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Motor Imagery Based Brain-Computer Interface for Cerebellar Impaired Patients

Young-Seok Choi

***Abstract*—**Cerebellar ataxia is a steadily progressive neurodegenerative disease associated with loss of motor control, leaving patients unable to walk, talk, or perform activities of daily living. Direct motor instruction in cerebella ataxia patients has limited effectiveness, presumably because an inappropriate closed-loop cerebellar response to the inevitable observed error confounds motor learning mechanisms. Could the use of EEG based BCI provide advanced biofeedback to improve motor imagery and provide a "backdoor" to improving motor performance in ataxia patients? In order to determine the feasibility of using EEG-based BCI control in this population, we compare the ability to modulate mu-band power (8-12 Hz) by performing a cued motor imagery task in an ataxia patient and healthy control.

***Keywords*—**Cerebellar ataxia, Electroencephalogram, brain-computer interface, motor imagery.

I. Introduction

CEREBELLAR ataxia is a rare neurodegenerative disease associated with loss of motor control. Some independence could be restored through the use of a brain-computer interface (BCI), which has been used to decode brain signals to drive a computer controller, a wheelchair joystick, or even a prosthetic arm for other patients with motor impairments.

BCIs use electrophysiological measures of brain function to enable individuals to communicate directly with their external world, bypassing normal neuromuscular pathways.

Recently, noninvasive BCIs have used a variety of electroencephalogram (EEG) based features to communicate the intent of the user, such as slow cortical potentials and event-related desynchronization via motor imagery. This noninvasive EEG-BCI has been a highly active research topic in neuroscience, engineering, and signal processing. One of the reasons for this development is the remarkable advances of BCI systems with respect to usability, information transfer, and robustness for which modern machine learning and signal processing techniques have been instrumental [1].

One of the most important characteristics of the EEG recorded over the sensorimotor cortex is linked to possible modulation of EEG rhythms through simple motor imagery, e.g., imagining a flexion of the right or left elbow. A widely used rhythm for control is the "mu" rhythm (8-12 Hz). The reason for utilizing this is that it shows an increase in power during relaxation (event-related synchronization, ERS), and similarly, a decrease during real and imaginary motor movement performance (event-related desynchronization, ERD) [2]. This characteristic can be utilized to control a cursor in at least one dimension. The two electrodes shown to have the largest weight of mu rhythm are located at C3 and C4 or adjacent positions, but recruitment of more electrodes could be necessary for control of more sophisticated movements.

EEG mapping may be distorted in the setting of neurologic disease, which may affect the ability of ataxia patients to use EEG-based BCI. EEG is a rough measure of neural activity, based on the voltages generated by the firing of large populations of neurons, as recorded over time from the scalp at discrete sites. Although the literature states that the EEG pattern is "normal" in cerebellar degeneration, this presumably refers to the lack of heightened epileptogenic potential, which does not necessarily indicate that the EEG is comparable to that of unaffected individuals. We hypothesize that cortical regions of the corticocerebellar circuit may show functional abnormalities when they are connected to areas of primary cerebellar degeneration. The strong interconnections between the cerebellum and the cerebral cortex most likely contribute to the distortion in the processing of sensory feedback.

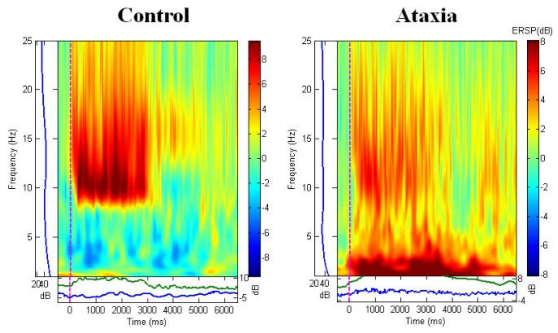
Electrophysiological studies in ataxia are rarely performed as early EEG studies were reportedly normal [3], [4]. There are, however, some disease-specific differences in visual evoked potentials, [5] and auditory evoked potentials, [6] suggestive of white matter disease located outside of the cerebellum and its direct connections. Seizures are not a common clinical manifestation, except in certain rare subtypes, such as acetazolamide-responsive paroxysmal ataxia [7]; EEG findings are consistent with the epileptic phenotype.

Electrophysiological biomarker studies in other non-epileptic brain conditions demonstrate that EEG measures are useful for detecting clinically relevant, disease-specific differences [8]. However, electrophysiological studies in ataxia are not routinely performed in the clinical setting, given that early EEG studies were reportedly normal [9]. There are, however, some disease-specific differences in visual evoked potentials, auditory evoked potentials, and auditory brain stem response suggesting that there may be white matter disease located outside of the cerebellum and its direct connections.

There is some evidence that motor imagery is affected in cerebellar ataxia. In a study of people with unilateral cerebellar stroke, patients attempting motor imagery showed decreased motor evoked potential facilitation in the associated motor cortex [10]. In a second study, patients that had apparently recovered from a unilateral cerebellar stroke showed a marked slowing of motor performance in both hands (ipsi- and contralateral to lesion). This effect was accompanied by a

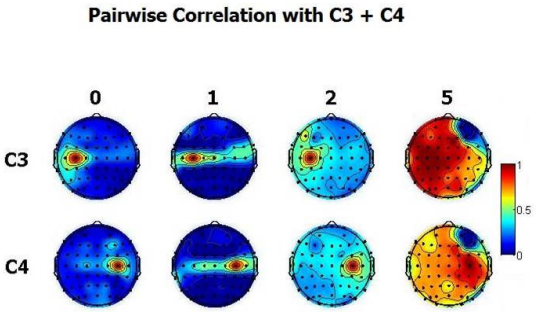
Y.-S. Choi is with the Department of Electronic Engineering, Gangneung-Wonju National University, 7 Jukheon-gil, Gangneung, 210-702 Republic of Korea (phone: +82-33-640-2429; fax: +82-33-646-0740; e-mail:yschoi@gwnu.ac.kr).

similar slowing of motor imagery, suggesting that the cerebellum, traditionally implicated in the control of motor execution, is also involved in nonexecutive motor functions such as the planning and internal simulation of movements [11].

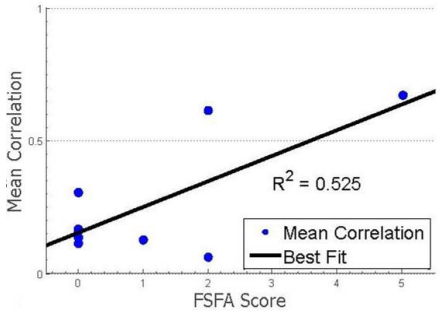


(a) (b)

Fig. 1 The event related spectral power (ERSP) differences between the averages of the two task condition for the control (a) and ataxia (b) subjects from the C3 electrode



(a)



(b)

Fig. 2 (a) Pairwise correlation of bandpass-filtered signals between each electrode and C3/C4. Increasing ataxia severity (FSFA scores [0-5] listed in the top row) is associated with broadened spatial distribution of correlation, (b) Linear regression of mean correlation against FSFA, R2 = 0.525

In order to demonstrate feasibility in this first study of non-invasive, EEG-based BCI in cerebellar ataxia, we assessed the ability to modulate mu-band power during a cued motor imagery task. We also examined possible differences in performance associated with ataxia, as these differences might necessitate modification in BCI decoding algorithms.

II. Methods

Four cerebellar ataxia patients (Functional Staging for Ataxia FSFA score 1-5 [12]) and five control subjects provided informed, written consent according to a research protocol approved by the Johns Hopkins University Institutional Review Boards. Subjects had never previously used an EEG-based BCI interface.

During each trial, subjects were visually cued either to enter a state of relaxation (target appears at the top of a computer screen) or to imagine motor movement (target appears at the bottom). A three-state (move up, move down, remain still) EEG-based BCI was used to control the position of a cursor in one dimension on a computer screen. EEG Signals were acquired using a QuickCap 64-channel EEG cap (modified 10-20 system, referenced between Cz and CPz, and grounded anteriorly to Fz; Compumedics, El Paso, TX). The amplifier and signal processing modules were connected through client-server architecture, with a Neuroscan SynAmps2 64-channel amplifier system from Compumedics (El Paso, TX) acting as the server, and the signal processing module running on a separate client computer. Data were sampled at 250 Hz, with a band-pass filter applied between 0.1 and 30 Hz, and transmitted over a TCP/IP protocol to the client PC for storage and real-time signal processing using a custom BCI platform.

EEG signals were spatially filtered using common average referencing. The C3 and C4 electrodes, which generally overlap with the hand-area of the primary motor cortex, were then used in an autoregressive (AR) model to determine the power spectrum [13] as

$$y_E[n] = \sum_{k=1}^{K} a_k y_E[n-k] + e[n], \quad (1)$$

where E denotes the electrode of interest, a_k denote the AR coefficients, is the order of AR model, and is an independent identically distributed stochastic sequence sith zero means and variance σ^2 [14]. Here, K was set to 15. In addition, Burg's spectral estimation method was used to estimate the time-varying AR coefficients. Then, the power spectral density (in dB) of the AR processes was obtained by

$$P(\omega) = \frac{\sigma^2}{|1 - \sum_{k=1}^{K} a_k e^{-ik\omega}|} \quad (2)$$

and the mu-band (8-12 Hz) power was determined at time instants t_k as the mean of $P(\omega_\mu)$, where, ω_μ denotes the mu-band frequency range.

The sum of the mu band power of the C3 and C4 electrodes were used to train a two stage hierarchical linear classifier. A gating classifier G was designed to identify significant modulations of power due to intention, which is given by

$$G(t_k) = \begin{cases} 1 \text{ if } \left| w_{1G} P_{C3}(t_k) + w_{2G} P_{C4}(t_k) + B_G \right| > T_G \\ 0 \qquad\qquad\qquad \text{otherwise} \end{cases} \quad (3)$$

where w_{1G} w_{2G}, B_G, and T_G are the weights, bias, and threshold, respectively, as determined online. A second movement classifier was designed to distinguish between the relaxation and the motor imagery task,

$$M(t_k) = \begin{cases} +1 \text{ if } \left| w_{1M} P_{C3}(t_k) + w_{2M} P_{C4}(t_k) + B_M \right| > T_M \\ -1 \qquad\qquad\qquad \text{otherwise} \end{cases} \quad (4)$$

where w_{1G} w_{2G}, B_M, and T_M are he weights, bias, and threshold, respectively, as determined online. Finally, the output of classifiers was the product of the two classifiers,

$$F(t_k) = G(t_k) \times M(t_k) \quad (5)$$

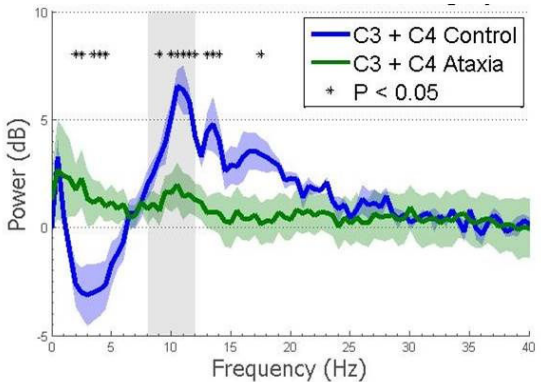
where +1 corresponds to relaxation, -1 to motor imagery, and 0 to no task. If the cursor reached a target 7 steps away from the center where trials start, within 15 seconds, the trial was considered a success. To achieve a trial success case, the sum of $F(t_k)$ over all t_k within that trial must equal +7 in the relaxation trials, and -7 in the motor imagery trials before 15 seconds elapse. Subjects underwent 16 trials each set (8 relaxation and 8 movement imagery trials), with a pseudo-randomized order of presentation within each set.

To determine spatial correlation, the signal amplitude over the duration of the experiment was band-pass filtered (Butterworth) from 8-12 Hz. Correlations between the time-domain signals at each channel were taken pairwise with C3, then C4. To determine spectral correlation, the Spearman correlation between the mu-band power and successive 1 Hz bins was taken.

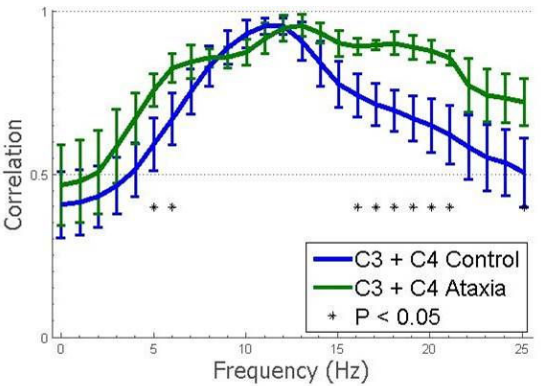
III. Results

Our primary endpoint was to evaluate the possibility that ataxia patients could achieve control of a BCI using cued motor imagery. Indeed, subjects were able to achieve mean trial success of greater than 13.21% (chance performance rate) on their first session. Chance performance was calculated as the probability of reaching the target based on an equal probability of performing any of the three possible movements with each step, up to the maximum allowable 30 steps. The average successful chance trial duration was determined as the first moment of chance successes rates for the allowable step counts.

Fig. 1 suggests that the representative ataxia patient and control subject show an increased difference in power in the mu-band (8-12 Hz), between relaxation and motor imagery. Darker shades of red represent an increase in power during the relaxation task over the motor imagery task at the same time point and frequency band within the trial. The blue trace on the left indicates average power of each frequency. The bottom blue trace indicate minimum power over the range of frequencies at each time point, while the bottom green trace indicates maximum power at each time point. This difference is greater in the control subject as compared to the ataxia patient. Although trials continued on beyond 4 seconds, the power difference between the two tasks is not as obvious, due to the inability of subjects to maintain the required imagination throughout the task duration. The ataxia subject had higher power activity in low frequency bands, which is most likely due to movement artifacts.



(a)



(b)

Fig. 3 (a) C3/C4 mean power shift ± SEM. Ataxia patients show decreased amplitude and lack of frequency specificity, (b) C3/C4 mean spectral power correlation with mu band ± SEM. Ataxia patients show broader correlation

Fig. 2 shows the pairwise correlation of bandpass filtered signals between each electrode and C3 and C4. Control subjects achieved the expected modulation, which was modulation, which was isolated to the mu peak and was located primarily in C3 and C4. However, ataxia patients showed a different control profile in Fig. 2 (a), showing higher correlation of C3 and C4 activity with surrounding brain regions. Increasing ataxia severity (FSFA scores listed in the top of figure) is associated with broadened spatial distribution of correlation. With more cases, it is clear that the spatial correlation is broadened with increasing FSFA score (R2=0.525).

Fig. 3 shows, the spectral correlation over frequency ranges for both control and ataxia groups. Power modulation was not isolated to mu-band range, but was correlated with broad changes in other spectral bands. Futhermore, spectra of ataxia

subjects showed a smaller mu band peak during relaxation. Here, p-value was used.In Fig. 3 (a); it shows C3/C4 mean power shift standard error of mean (SEM) and resulting p-value. Ataxia subjects show decreased amplitude and lack of frequency specificity. Fig. 3 (b) shows C3/C4 mean spectral power correlation with mu band SEM. From figure, we come to know that ataxia patients show broader correlation.

IV. Conclusions

Our works demonstrate that despite the theoretical possibility that patients with severe ataxia may have impaired motor imagery and abnormal cortical rhythms, an ataxia patient is capable of generating sufficient changes in cortical rhythms to achieve voluntary control of an EEG based BCI using cued motor imagery.

Although BCI control is possible for ataxia patients, electrophysiology is abnormal. Impaired synchronization of circuits could result in spectral smearing of the resulting signal, explaining the less pronounced mu-band peak during ERS as well as the increased bandwidth modulated during ERD. This reduced amplitude could lead patients to use alternative strategies to achieve BCI control, such as compensatory modulation of more distal brain regions. This could provide the necessary power shift and would explain the observed broadening of spatial correlation. Intriguingly, these EEG differences correlate with disease severity, suggesting that EEG modulation could be used as a biomarker of disease progression or to train motor imagery.

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