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Neurofeedback: An Alternative and Efficacious Treatment for Attention Deficit Hyperactivity Disorder

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Current research has shown that neurofeedback, or EEG biofeedback as it is sometimes called, is a viable alternative treatment for Attention Deficit Hyperactivity Disorder (ADHD). The aim of this article is to illustrate current treatment modalities(s), compare them to neurofeedback, and present the benefits of utilizing this method of treatment to control and potentially alleviate the symptoms of ADHD. In addition, this article examines the prevalence rates and possible etiology of ADHD, the factors associated with ADHD and brain dysfunction, the current pharmacological treatments of ADHD, Ritalin, and the potential risks and side effects. Behavior modification and cognitive behavioral treatment for ADHD is discussed as well. Lastly, a brief history of the study of neurofeedback, treatment successes and clinical benefits, comparisons to medication, and limitations are presented.

KEY WORDS: neurofeedback; attention deficit hyperactivity disorder; ADHD; EEG biofeedback; ritalin.

Attention Deficit Hyperactivity Disorder (ADHD) is a psychological disorder that presents with a persistent pattern of inattention and/or hyperactivity–impulsivity that is a predominant characteristic of an individual's behavior. According to the Diagnostic Statistical Manual of Mental Disorders, fourth edition, Text Revision (*DSM-IV-TR*; American Psychiatric Association, 2000), the symptoms must have been present and caused impairment before the age of 7, evident in two or more settings, such as school and home, and the impairment contributes to social, academic, or occupational dysfunction. These symptoms of inattention and/or hyperactivity-impulsivity must be present for at least 6 months. The *DSM-IV-TR* breaks ADHD into three subtypes: (1) Combined Type, which includes symptoms of both inattention and hyperactivity–impulsivity; (2) Predominantly Inattentive Type, which includes symptoms of inattention; and (3) Predominantly Hyperactive–Impulsive Type, which includes symptoms of hyperactivity–impulsivity. This disorder has become one of the most frequently treated disorders in childhood (Cantwell, 1996) and in the last 10 years the number of children being diagnosed with this disorder has risen to four times the original prevalence rate (Brownell & Yogendran, 2001).

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According to the Multimodal Treatment Study of ADHD, which is funded by the National Institute of Mental Health and the Office of Special Education Programs of the U.S. Department of Education, this disorder affects approximately 3–5% of school-aged children (The MTA Cooperative Group, 1999). Hyperactivity is predominantly seen in younger children, and it is expected to decrease in early adolescence, possibly disappearing by late adolescence. However, between 30 and 70% of children diagnosed with ADHD have symptoms that endure into adolescence and adulthood (Bellak & Black, 1992; Weiss & Hechtnian, 1993). This has been found to be due to a failure of those children diagnosed with ADHD to show normal right-greater-than-left asymmetry in the frontal lobes of the brain when assessed using an Electroencephalograph (EEG; Hynd, Hem, Voeller, & Marshall, 1991). The frontal lobe controls the manner in which feedback from the environment is interpreted, the ability to respond to different situations in a variety of ways, risk-taking, compliance and noncompliance with rules, and associated learning (using external cues to help guide behavior). To further substantiate this finding, Mann, Lubar, Zimmerman, Miller, and Muenchen (1992) found that boys diagnosed with ADHD showed significantly greater slow wave activity (theta) and less fast wave activity (beta) predominantly in the frontal regions of the brain.

When an individual without ADHD is presented with an attentional task, such as reading, simple arithmetic, or listening to a story, his/her EEG usually shifts to the beta frequency band with an increase in the frontal, more specifically, the right frontal region. In comparison, individuals diagnosed with ADHD shift down into a slow frequency (theta) without any significant increase in frontal activity (Lubar, 1991; Mann et al., 1992). The slow frequency activity is related to the mental wandering, nonvigilance, and unfocused thought. Individuals with ADHD tend to drift in their thoughts as opposed to being able to centralize their attention and grasp information in a way that those without this diagnosis are able to do.

It has been widely held that ADHD is caused by a chemical imbalance in the brain, genetic abnormalities, exposure to toxins during pregnancy and during childhood, complications during pregnancy such as oxygen deprivation, low birth weight, and low Omega-3 fatty oils (Elia, Ambrosini, & Rapoport, 1999). Several correlational studies have attempted to identify the cause(s) of ADHD, such as food additives and dietary sugar (Feingold, 1973, 1975), blood lead levels (David, 1974), allergies (Marshall, 1989), and smoking and/or alcohol use during pregnancy (Barkley, 1990). Additional studies have found central symptoms of ADHD to be associated with metabolic (Zametkin & Rapoport, 1987), circulatory (Amen, Paldi, & Thisted, 1993), and electrophysiological abnormalities (Chabot, Merkin, Wood, Davenport, & Serfontein, 1996; Chabot & Serfontein, 1996; Mann et al., 1992). In addition to the central characteristics of ADHD, inattention and hyperactive–impulsive symptoms, secondary characteristics include learning disorders, tic disorders, conduct disorders, anxiety, depression, and other mood disorders (Spencer, Biederman, & Wilen, 1999). The prevalence of the secondary symptoms co-occurring with the central symptoms ranges from 50 to 90% (Barkley, 1998; Spencer et al., 1999). Considering the intense impact on academic, social, family, and vocational functioning, a great amount of effort has been directed at developing effective treatments for ADHD. Before any of these treatments can have an effect, the basis of ADHD must be explored and identified.

Attention Deficit Hyperactivity Disorder has been associated with dysfunction in dopaminergic, and possibly, noradrenergic cortico-subcortical networks related to executive

functioning and the regulation of behavior. Additionally, children with ADHD appear to have a central nervous system (CNS) dysfunction that has been characterized by a maturational-lag (Mann et al., 1992) or cortical underarousal (Lubar, 1991). Research over the last 30 years has identified differences between ADHD and non-ADHD children having a surplus of slow wave activity, mostly in the delta and theta bands, and deficiencies of alpha and beta activities (Chabot & Serfontein, 1996; Clarke et al., 1998; Clarke, Barry, McCarthy, Selikowitz, & Brown, 2001a, 2001b, 2002; Lazzaro et al., 1998; Mann et al., 1992; Satterfield, Cantwell, Lesser, & Podosin, 1972). Beta activity has been found to increase during both physical and mental activities (Ackerman, Dykman, Oglesby, & Newton, 1994, 1995), and the research has shown that children diagnosed with ADHD have lower beta activity during cognitive tasks (Lubar, 1991; Mann et al., 1992). Noting all the neurophysiological factors mentioned above, ADHD is considered a psychiatric disorder, diagnosed on the basis of behavioral evidence. The primary treatment for ADHD consists of medication. The most popular of these medications is Ritalin (Methylphenidate).

ATTENTION DEFICIT HYPERACTIVITY DISORDER AND MEDICATION

The treatment of ADHD with medication is used under the assumption that symptoms can be assuaged if neuroendrocrine and or neurotransmitter changes can be achieved (Barkley, 1990; Bradley, 1937; Solanto, 1998; Volkow et al., 2001). Stimulant medications, such as Ritalin, Concerta, Methylin, Dexedrine, and Adderall are prescribed for 600,000 to 1 million school children in the United States. Published studies indicate that between 70 and 80% of ADHD children respond favorably to psychostimulants, as compared to over 35% that improve with placebos (Barkley, 1990). Medication has been found to have no effect on 25–40% of children with this disorder. That equates to approximately 150,000 to 400,000 children who cannot be helped by standard means of treatment (Swanson et al., 1993).

Neurologically, those individuals identified as hypoaroused, which is identified by high theta and low beta and delta activity, regardless of whether they have been diagnosed with combined or inattentive types of ADHD, may be more likely to respond to stimulant medications than those identified with a maturational-lag, which is identified by increased slow wave activity (e.g., delta and theta) and deficiencies in fast wave activity (e.g., beta; Clarke et al., 2002b). Monastra, Monastra, and George (2002) found that after a year of pharmacological treatment, the beneficial effects of Ritalin were eliminated when participants were retested without medication 1 year later. This finding is consistent with the summarized research on stimulant therapy and ADHD (Barkley, 1998). It appears that stimulant therapy "would appear to constitute a type of prophylactic intervention, reducing or preventing the expression of symptoms without causing an enduring change in the underlying neuropathy of ADHD" (Monastra et al., 2002, p. 245).

Until recently, the mechanisms of action regarding Ritalin were not known, though it has been prescribed for the last 50 years. It has been recently found that Ritalin and other stimulants interrupt the recycling or "reuptake" of dopamine in the brain by blocking dopamine transporters. Dopamine transporters mediate the uptake of dopamine into neurons and are a major target for various pharmacologically active drugs and environmental toxins. By blocking these dopamine transporters the brain is better able to transmit a clearer signal,

which provides the individual with an increased ability to focus their attention so he/she is not as easily distracted (Solanto, 1998; Volkow et al., 2001).

Though we now know the mechanisms of action for stimulants, pharmacological treatment for ADHD has failed to show that the wide range of clinical problems that accompany this disorder, such as cognition, academic achievement, and social skills, are attenuated by this type of treatment (Bennett, Brown, Craver, & Anderson, 1999; Brown & Sawyer, 1988; National Institute of Health, 1998). Additional problems that limit the effectiveness of medication include long-term compliance rates, especially among families of low socioeconomic status (Barkley, 1990) and adolescents, whether the medication has been helpful or not. It has been estimated that only 30–40% of children with ADHD "grow out of it" by late adolescence or early adult years. The remaining 60–70% continue to experience significant ADHD symptoms that impact their academic, vocational, emotional, and/or social functioning (Weiss & Hechtnian, 1993). Medication does not lead to significant improvement in reading, athletic or gaming skills, pro-active social skills, or learning other than improved attending. There is no evidence of long-term adjustment such as improved academic achievement or reduction in antisocial behaviors or negative interactions with law enforcement (Swanson et al., 1993).

ATTENTION DEFICIT HYPERACTIVITY DISORDER MEDICATION SIDE EFFECTS

There are several known side effects that occur in 20–50% of individuals taking psychostimulant medication, such as heachaches, anxiety, irritability, stomach aches, decreased appetite, insomnia, and headaches (Goldstein & Goldstein, 1990). According to Whalen and Henker (1991), there are a variety of side effects and limitations of stimulant medication, such as growth stunts unless medication vacations are taken, short length of action (4–5 hr) that requires careful planning of administration to coincide with school and overnight trips, cardiovascular problems, self-esteem issues of having to take medication, tics, Tourette's Syndrome, and in a small group of those who take the medication, negative physiological side effects similar to those seen in amphetamine use (i.e., aggression, nausea, itching, etc.).

To date no medication has been found that creates long-term improvement in children with ADHD. The major area of difficulty is that once the treatment is ceased, these children return to the original level of deficit (Barkley, 1992, 1998). Several studies have shown that long-term sustained benefits of EEG biofeedback or neurofeedback show significant improvement on behavioral and neuropsychological measures, as well as increased cortical arousal in individuals diagnosed with ADHD (Linden, Habib, & Radojevic, 1996; Lubar et al., 1995; Monastra et al., 2002; Rossieter & LaVaque, 1995; Thompson & Thompson, 1998).

OTHER TREATMENT OPTIONS FOR ATTENTION DEFICIT HYPERACTIVITY DISORDER

Behavior modification is another widely used treatment for ADHD. This type of treatment usually includes both parents and teachers. Utilizing behavior modification with stimulant medication creates a more comprehensive approach to treatment. However, a

substantial number of children do not respond to this type of treatment for several reasons: (1) training is difficult to generalize to different situations, (2) there is little to no carryover from home to the classroom and vice versa, (3) once treatment is terminated the behaviors usually return, and (4) it requires a high degree of cooperation between parents and teachers (Barabasz, 1987). Firestone, Kelly, Goodman, and Davey (1981) found that 50% of parents fail to continue behavior modification treatment with an ADHD child. Cognitive–Behavioral Therapy has also been used with children with ADHD. This type of treatment centers around self-talk coping and is expected to generalize to a wide range of situations. However, Cognitive–Behavioral Therapy has shown to have few positive outcomes or has failed to demonstrate any lasting effects (Conte, 1991; Gaddes & Edgell, 1994).

ATTENTION DEFICIT HYPERACTIVITY DISORDER AND NEUROFEEDBACK

Neurofeedback works to help the participant modify brainwave activity to improve attention, reduce impulsivity, control hyperactive behaviors, and produce long-term change (Table I) In 1976, Lubar and Shouse utilized operant conditioning techniques to reinforce specific types of electrophysiological activity for the purpose of treating symptoms of ADHD. They provided participants with visual and auditory "feedback" for certain neuronal responses and showed reduced hyperactive behavior and improved attention.

This was later followed by two controlled studies. The first was composed of 46 participants who self-selected either a stimulant (Ritalin) or neurofeedback treatment group. The participants underwent 20 neurofeedback sessions over a 3-month period. Pre- and posttreatments assessments were conducted utilizing behavior rating scales and the Test of Variables of Attention (TOVA). Results showed that both groups made significant improvement on the dependent measures, and no significant difference was detected between the two groups. In addition, the results generalized beyond the experimental environment and improvement was observed as symptoms reduced in the participants' daily lives (Rossieter & LaVaque, 1995). On the basis of this finding, the researchers concluded that neurofeedback could be an effective tool for treating ADHD. The second controlled study utilized a randomized design and compared the effects of 40 neurofeedback sessions with a control group. Results showed improvement on a measure of intelligence and reduced ADHD symptoms on a behavior rating scale in the neurofeedback group (Linden et al., 1996).

A review of over 200 children who were treated with neurofeedback for ADD/ADHD concluded that this mode of treatment provided significant and "dramatic" clinical improvements (Chartier & Kelly, 1991). Gaddes and Edgell (1994) reported that 80% of children with ADHD who were treated with neurofeedback showed significant measurable improvements in IQ tests, standardized tests of achievement, and teacher/parent ratings of behavior, and the effects were maintained at long-term follow-up. These results show that neurofeedback provides attentional and intellectual improvements. A study designed to assess the effectiveness of neurofeedback and Ritalin in two separate groups was conducted and found that both the medication and neurofeedback groups showed significant improvements on all four scales of the TOVA (Fuchs et al., 2003). This result, as well as those reported previously, supports the use of neurofeedback as an efficacious treatment for ADHD.

Table I. Efficacy Studies Utilizing Neurofeedback

Citation	Research design	Treatment modality	Outcome
Lubar and Shouse (1976)	Operant conditioning techniques to reinforce specific types of electrophysiological activity to treat central ADHD symptoms	Neurofeedback used to increase SMR or beta while decreasing theta	Reduction of impulsivity and hyperactivity symptoms while improving attention
Chartier and Kelly (1991)	Reviewed the effects of neurofeedback for ADD/ADHD on over 200 children	Review of literature	They found that neurofeedback training provided significant and sometimes "dramatic" clinical improvements in children with ADD/ADHD
Rossieter and LaVaque (1995)	Allowed participants to decide whether to attend a neurofeedback group or a stimulant therapy group (titrated Ritalin)	Twenty sessions of neurofeedback were provided over a 3-month period. Pre- and post-assessment were taken using the Test of Variables of Attention (TOVA)	Both groups showed a benefit from treatment and no significant differences were found between Ritalin and the neurofeedback group
Linden, Habib, and Radojevic (1996)	Utilized a random design to compare two groups of individuals with ADHD	The effects of 40 sessions of neurofeedback focusing on decreasing theta and increasing beta while utilizing a "waiting list" control group. No medication was provided for either group	Results showed improvement in the Kaufman-Brief Intelligence Test scores and parental behavioral reports of inattention, hyperactivity, aggressive, and defiant behaviors for the experimental group when compared to the control group
Monastra, Monastra, and George (2002)	One-year outpatient program with 100 children aged 6–19	Treatment included Ritalin, parent counseling, and academic support at school. Fifty-one participants also received neurofeedback	Significant improvement was noted on the TOVA and the Attention Deficit Disorders Evaluation Scale (ADDES; McCarney, 1995). Only those who received neurofeedback sustained gains when tested without Ritalin
Fuchs et al. (2003)	Compared neurofeedback treatment (increase SMR or beta 1 and decrease theta and beta 2) with stimulant medication (Ritalin)	Participants were assigned to either neurofeedback or medication group based upon parents' preference	Both medication and neurofeedback showed improvements on TOVA subscales. Ratings were significantly reduced in both groups by parents and teachers on the IOWA-Conners Behavior Rating Scale

To further illustrate the effects of neurofeedback on ADHD, Monastra et al. (2002) reported that those who received neurofeedback showed greater attention and less hyperactive/impulsive behaviors at home when compared to those who received medication, specifically Ritalin. Also in this study, teachers rated students who received neurofeedback as more attentive and less hyperactive/impulsive. After a medication "washout," where the participants did not receive medication any longer, those individuals who participated in neurofeedback showed sustained improvements at home and at school, whereas those who were in the "washout" group did not. Parenting strategies was also an examined variable in this study. At 1-year follow-up, the individuals whose parents used consistent reinforcement strategies at home and were in the neurofeedback group continued to show a significant reduction in symptoms, as compared to those in the medication group.

On the basis of the data presented above, it is apparent that neurofeedback is an efficacious treatment for ADHD and a viable alternative to the use of psychostimulant medication, and it is considered the only type of treatment with sustained improvement of central ADHD symptoms in the absence of stimulant therapy (Monastra et al., 2002; Rossieter & LaVaque, 1995). Neurofeedback is also considered to be the treatment of choice where "medication is ineffective, only partially effective, has unacceptable side effects, or where medication compliance is low" (Rossieter & LaVaque, 1995, p. 11).

Like all things, neurofeedback is not without limitations. For sustained, long-term change to occur it may require up to 60 sessions or 6 months of treatment. However, when one considers the life altering change that can be made and the improvement in quality of life, a 6-month time frame may not seem too dissonant. When compared to a medication program, the cost is higher for neurofeedback in the short-term, but successful long-term change has been found in as few as 20 sessions in 30% of ADHD cases treated with neurofeedback. Considering long-term use of medication, if neurofeedback results in lasting symptom reduction and the individual does not "outgrow" the disorder, as 60–70% do not, then neurofeedback is a cost-effective alternative (Rossieter & LaVaque, 1995). This paper did not address the relationship between EEG changes and behavior changes. Future research should be focused on illuminating this effect. Finally, it is important to consider the research that has presented neurofeedback as a tool for long-term symptom reduction, showing prolonged effects ranging from 1 to 10 years (see Lubar, 1995; Tansey, 1993).

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