

## Combined Psychophysiological Assessment of ADHD: A Pilot Study of Bayesian Probability Approach Illustrated by Appraisal of ADHD in Female College Students

Raina Robeva,<sup>1,4</sup> Jennifer Kim Penberthy,<sup>2</sup> Tim Loboschewski,<sup>3</sup>  
Daniel Cox,<sup>2</sup> and Boris Kovatchev<sup>2</sup>

---

*Manifestations of ADHD are observed at both psychological and physiological levels and assessed via various psychometric, EEG, and imaging tests. However, no test is 100% accurate in its assessment of ADHD. This study introduces a stochastic assessment combining psychometric tests with previously reported (Consistency Index) and newly developed (Alpha Blockade Index) EEG-based physiological markers of ADHD. The assessment utilizes classical Bayesian inference to refine after each step the probability of ADHD of each individual. In a pilot study involving six college females with ADHD and six matched controls, the assessment achieved correct classification for all ADHD and non-ADHD participants. In comparison, the classification of ADHD versus non-ADHD participants was <85% for any one of the tests separately. The procedure significantly improved the score separation between ADHD versus non-ADHD groups. The final average probabilities for ADHD were 76% for the ADHD group and 8% for the control group. These probabilities correlated ( $r = .87$ ) with the Brown ADD scale and ( $r = .84$ ) with the ADHD-Symptom Inventory used for the screening of the participants. We conclude that, although each separate test was not completely accurate, a combination of several tests classified correctly all ADHD and all non-ADHD participants. The application of the proposed assessment is not limited to the specific tests used in this study—the assessment represents a general paradigm capable of accommodating a variety of ADHD tests into a single diagnostic assessment.*

---

**KEY WORDS:** attention-deficit/hyperactivity disorder (ADHD); electroencephalography (EEG); Bayesian probability/statistics; diagnostic assessment.

### INTRODUCTION

Attention-deficit/hyperactivity disorder (ADHD) is a commonly diagnosed disorder and is associated with many negative consequences, such as poor school achievement, more

<sup>1</sup>Department of Mathematical Sciences, Sweet Briar College, Sweet Briar, Virginia.

<sup>2</sup>Center for Behavioral Medicine Research, University of Virginia Health System, Charlottesville, Virginia.

<sup>3</sup>Department of Psychology, Sweet Briar College, Sweet Briar, Virginia.

<sup>4</sup>Address all correspondence to: Raina Robeva, PhD, Department of Mathematical Sciences, Sweet Briar College, Sweet Briar, Virginia 24595; e-mail: robeva@sbcc.edu.

visits to the emergency room, and more automobile accidents. Currently, however, ADHD cannot be strictly defined, and precisely and objectively measured (Barkley, 1998).

As a result, recorded prevalence rate for ADHD varies substantially, partly because of changing diagnostic criteria over time (the *Diagnostic and Statistical Manual for Mental Disorders (DSM)* has gone through four different editions during the last 35 years: *DSM-I*, *DSM-II*, *DSM-III*, and *DSM-IV*) and partly because of variations in ascertainment and the frequent use of referred samples to estimate rates. Reported rates also vary substantially in different geographic areas and across countries (American Academy of Pediatrics, 2000). Although the *DSM-IV* (American Psychiatric Association [APA], 1994) estimates the prevalence of ADHD in school-age children as between 3 and 5%, other community survey studies suggest it may be as high as 16% (Barbarese et al., 2002). ADHD occurs more commonly in males than in females, with ratios ranging from 4:1 to 9:1 (Monastra et al., 1999). Overall, ADHD is the most common neurobehavioral disorder of childhood, and is also among the most prevalent chronic health conditions affecting school-aged children (American Academy of Pediatrics, 2000).

Despite the predominance of research on young ADHD males, several findings indicate that ADHD may more correctly be identified as a lifespan developmental problem that does not limit itself to any one population (Gittleman, Mannuzza, Shenker, & Bonagura, 1985; Weiss, 1985). Early signs of ADHD may be observed in children before noticeable behavioral problems develop (Coldren & Corradetti, 1997), and these difficulties can apparently continue on into adulthood for as much as 70% of the overall ADHD patient population (Biederman, Wilen, Spence, & Faraone, 1996; Shaffer, 1994). One reason why adults have been largely ignored is that the ADHD symptomatology changes over time in such a way that although the attentional deficits are usually maintained, corresponding behavioral problems become less obviously linked to the disorder itself (Hart, Lahey, Loeber, Applegate, & Frick, 1995; Murphy & Barkley, 1996). Whereas the hyperactivity of children is easy to observe, this symptom appears to wane in adults. However, symptoms of impulsivity and inattention may remain in adulthood, and may disrupt social and occupational functioning, which may in turn facilitate associated symptoms of depression, anxiety, and conduct disorders. When these secondary symptoms occur, they may mask ADHD (Biederman et al., 1994; Murphy & Barkley, 1996; Shaffer, 1994). These associated problems are often misdiagnosed as being unrelated to the earlier diagnosis of ADHD when, in fact, they are an outgrowth of these difficulties (Biederman et al., 1994; Denckla, 1993; Epstein, Shaywitz, Shaywitz, & Wollston, 1991; Shaywitz & Shaywitz, 1991).

In addition to a paucity of research on the adult population, a recent conference on gender differences in ADHD (Eugene, 1996) also came to the general conclusion that females with ADHD are seriously underdiagnosed and remain largely unexamined at all ages. Although at younger ages, males and females may show many of the same, noticeable behavioral manifestations of ADHD, females are not as quickly diagnosed nor are they as likely to be treated for the condition (Biederman & Seidman, 1997; Goldstein, 1997; Silverthorn, Frick, Kuper, & Ott, 1996). With increasing age, both males and females with ADHD show decrease in situational hyperactivity or fidgeting (i.e., the overt behavioral symptoms). However, females generally seem to develop comorbidities such as anxiety/emotional disorders with increasing age, compared to males, who seem more likely to have a higher comorbid rate of conduct disorder, either in childhood or developing during adolescence (Biederman, et al., 1994; Epstein et al., 1991; Horner & Scheibe, 1997). Thus, ADHD is a valid clinical problem for females.

In addition to ADHD's psychological/behavioral manifestations of inattention, impulsivity, and hyperactivity, blood flow models, high-resolution SPECT imaging, and a number of EEG studies have led to the acceptance, by most investigators, that ADHD is a physiologically based disorder with a multifactorial etiology that includes neurobiology as an important factor (Ahn et al., 1980; Comings, 1997; Amen & Carmichael, 1997; Landau, Lorch, & Milich, 1992). Although a detailed review of EEG findings related to ADHD falls out of the scope of this manuscript, two specific phenomena, the EEG transition consistency and the alpha attenuation, will need to be introduced in order to clarify our further presentation.

The EEG Consistency Index (CI) was previously introduced as a measure quantifying the consistency of transition while participants shift from one cognitive task to another (Cox et al., 1999). The CI is based on a specific mathematical representation of EEG as a three-dimensional (frequency, spatial-EEG electrode position, and time) data flow. In a series of studies the CI has been shown to be a reliable and reproducible marker of ADHD that works best for boys younger than 12 years (Cox et al., 1999; Kovatchev et al., 2001). The sensitivity of the CI is decreased for females and additionally declined with increased age in both males and females (Kovatchev et al., 2001; Merkel et al., 2000). Mirroring diagnostic and treatment patterns, the CI has had relative difficulty being applied to the adult female population.

In addition to the CI, alpha attenuation refers to the second important component of this developing model. From the EEG spectrum, alpha waves refer to the range between 7.5 and 13 Hz. Alpha is usually best seen in the posterior regions of the head and on each side, being higher in amplitude on the dominant side. It is brought out by closing the eyes and by relaxation, and reduced by eye opening or alerting by any mechanism such as thinking or calculating. It is the major rhythm seen in normal relaxed adults, and is present during most of life, especially beyond the 13th year, when it dominates the resting of most persons. It is associated behaviorally with a relaxed state of unfocused attention and is present between active cognitive tasks. Studies have found low alpha activity associated with ADHD in children and young adults, and some researchers have proposed that these findings may support a maturational lag theory of ADHD. For example, Crawford and Barabasz (1996) tested seven ADHD and seven non-ADHD participants (ages 9–16) and found the presence of low frequency alpha activity in the frontal right hemisphere regions while listening to a story with their eyes closed and low alpha in central and temporal regions while doing arithmetic. Other findings include weaker stimulus-locked alpha attenuation in hyperactive as compared to non-hyperactive children (Grunewald-Zuerbier, Grunewald, & Rask, 1975). The finding that the alpha rhythm attenuates (or "blocks") with eye opening, combined with the gradual increase of this rhythm until adult levels are attained in late childhood, suggest that a weaker alpha attenuation may be another marker of ADHD observed in young adults. Thus, we hypothesized that alpha attenuation during active tasks would be weaker in young adults with ADHD versus controls.

In summary, there are several psychological and physiological markers that identify children with ADHD and could possibly signify ADHD in young adults. None of these markers is definitive, in terms of achieving 100% accuracy of classification, or reliable, in terms of clear reproducibility across time and across studies. In general, no ADHD test claims perfect sensitivity and specificity. As a result, physicians and researchers are forced

to use batteries of [imperfect] tests. Indeed, even with the use of current markers available, a substantial portion of individuals with ADHD remains largely undetected.

We here propose a method that meaningfully combines several tests into a single assessment and presents clear benefits when compared to its individual components. Our strategy is designed to incorporate various tests and markers for ADHD, none of which could claim perfect sensitivity and specificity to ADHD. The formal framework of that combined assessment employs a Bayesian approach that allows for linking of disparate assessment instruments into one unified stochastic assessment.

Bayesian methods have been used for statistical inference for more than a hundred years; however, recently there is an impressive expansion of their application to a variety of areas. As Carlin and Louis write in the second edition of their book: “The allocation of billions of U.S. federal dollars now depends on Bayesian estimate of population characteristics . . . The FDA now not only permits, but also encourages Bayesian designs and analyses . . . even the Microsoft Office Assistant is based on a Bayesian artificial intelligence algorithm. No longer merely of academic interest, Bayes methods have assumed their place in the toolkit of the well-equipped applied statistician (Carlin & Louis, 2000).”

This study proposes a stochastic assessment capable of combining several disparate measures into a single coherent assessment with a compounded classification accuracy that is significantly greater than the accuracy of each individual measure. In addition, we introduce a new EEG-based measure of alpha attenuation during active tasks that differentiates ADHD from control college-age females.

## METHODS

### Participants

Six female college students ranging in age from 18 to 22 years with an average age of 20.7 ( $SD = 1.5$ ) diagnosed with ADHD and currently taking ADHD medication, and six age-matched controls ranging in age from 18 to 21 years with an average age of 19.5 ( $SD = 1.4$ ) were selected for the study. All participants attended a small liberal arts woman’s college in the south. Women were included in the ADHD group if they (a) had a current diagnosis of ADHD (with or without hyperactivity); (b) were currently taking and have been taking for at least three years any type of psychostimulant medication (with the exception of Cylert<sup>®</sup> or Pemoline) to treat their ADHD; (c) were not taking medication to treat anxiety or depression; and (d) did not have any significant health problems or disorders that might affect the brain or EEG recordings (e.g., Tourette’s Syndrome, epilepsy). According to the Utah standards (Wender, 1995), an original diagnosis has to be made during childhood and all of our ADHD met this criterion.

Participants in the control group ( $N = 6$ ) were included if they (a) had no known history of ADHD or disruptive behavioral disorders; (b) had never been prescribed stimulant medication nor taken it for recreational purposes; (c) were not taking any medication to treat anxiety or depression; (d) did not have any significant medical disorders which might impact the brain or EEG data collection/interpretation, and (e) demonstrated the lack of ADHD symptoms on our screenings.

No participants in our selected sample had any reported learning disabilities.

## Procedure

The study was approved by the Institutional Review Board. Interested participants ( $N = 60$ ) responded to a campus-wide e-mail-recruiting message with general contact and demographic information, as well as their ADHD diagnostic status. All participants were first given a general screening questionnaire that included sections on ADHD history, learning disabilities, drug/alcohol use, college activities and academic standing, current medications, and history of medical/psychological problems.

Participants who presented with an ADHD diagnosis were given the Brown (Brown ADD scales, 1996) and ADHD—Symptom Inventory (ADHD-SI; Cox et al. 1999) tests (described in the next section) to confirm their ADHD status. As a result of this screening a total of six women matching the inclusion criteria above were selected for this group. All women in this sample were classified as ADHD combined type. An additional five ADHD women were screened but not included due to intermittent and inconsistent adherence to proscribed medical regimen ( $N = 1$ ), additional medication for anxiety/depression ( $N = 2$ ), and significant health problems that might effect the EEG recordings ( $N = 2$ ). One of these five women had a learning disability (dyslexia), but she was on a number of medications for anxiety/depression and was excluded on that basis. No other participants reported learning disabilities.

The control sample was matched based on similarity to our already defined ADHD sample. To confirm the lack of ADHD symptoms and to rule out the possibility of ADHD, the potential control participants were also screened with the Brown ADD test and ADHD-SI. In the cases where more than one individual matched our ADHD participants, we selected those with lowest Brown and ADHD—SI scores from the pool of potential control participants.

## Screening Measures

The following measures were used as external confirmation of the self-reported diagnoses and inclusion in the ADHD group as well as to demonstrate lack of ADHD symptoms for the control group.

### *Brown Attention-Deficit Disorder Scale (adult version)*

The Brown ADD scale (Brown ADD scales, 1996) is a 40-item self-report scale designed to serve as a preliminary screen for ADD and to assess for additional cognitive and affective impairments often associated with Attention-deficit disorders. Individuals rate the occurrences of behavior on a 4-point scale ranging from 0 (*never*) to 3 (*almost daily*). Cluster scores are available for problems with activation, attention, effort, affect, and memory as well as a total score. Individuals who have a total score of 40 or less are possible, but not likely to have ADD; scores of 41–54 indicate a probability of an ADD and scores 55 or higher indicate a high probability of an ADD. The testing manual (Brown, 1996) reports several studies that support the internal and construct validity of the instrument as well as its discriminative power. The sample of ADHD participants had total scores ranging from 55 to 94; and the control sample had total scores ranging from 6 to 32 (Table I - Screening).

**Table I.** Screening Scores, Scores From All Tests Included in the Sequential Stochastic Assessment, and Conditional Probabilities of Earning These Scores Given ADHD for All Participants

Group	Screening		Steps of the sequential stochastic assessment					
			A: WURS		B: Consistency index		C: Alpha blockade index	
			Score	$P_{\downarrow}^{\text{WURS}}$	% CI	$P_{\downarrow}^{\text{CI}}$	% ABI	$P_{\downarrow}^{\text{ABI}}$
ADHD	121	94/17	33	.52	37.5	0.53	19.7	.51
	401	58/33	37	.55	12.5	0.84	15.4	.61
	427	63/38	45	.61	<b>50.0</b>	<b>0.42</b>	16.0	.60
	509	55/21	<b>25</b>	<b>.42</b>	37.5	0.53	8.6	.78
	874	91/21	46	.61	0.0	1.00	8.2	.80
	712	64/29	56	.69	12.5	0.84	<b>72.4</b>	<b>.17</b>
Control	45	20/9	3	.05	100.0	0.00	20.1	.50
	191	15/3	12	.20	50.0	0.42	39.2	.38
	194	8/2	8	.13	62.5	0.31	25.8	.46
	268	6/1	2	.03	<b>37.5</b>	<b>0.53</b>	20.7	.50
	500	32/12	<b>35</b>	<b>.54</b>	87.5	0.10	<b>11.2</b>	<b>.72</b>
	640	17/4	9	.15	87.5	0.10	80.2	.12
Fisher's exact test			Exact significance (two-sided) = .041		Exact significance (two-sided) = .041		Exact significance (two-sided) = .041	

Note. The cells with bold numbers indicate participants who were misclassified by a particular test.

<sup>a</sup>Generated randomly to ensure confidentiality.

### ADHD Symptom Inventory (ADHD-SI)

The ADHD-Symptom Inventory is an 18-item scale developed from *DSM-IV* criteria for ADHD and was introduced by Cox et al., 1999. The ADHD-SI is a measure of symptom severity, and is scored on a 3-point Likert scale with higher scores representing higher severity of symptoms. The ADHD-SI correlates highly with other rating scales assessing hyperactivity and inattentive behavior and exhibits good discriminative power (Cox et al., 1999, Merkel et al., 2000). On this measure, the ADHD group scores ranged from 17 to 38 in comparison to the range of 1–12 for the controls (see Table I - Screening).

### Measures for the Combined Stochastic Assessment

To validate the proposed assessment, we used a combination of one established psychometric test (Wender-Utah Rating Scale [WURS]; Ward, Wender, & Reimherr, 1993), one previously reported EEG marker for ADHD (the Consistency Index, Cox et al., 1999; Merkel et al., 2000; Kovatchev et al., 2001), and one new EEG-based marker, the Alpha Blocking Index (ABI), introduced in this manuscript.

#### Psychometric Test—Wender-Utah Rating Scale

The WURS test is a 61-item retrospective self-report scale with adequate reliability and validity (Ward, Wender, & Reimherr, 1993). Individuals rate the severity of ADHD symptoms experienced when they were children using a 5-point Likert scale. Using a cutoff score of 36, Ward et al. report correctly identifying 96% of the ADHD adult sample from normal controls. Using a cutoff score of 46 correctly identified 86% of ADHD individuals, 99% of normal controls and 81% of individuals classified as depressed. Twenty-five of

the 61 items were empirically determined to be the most valid discriminators of ADHD versus non-ADHD individuals. Scores on this “short form” are derived by summing the individual’s ratings on these 25 items (Ward et al., 1993). The score from the WURS (short form) ranges from 0 to 100, with scores  $>30$  indicating ADHD. For adults, WURS has been shown to be a valid retrospective screening and dimensional measure of childhood ADHD symptoms (Stein, Fischer, & Szumowski, 1999, 2000), to replicate and correlate with Connors Abbreviated Parent and Teacher Questionnaire and demonstrate internal consistency reliability (Fossati et al., 2001), and to exhibit good construct validity (Weyandt, Linterman, & Rice, 1995).

### *EEG Session*

Participants were seated in front of the computer and an appropriately sized EEG cap was placed over their heads. Electrode placement was in accordance with the international 10–20 system. Eight electrode sites were prepared: a ground in front of Cz, a right earlobe reference electrode, and Cz, Pz, P3, P4, F3, F4, T5, T6. EEG signals were amplified and processed by the Lexicor Neurosearch-24 system. The EEG data collection used standard settings of a clinical EEG acquisition with 5 k $\Omega$  impedance criterion measured by a Prep-Check electrode impedance meter. The total frequency range was 0.5–32 Hz. The relative power of the following frequency bands was computed: theta (4–7.5 Hz), low alpha (7.5–10 Hz), high alpha (10–13 Hz), and beta (13–22 Hz). The residual power was carried by frequencies below 4 Hz or above 22 Hz.

Following all screenings and assignment to categories of ADHD or control, all participants took part in a structured EEG session. Individuals in the ADHD group were tested off their medication; medication was discontinued at least 36 hr prior to the EEG testing. EEG was recorded while the participants engaged in a rapid sequence of six 3-min computer-tracing tasks separated by five 2-min rest intervals. The tracing used the “MacLaboratory for Psychology Research” program and had increasing difficulty with each task. To acclimate to the testing environment, the participants were initially asked to engage in a simple computer-tracing task (i.e., they used the computer mouse to trace a simple geometric shape). During subsequent phases, they had to move the mouse at an increasingly rapid pace, while maintaining the trace. This was followed by a mirror-tracing phase where participants continued the tracing, while the computer mouse and cursor work in opposite directions. So, if the participant moved the mouse up and to the left, the cursor moved down and to the right on the screen. To be accurate at this test, a substantial amount of concentration and mental effort was required.

### *EEG Consistency Index*

As previously reported, the CI is an EEG-based measure of ADHD (Cox et al., 1999; Kovatchev et al., 2001; Merkel et al., 2000). The CI ranges from 0 to 100%; a CI  $< 40\%$  indicates ADHD (Kovatchev et al., 2001). The CI of each participant was computed using data from the first two adjacent disparate tracing tasks, slow forward and fast forward tracing, which followed the initial accommodation period. We used our previously published algorithm with threshold parameter of 1.0 and no cutoff (Kovatchev et al., 2001). These settings correspond to the procedures employed by our previous studies (Cox et al., 1999;

Kovatchev et al., 2001; Merkel et al., 2000). A brief description of the CI follows. Details can be found in Kovatchev et al., 2001.

The CI is based on the notion that the EEG data stream can be represented by a three-dimensional numeric array—at any given moment one dimension is frequency of brain waves, another is spatial—the location of the electrode on a participant’s head, and the third is time. ADHD can cause disruption in the frequency or spatial dimension or in both. This disruption is most evident when the tested participant transitions from one cognitive task to another, the two tasks being separated by a rest period of approximately 3–5 min. The transition is deemed “consistent” if the differences between the means of the power spectra from the adjacent tasks shift coherently from low to high or vice versa, for example, a consistent transition would mean that most frequency bands and most channels would display similar unidirectional shifts, while an inconsistent transition will result in scattered power changes across the EEG bands and channels.

Given this heuristic explanation, the algorithm for computing the CI works as follows:

- (1) Discrete spectra, including residual power, are calculated for all EEG channels through a standard FFT algorithm. The relative power of the theta, low alpha, high alpha, and beta frequency bands was computed.
- (2) *Power change distances* (PCD) between two contiguous tasks separated by a break (slow forward and fast forward tracing in our study) are computed for each EEG band and channel. Each PCD is normalized using the formula on the below, where  $M1$  and  $M2$  are the mean powers at two contiguous tasks,  $SD1$  and  $SD2$  are their standard deviation, and  $N1$  and  $N2$  are the epoch counts at these tasks.

$$\text{PCD} = \frac{M1 - M2}{\sqrt{\frac{SD1^2}{N1} + \frac{SD2^2}{N2}}}$$

- (3) PCD undergo filtering to eliminate changes below a “noise threshold” of 1.0: The PCD values that are larger by an absolute value than the threshold will be marked with 1 or  $-1$  depending on their direction, whereas all PCD below threshold will be marked by zero. This procedure transforms the PCD sequence into a sequence of 1, 0,  $-1$  that indicates, for each EEG band and channel, whether a significant power change was observed while the person shifted from the first task to the next. *The CI is defined as the count of nonzero components of this sequence.* The maximum CI equals the number of EEG channels multiplied by the number of EEG bands used during spectrum discretization. For our study, with eight-channel EEG equipment and four bands, the CI ranges from 0 to 32. To make the results comparable across different experiments, the CI is expressed in terms of percentage from its maximum value.

#### *Alpha Blockade Index (ABI)*

We used EEG data from the six tasks and the five-separating rest periods to quantify the suggested weaker attenuation of alpha in ADHD (Grunewald-Zuerbier, et al., 1975).

The ABI was designed as a measure that quantifies this attenuation. Based on the studies summarized in the Introduction section, it was expected that an ADHD person going through a rapid sequence of task and rest periods would display less alpha attenuation compared to a control. In numerical terms this translates into a lower average absolute difference between the alpha powers in sequential task and rest periods.

In particular, during the EEG session a sequence of high-alpha powers  $\alpha_1, \alpha_2, \dots, \alpha_k$  is obtained for each person, corresponding to the sequence of rapidly changing tasks and rest periods. Then the ABI for a person is computed using the formula

$$\text{ABI} = \frac{10}{k-1} \cdot \sum_2^k |\alpha_k - \alpha_{k-1}|,$$

where  $k$  is the total number of task and rest periods. ABI values which are greater than 100 are reset to 100, which results in an index expressed in percentage points between 0 and 100%. A lower ABI is associated with weaker attenuation of alpha during active periods, as compared to rest periods and could therefore be indicative of ADHD. On the basis of the results in this study, we suggest a cutoff of 20%, that is  $\text{ABI} < 20\%$  would indicate ADHD.

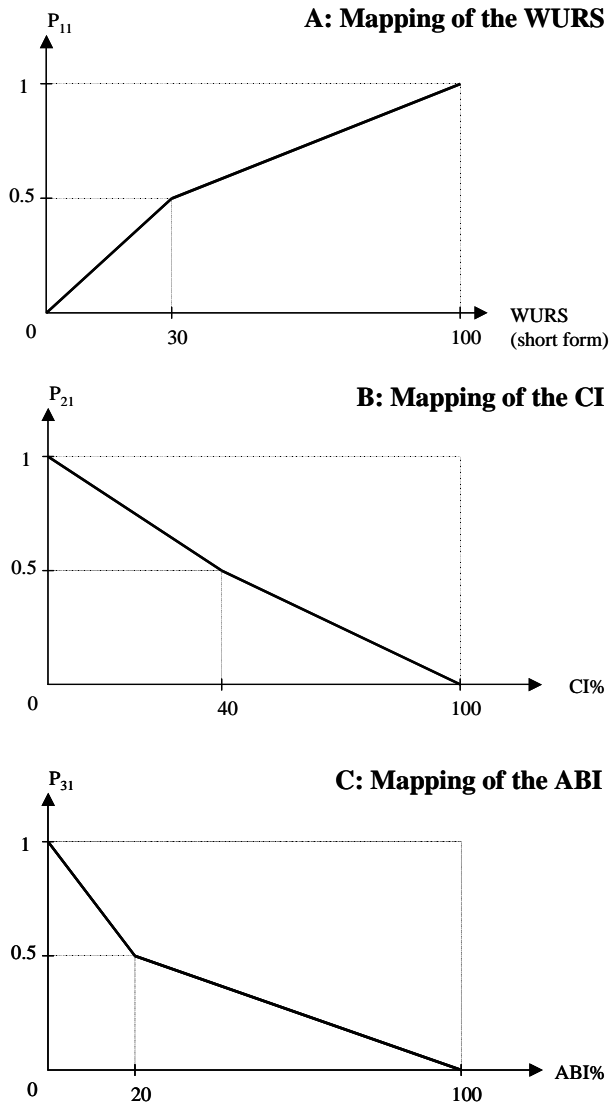
A brief justification of this formula follows: The ABI is essentially equal to the average of the absolute values of the differences in alpha power between two consecutive tasks/rest periods, that is, the ABI is essentially equal to

$$\frac{1}{k-1} \sum_2^k |\alpha_k - \alpha_{k-1}|,$$

where  $k$  is the total number of tasks and rests. The coefficient 10 and the reset of values above 100 to 100 are only used to calibrate the ABI to the range 0–100, similarly to the calibration of the CI (Kovatchev et al., 2001). This calibration does not alter the classification of ADHD/non-ADHD based on the ABI (with a suggested cutoff of 20), or any statistical tests reflecting the power of that classification. Note also, that the formula could be applied to any sequence of short tasks separated by short breaks with suggested length 3 and 2 min, respectively. In this study  $k = 6(\text{tasks}) + 5(\text{restperiods}) = 11$ .

### Standardizing the Scores of Different Tests

In order to integrate the results from the psychometric test, the CI, and the ABI results into a single assessment of ADHD, we first needed to standardize the output of these tests. To do so, we translate the output of each test into a “probability for ADHD.” The idea is that at each test (step) of the assessment, each participants receives a certain test score and the magnitude of this score depends on whether the participants has ADHD, as well as on the severity of the disorder. In other words, the probability of earning a certain score on a test depends on the participants’ condition, ADHD or non-ADHD. In addition, each test has a suggested cutoff value, and scores greater (or lower) than this cutoff value are accepted as indicators of ADHD. Thus, we can use a linear mapping of a test score into a probability for ADHD ranging from 0 to 1 with the test cutoff value mapped to .5, and the test maximal



**Fig. 1.** Mapping of probabilities to test scores of WURS, CI, and ABI, respectively. The mapping uses separate linear interpolations within each of the two tests score intervals: from minimal score to cutoff, and from cutoff to maximal score.

(or minimal) value indicating ADHD mapped to 1. Figure 1 presents the mapping of test scores to probabilities for the tests employed in this study.

Specifically, the calculations of the probabilities for ADHD are carried out as follows:

- (1) Figure 1(A): The score on the WURS (short form) ranges from 0 to 100 with scores  $>30$  indicating ADHD. Thus the [conditional] probability for earning a

score of  $K_1$  on this test, given ADHD is  $P_1^{\text{WURS}} = K_1/60$  if  $K_1 \leq 30$  and  $P_1^{\text{WURS}} = (K_1 + 40)/140$ , if  $K_1 > 30$ . The probability of earning a score of  $K_1$ , given non-ADHD is  $P_2^{\text{WURS}} = 1 - P_1^{\text{WURS}}$ .

- (2) Figure 1(B): The CI ranges from 0 to 100% with a CI < 40% indicating ADHD. Thus the probability of having CI =  $K_2$ , given ADHD is  $P_1^{\text{CI}} = (80 - K_2)/80$ , if  $K_2 \leq 40$  and  $P_1^{\text{CI}} = (100 - K_2)/120$ , if  $K_2 > 40$ . The probability of CI =  $K_2$ , given non-ADHD, is  $P_2^{\text{CI}} = 1 - P_1^{\text{CI}}$ .
- (3) Figure 1(C): The ABI ranges from 0 to 100% with ABI < 20% indicating ADHD. Thus the probability of ABI =  $K_3$ , given ADHD is  $P_1^{\text{ABI}} = (40 - K_3)/40$ , if  $K_3 < 20$  and  $P_1^{\text{ABI}} = (100 - K_3)/160$ , if  $K_3 > 20$ . The probability of ABI =  $K_3$ , given non-ADHD, is  $P_2^{\text{ABI}} = 1 - P_1^{\text{ABI}}$ .

### Combined Psychophysiological Assessment of ADHD

We use the classic theory of Bayesian inference (Carlin & Louis, 2000). The general idea of this approach is that before an experiment we have a prior probability of certain outcome (e.g., ADHD), then the experimental data update this probability resulting in a posterior probability of the outcome.

Heuristically, justification of this paradigm would be the following: any test, for ADHD or otherwise, produces a score that is contingent upon certain (psychological, biologic, etc.) characteristics of the tested participants. Therefore, a participant with a certain condition (ADHD) is expected to yield a higher (or a lower score depending on the direction of the test), compared to a subject without that condition. However, the relationship between the condition and the test score is not always exact—it may happen that a participant without ADHD scores high on the WURS scale, or vice versa. Thus, this relationship is probabilistic and is best quantified as a conditional probability of earning certain score, given a preexisting condition, which is a number between 0 and 1 (or 0 to 100%), that is one of continuum-many possible values.

When we utilize the Bayesian method in this study, applying it to a series of experiments, the posterior probability of each experiment is fed into the next experiment, yielding a sequential refinement of the outcome probability. The refinement procedure uses Bayes' formula to link the sequential steps as follows: At step 0, a prior probability for ADHD  $P_{\text{ADHD}}^0 = 0.5$  is assigned to each participant regardless of whether she is ADHD or control (see Discussion section for consideration of the choice of prior probabilities). Then  $P_1^{\text{WURS}}$  and  $P_2^{\text{WURS}}$  are used to calculate a posterior probability  $P_{\text{ADHD}}^1$  for ADHD, given this participant's WURS score via the formula:

$$P_{\text{ADHD}}^1 = \frac{P_1^{\text{WURS}} \cdot P_{\text{ADHD}}^0}{P_1^{\text{WURS}} \cdot P_{\text{ADHD}}^0 + P_2^{\text{WURS}} \cdot (1 - P_{\text{ADHD}}^0)}$$

From here on the procedure is recursive—after each step the posterior probability becomes a prior probability for the next step; for example, taking into consideration the CI scores, in the formula above  $P_{\text{ADHD}}^0$  is replaced by  $P_{\text{ADHD}}^1$ ,  $P_1^{\text{WURS}}$  is replaced by  $P_1^{\text{CI}}$ , and  $P_2^{\text{WURS}}$  is replaced by  $P_2^{\text{CI}}$ , producing as a result  $P_{\text{ADHD}}^2$ , which now incorporates results from both WURS and CI. The final result of the computations will be the probability  $P_{\text{ADHD}}^3$  incorporating results from all three tests that were conducted.

## Statistical Analysis

Fisher's exact probability tests were used to evaluate the ability of each test to classify ADHD versus non-ADHD participants. t-Tests, repeated measures ANOVA, and correlations were used to analyze the probabilities of ADHD computed at and after each test of the combined assessment.

## RESULTS

### Steps of the Assessment

Table I presents the scores from each test (step of the combined assessment) as well as the probability of earning these scores given ADHD as derived from the standardized scores for each test. The cells with bold numbers in Table I identify participants who were misclassified by the test, for example, had  $<.5$  probabilities for ADHD while being in the ADHD group, or  $>.5$  probabilities for ADHD while being controls. It is evident that each of the three tests misclassified one control and one ADHD participant, which lead to identical significance levels of  $p = .041$  for all of the Fisher's exact probability tests. However, the three tests misclassified *different* participants, thus we can expect that a combination of all tests would be able to distinguish clearly ADHD participants from controls. Specifically, Table I—Steps of the Combined Stochastic Assessment presents the WURS (short form), the CI, and the ABI scores of all participants together with their respective conditional probabilities  $P_1^{\text{WURS}}$ ,  $P_1^{\text{CI}}$ , and  $P_1^{\text{ABI}}$  to earn this score on each of the specified test, given ADHD.

### Combined Psychophysiological Assessment

Table II presents the process of the Bayesian assessment of each participant's probability for ADHD as updated after each test. The initial probabilities for all participants are  $.5$ , for example, we do not assume anything in the beginning of the assessment. At each step a probability of ADHD greater than  $.5$  classifies the participant as ADHD, whereas a probability  $<.5$  classifies the participant as a control. It is evident that the classification improves with each additional step, achieving correct classification for all participants after the final ABI test. After the first step (WURS) one ADHD and one control participant are misclassified. At Step 2, the CI corrects the classification of the misclassified control participant. At the last step, the ABI corrects the classification of the one misclassified ADHD participant, without compromising the classification of the rest of the participants.

The average probabilities for ADHD increase along the steps of the combined assessment from 57 to 76% in the ADHD group, and decrease from 18 to 8% in the control group, thus increasing the separation between the groups as the evaluation progresses (Table II). To confirm that this increase was significant, we conducted a  $2 \times 2$  repeated measures ANOVA (ADHD-control)  $\times$  (Pre-post combined assessment). This analysis resulted in a significant interaction effect,  $F = 12.005$ ,  $p = .006$ ,  $df = 1, 10$ , demonstrating that even with this small sample size the application of the combined assessment resulted in significantly better separation of the two groups. The increase of the separation was further confirmed by the increase of the  $t$  values of the  $t$  tests comparing ADHD versus control probabilities for ADHD at each step (Table II).

**Table II.** Sequential Psychophysiological assessment

Screening		Sequential probabilities of ADHD			
Group	ID	Step 0: Initialization ( $P^0_{ADHD}$ )	Step 1: WURS ( $P^1_{ADHD}$ )	Step 2: CI ( $P^2_{ADHD}$ )	Step 3: ABI ( $P^3_{ADHD}$ )
ADHD	121	.5	.52	0.55	0.56
	401	.5	.55	0.87	0.91
	427	.5	.61	0.52	0.62
	509	.5	<b>.42</b>	<b>0.45</b>	0.75
	874	.5	.61	1.00	1.00
	712	.5	.69	0.92	0.71
Control	45	.5	.05	0.00	0.00
	191	.5	.20	0.15	0.10
	194	.5	.13	0.07	0.06
	268	.5	.03	0.04	0.04
	500	.5	<b>.54</b>	0.12	0.26
	640	.5	.15	0.02	0.00
<i>t</i> -Value; significance		—	$t = 4.1; p = .02$	$t = 4.7; p = .001$	$t = 7.3; p < .0001$

*Note.* At Step 0 all participants were assigned equal probabilities of ADHD, which were then refined by the sequential tests. The cells with bold numbers indicate participants who were misclassified at each step.

There was a highly significant correlation between the final probabilities for ADHD and the behavioral scales used as screening measures: Brown ADD scale:  $r = .87, p < .001$ ; ADHD-SI:  $r = .84, p < .005$ . In addition, the partial correlations between the final probabilities for ADHD and the screening scales, controlling for the first step of the assessment (WURS) were Brown ADD scale:  $r = .60, p = .027$  and ADHD-SI:  $r = .44, p = .083$ . This indicates that despite a significant initial correlation between the screening and Step 1 of the assessment ( $r = 0.8$ ), Steps 2 and 3 had additional sizable contribution over Step 1. Thus, a combined approach not only separates control from ADHD participants better than its components, but also yields a better agreement (collinearity) between the final ADHD score and the baseline diagnosis.

## DISCUSSION

Despite the well-documented persistent patterns of inattention and/or hyperactivity-impulsivity in ADHD, the mechanisms and etiology of the disorder remain methodologically difficult to study, with different studies yielding inconsistent results (Barkley, 1998). Zametkin and Rapoport (1987) identified 11 separate neuroanatomical hypotheses that have been proposed for the etiology of ADHD. Overall, numerous studies have compared ADHD to non-ADHD children using various neuroimaging technologies and EEG acquisition and analysis techniques, and many studies have repeatedly found differences; for example, decreased metabolic activity in suspected attentional areas of the brain (Zametkin et al., 1990), increased power of theta relative to beta in ADHD (Mann, Lubar, Zimmerman, Miller, & Muenchen, 1991; Monastra et al., 1999), but the specific differences have been inconsistent. In most studies the conclusion was that either delayed maturation or defects in cortical activation play roles in the pathophysiology of ADHD, however no single clear-cut psychometric, or neurobiological assessment of ADHD has been developed to date. Hence, a viable alternative to a single definitive measure might be a combination of measures, equipped with

a method for refining the results from one test with the results from another and yielding a compounded assessment that works better than each of its separate components.

The concept of combining test outcome with intuitive knowledge and expert opinion is well developed mathematically. Bayesian methods provide a way to combine probabilistic and experimental-data reasoning, as well as very convenient tools for creating evaluation procedures that refine the outcome assessment with every subsequent step. These procedures are especially useful in situations where there is no single conclusive assessment, but rather a number of imperfect tests that marginally address the outcome of interest.

In this manuscript we present a general model of Bayesian inference and apply this model to assess ADHD in a small sample of female college students. The students were first classified in ADHD versus non-ADHD groups and then were studied using a sequence of psychometric and EEG tests. We used the Brown ADD scale and the ADHD-SI in the screening process to confirm existing ADHD diagnosis for the ADHD group and to assure lack of ADHD symptoms for the control group. The Bayesian stochastic assessment was further built using the individual WURS, CI, and ABI scores of the participants in both groups. It should be stressed however that the choice of screening measures and/or further tests, as well as their number, is not binding. For example, instead of using the Brown ADD scale for screening, we could have used WURS. Subsequently, we could have included the Brown scores in place of WURS in the combined assessment (applying appropriate standardization). In other words, the proposed assessment does not aim at replacing any established practices for screening and diagnosing of ADHD but instead at demonstrating that the outcomes of different tests could be linked in a manner that is guaranteed to work better than each of the individual test.

To combine the results of such generally disparate tests, we propose a method for standardization of test results. Since the plan is to employ Bayesian probability theory, a natural scale for standardization is offered by the conditional probability of earning a certain test score, given ADHD. In many tests certain cutoff points are recommended, which are useful for classification. By tradition used in a number of statistical analyses (e.g., discriminant analysis or logistic regression) the probability cutoff is 0.5, for example a person with a probability of ADHD  $> .5$  would be classified as ADHD, whereas a person with a probability  $< .5$  would be classified as non-ADHD. Thus, when converting each test's scores into probabilities, we need to keep in mind the cutoff values suggested by the test author, and map these values to cutoff probabilities of .5. For example, in our studies, the CI has a suggested cutoff of 40% with a CI  $< 40\%$  usually indicating ADHD. Thus, 40% should be mapped to a probability of .5, CI = 0 should be mapped to a probability of 1, and CI = 100% should be mapped to ADHD probability of 0 (Fig. 1(B)). If a test does not have a suggested cutoff value, the whole range of the test score could be mapped to the interval [0, 1] paying attention to the direction of the test, for example, if a higher test value indicates ADHD, then the highest possible test score should be mapped to 1 and the lowest possible test score should be mapped to 0.

Heuristically, mapping the suggested cutoff value for each test to .5 quantifies the notion that an event is more likely to happen when its odds are bigger than 50/50 and less likely to happen when the odds are less than 50/50. This emphasizes the stochastic nature of ADHD classification and that, even in a case of very high or very low probability for ADHD in the Bayesian model, the possibility of misclassification remains. This approach is in agreement with the standard practices for interpreting the results from certain psychometric ADHD

tests. For example, the Brown scale introduces a threshold interpretation of the total score broken down into three regions: (1) ADD possible but not likely, (2) ADD probable but not certain, and (3) ADD highly probable (Brown ADD Scales; Adult, 1996).

An important feature of the proposed combined assessment is that it is test-order-invariant. In other words, the sequence in which the tests are introduced into the recursive Bayesian algorithm *does not alter the final result*. For example, we could choose to replace the sequence WURS\_CI\_ABI in the paper with ABI\_WURS\_CI, or CI\_WURS\_ABI, or any other permutation of the tests, but the final ADHD probabilities in Table II will remain unchanged. The same is true for any selection of tests and for any number of tests. This generalization could be strictly proved to establish the following mathematical fact: *For any number of tests, the final Bayesian probability for ADHD calculated by the proposed stochastic assessment does not depend on the order in which the tests are included in the assessment*. The proof is available from the authors upon request.

It should be noted, however, that the probabilities calculated at the intermediate steps in the assessment will change. For example, if we use the sequence ABI\_WURS\_CI, and compare it with the one we have used in the paper (WURS\_CI\_ABI), the results in Table II for  $P_{ADHD}^1$  and  $P_{ADHD}^2$  will be different:  $P_{ADHD}^1$  will be the probability for ADHD based on one test (ABI) and  $P_{ADHD}^2$  will be the probability for ADHD based on the ABI and WURS tests.

The choice of  $P_{ADHD}^0 = 0.5$  for the initial probability in the assessment is an indicator of no prior assumptions or knowledge regarding the ADHD or non-ADHD status of a particular participant. In other words, when *no* information is available, that is before any tests are performed or any demographic/prevalence data are considered, the chances that a particular participant has or does not have ADHD are 50/50. With this choice of initial probability, the Bayesian probability for ADHD calculated after the first test will always be exactly equal to the conditional probability [given ADHD] on that first test. Thus, an alternative approach is to set the initial probability to be equal to the probability of earning a certain score on the first test, given ADHD. For example, a physician who knows nothing about a new patient having or not having ADHD may assume that the patients' probability of ADHD is equal to the population prevalence of the disease at the moment this new patient walks into the office, for example, if the patient is a school-age boy, the physician may assume that the prior probability of ADHD for that boy is 5%. In terms of our stochastic assessment, this physician's assumption is equivalent to taking a prior probability of ADHD of 50% (no knowledge at all) and running a first "demographic test" when the boy enters the office with probabilities of 5 and 95% for ADHD and non-ADHD respectively. As a result of this first test the Bayesian probability for ADHD will become 5%, that is, exactly equal to the prevalence of the disorder. Further tests will refine this probability and will end up with a definitive diagnosis.

In many cases it might be useful to identify an intermediate "gray zone" between non-ADHD and ADHD. In the "gray zone" no classification is apparent. This corresponds to the elusive and continuous nature of the disorder and is, again, best quantified in terms of an interval of probabilities, for example, if  $.4 < P_{ADHD} < .6$ , no definitive classification can be made. Each of the tests discussed in this manuscript has its own gray zone, which results in uncertain classification of participants. The procedure that we present narrows the gray zone with each step. In this particular study the method achieved perfect separation between ADHD women and controls. In general, we would expect that in a larger, or

less carefully selected participant sample, the three-step assessment would also have its own “gray zone.” Indeed a post hoc analysis of six additional participants, all of whom were classified as controls by the Brown scores, revealed that two of these participants had final probabilities of ADHD  $>.5$ . Interestingly, these two participants had relatively high WURS (short form) scores of 27 and 25 indicating possible ADHD, and for both of them the CI and the ABI were in the ADHD zones. This discrepancy between the results of all tests goes along with the claim of the Brown scores: “a score  $<40$  indicates that ADD is *possible*, but not likely” (Brown ADD Scales; Adult, 1996). It also illustrates that a combination of tests may point in the direction of identifying possible ADHD cases that may have remained undetected when only a single, although well established, assessment is used.

Neuropsychological EEG studies have attributed certain changes in powers of frequency band under specific testing conditions. The presence of beta activity is considered by most psychophysicists to reflect active mental processing, whereas alpha is associated with relaxation and delta and theta with under arousal. The attenuation of alpha and theta activity and the presence of beta activity signify “active mental processing” (Andreassi, 1989). In an attempt to quantify alpha attenuation, this manuscript introduces the Alpha Blockade Index as another possible EEG-based measure of ADHD. As opposed to the CI, which is based on all EEG bands, the ABI uses only the power of high-alpha (10–13 Hz) brain waves. Nevertheless, the ABI contributed to the better classification of ADHD versus control participants, increasing the separation between the ADHD and control groups in terms of  $t$  values (Table II, step 3). Although the small sample size of this study does not allow for a definitive validation of the ABI, upon further investigation, this index may prove to be another EEG measure that is sensitive to ADHD in young adults, females in particular. The use of the ABI also reiterates that our Bayesian method is general in nature and is capable of combining well-established measures (e.g., WURS) with newer procedures (e.g., CI), and experimental measures (e.g., ABI).

A major limitation of this study is its small sample size. Despite the small sample size, however, all presented results are statistically significant. On the other hand, we would like to stress that the purpose of this paper is to present a mathematical method and a detailed computational algorithm that make possible the integration of seemingly disparate ADHD tests. Thus, this paper is methodological, rather than a clinical-study report. The application of the presented methods is not limited to the tests that we chose for an illustration of our combined stochastic procedure: any of these tests can be replaced by another test and the number of sequential tests that can be included in the assessment is not restricted. In addition, the general idea of the proposed combined assessment could be used for initial screening and diagnosis of other disorders, as well as for treatment and evaluation of the effects of treatments, such as medication or additional therapies.

## ACKNOWLEDGMENTS

This study was supported by grant J-451 “Encephalographic and Psychometric Differences Between Female College Students With and Without Attention Deficit/Hyperactivity Disorder” from the Thomas F. Jeffress and Kate Miller Jeffress Memorial Trust, Richmond, VA, and by the grant “Quantifying Cognitive and Attentional Impairments: A Physiological Procedure” from the Carilion Biomedical Institute, Roanoke, VA.

## REFERENCES

- Ahn, H., Prichep, L., John, E. R., Baird, H., Treptin, M., & Kaye, H. (1980). Developmental equations reflect brain dysfunctions. *Science*, *210*, 1259–1262.
- Amen, D. G., & Carmichael, B. D. (1997). High-resolution SPECT imaging in ADHD. *Annals of Clinical Psychiatry*, *9*, 81–86.
- American Academy of Pediatrics. (2000). Clinical practice guideline: Diagnosis and evaluation of the child with Attention-Deficit/Hyperactivity Disorder. *Pediatrics*, *105*, (5), 1158–1170.
- American Psychiatric Association (APA). (1994). *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.). Washington, DC: American Psychiatric Association.
- Andreassi, J. L. (1989). *Psychophysiology: Human behavior and physiological response*. Hillsdale, NJ: Erlbaum.
- Barbareis, W. J., Katusic, S. K., Colligan, R. C., Pankratz, V. S., Weaver, A. L., Weber, K. J., et al. (2002). How common is attention-deficit/hyperactivity disorder? Incidence in a population-based birth cohort in Rochester, Minn. *Archives of Pediatrics and Adolescent Medicine*, *156*(3), 217–224.
- Barkley, R. A. (1998). *Attention deficit hyperactivity disorder: A handbook for diagnosis and treatment* (2nd ed.). New York: Guilford Press.
- Biederman, J., & Seidman, L. J. (1997). “A pilot study of neurological function in girls with ADHD”: Reply. *Journal of the American Academy of Child and Adolescent Psychiatry*, *36*, 1162–1163.
- Biederman, J., Faraone, S. V., Spencer, T., Wilens, T., Mick, E., & Lapey, K. A. (1994). Gender differences in a sample of adults with attention deficit hyperactivity disorder. *Psychiatry Research*, *53*, 13–29.
- Biederman, J., Wilens, T. E., Spence, T. J., & Faraone, S. (1996). Diagnosis and treatment of adult attention-deficit/hyperactivity disorder. In M. H. Pollack, M. W. Otto, & J. F. Rosenbaum (Eds.), *Challenges in clinical practice: Pharmacologic and psychosocial strategies* (pp. 380–407). New York: Guilford Press.
- Brown ADD Scales. (1996). Copyright © 1996 by The Psychological Corporation, Harcourt Brace & Company.
- Brown, T. E. (1996). *Brown Attention-Deficit Disorder Scales: Manual*. San Antonio, TX: The Psychological Corporation.
- Carlin, B. P., & Louis, T. A. (2000) Bayes and empirical Bayes methods for data analysis (2nd ed.). Washington DC: Chapman & Hall/CRC.
- Coldren, J. T., & Corradetti, K. (1997). Conceptual relations between attentional process in infants and children with attention deficit/hyperactivity disorder: A problem solving approach. In J. A. Burack & J. T. Enns (Eds.), *Attention, development, and psychopathology* (pp. 147–167). New York: Guilford Press.
- Comings, D. E. (1997). Genetic aspects of childhood behavioral disorders. *Child Psychiatry and Human Development*, *27*, 139–150.
- Cox, D. J., Kovatchev, B. P., Morris, J. B., Phillips, C., Hill, R., & Merkel, L. (1999). Electroencephalographic and psychometric differences between boys with and without Attention-Deficit/Hyperactivity Disorder (ADHD): A pilot study. *Applied Psychophysiology and Biofeedback*, *23*, 179–188.
- Crawford, H., & Barabasz, M. (1996). Quantitative EEG magnitudes in children with and without attention deficit disorder during neurological screening and cognitive tasks. *Child Study Journal*, *26*, 71–86.
- Denckla, M. B. (1993). The child with developmental disabilities grown up: Adult residua of childhood disorders. *Neurologic Clinics*, *11*, 105–125.
- Epstein, M. A., Shaywitz, S. E., Shaywitz, B. A., & Wollston, J. L. (1991). The boundaries of attention deficit disorder. *Journal of Learning Disabilities*, *24*, 78–86.
- Eugene, A. L. (1996). Sex differences in ADHD: Conference summary. *Journal of Abnormal Child Psychology*, *24*, 555–569.
- Fossati, A., Di Ceglie, A., Acquarini, E., Donati, D., Donini, M., Novella, L., et al. (2001). The retrospective assessment of childhood attention-deficit hyperactivity disorder in adults: Reliability and validity of the Italian version of the Wender Utah Rating Scale. *Comprehensive Psychiatry*, *42*, 326–336.
- Gittleman, R., Mannuzza, S., Shenker, R., & Bonagura, N. (1985). Hyperactive boys almost grown up: I. Psychiatric status. *Archives of General Psychiatry*, *42*, 937–947.
- Goldstein, S. (1997). A pilot study of neurological function in girls with ADHD: Comment. *Journal of the American Academy of Child and Adolescent Psychiatry*, *36*, 1162.
- Grunewald-Zuberbier, E., Grunewald, G., & Raske, A. (1975). Hyperactive behavior and EEG arousal reactions in children. *Electroencephalographic and Clinical Neurophysiology*, *42*, 149–159.
- Hart, E. L., Lahey, B. B., Loeber, R., Applegate, B., & Frick, P. J. (1995). Developmental change in attention-deficit hyperactivity disorder in boys: A four year longitudinal study. *Journal of Abnormal Child Psychology*, *23*, 729–749.
- Horner, B. R., & Scheibe, K. E. (1997). Prevalence and implications of attention-deficit hyperactivity disorder among adolescents in treatment for substance abuse. *Journal of the American Academy of Child and Adolescent Psychiatry*, *36*, 30–36.

- Kovatchev, B. P., Cox, D. J., Hill, R., Reeve, R., Robeva, R. S., & Loboschefska, T. (2001). A psychophysiological marker of Attention Deficit/Hyperactivity Disorder- Defining the EEG consistency index. *Applied Psychophysiology and Biofeedback, 26*, 127–139.
- Landau, S., Lorch, E. P., & Milich, R. (1992). Visual attention to and comprehension of television in attention-deficit hyperactivity disorder and normal boys. *Child Development, 63*, 928–937.
- Mann, C. A., Lubar, J. F., Zimmerman, A. W., Miller, C. A., & Muenchen, R. A. (1991). Quantitative analysis of EEG in boys with attention-deficit-hyperactivity disorder: Controlled study with clinical implications. *Pediatric Neurology, 8*, 30–36.
- Merkel, R. L., Cox, D. J., Kovatchev, B. P., Morris, J., Seward, R., Hill, R., et al. (2000). The EEG consistency index as a measure of Attention Deficit/Hyperactivity Disorder and responsiveness to medication: A double blind placebo controlled pilot study. *Applied Psychophysiology and Biofeedback, 25*, 133–142.
- Monastra, V. J., Lubar, J. F., Linden, M., Van Deusen P., Green, G., Wing, W., et al. (1999). Assessing attention deficit hyperactivity disorder via quantitative electroencephalography: An initial validation study. *Neuropsychology, 13*(3), 424–433.
- Murphy, K., & Barkley, R. A. (1996). Attention deficit hyperactivity disorder adults: Comorbidities and adaptive impairments. *Comprehensive Psychiatry, 37*, 393–401.
- Shaffer, D. (1994). Attention deficit disorder in adults. *American Journal of Psychiatry, 151*, 633–638.
- Shaywitz, B. A., & Shaywitz, S. E. (1991). Comorbidity: A critical issue in attention deficit disorder. *Journal of Child Neurology, 6*(Suppl. 1), 13–22.
- Silverthorn, P., Frick, P. J., Kuper, K., & Ott, J. (1996). Attention deficit hyperactivity disorder and sex: A test of two etiological models to explain the male predominance. *Journal of Clinical Child Psychology, 25*, 52–59.
- Stein, M. A., Fischer, M., & Szumowski, E. (1999). Evaluation of adults for ADHD. *Journal of the American Academy of Child and Adolescent Psychiatry, 38*, 940–941.
- Stein, M. A., Fischer, M., & Szumowski, E. (2000). Evaluation of adults for ADHD: Erratum. *Journal of the American Academy of Child and Adolescent Psychiatry, 39*, 674.
- Ward, M. F., Wender, P. H., & Reimherr, F. W. (1993). The Wender Utah Rating Scale: An aid in the retrospective diagnosis of childhood Attention-deficit-hyperactivity disorder. *American Journal of Psychiatry, 150*, 885–890.
- Weiss, G. (1985). Follow up studies on outcome of hyperactive children. *Psychopharmacology Bulletin, 21*, 169–177.
- Wender, P. H. (1995). *Attention-deficit hyperactivity disorder in adults*. Oxford University Press: New York.
- Weyandt, L. L., Linterman, I., & Rice, J. A. (1995). Reported prevalence of attentional difficulties in a general sample of college students. *Journal of Psychopathological and Behavioral Assessment, 17*, 293–304.
- Zametkin, A. J., Nordahl, T. E., Gross, M., King, A. C., Semple, W. E., Rumsey, J., et al. (1990). Cerebral glucose metabolism in adults with hyperactivity of child onset. *New England Journal of Medicine, 323*, 1361–1366.
- Zametkin, A. J., & Rapoport, J. L. (1987). Neurobiology of attention deficit disorder with hyperactivity: Where have we come in 50 years? *Journal of the American Academy of Child and Adolescent Psychiatry, 26*, 676–686.

Copyright of Applied Psychophysiology & Biofeedback is the property of Kluwer Academic Publishing and its content may not be copied or emailed to multiple sites or posted to a listserv without the copyright holder's express written permission. However, users may print, download, or email articles for individual use.