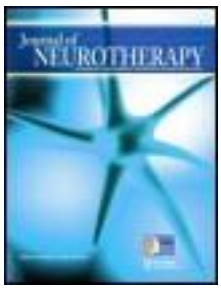

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Changes in EEG Spectrograms, Event-Related Potentials and Event-Related Desynchronization Induced by Relative Beta Training in ADHD Children

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Changes in EEG Spectrograms, Event-Related Potentials and Event-Related Desynchronization Induced by Relative Beta Training in ADHD Children

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ABSTRACT. *Background*. During the last three decades EEG-based biofeedback (neurofeedback) was used as an alternative treatment for reducing symptoms of ADHD. The goal of this study was to objectively assess the efficacy of biofeedback training by comparing spectrograms, ERPs and ERDs, measured before and after 20 sessions of neurotherapy in a group of ADHD children.

Method. Electroencephalogram (EEG), Event related potentials (ERPs) and event related synchronisation/desynchronisation (ERD/ERS) were recorded and computed in auditory GO/NOGO task before and after 15-22 sessions of EEG biofeedback. Eighty-six ADHD children participated in the study. Each session consisted of 30 min of relative beta training. The patients were divided into two groups (good performers and poor performers) depending on their ability to elevate beta activity during sessions.

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Results. Amplitude of late positive components of evoked potentials in response to NOGO stimuli increased, and event-related synchronisation in alpha frequency band measured at central areas decreased after the whole set of sessions of neurofeedback training in the group of good performers but did not change for the poor performers group. Evoked potential differences between post- and pre-treatment conditions for good performers were distributed over frontal-central areas, reflecting activation of frontal cortical areas associated with beta training.

Conclusion. Relative beta training with electrodes located above the frontal areas was associated with an increase of the late positive NOGO component. This activation likely indicates recovery of normal functioning of the executive system. doi:10.1300/J184v11n02_02

KEYWORDS. Attention Deficit Hyperactivity Disorder, executive functions, event-related potentials, event-related synchronisation, GO/NOGO paradigm, EEG biofeedback (neurofeedback), beta training

INTRODUCTION

Attention Deficit/Hyperactivity Disorder (ADHD) is a childhood psychiatric disorder which, when carefully defined, affects around 4% of the school-age population. During the last three decades EEG based biofeedback (neurofeedback) has been used as an alternative treatment for reducing symptoms of ADHD. The protocols of neurofeedback were based on an empirical observation of slowing EEG rhythms in ADHD children. This slowing is represented by increase of EEG power in theta band and corresponding decrease of EEG power in beta band (Mann et al., 1992; Jansen et al., 1995; Shabot and Serfontein, 1996; Clarke et al., 2001, Monastra et al., 1999). A conventional neurofeedback protocol for reducing inattention and impulsivity consists of enhancement of beta activity and suppressing theta activity (Lubar et al., 1995; Linden et al., 1996).

Parameters of executive functions (the number of omission errors as an index of inattention and the number of commission errors as an index of impulsivity) as measured by TOVA (the Test of Variables of Attention) were shown to change towards normative scores after intensive EEG training (Lubar et al., 1995; Othmer et al., 2000, Monastra et al., 2002, Fuchs et al., 2003). Executive functions of the brain are traditionally assessed by measuring physiological parameters (by PET, fMRI, ERPs components) in various modifications of GO/NOGO paradigm. In various tasks of the GO/NOGO paradigm, ADHD children were shown to exhibit lower amplitude P300 components in comparison to normal groups (Kropotov et al., 1999; Overtoom et al., 1998; van Leeuwen et al., 1998).

The goal of this study was to objectively assess the efficacy of biofeedback training by comparing spectrograms, ERPs and ERDs, measured before and after 20 sessions of neurotherapy in a group of ADHD children.

METHODS

Subjects

Eighty-six children with ADHD symptoms (77 boys and 9 girls, age 9 to 14 years, mean 11.4) voluntarily participated in this study. All subjects were Russian-speaking schoolchildren who attended normal secondary schools in St.-Petersburg, Russia. Children with ADHD were referred to the Neurotherapy Center at the Clinics of the Institute of the Human Brain of Russian Academy of Sciences in St.-Petersburg, Russia. Patients were evaluated by a psychiatrist L. S. Chutko, PhD, MD) and received a primary DSM-IV (American Psychiatric Association, 1994) diagnosis of Attention Deficit Hyperactivity Disorder.

Pre-Treatment Assessment

An adapted version of SNAP-4 parents' questionnaire (Swanson, 1992) was used for

subjective estimation of the level of attention deficit, hyperactivity, and impulsiveness. These subjective assessments of behavior were calculated from parental responses compared to the normative values.

All patients performed the auditory two-stimulus GO/NOGO task, a continuous performance task consisting of 480 trials. Two tones–high frequency tone of 1300 Hz (referred to as H) and low frequency tone of 1000 Hz (referred to as L) were used as stimuli. Pairs of stimuli, LL and LH, were presented at random with a 50% probability. The stimuli duration was 100 ms and the sound intensity was 75 dB. Intervals between stimuli within pairs and between pairs were 800 and 1500 ms, respectively. The task of a subject was to press a button with the right hand in response to the LL pair (target). The duration of the task was 20 min with 2-3 short intervals of 1-2 min for rest.

EEG, ERPs and ERD/ERS Recordings

EEG, ERPs and ERD/ERSs in all patients were registered during performance of the auditory GO/NOGO task described above. The electroencephalogram (EEG) was recorded by the Telepat-104 24-channel EEG system (Potential, Ltd., Russia) and by the Mitsar 21-channel EEG system (Mitsar, Ltd., Russia). Nineteen silver-chloride electrodes were placed on the skull according the standard 10-20 system. Input signals were referenced to the tip of the nose, amplified, with a band pass of 0.5-30 Hz, and sampled at the rate of 250 Hz. The ground electrode was placed on the forehead. Impedance was kept below 10 KOhms.

We used a weighted average montage according to Lemos (Lemos and Fisch, 1991). Some results of this study with linked-ears montage are presented in Kropotov et al. (2005). Eye movement artefacts were corrected by means of Independent Component Analysis (Ille et al., 2002). ERPs were computed off line. The epoch of analysis included 300 ms before the first stimulus and 900 ms after the second stimulus. Event related desynchronization (ERDs) were computed according to a standard procedure described by Kalcher and Purtscheller, 1995, in several steps:

1. The signal for a given frequency range is detected for each EEG fragment (trial) by means of digital bandpass filters.
2. To reduce the influence of ERP components on ERD/ERS, averaged ERPs are computed over the trial to be then subtracted from each trial.
3. To assess EEG signal power dynamics for a given frequency range, for each time readout (bin) the values are squared and averaged over all trials.
4. To reduce data dispersion, the EEG power dynamics is smoothed by moving average with averaging epoch width (optimally 100 msec or 25 bins).
5. ERD/ERS is calculated as percent of signal power change for each bin in relation to average power during the prestimulus interval (R):

$$((P(i) - R)/R) \times 100\%$$

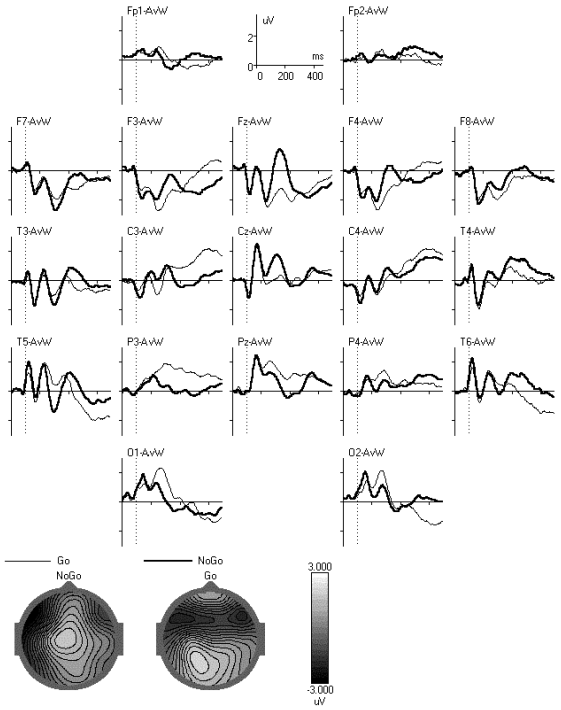
The signals obtained are then averaged similarly to ERPs.

Figure 1 presents the averaged ERPs recorded in auditory GO/NOGO task in response to second stimuli (GO and NOGO) before the neurofeedback sessions in the good performer's group. At least 4 different components of ERPs could be visually separated: N100, P200, N200 and P300. The P300 to GO stimuli will be referred to as GO component, while the P300 to NOGO stimuli will be referred to as NOGO component. As one can see from the figures, the NOGO component had more anterior distribution compared to the GO component. All patients performed GO/NOGO task twice: a pre-training testing usually took place 1-7 days before biofeedback training and a post-training test 1-7 days after the last training session.

Statistical Analysis

Three frequency bands were chosen for analyzing spectra power changes associated with neurofeedback sessions: theta (4-7.5 Hz), alpha (8-13 Hz) and beta 1 (13-21 Hz). Two-way ANOVAs for repeated measurement with factors *Treatment* (before and after 20 sessions of training) and *Location* (19 electrodes) were cal-

FIGURE 1. Grand average ERPs in the two stimulus auditory GO/NOGO test in 37 good-performers after 20 sessions of relative beta training. Horizontal axis: time in ms; vertical axis: averaged scalp potentials recorded in different electrode locations (Fp1, Fp2, ..., O1, O2). Thin line: Go components, thick line: NOGO components. At the bottom: maps of grand average ERP for Go and NoGo stimuli taken at 310 ms after stimulus. Mapping scale is presented in the middle.



culated to evaluate neurofeedback-induced changes.

Three time segments corresponding to early ERPs components were selected for analysis of ERP changes during neurofeedback: N100 (80-130 ms) and P200 (130-180 ms), and late ERP complexes (180-420 ms after the second stimulus) for both conditions (GO and NOGO). A time segment 200-1200 ms after the first stimulus was selected for analysis of alpha ERD/ERS.

Two-way ANOVAs for repeated measurement with factors *Treatment* (before and after training) and *Location* (19 electrodes) were calculated to evaluate differences between alpha ERD/ERS and ERP components for GO and NOGO conditions separately. The Greenhouse-Geisser procedure was used to compensate for violations of sphericity.

Procedure of Neurofeedback

EEG training was performed on the Telepat-104 or Mitsar EEG system. We used a bipolar montage with Fz-C3 or C4-Pz in the standard 10-20 system. Left-side (C3) and right-side (C4) training involved rewarding activity in the 15-18 Hz and 12-15 Hz, respectively. These two protocols were used in succession during a single training session: 20 min of relative 15-18 Hz training, 7-10 min of relative 12-15 Hz training. This protocol is based on the results of investigation of ERS in GO/NOGO test. The studies have shown increase of beta activity within 13-18 Hz at frontal areas in a group of normal children. This increase was significantly lower in children who were diagnosed with ADHD/ADD. On the basis of this finding we decided to reward increased beta activity in frontal areas.

The biofeedback procedure included the following computations: Power spectrum was calculated for a 1 s epoch every 250 ms using fast Fourier transformation. The ratio of the trained beta rhythm power by the power of low (1-11 Hz) and high (19-30 Hz) frequencies served as biofeedback parameter.

Visual feedback was provided by a blue bar against a grey background on a computer screen. The height of the bar followed the dynamics of the biofeedback parameter. The patient's task was to keep the bar above a threshold.

Video mode was used as another kind of visual presentation of the biofeedback signal. In this mode, the biofeedback parameter controlled the level of a noise generated by a separate electronic unit called Jammer, a unit designed specifically for this purpose in the laboratory. The amplitude of the noise was maximal if the biofeedback parameter was minimal and decreased gradually to zero when the parameter approached a threshold. This noise was mixed with the video signal and was fed to a TV. Thus the patient was able to control the quality of the picture on the screen with his or her brainwaves: when the biofeedback parameter was higher than threshold, the picture on the screen was clear, otherwise it was blurred. Usually during the first 5-8 sessions patients performed training with the bar; then training in the video mode started.

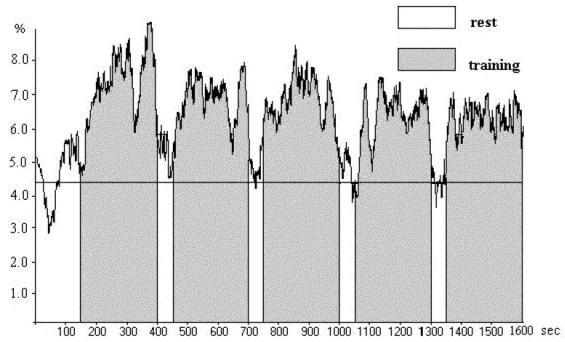
The threshold for the biofeedback parameter was defined by the prefeedback baseline mean measure taken during a 2.5 min of feedback-free period with eyes opened at the beginning of the first session to ensure that the biofeedback parameter exceeded threshold about 50% of the time.

The patient was instructed about the rationale of the procedure, as well as about the dependence of the biofeedback signal on brain activity and attention. The number of training sessions for each patient varied depending upon several factors such as age, type of ADHD, learning curves, parent reports, and varied from 15 to 22 sessions (mean 17). Termination criteria was (1) stabilization of training performance as assessed by the dynamics of the trained parameter (see below Methods 2.6) during the last 3-5 sessions, (2) stabilization of patient's behavior according to parental reports. Sessions were administrated two to five times per week for five to eight weeks.

Assessment of Performance During Training

The dynamics of the biofeedback parameter were analyzed for each patient and for each session. Figure 2 shows a typical training curve for a single patient taken at his 15th session. One can see that the patient was able to elevate the parameter during periods of training while the parameter dropped to pre-training levels during rest periods.

FIGURE 2. Dynamics of a relative beta power during a single training session in an ADHD boy. Horizontal axis: time in ms; vertical axis: beta relative in percent. Grey areas indicate training periods, white areas–resting periods.



Furthermore, the quality of patient's performance, i.e., the ability of a patient to increase the biofeedback parameter during training periods was assessed. We considered the training session to be successful if a patient was able to increase the biofeedback parameter during training periods more than 25% in comparison to resting periods. Patients were referred to as good performers if they were successful in more than 60 % of the sessions. Seventy-one patients (82.5%) belonged to the good performance group. Fifteen patients (17.5%) belonged to the poor performance group. This group acted as one form of control group in data analysis.

RESULTS

SNAP-4. According to SNAP-4 parents' questionnaire, the average degree of inattention in good performers decreased from 2.3 to 1.75 ($p < 0.01$), whereas mean impulsiveness/hyperactivity decreased from 1.45 to 1.20 ($p < 0.05$) after training (Table 1). No statistically reliable changes were found for SNAP-4 scores in the poor performers group (Table 2).

ERPs. To obtain reliable ERPs for each condition we needed a sufficient number of trials for averaging. Recall that the background EEG

TABLE 1. Mean standard scores for SNAP-4 measures before and after biofeedback course for 71 good performers.

Dependent measure	Before treatment	After treatment
Inattention	2.30 ±0.3	1.75 ±0.4**
Hyperactivity	1.45 ±0.3	1.10 ±0.4*

*p<0.05, ** p<0.01 compared to the "before" treatment condition.

TABLE 2. Mean standard scores for SNAP-4 measures before and after biofeedback course for 15 poor performers.

Dependent measure	Before treatment	After treatment
Inattention	2.10 ±0.3	2.15 ±0.3
Hyperactivity	1.30 ±0.3	1.25 ±0.3

was about 50-70 μV while GO and NOGO components are about 10 μV, so to get a good signal to noise ratio more than 70 trials were required. The total number of trials for each category was 240. Children with ADHD made errors in about 20% of trials and had quite a lot of muscle and movement artefacts during EEG recording. For these reasons the number of patients with reliable ERPs was 50 (37 good performers and 13 poor performers).

For the GO condition in the good performers, two-way ANOVA (before/after treatment) for 19 electrodes revealed no significant difference in the amplitude of late ERPs complex in 180-420 ms interval after the second stimulus ($F(1,72) = 2,42$, $p < 0.13$). However a significant interaction of two factors (before/after treatment) and electrode localization were observed ($F(18,13) = 43,42$, $p < 0.0001$). For NOGO condition two-way ANOVA (before/after treatment) for 19 electrodes revealed a significant difference in the amplitude of late additional complex in the 180-420 ms interval after the second stimulus ($F(1,72) = 7,65$, $p < 0.008$) parallel with significant interaction of factors (before/after treatment) and electrode localization ($F(18,13) = 26.62$, $p < 0.00001$).

Figure 3 compares ERP differences induced by 20 sessions of neurofeedback in two groups: 37 good performers and 13 poor performers. In contrast to good performers two-way ANOVA (before/after treatment) in poor performers did not reveal any significant difference in amplitude of late additional complex for NOGO condition (for 19 electrodes $F(1,24) = 0.69$, $p < 0.4$).

ERD/ERSs. In the good performers, two-way ANOVA (before/after treatment) for 19 electrodes revealed a significant difference in the amplitude of ERS in alpha frequency band (9-13 Hz) in 200-1200 ms interval after the first stimulus ($F(1,36) = 11.62$, $p < 0.017$).

Figure 4 compares alpha ERS differences after 20 sessions of neurofeedback in two groups, 37 good performers and 13 poor performers. In contrast to good performers two-way ANOVA (before/after treatment) for 19 electrodes in poor performers did not reveal any significant difference in ERS in the alpha band ($F(1,12) = 6.07$, $p < 0.30$).

EEG spectra power. A two-way ANOVA (before/after treatment) for 19 electrodes re-

FIGURE 3. Comparison of ERP differences (before-after) induced by 20 sessions of neurofeedback in the groups of good (A) and poor performers. At the left: grand average ERP differences for Go (thin line) and NoGo (thick line) stimuli taken for Fz location. At the right: maps of grand average ERP differences for Go and NoGo stimuli taken at 310 ms after stimulus. Mapping scale is presented in the middle.

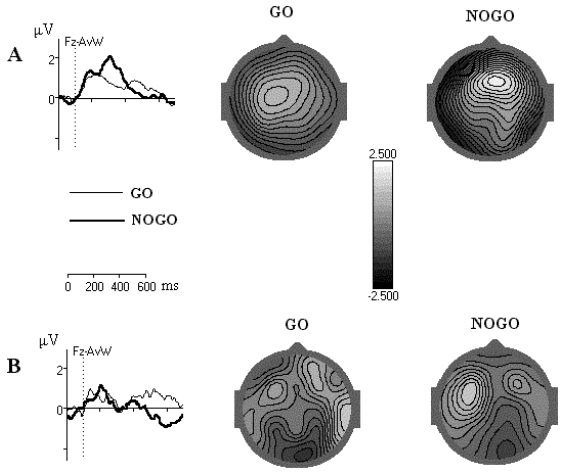
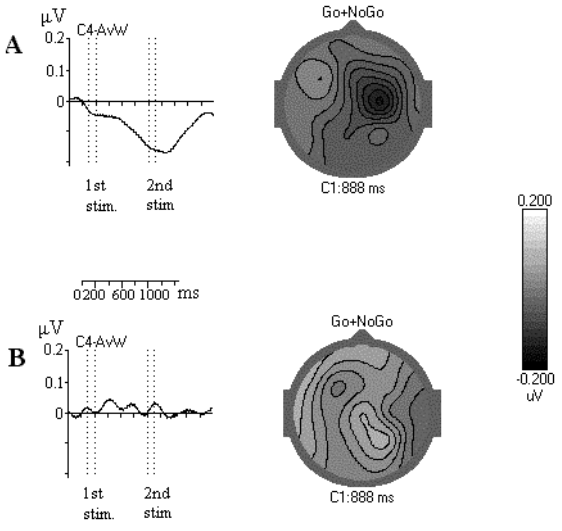


FIGURE 4. Comparison of event related synchronisation (ERS) differences (after-before) induced by 20 sessions of neurofeedback in the groups of good (A) and poor performers. At the left: grand average alpha ERS differences for 1st stimulus taken for C4 location. At the right: maps of grand average alpha ERS differences taken after second stimulus. Mapping style is presented on the right.



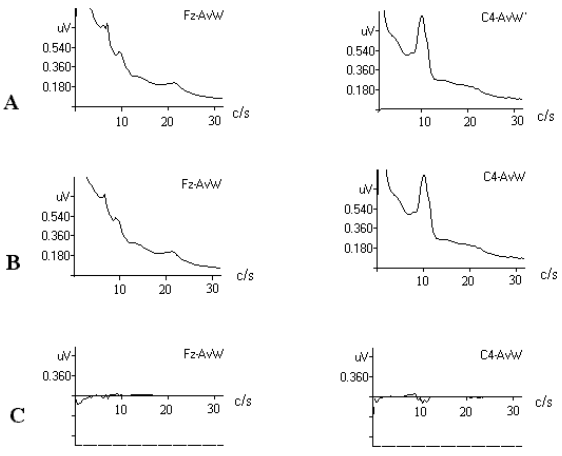
vealed no significant difference in EEG spectra power for either performance group. F-statistics were as follows: in good performers' group for theta band ($F(1,72) = 0.53$, $p < 0.9$)), for alpha band ($F(1,72) = 0.75$, $p < 0.74$)), and for beta 1 band ($F(1,72) = 2.30$, $p < 0.22$)); in poor performers group for theta band ($F(1,24) = 0.09$, $p < 0.77$)), for alpha band ($F(1,24) = 0.15$, $p < 0.70$)), and for beta 1 band ($F(1,24) = 0.41$, $p < 0.53$). Figure 5 shows the dynamic of EEG spectra powers after 20 sessions of neurofeedback in the groups of good performers.

DISCUSSIONS

Selection of neurofeedback protocol. In our study we selected a protocol that implemented a relative beta power as a biofeedback parameter. This parameter was defined as a ratio of EEG power in beta frequency range to the EEG power in the rest of the frequency range. Most of conventional protocols use simultaneous elevation of beta activity and suppression of theta activity (Lubar et al., 1995; Linden et al., 1996; Othmer et al., 2000).

Theoretically our protocol differs from conventional protocols because elevation of the biofeedback parameter in our study could be achieved by increasing beta power, and/or by decreasing theta as well alpha power. However, as the results of the present study indicate, the application of our protocol turns out to be as effective as conventional protocols. Indeed, 82.5% of our patients were able to significantly increase (for more than 30%) their biofeedback parameter in more than a half of sessions.

FIGURE 5. EEG spectra powers before (A) and after (B) 20 sessions of neurofeedback and their differences (C) in the group of good performers.



Effect on late positive NOGO component. The main goal of this study was to observe changes in ERPs induced by neurofeedback. We found that relative beta training does not change early N1 and P2 components of ERPs (with latencies of 80-180 ms). This finding can be considered as evidence against any relative beta training effect on early stages of auditory information processing.

Late ERP components that follow N1 and P2 components have been traditionally associated with executive functions–engagement and disengagement operations in control of behavior (Simson et al., 1977; Roberts et al., 1994; Kropotov et al., 1997, 1999). Our study shows that neurofeedback sessions of relative beta training led to a statistically significant enhancement of NOGO component.

Executive functions, such as attention and motor control, are known to be maintained by neuronal circuits including the frontal lobes and the basal ganglia thalamo-cortical pathways (Castellanos, 1997; Kropotov et al., 1997, 1999). These circuits also participate in self-regulation of the frontal cortex (Alexander et al., 1986; Brunia, 1992). On the other hand, ADHD children are reported to exhibit abnormalities in both the frontal cortex and the basal ganglia, including lower metabolic activity, smaller sizes and higher concentration of DAT (dopamine transporter) receptors in the basal ganglia (Lou et al., 1984; Zametkin et al., 1990, 1993; Castellanos, 1997; Dresel et al., 2000).

The decrease of metabolic activity in the frontal cortex of ADHD children seems to be associated with thalamo-cortical dysrhythmia: increase of theta activity and decrease of beta activity in ADHD children (Mann et al., 1992; Janzen et al., 1995; Shabot and Serfontein, 1996; Monastra et al., 1999; Clarke et al., 2001). This association is directly supported by the recent studies that found a strong positive correlation between perfusion measured by PET and EEG power in beta band, so that decreased level of beta activity in frontal region would correspond to a lower level of metabolic activity of this area (Cook, 1998).

According to this assumption, relative beta training with electrodes located above the frontal areas is associated with activation of the underlying frontal cortex. In our study the difference ERPs waves ("ERPs after" minus "ERPs before") associated with neurofeedback training are distributed over the frontal lobes. Consequently, the statistically significant increase of the late ERP component in response to NOGO stimuli after beta training might be a correlate of training-related activation of the frontal cortex. This activation seems to indicate the recovery of normal functioning of the executive system.

Effect on ERS. EEG oscillations in alpha frequency band have been long considered as idling rhythms (Niedermeyer, 1997; Pfurtsheller et al., 1996). Alpha oscillations over the sensorimotor strip (measured at the scalp in C3, C4) are named mu or sensorimotor rhythms. They are negatively correlated with the level of metabolic activity in the corresponding sensorimotor areas, so that decrease of EEG power in the alpha frequency band could be regarded as indication of activation of the cortical area.

In the present study we found that 20 sessions of relative beta training led to increased alpha desynchronization in the ADHD population. This desynchronization occurs with the first stimulus and appears to be related to the motor preparatory set associated with activation of the left (contra-lateral) sensorimotor area. After 20 sessions of neurofeedback this type of activation increases in the group of good performers, but not in the group of poor performers.

Effects on spectrograms. It should be stressed here that all neurofeedback-related changes were found only in EEG reactions (ERPa and ERDs), but not in the background EEG spectrograms. There may be two reasons for that: First, spectrograms are more variable than ERPs/ERDs, i.e., inter-individual variations in spectrograms and, consequently, standard deviations are bigger than those for ERPs/ERDs. So, the power of the statistical criteria for assessment changes in spectrograms is inferior to the comparison of ERPs/ERSs and, consequently, does not reveal any statistically significant change. Second, neurofeedback may indeed change only reactivity of the brain to certain stimuli in certain conditions. To select between two options more data must be collected.

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