresis activate their motor system during tasks without demand to move the limb. Based on the results of functional brain imaging studies, we hypothesized that motor excitability might be suppressed during motor imagery (MI) or action observation (AO). Motor imagery may be defined as being “a dynamic state during which the representation of a given motor act is internal rehearsal without any overt motor output” [18]. Functional imaging studies in healthy subjects have demonstrated that similar motor areas are activated as during real execution of the movement (e.g., Refs. [19–22]). Comparable to MI, AO leads to activations in parietal and premotor cortex [23–25].

TMS investigations have shown that both MI and AO enhance corticospinal excitability in a muscle-specific manner [26,27] and to a similar degree [28]. Patients with motor conversion disorder participating in a mental rotation task had more difficulties with an explicit MI than with the implicit MI [29]. In another experiment, the authors described a specific slowing of motor initiation but not a general slowing involving motor execution [30]. Recent TMS studies with patients suffering from psychogenic upper limb paresis demonstrated that MI of finger movements with the affected limb induced a decrease of motor excitability, suggesting an inhibition of the motor system during that task [31,32]. The present study explored if a similar phenomenon can also be found in psychogenic lower limb paresis and if abnormalities occur to a comparable degree in MI and AO, respectively.

## Methods

### Patients

We studied 10 patients with a flaccid psychogenic paresis (mean age: 43±8 years, mean duration of symptoms: 25 months, range: 4–120 months, four women). Four patients complained about a paraparesis, three patients presented with a right-sided paresis, the other three with a left-sided paresis. In all of them, ankle dorsiflexion was more severely impaired than other movements. In all but one patient ankle dorsiflexion was plegic. More detailed clinical information

is supplied in Table 1. Before participating in the study, all patients had undergone an extensive diagnostic work-up. Reflexes, somatosensory evoked potentials, motor evoked potentials, nerve conduction studies, electromyography, magnetic resonance imaging of brain and spinal cord, cerebrospinal fluid examination and blood tests for vitamin deficits or hypothyroidism had all been normal. Eight patients complained about sensory deficits to a varying degree, the other two patients did not suffer from sensory impairments. Five patients reported a physical injury prior to symptom onset. This percentage (42%) is comparable with a recently reported number [1]. Remarkably, all the physical injuries involved a psychic trauma or affected psychosocially prestressed individuals. Furthermore, all patients included showed relevant psychosocial strains and/or psychiatric comorbidity (Table 1).

We had the opportunity to perform MI experiments in two patients with a psychogenic fixed dystonia. In order to maintain homogeneity in the group of patients with flaccid paresis, we did not include results from these two patients in the group data but report them as single cases. More clinical details are summarized in Table 1. Our healthy control group consisted of 10 age-matched subjects (42±9 years, four women).

Both groups participated in MI and AO experiments.

All subjects had given informed consent prior to inclusion. The study was approved by the Ethical committee of the University of Constance. Exclusion criteria included pregnancy, metallic implants in the brain and heart pace makers.

### TMS

Recordings were taken with surface electrodes (belly-tendon montage) from the tibialis anterior muscle. TMS was performed with a circular coil (outer diameter: 14 cm) (The Magstim, Dyfed, UK) which was connected to a magnetic stimulator (Magstim 200 HP). To apply paired pulses, the coil was connected to a Bistim device which triggered two magnetic stimulators. The optimal coil position where MEPs could be evoked with the lowest stimulus intensity was marked with ink to ensure an exact repositioning of the coil throughout the experiment. At this coil position the motor threshold (MT) was determined. MT was defined as the stimulus intensity needed to produce MEPs with a size of 50–100  $\mu$ V in five out of 10 consecutive trials during complete muscle relaxation [33]. TMS single pulses were applied with an intensity of 115% of the individual MT at rest. Short interval intracortical inhibition (SICI) and intracortical facilitation (ICF) were examined in a conditioning-test pulse TMS paradigm [34] at complete rest. The first conditioning shock had an intensity of 75% of MT. The intensity of the second pulse was adjusted to produce an MEP of approximately 0.3 mV peak-to-peak size. The following interstimulus intervals were tested: 2, 3, 10 and 15 ms. To increase the power and to obtain a representative mean value for SICI and ICF we combined conditioned MEP

Table 1  
Clinical details of the patient group

| Patient | Age | Sex | Symptoms/affected limb(s)                                                                                                         | Duration (months) | Sensory deficits | Pain                                                     | Physical injury                             | Psychiatric morbidity (ICD-10)                                                                                                                                 | Psychosocial aspects                                                                                                                   |
|---------|-----|-----|-----------------------------------------------------------------------------------------------------------------------------------|-------------------|------------------|----------------------------------------------------------|---------------------------------------------|----------------------------------------------------------------------------------------------------------------------------------------------------------------|----------------------------------------------------------------------------------------------------------------------------------------|
| 1       | 51  | M   | Flaccid, both legs, plegic                                                                                                        | 19                | None             | None                                                     | Surgical intervention                       | Dissociative motor disorder F44.4                                                                                                                              | Experienced life threatening medical condition (pulmonary embolism)                                                                    |
| 2       | 44  | M   | Flaccid, right leg (degree of strength: 4-), ankle dorsiflexion plegic                                                            | 11                | +                | Lower back                                               | Catheter intervention                       | Mixed dissociative disorder F44.7, Persistent somatoform pain disorder F45.4, Recurrent depressive disorder F33.2, subsyndromal PTSD F43.8                     | Traumatic childhood experiences, anxious [avoidant] personality traits multiple surgical interventions experienced as life threatening |
| 3       | 35  | M   | Flaccid, Left leg and left arm, plegic                                                                                            | 15                | ++               | Headache                                                 | None                                        | Mixed dissociative disorder F44.7, Mixed anxiety and depressive disorder F41.2                                                                                 | Health problems since childhood, traumatic life event, current relational conflict                                                     |
| 4       | 28  | M   | Flaccid, both legs, pronounced on the right side (degree of strength: 2), ankle dorsiflexion plegic                               | 22                | +++              | Lower back, generalized myalgias                         | None                                        | Mixed dissociative disorder F44.7                                                                                                                              | Relational conflict, anxious [avoidant] personality traits                                                                             |
| 5       | 47  | M   | Flaccid, right leg, paretic (degree of strength: 3), ankle dorsiflexion plegic                                                    | 12                | ++               | Right leg, back                                          | None                                        | Mixed dissociative disorder F44.7, subsyndromal PTSD F43.8                                                                                                     | Several stressful live events, anxious [avoidant] personality traits                                                                   |
| 6       | 44  | M   | Flaccid, both legs, plegic                                                                                                        | 7                 | +++              | Headache, back, upper and lower extremities, fluctuating | Current impulse <sup>a</sup>                | Mixed dissociative disorder F44.7, Persistent somatoform pain disorder F45.4                                                                                   | Avoidant coping strategy                                                                                                               |
| 7       | 44  | F   | Flaccid, left leg and left arm, (degree of strength: 3), ankle dorsiflexion plegic                                                | 36                | ++               | Cervical pain, headache                                  | Traffic accident, cervical spine distortion | Mixed dissociative disorder F44.7, subsyndromal PTSD F43.8                                                                                                     | Avoidant coping strategy                                                                                                               |
| 8       | 54  | F   | Flaccid, left leg, ankle dorsiflexion almost plegic (degree of strength: 1), plantar flexion paretic (degree of strength: 4-)     | 9                 | +                | None                                                     | Ankle joint distortion                      | Mixed dissociative disorder F44.7, Neurasthenia F48.0, Specific phobias F41.2                                                                                  | Anankastic (performance-oriented) personality traits, current conflict at work                                                         |
| 9       | 48  | F   | Flaccid, both legs, plegic                                                                                                        | 120               | none             | Headache, shoulder, back                                 | Accident (bicycle)                          | Mixed dissociative disorder F44.7, Persistent somatoform pain disorder F45.4, Postconcussional syndrome F07.2, dependence syndrome (sedatives/hypnotics) F13.2 | Traumatic childhood experiences, chronic illness behavior                                                                              |
| 10      | 28  | F   | Flaccid, right leg (degree of strength: 2), ankle dorsiflexion plegic                                                             | 4                 | ++               | Lower back                                               | None                                        | Dissociative motor disorder F44.4, Persistent somatoform pain disorder F45.4, Dysthymia F34.1                                                                  | Experienced abuse and disregard in childhood, current psychosocial stress (persistent)                                                 |
| 11      | 28  | M   | Fixed dystonia of the right leg, in particular the ankle joint. Foot in plantar flexion and supination. Ankle dorsiflexion plegic | 22                | +                | Shoulder, back                                           | None                                        | Mixed dissociative disorder F44.7, Persistent somatoform pain disorder F45.4                                                                                   | Several traumatic life events in childhood and youth, current relational conflicts                                                     |
| 12      | 37  | M   | Fixed dystonia, left leg and left arm, both in a stretched position, unable to bend them passively or actively                    | 5                 | +++              | None                                                     | None                                        | Mixed dissociative disorder F44.7, subsyndromal PTSD F43.8                                                                                                     | Suffered act of violence, experienced as life threatening                                                                              |

M, male; F, female; +, small sensory deficit; ++, moderate sensory deficit; +++, loss of sensation.

<sup>a</sup> Electric shock.

values at interstimulus intervals of 2/3ms and 10/15 ms and took the mean conditioned MEP values at 2/3 ms and 10/15 ms as a measure of SICI and ICF, respectively. Recordings were stored on a Viking IV (Nicolet, Kleinostheim, Germany) and analysed offline. MEP amplitudes were measured peak-to-peak.

## Experiments

### Motor imagery

Subjects were asked to imagine a dorsiflexion of the foot. In detail, they were instructed to imagine a phasic movement lasting for 2 s, followed by a tonic phase for another 2 s. Performance was practiced prior to the initiation of the experiment. Two seconds after the command to start the imagery the TMS pulse was given. After each TMS pulse, subjects were asked to stop imagery for a few seconds in order to avoid mental fatigue. The frequency of TMS was approximately 0.1 Hz. Auditory feedback via a loudspeaker ensured that, during imagery, no muscle contraction occurred. Recording of each trial started 30 ms prior to the TMS pulse and finished after 200 ms. Trials with electromyographic (EMG) contamination produced by involuntary muscle activity were excluded from further analysis.

### Action observation

Two videos were demonstrated. A person, viewed from a first person perspective, was shown in a sitting position with the right leg crossed over the left. In video 1, the person did not move. In Video 2, the person repetitively performed a dorsiflexion of his right foot. The movement duration was 2 s, the position of the dorsiflexed foot was kept for another 2 s, then relaxed. TMS was applied as soon as the foot was in the maximally dorsiflexed position. The sequence of videos was randomized across subjects. Subjects were asked to closely watch the videos without attempting to imitate the movements.

### Experiment 1: MI

Patients and healthy subjects were asked to imagine a dorsiflexion of the foot. In patients with a unilateral paresis, both sides were studied, in patients with a paraparesis the right foot was studied. Healthy subjects were tested on both sides. Each imagery task was repeated eight times. In addition, 16 stimuli were given at rest. Results are expressed as percentage of the MEP amplitude at rest.

### Experiment 2: comparison of MI and AO

In a subgroup of patients ( $n=5$ ) MI and action observation were investigated in order to evaluate motor excitability

changes of the two different tasks in the same subjects. Healthy age-matched control subjects ( $n=5$ ) participated in the same procedures.

MI and AO tasks were repeated eight times. Additionally, eight stimuli were given at rest. Results are expressed as percentage of the MEP amplitude at rest.

### Experiment 3: Motor excitability at rest

To explore motor excitability at rest, MTs were determined and MEP amplitudes were measured in all patients. In a subgroup of five patients, the paired pulse paradigm testing motor cortex excitability was applied. The same number of healthy subjects was studied with these techniques.

### Statistical analysis

An analysis of variance (ANOVA) was used after testing for normal distribution. In Experiments 1 and 3, results of the patient group were compared with the control group using unifactorial ANOVAs.

In Experiment 2, a two-factorial ANOVA with the factors GROUP (two levels: patient group and control group) and the factor INTERVENTION (two levels: MI and AO) was calculated. When significant  $p$  values occurred in the ANOVA, Bonferroni corrected post hoc  $t$ -tests were applied. The level of significance was defined as  $P<.05$ .

## Results

### Experiment 1

There was a significant difference of motor excitability changes induced by MI [ $F(1,19)=26.1$ ;  $P<.0001$ , Table 2]. The control group exhibited an increase of motor excitability during MI. In contrast, the patients did not show an increase but a decrease of motor excitability (Fig. 1). The decreased MEP amplitudes in the patient group were also significantly different from mean baseline MEP amplitudes ( $P=.028$ ). In

Table 2  
TMS results in the patient group ( $n=10$ ) and the control group ( $n=10$ )

|                       | Patient group | Control group |
|-----------------------|---------------|---------------|
| Motor threshold       | 64.8±10.8     | 67.3±10.7     |
| MEP size at rest (mV) | 0.39±0.34     | 0.37±0.35     |
| ICI                   | 42.9±37.9     | 40.7±17.8     |
| ICF                   | 150.4±55.9    | 164.7±57.7    |
| MI                    | 74.8±16.4     | 210.8±76.8    |
| AO                    | 128.9±15.4    | 132.8±15.2    |

Intracortical inhibition and facilitation and action observation data were recorded in a subgroup of five patients, all other parameters in the group of 10 patients. ICI and ICF are expressed as percentage of the MEP size induced by single TMS. MI and AO results are shown as percentage of the MEP size at rest. Motor threshold is expressed as percentage of the maximum stimulator output. Results are shown as mean±S.D.

the six patients with unilateral symptoms, MEP increase during MI with the non-affected leg was similar to the results obtained in the control group. In these six patients, MEP size increased to  $232.4 \pm 105.4\%$  (mean  $\pm$  S.D.) of MEP amplitude at rest.

### Experiment 2

The ANOVA indicated a significant effect for the factor GROUP [ $F(1,19)=14.7$ ,  $P=.0014$ ] and for the interaction GROUP  $\times$  INTERVENTION [ $F(1,19)=12.3$ ,  $P=.003$ ] but not for the factor INTERVENTION [ $F(1,19)=0.4$ ,  $P=.54$ ]. Post hoc analysis revealed a significant difference of MI effects in patients and control subjects ( $P=.005$ ) and a significant difference between MI and AO effects in the patients ( $P=.023$ ). In contrast, AO effects were similar in patients and healthy subjects ( $P=.57$ ), and there was only a non-significant trend towards a stronger excitability increase during MI than during AO in the control group ( $P=.19$ ) (Fig. 2).

### Experiment 3

There was no significant difference between patients and healthy subjects regarding MTs [ $F(1,19)=0.74$ ;  $P=.4$ ] and MEP amplitudes at rest [ $F(1,19)=0.25$ ;  $P=.87$ ]. Moreover, neither ICI values [ $F(1,19)=0.014$ ;  $P=.91$ ] nor ICF results [ $F(1,19)=0.158$ ;  $P=.7$ ] indicated a difference between the two groups.

### Results in two patients with a psychogenic fixed dystonia

During MI, both patients showed an excitability pattern comparable to the results of the group with flaccid paresis. In patient 11, MEP amplitude during MI was 86.8% of MEP amplitude at rest. In patient 12, MEP amplitude during MI was 77.1% of MEP amplitude at rest. Motor thresholds (patient 11: 61% of maximum stimulator output intensity; patient 12: 56%) were somewhat lower than those in the two groups but still within a single standard deviation of the

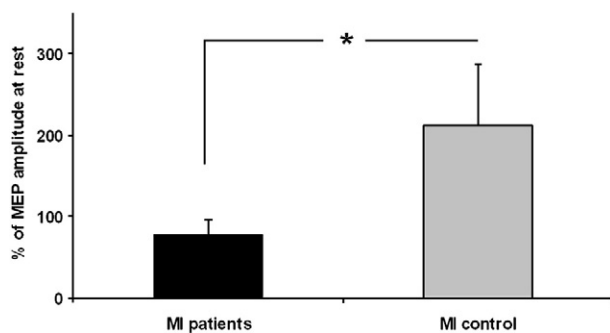


Fig. 1. Comparison of MEP amplitudes evoked during MI of ankle dorsiflexions in patients with psychogenic lower limb paresis ( $n=10$ ) and an age-matched healthy control group ( $n=10$ ). Error bars indicate standard deviations.  $*P<.05$ .

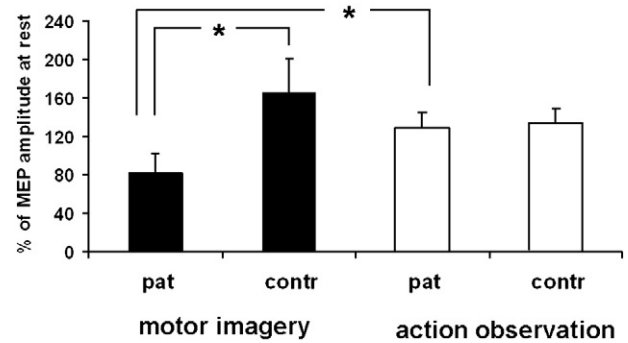


Fig. 2. Comparison of TMS results during motor imagery (black bars) and during action observation (open bars) in patients with psychogenic lower limb paresis ( $n=5$ ) and an age-matched healthy control group ( $n=5$ ). Error bars indicate standard deviations.  $*P<.05$ .

mean group value. In patient 11, ICI and ICF were tested at rest. He had a somewhat lower ICI (54.9% of MEP amplitude evoked by single pulse stimulation) and an increased ICF (305% of MEP amplitude evoked by single pulse stimulation) as compared to the control group.

### Discussion

Our study demonstrates that patients with a psychogenic lower limb paresis exert an abnormally low excitability pattern during imagery of ankle dorsiflexions. The MEP amplitude reduction during MI was observed in every single patient and indirectly indicates an “active” down-regulation of motor excitability. In contrast, all healthy subjects showed an excitability increase during MI. We hypothesize that the patients’ inhibition of excitability is an electrophysiological correlate of the inability to perform voluntary movements. It is highly improbable that the results are due to an inability to perform MI. If that were the case, MEP amplitudes should have been similar to those obtained during rest. However, recently it has been demonstrated that limb amputation and disuse may weaken the ability to generate vivid images of movements [35]. Therefore, in future studies, we will address the evaluation of imagery capabilities in more detail by introducing scales to assess MI vividness. The current results are comparable to those in patients with a psychogenic upper extremity paresis [31,32], suggesting that the pathophysiological mechanisms for upper and lower limb motor conversion disorder are similar. Moreover, since the two patients with dystonic symptoms displayed the same abnormal inhibition of excitability as the patients with flaccid paresis, our finding represents a more generalized mechanism that occurs even in opposite clinical pictures. The results also suggest that the activation of inhibitory neurons is task-dependent since motor excitability measures at rest did not indicate a difference between the two groups. In brief, motor cortex excitability, examined by the paired pulse paradigm, as well as corticospinal excitability, tested by MTs and MEP amplitudes at rest, was similar in both



groups. This finding corresponds to other TMS studies in motor conversion disorder [3–7] and indicates that the “passive” motor system excitability is normal. However, this seems only to be true for patients with flaccid psychogenic paresis. In patients with psychogenic dystonia, abnormalities of cortical excitability at rest have been described, e.g., different groups have reported a reduction of Intracortical Inhibition [36–38]. We also found a reduced Intracortical Inhibition and an increased Intracortical Facilitation in a patient with fixed dystonia (Patient 11, Table 1), thus reproducing the results published by others. Avanzino et al. [37] suggested that enhanced cortical excitability might represent an underlying predisposing trait to different clinical forms of dystonia.

Where does the inhibition of motor excitability during MI occur? We hypothesize that motor cortex and its output are down-regulated by other higher-order brain areas. However, the current results do not allow to draw definite conclusions regarding such areas involved in this inhibition. Considering brain imaging results, one could speculate that an inhibitory neuronal circuitry from prefrontal cortex to the basal ganglia and from there to the motor cortex becomes activated during MI and during the attempt to move voluntarily. An alternative might be a loop from prefrontal to premotor cortex [15] and from there to the primary motor cortex. Based on our results, the question whether a motor conversion disorder rather interferes with the preparation, or the execution of voluntary movements, or both, cannot be answered with certainty [16]. Most patients complained about a more or less severe sensory impairment in the affected leg. Some patients described an inability to localize the position of the leg without visual feedback. Most probably, the mental image of that limb was impaired or even lacking in these patients. This certainly also contributes to an impaired ability to imagine movements. Brain imaging studies had found a hypoactivation of parietal cortex [8,12], potentially a correlate of an impaired sensation. However, lack of sensation cannot be the only reason for our finding, since the decrease of motor excitability even below the resting condition points to an active inhibition. Interestingly, such an inhibition was not found during the AO task. The patients displayed a “normal” excitability increase during observation of another person performing movements. This suggests that inhibitory activity depends on the perspective and on the question whether the patient focuses his attention on himself or on a different person. Another reason for the discrepancy between AO and MI might be that AO activates the motor system in an implicit, “bottom-up” manner, whereas our MI task activates the motor system in an explicit, “top-down” way. The differential findings might have therapeutic implications: Currently, we hypothesize that patients with motor conversion disorder might benefit more from a treatment with AO elements than with MI aspects. This hypothesis should be tested in a controlled clinical trial. However, one

can also consider an alternative approach, consisting of strategies aimed at improving the expression of MI.

Using an AO task, Burgmer et al. [39] examined brain activation patterns in four patients with psychogenic arm paresis and reported a decrease of motor cortex activation. This result is in some contrast to our findings. The reason for this discrepancy is unclear. Differences in methods (TMS versus functional magnetic resonance imaging), patient characteristics (e.g., affection of upper versus lower extremity) and the observed movements might be responsible.

In our study with TMS we cannot differentiate between feigning and true motor conversion disorder. Sohn et al. [40] have demonstrated that the volitional suppression of an intended movement may also induce a reduction of MEP amplitudes. However, based on clinical observations of the patients’ behavior and their psychopathological profile during the inpatient rehabilitation period, feigning was not suspected in any of the patients.

In summary, our study demonstrated an abnormal down-regulation of motor excitability during MI but a normal excitability increase during AO. These results do not only enlarge our knowledge about pathophysiological changes in patients with motor conversion disorder but surprisingly also helped individual patients to accept the diagnosis and a psychosomatic therapeutic approach. Recently, such a finding has also been reported by others [15].

## Acknowledgments

We thank Ariel Schoenfeld for helpful comments.

## References

- [1] Stone J, Carson A, Aditya H, Prescott R, Zaubi M, Warlow C, et al. The role of physical injury in motor and sensory conversion symptoms: a systematic and narrative review. *J Psychosom Res* 2009;66:383–90.
- [2] Krem MM. Motor conversion disorders reviewed from a neuropsychiatric perspective. *J Clin Psychiatry* 2004;65:783–90.
- [3] Schriefer TN, Mills KR, Murray NMF, Hess CW. Magnetic brain stimulation in functional weakness. *Muscle & Nerve* 1987;10:643.
- [4] Pillai JJ, Markind S, Streletz LJ, Field HL, Herbison G. Motor evoked potentials in psychogenic paralysis. *Neurology* 1992;42:935–6.
- [5] Jellinek DA, Bradford R, Bailey I, Symon L. The role of motor evoked potentials in the management of hysterical paraplegia: Case report. *Paraplegia* 1992;30:300–2.
- [6] Morota N, Deletis V, Kiprovski K, Epstein F, Abbott R. The use of motor evoked-potentials in the diagnosis of psychogenic quadriplegia. *Pediatr Neurosurg* 1994;20:203–6.
- [7] Cantello R, Boccagni C, Comi C, Civardi C, Monaco F. Diagnosis of psychogenic paralysis: the role of motor evoked potentials. *J Neurol* 2001;248:889–97.
- [8] Tiihonen J, Kuikka J, Viinamäki H, Lehtonen J, Partanen J. Altered cerebral blood flow during hysterical paresthesia. *Biol Psychiatry* 1995;37:134–5.
- [9] Marshall JC, Halligan PW, Fink GR, Wade DT, Frackowiak RSJ. The functional anatomy of a hysterical paralysis. *Cognition* 1997;64: B1–8.
- [10] Vuilleumier P, Chicherio C, Assal F, Schwartz S, Slosman D, Landis T. Functional neuroanatomical correlates of hysterical sensorimotor loss. *Brain* 2001;124:1077–90.

- [11] Mailis-Gagnon A, Giannoylis I, Downar J, Kwan CL, Mikulis DJ, Crawley AP, et al. Altered central somatosensory processing in chronic pain patients with “hysterical” anesthesia. *Neurology* 2003;60:1501–7.
- [12] Ghaffar O, Staines R, Feinstein A. Unexplained neurologic symptoms: an fMRI study of sensory conversion disorder. *Neurology* 2006;67:2036–8.
- [13] Stone J, Zeman A, Simonotto E, Meyer M, Azuma R, Flett S, et al. fMRI in patients with motor conversion symptoms and controls with simulated weakness. *Psychosom Med* 2007;69:961–9.
- [14] Cojan Y, Waber L, Caruzzo A, Vuilleumier P. Motor inhibition in hysterical conversion paralysis. *Neuroimage* 2009;47:1026–37.
- [15] Nowak DA, Fink GR. Psychogenic movement disorders: aetiology, phenomenology, neuroanatomical correlates and therapeutic approaches. *Neuroimage* 2009;47:1015–25.
- [16] Scott RL, Anson JG. Neural correlates of motor conversion disorder. *Motor Control* 2009;13:161–84.
- [17] Ballmaier M, Schmidt R. Conversion disorder revisited. *Funct Neurol* 2005;20:105–13.
- [18] Decety J, Grezes J. Neural mechanisms subserving the perception of human actions. *Trends Cogn Sci* 1999;3:172–8.
- [19] Deiber MP, Ibanez V, Honda M, Sadato N, Raman R, Hallett M. Cerebral processes related to visuomotor imagery and generation of simple finger movements studied with positron emission tomography. *Neuroimage* 1998;7:73–85.
- [20] Jeannerod M, Frak V. Mental imaging of motor activity in humans. *Curr Opin Neurobiol* 1999;9:735–9.
- [21] Kuhlitz-Buschbeck JP, Mahnkopf C, Holzknecht C, Siebner H, Ulmer S, Jansen O. Effector-independent representations of simple and complex imagined finger movements: a combined fMRI and TMS study. *Eur J Neurosci* 2003;18:3375–87.
- [22] Hanakawa T, Dimyan MA, Hallett M. Motor planning, imagery, and execution in the distributed motor network: a time-course study with functional MRI. *Cereb Cortex* 2008;18:2775–88.
- [23] Decety J, Grezes J, Costes N, Perani D, Jeannerod M, Procyk E, et al. Brain activity during observation of actions. Influence of action content and subject’s strategy. *Brain* 1997;120:1763–77.
- [24] Lui F, Buccino G, Duzzi D, Benuzzi F, Crisi G, Baraldi P, et al. Neural substrates for observing and imagining non-object-directed actions. *Soc Neurosci* 2008;3:261–75.
- [25] Suchan B, Melde C, Herzog H, Hömberg V, Seitz RJ. Activation differences in observation of hand movements for imitation or velocity judgement. *Behav Brain Res* 2008;188:78–83.
- [26] Maeda F, Kleiner-Fisman G, Pascual-Leone A. Motor facilitation while observing hand actions: specificity of the effect and role of observer’s orientation. *J Neurophysiol* 2002;87:1329–35.
- [27] Alaerts K, Heremans E, Swinnen SP, Wenderoth N. How are observed actions mapped to the observer’s motor system? Influence of posture and perspective. *Neuropsychologia* 2008;47:415–22.
- [28] Patuzzo S, Fiaschi A, Mangano P. Modulation of motor cortex excitability in the left hemisphere during action observation: a single- and paired-pulse transcranial magnetic stimulation study of self- and non-self-action observation. *Neuropsychologia* 2003;41:1272–8.
- [29] Roelofs K, Naring GWB, Keijsers GPJ, Hoogduin CAL, van Galen GP, Maris E. Motor imagery in conversion paralysis. *Cognit Neuropsychiatry* 2001;6:21–40.
- [30] Roelofs K, van Galen GP, Keijsers GPJ, Hoogduin CAL. Motor initiation and execution in patients with conversion paralysis. *Acta Psychologica* 2002;110:21–34.
- [31] Liepert J, Hassa T, Tuscher O, Schmidt R. Electrophysiological correlates of motor conversion disorder. *Mov Disord* 2008;23:2171–6.
- [32] Liepert J, Hassa T, Tuscher O, Schmidt R. Abnormal motor excitability in patients with psychogenic paresis - a TMS study. *J Neurol* 2009;256:121–6.
- [33] Rossini PM, Barker AT, Berardelli A, Caramia MD, Caruso G, Cracco RQ, et al. Non-invasive electrical and magnetic stimulation of the brain, spinal cord and roots: basic principles and procedures for routine clinical application. Report of an IFCN committee. *Electroencephalogr Clin Neurophysiol* 1994;91:79–92.
- [34] Kujirai T, Caramia MD, Rothwell JC, Day BL, Thompson PD, Ferbert A, et al. Corticocortical inhibition in human motor cortex. *J Physiol* 1993;471:501–19.
- [35] Malouin F, Richards CL, Durand A, Descent M, Poire D, Fremont P, et al. Effects of practice, visual loss, limb amputation, and disuse on motor imagery vividness. *Neurorehabil Neural Repair* 2009;23:449–63.
- [36] Espay AJ, Morgante F, Purzner J, Gunraj CA, Lang AE, Chen R. Cortical and spinal abnormalities in psychogenic dystonia. *Ann Neurol* 2006;59:825–34.
- [37] Avanzino L, Martino D, van de Warrenburg BP, Schneider SA, Abbruzzese G, Defazio G, et al. Cortical excitability is abnormal in patients with the “fixed dystonia” syndrome. *Mov Disord* 2008;23:646–52.
- [38] Quartarone A, Rizzo V, Terranova C, Morgante F, Schneider S, Ibrahim N, et al. Abnormal sensorimotor plasticity in organic but not in psychogenic dystonia. *Brain* 2009;132:2871–7.
- [39] Burgmer M, Konrad C, Jansen A, Kugel H, Sommer J, Heindel W, et al. Abnormal brain activation during movement observation in patients with conversion paralysis. *Neuroimage* 2006;29:1336–43.
- [40] Sohn YH, Dang N, Hallett M. Suppression of corticospinal excitability during negative motor imagery. *J Neurophysiol* 2003;90:2303–9.