Biofeedback and Self-Regulation, Vol. 21, No. 1, 1996

A Controlled Study of the Effects of EEG Biofeedback on Cognition and Behavior of Children with Attention Deficit Disorder and Learning Disabilities[1]

Michael Linden,[2] Thomas Habib, and Vesna Radojevic
Mission Psychological Consultants, San Juan Capistrano

Eighteen children with ADD/ADHD, some of whom were also LD, ranging in ages from 5 through 15 were randomly assigned to one of two conditions. The experimental condition consisted of 40 45-minute sessions of training in enhancing beta activity and suppressing theta activity, spaced over 6 months. The control condition, waiting list group, received no EEG biofeedback. No other psychological treatment or medication was administered to any subjects. All subjects were measured at pretreatment and at posttreatment on an IQ test and parent behavior rating scales for inattention, hyperactivity, and aggressive/defiant (oppositional) behaviors. At posttreatment the experimental group demonstrated a significant increase (mean of 9 points) on the K-Bit IQ Composite as compared to the control group ($p < .05$). The experimental group also significantly reduced inattentive behaviors as rated by parents ($p < .05$). The significant improvements in intellectual functioning and attentive behaviors might be explained as a result of the attentional enhancement affected by EEG biofeedback training. Further research utilizing improved data collection and analysis, more stringent control groups, and larger sample sizes are needed to support and replicate these findings.

[1]This research was supported by an equipment grant by Autogenics Systems. Portions of this paper were presented at the annual convention of the Association of Applied Psychophysiology and Biofeedback, March, 1993 in Los Angeles and at the annual meeting of the Biofeedback Society of California, November, 1992 in Monterey, California. The authors gratefully acknowledge Todd Fischer and Paul Clopton for their valuable assistance in statistical analysis for this article.
[2]Address correspondence to Michael K. Linden, Ph.D., Mission Psychological Consultants, 30270 Rancho Viejo Road, Suite C, San Juan Capistrano, California 92675.

0363-3586/96/0300-0035$09.50/0

Descriptor Key Words: EEG biofeedback; attention deficit disorder; attention deficit hyperactivity disorder; intelligence; learning disabilities.

Attention Deficit Disorder (ADD) is one of the most common psychiatric disorders of childhood. Estimates of the prevalence of ADD range from 5% to 15% of school-aged children (Barkley, 1990; Linden, 1988, 1991). Together with the associated disorders of Hyperactivity, Oppositional Defiant Disorder, and Learning Disabilities, about 20% of all children may be affected. The ramifications of these disorders are widespread, not only affecting school performance, but also compromising parenting at home and peer relations.

Previously ADD was of concern only for children. However, it is now believed that 70% of children do not outgrow the attention and learning problems associated with ADD (Lambert, Hantsough, Sassone, & Sandoval, 1987). Research over the last 15 years (Lubar & Lubar, 1984; Zametkin, Nordal, Gross, King, Semple, Rumsey, Hamburger, & Cohen, 1990; Linden, 1991; Lubar, 1992) further suggests that ADD may have a genetic and biological component which can be measured physiologically using Event-Related Brain Potentials, EEGs, and PET scans.

Learning disabilities (LD) have been noted since the advent of formalized education. Recently a biological basis for learning disabilities has been explored. Biological factors such as premature birth, birth by an older mother, and closed head injury may contribute to LD (American Psychiatric Association, 1980). In most cases the disorder persists throughout childhood and adolescence, and many individuals continue to show some residual symptoms of the disturbance in adult life. The finding that ADD and LD do not resolve by adulthood and that incidence rates are much greater among family members (American Psychiatric Association, 1980) also supports a genetic hypothesis.

Until recently, the treatment of ADD children has been limited primarily to pharmacological and behavioral approaches. The use of stimulant medications such as methylphenidate (Ritalin) is the most common treatment for ADD even though its effects are temporary (e.g., the half-life of Ritalin is approximately four hours). Furthermore, numerous side effects of Ritalin, such as loss of appetite, inhibited growth, insomnia, depression, and motor tics are common.

Another approach to management has been the use of behavioral treatment. However, behavior modification techniques are complicated and time consuming, and lack of consistency and follow-through can reduce the intervention's effectiveness. A nonpharmacological treatment with lasting results and minimal side effects has been unavailable for these disorders.

Research efforts into new treatment options are vital considering the extent and intractability of these disorders.

In the past decade, EEG has emerged as another potential diagnostic assessment and treatment for ADD and LD. Winkler, Dixon, and Parker (1970), in a study of 24 children exhibiting scholastic and behavioral problems and 24 normal controls, revealed that there was more diffuse, rhythmically slow wave, specifically theta activity (4-8 Hz), and less faster wave beta sensorimotor rhythm (SMR) activity (12-20 Hz), and a greater incidence of abnormal transient discharges in the problematic group. Satterfield (1973) found that EEG and electrodermal responses covaried with the level of arousal in ADD children, with hyperactive children having underarousal. This finding of underarousal correlates with low amplitude in EEG beta frequencies found in this population.

The first outcome study of EEG biofeedback for hyperactive and LD children (Nall, 1973) used 48 children, and found no significant academic or behavioral differences between contingent and noncontingent alpha feedback (8-13 Hz) group and a no-treatment control condition. However, alpha feedback mainly assists in relaxation, and may not be accountable for increasing concentration (beta; 16-20 Hz) and decreasing daydreaming (theta).

Lubar and Shouse (1976) conducted the initial EEG biofeedback study using specific beta activity enhancement, theta suppression, and academic training with four ADD and LD children. The academic training consisted of reading, arithmetic, and spatial tasks to improve the children's attention. The single-blind ABA design indicated that the biofeedback normalized physiological indicators of low arousal (P300 auditory evoked potential, electrodermal response) and brainwave patterns, and increased grades and achievement scores. Although this study was descriptive in nature and had a limited sample size and absence of appropriate control groups, it promoted the initial rationale and basic methodology for exploring EEG biofeedback as a potential treatment modality for ADD and LD children.

Event-related potentials (ERPs) are EEGs recorded during a visual or auditory task in which one has to discriminate frequent from infrequent stimuli. This type of recording is measured between the occurrence of the stimuli and the subjects' response. Studies of ERPs with ADD children (Satterfield & Braley, 1977; Linden, 1991) found high amplitude early components and slow latency late components, which correlate with findings of low SMR/beta and high theta.

The underarousal and underactivity theories of ADD received additional support from Zametkin et al. (1990), who found frontal hypometabolism in the PET scans of adults with ADD. EEG biofeedback which activates the Beta/SMR frequencies related to increased motor con-

trol and arousal and suppresses the alpha/theta frequencies related to sensory overstimulation is hypothesized to directly impact these brain abnormalities and facilitate the attentional and behavioral improvements reported in previous research with ADD children.

Muehl, Knott, and Benton (1965) reported that 63% of children with learning disabilities had EEG abnormalities, as compared with 20% of controls matched for age and IQ scores.

The initial investigation using EEG biofeedback for LD was a case study of one child with a developmental reading disorder, oculo-motor-vestibular dysfunctions, and hyperactivity. Although successful results were reported (Tansey & Bruner, 1983), the generalizability of this research study is limited.

Lubar and Lubar (1984), in a study of six LD children who received variable numbers of SMR and beta EEG biofeedback sessions either alone or in combination with academic training, found improvements in learning and academic performance as measured by standard achievement test scores. However, the absence of control conditions, the presence of a small sample size, and the lack of consistency in the treatment protocol render these results tentative.

Lubar, Bianchini, Calhoun, Lambert, Brody, and Shabsin (1985) collected EEG data on 69 LD and 34 normal children. The authors found that LD children exhibited significantly more slow theta brainwave activity and more EMG activity recorded from scalp electrodes. Furthermore, it was possible to predict group membership (LD or normal) with greater than 95% accuracy using discriminant analysis of the EEG data.

Tansey (1985, 1990, 1991) studied the effects of EEG biofeedback on LD children and reported successful results, including WISC-R Full Scale IQ increases averaging 20 points. However, these studies also suffered from similar methodological problems; e.g., absence of appropriate control conditions. The mechanisms by which this treatment affects brain impairments are assumed to have their basis in neurochemical and/or neuroelectrical input under the subjects' direct control. According to Tansey (1991), while traditional treatments for LD take an indirect approach to underlying central nervous system (CNS) impairments, EEG biofeedback may directly impact the LD child's impaired CNS by normalizing their brain wave signatures.

Previous studies of EEG have provided some clarification in the mechanisms underlying ADD and LD. In addition, EEG biofeedback treatment outcome studies for ADD and/or LD have reported promising results not only in significant reductions in hyperactive, inattentive, and disruptive behaviors, but also improvements in academic performance and IQ scores. However, all of the previous studies of EEG biofeedback treatment lack

appropriate control groups, the use of standard protocols for training, and evaluation of EEG biofeedback as an independent treatment component. Finally, the majority of previous research suffers from small sample sizes. Objectives of the current study were to (1) decipher the effects of EEG biofeedback when compared with a waiting list control group receiving no treatment, (2) control for standardized amount of sessions, and (3) replicate previous research using similar treatment and dependent measures. It was predicted that the group receiving EEG biofeedback would show more improvement than the controls on all measures under investigation.

METHODS

Subjects

Eighteen subjects were randomly assigned from community referrals to an outpatient clinic, Mission Psychological Consultants (MPC). The subjects were children aged 5-15 with a primary diagnosis of ADD/ADHD (n = 12) and some also with LD (n = 6). All subjects were diagnosed using DSM-III-R criteria (American Psychiatric Association, 1987) collected from the following sources: (1) family history, (2) teacher behavior rating scales, (3) parent behavior rating scales,.(4) family interview, (5) developmental history, and (6) psychoeducational testing including intelligence and achievement tests. Children who had mental retardation, depression, anxiety disorders, and adjustment disorder as a principal diagnosis were excluded.

Procedure

The 18 subjects were randomly assigned to one of two groups: an experimental group, which underwent 40 sessions of the EEG biofeedback treatment over six months (n = 9), or a waiting-list control group, which underwent no EEG biofeedback treatment during the same period of time (n = 9). Equal numbers of ADD/ADHD (n = 6) and ADD/ADHD with LD (n = 3) subjects were represented in each treatment group, and statistical analysis indicated that the two groups were equivalent on diagnostic representation. A waiting list control group was utilized because the option of performing placebo EEG biofeedback for a six-month time period was decided to be unethical by both the human subjects committee and the biofeedback manufacturer consultants to this study.

To control for other treatment effects, all subjects in this study were not prescribed or taking any medication for ADD (e.g., Ritalin) or involved in other treatments (e.g., counseling, tutoring, etc.) for their disorders during the six-month duration of their participation in this study. The waiting-list control subjects were offered the experimental treatment after completion of the six-month waiting period as an incentive to remain in the research study without starting any other treatments.

The dependent measure of intelligence (IQ) and parent behavior rating scales were collected prior to and after six months of treatment. The IQ examiners and research assistants that scored the rating scales were blind to subjects' group assignment. The principal investigator randomly assigned the subjects into the two groups.

The EEG biofeedback sessions were 45 minutes in length and consisted of electrode attachment and 3 10-minute EEG biofeedback segments: (1) standard training (biofeedback with eyes open while attending to visual and auditory feedback), (2) a reading task (biofeedback during reading age appropriate books), and (3) an auditory listening task (biofeedback while an assistant read age appropriate material to them). During the reading and listening sessions, if the subject stopped receiving feedback rewards (points or tones) the task was temporarily stopped and the subject was instructed to concentrate until the rewards began, and then continue reading or listening. Some of the feedback rewards supplied by the Autogenic instrumentation were presented in a video game format. At the conclusion of each training day, the subjects were given small rewards (e.g., baseball cards, stickers), which were provided based on their levels of cooperation, effort, and performance.

The subjects attended two sessions per week, either after school or on the weekends. The first author trained all the EEG neurotherapists who conducted individual sessions for each subject. The neurotherapists, who were constantly in the room sitting next to the subject, instructed the children in learning the feedback process, recorded the subjects' EEG data after each task, and monitored their EEG recordings to ensure accurate EEG feedback. The children were encouraged to become aware of their brainwave activity and develop their own strategies to obtain the highest amount of reinforcement; however, the neurotherapists were available to assist the subjects in developing and recalling strategies when necessary.

The visual and auditory feedback was provided via color VGA monitors and audio speakers within the computer. Biofeedback was performed to suppress (decrease) the theta (4-8 Hz) and enhance (increase) the beta (16-20 Hz) bands of the EEG. Audio (tones or beeps) and visual (graphs, game movement, or points) feedback was attained by the subjects when three conditions were met simultaneously: (1) the beta amplitude was above

its threshold; (2) the theta amplitude was below its threshold; and (3) the EMG (muscle artifact) was below its threshold. The EEG brainwaves were shaped to decrease the theta/beta ratio relative to the subject's performance by gradually decreasing the thresholds for theta and increasing the thresholds for beta as the training sessions progressed. This technique was performed in a similar manner for each child throughout the 40 sessions, regardless of the child's age or diagnosis.

Biofeedback subjects were connected to the biofeedback system using three Grass gold-plated electrodes attached to their scalp at the bipolar placements of Cz and Pz and grounded at the right ear. Omni Prep was used to prepare the skin and 10-20 or EC-2 electrode paste was used to connect the electrodes to the skin. All electrode skin contact was checked by a Checktrode meter and kept below 10 kΩ impedance in order to ensure accurate readings. Muscle tension artifact was controlled by inhibiting biofeedback reinforcement if the EMG recorded from the scale exceeded 15 μV.

Biocomp and Autogenic A620 instrumentation were used to collect the EEG data and record it on hard disk. The initial four subjects began using Biocomp equipment specially modified to record and provide feedback of EEG beta and theta activity. However, as soon as the Autogenic equipment became available, these initial four subjects' and the remaining sample's EEG biofeedback training was conducted on the then acquired Autogenic instrumentation, which was developed specifically for use with ADD children.

The specifications of the A620 Autogenic instrumentation is as follows. The EEG was filtered using a high pass filter at 0.5 Hz. The gain was 50,000, the differential input impedance was 200 kΩ, and the common mode rejection ratio was greater than 110 db. The sampling rate was 128 per second with an A/D resolution of 0.05 μV.

The ratio of theta:beta was used to assess changes in the EEG over time as a basis to modify the shaping procedures, by guiding the beta threshold settings higher and the theta settings lower, and to assess improvement. This ratio was used instead of the actual theta and beta levels because it controls for the gradual EEG differences between the younger and older ADD subjects (i.e., as individuals become older, both their theta and beta amplitudes decrease, but their theta:beta ratio remains more consistent). Unfortunately, because of software restrictions and revisions on both the Biocomp and Autogenics equipment, the EEG data and thus threshold settings were inconsistent. Therefore, these EEG data were not conducive to statistical analysis because the comparisons would be meaningless due to the variability in the equipment.

Dependent Measures

Intelligence (IQ). The dependent measure for IQ was the Composite IQ score on the Kaufman—Brief Intelligence Test (K-BIT). The K-BIT is a brief, individually administered measure of verbal and nonverbal intelligence. The test requires approximately 20 to 30 min to administer and has two subtests: (1) Vocabulary (including Part A, Expressive Vocabulary, and Part B, Definitions) and (2) Matrices. Age-normed standard scores with a mean of 100 and a standard deviation of 15 are provided for the overall score on the K-BIT, known as the K-BIT IQ Composite. The K-BIT's standard scores are comparable to other intelligence tests such as the Weschler Intelligence Scale for Children—Revised (WISC-R) (Kaufman & Kaufman, 1990).

The construct validity of the K-BIT IQ Composite was compared to both brief intelligence tests such as the Slosson Intelligence Test and comprehensive tests such as the WISC-R. The K-BIT IQ Composite correlated highly with the WISC-R Full Scale IQ (r = .80), supporting the construct validity of the K-Bit IQ Composite (Kaufman & Kaufman, 1990).

The K-BIT was selected because its use for repeated measures (test-retest) was better than the longer Weschler IQ tests because of its brevity (Kaufman & Kaufman, 1990). IQ was measured in order to compare the results of the current study to previous studies of EEG biofeedback with ADD children. According to Kaufman and Kaufman (1990), the K-BIT is recommended for use in measuring global intelligence of various groups for research purposes. Split-half reliability for the K-BIT IQ Composite is acceptable, with values ranging from, r = .88 to r = .94, for ages 5 to 15 years. The test-retest reliability, quite important in this study of treatment changes over time, varies between r = .92 and r = .93, for ages 5 to 15. Overall, the test-retest correlation coefficients corroborate the split-half results, offering strong support for the reliability of the K-BIT IQ Composite score (Kaufman & Kaufman, 1990). Because of the high reliability, the K-BIT IQ Composite has low standard errors of measurement between 3.7 and 5.2 for the ages of interest in this study, 5 to 15 years.

Behavioral Ratings. This dependent measure was composed of three scales from two common behavior rating scales. The parent IOWA-Conners behavior rating scale was used for measures of inattentive and overactive (I/O or ADHD) behaviors seen in ADD patients, and aggressive and defiant (A/D) behaviors seen in ODD patients. In addition, the parent SNAP behavior rating scale index for inattentive behaviors was used to assess the presence of ADD behavior without hyperactivity. These behavior rating scales were selected for their ability to differentiate subgroups of ADD children, and most importantly their ability to be used for repeated meas-

ures (i.e., the scales are routinely used as repeated measures of medication effectiveness).

The IOWA-Conners Behavior Rating Scale is a 10-item scale designed to provide a standard measure of attention deficit/hyperactivity and aggression in children. There are two subscales, each consisting of 5 items: the Inattention/Overactivity scale and the Aggressive scale. The items contained on the two rating subscales were derived from the Conners Teacher Rating Scale, the most commonly used rating scale for research and treatment of hyperactivity and related disorders (Atkins & Milich, 1987). Items are scored by a child's classroom teacher or parent as occurring on a continuum, 0 = "not at all," 1 = "just a little," 2 = "pretty much," or 3 = "very much."

The test-retest reliability for the IOWA-Conners is $r = .89$ for the inattentive/overactivity subscale (I/O) and $r = .86$ for the aggressive subscale (A/D). Internal stability coefficients are $r = .80$ (I/O) and $r = .87$ (A/D). Validity of the IOWA-Conners has been established with comparison to other behavioral rating scales and methods. Significant correlations were reported for the I/O scale with the Hyperactivity factor of the Conners Teacher rating Scale, and for the A/D scale with the Conduct Problem factor of the Conners Teacher Rating Scale. Convergent and discriminant validity for these scales was evidenced with classroom observations of clinic referred boys (Milich & Fitzgerald, 1985) and playroom observation data (Milich, Loney, & Landau, 1982).

The SNAP questionnaire was developed by Swanson, Nolan, and Pelham (1981). It consists of 46 items taken from the DSM-III symptoms of ADHD, the DSM-III-R symptoms of ADHD and Oppositional Defiant Disorder (ODD), and the Carlson/Lahey items for ADD without hyperactivity or undifferentiated ADD (sometimes referred to as ADD in comparison to ADHD). The Inattention subscale contains 5 items. The items are scored in the same manner as the above IOWA-Conners Rating scale. The reliability and validity of the Inattention subscale of the SNAP are similar to the IOWA-Conners.

RESULTS

Pretreatment Analyses

Multivariate analysis of variance (MANOVA) was conducted on the pretreatment dependent measures of IQ, inattention, overactivity (hyperactivity), and aggressive-defiant (ODD) behavior. The two conditions did not significantly vary on any of these measures, suggesting that the groups were

Table I. Mean IQ and Parent Rating Scores for the Experimental and Control Groups before and after Treatment

Variable	EEG Biofeedback group		Control group	
	Pre	Post	Pre	Post
IQ	101.1	110.4*	99.1	100.0
Inattention	11.33	8.11*	12.00	12.44
Hyperactivity	8.56	4.77	9.83	9.00
Aggressive/defiant	5.66	3.00	8.45	7.45

*$p < .05$.

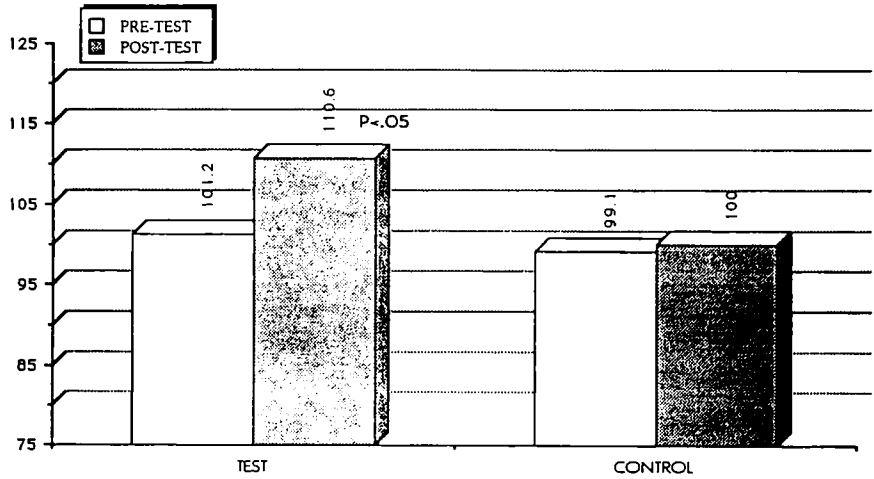

Fig. 1. EEG biofeedback effects on inattentive behavior.

equivalent at entry into the study. Since the mean age for each group was identical, 9 years and 2 months, no analysis was necessary for age effects.

Treatment Outcome Analyses

A 2 (Experimental vs. Control) × 2 (pre vs. post) MANOVA for the IQ and behavioral rating variables did not demonstrate significant main or interaction effects. However, a trend emerged approaching significance for the main effect of time. Since specific predictions were made (that the Experimental group would outperform the Control group), follow-up ANOVAs were employed to test the trials effect further. The variable of IQ was significantly enhanced at posttreatment for the EEG Biofeedback group; $F(1, 16) = 6.41$, $p = .02$ (see Table I and Figure 1). The Experimental group had an average increase in IQ of 9 points greater than the Waiting

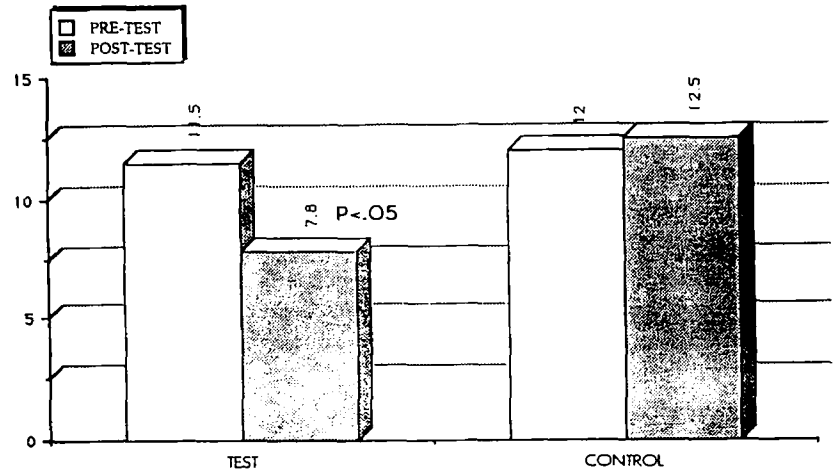

Fig. 2 . EEG biofeedback effects on K-bit IQ composite scores.

List Control Group. The Inattentive behaviors were significantly reduced at posttreatment for the EEG Biofeedback group: $F(1, 16) = 5.27$, $p = .04$ (see Table I and Figure 2). However, the two groups were not significantly different on Aggressive/Defiant behaviors at posttreatment. Although the two groups did not statistically differ on hyperactive behaviors at posttreatment, the EEG Biofeedback group's hyperactive behaviors decreased below the cutoff score that is typically used for a positive finding of hyperactivity, documenting the clinical significance of this improvement.

To evaluate if our sample size was adequate to detect significant differences, a power analysis was calculated for the four dependent measures according to the method described by Kirk (1968). For our data, at the .05 alpha level and a difference of 0.5 standard deviation, the power coefficients were as follows: IQ > .99, Hyperactivity = .68, Inattention > .99, and Aggressive/Defiant > .99. All but Hyperactivity exceeded the de facto standard for adequate power, .80.

DISCUSSION

The results of this study are consistent with previous research conducted by Lubar and associates (1976, 1984, 1985, 1991) and Tansey (1983, 1984, 1985, 1990). The present study included a larger sample size with adequate power to detect significant differences in IQ and most of our behavioral ratings. With respect to previous research in this area, the current study also utilized improved methodology: (1) a control group which

received no treatment of any kind, (2) the same type and amount of EEG biofeedback for all treated subjects (beta enhancement & theta suppression), and (3) similar dependent measures used in previous research for comparison purposes.

The significant improvements in intellectual functioning as measured by increases in K-BIT IQ scores may be the result of the EEG biofeedback treatment group having an increased ability to attend and concentrate, and therefore perform better on the tasks that compose the K-BIT. However, the increase in attention may also have been influenced by rewarding these behaviors over 40 hours of focused concentration irrespective of the neurofeedback. Attentional training through behavioral methods cannot be ruled out based on the current design.

The promising results of this treatment study of EEG biofeedback on cognitive and behavioral measures for ADD/ADHD children need to be replicated. The results may have been different or more significant if the selection criteria for subjects included abnormalities in the EEG, that is, children possessing the high theta and low beta EEG brainwave patterns. The subjects in this study were not selected for inclusion based on their EEG patterns because reliable normative data were not available, and independent traditional assessment techniques were suggested to be more reliable.

Unfortunately, we were not able to analyze the EEG data because of equipment restrictions, which produced unreliable data at times. It is not known whether EEG changes would have been present in all cases since we do not have all of the recorded data to support our impressions. We had the strong sense while training the subjects that most of them had learned to enhance beta amplitudes and decrease theta amplitudes. In addition, from visual inspection it is estimated that most of the subjects' theta thresholds decreased and beta thresholds increased during the 40 training sessions. However, in the absence of EEG data, it is not possible to fully conclude that the EEG biofeedback training was the responsible element in the changes observed. Although the significant increases in IQ and improvements in behavior ratings of our study are consistent with previously reported EEG biofeedback training studies, where learning of the intended EEG changes was demonstrated, these results may have occurred for a number of reasons not related to the biofeedback training itself. Future studies should use consistent EEG biofeedback equipment hardware and software versions in order to be able to analyze the EEG data and compare beta and theta amplitudes, ratios, and thresholds.

As previously stated, the children in the current study did not receive any other treatment, including medication, during their participation in the study. The effects of a multimodality treatment including EEG biofeedback

as part of the protocol may have been of greater benefit than the current study demonstrated. Moreover, the K-BIT test may not be as sensitive as the WISC-R or WISC-III or other intelligence, achievement, or neuropsychological tests in measuring attentional changes, and thus the cognitive changes occurring as a result of the EEG biofeedback may not have been adequately measured. Furthermore, the current study used beta EEG training, not SMR training or a combination of both. In comparison, Tansey (1983, 1984, 1985, 1990) reported utilizing mainly SMR training, while Lubar and associates (1976, 1984, 1985) have used both beta and SMR training, especially with ADHD children. Our results may have been different if SMR training had been used in addition to beta training. Finally, the subjects in this study received 40 sessions of EEG biofeedback, whereas most children in previous research and clinical treatment received a range of between 40 and 70 sessions. Certain children in the current study may not have received enough training to acquire the necessary benefits of EEG training. Individualizing EEG protocols based on each child's ADD symptoms and brainwave activity may be important in future studies and particularly in clinical settings.

The question of reactivity of the measures in reference to this study's results has to be considered. Although the parent behavioral ratings were not completed blind to treatment group, the parents were not given information about their previous rating scores, and, according to their reports, memory deficits for pretreatment ratings usually occurred. Nonetheless, these ratings may have been affected by the parents' expectations for improvement.

Future research designs to evaluate EEG biofeedback treatment should consider a double-blind design including a placebo EEG biofeedback treatment. The placebo treatment should utilize similar instrumentation, instructions, and interpersonal contact with therapists. This was our initial research plan, but the difficulties in keeping the treatment group concealed from all parties involved, and obtaining enough subjects whose parents were willing to participate for six months to one year with the chance that their children may be receiving a placebo treatment, made it unrealistic to implement in our private practice setting. In fact, even the offer of complimentary treatment and complete diagnostic evaluation was insufficient incentive to obtain more than a few subjects. Moreover, there has been some debate whether a placebo EEG biofeedback treatment for a six-month period would be ethical.

Future research using a four-group design including not only a nontreatment control group and an EEG biofeedback group, but also two alternate treatment groups (one receiving medication and one receiving a similar behavioral treatment, such as social attention, attention training, or

biofeedback-assisted relaxation training) would be useful in evaluating the independent contribution of these separate treatment components in cognitive and behavioral improvements for ADD. Combinations of these and other treatments, such as behavior modification, social skills training, and psychotherapy could lead to additional information about the best possible multimodal treatment combination. Further research utilizing multitreatment group designs, larger sample sizes, improved EEG data analyses, and perhaps a double-blind placebo group would be beneficial in order to validate and replicate the findings of this study.

In conclusion, the findings of this initial controlled study of EEG biofeedback effects on ADD/ADHD and LD children are encouraging. EEG biofeedback treatment, if performed in private practice settings, should be done in conjunction with other modalities of treatment in order to comprehensively impact the cognitive, behavioral, emotional, social, and environmental aspects of ADD/ADHD and LD. In summary, the application of EEG biofeedback may prove to be an essential treatment component for specific diagnostic groups comprised within the spectrum of disruptive behavior disorders (e.g., ADD, ADHD, ODD) and learning disabilities.

REFERENCES

American Psychiatric Association (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: Author.

American Psychiatric Association (1987). *Diagnostic and statistical manual of mental disorders* (3rd ed. rev.). Washington, DC: Author.

Atkins, M., & Milich, R. (1987). IOWA-Conners Teacher Rating Scale. In M. Hersen & A. Bellack (Eds.), *Dictionary of behavioral assessment techniques* (pp. 273-275). New York: Pergamon.

Barkley, R. A. (1990). *Attention deficit hyperactivity disorder: A handbook for diagnosis and treatment*. New York: Guilford.

Kaufman, A., & Kaufman, N. (1990). *K.BIT: Kaufman Brief Intelligence Manual.* Circle Pines, MN: American Guidance Service.

Kirk, R. E. (1968). *Experimental design: Procedures for the behavioral sciences.* Belmont, CA: Brooks/Cole.

Lambert, N., Hartsough, C., Sassone, D., & Sandoval, J. (1987). Persistence of hyperactivity symptoms from childhood to adulthood. *American Journal of Orthopsychiatry, 57*, 22-32.

Linden, M. (1988). *An auditory event related potential evaluation of subgroups of hyperactive children to assess underlying arousal level.* Unpublished doctoral dissertation, California School of Professional Psychology, San Diego.

Linden, M. (1991). Event related potentials of subgroups of attention deficit disorder children and implications for EEG biofeedback. *California Biofeedback, 7*, 7-12.

Lubar, J. (1991). Discourse on the development of EEG diagnostics and biofeedback for attention-deficit/hyperactivity disorders. *Biofeedback and Self-Regulation, 16*, 201-225.

Lubar, J. (1992). Address delivered to CHADD, Chicago, IL, October 14, 1992.

Lubar, J., Bianchini, K., Calhoun, W., Lambert, E., Brody, Z., & Shabsin, H. (1985). Spectral analysis of EEG differences between children with and without learning disabilities. *Journal of Learning Disabilities, 18*, 403-408.

Lubar, J., & Lubar, J. (1984). Electroencephalographic biofeedback of SMR and beta for treatment of attention deficit disorders in a clinical setting. *Biofeedback and Self-Regulation, 9*, 1-23.

Lubar, J., & Shouse, M. (1976). EEG and behavioral changes in a hyperactive child concurrent with training of the sensorimotor rhythm (SMR). A preliminary report. *Biofeedback and Self-Regulation, 1*, 293-306.

Milich, R., & Fitzgerald, G. (1985). A validation of inattention/overactivity and aggressive ratings with classroom observations. *Journal of Consulting and Clinical Psychology, 53*, 139-140.

Milich, R., Loney, J., & Landau, S. (1982). Independent dimensions of hyperactivity and aggression: A validation with playroom observation data. *Journal of Abnormal Psychology, 91*, 183-198.

Muehl, S., Knott, J., & Benton, A. (1965). EEG abnormality and psychological test performanced in reading disability. *Cortex, 1*, 434-439.

Nall, A. (1973). Alpha training and the hyperactive child: Is it effective? *Academic Therapy, 9*, 5-19.

Satterfield, J. (1973). EEG issues in children with minimal brain dysfunction. *Seminars in Psychiatry, 5*, 35-46.

Satterfield, J., & Braley, B. (1977). Evoked potentials and brain maturation in hyperactive and normal children. *EEG and Clinical Neurophysiology, 43*, 43-51.

Swanson, J., Nolan, W., & Pelham, W. (1981). The SNAP rating scale. *Resources in Education.*

Tansey, M. (1984). EEG sensorimotor rhythm biofeedback training: Some effects on the neurologic precursors of learning disabilities. *International Journal of Psychophysiology, 1*, 163-177.

Tansey, M. (1985). Brainwave signatures—An index reflective of the brain's functional neuroanatomy: Further findings on the effects of EEG SMR biofeedback training on the neurologic precursors of learning disabilities. *International Journal of Psychophysiology, 3*, 85-89.

Tansey, M. (1990). Righting the rhythms of reason: EEG biofeedback training as a therapeutic modality in a clinical office setting. *Medical Psychotherapy, 3*, 57-68.

Tansey, M. (1991). Wechsler (WISC-R) changes following treatment of learning disabilities via EEG biofeedback training in a private practice setting. *Australian Journal of Psychology, 43*, 147-153.

Tansey, M., & Bruner, R. (1983). EMG and EEG biofeedback training in the treatment of a 10-year old hyperactive boy with a developmental reading disorder. *Biofeedback and Self-Regulation, 8*, 25-37.

Winkler, A., Dixon, J., & Parker, J. (1970). Brain function in problem children and controls: Psychometric, neurological, electroencephalogic comparisons. *American Journal of Psychiatry, 127*, 94-105.

Zametkin, A., Nordal, T., Gross, M., King, A., Semple, W., Rumsey, J., Hamburger, S., & Cohen, R. (1990). Cerebral glucose in adults with hyperactivity of childhood onset. *New England Journal of Medicine, 323*, 1361-1366.