

Introduction

Writer's cramp (WC) is a type of focal dystonia [1] involving the sensorimotor circuit, probably with a heterogeneous biological, environmental and psychological background. Many kinds of intervention have been already attempted, with limited effectiveness and beneficial duration for the patients.

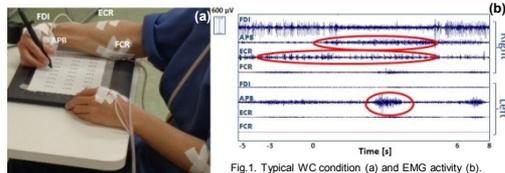


Fig. 1. Typical WC condition (a) and EMG activity (b).

EEG based Brain Computer Interface (BCI) [2] has been recently shown to be a promising tool for the rehabilitation from this disease (see Fig.2) [3]. Nevertheless, no exhaustive EEG study is yet available to determine which feature should drive the neurofeedback to achieve the most effective results.

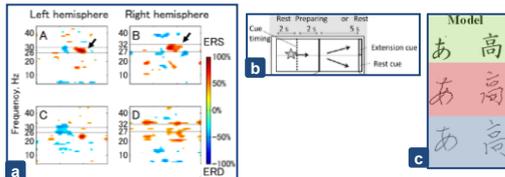


Fig. 2. Summary from BCI pilot study on WC patient (permission from Hashimoto et al. [3]). EEG features before and after training (a). BCI paradigm (b). Functional outcomes (c).

Therefore, the purpose of this study is to identify the abnormal EEG pattern(s) that can be considered biomarker(s) for the WC condition and be targeted by a suitable BCI in future.

Methods

- **EEG acquisition:**
 - 31 EEG channels using International 10/20 System.
 - monopolar derivation with reference on FCz.
- **EEG preprocessing:** band-pass elliptic filter in (5,45) Hz.
- **Participants:** 7 WC patients (age 50±15, 4M+3F, onset age 45±15).
- **Paradigm:**
 - Two different tasks:
 1. Execution of pinch grip with right hand (ME) for 5s.
 2. Kinesthetic imagination of the same movement (MI) for 5s.
 - 100 trials per task (randomized between ME and MI, see Fig.3).
 - rest between trials randomly between 5 and 6 s.
- **Offline analysis:**
 - Time-frequency analysis over the 5-45 Hz frequency band via spectrogram (500 ms Hamming window with 250 ms overlap).
 - Event-related (de)synchronization(ERD/ERS) as in [4].
 - Significance via explained variance (results shown with $R^2 \geq 0.05$).

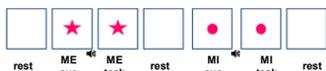


Fig. 3. Paradigm for the current study. Randomly presented stimuli for ME and MI pinch grip on a real pen.

Results

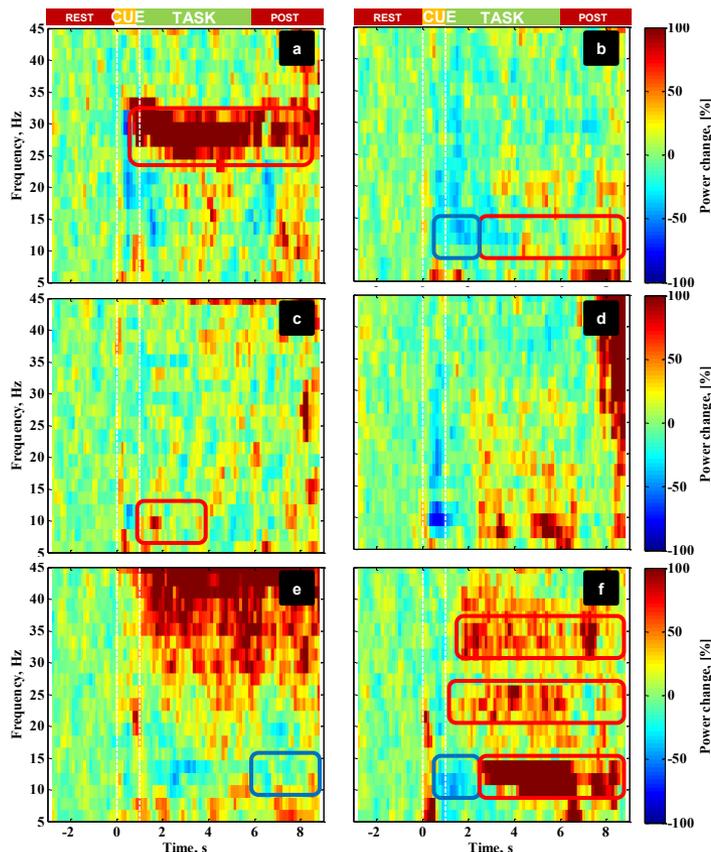


Fig. 4. Abnormal EEG patterns. Blue represents ERD, red ERS. Rectangles include only significant values ($R^2 \geq 0.05$). (a) high β ERS during task. (b) Abrupt switch between ERD and ERS during task. (c) Delayed ERD in μ band at task beginning. (d) absence of significant μ or low β ERD. (e) absence of consistent β rebound (ERS) at the termination of the task. (f) Coexistence of multiple abnormal patterns. Quantification of ERD/ERS (mean±std) reported in Table 1.

	PATTERN (a)	PATTERN (b)	PATTERN (c)	PATTERN (d)	PATTERN (e)
ME	15.2±6.7	-14.2±8.3(ERD) 9.5±7.6(ERS)	27.1±6.5	1±2.3	-7.1±9.9
MI	70.9±35.6	-10.1±10.7(ERD) 64.1±38.6(ERS)	1.91±2.5	0.3±1.2	0.6±11.5

Table 1. Quantification of abnormal patterns shown in Fig. 4. Values represent %ERD (negative values) or %ERS (positive values). Each patterns is quantified by mean ± standard deviation of significant ($R^2 \geq 0.05$) power changes as in [4].

	Abnormal high β	Abnormal ERD during task	Abnormal ERS at termination
ME	2	7	6
MI	3	5	6

Table 2. Summary of results. With reference to Fig.4, "Abnormal high β " includes only pattern (a). "Abnormal ERD during task" includes patterns (b) to (d). "Abnormal ERS at termination" includes pattern (e).

Discussion

Expectations from well-known literature on healthy subjects:

- (1) Significant power decrease (ERD) during the task in μ band (around 10 Hz) and β band (around 20 Hz) over sensorimotor cortices, especially the contralateral, compared to a baseline period selected during the rest before the task.
- (2) Significant power increase (rebound/ERS) in β band within one second from the termination of the task.

Findings on WC patients (see Table 2 and Fig. 4):

- 1) "Abnormal high β " oscillations: significant abnormal power increase in high β (above 25 Hz) band (pattern a) during ME (2 patients) and MI (3 patients).
- 2) "Abnormal ERD during task": switch from ERD to ERS (pattern b), delayed ERD (pattern c) or complete suppression of ERD (pattern d) in μ or low β band during ME task (7 patients) and MI task (5 patients).
- 3) "Abnormal ERS at termination": absence of β rebound/ERS at the termination of the task (6 patients, both ME and MI).

Limitations of the present study:

- limited number of patients
- absence of age-matched control group

Conclusions

- Besides the abnormality in the high β band, previously suggested in the literature [3], other abnormal patterns - still far from the healthy case - could be identified in WC patients.
- Heterogeneity of phenomenology in WC condition could be found in their EEG recordings.

Future perspectives:

The investigation on the relationship between the different frequency components (cross-frequency coupling) and their behavior during the occurrence of the dystonic symptoms could provide a support for these first interesting outcomes and clarify whether pathological or compensatory mechanisms led to the observed abnormal EEG patterns. Later, BCI should trigger the pathological mechanism that could be either in the low frequency band (μ and low β) or the high frequency band (high β).

References:

- [1] M. Hallett, *Pathophysiology of writer's cramp*, Hum.Mov.Sci.2006.
- [2] S. Silvoni et al., *Brain-computer interface in stroke: a review of progress*, Clin. EEG Neurosci.2011.
- [3] Y. Hashimoto et al., *Functional recovery from chronic writer's cramp by brain-computer interface rehabilitation: a case report*, BMC Neurosci., 2014.
- [4] G. Pfurtscheller and F.H. Lopes da Silva, *Event-related EEG/MEG synchronization and desynchronization: basic principles*, Clin. Neurophysiol., 1999.