Is Breathing Rate a Confounding Variable in Brain-Computer Interfaces (BCIs) Based on EEG Spectral Power?

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***Abstract*— Brain-computer interfaces (BCIs) enable paralyzed patients to interact with the world by directly decoding brain activity. We investigated if systematic changes in breathing rate affect EEG bandpower features that are commonly used in BCIs. This is of particular interest for the development of cognitive BCIs for patients with artificial ventilation, e.g. for those in late stages of amyotrophic lateral sclerosis (ALS). If subjects can alter the spectrum of the EEG by changing their breathing rate, decoding results obtained with healthy subjects may not generalize to this patient population. We recorded a high-density EEG from twelve healthy subjects, who were instructed to alternate between fast and slow breathing. We do not find any statistically significant modulation of EEG bandpower. As such, changes in breathing rate are unlikely to substantially bias the performance of BCIs based on EEG bandpower features.**

I. INTRODUCTION

Brain-computer interfaces (BCIs) enable subjects to communicate by self-regulating their brain activity [10]. In BCIs based on electroencephalography (EEG), it is well known that electromyogenic (EMG) activity, arising from muscles covering the skull, as well as electrooculographic (EOG) activity, arising from eye movements and eye blinks, are potential confounding variables [2]; that is, subjects may (unintentionally) use these non-cortical contributions to the EEG to control the BCI. Because the spectral- and spatial characteristics of EMG and EOG artifacts differ from those of cortical sources, it is possible to attenuate the contribution of these non-cortical sources to the EEG [9]. However, cortical sources may also be affected by confounding factors. There is a trend in the BCI community towards systems that are based on high-level cognitive tasks [11], [5]. Because alternating between tasks with variable cognitive demands has an effect on breathing patterns [12], and changes in breathing patterns can be detected in the EEG [13], it is conceivable that task-induced changes in breathing rate bias the decoding performance of cognitive BCIs. Because these effects may manifest in cortical sources, it may not be possible to distinguish task-induced changes of the EEG from those that are mediated by breathing rate. This is of particular concern in the development of BCIs for patients who are artificially ventilated, e.g. those in late stages of amyotrophic lateral sclerosis (ALS). If breathing rate is a confounding variable in cognitive BCIs, decoding results obtained with healthy subjects may not transfer to patient populations.

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We studied this problem by instructing healthy subjects to up- and down-regulate their natural breathing rate while recording a high-density EEG. We then investigated if changes in breathing rate modify the spectral power of the EEG. Correcting for multiple comparisons, we do not find any statistically significant modulation of EEG power. The strongest effects in the α (8–14 Hz) and in the γ (35–85 Hz) range only explain up to 4% of the EEG's variance in spectral power. We conclude that, when decoding subjects' intentions from EEG bandpower changes, variations in breathing rate are unlikely to substantially bias the performance of cognitive BCIs.

II. METHODS

A. Experimental Paradigm

The experimental paradigm consisted of two blocks. In the initial control block, we recorded the participants' natural breathing patterns. In the following experimental block, we instructed subjects to up- and down-regulate their breathing rate.

The control block consisted of three phases of recordings, each of five minutes length. In the first five minutes, in which we recorded a subject's baseline activity, only a white cross was shown in the middle of the black screen and the subject was asked to relax with eyes open. Information about the subject's natural breathing rate was acquired during this phase. For the second phase of five minutes, the subject was again asked to relax with eyes open and to watch a moving stimulus in the middle of the screen. The stimulus was a gray ball that pulsated at the subject's natural breathing rate, as measured in the first phase. For the last five minute recording phase, the subject was shown the same pulsating gray ball, but now he or she was asked to breathe according to the ball's movement, inhaling when the ball grew and exhaling when it contracted. The second block of recordings consisted of three sessions with 20 one-minute trials per session; ten for fast breathing and ten for slow breathing. Subjects were instructed to breathe according to the pulsating gray ball. Its oscillating frequency was varied between trials of fast- and slow breathing as described in Section II-C.1. The order of trials was randomized. The trials were separated by rest periods of approximately one second. The participants sat in a comfortable chair approximately 1.25 m away from a computer screen.

B. Data acquisition

Fourteen subjects participated in this study (28.7 $\pm$ 6.8 years of age (mean $\pm$ SD), two women and twelve men).

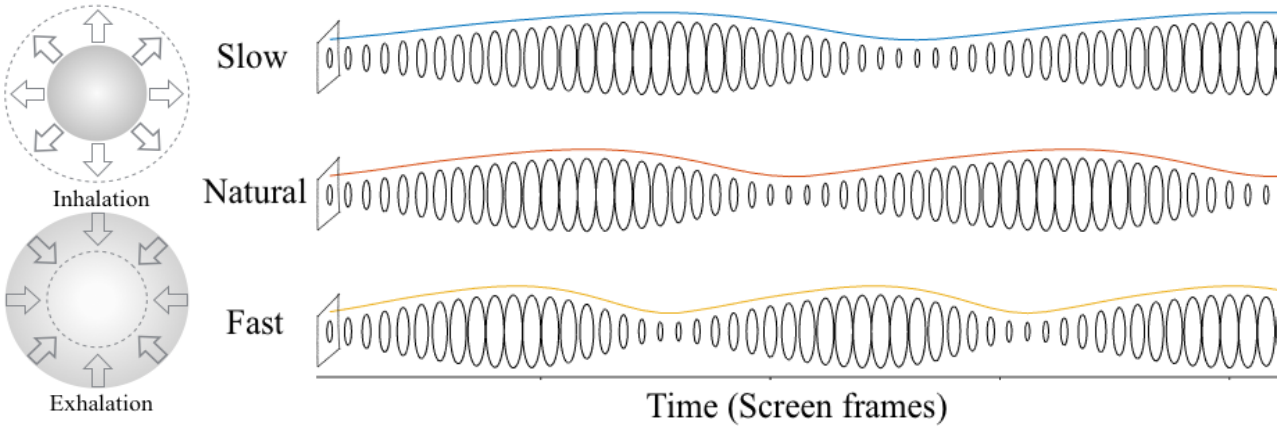

Fig. 1. Schematic representation of the stimulus, the ball, as it expanded and contracted through time. The lines above the circles are formed by the slow wave, natural breathing wave and fast wave, respectively. The lines were not viewed by the participants, only the changing circles were displayed on the screen. Each circle corresponds to a different screen frame. When viewed by the participant the circle appeared to expand and contract, to which the participants had to inhale and exhale accordingly.

Two subjects had to be discarded due to technical problems. All participants signed a consent document prior to the start of the experiment, in agreement with guidelines of the Max Planck Society and the principles outlined in the Helsinki Declaration of 1975, as revised in 2000. At the end of the session each participant filled out a questionnaire. We recorded the subjects' EEG using a 124-channel cap of act-iCAP active electrodes connected to a QuickAmp amplifier (recording at a sampling frequency of 500 Hz). We used a respiration belt to measure the subjects' breathing patterns. The breathing rate was also recorded by the QuickAmp amplifier at the same sampling frequency (500 Hz). The electrodes, amplifier and breathing belt were provided by BrainProducts GmbH, Gilching, Germany. Electrodes were placed according to the extended 1020 system with electrode Cz as the initial reference. All recordings were converted to common average reference. The experimental paradigm was programmed using the BCI2000 toolbox [4].

C. Data analysis

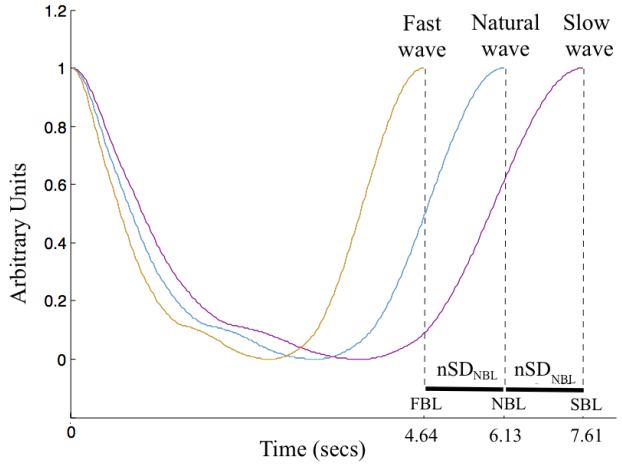

Fig. 2. Breathing Waveform Patterns calculated according to Section II-C.1. These curves were used to specify the how the radius of the ball changed, and thus how the participant ought to breath.

1) Breathing data analysis: The breathing data served two purposes. First, it was used to cue the participant on how to breathe. Second, it served as evidence that the participants followed our instructions. For each participant, the first five minutes of breathing recording were processed between the first and second control phases. In order to remove any linear trend, a linear model was fitted to the signal and subtracted. The resulting signal was then standardized. We separated each breathing cycle by localizing peaks on this signal. We used a manually chosen threshold to detect the peaks for each participant. The distance between peaks was averaged to obtain the average natural breathing length (NBL) of the participant, measured in seconds/breath. The standard deviation of the breathing length was also calculated. This was needed in order to determine the slow and the fast breathing lengths (SBL and FBL) characteristic of a particular subject. The slow breathing length was created by summing n times the standard deviation to the natural breathing length: $\text{SBL} = \text{NBL} + \text{nSD}_{\text{NBL}}$ (see Fig. 1). In a similar manner the fast breathing length can be expressed as $\text{FBL} = \text{NBL} - \text{nSD}_{\text{NBL}}$. The value of n was chosen as the maximum value from the set $\{2, 1.5, 1.5, 1.5, 1\}$ such that both the slow breathing length and fast breathing length remained within a natural, comfortable limit (between 10 and 40 breaths per minute) [7].

We then constructed the average breathing waveform pattern of the participant. This pattern conveys the way in which the person breathes, how long they inhale and how long they exhale. To calculate it, we first splined the signal of each breathing cycle so that it would be as long as the participant's NBL. Then we averaged all breathing cycles. The result was an average waveform pattern. We smoothed this waveform by fitting a least squares approximation using eight cosine-like functions. With these functions we were able to respect the participant's own proportion of inhaling versus exhaling time (which has an effect on arousal, relaxation and mindfulness [8]), while at the same time creating an approximation with smooth transitions between cycles. The smooth waveform

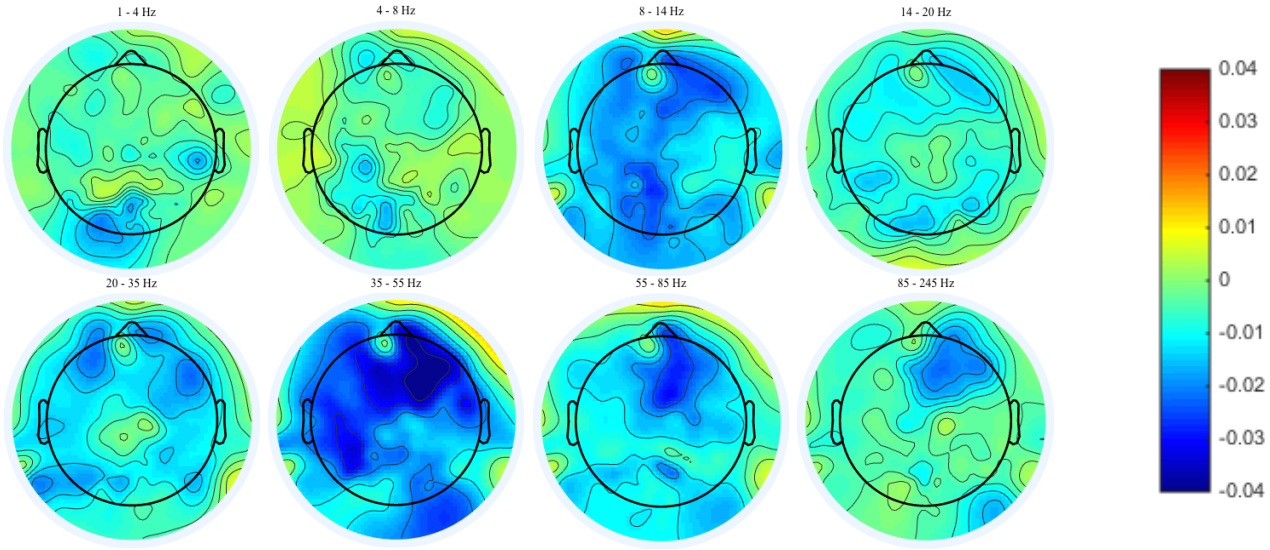

Fig. 3. Topographies of artifact-corrected group-average signed r^2 values in different frequency bands (n = 12).

pattern was used in the BCI application to dictate the expansion and contraction of the visual stimulus. This required one last processing step. The smooth waveform was splined again so that it spanned the same length of screen frames as the NBL of the participant. A slow wave and a fast wave were also created by changing the length of the smooth waveform breathing pattern. The slow wave was made as long as the SLB; likewise for the fast wave (see Fig. 1).

The same process was applied to the breathing data obtained from the experimental conditions. Each one minute trial data was fitted to a linear model in order to remove the linear drift. Afterwards, it was standardized. All 30 standardized trials for each participant and each condition were then concatenated into one vector and the rest of the analysis was carried out in the same way as above. The average breath lengths during slow and fast conditions were calculated. To determine whether or not each participant up-regulated and down-regulated his or her breathing rate, a t-test was used to test the null hypothesis of equal mean breathing lengths between conditions. The mean waveform breathing pattern for each condition was also calculated in order to conduct a visual inspection of the different breathing patterns per condition.

2) EEG data analysis: We used an artifact correction procedure based on independent component analysis (ICA) to remove prominent muscular components of the EEG signal as well as non-physiological artifacts. The artifact correction is described in detail in [5]: We first filtered the data using a high-pass Butterworth filter of order three with a cut-off frequency of 3 Hz. Then, we computed a principal component analysis by which we reduced the signal to 64 dimensions. We then separated it into independent components (ICs) using the SOBI-algorithm [1] and sorted the resulting ICs according to their neurophysiological plausibility [14]. This provided us with a topography, a spectrum, and a time-series of each IC. We determined by visual inspection whether or not each IC was of cortical origin. Any of the following four reasons were considered sufficient to reject an IC: (1) The spectrum did not show the $1/f$-behaviour typical of a cortical source. In particular, we rejected ICs that showed a monotonic increase in spectral power starting around 20 Hz, which is characteristic of muscular activity [2]. (2) The time-series showed eye blinks. (3) There was no dipolar pattern in the topography. (4) Noise, such as large spikes or 50 Hz line noise, were visible in the time-series. The sources that were kept were then projected back onto the scalp. Following the artifact correction, features from each trial were computed by a Fast Fourier Transform using a Hanning window. The features represented the average log-bandpower of the signal in the following frequency bands: 1–4 Hz (δ), 4–8 Hz (θ), 8–14 Hz (α), 14–20 Hz (low β), 20–35 Hz (high β), 35–55 Hz (low γ), 55–85 Hz (mid γ), and 85–245 Hz (high γ).

Next, we computed *signed* r^2 values to investigate the effect of the experimental conditions on EEG bandpower. r^2 values range from zero to one and represent the percentage of the variance of the features explained by up/down-regulation of breathing rate; the sign comes from the sign of the correlation coefficient r. For each participant, each channel, and each frequency band, a signed r^2 value was obtained using the features from all 60 trials. A permutation test (10,000 permutations) rendered a p-value associated with each of these signed r^2 values. We then averaged the signed r^2 values of all participants, ending up with one group signed r^2 for each channel and each frequency band.

We tested for each channel and frequency band if it displayed a statistically significant group-average signed r^2. To do so, we pooled together the twelve individual p-values of all participants for a given channel and frequency band. Because p-values are drawn from a uniform distribution with range zero to one if the null-hypothesis is true, we quantified the probability of the null-hypothesis by measuring the distance of the cumulative distribution function (CDF) of the original p-values with CDFs obtained by 1000

times randomly drawing twelve samples from a uniform distribution between zero and one. This gave us one p-value for each pair of channel and frequency band, to which a false discovery rate (FDR) correction ($\alpha = 0.01$) for multiple tests was applied [15].

III. Results

A. Breathing results

We confirmed that the participants modulated their breathing rate by, first, analyzing the results of the t-tests and, second, by evaluating the subjects' the responses in the questionnaire. All twelve t-tests rejected the null-hypothesis (mean of average breathing lengths between slow and fast trials equals zero) at significance level $\alpha = 0.05$. The participants reported that they were able to breathe according to the ball's rhythm. Their answer was 7.89 $\pm$ 1.14 (mean $\pm$ SD) on a scale from zero to ten, were zero stood for "I could not follow it at all" and ten stood for "I followed it perfectly".

B. EEG results

Fig. II-C.1 shows the group-average signed r^2-profile of log-bandpower modulation across different frequency bands. Following the FDR correction for multiple comparisons, none of the group-average signed r^2 were found to be statistically significant. Nonetheless, there is structure in the bandpower modulation. First, most of the signed r^2 values are negative. This implies that there tends to be less EEG power in the fast breathing-rate condition than in the slow breathing-rate condition. Second, bandpower modulation is most pronounced in the α and in the low γ range. In both frequency bands, there is a spatially contiguous frontal component. In the case of the low γ band, this frontal component remains visible in the medium and the high γ range. However, fast vs. slow breathing explains at most 4% of the variance in EEG power across trials for any given electrode and frequency band.

IV. Discussion

In this study, we did not find a statistically significant modulation of EEG bandpower by instructing subjects to alternate between fast and slow breathing. However, we observed spatially and spectrally coherent patterns of bandpower modulation, which contradicts the null-hypothesis of no bandpower modulation. As such, it is conceivable that a reproduction of this study on a larger number of subjects may find statistically significant modulations. In any case, the effects we observe are very weak. We hence conclude that it is unlikely that changes in breathing rate, that are induced by varying cognitive demands in BCI paradigms, have a substantial impact on the obtained decoding results. We note, however, that we only investigated bandpower changes of the EEG. It remains conceivable that BCIs based on event-related potentials (ERP) are affected by changes in breathing rate. In this work we carefully removed the artifacts of the EEG signal, therefore another question that remains open is whether or not breathing rate can be a confound for BCI studies that do not perform artifact correction.

ACKNOWLEDGMENT

We would like to thank Bernd Battes for his support in recording the experimental data.

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