

Electroencephalography Based Brain-Computer Interface for Cerebellum Impaired Patients

Young-Seok Choi

Abstract—In healthy humans, the cortical brain rhythm shows specific mu (~6-14 Hz) and beta (~18-24 Hz) band patterns in the cases of both real and imaginary motor movements. As cerebellar ataxia is associated with impairment of precise motor movement control as well as motor imagery, ataxia is an ideal model system in which to study the role of the cerebello-cortical circuit in rhythm control. We hypothesize that the EEG characteristics of ataxic patients differ from those of controls during the performance of a Brain-Computer Interface (BCI) task. Ataxia and control subjects showed a similar distribution of mu power during cued relaxation. During cued motor imagery, however, the ataxia group showed significant spatial distribution of the response, while the control group showed the expected decrease in mu-band power (localized to the motor cortex).

Keywords—Brain-computer interface, EEG, modulation, ataxia.

I. INTRODUCTION

CEREBELLAR ataxia is a rare movement disorder characterized by cerebellar degeneration. As ataxia progresses, motor movement control gradually worsens, often to the point where patients can no longer walk, talk, or perform activities of daily living [1]. Rare diseases such as cerebellar ataxia are often overlooked because relatively few individuals are affected; however, ataxia could provide us with useful insight into a human lesion model in the control center of the motor system. This will help to improve our basic understanding of motor imagery and control. Because degeneration appears to be the most severe in the cerebellum [2], it is possible that the output of the presumably intact motor cortex is capable of being correctly interpreted to form the intended movements of the patient. In exploring therapeutic interventions, we hope to bypass the corrupted movement commands of the defective cerebellum. Doing so would allow us to reconstruct the original intention of motor cortex commands, and thus regain motor control.

Furthermore, recent studies [3] show that unilateral cerebellar stroke affects not only motor performance, but also motor imagery. This suggests that the cerebellum is also involved in nonexecutive motor functions, such as the planning and internal simulation of movements. Our preliminary results show that EEG abnormalities in patients with cerebellar ataxia can partially normalize with learning, and that short-term EEG-BCI feedback facilitates these changes. Encouraged by these promising findings, we predict that long-term practice of

a motor imagery task using EEG-BCI will be associated with long-term changes in the typical EEG pattern.

Electroencephalography (EEG) is a measure of neural activity, based on the voltages generated by the firing of large populations of neurons. These easily-recognizable electrical rhythms are recorded from specific areas on the scalp, and may be indicative of the state of the brain. 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). The most widely used rhythm for motor control is the “mu” rhythm (~8-12 Hz). Mu rhythm shows an increase in power during relaxation (event-related synchronization) and a decrease during real and imaginary motor movement performance (event-related desynchronization) [4]. This characteristic can be utilized with a BCI interface to allow mental control of a computer cursor in at least one dimension [5]. 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 more sophisticated control. This BCI method has been successfully tested with normal subjects, as well as patients suffering from various forms of locked-in syndrome [6]. Prior to our study, it had not been tested with Ataxia patients, in part due to the sparseness of the subject population, which is estimated at about 0.05% of Americans [1]. By having ataxia patients control a simple cursor task through EEG modulation, we tested the hypothesis that BCI is an effective means of restoring limited motor function. Since a byproduct of cerebellar feedback pathway degeneration is a lack of localization of motor modulation, a relative decrease or increase in power in the mu band would manifest itself not only over the hand area, but would appear as a more globally diffuse change in mu power. Based on the clinically evident impairment of motor movements and reported impairment of motor imagery, we predicted that ataxia patients would not initially perform as well as controls at the BCI task, but would eventually regain some level of motor control.

II. METHODS

Four cerebellar ataxia patients and six control subjects were selected to perform the task. Each subject sat in front of a computer screen, and was asked to imagine movement or relaxation without actually moving. EEG signals were collected with a 64-electrode scalp cap connected to a SynAmps amplifier system (Neuroscan, Charlotte, NC).

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).

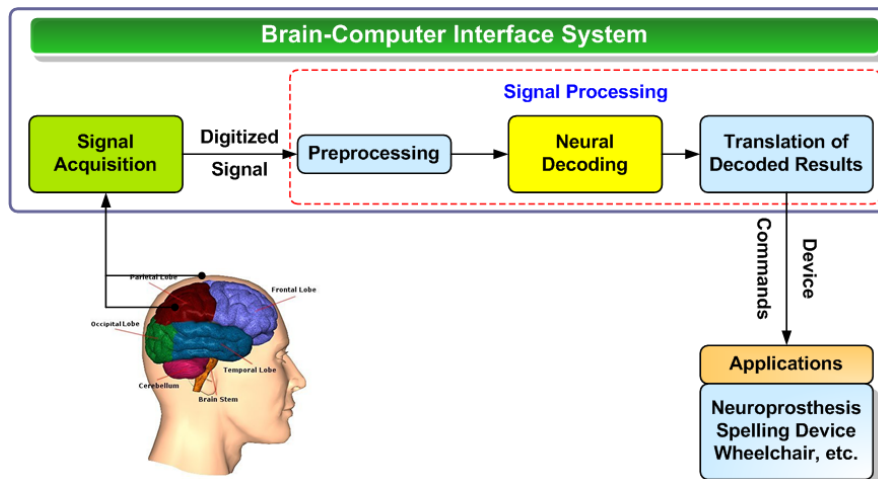


Fig. 1 General process of BCI system

EEG Signals were acquired via a QuickCap 64-channel EEG cap (modified 10-20 system), referenced between Cz and CPz, and grounded anteriorly to Fz. 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. An EMG electrode was attached to the flexor and extensor muscles on the subject's forearm, and routed through the SynAmps headbox. EEG signals were sampled at 250 Hz and transmitted over a TCP/IP protocol to the client PC for storage and real-time signal processing using a custom BCI platform. The output of the amplifier was connected to a PC, which spatially filtered the signals using common average referencing. The C3 and C4 electrodes, which coincide with the hand-area of the primary motor cortex, were then used in an autoregressive model in order to determine the power spectrum.

A general system for BCI consists of several signal processing steps, shown in Fig. 1. During relaxation, all of the subjects tested exhibited a peak generally centered between 6-14 Hz. By imagining bilateral movements, subjects could temporarily suppress the power in this frequency band with variable effectiveness. Based on the combined amplitude of the peaks in the C3 and C4 electrodes, a movement classification was made, which was then output to a cursor control program. In this program, a target was displayed either toward the top (re-laxation trial) or the bottom (movement trial) of the computer screen, with the imagery-controlled cursor in the middle. Based on whether the power spectrum indicated the subject was imagining movement or relaxation, as determined every 500 milliseconds, the cursor moved either down or up by a fixed increment, with seven steps from the origin to the target. A trial ended either when the cursor reached the target or when 15 seconds had passed. A variable number of blocks were run, generally between 6 and 9.

The exact number of blocks depended on whether the subject achieved a consistent level of success that did not improve after subsequent blocks. Each block consisted of 16 trials (8

relaxation and 8 movement trials), which were presented in random order throughout each block.

III. RESULTS

Our comparisons showed no obvious trends in the data to suggest that performance alone is enough to classify subjects into control or ataxic categories (Fig. 2).

Fig. 3 shows the power spectral trends (based on control or ataxic status) that were recorded from electrodes C3 and C4. The results of this analysis suggest that in contrast to controls, ataxic subjects do not have a significant mu peak during relaxation. Furthermore, we observed a difference in the beta band (~18-24Hz) between relaxation and motor imagery that appeared in ataxic patients, but was absent in control data.

Fig. 4 shows the averaged mu band scalp maps. These maps demonstrate that while control subjects greatly increased mu power in the area covering the motor strip during relaxation, ataxic patients appeared to have a relatively smaller increase in mu power during relaxation. In ataxia patients, the more modest increase in power is a less localized phenomenon; for example, there appears to be a greater change in power over the frontal lobes in ataxic subjects only in addition to changes over the motor cortex.

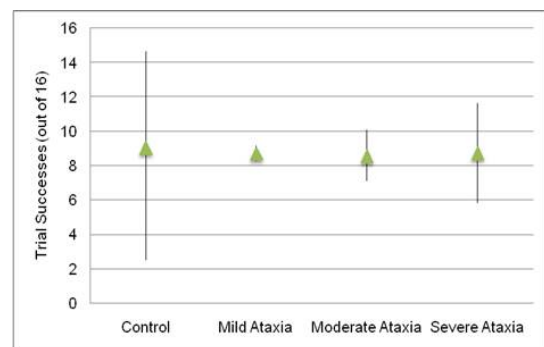


Fig. 2 Trial success rates show the variability in subject success rates grouped by condition. Triangles indicate the mean success rate of all subjects for each condition, with lines representing the range of average successes for each subject in that category

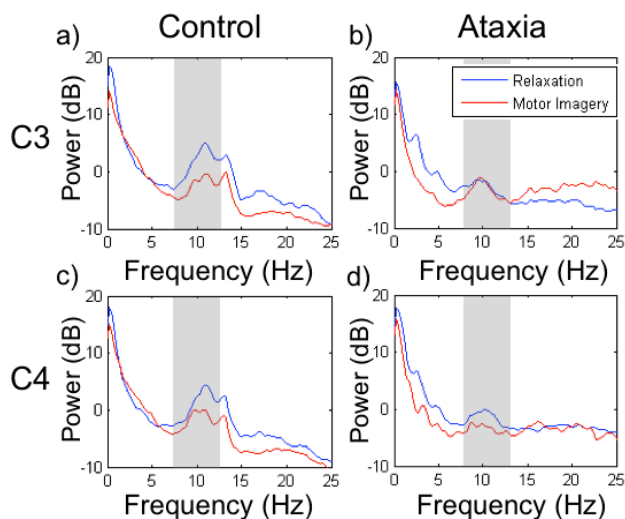


Fig. 3 The power spectrum averaged over all successful trials are displayed according to subject type (controls, 6 subjects: a,c, ataxia patients, 4 subjects: b,d), along with the channel of interest (C3: a,b, C4: c,d). Blue lines represent the power spectrum from trials in which the goal was to move the cursor upward by relaxing, while red lines represent trials in which the goal was to move the cursor downward by imagining movements. The control subjects have a more prominent peak during relaxation in the mu band (highlighted grey) than ataxia patients, as well as a larger separation between trial conditions

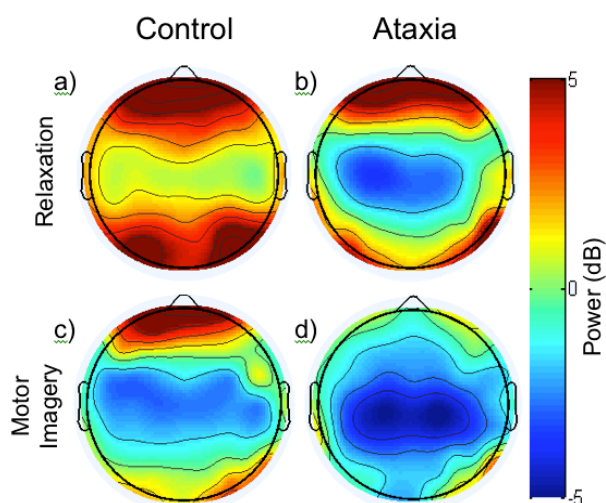


Fig. 4 Topographic maps of mu average power during relaxation (a, b) or movement imagery (c, d) in control subjects (a, c) and ataxia patients (b, d). These maps demonstrate that while control subjects increased mu power during relaxation with a focus in the area covering the motor strip, ataxia patients appeared to have a relatively smaller, but more global increase in power during relaxation

IV. DISCUSSION

Our results show clear differences between the EEG signals of ataxic patients and controls during a motor imagery task. Success and learning rates do not appear to be directly related to the clinical diagnosis of the subject. This implies that to use BCI for diagnostic purposes, some degree of analysis must be performed on the EEG data. On average, ataxic patients have a smaller increase in mu band power during imagined relaxation,

compared to controls. This suggests that ataxic patients are unable to properly modulate the synchronous firing of large groups of neurons in the motor cortex due to a deterioration of feedback pathways from the cerebellum. While overall activity in this area may be increased, this lack of increased synchronization results in the non-existence of a prominent mu peak.

Contrary to our initial expectations, the ataxic subjects were able to perform the task with no obvious decrease in performance compared to controls. This suggests that despite not having a significant mu peak in the same frequency range, individual subjects are capable of creating a mu power separation between relaxation and movement imagination, but each with different peak frequencies. This causes these peaks to disappear after spectrum averaging. One of our hypotheses was that due to decreased cerebellar feedback modulation, motor imagination would not be localized to just the area of cortex corresponding to the part of the body where the movement was visualized. Instead, we expected to observe more global increases in neuronal synchronization in ataxia patients, which was demonstrated in our results. This indicates that ataxia patients are capable of some amount of compensation for a loss of feedback by increasing global activity. Those who cannot generate large, focused increases in power are able to create more diminished, but well-distributed increases in power to achieve the same impact. In this case, it represents the control of a BCI.

V. CONCLUSIONS

While using our current setup allows ataxic subjects to control a BCI with similar efficacy to control participants, it is clear that the neural method of control is different between the populations. It may be possible to take advantage of these differences to create a BCI that ataxia patients would be able to control more easily, perhaps by determining the average power over several central electrodes, instead of just C3 and C4. By creating a BCI specific to ataxia patients, we may be able to increase their ability to naturally control an end-effector, and with continuous training, improve their motor control skills. For severe ataxia patients, who are wheelchair bound, and unable to even maintain a steady grip on a cup, this could mean a significant improvement in quality of life.

Our preliminary data show robust and consistent differences between the EEG patterns of ataxia patients and control subjects during a motor imagery task. Exploring these differences may be vital to gaining a better understanding of cerebellar ataxia, which currently has no cure, and its impact on the human brain. The EEG differences we have uncovered could be used as a diagnostic tool, and may find a role in rehabilitation therapy.

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