Eur Child Adolesc Psychiatry (2011) 20:481–491
DOI 10.1007/s00787-011-0208-y

ORIGINAL CONTRIBUTION

Neurofeedback in ADHD: a single-blind randomized controlled trial

Ali Reza Bakhshayesh · Sylvana Hänsch · Anne Wyschkon · Mohammad Javad Rezai · Günter Esser

Received: 30 June 2010 / Accepted: 28 July 2011 / Published online: 13 August 2011
© Springer-Verlag 2011

Abstract Neurofeedback treatment has been demonstrated to reduce inattention, impulsivity and hyperactivity in children with attention deficit/hyperactivity disorder (ADHD). However, previous studies did not adequately control confounding variables or did not employ a randomized reinforcer-controlled design. This study addresses those methodological shortcomings by comparing the effects of the following two matched biofeedback training variants on the primary symptoms of ADHD: EEG neurofeedback (NF) aiming at theta/beta ratio reduction and EMG biofeedback (BF) aiming at forehead muscle relaxation. Thirty-five children with ADHD (26 boys, 9 girls; 6–14 years old) were randomly assigned to either the therapy group (NF; $n = 18$) or the control group (BF; $n = 17$). Treatment for both groups consisted of 30 sessions. Pre- and post-treatment assessment consisted of psychophysiological measures, behavioural rating scales completed by parents and teachers, as well as psychometric measures. Training effectively reduced theta/beta ratios and EMG levels in the NF and BF groups, respectively. Parents reported significant reductions in primary ADHD symptoms, and inattention improvements in the NF group were higher compared to the control intervention (BF, $d_{corr} = -.94$). NF training also improved attention and reaction times on the psychometric measures. The results indicate that NF effectively reduced inattention symptoms on parent rating scales and reaction time in neuropsychological tests. However, regarding hyperactivity and impulsivity symptoms, the results imply that non-specific factors, such as behavioural contingencies, self-efficacy, structured learning environment and feed-forward processes, may also contribute to the positive behavioural effects induced by neurofeedback training.

Keywords Biofeedback · Neurofeedback · EMG biofeedback · ADHD · Single-blind

A. R. Bakhshayesh · M. J. Rezai
Department of Psychology, Faculty of Humanities, Yazd University, Yazd, Iran

S. Hänsch · A. Wyschkon · G. Esser (✉)
Department of Clinical Psychology/Psychotherapy and Academy of Psychotherapy and Intervention Research of Potsdam University, Karl-Liebknecht-Straße 24/25, OT Golm, 14476 Potsdam, Germany
e-mail: gesser@uni-potsdam.de

Introduction

Attention deficit/hyperactivity disorder (ADHD) is among the most prevalent childhood disorders [1, 2], affecting approximately 3–5% of school-aged children [3, 4]. Children suffering from ADHD are diagnosed by the following primary symptoms: inattention, hyperactivity and/or impulsivity. European clinical guidelines for hyperkinetic disorder recommend a multimodal treatment, encompassing medication, cognitive-behavioural treatments and parent training [5]. As reviewed by Barkley [6], Greenhill [7] and Swanson [8], patients who respond to stimulants typically demonstrate improved performance and functioning across multiple aspects of ADHD. The outcome of the Multimodal Treatment Study for Children with ADHD [3] suggests that while pharmacological treatments for ADHD are effective in treating core ADHD symptoms, combining such treatments with social skills and parent training yielded additional improvements in secondary areas of psychosocial functioning (e.g., learning, behavioural, emotional, social and family problems) [9]. However, there is no evidence that these clinical improvements continue in

the absence of sustained, long-term treatment with stimulant medication.

EEG biofeedback was developed as an additional or alternative treatment option for children, proceeding from a perspective that ADHD is a neurologically based disorder limiting capacity for attention and behavioural control [10, 11]. Neurofeedback treatments within child and adolescent psychiatry began about 30 years ago [12]. Two training protocols—theta/beta training and training of Slow Cortical Potentials (SCPs)—are typically used in children with ADHD. Findings from EEG and Event-Related Potential (ERP) studies provide the rationale for applying these paradigms in ADHD. In the resting EEG (relaxed awake state, usually with eyes closed), increased slow wave activity (theta, 4–8 Hz) and/or reduced alpha (8–13 Hz) and beta (13–30 Hz) activity, especially in central and frontal regions, might be associated with ADHD (for review, see [11, 13]). This indicates cortical underarousal, particularly in mixed subtypes [14–22]. Thus, it seems plausible that in a paradigm often applied in ADHD, the goal is to decrease activity in the theta band and to increase activity in the beta band (or to decrease theta/beta ratio) at the vertex (electrode Cz), i.e., activating and maintaining a state of cortical arousal ("tonic activation").

In order to expand to the neurophysiologic heterogeneity of ADHD, it should be mentioned that a second pattern of excessive "beta" activity or "hyperarousal" over frontal regions has also been found in patients with ADHD [e.g., 16, 23, 24]. Indeed, EEG analysis has revealed increased relative beta power, decreased relative alpha power and decreased theta/beta power ratios compared to healthy peers [25, 26]. It is discussed whether these "ADHD" patients constitute a different clinical syndrome.

Neurofeedback is a biofeedback method based on the rationale that there is a relationship between surface EEG and the underlying thalamocortical mechanisms responsible for its rhythms and frequency modulations [27]. As reviewed by Sterman [28], variations in alertness and behavioural control appear directly related to thalamocortical generator mechanisms. The principle of NF is that over time, participants learn operant control of their EEG and change from an "abnormal" state to one resembling that of typically developing children. This process is thought to eventually remediate the symptoms associated with ADHD [29]. Monastra et al. [25] conclude that EEG biofeedback is "probably efficacious" for the treatment of ADHD. Case studies and controlled-group studies of EEG biofeedback have demonstrated beneficial effects on measures of intelligence, behavioural rating scales assessing the frequency of the core symptoms of ADHD, computerized tests of attention and QEEG measures of cortical arousal for theta/beta training and SCP training [30–35]. Comparisons with a gold standard treatment for ADHD (stimulant medication) indicated that EEG biofeedback yielded equivalent results [36–38]. However, in the same year as Monastra et al. [25], Loo and Barkley published a review concluding that "…the promise of EEG biofeedback as a legitimate treatment cannot be fulfilled without studies that are scientifically rigorous" [19, p. 73]. The main shortcomings they raised were the lack of well-controlled, randomized studies, small group sizes and the lack of proof that the EEG feedback is solely responsible for the clinical benefit and not unspecific factors such as the additional time spent with a therapist or "cognitive training" [11, 19]. They also criticized the mixed multiple intervention strategies and the disregard for long-term outcomes.

Although many studies have confirmed the effect of neurofeedback in ADHD treatment [for review, see 11, 39–41], Heinrich et al. [11] recommended conducting randomized controlled trials in future studies to disentangle specific and unspecific effects of NF at the clinical level (p. 12).

Controlled studies

In a meta-analytic approach, Arns et al. [41] summarized two types of controlled studies: studies with passive or semi-active control groups, such as waiting list control group and cognitive training, and studies using an active control group such as stimulant medication.

Overall, the study by Gevensleben et al. [42, 43] is the most methodologically sound study to date, including randomization, a large sample size and a multi-centre approach. This study showed a medium ES for hyperactivity (ES = .55) and a large ES for inattention (ES = .97). Only Gevensleben et al. [42] and Holtmann et al. [44] used control groups that were thoroughly and equally trained in an attention-demanding task (computerized cognitive training) to control for unspecific effects in a randomized design. Drechsler et al. [31] used a control group undergoing group therapy but failed to conduct randomization. In all of these studies, neurofeedback compared to a semi-active control group still had medium to large ES for inattention and impulsivity, and small to medium ES for hyperactivity (measurements: rating scale data for hyperactivity and inattention and commission errors on a CPT test as a measure of impulsivity). Computerized cognitive training can be considered to be a suitable control, providing an equal level of cognitive training and client–therapist interaction. But is it the best option?

Loo and Barkley [19] discussed NF as another form of cognitive-behavioural training that merely happens to employ electrodes placed on the head. They suggest that the treatment effect may be related not to the electrophysiology, but rather to the immediate, salient rewards

provided for successful performance, which are particularly effective in ADHD children [45]. Doehnert et al. [46] recommended testing mock—(placebo training, see also [11]) or muscular feedback providing similar immediate feedback to rule out such an explanation.

The present study

The present study aimed to control for unspecific effects (e.g., the fact that training is an attention-demanding task) and confounding variables (e.g., parental engagement, motivational effects, [46]). More specifically, the aim was to control for motivational aspects by using the same immediate feedback scheme. Thus, we chose an innovative single-blind randomized controlled trial: EMG biofeedback training. EMG biofeedback and neurofeedback are conceived as similarly as possible. From a methodological perspective, a yoked-control design would be best to disentangle specific and unspecific effects of NF, but such a mock NF is limited by ethical considerations [39].

We hypothesized that improvements in the NF group would exceed the treatment effects in the control group (BF) regarding behavioural changes as rated by parents and teachers, and improvement on cognitive performance (test of attention). Moreover, we assumed that participants receiving neurofeedback training improve their ability to regulate their cortical activation over time, as represented by decreased activity in the theta band and increased activity in the beta band (i.e., decreased theta/beta ratio).

Method

Participants

Thirty-eight children with hyperkinetic disorder, aged 6–14 years ($M = 9.34$, SD $= 1.92$), participated in this study. Sample size was estimated to be large enough to detect a medium effect size of $f = 0.25$ for the within-between interaction with a power of 0.8 (two-sided, 0.05 level of significance, G-Power assumes a medium effect size for the within-between factor ANOVA interaction and a correlation of 0.5 among the repeated measures, although this sample (18 + 17) would be too small to reliably detect a medium effect size of $d = 0.5$ in terms of *change scores* or *outcome scores* [42, 68]). Informed consent was obtained from the children and parents and in accordance with the ethical standards of the 1964 Declaration of Helsinki. The study was approved by the local ethics committee of the participating universities. The children were recruited from the Institute of Psychotherapy and Intervention Research at the University of Potsdam. The sample consisted of children, who had:

(a) a primary diagnosis of hyperkinetic disorder (disturbance of activity and attention; ICD-10:F90.0) or attention deficit without hyperactivity (ICD-10:F98.8),
(b) an IQ > 80 (CPM, SPM), and
(c) no known neurological or gross organic diseases and no hyperkinetic conduct disorders (ICD-10:F90.1) or pervasive developmental disorders.

Children currently taking stimulant medication were not excluded from the study, but their parents were asked to keep medication levels constant throughout the training period in order to avoid interference effects.

Design and procedure

Children were randomly assigned to one of the two treatment groups (NF: $n = 18$; BF: $n = 17$, ratio NF-group, BF group = 1:1). Children and parents were not explicitly informed about the randomized treatment conditions (NF vs. BF). Pre- and post-treatment assessment consisted of a structured standardized clinical interview [47] to determine a diagnosis and assess comorbid conditions; parent and teacher questionnaires about ADHD-related behaviours (German ADHD Rating Scale, [48]), a paper-and-pencil attention test (bp/d2, [54, 55]), non-verbal intelligence tests (CPM/SPM, [49]), continuous performance tests (CPT, [52]) and standardized behavioural observations in the classroom. Clinical diagnosis was confirmed by an independent psychotherapist using the Diagnostic Checklist for Hyperkinetic Disorders [48]. There were no significant differences between the NF and BF groups on the demographic, psychological and clinical variables prior treatment (see Table 1).

The treatment phase began immediately after pre-treatment assessment. Both trainings consisted of 30 sessions [50] (training period: September 2005–February 2007). Treatment sessions were held 2–3 times per week. Additionally, a psychotherapist met with all parents twice per month for a total of 4 sessions (psychoeducation, effective instructions, rewarding desired behaviour, logical consequences). Only the parents, whose child dropped out after 25 sessions, missed one appointment. Post-treatment assessment was conducted by an independent psychotherapist.

Treatment phase

The treatment phase lasted 10–15 weeks and was identical for both groups in terms of allotted time. Each session lasted 30 min. Both groups experienced similar treatment conditions except for the location of electrodes. The interconnection between the device, body, computer, the relevant software (Bio Trace+®) and the monitor screen was explained. Children received instructions on a

Table 1 Demographic and clinical characteristics of the NF group and EMG biofeedback group: at the pre-training level, there were no significant differences between the groups

	Neurofeedback ($n = 18$)	EMG Biofeedback ($n = 17$)	
Gender (boys/girls)	13/5 (72%/28%)	13/4 (76%/24%)	n.s.
Age			
Mean (SD)	9.6 (2.2)	9.1 (1.6)	n.s.
Range	7–14	6–12	
IQ (CPM, SPM)			
Mean *t*-scores (SD)	50.7 (12.1)	53.2 (9.4)	n.s.
Diagnosis (ICD-10)			
Disturbance of activity and attention (F90.0)	14 (78%)	15 (88%)	n.s.
Inattentive type (F98.8)	4 (22%)	2 (12%)	
Stimulant medication	4 (22%)	3 (18%)	n.s.
Associated disorders	7 (39%)	2 (12%)	n.s.
Conduct disorder (F92.0)	1 (6%)	−(0%)	
Emotional disorder	2 (11%)	−(0%)	
Enuresis	1 (6%)	1 (6%)	
Motor skills disorder/ dyslexia	3 (17%)	2 (12%)	

Dropouts are not included in the table

computer screen to familiarize them with the exercises based on their thoughts or relaxation and their concentration. Children were trained playing three different games: smiley, monkey and ball (see Fig. 1). In the first game, the children were asked to make the face smile and keep it smiling for 3 min. Next, children played the monkey game. Here, children received a point if they succeeded in making the monkey climb a tree and eat some food. The goal of the ball game was move a ball to the top of a pyramid, and to keep it there until the ball blinked. There was a 30-s break between the different games. Each game consisted of three trials lasting 3 min each.

During the treatment sessions, the children's success in completing the exercises was reinforced. Once patients had kept the face smiling or the ball blinking for at least two-thirds of the exercises or had gained three points in one trial in the monkey game, they were rewarded with a Smiley voucher (token economy). After collecting three Smiley vouchers, the child was given a small reward (toy or chocolate). The treatment phase (i.e., therapy process, feedback and rewards) was identical for both groups.

In the first 2 min of each session (first trial of the smiley game), the baseline was determined, by measuring the theta/beta ratio (NF) or the EMG amplitude (BF). During the games, subjects received both positive auditory and

Fig. 1 Screenshots from the different games for Neurofeedback and EMG Biofeedback: smiley, monkey and ball

visual feedback. Children in the NF group were rewarded when the theta amplitude (4–8 Hz) was below the baseline while the beta amplitude (16–20 Hz) was above the baseline. Children in the BF group were rewarded when keeping the EMG amplitude below the baseline.

Theta/beta neurofeedback training

A Nexus® amplifier was used for neurofeedback training. The connection between the electrodes and skin was continuously monitored throughout the session. Nexus uses DC offset checking which is done online and does not interfere with the signals, instead of using an impedance check which interferes with the EEG signals. In order to reduce skin impedance, an opaque adhesive paste (Ten20) was applied. Artefacts were controlled automatically. The thresholds were fixed (theta: 4–8 Hz, beta: 16–20 Hz). The present study employed a theta/beta protocol; thus, active electrodes were located on CPz and FCz, based on the international 10/20 system. The reference electrode was installed on the mastoid ([21], p. 426 and [51], pp. 110–111). Children in the NF group were instructed to use their concentration when playing the different computer games.

EMG biofeedback training

In the BF group, electrodes were placed on the frontalis musculature to measure EMG amplitudes. The children were instructed to use relaxation in order to play the games.

Variables and measurement instruments

Behavioural ratings

Children's behaviour was assessed by their parents and teachers using the ADHD rating scale (FBB-HKS; [48]). FBB-HKS is part of the Diagnostic System for Mental Disorders in Childhood and Adolescence (DISYPS-KJ, [48]) and based on the symptom criteria of ICD-10 and DSM-IV. The scale is frequently used in Germany for evaluating medical or cognitive behavioural treatment. The German ADHD rating scale includes 20 items. The three subscales (a) Inattention (9 items), (b) Hyperactivity (7 items) and (c) Impulsivity (4 items) are all assessed for severity (severity score) and experienced difficulties (problem score). Parents and teachers assessed the behaviour of pre- and post-treatment. The FBB-HKS severity scores, ranging from 0 to 3, constitute the primary outcome measures of this study.

Neuropsychological evaluation

Continuous Performance Task (CPT) The CPT [52] is a computerized test used to measure selective attention, attention duration and impulsive behaviours. The CPT consisted of 400 stimuli (letters) that were presented at the centre of the screen for 200 ms each, with an inter-stimulus interval of 1,400 ms. Children were instructed to respond to a target: letter O followed by the letter X (probabilities for sequences O–X and O-not-X were 10% each). CPT performance was measured by scoring reaction times of hits, variability in reaction times, omission errors (relevant stimuli neither seen nor reacted to) and commission errors (irrelevant stimuli reacted to) [53].

Paper-and-pencil attention tests (bp/d2) The bp-test [54] was administered to 6- to 9-year-old children and the d2-test [55] to children older than 9 years. The bp-test is a standard paper-and-pencil test for measuring short-term selective attention originally standardized for 8-year-old children [56]. The participant has to cross out target letters (b and p) from among other letters (g, q, d and h). In total, there are 12 lines of letters that are randomly ordered, and the participant is given 25 s to complete each line.

In the d2-test, a paper-and-pencil test measuring focused and selective attention, participants cross out target letters on a worksheet, working line by line. The test has been standardized and used for participants aged between 9 and 60 years. The participant is asked to cross out the relevant stimuli (d with two lines) from among the irrelevant ones (d with one, three or four lines and p). Fourteen lines of letters are presented, and subjects are given 20 s for each line.

The following dependent variables were derived from the tests: Correct (R): letters crossed out correctly, indicating the participant's speed. Omission errors (error 1, E1) were scored for each target letter missed (b and p or d letters not crossed out); and Commission errors (error 2, E2): were scored for responses to non-target letters. In both tests, total concentration scores were computed (bp-test: Percentage of errors (E*100/R), d2: total of correct responses minus the total number of commission errors).

A recent validation study of the bp-test with 150 6- to 11-year olds showed high reliability for correctness score (Cronbach's Alpha = .97), and good coefficients for error score and percentage of errors (Cronbach's Alpha = .87). Moreover, concurrent validity (r_{tc} = .590 with d2) was demonstrated [54]. Validity and reliability of the d2 have been verified [55].

Data analysis

Psychometric tests and EEG data

Data were analysed by calculating repeated measures ANOVA. EEG data were evaluated at three assessment points. For all statistical procedures, significance was set at $p < .05$.

Effect sizes

Effect sizes (d_{corr}, [57]) measure the magnitude of the effect and vary from 0.2 (small effect) to 0.5 (medium effect) and 0.8 (large effect). Klauer's d_{corr} is computed as the difference between d_{pre} and d_{post}, as the effect size of the post-treatment advantage for intervention corrected for any pre-treatment group differences. As standard deviation, the pooled deviation of both groups is used $\left(S_p = \sqrt{\frac{\left((N_{EG}-1)\times s_{EG}^2+(N_{GG}-1)\times s_{GG}^2\right)}{N_{EG}+N_{GG}-2}}\right)$.

Results

From the thirty-eight children with hyperkinetic disorder, who were initially assessed and randomly assigned to a training group, three children were excluded (NF: $n = 1$, BF: $n = 2$) due to loss of motivation ($n = 2$) or protocol violation ($n = 1$). One child dropped out after 25 sessions but was treated like children with 30 completed sessions. Hence, 35 children were included in the analysis.

EEG and EMG data elicited from treatment sessions

The 30 treatment sessions in both groups were divided into three 10-session sections in order to provide a better understanding of the observed change. Distorted data caused by movement and muscle artefacts (thresholds were fixed: theta 4–8 Hz, beta 16–20 Hz), electrode disconnection and computer breakdown were excluded. Table 2 shows the group means, standard deviations, the results of one-way ANOVAs with repeated measures and the effect sizes for theta/beta ratios and EMG amplitudes in the neurofeedback and EMG biofeedback group, respectively.

For NF, the theta/beta ratio decreased significantly across training for the smiley and the ball game. The BF group shows a highly significant difference in the EMG amplitude for baseline, the smiley and the ball game. Additionally, EMG amplitude for the monkey game decreased significantly, although the significance level (95%) is lower than that of the other two training games.

German ADHD rating scales (parents)

With mean FBB-HKS total scores of around 1.5, ADHD symptoms were moderately pronounced in both groups prior treatment. Data were analysed by calculating 2 (×2)—ANOVAs, with Treatment group (NF vs. BF) as between-subjects variable and Time (pre- vs. post-treatment) as within-subjects variable. There was a significant main effect for Time: the total score as well as the subscales decreased after treatment (see Table 3). Improvement of the NF group in the FBB-HKS total score (primary outcome measure) was superior to the EMG group ($F(1,33) = 3.72$; $p = .062$), but the interaction failed to reach statistical significance. This effect reached a medium effect size of −.77 (d_{corr}, [57]).

Table 2 Theta/beta ratios and EMG amplitudes (group means, standard deviations, ANOVA results) at baseline and three training conditions (Smiley, Monkey and Ball) in the neurofeedback ($n = 18$) and EMG biofeedback ($n = 17$) group

Group	Neurofeedback					EMG biofeedback				
TS	*M* (SD)	Treatment time			ES (d_{corr})	*M* (SD)	Treatment time			ES (d_{corr})
		df	*F*	*p*			*df*	*F*	*p*	
BL-1	2.838 (0.567)	2	14,799***	.000	.62	8.281 (0.821)	2	10,850***	.001	.55
BL-2	2.854 (0.076)					7.920 (0.280)				
BL-3	2.744 (0.073)					7.331 (0.543)				
TC1-1	2.743 (0.102)	2	5,827*	.011	.39	8.132 (1.092)	2	9,713***	.001	.52
TC 1-2	2.844 (0.086)					7.623 (0.372)				
TC 1-3	2.694 (0.069)					6.940 (0.533)				
TC 2-1	2.781 (0.133)	2	1,284	.301	.13	7.182 (1.078)	2	4,891*	.020	.35
TC 2-2	2.785 (0.094)					6.936 (0.242)				
TC 2-3	2.719 (0.117)					6.355 (0.477)				
TC 3-1	2.769 (0.091)	2	4,785*	.022	.35	7.315 (0.529)	2	10,217***	.001	.53
TC 3-2	2.801 (0.075)					6.964 (0.477)				
TC 3-3	2.697 (0.064)					6.373 (0.487)				

TS Training section, *M* means, *SD* standard deviations, *ES* effect sizes, *BL-1 to BL-3* baseline for the three sections of training, *TC1-1 to TC1-3* training condition 1 (Smiley) for the three sections of training, *TC2-1 to TC2-3* Training condition 2 (Monkey) for the three sections of training, *TC3-1 to TC3-3* training condition 3 (Ball) for the three sections of training; each section consisted of 10 consecutive sessions; * $p \leq .05$; *** $p \leq .001$

The predicted Treatment × Time interaction was significant only for Inattention ($F(1,33) = 6.43$; $p < .05$). This effect reached a large effect size of −.94 (d_{corr}). Moreover, there were no significant differences between Treatment groups.

German ADHD rating scales (teachers)

The results of the ANOVA showed a significant effect of Time for Inattention (NF: $M_{pre} = 1.42 \pm 1.12$; $M_{post} = 0.92 \pm 0.81$; BF: $M_{pre} = 1.06 \pm 0.78$; $M_{post} = 1.06 \pm 0.53$; $F(1,33) = 6.91$; $p = .013$), Hyperactivity (NF: $M_{pre} = 1.17 \pm 0.96$; $M_{post} = 0.69 \pm 0.64$; BF: $M_{pre} = 1.01 \pm 0.81$; $M_{post} = 0.86 \pm 0.59$; $F(1,33) = 6.48$; $p = .016$) and total mean scores (NF: $M_{pre} = 1.38 \pm 0.74$; $M_{post} = 1.04 \pm 0.53$; BF: $M_{pre} = 1.38 \pm 0.57$; $M_{post} = 1.31 \pm 0.57$; $F(1,33) = 5.86$; $p = .021$). There were no significant differences between Treatment groups. The NF group showed an improvement of up to 40%, whereas the BF group showed less improvement on the primary ADHD symptoms. There was no significant interaction between Treatment group and Time. However, there was a significant trend for Impulsivity ($F(1,33) = 3.574$; $p = .068$). The effect sizes showed larger improvements for the NF than for the BF group.

Paper-and-pencil attention tests

Table 4 displays the *t*-scores (mean scores), standard deviations, effect sizes and results of the ANOVA. A 2 (×2)—ANOVA examined the effects of Treatment group and Time. Higher *t*-scores represent better performance. There was no significant effect of Treatment group. The results showed significant improvements on all scales (speed, error and total score) in the paper-and-pencil attention tests after treatment. The Treatment group × Time interactions reached significance for all scales, indicating that NF group improved more compared to the BF group. The medium to large effect sizes support these results.

Continuous Performance Task (CPT)

In the Continuous Performance Task, low *t*-scores indicate better achievement, representing high/fast performance, functioning with high continuity, and few errors. Knye et al. [53] show that commission errors are a particularly sensitive measurement of impulsivity and inattention. There were no significant differences between Treatment groups. The ANOVAs showed significant differences in commission errors between pre- and post-treatment ($F(1,33) = 11.865$; $p = .002$). There was also an expected significant interaction between Treatment group and Time for reaction time ($F(1,33) = 7.359$; $p = .011$), with a medium effect size of −.70 (d_{corr}). Overall, performance in the BF group decreased, while performance of the NF group improved after treatment. The effect sizes vary from $d_{corr} = -.32$ (reaction time variability) to $d_{corr} = -.79$ (reaction time).

Discussion

The present study evaluated neurofeedback training compared to biofeedback training in children with hyperkinetic disorders, in order to gain further information about the efficacy of neurofeedback. In contrast to previous studies, the control treatment (i.e., biofeedback training) was designed to resemble neurofeedback as closely as possible.

Furthermore, the present study attempted to constructively rectify methodological issues of past studies by [19, 58]; obtaining multidimensional diagnosis of an independent (blind) psychotherapist, using subjective and objective

Table 3 Parents' rating GRS: the results of two-way ANOVA with repeated measures for the comparison of the neurofeedback ($n = 18$) and EMG biofeedback ($n = 17$) group regarding hyperkinetic symptoms

Scale	MT	*M* (SD)		Treatment time			Treatment group			Treatment group × time			ES (d_{corr})
		NF	BF	*df*	*F*	*p*	*df*	*F*	*p*	*df*	*F*	*p*	
AD	Pre	1.978 (0.789)	1.713 (0.583)	1	4.49*	.042	1	0.70	.794	1	6.43*	.016	−.94
	Post	1.400 (0.614)	1.756 (0.667)										
H	Pre	1.289 (0.764)	1.147 (0.560)	1	17.76***	.000	1	0.00	.967	1	1.60	.215	−.51
	Post	0.644 (0.440)	0.800 (0.602)										
I	Pre	1.650 (0.695)	1.594 (0.638)	1	18.08***	.000	1	0.17	.679	1	1.23	.275	−.39
	Post	0.978 (0.506)	1.200 (0.899)										
TS	Pre	1.689 (0.641)	1.512 (0.469)	1	12.59***	.001	1	0.07	.796	1	3.72	.062	−.77
	Post	1.072 (0.408)	1.329 (0.691)										

GRS German ADHD rating scale, *AD* attention deficit mean score, *H* hyperactivity mean score, *I* impulsivity mean score, *TS* total mean score, *Pre* pre-test, *Post* post-test, *MT* measurement time, *M* mean, *SD* standard deviation, *ES* effect size, *NF* neurofeedback group, *BF* EMG biofeedback group, * $p \leq .05$; *** $p \leq .001$

Table 4 Paper-and-pencil attention tests and CPT (*t*-score): Speed, error, total scores and reaction time, variability and omission/commission errors in the pre- and post-measurement comparing neurofeedback ($n = 18$) versus EMG biofeedback ($n = 17$)

Scale	MT	*M* (SD)		Treatment time			Treatment group			Treatment group × time			ES (d_{corr})
		NF	BF	*df*	*F*	*p*	*df*	*F*	*p*	*df*	*F*	*p*	
S	Pre	42.67 (10.77)	43.24 (10.43)	1	76.5***	.000	1	1.8	.189	1	14.17***	.001	.88
	Post	60.83 (11.00)	50.47 (14.00)										
E	Pre	51.56 (10.57)	51.76 (14.22)	1	11.23**	.002	1	0.945	.338	1	4.39*	.044	.68
	Post	61.00 (6.64)	53.94 (13.71)										
TCS	Pre	47.50 (10.00)	46.29 (13.20)	1	31.75***	.000	1	3.91	.056	1	8.3**	.007	.99
	Post	63.50 (8.60)	51.47 (13.00)										
RT	Pre	59.72 (5.55)	60.29 (6.24)	1	<0.01	.959	1	3.29	.079	1	7.36*	.011	−.79
	Post	56.67 (7.67)	63.24 (7.06)										
RTV	Pre	50.83 (8.27)	49.41 (8.27)	1	3.14	.086	1	<0.01	.971	1	0.59	.450	−.32
	Post	46.39 (8.01)	47.65 (9.03)										
OM	Pre	54.72 (9.31)	55.88 (12.65)	1	1.29	.264	1	1.88	.180	1	2.38	.133	−.54
	Post	48.89 (10.08)	56.76 (14.25)										
COM	Pre	53.06 (11.26)	54.71 (9.92)	1	11.87**	.002	1	3.59	.067	1	2.50	.123	−.70
	Post	41.94 (10.73)	50.59 (9.33)										

CPT Continuous Performance Task, *MT* measurement time, *SD* standard deviations, *ES* effect sizes, *NF* Neurofeedback group, *BF* EMG biofeedback group, *S* speed, *E* error, *TCS* total concentration score, *RT* reaction time, *RTV* reaction time variability, *OM* omission error, *COM* commission error, *Pre* pre-test, *Post* post-test; * $p \leq .05$; ** $p \leq .01$; *** $p \leq .001$

outcome parameters (parent and teacher ratings, psychometric tests, EEG/EMG data), a power-analysis-based sample size and a randomized group assignment.

NF exceeded BF on one subscale (Inattention) of the parent ratings, but not on any teacher ratings. The effect sizes further indicate large improvement of attention (parents) and medium improvement of hyperactivity (parents) and impulsivity (teacher) in the neurofeedback group compared to the control group. Overall, results of the ADHD rating scales show significant improvements after treatment on all subscales of parents' and in three of four subscales of teachers' ratings.

Heinrich et al. [32], who employed a slow cortical potential protocol (SCP), found ADHD rating scale score decreased by a quarter. In our study, neurofeedback training reduced parent's ratings by 39%, and teacher's ratings by 26%. The findings of the present study therefore corroborate studies reporting the perceived alleviation of symptoms as a result of the neurofeedback method assessed via rating scales [30, 33, 36, 38, 59 among others].

The ANOVA results show some improvements in the children's performance in paper-and-pencil attention tests (bp/d2) after treatment. As predicted, the neurofeedback group outperformed the EMG biofeedback group on all test parameters, with medium to large effects.

Overall, one of the four CPT indicators (commission errors) improved significantly over time, indicating that all children showed fewer impulsive reactions. Only one significant interaction (RT) showed the hypothesized positive change in NF compared to BF. The other non-significant Treatment group × Time interactions showed no advantage of NF over BF. This was surprising, as Heinrich et al. [32] found a decrease in impulsivity errors (CPT-OX) in SCP neurofeedback training in a waiting list control group design.

As discussed in the previous studies, such unpredicted results could be caused by inter-individual differences (e.g., non-responders in neurofeedback training groups [46]). Thus, training protocols have to be optimized to select the best possible paradigm for an individual or a certain clinical or spectral EEG profile subtype.

Another reason for non-significant differences between different treatment groups might be the choice of control group, which will be discussed next.

EMG biofeedback—a suitable placebo?

The chosen protocol offers advantages but also bears risks. EMG biofeedback was used as the reinforcer-control condition due to its similarity to neurofeedback. Treatment conditions were identical for both Treatment groups (computer games, duration of training, number of sessions, rewards, diagnostic assessment and psychotherapist) except for the placement of electrodes. Across groups, the child was attended to by one therapist in a structured setting, receiving continuous and consistent feedback [44]. To further increase similarities between the treatment groups, electrodes for BF were placed on the frontalis musculature.

Additionally, BF, compared to a waiting list control group, offers control over effects of therapy expectations. As spontaneous remission of ADHD is unlikely, one might risk instead a high percentage of drop-outs within the waiting group.

There are conflicting results in the application of relaxation/EMG biofeedback in ADHD that poses the question, whether EMG biofeedback is really just a control condition. In the seventies and eighties, studies demonstrated in part the efficacy of relaxation training in the treatment of hyperkinetic disorders, particularly in the reduction of restlessness ([60], for review, see [61]). Cobb and Evans [62] concluded that there was no evidence that biofeedback was superior to "more conventional treatments" in learning or behavioural disorders. Omizo and Michael [63] randomly assigned hyperactive boys, aged 10–12 years, to either four sessions of EMG biofeedback-induced relaxation ($n = 16$) or sham treatment ($n = 16$) of equal length. The relaxation induced significant improvements in attention and impulsivity compared to sham control (ES = 1.0–1.3, $p < 0.01$). Despite these encouraging results, EMG biofeedback was not applied in more recent studies in ADHD treatment. In the present single-blind study, BF group showed significant reduction in hyperactivity ($M_{pre} = 1.15 \pm 0.56$ vs. $M_{post} = 0.8 \pm 0.60$, $t(16) = 1.84$, $p = .05$) and impulsivity ($M_{pre} = 1.59 \pm 0.63$ vs. $M_{post} = 1.20 \pm 0.90$; $t(16) = 1.91$, $p = .05$) in parent ratings. This could be one reason for the lack of significant group differences in those ADHD symptoms. Maybe the ongoing DFG-funded study on ADHD and Peripheral electromyographic (EMG) biofeedback at the Central Institute of Mental Health (Germany, Dr. Holtmann) can address this question in detail.

Methodological considerations

The following shortcomings of our study should also be mentioned: uniform treatment of all children without taking into account individual neurophysiologic profiles, the combination with medication and lack of follow-up. Although 6-month follow-up measurement was conducted, the results were not published in this paper. Concerning medication, there were no pre-testing differences between both groups, but non-specific effects might be possible nonetheless.

Conclusion

Neurofeedback training improved hyperkinetic symptoms overall, but we were unable to prove that the effects of neurofeedback training were superior to those of EMG biofeedback training with regard to hyperactivity and impulsivity symptoms on rating scales. Specific improvements were found for inattention symptoms on parent rating scales and for reaction time in neuropsychological tests.

Comparable benefits, specifically on scales of cognitive regulation (inattention and metacognitive abilities), were found by Drechsler et al. [31], but they did not detect any advantage for behavioural regulation (e.g., inhibitory control, hyperactivity) for the feedback training compared to a group training programme. Heywood and Beale [64] used sham treatment in a single-blind uncontrolled case study, revealing possible placebo effects in EEG biofeedback treatment in ADHD. Children and parents were aware that some biofeedback sessions would not be the "real" biofeedback, but they were unaware which sessions were placebo sessions. The primary finding of this study was that when all seven participants were included in analyses controlling for overall trends, EEG biofeedback was no more effective than a placebo control condition involving non-contingent feedback, and neither procedure resulted in improvements relative to baseline levels. This is in line with Logeman et al. [65], who incorporated a sham group-controlled double-blind design with 27 students to control for unspecific effects and to investigate the effect of neurofeedback above placebo. No interaction proved to be significant at the behavioural level. The findings suggest that neurofeedback may have no effect on behaviour when accounting for unspecific factors. However, the specific form of neurofeedback and application of the design may have diminished the effect of neurofeedback.

It should be discussed whether the therapeutic alliance itself may be the factor that results in a change in brainwave activity [66]. Other extraneous factors include behavioural contingencies, self-efficacy, relaxation, structured learning environment, routines and feed-forward processes like constructing response strategies [64].

Future directions

An important aspect for future research will be to identify predictors and mediators of response and to clarify the complex relationship between non-specific factors and specific effects of neurofeedback. One step towards this goal was to discuss EMG biofeedback as an innovative condition, to control for the amount of feedback and reinforcement. Hyperactivity could potentially be positively affected by EMG biofeedback and relaxation training [67]. Therefore, it would be beneficial to find a neutral approach, which should not, however, reduce the similarities between the two methods. For example, in the placebo group, a pre-recorded "average" neurofeedback session might be used. Additionally, in order to enable the transfer into children's daily life, neurofeedback should be

embedded within school-related activities [51]. The current study attempted this for part of the training, but documentation was insufficient for empirical analysis.

Conflict of interest The authors declare that they have no conflict of interest.

References

1. Doepfner M (2002) Hyperkinetische Störungen. In: Esser G (ed) Lehrbuch der klinischen Psychologie und Psychotherapie des Kindes- und Jugendalters, pp 172–196. Thieme, Stuttgart
2. Scahill L, Schwab-Stone M (2000) Epidemiology of ADHD in school-age children. Child Adolesc Psychiatr Clin N Am 9(3):541–555
3. MTA Cooperative Group (1999) A 14-month randomized clinical trial of treatment strategies for attention-deficit/hyperactivity disorder. Arch Gen Psychiatry 56(12):1073–1086
4. Pelham WE, Gnagy EM (1999) Psychological and combined treatments for ADHD, mental retardation and developmental disabilities. Res Rev 5(3):225–236
5. Taylor E, Döpfner M, Seargant J et al (2004) European clinical guidelines for hyperkinetic disorder—first upgrade. Eur Child Adolesc Psychiatry 13(Suppl 1):17–30
6. Barkley RA (2006) Attention-deficit hyperactivity disorder: a handbook of diagnosis and treatment. Guilford Press, New York
7. Greenhill LL (2002) Stimulant medication treatment of children with attention deficit hyperactivity disorder. In: Jensen PS, Cooper JR (eds) Attention deficit hyperactivity disorder: state of the science—best practices. Civic Research Institute, Kingston, pp 9.1–9.27
8. Swanson JM (1993) Effect of stimulant medication on hyperactive children: a review of reviews. Except Child 60:154–162
9. Jensen PS, Hinshaw SP, Swanson JM et al (2001) Findings from the NIMH Multimodal Treatment Study of ADHD (MTA): implications and applications for primary care providers. Dev Behav Pediatr 22:60–73
10. National Institutes of Health Consensus Statement (1998) Diagnosis and treatment of attention deficit hyperactivity disorder
11. Heinrich H, Gevensleben H, Strehl U (2007) Annotation: neurofeedback—train your brain to train behaviour. J Child Psychol Psychiatry 48(1):3–16
12. Lubar JF, Shouse MN (1976) EEG and behavioural changes in a hyperkinetic child concurrent with training of the sensorimotor rhythm (SMR): a preliminary report. Biofeedback Self Regul 1:293–306
13. Barry RJ, Clarke AR, Johnstone SJ (2003) A review of electrophysiology in attention-deficit/hyperactivity disorder: II. Event-related potentials. Clin Neurophysiol 114:184–198
14. Chabot RJ, Serfontein G (1996) Quantitative electroencephalographic profiles of children with deficit disorder. Biol Psychiatry 40:951–963
15. Clarke AR, Barry RJ, McCarthy R, Selikowitz M (2001) Age and sex effects in the EEG: differences in two subtypes of attention deficit hyperactivity disorder. Clin Neurophysiol 112:815–826
16. Clarke AR, Barry RJ, McCarthy R, Selikowitz M (2001) Electroencephalogram differences in two subtypes of attention-deficit/hyperactivity disorder. Psychophysiology 38:212–221
17. El-Sayed E, Larsson JO, Persson HE, Rydelius PA (2002) Altered cortical activity in children with attention deficit/hyperactivity disorder during attentional load task. J Am Acad Child Adolesc Psychiatry 41:811–819
18. Hill RW, Castro E (2002) Getting rid of Ritalin: how neurofeedback can successfully treat attention deficit disorder without drugs. Hampton Roads Publishing Company, USA
19. Loo SK, Barkley RA (2005) Clinical utility of EEG in attention deficit hyperactivity disorder. Appl Neuropsychol 12(2):64–76
20. Lubar JF (1991) Discourse on the development of EEG diagnostics and biofeedback treatment for attention deficit/hyperactivity disorders. Biofeedback Self Regul 16:202–225
21. Lubar JF (2003) Neurofeedback for the management of attention deficit disorders. In: Schwartz MS, Andrasik F (eds) Biofeedback: a practitioner's guide, 3rd ed. Guilford Press, New York, pp 409–437
22. Mann CA, Lubar JF, Zimmermann AW, Miller CA, Muenchen RA (1992) Quantitative analysis of EEG in boys with attention-deficit hyperactivity disorder: controlled study with clinical implications. Pediatr Neurol 8(1):30–36
23. Chabot RJ, Orgill AA, Crawford G, Harris MJ, Serfontein G (1999) Behavioral and electrophysiologic predictors of treatment response to stimulants in children with attention disorders. J Child Neurol 14(6):343–351
24. Clarke AR, Barry RJ, McCarthy R, Selikowitz M (2001) Excess beta activity in children with attention-deficit/hyperactivity disorder: an atypical electro-physiological group. Psychiatry Res 103:205–218
25. Monastra VJ (2005) Electroencephalographic biofeedback (neurotherapy) as a treatment for attention deficit hyperactivity disorder: rationale and empirical foundation. Child Adolesc Psychiatr Clin N Am 14:55–82
26. Monastra VJ, Lynn S, Linden M, Lubar JF, Gruzelier J, LaVaque T (2005) Electroencephalographic biofeedback in the treatment of attention-deficit/hyperactivity disorder. Appl Psychophysiol Biofeedback 30(2):95–114
27. Vernon DJ (2005) Can neurofeedback training enhance performance? An evaluation of the evidence with implications for future research. Appl Psychophysiol Biofeedback 30(4):347–364
28. Sterman MB (1996) Physiological origins and functional correlates of EEG rhythmic activities: implications for self-regulation. Biofeedback Self Regul 21(1):3–49
29. Thompson L, Thompson M (1998) Neurofeedback combined with training in metacognitive strategies: effectiveness in students with ADD. Appl Psychophysiol and Biofeedback 23(4):243–263
30. Carmody DP, Radvanski DC, Wadhwani S, Sabo MJ, Vergara L (2001) EEG biofeedback training and attention-deficit/hyperactivity disorder in an elementary school setting. J Neurotherapy 4(3):5–27
31. Drechsler R, Straub M, Doehnert M, Heinrich H, Steinhausen HC, Brandeis D (2007) Controlled evaluation of a neurofeedback training of slow cortical potentials in children with attention-deficit/hyperactivity disorder (ADHD). Behav Brain Funct 3:35–48
32. Heinrich H, Gevensleben H, Freisleder FJ, Moll GH, Rothenberger A (2004) Training of slow cortical potentials in attention-deficit/hyperactivity disorder: evidence for positive behavioral and neurophysiological effects. Biol Psychiatry 55:772–775
33. Linden M, Gevirtz R, Isenhart R, Fisher T (1996) Event related potentials of subgroup of children with attention deficit hyperactivity disorder and the implications for EEG biofeedback. J Neurotherapy 1(3):40–49
34. Strehl U, Leins U, Goth G, Klinger C, Hinterberger T, Birbaumer N (2006) Self-regulation of slow cortical potentials: a new treatment for children with attention-deficit/hyperactivity disorder. Pediatrics 118:1530–1540
35. Gevensleben H, Holl B, Albrecht B, Schlamp D, Kratz O, Studer P, Wangler S, Rothenberger A, Moll GH, Heinrich H (2010) Distinct EEG effects related to neurofeedback training in children with ADHD: a randomized controlled trial. Int J Psychophysiol 74:149–157
36. Fuchs T, Birbaumer N, Lutzenberger W, Gruzelier JH, Kaiser J (2003) Neurofeedback treatment for attention-deficit/hyperactivity

disorder in children: a comparison with methylphenidate. Appl Psychophysiol Biofeedback 28(1):1–12
37. Monastra V, Monastra D, George S (2002) The effects of stimulant therapy, EEG-biofeedback and parenting style on the primary symptoms of attention-deficit/hyperactivity disorder. Appl Psychophysiol Biofeedback 27:231–249
38. Rossiter TR, La Vaque TJ (1995) A comparison of EEG biofeedback and psychostimulants in treating attention deficit/hyperactivity disorders. J Neurotherapy 1(1):48–59
39. Holtmann M, Stadler C, Leins U, Strehl U, Birbaumer N, Poustka F (2004) Neurofeedback in der Behandlung der Aufmerksamkeits-Hyperaktivitätsstörung (ADHS) im Kindes- und Jugendalter. Zeitschrift für Kinder- und Jugendpsychiatrie und Psychotherapie 32(3):187–200
40. Holtmann M, Stadler C (2006) Electroencephalographic biofeedback for the treatment of attention-deficit/hyperactivity disorder in childhood and adolescence. Expert Rev Neurother 6:533–540
41. Arns M, de Ridder S, Strehl U, Breteler M, Coenen A (2009) Efficacy of neurofeedback treatment in ADHD: the effects on inattention, impulsivity and hyperactivity: a meta-analysis. Clin EEG Neurosci 40:180–189
42. Gevensleben et al. (2009) Is neurofeedback an efficacious treatment for ADHD? A randomised controlled clinical trial. J Child Psychol Psychiatry. doi:10.1111/j.1469-7610.2008.02033.x
43. Gevensleben H, Holl B, Albrecht B, Schlamp D, Kratz O, Studer P, Rothenberger A, Moll GH, Heinrich H (2010) Neurofeedback training in children with ADHD: 6-month follow-up of a randomized controlled trial. J Eur Child Adolesc Psychiatry 19:715–724
44. Holtmann M, Grasmann D, Cionek-Szpak E et al (2009) Spezifische Wirksamkeit von Neurofeedback auf die Impulsivität bei ADHS. Kindheit und Entwicklung. 18(2):95–104
45. Oosterlaan J, Sergeant JA (1998) Effects of reward and response cost on response inhibition in AD/HD, disruptive, anxious and normal children. J Abnorm Child Psychol 26:161–174
46. Doehnert M, Brandeis D, Straub M, Steinhausen H-C, Drechsler R (2008) Slow cortical potential neurofeedback in attention deficit hyperactivity disorder: is there neurophysiological evidence for specific effects? J Neural Transm 115:1145–1456
47. Esser G, Blanz B, Geisel B, Laucht M (1989) Mannheimer Elterninterview, MEI, Beltz Test. GmbH, Weinheim
48. Doepfner M, Lehmkuhl G (2000) Diagnostik- System für psychische Störungen im Kindes- und Jugendalter (DISYPS- KJ) nach ICD-10 und DSM-IV, klinische Diagnostik- Elternurteil- Erzieher- und Lehrerurteil- Selbsturteil, Manual. Huber, Bern
49. Kratzmeier H, Horn R (1988) Standard progressive matrices (SPM) manual. Beltz Test Gesellschaft, Weinheim
50. Rasey HW, Lubar JF, McIntyre A, Zoffuto AC, Abbott PL (1996) EEG biofeedback for the enhancement of attentional processing in normal college students. J Neurotherapy 1(3):15–31
51. Lubar JF, Lubar JO (1999) Neurofeedback assessment and treatment for attention deficit/hyperactivity disorder. In: Evans JR, Abarbanel A (eds) Introduction to quantitative EEG and Neurofeedback. Academic Press Inc, San Diego, pp 103–143
52. Van Leeuwen TH, Steinhausen HC, Overtoom CC, Pascual-Marqui RD, van't Klooster B, Rothenburger A, Sergeant JA, Brandeis D (1998) The continuous performance test revisited with neuroelectric mapping: impaired orienting in children with attention deficits. Behav Brain Res 94:243–263
53. Knye M, Roth N, Westhus W, Heine A (2004) Continuous Performance Task (CPT)-manual. Hogrefe, Göttingen
54. Esser G, Wyschkon A, Ballaschk K (2008) Basisdiagnostik Umschriebener Entwicklungsstörungen im Grundschulalter. Hogrefe, Göttingen
55. Brickenkamp R (2002) Test d2, Aufmerksamkeits-Belastungs-Test, 9th edn. Hogrefe, Göttingen
56. Esser G, Geisel B (1978) Test zur Erfassung der Konzentrationsfähigkeit 8jähriger Kinder bp-Test. Kurzfassung
57. Klauer K (1993) Denktraining für Jugendliche. Ein Programm zur intellektuellen Förderung. Handanweisung. Hogrefe, Göttingen
58. Leins U (2004) Train your brain: neurofeedback für Kinder mit einer Aufmerksamkeits-/Hyperaktivitätsstörung (ADHS). Dissertation, Universität Tübingen
59. Monastra VJ, Lubar JF, Linden M (2001) The development of a quantitative electroencephalographic scanning process for attention deficit- hyperactivity disorder: reliability and validity studies. Neuro-psychology 15(1):136–144
60. Arnold LE (2001) Alternative treatments for adults with attention-deficit hyperactivity disorder (ADHD). Ann N Y Acad Sci 931:310–341
61. Lee SW (1991) Biofeedback as a treatment for childhood hyperactivity: a critical review of the literature. Psychol Report 68(1):163–192
62. Cobb DE, Evans JR (1981) The use of biofeedback techniques with school-aged children exhibiting behavioural and/or learning problems. J Abnorm Child Psychol 9:251–281
63. Omizo MM, Michael WB (1982) Biofeeback-induced relaxation training and impulsivity, attention task, and locus of control among hyperactive boys. J Learn Disabil 15:414–416
64. Heywood C, Beale I (2003) EEG biofeedback vs placebo treatment for attention-deficit/hyperactivity disorder: a pilot study. J Atten Disord 7(1):43–55
65. Logemann HNA, Lansbergen MM, Van Os TWDP, Böcker KBE, Kenemans JL (2010) The effectiveness of EEG-feedback on attention, impulsivity and EEG: a sham feedback controlled study. Neurosci Lett 479:49–53
66. Baydala L, Wikman E (2001) The efficacy of neurofeedback in the management of children with attention deficit/hyperactivity disorder. Paediatr Child Health 6(7):451–455
67. Braud LW, Lupin MN, Braud WG (1975) The use of electromyographic biofeedback in the control of hyperactivity. J Learn Disabil 8:21–26
68. G-Power http://www.psycho.uni-duesseldorf.de/aap/projects/gpower.de