Poster 692

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Abnormal EEG oscillations in β and γ bands in writer's cramp

Cisotto G.[1,2], Kita K.[2,3], Uehara K.[2], Yoshinaga K.[2], Hashimoto Y.[4], Sakamoto T.[5], Ushiba J.[6], Hanakawa T.[2]

[1]Dept. Information Engineering, University of Padua, Italy, [2]Integrative Brain Imaging Center, National Center of Neurology and Psychiatry, Tokyo, Japan, [3]Center for Frontier Medical Engineering, Chiba University, Chiba, Japan, [4]Kitami Institute of Technology, Hokkaido, Japan, [5]Department of Neurology, National Center, Hospital of Neurology and Psychiatry, Tokyo, Japan, [6]Department of Biosciences and Informatics, Keio University, Tokyo, Japan

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.

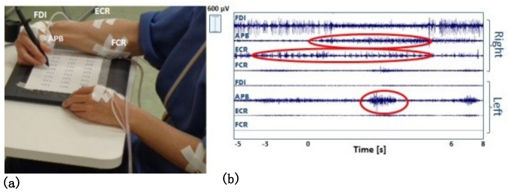

Fig.1. Typical WC pathological posture (a) and dystonic EMG activity (b).

EEG based Brain Computer Interface (BCI) has been recently shown to be a promising tool for the rehabilitation from this disease [2,3].

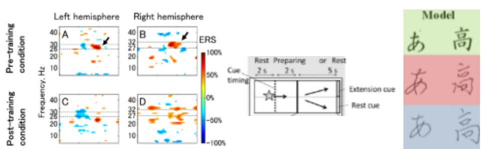

Fig.2. Summary from BCI pilot study on WC patient (permission from Hashimoto et al.). EEG features before and after training (a). BCI paradigm (b). Functional outcomes (c).

Nevertheless, no exhaustive EEG study is yet available to determine which feature should drive the neurofeedback to achieve the most effective results.

Methods

- 11 WC patients (age 45±12.6, 6M+5F)
- 10 healthy subjects (age 39.7.1±12.9, 5M+5F)
- Task: pen-holding execution with right hand (100 trials)
- Time-frequency analysis over the 5-45 Hz frequency band via spectrogram and event-related (de)synchronization (ERD or ERS) computation as in [4].
- Significance was assessed by cluster-based statistics (α=0.05, 75-percentile).

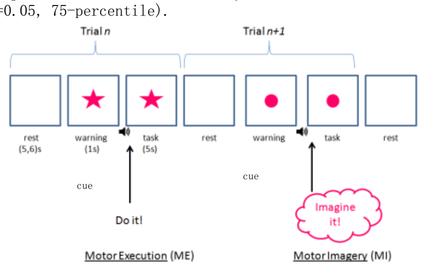

Fig.3. Paradigm for the current study. Randomly presented stimuli for motor execution (ME) and imagery (MI) of a pen-holding task, using a real pen.

Results

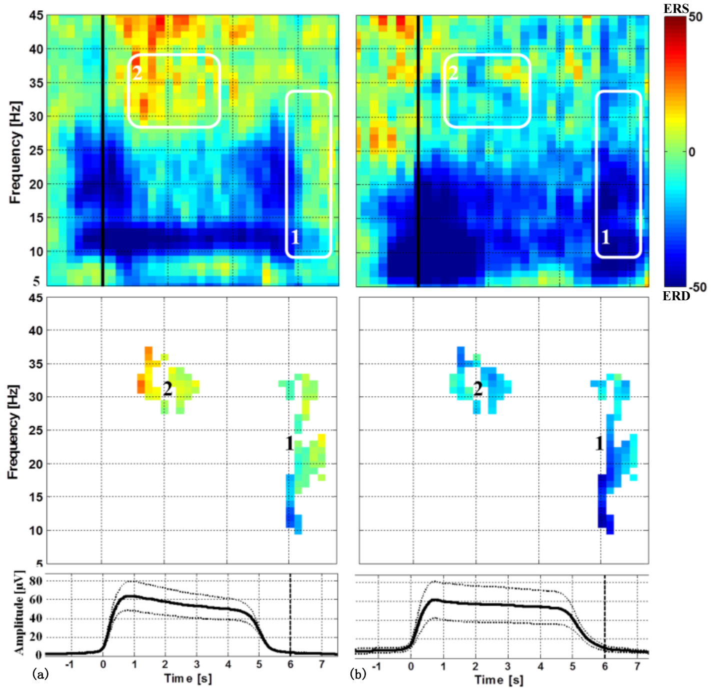

Fig.3. Grand-averaged, time-frequency distributions of power changes at C3. Colour codes for the percentage strength of ERD (blue) and ERS (red). a) Healthy subjects (HS); b) WC patients. Below, average (solid line) and standard error (dashed line) of first dorsal interosseous (FDI) activation in each group. White boxes identify clusters where significant between-groups difference existed.

Results on healthy subjects confirmed previous literature: μ ERD was seen from ~1s before movement to the end of it; β ERD was found at beginning and termination of muscle contraction, with β rebound (ERS) occurring after termination. Moreover, low γ band ERS was found during muscle contration (task execution phase).

In case of WC patients, sustained μ and β ERD was found, with no β rebound within 1s after the termination of movement. Moreover, low γ band showed ERD during the muscle contration, too.

Cluster No.	Cluster Width [bins]	HS Mean ± se [%]	WC Mean ± se [%]	Time	Frequency
1	55	-4.16 ± 1.06	-23.19 ± 0.98	[5.25 7]s	10-34 Hz
2	34	10.96 ± 0.72	-15.99 ± 0.56	[0.75 3]s	28-38 Hz

Table 1. Mean and standard error of power changes in every cluster, in both groups (third and fourth columns). Cluster number (first column) refers to Fig. 3. Time and frequency ranges (fifth and sixth columns) refer to the smallest rectangular box (white boxes in Fig.3) that includes the correspondent cluster.

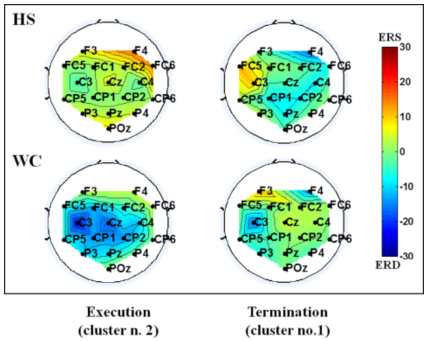

Fig.4. Grand-averaged power changes topography in every cluster, both in WC and HS group. Plots were grouped by movement phase (execution and termination). Cluster number and colours coding for ERD and ERS is as in Fig. 3.

Cluster-based statistical analysis confirmed two statistically significant clusters that distinguish healthy subjects from patients (see Table 1): they represent abnormality in low γ band during execution and in β band after termination of the movement, respectively.
Topography revealed a focalized ERD at C3 (contra-lateral electrode), for both clusters.

Discussion

(1) Long-range brain-muscle communications have been shown to rely on ~20Hz β oscillations [5]. Then, the abnormality found in this band may relate to the impaired balance of excitation-inhibition mechanisms during contraction and relaxation in WC patients.

(2) Short-range communications within the sensorimotor circuit have been shown to exploit γ oscillations[6]. Therefore, abnormal γ band behaviour could suggest pathological processing of sensorimotor information in WC patients.

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 preliminary interesting outcomes and clarify whether pathological or compensatory mechanisms led to the observed abnormal EEG patterns.
Indeed BCI should then trigger the pathological mechanism that could be either in the low frequency band (μ and low β) or the high frequency band (low γ band).

References:

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