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Slow cortical potential neurofeedback in attention deficit hyperactivity disorder: is there neurophysiological evidence for specific effects?

Mirko Doehnert · Daniel Brandeis · Marc Straub · Hans-Christoph Steinhausen · Renate Drechsler

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Abstract This study compared changes in quantitative EEG (QEEG) and CNV (contingent negative variation) of children suffering from ADHD treated by SCP (slow cortical potential) neurofeedback (NF) with the effects of group therapy (GT) to separate specific from non-specific neurophysiological effects of NF. Twenty-six children (age: 11.1 ± 1.15 years) diagnosed as having ADHD were assigned to NF ($N = 14$) or GT ($N = 12$) training groups. QEEG measures at rest, CNV and behavioral ratings were acquired before and after the trainings and statistically analyzed. For children with ADHD-combined type in the NF group, treatment effects indicated a tendency toward improvement of selected QEEG markers. We could not find the expected improvement of CNV, but CNV reduction was less pronounced in good NF performers. QEEG changes were associated with some behavioral scales. Analyses of subgroups suggested specific influences of SCP training on brain functions. To conclude, SCP neurofeedback improves only selected attentional brain functions as measurable with QEEG at rest or CNV mapping. Effects of neurofeedback including the advantage of NF over GT seem mediated by both specific and non-specific factors.

Keywords SCP neurofeedback · ADHD · QEEG · CNV

M. Doehnert and D. Brandeis have contributed equally to this paper.

M. Doehnert · D. Brandeis · M. Straub · H.-C. Steinhausen · R. Drechsler (✉)
Department of Child and Adolescent Psychiatry, University of Zurich, Neumuensterallee 9, 8032 Zurich, Switzerland
e-mail: renate.drechsler@kjpd.uzh.ch

M. Doehnert
Department of Child and Adolescent Psychiatry, University of Leipzig, Leipzig, Germany

D. Brandeis
Center for Integrative Human Physiology, University of Zurich, Zurich, Switzerland

Introduction

Attention-deficit hyperactivity disorder (ADHD) is the most common psychiatric disorder of childhood with a prevalence of approximately 3–7% in school aged children. The main symptoms of the disorder are inattention and hyperactivity/impulsivity. Children with ADHD are significantly impaired in their functioning, and often also suffer from a number of associated problems or comorbidities, like poor academic performance, learning disorders, conduct disorders, etc. (Barkley 2006).

There is increasing interest in neurofeedback treatment of ADHD, which aims to improve behavioral self-control through electroencephalogram (EEG) biofeedback. This is partly because a sizable (25%) proportion of ADHD patients do not respond to pharmacological treatment, and additionally because many patients are seeking long-term efficacy and alternatives to pharmacological treatment (Holtmann and Stadler 2006; Heinrich et al. 2007).

The rationale for improving the core symptoms of ADHD through neurofeedback is based on the close relation between specific neurophysiological EEG rhythms or ERP (event-related potential) components, and the underlying (presumably thalamocortical) regulation of alertness, attention and behavioral control. Since ADHD neuropathology is known to alter both EEG rhythms and ERP components, neurofeedback training, directed at learning to

normalize them, may yield sustaining clinical benefits (Monastra et al. 2005).

Several EEG rhythms which reflect maturation and arousal or attention during wakefulness show subtle abnormalities in ADHD. The most common neurophysiological abnormalities in the spontaneous EEG of ADHD subjects are increased slow wave activity (mainly theta) and/or reduced alpha and beta activity in the resting EEG (a relaxed awake state, usually with eyes closed). Slowing due to increased theta was found in all ADHD- and age-subgroups, although especially prominent in ADHD-combined type, while decreased alpha characterized only ADHD boys (Clarke et al. 2001). Slowing is not only the most reliable EEG marker of ADHD but also characterizes immaturity and lower arousal, especially over central and frontal scalp regions (Barry et al. 2003a). Quantitative EEG markers of such slowing (especially theta/beta-ratio at Cz) are even considered to be an useful addition in the assessment of ADHD. Although recent estimates of their diagnostic value (Magee et al. 2005: sensitivity 89.0% and specificity 79.6%) are not quite as optimistic as in earlier work (Monastra et al. 1999: sensitivity 86%, specificity 98%), EEG slowing may still compare favorably to behavioral rating scales as a more specific marker of ADHD in clinical samples (Quintana et al. 2007).

Since most patients diagnosed with ADHD demonstrate EEG slowing, classical neurofeedback treatments specifically target EEG slowing by decreasing theta and increasing beta activity (theta/beta training). Most studies reported positive effects (Lubar et al. 1995; Thompson and Thompson 1998, etc.) comparable to stimulant treatment (Rossiter and La Vaque 1995; Monastra et al. 2002; Fuchs et al. 2003; Rossiter 2004).

Neurophysiological abnormalities also characterize the event-related potential (ERP) of patients with ADHD. Most common are attenuations of late ERPs such as the different P300 components which reflect attention, inhibition and cognitive control and of slow cortical potentials (SCPs) like the contingent negative variation (CNV) during preparation and activation (Barry et al. 2003b). Source localization suggests that both anterior and posterior attention networks are involved (Brandeis et al. 1998; van Leeuwen et al. 1998). Reductions of the CNV during cognitive preparation following a warning stimulus are common in ADHD patients (van Leeuwen et al. 1998; Hennighausen et al. 2000; Perchet et al. 2001; Banaschewski et al. 2003, 2004), and most prominent for pure ADHD cases without comorbid behavioral problems due to ODD/CD (Banaschewski et al. 2003). The findings of a decreased CNV are in line with dysfunctional regulation of energetical resources in ADHD (Sergeant 2000), and with negative SCP shifts representing higher neural excitability (Birbaumer et al. 1990).

SCPs such as the CNV which are both related to attentional preparation and reduced in ADHD, are thus another obvious target for neurofeedback control in these patients. Accordingly, training to regulate SCPs on a trial to trial basis following a cue forms the core of SCP neurofeedback treatment for ADHD patients.

Heinrich et al. (2004) published a first controlled study on SCP neurofeedback in ADHD. They compared 13 ADHD-children (age 7–13 years) assigned to 25 sessions of about 50 min SCP training with nine ADHD-children in a waiting control group. Only SCP training reduced ADHD symptoms and increased the CNV during an attention test (cued continuous performance test, CPT). Subsequently, Leins et al. (2006) compared SCP with frequency (theta/beta) neurofeedback training. Both groups of 19 ADHD-children (age 8–13 years) learned to regulate their brain activity in the 30 training sessions, and improved similarly according to attentional and cognitive tests, as well as parent and teacher reports of behavioral symptoms. Clinical effects for both groups remained stable 6 months after training.

Controlled studies of both frequency and SCP neurofeedback training thus demonstrate clinically relevant improvements. These appear similar and comparable to stimulant treatment in size, and are absent in waiting control groups. Some specificity is also indicated by findings suggesting normalization of neurophysiological EEG and ERP markers of ADHD (Monastra et al. 2002; Heinrich et al. 2004; Kropotov et al. 2005; Strehl et al. 2006), and by improved outcomes for those patients who achieved better control over their brain activity (Kropotov et al. 2005; Strehl et al. 2006). However, whether SCP and EEG frequency neurofeedback target the same attention network, and whether successful SCP training also improves EEG frequency markers of attention and vice versa is unclear. Also, specificity remains a critical issue. No study of neurofeedback treatment for ADHD has yet demonstrated superiority over control training matched for time and attention invested, or has controlled for other non-specific modulators such as non-specific motivational effects, the structured treatment setting and the degree of therapist–patient interaction. Similarly the normalization of distinct neurophysiological markers of ADHD such as increased EEG frequency and CNV amplitude may be correlated and mediated by non-specific common factors.

Here we thus compare the neurophysiological effects of SCP neurofeedback and group therapy. Behavioral and neuropsychological comparisons from this study have been reported elsewhere (Drechsler et al. 2007).

Our hypotheses were as follows. First, given our prior report that participants treated with neurofeedback improved and compared favorably to the control group (Drechsler et al. 2007) on behavioral and neuropsychological measures,

we predicted similar advantages for neurofeedback during the separate EEG session for the behavioral measures of the CPT. Second, neurofeedback subjects should show more improvements than the controls regarding attentional ERP makers, and particularly for the CNV to cues, as reported previously by Heinrich et al. (2004), since enhancing the CNV-like SCP was part of the neurofeedback training. Third, improvement might affect not only SCP activation but also reduces slowing in the resting quantitative EEG (QEEG) due to overlapping attentional networks for activation and state regulation. These EEG and ERP changes should not only represent specific findings of the neurofeedback group, but also be stronger for subjects who achieved better control of their brain activity, and therefore furnish additional evidence for specific effects of SCP neurofeedback training in children with ADHD.

Materials and methods

Subjects

Twenty-six ADHD-children, aged 9–12 years, participated in this study on the basis of informed consent by the child and parent and in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki. Fourteen children (nine with ADHD-combined subtype) participated in the neurofeedback group (NF), while 12 children (eight with ADHD-combined subtype) formed the control group who completed a group therapy (GT). Group assignment was partly based on parental preferences and certain therapeutic and practical aspects had to be respected. Group characteristics are listed in Table 1. These children were selected from the 30 participants of a larger training study (Drechsler et al. 2007) as those who had completed EEG mapping assessments before and after the training.

Clinical diagnosis and subtyping was confirmed by HYPESCHEME, a computerized operational criteria checklist and diagnostic algorithm for DSM-IV and ICD-10 which includes a diagnostic interview (PACS, parental account of children's symptoms; Taylor et al. 1986; Curran et al. 2000), Conners Parent Rating Scale (CPRS; Conners et al. 1998a) and Conners Teachers Rating Scale (CTRS; Conners et al. 1998b). All patients fulfilled diagnostic criteria according to DSM-IV and ICD-10. Children could continue stimulant medication during the training course, but interrupted medication at least 48 h before pre- and post-neurofeedback assessment.

SCP training

We used the visual neurofeedback system "GoeFI" (Goettinger Feedback) for SCP training (Heinrich et al. 2004; Drechsler et al. 2007). In brief, feedback was provided through several animations made especially for children. During training, children had to change the color of an object on the screen to red in negativity trials, and to blue in positivity trials by modulating their brain electrical activity. They were told that the color red may be achieved by increasing attention, whereas the color blue may be associated with decreased activation.

The training was divided into two phases and provided the equivalent of 30 typical training units in 15 sessions. In the first phase the children performed 20 units (10 double sessions of two units a day) over 2 weeks during school holidays. The second phase, after a 5 week break, consisted of five double sessions (10 units). During the 5-week break, the children and parents were instructed to do daily transfer training, during which no feedback was provided, and the children were instructed to practice their strategies for generating negativities and positivities using cards which displayed typical pictures for activation versus deactivation.

Each double training session consisted of two units of 45 min, which took place in the same day. About 180 trials per session were performed in the following order: 40

Table 1 Group characteristics

	Neurofeedback ($n = 14$)	Group therapy ($n = 12$)	P
Age mean (SD)	10.8 (1.3)	11.4 (0.9)	NS
Male/female	12/2	10/2	NS
IQ (HAWIK-III)	101.2	110.3	NS
CPRS (global index)	73.8	65.2	$t = 2.24$, $P = 0.035$
CTRS (global index)	63.5	63.9	NS
ADHD subtype			NS
Combined	9	8	
Inattentive	4	4	
Hyperactive	1	0	
Stimulant medication yes/no	6/8	6/6	NS

feedback, 30 transfer, 40 transfer with cards, break, 40 feedback, 30 transfer.

Negativity and positivity trials (50% each) were presented randomly. A trial lasted for 8 s (baseline 2 s, feedback 6 s). Feedback was calculated from Cz (reference: mastoids). Eye movements were corrected online using electrooculogram (EOG) electrodes above and below the left eye.

Group therapy

The group therapy was conducted by two experienced clinical psychologists with groups of five to six ADHD-children. The training was based on principles of cognitive-behavioral therapy. Twelve 90-min-sessions were provided. Training consisted of the following: emotional control, feedback, planning and organizing, time management, using coping strategies, etc. Additionally, role plays, homework and a parents meeting were used.

Pre-/post-measurement

Electroencephalogram was recorded from 46 EEG and 2 EOG electrodes mounted in a cap, using FCz as recording reference and AFz as ground. Impedances were below 20 kOhm. The sampling rate of the EEG was 256 Hz, low frequency filter 0.1 Hz, high frequency filter 70 Hz.

Resting EEG was recorded in a 3 min eyes-closed resting condition, and referred to the average reference. At least 24 epochs of 2.5 s were selected by semiautomatic artefact rejection of epochs with amplitudes exceeding ± 150 μV followed by visual appraisal of every epoch for the absence or presence of artefact. The selected epochs were Fourier transformed and averaged. We focused on theta (3.5–7.5 Hz), alpha (7.5–12.5 Hz) and beta (12.5–25 Hz) frequency bands. Since Chabot et al. (2001) found most significant abnormalities around 5 Hz (theta band) and at 11 Hz (alpha band) in ADHD-children, we also evaluated similar sub-bands (4–6 Hz, 10–12 Hz). Topographically, we focused on Cz for theta and the theta/beta-ratio (Barry et al. 2003a), and for alpha on occipital (Oz, O1, O2, OI1, OI2) sites due to maximal alpha activity, and on other posterior sites (Pz, P3, P4, POz, PO1, PO2, CP1, CP2, CP5, CP6, Cz, C1, C2, T5, T6) with reduced alpha. Mean power of these frequency bands and differences between pre- and post-measurement were calculated.

Next, ERPs were recorded in cued continuous performance tests (CPT; van Leeuwen et al. 1998). The CPT consisted of 400 stimuli (letters) that were presented at the center of a monitor at the viewing distance of 120 cm for 150 ms each, with an interstimulus interval of 1,650 ms. Children were instructed to respond to a letter X occurring after the cue letter O (probabilities for the sequence O–X and O-not-X were 10% each). Total task duration was 11 min. The task was practiced and comprehension ascertained just prior to task performance. If necessary, subjects were told to minimize eye movements or blinks. To increase attentional load, an additional flanker variant of the same CPT was performed with an irrelevant, incompatible letter flanking the critical central (OHO…XGX…XOX…OXO).

The EEG was digitally lowpass-filtered at 30 Hz. Following ICA-based ocular artifact reduction, the average reference was computed and artefacts were rejected before averaging. All averages contained a minimum of 20 accepted sweeps.

The mean amplitude of the CNV in the 1,000–1,600 ms interval at Cz in cue trials were computed. Subjects with a hit rate of less than 50% ($n = 3$) were excluded because it was assumed these subjects had not really understood the task despite practice.

CPT performance was measured by scoring number of hits (targets), omission, commission and impulsivity errors.

Behaviorally, children were assessed using German versions of CPRS, CTRS, ADHD rating scale (FBB-HKS; Doepfner and Lehmkuhl 2000), BRIEF (Gioia et al. 2000) and neuropsychological tasks at pre- and post-measurement. FBB-HKS is part of the Diagnostic System for Mental Disorders in Childhood and Adolescence (DISYPS-KJ) and based on the symptom-criteria of ICD-10 and DSM-IV. Its three subscales (inattention, hyperactivity and impulsivity) are all assessed for severity (severity score) and experienced difficulties (problem score), thus yielding six scores to analyze.

Statistics

First, the behavioral scores and the quantitative EEG before training were examined for correlations and for differences between neurofeedback and group therapy subjects. The main analyses focused on changes with training, focusing on the frequency bands and ratios described above. Changes of EEG power with training were displayed topographically as *t*-maps, and analyzed using repeated-measures analyses of variance (ANOVA: training group by time, plus an additional electrode factor when multiple electrodes were involved). Significant group by time interactions were followed up by *t* tests which were reported if significant at the $P < 0.01$ level. The analyses of ERP changes focused on the CNV in the CPT and its relation to CPT performance. Again, changes were analyzed using ANOVA and significant group by time interactions were followed up by *t* tests.

To clarify associations between changes of neurophysiological and behavioral data for neurofeedback subjects, repeated-measures analyses of variance and Pearson's

correlations were performed. Furthermore we analyzed associations between significant changes in QEEG, CNV and CPT measures for neurofeedback subjects.

While we had a few strong a-priori hypotheses, the many possibilities for additional tests at other frequency bands and electrodes and subgroups raises the statistical problem of correction for multiple tests. It is clear that our small group study would be severely underpowered if all plausible hypotheses and topographies were tested with strict Bonferroni correction.

We therefore tested only the few a -priori hypotheses from different tests (EEG, CPT) without correcting for multiple testing beyond the ANOVA, as is common. These hypotheses were

1. CNV amplitude increases at Cz (ANOVA interaction for specific effects, and *t* tests for neurofeedback effects)
2. Theta und theta/beta-ratio at Cz decrease, especially in ADHS-combined type (ANOVA interaction for specific effects, and *t* tests for neurofeedback effects)
3. CPT performance, especially hit rate, errors, reaction time, and its standard deviation, improves significantly (ANOVA interaction for specific effects, and *t* tests for neurofeedback effects).

To explore alternative topographies (like effects not at Cz), we applied a typical correction (0.01) considering the high correlation across electrodes. All other tests (different frequency bands and subgroups) are considered as exploratory and are clearly reported so. Since we did not expect stronger effects outside the hypothesized regions of interest, we did not apply a correction for multiple testing to these exploratory tests.

Neurofeedback subgroup analyses

The neurofeedback group was further subdivided into good and poor performers according to their final self-regulation abilities on transfer trials.

Individual mean SCP amplitudes were calculated for every trial type and session during positivity and negativity tasks with the difference between positivity and negativity representing the ability for differentiation. The mean negativation and the mean difference during transfer trials of the second part of the neurofeedback training (sessions 7–14; Drechsler et al. 2007) were used to characterize successful self regulation. Groups of good and poor performers were created by median split using these individual means. Repeated-measures ANOVAs and Pearson's correlations were computed to examine the associations between significant changes in performance, self-regulation abilities and CNV and EEG differences (post minus pre-measurement).

Parental support was categorized into "high" or "low" according to the involvement of the parents in the training and according to parents' self-evaluations during an interview (Drechsler et al. 2007). Eight children were categorized into a "high support" group and the remaining six into a "low support" group. In order to quantify the impact of parental support on neurophysiological measures (QEEG, CNV) repeated-measures ANOVAs were conducted. Relations between parental support and neurophysiological changes were analyzed by correlations.

Results

Changes in quantitative EEG at rest

Mapping EEG band power revealed typical topographies before treatment, but no significant changes (*t*-maps) in the conventional theta, alpha or beta power with treatment (Fig. 1).

Concerning the theta frequency band, direct group comparison revealed no significant group by time interactions for the full theta band at Cz in an ANOVA ($F_{(1,24)} < 1$). No significant group by time interactions were found for ANOVAs at the expected site (Cz) in the full and in the core theta band between 4 and 6 Hz. We explored alternative topographies and found only a single trend for core theta at Oz ($F_{(1,24)} = 3.181$; $P = 0.087$), reflecting a significant mean power decrease for the neurofeedback group (Fig. 2; $t(13) = 3.411$, $P = 0.005$) but not the control group ($t(11) = 0.512$, $P = 0.619$).

No significant group differences in pre-measurement theta measures between neurofeedback and group therapy were found (theta power and theta/beta-ratio, for all derivations $P > 0.01$). However, exploratory analyses of pre-measurement theta power and theta/beta-ratio, respectively, correlated with the behavioral scales across groups: theta/beta-ratio was significantly correlated with the hyperactivity problem score (FBB-HKS; $r = 0.408$, $P = 0.039$), and with social problems (CPRS; $r = 0.415$, $P = 0.035$); midline core theta power (4–6 Hz) was significantly correlated with CPRS-scale social problems (Cz: $r = 0.580$, $P = 0.002$; Pz: $r = 0.434$, $P = 0.027$; POz: $r = 0.406$, $P = 0.040$; Oz: $r = 0.450$, $P = 0.021$) and there were trends for associations between theta/beta-ratio and hyperactivity severity score (FBB-HKS; $r = 0.353$, $P = 0.077$), between theta/beta-ratio and total problem score (FBB-HKS; $r = 0.344$, $P = 0.085$), and core theta power at Cz and hyperactivity problem score (FBB-HKS; $r = 0.338$, $P = 0.092$).

Changes of the theta/beta-ratio at Cz revealed no significant group by time interaction for the full groups, but this interaction became significant for subjects with ADHD-combined subtype ($N = 9$; $F_{(1,15)} = 5.036$, $P = 0.040$), as

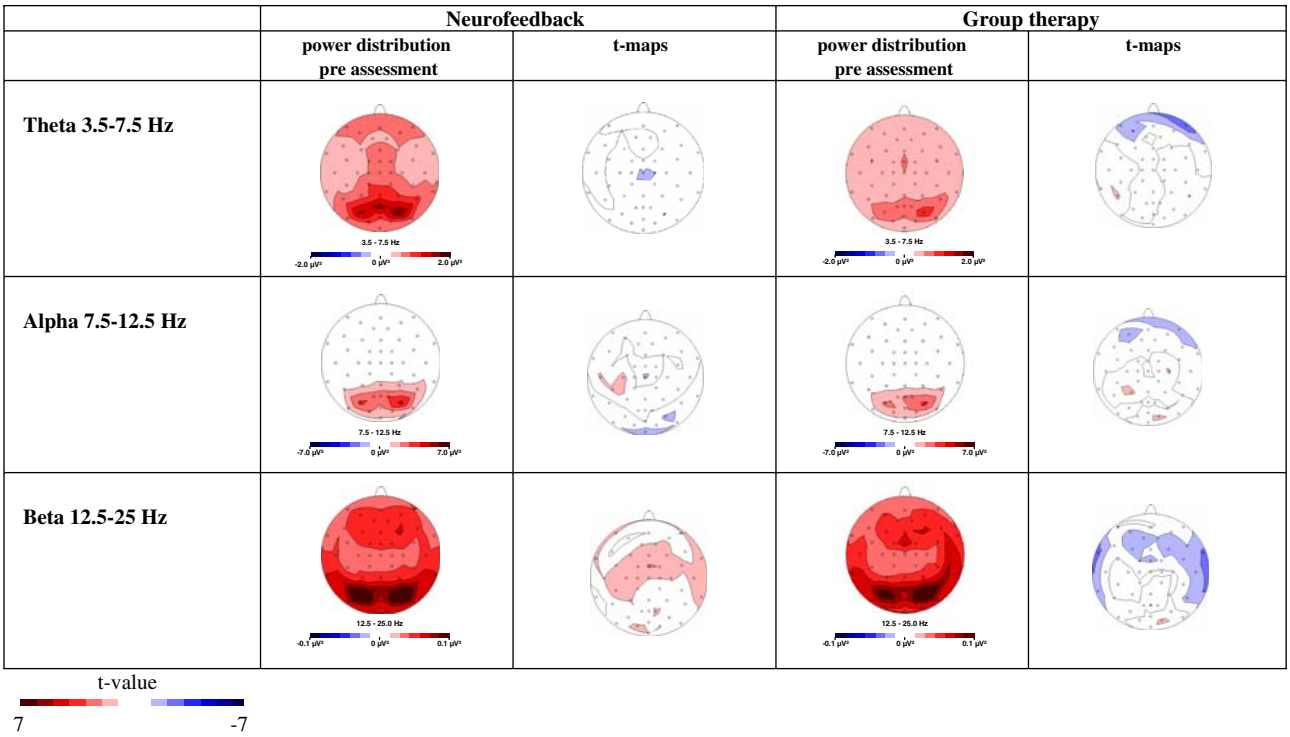

Fig. 1 Power maps (pre-assessment) and *t*-maps for subjects of both groups and different frequency bands (*t*-maps: *red* increase of power, *blue* decrease of power with treatment) (For colors please refer to the online version.)

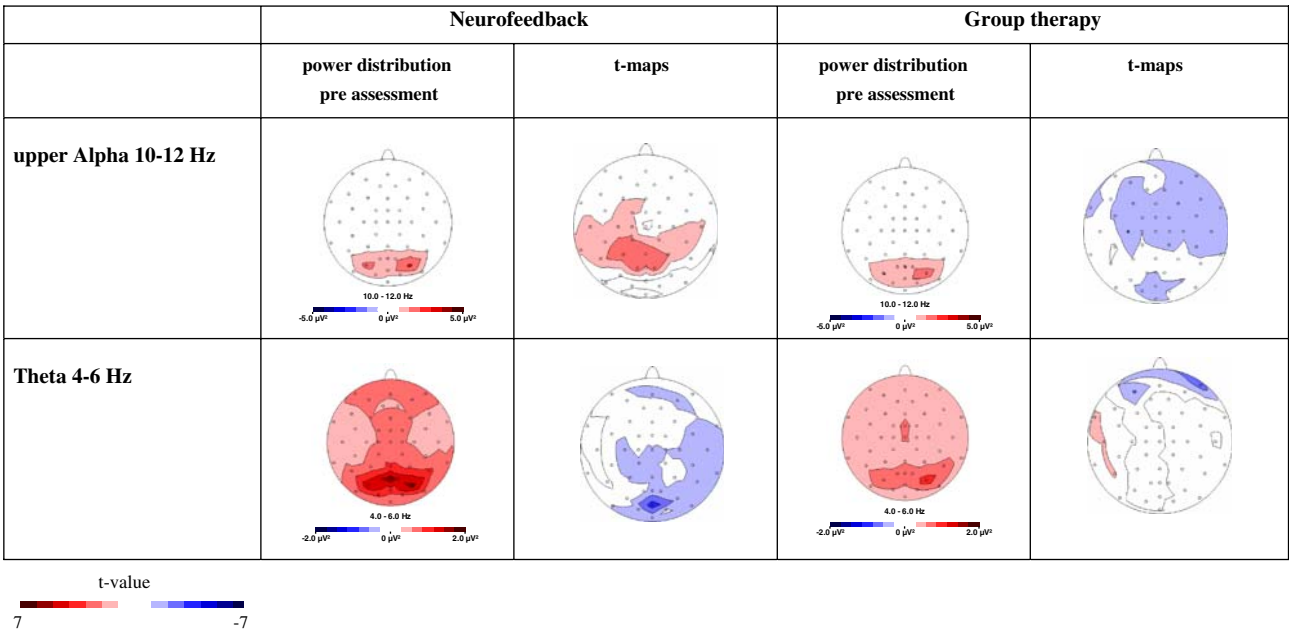

Fig. 2 Power maps (pre-assessment) and *t*-maps for subjects of both groups and different parts of frequency bands (*t*-maps: *red* increase of power, *blue* decrease of power with treatment) (For colors please refer to the online version.)

those in the neurofeedback group significantly decreased their theta/beta-ratio ($t(8) = 2.528$, $P = 0.035$).

Exploratory analysis of changes in alpha activity regarding the *t*-maps revealed some increase of activity for the upper alpha (10–12 Hz) over centro-parietal areas in the neurofeedback group (Fig. 2). Direct comparison between both treatments (NF vs. GT) revealed significant group by time interactions ($F_{(1,24)} = 5.003$, $P = 0.035$) for

upper alpha (10–12 Hz) over the selected central and posterior areas, but not over the occipital ones (Oz, O1, O2, OI1, OI2). The paired t test displayed several significant differences at central, parietal and occipitoparietal electrodes at the $P < 0.05$ level, but none at the $P < 0.01$ level.

No significant group differences were found in pre-measurement alpha power ($P > 0.01$ for all derivations).

Changes in CNV during CPT performance

Contingent negative variation (CPT-standard and CPT-flanker) map and waveshape measurements suggested significantly smaller CNV amplitudes at post-measurement instead of the expected improvement in both groups (Fig. 3), but in accordance with a lack of significant improvements in the CPT performance (Tables 4 and 5).

The MANOVA (measures: CPT-standard-CNV and CPT-flanker-CNV at Cz) revealed a main effect for time ($F_{(2,22)} = 16.882$, $P = 0.000$) reflecting this CNV amplitude reduction, but no significant group by time interaction ($F_{(2,22)} = 1.682$, $P = 0.209$), even though inspection of the means (Fig. 3) suggested a less pronounced CNV reduction for the neurofeedback group.

Paired t test indicated significant mean CNV amplitude reductions from pre- to post-test for both CPT variants and both groups (Tables 4 and 5).

No significant group differences in CNV amplitude were present before treatment (pre-measurement; CPT: $t(22) = -0.379$, $P = 0.709$/CPT-flanker: $t(22) = -0.069$, $P = 0.946$).

Neurofeedback subgroup analyses

Most of the subjects learned to differentiate between positivation and negativation. They especially learned to increase cortical activation (negativation) whereas positivation did not improve significantly over time but seemed to occur quite spontaneously from the beginning (Drechsler et al. 2007). Table 2 shows the mean amplitudes of negativation during transfer trials and separately for good and poor performers. Half of the subjects learned to transfer negativation.

The CNV-changes (amplitude reduction) did not interact with transfer training performance. Only a trend for main effect of time was observed (MANOVA negativation: group × time $F_{(2,10)} < 1$, group $F_{(2,10)} < 1$, time $F_{(2,10)} = 3.953$, $P = 0.054$; MANOVA differentiation: group × time $F_{(2,10)} < 1$, group $F_{(2,10)} < 1$, time $F_{(2,10)} = 3.988$, $P = 0.053$). However, there were differential correlations between CNV-changes and transfer training performance (negativation and differentiation) within the subgroups. CNV-changes (CPT-standard) tended to correlate with abilities to transfer negativation for good performers ($r = 0.804$, $P = 0.054$; for poor performers: $r = 0.474$, $P = 0.283$), and CNV-changes in the CPT-flanker correlated significantly with abilities to transfer

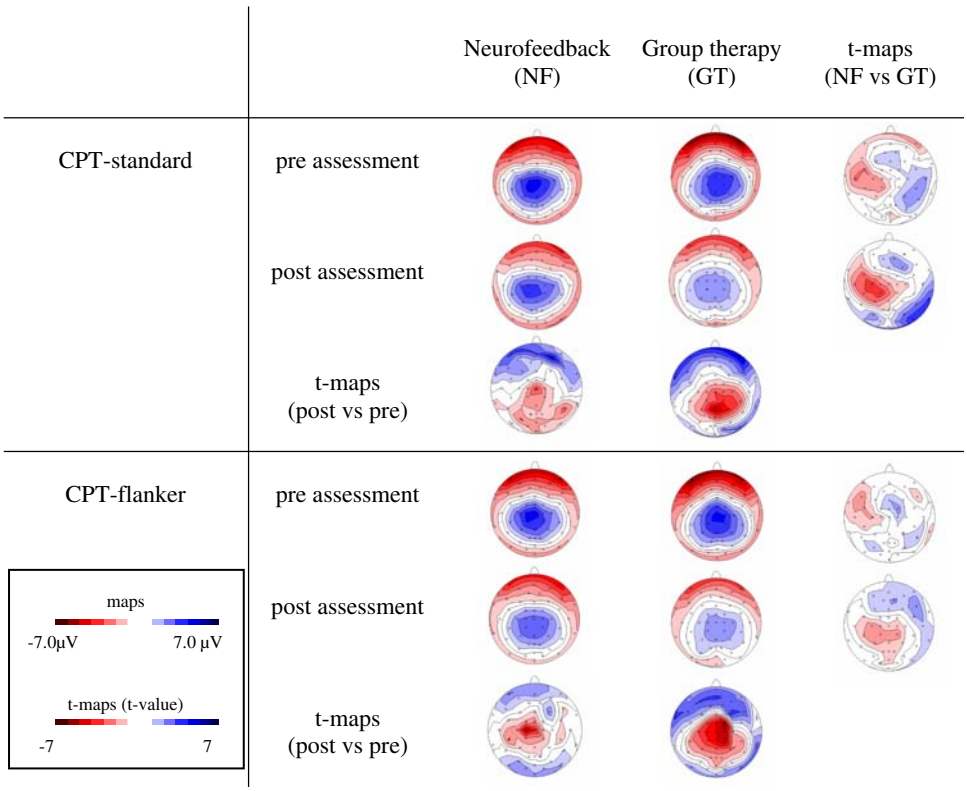

Fig. 3 Maps of the CNV (1,000–1,600 ms, CPT-standard and CPT-flanker) for pre and post assessment and both groups, t-maps depict the differences between post and pre assessment and between both groups, respectively. (For colors please refer to the online version)

Table 2 Mean amplitudes of negativation during transfer condition and classification into good versus poor performer

Negativation—transfer condition		
Subject no.	Mean amplitudes sess. 7–14 (μV)	Performance
2005	−3.24	Good performer (−0.14 to −4.15 μV)
2013	−2.71	
2014	−0.14	
2015	−0.42	
2016	−2.20	
2017	−4.15	
2019	−1.89	
2001	0.27	Poor performer (0.27 to 5.31 μV)
2002	3.72	
2003	2.53	
2007	2.29	
2008	1.62	
2010	1.01	
2018	5.31	

differentiation for good performers ($r = -0.883$, $P = 0.047$; for poor performers: $r = -0.016$, $P = 0.973$). In both cases better neurofeedback training performance was associated with less reduction of CNV amplitude for good performers only.

Theta/beta-ratio at Cz also did not interact with the training performance in transfer conditions. Furthermore we could not find any significant correlations between theta/beta-ratio changes in either one of the neurofeedback subgroups.

Changes in neurophysiological measures (CNV and QEEG: core theta at Oz, upper alpha at Pz, theta/beta-ratio at Cz) and CPT performance did not interact with parental support and no significant correlations between these changes of neurophysiological measures and parental support were found.

Correlating changes in QEEG and behavior scales for the neurofeedback group

Table 3 shows the results: Theta/beta-ratio reductions at Cz correlated with reductions on the CPRS-scale hyperactivity ($r = 0.643$, $P = 0.013$) and tended to correlate with reduced problem score of FBB-HKS subscale hyperactivity ($r = 0.510$, $P = 0.063$).

Further exploratory analyses revealed the following: Increased alpha power at Oz (7.5–12.5 Hz) tended to correlate with decreased hyperactivity in parents rating (CPRS-R:L). Upper alpha activity increases at Pz (10–12 Hz) tended to correlate with reduced impulsivity (FBB-HKS problem score, $r = -0.484$, $P = 0.080$).

Theta power reductions (3.5–7.5 Hz) at Cz correlated with FBB-HKS hyperactivity subscale problem score reduction ($r = 0.654$, $P = 0.011$). They also tended to be associated with lower values on the CPRS hyperactivity subscales ($r = 0.517$, $P = 0.058$), a reduced severity score of the FBB-HKS hyperactivity subscale ($r = 0.469$, $P = 0.091$), and a reduced severity score of the FBB-HKS impulsivity subscale ($r = 0.480$, $P = 0.083$).

CPT-standard and CPT-flanker performance

CPT performance [measures: RT and its standard deviation (RT–SD), target hit rate and total errors] indicated no significant group (NF vs. GT) by time interactions for both CPT variants. The only significant difference between

Table 3 Pearson's correlations between changes in QEEG and behaviour scales for neurofeedback subjects

	CPRS-R:L Hyperactivity	CTRS-R:L Hyperactivity	FBB-HKS Hyperactivity[a]	FBB-HKS Impulsivity[a]
Alpha power increase at Oz	**−0.485**$^{+}$	0.088	−0.164	−0.139
			−0.302	0.082
Upper alpha power increase at Pz	0.328	−0.176	−0.232	−0.117
			0.110	**−0.484**$^{+}$
Theta power decrease at Cz	**0.517**$^{+}$	−0.197	**0.469**$^{+}$	**0.480**$^{+}$
			0.654*	0.352
Core theta power decrease at Oz	0.358	−0.231	−0.251	−0.021
			0.016	−0.076
Theta/beta-ratio reduction at Cz	**0.643***	0.060	0.436	0.403
			0.510^{+}	0.243

CPRS-R:L Conners Parent Rating Scale, *CTRS-R:L* Conners Teacher Rating Scale, *FBB-HKS* German version of ADHD rating scale

[a] First line: severity score, second line: problem score

* $P < 0.05$, $^{+}$ $P < 0.1$

Table 4 CPT-standard performance and CNV mean amplitudes in the pre- and post-measurement comparing neurofeedback versus group therapy

CPT-standard performance								
	Neurofeedback ($N = 12$)				Group therapy ($N = 11$)			
	Pre (SD)	Post (SD)	*t* test/sign.	Effect size *d*	Pre (SD)	Post (SD)	*t* test/Sign.	Effect size *d*
RT (ms)	408.50 (52.11)	450.67 (83.93)	NS	−0.60	458.73 (80.40)	468.73 (86.70)	NS	−0.12
RT–SD	146.67 (44.19)	198.67 (85.41)	$t = -2.30/P = 0.042$	−0.76	173.36 (70.64)	170.36 (64.85)	NS	0.04
Targets	37.58 (1.98)	35.00 (4.22)	NS	−0.78	36.18 (4.85)	36.55 (3.17)	NS	0.09
ERR-total	5.00 (3.10)	7.67 (6.93)	NS	−0.50	5.36 (7.12)	6.00 (5.35)	NS	−0.10
ERR-omiss	2.41 (1.98)	5.00 (4.22)	NS	−0.79	3.82 (4.85)	3.45 (3.17)	NS	0.09
ERR-comiss	2.58 (2.07)	2.67 (3.50)	NS	−0.03	1.54 (3.24)	2.54 (2.81)	NS	−0.33
ERR-impuls	1.75 (1.36)	1.58 (1.73)	NS	0.11	1.09 (2.39)	1.18 (1.60)	NS	−0.04
CNV (μV)	−3.81 (1.61)	−2.85 (1.68)	$t = -3.017/P = 0.011$	−0.58	−3.49 (2.31)	−1.78 (1.66)	$t = -2.818/P = 0.018$	−0.85

SD standard deviation, *RT* reaction time, *ERR-total* total errors, *ERR-omiss* omission errors, *ERR-comiss* commission errors, *ERR-impuls* impulsivity errors

Table 5 CPT-flanker performance and CNV mean amplitudes in the pre- and post-measurement comparing neurofeedback versus group therapy

CPT-flanker performance								
	Neurofeedback ($N = 12$)				Group therapy ($N = 11$)			
	Pre (SD)	Post (SD)	*t* test/ Sign.	Effect size d	Pre (SD)	Post (SD)	*t* test/sign.	Effect size d
RT (ms)	498.17 (109.17)	490.42 (44.54)	NS	0.09	486.55 (83.94)	537.27 (90.12)	NS	−0.58
RT-SD	167.00 (49.41)	178.08 (43.47)	NS	−0.24	175.18 (51.04)	197.91 (37.16)	NS	−0.51
Targets	35.33 (4.52)	36.00 (2.59)	NS	−0.18	35.82 (3.74)	36.00 (3.55)	NS	−0.05
ERR-total	15.58 (17.02)	7.08 (3.75)	NS	0.69	9.54 (8.41)	7.09 (5.68)	NS	0.34
ERR-omiss	4.67 (4.52)	4.00 (2.59)	NS	0.18	4.18 (3.74)	4.00 (3.55)	NS	0.05
ERR-comiss	10.92 (16.41)	3.08 (2.27)	NS	0.67	5.36 (5.54)	3.09 (3.53)	NS	0.49
ERR-impuls	3.08 (2.15)	1.58 (1.73)	NS	0.77	3.63 (4.52)	1.18 (1.60)	$t = 2.469/P = 0.033$	0.72
CNV (μV)	−3.97 (1.51)	−2.49 (1.34)	$t = -3.590/P = 0.004$	−1.04	−3.91 (2.15)	−1.96 (1.52)	$t = -4.093/P = 0.002$	−1.04

For abbreviations see Table 4

pre- and post-measurement was an increase of RT–SD in CPT-standard performance for the neurofeedback group (Table 4).

Total error rate and RT (CPT-standard) did not improve with either training, and both groups even made more errors and responded slower after treatment, although this deterioration was not significant (Table 4).

A similar lack of significant changes was observed for CPT-flanker performance, except that both groups improved with regard to their impulsivity errors (effect size 0.72 and significant *t* test for group therapy subjects; effect size 0.77 for neurofeedback subjects; Table 5).

The neurofeedback subgroup analyses revealed no significant group by time interactions for both CPT variants, and no group differences between good and poor neurofeedback performers (as defined by negativity as well as differentiation on transfer trials) for either CPT version.

Correlating changes in QEEG, CNV and CPT measures for neurofeedback subjects

The theta/beta-ratio reduction in the ADHD-combined did not show significant correlations with the CNV reduction and CPT measures.

Further exploratory analyses revealed the following: The increase in QEEG upper alpha activity (10–12 Hz at Pz) correlated with the (also significant) increase in standard deviation of CPT reaction time ($r = 0.623$, $P = 0.030$).

The significant CNV amplitude reduction in the CPT-flanker tended to correlate with the significant increase in QEEG upper alpha activity (10–12 Hz at Pz: $r = 0.543$, $P = 0.055$). Overall, the CNV reduction in the CPT-standard (but not in the CPT-flanker) correlated with increases in full band theta ($r = -0.662$, $P = 0.010$) and full band alpha ($r = -0.732$, $P = 0.003$).

Discussion

The present study focused on neurophysiological effects of a SCP neurofeedback training for children with ADHD. These EEG and ERP effects were compared to those in a control group of ADHD-children involved in a group training program of comparable intensity. We were especially interested in the evaluation of specific versus non-specific effects of SCP neurofeedback training. In addition to probing specificity through comparison with the control group, we thus examined associations between individual training success and neurophysiological changes. Behavioral and neuropsychological aspects of this study have been reported by Drechsler et al. (2007).

Our results are roughly in line with former studies on behavioral improvements after neurofeedback training (e.g., Lubar et al. 1995; Monastra et al. 2002; Heinrich et al. 2004; Leins et al. 2006; Strehl et al. 2006). Training modalities and outcome were comparable.

Contrary to our hypotheses the ADHD-children who performed the SCP neurofeedback training showed no significant resting EEG changes when directly compared with subjects of the control group. However, exploratory analyses revealed that neurofeedback subjects displayed a trend for decreased core theta power at occipital electrodes. Furthermore we found a slight increase of upper alpha activity, which tended to correlate with an improvement of impulsive behavior (parents' ratings) but also with lower CNV amplitude and with higher standard deviations of CPT reaction time. We interpret this increase of upper alpha activity as a sign of relaxation or detachment, with increased fluctuation of alertness and attention possibly due to impaired motivation.

Theta/beta-ratio at Cz, considered as the most common neurophysiological marker of ADHD by many, was improved after SCP neurofeedback for those subjects with ADHD-combined type, and correlated with improved hyperactivity in parents' ratings. Pre-assessment theta power and theta/beta-ratio, respectively, were associated with ADHD symptoms (especially hyperactivity), which underlines the validity of these electrophysiological markers.

In the EEG session, we could not find any improvements in CPT performance and CNV amplitude except for the reduction of impulsivity errors, which was found in both groups but only for the more complex flanker version and thus suggests practice effects. Instead, significant CNV reduction accompanied the nonsignificant decrements in performance for both groups. We interpret this unexpected result as a motivational problem. It is well known that children suffering from ADHD may perform new tasks quite well. However, once they have to perform boring task batteries repeatedly and without additional motivation or reward, one often observes a tendency to deterioration. We hypothesize that "executive-task aversion" (Sonuga-Barke 2005) might increase with failure or negative experiences on executive tasks, and further reduces the extent to which tasks are intrinsically motivating. Testing this hypothesis would require research with systematic variation of motivational modulators. In other words, the possible effect of the training on the CNV was maybe smaller than the effect of boredom to which children with ADHD seem particularly prone.

Subgroup analyses revealed an interesting effect within the subgroup of good performers, where better abilities in SCP self-regulation during neurofeedback training were associated with higher CNV amplitude. The effect suggests specific influences of SCP neurofeedback training on brain functions; although there was no difference of mean changes in CNV amplitude between good and poor performers (which may be due to widely differing strategies among poor performers). The unexpected lack of CNV improvements may reflect important motivational and non-specific effects. This is consistent with our finding that parental support was an important aspect of the neurofeedback training which strongly modulated behavioral outcome measures (Drechsler et al. 2007).

The decreased theta/beta-ratio after SCP training also points to specific neurophysiological effects of SCP neurofeedback training in children with ADHD. This EEG acceleration and its occipital topography might be considered more typical for theta/beta frequency feedback training which aims at tonic aspects of arousal, while SCP training aims at phasic regulation of excitability (Heinrich et al. 2007). On the other hand, it seems likely that both trainings affect overlapping attentional networks for activation and state regulation, and the topography of neurofeedback effects has hardly been examined before.

Our results also support the notion that neurofeedback training should be regarded as a kind of behavioral psychotherapy, where positive expectations and the experience of self-efficacy are important nonspecific variables (Grawe et al. 2001). To inform the patients about the neurobiological background of the disease and offer to treat them with a neurobiological method is likely to induce positive expectations. The therapeutic relationship and setting may contribute to the positive effects of the training. All these aspects suggest that neurofeedback should be efficacious apart from specific effects due to learned self-regulation of brain activity.

Some shortcomings of our study such as the small groups, the lack of random assignment and a tendency for pre-measurement group differences should also be mentioned. Considering that about half of the neurofeedback subjects were non-responders, it is understandable that the effects for the total neurofeedback group were limited. An important task for future research will be to identify those

ADHD patients with a poor response, given the commitment needed, and to find improved forms of training for them.

It is less clear whether the intense schedule was problematic. The neurofeedback training was performed during school holidays, but the long training double sessions (2 × 45 min) can still be regarded as inconvenient for ADHD-children with their concentration problems. However, the CNV reductions point to a major problem in transferring the behavioral improvements to the lengthy EEG measurements. The EEG-tasks were not exciting, had to be performed alone in a sound insulated testing room, and subjects were not systematically reinforced. We thus consider motivational aspects responsible for the limited results, especially concerning the CPT performance and ERPs. Barkley (2006, p. 323) describes ADHD as a "MDD", motivation deficit disorder. Subjects with ADHD have difficulties in creating and sustaining self-motivation. Self-motivation provides the drive to continue persistently toward tasks and future goals in the absence of externally provided reinforcement or punishment. Children with ADHD are more dependent on these consequences and on sophisticated reinforcement strategies. Further studies should consider these special motivational problems in repeated tests.

We noted improvement in learning negativation until the last sessions of neurofeedback training, suggesting that more sessions could have improved the ability to increase negativity. Thompson and Thompson (1998) report that children with learning difficulties and hyperactive behavior required more than 40 sessions and their improvements may only emerge after 50–60 sessions. They seem to take longer to settle down and generalization of the gains to the classroom also takes longer; maybe this is applicable to ADHD-children generally.

The control group undergoing a group therapy matched in intensity and duration may not be the most appropriate way to control for neurofeedback therapy, partly because of the different (single vs. group) setting where only neurofeedback subjects spent over 30 therapy units exclusively with their therapist. However, the use of optimal control conditions (such as mock neurofeedback) is limited by ethical considerations (Holtmann et al. 2004).

Loo and Barkley (2005) discussed neurofeedback as another form of cognitive-behavioral training that just happens to employ the use of electrodes placed on the head. They suggest that the treatment effect may have nothing to do with the electrophysiology, but rather with the immediate, salient rewards provided for successful performance which are particularly effective in ADHD-children (Oosterlaan and Sergeant 1998). Our results (changes in QEEG, correlations between these changes and behavioral improvements etc.) can only partly rule out such an explanation, which should be tested with different control conditions such as mock- or muscular feedback providing similar immediate feedback.

We conclude that both specific and non-specific effects are responsible for the behavioral effects of SCP neurofeedback. Further studies should clarify the complex relation between non-specific factors and specific effects of neurofeedback.

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