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Electroencephalographic Biofeedback in the Treatment of Attention-Deficit/Hyperactivity Disorder

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Historically, pharmacological treatments for attention-deficit/hyperactivity disorder (ADHD) have been considered to be the only type of interventions effective for reducing the core symptoms of this condition. However, during the past three decades, a series of case and controlled group studies examining the effects of EEG biofeedback have reported improved attention and behavioral control, increased cortical activation on quantitative electroencephalographic examination, and gains on tests of intelligence and academic achievement in response to this type of treatment. This review paper critically examines the empirical evidence, applying the efficacy guidelines jointly established by the Association for Applied Psychophysiology and Biofeedback (AAPB) and the International Society for Neuronal Regulation (ISNR). On the basis of these scientific principles, EEG biofeedback was determined to be "probably efficacious" for the treatment of ADHD. Although significant clinical improvement was reported in approximately 75% of the patients in each of the published research studies, additional randomized, controlled group studies are needed in order to provide a better estimate of the percentage of patients with ADHD who will demonstrate such gains in clinical practice.

KEY WORDS: attention-deficit/hyperactivity disorder; ADHD; EEG biofeedback; neurotherapy; efficacy; review.

INTRODUCTION

Attention-Deficit/Hyperactivity Disorder is an enduring mental disorder, characterized by persistent symptoms of inattention alone or in combination with hyperactivity and impulsivity (American Psychiatric Association, 1994). Prevalence of this disorder in the United States is reported to be approximately 7% (Gadow & Sprafkin, 1997; Pelham,

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Gnagy, Greenslade, & Milich, 1992; Wolraich, Hannah, Pinnock, Baumgaertel, & Brown, 1996) with international prevalence rates ranging from 2 to 29% (Barkley, 1998). The severity of these symptoms is known to significantly impair a person's ability to function effectively at home, school, and in the workplace.

Without effective treatment, children and adolescents with ADHD are at greater risk to develop academic, behavioral, mood, and anxiety disorders (Biederman et al., 1996), incur accidental injury (Hartsough & Lambert, 1985; Lahey et al., 1998), and struggle with substance abuse disorders (Claude & Firestone, 1995; Mannuzza et al., 1991). Similarly, when not systematically treated, adults with a childhood history of ADHD have academic histories marked by lower average marks, more expulsions, a higher rate of retention in a grade, and fewer completed grades (Weiss & Hechtman, 1993; Mannuzza et al., 1993, 1998). These patients are more likely to have a higher incidence of substance abuse, psychiatric disorders, and criminal behavior, and have an employment history of more jobs, more frequent "layoffs" and an overall job status that was lower than that of peers of similar intelligence without ADHD (Weiss & Hechtman, 1993; Murphy & Barkley, 1996).

Because of the severity and enduring nature of the functional impairments associated with ADHD, a substantial amount of scientific effort has been directed at understanding the causes of ADHD and the identification of effective treatments. Beginning with the early clinical impressions of Still (1902) and Tredgold (1908), researchers have wondered if children with problems of behavioral inhibition and lack of sustained attention suffered from some type of undiagnosed brain disease or injury. Over the past century, the preponderance of scientific findings now supports the position that ADHD is an inherited disorder, whose core symptoms are founded in neuroanatomical, neurochemical, and neurophysiological characteristics that adversely affect neuronal functioning at the cortical level.

GENETICS, NEUROANATOMY, AND ADHD

During the past decade, numerous scientific studies of twins have revealed a heritability index of approximately .75 for ADHD (Levy, Hay, McStephen, Wood, & Waldman, 1997; Silberg et al., 1996; Willcut, Pennington, & DeFries, 2000). Similarly elevated incidence rates are evident in studies of families including a member with ADHD. In families that include a child with ADHD, over 30% of the siblings also have ADHD (Biederman et al., 1992; Biederman, Keenan & Faraone, 1990; Welner, Welner, Stewart, Palkes, & Wish, 1977). In those families that include an adult with ADHD, the likelihood that at least one child will have this disorder is 57% (Biederman et al., 1995).

Efforts to identify those genes that contributed to these patterns of inheritance have focused primarily on dopaminergic alleles. This appears to be due to recent advances in molecular biology, which has revealed that stimulant medications produce their clinical effects by occupying dopamine reuptake transporters, thereby increasing the availability of dopamine at the synaptic level (Ding et al., 1997; Volkow et al., 1995). In genetic studies conducted to date, there is evidence implicating that anomalies of the dopamine receptor-4 gene (DRD4: Smalley et al., 1998), the dopamine receptoer-2 gene (DRD2; Comings et al., 1996), and the dopamine reuptake transporter (DAT1) gene (Cook et al., 1995) occur significantly more frequently in patients with ADHD. The hypothesis derived from these studies was that such anomalies would limit the number of dopamine receptors and result in reduction of the size of dopamine-rich brain regions.

Neuroanatomical studies of patients with ADHD supported such a hypothesis by providing evidence of structural differences between patients with ADHD and healthy age peers. Because the core symptoms of ADHD are comprised of impaired behavioral control and lack of sustained attention, neuroimaging studies have focused on those structures involved in the control of movement (e.g. the basal ganglia and the cerebellum) and attentional functions (e.g. the anterior cingulate gyrus, the right frontal region, the anterior and posterior regions of the corpus callosum, and the caudate). As would be anticipated on the basis of genetic findings, reports of reduced size in each of these regions has been reported and replicated in the scientific literature (Aylward et al., 1996; Castellanos, 1997; Castellanos et al., 1994; Giedd et al., 1994; Hynd, Hern, Novey, & Eliopulos, 1993; Hynd, Semrud-Clikeman, Lorys, & Novey, 1990; Mostofsky, Reiss, Lockhart, & Denckla, 1998; Semrud-Clikeman, Filipek, Biederman, & Steingard, 1994).

Further indication of the significance of the prefrontal cortex, the basal ganglia circuitry, and the cerebellum in regulating attention and behavioral control is evident in the results of studies utilizing single photon emission tomography (SPECT) and positron emission tomography (PET). Initially, Lou, Henriksen, and Bruhn (1984) reported hypoperfusion in the prefrontal cortex and basal ganglia during PET examination of patients with ADHD. Subsequently, Zametkin et al. (1990) and Ernst et al. (1994) reported decreased glucose metabolism in these regions, indicating cortical underarousal. Researchers using SPECT imaging (e.g. Kim, Lee, Shin, Cho, & Lee, 2002; Sieg, Gaffney, Preston, & Hellings, 1995) likewise noted decreased cerebral blood flow in the right lateral prefrontal cortex, the right middle temporal cortex, and the orbital and cerebellar cortices (bilaterally) in patients diagnosed with ADHD.

QUANTITATIVE ELECTROPHYSIOLOGY AND ADHD

Consistent with the results of neuroimaging studies, neurophysiological researchers have primarily found evidence of underactivity over frontal and central, midline cortical regions in approximately 85–90% of patients with ADHD (Chabot, Merkin, Wood, Davenport, & Serfontein, 1996; Clarke, Barry, McCarthy, & Selikowitz, 2001a; Mann, Lubar, Zimmerman, Miller, & Nuenchen, 1992; Monastra et al., 1999). The primary electrophysiological indicators of underactivity that have been identified via quantitative electroencephalographic (QEEG) analysis of patients with ADHD include: elevated relative theta power, reduced relative alpha and beta power, and elevated theta/alpha and theta/beta power ratios, predominately over frontal and central, midline regions.

A secondary pattern of excessive activity or "hyperarousal" over frontal regions has also been revealed in patients with ADHD (e.g. Chabot & Serfontein, 1996; Clarke, Barry, McCarthy, & Selikowitz, 2001b). This pattern has been particularly evident in those patients who have not responded optimally to stimulant medications (Chabot, Orgill, Crawford, Harris, & Serfontein, 1999). In such patients, QEEG analysis has revealed increased relative beta power, decreased relative alpha power, and decreased theta/beta power ratios across all cortical recording sites in comparison to healthy peers.

Additionally, when compared to other patients diagnosed with ADHD, patients with "hyperaroused" profiles demonstrated greater relative beta activity, decreased relative theta activity, decreased theta/beta ratios, and decreased relative delta over frontal and central regions of the cortex. Although it is not clear whether those "ADHD" patients who

demonstrate cortical hyperarousal constitute a different clinical syndrome, EEG biofeedback protocols have been developed to treat ADHD patients who present with either hypoarousal or hyperarousal over frontal or central midline regions.

THE RATIONALE FOR EEG BIOFEEDBACK FOR ADHD

The rationale for EEG biofeedback is derived from substantial neurophysiological research which clarified the relationship between surface EEG and the underlying thalamocortical mechanisms that are responsible for its rhythms and frequency modulations. As reviewed by Sterman (1996), variations in alertness and behavioral control appear directly related to specific thalamocortical generator mechanisms and that such variations are evident in distinctive EEG frequency rhythms that emerge over specific topographic regions of the brain. He hypothesized that neuropathology (such as ADHD) could alter these rhythms and that EEG feedback training directed at normalizing these rhythms may yield sustaining clinical benefits. Consistent with this hypothesis, each of the large scale QEEG studies of patients with ADHD that have been conducted since 1996 (e.g. Chabot et al., 1996; Chabot & Serfontein, 1996; Clarke et al., 2001a, 2001b; Clarke, Barry, McCarthy, & Selikowitz, 1998; Monastra et al., 1999; Monastra, Lubar, & Linden, 2001) has reported abnormal QEEG findings in patients with ADHD.

Further impetus for the development of EEG biofeedback for ADHD is derived from studies demonstrating adverse side effects and insufficient response to existing medical treatments. Although both stimulant (e.g. methylphenidate, dextroamphetamine and pemoline) and nonstimulant medications (e.g. atomoxetine and impramine) have been shown to be efficacious for the treatment of the core symptoms of ADHD in controlled, group studies, approximately 25% of ADHD patients demonstrate either an adverse response or no response (Greenhill, Halperin, & Abikoff, 1999; Swanson, McBurnett, Christian, & Wigel, 1995). In addition, as noted by Pelham and Murphy (1986), only a minority of ADHD patients show sufficient improvement to be considered within the normal range following medication treatments and there is great variability in the degree of improvement noted in those patients who do respond to medication (Pelham & Smith, 2000). Typically improvements are noted in some functional domains but not in others. As concluded by Pelham (2002), "other interventions are needed for nonresponders or incomplete responders to medication" (pp. 12–13).

TREATMENT PROTOCOLS

As previously noted, the vast majority of patients diagnosed with ADHD demonstrate excessive cortical slowing during QEEG, PET, or SPECT examinations. A smaller percentage exhibit cortical "hyperarousal." During the past three decades, specific EEG biofeedback treatments that target cortical slowing or hyperarousal have been developed and evaluated in controlled case and group studies. In each of these studies, patients have participated in training procedures in which they were reinforced (via tone or visual display) for producing a specific change in cortical activity (e.g. reducing the amplitude of activity at slower EEG frequencies; increasing activity in faster frequencies). Typically, the patient had to maintain this desired change for a period of 0.5 s in order to be "rewarded." It was hypothesized that if patients could begin to "normalize" the level of activity in

regions responsible for attention and behavioral control, they would begin to demonstrate developmentally appropriate abilities to attend and maintain behavioral control.

The initial demonstration that biofeedback could yield changes in cortical activity and that such modifications resulted in observable improvements in behavior/functioning was provided by Sterman and his colleagues (Sterman, Wywricka, & Roth, 1969; Wywricka & Sterman, 1968). Much of Sterman's groundbreaking research examined the electrophysiological characteristics of behavioral inhibition (Roth, Sterman, & Clemente, 1967; Sterman & Wywricka, 1967; Wywricka & Sterman, 1968). His methodical examination of EEG patterns associated with inhibition led to the identification of the "Sensorimotor Rhythm" (or SMR), which is generated over the Rolandic Cortex. Although initially identified as a range of activity between 12 and 20 cycles per second, the "peak activity" of the SMR was noted at 12–14 Hz. Sterman et al. (1969) and Wywricka and Sterman (1968) found that laboratory animals could be trained to produce this rhythm voluntarily and applied these findings in the treatment of individuals with a specific type of impaired behavioral control (epilepsy). As reviewed by Sterman (2000) and Monastra (2003), this application of EEG biofeedback has been demonstrated to be particularly helpful in the treatment of seizure disorders in patients who have not responded to pharmacological treatments.

The initial application of SMR training for the treatment of patients with ADHD was reported by Lubar and Shouse (1976). Their initial demonstration of clinical response in a hyperactive child stimulated considerable interest in SMR training as a potentially efficacious treatment for ADHD. Subsequently, in response to scientific understanding of the role of the frontal lobes in sustained attention, and mounting evidence of excessive cortical slowing over central, midline and frontal regions in ADHD patients, Lubar and his colleagues (e.g. Lubar & Lubar, 1984) expanded their EEG biofeedback treatments to include efforts to increase production of EEG activity in a faster frequency range ("beta": 16–20 Hz), while suppressing activity at slower speeds ("theta": 4–8 Hz). These two primary training approaches (SMR enhancement; theta suppression/beta enhancement) provide the foundation for each of the protocols that have been examined in the controlled group studies of EEG biofeedback in the treatment of ADHD conducted to date. Although recent QEEG findings of a neurophysiological "subtype" of ADHD patients, characterized by excessive "beta" activity over frontal regions (Chabot & Serfontein, 1996: Clarke et al., 2001a, 2001b), have prompted interest in the development of protocols to suppress excessive beta appearing frontally, no controlled group studies examining this type of EEG biofeedback have been reported to date.

At this time, three EEG biofeedback treatment protocols have been primarily examined in controlled-group studies. Reflecting neuroanatomical findings, these protocols target cortical regions responsible for attention and behavioral inhibition. A brief description of each follows:

PROTOCOL 1: SMR ENHANCEMENT/THETA SUPPRESSION

In this type of EEG biofeedback, patients are encouraged to develop control over behaviors of hyperactivity and impulsivity by learning to increase their production of the SMR (12–15 Hz) over one of two sites (C3 or C4), while simultaneously suppressing the production of theta (4–7 or 4–8 Hz) activity. Typically, recordings are obtained from one active site, referenced to linked earlobes, with a sampling rate of at least 128 Hz. Auditory

(tones) and visual feedback (counter display; movement of puzzle pieces, graphic designs, or animated figures) is provided based on patient success in controlling microvolts of theta or SMR, or the percentage of time that theta is below or SMR is above (SMR) pretreatment "thresholds." This type of training was included in the first controlled, group study of EEG biofeedback for ADHD (Rossiter & LaVaque, 1995).

PROTOCOL 2: SMR ENHANCEMENT-BETA-2 SUPPRESSION

A secondary type of SMR training has also been examined in a controlled, group study (Fuchs, Birbaumer, Lutzenberger, Gruzelier, & Kaiser, 2003). In this protocol, patients with ADHD, Predominately Hyperactive-Impulsive Type are trained to increase SMR (12–15 Hz) activity, while simultaneously decreasing beta-2 (22–30 Hz) activity. Recordings are obtained at C4 with linked ear reference. Sampling rate is at least 128 Hz. In Fuchs et al.'s protocol, patients with a Combined Type of ADHD receive this type of training during half of each session. During the other half of each session, a theta suppression-beta-1 enhancement protocol (described below) is followed (training site: C3). As with the "first" SMR protocol, feedback is contingent on patient success in controlling microvolts of theta, SMR, beta-1 or beta-2.

PROTOCOL 3: THETA SUPPRESSION/BETA-1 ENHANCEMENT

This protocol has been investigated in three of the five controlled, group studies published to date (Linden, Habib, & Radojevic, 1996; Monastra, Monastra, & George, 2002; Rossiter & LaVaque, 1995). In this training procedure, patients are encouraged to increase production of beta-1 activity (16–20 Hz), while suppressing theta activity (4–8 Hz). Recordings are obtained at Cz with linked ear reference (monopolar); at FCz-PCz with ear reference, or at Cz-Pz with ear reference. Fuchs et al. (2003) used a variation of this protocol with patients diagnosed with ADHD, Predominately Inattentive Type, training theta suppression and beta enhancement at C3. Sampling rate is at least 128 Hz. Feedback is provided contingent on patient success in controlling microvolts of beta or theta.

A combination of two of these procedures (Protocol 1 and 3) has also been reported in a controlled, group study (Carmody, Radvanski, Wadhwani, Sabo, & Vergara, 2001). In this procedure, patients are encouraged to increase production of a restricted range of beta-1 activity (16–18 Hz) while suppressing activity at 2–7 Hz. Recordings are obtained at C3 or Cz with linked ear reference (monopolar). Students who displayed increased aggression or agitation within the first 13–35 sessions of this type of training were considered to be "overstimulated." Such patients were then treated with a SMR training protocol, in which they were reinforced for increasing activity at 13–15 Hz and suppressing activity at 2–7 Hz.

REVIEW OF THE SCIENTIFIC LITERATURE: CASE STUDIES

As noted in reviews by Lubar (2003) and Monastra (2003), there are numerous case studies demonstrating clinical benefits in patients diagnosed with ADHD. Historically, training has followed Protocol 1 or Protocol 3, with slight variation in the size of the SMR or theta bands. The initial report (Lubar & Shouse, 1976) presented the results of an SMR training protocol (Protocol 1) in the treatment of an 11-year-old boy diagnosed with hyperkinesis. In their study, Lubar and Shouse demonstrated an electrophysiological

Table I. Summary of the Primary Findings of Studies Examining the Efficacy of EEG Biofeedback for the Treatment of ADHD: Selected Case Studies

	Participants			Method		Sessions		Outcome		
Researchers	*N*	Ages	Dx	Design	Protocol	*N*/week	Total	QEEG	Beh.	CPT
Lubar & Shouse (1976)	1	11	H	1	1	3	158	(+)	(+)	(+)
Lubar et al. (1995)	17	8–15	C	0	2	5	30–45	(+)	NR	NR
Thompson & Thompson (1998)	111	5–63	I/H/C	0	1, 3	2	40	(+)	NR	(+)
Kaiser & Othmer (2000)	186	5–67	I/H/C	0	1[a]	1–5	20–40+	NR	NR	(+)
Heywood & Beale (2003)	7	7–12	I/H/C	2	1	NR	80	NR	(+*)	NR

Note. (+): statistically significant change in score on dependent measure; (+*): statisically significant change only among participants who completed treatment; NR: not reported. Dx code: I = ADHD, predominately inattentive; H = ADHD, predominately hyperactive; C = ADHD, combined; design: 0 = Single/multiple case study, repeated measurement; 1 = Single/multiple case study, ABAB (Reversal); 2 = Single/multiple case Study, ABAB (Reversal) with placebo control.
[a] Variation of SMR enhancement/theta suppression protocol.

training effect in the laboratory that was associated with a decrease in off-task and oppositional behaviors, as well as, increased cooperation and completion of school work in the classroom. By using an ABA case study design, Lubar and Shouse showed that these clinical gains paralleled patient increases and decreases in control over SMR activity during training sessions.

Since the publication of this initial case study, there have been other notable case reports, the most extensive being Thompson and Thompson's (1998) study of 111 patients diagnosed with Attention Deficit Disorder (with and without hyperactivity) and Kaiser and Othmer's (2000) investigation of EEG biofeedback in 1089 patients (186 diagnosed with ADHD). Clinical gains included improved scores on tests of attention and impulse control (Kaiser & Othmer, 2000; Thompson & Thompson, 1998), as well as, an average increase of 12 points on the Wechsler Full Scale Intelligence Quotient (Thompson & Thompson). Table I summarizes the characteristics of several important case-studies.

Initial reports of the enduring nature of EEG biofeedback have been provided by Tansey (1993) and Lubar (2003). Tansey (1993) reported the results of a 10-year follow-up study, which indicated that a child (initially treated in the fourth grade) was able to maintain sustained control over hyperactive symptoms during adolescence and early adulthood. Lubar (2003) conducted a retrospective study of 52 patients who had completed EEG biofeedback treatment for ADHD over a 10-year period. During telephone interviews conducted by an independent surveyor, parents (or older patients) were asked to rate the degree of improvement in 16 targeted symptoms (e.g. fidgeting, restlessness, overactivity, inattention, failure to complete tasks, outbursts of temper, low frustration tolerance, relationships with others) since the completion of treatment. The results of the survey were positive, with primary improvements noted in sustained control over symptoms of hyperactivity, emotional lability, rate of completing homework, and report card marks.

CRITIQUE OF CASE STUDIES

As in any field of applied clinical research, case studies are necessary in the development of new treatments. Through such studies, researchers are able to identify potentially beneficial intervention strategies, as well as, any potential patient risks. Such is the case with EEG biofeedback for ADHD. During the last 25 years, several training protocols have

been developed and examined in case studies using a variety of procedures to determine treatment effects. Examination of the outcomes of these case studies reveals consistently positive results. Each of the studies reported to date indicated improvements in symptoms of attention and behavioral control in patients diagnosed with hyperkinesis or ADHD (Inattentive; Hyperactive-Impulsive; or Combined Types) following treatment with EEG Biofeedback. No significant adverse effects were reported in the case studies, although deterioration of clinical effects and relapse has been reported in those case studies in which training has been discontinued prior to completion of treatment (e.g. Lubar & Shouse, 1976).

Despite the positive outcomes reported in case studies, information derived from such studies is considered insufficient to demonstrate the efficacy of any treatment (Chambless & Hollon, 1998; LaVaque et al., 2002; Nuwer, 1997). Although meta-analysis studies have concluded that there is often no difference in terms of outcome between the results of case-controlled and prospective-randomized studies (Benson & Hartz, 2000), case studies do not provide a method for examining nonspecific factors that may influence the effectiveness of a particular treatment in applied clinical settings.

In evaluating the efficacy of any type of treatment, it is important to use research designs that can clarify the degree that beneficial effects are due to factors other than the specific treatment that was administered (in this case, EEG biofeedback). Such nonspecific factors include therapist characteristics (e.g. degree of compassion, understanding, displayed knowledge or confidence), patient characteristics (e.g. patient intelligence and capacity to learn new skills, severity of the disorder, the degree of hope or expectancy, variations in patient motivation for participating in the study), and treatment characteristics (e.g the administration of a pill; the use of computerized EEG equipment), patient exposure to other therapeutic experiences, other than the treatment under investigation (e.g. counseling, tutoring, variations in parenting styles), and maturation. Without controls for such factors, the percentage of patients likely to respond to any treatment is difficult to estimate.

Two controlled, case studies can serve to illustrate the importance of motivation and capacity to learn new skills in assessing efficacy. In the mid-1990's Lubar and his colleagues reported the outcome of a series of studies, including one examining the efficacy of EEG biofeedback (Lubar, Swartwood, Swartwood, & Timmerman, 1995). In studying the effects of theta suppression/beta enhancement (Protocol 3) in 17 children diagnosed with ADHD, these researchers reported that two groups emerged. One group of children ($n = 6$) were unable to demonstrate a training effect on any of the EEG measures obtained during training. Another group ($n = 11$) was able to "learn" to increase cortical activation (by lowering the theta/beta power ratio). Although the association between learning to control cortical activation via EEG biofeedback and degree of clinical response was not directly assessed, Lubar et al.'s (1995) paper illustrates the importance of directly assessing neurophsyiological indicators of learning in any evaluation of the efficacy of EEG biofeedback.

Heywood and Beale's (2003) more recent study of EEG biofeedback in seven children diagnosed with ADHD provides further impetus for examining such nonspecific factors. In their study, they provided a "bona fide" biofeedback training protocol designed to promote an increase of SMR and decrease in theta and beta 2 at Cz. The placebo control training included "noncontingent" EEG biofeedback in which a series of randomly determined bandwidths were reinforced or inhibited (e.g. 12–29 Hz; 2–6 Hz; 2–18 Hz). In addition, Heywood and Beale (2003) used a randomized design with an embedded ABAB reversal, to

control for maturation and treatment sequence effects. Children were not informed whether the training was bona fide or placebo.

Examination of their findings revealed that five of the children completed training, two did not. Analysis of results indicated that when the data were analyzed for children who completed treatment, a significant positive effect was noted on neurophysiological and behavioral measures of attention. However, when the data from the two children who discontinued treatment were included, and control for overall trend was added to the analysis, the overall size of these gains diminished. Although it is difficult to draw conclusions regarding the efficacy of EEG biofeedback from such a small study, Heywood and Beale's work illustrates the importance of reporting the results of "nonresponders" and controlling for nonspecific factors and trend effects in studies examining the efficacy of EEG biofeedback.

Overall, the results of case studies illustrate the potential benefits of EEG biofeedback in the treatment of patients with ADHD. However, it is clear from a review of case studies that there is a percentage of patients who will not "learn" how to regulate cortical activity and reduce core ADHD symptoms via EEG biofeedback. In reported case studies, that percentage is comparable to the number of patients who do not respond to stimulant medications and ranged from 29% (Heywood & Beale, 2003) to 35% (Lubar et al., 1995). In addition, the results of the Heywood and Beale (2003) study provides evidence that nonspecific factors (e.g. expectancy, maturation) need to be evaluated/controlled in efficacy studies of EEG biofeedback for ADHD.

REVIEW OF THE SCIENTIFIC LITERATURE: CONTROLLED-GROUP STUDIES

To date, five controlled-group studies have been reported in peer-reviewed journals (Carmody et al., 2001; Fuchs et al., 2003; Linden et al., 1996; Monastra et al., 2002; Rossiter & LaVaque, 1995) [see Table II]. Each of these studies sought to examine the effects of EEG biofeedback in the treatment of patients diagnosed with ADHD, while attempting to control for certain factors (e.g. age, intelligence, severity of symptoms prior to initiating treatment).

Table II. Summary of the Primary Findings of Studies Examining the Efficacy of EEG Biofeedback for the Treatment of ADHD: Controlled Group Studies

	Participants			Method		Sessions		Outcome		
Researchers	*N*	Ages	Dx	Design	Protocol	*N*/week	Total	QEEG	Beh.	CPT
Rossiter & LaVaque (1995)	46	8–21	I/C	1	1,3	3–5	20	NR	(+)	(+)
Linden et al. (1996)	18	5–15	I/H/C AD/LD	0	3	2	40	NR	(+)	(+)
Carmody et al. (2001)	16	8–10	I/H/C/N	0	1, 3[a]	3–4	36–48	(−)	(+)	(+)
Monastra et al. (2002)	100	6–19	I/C	1	3	1	34–50	(+)	(+)	(+)
Fuchs et al. (2003)	34	8–12	I/H/C	1	2	3	36	NR	(+)	(+)

Note. (+): statistically significant change in score on dependent measure; (−): no statistically significant change in score on dependent measure; NR: not reported. Dx code: I = ADHD, predominately inattentive; H = ADHD, predominately hyperactive; C = ADHD combined; AD/LD = ADHD + learning disorder; N = No psychiatric disorder. design: 0 = controlled group study, random assignment, waiting list control; 1 = Controlled group study, nonrandom assignment, bona fide treatment comparison (stimulants).

[a] Variation of theta/suppression/SMR and beta enhancement protocols.

Maturation effects were also controlled in each of these studies and comparisons with a "bona fide" treatment that has been classified as efficacious (i.e. stimulant medication) were included in three of the five studies in order to control for placebo and trend effects.

The first of the controlled studies was published by Rossiter and LaVaque (1995). This study sought to compare the effects of EEG biofeedback and stimulant medication (methylphenidate or dextroamphetamine) on a continuous performance test (Test of Variables of Attention, Greenberg & Dupuy, 1993) and a standardized behavioral rating scale which assessed ADHD symptoms, as well as indicators of other types of behavioral problems (Behavior Assessment System for Children). After initial pretesting, patients were matched for age, intelligence, gender, and diagnosis and treated with one of two EEG biofeedback protocols (Protocol 1 or Protocol 3) or with stimulant medication (as prescribed, monitored, and adjusted by the patient's physician). A total of 46 patients (aged 8–21) participated in the study. Two groups of 23 patients received the treatment of their (or parent's) choice (either medication or 20 sessions of EEG biofeedback). Patients participating in EEG biofeedback were seen three to five times per week (45–50-min sessions that included 30 min of feedback)

The results of this study indicated significant improvement on the T.O.V.A. and several subscales of the BASC (e.g. Hyperactivity, Attention Problems, and Externalizing Behaviors) in patients who completed EEG biofeedback. In addition, comparison with a bona fide treatment for ADHD (stimulant medication) revealed no difference in the efficacy of these treatments after 20 sessions. Similarly, there was no significant difference in the percentage of patients who showed significant improvement with EEG biofeedback (83%) and stimulant medication (87%).

The second published controlled group study was reported by Linden et al. (1996). In this study, 18 children (ages 5–15) diagnosed with ADHD were randomly assigned to either a "waiting list" condition (and received no psychological treatment or medication) or EEG biofeedback (Protocol 3). Groups were comprised of an equal number of children diagnosed with ADHD alone ($n = 6$) or in combination with a learning disorder ($n = 3$), for a total of nine children in each group. Power analysis conducted prior to initiating the study indicated sufficient sample size to detect significant group statistical differences. The study was conducted over a six month time period. Patients receiving EEG biofeedback participated in 40, 45-min training sessions. Medications for ADHD were not prescribed.

The results of the Linden et al. (1996) study reflected a significant increase on a measure of intelligence (Kaufman Brief Intelligence Scale: Kaufman & Kaufman, 1990) and a reduction in symptoms of inattention on the IOWA-Conners Behavior Rating Scale in the group of children who received EEG biofeedback. No adverse results were reported.

A randomized, waiting list, controlled group study was also conducted by Carmody et al. (2001) in a school setting. In their study, 16 children (ages 8–10) were randomly assigned to an active treatment condition (EEG biofeedback) or a waiting list. Eight of the children were diagnosed with ADHD; eight were not diagnosed with ADHD or any other psychiatric disorder. Although pharmacological treatment had been recommended for all of the children diagnosed with ADHD, none of their parents selected that type of treatment.

Carmody et al. (2001) utilized a variation of Protocols 1 and 3 in their study. During the active phase of treatment, participants received 3–4 EEG biofeedback sessions per week, completing between 36 and 48 sessions during a six month period. Dependent variables of interest included several QEEG measures ("delta-theta" amplitude; beta amplitude; SMR amplitude), home and school versions of a behavioral rating scale assessing

frequency of ADHD symptoms (ADDES; McCarney, 1989) and a continuous performance test (T.O.V.A., Greenberg & Dupuy, 1993).

The results of the Carmody et al. (2001) study indicated that children with ADHD who were treated with EEG biofeedback reduced symptoms of impulsivity on the T.O.V.A. and were rated as more attentive by their teachers on the School Version of the ADDES. However, no consistent pattern of improvement was evident on the QEEG measures selected by this research team.

Monastra et al. (2002) published the largest, controlled-group study in the literature. Similar to Rossiter and LaVaque (1995), the effects of EEG biofeedback were compared with a "bona fide" treatment (Ritalin). In their study, 100 patients (aged 6–19) participated in a multimodal treatment program that included: stimulant medication (dosage titrated based on the results of behavioral measures and the T.O.V.A.), a 10 week parenting program (Monastra, 2004) with subsequent, individualized parent-counseling provided as needed, and academic support at school (via an Individual Education Plan or 504 Accommodation Plan). Patients were also given the opportunity to receive EEG biofeedback (Protocol 3) as part of their treatment program. Fifty-one families chose to include EEG biofeedback (49 did not). The average dose of Ritalin administered to the patients of both groups was 25 mg (10 mg after breakfast, 10 mg after lunchtime, 5 mg after school). The range was 15–45 mg per day.

EEG biofeedback sessions were conducted on a weekly basis (45–50 min) and continued until the patient could demonstrate a level of cortical activity on the QEEG scan that was within 1.0 standard deviation of age peers (Monastra et al., 1999 database) and could maintain this level of arousal for three consecutive, training sessions (40 min each). The average number of sessions needed to reach this goal was 43 (range 34–50). All of the participants who received EEG biofeedback achieved this goal.

Pretreatment screening included tests of intelligence, behavioral rating scales, a continuous performance test, and a QEEG assessment (Monastra et al., 1999). All of the participants needed to demonstrate evidence of cortical slowing on the QEEG measure in order to be included in the study. There were no significant differences on pretreatment measures between patients who received EEG biofeedback as part of their treatment and those who did not.

Posttreatment evaluation was conducted 1 year after initial evaluation, under two conditions. First, participants were evaluated while continuing to take stimulant medication. Subsequent to this assessment, medication was discontinued for 1 week, and participants were evaluated following this medication "wash-out." All participants remained in the study for the year required to complete this research.

Results of the Monastra et al. (2002) study supported the efficacy of stimulant medication as well as EEG biofeedback, and indicated that parenting style was a moderating factor in both treatments. In their study, significant improvement was noted in both groups on posttreatment evaluations that were conducted while the patients were using medication. However, following a week-long medication wash-out, relapse was noted on behavioral and CPT measures in each of the participants who had not received EEG biofeedback and no sustained improvement was noted on the QEEG measure among the members of that group.

In contrast, patients who received EEG biofeedback as part of their treatment demonstrated sustained improvement on the T.O.V.A. and on behavioral measures, and maintained gains on QEEG measures of cortical arousal even when tested after a 1-week medication washout. In both the EEG biofeedback and the "non-biofeedback" groups, parents who

were systematically using the strategies taught in the parenting program had children who displayed fewer attentional and behavioral control problems at home.

The fifth controlled-group study was reported by Fuchs et al. (2003). In this study, a comparison between EEG biofeedback and a bona fide treatment for ADHD (stimulant medication) was investigated. A total of 34 children (aged 8–12) participated in the study. Twelve were treated with Ritalin (mean dose: 10 mg, t.i.d.; Range: 10–60 mg per day). Neurofeedback sessions (Protocol 2) were conducted three times per week (30–60 min in duration). All participants were treated for a 12-week period. Assignment to the treatment groups was based on parental preference.

As with the other controlled-group studies, pretreatment measures included a test of intelligence (WISC-R), computerized tests of attention (T.O.V.A.; the Attention Endurance Test, Brickenkamp, 1994), and behavioral rating scales (IOWA-Conners Behavior Rating Scale). Statistical analysis of pretreatment measures indicated that the groups were comparable in terms of intelligence and severity of impairment associated with ADHD. Posttreatment analysis revealed that both EEG biofeedback and Ritalin were associated with significant improvements on computerized tests of attention and on behavioral rating scales. The degree of improvement noted in patients treated with EEG biofeedback was comparable with that noted in patients treated with Ritalin. No adverse effects were reported.

CRITIQUE OF CONTROLLED-GROUP STUDIES

Controlled-group studies of EEG biofeedback in the treatment of ADHD have demonstrated beneficial effects of EEG biofeedback on measures of intelligence, on behavioral ratings scales assessing the frequency of the core symptoms of ADHD, on computerized tests of attention, and on QEEG measures of cortical arousal. In contrast to case-studies, these studies have compared patient outcomes obtained following EEG biofeedback training with those noted following a bona fide treatment of ADHD (stimulant medication), as well as a waiting list control. These consistent reports of significant, beneficial effects in controlled, group studies following the use of a nonpharmacological treatment (EEG biofeedback), represents a significant step in the identification of effective psychological treatments for ADHD. To date, no other type of psychological treatment has been demonstrated to exert a significant effect on the *core symptoms* of ADHD (i.e. inattention, hyperactivity, and impulsivity).

Despite the positive results of studies examining the efficacy of EEG biofeedback, data from controlled-group studies that randomly assign participants to EEG biofeedback or comparison groups (e.g., stimulant medication, noncontingent biofeedback, or a waiting list control group that has comparable amount of therapist contact) are needed in order to clarify the percentage of patients diagnosed with ADHD who will respond to EEG biofeedback in clinical practice. Although the studies reported to date have indicated a positive response in over 75% of patients treated with EEG biofeedback, these studies have been conducted by highly experienced therapists, with patients who volunteered to receive this type of treatment. As such, the number of "treatment responders" and the degree of clinical improvement reported in these studies may exceed results obtained in clinical practice.

As noted previously, differences in patient characteristics (e.g. motivation for change, expectancy or hope that a new treatment will "work," interest in learning new skills) and in therapist characteristics (e.g. degree of compassion, understanding of protocols, level of confidence displayed in sessions, ability to conduct treatment sessions with a high degree

of fidelity) can affect response to treatment. Randomized, controlled-group studies that monitor and control for such factors are still needed. Such studies will facilitate a better understanding of the percentage of patients likely to respond to EEG biofeedback for ADHD in clinical practice.

ASSESSMENT OF EFFICACY

The Guidelines for Evaluation of Clinical Efficacy of Psychophysiological Interventions (LaVaque et al., 2002), which have been accepted by the Association for Applied Psychophysiology & Biofeedback (AAPB) and the International Society for Neuronal Regulation (ISNR), specify five types of classification for the effectiveness of biofeedback procedures, ranging from "Not empirically supported" to "Efficacious and Specific." The requirements for each classification level is summarized below.

Criteria for Levels of Evidence of Efficacy

Level 1: *Not empirically supported*. This classification is assigned to those treatments that have only been described and supported by anecdotal reports and/or case studies in non-peer reviewed journals.

Level 2: *Possibly efficacious*. This classification is considered appropriate for those treatments that have been investigated in at least one study that had sufficient statistical power, well identified outcome measures, but lacked randomized assignment to a control condition internal to the study.

Level 3: *Probably efficacious*. Treatment approaches that have been evaluated and shown to produce beneficial effects in multiple observational studies, clinical studies, wait list control studies, and within-subject and between-subject replication studies merit this classification.

Level 4: *Efficacious*. In order to be considered "efficacious," a treatment must meet the following criteria:

(a) In a comparison with a no-treatment control group, alternative treatment group, or sham (placebo) control *utilizing randomized assignment*, the investigational treatment is shown to be statistically significantly superior to the control condition or the investigational treatment is equivalent to a treatment of established efficacy in a study with sufficient power to detect moderate differences;
(b) The studies have been conducted with a population treated for a specific problem, from whom inclusion criteria are delineated in a reliable, operationally defined manner;
(c) The study used valid and clearly specified outcome measures related to the problem being treated;
(d) The data are subjected to appropriated data analysis;
(e) The diagnostic and treatment variables and procedures are clearly defined in a manner that permits replication of the study by independent researchers, and
(f) The superiority or equivalence of the investigational treatment have been shown in at least two independent studies" (LaVaque et al., 2002, p. 280).

Level 5: *Efficacious and Specific*. To meet the criteria for this classification, the treatment needs to be demonstrated to be statistically superior to a credible sham therapy, pill, or bona fide treatment in at least two independent studies.

Review of the scientific literature revealed both controlled case and group studies on the effects of EEG biofeedback in treating the core symptoms of ADHD. These studies examined the efficacy of well-defined treatment protocols in the treatment of patients diagnosed with hyperkinesis, as well as, those diagnosed with each of the primary subtypes of ADHD (Inattentive, Hyperactive-Impulsive, or Combined). The results of these studies indicated improvement on standardized tests of intelligence, attention, and behavioral control following EEG biofeedback. Increased level of cortical arousal was also reported during QEEG examination of patients treated with EEG biofeedback. Comparisons with a bona fide treatment for ADHD (stimulant medication) indicated that EEG biofeedback yielded equivalent or superior results. The results of randomized, controlled group studies using a waiting list control also indicated the superiority of EEG biofeedback. Such findings suggest the efficacy of EEG biofeedback in the treatment for ADHD.

However, because of the small sample size in the two, randomized group studies reported, and the absence of control for patient and therapist characteristics that could influence outcome in any of the five, controlled-group studies, our determination (based on AAPB/ISNR Guidelines) is that EEG biofeedback is probably efficacious for the treatment of ADHD. Although it is clear from the outcomes of each of the published case and controlled studies of EEG biofeedback for ADHD, that significant, beneficial effects have consistently been reported in patients/families who volunteered to receive this type of treatment, additional controlled, group studies (with random assignment to treatment condition) are needed in order to promote a clearer understanding of the number of patients and degree of improvement that can be anticipated in clinical practice.

CLINICAL TREATMENT GUIDELINES

Selection Criteria

The following exclusion criteria have been utilized in case and controlled-group studies of biofeedback for ADHD. The treatment protocols described in this paper have not been systematically evaluated in individuals with the following characteristics:

- Age under 6 years,
- mental retardation,
- presence of another medical or psychiatric condition known to adversely affect attention or behavioral control (e.g. anemia; hypoglycemia; diabetes; psychosis, severe depression, or bi-polar disorder),
- history of neurological disease (including seizure; traumatic brain injury),
- substance abuse or dependence, and
- families with significant marital discord that interferes with participation in the treatment process.

Training Protocols

The following protocols for EEG biofeedback training for ADHD have been supported by controlled group studies. However, given recent findings indicating at least two neurophysiological subtypes of ADHD, QEEG evaluation of the patient may be

helpful prior to initiating any of these training procedures. Such databased comparisons of a patient with healthy age peers can be useful in defining the location and type of EEG abnormality(ies) and contribute to the selection of a particular treatment protocol:

Protocol 1: SMR Enhancement/Theta Suppression	
Electrode placement	C3 or C4 (linked ear lobe reference)
Reward frequency	12–15 Hz
Inhibit frequency	4–7 or 4–8 Hz
Duration of behavior needed to obtain reward (auditory/visual)	0.5 s.
Sampling rate	128 Hz (minimum)
Rate of reward	Initial settings for REEG and IEEG should provide approximately 15–20 auditory/visual "rewards" per minute
Clinical goals	Improve behavioral control, reduce symptoms of hyperactivity and impulsivity

Protocol 2: SMR Enhancement/Beta-2 Suppression	
Electrode placement	C4 (linked ear lobe reference)
Reward frequency	12–15 Hz
Inhibit frequency	22–30 Hz
Duration of behavior needed to obtain reward (auditory/visual)	0.5 s.
Sampling rate	128 Hz (Minimum)
Rate of reward	Same as Protocol 1
Clinical goal	Improve behavioral control, reduce symptoms of hyperactivity and impulsivity

Protocol 3: Theta Suppression/Beta Enhancement	
Electrode placement	Cz or C3 (linked ear lobe reference) FCz-PCz (ear lobe reference) Cz-Pz (ear lobe reference)
Reward frequency	16–20 Hz
Inhibit frequency	4–8 Hz*
Duration of behavior needed to Obtain reward (auditory/visual)	0.5 s.
Sampling rate	128 Hz (Minimum)
Rate of reward	Same at Protocol 1.
Clinical goal	Improve attention and behavioral control, primarily in patients with cortical slowing.

*Lubar (2003) also reports a training protocol that reinforces suppression of an expanded "alpha" range (6–10 Hz) when treating adolescents and adults with this protocol. However, this has not been evaluated in controlled, clinical group studies.

Treatment Schedules

Positive response has been noted with both massed (3–5 sessions per week) and spaced (1 session per week) training. Sessions range in duration from 30–45 min of biofeedback training. In each session, a "baseline" or "warm-up" condition is initially conducted, during which no feedback is provided (2–5 minutes in duration). Subsequently, "training" segments are conducted and EEG biofeedback is provided. The duration of these training segments varies, often beginning with 5-min periods, gradually increasing to 9–10 min depending on patient learning curves and clinical response.

Course of treatment is variable, ranging from 20 to 50 sessions. Calculation and review of quantitative indicators of patient progress at the conclusion of each session (e.g. microvolts of theta, beta-1, beta-2, or SMR; percentage of time that the patient exceeds reward or inhibitory thresholds) and examination of the graphic depiction of such data is critical in shaping the training protocol. Additionally, information derived from continuous performance tests and behavioral rating scales is useful in evaluating patient progress.

Adverse Effects

Although no side-effects were reported in case or controlled studies, those clinical researchers who have examined the effects of EEG biofeedback in conjunction with stimulant medication have noted increased irritability, moodiness, and hyperactivity in patients who are being treated with both types of treatments concurrently (Lubar, 2003; Monastra, 2003). This type of occurrence appears during the mid to late phases of biofeedback training, most commonly in patients who are demonstrating improved cortical activation via EEG biofeedback. Reduction in the dosage of stimulant medication (with or without introduction of a "nonstimulatory" ADHD medication) has been associated with elimination of this type of side effect. Other side effects (headaches; dizziness) can occur in 1–3% of patients, but typically respond to a brief resting period (30 min) or consumption of food.

Adjunctive Treatments

Because ADHD has been associated with significant impairment of educational performance, collaboration between the clinician and teachers is recommended, in order to promote the child's success at school during the treatment process. In the United States, students with ADHD are entitled to either an Individual Education Plan (I.E.P.) or the development of a 504 Accommodation Plan, depending on their need for special education services. Such plans are intended to identify specific areas of functional impairment associated with ADHD (e.g. disorganization, difficulty completing school assignments in class and at home, and poor study skills) and define interventions to provide accommodation and remediation for these problems. Whether specified via an I.E.P., an Accommodation Plan, or an informal arrangement with teachers, efforts to ensure that patients are receiving assistance at school typically improve classroom performance, reduce stress at home and create a less conflictual learning environment for the child.

Parent counseling is also recommended in the treatment of children with ADHD. Specifically, teaching parents strategies for systematically using reinforcement principles

has been shown to enhance EEG biofeedback treatments for ADHD (Monastra et al., 2002). Similarly, social skills training programs, utilizing contingency management strategies (i.e. point/token reward systems, time-out, and response cost) have been shown to promote improved classroom functioning and the development of positive peer relationships in children diagnosed with ADHD (see review by Pelham, 2002).

None of the traditional psychotherapeutic techniques that have been effective in treating other disorders has been determined to be efficacious in the treatment of the core symptoms of ADHD (i.e. inattention, hyperactivity, and impulsivity). As reported in the NIH Consensus Statement on the Diagnosis and Treatment of ADHD (1998), insight-oriented treatments do not exert a significant effect on these symptoms. Similarly, more recently developed "cognitive-behavioral" treatments (e.g. self-monitoring, verbal self-instruction, problem-solving training, self-reinforcement) have also failed to promote improvement in the primary symptoms of ADHD or significant changes in the behavior or academic functioning of children diagnosed with ADHD (Abikoff et al., 1988; Bloomquist, August, & Ostrander, 1991; Brown, Borden, Wynne, Spunt, & Clingerman, 1987; National Institute of Health [NIH], 1998). However, children and teens who are being subjected to parental neglect or abuse will often respond favorably to individual and family therapy (as well as community-based interventions from Child Protective Agencies) that address these issues.

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