

Neurofeedback Treatment for Attention-Deficit/ Hyperactivity Disorder in Children: A Comparison With Methylphenidate

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Clinical trials have suggested that neurofeedback may be efficient in treating attention-deficit/hyperactivity disorder (ADHD). We compared the effects of a 3-month electroencephalographic feedback program providing reinforcement contingent on the production of cortical sensorimotor rhythm (12–15 Hz) and beta1 activity (15–18 Hz) with stimulant medication. Participants were N = 34 children aged 8–12 years, 22 of which were assigned to the neurofeedback group and 12 to the methylphenidate group according to their parents' preference. Both neurofeedback and methylphenidate were associated with improvements on all subscales of the Test of Variables of Attention, and on the speed and accuracy measures of the d2 Attention Endurance Test. Furthermore, behaviors related to the disorder were rated as significantly reduced in both groups by both teachers and parents on the IOWA-Connors Behavior Rating Scale. These findings suggest that neurofeedback was efficient in improving some of the behavioral concomitants of ADHD in children whose parents favored a nonpharmacological treatment.

KEY WORDS: attention-deficit/hyperactivity disorder (ADHD); neurofeedback; electroencephalogram; methylphenidate; children.

INTRODUCTION

Attention-deficit/hyperactivity disorder (ADHD) is a behavioral disorder characterized by inattentiveness, impulsivity, and hyperactivity, affecting 3–5% of school-aged children (The MTA Cooperative Group, 1999). Current etiological theories have linked ADHD to abnormalities in dopaminergic and, possibly, noradrenergic cortico-subcortical networks relevant for executive functions and the regulation of behavioral responses. Event-related potential studies have demonstrated that children with ADHD compared with healthy

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controls have smaller amplitudes and longer latencies for a variety of components including N1, N2, mismatch negativity, readiness potential, and P3b that may indicate attentional and information processing deficits (Jonkman et al., 1997b; Kemner et al., 1996; Klorman, 1991; Loiseau, Stamm, Maitinsky, & Whipple, 1980; Novak, Solanto, & Abikoff, 1995; Satterfield, Schell, & Nicholas, 1994; Schlottke, 1988; Stamm et al., 1982; Steger, Imhof, Steinhausen, & Brandeis, 2000). Investigations of spectral activity in the electroencephalogram (EEG) have consistently reported an abnormal abundance of slow frequencies such as theta (4–7 Hz), especially over frontal areas, and reduced power in faster bands such as alpha and beta (8–12 and 12–22 Hz, respectively; Chabot, Merkin, Wood, Davenport, & Serfontein, 1996; Clarke, Barry, McCarthy, & Selikowitz, 1998; Lazzaro et al., 1999; Mann, Lubar, Zimmerman, Miller, & Muenchen, 1992; Monastra et al., 1999). Consistent with these findings, both functional and volumetric brain imaging have indicated a dysfunction of fronto-striatal systems in ADHD (Castellanos et al., 1996; Gustafsson, Thernlund, Ryding, Rosen, & Cederblad, 2000; Rubia et al., 1999; Swanson, Castellanos, Murias, LaHoste, & Kennedy, 1998) that may account for deficits of higher order motor control, arousal, behavioral inhibition, and attention (Hale, Hariri, & McCracken, 2000; Pliszka, Liotti, & Woldorff, 2000). EEG, brain morphometric, neurochemical, and molecular genetic findings taken together suggest that ADHD may be related to aberrant early brain development (Nopoulos et al., 2000; Zametkin & Liotta, 1998).

Neurofeedback training to increase the power of sensorimotor rhythm (SMR, 12–15 Hz) and low beta activity (15–18 Hz) has been reported to improve ADHD (J. F. Lubar & Lubar, 1999; J. F. Lubar, Swartwood, Swartwood, & O'Donnell, 1995; J. O. Lubar & Lubar, 1984; Nash, 2000; Rossiter & La Vaque, 1995; L. Thompson & Thompson, 1998). Improvements in intellectual functioning and attentive behaviors have been interpreted as a result of the attentional enhancement related to EEG neurofeedback training. The purpose of this study was to compare the efficacy of SMR/low beta neurofeedback with standard pharmacotherapy for children suffering from ADHD. Stimulants like methylphenidate have been demonstrated to improve abnormal behaviors of ADHD in a multitude of well-controlled studies in large samples and across long periods of time (McBride, 1988; The MTA Cooperative Group, 1999; Spencer et al., 1996). Moreover, methylphenidate has been found to temporarily remediate most of the ADHD-related deficits as reflected in EEG correlates of cognitive function (Jonkman et al., 1997a; Klorman, 1991; Klorman et al., 1990; Novak et al., 1995; Taylor, Voros, Logan, & Malone, 1993). The present investigation assessed performance on tests of attentional functions, behavioral indices of ADHD as rated by parents and teachers, and measures of intelligence in two groups of children with ADHD both before and after 3-month treatment periods with either neurofeedback or methylphenidate.

METHODS

Participants

Participants were $N = 34$ children selected among new admissions to a pediatric outpatient clinic between 1997 and 1999. All children belonged to rural southern German families of heterogeneous socioeconomic status. Inclusion criteria were a) a primary diagnosis of ADHD based on semistructured interviews with parents and children using *DSM-IV* criteria for simple activity and attention disorders of the inattentive, hyperactive–impulsive,

and combined subtypes (American Psychiatric Association, 1994); b) a Wechsler intelligence quotient >80; and c) at least one substandard score (<85) on the Test of Variables of Attention (TOVA; Greenberg, 1987). Diagnoses were made independently by two experienced clinicians: a child neurologist or pediatrician and a psychologist specialized in child and adolescent clinical psychology. None of the patients had received any kind of treatment (medication or other) for ADHD prior to their inclusion in the study.

Assignment to the treatment groups was based on the parents' informed choice. About twice as many parents preferred neurofeedback over stimulants. Thus 22 participants (1 female, 21 males, $M = 9.8$ years, $SD = 1.3$ years) were assigned to a neurofeedback training program. They did not receive any psychoactive medication during the entire study period. The methylphenidate group was composed of 12 children (all male, $M = 9.6$ years, $SD = 1.2$ years) who were typically administered three times 10 mg methylphenidate (Ritalin[®]) on school days only during the entire treatment period. Dosing decisions were made after the initial examination. Individual dosages were adjusted during the treatment period and varied between 10 and 60 mg/day. Both groups were treated for 12 weeks. All participants of the neurofeedback group completed the treatment schedule, whereas one participant of the methylphenidate group dropped out because of excessive side effects (motor and verbal tics). This left a total of 33 participants for analysis.

Neurofeedback Training

Electroencephalographic (EEG) neurofeedback training was conducted over a period of 12 weeks with three training sessions per week conducted on weekday afternoons. Training was administered to all clients by the same therapist (T. Fuchs) using the Neurocybernetics EEG Biofeedback System (EEG Spectrum, Encino, CA, USA). The procedure followed the paradigm described by J. F. Lubar et al. (1995). Participants were seated in a comfortable armchair in a quiet room. EEG was recorded from one electrode at position C3 or C4 (International 10-20 System; Jasper, 1958) referenced to linked earlobes (mastoid ground electrode, sampling rate: 128 Hz). The ongoing EEG was band-pass filtered in the following four frequency ranges: theta (4–7 Hz), sensorimotor rhythm (SMR, 12–15 Hz), beta1 (15–18 Hz), and beta2 (22–30 Hz). C4 and SMR were used in the children of the hyperactive–impulsive subtype, and C3 and beta1 were used in the children of the primarily inattentive subtypes. Children of the combined subtype were treated during half of the sessions like hyperactive–impulsive and during the other half like the inattentive subtype. The rationale behind this distinction was that SMR reflects inhibition of the thalamo-cortical loop and that hyperactivity may be related to a right-hemispheric overresponsiveness (Sterman, Wyrwicka, & Howe, 1969). Attentional deficits (decreases in vigilance) on the other hand may be reflected by predominantly left-hemispheric slow theta activity and a relative absence of beta activity. The aim of neurofeedback training was to increase the power in the SMR or beta1 bands (“reward bands”) and simultaneously to decrease the power in the theta and beta2 bands (“inhibit bands”). Information about the power in each of these frequency bands was monitored by the therapist throughout the session and fed back audiovisually to the clients via a personal computer.

Neurofeedback training consisted of 30–60 min of visual and auditory feedback per session, interrupted for short breaks if required by the participant. At the beginning of a training session, threshold levels were determined for each participant from 2-min baseline

amplitude measures of activity in the four frequency bands. These levels ranged between 0.5 and 1 μV below or above the baseline values for inhibit or reward bands, respectively. Reward criteria were set so that reward thresholds had to be exceeded in 60% of sampled events in a 500-ms period, and spectral amplitudes had to range below inhibit thresholds in 30% of sampled events to receive a reward. When participants consistently achieved the defined goals (e.g. remained above the reward threshold for 70% of events for two consecutive trials), their thresholds were made more difficult. Visual feedback was provided by a variety of means that translate the EEG amplitude in the reward and inhibit bands into the brightness, size, and/or velocity of objects on a computer monitor. An example would be the pacman-type game “mazes” in which an icon moved through a maze eating dots. The power in the reward bands (12–15 or 15–18 Hz) determined the speed and brightness of the icon: the higher the power, the faster and brighter the icon. When the reward criterion was attained, scores were indicated by an audiovisual signal (a beeping noise and a counter increasing its value). Conversely, when the power in the inhibit bands (4–7 or 22–30 Hz) exceeded its limit, the icon stopped moving and turned black. When the icon reached the end of the maze, a bar chart appeared showing the performance and there was a short break before the next maze started.

Test Materials

Both groups were examined both prior and subsequent to the 12-week treatment period. The following tests were administered.

The TOVA (Greenberg, 1987) is a computerized visual continuous performance task. The test stimulus is a square containing a small rectangle near the top or bottom edge. The square with the small rectangle at the top is the target that participants are instructed to respond to by pressing a hand-held microswitch. They are not to respond to the square with the small rectangle at the bottom. The stimuli are presented for 100 ms each with 2-s interstimulus onset intervals. The duration of the test is 22.5 min. Targets are present on 22.5% of the trials during the first half of the test and 77.5% of the trials during the second half. Standard scores ($M = 100$, $SD = 15$) were calculated based on single-year age norms for the following subscales: impulsivity (based on the number of commission errors, i.e. responses to nontargets), inattention (based on the number of omission errors, i.e. missed targets), response time (based in the mean response latency), and variability (based on the variance of response times). Higher scores indicate better/more stable performance. The TOVA is sensitive to attentional deficits and has been found to discriminate well between children with ADHD and controls (Forbes, 1998; Wada, Yamashita, Matsuishi, Ohtani, & Kato, 2000).

The Attention Endurance Test (d2; Brickenkamp, 1994) is a paper-and-pencil task requiring the participants to identify targets (the letter d with two apostrophe marks that may be located either both above, both below, or one above and one below the d) and to ignore distracters (d's with one, three, or four marks or p's with one or two marks). The stimuli are arranged in 14 rows containing 47 letters each. Each row may be scanned for 20 s after which the experimenter tells the participant to move to the subsequent row. Percent rank scores are calculated on the basis of age norms for the following scales: speed (based on the number of correct responses), accuracy (based on the number of errors), total (based on the number of correct responses minus errors), and variability (based on the difference

in correct responses between the row with the highest and the row with the lowest number of correct responses). Higher scores indicate better/more stable performance.

A German version of the IOWA-Connors Behavior Rating Scale (Atkins & Milich, 1987) was completed by a teacher and by both parents of each of the participants both prior to the onset and after the completion of the training period. Teachers were not informed about the type of treatment the children received, whereas for parents blinding was obviously not possible. The scale comprises ten 4-point items designed to measure inattentiveness, hyperactivity, and aggression in the children's everyday behavior. Scores range from 0 to 30, with high scores indicating a high level of ADHD-like symptoms. Although the scale's authors reported a high validity of the scale with 85% correct classification of children with ADHD (Connors, Sitarenios, Parker, & Epstein, 1998), there have been contradictory findings (Ullmann, Sleanor, & Sprague, 1985).

In addition to the ADHD-related measures above, the Wechsler Intelligence Scale for Children-Revised (Hamburg-Wechsler Intelligenztest für Kinder, HAWIK; Tewes, 1983; Wechsler, 1974) was administered. At the time of data collection, this was the most recent German version of this measure of general intelligence. Intelligence quotients ($M = 100$, $SD = 15$) are calculated on the basis of age norms both for the complete test and for the performance and verbal subscales separately.

Statistical Analysis

Treatment (pre- vs. posttreatment) and Group (neurofeedback vs. methylphenidate) were entered as within- and between-participant factors, respectively, in separate repeated-measures analyses of variance (ANOVA) for each dependent variable. The nature of the main effects or interactions was further explored with post hoc t tests where a Bonferroni-correction of the alpha level was used for multiple tests. Effect sizes were calculated as Cohen's d (Cohen, 1988), that is, as the difference of group means divided by the root mean square of the two standard deviations. Power values were calculated for an alpha level of 0.05 for the observed mean differences and standard deviations for each pre- versus posttreatment comparison. In addition, equivalence analysis (Rogers, Howard, & Vessey, 1993) was conducted to test whether the treatment-related changes in both groups could be regarded as statistically equivalent (the equivalence interval was chosen as $\pm 20\%$ of the mean change in the medication group).

RESULTS

TOVA

The effects of both types of treatment on the four TOVA subscales are shown in Fig. 1. There were no pretreatment differences between both groups on any of the TOVA variables. For impulsivity, a main effect of Treatment, $F(1, 31) = 32.9$, $p < .001$, was identified. There were no effects of Group and no Treatment \times Group interaction. Highly significant improvements on the impulsivity scale were found for both neurofeedback, $t(21) = 5.0$, $p < .001$, Cohen's $d = 1.21$, power = 1.0, and methylphenidate, $t(10) = 3.4$, $p = .007$, Cohen's $d = 1.03$, power = 0.93, treatments. Similarly, there was a main effect

Test of Variables of Attention (TOVA)

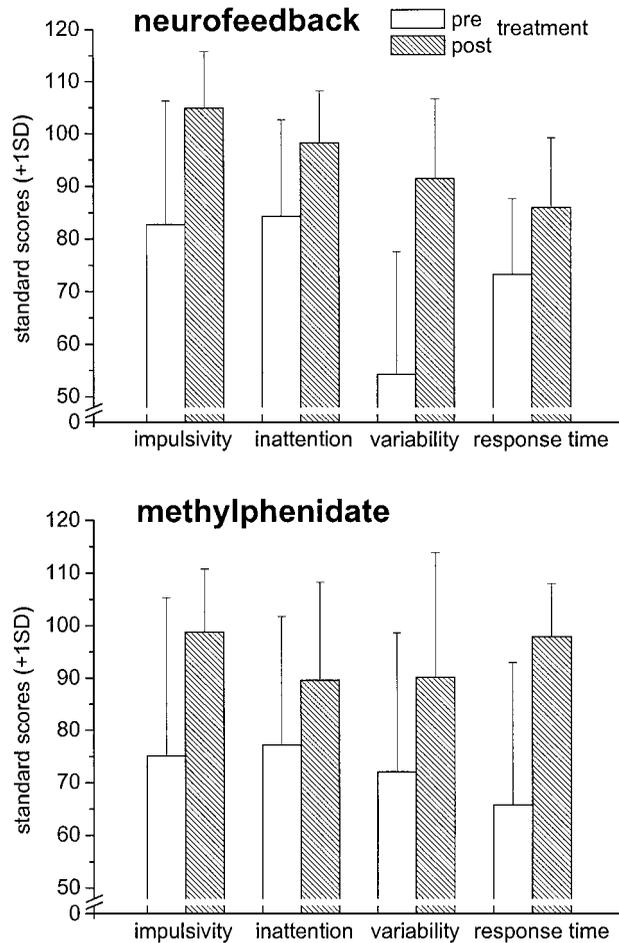


Fig. 1. Pre- and posttreatment standard scores (means + 1SD) of the TOVA subscales for the neurofeedback ($N = 22$) and methylphenidate ($N = 11$) groups.

of Treatment for inattention, $F(1, 31) = 29.4$, $p < .001$, but no effect of Group or interaction. Inattention was reduced both by neurofeedback, $t(21) = 5.0$, $p < .001$, Cohen's $d = 0.95$, power = 0.99, and by methylphenidate, $t(10) = 3.1$, $p = .01$, Cohen's $d = 0.57$, power = 0.71.

For response time variability, we observed both a main effect of Treatment, $F(1, 31) = 41.4$, $p < .001$, and an interaction Treatment \times Group, $F(1, 31) = 4.9$, $p = .034$, but no main effect of Group. Variability was greatly improved by both neurofeedback, $t(21) = 6.7$, $p < .001$, Cohen's $d = 1.45$, power = 1.0, and methylphenidate, $t(10) = 3.6$, $p = .005$, Cohen's $d = 0.72$, power = 1.0. For the interaction, post hoc tests did not yield significant effects at the Bonferroni-corrected alpha level. For response time, both a main effect of

Treatment, $F(1, 31) = 42.6$, $p < .001$, and an interaction Treatment \times Group, $F(1, 31) = 8.1$, $p = .008$, but no main effect of Group were observed. Although response time scores were improved by both neurofeedback, $t(21) = 3.6$, $p = .002$, Cohen's $d = 0.92$, power = 0.99, and methylphenidate, $t(10) = 4.7$, $p = .001$, Cohen's $d = 1.57$, power = 1.0, this effect was more pronounced in the methylphenidate group (mean difference = 32.1, $SD = 22.6$) than in the neurofeedback group (mean difference = 12.6, $SD = 16.3$), $t(31) = 2.8$, $p = .008$. No equivalence test reached significance for any of the four TOVA variables.

d2 Attention Endurance Test

The d2 was not administered to three participants in the neurofeedback group because there are no age norms for children under 9 years of age. The means and standard deviations for the four subscales pre- and posttreatment including t test results for the treatment effects are presented in Table I. There were no pretreatment differences between both groups on any of the d2 measures. Significant main effects of Treatment were found for both speed, $F(1, 28) = 13.8$, $p = .001$, accuracy, $F(1, 28) = 4.8$, $p = .037$, and the combined total score, $F(1, 28) = 15.7$, $p < .001$, indicating comparable effects of both neurofeedback and methylphenidate. There were no main effects of Group or interactions for these three subscales. There were no significant main effects or interactions for the variability subscale. Equivalence tests were nonsignificant.

IOWA-Connors Behavior Rating Scale

Ratings given by mothers and fathers on the IOWA-Connors Behavior Rating Scale were highly positively correlated both pre- and posttreatment ($r = 0.81$ and 0.82 , respectively, both $p < .001$) and were therefore collapsed into a combined "parents" score. Ratings by teachers and parents were also highly positively correlated pretreatment (teacher–mother:

Table I. Pre- and Posttreatment Mean Percent Rank Scores and Standard Deviations for the Subscales of the d2 Attention Endurance Test (Brickenkamp, 1994) for the Neurofeedback ($N = 19$) and Methylphenidate ($N = 11$) Groups and t Test Results for the Treatment Effects

	Pretreatment		Posttreatment		t test	p	ES	Power
	M	SD	M	SD				
Speed								
NF	38.3	(30.3)	55.1	(25.9)	$t(18) = 3.2$.005	0.60	0.80
MPH	42.8	(32.4)	58.6	(21.8)	$t(10) = 2.2$.054	0.57	0.53
Accuracy								
NF	31.5	(23.6)	45.3	(24.5)	$t(18) = 1.9$.068	0.57	0.77
MPH	32.0	(18.8)	43.7	(23.1)	$t(10) = 1.3$	<i>ns</i>	0.56	0.51
Total								
NF	36.4	(27.1)	55.8	(22.2)	$t(18) = 3.6$.002	0.78	0.94
MPH	37.8	(12.5)	50.6	(15.7)	$t(10) = 2.4$.035	0.90	0.87
Variability								
NF	46.8	(31.3)	50.3	(25.5)	$t(18) = 0.4$	<i>ns</i>	0.12	0.12
MPH	42.7	(22.6)	48.7	(18.3)	$t(10) = 0.6$	<i>ns</i>	0.29	0.21

Note. SD : standard deviation; ES : effect size (Cohen's d); NF : neurofeedback; MPH : methylphenidate; *ns*: not significant.

Conners Behavior Rating Scale

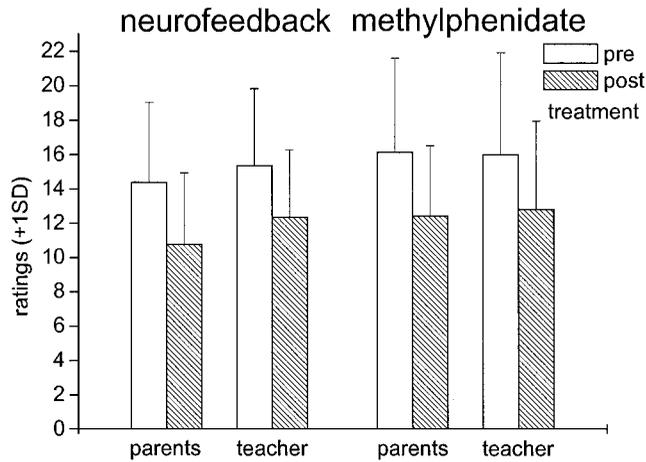


Fig. 2. Pre- and posttreatment teachers' and parents' ratings (means + 1SD) on the IOWA-Conners Behavior Rating Scale for the neurofeedback ($N = 22$) and methylphenidate ($N = 11$) groups.

$r = 0.72$, teacher–father: $r = 0.70$, both $p < .001$) and moderately positively correlated post-treatment (teacher–mother: $r = 0.37$, $p = .037$, teacher–father: $r = 0.39$, $p = .26$). The effects of both types of treatment on the IOWA-Conners Behavior Rating Scale for parents and teachers are depicted in Fig. 2.

Both groups did not differ on the IOWA-Conners Scale ratings pretreatment. There were main effects of Treatment for both the parents' and teachers' ratings, $F(1, 31) = 27.8$, $p < .001$, and $F(1, 31) = 19.9$, $p < .001$, respectively, but no main effects of Group or Treatment \times Group interactions were found. Both treatments resulted in improved parents' ratings, neurofeedback: $t(21) = 4.5$, $p < .001$, Cohen's $d = 0.82$, power = 0.98; methylphenidate: $t(10) = 3.3$, $p = .007$, Cohen's $d = 0.76$, power = 0.75, and teachers' ratings, neurofeedback: $t(21) = 3.5$, $p = .002$, Cohen's $d = 0.71$, power = 0.94; methylphenidate: $t(10) = 3.5$, $p = .005$, Cohen's $d = 0.58$, power = 0.53. Equivalence tests were nonsignificant.

Wechsler Intelligence Scale for Children-Revised

The means and standard deviations for the three intelligence scores pre- and posttreatment including t test results for the treatment effects are presented in Table II. There were no pretreatment differences between both groups in any of the intelligence scores. Main effects of Treatment were identified for the full scale intelligence quotient, $F(1, 31) = 11.2$, $p < .001$, indicating an improvement of this intelligence score by both neurofeedback and methylphenidate. Analysis of the subscores demonstrated that this effect was accounted for mainly by the performance score, $F(1, 31) = 32.8$, $p < .001$, but not by the verbal score where no effect of Treatment was observed. There were no further main effects or interactions for any of the three scores. Equivalence tests were nonsignificant.

Table II. Pre- and Posttreatment Mean Intelligence Quotients and Standard Deviations for the Full Scale and the Performance and Verbal Subscales of the Wechsler Intelligence Scale for Children-Revised (Tewes, 1983; Wechsler, 1974) the Neurofeedback ($N = 22$) and Methylphenidate ($N = 11$) Groups and t Test Results for the Treatment Effects

	Pretreatment		Posttreatment		t test	p	ES	Power
	M	SD	M	SD				
Full scale								
NF	101.0	12.2	104.7	11.6	$t(21) = 2.9$.009	0.31	0.38
MPH	99.8	10.94	102.7	10.2	$t(10) = 2.7$.022	0.26	0.18
Performance								
NF	99.1	12.3	104.2	12.5	$t(21) = 4.6$	<.001	0.41	0.58
MPH	97.1	11.9	103.2	11.5	$t(10) = 3.6$.004	0.52	0.47
Verbal								
NF	102.2	11.4	104.2	10.4	$t(21) = 1.4$	<i>ns</i>	0.18	0.20
MPH	102.0	10.9	101.7	9.8	$t(10) = 0.3$	<i>ns</i>	0.03	0.06

Note. SD : standard deviation; ES: effect size (Cohen's d); NF: neurofeedback; MPH: methylphenidate; *ns*: not significant.

DISCUSSION

Both a 3-month neurofeedback program contingent on the suppression of theta/high beta and on the enhancement of SMR/low beta activity in EEG and pharmacotherapy with methylphenidate were successful in remediating ADHD symptomatology in children. Significant improvements were observed for all four subscales of the TOVA, providing support for the efficacy of both treatments, considering the high sensitivity of this test for attentional deficits (Forbes, 1998; Wada et al., 2000) and its low susceptibility to practice effects (Greenberg, 1987). Also, larger effect sizes and power values were obtained for the TOVA than for any of the other measures. Both groups showed improvements on the d2 Attention Endurance Test accuracy and speed scores and on the composite total score, demonstrating that the children were able to work on a larger number of items while making fewer mistakes after treatment. Both interventions also led to improvements on the IOWA-Connors Behavior Rating Scale. Both parents and teachers rated the occurrence of ADHD-related behaviors as significantly reduced posttreatment. For d2 and Connors scores, moderate effect sizes and power values were obtained. In contrast, the improvements on the intelligence scales were rather small compared to other studies (Linden, Habib, & Radojevic, 1996; J. F. Lubar et al., 1995; L. Thompson & Thompson, 1998). It is likely that the observed changes were attributable to practice effects. Although both treatments led to significant improvements in many variables, equivalence tests remained nonsignificant for all of the dependent measures. Proving the equivalence of both treatments would probably require a much larger sample.

Previous research into SMR/beta neurofeedback has suffered from lack of random assignment, standardized measures of target symptoms, assessment of EEG changes, control groups, and sample sizes (Birbaumer & Flor, 1999; Linden et al., 1996; Lohr, Meunier, Parker, & Kline, 2001). The present study overcame some of these problems by including a comparison group and by applying both objective performance tests and external ratings by teachers and parents. We are aware of the problems associated with not randomizing group membership, such as possible differences in treatment motivation and effects of expectancies, especially on parents' ratings. However, it would not have been feasible

to administer either treatment without the parents' consent. In our experience, giving the parents the option to choose resulted in high levels of motivation and compliance in both groups. Using an untreated, waiting list control group (Linden et al., 1996) would have been unethical considering that a standard medical treatment exists for ADHD (La Vaque & Rossiter, 2001), and placebo or sham neurofeedback is impossible because it is soon recognized by both therapists and patients (Kotchoubey et al., 2001). Therefore the present comparison group received the standard medical treatment with methylphenidate whose therapeutic efficacy has been proven in numerous well-controlled investigations (McBride, 1988; The MTA Cooperative Group, 1999; Spencer et al., 1996). To compare possible differences in the placebo effects between treatments, future studies should include measures of treatment satisfaction (Kotchoubey et al., 2001).

Unfortunately the actual power changes in EEG frequency bands as a result of neurofeedback were not monitored and analyzed. However, a study comparing neurofeedback "responders" and "nonresponders" reported that about two thirds of the ADHD patients significantly reduced their theta activity as a result of training over approximately 40 sessions, and improvement on the TOVA was more pronounced in participants with significant EEG changes than in those without (J. F. Lubar et al., 1995). Supposing on the basis of this evidence that a fraction of patients did not achieve control over their spectral EEG activity, identifying and excluding these participants might have further enhanced the treatment effects in this study. In clinical settings, individuals who do not respond to one form of treatment could be assigned to another, more appropriate treatment. This applies not only to neurofeedback but also to methylphenidate, where response rates of below 70% have been reported (McBride, 1988). Regrettably, long-term follow-ups were not possible because children returned to mostly distant rural homes. However, the average or above-average scores in all instruments after treatment argue against placebo as the main factor. In summary, neurofeedback was efficient in improving some of the behavioral concomitants of ADHD in children whose parents have a positive attitude toward a nonpharmacological treatment. The findings are promising and may stimulate further research into the efficacy of neurofeedback methods in ADHD. Future studies should use larger samples and assess symptomatology at long-term follow-ups to demonstrate the clinical significance of these results.

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REFERENCES

- American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders (DSM-IV)* (4th ed.). Washington, DC: Author.
- Atkins, M., & Milich, R. (1987). IOWA-Conners Teacher Rating Scale. In M. Hersen & A. Bellack (Eds.), *Dictionary of behavioral assessment techniques* (pp. 273–275). New York: Pergamon.
- Birbaumer, N., & Flor, H. (1999). Applied psychophysiology and learned physiological regulation. *Applied Psychophysiology and Biofeedback*, 24, 35–37.

- Brickenkamp, R. (1994). *Test d2, Aufmerksamkeits-Belastungs-Test* (8th ed.). Göttingen: Hogrefe.
- Castellanos, F. X., Giedd, J. N., Marsh, W. L., Hamburger, S. D., Vaituzis, A. C., Dickstein, D. P., et al. (1996). Quantitative brain magnetic resonance imaging in attention-deficit hyperactivity disorder. *Archives of General Psychiatry*, *53*, 607–616.
- Chabot, R. J., Merkin, H., Wood, L. M., Davenport, T. L., & Serfontein, G. (1996). Sensitivity and specificity of QEEG in children with attention deficit or specific developmental learning disorders. *Clinical Electroencephalography*, *27*, 26–34.
- Clarke, A. R., Barry, R. J., McCarthy, R., & Selikowitz, M. (1998). EEG analysis in Attention-Deficit/Hyperactivity Disorder: A comparative study of two subtypes. *Psychiatry Research*, *81*, 19–29.
- Cohen, J. (1988). *Statistical power analysis for the behavioral sciences* (2nd ed.). Hillsdale, NJ: Erlbaum.
- Conners, C. K., Sitarenios, G., Parker, J. D., & Epstein, J. N. (1998). Revision and restandardization of the Conners Teacher Rating Scale (CTRS-R): Factor structure, reliability, and criterion validity. *Journal of Abnormal Child Psychology*, *26*, 279–291.
- Forbes, G. B. (1998). Clinical utility of the Test of Variables of Attention (TOVA) in the diagnosis of attention-deficit/hyperactivity disorder. *Journal of Clinical Psychology*, *54*, 461–476.
- Fuchs, T. (1999). *Aufmerksamkeit und Neurofeedback*. Regensburg: Roderer.
- Greenberg, L. M. (1987). An objective measure of methylphenidate response: Clinical use of the MCA. *Psychopharmacology Bulletin*, *23*, 279–282.
- Gustafsson, P., Thernlund, G., Ryding, E., Rosen, I., & Cederblad, M. (2000). Associations between cerebral blood-flow measured by single photon emission computed tomography (SPECT), electro-encephalogram (EEG), behaviour symptoms, cognition and neurological soft signs in children with attention-deficit hyperactivity disorder (ADHD). *Acta Paediatrica*, *89*, 830–835.
- Hale, T. S., Hariri, A. R., & McCracken, J. T. (2000). Attention-deficit/hyperactivity disorder: Perspectives from neuroimaging. *Mental Retardation and Developmental Disability Research Review*, *6*, 214–219.
- Jasper, H. H. (1958). Report of the committee on methods of clinical examination in EEG. Appendix: The ten twenty electrode system of the international federation. *Electroencephalography and Clinical Neurophysiology*, *10*, 371–375.
- Jonkman, L. M., Kemner, C., Verbaten, M. N., Koelega, H. S., Camfferman, G., van der Gaag, R. J., et al. (1997a). Effects of methylphenidate on event-related potentials and performance of attention-deficit hyperactivity disorder children in auditory and visual selective attention tasks. *Biological Psychiatry*, *41*, 690–702.
- Jonkman, L. M., Kemner, C., Verbaten, M. N., Koelega, H. S., Camfferman, G., van der Gaag, R. J., et al. (1997b). Event-related potentials and performance of attention-deficit hyperactivity disorder: Children and normal controls in auditory and visual selective attention tasks. *Biological Psychiatry*, *41*, 595–611.
- Kemner, C., Verbaten, M. N., Koelega, H. S., Buitelaar, J. K., van der Gaag, R. J., Camfferman, G., et al. (1996). Event-related brain potentials in children with attention-deficit and hyperactivity disorder: Effects of stimulus deviancy and task relevance in the visual and auditory modality. *Biological Psychiatry*, *40*, 522–534.
- Klorman, R. (1991). Cognitive event-related potentials in attention deficit disorder. *Journal of Learning Disabilities*, *24*, 130–140.
- Klorman, R., Brumaghim, J. T., Salzman, L. F., Strauss, J., Borgstedt, A. D., McBride, M. C., et al. (1990). Effects of methylphenidate on processing negativities in patients with attention-deficit hyperactivity disorder. *Psychophysiology*, *27*, 328–337.
- Kotchoubey, B., Strehl, U., Uhlmann, C., Holzapfel, S., König, M., Fröscher, W., et al. (2001). Modification of slow cortical potentials in patients with refractory epilepsy: A controlled outcome study. *Epilepsia*, *42*, 406–416.
- La Vaque, T. J., & Rossiter, T. (2001). The ethical use of placebo controls in clinical research: The Declaration of Helsinki. *Applied Psychophysiology and Biofeedback*, *26*, 23–37.
- Lazzaro, I., Gordon, E., Li, W., Lim, C. L., Plahn, M., Whitmont, S., et al. (1999). Simultaneous EEG and EDA measures in adolescent attention deficit hyperactivity disorder. *International Journal of Psychophysiology*, *34*, 123–134.
- Linden, M., Habib, T., & Radojevic, V. (1996). A controlled study of the effects of EEG biofeedback on cognition and behavior of children with attention deficit disorder and learning disabilities. *Biofeedback and Self-Regulation*, *21*, 35–49.
- Lohr, J. M., Meunier, S. A., Parker, L. M., & Kline, J. P. (2001). Neurotherapy does not qualify as an empirically supported behavioral treatment for psychological disorders. *The Behavior Therapist*, *24*, 97–104.
- Loiselle, D. L., Stamm, J. S., Maitinsky, S., & Whipple, S. C. (1980). Evoked potential and behavioral signs of attentive dysfunctions in hyperactive boys. *Psychophysiology*, *17*, 193–201.
- Lubar, J. F., & Lubar, J. O. (1999). Neurofeedback assessment and treatment for attention deficit/hyperactivity disorders. In J. R. Evans & A. Abarbanel (Eds.), *Introduction to quantitative EEG and neurofeedback* (pp. 103–143). San Diego: Academic Press.
- Lubar, J. F., Swartwood, M. O., Swartwood, J. N., & O'Donnell, P. H. (1995). Evaluation of the effectiveness of EEG neurofeedback training for ADHD in a clinical setting as measured by changes in T.O.V.A. scores, behavioral ratings, and WISC-R performance. *Biofeedback and Self-Regulation*, *20*, 83–99.

- Lubar, J. O., & Lubar, J. F. (1984). Electroencephalographic biofeedback of SMR and beta for treatment of attention deficit disorders in a clinical setting. *Biofeedback and Self-Regulation*, *9*, 1–23.
- Mann, C. A., Lubar, J. F., Zimmerman, A. W., Miller, C. A., & Muenchen, R. A. (1992). Quantitative analysis of EEG in boys with attention-deficit-hyperactivity disorder: Controlled study with clinical implications. *Pediatric Neurology*, *8*, 30–36.
- The MTA Cooperative Group. (1999). A 14-month randomized clinical trial of treatment strategies for attention-deficit/hyperactivity disorder. *Archives of General Psychiatry*, *56*, 1073–1086.
- McBride, M. C. (1988). An individual double-blind crossover trial for assessing methylphenidate response in children with attention deficit disorder. *Journal of Pediatrics*, *113*, 137–145.
- Monastero, V. J., Lubar, J. F., Linden, M., VanDeusen, P., Green, G., Wing, W., et al. (1999). Assessing attention deficit hyperactivity disorder via quantitative electroencephalography: An initial validation study. *Neuropsychology*, *13*, 424–433.
- Nash, J. K. (2000). Treatment of attention deficit hyperactivity disorder with neurotherapy. *Clinical Electroencephalography*, *31*, 30–37.
- Nopoulos, P., Berg, S., Castellanos, F. X., Delgado, A., Andreasen, N. C., & Rapoport, J. L. (2000). Developmental brain anomalies in children with attention-deficit hyperactivity disorder. *Journal of Child Neurology*, *15*, 102–108.
- Novak, G. P., Solanto, M., & Abikoff, H. (1995). Spatial orienting and focused attention in attention deficit hyperactivity disorder. *Psychophysiology*, *32*, 546–559.
- Pliszka, S. R., Liotti, M., & Woldorff, M. G. (2000). Inhibitory control in children with attention-deficit/hyperactivity disorder: Event-related potentials identify the processing component and timing of an impaired right-frontal response-inhibition mechanism. *Biol Psychiatry*, *48*, 238–246.
- Rogers, J. L., Howard, K. I., & Vessey, J. T. (1993). Using significance tests to evaluate equivalence between two experimental groups. *Psychological Bulletin*, *113*, 553–565.
- Rossiter, T. R., & La Vaque, T. J. (1995). A comparison of EEG biofeedback and psychostimulants in treating attention deficit hyperactivity disorders. *Journal of Neurotherapy*, *1*, 48–59.
- Rubia, K., Overmeyer, S., Taylor, E., Brammer, M., Williams, S. C., Simmons, A., et al. (1999). Hypofrontality in attention deficit hyperactivity disorder during higher-order motor control: A study with functional MRI. *American Journal of Psychiatry*, *156*, 891–896.
- Satterfield, J. H., Schell, A. M., & Nicholas, T. (1994). Preferential neural processing of attended stimuli in attention-deficit hyperactivity disorder and normal boys. *Psychophysiology*, *31*, 1–10.
- Schlotzke, P. F. (1988). Zwischen “Zappelphilipp” und “Hans Guck-in-die-Luft”: Kinder mit Aufmerksamkeitsstörungen. *Acta Paedopsychiatrica*, *51*, 209–219.
- Spencer, T., Biederman, J., Wilens, T., Harding, M., O'Donnell, D., & Griffin, S. (1996). Pharmacotherapy of attention-deficit hyperactivity disorder across the life cycle. *Journal of the American Academy of Child and Adolescent Psychiatry*, *35*, 409–432.
- Stamm, J., Birbaumer, N., Lutzenberger, W., Elbert, T., Rockstroh, B., & Schlotzke, P. F. (1982). Event-related potentials during a continuous performance test vary with attentive capacities. In A. Rothenberger (Ed.), *Event-related potentials in children. Basic concepts and clinical application* (pp. 273–294). Amsterdam: Elsevier.
- Steger, J., Imhof, K., Steinhausen, H., & Brandeis, D. (2000). Brain mapping of bilateral interactions in attention deficit hyperactivity disorder and control boys. *Clinical Neurophysiology*, *111*, 1141–1156.
- Sterman, M. B., Wyrwicka, W., & Howe, R. (1969). Behavioral and neurophysiological studies of the sensorimotor rhythm in the cat. *Electroencephalography and Clinical Neurophysiology*, *27*, 678–679.
- Swanson, J., Castellanos, F. X., Murias, M., LaHoste, G., & Kennedy, J. (1998). Cognitive neuroscience of attention deficit hyperactivity disorder and hyperkinetic disorder. *Current Opinion in Neurobiology*, *8*, 263–271.
- Taylor, M. J., Voros, J. G., Logan, W. J., & Malone, M. A. (1993). Changes in event-related potentials with stimulant medication in children with attention deficit hyperactivity disorder. *Biological Psychology*, *36*, 139–156.
- Tewes, U. (1983). *Hamburg-Wechsler-Intelligenztest für Kinder—Revision*. Bern: Huber.
- Thompson, L., & Thompson, M. (1998). Neurofeedback combined with training in metacognitive strategies: Effectiveness in students with ADD. *Applied Psychophysiology and Biofeedback*, *23*, 243–263.
- Ullmann, R. K., Sleator, E. K., & Sprague, R. L. (1985). A change of mind: The Conners abbreviated rating scales reconsidered. *Journal of Abnormal Child Psychology*, *13*, 553–565.
- Wada, N., Yamashita, Y., Matsuishi, T., Ohtani, Y., & Kato, H. (2000). The test of variables of attention (TOVA) is useful in the diagnosis of Japanese male children with attention deficit hyperactivity disorder. *Brain Development*, *22*, 378–382.
- Wechsler, D. (1974). *Manual for the Wechsler intelligence scale for children*. New York: Psychological Corporation.
- Zametkin, A. J., & Liotta, W. (1998). The neurobiology of attention-deficit/hyperactivity disorder. *Journal of Clinical Psychiatry*, *59*, 17–23.

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