# Assessing Attention Deficit Hyperactivity Disorder via Quantitative Electroencephalography: An Initial Validation Study

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Spectral analysis of the electrophysiological output at a single, midline prefrontal location (the vertex) was conducted in 482 individuals, ages 6–30 years old, to test the hypothesis that cortical slowing in the prefrontal region can serve as a basis for differentiating patients with attention deficit hyperactivity disorder (ADHD) from nonclinical control groups. Participants were classified into 3 groups (ADHD, inattentive; ADHD, combined; and control) on the basis of the results of a standardized clinical interview, behavioral rating scales, and a continuous performance test. Quantitative electroencephalographic (QEEG) findings indicated significant maturational effects in cortical arousal in the prefrontal cortex as well as evidence of cortical slowing in both ADHD groups, regardless of age or sex. Sensitivity of the QEEG-derived attentional index was 86%; specificity was 98%. These findings constituted a positive initial test of a QEEG-based neurometric test for use in the assessment of ADHD.

Attention deficit hyperactivity disorder (ADHD) is a psychiatric disorder that has been historically characterized by the behavioral symptoms of inattention, impulsivity, and hyperactivity (American Psychiatric Association, 1980, 1987, 1994). Estimates of the prevalence of this disorder range from 5% to 15% of the school-age population (American Psychiatric Association, 1994; Barkley, 1990; Rie & Rie, 1980), occurring more commonly in boys than girls (ratios range from 4:1 to 9:1). Onset typically occurs prior to age 7. The condition is a relatively enduring one (Barkley, 1997a), and children diagnosed with ADHD are at increased risk for emergence of comorbid psychiatric disorders, as reported by

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Correspondence concerning this article should be addressed to Vincent J. Monastra, Clinical Director, The Family Psychology Institute, 2102 East Main Street, Endicott, New York 13760. Biederman et al. (1990), Mannuzza and colleagues (Mannuzza, Klein, Bessler, Malloy, & LaPadula, 1993; Mannuzza et al., 1991), and Morrison (1980).

Consistent with the behavioral formulation of ADHD, psychometric procedures initially focused on assessing this disorder from a behavioral perspective. Behavioral rating scales, such as the Child Behavior Checklist (Achenbach & Edelbrock, 1983), the Conners' rating scales (Conners, 1973), the ADHD Rating Scale (DuPaul, 1991), and the Attention Deficit Disorder Evaluation Scale (McCarney, 1989), were developed and provided a database for comparing the behavioral observations of parents and teachers with normative populations. Similarly, performance tests measuring capacity for vigilance and impulse control during visual and auditory tracking tasks (continuous performance tests) were developed (Conners, 1994; Gordon, 1983; Greenberg, 1994; Sanford, 1994), providing a more objective measure of the core symptoms of inattention and impulsivity. As Barkley (1990), Trommer, Hoeppner, Lorber, & Armstrong (1988), and Cantwell (1996) concluded, these measures are useful in the assessment process, particularly when combined with a thorough review of medical, developmental and family histories and an examination of intellectual functions and academic achievement. However, as Barkley, Trommer et al., and Cantwell noted, these tests cannot be considered diagnostic for ADHD because of the rater bias associated with rating scales and the high false-negative rate reported with continuous performance tests. In order to improve

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diagnostic accuracy, the development of additional assessment procedures appeared necessary.

As reviewed by Barkley (1997b), the primary deficits associated with ADHD support a hypothesis that anatomical and biochemical abnormalities of the prefrontal cortex constitute the physical basis of this disorder. Physical examination of this cortical region has been conducted with neurodiagnostic procedures (e.g., positron emission tomography [PET] and single photon emission tomography [SPECT]). The results of these examinations have provided evidence of hypoperfusion and low metabolic activity in the prefrontal and caudate nuclei regions (Amen, Paldi, & Thisted, 1993; Lou, Henriksen, & Bruhn, 1984; Zametkin et al., 1990; Zametkin & Rapoport, 1987). In addition, neuroimaging procedures (e.g., magnetic resonance imaging [MRI]) have revealed anatomical differences in the caudate nucleus (Casey et al., 1997; Hynd et al., 1993) and corpus callosum (Hynd et al., 1991). Overall, as noted by Casey et al. (1997), these studies have provided clear evidence of the importance of the frontostriatal circuitry (specifically, in the right hemisphere) in understanding the neurological basis of ADHD.

Three types of research initiatives, stimulated by the results of these neurological studies, emerged in an effort to improve diagnostic accuracy. Each research initiative examined procedures that assess the functional performance or electrophysiological activity of the frontal lobes. These research efforts have included neuropsychological studies assessing the performance of individuals with ADHD on tests associated with frontal lobe functions (reviewed by Barkley, Grodzinsky, & DuPaul, 1992), quantitative electroencephalographic (QEEG) studies examining event related potentials in individuals with ADHD (e.g., Kuperman, Johnson, Arndt, Lindgren, & Wolraich, 1996; Linden, Gevirtz, Isenhart, & Fisher, 1996; Loiselle, Stamm, Maitinisky, & Whipple, 1980; Satterfield, Schell, Nicholas, Satterfield, & Freese, 1990), and QEEG studies using computerized power spectral analysis (PSA) to study patterns of cortical activation (e.g., Capute, Niedermeyer, & Richardson, 1968; Chabot, Merkin, Wood, Davenport, & Serfontein, 1996; Klinkerfuss, Lange, Weinberg, & O'Leary, 1965; Lubar, 1991; Mann, Lubar, Zimmerman, Miller, & Muenchen, 1992).

The present study proceeded from an examination of the QEEG studies conducted using PSA. Such procedures involve the collection of multiple, short periods of digitized electroencephalographic (EEG), which are subjected to a fast Fourier transformation (FFT) algorithm (Cooley & Tukey, 1965). The FFT-derived data are then averaged over all trials for a given experimental condition. The overall electrophysiological power (pW) can then be determined and compared for various frequency bands at each active electrode site. Common frequency bands investigated by researchers have included delta (0.1 to 4 Hz), theta (4 to 8 Hz), alpha (8 to 12 Hz), sensorimotor rhythm (12 to 16 Hz), and beta (16 to 20 Hz).

Systematic, multisite spectral analysis studies comparing QEEG data of patients with ADHD and nonclinical controls have revealed certain cortical locations that differentiated

the EEG protocols of ADHD versus control groups. Mann et al. (1992) showed significant increases in slow-wave activity (4.00 to 7.75 Hz) in prefrontal, midline regions, with decreased posterior beta activity (12.75 to 21.00 Hz) when EEG recordings were obtained during academic challenges. Similarly, Janzen, Graap, Stephanson, Marshall, and Fitzsimmons (1995) noted increased theta activity in frontal, central, and posterior regions. Lubar (1995; Lubar, Swartwood, Swartwood, & Timmermann, 1996) examined the relationship between ADHD and a ratio derived by dividing the electrophysiological output (pW) produced in a frequency band defined as 4 to 8 Hz by the output produced in frequencies from 13 to 21 Hz. This theta-beta power ratio was calculated as individuals completed the following tasks: eyes open baseline, eyes closed baseline, reading silently, completing visuomotor tasks, and listening. Lubar and his colleagues hypothesized that evidence of excessive cortical slowing (i.e., a higher ratio of slow-wave activity relative to fast EEG activity) would be noted in individuals with ADHD. Their findings supported this hypothesis. Significant group differences were noted in the theta-beta power ratios obtained at multiple cortical sites, with CZ and FZ appearing the most promising for consideration in the development of an assessment procedure on the basis of spectral analysis.

Chabot and Serfontein (1996) expanded this research in their examination of 310 "normal" and 407 attention deficit disorder-attention deficit hyperactivity disorder patients. Initially, by using a discriminant function analysis of multiple QEEG characteristics, they correctly identified approximately 95% of the normal and 93% of the ADD-ADHD patients. In their subsequent study, Chabot et al. (1996) sought to examine the sensitivity and specificity of their procedure in an examination of 407 children with attentional disorders and 242 children with learning disorders. Similar to their earlier findings, Chabot et al. (1996) reported 93% correct classification of the children with ADHD and 90% of children with learning disorders when a discriminant function analysis of nine QEEG measures was conducted.

On the basis of the previous QEEG studies that used power spectral analysis, our research team sought to develop and test a simplified neurometric procedure for use in the assessment of ADHD. Prior findings (Lubar, 1995; Lubar et al., 1996) have indicated that the highest degree of differentiation between ADHD and non-ADHD participants was noted at the vertex; thus, CZ was selected for placement of the active electrode. Because critiques of prior QEEG studies (Levy, 1994) noted that the statistical differences between groups could have occurred as a function of multiple statistical comparisons, only one active site was used. Previous studies have indicated differentiation between groups when participants were involved in scholastic tasks (e.g., reading, listening, drawing), and difficulty sustaining attention during completion of these types of tasks frequently results in referral of children for evaluation; therefore, QEEG recordings were obtained while children completed reading, listening, and drawing tasks.

In order to minimize experimenter bias, evaluations were conducted by members of our research team at eight independent locations. To reduce error due to low interrater reliability rates for ADHD (reviewed by Barkley, 1990), classification as ADHD or non-ADHD was based on data derived from a combination of a structured clinical interview, a behavioral rating scale, and a continuous performance test. Because the most commonly used behavioral rating scales provide standard scores for inattentive or combined hyperactive-impulsive symptoms, only two of the ADHD subtypes (i.e., ADHD, inattentive, and ADHD, combined) were examined. Predominately hyperactive or impulsive types of ADHD patients who did not test positive for inattention were not examined in this study because of our effort to reduce classification error. A continuous performance test was added to the screening process used by Mann et al. (1992) and Chabot et al. (1996) because such procedures have been shown to reflect frontal lobe functioning by SPECT (Rezai et al., 1993), are useful in identifying individuals with attentional deficits, and have been associated with low false-positive rates for ADHD (Greenberg, 1994).

Given the findings of previous QEEG studies, we hypothesized that significant differences in the theta-beta power ratios would be noted, with patients diagnosed with ADHD exhibiting higher ratios than nonclinical controls. In order to initially test the classification accuracy of a neurometric test based on the theta-beta power ratio, critical values of the power ratio were to be calculated on the basis of the mean and standard deviation of the control groups. It was hypothesized that classification of participants into ADHD and non-ADHD groups could be made on the basis of these critical values, given the location of our QEEG recording site and the neuroanatomical and biochemical research data supporting the role of the prefrontal cortex in ADHD. Our goal was to conduct the initial validation study involving a specific neurometric indicator of cortical slowing, in order to begin the process of developing an inexpensive, nonintrusive electrophysiological measure of frontal lobe functioning that could contribute to the existing assessment procedures for the diagnosis of ADHD.

## Method

#### **Participants**

Four hundred and eighty-two individuals were evaluated using behavioral rating scales, continuous performance tests, computerized PSA of QEEG recordings, and structured clinical interviews. Two hundred and seven of the participants were girls, female adolescents, and women; 275 were boys, male adolescents, and men. In an effort to minimize experimenter bias and obtain data from multiple geographic regions, eight research centers in the following states participated in the project: New York, Georgia, Ohio, Tennessee, Missouri, Nevada, and California. The geographic distribution of participants was as follows: Eastern Region (New York) = 24%, Central Region (Ohio, Tennessee, Missouri) = 29%, Southern Region (Georgia) = 36%, and Western Region (California, Nevada) = 11%. Participants were recruited by correspondence with schools, physicians, and mental health professionals located near each of the participating research centers, as well as through newspaper solicitation.

Because of the importance of establishing clinical and control

groups free of other neurological conditions, caretakers completed Barkley's (1991a) ADHD Clinical Parent Interview for participants aged 6 to 20 years. Participants aged 21 to 30 years completed the adult version of this structured interview. Detailed information regarding medical and developmental history was obtained through this interview. Individuals with other neurological disorders (e.g., epilepsy, autism) were not included in this study. All participants were under the care of physicians. None reported treatment for any neurological condition.

To control for medication effects, none of the members of the control group were evaluated while using any medication. For those participants being treated with Ritalin, testing was completed after a medication-free period of at least 12 hr. Given the clinical action of this medication as well as published research (Lubar et al., 1996) that has indicated no effect of stimulant medication on the QEEG recordings obtained from 19 sites (including CZ), we considered our clinical groups to be medication free as well. Evaluations were conducted between the hours of 9:00 a.m. and 3:00 p.m.

Classification of individuals into clinical and nonclinical groups was accomplished through a screening procedure that included Barkley's ADHD Clinical Parent Interview (or Adult Interview; Barkley, 1991a), behavioral rating scales (Attention Deficit Disorders Evaluation Scale [ADDES; McCarney, 1989], ADD-H: Comprehensive Teacher's Rating Scale [Ulmann, Sleator, & Sprague, 1984], or other ADHD rating scales), and a continuous performance test (Conners' Continuous Performance Test [Conners, 1994], Gordon Diagnostic System [Gordon, 1983], Test of Variables of Attention Continuous Performance Test [Greenberg, 1994], and Intermediate Visual and Auditory Continuous Performance Test [Sanford, 1994]). To be placed in one of the clinical groups, participants had to meet the Diagnostic and Statistical Manual of Mental Disorders (4th ed.; DSM-IV; American Psychiatric Association, 1994) criteria for ADHD on the basis of the report of the referral source and had to test positive for ADHD on both behavioral and continuous performance test measures. The specific criteria for group placement were as follows: For ADHD, inattentive type (ADHD-I), participants had to meet DSM-IV criteria according to a referring source (school, physician), meet DSM-IV criteria according to caretaker or self-report on the Barkley Interview, obtain a positive score for inattention on the ADDES or other ADHD rating scale and score in the nonclinical range on the Impulsive and Hyperactive scales, and obtain a positive overall rating for ADHD on a continuous performance test. For ADHD, hyperactive-combined type (ADHD-H/C), participants had to meet the same first two requirements as for ADHD, inattentive type, to obtain a positive score for impulsivity or hyperactivity on the ADDES or other rating scale, and to obtain a positive overall rating for ADHD on a continuous performance test. Finally, for nonclinical controls, participants did not meet DSM-IV criteria for any psychiatric disorder on the basis of caretaker or self-report on the Barkley Interview, their caretaker or self-report scores on the ADDES or other ADHD rating scale were in the nonclinical range for inattention, impulsivity, and hyperactivity, and their continuous performance test overall ratings were negative for ADHD. Distribution of participants by age and diagnosis is presented in Table 1.

# Materials

QEEG recordings were obtained using Autogenics A-620 Electroencephalograph (Wood Dale, IL) with associated Assessment Software (Wood Dale, IL) for computerized analysis of EEG data. This system provides researchers with a quantitative analysis of electrophysiological recordings in multiple frequency bands. For

Table 1Distribution of Participants by Age and Diagnosis

Diagnosis	Age (years)			
	611	12–15	16-20	21-30
ADHD-I	64	48	51	13
ADHD-H/C	149	43	21	8
Control	30	34	10	11
Total	243	125	82	32

*Note.* ADHD-I = attention deficit hyperactivity disorder, inattentive; ADHD-H/C = attention deficit hyperactivity disorder, hyperactive–combined type.

the purpose of this study, 4-8 Hz defined the theta band, and 13-21 Hz defined the beta band. Similar to other PSA studies, multiple short periods (90 s) of digitized EEG were obtained. An FFT algorithm was computed by the A-620 Assessment System and averaged over four trials. The overall electrophysiological power (pW) was computed for the theta and beta bands by the A-620 Assessment System and then manually entered into the statistical program Statistica (StatSoft, 1995) for data analysis and graphic presentation.

## Procedure

Participants meeting selection criteria for involvement in this study were evaluated using the following QEEG procedure:

1. The vertex (CZ) was located using the International 10–20 System of electrode placement (Andreassi, 1989).

2. The area was cleaned using Omni prep (or equivalent) and isopropyl alcohol. A small amount of conductive paste (e.g., Ten20) was applied to the scalp and to a Grass Gold Disc Electrode (Astro-Med, Inc., West Warwick, RI) with hole (E5GH), and the sensor was attached to the scalp. A similar cleaning procedure was used for preparing the earlobes and one pair of Gold Disc Electrodes in Ear Clip (Grass E34D) was attached to each earlobe. Quality of preparation was assessed by way of an Autogenics Electrode Tester (Wood Dale, IL). Impedance readings were to be below 10K $\Omega$ . Offset potential was to be below 10  $\mu$ V before recordings were obtained.

3. Band frequencies were defined on the Assessment Software with 4-8 Hz defining theta and 13-21 Hz defining beta. Once the sensors were tested and band frequencies defined, the participant's EEG activity at CZ was recorded during four tasks. The first task was eyes fixed-baseline. The child or adult was seated in front of the computer monitor display and instructed to focus his or her gaze on the monitor's "on/off" indicator light. EEG recordings were obtained for 90 s. After the task was completed, the EEG record was reviewed in 2-s intervals (epochs), in order to manually filter out epochs containing excessive electromyograph (EMG) artifact (e.g., body movement, eye rolls or blinks). A minimum of 15 low-artifact epochs (i.e., no evidence of eye rolls or blinks and overall EMG output below 15 µV) was required for completion of this assessment task. The next 90-s task was reading. Material that was age or grade appropriate was selected (e.g., school reading texts, and reading tasks from the Kaufman Test of Educational Achievement (Kaufman & Kaufman, 1985), the Peabody Individual Achievement Test (Dunn & Markwardt, 1970), or other age-related reading tests) and read silently by the participant. Again, after completion of this task, the EEG was reviewed in 2-s intervals to eliminate epochs with excessive EMG activity or eye movement or blink artifact. A minimum of 15 low-artifact epochs was required for completion of this assessment task. A 90-s listening task occurred next. Age appropriate material was selected and read by the clinician (as described for the reading task). EEG review was conducted as with the first two tasks. The final task was drawing. A stable drawing surface was placed in front of the child or adult. He or she was instructed to copy geometric figures from one of the following tests: Beery Developmental Test of Visual-Motor Integration (Beery & Buktenica, 1967), Benton Visual Retention (Benton, 1955), or McCarthy Scales of Children's Abilities (McCarthy, 1972). EEG was recorded for 90 s, with review as with the previous tasks.

#### Results

# Cortical Slowing and ADHD

The initial statistical analyses were conducted in order to test the hypothesis that participants identified with ADHD (either inattentive or combined types) would display significantly higher levels of slow-wave (i.e., theta, 4–8 Hz) relative to fast-wave EEG activity (i.e., beta, 13–21 Hz). The calculation of these theta-beta power ratios was performed by the A-620 Assessment Software for each participant on each of four tasks. The resulting ratio data was then transferred to StatSoft's Statistica program for statistical analysis and graphic presentation of data.

The planned statistical analysis consisted of an analysis of variance (ANOVA) with repeated measurement of the theta-beta ratio during four tasks (baseline, reading, listening, and drawing). Between-subject comparisons were made to examine the effects of age and diagnosis on the theta-beta power ratio. Within-subject comparisons were studied in order to evaluate task effects. Tukey's honest significant difference (HSD) test was selected for post hoc testing of significant main or interactional effects. An alpha level of .01 was used for all statistical tests.

A summary of the ANOVA analysis of all effects is provided in Table 2. Consistent with our hypothesis, statistical analysis revealed that theta-beta power ratios were significantly affected by age and diagnosis (p < .001). In addition, data analysis indicated that the power ratio was affected by type of task (p < .001). There was no evidence that the degree of cortical slowing was related to the sex of the participant (Rao's R = .646, p = .63). Similarly, there was no indication that the effects of age, diagnosis, or task were confounded by the sex of the participant.

Post hoc comparisons of the main effects (age and diagnosis) consisted of examination of the theta-beta power ratios on each of the four tasks. Consistent with maturational models of cortical development, the level of cortical slowing noted in our PSA study was highest in the youngest age

Table 2

Summary of all Analyses of Variance Effects

Effect	dfs	F	р
Diagnosis	2, 580	29.47	<.001
Age	3, 580	28.95	<.001
Task	3, 1740	7.87	<.001
Diagnosis $\times$ Age	6, 580	2.96	<.001
Diagnosis $\times$ Task	6, 1740	1.24	<.280
$Age \times Task$	9, 1740	0.86	<.560
Diagnosis $ imes$ Age $ imes$ Task	18, 1740	1.09	<.360

Table	3
Mean	Theta-Beta Power Ratios

Age (years)		Diagnosis	
	ADHD-I	ADHD-H/C	Control
6-11	8.485	7.698	3.027
12-15	4.494	5.547	2.059
16-20	3.617	4.188	1.999
21-30	2.454	4.125	1.495

Note. ADHD-I = attention deficit hyperactivity disorder, inattentive; ADHD-H/C = attention deficit hyperactivity disorder, hyperactive–combined type.

group (ages 6–11 years old). When compared with each of the other age groups, the participants aged 6–11 years had significantly higher theta-beta ratios (Tukey HSD tests, p < .001) on each of the four tasks. Although continued reduction in the theta-beta ratios was associated with increased age, as reflected in Table 3 and Figures 1, 2, 3, and 4, post hoc comparison of these age effects was not significant. The primary improvement in the level of cortical arousal was apparent by ages 12–15 years old and persisted through the age of 30 years old.

Post hoc analysis of the effect of diagnostic classification revealed a consistent pattern of differentiation of both ADHD groups from the nonclinical control group on all tasks. Examination of between-group differences using Tukey's HSD test revealed statistically significant differentiation between both of the ADHD groups and the control group on the baseline (p < .001), reading (p < .001), listening (p < .001), and drawing (p < .001) tasks. Withinsubject comparisons across task revealed that individuals classified as either ADHD, inattentive or ADHD, combined type showed significantly higher power ratios on the drawing task relative to their ratios on the other tasks (p < .01). No such pattern was observed in participants from the nonclinical control group. Although differentiation between the two ADHD groups was suggested by the graphic depiction of the mean theta-beta ratio data (see Figures 1-4), statistically significant differences were noted only on the drawing task. On the drawing task, mean power ratios for the ADHD, combined group were significantly greater than those demonstrated by the ADHD, inattentive group (p < .01).

# The Theta-Beta Power Ratio as a Test for ADHD

The second hypothesis of this study was that critical values derived from the means and standard deviations of the theta-beta power ratio of the control groups could serve as a basis for differentiating participants with ADHD from nonclinical control participants. In order to define critical values for ADHD, the mean theta-beta ratio was first calculated for each of the four control groups, collapsing across all tasks. Critical values for ADHD were defined as 1.0, 1.5, and 2.0 SDs above the mean for each of the control groups. A summary table of these critical values is provided in Table 4.

After calculating critical values for ADHD, an overall power ratio score was derived for each participant. This ratio score was obtained by averaging the theta-beta power ratios for each participant on the four tasks. Participants were classified as ADHD or non-ADHD on the basis of the power ratio alone by using cutoffs of 1.0, 1.5, and 2.0 SDs from the mean of each of the nonclinical control groups. Because the goal of this initial study was to examine whether an attentional index derived from QEEG data (i.e., the thetabeta power ratio averaged over four tasks) could differentiate individuals with ADHD from nonclinical controls, accurate classification was considered to occur when the theta-beta ratio score was in agreement with classification as ADHD or non-ADHD on the basis of behavioral rating



*Figure 1.* Plot of the mean theta-beta power ratios for the two-way interaction, Age (Years)  $\times$  Diagnosis, during the eyes-fixed baseline task. DX = diagnosis; ADHD(I) = attention deficit hyperactivity disorder, inattentive type; ADHD(C) = attention deficit hyperactivity disorder, hyperactive-combined type.



*Figure 2.* Plot of the mean theta-beta power ratios for the two-way interaction, Age (Years)  $\times$  Diagnosis, during the reading task. DX = diagnosis; ADHD(I) = attention deficit hyperactivity disorder, inattentive type; ADHD(C) = attention deficit hyperactivity disorder, hyperactive-combined type.

scales and continuous performance tests. False-positive classification occurred when the theta-beta ratio score indicated ADHD in a participant classified as non-ADHD on the behavioral rating scales and continuous performance tests. False-negative classification occurred when the theta-beta ratio score indicated non-ADHD in a participant classified as ADHD in the screening process. A summary of the accuracy rates is provided in Table 5.

Examination of the accuracy rates provided in Table 5 reveals a high degree of consistency between classification derived from our index of cortical slowing and those obtained through behavioral rating scales and continuous performance tests. When 1 *SD* above the mean for control

groups was used as a critical value, the rate of diagnostic agreement was above 85% for each group (M = 88%). At 1.5 SDs, the agreement rate ranged from 81% to 91% (M = 84%). At 2.0 SDs, the agreement rate dropped to 76%, with 23% of the errors resulting from false-negative ratings.

Additional analysis of classification accuracy was conducted in order to examine the sensitivity and specificity of the QEEG-derived attentional index. In this analysis, a participant whose attentional index was 1.5 SDs greater than the mean of the age appropriate nonclinical control group was considered positive for ADHD. Examination of the percentage of participants classified with either type of ADHD who tested positive on the QEEG revealed a



*Figure 3.* Plot of the mean theta-beta power ratios for the two-way interaction, Age (Years)  $\times$  Diagnosis, during the listening task. DX = diagnosis; ADHD(I) = attention deficit hyperactivity disorder, inattentive type; ADHD(C) = attention deficit hyperactivity disorder, hyperactive-combined type.



*Figure 4.* Plot of the mean theta-beta power ratios for the two-way interaction, Age (Years)  $\times$  Diagnosis, during the drawing task. DX = diagnosis; ADHD(I) = attention deficit hyperactivity disorder, inattentive type; ADHD(C) = attention deficit hyperactivity disorder, hyperactive-combined type.

sensitivity rating of 86%. Specificity of the QEEG measure (i.e., the percentage of non-ADHD participants testing negative for ADHD) was 98%. The overall positive predictive power of the measure was 99%, meaning that only 1% of the individuals who tested positive on the measure did not have ADHD. Consequently, the results of our evaluation of test sensitivity and specificity were considered supportive of the use of the theta-beta power ratio in assessing ADHD.

#### Discussion

The essential findings of this study were as follows. First, a significant association was noted between age and a neurometric indicator of cortical slowing (the theta-beta power ratio obtained at the vertex using a referential montage). Second, scores on this indicator were significantly higher in patients with attention deficit disorders (both ADHD-I and ADHD-H/C) than nonclinical controls for ages 6 through 30 years old. Third, critical values derived from the neurometric scores of the nonclinical controls could serve as a basis for accurate classification of the participants of the study. Fourth, this indicator of cortical slowing yielded similar accuracy rates, regardless of the sex of the participant.

In summary, these findings provide initial guidelines for

Table 4Critical Values for Attention Deficit Hyperactivity Disorderon the Basis of Power Ratios

		SD	
Age (years)	1.0	1.5	2.0
6-11	4.36	5.03	5.69
12-15	2.89	3.31	3.72
16-20	2.24	2.36	2.48
21-30	1.92	2.13	2.34

clinical researchers seeking to examine the validity of a simplified QEEG indicator as a laboratory test for ADHD. The present study clarified certain electrophysiological parameters and assessment procedures that can be used to accurately classify ADHD patients and nonclinical controls. The level of accuracy obtained using our neurometric indicator was similar to that presented by the developers of behavioral and continuous performance tests for ADHD. In addition, the present findings yielded levels of accuracy similar to those reported by researchers using discriminant function analysis of multichannel EEG recordings.

These findings are consistent with the results of neurological assessment procedures (PET, SPECT, MRI), as well as emerging neuropsychologically based models associating ADHD with prefrontal cortical functioning (Barkley, 1997b). In addition, our findings, similar to those presented by Mann et al. (1992), Lubar (1995; Lubar et al., 1996), and Chabot and his associates (Chabot et al., 1996; Chabot & Serfontein, 1996), are supportive of the development of QEEG-based assessment procedures for evaluating ADHD. Because the preponderance of neurological, biochemical, and electrophysiological research has supported the conclusion that ADHD is a health impairment, it appears imperative that assessment procedures be developed to assess the physical

Table 5Accuracy of Classification Using theTheta-Beta Power Ratio

Criterion			
	Correct (%)	False+ (%)	False- (%)
1.0 SD	88	3	9
1.5 SD	84	2	14
2.0 SD	76	1	23

*Note.* False+ = false-positive classification; False- = false-negative classification.

as well as the neuropsychological and behavioral symptoms of this disorder.

Because QEEG procedures are relatively nonintrusive, inexpensive, and can provide information about cortical processes that are difficult to obtain from neuroimaging scans (e.g., degree of coherence and symmetry in activity between different cortical regions), their application in developing an understanding of ADHD appears promising. QEEG researchers like Mann et al. (1992), Lubar (1995; Lubar et al., 1996) and Chabot and his colleagues (Chabot et al., 1996; Chabot & Serfontein, 1996) have shown that multichannel EEG recordings and an examination of QEEG characteristics, such as electrophysiological power, power ratios, coherence, and symmetry, can be useful in differentiating individuals with ADHD from nonclinical controls and from peers with learning disorders. Our study sought to examine the sensitivity and specificity of a QEEG scan for ADHD on the basis of the electrophysiological output from a single channel recording at the vertex.

Similar to the findings of Mann et al. (1992), Lubar (1995; Lubar et al., 1996), and Chabot and Serfontein (1996), the results of our study provided further evidence of cortical slowing in participants with ADHD. Mann et al. examined electrophysiological power from 19 sites and concluded that participants with ADHD exhibited higher theta (4.00-7.75 Hz) activity at several frontal and central locations. Lubar (1995; Lubar et al., 1996) reported significantly higher theta-beta power ratios at several central and frontal locations (including the vertex). Chabot and Serfontein reported two neurophysiological subtypes for ADHD; one type was characterized by theta-alpha excess (with normal alpha mean frequency), and the other type by theta-alpha excess coupled with decreased alpha mean frequency. Again, the primary locations of interest were within frontal and central locations. Our finding of significantly higher theta-beta power ratios at the vertex and high rates of classification accuracy using this neurometric is consistent with these findings and supports further examination of a simplified scanning procedure for ADHD.

The current findings provide a first step in the identification of a neurometric test for ADHD that is far less intrusive and expensive than other procedures. Given our results, we hypothesize that the use of such an indicator, in conjunction with behavioral and continuous performance test measures, will serve to increase overall diagnostic accuracy by reducing error rates associated with nonneurologically based conditions with similar behavioral symptoms. In order to continue the process of test development, a series of studies is required.

First, to ensure valid comparisons across clinical research centers, standardization of the assessment process is required. This will necessitate the development of software programs for stimuli presentation, as well as neurometric data processing. Second, issues of test-retest reliability need to be addressed. Third, examination of the ability of the neurometric assessment process developed in this study to accurately classify participants not involved in our initial standardization study is needed for cross-validation purposes. Finally, in order to assess test specificity, examination of the accuracy of this indicator to differentiate conditions such as oppositional defiant disorder or affective disorders from ADHD is required.

Comparisons with behavioral and cognitive tests (continuous performance tests) developed to assess ADHD likewise seem desirable in order to examine the issue of construct validity. Although both types of measures were obtained in our current study, they were used for classification purposes. Consequently, comparisons between behavioral, cognitive, and neurometric measures were not made during this study because the degree of correlation would be artificially inflated. However, such comparisons are planned in our ongoing research. Our goal remains not to supplant behavioral or cognitive measures but to add a neurometric laboratory test to aid in the diagnostic process.

Two additional research directions are derived from what was not demonstrated in our study. Specifically, we are aware that the current findings did not reveal significant differences between the subtypes of ADHD on any task other than drawing and only examined individuals aged 6 to 30 years. Several modifications in our approach to assessment for subtypes and patients above the age of 30 years old are planned.

In order to attempt differentiation of subtypes, analysis of the sensorimotor rhythm (12-15 Hz) is planned. Sensorimotor rhythm represents inhibitory activity generated in pathways originating in the cerebellum and terminating on motor neurons in the sensorimotor cortex (Sterman, 1996). Because sensorimotor rhythm training (Lubar, 1995) has yielded positive results in the treatment of two primary clinical features of ADHD-H/C (impulsivity and hyperactivity), examination of this frequency band may prove useful in differentiating ADHD-H/C from ADHD-I subtypes. Likewise, because patients with ADHD, hyperactive or combined type, show multiple indicators of impaired motor control (both in lack of motor inhibition and frequently in impaired handwriting ability), replication of the current PSA procedure during performance of graphomotor tasks would provide an indication of the consistency of present findings across samples and contribute to an understanding of certain of the neuropsychological differences between subtypes.

Finally, in order to identify neurometric indicators for ADHD in individuals beyond age 30 years old, improved methods for initial classification seem required. Specifically, behavioral assessment procedures for individuals over the age of 18 years old have typically relied exclusively on self-report. As indicated by Barkley (1997a), the self-report of individuals with ADHD may underestimate symptom severity. Consequently, the inclusion of ratings provided by relatives (e.g., using the ADDES, Adult Version; McCarney, 1996) and an examination of a large sample size of adults may prove useful in determining whether our neurometric index of cortical slowing will continue to differentiate persons with ADHD through adulthood. In addition, expansion of our neurometric examination to include other slow-wave frequencies (e.g., 6–10 Hz) is planned.

#### References

- Achenbach, R. M., & Edelbrock, C. S. (1983). Manual for the Child Behavior Checklist and Revised Child Behavior Profile. Burlington: University of Vermont, Department of Psychiatry.
- 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.
- American Psychiatric Association. (1980). Diagnostic and statistical manual of mental disorders (3rd ed.). Washington, DC: Author.
- American Psychiatric Association. (1987). *Diagnostic and statistical manual of mental disorders* (3rd ed., rev.). Washington, DC: Author.
- American Psychiatric Association. (1994). Diagnostic and statistical manual of mental disorders (4th ed.). Washington, DC: Author.
- Andreassi, J. (1989). Psychophysiology, human behavior and physiological response. New York: Erlbaum.
- Barkley, R. A. (1990). Attention-deficit hyperactivity disorder: A handbook for diagnosis and treatment. New York: Guilford Press.
- Barkley, R. A. (1991a). ADHD Clinical Interview (Parent or Adult Form). In R. A. Barkley (Ed.), Attention-deficit hyperactivity disorder: A clinical workbook (pp. 5–43). New York: Guilford Press.
- Barkley, R. A. (1991b). The ecological validity of laboratory and analogue assessment methods of ADHD symptoms. *Journal of Abnormal Child Psychology*, 19, 149–178.
- Barkley, R. A. (1997a). Age dependent decline in ADHD: True recovery or statistical illusion? *The ADHD Report*, 5 (1), 1–5.
- Barkley, R. A. (1997b). Behavioral inhibition, sustained attention, and executive functions: Constructing a unifying theory of ADHD. *Psychological Bulletin*, 121, 65–94.
- Barkley, R. A., Grodzinsky, G., & DuPaul, G. J. (1992). Frontal lobe functions in attention deficit disorder with and without hyperactivity: A review and research report. *Journal of Abnor*mal Child Psychology, 20, 163–188.
- Beery, K. E., & Buktenica, N. A. (1967). The Beery Developmental Test of Visual-Motor Integration. Columbus, OH: Modern Curriculum Press.
- Benton, A. L. (1955). *Visual Retention Test.* New York: The Psychological Corporation.
- Biederman, J., Faroane, S. V., Spencer, R., Wilens, R., Norman, D., Lapey, K. A., Mick, E., Krifcher-Lehman, B., & Doyle, A. (1990). Patterns of psychiatric comorbidity, cognition and psychosocial functioning in adults with attention deficit disorder, residual type. *Comprehensive Psychiatry*, 31, 416–425.
- Cantwell, D. P. (1996). Attention deficit disorder: A review of the past ten years. Journal of the American Academy of Child and Adolescent Psychiatry, 35, 978–987.
- Capute, A. J., Niedermeyer, E. F. L., & Richardson, F. (1968). The electroencephalogram in children with minimal brain dysfunction. *Pediatrics*, 4, 1104–1114.
- Casey, B. J., Castellanos, F. X., Giedd, J. N., Marsh, W. L., Hamburger, S. D., Schubert, A. B., Vauss, Y. C., Vaituzis, A. C., Dickstein, D. P., Sarfatti, S. E., & Rapoport, J. L. (1997). Implication of right frontostriatial circuitry in response inhibition and attention-deficit/hyperactivity disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, 36(3), 374–383.
- 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. (1973). Rating scales for use in drug studies with children. Psychopharmacology Bulletin, 9, 24–84.
- Conners, C. K. (1994). Conners' Continuous Performance Test. Toronto, Canada: Multi-Health Systems.
- Cooley, J. W., & Tukey, J. W. (1965). An algorithm for the machine calculation of complex Fourier series. *Mathematics of Computation*, 19, 267–301.
- Dunn, L. M., & Markwardt, F. C. (1970). The Peabody Individual Achievement Test. Circle Pines, MN: American Guidance Service.
- DuPaul, G. J. (1991). ADHD Rating Scale. In R. A. Barkley (Ed.), *Attention-deficit hyperactivity disorder: A clinical workbook* (pp. 46–48). New York: Guilford Press.
- Gordon, M. (1983). *The Gordon Diagnostic System*. Dewitt, NY: Gordon Systems.
- Greenberg, L. M. (1994). Test of Variables of Attention Continuous Performance Test. Los Alamitos, CA: Universal Attention Disorders.
- Hynd, G. W., Hern, K. L., Novey, E. S., Eliopulos, D., Marshall, R., Gonzalez, J. J., & Voeller, K. K. (1993). Attention deficithyperactivity disorder and asymmetry of the caudate nucleus. *Journal of Child Neurology*, 8, 339–347.
- 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.
- Kaufman, A. S., & Kaufman, N. L. (1985). The Kaufman Test of Educational Achievement. Circle Pines, MN: American Guidance Service.
- Klinkerfuss, G. H., Lange, P. H., Weinberg, W. A., & O'Leary, J. L. (1965). Electroencephalographic abnormalities of children with hyperkinetic behavior. *Neurology*, 15, 883–891.
- Kuperman, S., Johnson, B., Arndt, S., Lindgren, S., & Wolraich, M. (1996). Quantitative EEG differences in a nonclinical sample of children with ADHD and undifferentiated ADD. Journal of the American Academy of Child and Adolescent Psychiatry, 35, 1009–1016.
- Levy, F. (1994). Neurometrics: Review and comments. *The ADHD Report*, 2(5), 1–3.
- Linden, M., Gevirtz, R., Isenhart, R., & Fisher, T. (1996). Event related potentials of subgroups of children with attention deficit hyperactivity disorder and the implications for EEG biofeedback. *Journal of Neurotherapy*, *1*, 1–11.
- Loiselle, D. I., Stamm, J. S., Maitinisky, S., & Whipple, S. C. (1980). Evoked potential and behavioral signs of attentive dysfunctions in hyperactive boys. *Psychophysiology*, 17, 193– 201.
- Lou, H. C., Henriksen, L., & Bruhn, P. (1984). Focal cerebral hypoperfusion in children with dysphasia and/or attention deficit disorder. Archives of Neurology, 41, 825–829.
- Lubar, J. F. (1991). Discourse on the development of EEG diagnostics and biofeedback for attention-deficit/hyperactivity disorders. *Biofeedback & Self Regulation*, 16, 201–225.

- Lubar, J. F. (1995). Neurofeedback for the management of attention deficit hyperactivity disorders. In M. S. Schwartz (Ed.), *Biofeedback: A practitioners guide* (pp. 493–522). New York: Guilford Press.
- 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., & Muenchen, R. (1992). Quantitative analysis of EEG in boys with attentiondeficit-hyperactivity disorder: Controlled study with clinical implications. *Pediatric Neurology*, 8, 30–36.
- Mannuzza, S., Klein, R. G., Bessler, A., Malloy, P., & LaPadula, M. (1993). Adult outcome of hyperactive boys: Educational achievement, occupational rank and psychiatric status. Archives of General Psychiatry, 50, 565–576.
- Mannuzza, S., Klein, R. G., Bonagura, N., Malloy, P., Giampino, H., & Addalli, K. A. (1991). Hyperactive boys almost grown up: Replication of psychiatric status. Archives of General Psychiatry, 48, 77–83.
- McCarney, S. B. (1989). Attention Deficit Disorders Evaluation Scale. Columbia, MO: Hawthorne Press.
- McCarney, S. B. (1996). The Adult Attention Deficit Disorders Evaluation Scale. Columbia, MO: Hawthorne Press.
- McCarthy, D. (1972). *McCarthy Scales of Children's Abilities*. New York: The Psychological Corporation.
- Morrison, J. R. (1980). Childhood hyperactivity in an adult psychiatric population: Social factors. *Journal of Clinical Psychiatry*, 41, 40–43.
- Rezai, K., Andreasen, N. C., Alliger, R., Cohen, G., Swayze, V., & O'Leary, D. S. (1993). The neuropsychology of the prefrontal cortex. Archives of Neurology, 50, 636–642.

- Rie, H. E., & Rie, E. D. (Eds.). (1980). Handbook of minimal brain dysfunction: A critical view. New York: Wiley.
- Sanford, J. A. (1994). Intermediate Visual and Auditory Continuous Performance Test. Richmond, VA: BrainTrain.
- Satterfield, J. H., Schell, A. M., Nicholas, T. W., Satterfield, B. T., & Freese, T. E. (1990). Ontogeny of selective attention effects on event-related potentials in attention-deficit hyperactivity disorder and normal boys. *Biological Psychiatry*, 28, 879–903.

StatSoft. (1995). Statistica. Tulsa, Oklahoma: StatSoft.

- Sterman, M. B. (1996). Physiological origins and functional correlates of EEG rhythmic activities: Implications for selfregulation. *Biofeedback and Self-Regulation*, 21, 3-33.
- Trommer, B. L., Hoeppner, J. B., Lorber, R., & Armstrong, K. (1988). Pitfalls in the use of a continuous performance test as a diagnostic tool in attention deficit disorder. *Journal of Developmental and Behavioral Pediatrics*, 9, 339–345.
- Ulmann, R. K., Sleator, E. K., & Sprague, R. (1984). A new rating scale for diagnosis and monitoring of ADD children. *Psychophar*macology Bulletin, 20, 160–164.
- 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.
- Zametkin, A. J., & Rapoport, J. L. (1987). Noradrenergic hypothesis of attention deficit disorder with hyperactivity: A critical review. In H. V. Metsler (Ed.), *Psychopharmacology: The third* generation of progress (pp. 837–842). New York: Raven Press.

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