Electrophysiological Correlates of Motor Conversion Disorder

Joachim Liepert, MD,[1,2]* Thomas Hassa, MD,[3] Oliver Tüscher, MD,[2] and Roger Schmidt, MD[3,4]

[1]*Department of Neurorehabilitation, Kliniken Schmieder, Allensbach, Germany*
[2]*Department of Neurology, University of Freiburg, Germany*
[3]*Department of Neurorehabilitation, Kliniken Schmieder Gailingen, Germany*
[4]*Department of Neurorehabilitation, Kliniken Schmieder, Konstanz, Germany*

Abstract: In patients with a functional (psychogenic) paresis, motor conduction tests are, by definition, normal. We investigated whether these patients exhibit an abnormal motor excitability. Four female patients with a functional paresis of the left upper extremity were studied using transcranial magnetic stimulation (TMS). We investigated motor thresholds, intracortical inhibition and intracortical facilitation at rest. Corticospinal excitability was evaluated by single pulse TMS during rest and during imagination of tonic index finger adductions. Data obtained from the affected first dorsal interosseous muscle were compared with the unaffected hand and with a healthy age-matched control group. Three patients demonstrated a flaccid paresis, one patient had a psychogenic dystonia. Motor thresholds, short interval intracortical inhibi-tion and intracortical facilitation recorded from the affected side were normal. In healthy subjects, movement imagination produced an increase of corticospinal excitability. In the patients, motor imagery with the affected index finger resulted in a decrease of corticospinal excitability compared to rest, being significantly different from the unaffected side and from the control group. We suggest that suppression of corticospinal excitability during movement imagination is an electrophysiological correlate of the patients' inability to move voluntarily and provides some insight into the patho-physiology of this disorder. © 2008 Movement Disorder Society

Key words: functional paresis; movement imagery; trans-cranial magnetic stimulation; motor excitability

Patients with functional (psychogenic) paresis show an inability to move a body part voluntarily, a condition which mimics a true neurological disease. Within the framework of the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV) a paresis of this kind is categorized as motor conversion disorder within the category of somatoform disorders. To diagnose this condition, organic disorders like central or peripheral nerve lesions or muscle diseases have to be positively ruled out. Thus, by definition, the results of electrophysiological tests investigating central or peripheral neuronal conduction should be normal.[1] Psychiatric comorbidity is high and multifaceted, and often a psychological factor or triggering traumatic event can be detected.[2] Beyond the psychodynamic theories which hypothesize an unconscious conflict or some other form of psychosocial stress at the origin of the disorder, the inability to move voluntarily or to follow an external command to move may suggest, from a neurobiological perspective, that these patients have difficulties in accessing their motor program or that motor cortex excitability is reduced. In fact, results from a case study performed in a woman with a psychogenic hemiparalysis support this hypothesis. Functional brain imaging revealed that the attempt to move the paralyzed leg failed to activate the corresponding primary motor cortex. Instead, orbito-frontal and cingulate cortex areas were activated, possibly indicating an inhibitory influence from these areas on the motor cortex.[3]

In this study, we used Transcranial Magnetic Stimulation (TMS) to test whether motor cortex excitability at rest is abnormal in patients with functional paresis and also whether the excitability enhancement that usually occurs during motor imagery is modified. Motor imagery may be defined as being "a dynamic state during which the representation of a given motor act is

*Correspondence to: Dr. Joachim Liepert, Kliniken Schmieder, Department of Neurorehabilitation, Zum Tafelholz 8, D-78476 Allensbach, Germany. E-mail: j.liepert@kliniken-schmieder.de

internal rehearsal without any overt motor output."[4] Functional imaging studies have demonstrated that the same motor areas are activated as during real execution of the movement [e.g., refs. 5–7]. Similarly, TMS studies have shown that the pattern of motor excitability changes during motor imagery is comparable to that pattern which is observed during executed movements.[8–22] Effects of motor imagery on excitability have been tested in various groups of patients, e.g. stroke victims[23] and patients suffering from writer's cramp.[24] Based on the study by Marshall et al.,[3] we hypothesized that patients with a functional paresis would not be able to enhance motor excitability during imagery of movements with the affected body part, but that imagery of movements with a non-affected extremity would produce a similar excitability increase as in healthy subjects.

METHODS

Patients

We studied four subjects with a functional paresis of one upper extremity. In all of them, extensive diagnostic workups including MRI scans of the brain and the spinal cord, examination of the cerebrospinal fluid, somatosensory-evoked potentials, motor-evoked potentials, peripheral sensory and motor nerve stimulations as well as electromyography had produced normal results. All patients presented symptoms of a ICD-10 F44 Dissociative Disorder or a DSM-IV Conversion Disorder. In the following, the patients' history is described in greater detail.

Patient 1.

This 28-year-old female presented with a perceived spastic paresis of her left arm, which had occurred "during sleep" six months prior to our investigation and had since then remained unchanged. Clinical examination demonstrated an increase of muscle tone in the left arm, which was held in a flexed elbow position and the fingers clenched to a fist. The patient was unable to move the arm voluntarily. From a psychiatric point of view she presented a subthreshold depressive symptomatology. She had a medical history of serious organic diseases, and she had once already developed conversions symptoms some years prior to the actual episode.

Patient 2.

This 26-year-old female reported a sudden and complete loss of left-sided hand and finger movements and a loss of sensation for touch and temperature in the left hand. The symptoms started overnight six months prior to the TMS investigation. Since then, severity of symptoms had oscillated. At the time of our investigation, the hand was again paralyzed. Clinical examination showed a completely flaccid paresis for all hand and finger functions but normal tendon reflexes. No actual psychosocial stressor could be identified. While mostly trying to avoid any reference to psychosocial problems, the patient, however, repeatedly showed vegetative and emotional reactions as usually seen as sequelae to traumatic life events.

Patient 3.

This 39-year-old female complained about a right-sided hemiparesis that had developed within one day, three months prior to our investigation. Since then, symptoms had been oscillating. At the time of our study the clinical investigation indicated a reduced strength for all right-sided finger and hand movements (degree of strength according to the Medical Research Council scale: 4); tendon reflexes were equal on both sides. From a psychiatric point of view she showed residual signs of a posttraumatic stress disorder, developed after a rape suffered in early childhood and not completely remitted.

Patient 4.

This 48-year-old female reported an impairment of her left hand which had started two weeks prior to the TMS study. She felt a reduced motor control which prevented or disturbed voluntary left-sided finger movements but also produced a "tremor" (frequency: 5–6 Hz) of the left arm or hand lasting for ∼10 s. Interestingly, she had had problems with the control of her right arm some weeks earlier. However, the right-sided symptoms had vanished by the time of our investigation. Clinical examination showed a variable degree of strength in her left hand and arm (between 4 and 5 on the MRC scale) without sensory deficits or reflex differences. The patient, currently in a leading health care position, presented an adjustment disorder with anxiety. Conversion disorder developed in the aftermath of an acute episode of a known Crohn's disease that had arisen in a moment of life characterized by severe psychosocial stress.

The electrophysiological studies were also performed in an age-matched healthy control group (six women, two men, mean age: 35 ± 8.1 years; range: 26–47 years).

Patients and healthy subjects were included after having given informed consent. The study was approved by the University of Constance Ethical Committee.

TMS

TMS measurements were carried out bilaterally in all patients. In six of the healthy subjects motor evoked potentials (MEPs) were obtained from the left side, in the other two subjects MEPs were recorded from the right side.

Recordings were taken with surface electrodes (belly-tendon montage) from the first dorsal interosseous muscle (FDI) bilaterally. The ulnar nerve was stimulated electrically at the wrist with supramaximal intensities to elicit M responses and F waves. TMS was performed with a figure-of eight coil (The Magstim Comp., 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 coil was held with the grip pointing posteriorly and perpendicular to the central sulcus. Resting motor threshold was defined as the stimulus intensity needed to produce MEPs with a size of 50–100 μV in 5 of 10 consecutive trials during complete muscle relaxation.[25] 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.

Total MEP latencies and central motor conduction time (CMCT) were determined with suprathreshold (150% motor threshold) single TMS pulses. Short interval intracortical inhibition (SICI) and intracortical facilitation (ICF) were examined in a conditioning-test pulse TMS paradigm[26] at complete rest. The first conditioning shock had an intensity of 75% of motor threshold. The intensity of the second pulse was adjusted to produce an MEP of ~0.5 mV peak-to-peak size. The following interstimulus intervals were tested: 2, 3, 10, and 15 ms.

Patient 1 was unable to relax the FDI on the affected side. In order to improve comparability of results, recordings from the unaffected hand were made during voluntary innervation of the unaffected FDI. Her paired-pulse results were not included in the statistical analysis since pre-innervation of a muscle is known to modulate SICI and ICF.[27] TMS single pulses with an intensity that allowed obtaining MEPs of ~1 mV peak-to-peak size at rest were applied during motor imagery.

Subjects were asked to imagine a tonic adduction of the index finger of the "paretic" hand, a tonic adduction of the nonaffected index finger and a tonic adduction of both index fingers simultaneously. 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. The frequency of TMS was ~0.1 Hz. Auditory feedback via a loudspeaker ensured that, during imagery, no muscle contraction occurred. For each of the six motor imageries, 6 TMS pulses were applied. Trials with imagery were mixed randomly with 24 TMS pulses given during rest. Recordings were stored on a Viking IV (Nicolet, Kleinostheim, Germany) and analyzed off-line. M responses, F wave amplitudes and MEP amplitudes were measured peak-to-peak.

CMCT was determined according to the formula: Total MEP latency—([M response latency + F wave latency − 1]/2). In the paired pulse paradigm conditioned MEP amplitudes were expressed as a percentage of the mean MEP amplitude following single TMS pulses. Each interstimulus interval was tested eight times and unconditioned test MEPs 24 times in a random order. To increase the power and to obtain a representative mean value for ICI and ICF we combined conditioned MEP values at interstimulus intervals of 2/3 ms 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. The MEPs obtained during motor imagery were expressed as percentage of the MEPs obtained at rest.

Statistics

An analysis of variance (ANOVA) with the factor GROUP (three levels: affected hand, unaffected hand of the patients and healthy control group, between-subjects analysis) was performed to explore differences in motor thresholds. The same analyses were calculated for CMCT values, SICI and ICF, respectively.

Motor Imagery.

A two-factorial ANOVA with the factors CONDITION (three levels: motor imagery of the index finger ipsilateral to the recording, of the index finger contralateral to the recording and of both, within-subjects analysis) and the factor SIDE (two levels: affected hand and unaffected hand of the patients, within-sub-

jects analysis) was calculated. Results obtained in the control group were compared with results from the patients in a two-factorial ANOVA with the factors CONDITION (three levels: motor imagery of the index finger ipsilateral to the recording, of the index finger contralateral to the recording and of both, within-subject analysis) and GROUP (two levels: affected hand of the patients and healthy control group, between-subjects analysis). When significant P values occurred in the ANOVA post hoc Tukeys t-test were applied. The level of significance was defined as $P < 0.05$.

RESULTS

In all four patients, motor thresholds and CMCTs were equal on both sides and not significantly different from motor thresholds and CMCTs in the healthy control group (see Table 1). For the comparison of SICI and ICF between both sides and control group, respectively, Patient 1 was excluded. She presented a reduction of SICI and of ICF on the affected side (SICI: 73.1%; ICF: 89.6%) and, to a lesser extent, on the unaffected side (ICI: 42%; 83%). In the other three patients SICI and ICF values of the affected side were neither significantly different from the unaffected side nor from the control group (Table 1). However, it should be noted that the patient group tended to have less ICF on both sides.
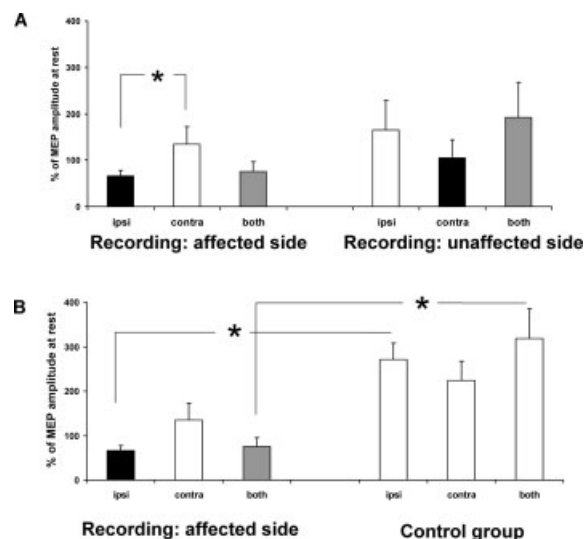
Motor Imagery.

ANOVA calculation indicated a significant effect of SIDE ($F_{[1,7]} = 8.2$; $P = 0.014$) and a significant interaction between SIDE and CONDITION ($F_{[1,7]} = 5.6$; $P = 0.02$) but no significant effect of CONDITION. Post hoc t tests indicated that MEP amplitudes evoked during motor imagery with the affected index finger were significantly different from those evoked during

FIG. 1. MEP amplitudes during imagination of tonic index finger adductions, expressed in percentage of the mean MEP amplitude obtained by single pulse TMS during rest. *: $P < 0.05$. **A**: recording from the first dorsal interosseous (FDI) muscle of the affected side in patients and from the control group. **B**: recording from the unaffected FDI in patients. Aff, imagined movement of the affected index finger; unaff, imagined movement of the unaffected index finger; Both, imagined movement of both index fingers; IL, imagined movement of the index finger ipsilateral to the FDI from which is being recorded; CL, imagined movement of the index finger contralateral to the FDI from which is being recorded.

imagery with the nonaffected finger while recording from the affected FDI ($P = 0.027$, Fig. 1A). The comparison between patients and healthy subjects showed a significant effect of the factor GROUP ($F_{[1,11]} = 77.7$, $P < 0.0001$) and a significant interaction between CONDITION and GROUP ($F_{[1,11]} = 7.1$; $P = 0.004$). Post hoc t tests indicated significant group differences for imagination of ipsilateral finger movement ($P < 0.0001$) and bilateral finger movement ($P = 0.0002$, Fig. 1B). Figure 2 demonstrates that, on an individual

TABLE 1. *Motor thresholds (MT), central motor conduction time (CMCT), short interval intracortical inhibition (SICI), and intracortical facilitation (ICF) in the patients and the age-matched healthy control group*
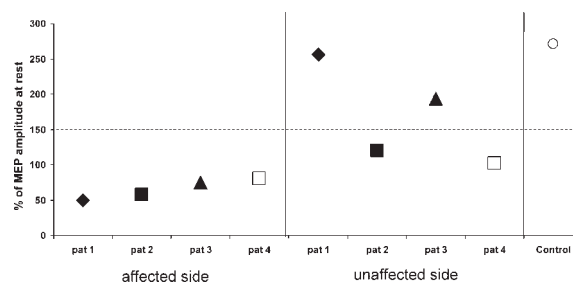
	Patients, AS	Patients, UAS	Control group
MT	38.8 ± 4.8	38.8 ± 5.1	38.4 ± 5.4
SICI	18.8 ± 6.7	23 ± 5.6	20.1 ± 8.8
ICF	125.6 ± 35.5	102 ± 3.7	178.1 ± 55.7
CMCT	5.6 ± 0.9	5.7 ± 0.8	5.5 ± 1

MT is presented as percentage of the maximum stimulator output intensity, CMCT values are shown in msec, SICI and ICF are demonstrated as percentage of the mean MEP amplitude evoked by single pulse TMS. Data are shown as mean ± standard deviation. AS, affected side; UAS, unaffected side.

FIG. 2. MEP amplitudes obtained during movement imagination in each patient. Individual results in the four patients in comparison to the mean result in the control group (open circle). The horizontal line indicates the 2.5 standard deviation of the control group. The patient data represent recordings from the first dorsal interosseous muscle ipsilateral to the imagined index finger movement.

level, the affected side of each patient was below the 2.5 standard deviation of the mean value of the control group. It is also noteworthy that motor imagery of index finger adductions with the healthy hand was below normal range in two patients even without being clinically affected.

DISCUSSION

This study demonstrates an abnormal decrease of corticospinal excitability during motor imagery when recording from the affected limb of patients with functional paresis. Normally, motor imagery induces an increase of excitability, as demonstrated in our control group and by other groups of investigators.[9–23] If our patients had not co-operated when being instructed to imagine an index finger adduction, one would expect that MEP amplitudes recorded during the imagination period would be identical to those obtained at rest. However, since MEPs were (on average) only 66% of the MEP size at rest, we suggest that motor excitability had been down-regulated. We hypothesize that this loss of excitability is not under voluntary control. The excitability changes during motor imagery resemble MEP changes observed during a "NoGo" task in healthy volunteers. In that experimental set up, subjects were asked to react quickly in response to an auditory "Go" signal by imagining squeezing hands but to imagine suppression of TMS-induced twitching movements after receiving another signal (the "NoGo" signal). Compared with the control condition (no imagination) MEPs amplitudes were significantly suppressed after the "NoGo" signal.[28] Using a slightly different study design, Coxon et al.[29] reported similar results. They also found greater intracortical inhibition in "Stop trials" compared to "Go trials" and concluded that volitional inhibition of a prepared action is exerted at a cortical level. This finding is supported by a functional magnetic resonance imaging study in which the correlates of an inhibited movement were identified. The authors described a multifocal network including premotor, primary sensorimotor, superior parietal, cingulate cortex, and cerebellar areas.[30] Using vibrotactile stimulation, brain imaging techniques have already described various abnormalities in functional sensory disorders. In patients with functional loss of sensation the application of sensory stimuli produced a decreased or no activation in the corresponding primary sensorimotor cortex.[31–33] During vibratory stimulation of both hands regional cerebral blood flow was reduced in the thalamus and basal ganglia contralateral to a functional sensorimotor impairment.[34] This abnormality vanished

with the loss of symptoms. These studies may be interpreted as indicators of an enhanced cortical and subcortical inhibition in the hemisphere contralateral to the functionally impaired limb. Our study extends these findings by demonstrating enhanced inhibition in the motor system.

From a neurological point of view the four patients we present here show a strong clinical heterogeneity. Therefore, it is even more remarkable that they all share a reduction of corticospinal excitability during motor imagery as a common feature. This may suggest that this finding is a basic property belonging to the development of a motor conversion symptom and its electrophysiological underpinnings. From a psychiatric point of view, however, the patients show a similar psychological condition that can be described as being caught in a situation of psychosocial impasse that one cannot leave by own means. The question may arise as to whether this method can be used to validly differentiate between "true" functional paresis and malingerers. Since it has been demonstrated that healthy subjects are able to suppress their corticospinal excitability to some extent[28] we doubt that such a differentiation is possible alone on grounds of a single diagnostic procedure. The diagnosis of functional paresis is first and foremost a clinical one, based on a comprehensive clinical evaluation that ideally integrates an exhaustive (biographical) anamnesis with a solid neurological and psychiatric evaluation.[35] This is all the more true as some sort of malingering may be found in patients with obvious conversion disorder. However, the present results might indicate the possibility to use TMS during motor imagery as an additional diagnostic tool that does not only exclude structural lesions but also supports the diagnosis of a functional paresis actively. In addition, our results may help to understand some of the mechanisms that lead to the inability to move voluntarily and thus help to further elucidate the nature of the pathophysiological processes underlying the disorder. In a recent study, high-frequency (15 Hz) repetitive TMS (rTMS) was applied to the motor cortex of patients with a motor conversion disorder in order to reduce symptoms. This type of stimulation is known to induce an increase of motor excitability. The authors reported beneficial results with motor improvements in three of four patients after up to 12 weeks of rTMS stimulation.[36] We suggest that our results can provide an explanation for the effectiveness of excitatory rTMS. Possibly, rTMS was able to reverse the abnormal inhibition of corticospinal excitability in these patients and thus support them in re-establishing motor programs.

Given the small sample size in our study, our results have to be considered preliminary. Future studies will not only explore effects of motor imagery in a larger group of subjects but will also investigate the underlying mechanisms in greater detail, e.g. by exploring connectivity between different motor areas.

REFERENCES

1. Cantello R, Boccagni C, Comi C, Civardi C, Monaco F. Diagnosis of psychogenic paralysis: the role of motor evoked potentials. J Neurol 2001;248:889–897.
2. Krem MM. Motor conversion disorders reviewed from a neuropsychiatric perspective. J Clin Psychiatry 2004;65:783–790.
3. Marshall JC, Halligan PW, Fink GR, Wade DT, Frackowiak RS. The functional anatomy of a hysterical paralysis. Cogn 1997;64: B1–B8.
4. Decety J, Grezes J. Neural mechanisms subserving the perception of human actions. Trends Cogn Sci. 1999;3:172–178.
5. 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.
6. Jeannerod M, Frak V. Mental imaging of motor activity in humans. Curr Opin Neurobiol 1999;9:735–739.
7. Kuhtz-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;18:3375–3387.
8. Pascual-Leone A, Nguyet D, Cohen LG, Brasil-Neto JP, Cammarota A, Hallett M. Modulation of muscle responses evoked by transcranial magnetic stimulation during the acquisition of new fine motor skills. J Neurophysiol 1995;74:1037–1045.
9. Abbruzzese G, Trompetto C, Schieppati M. The excitability of the human motor cortex increases during execution and mental imagination of sequential but not repetitive finger movements. Exp Brain Res 1996;111:465–472.
10. Yahagi S, Shimura K, Kasai T. An increase in cortical excitability with no change in spinal excitability during motor imagery. Percept Mot Skills 1996;83:288–290.
11. Kasai T, Kawai S, Kawanishi M, Yahagi S. Evidence for facilitation of motor evoked potentials (MEPs) induced by motor imagery. Brain Res 1997;744:147–150.
12. Kiers L, Fernando B, Tomkins D. Facilitatory effect of thinking about movement on magnetic motor-evoked potentials. Electroencephalogr Clin Neurophysiol 1997;105:262–268.
13. Fadiga L, Buccino G, Craighero L, Fogassi L, Gallese V, Pavesi G. Corticospinal excitability is specifically modulated by motor imagery: a magnetic stimulation study. Neuropsychologia 1999;37: 147–158.
14. Hashimoto R, Rothwell JC. Dynamic changes in corticospinal excitability during motor imagery. Exp Brain Res 1999;125:75–81.
15. Rossini PM, Rossi S, Pasqualetti P, Tecchio F. Corticospinal excitability modulation to hand muscles during movement imagery. Cereb Cortex 1999;9:161–167.
16. Facchini S, Muellbacher W, Battaglia F, Boroojerdi B, Hallett M. Focal enhancement of motor cortex excitability during motor imagery: a transcranial magnetic stimulation study. Acta Neurol Scand 2002;105:146–151.
17. Stinear CM, Byblow WD. Motor imagery of phasic thumb abduction temporally and spatially modulates corticospinal excitability. Clin Neurophysiol 2003;114:909–914.
18. Stinear CM, Byblow WD. Modulation of corticospinal excitability and intracortical inhibition during motor imagery is task-dependent. Exp Brain Res 2004;157:351–358.
19. Levin O, Steyvers M, Wenderoth N, Li Y, Swinnen SP. Dynamical changes in corticospinal excitability during imagery of unimanual and bimanual wrist movements in humans: a transcranial magnetic stimulation study. Neurosci Lett 2004;359:185–189.
20. Li S, Kamper DG, Stevens JA, Rymer WZ. The effect of motor imagery on spinal segmental excitability. J Neurosci 2004;24: 9674–9680.
21. Vargas CD, Olivier E, Craighero L, Fadiga L, Duhamel JR, Sirigu A. The influence of hand posture on corticospinal excitability during motor imagery: a transcranial magnetic stimulation study. Cereb Cortex 2004;14:1200–1206.
22. Fourkas AD, Avenanti A, Urgesi C, Aglioti SM. Corticospinal facilitation during first and third person imagery. Exp Brain Res 2006;168:143–151.
23. Cicinelli P, Marconi B, Zaccagnini M, Pasqualetti P, Filippi MM, Rossini PM. Imagery-induced cortical excitability changes in stroke: a transcranial magnetic stimulation study. Cereb Cortex 2006;16:247–253.
24. Quartarone A, Bagnato S, Rizzo V, et al. Corticospinal excitability during motor imagery of a simple tonic finger movement in patients with writer's cramp. Mov Disord 2005;20:1488–1495.
25. Rossini PM, Barker AT, Berardelli A, 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.
26. Kujirai T, Caramia MD, Rothwell JC, et al. Corticocortical inhibition in human motor cortex. J Physiol 1993;471:501–519.
27. Ridding MC, Taylor JL, Rothwell JC. The effect of voluntary contraction on cortico-cortical inhibition in human motor cortex. J Physiol 1995;487:541–548.
28. Sohn YH, Dang N, Hallett M. Suppression of corticospinal excitability during negative motor imagery. J Neurophysiol 2003;90: 2303–2309.
29. Coxon JP, Stinear CM, Byblow WD. Intracortical inhibition during volitional inhibition of prepared action. J Neurophysiol 2006; 95:3371–3383.
30. Hummel F, Saur R, Lasogga S, et al. To act or not to act. Neural correlates of executive control of learned motor behavior. Neuroimage 2004;23:1391–1401.
31. Tiihonen J, Kukka J, Viinamaki H, Lehtonen J, Partanen J. Altered cerebral blood flow during hysterical paraesthesia (letter). Biol Psychiatry 1995;37:134–135.
32. Mailis-Gagnon A, Giannoylis I, Downar J, et al. Altered central somatosensory processing in chronic pain patients with "hysterical" anesthesia. Neurol 2003;60:1501–1507.
33. Ghaffar O, Staines R, Feinstein A. Unexplained neurologic symptoms: an fMRI study of sensory conversion disorder. Neurol 2006;67:2036–2038.
34. Vuilleumier P, Chicherio C, Assal F, Schwartz S, Slosman D, Landis T. Functional neuroanatomical correlates of hysterical sensorimotor loss. Brain 2001;124:1077–1090.
35. Stone J, Carson, A und Sharpe, M. Functional symptoms and signs in neurology: assessment and diagnosis. J Neurol Neurosurg Psychiatry 2005;76Suppl 1:2–12.
36. Schonfeldt-Lecuona C, Connemann BJ, Viviani R, Spitzer M, Herwig U. Transcranial magnetic stimulation in motor conversion disorder: a short case series. J Clin Neurophysiol 2006;23: 472–475.