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.
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 (Ames, Paldi, & Thisted, 1993; Lou, Henriksen, & Bruhn, 1984; Zametkin et al., 1990; Zametkin & Rapoport, 1987). In addition, neuro-imaging 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 frontostral 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, Maitininsky, & 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, Niedermeier, & 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 function 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, noninvasive 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 (1991b) 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 [ADDERS; McCarney, 1989], ADD-H: Comprehensive Teacher’s Rating Scale [Ullman, Sleanor, & 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 ADDERS 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 ADDERS 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 ADDERS 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
listening task occurred next. Age appropriate material was selected
Again, after completion of this task, the EEG was reviewed in 2-s

Achievement (Kaufman & Kaufman, 1985), the Peabody Indi-
texts, and reading tasks from the Kaufman Test of Educational
was required for completion of this assessment task. A 90-s
movement or blink artifact. A minimum of 15 low-artifact epochs

record was reviewed in 2-s intervals (epochs), in order to manually
gaze on the monitor's "on/off' indicator light. EEG recordings
the computer monitor display and instructed to focus his or her

sensors were tested and band frequencies defined, the participant's
isopropyl alcohol. A small amount of conductive paste (e.g., Ten20)
software was defined on the Assessment Software
2. The area was cleaned using Omni prep (or equivalent) and

rati on each of the four tasks. Consistent with maturational
diagnosis) consisted of examination of the theta-beta power
was not confounded by the sex of the participant. 
addition, data analysis indicated that the power ratio was
significantly affected by age and diagnosis

ANOVA with repeated measurement of the
theta-beta ratio during four tasks (baseline, reading, listen-
ing, 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, statisti-
cal 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>
<thead>
<tr>
<th>Diagnosis</th>
<th>Age (years)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>6–11</td>
</tr>
<tr>
<td>ADHD-I</td>
<td>64</td>
</tr>
<tr>
<td>ADHD-H/C</td>
<td>149</td>
</tr>
<tr>
<td>Control</td>
<td>30</td>
</tr>
<tr>
<td>Total</td>
<td>243</td>
</tr>
</tbody>
</table>

Note. ADHD-I = attention deficit hyperactivity disorder, inatten-
tive; ADHD-H/C = attention deficit hyperactivity disorder, hyper-
active–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
andisopropyl 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
be below 10KQ. Offset potential was to be below 10 μV 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 Indi-
vidual 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, 1953), or McCarthy Scales of Children’s
Abilities (McCarthy, 1972). EEG was recorded for 90 s, with re-
view 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 signifi-
cantly 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, listen-
ing, 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
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Table 2
Summary of all Analyses of Variance Effects

<table>
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<tr>
<th>Effect</th>
<th>df</th>
<th>F</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diagnosis</td>
<td>2</td>
<td>580</td>
<td>29.47</td>
</tr>
<tr>
<td>Age</td>
<td>3</td>
<td>580</td>
<td>28.95</td>
</tr>
<tr>
<td>Task</td>
<td>3</td>
<td>1740</td>
<td>7.87</td>
</tr>
<tr>
<td>Diagnosis × Age</td>
<td>6</td>
<td>580</td>
<td>2.96</td>
</tr>
<tr>
<td>Diagnosis × Task</td>
<td>6</td>
<td>1740</td>
<td>1.24</td>
</tr>
<tr>
<td>Age × Task</td>
<td>9</td>
<td>1740</td>
<td>0.86</td>
</tr>
<tr>
<td>Diagnosis × Age × Task</td>
<td>18</td>
<td>1740</td>
<td>1.09</td>
</tr>
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</table>
Table 3

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>ADHD-I</td>
</tr>
<tr>
<td>6–11</td>
<td>8.485</td>
</tr>
<tr>
<td>12–15</td>
<td>4.494</td>
</tr>
<tr>
<td>16–20</td>
<td>3.617</td>
</tr>
<tr>
<td>21–30</td>
<td>2.454</td>
</tr>
</tbody>
</table>

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

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. Within-subject 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 theta–beta 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.

Rao R (24,1588)=1.12; p<.3144

Figure 1. Plot of the mean theta–beta power ratios for the two-way interaction, Age (Years) × 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.
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
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 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 4
Critical Values for Attention Deficit Hyperactivity Disorder on the Basis of Power Ratios

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>1.0 SD</th>
<th>1.5 SD</th>
<th>2.0 SD</th>
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<tbody>
<tr>
<td>6–11</td>
<td>4.36</td>
<td>5.03</td>
<td>5.69</td>
</tr>
<tr>
<td>12–15</td>
<td>2.89</td>
<td>3.31</td>
<td>3.72</td>
</tr>
<tr>
<td>16–20</td>
<td>2.24</td>
<td>2.36</td>
<td>2.48</td>
</tr>
<tr>
<td>21–30</td>
<td>1.92</td>
<td>2.13</td>
<td>2.34</td>
</tr>
</tbody>
</table>

Table 5
Accuracy of Classification Using the Theta-Beta Power Ratio

<table>
<thead>
<tr>
<th>Classification</th>
<th>Correct (%)</th>
<th>False+ (%)</th>
<th>False− (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.0 SD</td>
<td>88</td>
<td>3</td>
<td>9</td>
</tr>
<tr>
<td>1.5 SD</td>
<td>84</td>
<td>2</td>
<td>14</td>
</tr>
<tr>
<td>2.0 SD</td>
<td>76</td>
<td>1</td>
<td>23</td>
</tr>
</tbody>
</table>

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 noninvasive, 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 beta–alpha excess (with normal alpha mean frequency), and the other type by beta–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 neurologically 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


