A Comparison of EEG Biofeedback and Psychostimulants

in

Treating Attention Deficit/Hyperactivity Disorders

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The study compared treatment programs with EEG biofeedback or stimulants as their primary components. An EEG group (EEG) was matched with a stimulant group (MED) by age, IQ, gender and diagnosis. The Test of Variables of Attention (TOVA) was administered pre and post treatment. EEG and MED groups improved ($p < .05$) on measures of inattention, impulsivity, information processing, and variability, but did not differ ($p > 0.3$) on TOVA change scores. The EEG biofeedback program is an effective alternative to stimulants and may be the treatment of choice when medication is ineffective, has side effects, or compliance is a problem.

The purpose of the study was to examine the efficacy of 20 sessions of EEG biofeedback in reducing AD/HD symptoms and to compare the results with those obtained with psychostimulant medication. Psychostimulants are the most widely used treatment for AD/HD (Barkley, 1990). In order to be a widely accepted alternative to medication, EEG biofeedback must be able to produce equivalent symptom reduction.

Reports documenting the use of EEG biofeedback in the treatment of attention deficit hyperactivity disorder (AD/HD) began to appear in the literature in the mid 1970's (Lubar & Shouse, 1976). In recent years the use of this treatment has become more widespread and has received increasing attention from the professional community and the public. The increased professional interest may be due to a number of factors including the reported effectiveness of the treatment, the availability of relatively inexpensive, high quality, quantitative EEG equipment, an expanding number of opportunities for training in the use of EEG biofeedback, and the emergence of scientific interest groups that have facilitated the promulgation of information in this area.

With increasing exposure, EEG biofeedback has been subject to greater scrutiny from the biofeedback community as well as professions dealing with the diagnosis and treatment of AD/HD. Barkley (1992, p. 10) concluded that "there is not enough evidence from well controlled scientific studies at this time to support the effectiveness of EEG biofeedback for AD/HD children." He criticized studies that used small numbers of subjects, lacked appropriate control groups, used diagnostic criteria that were unspecified or ambiguous, confounded treatment effects by using multiple interventions (e.g., academic tutoring, self control training, etc.), and employed outcome measures susceptible to practice and/or placebo effects. Some of Barkley's criticisms are valid (Lubar, 1993) and are being addressed by controlled studies using larger numbers of subjects.

Linden, Habib, & Radcjevic (in press), using a waiting list control, demonstrated that 40 sessions of EEG biofeedback resulted in significant increases in IQ and reductions in parental reports of inattentiveness for the experimental, but not the control group. Cartozzo, Jacobs and Gevirtz (1995) found that 30 sessions of EEG biofeedback led to a significant reduction in theta (4-7 hz)

amplitude, increased attention span on the Test of Variables of Attention (TOVA), and improved scores on the Freedom from Distractibility (FD) factor from the Wechsler Intelligence Scale for Children-Revised (WISC-R). A pseudo-treatment control group showed a significant increase in theta amplitude and no improvement on the TOVA or the WISC-R Freedom from Distractibility factor. Scheinbaum, Zecker, Newton and Rosenfeld (1995) compared an EEG biofeedback group to a "cognitive control therapy" group. Only the EEG biofeedback group showed significant improvement on the TOVA at post-treatment testing. More controlled experimental studies are necessary to demonstrate that EEG biofeedback has an independent effect in reducing the symptoms of AD/HD. However, clinical outcome studies that compare EEG biofeedback with other forms of treatment, particularly psychostimulants, are also needed to establish the relative effectiveness of EEG biofeedback as a treatment for AD/HD.

Treatment of AD/HD has traditionally involved use of psychostimulants and/or behavioral interventions. Among the psychostimulants, methylphenidate, dextroamphetimine, and pemoline are the most commonly used medications, respectively. Between 70-80% of children with AD/HD appear to respond favorably to psychostimulants as compared to over 35% that improve with placebos (Barkley, 1990). The primary areas of improvement include attention span, impulse control and reduced motor activity. However, psychostimulants are not without their drawbacks. "To date research studies have not found any single treatment which provides for any long-lasting improvement in ADHD children, particularly once treatment is terminated, and that generalizes to other situations where the treatment wasn't given" (Barkley, 1992, p. 8). This is perhaps the most serious shortcoming of psychostimulants in treating AD/HD. The benefits are temporary unless the patient is willing to take the medication indefinitely (Barkley, 1990). In addition, side effects including decreased appetite, insomnia, anxiety, irritability, stomach aches and headaches occur in 20-50% of children treated with psychostimulants (S. Goldstein & M. Goldstein, 1990). In most cases, the side effects are mild and short term (Barkley, 1990). A potentially more serious, but infrequent, side effect involves the possible development or increase in tics produced by psychostimulants (Denckla, Bemporad, & MacKay, 1976).

Noncompliance with taking medication is a major factor limiting the effectiveness of psychostimulant medication. Long term compliance rates are typically poor and may be especially problematic among families of low socioeconomic status (Barkley, 1990). Many adolescents actively resist taking psychostimulants whether the medication has been helpful to them in the past or not. This might not be a serious problem if AD/HD children outgrew the disorder when they reached puberty as was previously believed. However, it is now estimated that only 30-40% of children with AD/HD have no residual symptoms of the disorder by their late adolescent or early adult years (Weiss & Hechtman, 1993). The remaining 60-70% continue to experience significant AD/HD symptoms that impair their emotional, social, academic and/or vocational functioning. The unwillingness of many adolescents to continue treatment with psychostimulants severely limits their treatment options.

Decisions regarding various treatments for AD/HD, including EEG biofeedback, are usually made in the context of limited health care resources. An informed decision requires information regarding the efficacy of EEG biofeedback compared to medication and other forms of treatment, the expected duration and cost of EEG biofeedback, how quickly response to

treatment can be assessed, and what long term outcome(s) can be expected with the competing forms of treatment. The present study may help address some of these issues.

The present study uses a clinical trials methodology to compare the efficacy of two treatment programs which have EEG biofeedback and psychostimulants as their primary components. Kazdin (1986) views analogue studies and clinical trials as bein- at the opposite ends of a continuum of research methodologies in assessing treatment. Analogue studies refer to investigations of treatment procedures in the context of highly controlled laboratory conditions that only approximate the clinical situation. Analogue research is best suited to investigate specific aspects of treatment, the mechanisms responsible for change, factors that influence treatment efficacy and similar issues requiring precise experimental control. Clinical trials are the most appropriate method for examining the effectiveness of alternative treatments under clinical conditions. Clinical trials utilize patients who have come to a clinic seeking services as opposed to college students or recruited volunteers. Because the research is conducted in a clinic setting, some compromises in research methodology and experimental controls often have to be made for practical and ethical reasons. Treatment is tailored to the individual and is determined on the basis of the patient's problems. Furthermore, it is the patient, rather than the clinician, who is ultimately responsible for choosing the treatment. In essence, a clinical trial provides treatment under many of the conditions where it would be applied in clinical practice. Thus, the results have the potential for broad applicability.

Since patients were drawn from a clinic population rather than being randomly assigned to treatment groups, they were matched on relevant demographic and treatment variables. Treatment(s) provided to each patient were based on the needs of the patient and were not limited to EEG biofeedback or medication. A multimodal approach to the treatment of AD/HD is generally considered preferable to reliance on any single intervention (Barkley, 1991; S. Goldstein & M. Goldstein, 1991; Lubar, 1995). Longitudinal studies of AD/HD suggest that the best long term outcomes are obtained with multiple interventions which change over time, but are based on the current needs of the patient (Weiss & Hechtman, 1993). By designing individualized treatment programs for both the EEG and MED patients, the results obtained are more likely representative of the outcomes that can be expected in clinical practice.

The Test of Variables of Attention (TOVA) was chosen as the instrument with which to compare matched groups of EEG biofeedback and medication treated patients because it is sensitive to the effects of both psychostimulants (Crosby, Corman, & Greenberg, 1992) and EEG biofeedback (S. F. Othmer & S.Othmer, 1992). The TOVA has the further advantage that, being computer administered and scored, it provides objective data that is relatively free of human bias. The same cannot necessarily be said of patient, parent, or teacher reports of behavioral changes whether obtained via interview or standardized questionnaires.

The purpose of the study was to: (1) demonstrate that 20 sessions of an EEG biofeedback program significantly reduce the cognitive and behavioral symptoms of AD/HD; (2) compare the results obtained with the EEG biofeedback program to those obtained with the psychostimulant medication program.

Method

Participants

The participants were 46 patients seen at two outpatient mental health clinics on a fee for service basis. They were referred by their parent(s), physician, school, or were self referred. The patients were evaluated by the first author and received a primary DSM-III-R (American Psychiatric Association, 1987) diagnosis of Attention Deficit Hyperactivity Disorder (ADHD) or Undifferentiated Attention Deficit Disorder (UADD). They included patients between 8 and 21 years of age, with IQs between 80 and 120, who were administered the Test of Variables of Attention (TOVA) pre and post treatment. Two treatment groups of 23 patients each were formed. The first group included all patients who received EEG biofeedback (EEG) as part of their treatment. The second group included patients who were treated with psychostimulants and did not receive EEG biofeedback (MED). The MED group was drawn from a larger pool of patients (N = 39) ages five to 45 and matched with the EEG group by age. Baseline evaluations were completed for both groups of patients before decisions regarding treatment(s) were made. The options of EEG biofeedback and/or a trial on psychostimulant medication were discussed with all patients regardless of their history of prior treatment with medication and/or expressed desire to receive EEG biofeedback. In some cases the choice of treatment was dictated by the availability of insurance coverage for EEG biofeedback and/or whether the patient's schedule could accommodate the three treatment sessions per week considered optimal. Among the EEG patients with a history of treatment with psychostimulants, failure to respond to medication, limited symptom reduction, unacceptable side effects, or an unwillingness to continue use of medication were cited as reasons for seeking an alternative to medication.

Instruments

Intelligence data were obtained using the Kaufman Brief Intelligence Test (KBIT) or the age appropriate Wechsler Scale (WISC-R or III, WAIS-R). In some cases, results obtained during a school evaluation during the previous year were used. The IQ data were needed because intelligence is a factor in interpreting the TOVA performance of children and younger adolescents (Greenberg and Dupuy, 1993).

The TOVA is a 22.5 minute fixed interval visual continuous performance test (CPT) that is not language based and requires no left-right discrimination. One of two easily discriminated visual stimuli is presented for 100 ms every two seconds. The TOVA was standardized on over 1500 individuals ranging from 4 to 90 years of age and provides separate norms for males and females. The TOVA yields four outcome measures used in assessing AD/HD: errors of omission, errors of commission, average response time for the correct responses, and the standard deviation of the response time for correct responses. These four variables are interpreted as measures of inattentiveness, impulsivity or failure to inhibit response, speed of information processing and variability in attention, respectively. Two additional variables, anticipatory responses and excessive commission errors are used to determine if the TOVA results are valid.

The TOVA pattern consistent with AD/HD changes from childhood through the adolescent years. For example, excessive omission errors are a sensitive measure for younger children, but it is unusual to find deviant omission errors in adolescents and adults. In contrast, commission

errors are often the only deviant finding for adults with AD/HD. These developmental trends necessitated that subjects be matched by TOVA normative age group. TOVA norms are in two year increments from ages four through nineteen and in ten year intervals for ages twenty and beyond. The TOVA has been shown to differentiate between ADHD, UADD, Conduct Disorder and normals (Waldman & Greenberg, 1992), be unaffected by the presence of a comorbid reading disorder (Dupuy & Greenberg, 1993), be sensitive to different dosage levels of psychostimulant medication (Crosby, Corman & Greenberg 1992) and to the effects of EEG biofeedback (S. S. Othmer & S. Othmer, 1992). The test is computer administered and scored, which reduces the likelihood of human bias with respect to both the testing and outcome data. The TOVA thus avoids some of the potential difficulties inherent in relying on parent, teacher and patient reports as the primary basis for both diagnosing AD/HD and assessing treatment effects.

The Behavior Assessment System for Children (BASC) is used to evaluate children and adolescents from four to eighteen years of age. It provides teacher, parent, and self reports plus direct classroom observations and a structured developmental history. The parent and teacher questionnaires are parallel forms and permit direct comparisons on a number of scales including anxiety, aggression, attention problems, atypicality, conduct problems, depression, hyperactivity, social skills, somatization, and withdrawal.

Although a combination of the BASC instruments were used clinically, only the 6 to 11 (138 items) and 12 to 18 (126 items) year old Parent Rating Scales completed by mothers were included in the study. They were available for the largest number of EEG subjects both pre and posttreatment. The parent rates items on a four-point scale indicating whether it never, sometimes, often, or almost always occurs. In addition to the clinical scales noted above, the BASC also utilizes three validity scales. The BASC provides separate scales for measuring hyperactivity and impulsivity (Hyperactivity) and inattentiveness and distractibility (Attention Problems). This is an advantage in the differential diagnosis of ADHD and UADD. Several broader composite scales were also used in the study. The Externalizing Problems composite is characterized by disruptive behavior problems such as aggression, hyperactivity, and delinquency. The Internalizing Problems composite includes scales that measure depression, anxiety, somatization, and similar problems that are not marked by acting out behavior. The Behavior Symptoms Index provides a global measure of psychopathology derived from the other clinical scales. Over 50% of children and adolescents with AD/HD are comorbid for other disorders. The rate of comorbidity is in the range of 30-50% for Conduct Disorder, 3560% for Oppositional Defiant Disorder, 2030% for Anxiety Disorders, 30% for Mood Disorders, and 20-25% for learning disabilities (Weiss & Hechtmann, 1993). It was expected that changes would occur on the broader measures of psychopathology as well as the scales directly related to AD/HD (S. Othmer, S. F. Othmer & Marks, 1991).

Evaluation

The baseline evaluation for the EEG and MED subjects included the TOVA and intelligence testing if current IQ data were not available from another source. The BASC was administered for 14 of the EEG group. The remaining 10 members of the EEG group were evaluated using the Personality Inventory for Children or the MMPI-2 with patients over 18 years of age.

A number of subjects in both the EEG (n = 5) and the Med (n = 4) groups were being treated with psychostimulants at the time of the baseline evaluation. With the exception of two EEG patients being treated with pemoline, all of the patients were taking methylphenidate or dextroamphetimine. Medication was discontinued two days prior to baseline testing. This was considered sufficient to produce results not contaminated by medication effects. Methylphenidate and dextroamphetimine have half-lives and produce behavioral effects for 12 hours or less (Barkley, 1990). Pemoline has a more variable half-life and may be effective for as long as 12-18 hours (Wender, 1987). Personality and behavioral assessment was completed at the same time as the TOVA testing. After baseline testing, medication was reinstated for the five EEG patients being treated with psychostimulants and continued at maintenance levels through the 20 EEG biofeedback sessions.

Posttreatment administration of the TOVA for the EEG group was carried out after 20 EEG biofeedback sessions had been completed. This occurred from four to seven weeks after biofeedback began. Among the EEG group, five of 23 patients were still being treated with psychostimulants. For those patients, medication was discontinued two days before post treatment TOVA's were administered.

The MED group was retested while medicated from one to five weeks after starting medication. The TOVA was re-administered 90 minutes after taking the short acting form of methylphenidate or dextroamphetimine or 2.5 hours after taking the long acting forms of the medications. At that point, the medications are at peak effectiveness (Greenberg & Dupuy, 1993).

Treatment

Both authors provided EEG biofeedback. EEG treatment protocols varied and depended on the age, presenting symptoms, and the baseline test results obtained from each patient. EEG protocols were sometimes changed during the course of treatment as targets for intervention changed, e.g., from improving attention span to reducing impulsivity. The protocols used were patterned after those ofJ. 0. Lubar and J. F. Lubar (1984) and S. F. Othmer and S. Othmer (1992). The Lubar protocols emphasize suppressing activity in the theta range (4-8 Hz) with children and adolescents through the age of fourteen, increasing beta (16-20Hz) or sensorimotor rhythm (SMR) output (12-15 Hz) with adults twenty and older, and a combination of theta suppression and beta or SMR enhancement in the fourteen to twenty age range. The goal of the Othmer protocols is to enhance beta (1518Hz) or SMR (12-15Hz) production for all ages. Suppression of theta (4-7Hz) and high beta (22-3014z) is of secondary importance.

NRS-24, NRS-1620, and NRS-2A digitizing EEG systems (Lexicor Medical Technology, Boulder, CO) were used to provide EEG biofeedback. These instruments differ primarily in the number of EEG channels available. They utilize data acquisition and patient feedback software (BioLex Version 2.0 or 2.2) that is functionally identical. EEG data were acquired using two bipolar electrodes, a forehead ground, and linked ear reference electrodes with the Lubar protocols. The Othmer protocols employed a single referential electrode, a reference electrode on the left ear, and a ground electrode on the right ean The active electrode (s) was placed at Cz (Othmer protocols) or midway between Cz and Fz and midway between Cz and Pz (Lubar protocols) using the 10-20 International System. Skin preparation was conducted according to

recommendations by the equipment manufacturer. Skin impedance during training sessions was less than 5K ohms (Lubar protocols) or less than IOK ohms (Othmer protocols).

EEG patients were seen three to five times a week for 45-50 minute treatment sessions that included 30 minutes of EEG biofeedback. Biofeedback sessions consisted of three 10 or two 15 minute segments. At least 10 minutes of the training time was spent in active focusing. That is, the patient was seated in front of a computer monitor with eyes open receiving both visual and auditory feedback. The patient was instructed to increase output in the beta or SMR band while inhibiting theta activity. No other activity was being carried on at the same time. Some patients engaged in reading or another cognitive challenge during part of the 30 minute biofeedback session. EEG biofeedback continued through 20 sessions over a period of four to seven weeks.

Patients were re-evaluated using the TOVA in conjunction with parent and teacher questionnaires to determine if there was a positive response to treatment. This determination was based primarily on the TOVA results where a change of 7.5 points (M = 100, SD = 15) in either direction is considered clinically significant (Greenberg & Dupuy, 1993). When there was evidence of improvement at the re-evaluation, it was recommended that EEG biofeedback be continued, usually for an additional 20 sessions. This wasto allow the patient to make additional progress and/or to provide the opportunity to "over learn" the skills involved and increase the likelihood that they would persist over time. Otherwise the EEG biofeedback component of the treatment program was discontinued and alternatives considered.

Patients in the MED group were started or restarted on methylphenidate (n=16) or dextroamphetimine (n=7) prescribed by their personal physicians following the baseline evaluation. After the patient had been on medication for a minimum of three days with no sionificant side effects, the TOVA was readministered. The response to medication was determined by re-testing the patient 90 minutes after taking the medication and comparing the results with the pre-treatment TOVA. When the response to the initial dose of medication did not appear to be optimal, patients were reevaluated using 2.5 mg or 5.0 mg increases or decreases in medication to determine the most effective maintenance dose.

Treatment was not limited to EEG biofeedback for the EEG patients or psychostimulants for the MED patients. Additional interventions were provided based on the needs of the individual patient. Ancillary treatments included school behavior modification programs aimed at improving the quality and consistency of behavior and/or schoolwork. Teachers completed behavioral and academic rating forms which were sent home daily or weekly and linked to, from four to six privileges dispensed by the parents. If patients were experiencing behavior problems at home, the parents were seen as needed to develop effective behavior management strategies. These included the use of Time Out, Corrective Practice, and other behavior modification techniques. During the time period that the study was conducted, no patients were involved in individual psychotherapy or family therapy. No academic tutoring programs or special education placements were implemented or terminated.

Results

EEG and MED patients were initially matched by TOVA age group. Analysis of relevant pretreatment demographic and treatment variables indicates that age matching produced

treatment groups that were equivalent in most respects (Table 1). They did not differ in age (t = 0.19, *df = 44, p = .85*), gender distribution (X2 = 1.23, *df= 1,* P = .26), intelligence (t = 0.06, *df= 44, p = .95*), frequency of ADHD vs UADD as the primary diagnosis (X2 = 0. 11, *df = 1, p =* .74), frequency of secondary/ tertiary diagnoses (X2 = 1. *04, df= 44, p = .3* 1), or frequency of Learning Disability and/or Emotionally Disturbed placements (X2 = 1.*04 , df = 1, p =* .31). The EEG and MED groups were not significantly different on baseline TOVA measures of attentiveness *(t = 1.02, df= 44,* P = .31), impulsivity (t = .28, *df = 44, p = .78*), processing speed (t = .03, *df = 44, p = .97*) or variability in attention(t = .60, *df = 44,* p = .55). However, more of the EEG (n = 17) than MED (n = 10) patients had previously been treated with psy~ chostimulents (X2 = 4.39, *df = 1, p =.04*).

The EEG (2/23) and MED (4/23) groups did not differ in the frequency of parents receiving behavior management training (X2 = *0.77, df* = 1, p = .38). However, patients in the MED group (13/23) were more likely than those in the EEG group (5/23) to be involved in a school behavior modification program (x2 = 5.84, *df = 1,* P = .02) during treatment. The relatively low frequency of school behavior modification for the EEG group is due to the fact that many of the EEG patients were treated during the summer months when school was not in session.

Table 1

EEG and Medication Group Demographic Variables

Variable	EEG	MED
Age (years)		
M	12.9	12.7
SID	2.9	3.2
Gender (n)		
Male	17	20
Female	6	3
Intelligence		
M	102.4	102.6
SID	9.9	9.4
Primary Diagnosis (n)		
ADHD	17	16
UADD	6	7

Secondary Diagnosis (n)	14	12
Treatment History (n)		
Special Education	8	7
Psychostimulents	17	10

Note. n = 23 for EEG and MED groups

The first purpose of the study was to demonstrate improvement in TOVA outcome variables following *20* sessions of the EEG program. Means and standard deviations for the pre and posttreatment TOVA variables are presented in Table 2. The TOVA data for the EEG group were analyzed using a one tailed t-test for dependent measures. It was predicted a priori that all four TOVA variables would demonstrate significant improvement following treatment. These predictions were confirmed. The EEG group showed increased attentiveness (t = 3.*01, df = 22, p = .003),* reduced impulsivity (t = 2.*47, df = 22, p = .01),* increased processing speed (t = 1.*85, df = 22, p*= .04), and decreased variability in attention (t *=4.67,df=22,p=.0001).* It was also predicted a priori that the EEG group would show significant behavioral changes on five BASC scales (Table 3). The prediction was confirmed. A one tailed t-test for dependent measures indicated significant reductions on the Hyperactivity (t = 2.84, *df = 13, p = .007),* Attention Problems (t = 2.*81, df = 13, p = .007),* Externalizing Problems (t = 4,21, *df = 13, p* = .0005), Internalizing Problems (t = 5.01, *df* = 13, p = .0001), and Behavior Symptoms Index *(t = 4.4 1, df= 13, p = .0004)* scales.

The second purpose of the study was to compare the effectiveness of the EEG biofeedback program with that of a medication pro gram in reducing the symptoms of AD/HD. It was predicted a priori that both treatment programs would result in significant improvement on TOVA outcome measures. This hypothesis was confirmed. The MED group (Table 2) showed improved attention (t = 2.*50, df = 22,* P .01), reduced impulsivity (t = 3.*79, df = 22,* P .0005), improved processing speed (t = 3,72, *df = 22, p = .0006*), and reduced variability in attention (t = 4.*08, df = 22, p = .0003).* It was further predicted that there would be no significant differences between the EEG and MED groups in the degree of improvement shown. The results confirmed this hypothesis (two tailed t-test for independent measures). There were no significant differences between the EEG and MED groups on change scores (posttest score minus pretest score) for errors of omission (t = 0.*93, df = 44, p = 0.36),* errors of commission (t = 0. *03, df= 44,* P = 0.98), average response time (t = 0.79, *df = 44, p = 0.43),* standard deviation of response time (t = 0.*39,df = 44, p = 0.70),* or the sum of the change scores on the four TOVA variables (t = 0. 11, *df 44, p = 0.91).*

Table 2

TOVA Results For EEG And Medication Groups

	TOVA EEG		MED	
Variables				
	Mean	SD	Mean	SD
Omission				
Pre	86.96	24.21	93.30	17.39
Post	102.91	5.86	103.13	11.92
Change	15.96	25.42	9.83	18.86
Commission				
Pre	95.43	16.12	94.17	14.15
Post	104.78	13.42	103.65	13.27
Change	9.35	18.13	9.48	12.00
Response Time				
Pre	84.35	18.37	84.52	17.35
Post	89.52	19.27	92.48	14.68
Change	5.17	13.43	7.96	10.27
Variability				
Pre	84.09	16.12	87.26	19.33
Post	97.30	16.90	102.30	15.88
Change	13.22	13.57	15.04	17.70

Note. Test Of Variables of Attention scores are standard scores with M = 100, S D 15.

N = 23 for EEG and MED GROUPS

When the data were analyzed on the basis of the outcomes for individual patients rather than treatment group means, there was no difference between the EEG *(19/23)* and MED *(20/23)* groups in the number of patients who showed significant improvement on the TOVA (X2 = 0. *17, df= 1, p = 0,68).*

Discussion

The study demonstrated that a treatment program with EEG biofeedback as the major component led to significant reduction in both cognitive and behavioral symptoms of AD/HD after *20* treatment sessions completed over a period of four to seven weeks. The EEG group manifested significant improvement in attention, impulse control, speed of information processing and consistency of attention on the TOVA. BASC questionnaires completed by mothers confirmed the reduction in AD/HD symptoms and also indicated a decline in internalizina and externalizing psychopathology. In every case where parents and/or teachers reported significant improvement in behavior or school performance, corresponding improvement in the TOVA performance was observed. This confirms that improvement was not limited to TOVA test scores, but had generalized beyond the clinic and was observed as symptom reduction in the patients' daily lives. More importantly, the EEG biofeedback program led to improvement on all four TOVA outcome variables that was equivalent to that obtained with the medication pro-ram. The EEG program is an effective treatment for AD/HD and a viable alternative to the use of psychostimulant medication. The results indicating significant reduction of AD/HD symptoms with EEG biofeedback are consistent with those reported by Lubar (1991), S. F. Othmer and S. Othmer (1992), Linden, Habib & Radcjevic (in press), Cartozzo, Jacobs & Gevirtz (1995) and Scheinbaum, Zecker, Newton & Rosenfeld (1995). Moreover, the improvement was evident in far fewer than the 40-80 sessions sometimes cited as the expected course of treatment (Barkley, 1992). This allows for conservation of health care resources by identifying patients who are not responding to treatment earlier in the treatment process.

Table 3

EEG Group BASC Data.

BASC	Pre Treatment		Post Treatment	
Scales	Mean	SD	Mean	SD
Hyperactivity	63.43	15.02	54.62	11.00
Attention Problems	71.29	8.65	64.69	11.28
Externalizing Problems	62.71	12.07	55.53	10.10
Internalizing Problems	61.50	13.57	51.23	10.92
Behavior Symptoms Index	67.14	12.20	56.15	10.87

Note. Behavior Assessment System for Children scores are T scores with M 50, S D = 10. n 14.

The EEG biofeedback program is an effective treatment for AD/HD and may be the treatment of choice in cases where medication is ineffective, only partially effective, has unacceptable side effects, or where compliance with taking medication is low. In addition, 60-70% of children with AD/HD continue to have symptoms of the disorder into their adolescent and adult years (Weiss & Hechtman, 1994). Since psychostimulants do not result in any lasting reduction of AD/HD symptoms, their use must be continued indefinitely if the symptoms are to be controlled. By the time many children reach adolescence, they are no longer willing to take psychostimulants whether they had responded favorably in the past or not. For this reason, there is a substantial population of AD/HD adolescents and young adults for whom medication is not an acceptable treatment option. The EEG biofeedback program provides an alternative for this group of patients.

Among patients who have a good response to medication, the choice between EEG biofeedback and medication is not as clear cut. The EEG program is more expensive in the short run than the medication program. However, the cost differential may be declining due to better pretreatment assessment and more efficient treatment protocols . S. Othmer (1994) reports that training is successfully completed in 20 sessions for at least 30% of AD/HD patients. The EEG biofeedback program is a cost effective alternative to the long term use of medication if it results in lasting symptom reduction, particularly if the patient is one of the 60-70% who will not "outgrow" the disorder. One to ten year follow-up of successfully treated patients suggests that EEG biofeedback leads to long term symptom reduction (Othmer, S., Othmer, S. F., & Marks, 1991; Lubar, 1995; Tansey, 1993). These reports are encouraging but need to be confirmed by systematic follow-up studies with larger samples of patients using objective assessment procedures such as the TOVA, standardized academic achievement tests, etc.

EEG biofeedback is not a "cure" for AD/HD. Nevertheless, there is an increasing body of evidence to support Lubar's (1995) conclusion that EEG biofeedback, often delivered in the context of a multimodal treatment program, leads to "normalization" of behavior and can enhance the long-term academic performance, social functioning, and overall life adustment of the AD/HD patient.

References

Barkley, R. A. (1992). Is EEb biofeedback treatment effective for ADHD children? *Ch.A.D.D.er Box, 5-11.*

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

Cartozzo, H. A., Jacobs, D., & Gevirtz, R. N.(1995). EEG biofeedback and the remediation of ADHD symptomatology: A controlled treatment outcome study. *Proceedings of the 26th Annual Meeting of the Association for Applied Psychophysiology and Biofeedback,* USA, 21-25.

Crosby, R., Corman, C., & Greenberg, L. (1992). *The assessment ofmedication effects in attention deficit disorder using the Test 'of Variables of Attention.* Unpublished manuscript

Denckla, M. B., Bemporadj R., & MacKay, M. C. (1976). Tics following methylphenidate administration. *Journal of the American Medical Association,* 235, 1349-1351.

Dupuy, T. R., & Greenberg, L. M. (1993). *TO. VA. manual Test of Variables of Attention computer program version 6.x* (Available from Universal Attention Disorders, 4281 Katella Avenue, #215, Los Alamitos, CA 90720)

Goldstein, S., & Goldstein, M. (1990). *Managing attention disorders in children: A guidefor practitioners.* New York: John Wiley & Sons.

Greenberg, L. M., & Dupuy, T R. (1993). *Interpretation manualfor the Test of Variables of Attention computer program.* (Available from Universal Attention Disorders, 4281 Katella Avenue, #215, Los Alamitos, CA 90720)

Kazdin, A. E. (1986). The evaluation of psychotherapy: Research desio, and methodology. In S. L. Garfield & A. E. Bergin (Eds.), *Handbook of psychotherapy and behavior change* (pp. 23-68). New York: John Wiley.

Linden, M., Habib, T., & Radojevic, V (in press). A controlled study of EEG biofeedback effects on cognitive and behavioral measures with attention-deficit disorder and learning disabled children, *Biofeedback and Sey-ftulation.*

Lubar, J. F (1995). Neurofeedback for the management of attention-deficit/hyperactivity disorder. In Schwartz, M. S. & Associates (Eds.), *Biofeedback: A PractitionersGuide* (2nd ed.), (pp. 493-522). New York: Guilford Press.

Lubar, J. F. (1993). Innovation or inquisition: The struggle for ascent in the court of science: *Neurofeedback and ADBD. Biofeedback,* 21,23-30.

Lubar, J. F. (1991). Discourse on the development of EEG diagnostics and biofeedback for attention-deficit/hyperactivity disorders. *Biofeedback and Se~f-Regulalion, 16,* 202225.

Lubar, J. F., & Shouse, M. N. (1976). EEG and behavioral changes in a hyperkinetic child concurrent with training of the sensorimotor rhythm (SMR): A preliminary report. *Biofeedback and Se4r-Regulation, 3,* 293-306.

Lubar, J. 0. & Lubar, J. F. (1984). Electroencephalographic biofeedback of SMR and beta for treatment of attention deficit disorders in a clinical setting. *Biofeedback and Se~f-Regulation, 9,* 1-25.

Othmer, S. (1994). *A discussion of alphaltheta training and SMR/beta training and their respective roles.* (Available from EEG Spectrum, Inc., 16100 Ventura Blvd, Suite 10, Encino, CA 91436)

Othmer, S., Othmer, S. F., & Marks, C. S. (1991). *EEG biofeedback trainingfor attention deficit disorder, specific learning disabilities, and associated conduct problems,* (Available from EEG Spectrum, Inc., 16100 Ventura Blvd., Suite 10, Encino, CA 91436)

Othmer, S.F.,& Othmer, S. (1992). *Evaluation and remediation of attentional deficits.* (Available from EEG Spectrum, Inc., 16100 Ventura Blvd., Encino, CA 91436)

Scheinbaum, S., Zecker, S., Newton, C. J., & Rosenfeld, P. (1995). A controlled study of EEG biofeedback as a treatment for attendon-deficit disorders. *Proceedings of the 26th Annual Meeting of the Association for Applied Psychophysiology and Biofeedback, U.S.A, 131*134.

Tansey, M. A. (1993). Ten-year stability of EEG biofeedback results for a hyperactive boy who failed fourth grade perceptuallyimpaired class.*Biofeedback and Self-Regulation, 18, 33-44.*

Waldman, 1. D., & Greenberg, L. M. (1992). *Inattention and impulsivity discriminate among disruptive behavior disorders.* Unpublished manuscript.

Weiss, G., & Hechtnian, L. T. *(1993). Hyperactive children grown up: ADHO in children, adolescents, and adults* (2nd edition). New York: Guilford Press.

Wender, P. H. *(1987). The hyperactive child, adolescent, and adult: Attention deficit disorder through the 1ifespan.* New York: Oxford University Press.

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EEG Biofeedback for the Enhancement of Attentional Processing in Normal College Students

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College students diagnosed as free of any neurological or attention deficit disorder received EEG biofeedback to enhance beta (16-22 hertz) activity while simultaneously inhibiting high theta and low alpha (6-10 hertz) activity in order to evaluate improvements in attentional measures. *Following short-term treatment (mean number of sessions=20), subjects were evaluated as either learners or non-learners based upon standard pre- versus post-treatment neurofeedback measures. Attention quotients taken from pre and post-treatment measurements using the Intermediate Visual and Auditory (IVA) Continuous Performance Test identified significant improvements in attentional measures in learners, while non-learners showed no significant improvements. Results suggest that some "normal" young adults can learn to increase EEG activity associated with improved attention. Twenty sessions, however, even for this population may represent the lower limit for achieving significant improvement.*

Over the past two decades, the use of electroencephalographic (EEG) biofeedback has been shown to be beneficial for the enhancement of attentional processes in children and adults with attention-deficit disorder (ADD) and attention-deficit hyperactivity disorder (ADHD). For these two groups the EEG biofeedback procedure has been used to help individuals normalize neurological functioning, thereby enabling them to process information and deal with sensory stimulation more effectively (Lubar, 1985, 1991, 1995a, 1995b; Lubar & Lubar, 1984; Lubar, Swartwood, Swartwood, & O'Donnell, 1995; Mann, Lubar, Zimmerman, Miller, & Muenchen, 1992; Senf, 1988; Tansey, 1984, 1990).

Given the effectiveness of using EEG biofeedback for enhancing performance in individuals with ADHD and ADD, the next research question that needs to be answered is whether this EEG training can be used for the enhancement of attentional processing in normal individuals. The objective of this study was to determine whether the EEG biofeedback techniques effectively used to improve attentional processing for individuals with ADHD and ADD will have similar benefits for normal subjects.

Method

Subjects

Seven subjects were recruited from the undergraduate population at the University of Tennessee for participation in this project. Subjects were required to meet the following inclusion criteria: 1) between the ages of 1845, 2) classified as a freshman or sophomore, 3) cumulative grade point average between 2.0 and 2.'5 (on a 4.0 point scale), 4) free from any diagnosed learning disorder, and 5) no previous history of EEG biofeedback training. All subjects signed a consent form that had been approved by the Committee on Research Participation at the University of Tennessee.

Three of the initial subjects failed to complete participation. The final sample of four included two men and two women.

Procedure

Following an initial evaluation meeting with one of the researchers to determine subject appropriateness based upon inclusion criteria, pre-treatment evaluations were conducted over a one week span. These evaluations included the following: Intermediate Visual and Auditory (IVA) Attention 7bst, Autogen A620 Neurofeedback Assessment, Quantitative Monopolar 19 channel EEG, and the Wechsler Adult Intelligence Scale-Revised (WAIS-R).

The IVA attention test is a computerized, continuous performance test designed for individuals ages eight through adult, combining visual and auditory stimuli using various computer displayed patterns. This evaluation yielded the following scores: full attention quotient, auditory attention quotient, visual attention quotient, mean response time for auditory stimuli, and mean response time for visual stimuli. Subjects were scheduled individually for the administration of this test and given general instructions concerning testing procedures. Because all test instructions and procedures for this test were automated and provided directly by the computer both in spoken and written form, the experimenter initiated the procedure for the subject and left the room.

The Autogen A620 (Stoelting-Autogenics Corp.) neurofeedback assessment was used to determine power spectrum and ratio information within various electroencephalographic (EEG) domains. This assessment was conducted using a dual sensor placement at locations FCZ and CPZ of the International 10-20 system for electrode placement. These locations are 10 percent of the Nasion-Inion distance measured from CZ anteriorly and posteriorly The sites were physically prepared using Omniprep solution to cleanse the surface and provide improved conductance. A quarter-inch high mound of Elefix (Weaver and Company) conductive cream was placed at each site with electrodes pushed through this mound to the scalp surface at their respective locations. The electrodes (Gram Instrument Co.), gold-plated silver with a central hole for the paste to extrude, were held in place using a medium-sized cotton ball. A single earclip electrode, using Electrogel to improve conduction, was placed on the left ear of the subject following a cleansing using Oniniprep solution. Following this preparation procedure, subjects were instructed to fixate on a location approximately 36 inches directly ahead of them for a period of one and one-half minutes during which data were collected.

A quantitative monopolar 19-channel EEG or "Brain Map" was used to provide percentage and power information within the following encephalographic domains: beta, alpha, theta, and delta. Information concerning the relationship between these domains was also obtained. The quantitative EEG was administered following the International 10-20 system for electrode placement and included the central locations. Prior to Electro Cap placement, an earclip electrode was placed on each ear using Electrogel to improve conductance following a preparation using Omniprep solution. Connections were made using an electrode cap (Electro Cap Company) and Electrogel was applied to each sensor using a small blunt hypodermic tube inserted through the sensor in order to improve conductance. Following preparation, data were collected under the following conditions: eyes open baseline, eyes closed baseline, a reading

condition, a drawing condition, while completing the Raven's Progressive Matrices, and a listening condition.

The Wechsler Adult Intelligence ScaleRevised (WAIS-R) was used to obtain intelligence quotient and subscale scores for all subjects. It was hypothesized that through an analysis of subscale scores, specific trends relating to performance on neurofeedback parameters and attentional improvements could be identified.

Following the pre-treatment evaluations, subjects received EEG biofeedback training (mean number of sessions=20) using the Autogen A620 Neurofeedback System to increase beta (16-22 hertz) activity while simultaneously inhibiting high theta and low alpha (6-10 hertz) activity (theta-alpha). Feedback was provided individually using a dual sensor placement (bipolar placement) at location PCZ and CPZ of the International 10-20 system for electrode placement. Following the physical preparation of the scalp using Ornniprep solution a quarter-inch high mound of Elefix (Weaver and Company) conductive cream was placed at each site. Each sensor was placed into its respective mound and held in place using a medium sized cotton ball. A single earclip electrode, using Electrogel to improve conduction, was placed on the left ear following a preparation using Omniprep solution. Measures of impedance remained below 5 ohms throughout training sessions.

All feedback sessions used the following training protocol: baseline-two minutes, feedback-seven minutes, reading plus feedback-seven minutes, feedback-seven minutes, and listening plus feedback-five minutes. During baseline, subjects were asked to sit quietly and focus their attention on a picture placed approximately two feet in front of them at eye level. Subjects received no feedback concerning their performance during baseline. During both feedback only conditions, subjects were provided with both visual and auditory feedback contingent upon the production of beta EEG while simultaneously inhibiting the production of theta-alpha EEG activity. The Autogen A620 criterion for reward was set at 50 samples occurring in 0.5 seconds and the EEG was sampled at 128 samples per second. During the reading condition, subjects read to themselves passages from Fiction 100: An Anthology of Short Stories (fifth edition) or other college level material. During the listening condition, subjects listened to the experimenter read passages similar to those used during the reading condition. For both the reading and listening conditions subjects received only auditory feedback. Following the completion of each session, sensors were removed and all pastes and gels were cleaned.

Following the treatment phase subjects were once again administered the tests utilized during pre-treatment. These tests were once again completed over a period of two weeks. All post-tests were administered at approximately the same time of day as the pre-tests in an attempt to limit any differences among scores due to daily fluctuations in EEG and other effects of circadian rhythm.

Results

Results were calculated using four of the original seven subjects. It was necessary to remove three subjects from the analysis due to non-compliance issues in one or more of the following areas: failure to regularly attend neurofeedback sessions, failure to complete required number of sessions, and failure to complete post-training assessments.

In order to assess the effects of neurotherapy for those subjects who were exposed to all procedures, Sperman RHO Correlations were obtained based upon changes from baseline in the following Autogen A620 parameters: % of EEG thetaalpha (IEEG%), % of EEG beta (REEG%), [tV levels of EEG theta-alpha (IEEG [N), [tV levels of EEG beta (REEG VV), and thetaalpha/beta ratios (I/R). Subject I (SI) obtained the following Sperman RHO Correlations: IEEG%=-0.467, REEG%= -0.078, IEEG [LV=-0.183, REEG [tV=0.037, and I/R=-0.332. For subject 2 (S2) the Sperman RHO Correlations were -0.450, 0.497, -0.344, -0.282, and -0.149 respectively. For subject 3 (S3) the Sperman RHO Correlations were 0.214, -0-233, 0.088, -0.032, and 0.193 respectively For subject 4 (S4) the Sperman RHO Correlations were 0.419, -0.324, 0.429, 0.355, and 0.305 respectively All correlations are summarized per subject in Figure 1.

Among the parameters documented, learning is indicated by negative correlations in % of EEG theta-alpha, theta-alpha uV levels, and theta/beta ratios, and positive correlations in % of EEG beta and beta [tV levels. Based upon these observations, S1 and S2 demonstrated learning in four out of five parameters. S3 demonstrated no learning using these parameters, and S4 demonstrated learning in only one out of the five parameters.

Improvements in attentional processing were directly assessed using the Intermediate Visual and Auditory (IVA) Attention Test. Based upon a standard deviation equal to 15 points as signifying improvement between pre- and post-tests, S1 and S2 achieved Full Attention Quotient (FAQ) deviation scores of 2.2 and 3.73 respectively, while S3 and S4 achieved FAQ scores of -.93 and .13 respectively. In order to provide more specific analysis, the FAQ was divided into two sub-quotients, the Auditory Attention Quotient (AAQ) and the Visual Attention Quotient (VAQ), both of which yielded deviation scores. S1 and S2 achieved AAQ deviation scores of 1.33 and 1.67 and VAQ deviation scores of 2.0 and 1.33, all respectively S3 and S4 achieved AAQ deviation scores of -2.0 and -.13 and VAQ deviation scores of .60 and .26, all respectively

An additional IVA measurement that provides further information related to attentional processes is mean reaction time. This measurement was divided into two portions: mean auditory reaction time (MNA) and mean visual reaction time (MNV). Comparing the difference between pre- and post-test administration, S1 and S2 demonstrated MNA improvements of 74 and 52 milliseconds respectively. For S3 and S4, MNA difference scores were -68 and -33 milliseconds respectively. The difference measurements between pre- and post-tests for MN`V were 75, 68, and 44 milliseconds for S1, S2, and S4 respectively and -23 for S3.

For the purpose of WAIS-R analysis, subjects were classified as either "Learners" or "Non-Learners" based upon the results from the Sperman RHO Correlations for

neurofeedback parameters, improvements in **attentional processing measured by scores obtained from the IVA, and improvements** in mean reaction time from the IVA. Using these results, S1 and S2 were labeled as Learners and S3 and S4 were labeled as Non-Learners.

Results from WAIS-R administration revealed that all four subjects had Full Scale IQ scores in the average or above average range (98-117), and all four subjects manifested significant scatter among subscale scores. None manifested a pattern of scatter that **would suggest** a processing

anomaly of a nature likely to be associated with either a specific learning disability, an Attention Deficit Disorder, or unwillingness to exert effort in response to at least circumscribed task demands. It would appear, then, that all were intellectually capable of succeeding at college level work, but that psycho-social factors reflected in scatter differences between the two groups might be related to their successes or failures as learners.

Differences between Learners'and NonLearners' scatter on WAIS-R subtests were explored. First the subtests on which each subject's observed performance differed from that expected from his or her Full Scale IQ score were identifled. A difference was considered to have been manifested when the subject's performance was three or more scaled scores less or greater than his or her mean scaled score for the eleven subtests. Then subtests on which Learners and Non-Learners consistently differed from each other in terms of expected or unexpected performance could be identified.

There was no consistent difference between Learners and Non-Learners in terms of number of subtests on which observed performances differed from expected performances. There was only one subtest in which differences were associated with learning status: Vocabulary. Here both Learners showed poorer observed than expected performances whereas the observed performance of the Non-Learners

did not differ from those which were expected for them. In fact, the scaled scores (6 and 7) on Vocabulary for both Learners were at levels that suggest impairment.

Initial results from the Autogen A620 neurofeedback assessment and the quantitative monopolar 19-channel EEG revealed no indications of attentional or neurological abnormalities for any of the subjects. Comparing pre- and post-treatment measurements, no distinct pattern of change was observed for either assessment. Distinct patterns of change were also not observed in pre- versus post-treatment WAIS-R scores. Because no distinct patterns were observed, data are not included.

Discussion

Based upon the results of the neurofeedback parameters which demonstrated that S1 and S2 improved in four out of the five measurements, and S3 and S4 improved in zero and one measurement, respectively, subjects were classified as "Learner" or "Non-Learner." Based upon the improvements in attentional performance among Learners, the results suggest that some college students can learn to increase EEG activity assessed by objective measures.

The first possible explanation for why Non-Learners were unable to perform as well as Learners may have to do with the relatively low number (mean=20) of EEG biofeedback sessions all participants received. The number of sessions required to show significant improvements has been consistently shown to be between 30 and 50 (Lubar, 1995a, 1995b; Lubar & Lubar, 1984). The mean of 20 feedback sessions received by these subjects may represent, even for a college population, the lower limit for any non-handicapped student achieving significant improvements.

Scores obtained by Learners and NonLearners on the WAIS-R Vocabulary subtest are suggestive of potential differences existing between these groups that may have led to their ability or inability to perform successfully during neurofeedback training. Ordinarily scaled scores on Vocabulary are regarded as the single best estimate of overall intellectual ability because of their high correlation with Full Scale IQ and the fact that they are relatively robust to interference from functional psychopathologies. When impaired performance is seen specifically on this subtest, it usually reflects a history of social deprivation with regard to spoken language. That is, there has usually been a marked impoverishment in the language used in the individual's familial and personal social environments. It would appear likely then, that the Learners came to college from markedly language impoverished environments. Their underdeveloped vocabularies would be specific handicaps for college achievement commensurate with their general intellectual abilities. The fact that they are in college, however, suggests that they have been ambitious to achieve more academically than their social environments might ordinarily support. The inferred high ambition to learn may account for their accomplishment during neurofeedback in a very circumscribed number of trials.

Based upon this evaluation, further investigations assessing the role of motivation and level of ambition on EEG biofeedback performance seems warranted. The important relationship between psychological factors and learning in a neurofeedback paradigm needs to be investigated because it may lead to the development of predictive measures for success. It may be that there are some individuals with normal intelligence for whom neurofeedback is not an appropriate intervention wherein for others it may be the most effective way to enhance peak academic performance. Finally, based on these preliminary results we strongly suggest that at least 30 sessions of training are necessary, even for "normal" populations, to enhance attention for complex tasks.

References

Lubar, J. F (1985). Changing EEG activity through biofeedback applications for the diagnosis and treatment of learning disabled children. *Theory Into Practice, Ohio State University, 24, 106-111.*

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

Lubar, J. F (1995a). Neurofeedback treatment of attention deficit hyperactivity disorder: Research and clinical implication. *Biobehavioral Self-Regulation in the East and West (pp.* 312-323). 'Ibkyo: Springer-Verlap.

Lubar, J. F (1995b). Neurofeedback for the management of attention deficit-hyperactivity disorders. In M. S. Schwartz (Ed.), *Biofeedback: A practitioner's guide* (2nd ed., pp. 493-522). New York: Guilford Publications, Inc.

Lubar, J. E, Swartwood, M. 0., Swartwood, J. N., & O'Donnell, P. (1995). Evaluation of the effectiveness of EEG neurofeedback training for ADHD in a clinical setting as measured by changes in T.OXA. scores, behavioral ratings, and WICS-R performance. *Biofeedback and SelfRegulation, 20,* 83-99.

Lubar, J. 0., & Lubar, J. F. (1984). Electroencephalographic biofeedback of SMR and beta for treatment of attention deficit disorders in a clinical setting. *Biofeedback and Self-Regulation, 9, 1*23.

Mann, C. A., Lubar, J. F, Zimmerman, A. W, Miller, B. A., & Muenchen, R. A. (1992). Quantitative analysis of EEG in boys with attention deficit/hyperactivity disorder (ADHD). A controlled study with clinical implications. *Pediatric Neurology, 8, 30-36.*

Senf, G. M. (1988, November/December). Neurometric brainmapping in the diagnosis and rehabilitation of cognitive dysfunction. *Cognitive Rehabilitation, 20*37.

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

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

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Neurometrics and ADHD: Stimulant Responsivity and Subtype Analysis

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Abstract: *Previous findings reported by our research team have described specific QEEG characteristics that can contribute to the assessment of patients suspected of ADHD. The current paper summarizes our subsequent investigations of ADHD subtypes. Specifica11y, we sought to determine whether stimulant responders could be differentiated from "non. responders" on the basis of our "Attention Index" and whether patients with an Inattentive type of ADHD could be distinguished from patients with a Combined type on the basis of an examination of the SMR. Our examination of 144 patients diagnosed with ADHD revealed that 93% of stimulant responders showed "cortical slowing" on our QEEG Scan; whereas none of the "non-responders" exhibited such a pattern. A second study, examining 55 patients diagnosed with ADHD, Inattentive Type and 171 diagnosed with ADHD, Combined Type, revealed significantly higher "theta/SMR" power ratios in the Combined group. particularly during a graphomotor task. These findings highlight the potential clinical relevance of QEEG applications both in the selection of pharmacological treatment and in broadening our understanding of ADHD subtypes"*

Introduction

Approximately six years ago, a research team led by Dr. Joel Lubar and I began a series of experiments examining electrophysiological characteristics of patients diagnosed with ADHD. Earlier investigations by Dr. Lubar and his students at the University of Tennessee (Mann et al, 1992) had revealed a pattern of cortical hypo-arousal over frontal regions, which supported the hypothesis that ADHD was a neurodevelopmental disorder with physical characteristics that were identifiable via PET (Zametkin et al, 1990), MRI (Lou et al, 1984; Hynd et al, 1991), and SPECT imaging (Amen, Paldi and Thisted, 1993). While these studies greatly facilitated the recognition that ADHD was a "health impairment" rather than a defect of moral development or motivation, clinicians were still unable to incorporate any of these techniques in the assessment process due to the lack of normative databases for comparative purposes. In order to develop an electro-physiological assessment procedure, additional studies were needed.

The tasks undertaken by our research team included: determination of specific QEEG characteristics that could serve as the basis for a "neurometric", development of a normative

database, and investigation of the reliability and validity of a QEEG "Scan" for ADHD. Based on Mann et al's (1992) findings, we began a systematic examination of power ratios at the vertex. Specifically, we chose to examine the ratio of power recorded at 4 to 8 Hz to that recorded at 13-21 Hz. In addition to a traditional "eyes fixed" baseline recording, we examined patients during tasks commonly performed in school and work settings (reading, listening), as well as on a task requiring attention to visual detail, motor planning and coordination (copying geometric shapes).

EEG recordings were obtained during four, 90-second tasks; power ratios were calculated for each task and then averaged for the four tasks. This statistically derived "Attention Index" was then examined in a series of studies that have been published (Monastra et al, 1999) and presented during the past three annual AAPB conferences, as well as at the national conferences of The American Psychological Association and C.H.A.D.D. The results of our initial six studies, involving approximately 1000 individuals, have indicated that our QEEG "Scan" provides a reliable, valid measure of a frequently reported neurophysiological symptom of ADHD. Despite the success of our research project, as well as a similarly successful series of investigations conducted by New York University's Brain Research Laboratory (Chabot & Serfontein, 1996; Chabot, Merkin, Wood, Davenport and Serfontein, 1996), critics of QEEG approaches have generally minimized the significance of such findings.

While behaviorally oriented researchers (e.g. Barkley, 1998) have begun to recognize the contribution of QEEG in strengthening "the evidence for genetic and developmental neurological factors as likely causal of this disorder while greatly reducing the support for purely social or environmental factors" (Barkley, 1998, p. 177), Barkley (1994) in his commentary on neurometrics, notes that "it will be a formidable task, indeed, to find an instrument, Neurometrics or otherwise, that could exceed the type of predictive accuracy of drug response based on sound clinical diagnosis alone" (p.3). While behavioral rating scales are generally considered useful in order to provide a basis for determining the degree of deviance from developmental norms, the potential contribution of neurological or electro-physiological measures has been repeatedly questioned. As one critical journal reviewer expressed to me, "Using an EEG to determine ADHD is as useless as a blood test to determine obesity". What that reviewer seemed to misunderstand is that similar to a blood test of an obese person, QEEG procedures have the potential to contribute to a better understanding of the causes of a patient's symptoms and to assist us in developing more effective treatment procedures. Without a blood test, an obese patient whose symptoms are caused by "hypo-thyroidism" could waste a lot of time, money and energy on cognitive behavioral therapy, anti-depressants, high protein/ low carbohydrate diets, fat elimination pills and a gym full of exercise equipment. Similarly, without valid assessment procedures to evaluate the underlying physical causes of ADHD, patients whose symptoms may be due to other chronic medical or psychiatric conditions may waste time, money and energy on a variety of treatments for ADHD.

Consequently, during the past two years our clinical research efforts shifted into an examination of core etiological and clinical issues. The initial questions centered on an examination of the electrophysiological characteristics of stimulant "non-responders", as well as, ADHD subtypes, since an important clinical application of QEEG assessment procedures would be assisting physicians in the determination of potentially effective pharmacological interventions.

Application of the QEEG Scan: Identifying Stimulant Responders vs Non-Responders

DSM-IV specifies inattention, impulsivity and hyperactivity as the essential behavioral symptom clusters for ADHD. There is no requirement that a patient display evidence of any type of abnormality on neurological or neurophysiological examination, despite the fact that psychopharmacological interventions constitute the primary type of treatment for patients diagnosed with this disorder (Barkley, 1998). However, as noted by Barkley (1998), approximately 30% of ADHD patients aged 5 through 12 and as many as 50% of adolescent patients with ADHD do not respond positively to stimulant therapy. Since there are no behavioral or psychological indicators that can be used to assist physicians in their decision-making, we sought to examine whether an electrophysiological measure could be used for such a purpose. The goal of our initial study was to examine whether ADHD patients who would respond positively to stimulant medications could be differentiated from stimulant "non-responders" on the basis of our QEEG Scan.

Our hypothesis was that stimulant responders would exhibit evidence of cortical slowing at the vertex (i.e., theta/beta ratios 1.5 S.D. above age peers), whereas, non-responders would exhibit either no evidence of such slowing or display evidence of "hyper-arousal". Initial evidence supportive of such a differentiation was suggested by the findings of Simeon, Ferguson and Fleet (1986) in their study of ADHD patients who responded positively to Buproprion,™ as well as by Chabot and Serfontein's (1996) identification of electrophysiological "subtypes" among ADHD patients (both hypo-arousal and hyper-arousal were noted). Our study examined 144 patients aged 6 to 20 who had been diagnosed with ADHD by their physicians using DSM-IV criteria. The male to female ratio was approximately 4:1 (115 males; 29 females). All patients had been screened for medical conditions that could contribute to attentional problems (e.g. thyroid disorders, anemia, hypo-glycemia).

Following a thorough clinical and medical assessment to determine a diagnosis of ADHD, all patients began a clinical trial of stimulant medication (Ritalin™ or methylphenidate) starting with lowest dose (5 mg; b.i.d.). Dosage was titrated to a maximum of 20 mg per dose, as needed. Stimulant response was considered positive if parent and teacher ratings improved to the non-clinical range on the ADDES (McCarney, 1995) and the results of either the T.O.V.A.(Greenberg, 1994) or the Conners' CPT (Conners, 1994) were within the "non-clinical" range according to computerized test interpretation. If the patient showed no significant positive response (based on parent and teacher ratings) to the maximum 20 mg dose of Ritalin, the initial stimulant was discontinued and a second stimulant was introduced (Adderall™). Dosage was titrated from 5 mg to a maximum of 20 mg. If no significant improvement was noted, the patient was considered a stimulant "non-responder". Dependent measures used in this study included a behavioral rating scale (The Attention Deficit Disorders Evaluation Scale (McCarney, 1995), the T.O.V.A. (Greenberg, 1994), the Conners' Continuous Performance Test (Conners, 1994) and the QEEG "Scan" (Monastra et al, 1999). Each of these tests was administered as part of the initial diagnostic process and the ADDES and Continuous Performance Tests were re-administered in order to assess stimulant response.

The QEEG "Scan" was only obtained prior to initiation of medication. The results of the study

indicated that a patient's response to stimulant could not be predicted on the basis of the number of ADHD symptoms, the frequency of ADHD symptoms on the ADDES, or the degree of impairment on the T.O.V.A. or Conners' CPT. Rather, as anticipated, when a criteria of 1.5 S.D. above the mean for age peers on the QEEG Scan was used to identify ADHD patients with cortical slowing, a high degree of predictive accuracy was achieved.

In our examination of 144 patients diagnosed with ADHD, 103 demonstrated cortical slowing; 41 did not. Although all were treated with the same pharmacological agents, 93% (96 of 103) of ADHD patients exhibiting cortical slowing showed positive response to stimulant therapy. None of the patients who showed no evidence of cortical slowing demonstrated positive response on the dependent measures. In addition, each of the "stimulant non-responders" exhibited at least three of the following "side effects" in response to stimulant therapy: headaches, increased irritability, sedation, rapid speech, increased intrusive and impulsive behaviors, or increased hyperactivity.

This observation of differential response to stimulant medication among patients diagnosed with ADHD could not have been anticipated on the basis of any of the commonly used strategies for assessment. As noted in the research of Chabot and his colleagues, the SPECT findings summarized by Amen (1998) and our initial research, it appears that there are biologically distinct subtypes of ADHD that can be differentiated via neuro-imaging and electro-physiological techniques. In general, our findings are consistent with those reported by Chabot, Amen and others who have noted cortical "slowing" in certain ADHD patients, and cortical "hyper-arousal" in others. Furthermore, our research suggests that only patients with ADHD who exhibit cortical slowing over frontal regions are likely to respond to stimulant therapy. Consequently, inclusion of a QEEG screening to examine patients for this pattern may prove useful in optimizing pharmacological response and reducing the discomfort caused to patients.

ADHD Subtypes: An Electrophysiological Perspective

In recent years, considerable scientific debate has been engendered regarding the relative importance of "inattention" vs. "hyperactivity/impulsivity" in determining a diagnosis of ADHD. Barkley (1998) has proposed that "behavioral disinhibition or poor regulation and inhibition of behavior are the hallmarks of ADHD" (p. 69) and questioned, "whether the requirement for significant inattention to diagnose ADHD is even necessary" (p. 65). By contrast, Brown (1999) has argued that problems of sustained attention are common among the various ADHD subtypes and that inattention is at the core of ADHD symptomatology.

In order to examine the question of whether "inattention," "behavioral disinhibition", or (as DSM-IV implies) both characteristics are essential symptoms of ADHD, an electrophysiological examination of 226 patients diagnosed with ADHD by their physicians was conducted at our clinic. While QEEG studies of patients with ADHD have examined a wide range of electrophysiological characteristics, primary interest has been focused on frontal and central theta (4-8 Hz), beta (ranging from 13-21Hz), and the sensorimotor rhythm (SMR: 12-14 Hz) over the motor cortex (C3; C4). From a functional perspective, frontal regions have been associated with "executive" functions (reasoning; concentration; sustained attention) and the

motor cortex with volitional control over movements.

Frontal and central "hypo-arousal" has been consistently identified in ADHD patients regardless of subtype (Mann et al, 1992; Janzen et al, 1995; Lubar et al, 1995; 1996) and no significant difference in the "theta/beta power ratio" was noted between patients with "Inattentive" and "Combined" types of ADHD in Monastra et al's (1999) study, although both groups of ADHD patients exhibited significantly more "slowing" over the frontal region than "non-clinical controls". Operant conditioning studies seeking to enhance SMR (e.g. Lubar & Shouse, 1976; Shouse & Lubar, 1979; Tansey & Bruner, 1983) have noted improved behavioral regulation following increased production of SMR over motor regions. Based on these findings, an examination of the electrophysiological characteristics of two groups of ADHD patients (Predominately Inattentive and Combined Types) and a non-clinical control group was conducted. Both "hypo-arousal" and impairment of regulatory activity over the motor cortex were examined.

Our hypotheses were as follows. First, if "inattention" was a common symptom among patients with "inattentive" and "hyperactive" symptoms, then evidence of cortical slowing should be noted at the vertex in both ADHD groups (as was noted in Monastra et al, 1999). Secondly, if impaired motor planning and regulation was a common characteristic of ADHD patients, then suppression of SMR activity should be noted in both ADHD groups relative to non-clinical controls. Finally, given the marked impairment in behavioral control that is evident in ADHD patients termed "hyperactive," evidence of greater SMR suppression should be noted in the "Combined" group, particularly when involved in completing a motor task requiring attention to detail and motor planning.

To test these hypotheses, 226 patients, aged 6 to 15, who had been diagnosed with ADHD by their physicians using DSM-IV criteria participated in this study. Fifty-five were diagnosed with ADHD, Predominately Inattentive Type; 171 with ADHD, Combined Type. In addition, 18 individuals who exhibited no evidence of significant impairment of attention or behavioral regulation were examined. The Male:Female ratio was approximately 4:1 (193 males; 51 females).

All participants had been evaluated by their physicians who determined that they had no other medical condition that could cause impairment of attention or behavioral control. QEEG examination consisted of an analysis of theta/beta power ratios (i.e., power recorded at 4-8 Hz compared to that recorded at 13-21 Hz) and theta/SMR power ratios (SMR defined as 12-15 Hz activity) during four, 90 second tasks: eyes open baseline, reading, listening and copying geometric shapes from the Benton Visual Retention Test. Recordings were obtained at the vertex. The resulting statistical analyses confirmed each of our hypotheses.

First, significantly higher theta/beta power ratios were recorded in both of the ADHD groups than in the "control" group on each of the four tasks ($p < .01$). No difference was noted between the ADHD groups indicating that both groups showed a similar pattern of "hypo-arousal" at the vertex. Secondly, both ADHD groups exhibited significantly higher theta/SMR ratios when compared to the "control" group, suggesting that impaired behavioral regulation was a characteristic of the two ADHD groups. Finally, as anticipated, differentiation between the two

ADHD groups was only noted on the graphomotor task, with the ADHD, Combined group exhibiting a significantly higher theta/SMR ratio than the ADHD, Inattentive group. Again, this was predicted since the ADHD, Combined group displayed much more dramatic behavioral characteristics of hyperactivity and impulsivity. Overall, this second study highlights the contribution that QEEG procedures can make in efforts to understand the nature of neuro-developmental disorders like ADHD.

An examination of the symptoms of ADHD reveals that imbedded in the DSM-IV behavioral descriptions of "Inattention", are symptoms of impairment of behavioral regulation (e.g. "Makes careless mistakes in schoolwork", "does not follow through on instructions", "has difficulty organizing tasks and activities", and "often loses things"). Each of these symptoms of inattention are manifested in the form of impaired motor planning and functioning. Similarly, several of the symptoms listed under "Hyperactivity" (e.g. "often leaves seat in classroom", runs about or climbs excessively in situations in which it is inappropriate", "often talks excessively") would certainly not be considered to be "attentive" behaviors during instruction. Consistent with such an overlapping perspective of "behavioral" symptoms, QEEG data reveal evidence of both "hypo-arousal" and impairment in the production of the type of EEG activity associated with behavioral inhibition. Consequently, these findings support a perspective that ADHD can be viewed as a neurophysiological disorder characterized by evidence of impaired attention and behavioral regulation.

Summary

During the past two decades, significant advances have been made in developing normative QEEG databases for use in clinical research. However, despite progress made in the field, critics of QEEG approaches have questioned the application of this data, typically noting that psychiatric diagnosis is made on the basis of behavioral symptoms and clinical history. While the DSM-IV currently defines ADHD exclusively in behavioral terms, the clinical practice of primary care physicians reflects a position that ADHD is a medical condition that will respond to interventions that primarily target dopaminergic neural pathways. Such a perspective appears to reflect recognition of emerging neurological and neurophysiological models of the biological foundations of ADHD.

Proceeding from a neurophysiological perspective on ADHD, this paper describes several ways in which QEEG data can be applied in clinical settings. First, by using QEEG indicators of "hypo-arousal" practitioners may be able to reduce the number of patients who display adverse side effects and no positive response to stimulant therapy. In addition, as suggested by Amen (1998), ADHD patients who exhibit "hyper-arousal" may prove responsive to a variety of "non-stimulant" medications (e.g. SSRI) and the use of QEEG screening may facilitate the development of an appropriate pharmacological treatment plan for such patients. Secondly, electrophysiological findings support the perspective that impaired attention and behavioral regulation are common to two of the primary subtypes of ADHD. Targeting both of these QEEG indicators provides a promising direction for clinicians and clinical researchers seeking to develop neurotherapeutic and pharmacological treatments that promote sustained clinical improvement in patients diagnosed with ADHD.

References

Amen, D.G. (1998). *Change your brain, Change your life*. New York: Times Books.

Amen, D.G. Paldi, J.H. & Thisted, R.A. (1993). Evaluating ADHD with brain SPECT imaging. *Journal of the American Academy of Child and Adolescent Psychiatry, 32*, (5), 1080-1081

Barkley, R.A.(1994). Neurometrics: Review and Comments. *The ADHD Report, 5* (2), 3.

Barkley, R.A. (1998). *Attention-Deficit Hyperactivity Disorder: A handbook for diagnosis and treatment.* New York: The Guildford Press.

Brown, T.E. (1999). Does ADHD diagnosis require impulsivity-hyperactivity? A response to Gordon & Barkley. *The ADHD Report, 7* (6), 1-7.

Chabot, R.A. Merkin, H. Wood, L.M. Davenport, T.L. & Serfontein, G. (1996). Sensitivity and specificity of QEEG in children with attention deficit or specific developmental learning disorders. *Clinical Electroencephalography, 27*, 26-34.

Chabot, R.A. & Serfontein, G. (1996). Quantitative electroencephalographic profiles of children with attention deficit disorder. *Biological Psychiatry, 40*, 951-963.

Conners, C.K. (1994). *Conners' Continuous Performance Test*. Toronto, Canada: Multi-Health Systems.

Greenberg, L.M. (1994) *Test of Variables of Attention Continuous Performance Test*. Los Alamitos, CA: Universal Attention Disorders.

Hynd, G.W. Semrud-Clikeman, M. Lorys, A.R. Novey, E.S. Eliopulos, D. & Lyytinen, H. (1991). Corpus callosum morphology in attention deficit-hyperactivity disorder: Morphometric analysis of MRI. Special Series: Attention deficit disorder. *Journal of Learning Disabilities, 24*, 141-146.

Janzen, T. Graap, K. Stephanson, S. Marshall, W. & Fitzsimmons, G. (1995). Differences in baseline EEG measures for ADD and normally achieving preadolescent males. *Biofeedback & Self Regulation, 20*, 65-82.

Lou, H.C., Henriksen, L, & Bruhn, P. (1984). Focal cerebral hypoperfusion in chilren with dysphasia and/or attention deficit disorder. *Archives of Neurology, 41*, 825-829.

Lubar, J.F. Swartwood, M.O. Swartwood, J.N. & O'Donnell, P.H. (1995). Evaluation of the effectiveness of EEG neurofeedback training for ADHD in a clinical setting as measured by changes in T.O.V.A. scores, behavioral ratings and WISC-R performance. *Biofeedback and Self-Regulation, 20*, 83-99.

Lubar, J.F. & Shouse, M.N. (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.

Lubar, J.F. Swartwood, M.O. Swartwood, J.N. & Timmermann, D.L. (1996). Quantitative EEG and auditory event-related potentials in the evaluation of attention-deficit/hyperactivity disorder: Effects of methylphenidate and implications for neurofeedback training. *Journal of Psychoeducational Assessment*, 143-160.

Mann, C. Lubar, J. Zimmerman, A. Miller, C. & Meunchen, R. (1992). Quantitative analysis of EEG in boys with attention-deficit-hyperactivity disorder: Controlled study with clinical implications. *Pediatric Neurology, 8*, 30-36.

McCarney, S.B. (1995). *The Attention Deficit Disorders Evaluation Scale*. Columbia, MO: Hawthorne Press.

Monastra, V.J. Lubar, J.F. Linden, M. VanDeusen, P. Green, G. Wing, W. Phillips, A. & Fenger, T.N. (1999) Assessing attention deficit hyperactivity disorder via quantitative electroencephalography: An initial validation study. *Neuropsychology, 13* (3) 424-433.

Shouse, M.N. & Lubar, J.F. (1979). Operant conditioning of EEG rhythms and Ritalin in the treatment of hyperkinesis. *Biofeedback and Self-Regulation, 4*, 301-312.

Simeon, J.G. Ferguson, H.B. & Fleet, J.V.W. (1986). Buproprion effects in attention deficit and conduct disorders. *Canadian Journal of Psychiatry, 31*, 581-585.

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

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