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Clinical Utility of EEG in Attention Deficit Hyperactivity Disorder

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Electrophysiological measures were among the first to be used to study brain processes in children with attention deficit hyperactivity disorder (ADHD; Diagnostic and Statistical Manual of Mental Disorders [4th ed.], American Psychiatric Association, 1994) and have been used as such for over 30 years (see Hastings & Barkley, 1978, for an early review). More recently, electroencephalography (EEG) has been used both in research to describe and quantify the underlying neurophysiology of ADHD, but also clinically in the assessment, diagnosis, and treatment of ADHD. This review will first provide a brief overview of EEG and then present some of the research findings of EEG correlates in ADHD. Then, the utility of EEG in making an ADHD diagnosis and predicting stimulant response will be examined. Finally, and more controversially, we will review the results of the most recent studies on EEG biofeedback (neurofeedback) as a treatment for ADHD and the issues that remain to be addressed in the research examining the efficacy this therapeutic approach.

Key words: EEG biofeedback, diagnosis, treatment, neurotheraphy

Electroencephalography (EEG) measures reflect the correspondence between intracranial electrical currents and the resulting voltages on the scalp reflecting certain facets of brain electrical function and processing, such as how electrically active various brain regions are and how responsive they may be to stimuli or during cognitive tasks. Early EEG studies found that children with attention deficit hyperactivity disorder (ADHD) exhibit EEG abnormalities such as excess slow wave activity and epileptiform spike and wave activity (Satterfield, Cantwell, & Satterfield, 1974). These findings were interpreted as indicating abnormal brain processes among children with ADHD, specifically a maturational delay marked by underarousal. Recent advances in technology have resulted in more accurate quantification of EEG activity by allowing computation of amplitude and power values for specific frequency bands of activity, source localization, and brain electrical activity mapping. Electrophysiological techniques (event-related potential [ERP] and EEG) are noninvasive, are less sensitive to movement artifact, do not include radioactive isotopes, and offer excellent temporal resolution (EEG can measure changes in the brain to the millisecond). The spatial resolution (i.e., where the EEG signal is coming from), however, is sometimes difficult to determine because electrical currents recorded from the cortex do not always bear a direct relation to any specific underlying brain structure and are affected by many sources of electrical artifacts.

When examining EEG activity, scientists and clinicians often look at the activity within a specific frequency band. Frequency refers to the number of oscillations (or cycles) within a given time period (e.g., four cycles per second). Note that EEG waveforms are a mixture of several different frequency bands, which are transformed and quantified for further analysis. In addition, although it is possible to decompose the EEG signal into different frequency bands, they are part of a dynamic milieu that acts in concert. Thus, certain cognitive or behavioral characteristics have been associated with a frequency band, but it is also the relationship among frequencies in other areas of the brain that produce complex behaviors. Details about the specific frequency bands are presented in the table below along with a brief summary of some findings concerning ADHD and its subtypes.

In addition to looking at activity in the individual frequency bands, theta/beta and theta/alpha ratios have also been examined and are thought to reflect level of

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Table 1. *Summary of EEG Frequency Bands and ADHD*

Frequency Band	Delta	Theta	Alpha	Beta
Cycles per second (Hz)	< 4	4–7	8–12	>13
Associated feeling states	Sleep, unconscious	Drowsiness, unfocused	Eyes closed; relaxed, but alert	Mental activity, concentration
Findings in ADHD	Mixed findings: Increased in some ADHD, normal or decreased levels in others	Increased in frontal and central area, continues into adulthood	Mixed findings: May depend on age, gender, or subtype	Decreased in some but not all ADHD children, may normalize with in adults
ADHD subtype	Increased in ADHD–Combined Type	Increased in ADHD–Combined Type	Increased in ADHD–Inattentive Type	Decreased in ADHD–Combined Type

Note: ADHD = attention deficit hyperactivity disorder.

cortical arousal and maturational delay, respectively. Though these may seem redundant measures of the individual frequency bands, they have been proposed to be a better way to capture the relative levels of these offsetting brain activation patterns (Monastra, Lubar, & Linden, 2001).

EEG Correlates in ADHD Children

Comparisons of ADHD and Normal Children

Early reviews of studies of electrophysiological measures collected on hyperactive or ADHD children concluded that the disorder was most likely associated with problems of underreactivity to stimulation and task demands with less evidence supporting resting underarousal in the disorder (Hastings & Barkley, 1978; Rosenthal & Allen, 1978). Recent studies have helped to support, clarify, and further refine these early studies (for a comprehensive review of EEG findings, see Barry, Clarke, & Johnstone, 2003). Current research findings show that most children with ADHD display fairly consistent EEG differences in brain electrical activity when compared to normal children, particularly regarding frontal and central theta activity, which is associated with underarousal and indicative of decreased cortical activity (Chabot & Serfontein, 1996; Clarke, Barry, McCarthy, & Selikowitz, 1998, 2001a; El-Sayed, Larsson, Persson, & Rydelius, 2002; Lazzaro et al., 1998). In the largest EEG study of ADHD to date (with a sample of over 400 children), Chabot and Serfontein (1996) found that children with ADHD displayed increased theta power, slight elevations in frontal alpha power, and diffuse decreases in beta mean frequency. Increased theta power is the most consistent finding in this ADHD EEG literature, indicating that cortical hypoarousal is a common neuropathological mechanism in ADHD.

It has also been suggested that the theta/beta ratio is associated with cortical arousal that has also been shown to consistently differentiate between ADHD and normal samples (Bresnahan & Barry, 2002; Clarke et al., 2001a; Monastra et al., 2001). Furthermore, this measurement has been shown to be stable over time; EEG recording from a single electrode at the vertex (Cz) yielded that a 1-month reliability of the theta/beta ratio was .96, $p < .05$ (Monastra et al., 2001). The theta/beta ratio has been found to discriminate between individuals with ADHD and normal controls across the age range (Bresnahan, Anderson, & Barry, 1999) and theoretically makes sense given that frequency bands are part of a milieu rather than occurring in isolation.

Aside from the findings for theta and the theta/beta ratio, results for other frequency bands such as beta and alpha have been more variable among children with ADHD. The findings for beta (indicative of heightened cortical arousal) activity have been less consistent, with several studies finding decreased beta activity in frontal and central regions (Chabot & Serfontein, 1996; Clarke et al., 1998, 2001a; Lazzaro et al., 1998; Mann, Lubar, Zimmerman, Miller, & Muenchen, 1992) and others not (Janzen, Graap, Stephanson, Marshall, & Fitzsimmons, 1995; Kuperman, Johnson, Arndt, Lindgren, & Wolraich, 1996; Satterfield, Schell, Backs, & Hidaka, 1984). There also appears to be a small group of ADHD children (~15%) who show an excess of beta activity (Chabot & Serfontein, 1996; Clarke, Barry, McCarthy, & Selikowitz, 2001c). With the exception of slightly elevated levels of temper tantrums and moodiness, this group of children appears to be behaviorally similar to other ADHD children, although they are more likely to be ADHD–Combined Type (Clarke et

al., 2001c). Similarly, studies have found mixed results for alpha power, with some studies showing it to be increased (Chabot & Serfontein, 1996; Clarke et al., 2001a), others showing it to be decreased (El-Sayed et al., 2002), and still other studies finding it to be similar (Bresnahan et al., 1999) between children with ADHD and normal controls. Gender and age may play a role in these discrepant findings, as well as differences in methods such as sampling (i.e., community vs. clinical samples), diagnostic procedures, and EEG data collection and processing.

EEG Differences Among ADHD Subtypes

Another possible explanation for the variability in EEG findings may lie in patterns of EEG activity according to the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed. [*DSM–IV*], American Psychiatric Association, 1994) ADHD (Inattentive, Hyperactive–Impulsive or Combined) subtypes. Subtype differences, however, have not been well studied, with only a handful of groups examining this question. Chabot and Serfontein (1996) looked at different groups of children with attention disorders (normal, attention problems [but subthreshold ADHD], ADHD) and found that differences were more quantitative than qualitative, with the children having the most severe symptoms showing the greatest EEG abnormality. Another group that has systematically studied subtype differences found that children with ADHD–Combined Type (CT) exhibited more absolute and relative theta, and higher theta/alpha and theta/beta ratios when compared to those with ADHD–Inattentive (I) type (Clarke et al., 1998, 2001a; Clarke et al., 2003). The differentiation between ADHD subtypes by the theta/beta power ratio was not independently replicated (Monastra et al., 2001); thus more study is required. Nonetheless, these results suggest that it is the ADHD–CT children who show the classic pattern found in earlier studies of greater underarousal and maturational delay than ADHD–I children. In contrast, ADHD–I children exhibited more relative alpha in the posterior regions than those with ADHD–CT, which is consistent with reports of slower cognitive processing and increased rates of daydreaming among these kids. Developmental studies suggest that there are actually two distinct components in ADHD that may be quantifiable with EEG. The first is a hyperactive–impulsive component that appears to normalize with increasing age and the second is an inattentive component that does not normalize with increasing age and therefore represents a deviation from normal development (Barry et al., 2003). This hypothesis is consistent with the clinical phenomenology of ADHD, where the hyperactive symptoms often decrease substantially with age, but inattention and disorganization remain problematic longer into development (Hart, Lahey, Loeber, Applegate, & Frick, 1995).

It is likely, however, that the use of the *DSM–IV* approach to ADHD subtyping will not be the most fruitful means of clinical description since diagnosis does not inform treatment or predict treatment response (Pelham, 2001). Furthermore, the different ADHD subtypes are based on behavioral criteria with no consideration of underlying pathophysiology. An alternate way of classifying subgroups of ADHD children may be according to their EEG patterns, which may reflect CNS abnormality. In studies examining whether there are distinct EEG defined subgroups of ADHD children, studies have found that both the ADHD–CT and ADHD–I have at least two distinct clusters with similar EEG profiles: a hypoaroused group and a maturational lag group (Clarke, Barry, McCarthy, Selikowitz, & Brown, 2002). Further work is needed to replicate these subgroups in other ADHD samples and to determine whether this approach to ADHD diagnosis can aid in treatment response and prediction. These findings may be consistent with research that suggests that a subset of ADHD children (perhaps 30%–50%) now classified as Inattentive Type may have qualitatively different problems with attention, cognitive, social, and academic functioning as well as treatment response profiles (Milich, Ballentine, & Lynam, 2001). This subset is referred to as having Sluggish Cognitive Tempo (SCT; McBurnett, Pfiffner, & Frick, 2001) and some have suggested that it may constitute a separate, distinct disorder from ADHD. More work is needed to determine whether one of these EEG defined subgroups is associated with the SCT subgroup.

EEG Findings Among Adolescents and Adults With ADHD

Studies that have examined developmental trends among normal and ADHD individuals have also documented EEG abnormalities across the lifespan in ADHD samples. Developmental studies of EEG among normal samples have found decreasing theta and increasing beta activity with age, with alpha activity initially increasing into adolescence and then decreasing into adulthood (Bresnahan & Barry, 2002; Gasser, Verleger, Bacher, & Sroka, 1988; John et al., 1983). The theta/beta ratio also decreases with increasing age among normal samples (Bresnahan et al., 1999;

Monastra et al., 2001). Similar to children with ADHD, adolescents with ADHD had higher levels of theta activity and higher theta/beta ratios across development, which remained abnormally high into adulthood. Beta activity was also significantly reduced among adolescents with ADHD when compared to normal samples; however, this normalized into adulthood, except in posterior locations (Bresnahan et al., 1999; El-Sayed et al., 2002; Hermens et al., 2004). Thus, these findings have led some to hypothesize that frontal–central theta activity is related to impulsivity and frontal–central beta activity with hyperactivity. This might mirror the clinical phenomenology of decreased gross motor activity as ADHD children get older; however, at the level of behavior ratings, hyperactivity and impulsivity form a single dimension of child behavior, not two distinct domains as suggested by these results (Achenbach, 2001; DuPaul, Power, Anastopoulos, & Reid, 1999). Attentional systems in the posterior regions of the brain (Levy & Swanson, 2001; Mirsky, 1996; Posner & Dehaene, 1994) may be related to EEG abnormalities in the alpha or beta bands seen in parietal regions.

Diagnostic Utility of EEG in ADHD

Sensitivity and specificity of EEG. To study the diagnostic utility of any new instrument (in this case EEG), one should compare its ability to correctly identify those with a diagnosis (made with the "gold standard") and those with no diagnosis. Sensitivity tells you what percent of ADHD children have an abnormal EEG and specificity tells you what percent of non-ADHD children have a normal EEG. In several studies, the EEG has demonstrated good sensitivity (90%–97%) and specificity (84%–94%; Chabot, Merkin, Wood, Davenport, & Serfontein, 1996; Monastra et al., 2001; Monastra et al., 1999). This means that, when you have a group of children with ADHD, a high percentage of kids will have a corresponding abnormal EEG (increased theta or high theta/beta ratio); in a comparison group of children with no ADHD, a high percentage of those children will have a normal EEG (lower levels of theta or lower theta–beta ratio). But more important from the standpoint of clinical diagnosis is positive (PPP) and negative predictive power (NPP). PPP tells you whether an abnormal EEG can correctly predict which children will receive a diagnosis of ADHD and NPP tells whether a normal EEG correctly predicts who will be normal or non-ADHD. The PPP and NPP for EEG have been reported to be 98% and 76%, respectively (Monastra et al., 2001), meaning that when there is an abnormal EEG (high theta/beta ratio in this case) it is highly likely that the child is ADHD. However, when the EEG is within the normal range, 24% of those children go on to be diagnosed as ADHD using other clinical methods. Most clinicians would consider this to be an unacceptably high rate of misdiagnosis for clinical purposes. Furthermore, a two-group comparison (ADHD vs. normal) of EEG diagnostic validity is not the most appropriate way to examine predictive power because most ADHD children referred to clinics have at least one if not two other comorbid disorders. Thus, the issue facing the clinician is not whether the referred case is disordered or normal, but rather, which disorder or set of disorders the case manifests among the various possible disorders (e.g., learning, depression, anxiety, etc.) occurring in a clinical practice.

Differentiating ADHD and other comorbid disorders. More instructive, therefore, are studies examining whether EEG can discriminate among ADHD, learning disorders, and other psychiatric disorders. Chabot and Serfontein published two papers (Chabot et al., 1996; Chabot & Serfontein, 1996) reporting discrimination between normal children and those with learning disabilities (LD) and ADHD. When comparing an ADHD sample to a normative database of normal and LD children (John et al., 1983), EEG was sensitive (93%–97%) and fairly specific (84%–90%) in differentiating ADHD from LD. Including the possibility that children may be normal lowers these values considerably with correct classification rates of 76% of normals, 89% of ADHD–ADD, and 70% of LD children (Chabot et al., 1996). There are also methodological issues that may affect the generality of these results. Children were diagnosed ADHD using only parent and teacher behavior rating scales, which may have led to misdiagnosis. In addition, the ADHD sample was compared to a normative database where LD children were not specifically screened for ADHD and included a very broad and heterogeneous group of children with learning difficulties (either low IQ or normal IQ with an achievement score less than 90).

When large samples of ADHD and LD children were directly compared, the discriminant validity of EEG appear high enough to be potentially useful (Chabot & Serfontein, 1996). Though the classification of ADHD alone and LD alone was good (97% and 84%, respectively), classification of ADHD children with and without learning disorders was not reliable (i.e., a split half replication was less than 60%). The best initial classification they obtained was 65% of ADHD only and 70% of ADHD + LD children. Although these classification

rates are significantly higher than chance, they still result in unacceptably high rates (20%–35%) of misclassification and therefore misdiagnosis. These studies do not support the clinical utility of EEG alone in differentiating between ADHD patients with and without learning disabilities.

In addition to learning disorders, most ADHD children referred to clinics have at least one if not two other comorbid psychiatric disorders, such as other disruptive behavior disorders, depression, anxiety, and substance abuse disorders (Barkley, 1998). It is hard to anticipate how these other disorders might affect the EEG measures and their capacity to discriminate ADHD from normal cases as well as those involving other disorders. Unpublished data (V. Monastra, personal communication, August, 2004) suggests that EEG has a sensitivity of 78% and specificity of 95% (with an overall classification rate of 86%) when differentiating between ADHD and oppositional, anxiety, and mood disorders. More systematic work in this area is needed. To date, all of the studies conducted thus far have examined children with ADHD who do not have other comorbid psychiatric conditions or where the proportion of comorbid disorders goes unspecified. Similarly, none of the studies to date have examined whether EEG can differentiate or accurately classify children having the different ADHD subtypes. Until EEG research addresses its utility in this context of diagnostic comorbidity, it should not be used clinically in the diagnosis of ADHD.

EEG and medication response. Relatively few EEG studies have examined medication response among ADHD children. ADHD children who are medication responders have been reported to have excessive slow wave activity (Clarke, Barry, McCarthy, & Selikowitz, 2002; Satterfield et al., 1984), supporting the theory that ADHD children are cortically hypoaroused. In addition, stimulant medication appears to "normalize" the EEG patterns and evoked potentials of children with ADHD (Jonkman et al., 1997; Verbaten et al., 1994; Winsberg, Javitt, & Silipo, 1997) and to decrease slow wave EEG (theta) activity and increase fast wave (beta) activity depending on the task and electrode location (Clarke, Barry, Bond, McCarthy, & Selikowitz, 2002; Loo, Teale, & Reite, 1999; Lubar, White, Swartwood, & Swartwood, 1999; Swartwood et al., 1998). Using EEG alone or a combination of behavioral and EEG measures, several studies have reported correct identification of 70%–80% of stimulant responders (Chabot, di Michele, Prichep, & John, 2001; Chabot et al., 1996; Prichep & John, 1992; Suffin & Emory, 1995). Similar predictive power rates (PPP and NPP ~70%) have been reported using ERP components such as the P3 (Sangal & Sangal, 2004). Though this may seem to be a relatively impressive predictive power of the EEG for predicting medication response, it is actually no better than the base rate one would have guessed in the absence of any EEG information. Research repeatedly finds that ~70% of ADHD children placed on a single stimulant demonstrate a positive response (Barkley, DuPaul, & McMurray, 1991; Cantwell, 1996; Findling, Short, & Manos, 2001). Unless the EEG can significantly surpass the prediction from the base rate, its utility in this respect is unimpressive.

Summary

Collectively, the EEG findings in children, adolescents, and adults with ADHD are increased slow-wave activity in frontal regions, suggesting cortical hypoarousal, especially in the ADHD Combined subtype. Several researchers have reported that EEG measures discriminate well between children with and without ADHD and others have asserted that the EEG works well in determining medication responders from nonresponders. There is preliminary evidence that EEG can differentiate ADHD subtypes, at least at the group level of comparison, but the requisite information on accuracy of individual classification is lacking.

Our conclusion, then, is that EEG alone, if used for diagnosis or prediction of treatment (i.e., stimulant) response, results in unacceptably high rates of misdiagnosis and misclassification. Although rates of 70%–80% classification are interesting at the research level and may be comparable to other assessment tools alone (e.g., rating scales or computerized tests), in a clinical setting, it means that 20%–30% of children will not receive the correct diagnosis or treatment. This suggests that the use of diagnostic instruments such as a structured or semistructured clinical interview, well-standardized behavior rating scales of ADHD symptoms, and information collected from multiple sources (parent, teacher, child) are still required. Because such measures must still be collected in evaluating anyone for ADHD, regardless of whether an EEG has been conducted, the EEG findings remain an interesting but nonessential piece of information in the diagnostic process. Though these findings indicate some promise for EEG as a diagnostic tool, additional systematic research to empirically validate its classification accuracy is needed.

EEG Biofeedback (Neurofeedback): The EEG as Treatment Device

Given the excess of theta and decreased beta activity observed among children with ADHD, it is easy to understand the theoretical basis for examining whether altering these problems through treatment would result in improvements in ADHD symptoms. This is the basic goal of EEG biofeedback, neurofeedback, or neurotherapy—to train the patient to decrease their slow wave activity and/or increase their fast wave EEG activity, often using behavioral principles such as operant conditioning (i.e., positive reinforcement) in the process. Typically, a neurofeedback therapist places one to three electrodes on the patient's head, which are connected to a computer. The computer detects the EEG information and provides a visual or auditory display of activity in the targeted frequency band(s). When the person is producing the desired EEG pattern (there are differential training programs for alpha or theta reduction and sensorimotor rhythm [SMR] or beta increase), the computer will give a positive response or reward, usually in the form of points earned. The person is then given a reward (e.g., money or other reinforcers) for earning a certain amount of points within each session. After many sessions of training, between 20 and 50 as currently practiced, it is hypothesized that a person will be able to produce the desired EEG brain waves on their own through increased awareness of their own physiological processes. Such conditioned EEG changes have been reported to be associated with improved or normalized symptoms of ADHD, to generalize outside the treatment setting (such as at home, school, or work) even when the treatment is withdrawn (Monastra, Monastra, & George, 2002) and to be maintained into adulthood in most treated cases (Lubar, 1991). Of note is the fact that no other treatment approach for ADHD has been able to demonstrate such generalization or maintenance effects (Pelham, Wheeler, & Chronis, 1998; Smith, Barkley, & Shapiro, in press).

This treatment has stirred up quite a controversy between the clinical and scientific communities working with ADHD. Recent reviews of EEG biofeedback have generally concluded that preliminary studies of EEG biofeedback are promising, but require further study in rigorous scientifically controlled studies (Arnold, 2001; Nash, 2000; Ramirez, Desantis, & Opler, 2001). Proponents of EEG biofeedback feel that their studies have been overly criticized and that the scientific community has been unfairly biased against neurofeedback treatment, despite large numbers of participants who have reportedly experienced positive outcomes. Critics of EEG biofeedback, however, contend that the published studies have suffered from significant methodological weaknesses that make interpretation of the results and conclusions about the actual effect of EEG biofeedback impossible.

Many of these flaws were identified a decade ago by Barkley (1992) and the same problems with scientific methodology that existed then continue to exist with these newer studies. The flaws included no control groups, the confounding of several different treatments within the EEG biofeedback group, use of small numbers of participants, diagnostic uncertainty about the children in the study, lack of placebo control procedures, absence of blindness of the evaluators to the treatment received by the cases, and practice effects with the measures being used to evaluate the ADHD children. Crucial yet lacking in most studies of EEG biofeedback has been the randomized assignment of cases to treatment and no-treatment (or placebo) groups. Instead, treatment groups are often constructed retrospectively from a series of clinical cases that have been previously treated or not with EEG biofeedback. Furthermore, there may exist a conflict of interest in these findings because EEG biofeedback studies are typically conducted by clinicians who are being paid to provide the treatment and are published in neurotherapy journals that do not have rigorous peer review. As Chambless and Hollon (1998) pointed out in their guidelines for defining empirically supported therapies, treatment efficacy must be demonstrated in controlled research where it is "reasonable to conclude that benefits observed are due to the effects of the treatment and not to chance or confounding factors such as the passage of time, the effects of psychological assessment, or the presence of different types of clients in the various treatment conditions." Thus, these are not petty or simply annoying issues that can be ignored. They are central to any demonstration of treatment efficacy. In the following paragraphs, we will review the most recent controlled studies of EEG biofeedback and offer a summary of where the state of EEG biofeedback lies currently.

EEG Biofeedback Versus No Treatment

The first controlled study was completed by Linden, Habib, and Radojevic (1996) and utilized small samples of ADHD patients; nine cases were randomly assigned to receive EEG biofeedback and nine were placed on a wait-list. Importantly, no other treatment was provided simultaneously including stimulant medication. The dependent measures included an IQ test

and two parent rating scales of ADHD symptoms and aggression. The neurofeedback group showed a significant increase in IQ and a significant decrease in parent ratings of inattention. There was no significant effect of EEG biofeedback on hyperactive–impulsive or aggressive behavior ratings.

This study is often cited as support for EEG biofeedback and does incorporate some important methodological controls such as random assignment, wait-list control, and treatment integrity. Noteworthy, however, is that no pre- and posttreatment comparisons in EEG power were reported to show that the treatment had altered the EEG parameters associated with ADHD. Also important to consider is that (a) no placebo control group was used to control for therapist time, attention, and other demand characteristics of the treatment environment; (b) parents evaluating the children before and after therapy were not blind to the treatment condition (nor were the children); and (c) the improvement on the IQ test is irrelevant to the demonstration of efficacy of this treatment for ADHD. IQ is not a measure of ADHD. Just as important, the overall or omnibus statistical analysis (multivariate analysis of variance, or MANOVA) of IQ and behavior ratings reported in this article did not find a significant effect of treatment group but rather a nonsignificant "trend" for time (pre- to posttreatment), meaning that all children, regardless of whether they had treatment or not, showed similar levels of improvement from the pretreatment to posttreatment evaluations. Though this may have been due to low power because of the small sample size, follow-up univariate tests of a nonsignificant MANOVA are not recommended and increase the risk of Type 1 (false positive) error (Weinfurt, 1998).

EEG Biofeedback Versus Placebo Biofeedback

Most EEG biofeedback studies suffer a glaring oversight and that is the failure to incorporate a placebo control condition. There have been many reasons put forth for not using a placebo control, such as difficulty designing a sham biofeedback that is not detectable by clinicians and patients, ethics of giving a placebo treatment for 6 months when other effective treatments are available, and feasibility of doing a placebo control condition within the context of a private clinical practice, which is where these studies have been conducted. Nonetheless, there is no other way to control for the effects of patient–therapist time, expectations generated by applying electrodes and being connected to a computer, ancillary support given to parents, and motivation and investment needed to complete treatment.

Only one study has used a placebo control group and is noteworthy for the degree of scientific rigor in its design. This was the unpublished paper by Fine, Goldman, and Sandford presented at the American Psychological Association meeting in 1994. In this study, 71 patients were randomly assigned to biofeedback, a no-treatment wait-list control group, or a placebo control group involving computerized cognitive training protocol. The authors collected 51 different measures, including 30 lab measures and parent ratings. Examiners doing the testing were blind to the treatment group assignment of these children; however parents were not. There were significant group differences on 12 measures, eight of which came from parent ratings. Of the four lab measures, just one favored the biofeedback group whereas the other groups did better on the remaining three. On the parent ratings, both treatment groups exceeded the wait-list control group on eight subscales from the three global rating scales. The biofeedback group was slightly better than the placebo group on two scales whereas the opposite was the case on the third rating scale, that being the Child Behavior Checklist (Fine, Goldman, & Sandford, 1994). In what is the most methodologically sound study on EEG biofeedback treatment outcome, using random assignment to groups and a placebo control group with examiner blindness to treatment assignment, no compelling evidence of efficacy for EEG biofeedback was evident.

Additionally, Heywood and Beale (2003) employed a single-subject design with a placebo control condition applied to a small sample of children ($N = 7$). The effects of EEG biofeedback were contrasted with a placebo (noncontingent) feedback condition. Outcome measures included parent and teacher behavior ratings as well as several cognitive tests (auditory and visual continuous performance tests, or CPTs, paired associate learning task, and verbal fluency task) during each of the conditions. Behavioral ratings and performance on cognitive tasks during active and placebo feedback conditions were compared and the results appear to support the effects of active EEG biofeedback on the dependent measures. These effects disappear, however, when controlling for overall trend of the data (which helps to account for maturation and nonspecific treatment effects) and including treatment noncompleters (known as an intent-to-treat design). Furthermore, the effects of active and placebo biofeedback do not result in changes in the treatment outcome measures that differ significantly from baseline measures. Thus, one might mistakenly conclude that there is a significant

treatment effect of EEG biofeedback only if maturation and nonspecific effects as well as treatment noncompleters are ignored.

Overall, of the three treatment outcome studies comparing EEG biofeedback to either no-treatment or placebo control conditions, two fail to support an active treatment effect. These studies are, methodologically speaking, the three strongest. Though the small sample sizes in the Linden et al. study may have limited statistical power for comparisons, the Fine et al. study had large sample sizes providing sufficient statistical power to detect differences between conditions had they been present.

EEG Biofeedback Versus Other Treatments (Medication and Psychological)

There have been three studies that have compared neurotherapy to other treatments, all including psychostimulant medication that is the gold standard in treatment for ADHD. If EEG biofeedback treatment demonstrated treatment effects that are similar (or not significantly inferior) to stimulant medication treatment, this might be taken as an indicator of equivalence in efficacy (Chambless & Hollon, 1998). Unfortunately, none of these studies used random assignment. Instead, they reconstructed their treatment groups after the fact of treatment (months or years) using samples of clinically treated patients or allowed patients to self-select into the treatment they preferred. Also, these studies failed to report psychiatric or learning disorders that often are comorbidities with ADHD, and did not incorporate evaluators who were blind to the patient's treatment condition. In addition, only one study tested EEG biofeedback by itself without confounding it with additional treatments.

Rossiter and LaVaque (1995) were the first to compare EEG biofeedback to stimulant medication in groups (23 in each group) of children and adults with ADHD (ages 8 to 21 years). Rather than randomly assigning cases to each treatment group, the authors matched the cases of those who previously received EEG treatment against those who had received stimulant therapy (ages 5–45 years) using age as a matching criterion. Again, the absence of random assignment to treatment groups is an important methodological oversight here because such randomization helps to minimize inherent biases such as self-selection into the various treatments and experimenter bias in choosing the patients in treatment group assignment that confound efforts to draw conclusions from group comparisons. The patients were assigned to treatment groups based in part on their preferences, in part on whether they had previously failed stimulant therapy, and in part on insurance coverage for biofeedback. Furthermore, medication (to five of the EEG cases) and additional treatments were provided to all cases, confounding the two treatment groups and making interpretation of individual treatment effects (medication and EEG biofeedback) impossible. The authors reported using the Test of Variables of Attention (TOVA), a continuous performance test assessing inattention and impulsiveness, and a parent rating scale of behavioral problems, though not the same one for all participants. Cases and their parents were not blind to their treatment condition, nor were the examiners testing the cases on the lab measures blind to such assignment. Also, no information is provided as to just how these cases were selected from the larger pool of clients likely treated in this practice. Were all available cases within a specified period of time reclassified into these post hoc treatment groups or just some? If not all, why were some chosen for inclusion in these analyses and others not?

From pre- to posttest, the EEG biofeedback group showed significant improvement on the TOVA and on parent ratings of inattention, hyperactivity, and internalizing symptoms. So did the medication group, with no differences between them in the degree of change shown. Important to note is that, here again, pre- and posttreatment EEG measures were not reported so as to show that the biofeedback had changed the important parameters of the EEG believed to mediate the changes in ADHD symptoms. Although the authors conclude that, for children who do not respond to medications, EEG biofeedback is a good treatment choice, the significant scientific design problems (i.e., absence of random assignment to treatments, confounding of treatments, and lack of reported EEG changes) prohibit making such a conclusion.

In their study on EEG biofeedback, Monastra et al. (2002) reported results from samples of 51 ADHD children (6 to 19 years old) who received comprehensive clinical care (CCC; medication, parent counseling, academic support) with EEG biofeedback for 1 year and 50 who had received CCC alone (no biofeedback). Again, patients were not randomly assigned to the treatment groups. The fact that groups were found to not differ on pretreatment scores on either the measures of ADHD (ratings) or EEG measures is not very reassuring given that many other variables can operate to bias treatment studies such as this one absent random assignment to treatment groups before initiating therapy. Results of the study indicate that children in the CCC + EEG bio-

feedback (CCC + B) group were better at posttreatment on behavior ratings of attention and hyperactivity–impulsive behaviors (on and off medication), as well as on the TOVA (only when tested off medication), when compared to the CCC group. In addition, at posttreatment those in the EEG group had lower theta/beta ratios than the CCC group. These results indicate improved functioning in the CCC + B group even when off medication; however, the significant differences are primarily due (surprisingly) to virtually no improvement in the CCC group. Close examination of the pre- and posttreatment behavior rating scale scores (on and off medication) indicate that the CCC alone group appear to have received a degraded version of the CCC or are treatment nonresponders. This atypical patient group as a comparison coupled with the lack of random assignment, variation in individual treatment components, failure to control for the amount of time spent with a therapist, and lack of information as to just how patients were chosen from the larger treated pool prohibits interpretation concerning efficacy of any specific treatment component (EEG biofeedback without all of the other treatments).

The Fuchs et al. (Fuchs, Birbaumer, Lutzenberger, Gruzelier, & Kaiser, 2003) study is the only study thus far that involves a direct comparison between EEG biofeedback (N = 22) and stimulant medication (N = 11) where the treatments are not confounded (i.e., stimulants given to the EEG biofeedback group). As with the previous studies, the sample description lacks important information regarding ADHD subtype and psychiatric or learning disorder comorbidity. In addition, the readministration of the WISC intelligence test within such a short time period (12 weeks) invalidates the results of the posttreatment test. Methodological issues (no random assignment, no control for additional therapist time, small sample size, no information on the larger pool of treated patients from which these cases were selected and why) notwithstanding, these results may suggest that EEG biofeedback and methylphenidate result in similar levels of short-term change in ADHD behaviors. Yet those methodological issues are crucial to being able to say anything about such a treatment effect. Replication of these findings with increased scientific controls and larger sample sizes (at least 25–30 respondents per condition; Chambless & Hollon, 1998) will be a necessary step toward establishing EEG biofeedback as an equivalent treatment to medication. To demonstrate that EEG changes are responsible for treatment effects, reporting of actual EEG changes and correlation with treatment outcome must be shown.

Is EEG Conditioning the Active Ingredient in Biofeedback?

The reason we come back over and over again to scientific methodology is that proper experimental controls makes it possible to discern whether training EEG patterns is the active ingredient in the treatment. In fact, one of the biggest issues that the EEG biofeedback treatment literature needs to address is whether or not it is actually the training of the EEG patterns that leads to improvement in ADHD symptoms. Though the goal of EEG biofeedback is the "unconscious conditioning of underlying neurological systems [to] learn balance through reinforced practice" (Monastra, 2004), none of the studies thus far have demonstrated that the EEG changes are the actual mediator of treatment outcome. In an earlier EEG biofeedback study (Lubar, Swartwood, Swartwood, & O'Donnell, 1995), ~60% of children showed EEG changes with biofeedback treatment. The children who showed EEG changes (decreased theta) also exhibited significantly greater improvement on the TOVA (three of four scales improved) when compared to those whose EEG did not change (one of four scales improved). And yet, there are significant overall treatment effects in several studies. This indicates that other nonspecific or unintentional factors are present in the treatments that are helping bring about behavioral and cognitive improvement. But if it isn't EEG change, what else might be at work to elicit the behavioral and cognitive improvements reported in these studies?

First, there are several nonspecific factors that may result in ADHD symptom improvement. Children in EEG biofeedback conditions, based on the study descriptions, received additional time with a therapist ranging from 17 to 40 hours across studies than did cases not receiving biofeedback. Failure to control for the amount of treatment time means that the EEG biofeedback group may have improved simply because they spent more time with a therapist, are more invested in treatment and therefore more motivated to change, or may have more stability and support from mental health professionals rather than the EEG biofeedback per se, or may simply have been more likely to want to please the therapist.

Another possibility is that biofeedback is simply another form of cognitive–behavioral training that just happens to employ the use of electrodes placed on the head. Under this scenario, it is not anything to do with the electrodes or EEG that necessarily produces the treatment effect. Instead, it is whatever conscious cognitive or behavioral actions the individual is actively

employing to alter the EEG activity that is being conditioned. For instance, in some studies, children are told to focus their concentration on some object or on some imagined condition, such as being a heavy rock. In others they are told to find some mental activity that results in a change in their performance of the videogame being used to give them feedback about their EEG status. In others, they are told simply to try to do better at the videogame. Even advocates of EEG biofeedback concede that "attentional training through behavioral methods cannot be ruled out" (Linden et al., 1996) and that the "factors that are essential in teaching attention/concentration remain an empirical question" (Monastra, 2004). Noteworthy here is that cognitive behavioral training has not been found to be effective in treating ADHD (DuPaul & Eckert, 1997; Pelham et al., 1998). Yet, rarely have children had to perform this sort of sustained practice (for 30–50 hr) and received such salient rewards (up to $150) for successful performance. Research studies have repeatedly shown that ADHD children's performance on cognitive tests can be normalized with immediate and salient reinforcers (Firestone & Douglas, 1975; Oosterlaan & Sergeant, 1998). This is also likely to lead to stimulus generalization when children come into the lab for posttreatment EEG and TOVA assessments (Heywood & Beale, 2003). Thus, when children are completing the posttreatment session, they may be expecting similar rewards for performance; these behaviors may not continue, however, in other settings and if performance is not continually rewarded. Similarly, it has been suggested that altered breathing patterns may minimize theta activity, which may be a separate but correlated mechanism for treatment effects thought to be the result of EEG biofeedback (Heywood & Beale, 2003). This is consistent with some neurotherapy treatment protocols that encourage the patient to relax, which most likely leads to deeper breathing and increased oxygenation of blood cells in the brain. Perhaps it is the conditioning of deeper breathing and therefore increased cerebral perfusion that improves ADHD symptoms.

If it is not the reinforced conditioning of EEG activity per se, then the use of computers, electrodes, and amplifiers are unnecessary, similar to what was found for the EMG treatments in the 1970s—teaching muscle relaxation proved sufficient. This should lead to a less expensive, more targeted treatment focused on the active ingredient of this treatment. If, as proponents of EEG biofeedback state, it is the EEG conditioning that produces the balance among the underlying neurological systems, then additional studies are needed to demonstrate this specific effect. While EEG biofeedback studies with seizure patients have demonstrated correlation between EEG changes and clinical symptomatology (Sterman, 2000), this has not yet been demonstrated in ADHD.

Summary

Although the existing studies of EEG biofeedback claim promising results in the treatment of ADHD, the promise of EEG biofeedback as a legitimate treatment cannot be fulfilled without studies that are scientifically rigorous. Undoubtedly, treatments for ADHD would benefit greatly from a nonmedication alternative that is efficacious and cost effective. But there is much work to be done to demonstrate that EEG biofeedback provides that alternative and that actually changing the EEG is the mechanism of change in ADHD symptoms (as opposed to just more time with a therapist). Without such demonstrations, the changes in behavior cannot in fact be attributed to this specific treatment mechanism. It must also be shown that treatment effects can generalize to nontreatment settings and can persist over time. Even with such demonstrations it must also be shown that treatment is cost effective in managing the symptoms of ADHD relative to the prevailing empirically supported approaches.

Future Directions for Research on the Clinical Utility of EEG in ADHD

If EEG is to be used as a diagnostic tool for ADHD, there has to be much greater clarity on its ability to differentiate ADHD from normal children, ADHD subtypes from each other, and to assess for differential diagnoses as well as ADHD comorbidities. Work documenting correlations between EEG and ADHD symptoms and subtypes is needed. Two studies (Chabot & Serfontein, 1996; Clarke, Barry, McCarthy, & Selikowitz, 2001b) have identified EEG-defined subtypes within ADHD, with one group exhibiting a higher than normal beta power in both samples. Replication of these subtypes and greater description of how they relate to current diagnostic subgroups and treatment outcome seems warranted, particularly among the excess beta group. Though some work has been done to examine the diagnostic utility of EEG in ADHD, more systematic study needs to be done using rigorous diagnostic procedures (i.e., structured or semistructured diagnostic interviews), careful identification of comorbid diagnoses (including specific learning disorders) and impact of these disorders on EEG characteristics. In addition, studies examining EEG correlates of stimu-

lant response should incorporate double-blind medication titration and reporting of EEG differences according to varying doses of medication.

As for EEG biofeedback as a treatment for ADHD, there are clearly many issues that need to be addressed adequately in future research. The first and foremost is addressing the methodological problems that have plagued this treatment outcome research from the start. Proper scientific controls are crucial to demonstrating that there is a real treatment effect due to EEG biofeedback and that EEG conditioning is the effective ingredient within the treatment. This will require clinical trials that incorporate random assignment to treated and untreated groups, placebo conditions, larger sample sizes, evaluators that are blind to treatment condition, clear and comprehensive sample description (particularly with regard to psychiatric and learning disorder comorbidity), appropriate data analytic (statistical) procedures, and documentation of EEG changes that correlate with treatment outcome. These methodological difficulties compromise the internal validity of most of the studies reviewed here, making interpretation of the results and conclusions about the actual effect of treatment impossible.

Finally, side effects of EEG biofeedback must be monitored systematically and reported in studies. All truly effective treatments produce some side effects in some percentage of the population. This has to be so because individuals differ in their physiological makeup, particularly brain organization and functioning. Those individual differences are sufficient to result in the treatment producing adverse effects in a subset of the population. Moreover, clinical ineptitude in the delivery of the treatment in some cases and as a consequence of comorbid disorders in other cases always ensures that some patients will not respond well to the intervention as delivered. This is as true for behavioral interventions as it is for medications. Hence any claim that a treatment is effective yet has absolutely no associated side effects is oxymoronic. The former cannot exist without the latter. This may be a telling piece of information about whether neurofeedback is actually effective for the management of ADHD.

Conclusion

The clinical utility of EEG in ADHD has yet to be proven. Though there are some promising results that require further study, the threshold for using EEG clinically has not been met. Of the possible uses reviewed here (diagnostic utility, prediction of stimulant response, and EEG biofeedback), the diagnostic utility of EEG appears most promising although considerable work is needed for this promise to be realized. The EEG biofeedback studies with the most rigorous methodologies to date have not supported the efficacy of EEG biofeedback when compared to no-treatment control or placebo feedback. Methodological flaws of previous EEG studies have hampered firm conclusions regarding its usefulness and precision. Though the field of ADHD would benefit greatly from a single diagnostic test and an effective nonmedication treatment alternative, we cannot recommend the use of EEG in a clinical setting based on the current empirical data.

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