

Rapid Communication

Electroencephalographic spectral power in writer's cramp patients: Evidence for motor cortex malfunctioning during the cramp

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We investigated cortical activation as reflected in task-related spectral power (TRPow) changes in 8 writer's cramp patients during writing on a digital board and during isometric contraction and compared them to those of 8 age-matched healthy subjects. Scalp EEG was recorded over the contralateral primary sensorimotor area (SM1_c), and from the ipsilateral sensorimotor area (SM1_i). The electromyogram (EMG) was recorded from the Extensor Digitorum Communis (Extensor), Flexor Digitorum Superficialis (Flexor), and First Dorsal Interosseous (FDI) muscles. We analyzed (1) handwriting performance, (2) changes in the TRPow confined to alpha and beta band, and (3) the EMG spectral power during both tasks, writing and isometric contraction. During writing, all patients developed writer's cramp. The handwriting in writer's cramp patients was associated with significantly less reduction of the beta-range TRPow and lower frequency of the TRPow reduction compared to controls. No significant differences between patients and controls for the alpha band TRPow reduction during handwriting were observed. During writing, the patients showed higher EMG spectral power than the controls but this difference was at the border of significance. The present results indicate disorder in the motor execution system, in writer's cramp patients, associated with impaired functional beta-network state of the contra- and ipsilateral sensorimotor cortices, most probably due to inadequate modulation of the intracortical inhibition associated with writing.

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Introduction

The amplitude decrease in alpha and beta band cortical oscillations called event-related desynchronization (ERD)

(Pfurtscheller and Aranibar, 1977) was shown to consistently reflect the functional activation of the sensorimotor areas during the preparation for a voluntary movement (Babiloni et al., 1999; Feige et al., 1996; Pfurtscheller and Lopes da Silva, 1999; Stancak et al., 2000).

Amplitude decrease in alpha- and beta-frequency range was described not only during motor preparation but also during continuous motor performance: Manganotti et al. (1998) have investigated task-related spectral power (TRPow) changes during sequential finger movements. They found that the movement sequences were associated with TRPow decreases in alpha- and beta-frequency bands over the bilateral sensorimotor and parietal areas, with contralateral preponderance. The spatial extent and the magnitude of the spectral power decreases were greater for complex movements of higher complexity than for simpler ones. Using ECoG in a visual-motor decision task Crone (2000) and Crone et al. (1998a,b) focused on spectral power changes during execution of motor output rather than on its planning. These authors also used the term ERD for the amplitude decrease in alpha- and beta-frequency range while their subjects made sustained isometric muscle contraction in different body parts in response to randomized visual stimuli depicting each action.

Writer's cramp represents a task-specific kind of focal dystonia (Marsden and Sheehy, 1990) characterized by co-contraction of agonist and antagonist muscles and recruitment of muscles usually not involved in writing (Berardelli et al., 1998).

Among the many electrophysiological abnormalities shown to play a role in the pathophysiology of dystonia, the most important one is the deficient inhibition (Hallett, 1998): The mechanism of the co-contraction of the antagonists was shown to be related to deficient reciprocal inhibition at spinal and subcortical levels of the central nervous system (Rothwell et al., 1983). Deficient inhibition was described at cortical level as well: Feve et al. (1994) and Deuschl et al. (1995) have demonstrated reduced amplitude of the negative slope of the movement-related cortical

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potentials, which is thought to be generated in the motor cortex. Yazawa et al. (1999) recorded Bereitschaftspotentials preceding voluntary muscle contraction and relaxation and compared them to those in controls. They demonstrated that the cortical preparatory process for voluntary muscle relaxation is abnormal in focal hand dystonia as well. Abnormally reduced activation in motor cortical areas for both voluntary movement contraction and relaxation was also shown by event-related fMRI (Oga et al., 2002). Using a contingent negative variation paradigm, Hamano et al. (1999) suggested that motor programming is specifically abnormal for the affected body part, including the asymptomatic contralateral limb. This finding suggested deficient cortical motor control as well.

Experimental evidence about increased excitability of the primary motor cortex due to deficient intracortical inhibition (ICI) was provided also by rTMS (repetitive transcranial magnetic stimulation): In a “double pulse paradigm”, Ridding et al. (1995) have shown less inhibition bilaterally in patients with hand dystonia than in normal subjects. The finding of Siebner et al. (1999) that 1-Hz rTMS can reinforce deficient cortical inhibition and improve handwriting supports also the notion that reduced ICI plays a role in the pathophysiology of focal hand dystonia. These rTMS findings coincide well with findings from PET studies: Investigating voluntary movements in dystonic patients, Ceballos-Baumann et al. (1995) have shown abnormal suppression of regional cerebral blood flow in the caudal supplementary motor area and sensorimotor areas bilaterally. Abnormal suppression of regional cerebral blood flow in the contralateral sensorimotor cortex and SMA was also shown by Ibanez et al. (1996).

Toro et al. (2000) investigated the movement-related desynchronization related to self-paced index finger abduction movements in patients with writer’s cramp and controls. They found that the movements of the affected and unaffected hand in the patients were accompanied by less reduction in the beta (20 to 30 Hz) spectral power compared to controls. Further, the alpha spectral power reduction did not show significant differences between patients and controls. The authors interpreted the beta-range abnormality as a manifestation of the abnormal motor command at cortical level. The abnormality found by Toro et al. (2000) was demonstrated for the movement pre-execution state. It was of interest to investigate whether there are abnormalities in the oscillatory cortical activity confined to alpha and beta-range during execution of a motor task. This was the first aim of the present study, which investigated whether writer’s cramp patients differ from normal subjects in terms of oscillatory aspects of regional cortical activation. For this purpose, we investigated the EEG spectral power changes in alpha- and beta-frequency band during experimentally induced cramp by writing on a digital board. To test whether the spectral power changes are restricted to the symptomatic setting only as shown by Chen et al. (1997) using TMS in a “double pulse” paradigm or whether the physiological abnormality is more generalized, we introduced a second experimental task: maintained motor contraction task, i.e., a task not inducing any cramp in the writer’s cramp patients. The basic hypothesis was that the beta-power suppression will be less in writer’s cramp patients during writing.

Since both investigated tasks represented steady-state paradigms for the task-related spectral power changes, we preferred to use the term task-related power (TRPow) coined by Mangano et al. (1998). The TRPow changes were related to behavior and to the EMG spectral power. The latter was used as a measure for the

strength of the muscle contraction. The results from the writers’ cramp patients were compared to the results from age-matched controls. Recordings were made over the two cortical areas shown to be mostly involved in the writer’s cramp impairment: the contralateral sensorimotor area (SM1_c), and the ipsilateral primary sensorimotor (SM1_i) area (Chen et al., 1995; Deuschl et al., 1995; Nakashima et al., 1989).

Methods

Subjects

Eight right-handed patients with writer’s cramp participated in the study (two female and six male; age 42.2 ± 11.7 years). Clinical details about the writer’s cramp patients are shown in Table 1. The mean duration of the symptoms was 7.7 ± 5.96 years. Six of the patients had simple writer’s cramp. Two of the patients (P4 and P7) had dystonic writer’s cramp according to the definition of Marsden and Sheehy (1990), i.e., they had symptoms not only during writing but also during other motor activities (cf. Table 1). None of the patients had been treated with botulinum toxin or any other drug 6 months prior to the study.

Eight healthy volunteers (two female and six male; mean age 41.5 ± 10.9 years) acted as age-matched control subjects. None of the control subjects had any history of neurological disease.

All subjects participated according to the declaration of Helsinki, with informed consent and the approval of the local ethics committee. The handedness was tested according to a modified Oldfield questionnaire (Oldfield, 1971). The subjects had no previous experience with similar experiments.

Experimental paradigm

Prior to the experimental session, the patients were interviewed and video-recorded during writing of the sentence used in standard investigations of handwriting “Die Wellen schlagen hoch” (“The waves rise up high”) (Mai and Marquardt, 1994; Siebner et al., 1999).

During the experimental session, the individual was sitting in an electrically shielded, dimly lit room. The dominant hand and arm were supported in a rigid cast.

Table 1
Writer’s cramp patients’ details

Patient	Duration (years)	Type of dystonia	Disability	Task specificity
P1	1.5	Mild finger flexion	Mild	Writing
P2	5	Finger flexion and mild wrist flexion	Mild	Writing
P3	4	DII extension	Mild	Writing
P4	7	Finger flexion	Mild	Also when cutting with knife
P5	2	Finger flexion	Mild/moderate	Writing
P6	6	Finger flexion	Mild/moderate	Writing
P7	10	DI–DII extension	Moderate	Also when putting on make-up
P8	20	DI–DIII flexion	Moderate	Writing

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