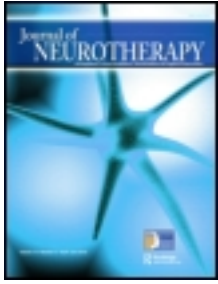


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A Position Paper on Neurofeedback for the Treatment of ADHD

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SCIENTIFIC FEATURES

A Position Paper on Neurofeedback for the Treatment of ADHD

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ABSTRACT. This position paper provides the current evidence supporting the use of neurofeedback in the treatment of ADHD and recommendations on the implementation of neurofeedback in clinical practice. The paper also provides basic information regarding the diagnosis and psychophysiological etiology of ADHD. The paper does not focus on a specific age range of a clinical population. Unless otherwise noted, we are referring to all subtypes of ADHD (inattentive, hyperactive only, and combined). Conclusions and recommendation are based on the most recent research; however, we also refer to relevant historical studies that support our position on neurofeedback. The readers are strongly advised to research behavioral diagnostic criteria and testing methods elsewhere. This paper is not intended as a comprehensive educational tool for diagnosis or treatment of ADHD. Our purpose is to demonstrate the rationale and to reference the necessary support for neurofeedback in order to be recognized as a legitimate, scientific, and evidence-based intervention for the treatment of ADHD.

KEYWORDS. ADHD, efficacy rating, neurofeedback, position, treatment

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SUMMARY AND RECOMMENDATIONS

This position paper provides basic information regarding the diagnosis and psychophysiological etiology of attention-deficit/hyperactivity disorder (ADHD) and the evidence for treatment of ADHD to include the modality of neurofeedback. We have summarized the primary historical research as well as the more recent investigations in which neurofeedback has been utilized in the ADHD population. Our conclusions find that neurofeedback not only is a suitable intervention for those diagnosed with ADHD, but also may be the preferred choice of interventions for some individuals. Solid scientific evidence and demonstrated clinical data, collected from multiple studies across the world, are the basis for our conclusions. The following are our recommendations:

1. Neurofeedback is a safe and efficacious treatment intervention for ADHD, meeting the rating of Level 5: Efficacious and Specific.
2. Neurofeedback in the treatment of ADHD has been shown to have long-term effects, lasting from 3 to 6 months. More research is required to investigate the effects after 3 to 5 years of treatment similar to the National Institute of Mental Health Collaborative Multisite Multimodal Treatment Study of Children with ADHD (NIMH-MTA) trial.
3. The effects of neurofeedback appear to have similar effects to stimulant medication for inattention and impulsivity, but more controlled and randomized studies are required to further support this observation.
4. Additional research is required to investigate the working mechanism of neurofeedback.
5. Given that neurofeedback currently requires multiple treatment sessions, further research should be directed toward improving neurofeedback treatment to require fewer treatment sessions (e.g., LORETA neurofeedback, Independent

Component Analysis (ICA) neurofeedback, Z-score neurofeedback).

6. Neurofeedback is efficacious when inattention and impulsivity are the main problems. When the main complaint is hyperactivity, medication is possibly a better choice given the limited success of neurofeedback in this domain. Controlled and randomized studies are required to further substantiate this claim.
7. No differences in neurofeedback efficacy have been found between medicated and nonmedicated children; therefore, neurofeedback can be utilized in combination with a medication regimen.
8. Licensed health care providers should take necessary educational prerequisites to understand the methods and proper implementation of neurofeedback and its appropriateness for the treatment of ADHD.
9. When appropriately trained in the planning, implementation, and monitoring of neurofeedback, the licensed health care professional should consider including neurofeedback as a potential modality of treatment.

NEUROFEEDBACK FOR THE TREATMENT OF ADHD

ADHD has become one of the most common neurodevelopmental and psychiatric disorders of childhood (Rowland, Lesesne, & Abramowitz, 2002). The general rate of prevalence is reported between 3% and 10% of school-age children (Erk, 1995). Currently, the disorder is primarily diagnosed by referring to the criteria of the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., text rev. [DSM-IV-TR]; American Psychiatric Association [APA], 2000) or the International Statistical Classification of Mental Disorders (World Health Organization, 1992). ADHD is not only the most common of the childhood psychiatric disorders but also the best researched disorder (Rowland et al., 2002). Depending on how it is characterized and diagnosed, it is estimated to affect as many as 3% to 7% of

the school-age children (American Psychiatric Association, 2000). The APA has also reported that the prevalence of adolescent and adult ADHD is not well known. According to the *DSM-IV-TR* (APA, 2000), the disorder presents itself in three primary subtypes: combined type, predominantly inattentive, predominantly hyperactive-impulsive type.

Neurofeedback is a type of operant conditioning in which an individual modifies the frequency, amplitude, or other characteristic of his or her own EEG. As early as 1941, Jasper and Shagass demonstrated that the EEG alpha rhythm could be classically conditioned. Furthermore, it has been demonstrated that humans and animals can control their EEG through feedback (S. S. Fox & Rudell, 1968; Hetzler, Rosenfeld, Birkel, & Antoinetti, 1977; Rosenfeld, Rudell, & Fox, 1969; Serman, 1996; Strehl et al., 2006; Thatcher, 2000) and that the skill to modulate EEG activity in the required direction is preserved over time (6 months: Leins et al., 2007; 2 years: Gani, Birbaumer, & Strehl, 2009). The application requires an individual to have at least three electrodes attached to the head, which records, analyzes, and gives near instantaneous feedback based on the electrical activity of the brain; thus giving us the term—neurofeedback.

Current Treatments and Future Perspectives for ADHD Treatments

Recently, the 8-year follow up results from an NIMH-sponsored trial on different treatments for ADHD have been published; the NIMH-MTA (Molina et al., 2009). This study compared four different treatments in 579 children. These participants were randomly assigned to (a) systemic medication management, (b) multicomponent behavior therapy, (c) a combination of (a) and (b), and (d) usual community care. After 14 months, the first results initially showed that the medication and combined groups had the greatest improvements in ADHD and ODD symptoms. Half of these effects had dissipated 10 months after the treatment was completed. More important, after an

8-years follow-up, there were no longer any differences found between these four groups, indicating that the initial treatment subject randomization did not predict functioning 6 to 8 years later. This multicenter large-scale study clearly demonstrates a lack of long-term effects for stimulant medication, multicomponent behavior therapy or multimodal treatment (Molina et al., 2009). Furthermore, 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 treatment modality that has sufficient long-term efficacy for children with ADHD and that there is a need for new treatments with better long-term outcomes. Given that the skills and the treatment effects of neurofeedback have the potential to last for longer than 6 months (Leins et al., 2007) or even more than 2 years (Gani et al., 2009), this makes neurofeedback a very interesting and promising treatment for ADHD.

Neurofeedback is growing in popularity, yet there is considerable controversy in using it to treat some neurologically and psychologically based disorders. However, neurofeedback is currently utilized in clinical settings as an intervention to treat a number of neurological and psychological impairments. The published literature contains a significant amount of support for the use of neurofeedback techniques and the more recent literature is most impressive; however, it also has to be emphasized that neurofeedback is not a panacea and is at this moment only a well-investigated treatment of ADHD and epilepsy. In the following sections, we briefly review the history of neurofeedback in the treatment of ADHD, critically appraise its efficacy, and put this treatment into perspective compared to other treatment modalities.

HISTORICAL PERSPECTIVE OF NEUROFEEDBACK IN ADHD

The growing number of individuals diagnosed with ADHD and the desire to avoid

stimulant medication have brought increasing attention to neurofeedback treatment in recent years. Because standardized testing has become an integral part of the educational system, ADHD is more apparent and well documented. Despite the increasing number of published studies, there is controversy regarding the efficacy of neurofeedback for treating ADHD.

Lubar and Shouse were the first to publish on the use of neurofeedback in ADHD in 1976. In their initial study they tested the idea that neurofeedback training (Sensorimotor Rhythm training [SMR] frequency of 12–14 Hz), over the sensorimotor strip of the brain, could be used to help children with hyperkinesis. In this case study, the child was rewarded to produce SMR activity and at the same time inhibit theta activity (4–7 Hz). During neurofeedback, the child increased SMR by three times the amount of the initial recording along with a decreased classroom self-stimulation and out-of-seat behavior. Simultaneously, the child increased his sustained attention and schoolwork. This study employed an ABA design in which after this initial uptraining of SMR (A) the procedure was reversed in a blind fashion and the child was trained to inhibit the SMR and to increase the theta frequencies (B). After 35 sessions, the child had regressed completely to baseline measures in both his EEG and school performance. To validate the initial procedure, the child was trained again in the original protocol design with reinforcement for SMR and no reinforcement for production of theta (A). After 28 sessions of this protocol, the child's previous successes were regained. Finally, the medication was removed to measure sustained attention and the improvements were maintained (Lubar & Shouse, 1976). Follow-up after several years demonstrated that the child continued to do well without the readministration of the medication (Lubar, 1991). Although this study showed significant findings in a controlled ABA design, its power is limited because it is a case study.

In 1979, Shouse and Lubar replicated the previous 1976 study with a hyperkinetic group ($N=4$) of children. The same

experimental ABA design was used with the addition of the gradual withdrawal of Ritalin (Lubar & Shouse, 1976). This replication succeeded in that these children were able to regulate their EEG by changing their SMR levels (a doubling of SMR activity) along with behavioral improvements. These two initial studies inspired many subsequent studies, which investigated neurofeedback as a treatment for ADHD.

The first published controlled group study was conducted by Linden et al., who utilized a randomized design, comparing the effects of neurofeedback to a waiting list control group in 18 participants (Linden, Habib, & Radojevic, 1996). In this study, participants were required to increase beta and decrease theta. The results reflected improvements in measures of ADHD symptoms and IQ measures.

Four studies compared neurofeedback treatment with stimulant medication (Fuchs, Birbaumer, Lutzenberger, Gruzelier, & Kaiser, 2003; Monastra, Monastra, & George, 2002; Rossiter, 2004; Rossiter & La Vaque, 1995). These studies all used a variation of theta/beta ratio neurofeedback protocols at fronto-central locations and found that this technique demonstrated significant changes. Of interest, the effects of these studies demonstrate similar treatment responses between stimulant medication and neurofeedback. In the Monastra et al. (2002) study, all participants were medicated; however, when the medication was removed at the end of treatment, only the participants who had completed neurofeedback were able to sustain their improvements. The posttreatment quantitative electroencephalography (QEEG) measurements also showed a significant decrease in cortical slowing of the individuals who had completed the neurofeedback (Monastra et al., 2002).

The Monastra et al. (2002) study differed from the other three studies in that the participants were preselected based on a deviating theta/beta ratio. This most likely resulted in selecting the ADHD children who were to respond to theta/beta neurofeedback protocols. Furthermore, randomized allocation to treatment groups was

not used in these four studies. Randomized allocation has been shown to lead to an overestimation of the clinical effects, especially on hyperactivity, because clients can select the treatment they prefer (Arns, de Ridder, Strehl, Breteler, & Coenen, 2009). These are essential drawbacks in all four studies; consequently, no firm conclusions can be drawn on the comparability between neurofeedback and the use of medication in the treatment of ADHD. Nonetheless, these studies suggest that neurofeedback has potentially comparable effects to medication, but further controlled research is required to substantiate that conclusion.

Lévesque, Beauregard, and Mensour (2006) demonstrated in a randomized controlled study not only improvements on inattention and hyperactivity but also, above all, a normalization of brain activity in the anterior cingulate cortex measured with functional magnetic resonance imaging (fMRI) for the neurofeedback group only, suggesting neurofeedback does normalize underlying networks that have been shown to deviate in children with ADHD.

Heinrich, Gevensleben, Freisleder, Moll, and Rothenberger (2004) were the first to report positive results after Slow Cortical Potential (SCP) neurofeedback in the treatment of ADHD. SCP neurofeedback is different from the aforementioned approaches 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 in lieu of rewarding changes in specific frequency bands. Intriguingly, both the SCP neurofeedback and SMR neurofeedback approaches have been successfully used in treating epilepsy (for an overview, also see Egner & Serman, 2006), and both are suggested to regulate cortical excitability (Kleinnijenhuis, Arns, Spronk, & Breteler, 2007). Several studies have compared theta/beta training and SCP training both within-subject and between-subjects, and both neurofeedback approaches show comparable effects on the different aspects of ADHD such as inattention, hyperactivity, and impulsivity (Arns, de Ridder, Strehl, Breteler, & Coenen, 2009; Gevensleben,

Holl, Albrecht, Schlamp, et al., 2009; Leins et al., 2007).

Strehl and colleagues (Leins et al., 2007; Strehl et al., 2006) conducted a randomized controlled prospective study in which clients were randomized to either theta/beta neurofeedback or SCP neurofeedback. Both interventions showed similar efficacy in the treatment of ADHD, which were sustained over 6 months. Furthermore, the self-regulation skills were also preserved at follow-up.

Holtmann et al. (2009) conducted a study with a randomized controlled design where children were randomized either to theta/beta neurofeedback protocol or to a control group consisting of Captain's Log training. Thus both groups were exposed to an attention training program for the same amount of time (neurofeedback or Captain's Log), thereby controlling for indirect attention training. This study found a specific and clinically relevant improvement of impulsivity on a Go-NoGo task for the neurofeedback group alone. An additional observation worth noting is that in many studies parent rating scales were utilized. The rating scales improved for inattention, hyperactivity, and impulsivity; however, there was no significant interaction, suggesting the effects were similar for both groups.

In 2009, one of the largest multisite randomized controlled trials on neurofeedback in ADHD was published by Gevensleben (Gevensleben, Holl, Albrecht, Schlamp, et al., 2009). This study incorporated data from 94 participants and overcame many of the criticism from the past while incorporating strong methodological aspects, such as randomization, multicenter study, a large sample size, and a credible sham control consisting of attention training. Post-QEEG data from this sample already showed that the neurofeedback trained group—but not the control group—showed reduced EEG theta power (Gevensleben, Holl, Albrecht, Vogel, et al., 2009), thereby demonstrating the specificity of this intervention.

As suggested by Loo and Barkley (2005), possible alternative explanations for the efficacy of neurofeedback consist of the fact that children who are exposed to neurofeedback

spent more time with the therapist, as compared to a control group. Another explanation put forward by Loo and Barley is that neurofeedback training, itself, could be considered a form of cognitive-behavioral training. These observations suggest that it is not the actual training of EEG activity per se, causing the treatment effects. The two studies by Gevensleben et al. (Gevensleben, Holl, Albrecht, Schlamp, et al., 2009; Gevensleben, Holl, Albrecht, Vogel, et al., 2009) and the Holtmann et al. (2009) study controlled for both aspects. The (semiactive) control groups that were employed can be considered credible placebo control.

RATIONALE FOR NEUROFEEDBACK IN ADHD

Psychophysiological Basis of Diagnosis

Cerebral blood flow and metabolism, measured in fMRI, positron emission tomography, and single-photon emission computed tomography studies all support the electrical and chemical signaling systems that are the currency of information transfer in the brain. In contrast, the EEG, a measure of the electrical activity of the brain, may be a more direct indicator of brain function. Indeed, the EEG offers information that is of greater temporal resolution but at the cost of less spatial resolution. EEG research on ADHD has been conducted at many levels and suggests that EEG parameters can effectively distinguish between children with ADHD and normal controls. The EEG reflects the electrical activity of large populations of synchronized neurons, mostly cortical pyramidal neurons. Therefore, some diseases can be more easily identified with EEG than with functional imaging, especially when the disease manifests into a form of altered electrical brain activity, as in ADHD (Van der Stelt, van der Molen, Gunning, & Kok, 2001).

QEEG spectral analysis of the EEGs of children with ADHD has frequently shown increased levels of slow waves (predominantly theta) and decreased levels of relative beta activity when compared to the EEGs

of normal controls (Barry, Clarke, & Johnstone, 2003). In general, the abnormalities seem to be more pronounced in children with the combined type of ADHD than the inattentive ADHD (Barry et al., 2003; Chabot & Serfontein, 1996). A large-scale multicenter study (Monastra et al., 1999) as well as a meta-analysis (Boutros, Fraenkel, & Feingold, 2005) have concluded that excess theta is a robust biomarker for ADHD. The literature is less consistent about the decreased *absolute* beta in ADHD (Callaway, Halliday, & Naylor, 1983; Dykman, Ackerman, Oglesby, & Holcomb, 1982; Mann, Lubar, Zimmerman, Miller, & Muenchen, 1992; Matsuura et al., 1993), which was not found by several other studies (Barry, Clarke, Johnstone, McCarthy, & Selikowitz, 2009; Clarke, Barry, McCarthy, & Selikowitz, 2001; Lazzaro et al., 1999; Lazzaro et al., 1998) and was found to be increased in one study (Kuperman, Johnson, Arndt, Lindgren, & Wolraich, 1996).

These EEG deviations in ADHD have been interpreted as a maturational lag by some (Barry et al., 2003; Satterfield, Cantwell, Saul, Lesser, & Podosin, 1973), whereas others have interpreted the excess theta to be reflective of a labile vigilance regulation (Hegerl et al., 2008) or hypoarousal (Barry et al., 2009; Satterfield et al., 1973) with the latter two models also explaining why stimulant medication works in the treatment of ADHD.

Several studies have also investigated the differences in brain activity between responders and nonresponders to stimulant medication. Most of these studies have shown that distinct neurophysiological groupings within the behaviorally diagnosed subtypes exist. Most studies have shown that increased theta and/or theta/beta ratios are related to a favorable treatment outcome, and these do not relate to *DSM* subdiagnosis (Arns, de Ridder, Strehl, Breteler, & Coenen, 2008; Clarke, Barry, McCarthy, & Selikowitz, 2002; Clarke, Barry, McCarthy, Selikowitz, & Brown, 2002; Suffin & Emory, 1995) suggesting that within behavioral homogenous groups such as ADHD, neurophysiological subgroups exist and respond differentially to treatment.

These investigations provide sufficient evidence to conclude that not only is the electrical activity of the brain reflective of the condition of ADHD but dysregulation contributes to the presence of the condition. From this, it can be reasoned that operant conditioning to decrease dysregulation and alter electrical activity would not only be possible but would stand as a treatment option for the disorder.

STATUS OF NEUROFEEDBACK FOR ADHD

Treatment of ADHD with neurofeedback has gained promising empirical support in recent years (Arns et al., 2009; D. J. Fox, Tharp, & Fox, 2005; Lubar, 2003; Monastra et al., 2002). In addition, neurofeedback results in normalizations of neurophysiological patterns with QEEG (Doehnert, Brandeis, Straub, Steinhausen, & Drechsler, 2008; Gevensleben, Holl, Albrecht, Schlamp, et al., 2009), event-related potential (Heinrich et al., 2004; Holtmann et al., 2009; Kropotov et al., 2005), and fMRI (Levesque et al., 2006).

Historical Studies Suggesting Levels of Efficacy

Anecdotal reports and case studies are often the first steps to validating an intervention. However, to be fully accepted as empirical support they must pass rigorous clinical trials (La Vaque et al., 2002). In 2002, according to the efficacy task force criteria, established by the Association for Applied Psychophysiology and Biofeedback and the International Society for Neurofeedback and Research, neurofeedback applications based on the reviewed literature achieved Level 3: Probably Efficacious (La Vaque et al., 2002). The publication by Monastra et al. (2002) and the reviewed literature were catalysts for this recommendation. In 2002 it was suggested that to achieve “efficacious and specific levels,” studies should be conducted that will demonstrate neurofeedback to be “statistically

superior to credible sham therapy, pill, or alternative bona fide treatment in at least two independent research settings” (La Vaque et al., 2002).

Recent Studies Suggesting Levels of Efficacy

In 2005 Monastra et al. critically reviewed the literature and applied the APA guidelines for rating clinical efficacy (see Table 1). It was concluded that neurofeedback treatment for ADHD could be considered as Level 3: Probably Efficacious. However, in that same year Loo and Barkley (2005) published a review article in which they concluded that “the promise of EEG Biofeedback as a legitimate treatment cannot be fulfilled without studies that are scientifically rigorous” (p. 73). The main concerns they raised were the lack of well-controlled, randomized studies; the small group sizes; and the lack of proof that the EEG feedback is solely responsible for the clinical benefit and not nonspecific factors such as the additional time spent with a therapist or “cognitive training.” In 2006, Holtmann and Stadler concluded that EEG biofeedback has gained promising empirical support in recent years, but there is still a strong need for more empirically and methodologically sound evaluation studies.

Since 2005, new research has been published investigating the clinical efficacy of neurofeedback for the treatment of ADHD. A recently published meta-analysis on neurofeedback in ADHD by Arns et al. (2009) concluded that neurofeedback for ADHD met Level 5: Efficacious and Specific and that neurofeedback had large effect sizes (large clinically relevant effect) on the domains of impulsivity and inattention and a medium (medium clinically relevant) effect size on hyperactivity. The meta-analysis included 15 studies and 1,194 clients with ADHD, and of these six studies randomization was used. Two randomized controlled trials both published in 2009 were the key studies on the basis of which the efficacy rating could be lifted to Level 5, the randomized multicenter study from Gevensleben, Holl,

TABLE 1. Levels of efficacy (American Psychological Association).

Level 1	Not Empirically Supported	Supported only through anecdotal evidence or non-peer-reviewed case studies
Level 2	Possibly Efficacious	Shown to have a significant impact in <i>at least one</i> study, but the study lacked a randomized assignment between controls
Level 3	Probably Efficacious	Shown to produce positive effects in <i>more than one</i> clinical, observational wait list or within-subject or between-subject study
Level 4	Efficacious	Shown to be more effective than a no-treatment or placebo control group; the study must contain valid and clearly specified outcome measures, and it must be replicable by at least two independent researchers demonstrating the same degree of efficacy
Level 5	Efficacious and Specific	Shown to be statistically superior to credible placebo therapies or to actual treatments, and it must be shown as such in two or more independent studies

Albrecht, Vogel, et al. (2009) and the randomized study from Holtmann et al. (2009). Both of these studies used sound methodological designs, employed randomization, and implemented semiactive control groups, which can be considered a credible placebo control. The study by Gevensleben, Holl, Albrecht, Schlamp, et al. (2009) consisted of a large sample size ($N=94$). Therefore, in line with the suggestion made by LaVaque et al. (2002), neurofeedback was shown to be superior to a credible placebo control, which was demonstrated in two independent research settings, thereby meeting Level 5.

Long-Term Effects

Several studies have investigated long-term outcomes. In 2003 Lubar published data on 52 cases, which were followed for up to 10 years after neurofeedback treatment in a single clinical setting. The data involved a phone interview given by an interviewer who was uninformed to the treatment, using the Conner's rating scale of 16 behavioral categories. Most participants rated themselves as "very much improved or more change." Because the interview was "blinded," it was performed objectively and had the advantage that the participants interviewed were chosen randomly from a

group of more than 1,000 cases but represented those who had been out of treatment the longest. Unfortunately, this was an uncontrolled study with no control group, thereby it cannot be ruled out that these effects were simply because of maturation. However, the following studies employed a control group, and follow-up was performed for both the experimental and control groups. Heinrich et al. (2004) performed a 3-month follow-up for the SCP group and found all measures improving further (Arns et al., 2009). The study of Strehl and colleagues that measured scores in impulsivity, inattention, and hyperactivity in a 6-month follow-up were shown to improve even further as compared to the end of treatment (Leins et al., 2007). A 2-year follow-up for this study 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 (Gani et al., 2008). In addition, EEG-self regulation skills turned out to be still preserved, indicating that these children were still able to regulate their brain activity successfully. The 6-month follow-up data from the large multicenter study by Gevensleben, Holl, Albrecht, Schlamp, et al. (2009) are currently being reviewed for publication, but according to these data, "behavioral improvements induced by NF training in

children with ADHD were sustained at 6-month follow-up” (H. Heinrich, personal communication, January 14, 2010).

Taken from the limited data available, it can be concluded that the clinical effects of neurofeedback remain stable and may improve further over 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, larger scale, controlled studies with longer follow-up are required to investigate this claim further.

Pre- and Post-QEEG Differences

It is often stated that studies fail to report pre- and post-QEEG differences because the EEG is the basis of treatment in neurofeedback (e.g., see Loo & Barkley, 2005). However, this is not a credible reason to criticize the clinical efficacy of neurofeedback or any other treatment. The primary question is, “Does it work?” The secondary question is, “How does it work?” Several studies found a normalization of neurophysiological patterns with QEEG (Doehnert et al., 2008; Gevensleben et al., 2009), event-related potential (Heinrich et al., 2004; Holtmann et al., 2009; Kropotov et al., 2005), and fMRI (Levesque et al., 2006). For rating clinical efficacy this is of no concern, therefore we do not discuss this issue further in this paper.

Neurofeedback versus Stimulant Medication

As was shown in the overview of neurofeedback studies, four studies directly compared neurofeedback to stimulant medication in the treatment of ADHD. Although the results of these four studies hint at the fact that neurofeedback demonstrates similar effects as compared to stimulant medication, these studies suffer some methodological issues making it impossible to draw that conclusion, at present. Better controlled studies (at least employing randomized

group assignments) are required to further support that conclusion.

Recently, a meta-analysis was conducted on two types of stimulant medication in the treatment of ADHD by Faraone and Buitelaar (2009). They compared the effects of methylphenidate and amphetamines on the domains of inattention and impulsivity/hyperactivity. They found that amphetamines are more efficacious in general as compared to methylphenidate. Furthermore, there was a significant publication bias for methylphenidate studies, which means that less efficacious studies on medication have not been published. The effect size (ES) for hyperactivity-impulsivity was 1.01 and the ES for inattention was 0.84.

These data allow an indirect comparison of the efficacy of neurofeedback and medication, as an ES is a standardized measure that is obtained from a comparison of the effects of multiple studies. Comparing these data to the meta-analysis on neurofeedback (Arns et al., 2009), it means that neurofeedback and methylphenidate have similar effects on inattention (ES NF = 0.81; ES MPH = 0.84) and for impulsivity/hyperactivity, medication has a higher ES (ES NF = 0.4/0.69; ES MPH = 1.01). The meta-analysis on neurofeedback also concluded that the effects on hyperactivity were most susceptible to indirect treatment effects and for controlled studies the ES was just significant. Therefore, based on the current state of research we conclude that neurofeedback is best indicated when the main clinical problems are inattention and impulsivity. When the main clinical problems are in the hyperactivity domain, medication is most likely a better treatment option. Lubar also previously suggested this in 1991:

Children with pure ADHD respond extremely well to EEG biofeedback training. Children with hyperkinesis, especially if they are good responders to stimulant medication, are also candidates for biofeedback treatment, but they may also require medication at least during the initial part of the treatment in order to obtain good control of their disorder. (p. 205)

CONCLUSIONS

One of the weaknesses within the neurofeedback studies is that they do not graph learning curves, so there is minimal evidence of day-to-day changes documented, in the parameters being trained in the neurofeedback programs that are being used. It should be noted that learning curves were presented in the Lubar studies described, and additionally, has been suggested by Lubar that in all studies the trained parameters be graphed over sessions to determine, if in fact, learning has taken place. One of the primary criticisms remains that there are many studies that employ small sample sizes. However, because of the publication of some very recent and sound methodological randomized controlled trials and a meta-analysis, many potential confounding factors have been addressed, and the clinical effects of neurofeedback in the treatment of ADHD can be regarded as clinically meaningful. The two independent randomized controlled trials, from Gevensleben, Holl, Albrecht, Vogel, et al. (2009) and Holtmann et al. (2009), have shown neurofeedback to be superior to a (semiactive) control group. The semiactive control group in these studies can be regarded as a credible sham control providing an equal level of cognitive training and client-therapist interaction. Therefore, in line with the guidelines for rating clinical efficacy, it can be concluded that neurofeedback treatment for ADHD can be considered Efficacious and Specific (Level 5).

The Monastra study (Monastra et al., 2002) employed preselection of participants based on deviating theta/beta ratios. This study was also excluded from the meta-analysis because it showed the highest ES for inattention (2.22) and hyperactivity (1.22) and therefore, most contributed to the heterogeneity of variance, suggesting this study showed higher efficacy due to preselection of deviating theta/beta ratio (Arns et al., 2009). This large study demonstrated that QEEG-based preselection could potentially improve the therapeutic outcome. Given that the three investigated neurofeedback treatment protocols (fronto-central theta/beta, central SMR/theta, and slow cortical potentials) have

shown similar efficacy, we advise that clinicians performing QEEG-based neurofeedback in the treatment of ADHD to utilize these protocols based on QEEG findings when determined appropriate to remain in line with the evidence-based literature (Arns et al., 2009).

These findings have led the current authors to deem it necessary to provide an updated position paper on neurofeedback for the treatment of ADHD that is accepted by the International Society for Neurofeedback and Research. Our conclusions are that neurofeedback not only is a suitable intervention for those diagnosed with ADHD but also may be the preferred choice of interventions for some individuals. The conclusions are based on solid scientific evidence and demonstrated by clinical data collected from multiple sites and studies across the world. Therefore this position paper, in our opinion, demonstrates the rationale and the necessary support for neurofeedback to be recognized, not only as legitimate and scientific but also an evidence based intervention for the treatment of ADHD.

The following is recommended:

1. Neurofeedback is a safe and efficacious treatment intervention for ADHD meeting the rating of Level 5: Efficacious and Specific.
2. Neurofeedback in the treatment of ADHD has been shown to have long-term effects, lasting from 3 to 6 months. More research is required to investigate the effects after 3 to 5 years (of treatment?) similar to the NIMH-MTA trial.
3. The effects of neurofeedback appear to have similar effects to stimulant medication for inattention and impulsivity, but more controlled and randomized studies are required to further support this observation.
4. Additional research is required to investigate the working mechanism of neurofeedback.
5. Given that neurofeedback currently requires multiple treatment sessions, further research should be directed toward improving neurofeedback treatment to

- require fewer treatment sessions (e.g., LORETA neurofeedback, ICA neurofeedback, Z-score neurofeedback).
6. Neurofeedback is efficacious when inattention and impulsivity are the main problems. When the main complaint is hyperactivity, medication is possibly a better choice given the limited success of neurofeedback in this domain. Controlled and randomized studies are required to further substantiate this claim.
 7. No differences in neurofeedback efficacy have been found between medicated and nonmedicated children; therefore, neurofeedback can be utilized in combination with a medication regimen.
 8. Licensed health care providers should take necessary educational prerequisites to understand the methods and proper implementation of neurofeedback and its appropriateness for the treatment of ADHD.
 9. When appropriately trained in the planning, implementation, and monitoring of neurofeedback, the licensed health care professional should consider including neurofeedback as a potential modality of treatment.

REFERENCES

- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders, 4th edition, text revision*. Washington, DC: Author.
- 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. *Clinical EEG and Neuroscience, 40*, 180–189.
- Arns, M., Gunkelman, J., Breteler, M., & Spronk, D. (2008). EEG phenotypes predict treatment outcome to stimulants in children with ADHD. *Journal of Integrative Neuroscience, 7*, 421–438.
- Barry, R. J., Clarke, A. R., & Johnstone, S. J. (2003). A review of electrophysiology in attention-deficit/hyperactivity disorder: I. Qualitative and quantitative electroencephalography. *Clinical Neurophysiology, 114*, 171–183.
- Barry, R. J., Clarke, A. R., Johnstone, S. J., McCarthy, R., & Selikowitz, M. (2009). Electroencephalogram theta/beta ratio and arousal in attention-deficit/hyperactivity disorder: Evidence of independent processes. *Biological Psychiatry, 66*, 398–401.
- Boutros, N., Fraenkel, L., & Feingold, A. (2005). A four-step approach for developing diagnostic tests in psychiatry: EEG in ADHD as a test case. *Journal of Neuropsychiatry and Clinical Neuroscience, 17*, 455–464.
- Callaway, E., Halliday, R., & Naylor, H. (1983). Hyperactive children's event-related potentials fail to support underarousal and maturational-lag theories. *Archives of General Psychiatry, 40*, 1243–1248.
- Chabot, R. J., & Serfontein, G. (1996). Quantitative electroencephalographic profiles of children with attention deficit disorder. *Biological Psychiatry, 40*, 951–963.
- Clarke, A. R., Barry, R. J., McCarthy, R., & Selikowitz, M. (2001). Electroencephalogram differences in two subtypes of attention-deficit/hyperactivity disorder. *Psychophysiology, 38*, 212–221.
- Clarke, A. R., Barry, R. J., McCarthy, R., & Selikowitz, M. (2002). EEG analysis of children with attention-deficit/hyperactivity disorder and comorbid reading disabilities. *Journal of Learning Disabilities, 35*, 276.
- Clarke, A. R., Barry, R. J., McCarthy, R., Selikowitz, M., & Brown, C. R. (2002). EEG evidence for a new conceptualisation of attention deficit hyperactivity disorder. *Clinical Neurophysiology, 113*, 1036–1044.
- 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? *Journal of Neural Transmission, 115*, 1445–1456.
- Dykman, R. A., Ackerman, P. T., Oglesby, D. M., & Holcomb, P. J. (1982). Autonomic responsivity during visual search of hyperactive and reading-disabled children. *The Pavlovian Journal of Biological Science, 17*(3), 150–157.
- Egner, T., & Serman, M. B. (2006). Neurofeedback treatment of epilepsy: From basic rationale to practical application. *Expert Review of Neurotherapeutics, 6*, 247–257.
- Erk, R. R. (1995). The evolution of attention deficit disorders terminology. *Elementary School Guidance & Counseling, 29*, 243–248.
- Faraone, S. V., & Buitelaar, J. (2009). Comparing the efficacy of stimulants for ADHD in children and adolescents using meta-analysis. *European Child & Adolescent Psychiatry, 14*(1), 353–364.
- Fox, D. J., Tharp, D. F., & Fox, L. C. (2005). Neurofeedback: An alternative and efficacious treatment for attention deficit hyperactivity disorder. *Applied Psychophysiology and Biofeedback, 30*, 365–373.

- Fox, S. S., & Rudell, A. P. (1968). Operant controlled neural event: Formal and systematic approach to electrical coding of behavior in brain. *Science*, *162*, 1299–1302.
- Fuchs, T., Birbaumer, N., Lutzenberger, W., Gruzelier, J. H., & Kaiser, J. (2003). Neurofeedback treatment for attention-deficit/hyperactivity disorder in children: A comparison with methylphenidate. *Applied Psychophysiology and Biofeedback*, *28*, 1–12.
- Gani, C., Birbaumer, N., & Strehl, U. (2009). Long term effects after feedback of slow cortical potentials and of theta-beta-amplitudes in children with attentiondeficit/hyperactivity disorder (ADHD). *International Journal of Bioelectromagnetics*, *10*, 209–232.
- Gevensleben, H., Holl, B., Albrecht, B., Schlamp, D., Kratz, O., Studer, P., et al. (2009). Distinct EEG effects related to neurofeedback training in children with ADHD: A randomized controlled trial. *International Journal of Psychophysiology*, *74*, 149157.
- Gevensleben, H., Holl, B., Albrecht, B., Vogel, C., Schlamp, D., Kratz, O., et al. (2009). Is neurofeedback an efficacious treatment for ADHD? A randomised controlled clinical trial. *Journal of Child Psychology and Psychiatry*, *50*, 780–789.
- Hegerl, U., Stein, M., Mulert, C., Mergl, R., Olbrich, S., Dichgans, E., et al. (2008). Eeg-Vigilance differences between patients with borderline personality disorder, patients with obsessive-compulsive disorder and healthy controls. *European Archives of Psychiatry and Clinical Neuroscience*, *258*, 137–143.
- Heinrich, H., Gevensleben, H., Freisleder, F. J., Moll, G. H., & Rothenberger, A. (2004). Training of slow cortical potentials in attention-deficit/hyperactivity disorder: Evidence for positive behavioral and neurophysiological effects. *Biological Psychiatry*, *55*, 772–775.
- Hermens, D. F., Rowe, D. L., Gordon, E., & Williams, L. M. (2006). Integrative neuroscience approach to predict ADHD stimulant response. *Expert Review of Neurotherapeutics*, *6*, 753–763.
- Hetzler, B. E., Rosenfeld, J. P., Birkel, P. A., & Antoinetti, D. N. (1977). Characteristics of operant control of centrally evoked potentials in rats. *Physiology & Behavior*, *19*, 527–534.
- Holtmann, M., Grasmann, D., Cionek-Szpak, E., Hager, V., Panzner, N., Beyer, A., et al. (2009). Spezifische wirksamkeit von neurofeedback auf die impulsivität bei ADHS. *Kindheit Und Entwicklung*, *18*, 95–204.
- Jasper, H., & Shagass, C. (1941). Conditioning the occipital alpha rhythm in man. *Journal of Experimental Psychology*, *28*, 373–387.
- Kleinnijenhuis, M., Arns, M., Spronk, D., & Breteler, R. (2007). Comparison of discrete-trial based SMR and SCP training and the interrelationship between SCP and SMR networks: Implications for brain-computer interfaces and neurofeedback. *Journal of Neurotherapy*, *11*(4), 19–35.
- Kropotov, J. D., Grin-Yatsenko, V. A., Ponomarev, V. A., Chutko, L. S., Yakovenko, E. A., & Nikishena, I. S. (2005). Erps correlates of EEG relative beta training in ADHD children. *International Journal of Psychophysiology*, *55*, 23–34.
- Kuperman, S., Johnson, B., Arndt, S., Lindgren, S., & Wolraich, M. (1996). Quantitative EEG differences in a nonclinical sample of children with ADHD and undifferentiated ADD. *Journal of the American Academy of Child and Adolescent Psychiatry*, *35*, 1009–1017.
- La Vaque, T. J., Hammond, D. C., Trudeau, D., Monastra, V. J., Perry, J., Lehrer, P., et al. (2002). Template for developing guidelines for the evaluation of the clinical efficacy of psychophysiological interventions. *Applied Psychophysiology and Biofeedback*, *27*, 273–281.
- 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.
- Lazzaro, I., Gordon, E., Whitmont, S., Plahn, M., Li, W., Clarke, S., et al. (1998). Quantified EEG activity in adolescent attention deficit hyperactivity disorder. *Clinical Electroencephalography*, *29*(1), 37–42.
- Leins, U., Goth, G., Hinterberger, T., Klinger, C., Rumpf, N., & Strehl, U. (2007). Neurofeedback for children with ADHD: A comparison of SCP and theta/beta protocols. *Applied Psychophysiology and Biofeedback*, *32*, 73–88.
- Lévesque, J., Beauregard, M., & Mensour, B. (2006). Effect of neurofeedback training on the neural substrates of selective attention in children with attention-deficit/hyperactivity disorder: A functional magnetic resonance imaging study. *Neuroscience Letters*, *394*, 216–221.
- 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.
- Loo, S. K., & Barkley, R. A. (2005). Clinical utility of EEG in attention deficit hyperactivity disorder. *Applied Neuropsychology*, *12*, 64–76.
- Lubar, J. F. (1991). Discourse on the development of EEG diagnostics and biofeedback for attention-deficit/hyperactivity disorders. *Applied Psychophysiology and Biofeedback*, *16*, 201–225.
- Lubar, J. F. (2003). Neurofeedback for the management of attention deficit disorders. In M. S. Schwartz &

- F. Andrasik (Eds.), *Biofeedback: A practitioner's guide* (pp. 409–437). New York: Guilford.
- Lubar, J. F., & Shouse, M. N. (1976). EEG and behavioral changes in a hyperkinetic child concurrent with training of the sensorimotor rhythm (SMR): A preliminary report. *Biofeedback and Self Regulation, 1*, 293–306.
- 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.
- Matsuura, M., Okubo, Y., Toru, M., Kojima, T., He, Y., Hou, Y., et al. (1993). A cross-national EEG study of children with emotional and behavioral problems: A WHO collaborative study in the western pacific region. *Biological Psychiatry, 34*, 59–65.
- Molina, B. S., Hinshaw, S. P., Swanson, J. M., Arnold, L. E., Vitiello, B., Jensen, P. S., et al. (2009). The MTA at 8 years: Prospective follow-up of children treated for combined-type ADHD in a multisite study. *Journal of the American Academy of Child and Adolescent Psychiatry, 48*, 484–500.
- Monastra, 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.
- Monastra, V. J., Lynn, S., Linden, M., Lubar, J. F., Gruzelier, J., & La Vaque, T. J. (2005). Electroencephalographic biofeedback in the treatment of attention-deficit/hyperactivity disorder. *Applied Psychophysiology and Biofeedback, 30*, 95–114.
- Monastra, V. J., Monastra, D. M., & George, S. (2002). The effects of stimulant therapy, EEG biofeedback, and parenting style on the primary symptoms of attention-deficit/hyperactivity disorder. *Applied Psychophysiology and Biofeedback, 27*, 231–249.
- Rosenfeld, J. P., Rudell, A. P., & Fox, S. S. (1969). Operant control of neural events in humans. *Science, 165*, 821–823.
- Rossiter, T. (2004). The effectiveness of neurofeedback and stimulant drugs in treating AD/HD: Part I. Review of methodological issues. *Applied Psychophysiology and Biofeedback, 29*, 95–112.
- 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.
- Rowland, A. S., Lesesne, C. A., & Abramowitz, A. J. (2002). The epidemiology of attention-deficit/hyperactivity disorder (ADHD): A public health view. *Mental Retardation and Developmental Disabilities Research Reviews, 8*, 162–170.
- Satterfield, J. H., Cantwell, D. P., Saul, R. E., Lesser, L. I., & Podosin, R. L. (1973). Response to stimulant drug treatment in hyperactive children: Prediction from EEG and neurological findings. *Journal of Autism and Child Schizophrenia, 3*, 36–48.
- Shouse, M. N., & Lubar, J. F. (1979). Operant conditioning of EEG rhythms and Ritalin in the treatment of hyperkinesis. *Biofeedback and Self Regulation, 4*, 299–312.
- Serman, M. B. (1996). Physiological origins and functional correlates of EEG rhythmic activities: Implications for self-regulation. *Biofeedback and Self Regulation, 21*, 3–33.
- 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*, e1530–e1540.
- Suffin, S. C., & Emory, W. H. (1995). Neurometric subgroups in attentional and affective disorders and their association with pharmacotherapeutic outcome. *Clinical Electroencephalography, 26*, 76–83.
- Thatcher, R. W. (2000). EEG operant conditioning (biofeedback) and traumatic brain injury. *Clinical Electroencephalography, 31*(1), 38–44.
- Van der Stelt, O., van der Molen, M., Gunning, W. B., & Kok, A. (2001). Neuroelectrical signs of selective attention to color in boys with attention-deficit hyperactivity disorder. *Cognitive Brain Research, 12*, 245–264.
- World Health Organization. (1992). *The ICD-10 Classification of mental and behavioural disorders: Clinical descriptions and diagnostic guidelines*. Geneva: Author.