Journal of the International Neuropsychological Society (1999), **5**, 222–233.

Neuropsychological performance of adults with attention deficit hyperactivity disorder (ADHD): Diagnostic classification estimates for measures of frontal lobe/executive functioning

DAVID W. LOVEJOY,[1,2] J.D. BALL,[3] MATTHEW KEATS,[3] MICHAEL L. STUTTS,[3] EDWARD H. SPAIN,[3] LOUIS JANDA,[4] AND JENNIFER JANUSZ[2]

[1]Hartford Hospital and The Institute of Living
[2]The Virginia Consortium Program in Clinical Psychology
[3]Eastern Virginia Medical School
[4]Old Dominion University

(RECEIVED December 19, 1997; REVISED June 12, 1998; ACCEPTED June 22, 1998)

Abstract

ADHD adults ($N = 26$) were compared to normal controls ($N = 26$) on 6 neuropsychological measures believed sensitive to frontal lobe–executive functioning. MANOVA analyses and subsequent univariate tests indicated that most of the neuropsychological measures discriminated between the two groups. To address clinical significance, diagnostic classification rates were also generated for each measure individually, and for the battery as a whole. Levels of positive predictive power (PPP) for each of the 6 measures (83–100%) indicated that abnormal scores on these tests were good predictors of ADHD. However, estimates of negative predictive power (NPP) suggested that normal scores poorly predicted the absence of ADHD. When classification rates were calculated for the overall battery classification accuracy improved substantially. Thus, neuropsychological tests can differentiate adults suffering from ADHD from adults without ADHD, while also providing good classification accuracy. Finally, the pattern of neurobehavioral impairments exemplified through the Summary Index scores was interpreted as consistent with conceptualizations of ADHD depicting mild neurologic dysfunction in networks associated with the frontal lobes. (*JINS*, 1999, *5*, 222–233.)

Keywords: Adult ADHD, Neuropsychological tests, Frontal lobe, Executive functioning

INTRODUCTION

Attention deficit hyperactivity disorder (ADHD) is marked by developmentally inappropriate degrees of inattentiveness, hyperactivity, and impulsivity. It is among the most common of childhood psychological problems, with an incidence of 5 to 7% for boys and 2 to 4% for girls (for review, see Barkley, 1990). Symptoms of ADHD first appear early in childhood with a mean age of onset between infancy and 7 years (Barkley et al., 1988). At present, the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition* (*DSM–IV*; American Psychiatric Association, 1994) allows for four diagnostic types of ADHD including a predominantly inattentive type, a predominantly hyperactive–impulsive type, a type that combines inattention with hyperactivity, and a not-otherwise-specified type.

Once conceptualized as a childhood disorder that resolved in adolescence, recent research clearly indicates ADHD persistence into adulthood for 30 to 50% of childhood cases (Mannuzza et al., 1991; Shekim et al., 1990). Biederman et al. (1995a) note that ADHD represents a substantial public health problem given costs to society from ADHD-related impairments in cognitive, social, familial, scholastic, and occupational functioning. The authors further note that an estimated 2% incidence of ADHD in adults makes this a fairly common and underidentified adult disorder.

While a neurological etiology is not necessary for a diagnosis of ADHD, converging evidence supports the premise that many cases of ADHD have a neurophysiological

Reprint requests to: David W. Lovejoy, The Institute of Living, Butler Building, Department of Psychology, 400 Washington Street, Hartford, CT 06106. E-mail: dlovejo@harthosp.org

basis. Indeed, it is estimated that 30 to 50% of pediatric ADHD cases are hereditary (for review, see Barkley, 1990). Those cases that reach adulthood appear to have even stronger familial–genetic etiologic risk factors, as 84% of ADHD adults examined in a recent study had at least 1 child with the disorder (Biederman et al., 1995a). Contemporary etiologic conceptualizations of ADHD implicate frontal cortical regions and interconnected subcortical structures such as the substantia nigra, striatum, and reticular activating system (Boucugnani & Jones, 1989; Denckla, 1989, 1991, 1993, 1996; Hynd et al., 1991a; Mattes, 1980). Such conceptualizations are also consistent with functional paradigms of attention that emphasize the role of the frontal lobes (Luria, 1966, 1973; Mesulam, 1986, 1990; Posner & Dehaene, 1994; Posner & Presti, 1987).

An abundance of clinical and research evidence supports theories of underlying frontal lobe dysfunction. For instance, similarities have been noted between the neurobehavioral sequelae of traumatic brain injury involving the frontal lobes and ADHD symptomatology (Evans et al. 1987). These observations are also consistent with animal models demonstrating that frontal lesions often produce a range of behaviors similar to the symptoms of ADHD (Mattes, 1980). Stimulant medications, which are largely the first line of medication therapy for ADHD, have also proven useful in treating the inattention, distractibility, and impulsivity associated with certain forms of traumatic brain injury (Evans et al. 1987; Gualtieri & Evans, 1988; Kløve, 1987; Weinstein & Wells, 1981). It is believed that these medications serve to ameliorate deficiencies in catecholamines such as dopamine and norepinephrine which maintain rich connections with the frontal lobes (Shenker, 1992). Researchers (Satterfield & Dawson, 1971; Satterfield et al., 1973, 1974) have shown that ADHD individuals appear to have increased levels of slow-wave (theta wave) activity in anterior areas of the cerebral cortex, supporting a hypothesis that ADHD symptomatology reflects an underarousal of the cerebral cortex. Indeed, catecholamine deficiencies in frontal regions may play a role in such an underarousal, accounting in part for the paradoxical effects of stimulants in treating ADHD. Neuroimaging studies have also demonstrated reduced frontal perfusion of blood flow in orbital and prefrontal regions (Lou et al., 1984, 1989), metabolic deficiencies of blood glucose metabolism in frontal areas (Ernst et al., 1994; Zametkin et al., 1990) and anterior structural differences (Giedd et al., 1994; Hynd et al., 1990, 1991b) in ADHD groups as compared with normal controls.

Cognitive deficits have long been thought to underlie some of the difficulties in adaptation experienced by those with ADHD (Douglas, 1972). Denckla (1989, 1993) explained that the convergence in evidence indicating similarities between ADHD and a dysfunction of the frontal lobes has convinced some neuropsychologists to employ "frontal lobe" batteries with ADHD clients, and that these batteries often reveal patterns of impairment similar in nature to what one might anticipate in cases of traumatic brain injury that involve the frontal lobes. The neurocognitive abilities assessed by such batteries largely fall within the arena of executive functions. These abilities include selective and sustained attention, inhibition of verbal and nonverbal responses, strategic memorization, working memory, organization, self-monitoring, planning and sequencing of complex behaviors, and management of time and space. Such abilities are often conceptualized as extending above and beyond intelligence, allowing one to productively harness and utilize one's intellectual abilities. As Denckla (1993) points out, despite high levels of intelligence, executive dysfunction can affect a wide variety of abilities leading to daily inefficiency and impairment in academic, interpersonal, and occupational spheres.

Barkley (1997) recently put forth a unifying theory that attempts to further delineate the nature of the frontal lobe impairment and executive deficits observed in ADHD. In this theory, he states that ADHD is characterized by a primary impairment in response inhibition which, in turn, leads to secondary deficits in four executive functions (working memory, regulation of affect, internalization of speech, and reconstitution) that are dependent on inhibition for their execution. These secondary deficits serve to decrease the individual's ability to regulate motor control through "internally represented information and self-directed action." This cascade of executive impairments is believed to be directly attributable to a dysfunction of the prefrontal cortex and may create the appearance of poor sustained attention in those suffering from ADHD.

Because adult ADHD is a relatively new diagnostic entity, studies employing frontal lobe tasks have largely focused on pediatric ADHD populations. Barkley et al. (1992) reviewed 22 neuropsychological studies of frontal functions in children with attention deficit disorder with and without hyperactivity. They concluded that although statistically significant group differences were often found between ADHD and normal control groups, only a handful of tests seemed to offer reliability in differentiating groups across studies. These tests included: Continuous Performance Tasks (CPTs), the Stroop test, the Hand Movements test, and go–no-go tests. The authors also noted that all of the studies reviewed differed greatly in a number of methodological procedures making clear comparisons across studies difficult. Authors (Dodrill, 1997; Reitan & Wolfson, 1995) have also noted, with regard to studies that employ tests of frontal lobe functioning, that there are no known neuropsychological tests shown to be exclusively sensitive to frontal lesions. In addition, the application of frontal lobe tests to distinguish children with ADHD from those without the disorder seem to disregard consensus among neurodevelopmentalists that frontal lobe functioning is not fully developed until late adolescence or early adulthood (Golden, 1981; Luria, 1980; Weinberger, 1987).

More recent studies continue to show group differences on some measures of executive functioning but not on others (Cahn & Marcotte, 1995; Grodzinsky & Diamond, 1992; Reader et al., 1994). Consistent with the premise that familial ADHD status reflects a meaningful biologic subtype of ADHD (Faraone & Biederman, 1994), Seidman et al. (1995)

found that a family history of ADHD was a strong predictor of neuropsychological impairment, in that ADHD persons with a family history of ADHD showed greater impairment than ADHD persons without a family history on certain measures of executive functioning.

A small number of studies have expanded the focus of executive dysfunction and ADHD to adult populations. Hopkins et al. (1979) found that hyperactive adults performed significantly more poorly than a group of controls on measures such as the Matching Familiar Figures Test, the Embedded Figures Test, and the Stroop Test. Mungas (1983) found that after five word list learning trials on the Rey Auditory Verbal Learning Test, they were able to discriminate between groups of amnestics, severe head trauma victims, ADHD patients, schizophrenics, and nonpsychotic psychiatric patients with a delayed recall trial following a distracter list. The ADHD adults showed impairments similar to those of the nonpsychotic and schizophrenic inpatients, but performed better than amnestic and head trauma patients. Similarly, Holdnack et al. (1995) found mild-to-moderate acquisition deficits on the California Verbal Learning Test with average to above average verbal intellectual abilities. The ADHD participants in this study acquired less information than controls across learning trials and displayed an inconsistent pattern of semantic clustering. Jenkins et al. (1996) found that adults with complaints of diminished attention and concentration who had a prior history of childhood ADHD could be differentiated neuropsychologically on the PASAT–R, the California Verbal Learning Test, and the Controlled Oral Word Association Test. Trends toward worse performance for those with ADHD histories were also evident on Digits Backward, the Recurring Figures Test, and reciprocal motor alternation tasks. Finally, Rugle and Melamed (1993) showed that a group of nonsubstance abusing gamblers with childhood histories indicative of ADHD performed significantly more poorly on "higher order" measures of attention or frontally mediated executive functions, such as the Embedded Figures Test, the Wisconsin Card Sorting Test, and the Porteus Maze Test, than did a group of matched controls. Significant differences were not noted on neuropsychological measures such as the Trail Making Test Part B, or the List Learning Test with Categorical Clustering.

Although statistically significant differences have been found between ADHD and normal control groups on neuropsychological tests believed to measure frontal lobe functions, these findings do not offer clinicians adequate information for making diagnostic decisions. Retzlaff and Gibertini (1994) explained that statistical procedures used in the analysis of neuropsychological data are often not ecologically useful for the clinician, and that group statistical analyses are of limited value when making decisions about individual patients. Indeed, Barkley and Grodzinsky (1994) point out that group means may differ significantly on an instrument with only a minority of cases in one group having displayed impaired scores on that instrument. Elwood (1993) put forth that classification probabilities such as positive predictive power (PPP), negative predictive power (NPP), sensitivity, and specificity are useful in characterizing the diagnostic efficiency of instruments. PPP, the ratio of true positive cases to all test positives, refers to the chances that a person with impaired test score actually has the disorder in question. NPP, the ratio of true negatives to all test negatives, refers to the chance of a person without an impaired test score not having the disorder in question. The reciprocal of PPP is the false positive and the reciprocal of NPP is the false negative. Sensitivity is the proportion of individuals that have the disorder in question and obtain an impaired score on the instrument. Specificity is the proportion of individuals that do not have the disorder in question and obtain an unimpaired score on the instrument.

Two recent studies have examined the classification accuracy of neuropsychological tests of frontal lobe functions with pediatric ADHD populations (Barkley & Grodzinsky, 1994; Grodzinsky & Barkley, 1997). Barkley and Grodzinsky (1994) examined children diagnosed with hyperactivity (ADD + H), ADD without hyperactivity (ADD − H), LD, or controls. Although classification rates were unacceptable when ADD + H or ADD − H diagnoses were considered separately, PPP was interpreted as acceptable for two of the eight measures (the Continuous Performance Test scores and Controlled Oral Word Association Test) when the ADD groups were combined. Unfortunately, estimates for negative predictive power and false negatives for these measures remained unacceptable, leading to the conclusion that while abnormal scores were predictive of ADHD, normal scores were not predictive of an absence of ADHD. Correcting for methodological problems inherent in their earlier study, Grodzinsky and Barkley (1997) next examined the classification accuracy of eight frontal lobe tests with a larger sample of children diagnosed with ADHD–combined type. The findings indicated that abnormal scores on four of the tests (Stroop test, KABC Hand Movements subtest, COWAT–FAS version, and a computerized CPT) were good indicators of ADHD, but again, normal scores did not indicate an absence of ADHD.

The present study seeks to expand existing knowledge regarding neuropsychological correlates of ADHD and to further define the utility of neuropsychological measures for the diagnosis of adults with ADHD. By utilizing an adult population, the present study avoids problems associated with immature frontal lobe functioning in children. As Grodzinsky and Diamond (1992) pointed out, differences between those with ADHD and controls will be apparent only when the frontal functions of controls have reached full maturity. Additionally, utilizing adult participants avoids psychometric concerns that most measures shown to be sensitive to frontal lobe functions were designed for and subsequently validated with adults, rather than children (Welsh & Pennington, 1988). The present study also addresses some of the methodological difficulties present in earlier studies, such as small sample sizes and a failure to attend to learning disorders in ADHD or control samples. Since ADHD behavior is marked by its inconsistency, the present study employed

a "battery approach" with the hypothesis that ADHD behavior should be present somewhere within a group of tests, even if not always present on the same test.

It was predicted that the ADHD adult group would perform more poorly than non-ADHD adults on tests of executive–frontal lobe functioning, as reflected in significance testing of group means. It was also expected that test scores for ADHD adults would fall within clinically impaired ranges when compared to published normative data for these tests, and that an analysis of classification rates would suggest that these tests are useful in establishing a diagnosis of ADHD in adults. Finally, it was hypothesized that a Summary Index derived from the normative-adjusted test scores would yield optimal classification rates.

METHODS

Research Participants

Twenty-six ADHD participants were recruited with a brief informational notice and from treating psychiatrists. All ADHD participants met diagnostic criteria for ADHD as evidenced by a clinical interview performed by a board certified psychiatrist who specialized in ADHD and the participant's own endorsement of sufficient *DSM–IV* symptoms to meet criteria for either an *inattentive*, *hyperactive–impulsive*, or *combined* subtype of ADHD. Possible comorbid Axis I diagnoses, such as depression and anxiety, were also screened for as part of the clinical interview. In an effort to better identify individuals with clear evidence of ADHD, this study utilized only ADHD participants who were currently taking stimulant medications (methylphenidate or dextroamphetamine only) and who indicated on a questionnaire that stimulant medications were "very helpful" to them in addressing ADHD symptomatology. Individuals taking other psychoactive medications along with or instead of stimulants were excluded from the study.

Twenty-six control group participants were recruited from clinic waiting rooms in medical practice settings and from participant referrals of friends or spouses without ADHD symptoms. All participants in the control group endorsed three or fewer ADHD symptoms of either an inattentive or hyperactive–impulsive subtype of ADHD on a *DSM–IV* checklist, and had no history of taking stimulant medications for attentional difficulties.

Participants from both ADHD and control groups were matched on variables such as age, education, sex, and IQ. Estimated IQ was assessed with a short form of the Wechsler Adult Intelligence Scale–Revised (WAIS–R) which consisted of the Information and Picture Completion subtests. IQ indices based on these subtests have been shown to correlate .88 with Full Scale WAIS–R IQ scores in the WAIS–R standardized sample, and together this dyad has an average reliability coefficient of .90 (Kaufman, 1990).

Exclusion criteria for both groups included the following: (1) scores less than 85 on an estimate of intellectual functioning based on a short form of the WAIS–R, (2) a history of previously diagnosed psychiatric disorders other than ADHD or substance abuse difficulties, (3) history of neurologic disease or head injury, and (4) a previous diagnosis or special education remediation for specific learning disabilities.

Procedures

Eligible study participants were seen separately for 1-hr assessment appointments. At the time of assessment, the participants gave informed consent and were asked to complete a background questionnaire and a *DSM–IV* ADHD self-report questionnaire. Handedness was also assessed at this time by asking about the hand with which the participant prefers to write. All dependent measures were then administered in the same standardized order by the principal investigator or a research assistant. Each instrument was administered in accordance with published standardized administration procedures. The tests were then scored blind by the principal investigator using standardized scoring criteria from test manuals. Finally, test scores were transferred to a data summary sheet and participants' names were replaced with numerical codes for data analysis. All ADHD participants receiving stimulant medication were drug-free for a period of 12 hr before testing to be certain that they were not benefiting from medication at the time of testing.

Dependent Measures

The Stroop Neuropsychological Screening Test (SNST)

Believed to be sensitive to inhibition and impulsivity, the SNST (Trennery et al., 1988) has been shown to differentiate participants with frontal lesions from participants with temporal lesions, posterior brain damage, and control participants (Lezak, 1995; Trennery et al., 1988). Suggested clinical cut-off scores from the standardization sample (Trennery et al., 1988) for the color–word score were used in the Summary Index (20–21st percentile for age 18–49 and the 11th percentile for age 50 or over).

The Trail Making Test Parts A and B

The Trail Making Test (Reitan & Wolfson, 1985) is administered in two parts (A and B). Reviews of this measure (Lezak, 1995; Spreen & Strauss, 1991) indicate that Part B of this instrument is extremely sensitive to a wide range of brain impairing conditions. Trails B is the more complex of the two and is believed to tap executive functions such as shifting of attentional set, working memory, and novel reasoning. Although Trails A is less complex, it is the first of the two tests to be administered, and it may prove difficult for participants who have trouble orienting to new problem solving situations. The scores used from these measures include the total time to complete Part A and total time to complete Part B. *T* scores of 39, which fall 1 standard de-

viation below norm-referenced mean performance (Heaton et al., 1991), were used as clinical cut-off scores in the Summary Index.

The California Verbal Learning Test (CVLT)

The CVLT (Delis et al., 1987) is a list learning task believed to tap organization skills, concept formation, and working memory. While memory is often linked to the integrity of the temporal lobes, there is evidence to associate mild deficits in verbal list-learning to frontal lobe dysfunction (Stuss et al., 1994). The score from the Short-Delay Free Recall index was used in the present study, and provides insight into how well participants have organized, learned, and retained verbal information after five presentations of an initial list and an interference trial. Because recent research has shown that the CVLT often overestimates memory impairment by more than 1 standard deviation (Randolph et al., 1994), a clinical cut-off score of 2 standard deviations below norm-referenced mean performance was selected for use in the Summary Index.

The Controlled Oral Word Association Test (COWA)

The COWA (CFL version; Benton & Hamsher, 1983) is a test of word fluency that consists of three timed word-naming trials. This task measures spontaneous verbal production and organization while requiring participants to rely upon rule-governed strategies for retrieval of verbal information under strict time constraints. The score is the sum of all admissible words for the three letters. Reviews (Lezak, 1995; Spreen & Strauss, 1991) note that this test has been found to be sensitive to frontal lobe damage. A clinical cut-off score below the 16th percentile (*low-average–borderline* range) was utilized for the Summary Index (Ruff et al., 1996).

The WAIS–R Freedom From Distractibility Factor

Finally, two subtests (Arithmetic and Digit Span) that make up the Freedom From Distractibility Factor on the Wechsler Adult Intelligence Scale–Revised (WAIS–R) were also included in the battery. This factor is believed to measure attention–concentration skills and working memory. For the present study, a score calculated by averaging the age-adjusted scaled scores for the Digit Span and Arithmetic subtests was utilized. A score of 7, which is 1 standard deviation below mean performance, was selected as the cut-off. For the Summary Index, a difference score was calculated by subtracting the average of age-adjusted scaled scores for Information and Picture Completion from those of Digit Span and Arithmetic. This score was designed to reflect a personalized pattern of performance estimate such that varying degrees of divergence between subtests used to calculate a Full Scale IQ estimate and subtests used to calculate the Freedom From Distractibility factor of the WAIS–R could be identified.

The Summary Index

The scores that make up the Summary Index were calculated using the standardized normative samples for each of the six previously mentioned measures. As a result, the index represents an estimate of impairment that could be easily understood and applied across a variety of clinical and research contexts. It was constructed to reflect a "battery approach" to evaluating sensitivity, specificity, positive predictive power, and negative predictive power. Because ADHD is thought to manifest as an inconsistency in cognitive–attentional abilities (Leimkuhler, 1994), and those with ADHD can often rally their attention for brief periods of time (especially in a one-to-one testing situation) the index was developed to account for the possibility that these individuals may not consistently display cognitive difficulties on any one particular measure. Since cognitive weaknesses might be displayed inconsistently during a battery of tests, an index score reflective of the entire battery was thought to be more efficient in detecting ADHD in adults. The Summary Index was calculated by taking the number of scores in the clinically impaired range and dividing that by the number of tests administered, with greater numeric values corresponding to greater levels of impairment.

RESULTS

The sample ranged in age from 21 to 55, with a median age of 41. Male (50%) and female (50%) participants were equally represented. Educational level ranged from 12 to 20 years, with a median of 16 years. Estimated IQ scores ranged from 91 to 130, with a median score of 115. With regard to handedness, 19% of the ADHD group and 15% of the control group were left-handed. Finally, 65% of the ADHD group as compared to 7% of the control group had first degree relatives diagnosed with ADHD.

Comparison of Group Means

The group mean data is shown in Table 1. A series of univariate analyses of variance (ANOVAs) indicated that there were no significant differences between the two groups with regard to age [$F(1,50) = 0.12$, $p > .05$], education [$F(1,50) = .25$, $p > .05$], or estimated IQ [$F(1,50) = .02$, $p > .05$]. Chi-square tests revealed no significant differences in sex [$\chi^2(1, N = 52) = 0.0$, $p \geq .05$] or handedness [$\chi^2(1, N = 52) = 0.14$, $p \geq .05$]. ADHD adults did have significantly more first degree relatives that carry a diagnosis of ADHD than did non-ADHD adults [$\chi^2(1, N = 52) = 20.63$, $p < .0001$].

Raw scores for the six measures were analyzed utilizing a multivariate analysis of variance (MANOVA) to test overall group differences considering all six dependent measures collectively. The resulting analysis revealed highly significant group differences in neuropsychological functioning in relation to participants' group assignment [ADHD or non-ADHD; $F(5,46) = 8.90$, $p < .0001$; Wilks's Lambda = 0.51].

Table 1. Neuropsychological test results by diagnostic group with *F*-test values

	Group assignment				
	ADHD ($N = 26$)		Control ($N = 26$)		
Measure	*M*	*SD*	*M*	*SD*	*F*
COWA	29.35	10.30	39.92	7.14	18.52****
CVLT	11.00	2.91	12.38	2.32	3.60
Stroop	98.15	13.99	110.42	4.33	18.24****
Trails A	27.12	7.17	21.04	6.51	10.23**
Trails B	61.38	18.17	51.54	16.41	4.21*
WAIS–R Distractibility	10.81	2.02	12.19	1.53	7.76**
Overall MANOVA: [$F(5,46) = 8.90$, $p < .0001$; Wilks's Lambda = 0.51].					

Note. COWA = Controlled Oral Word Association Test; CVLT = California Verbal Learning Test; SNST = Stroop Neuropsychological Screening Test; Trails A = Trail Making Test Part A; Trails B = Trail Making Test Part B; WAIS–R Distractibility = Freedom From Distractibility Factor Score from the WAIS–R. Means with different superscripts differ significantly at $p < .05$.
*$p < .05$, **$p < .01$, ***$p < .001$, ****$p < .0001$.

Following the MANOVA analysis, a series of individual univariate analyses of variance (ANOVAs) were conducted to determine which specific test variables accounted for the overall significant MANOVA. The univariate follow-up tests revealed significant effects for ADHD status for five of the six neuropsychological measures used in the present study. The results indicated that the ADHD adults had significantly lower verbal fluency on the COWA [$F(1,50) = 18.52$, $p < .0001$], completed significantly fewer items on the color-word interference task of the Stroop Neuropsychological Screening Test [$F(1,50) = 18.24$, $p < .0001$], took significantly more time to complete the Trail Making Test–Part A [$F(1,50) = 10.23$, $p < .002$] and Part B [$F(1,50) = 4.21$, $p < .05$], and performed significantly more poorly on the Freedom From Distractibility Factor of the WAIS–R [$F(1,50) = 7.76$, $p < .008$] than did the non-ADHD controls. No significant differences were observed between groups for verbal recall on the Short Delay Free Recall index of the CVLT [$F(1,50) = 3.60$, $p > .05$].

Classification Accuracy for the Dependent Measures

Diagnostic classification estimates for the dependent measures are shown in Table 2. In addition to the classification rates, chi-square tests were conducted to test for differences between group assignment (ADHD *vs.* non-ADHD) on the presence or absence of clinical impairment using the norm-referenced cut-off criteria for each measure. Chi-square analyses were also performed for each level of the Summary Index. All chi-square values were significant for the dependent measures and Levels 1 through 3 of the Summary Index at the .05 level of confidence, with many reaching greater levels of significance.

With regard to the operating characteristics for these measures, despite lower levels of sensitivity (19–58%) or the ability of these measures to classify large numbers of those in the ADHD group, higher PPP (83–100%) and lower false positive rates (0–17%) indicated that when abnormal scores

Table 2. Operating characteristics of neuropsychological tests for ADHD adults relative to normal controls, with chi-square values indicating significant clinical differences between groups

Tests	OPP	Sens.	Spec.	PPP	NPP	FP	FN	χ^2
COWA	.75	.58	.92	.88	.69	.12	.31	16.2****
CVLT	.65	.38	.92	.83	.60	.17	.40	7.43**
SNST	.62	.23	1.0	1.0	.57	0.0	.43	9.1**
Trails A	.60	.19	1.0	1.0	.55	0.0	.45	7.46**
Trails B	.60	.23	.96	.86	.56	.14	.44	4.52*
WAIS Distractibility	.69	.38	1.0	1.0	.62	0.0	.38	16.3****

Note. OPP = overall predictive power; Sens = sensitivity; Spec = specificity; PPP = positive predictive power; NPP = negative predictive power; FP = false positives, FN = false negatives; COWA = Controlled Oral Word Association Test; CVLT = California Verbal Learning Test; SNST = Stroop Neuropsychological Screening Test; Trails A = Trail Making Test Part A; Trails B = Trail Making Test Part B; WAIS Distractibility = average of the age-adjusted scaled scores for the Arithmetic and Digit Span subtests. Measures with different superscripts indicate that groups differed significantly at $p < .05$, as indicated by chi-square analyses.
*$p < .05$, **$p < .01$, ***$p < .001$, ****$p < .0001$.

were obtained on these measures, they were good predictors of adult ADHD. NPP (55–69%) and false negative rates (31–45%), on the other hand, indicated that normal scores should not be interpreted, as they are not generally predictive of an absence of ADHD. Specific findings for each of the six measures were as follows:

1. Of the six neuropsychological measures, the Controlled Oral Word Association Test (COWAT), with a normative-referenced clinical cut-off score below the 16th percentile, showed the highest OPP (75%). This score reflects a specificity estimate of 92%, and a sensitivity estimate of 58%, indicating that 42% of adults with ADHD scored in the normal range. The PPP estimate for this measure indicated that 88% of those that obtained abnormal scores were correctly classified as having ADHD. The false positive rate (reciprocal of PPP) was also low, with only 12% of non-ADHD participants obtaining abnormal scores. However, the NPP estimate indicated that only 69% of those with normal scores were correctly classified as being non-ADHD. The false negative rate (reciprocal of NPP) indicated that 31% of those obtaining normal scores were in the ADHD group.
2. The Short-Delay Free Recall Index of the California Verbal Learning Test (CVLT), with a normative-referenced clinical cut-off of 2 standard deviations below mean performance, showed an OPP of 65%, reflecting a specificity estimate of 92%, and a sensitivity estimate of 38%, indicating that 62% of adults with ADHD scored in the normal range. The PPP estimate for this measure indicated that 83% of those that obtained abnormal scores were correctly classified as having ADHD. The false positive rate fell at 17%. The NPP estimate fell at 60% and the false negative rate for this measure indicated that 40% of those obtaining normal scores were in the ADHD group.
3. The OPP for the Stroop Neuropsychological Screening Test, using the normative-referenced clinical cut-off scores recommended by the test authors for brain impairment (21st percentile for ages 18–49 and 11th percentile for ages 50+), was 62%, reflecting a specificity estimate of 100%, and a sensitivity estimate of 23%. Thus, 77% of adults with ADHD scored in the normal range. The PPP estimate for this measure was 100%, indicating that 100% of those that obtained abnormal scores were correctly classified as having ADHD. Accordingly, the false positive rate was nonexistent at 0%. However, the NPP estimate was 57% and the false negative rate indicated that 43% of ADHD adults achieved normal scores on this measure.
4. Part A of the Trail Making Test, with a normative-referenced clinical cut-off score of 1 standard deviation below mean performance, showed an OPP at 60%, reflecting a specificity estimate of 100% and a sensitivity estimate of 19%, indicating that 81% of the ADHD adults scored within the normal range. The PPP estimate for this measure fell at 100%, indicating that 100% of those that obtained abnormal scores were correctly classified as having ADHD. Accordingly, the false positive rate was also low (0%). The negative predictive power estimate fell at 55%, with the false negative rate indicating that 45% of those achieving normal scores were in the ADHD group.
5. Part B of the Trail Making Test, with a normative-referenced clinical cut-off score of 1 standard deviation below mean performance, showed an OPP at 60%, resulting from a specificity estimate of 96% and a sensitivity estimate of 23%, indicating that 77% of adults with ADHD scored in the normal range. The PPP estimate for this measure fell at 86% and the false positive rate was 14%. The NPP estimate was 56% and the false negative rate indicated that 44% of those achieving normal scores were in the ADHD group.
6. The WAIS–R Freedom From Distractibility factor, with a normative-referenced clinical cut-off score at 1 standard deviation below mean performance, displayed an OPP of 69%. This overall classification rate was marked by a specificity estimate of 100% and a sensitivity estimate of 38%, indicating that the majority (62%) of ADHD adults scored within the normal range. The PPP estimate for this measure indicated that 100% of those that obtained abnormal scores were classified as having ADHD. Accordingly, the false positive rate was extremely low (0%). The NPP estimate indicated that 62% of those with normal scores were correctly classified as being non-ADHD and the false negative rate showed that 38% of the ADHD adults obtained normal scores on this measure.
7. Substantial increases in classification accuracy were observed for the battery as a whole, as compared with the classification rates for any of the six neuropsychological tests alone. Improved classification rates were evident for Summary Index scores that reflected one or more and two or more impaired tests out of the battery of six neuropsychological tests. One or more and two or more tests impaired out of the six tests in the battery, or Summary Index scores of .16 and .33 yielded the highest OPP (90 and 83%, respectively). These overall classification scores reflected higher specificity estimates (85 and 96%, respectively) and higher sensitivity estimates (96 and 69%, respectively). PPP estimates (86 and 95%, respectively) for these levels of impairment on the overall battery indicated that the vast majority of those that obtained abnormal scores were correctly classified as having ADHD. The false positive rates (14 and 5%, respectively) indicated that few non-ADHD participants obtained an abnormal score within the battery. The NPP estimates (96 and 76%, respectively) indicated that the majority of those with unimpaired scores were correctly classified as being non-ADHD, with false negative rates (reciprocal of NPP) of 4 and 24%, respectively. Estimates of specificity and PPP increased to 100% for

index scores reflecting three or more and four or more impaired tests within the battery, but, at this level of impairment on the battery, NPP began to drop to lower levels. Thus, Summary Index scores at these levels of impairment (three or more and four or more tests impaired out of six) are good indicators of ADHD, but poor indicators of an absence of ADHD. There appears to be no diagnostic utility for the battery at Summary Index levels reflecting five or six impaired tests. Diagnostic classification estimates for the Summary Index are shown in Table 3.

DISCUSSION

The comparison of group means revealed significantly poorer performance displayed by the adult ADHD group on measures of spontaneous verbal production and organization (COWA), the ability to cognitively inhibit distracting stimuli (SNST), shifting of attentional set, novel reasoning, and working memory (Trails A and B), and attention–concentration and working memory (WAIS–R Freedom From Distractibility). These significant differences are consistent with prior neuropsychological studies that have demonstrated similar group differences in populations of ADHD children and adolescents (Barkley et al., 1992; Barkley & Grodzinsky, 1994; Reader et al., 1994). These findings are also consistent with a number of studies focusing on adult ADHD populations. For instance, Jenkins et al. (1996) also found that ADHD adults performed significantly more poorly than controls on the COWA. Hopkins et al. (1979) also found that ADHD adults performed significantly worse than controls on a Stroop test. It was surprising that the Short-Delay Free Recall index of the CVLT failed to demonstrate significant differences between groups, as this measure was selected specifically to address the difficulties with encoding and learning of novel verbal information documented in both child and adult ADHD populations (Holdnack et al., 1995; Jenkins et al., 1996; Loge et al., 1990; Mungas, 1983). It was thought that, as a measure of retroactive interference, the Short-Delay Free Recall index would be particularly sensitive to impairments in the acquisition of novel verbal information. Perhaps because the Short-Delay Free Recall index is a measure of verbal learning that has occurred after five full rehearsal trials, it may have allowed for normal rehearsal and encoding to have taken place. It is possible that an initial recall index or a measure of semantic clustering would better reflect differences in verbal organization and learning.

The findings with regard to the Freedom From Distractibility factor of the WAIS–R also merit discussion. To date, research efforts have not yet examined how the cognitive difficulties reported by ADHD adults manifest on this measure, which is believed to be sensitive to difficulties with attention–concentration and working memory. The ADHD adult group's significantly worse performance, relative to controls on this measure, does offer support for the validity of a WAIS–R factor structure that taps such difficulties in this population. These findings are also consistent with research results that showed a trend toward worse performance on Digits Backwards as compared to Digits Forward (Jenkins et al., 1996).

Findings of statistically significant differences between ADHD and non-ADHD groups are not sufficient as diagnostic markers, since these differences do not necessarily reflect clinically significant findings. The clinical and diagnostic utility of these neuropsychological tests were expected to be better reflected by the classification rates based on norm-referenced cut-off scores. However, while each of the six neuropsychological tests considered separately displayed specificity estimates that ranged from 92 to 100%, estimates of sensitivity were much lower, with only one measure (COWA) rising above the 50% mark. These results indicate that while each of these measures classified most of the non-ADHD participants, they were unable to classify many (19–58%) of those who actually had ADHD. PPP estimates for the individual measures ranged from 83 to 100%. Accordingly, all of the measures had low false positive rates. These results indicate that despite low estimates of sensitivity, when impaired results were evident, a diagnosis of ADHD was highly probable. NPP estimates ranged from 55 to 69%. Accordingly, false negative rates were high for all

Table 3. Operating characteristics of the summary index for ADHD adults relative to normal controls, with chi-square values indicating significant clinical differences between groups

Index score	OPP	Sens.	Spec.	PPP	NPP	FP	FN	χ^2
1+ of 6 (.16)	.90	.96	.85	.86	.96	.14	.04	40.6****
2+ of 6 (.33)	.83	.69	.96	.95	.76	.05	.24	25.06****
3+ of 6 (.50)	.63	.27	1.0	1.0	.58	0.0	.42	10.8***
4+ of 6 (.66)	.54	.08	1.0	1.0	.52	0.0	.48	2.85
5+ of 6 (.82)	.50	0.0	1.0	0	.50	.50	.50	0.0
6+ of 6 (1.0)	.50	0.0	1.0	0	.50	.50	.50	0.0

Note. OPP = overall predictive power; Sens = sensitivity; Spec = specificity; PPP = positive predictive power; NPP = negative predictive power; FP = false positives, FN = false negatives; + = "or more". Measures with different superscripts indicate that groups differed significantly at $p < .05$, as indicated by chi-square analyses.
*$p < .05$, **$p < .01$, ***$p < .001$, ****$p < .0001$.

six measures, ranging from 31 to 45%, indicating that when scores on the neuropsychological tests fell within the unimpaired range, a clear and reliable diagnostic decision of ADHD or non-ADHD could not be made. Consistent with pediatric studies (Grodzinsky & Barkley, 1997), abnormal scores on these measures appear to be good predictors of adult ADHD, but normal scores should not be interpreted as they are not generally predictive of an absence of ADHD.

Sample composition is an important factor when considering various indices of diagnostic efficiency. Although base rates of a target group within a sample do not affect estimates of sensitivity and specificity, PPP and NPP vary with regard to base rates of the index group within the sample. Increases in PPP and decreases in NPP occur in conjunction with higher rates of target group representation within the sample. The prevalence of adult ADHD in the general population is estimated to be approximately 2% (Biederman et al., 1995a). As a consequence, one would expect that the use of these individual measures as screening tools in the general population would result in overestimates of PPP and underestimates of NPP. Because adult ADHD is a relatively new diagnostic entity, it is difficult to find estimates of the prevalence of adult ADHD in various clinic populations. However, the prevalence in the present study (50%) is quite similar to prevalence estimates in many pediatric clinic populations (Matier-Sharma et al., 1995). Thus, the operating characteristics reported for the neuropsychological measures in the present study are likely to be more accurate indicators of their utility in a clinical setting that addresses adult ADHD.

It was expected that a Summary Index derived from the six test scores would yield the greatest levels of sensitivity, specificity, positive predictive power and negative predictive power. In fact, classification accuracy did increase substantially when operating characteristics were calculated for the battery as a whole, as compared with the classification accuracy of any of the six neuropsychological measures alone. Improved classification rates were evident for Summary Index scores that reflected impairment in one or more or two or more tests out of the battery of six neuropsychological measures.

With one or more tests impaired in the battery (Summary Index Score = .16), estimates of sensitivity (96%), specificity (85%), PPP (86%), and NPP (96%) were all quite high. As impairment rose to two or more impaired tests within the battery (Summary Index Score = .33), sensitivity decreased somewhat (69%), specificity increased (96%), PPP increased (95%), and NPP dropped (76%). The estimates of PPP and NPP at these levels of impairment (one or more to two or more tests impaired) suggest that Summary Index scores of .16 and .33 appear to be good predictors of ADHD and that a normal score, or the absence of an impaired test in the battery, is also predictive of the absence of ADHD. PPP and estimates of specificity continued to rise to 100% for Summary Index scores that reflected three or more and four or more impaired tests in the battery, indicating that these scores were powerful predictors of ADHD group membership. However, NPP began to drop to unacceptable levels when more than two tests were impaired. There was no classification utility for index scores reflecting five or six impaired tests in the battery. Thus, ADHD is a disorder usually marked by subtle but not by serious impairment on measures of cognitive functioning.

The finding that the best classification utility was observed for the battery (Summary Index) approach, and that one or more or two or more impaired tests within the battery offered the best estimates of sensitivity, is consistent with current conceptualizations regarding ADHD. In that ADHD is thought to manifest more as an inconsistency in attentional difficulties rather than a consistent weakness (Leimkuhler, 1994), it stands to reason that no single neuropsychological measure within the battery would be reliably sensitive to weaknesses in attention–executive functioning. Indeed, the majority of sensitivity estimates for the individual measures in this study fell below the 50% mark (ranging from 19–57%). However, the sensitivity estimates for the Summary Index at one or more tests impaired (96%) and two or more tests impaired (69%) were far superior to those of any single measure alone. This finding suggests that, at these levels, the Summary Index was consistently sensitive to the inconsistent patterns of executive–attentional difficulties displayed in the neuropsychological battery. These findings also indicate that, in developing new measures for the detection of ADHD in adult populations, battery approaches that cover a range of attentional–executive abilities appear to be more sensitive to inconsistencies in attention–executive functioning than any one particular measure of cognitive capacity. Another related point relates to the nature of the neurologic impairment thought to underlie ADHD. That is, the cognitive impairment associated with ADHD is believed to reflect a more subtle functional abnormality (Murphy & Barkley, 1996). The fact that lower Summary Index scores (one or more and two or more tests impaired out of six) were most sensitive to ADHD is indeed consistent with more subtle impairments, and differs from impairment index approaches such as those developed for the Halstead-Reitan Neuropsychological Battery, where greater levels of impairment are associated with greater certainty of cerebral insult.

Findings from this study are also consistent with recent brain imaging data suggesting that those who suffer from ADHD have a dysfunction related to frontal areas of the cortex. The ADHD group's significantly poorer performance on measures of attention–executive functions in the context of their above average IQ estimates (*Mdn* = 115) offer support for Reader et al.'s (1994) observation that executive functions relate most to "how we use what we know," rather than the actual storage of information, or "what we know." However, neuropsychological tests utilized in this study are not solely sensitive to frontal impairments. As Seidman et al. (1997) point out, it is yet to be determined if the subtle frontal abnormalities seen in ADHD neuroimaging studies completely account for the neuropsychological deficits seen in this population.

There is strong reason to believe that the sample of ADHD adults used in the present study was representative of a biological subtype with a family history of the disorder, as opposed to manifestations of ADHD that appear to have stronger correlations with other etiologic factors such as psychosocial adversity (Biederman et al., 1995b). Indeed, inferences regarding dysfunction of frontal networks may be most valid in such a population. ADHD adults in the present study were selected on the basis of an "excellent" behavioral response to stimulant medications, judged both by the participant and the treating psychiatrist. In addition, 65% of the adult ADHD participants in this study had a first degree relative who also suffered from ADHD. Thus, this group shows a high likelihood of a genetic influence for ADHD symptoms. This finding is consistent with recent research (Biederman et al., 1995a) indicating that childhood cases of ADHD that continue through adulthood appear to have especially strong familial etiologic risk factors. It is interesting to note that Seidman et al. (1995) found that an ADHD family history is a strong predictor of significantly poorer neuropsychological performance, even within an ADHD population.

Limitations with regard to this study are worth noting. The present sample is well educated and above average in intelligence. It is possible that these findings would differ in populations with lower estimates of intelligence and less education. However, it is reasonable to assume that above average intelligence and educational attainment would only create advantages with regard to performance on many neuropsychological measures, making the presence of cognitive deficits in this sample more striking. This would imply that even stronger effects might have been found if intellectual functioning and educational attainment were lower. Although, it is also possible that floor effects on particular tests may obscure any relationships with regard to executive dysfunction and lower intelligence–education. The above average estimates of IQ in this study also argue against the premise that the deficits found in this study are attributable to broad cognitive weaknesses rather than ADHD symptomatology. Secondly, the operating characteristics reported in the present study may not generalize to other clinical populations, as additional diagnostic categories (e.g., learning disability, anxiety, and depression) were not simultaneously addressed in the present study. Although previous research indicates that the cognitive difficulties inherent to ADHD extend beyond cognitive impairments that may result from comorbid psychiatric diagnoses (Seidman et al., 1995, 1997), it is difficult to predict, for example, whether the neuropsychological measures utilized in this study would be diagnostically useful when differentiating between an anxiety disorder and ADHD. This becomes especially relevant when considering that there are significant comorbidities among ADHD and many such diagnoses. Finally, order effects may represent a possible limitation in the present study. That is, the neuropsychological measures were administered in a non-randomized predetermined order for each participant. However, it is unlikely that the test results were confounded by differential order effects or fatigue, as significant impairments in the ADHD group's performance were evident both at the beginning (COWA), the middle (Stroop), and at the end of the battery (WAIS–R Freedom From Distractibility).

Despite these limitations, the findings of this study indicate that neuropsychological performance, specifically with regard to attention–executive dysfunction, is impaired in adults with ADHD. Furthermore, both the individual measures and the Summary Index scores are useful in different degrees for diagnostic classification, especially in clinic populations where base rates of ADHD may be much higher than those in the general population. When each of the neuropsychological tests utilized in this study is considered independently, abnormal scores appear to be good predictors of adult ADHD, while normal scores should not be interpreted as they do not appear to be generally predictive of an absence of ADHD. Finally, diagnostic classification rates improve substantially when a battery approach is considered.

Although the neuropsychological measures used in the present study appear to offer useful diagnostic information in terms of classification rates, they should not be considered as diagnostic markers outside of a more thorough evaluation designed to rule out common comorbid conditions such as learning disabilities, depression, and anxiety. When used within a larger neuropsychological battery these measures appear to have distinct advantages in addition to offering valuable data regarding classification rates. Knowledge about the nature of cognitive impairments (e.g., difficulties with working memory, inhibiting distractions, shifting attentional sets) can be valuable when making recommendations for effective treatment plans. Finally, in contrast to self-report measures used to assess symptom intensity or psychiatric comorbidity, these measures avoid many of the confounding biases that arise when respondents rate their own behavior.

REFERENCES

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

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

Barkley, R.A. (1997). Behavioral inhibition, sustained attention, and executive functions: Constructing a unifying theory of ADHD. *Psychological Bulletin*, *121*, 65–94.

Barkley, R.A., Fischer, M., Newby, R., & Breen, M. (1988). Development of a multimethod clinical protocol for assessing stimulant drug responses in children with attention deficit disorder. *Journal of Clinical Child Psychology*, *17*, 14–24.

Barkley, R.A. & Grodzinsky, G.M. (1994). Are tests of frontal lobe functioning useful in the diagnosis of attention deficit disorders? *The Clinical Neuropsychologist*, *8*, 121–139.

Barkley, R.A., Grodzinsky, G.M., & DuPaul, G.J. (1992). Frontal lobe functions in attention deficit disorder with and without hyperactivity: A review and research report. *Journal of Abnormal Child Psychology*, *20*, 163–188.

Benton, A.L. & Hamsher, K. (1983). *Multilingual Aphasia Examination*. Iowa City, IA: AJA Associates.

Biederman, J., Faraone, S.V., Mick, E., Spencer, T., Wilens, T., Kiely, K., Guite, J., Ablon, S., Reed, E., & Warburton, R. (1995a). High risk for attention deficit hyperactivity disorder among children of parents with childhood onset of the disorder: A pilot study. *American Journal of Psychiatry*, *152*, 431–435.

Biederman, J., Milberger, S., Faraone, S., Kiely, K., Guite, J., Mick, E., Ablon, S., Warburton, R., & Reed, E. (1995b). Family environment risk factors for ADHD: A test of Rutter's indicators of adversity. *Archives of General Psychiatry*, *52*, 464–470.

Boucugnani, L.L. & Jones, W.W. (1989). Behaviors analogous to frontal lobe dysfunction in children with attention deficit disorder. *Archives of Clinical Neuropsychology*, *4*, 161–173.

Cahn, D.A. & Marcotte, A.C. (1995). Rates of forgetting in attention deficit hyperactivity disorder. *Child Neuropsychology*, *1*, 158–163.

Delis, D.C., Kramer, J.H., Kaplan, E., & Ober, B.A. (1987). *California Verbal Learning Test: Adult Version*. San Antonio, TX: The Psychological Corporation.

Denckla, M.B. (1989). Executive function, the overlap zone between attention deficit hyperactivity and learning disabilities. *International Pediatrics*, *4*, 155–160.

Denckla, M.B. (1991). Attention deficit hyperactivity disorder-residual type. *Journal of Child Neurology*, *6*, S44–S50.

Denckla, M.B. (1993). The child with developmental disabilities grown up: Adult residual of childhood disorders. *Neurologic Clinics*, *11*, 105–125.

Denckla, M.B. (1996). Biological correlates of learning and attention: What is relevant to learning disability and attention deficit hyperactivity disorder? *Journal of Developmental and Behavioral Pediatrics*, *17*, 114–119.

Dodrill, C.B. (1997). Myths of neuropsychology. *Clinical Neuropsychologist*, *11*, 1–17.

Douglas, V.I. (1972). Stop, look, and listen: The problem of sustained attention and impulsive control in hyperactive and normal children. *Canadian Journal of Behavioral Science*, *4*, 259–282.

Elwood, R.W. (1993). Clinical discriminations and neuropsychological tests: An appeal to Bayes' theorem. *Clinical Neuropsychologist*, *7*, 224–233.

Ernst, M., Zametkin, A.J., Matochik, J.A., Liebenauer L., Fitzgerald, G.A., & Cohen, R.M. (1994). Effects of intravenous dextroamphetamine on brain metabolism in adults with attention deficit hyperactivity disorder (ADHD). Preliminary findings. *Psychopharmacology Bulletin*, *30*, 219–225.

Evans, R.W., Gualtieri, C.T., & Patterson, D.R. (1987). Treatment of chronic closed head injury with psychostimulant drugs: A controlled case study and appropriate evaluation procedure. *Journal of Nervous and Mental Disease*, *175*, 106–110.

Faraone, S. & Biederman, J. (1994). Is attention deficit hyperactivity disorder familial? *Harvard Review of Psychiatry*, *1*, 271–287.

Giedd, J.N., Castellanos, F.X., Casey, B.J., Kozuch, P., King, C.A., Hamburger, S.D., & Rappaport, J.L. (1994). Quantitative morphology of the corpus callosum in attention deficit hyperactivity disorder. *American Journal of Psychiatry*, *151*, 665–669.

Golden, C.J. (1981). *The Luria-Nebraska children's battery: Theory and formulation*. In G.W. Hynd & J.E. Obrutz (Eds.), *Neuropsychological assessment and the school-age child* (pp. 277–302). New York: Grune and Stratton.

Grodzinsky, G.M. & Barkley, R.A. (1997). *The predictive power of frontal lobe tests for the diagnosis of Attention Deficit Hyperactivity Disorder*. Manuscript submitted for publication.

Grodzinsky, G.M. & Diamond, R. (1992). Frontal lobe functioning in boys with attention deficit hyperactivity disorder. *Developmental Neuropsychology*, *8*, 427–445.

Gualtieri, E.E. & Evans, R.W. (1988). Stimulant treatment for the neurobehavioral sequelae of traumatic brain injury. *Brain Injury*, *2*, 101–129.

Heaton, R.K., Grant, I., & Matthews, C.G. (1991). *Comprehensive norms for an expanded Halstead-Reitan battery: Demographic corrections, research findings, and clinical applications*. Odessa, FL: Psychological Assessment Resources.

Holdnack, J.A., Moberg, P.J., Arnold, S.E., Gur, R.C., & Gur, R.E. (1995). Speed of processing and verbal learning deficits in adults diagnosed with Attention Deficit Disorder. *Neuropsychiatry, Neuropsychology, and Behavioral Neurology*, *8*, 282–292.

Hopkins, J., Perlman, T., Hectman, L., & Weiss, G. (1979). Cognitive style in adults originally diagnosed as hyperactives. *Journal of Child Psychology and Psychiatry*, *20*, 209–216.

Hynd, G.W., Hern, K.L., Voeller, K.K., & Marshall, R.M. (1991a). Neurobiological basis of attention deficit hyperactivity disorder (ADHD). *School Psychology Review*, *20*, 174–186.

Hynd, G.W., Semrud-Clikeman, M., Lorys, A., Novey, E.S., & Eliopulos, D. (1990). Brain morphology in developmental dyslexia and attention deficit disorder/hyperactivity. *Archives of Neurology*, *47*, 919–926.

Hynd G.W., Semrud-Clikeman, M., Lorys, A.R., Novey, E.S., Eliopulos, D., & Lyytinen, H. (1991b). Corpus callosum morphology in attention deficit hyperactivity disorder: Morphometric analysis of MRI. *Journal of Learning Disabilities*, *24*, 141–145.

Jenkins, M., Malloy, P., Cohen, R., Salloway, S., Neeper, R., Penn, J., & Chang, K. (1996). *Attentional and learning dysfunction among adults with a history of childhood ADHD*. Paper presented at the 19th annual meeting of the International Neuropsychological Society, Veldhoven, The Netherlands.

Kaufman, A.S. (1990). *Assessing adolescent and adult intelligence*. Boston: Allyn & Bacon.

Kløve, H. (1987). Activation, arousal, and neuropsychological rehabilitation. *Journal of Clinical and Experimental Neuropsychology*, *9*, 297–309.

Leimkuhler, M.E. (1994). Attention deficit disorder in adults and adolescents: Cognitive, behavioral, and personality styles. In J.M. Ellison (Ed.), *Psychotherapists guide to neuropsychiatry* (pp. 175–216). New York: Plenum.

Lezak, M.D. (1995). *Neuropsychological assessment* (3rd ed.). New York: Oxford University Press.

Loge, D.V., Staton, D., & Beatty, W.W. (1990). Performance of children with ADHD on tests sensitive to frontal lobe dysfunction. *Journal of the American Academy of Child and Adolescent Psychiatry*, *29*, 540–545.

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

Lou, H.C., Henrikson, L., Bruhn, P., Borner, H., & Nielsen, J. (1989). Striatal dysfunction in attention deficit and hyperkinetic disorder. *Archives of Neurology*, *46*, 48–52.

Luria, A.R. (1966). *Higher cortical functions in man*. New York: Basic.

Luria, A.R. (1973). The frontal lobes and the regulation of behavior. In K.H. Pibram & A.H. Luria (Eds.), *Psychophysiology of the frontal lobes* (pp. 3–26). New York: Academic.

Luria, A.R. (1980). *Higher cortical functions in man* (2nd ed.). New York: Basic Books.

Mannuzza, S., Klein, R.G., Bonagura, N., Malloy, P., Giampino, T.L., & Addalli, K.A. (1991). Hyperactive boys almost grown up: V. Replication of psychiatric status. *Archives of General Psychiatry*, *48*, 77–83.

Matier-Sharma, K., Perachio, N., Newcorn, J.H., Sharma, V., & Halperin, J.M. (1995). Differential diagnosis of ADHD: Are objective measures of attention, impulsivity, and activity level helpful? *Child Neuropsychology*, *1*, 118–127.

Mattes, J.A. (1980). The role of frontal lobe dysfunction in childhood hyperkinesis. *Comprehensive Psychiatry*, *21*, 358–369.

Mesulam, M.M. (1986). Frontal cortex and behavior. *Annals of Neurology*, *19*, 320–325.

Mesulam, M.M. (1990). Large-scale neurocognitive networks and distributed processing for attention, language, and memory. *Annals of Neurology*, *28*, 597–613.

Mungas, D. (1983). Differential clinical sensitivity of specific parameters of the Rey Auditory-Verbal Learning Test. *Journal of Consulting and Clinical Psychology*, *51*, 848–855.

Murphy, K.R. & Barkley, R.A. (1996). Parents of children with Attention Deficit Hyperactivity Disorders: Psychological and attentional impairment. *American Journal of Orthopsychiatry*, *66*, 93–102.

Posner, M.I. & Dehaene, S. (1994). Attentional networks. *Trends in Neuroscience*, *17*, 75–79.

Posner, M.I. & Presti, D.E. (1987). Selective attention and cognitive control. *Trends in Neuroscience*, *10*, 13–17.

Randolph, C., Gold, J.M., Kozora, E., Cullum, M., Hermann, B.P., & Wyler, A.R. (1994). Estimating memory function: Disparity of Wechsler Memory Scale–Revised and California Verbal Learning Test indices in clinical and normal samples. *Clinical Neuropsychologist*, *8*, 99–108.

Reader, M.J., Harris, E.L., Schuerholz, L.J., & Denckla, M.B. (1994). Attention deficit hyperactivity disorder and executive dysfunction. *Developmental Neuropsychology*, *10*, 493–512.

Reitan, R.M. & Wolfson, D. (1985). *The Halstead-Reitan Neuropsychological Test Battery*. Tucson, AZ: Neuropsychology Press.

Reitan, R.M. & Wolfson, D. (1995). Category Test and Trail Making Test as measures of frontal lobe functions. *Clinical Neuropsychologist*, *9*, 50–56.

Retzlaff, P.D. & Gibertini, M. (1994). Neuropsychometric issues and problems. In R. Vanderploeg (Ed.), *Clinician's guide to neuropsychological assessment* (pp. 185–209). Hillsdale, NJ: Lawrence Erlbaum Associates.

Ruff, R.M., Light, R.H., Parker, S.B., & Levine, H.S. (1996). Benton Controlled Oral Word Association Test: Reliability and updated norms. *Archives of Clinical Neuropsychology*, *11*, 329–338.

Rugle, L. & Melamed, L. (1993). Neuropsychological assessment of attention problems in pathological gamblers. *Journal of Nervous and Mental Disease*, *181*, 107–112.

Satterfield, J.H., Cantwell, D.P., & Satterfield, B.T. (1974). Pathophysiology of the hyperactive child syndrome. *Archives of General Psychiatry*, *31*, 839–844.

Satterfield, J.H. & Dawson M.E. (1971). Electrodermal correlates of hyperactivity in children. *Psychophysiology*, *8*, 191–197.

Satterfield, J.H., Lesser, L.I., Saul, R.E., & Cantwell, D.P. (1973). EEG aspects in the diagnosis and treatment of minimal brain dysfunction. *Annals of the New York Academy of Science*, *205*, 274–282.

Seidman, L.J., Biederman, J., Faraone, S.V., Milberger, S., Norman, D., Seiverd, K., Benedict, K., Guite, J., Mick, E., & Kiely, K. (1995). Effects of family history and comorbidity on the neuropsychological performance of children with ADHD: Preliminary findings. *Journal of the American Academy of Child and Adolescent Psychiatry*, *34*, 1015–1024.

Seidman, L.J., Biederman, J., Faraone, S.V., Weber, W., & Ouellette, C. (1997). Toward defining a neuropsychology of attention deficit–hyperactivity disorder: Performance of children and adolescents from a large clinically referred sample. *Journal of Consulting and Clinical Psychology*, *65*, 150–160.

Shekim, W.O., Asarnow, R.F., Hess, E., Zaucha, K., & Wheeler., N. (1990). A clinical and demographic profile of a sample of adults with attention deficit hyperactivity disorder, residual state. *Comprehensive Psychiatry*, *31*, 416–425.

Shenker, A. (1992). The mechanism of action of drugs used to treat attention deficit hyperactivity disorder: Focus on catecholamine receptor activity. *Advances in Pediatrics*, *39*, 337–382.

Spreen, O. & Strauss, E. (1991). *A compendium of neuropsychological tests: Administration, norms, and scoring*. New York: Oxford University Press.

Stuss, D.T., Alexander, M.P., Palumbo, C.L., Buckle, L., Sayer, I., & Pogue, J. (1994). Organizational strategies of patients with unilateral or bilateral frontal lobe injury in word list learning tasks. *Neuropsychology*, *8*, 355–373.

Trennery, M.R., Crosson, B., DeBoe, J., & Leber, W.R. (1988). *Stroop neuropsychological screening test manual*. Odessa, FL: Psychological Assessment Resources.

Weinberger, D.R. (1987). Implications for normal brain development for the pathogenesis of schizophrenia. *Archives of General Psychiatry*, *44*, 660–669.

Weinstein, G.S. & Wells, C.E. (1981). Case studies in neuropsychiatry: Post-traumatic psychiatric dysfunction—diagnosis and treatment. *Journal of Clinical Psychiatry*, *42*, 120–122.

Welsh, M.C. & Pennington, B.F. (1988). Assessing frontal lobe functioning in children: Views from developmental psychology. *Developmental Neuropsychology*, *4*, 199–230.

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