CHAPTER 15

Enduring Effects of Neurofeedback in Children

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INTRODUCTION

The benefits of neurofeedback as a treatment for children with developmental disorders have been demonstrated. Neurofeedback has shown efficacy for a wide variety of developmental disorders such as autism, ADHD, epilepsy, and dyslexia (Coben & Padolsky, 2007; Egner & Sterman, 2006; Evans & Park, 1996; Hammond, 2007; Leins et al., 2007; Lubar, et al., 2005). Further, to our knowledge there is no study that has reported any detrimental side effects as a result of neurofeedback treatment.

Preliminary research suggests that neurofeedback is an effective therapy for reducing core symptoms in children with both autism and ADHD (Arns et al., 2009; Coben & Padolsky, 2007; Heinrich, Gevensleben, Freisleder, Moll & Rothenberger, 2004; Jarusiewicz, 2002). Neurofeedback is a therapy that teaches clients to regulate their brain activity to work in a new, more efficient way through the use of underlying operant conditioning paradigms. This treatment involves providing a subject with visual and/or auditory "feedback" for particular neural behaviors

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(Monastra, Monastra, & George, 2002). Through conditioning the subject is taught to inhibit EEG frequencies that are excessively generated and augment frequencies that are deficient. With continuous training and coaching, individuals are taught to maintain brainwave patterns concurrent with healthy neural functioning. Recently, Walker, Kozlowski, and Lawson (2007) presented evidence demonstrating the ability of neurofeedback training to successfully train neural functioning to more normal states as well as simultaneously demonstrating reductions in pathological symptoms. For more in-depth information regarding neurofeedback the interested reader is referred to Hammond (2007).

Neurofeedback was originally assessed as a useful therapy by Barry Sterman in 1970 at the Neuropsychiatric Institute of UCLA (Sterman et al., 1970). Later, Lubar and Shouse (1976) reported distinct positive EEG and behavioral changes in a hyperkinetic child with ADHD after training the sensorimotor EEG rhythm (SMR: 12–14 Hz). Since then there has been an increasing quantity of published research indicating positive effects of neurofeedback with a variety of disorders, including ADHD. Recently, Monastra et al. (2002) assessed more than 100 children with ADHD, and found that neurofeedback was capable of significantly reducing core symptoms of ADHD. Arns et al. (2009) performed a meta-analysis encompassing over 15 studies and a total sample size of more than 1100 children, and concluded that neurofeedback is an effective form of treatment for subjects with ADHD. Moreover, recent investigations have found the results of neurofeedback training to be comparable to the clinical gains achieved through medication in children (Fox, Tharp, & Fox, 2005). However, unlike medications, there has been no report of any unwanted or negative side effects as a result of treating ADHD with neurofeedback training.

The efficacy of neurofeedback for autistic children was initially assessed in a study by Jarusiewicz (2002) in which she reported a 26% decrease in autistic symptoms in an experimental group and a 3% reduction in a wait-list control group. More recently, Coben & Padolsky (2007) found similar, yet more impressive results, reporting a 40% decrease in core autistic symptoms as a result of neurofeedback therapy. Similar to the findings in regards to ADHD, to our knowledge there is no evidence of neurofeedback training producing any detrimental or unwanted side effects in children with autism spectrum disorders (ASD).

In this chapter we discuss evidence for long-term effects of neurofeedback. Over the course of three series of studies we examined the efficacy

of neurofeedback training for children with autism as well as children with ADHD. We hypothesized that neurofeedback creates effective as well as enduring positive clinical changes in children with autism and ADHD.

NEUROFEEDBACK AS A TREATMENT FOR CHILDREN WITH ADHD

Recently the 8-year follow-up results from a very large NIMH sponsored trial on different treatments for ADHD have been published [the NIMH Collaborative Multisite Multimodal Treatment Study of Children with Attention-Deficit/Hyperactivity Disorder (ADHD), abbreviated as MTA (Molina et al., 2009)]. This study compared four different treatments in 579 children. These were initially randomly assigned to: (1) systemic medication management; (2) multi-component behavior therapy; (3) a combination of (1) and (2); and (4) usual community care. The first results after 14 months initially showed that the medication and combined groups showed the greatest improvements in ADHD and ODD symptoms. However, half of these effects had dissipated 10 months after the treatment was completed. More importantly, after 8 years follow-up, there were no differences to be found between these four groups, indicating that the initial treatments to which the children were randomly assigned, did not predict functioning 6–8 years later. This multi-centre large-scale study hence clearly demonstrates a lack of long-term effects for either stimulant medication, multi-component behavior therapy or multimodal treatment (Molina et al., 2009). Furthermore, in general, response rates to stimulant medication in ADHD are estimated to be between 70 and 90% (see Hermens, Rowe, Gordon, & Williams (2006) for an overview).

These results clearly show that at present there is no commonly accepted treatment modality that has sufficient long-term efficacy for children with ADHD, and there is a need for new treatments with better long-term outcomes. In the next paragraphs we provide evidence that neurofeedback may be as effective and have more enduring effects than any of the presently commonly used treatment approaches for ADHD.

As noted above, in 1976 Lubar and Shouse were the first to report on EEG and behavioral changes in a hyperkinetic child after training the sensorimotor EEG rhythm (SMR: 12–14 Hz). In 2004, Heinrich et al. were the first to report positive results after Slow Cortical Potential (SCP) neurofeedback in the treatment of ADHD. SCP neurofeedback is different

from the above-mentioned neurofeedback approach in that changes in the polarity of the EEG are rewarded (i.e. positivity vs. negativity in the EEG), and a discrete reward scheme is used. Incidentally, both SCP neurofeedback and SMR neurofeedback approaches have been successfully used in treating epilepsy as well [for an overview also see Egner & Sterman, (2006)], and it has been suggested that both regulate cortical excitability (Arns et al., 2009; Kleinnijenhuis, Arns, Spronk, Breteler & Duysens, 2008). Several studies have compared theta-beta training and SCP training using both within-subject (Gevensleben et al., 2009b) and between-subject (Leins et al., 2007) designs, and both neurofeedback approaches showed comparable effects on different aspects of ADHD such as inattention, hyperactivity, and impulsivity. A recent meta-analysis investigating the effects of neurofeedback used data from 15 published studies with a total sample size of 1194 children with ADHD. Based on this study (Arns et al., 2009) it was concluded that neurofeedback for the treatment of ADHD met the evidence-based criteria for Level V: Efficacious and Specific. This study also addressed some of the criticisms made in the past. In this meta-analysis, long-term effects were not addressed at length.

Some studies have considered long-term effects of neurofeedback and found that the skill to modulate EEG activity in the required direction is still preserved over time [6 months: (Leins et al., 2007); and 2 years: (Gani, Birbaumer & Strehl, 2008)]. Given the treatment potential already mentioned, these long-term findings make neurofeedback a very interesting and promising treatment for ADHD. In the following paragraphs we will in a more quantitative way report on the long-term effects of neurofeedback.

LONG-TERM EFFECTS OF NEUROFEEDBACK

Some of the earliest studies of neurofeedback with ADHD considered long-term effects. Lubar (1991) reported follow-up results on the initial case mentioned above (Lubar & Shouse, 1976) demonstrating that the effects were sustained over time, and the child was still performing well without medication. In the Monastra, Monastra, and George (2002) study, all 100 ADHD children were medicated and 51 children also received neurofeedback. Interestingly, when the medication was removed at the end of treatment, only the subjects who had completed neurofeedback were able to sustain their improvements. The qEEG measurements also showed a significant decrease in cortical slowing of the individuals who

had completed neurofeedback, but not in the subjects who had only received medication.

Several controlled studies that investigated the effects of neurofeedback in ADHD reported follow-up results as well. Heinrich et al. (2004) performed a 3-month follow-up for a SCP training group and found all criterion measures improving further (Heinrich, personal communication: unpublished results; Arns et al., 2009). Strehl and colleagues showed that at 6-month follow-up scores in impulsivity, inattention, and hyperactivity were improved even further as compared to the end of treatment (Leins et al., 2007; Strehl et al., 2006). Furthermore, a 2-year follow-up for this study (Gani et al., 2008) showed that all improvements in behavior and attention turned out to be stable. Test results for attention and some of the parents' ratings once more improved significantly. In addition, EEG self-regulation skills turned out to be preserved, indicating that these children were still able to successfully regulate their brain activity.

In order to visualize these effects further we have plotted the effects for inattention and hyperactivity in Figure 15.1. The weighted average is an average of the scores of all three studies, and then weighted for the number of subjects per study. This figure clearly shows that the effects of neurofeedback improve further over time. Both measures are based on a DSM-based questionnaire. For impulsivity there were too few data to make a sensible comparison.

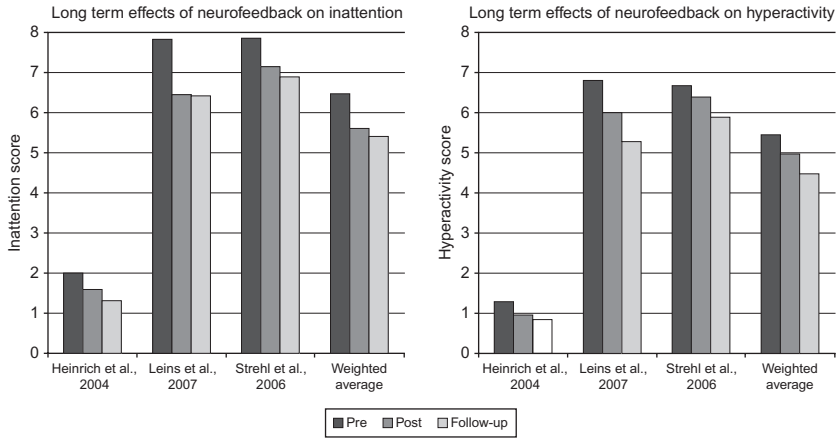

Figure 15.1 The effects of neurofeedback over time for three controlled studies for inattention (left) and hyperactivity (right). The study by Heinrich et al. performed 3 months follow-up and the other two studies performed 6 months follow-up. Note that the effects of neurofeedback tend to improve further over time (as opposed to the effects of medication, which are not sustained when the medication is stopped).

In 2009 one of the largest multi-site randomized controlled trials on neurofeedback in ADHD was published by Gevensleben (Gevensleben et al., 2009b). This study incorporated data from more than 100 subjects. Post-qEEG data from this sample showed that the neurofeedback-trained group – but not the control group – showed reduced EEG theta power (Gevensleben et al., 2009a), thereby demonstrating the specificity of this intervention. The 6-month follow-up data from this study (Gevensleben et al., 2010) showed that the beneficial effects that resulted from neurofeedback were maintained at follow-up.

Based on the totality of the limited data available, it may be concluded that the clinical effects of neurofeedback remain stable over time, and may improve further with time. This is in contrast to current treatments such as medication management and multicomponent behavior therapy [(as explained in the introduction based on the NIMH-MTA trial (Molina et al., 2009)]. However, more large-scale, controlled studies with longer follow-up will be required to solidify these conclusions.

Investigations have also been conducted in recent years on the long-term enduring effects of neurofeedback for conditions other than ADHD. For example, neurofeedback has been shown to be ameliorative in nature for subjects with autism and the lasting effects of this treatment have been increasingly examined.

NEUROFEEDBACK AS A TREATMENT FOR CHILDREN WITH ASD

The first study of Kouijzer and colleagues (Kouijzer, de Moor, Gerrits, Congedo, & van Schie, 2009) investigated the effects of neurofeedback in children with autism. It included 14 children aged from 8 to 12 years with a diagnosis of Pervasive Developmental Disorder – Not Otherwise Specified (PDD-NOS). Excluded were children with an IQ score below 70, children using medication, and children with a history of severe brain injury or co-morbidity such as ADHD or epilepsy. Participants were divided into treatment and wait-list control group according to the order of applying. The first seven participants who applied were assigned to the treatment group; the control group included seven participants who were recruited out of a larger group of children who applied later, and matched participants in the treatment group on diagnosis, sex, and intelligence test scores. During baseline (Time1), all participants were evaluated using qEEG and a range of executive function tasks, and parents completed

behavior questionnaires (CCC and Auti-R). After neurofeedback training (Time2), or a comparable time interval for the wait-list control group, qEEGs and data on executive functions and social behavior were re-collected. One year after ending treatment (Time3), follow-up data including qEEGs, executive function tasks, and behavior questionnaires were collected in the treatment group. Participants in the wait-list control group did not participate in the follow-up, because they had started neurofeedback training. Participants in the treatment group had neurofeedback training twice a week, until 40 sessions were completed. In each session participants were rewarded when inhibiting theta power (4–8 Hz) and increasing low beta power (12–15 Hz) at scalp location C4 according to a protocol including seven 3-min intervals of neurofeedback training separated by 1-min rest intervals.

After 40 sessions of neurofeedback, 70% of the participants in the treatment group had effectively decreased theta power, ps < 0.05 and rs $= -0.496$ to -0.771, and increased low beta power, ps < 0.05 and rs $= 0.218$ to 0.529. Repeated measures MANOVA on the executive functions data collected at Time1 and Time2 revealed a significant interaction between treatment and control group, indicating improvement of participants in the treatment group on tasks measuring attention skills, $F(1,11) = 8.437$, $p < 0.05$, $\eta_\rho^2 = 0.434$, cognitive flexibility, $F(1,11) = 5.602$, $p < 0.05$, $\eta_\rho^2 = 0.3373$; set-shifting, $F(1,11) = 5.081$, $p < 0.05$, $\eta_\rho^2 = 0.316$; concept generation/inhibition $F(1,11) = 4.890$, $p < 0.05$, $\eta_\rho^2 = 0.308$ (verbal inhibition) and $F(1,11) = 5.064$, $p < 0.05$, $\eta_\rho^2 = 0.315$ (motor inhibition), and planning, $F(1,11) = 7.198$, $p < 0.05$, $\eta_\rho^2 = 0.396$. Using repeated measures MANOVA to compare questionnaire data collected at Time1 and Time2 revealed a significant interaction effect between treatment and control group, indicating improvement in non-verbal communication, $F(1,12) = 5.505$, $p < 0.05$, $\eta_\rho^2 = 0.314$, and general communication, $F(1,12) = 5.379$, $p < 0.05$, $\eta_\rho^2 = 0.310$. Time2 Auti-R questionnaire data evaluating changes in behavior over the last six months showed significant improvement in social interactions, $F(1,12) = 17.775$, $p < 0.05$, $\eta_\rho^2 = 0.618$, communication skills, $F(1,12) = 29.054$, $p < 0.05$, $\eta_\rho^2 = 0.725$, and stereotyped and repetitive behavior, $F(1,12) = 7.782$, $p < 0.05$, $\eta_\rho^2 = 0.414$ for the treatment group, but not for the control group, ps > 0.05.

One-year follow-up data demonstrated enduring effects of neurofeedback treatment (Kouijzer, de Moor, Gerrits, Buitelaar, & van Schie, 2009). Repeated measures MANOVA on the executive function task

scores at Time2 and Time3 indicated maintenance of cognitive flexibility, planning skills, and verbal inhibition, $ps < 0.05$, improvement of attention, $F(1,6) = 16.248$, $p < 0.05$, $\eta_\rho^2 = 0.765$, and marginally significant improvement of motor inhibition, $F(1,6) = 4.560$, $p = 0.086$, $\eta_\rho^2 = 0.477$. No significant decreases in executive function skills were found after one year. Repeated measures MANOVA comparing Time1 and Time 3 data confirmed maintenance of these effects. Analysis revealed significant increases of all executive functions that improved after neurofeedback treatment, i.e. attention skills, $F(1,6) = 39.201$, $p < 0.05$, $\eta_\rho^2 = 0.887$, cognitive flexibility, $F(1,6) = 27.802$, $p < 0.05$, $\eta_\rho^2 = 0.848$ (set-shifting), and $F(1,6) = 18.540$, $p < 0.05$, $\eta_\rho^2 = 0.788$ (concept generation), inhibition, $F(1,6) = 15.458$, $p < 0.05$, $\eta_\rho^2 = 0.756$ (verbal inhibition) and $F(1,6) = 10.696$, $p < 0.05$, $\eta_\rho^2 = 0.681$ (motor inhibition), and planning, $F(1,6) = 21.420$, $p < 0.05$, $\eta_\rho^2 = 0.811$. Figure 15.2 shows Time1, Time2, and Time3 scores of the treatment group on tests for attention, cognitive flexibility, inhibition, and planning.

Analysis of behavior questionnaires filled out by parents at Time2 and Time3 showed no loss of non-verbal communication and general communication (CCC), $ps > 0.05$, social interactions, communication skills, and stereotyped and repetitive behavior (Auti-R), $ps > 0.05$. Comparing Time1 and Time3 behavior questionnaires (CCC) confirmed the positive effect for non-verbal communication, $F(1,6) = 7.125$, $p < 0.05$, $\eta_\rho^2 = 0.543$, but not for general communication, $F(1,6) = 2.745$,

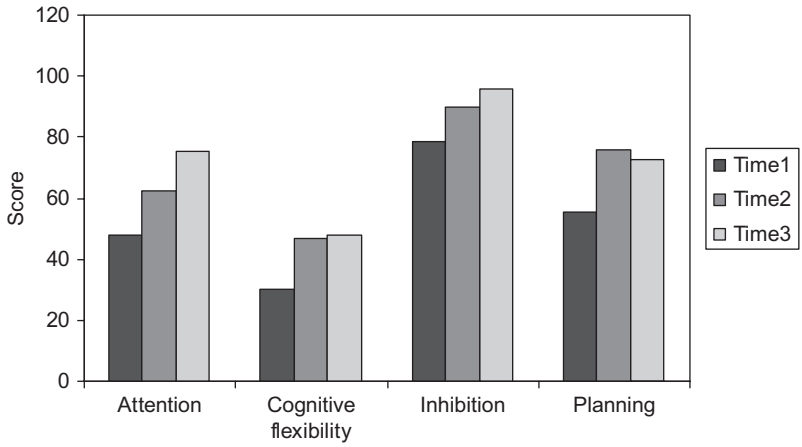

Figure 15.2 Time1, Time2, and Time3 data of the treatment group on executive function tasks.

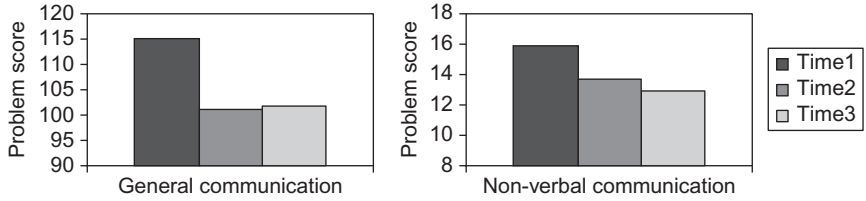

Figure 15.3 Time1, Time2, and Time3 data of the treatment group on social behavior.

$p = 0.149$, $\eta_\rho^2 = 0.314$. Figure 15.2 shows Time1, Time2, and Time3 questionnaire data (CCC) for general communication and non-verbal communication of the treatment group. Detailed information about the results of this study can be found in the original paper (Kouijzer, de Moor, Gerrits, Buitelaar et al., 2009).

In a second study of Kouijzer and colleagues (Kouijzer, van Schie, de Moor, Gerrits, & Buitelaar, 2010) several methodological improvements were implemented to better identify the effects of neurofeedback. A randomized wait-list control group design was used, and the study was conducted at the schools of the participants (N = 20). Participants were 8–12 years old and had diagnoses of autism, Asperger's disorder or PDD-NOS. Participants in the treatment group had 40 individual neurofeedback sessions using an individualized treatment protocol based on an initial qEEG. However, all treatment protocols included theta inhibition at frontocentral scalp locations. Treatment response was evaluated by qEEG measures taken during rest and task conditions, a range of executive function tasks, and social behavior questionnaires filled out by parents and teachers. All data were collected before (Time1) and after treatment (Time2) and at 6 months follow-up (Time3).

Results of the study showed that 60% of participants decreased theta power within 40 sessions of neurofeedback, ps < 0.05 and rs $= -0.387$ to -0.832. Additionally, repeated measures MANOVA on qEEG data revealed a significant interaction between treatment and control group, indicating a decrease in theta power in the treatment group in two out of four qEEG conditions, i.e. eyes closed, $F(1,14) = 4.883$, $p < 0.05$, $\eta_\rho^2 = 0.259$, and hand movement, $F(1,14) = 7.856$, $p < 0.05$, $\eta_\rho^2 = 0.359$. Repeated measures MANOVA on Time1 and Time2 executive function data showed a significant interaction between treatment and control group for cognitive flexibility, indicating improvement in cognitive flexibility in the treatment group compared to the control group,

$F(1,18) = 4.652$, $p < 0.05$, $\eta_\rho^2 = 0.205$. Repeated measures MANOVA showed a significant interaction effect for social interactions and communication skills, indicating that parents of participants in the treatment group reported significant improvement in social interactions and communication skills, $F(1,18) = 9.874$, $p < 0.05$, $\eta_\rho^2 = 0.367$, whereas less or no improvement was reported by parents of children in the control group. However, teachers of participants in the treatment group did not report any greater improvement in social behavior after neurofeedback treatment compared to reports of teachers of participants in the control group, $F(1,18) = 0.341$, $p = 0.566$, $\eta_\rho^2 = 0.019$.

Analysis of the 6-month follow-up data revealed enduring effects of neurofeedback treatment. Repeated measures MANOVA was used to compare the scores on executive function tasks at Time2 and Time3 and showed no significant changes, $F(1,18) = 0.186$, $p = 0.671$, $\eta_\rho^2 = 0.010$, suggesting that participants maintained the same levels of executive functioning for at least 6 months. Repeated measures MANOVA comparing Time1 and Time3 data confirmed the previously described effects by revealing a significant increase of cognitive flexibility for the treatment group but not for the control group, $F(1,18) = 5.499$, $p < 0.05$, $\eta_\rho^2 = 0.234$. Figure 15.4 shows Time1, Time2, and Time3 scores of the treatment and control group on cognitive flexibility.

Repeated measures MANOVA comparing the scores on behavioral questionnaires at Time2 and Time3 showed no effects of group or time,

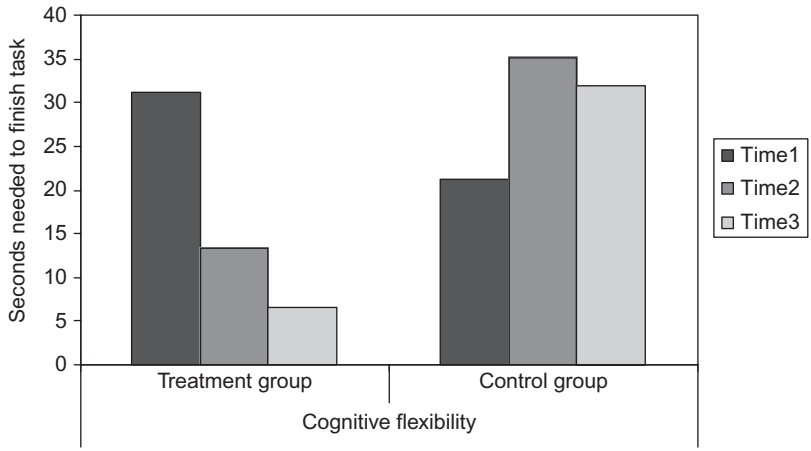

Figure 15.4 Time1, Time2, and Time3 data of treatment and control group on cognitive flexibility.

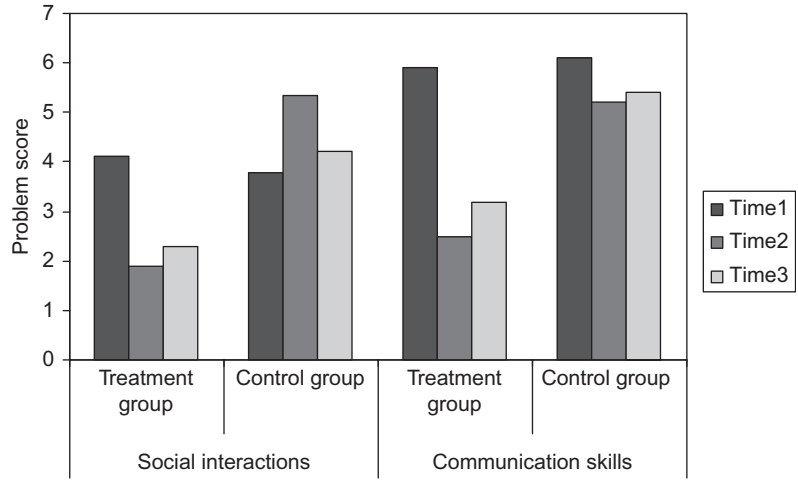

Figure 15.5 Time1, Time2, and Time3 data of treatment and control group on social behavior.

$F(1,18) = 1.099$, $p = 0.380$, $\eta_\rho^2 = 0.180$, indicating maintenance of the effects in social behavior that were reached 6 months earlier. Repeated measures MANOVA comparing Time1 and Time3 questionnaire data confirmed this effect by showing a significant interaction, suggesting decreases in problem scores on behavior questionnaires for the treatment group, but not for the control group, $F(1,18) = 4.871$, $p < 0.05$, $\eta_\rho^2 = 0.223$. Figure 15.5 shows Time1, Time2, and Time3 questionnaire data of social interactions and communication skills of treatment and control group. More detailed information about the results of this study can be found in the original paper (Kouijzer, de Moor, Gerrits, Buitelaar et al., 2009).

Both studies discussed above indicate maintenance of the effects in executive functions and social behavior from 6 months to 1 year after ending neurofeedback treatment.

ENDURING BEHAVIORAL AND NEUROPSYCHOLOGICAL BENEFITS OF NEUROFEEDBACK IN ASD

A similar study with findings that can be considered complementary to those of Kouijzer and colleagues was recently conducted by Coben at his New York clinic. This study assessed 20 patients with ASD in order to investigate long-term clinical effects of neurofeedback in terms of

behavioral and neuropsychological measures. The subject pool for this study was predominately male (male 16; female 4) and all Caucasian. The mean age was 9.53, with a range of 5–10. Most subjects (80%) were medication-free, with only one subject taking more than two medications. Handedness was mostly right-handed (N = 16) with one left-handed and three ambidextrous subjects.

Subjects were administered parent rating scales, including the Autism Treatment Evaluation Checklist (ATEC; Rimland & Eldelson, 2000), the Personality Inventory for Children (PIC-2; Lachar & Gruber, 2001), the Behavior Rating Inventory of Executive Function (BREIF; Gioia, Isquith, Guy & Kenworthy, 2000), and the Gilliam Asperger's Disorder Scale (GADS; Gilliam, 2001). Subjects were also administered neuropsychological assessments covering domains of attention/executive functioning, language, and visuo-spatial processing. After baseline assessments were collected all subjects underwent at least 40 sessions of neurofeedback training, with an average of 64.5 completed sessions among all subjects. Upon completion of therapy, subjects were re-evaluated and pre- and post-treatment scores were compared for significance. After re-evaluation, neurofeedback was withheld for 5–22 months (M = 10.1 months) while no other treatments were administered. Following this break in treatment, subjects were evaluated once again in the same fashion as previously described. Their latter scores were then compared to scores obtained at the end of active neurofeedback training (Time 2).

All statistical computations were performed in the statistical package SPSS. Scores prior to treatment on parent rating scales were compared for significance to scores obtained after treatment had ended. Analysis of pre- and post-scores obtained from the ATEC revealed significant changes following neurofeedback training (two-sample t test, $t = 11.302$, d.f. 19, $p < 0.000$). Likewise, changes in scores on the GADS prior to and following treatment were found to be significant (two-sample t test, $t = 8.332$, d.f. 19, $p < 0.000$). Significant changes were also found to be present following treatment among scores from the BRIEF (two-sample t test, $t = 5.370$, d.f. 19, $p < 0.000$) as well as the PIC-2 (two-sample t test, $t = 6.320$, d.f. 19, $p < 0.000$). Interestingly, when subjects were re-assessed following a period of no neurofeedback training (range 5–22 months), no significant changes were found on any parent rating scale administered (see Figure 15.6). This suggests that changes in parent ratings that were improved by neurofeedback training remained stable during this follow-up period.

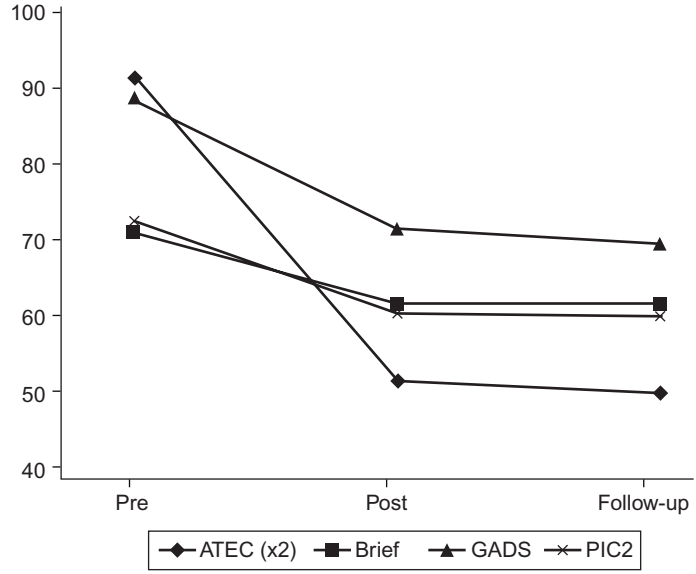

Figure 15.6 Clinical improvements among subjects as assessed by the parents rating scales of ATEC, BRIEF, GADS, and PIC-2 for pre-, post-treatment, and follow-up periods.

Neuropsychological evaluations encompassing the domains of attention, executive functioning, language, and visuo-spatial processing were also analyzed for significant differences. Significant changes from pre- to post-treatment scores were found among all three domains assessed: attention/executive functioning (two-sample t test, $t = -5.297$, d.f. 19, $p < 0.000$), language (two-sample t test, $t = -2.235$, d.f. 10, $p < 0.049$) and visuo-spatial processing (two-sample t test, $t = -5.308$, d.f. 18, $p < 0.000$). Interestingly, significant therapeutic changes were also found after subjects were re-evaluated after a lengthy (5–22 months) absence from neurofeedback training. These occurred in the areas of attention (two-sample t test, $t = -3.021$, d.f. 19, $p < 0.007$), language (two-sample t test, $t = -2.347$, d.f. 10, $p < 0.041$) and visuo-spatial processing (two-sample t test, $t = -3.568$, d.f. 18, $p < 0.002$) (see Figure 15.7). This would suggest that neurofeedback training not only led to objective gains in neuropsychological functioning, but that these enhancements in functioning continued to improve over the follow-up period when no treatment was being received.

The results of this present study were quite interesting. First, our findings add to the wealth of studies that have shown that from pre- to

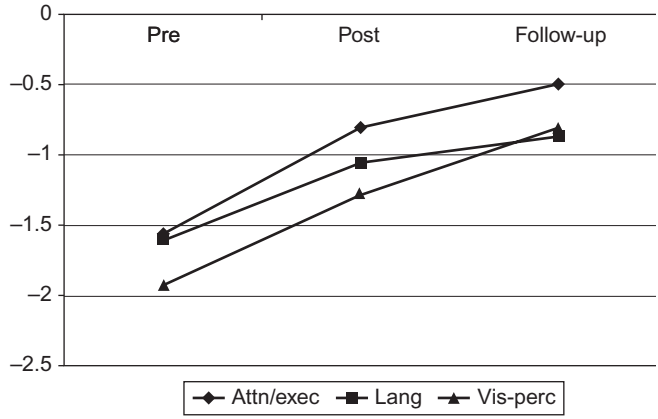

Figure 15.7 Graph showing the clinical improvements among the domains of attention/executive functioning, language, and visuo-spatial processing as assessed by neuropsychological evaluations at pre-, post-treatment and follow-up periods.

post-treatment conditions, neurofeedback is an effective therapy for treating individuals with autistic spectrum disorders. Additionally, these results show that this treatment was effective in limiting autistic behavioral deficits as well as deficits of a more neuropsychological nature. Furthermore, as our analysis shows, there were no significant increases in autistic pathology when subjects were re-evaluated after neurofeedback was withheld. This finding supports previously found evidence that neurofeedback is capable of creating stable changes within autistic subjects that are not likely to rapidly degrade when treatment ends (Coben & Padolsky, 2007; Jarusiewicz, 2002).

Of potentially even greater interest, this study found that during the period in which subjects were receiving no treatment, positive clinical neuropsychological gains were still being manifested within the domains of attention, executive functioning, language, and visuo-spatial processing. Thus, even without continued treatment subjects apparently were continuing to improve in these realms. An important implication of this finding is that neurofeedback may indeed change the autistic brain to work in novel and more efficient ways, and these changes may continue to progress even after the treatment has ended. This finding helps further the claim that neurofeedback creates specific neurophysiological changes within the autistic brain (Coben, Sherlin, Hudspeth & McKeon, 2009 [study under review]). This is in stark contrast to other commonly administered treatments for autism. For example, Lovaas et al. (1973) performed

a study in which Applied Behavioral Analysis (ABA) was administered to a group of children with autism. Upon completion of ABA training the experimenters reported positive gains in terms of clinical improvements in behavioral deficits. Subjects were then re-evaluated 1–4 years later, and subjects who did not continuously receive ABA training had significantly regressed. As our current findings demonstrate, there is no evidence of regression among any of our subjects receiving neurofeedback training. In terms of drug therapies there is no evidence to our knowledge that would indicate that medications result in enduring clinical gains for subjects with autism when medication is withheld. In fact, numerous studies indicate that prolonged medication use has detrimental effects on autistic individuals (Anderson et al., 2009; Malone et al., 2002).

In terms of the limitations of the current study, the participants consisted of a selected pool of subjects. Subjects were placed in groups by choice of the experimenter rather than by random assignment. When subjects are chosen in that manner there may be a degree of selection bias associated. We would also recommend that this experiment be replicated with more neuropsychological assessments and parent rating scales included in order to more widely assess the effects of neurofeedback training. This type of investigation could broaden the present findings, and help determine if there are other correlations or significant predictors we might not have considered. Also, we would recommend a study with a greater gap between the end of treatment and re-evaluation of subjects. Doing this, we believe, would help to assess nature and extent of any positive clinical gains found in subjects when they are no longer receiving treatment, as well as test more fully the limits of enduring effects of neurofeedback treatment.

DISCUSSION

The current chapter provided evidence that neurofeedback is a therapy capable of creating enduring changes in children with both autism and ADHD. This was found across all experiments reviewed. This coupling of multiple studies converging upon a singular finding, namely the enduring clinical effects of neurofeedback, serves to provide strong evidence that neurofeedback is effective for children with developmental disorders. Moreover, these findings provide evidence that neurofeedback is not only effective in children with developmental disorders, but also is capable of leading to long-lasting positive changes in these subjects.

A therapy that can lead to long-lasting effects for children with developmental disorders (and perhaps continuing improvement even after the treatment is stopped) is an enormous asset for children with developmental disorders. Most contemporary treatments require prolonged and lengthy treatment sessions. For example, ABA training can require up to 40 hours a week over several months to be effective (Howard et al., 2005). Furthermore, drug therapies usually require years of medication in order to maintain efficacy. In addition, some children require incremental increases in dosages over a period of years for medication use to be clinically viable. Our current results and those of others discussed in this chapter indicate that neurofeedback therapy can reach clinical efficacy relatively quickly, and positive gains can be retained for months after treatment has stopped. Outside of the clinical implications, there are ancillary benefits supporting the use of neurofeedback. For example, the financial aspects of this treatment should be considered. Presently, the United States alone spends upwards of $3.2 million for the care and treatment for a single individual with autism, a figure that equates to $35 billion annually (Ganz, 2006). Similarly, the overall cost for treatment of ADHD in the United States is $30 billion annually (Birnbaum et al., 2000). A treatment such as neurofeedback with positive effects that can endure over time has great potential to relieve some of the fiscal burdens associated with these disorders.

Results of the studies reviewed in this chapter also provide evidence for the safety of neurofeedback. All studies reported no instances of subjects worsening or showing any side effects while undergoing this treatment over an extended period of time. Moreover, there was no evidence of negative side effects when neurofeedback was ceased. In fact, the opposite was found across all studies. This, again, is contradictory to other interventions, most notable drug therapies, which have documented adverse reactions within this population and often have failed to demonstrate positive effects on primary symptoms (Kidd, 2002). For example, complaints of excessive weight gain, drowsiness, and fatigue have been reported by children with ASD and ADHD while taking Risperdal (risperidone) (McCraken et al., 2002); and it has been reported that children taking risperidone at relatively high doses may become susceptible to developing facial dystonia (Zuddas et al., 2000). Likewise, research into the administration of fluoxetine has been found to produce side effects such as restlessness, hyperactivity, agitation, decreased appetite, and insomnia (Cook et al., 1992). Investigations into other contemporary treatments (i.e. diet and chelation therapies) have failed to yield

adequate evidence in regard to their safety or efficacy (Doja & Roberts, 2005; Harrison-Elder et al., 2006; McDougle et al., 2000). Recently, Dr Susan Hyman and colleagues (2010) of the University of Rochester performed the single largest randomized study on the effects of casein- and whey-free diets as a treatment for autism. The results of this study found no therapeutic benefits in withholding whey or casein proteins from an autistic child's diet.

We speculate that the enduring effects of neurofeedback in children with developmental disorders are the result of this treatment's ability to change the brain in a therapeutic manner. Recently, Coben and colleagues reported specific neurophysiological changes in terms of coherence within and between specific neural regions following neurofeedback treatment for children with ASD (Coben, Sherlin, Hudspeth, & McKeon, 2009 [study under review]). We would argue that neurofeedback training causes specific neurophysiological changes within the brain, which in turn contribute to the long-lasting effects of this treatment, and this fosters the continued growth and development of cognitive functions. Moreover, we suggest that more research be conducted into the precise neural areas clinically affected by neurofeedback in an effort to more fully understand the efficacy of neurofeedback for children with developmental disorders.

In summary, results of the studies examined add to the growing wealth of investigations into the efficacy of neurofeedback as a treatment for children with developmental disorders. Moreover, these results have found this treatment to be effective over an extended period of time. Consistent with these results we recommend future studies be conducted that assess the enduring effects of neurofeedback over even longer treatment spans.

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