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Quantitative EEG and Auditory Event-Related Potentials in the Evaluation of Attention-Deficit/Hyperactivity Disorder: Effects of Methylphenidate and Implications for Neurofeedback Training

J. F. Lubar, M. O. Swartwood, J. N. Swartwood, D. L. Timmermann
University of Tennessee

Neurophysiological correlates of Attention Deficit Disorder with and without Hyperactivity (ADD/HD) and effects of methylphenidate are explored using electroencephalographic (EEG) and auditory eventrelated potentials (ERPs). In the first of four studies, a database of ADD/HD individuals of varying ages and matched adolescent/adult controls is presented. Study 2 compares controls and age-matched children with ADD, and children with ADHD on and off methylphenidate. Study 3 examines habituation of the auditory ERPs of controls and children with ADHD both on and off methylphenidate. The relationship between successful neurofeedback training and EEG changes is presented in Study 4. Overall, these studies support a neurologic basis for ADD/HD and raise questions regarding the role of methylphenidate in modulating cortical processing.

Attention Deficit/Hyperactivity Disorder (ADHD) is now recognized as one of the most complex psychiatric and neurologically based disorders of childhood with significant representation in adolescents and adults. ADHD rarely occurs in isolation; it is often comorbid with other conditions, including depression, oppositional defiant disorder, conduct disorder, obsessive-compulsive disorder, learning disabilities, anxiety disorders, and other significant psychological, psychiatric, and neurological problems (Barkley, 1981, Ross & Ross, 1982; Rutter, 1983; Whalen, 1983). Differential diagnosis of ADHD is therefore difficult. Currently, diagnosis of Attention Deficit Hyperactivity (ADHD) and Attention Deficit Disorder without Hyperactivity (ADD) is based on detailed developmental and school history, psychometric measures, rating codes, continuous performance tasks, and, occasionally, neuropsychological evaluation.

A multi-diagnostic approach is clearly appropriate in the differential diagnosis of ADD/HD because of the multidimensional aspects of this complex disorder. There has been a growing literature, dating back at least 50 years, that indicates that ADD/HD has a neurological basis. For example, in an early study by Jasper, Solomon, and Bradley (1938), children with "behavioral problems", which undoubtedly included ADD and ADHD as well as a number of other disorders, presented numerous electroencephalographic abnormalities, including slowing and, rarely, paroxysmal

activity, indicating that a seizure disorder may be part of the problem. More recently, Satterfield, Cantwell, Saul, Lesser, and Podosin (1973) described EEG slowing as one of the diagnostic signs in individuals with "minimal brain dysfunction syndrome," a previous categorization for many individuals with ADD/HD. Matousek, Rasmussen, and Gillberg (1984) evaluated the frequency distribution in children with minimal brain dysfunction (MBD syndrome) and reported slow EEG activity as one of its significant characteristics.

Early analysis of EEG data in individuals with ADD/HD (or their preceding diagnostic labels) was based primarily on qualitative analysis. Current quantitative analysis (QEEG) may employ several electrode placements or whole brain topographic mapping to compare individuals with normative data bases. A previous study by Mann, Lubar, Zimmerman, Miller, and Muenchen (1992) presented the first clear evidence that individuals with Attention Deficit Disorder without Hyperactivity (ADD) have significantly more 4 to 8 Hz theta activity and significantly less beta activity above 13 Hz in many cortical locations, particularly when challenged with academic tasks such as reading and drawing. In some cases these differences were as great as 30% in terms of the increased slow activity as compared with matched controls. One of the best reviews of the neurological basis for ADHD is by Riccio, Hynd, Cohen, and Gonzalez (1993), who present data to indicate that slowing is one of the primary EEG hallmarks of this disorder.

In a recent book, *Images of Mind,* Posner and Raichle (1994) presented a detailed description of the attentive mechanisms of the brain based on EEG and PET scan studies. They also provided data indicating that an area in the right posterior parietal cortex is involved in vigilance, and "executive attention" areas involved in motor and verbal responses are largely localized in the anterior cingulate gyrus between the cerebral hemispheres. The cingulate sends its output to the septal region, frontal cortex, and the supplemental motor areas that have been implicated in ADHD. Other authors have presented data to show that the right posterior parietal region is particularly important in organizing attentive mechanisms (Riccio et al., 1993; Voeller, 1991; Weinberg & Emslie, 1991).

Single proton emission tomography (SPECT) scan and PET scan studies have provided evidence that there is a metabolic disorder in ADD and ADHD individuals as well as electroencephalographic differences when compared to matched controls (Amen, Paldi, & Thistead, 1993). Zametkin and Rapoport (1987) have reviewed evidence that catecholamines, particularly dopamine and norepinephrine, are involved in attention deficit disorders. Of particular interest are findings that support the view that the prefrontal lobes are involved in ADHD (Zametkin et al., 1990). They report hypoprofusion of these brain regions, particularly the right frontal area and the posterior medial orbital cortex, in their PET scan studies of regional cerebral blood flow. These initial studies were done in adults who had a childhood history of ADHD. Previous studies have indicated that children with ADHD show decreased metabolic activity in prefrontal lobes and increased metabolic activity in primary sensory and sensorimotor cortex (Lou, Henriksen, & Bruhn, 1984).

Another method for evaluating neurological effects is based on eventrelated potentials (ERPs). These are time locked responses to specific stimuli. ERP components are believed to be associated with attentive mechanisms and cognitive processing (Andreassi, 1989). The search for differences in ERPs between children with and without ADHD has often resulted in contradictory findings, thus failing to provide a consistent physiological profile characteristic of the functioning of their central nervous systems (Buchsbaum & Wender, 1973; Callaway, Halliday, & Naylor, 1983; Hall, Griffin, Moyer, Hopkins, & Rappaport, 1976; Loiselle, Stamm, Maitinsky, & Whipple, 1980). For example, Prichep, Sutton, and Hakerman (1976) found higher amplitude P300 components in "hyperkinetic" children compared with normal controls, whereas Michael, Klorman, Salzman, Borgstedt, and Dainer (1981) found Smaller late positive components, and Loiselle et al. (1980) found no significant amplitude increment of the P300 component. Research on the early negative components also yields contradictory results, with findings of higher amplitudes of the negative waves such as the N1 component in hyperkinetic children (Callaway et al., 1983; Prichep et al., 1976) and findings of no significant amplitude increase in the N1 component in hyperkinetic children (Loiselle et al., 1980).

Although these and other studies yield conflicting data, if the syndrome of ADHD is the result of an aberrant development of the central nervous system, then physiological differences between age-matched ADHD and normal children should be readily apparent. An inherent flaw in previous research in this area has been the failure to obtain homogeneous groups of subjects, with children with learning disabilities and ADHD grouped together in the same clinical samples (Harmony et al., 1990). Additionally, there is clearly an age effect with regard to the ERP, with differential effects reported according to age (Satterfield, Schell, Nicholas, & Backs, 1988).

These studies and others provide a logical basis for the use of methylphenidate and other stimulants that are believed to affect both dopaminergic and adrenergic systems in the brain, ameliorating some of the symptoms of ADDADHD (Levy, 1991). However, in a recent study by Matochik et al. (1994) it was reported that cerebral metabolism in adults with ADHD was not affected by the administration of methylphenidate in 63 of 64 brain areas tested in PET scanning. Only in the putamen, a structure involved in the extra-pyramidal motor modulation of alpha motor neurons, was there evidence of an effect of this stimulant medication. This finding raises the interesting question of how does methylphenidate act if it doesn't affect cerebral metabolism? However, it must be pointed out that the Matochik et al. (1994) study was carried out in adults, and a similar study employing children still needs to be done. Clearly there is considerable evidence based on EEG and cerebral metabolic measures that supports the view that there is a neurological basis for Attention Deficit Disorder.

In the remainder of this paper we describe several studies that explore EEG and eventrelated potential (ERP) correlates of ADD/HD. In the first two studies, databases for evaluating individuals with Attention Deficit Disorder are shown, the relationship between quantitative EEG measures for individuals with the inattentive form of ADD as compared with the hyperactive form is investigated, and the effects of methylphenidate

on the EEG are evaluated. In the third study, auditory eventrelated potential data are presented indicating differences in the way in which children diagnosed with ADHD process information on and off methylphenidate as compared with matched controls. Finally, in the fourth study, data are shown to indicate that neurofeedback training designed to change EEG patterns associated with the inattentive form of ADD is related to the successful learning of the neurofeedback task and improvement in ADD symptomatology.

STUDY 1

Quantitative Differences between Individuals with ADD/ADHD of Different Ages and Adult Controls

Method

Subjects. This study consisted of 112 subjects independently diagnosed with ADD/HD; none of the subjects had received a diagnosis of learning disability. Of the subjects, 65 (59 males and 6 females) were between ages 8 and 11 (mean age = 10.9), 21 (15 males and 6 females) were between ages 12 and 14 (mean age = 13.1), and 26 (25 males and 1 female) were between ages 15 and 46 (mean age = 19.5). In addition, a control group of individuals (9 males and 2 females) between ages 16 and 55 (mean age = 41.3) were matched to the ADD/HD group in that approximate age range.

Procedure

Quantitative EEG measurements were obtained over the standard 19 locations normally used in clinical EEG. The subjects were fitted with an electrode cap containing the 19 electrodes in the standard 10-20 international EEG configuration. This cap was manufactured by Electro Cap International, Inc., and is used for quantitative EEG measurements in many hospital and clinic settings. The EEG amplifiers were contained in the Lexicor Medical Technology Neurosearch 24 instrument. The cap was fitted to each individual, and electrode gel was introduced into each electrode site. Electrode skin impedances were maintained below 5K ohms. All measurements were taken referentially with linked ears. The subject was seated in front of an easel where materials could be presented and measures were obtained under six different conditions. Each condition lasted for 200 seconds.

Measurements were taken during an eyes open baseline, where subjects were required to fixate on a point approximately 2 feet in front of them. This was done in order to minimize eye movement artifacts. Subjects were also asked to blink as little as possible. The second condition consisted of an eyes closed baseline, where subjects were told to close their eyes and to try to keep their eyes fixated forward to avoid eye movements

under the eyelids that could contaminate the EEG recordings. The third condition had the subjects read silently materials that were age or grade-appropriate. Condition 4 involved reproducing figures from the Berry Bender Visual Motor Gestalt Test (Bender, 1946). Condition 5 consisted of identifying missing portions from pictures taken from the Ravens Color Progressive Matrices (Raven, 1960). Condition 6 involved listening to materials read aloud by the therapist. These materials were usually a continuation of the story that the subjects had been reading silently to themselves in the third condition.

After completion of the testing, all of the EEG data was analyzed in order to reject artifacts due to eye movements, blinks, and muscle activity. The artifact-rejected data were than subjected to Fourier power spectral analysis, resulting in measurements of the percentage power of theta activity (48 Hz) and beta activity (1321 Hz). Data were then imported into Lotus or Excel programs for analysis and graphing.

Results

Figure 1 shows the mean theta/beta ratios for all of the eyes open conditions averaged together for each of the 19 electrode locations and for the different age groups. Table 1 presents the results from one way ANOVAs comparing theta/beta ratios during the eyes open condition for the adult controls with each of the ADD groups and for the ADD groups with each other.

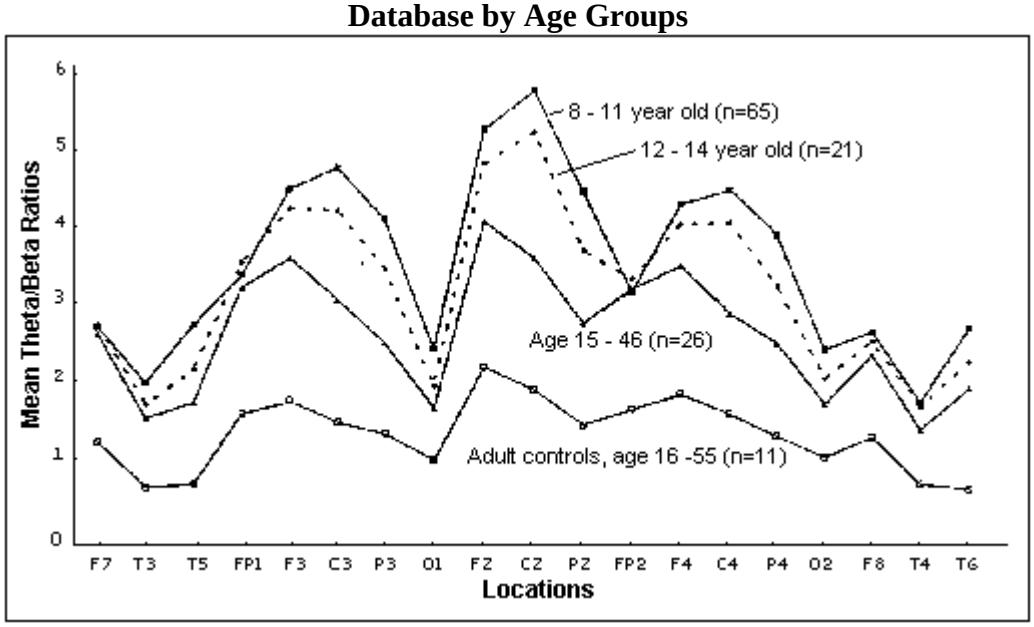

Figure 1. Mean Theta (4-8 Hz) / Beta (13-21 Hz) ratios to power ratios for 19 standard EEG locations according to the 10-20 International System. These ratios represent three groups with Attention Deficit Disorder and one adult control group; they were calculated for five eyes-open conditions averaged together.

Table 1. Results from One-Way ANOVAs Comparing Theta/Beta Ratio, Data Averaged across All Eyes-Open Conditions

10 - 20 Scalp Location	F-value ANOVA	F-prob. ANOVA	ADD- v. Controls $p < .05$	ADD- v. ADD+ No Ritalin $p < .05$	ADD- v. ADD+ Ritalin $p < .05$
Left hemisphere					
F7					
T5	3.52	0.017			■
FP1					
F3	6.57	0.0004	■	■	■
C3	3.66	0.014		■	■
P3	4.15	0.007	■	■	■
O1					
Central					
FZ	3.47	0.019			■
CZ	2.40	0.07			
PZ					
Right hemisphere					
FP2					
F4	5.80	0.001	■	■	■
C4	2.75	0.046			
O2					
F8					
T4					
T6					

* = Significance -- Note: Alpha level adjusted to $\boldsymbol{p} < .003$.

The most important aspect of Table 1 is the comparison of the controls with the ADD/HD individuals matched for age (shown in the first data column of Table 1). Clearly, there are many locations in which there are significant differences between Figure 1. Mean theta (48 Hz)/beta (1321 Hz) ratios to power ratios for 19 standard EEG locations according to the 10-20 International System. These ratios represent three groups with Attention Deficit Disorder and one adult control group; they were calculated for five eyes open conditions averaged together the two groups. The greatest differences were at central locations FZ, CZ, and PZ along the midline (see Figure 1). Significant differences corrected for multiple comparisons were also obtained in many other locations. Differences between the control group and the 8 to 11 year old or 12 to 14 year old group are interesting but not particularly meaningful, because this comparison is between children and older adolescent/adult controls. However, differences between the 8 to 11 year old group and the 12 to 14 year old group (see the last column in Table 1) are of some importance in that they indicate that the only significant locations were over the temporal lobe location T5 on the left side, PZ on the midline, and P4 over the right posterior-parietal region. This means that the 8 to 11 year old and 12 to 14 year old databases are very similar, and that they could be collapsed for future work, with the exception of the three locations mentioned above. However, both of these groups differ from the adolescent/adult ADD group in a number of locations, particularly for the comparison between 8 to 11 year olds and the oldest group.

Discussion

The importance of developing a database by age is that it will eventually be possible to compare new individuals with the data base on an age-matched basis. Other studies have clearly shown that EEG frequency and amplitude are very age dependent (Gasser,

Verleger, Bacher, & Sroka, 1988). Another advantage of this approach is that it allows for the evaluation of differences based on theta/beta ratios or other EEG measures for matched individuals within a particular age range, as will be shown in the next study.

STUDY 2

Differences between ADD Inattentive Type and ADD Hyperactive Type and the Effects of Methylphenidate

Method and Procedure

Subjects. This study consisted of four all male groups: Fifty one children between ages 8 and 11 with the inattentive type of ADD; 23 age matched non-ADD controls (mean age 9.8 years); and 23 children with the hyperactive type of ADD (ADHD) (mean age 9.7 years) measured under two conditions, with methylphenidate medication (Ritalin) given 30 minutes prior to testing and again 48 hours after total medication withdrawal. The medication dosage was titrated individually by each child's physician, based on optimal behavioral changes. Daily dosages ranged from 10 mg to 60 mg per day (mean = 20.7 mg), with larger dosages divided over several administrations each day. The actual dosages that were given prior to data collection ranged from 5 mg to 20 mg (mean = 14.1 mg). The subjects were not on any other medications during this study.

The procedures involved in this evaluation were identical to those used in the first study. Measurements were taken under the same baseline and academic conditions with the same instrumentation and electrode configurations.

Results

Figure 2 shows differences between the four groups described. Clearly the individuals with ADD inattentive type have the highest ratios of theta/beta activity; however, children with the hyperactive type of ADHD actually have somewhat lower theta/beta ratios than the matched controls. Interestingly, Ritalin has virtually no effect on theta/beta ratios in children with ADHD. Table 2 presents results from the statistical analyses for Figure 2.

Database of ADD-, ADD+ and Controls, Ages 9-11

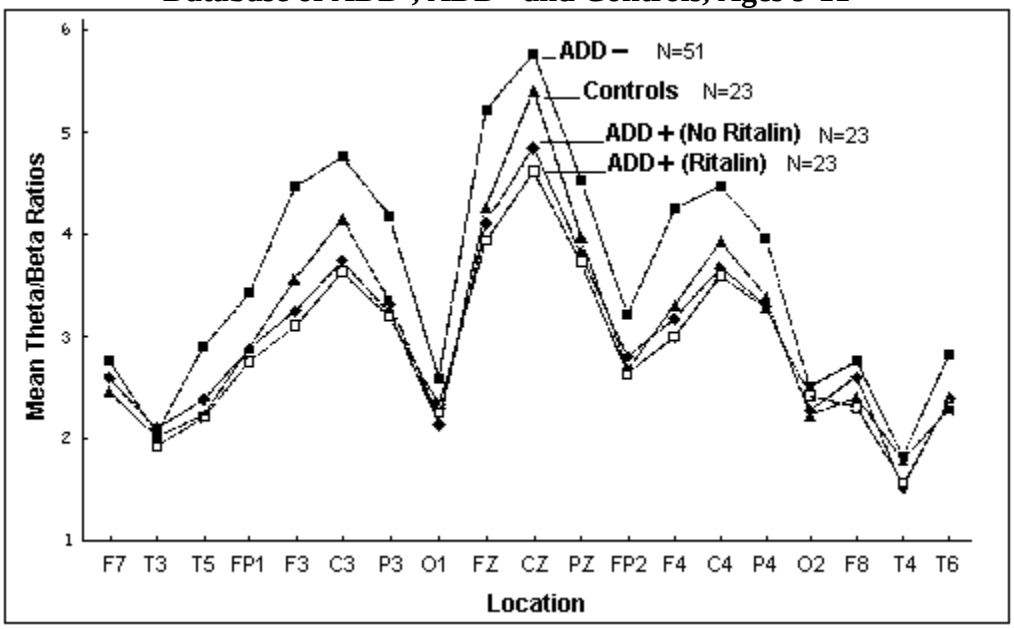

Figure 2. Theta-beta ratios for children with Attention Deficit Disorder with Hyperactivity and controls. The group with hyperactivity was tested under two conditions, with and without methylphenidate (Ritalin medication.)

Table 2. Significant Differences by Locations for ADD without Hyperactivity Compared with Other Groups Theta-Beta Ratios for ADD-, ADD+ without Ritalin, ADD+ with Ritalin, and Controls
Ages 9-11 Males Student-Newman-Keuls Test

10 - 20 Scalp Location	F-value ANOVA	F-prob. ANOVA	ADD- v. Controls $p < .05$	ADD- v. ADD+ No Ritalin $p < .05$	ADD- v. ADD+ Ritalin $p < .05$
Left hemisphere					
F7					
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Central					
FZ	3.47	0.019			■
CZ	2.40	0.07			
PZ					
Right hemisphere					
FP2					
F4	5.80	0.001	■	■	■
C4	2.75	0.046			
O2					
F8					
T4					
T6					

Discussion

There were a number of overall significant differences, particularly in the two central locations FZ and CZ and in locations T5, F3, C3, and P3 on the left side and F4 and C4 on the right side. Most of these locations are frontal and central; however, T5 is a left temporal location. One of the most important findings is that the inattentive ADDs differed significantly from their matched controls in frontal location F3, posterior location Pc, and right frontal location F4. In referring back to Figure 1 where the late adolescent and adult control group is compared with the matched ADD/HD groups, there were many more differences between the older matched group compared with the younger groups. Could this imply that the neurological deficit becomes greater in adulthood? The inattentive ADDs were also significantly different from the hyperactive ADHDs in the additional location C3. Even greater differences appeared when ADDs were compared with the ADHDs placed on Ritalin. However, there were no significant differences for any of the locations between the ADD group with and without Ritalin. This finding is most interesting in light of the Matochik et al. (1994) study cited earlier, in which no differences were found in 63 out of 64 locations in terms of the effects of Ritalin on cerebral metabolism. The current study indicates that there may be no significant effect of methylphenidate on these EEG measures as well.

STUDY 3

Habituation of Auditory Event-Related Potentials (ERPs) in Children with the Hyperactive Form of Attention Deficit Disorder with and without Ritalin Compared with Age-matched Controls

The purpose of the study was to provide age-matched, homogeneous samples of ADHD and control children. A habituation paradigm was used in order to determine differential effects of repeated presentation of a stimulus in control and ADHD subjects.

Method

Subjects. Seventeen males diagnosed with ADHD ranging in age from 9 to 11 years (mean age = 9.9 years) and 18 matched control children (mean age = 9.7 years) were used as subjects. Subjects in the ADHD group met the following criteria: (a) They had been physician diagnosed with ADHD according to *DSM-III-R* (1986) criteria; (b) they had no learning disabilities; (c) they were currently being treated with methylphenidate; and (d) they were rated by the parent in the clinically significant range on at least one subscale of the McCarney Attention Deficit Disorder Evaluation Scale (ADDES; McCarney, 1989). All subjects in the ADHD group were tested both on methylphenidate and again after being medication free for at least 48 hours.

Control group subjects were age matched to subjects in the ADHD group. Additionally, no control subject was rated on any subscale of the McCarney ADDES rating scale as

having symptoms consistent with a diagnosis of ADHD. Any subject who was rated as one standard deviation or more from the ADDES rating scale norms was excluded from the study.

Procedure

For collection of ERPs, the Lexicor NRS24 equipment was used in conjunction with Lexicor evoked potential software. The process of administering neurological and physiological tests was the same for both ADHD and control subjects, and event related potential data were collected at the same point in the testing regimen for all subjects. Electrode placements were made according to the International 1020 system and included the central midline locations, FZ, CZ, and PZ. Auditory ERP data were collected using an "oddball" paradigm. Subjects were instructed to close their forefinger and thumb together upon hearing an "odd" pitched tone. These "odd" pitched tones were interspersed among "common" tones. Odd tone presentations made up 20 % of the total tones presented. The tones were presented at random, one tone per second for a total of 200 seconds.

Data management. ERP data were averaged separately for each stimulus category, condition, and electrode placement for all artifact free trials. After artifact rejection for eye movements and muscle activity, data were stored on magnetic tape for later analyses.

In order to assess habituation effects, the ERP raw data were divided into thirds, with each third consisting of 65 seconds of data. Grand averages of ERP data were created for each subject group (controls, ADHD on medication, and ADHD off medication) using the Lexicor software. The latencies of the NI, P2, N2, and P3 components were determined from each group's grand average for the first and last thirds of the ERP recording. These latencies were used as time markers to identify the amplitude of each component for individual subjects. Paired *t* tests were used to determine if any change occurred in amplitude (habituation effects) between the first and last third of the recording session within each subject group.

Results

There were no significant differences in amplitude between the first and last third of the ERP recording for the control group. However, the ADHD group, both on and off medication, showed a decrease in amplitude for all four of the ERP components measured between the first and last third of the recording ($t < .05$). Using a Bonferroni adjusted alpha level of .004, only the P2 component showed a significant habituation effect in ADHD children. This effect occurred both on and off medication, although at different locations.

Table 3. Significant Amplitude Differences between the First and Last Third of the ERP Data for Each Group

	Location		
Wave Component	FZ	CZ	PZ
ADHD Group on Ritalin Data			
N1	*		
N2	*		
P2	**		*
P3			*
ADHD Group Off Ritalin Data			
N1		*	*
N2		*	*
P2			**
P3		*	
Control Group Data			
No significant differences			

* $p < .05$, ** $p < .004$ (Bonferonni adjusted p value).

Discussion

Since children with ADHD have poorer accuracy and slower reaction time than other children on performance tests, and their performance degrades over time with repeated presentation of stimuli (Greenberg, 1987), changes in cortical ERPs over time could be a potential index of neurophysiological processing difficulties for these children. Children diagnosed with ADHD show a greater degree of habituation to a repeated stimulus when compared with controls (Allen, 1986), and it is likely that this may be manifested by a degradation of some or all ERP components in the later portion of stimulus presentation. The possibility that children diagnosed with ADHD would differ from those without ADHD in terms of habituation to a repeated stimulus is supported by this study. The control group did not show any significant differences in amplitude between the first third and last third of the recording session. This finding indicates that children without a diagnosis of ADHD are able to maintain attention over several minutes while listening to repetitive tones. The children in the ADHD group, however, habituated over time as shown by the decrease in amplitude at one or more locations for all components of the ERP.

Interestingly, methylphenidate, although affecting the location where the decrease in amplitude occurs, does not appear to reduce the habituation effects; that is, methylphenidate does not cause the ERPs of the ADHD group to resemble more closely those of the control group in terms of amplitude habituation. Methylphenidate does affect the location where significant decreases in amplitude occur, however. The P2 component shows a significant difference in amplitude at PZ for the ADHD group off medication, and significant differences at FZ while on medication.

STUDY 4

Effect of EEG Neurofeedback Training on Quantitative EEG Method and Procedure

This study consisted of 17 children with ADD/HD between the ages of 8 and 15. Individual subjects received between 30 and 45 sessions of EEG neurofeedback training in an attempt to help them overcome some of the symptoms associated with their Attention Deficit Disorder. The training was carried out with two neurofeedback systems, the Lexicor system described previously employing the Biolex biofeedback program and the Autogen A620 Neurofeedback system developed by Stoelting Autogenics Corporation. The subjects were trained specifically to decrease either the microvolt levels or the percentage of theta activity in their EEGs, and to simultaneously increase the percentage of beta activity. For the purposes of neurofeedback training, theta was defined as 4 to 8 Hz and beta was trained between 16 and 20 Hz. Each child was seated in front of the neurofeedback instrument with recording electrodes located along the midline in a bipolar montage, with one electrode halfway between FZ and CZ (location FCZ) and the other electrode halfway between CZ and PZ (location CPZ) with ear reference.

Training was done under four specific conditions. Two of these training conditions involved trying to increase the number of rewards for changing the proportion of theta and beta activity; that is, decreasing theta and increasing beta activity. Two additional conditions combined academic tasks, including reading and listening, with neurofeedback. The baseline condition without feedback was also provided initially at the beginning of each session. Lubar, Swartwood, Swartwood, and O'Donnell (1995) also studied this group of children in another study in which pre and post measures were taken on the Test of Variables of Attention (TOVA), a continuous performance task (Greenberg, 1987), and the ADDES behavior rating scale (McCarney, 1989).

For the purposes of this study, the subjects were divided into two groups, those who were successful in learning and those who were unsuccessful. The successful learners were able to decrease theta activity (either in percentage or microvolt level) or to increase the percentage of beta activity. Dividing the subjects into groups resulted in 11 successful learners (9 males and 2 females) and 6 poor learners (all males). Pre-training and post-training multi-channel topographic brain maps were obtained for each subject using the paradigm presented in Studies 1 and 2.

Figure 3 shows the differences in theta/beta ratios between pre- and post- testing for those individuals who were successful in learning the task; Figure 4 shows the theta/beta ratios for those who were not successful. Figure 5 presents the relationship between these measures in terms of correlations over training trials.

Pre-Post Training Differences in Theta-Beta Ratios for 11 Good Learners

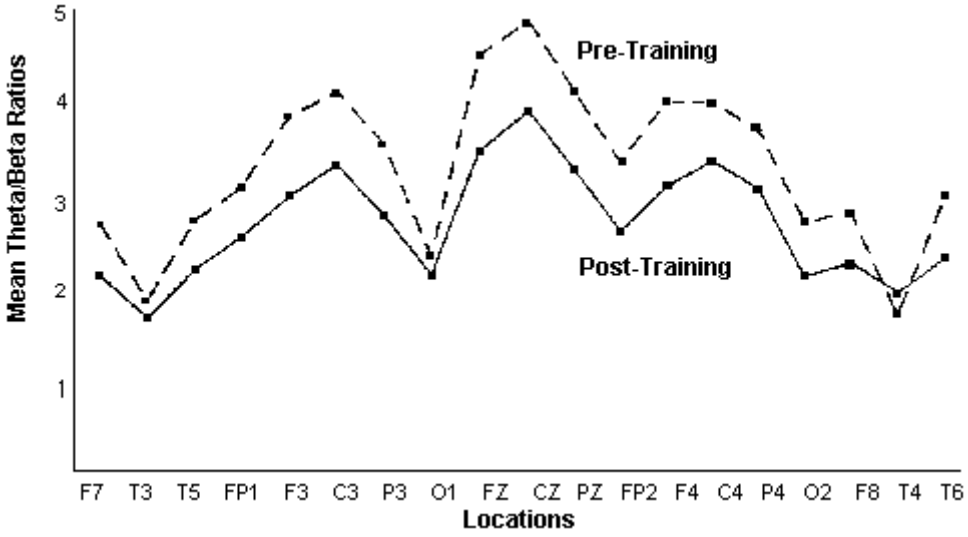

Figure 3. Pre and post training mean theta-beta power ratios for 11 children who were successful in learning neurofeedback paths.

Pre-Post Training Differences in Theta-Beta Ratios for 6 Poor Learners

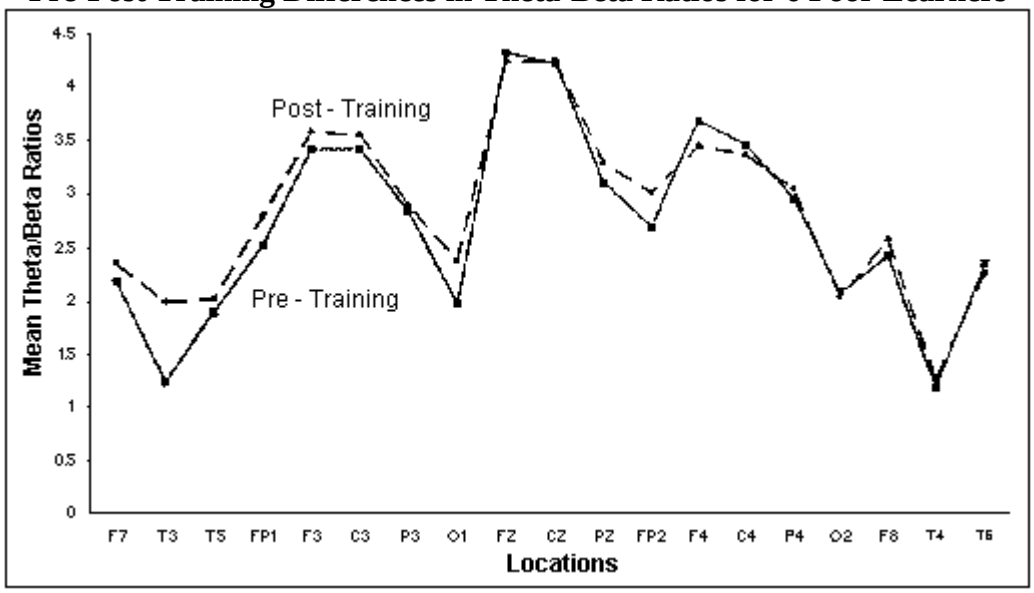

Figure 4. Pre and post training mean theta-beta power ratios for 6 children who were not successful in learning a neurofeedback task.

Relationship between Measured Learning Parameters and Quantitative EEG

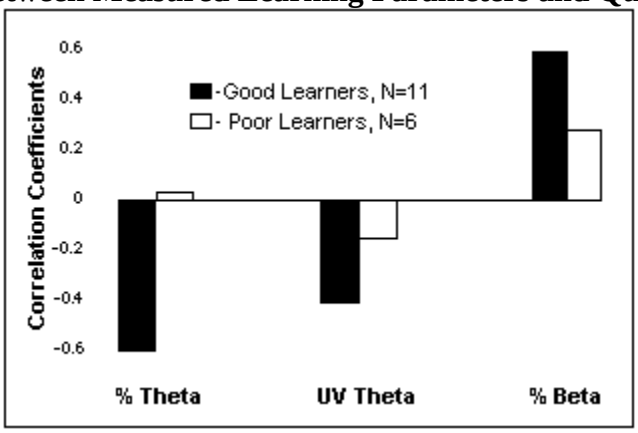

Figure 5. Relationship between neurofeedback measures relating success in learning and quantitative EEG changes.

For the individuals who were successful in learning the neurofeedback tasks, there was a significant decrease in the percentage of theta over sessions. The non-learners showed no significant change. The successful learners were also able to decrease the microvolt levels of theta, whereas the unsuccessful learners showed a very small decrease that was not significant. Finally the successful learners were able to increase the percentage of beta activity, whereas the nonlearners showed a small increase that was not significant.

Discussion

This study shows that success in neurofeedback training can be monitored by changes in the overall EEG; it also indicates that training in a single bipolar location along the midline generalizes to many cranial locations, as shown in Figure 3.

GENERAL DISCUSSION AND CONCLUSIONS

In this paper, we sampled a cross section of the literature that indicates that there is a neurological basis for ADD/HD, especially for individuals who experience the inattentive form of this disorder. This review includes research using both EEG and ERP measures. We presented four studies to further clarify the nature of electrophysiological correlates of ADD/HD. In the first study, we showed that there are age dependent differences in measures of theta/beta ratios, a commonly used statistic for evaluating differences between individuals with Attention Deficit Disorder and controls. We have also shown that there is a large and significant difference between a late adolescent and adult control group as compared with individuals who have Attention Deficit Disorder of the inattentive type in the same age range. The second study summarized theta/beta ratios under administration of methylphenidate and in control conditions without

methylphenidate for three groups of individuals matched in age (9-11). Two findings stand out in this study. First, individuals with the inattentive form of Attention Deficit Disorder (ADD) had the highest theta/beta ratios when compared with ADHD and control groups. However, contrary to expectations, individuals who have the hyperactive form of Attention Deficit Disorder actually have lower theta/beta ratios than do matched controls; in addition, methylphenidate does not change these ratios significantly.

The third study showed that children who have a diagnosis of ADHD show greater habituation in terms of event-related potential measures than do matched controls. Further, a normalizing effect of methylphenidate on this habituation effect was not shown, in that both the on and off-medication groups showed a significant habituation effect on the P2 component, with no such effect present in normal controls.

This increased habituation response to a novel stimulus in the ADHD children appears to be related to the fact that individuals with attention deficit disorders fail to sustain attention over time. As mentioned previously, the TOVA has been widely used, with success as high as 75% in differentiating individuals with ADHD from controls (Greenberg, 1987). In this task, the primary deficit in children with ADHD seems to be in variability of response and response time, with decreased performance over time. Both performance data and ERP data from the present study indicate that more rapid habituation of a response to a sensory event appears to be one of the hallmarks of ADHD.

The fourth study showed that there is a relationship between the ability to learn a neurofeedback task designed to change EEG activity and the degree of changing overall EEG parameters, specifically the theta/beta ratios that are based on either power or percentage power of activity in those two bands as compared with the full EEG spectrum. However, there has been some controversy regarding the use of neurofeedback as an adjunctive treatment for ADD/HD. Nevertheless, there is considerable recent evidence that this may be a very important modality of treatment to be integrated into a multi-modality program. A recent study by Lubar et al. (1995) shows that neurofeedback is highly effective, and previous studies reviewed by Lubar (1991) indicate that neurofeedback can play an important role in helping individuals with ADD/HD (particularly of the inattentive type) to overcome many of their symptoms. Previous studies by Lubar and Lubar (1984) have shown that neurofeedback training to increase sensorimotor rhythm (SMR) over the central cortex (12-15 Hz) while reducing theta activity or increasing beta activity resulted in improved school performance. In our earliest study of the effect of neurofeedback training (Lubar & Shouse, 1976; Shouse & Lubar, 1979) a small group of children were trained in a blind crossover paradigm first to increase rhythm (SMR), then to decrease it, and then to increase it again. In this study, medication was removed in a stepwise fashion at the end of training and the gains in classroom performance as well as EEG changes were maintained.

One of the questions raised by the current study as well as by the Matochik et al. study (1994) revolves around the effect of Ritalin on central nervous mechanisms underlying ADD/HD. A recent study by Swartwood (1994) included a larger group of children than described in the current study. Methylphenidate had little effect on the

electrophysiological parameters measured, including percentage of EEG theta, percentage of EEG beta, and visual ERPs. This finding is consistent with Matochik's finding of no significant effect on cerebral metabolism in 63 out of 64 locations. Nevertheless, methylphenidate can profoundly improve hyperactive behavior and in some cases significantly improve academic performance on a short term basis. Long term effects on certain academic or cognitive tests such as reading are much more limited (Barkley & Cunningham, 1978). Perhaps methylphenidate's primary effect is to increase the effect of sensory input on brainstem mechanisms so as to reduce stimulus seeking behaviors. One way this could be tested is to look at the effects of methylphenidate on brainstem auditory and visual evoked potentials. If methylphenidate affects input at the brainstem level, this may provide an explanation for understanding how stimulus seeking behavior is decreased by the medication. Clearly, the effect of methylphenidate is limited at the cortical level, although subcortical functioning may be affected.

The limited neurophysiological effects of methylphenidate are one rationale for using neurofeedback to change cortical and even executive processing and one of the reasons why neurofeedback may have a long-term carryover effect, even after treatment is completed. In a recently published study (Lubar, 1995), data on 51 cases followed for up to 10 years were evaluated using the Conners (1969) rating scale illustrating the long term carryover of gains experienced during neurofeedback training. The results of the current studies should not be interpreted to indicate that methylphenidate is not effective as a treatment for ADD/HD symptomatology, but the results do lend support to the notion that its effects on cortical functioning are limited. Neurofeedback, when integrated with medication, can lead to a much more profound and long lasting effect than either approach used in isolation.

Finally, it must be emphasized that there is no cure for Attention Deficit Disorder with or without Hyperactivity at the present time and that at the very best a multi-modality treatment properly applied with long term follow-up can lead to significant symptom reduction and amelioration of many of the academic and adjustment problems. However, future research should focus on investigating the cerebral mechanisms involved in this complex disorder.

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