To the Graduate Council:

I am submitting herewith a dissertation written by J. Noland White entitled "Neuropsychological and Electrophysiological Assessment of Adults with Attention Deficit Hyperactivity Disorder." I have examined the final copy of this dissertation for form and content and recommend that it be accepted in partial fulfillment of the requirements for the degree of Doctor of Philosophy, with a major in Education.

Teresa A. Hutchens, Ph.D., Major Professor

We have read this dissertation
and recommend its acceptance:

Marla P. Peterson, Ph.D.

Cheryl B. Travis, Ph.D.

Joel F. Lubar, Ph.D.

Accepted for the Council:

Interim Vice Provost and
Dean of The Graduate School

Neuropsychological and Electrophysiological Assessment of Adults with Attention Deficit Hyperactivity Disorder

A Dissertation

Presented for the Doctor of Philosophy Degree

The University of Tennessee

John Noland White Jr.

August 2001

Copyright © John Noland White Jr., 2001

All rights reserved

DEDICATION

This dissertation is dedicated to the loving memory of my grandmother, Julia W. Bradbury. From my earliest memories, she has been a paramount influence on my development, both as a student, and as an individual. Although she left this world just hours before my committee would congratulate me and call me "Doctor", I hope that somehow she knows that I made it. She will be in my thoughts and in my heart forever. I did it Granny!

ACKNOWLEDGEMENTS

Foremost, I would like to express my deep gratitude and respect for my major professor, Dr. Teresa A. Hutchens, whose leadership and encouragement have been invaluable. I am also greatly indebted to my mentor in neurophysiology, Dr. Joel F. Lubar, for his significant contribution to the success I have enjoyed at The University of Tennessee. I will remain forever thankful for each of you and your positive guidance on my pathway from student to scholar.

I'd also like to thank my other committee members, Dr. Marla P. Peterson and Dr. Cheryl B. Travis for their time, comments, and counsel throughout this process. I also gratefully acknowledge Dr. Joseph Sandford, the developer of the Integrated Visual and Auditory Continuous Performance Test, for his support of my research. I'd also like to thank my statistical consultant, Cary Springer.

I would like to acknowledge all of my fellow lab members, with special thanks to Kerry, Les, & Phil for their assistance during the pilot stages of this study. To my friends Alissa, Connie, Meta, and M.C., thank you for everything.

To Momma, Daddy & Marcella, Lynn & Ted, Julie & Rette, Mr. & Mrs. Snipes, and Carey, thank you for believing in me and keeping our family close despite the physical distance between us.

Lastly, and most importantly, I would like to thank my wife and daughter for their unconditional encouragement, patience, and love. I could not have reached this goal without you. Thank you for providing daily reminders of the truly important things in life and never losing faith in me.

ABSTRACT

The present study evaluated the neuropsychological performance of adults with and without Attention Deficit Hyperactivity Disorder (ADHD) during the Paced Auditory Serial Addition Test (PASAT), Wisconsin Card Sorting Test: Computerized Version 3 (WCST), and the Integrated Visual and Auditory Continuous Performance Test (IVA). The quantitative electroencephalograph (QEEG) was also collected during task performance to examine possible difference in cortical activity between groups and tasks. Results suggest that adults with ADHD demonstrate lower levels of performance on neuropsychological tasks that involve working memory, processing speed, and sustained attention, namely the PASAT and IVA. Furthermore, adult ADHD appears to be characterized by different neurophysiological markers than childhood ADHD and these markers vary according to the neuropsychological task being performed. In general, adults with ADHD demonstrate a QEEG pattern characterized by higher levels of high-alpha during the tasks that differentiated their performance from controls, specifically the PASAT and IVA.

TABLE OF CONTENTS

CHAPTER ONE **1**

Introduction **1**

Background 1

Diagnostic criteria 2

Differential diagnosis 6

Impact of Adult ADHD 8

Etiology 8

Traditional Assessment of ADHD 9

Neuropsychology and ADHD 10

Continuous Performance Tests 11

Cognitive Processing 16

Exploration of Attentional Mechanisms 22

Neuroanatomical Approaches to Attention 23

Neuroanatomical/Neurological Basis of ADHD 25

Neuroimaging Methods 26

Neurochemical and Neurophysiological Approaches to ADHD 31

Electroencephalographic (EEG) and Neurological Correlates of ADHD 35

Summary 39

Statement of the Problem 40

Research Hypotheses 40

CHAPTER TWO **41**

Methodology **41**

Participants 41

Instruments 45

Paced Auditory Serial Addition Task (PASAT) 45

Wisconsin Card Sorting Test (WCST) 45

Integrated Visual and Auditory Continuous Performance Test (IVA) 46

Electroencephalograph (EEG) Recording 46

Procedure 46

EEG Collection 47

Data Analysis 50

CHAPTER THREE **53**

Results **53**

Analysis of Demographic Variables 53

Initial Participant Groups 53

Realignment of Control Sample 55

Comparison of Realigned Controls to ADHD Sample 56

Analysis of Research Hypotheses 58

Comparison of non-ADHD and ADHD adults on neuropsychological measures 58

QEEG Band Power during Neuropsychological Task Performance 62

QEEG Power Ratio Measures during Neuropsychological Task Performance 67

Correlation of QEEG Power Ratios to Neuropsychological Task Performance 69

Prediction of Group Membership from Neuropsychological Task Scores 70

Exploratory Analyses 72

Comparison of Theta and High-alpha Activity Averaged Across Nine Central Sites During the Neuropsychological Tasks 73

Comparison of Adults with ADHD and Non-clinical Controls on QEEG "Index Value" across Baseline and Task Conditions 73

Separation of ADHD Participants into Subgroups 74

Comparison of QEEG Ratios in ADHD Subgroups 74

CHAPTER FOUR 78

Discussion 78

Background 78

Initial Comparison and Realignment of Control Sample 79

Comparison of Realigned Adult Controls to ADHD Sample 79

Neuropsychological Performance 80

Summary of Neuropsychological Measures 84

Quantitative Electroencephalography (QEEG) Analyses 85

Topographic Power 86

QEEG Ratio Measures 90

Relation of QEEG Ratio Measures to Task Performance 91

Summary of QEEG Measures 92

Exploratory Investigations 93

Examination of ADHD Subgroups 94

Study Limitations and Areas of Future Investigation 95

Limitations 95

Directions for Clinical Application 97

Directions for Future Research 98

Summary and Conclusions 99

REFERENCES 101

VITA 126

LIST OF TABLES

Table 1: Demographic Variable Means: Initial Comparison 54

Table 2: Demographic Variable Means of Control Subgroups 57

Table 3: Demographic Variable Means: Final Comparison Groups for Study 59

Table 4: Neuropsychological Performance of Control and ADHD Participants 60

Table 5: Paired-sample t-tests for High-alpha activity at Fz 66

Table 6: Correlation of QEEG Ratios at Fz with Neuropsychological Test Scores 71

Table 7: ADHD Subgroups' QEEG Ratios at Fz During Task Performance 77

ABBREVIATIONS

AAQ	IVA Auditory Attention Quotient
ADD	Attention Deficit Disorder
ADHD	Attention Deficit Hyperactivity Disorder
ANOVA	Analysis of Variance
CPT	Continuous Performance Test
CT	Computerized X-ray Tomography
Cz	Central Midline
deoxy-Hb	Deoxygenated hemoglobin
df	degrees of freedom
DSM	Diagnostic and Statistical Manual of Mental Disorders
EEG	Electroencephalography
fMRI	Functional Magnetic Resonance Imaging
Fz	Frontal Midline
FSAQ	IVA Full Scale Attention Quotient
FSRCQ	IVA Full Scale Response Control Quotient
GRTH	Generalized Resistance to Thyroid Hormone
HAB	High-alpha/Low-beta Ratio
Hz.	Hertz
IVA	Integrated Visual and Auditory Continuous Performance Test
M	Mean
MANOVA	Multivariate Analysis of Variance

MPFC	Medial Prefrontal Cortex
MRI	Magnetic Resonance Imaging
NIRS	Near Infrared Spectroscopy
NMR	Nuclear Magnetic Resonance
oxy-Hb	Oxygenated hemoglobin
PASAT	Paced Auditory Serial Addition Test
PET	Positron Emission Tomography
PPVT-III	Peabody Picture Vocabulary Test - 3rd Edition
QEEG	Quantitative Electroencephalography
rCBF	Regional Cerebral Blood Flow
RCQ	IVA Response Control Quotient
SD	Standard Deviation
SPECT	Single Photon Emission Computed Tomography
TB	Theta/Low-beta Ratio
WCST	Wisconsin Card Sorting Test

CHAPTER ONE

Introduction

Attention Deficit Hyperactivity Disorder (ADHD) is a developmentally disabling disorder of inattention, impulsivity, and hyperactivity. A topic of intense examination by medical, psychological, and educational professionals, ADHD has been scrutinized as a diagnostic category, both for its prevalence, and for its validity (Fergusson, Horwood, & Lynskey, 1993; Halperin et al., 1993; Reid & Katsiyannis, 1995). Behavioral implications have been shown to persist into adolescence and adulthood (Barkley, 1998b; Biederman et al., 1993; Biederman et al., 1996; Biggs, 1995; Cohen, Cohen, & Brook, 1993; Epstein, Conners, Sitarenios, & Erhardt, 1998; Gansler et al., 1998; Horton, 1996; Kovner et al., 1998; Lovejoy et al., 1999; Monastra et al., 1999; Riordan et al., 1999; Walker, Shores, Trollor, Lee, & Sachdev, 2000; Wender, 1995). Up to 50-65% of children diagnosed with ADHD will experience problems with behavior in general and ADHD symptoms as adults (Barkley, 1998b). ADHD is conceptualized as a medical/psychiatric condition and has been listed in the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM*) since 1987 (American Psychiatric Association, 1987; Reid & Katsiyannis, 1995); however, its quality and criteria reveal a history of nomothetic changes since 1968. It is currently found in the Fourth Edition of the *DSM* and is defined by criteria of inattention, distractibility, and/or impulsivity with various degrees of hyperactivity.

Background

In 1968, the American Psychiatric Association used the term "hyperkinetic syndrome" to describe this constellation of clinical behaviors (Shealey, 1994). At that

time, the defining focus was primarily a positive response to Ritalin and presumed neurological substrates of manifest behavior. Defining terminology more similar to the current application of ADHD diagnosis emerged in 1980 in the third edition of the *DSM* (American Psychiatric Association, 1980). At that time, a primary criterion was attention deficit and a dichotomy in the diagnostic label of Attention Deficit Disorder (ADD) was used: ADD With or ADD Without the presence of Hyperactivity. With the revision of the third edition of the *DSM* in 1987, primary criteria were the magnitude of expression of inattention and impulsiveness, continuing a focus on hyperactivity but with greater emphasis on the developmentally inappropriate degree and intensity (American Psychiatric Association, 1987). The diagnostic label was changed to Attention-Deficit Hyperactivity Disorder with the *DSM-III-R* and the diagnostic subtypes With and Without hyperactivity were removed. Attention-deficit without signs of impulsiveness and hyperactivity was then called Undifferentiated Attention-deficit Disorder. The fourth and current edition of the *DSM* (American Psychiatric Association, 1994) has maintained the label of ADHD with diagnostic criteria focused on a triad of discriminating inattention, impulsivity, and hyperactivity.

Diagnostic criteria

As defined in the *DSM-IV*, ADHD has been characterized by a persistent pattern of inattention and/or hyperactivity and impulsiveness, more frequent and severe than typically observed in developmental peers (American Psychiatric Association, 1994). The present category was designed to include three subtypes of ADHD, ADHD, predominantly inattentive (ADHD/I); ADHD, predominantly hyperactive-impulsive (ADHD/HI); and

ADHD, combined (ADHD/COM) (Power & DuPaul, 1996). Two clusters of symptoms, inattention and hyperactive-impulsive, have been included, with nine behaviors specified for each cluster. At least six of the nine behaviors in each cluster must be displayed in order to be considered sufficiently clinically significant for the diagnoses (Power & DuPaul, 1996). Specified symptoms must have been present before age 7 and shown to cause significant impairment across at least two of three settings: social, academic, and/or occupational. For a symptom to be applicable for diagnosis, it must have been present for at least six months and be inconsistent with an individual's developmental level (American Psychiatric Association, 1994).

Prevalence. ADHD has been reported to affect 3-5% of school-aged children (American Psychiatric Association, 1994) and is 4-9 more times more common in boys than in girls (Shealey, 1994). However, there have been studies reporting prevalence rates anywhere from 2% - 9.5% with an average prevalence rate of 4.9% when using clinical diagnostic criteria (Barkley, 1998d). Some research reports the prevalence of ADHD to be similar patterns across various ethnic and geographic cultures (Barbarin & Soler, 1993). Others reported prevalence rates ranging from 1.8% to 29% in school-aged children (Barkley, 1998d).

Gender Differences. One area of investigation in ADHD prevalence research has been related to gender with estimates ranging from three to nine times more common that a male will be diagnosed ADHD. Overall, research in this area has been more limited than would be expected given that prevalence has been reported to vary significantly by gender (Barkley, 1998a; Carlson, Tamm, & Gaub, 1997; Gingerich, Turnock, Litfin, &

Rosen, 1998). Typically, studies have included too few females to warrant separate data analyses. They have often been excluded in order to maximize sample homogeneity and eliminate sex as a confound (Carlson et al., 1997).

Epidemiological studies have shown a male-to-female prevalence ratio of 3:1 in community samples and between 6:1 and 9:1 in clinic-referred samples (Barkley, 1998a; Gaub & Carlson, 1997). Discrepancy in clinical as compared with community samples has been hypothesized to stem from referral bias (Barkley, 1998a; Gaub & Carlson, 1997; Gingerich et al., 1998). It has been suggested that, for males, the expression of the disorder is more disruptive for parents and teachers (Gingerich et al., 1998) with males tending to engage in more overt or impulsive behaviors. Parents or teachers may have had normative gender expectations that have influenced their rating of social acceptance (Carlson et al., 1997).

Data have shown that intensity and/or externalizing behaviors in normative males has tended to be greater than in females (Barkley, 1998a). Males have been reported to be more likely to engage in aggressive and antisocial behavior which may be more noticeable and hence, more likely to be referred for treatment or evaluation (Barkley, 1998a). Females have been shown to be more likely to manifest learning problems or internalizing symptoms, in contrast to males experiencing conduct problems or other externalizing symptoms (Barkley, 1998a; Gaub & Carlson, 1997). Females who exhibit problem behaviors have often been shown to be characteristic of older, comparable males at the time of referral (Gingerich et al., 1998). Poorer social functioning or level of social risk has appeared to be greater for females despite similar or less severe behavior

problems than males (Carlson et al., 1997). It has been suggested that externalizing behavior problems are more likely congruent with social expectations held of males (Gingerich et al., 1998). Such possible value judgments have formed unexpressed assumptions for research and have affected the kind of data have that been collected and what interpretations have been made of data (Bleier, 1986).

Another possible explanation in the apparent discrepancy of gender prevalence in ADHD has been the relative performance of females versus males in measures of attention, an often integral component of the diagnostic procedure for ADHD. Impaired attention appears to be associated with behavioral difficulties and poor academic performance in both males and females; however, females have performed better on tests of attention overall and the use of the same norms for both genders may result in greater numbers of false negatives among females (Pascualvaca et al., 1997). The resulting lower prevalence of ADHD may be an artifact of present diagnostic strategies; those females that are diagnosed may be those with more severe difficulties. A greater number of females may actually benefit from treatment than are currently being treated (Pascualvaca et al., 1997). Males have typically been the focus of ADHD research. When fragments of scientific focus are analyzed by prevailing modes of thought, without examination of those modes or alternate realities, the resulting reality is often altered which can affect our inquiry and technological tools chosen for the task (Namenwirth, 1986).

There is scant research addressing the number of adults who manifest ADHD. Barkley (1998d) reported three studies attempting to determine the incidence of diagnosis

difficult to distinguish symptoms of ADHD from developmentally age-appropriate behaviors, oppositional behavior, and other types of disorders. There have been numerous conditions identified that can co-occur with the diagnosis of ADHD (Auronowitz et al., 1994; Malone, Kershner, & Swanson, 1994; Semrud-Clikeman, Hynd, Lorys, & Lahey, 1993).

The American Psychiatric Association (1994) has indicated that there may be a higher prevalence of Mood Disorders, Anxiety Disorders, Learning Disorders, and Communication Disorders among children who are diagnosed with ADHD. Research with children, adolescents, and adults has suggested that ADHD has often occurred with conduct, major depressive, and anxiety disorders (Biederman et al., 1993; Biederman, Newcorn, & Sprich, 1991). Of those adults who were diagnosed with ADHD as children, it has been suggested that approximately 79% complain of difficulties with psychiatric symptoms such as anxiety, sadness, or somatic complaints (Barkley, 1998b). Furthermore, about 75% of them have reported interpersonal problems although the incidence of psychotic disorders has been no greater than that for a non-clinical control group (Barkley, 1998b).

Other conditions may actually produce the behaviors that are criterion-referenced in ADHD categories (Hutchens & White, 1998). Such conditions include metabolic disorders, head injury, depression, schizophrenia, learning disabilities, antisocial personality disorder, and psychoactive substance abuse, among others (Biederman et al., 1996; Horton, 1996; Hutchens & White, 1998). The possibility that there has been an

over-diagnosis of ADHD has stimulated an awareness of the potential for incomplete or inaccurate diagnoses (Fisher, 1997; Sabatino & Vance, 1994).

Impact of Adult ADHD

Besides the triad of symptoms associated with ADHD, adults may also have clinical symptoms commonly found with childhood ADHD (Biederman et al., 1996). Such symptoms that have been reported include stubbornness, low frustration tolerance, and chronic difficulties in social relations with peers, spouses, and authorities (Biederman et al., 1996). The prevalence of persisting ADHD in the adult population has been estimated to range from 2 – 7% to as high as 50 – 70% (Barkley, 1998b; Wender, 1995).

Attentional impairments may impact adults in areas other than those typically associated with such deficits. Functional domains that are most susceptible include memory, work situations, driving, and social behavior, e.g. not responding or acknowledging relevant cues (van Zomeren & Brouwer, 1994). The incidence of drug abuse has been reported to be as much as four times higher in adults with ADHD and as high as 33% of participants in alcohol treatment programs may evidence ADHD symptoms (Horton, 1996).

Etiology

From it's inception, it has been suggested that ADHD may have a biological basis (Weiss, Stein, Trommer, & Refetoff, 1993). It has been suggested that ADHD might be viewed as stemming from biologically-based deficiencies in behavior regulation rather than deficits in attention (Barkley, 1990). Other research has supported the neurophysiological aspects of ADHD (Lubar, 1991) and suggest that the primary

symptoms of ADHD are actually secondary outcomes resulting from a primary neurological disorder (Lubar, Swartwood, Swartwood, & O'Donnell, 1995; Riccio, Hynd, Cohen, Gonzalez, & et al., 1993). ADHD has been shown to be familial and heritable (Cook et al., 1995; Miller & Blum, 1996). Individuals with ADHD have often been reported to have family members that manifest the same symptoms. Examination of specific biological processes also reveals conditions in which ADHD symptomatology may be influenced. These examinations are often rooted in neuroanatomy or neurology.

Traditional Assessment of ADHD

Historically, the assessment of ADHD in children has been based on parent, teacher, and child interviews; parent and teacher rating scales; and parents' or, self-report measures. These measures are consistent with the behavioral formulation of ADHD diagnostic criteria and focus on assessing this disorder from a behavioral perspective (Monastra et al., 1999). Rating scales and self-report forms have also been developed for the diagnosis of ADHD in adults.

Rating scales include the Current Symptoms Scale and Childhood Symptoms Scale (Barkley & Murphy, 1998). The Current Symptoms Scale contains 18 symptom items for ADHD from the *DSM-IV*. The scale also includes items pertaining to age of onset of symptoms and areas in which there may be impairment in major life activities (Barkley & Murphy, 1998). Additionally, there are 8 items at the end of the scale for Oppositional Defiant Disorder (ODD) from the *DSM-IV*. Adults who report having this disorder as a child, along with their ADHD, are shown to be likely to retain some of their hostile-defiant behaviors into adulthood (Barkley & Murphy, 1998). The Childhood

Symptoms Scale (Barkley & Murphy, 1998) is used to indicate the clinical significance of a participant's retrospective recall of their childhood years (ages 5-12 years).

One example of such a rating scale is the Wender Utah Rating Scale (WURS). The Wender Utah Rating Scale (Wender, 1995) is a 61-item self-rated likert scale used to assess ADHD in adults. It is based on ADHD behaviors in childhood, medical conditions, and school performance. It was developed from observational data and, in order to correctly ascertain the presence of the adult symptoms of ADHD, is based on what the participant observes, what he or she relates others observe about their behavior, and if possible, the reports of a knowledgeable other (Wender, 1995).

Despite the widespread use of rating scales and behavior checklists, there has remained a quest for a more objective assessment of ADHD. Basically, there is an absence of behavioral specificity and overlap with other conditions producing the same behaviors, thus, they are not fully able to differentiate ADHD from some comorbid conditions. Rating scales are also subject to reporter and examiner bias and are thus subjective instruments. One attempt to make the assessment of ADHD more objective is in the examination of neuropsychological batteries.

Neuropsychology and ADHD

Unless a professional is highly experienced in working with adults with ADHD, questionnaires and self-report data may not be sufficient for a definitive or differential diagnosis (Biggs, 1995). Although no set battery of assessments has yet been identified, there have been recommendations of general domains of behavior to include. Such assessments are tests of general intelligence, continuous process/performance tests, other

types of tests of vigilance and attention, measures of impulsivity, memory tests, measures of strategic planning, and measures of executive functioning (Biggs, 1995).

Related types of behavior may also be involved in the ADHD constellation. Recently, researchers have examined verbal fluency in children as a means by which to differentially diagnose ADHD from two subtypes of dyslexia. Cohen, Morgan, Vaughn, Riccio, and Hall (1999) studied developmental differences in a sample of 130 children with no diagnosis, 42 children with developmental dyslexia (Language Disorder/Dysphonetic, $n = 35$; Visual-Spatial/Dyseidetic, $n = 7$), and 23 children with ADHD. Results of an Analysis of Variance (ANOVA) found that there was a significant performance effect ($F = 9.38$; $p < .001$) across the two Dyslexic subgroups and the ADHD group. Furthermore, performance on the verbal fluency test was significantly lower ($p < .05$) for the Language Disorder/Dysphonetic subgroup ($M = 75.86 \pm 29.93$) as compared to the Visual-Spatial/Dyseidetic subgroup ($M = 99.86 \pm 10.29$) and the ADHD group ($M = 97.13 \pm 16.04$), with the latter two groups' performance not differing significantly from each other or from average for this task (Cohen, Morgan, Vaughn, Riccio, & Hall, 1999). Thus, based on specific measures, ADHD can be differentiated from some conditions, but not from others.

Continuous Performance Tests

Measures of attention span and impulse control have also proven useful in the diagnosis of ADHD (Barkley, 1997b). Such measures include Continuous Performance Tests (CPTs). These are instruments in which the task involve the presentation of constantly changing stimuli with some clearly defined "target" stimulus or pattern that

occurs at a low frequency relative to the number of stimuli presented over the duration of the task (Reynolds, Lowe, Moore, & Riccio, 1998).

Research has demonstrated that hyperactive children produce greater errors of commission on continuous performance tests (CPTs), especially on computerized versions (Barkley, 1997a). These errors involve pressing the response key even when no target has appeared or a false target similar but not identical to the correct one is presented. Adults with ADHD also demonstrate more impulsive errors on CPTs (Barkley, 1997a). These errors of commission are related to symptoms of impulsiveness.

The original CPT, developed in 1956 by Rosvold and colleagues as a measure of sustained attention, was demonstrated to be highly sensitive to brain damage or dysfunction (Riccio, Moore, Lowe, & Reynolds, 1998; Rosvold, Mirsky, Sarason, Bransome, & Beck, 1956). In their version, participants pressed a key when a target letter appeared (e.g., X), or when the target letter was preceded by another letter (e.g., A – X) (Mirsky, 1996; Rosvold et al., 1956; Spreen & Strauss, 1998). The CPT was originally used to assess the capacity to sustain a focus of attention in individuals with absence seizures, complex partial seizures, closed head injuries, and a group of control participants (Mirsky, 1996; Rosvold et al., 1956).

There have been several commercially available CPTs including the Gordon Diagnostic System (GDS), the Test of Variables of Attention (TOVA), the Integrated Visual and Auditory Continuous Performance Test (IVA), the Raggio Evaluation of Attention Deficit Disorder (READD), and the Conners' Continuous Performance Test (Lowe, Reynolds, Riccio, & Moore, 1998). The numerous variations of the CPT may

impose different information processing demands on the individual and may affect the level of sustained attention and error pattern performance (Lowe et al., 1998). In spite of these variations and criticisms, CPTs have been shown to be valuable tools in the assessment of ADHD (Epstein et al., 1998; Forbes, 1998; Greenberg & Kindschi, 1996; Sandford, 1995). Others affirm their ability to eliminate ADHD from diagnostic consideration but question their ability to differentially identify it (Reynolds et al., 1998).

In a recent study of ADHD adults (n = 60) and non-ADHD adults (n = 72), Epstein et al. (1998) found that individuals with ADHD made more errors of commission (F (1, 129) = 11.4, p = .0009) and more errors of omission (F (1, 129) = 8.9, p = .003) than did controls. The CPT used in this study was the Conners. For the non-ADHD group, a sensitivity coefficient of 76.4% was reported, correctly identifying 55 of the 72 normals. For the ADHD group, 33 of the 60 were correctly identified producing a sensitivity coefficient of 55.0% (Epstein et al., 1998).

One CPT that is unique in its construction is the IVA. It is the first commercially available CPT to combine both auditory and visual CPTs of impulsivity and inattention. The entire test procedure is automated for standardized results and controlled by administration on computer. The four parts of the test include 1) Warm-up, 2) Practice, 3) Main CPT, and 4) Cool-down (Sandford, 1995). This design was chosen to aid in maximizing the performance of all participants by providing training and practice both in using the computer mouse and in performing the CPT task (Sandford, 1995). The test task requires the participant to click the mouse only when he or she sees or hears a "1" (target) and not to click when he or she sees or hears a "2" (foil). For adequate

performance, participants are intended to remember to click when they see a 1; click when they hear a 1; don't click when they see a 2; don't click when they hear a 2. The test was designed to promote errors of impulsivity and inattention by creating sets of responding and not responding, respectively; it has also challenged an individual's ability to shift cognitive sets (Sandford, 1995).

The IVA yields six global composite quotient scores and 22 other scales divided into four groups: Response Control, Attention, Attribute, and Validity. All quotient scores have a mean of 100 and a standard deviation of 15 (Sandford, 1995). There are two global full-scale scores, Full Scale Response Control Quotient (FRCQ) and Full Scale Attention Quotient (FSAQ). Each of these two main scores is based on six visual and auditory scales. Other scales included measure additional areas such as Fine Motor Regulation and validity.

Test-retest reliability for the IVA composite quotient scores range from .37 to .75. Analysis of the 22 IVA scale raw scores found that 20 had significant positive relationships, (.46 to .88). Thus, the test appears to be a stable measure for both global and specific scale performance (Sandford, 1995). The IVA has sufficient sensitivity (92%) and positive predictive power (89%) to be used in the evaluation of ADHD in children. Concurrent validity was examined by comparing children identified by IVA to be ADHD with other diagnostic instruments such as the Test of Variables of Attention (TOVA), the Gordon Continuous Performance Test (Gordon CPT), the Children's Attention Scale (CAS), and the CPRS-39 ADHD rating scale, with percent of agreement of diagnosis ranging from 90% to 100%. The percentage of false negatives was reported

to be 7.7% as compared to the aforementioned instruments ranging from 12.5% to 59.1% (Sandford, 1995).

Although there is a substantial literature examining CPT performance in childhood ADHD, research with adult samples have been sparse (Epstein et al., 1998). Those that have been conducted have used various methodologies, age ranges, and have produced inconsistent results. Attentional capabilities appear to increase with age and some CPTs may inherently have a low ceiling for commission errors (Epstein et al., 1998).

Concurrent validity for the use of CPTs in adult ADHD is supported by their use with other clinical populations that have symptomatology involving attention deficits such as Schizophrenia or Bipolar Disorder. In a study using the Integrated Variables of Attention (IVA) CPT, it was found that individuals with schizophrenia and bipolar disorder scored within the normal range in regard to response inhibition but were found to be generally impaired on measures of attention (Baerwald & Tryon, 1999b). Furthermore, the same individuals displayed a visual over auditory preference, indicating a modal attention asymmetry (Baerwald & Tryon, 1999a).

Recent research has reported that adults with ADHD have a distinct neuropsychological profile that approximates that seen in children. This profile is marked by relatively intact verbal reasoning and visual memory, but impaired performance on measures of verbal memory, motor and processing speed, visual scanning, and auditory and visual distractibility (Riordan et al., 1999). When control participants were compared against those with ADHD and those with ADHD and

comorbid depression (ADHD-D), neuropsychological performance was significantly lower for the two clinical groups. The researchers also reported that intellectual functioning was significantly higher for the control group and used Full Scale IQ as a covariate. The statistical analysis examined subjects as a between groups factor (ADHD, ADHD-D, controls) and various neuropsychological summary scales (verbal reasoning, visual memory, verbal memory, scanning, motor speed, processing speed, visual distractibility and auditory distractibility) as a within subject factor. MANCOVA yielded significant between groups effect, F (2,50) = 5.59, p = .006. However, a nonsignificant interaction between diagnosis and neuropsychological summary scales was found, F (14,88) = 1.54, p = .22, thus the ADHD and ADHD-D groups had similar neuropsychological profiles (Riordan et al., 1999). Furthermore, although the ADHD and ADHD-D performed significantly lower than controls, the differences were not that large which raised questions of clinical significance (Riordan et al., 1999).

Cognitive Processing

There have been studies examining neuropsychological deficits in adults with ADHD. Using a test battery, Horton (1996) reported finding that adults with ADD had few neuropsychological deficits except in the initial acquisition of verbal information on the Logical Memory Subtest of the Wechsler Memory Scale, and the Category Test. The limitations of the study were small sample size (n = 11), presence of comorbid conditions, and short forms of tests were used (Horton, 1996).

Additional research has indicated that hyperactive-impulsive behavior generally has an association with diminished IQ, particularly Verbal IQ (Barkley, 1997a). ADHD

symptoms have been found to be significantly related to IQ in both normal and ADHD samples (Barkley, 1997a). Moreover, intelligence has also been shown to account for a significant amount of variance in some tests of executive function, including the Wisconsin Card Sorting Test (Barkley, 1997a; Boone, 1999). It has been suggested that the role of intelligence in complex mental operations, such as attentional processes, is related to the speed of mental processing (Hutchens, 1989). This "information processing efficiency" (Shallice, 1988) is related to fluid intelligence, which develops as the result of biological maturation and reaches its peak in the late teens or early twenties (Heaton, Ryan, Grant, & Matthews, 1996).

One measure that has been used in the assessment of ADHD is the Paced Auditory Addition Test (PASAT). It is described as a measure of verbal working memory, attention, and speed of information processing (Barkley, 1997a; Brittain, La Marche, Reeder, Roth, & Boll, 1991; Gronwall, 1977; Gronwall & Sampson, 1974; Spreen & Strauss, 1998). This test is a serial-addition task used to assess capacity and rate of information processing, sustained attention, and divided attention (Spreen & Strauss, 1998).

The Levin et al. (1987) version of the PASAT consists of a pre-recorded tape, which delivers a series of 50 single digits in different random sequences for each of four trials. The auditory tape was produced by a computer-controlled synthesized speech program with electronically verified stimulus presentation at rates of 2.4, 2.0, 1.6, and 1.2 seconds (s); duration of .4s; inter-trial interval of 30 seconds (Brittain et al., 1991; Levin et al., 1987). The original version of the PASAT used the same set of numbers, 61 digits

each, across all four trials (Brittain et al., 1991; Gronwall, 1977; Gronwall & Sampson, 1974). The individual is instructed to add pairs of numbers such that each number is added to the one that immediately precedes it (Spreen & Strauss, 1998). The second number is added to the first stimulus item, the third to the second, the fourth to the third, and so on. For example, after the numbers "3, 5" the answer is "8"; if the next number is "7," this is added to the previous "5" to give the answer "12"; etc. The individual's response is not included in the addition sequence. To be correct, a response must be made before presentation of the next stimulus. Performance has been evaluated in terms of the percentage of correct responses, percentage of incorrect responses, or the total score across trials (Diehr, Heaton, Miller, & Grant, 1998; Lezak, 1995; Spreen & Strauss, 1998).

Correlation with mental ability has been reported as modest (Lezak, 1995; Spreen & Strauss, 1998). The PASAT's split-half reliability has been reported to be about .9, implying high internal consistency; Cronbach's alpha from scores on the four PASAT trials has been reported to be .90 (Spreen & Strauss, 1998). Performance levels have been reported to decline with age but are most prominent after age 50 (Lezak, 1995; Spreen & Strauss, 1998). Conflicting findings have been reported to the effect of gender (Lezak, 1995; Spreen & Strauss, 1998).

Barkley (1997) considers working memory as an executive function although some researchers view it as an element of attention and the PASAT has been used as a test of working memory. Despite its use in several neuropsychological battery approaches for ADHD, some researchers have reported that the PASAT has not been

successful in discriminating ADHD adults from controls (Corbett & Stanczak, 1999). In contrast, a recent study of working memory performance compared six adult males with ADHD to six adult males without ADHD who were matched in age and general intelligence (Schweitzer et al., 2000). In the Schweitzer et al. study (2000), men without ADHD performed better on the PASAT as compared to men with ADHD. Other studies have shown the PASAT to be highly sensitive to ADHD but it is also sensitive to other neuropsychiatric disorders (Katz, Wood, Goldstein, Auchenbach, & Geckle, 1998).

Another neuropsychological measure included in several batteries for the assessment of ADHD is the Wisconsin Card Sorting Test (WCST). The WCST assesses an individual's abstract reasoning ability and ability to shift cognitive strategies in response to changing environmental contingencies (Berg, 1948; Grant & Berg, 1948; Heaton, Chelune, Talley, Kay, & Curtiss, 1993; Heaton, Thompson, & Gomez, 1999). It assesses the ability to form abstract concepts, shift and maintain set, and utilize feedback (Spreen & Strauss, 1998).

The WCST consists of four stimulus cards and 128 response cards that depict various figures (crosses, circles, triangles, or stars), colors (red, blue, green, or yellow) and numbers of figures (one, two, three, or four) (Heaton et al., 1993). The stimulus cards are placed in front of an individual from left-to-right order: one red triangle, two green stars, three yellow crosses, and four blue circles on them. The participant is then given a deck of 64 response cards, which have designs similar to those on the stimulus cards. The participant is told to match each of the cards in the deck to one of the four key cards and is given feedback each time whether he or she is right or wrong (Heaton et al.,

1993; Spreen & Strauss, 1998). Performance is scored in six different ways including Categories Completed, Trials to Complete First Category, Percent Perseverative Errors, Failure to Maintain Set, Percent Conceptual Level Responses, and Learning to Learn (Heaton et al., 1993; Spreen & Strauss, 1998).

In addition to the manual form of the instrument, a computerized version (Wisconsin Card Sorting Test: Computer Version 3 for Windows Research Edition (WCST: CV3)) has been developed which provides a standardized automated administration and scoring procedure based on the work of Heaton et al. (1993) (Heaton et al., 1999). Demographically corrected normative data developed by Heaton et al. (1993) are provided in the WCST: CV3. This data was collected using the original, manually administered, 128-card version of the WCST but research has demonstrated general equivalence between computerized administration and card administration of the WCST in a small population of normal participants from Spain (N = 199) and in a sample of psychiatric patients (N = 30) (Artiloa i Fortuny & Heaton, 1996; Heaton et al., 1999; Hellman, Green, Kern, & Christenson, 1992).

The WCST has been used extensively in clinical and research applications as a measure of executive function (Heaton et al., 1993). Concurrent validity has been demonstrated through studies with participant groups having focal and diffuse brain injuries, seizure disorders, Parkinson's disease, multiple sclerosis, psychiatric disturbances such as schizophrenia, and ADHD (Heaton et al., 1993). Physiological evidence supports the activation of prefrontal cortex as a factor in adequate performance

on the WCST with the dorsolateral prefrontal area being especially important (Heaton et al., 1993; Milner, 1963).

Given the association of ADHD with possible pathology of the prefrontal cortex, the WCST has been one of the most frequently used measures in ADHD research (Grodzinsky & Barkley, 1999) with many studies finding differential performance of ADHD individuals as compared to controls (Barkley & Grodzinsky, 1994; Seidman, Biederman, Faraone, Weber, & Ouellette, 1997). However, as Grodzinsky & Barkley (1999) have stated, it's classification rates have not always been shown to be significant. Despite inconsistent findings in ADHD research, factor analyses has supported that attentional processes are involved in adequate performance on the WCST (Greve, Williams, Haas, Littell, & Reinoso, 1996).

Generalizability coefficients for WCST scores based on a single test administration have been reported to range from .39 to .72 and averaged .57 with a median of .60 (Heaton et al., 1993). Generalizability coefficients are analogous to traditional reliability coefficients but generalizability coefficients reflect the fidelity of true-score measurement as compared to reliability coefficients measure of test-item content homogeneity (Heaton et al., 1993). Compared to reliability coefficients, generalizability coefficients in this range appear to be only moderate in value; however, those of .60 or higher are regarded as demonstrating very good scale reliability (Heaton et al., 1993).

Age has been shown to have the strongest relationship to WCST performance (Heaton et al., 1993; Spreen & Strauss, 1998) with performance increasing from 5 years

through 19 years of age and remaining fairly stable between the ages 20 to 50. For adults, there is a gradual increase in performance from lower to higher levels of education and a modest relation between IQ and WCST scores (Heaton et al., 1993; Spreen & Strauss, 1998). The influence of gender has been controversial with some studies reporting that gender is not significantly related to WCST performance. However, there have been some investigations that have found that females tend to outperform males on the WCST (Heaton et al., 1993; Spreen & Strauss, 1998). Some researchers have suggested that Total Number of Errors, Percent Conceptual Level Responses, and Number of Categories Completed appear to be the most significant in differentiating clinical groups from controls (Heaton et al., 1993) while others have used Number of Categories Completed, Failure to Maintain Set, Perseverative Errors and Nonperseverative Errors (Seidman, Biederman, Weber, Hatch, & Faraone, 1998).

Besides identifying assessment protocols and/or batteries, researchers have repeatedly suggested that future studies should attempt to identify biological mechanisms that may help to diagnose ADHD and to differentiate different subtypes of ADHD (Monastra et al., 1999; Riordan et al., 1999). One biological substrate that has been identified in the quest for better diagnosis and differentiation of ADHD is the exploration attentional mechanisms.

Exploration of Attentional Mechanisms

There have been several different methods for exploring attentional mechanisms. These mechanisms have been examined from a neuroanatomical, neurochemical, or neurophysiological perspective (Riccio et al., 1993). The neuroanatomical approach has

focused on localization of brain areas that are thought to be involved in the regulation of attention or inhibition of motor activity. The neurochemical approach has examined the role of neurotransmitters that facilitate or inhibit communication among the neuronal systems implied in ADHD. The neurophysiological approach has attempted to examine the dynamic interaction between the neurochemical and anatomical components that together form a functional system (Riccio et al., 1993).

Neuroanatomical Approaches to Attention

Of the neuroanatomical areas implicated in ADHD, there have been several areas implicated, including the reticular activating system, a group of nuclei in the brainstem and thalamus; the superior colliculus, which is located in the midbrain; the thalamus; the anterior cingulate; and the cortex of the posterior parietal and the frontal lobes (Banich, 1997; Carter, 1998). Attention has been shown to require at least three basic elements of alerting and arousal, orientation, and focus (Banich, 1997; Carter, 1998; Kolb & Whishaw, 1996).

Arousal, a sudden increase in alertness, has been shown to be dependent on the Reticular Activating System (RAS). The cell bodies of the RAS are located in the brainstem and shown to have diffuse connections to most regions of the cortex and hypothalamus (Banich, 1997; Gilman & Newman, 1996; Waxman, 1996). An extension of arousal includes vigilance or sustained attention, the ability to maintain alertness continuously (Banich, 1997).

Orientation involves selective orientation of attention to the source of information (Carter, 1998; van Zomeren & Brouwer, 1994). Orientation has been

demonstrated to involve the superior colliculus and parietal cortex. The superior colliculus aids in shifting attention to new locations or objects by controlling eye movements responsible for bringing stimuli in the periphery quickly into foveal vision (Banich, 1997; Carter, 1998). The posterior parietal cortex is responsible for spatial aspects of attention and the allocation of attentional resources, which in turn assists with selective attention (Banich, 1997; Carter, 1998; Kolb & Whishaw, 1996).

Focus or selective attention has been shown to be facilitated by the lateral pulvinar, a part of the thalamus, which then shunts information about the target to the frontal lobes, which then lock on and maintain attention (Carter, 1998). This focus is facilitated by integration of emotional significance, a function of the cingulate cortex (Banich, 1997; Gilman & Newman, 1996; Waxman, 1996). The cingulate cortex has also been considered a "center" of executive attention.

There has also been another type of attentional system proposed in which the frontal lobe is involved with special regard to short-term memory functions (Kolb & Whishaw, 1996; Posner & Petersen, 1990). Several lines of evidence have supported this system including the activation of various frontal lobe sites across a variety of attentional tasks; the involvement of the frontal structures is proportional to the attentional effort; frontal lobe involvement is response selection is inversely related to practice; the finding that the frontal lobe has an important role in working memory for both sensory events and movements (Kolb & Whishaw, 1996; Posner & Petersen, 1990).

Neuroanatomical/Neurological Basis of ADHD

Researchers have repeatedly noted the similarities between the symptoms associated w/ ADHD and those manifested by lesions or injuries to the brain. As such, there have been many theories formulated in an attempt to clarify the relationship between specific brain structures and those behaviors evident in ADHD. These theories often arise from research and clinical observations of those individuals who have suffered some type of brain injury or through investigation of specific attentional mechanisms.

The frontal lobes in general and specifically the prefrontal cortex have been implicated in ADHD (Barkley, 1998c; Riccio et al., 1993; Samango-Sprouse, 1999). Historically, the frontal lobes have been implicated in the organization of human behavior and in the regulation of psychological processes (Luria, 1973a). This area of the cortex has been shown to play an important role in the regulation of vigilance and in the control of goal-linked behavior (Luria, 1973b). The prefrontal lobes are proposed to be even more involved in the executive functions of attending, integrating, formulating, executing, monitoring, modifying and judgment of all activities associated with the central nervous system (Fuster, 1997, 1999; Halstead, 1947; Stuss & Benson, 1986).

Clinical evaluation has revealed correlation of various brain injuries or pathologies that have been related to ADHD symptoms in both children and adults. Damage to the cingulate cortex results in disorders of temporal integration that are attributable to inattention and lack of interest (Fuster, 1999). Adults with brain injury often have clinical descriptions which include symptoms that are frequently observed with documented frontal lobe injury, including poor impulse control, decreased

flexibility, impaired attention, perseveration, and diminished divergent thinking (Levin & Krauss, 1994). Additionally, behavior changes following pediatric closed head injury (CBI), including social disinhibition and impulsivity, are likely related to frontal dysfunction (Scheibel & Levin, 1997).

Research has indicated that damage to the right dorsolateral prefrontal cortex has been linked to ADHD in children (Edwards-Lee & Saul, 1999; Eslinger, Biddle, & Grattan, 1997). Furthermore, in a study of 23 older adults, Casini & Ivry (1999) found that patients with dorsolateral frontal lobe lesions were significantly impaired on temporal and nontemporal perceptual tasks of two varying levels of attentional load. The authors suggested that the difficulty frontal participants experienced was related to demands of attention (Casini & Ivry, 1999).

Neuroimaging Methods

Contemporary neuroscience and neuropsychology have undergone many changes since the advent of neuroimaging technology. Neuropsychology was behaviorally based and inferential, conducted without the aid of *in vivo* brain imaging techniques (Bigler, Lowry, & Porter, 1997). Even still, there was an underlying assumption about the organic mechanisms underlying mental processes. In 1939, the physicist Hering described "new" devices that permitted direct measure, with photography of organs in layers and called this the "art of tomology" (as reported by, Fitzhugh-Bell, 1997). Although such imaging methods did not appear until the early 1970's, neuroscientists and neuropsychologists have been long been invested in ways of directly imaging the living brain (Posner & Raichle, 1997).

Electroencephalography (EEG). The first attempts to examine the electrical activity of the brain have been attributed to Richard Caton (1842-1926), an English physician (Duffy, Iyer, & Surwillo, 1989; Niedermeyer, 1999). Caton received a grant from the British Medical Association to explore electrical phenomena of the exposed cerebral hemispheres of rabbits and monkeys and published his first findings in 1875 (Duffy et al., 1989; Niedermeyer, 1999).

It was almost 50 years later that a German neuropsychiatrist, Hans Berger (1873-1941), first recorded electrical activity of the human brain in 1924 (Duffy et al., 1989; Niedermeyer, 1999). He coined the term "elektrenkephalogram", which is the German equivalent of electroencephalogram or EEG (Duffy et al., 1989; Niedermeyer, 1999). His findings were first published in 1929, reporting the alpha rhythm, the alpha blocking response, and beta activity (Niedermeyer, 1999). Berger described alpha waves of having a frequency of about 10 cycles per second that appeared to disappear with attention, and beta waves were reported to have frequencies greater than 15 cycles per second (Duffy et al., 1989). Berger also observed that the EEG had different features in neurological disorders, including epilepsy, trauma, and tumors (Duffy et al., 1989).

Widespread interest in electroencephalography and brain electrical activity fueled research efforts in the United States, Canada, Great Britain, Germany, France, and Belgium in the 1930s and1940s (Duffy et al., 1989; Niedermeyer, 1999). The development of clinical and experimental EEG continued to flourish until the 1960s, at which time some of the focus turned to automatic data analysis (Niedermeyer, 1999). Topical EEG analysis and computerized brain mapping became points of focus in the

1970s and 1980s (Duffy et al., 1989; Niedermeyer, 1999). Inexpensive and powerful microprocessors, and high capacity storage methods led to the development of computerized EEG or digital EEG in the 1980s and 1990s(Krauss & Webber, 1999; Lopes da Silva, 1999).

Computerized EEG led into the development of quantitative electroencephalography (QEEG) techniques. It has been shown that the electrical activity of each brain region is homeostatically regulated, resulting in predictable frequency composition of the background EEG. The power spectrum is traditionally divided among 4 separate frequency bands: Delta (0-4 Hz), Theta (4 to 8 Hz), Alpha (8 to13 Hz), and Beta (13 to 32 Hz). It has also been shown that the EEG power spectrum is independent of ethnic background (Hughes & John, 1999).

In QEEG, multichannel recording, usually 19 electrodes at standardized positions, i.e. the International 10-20 System (Jasper, 1958), of eyes-closed, eyes-open, or task condition EEG is artifact rejected and analyzed using the Fast Fourier Transform to quantify the power at each frequency of the EEG averaged across the entire sample. This is called the power spectrum (Hughes & John, 1999). Other factors that can be examined include frequency distribution, amplitude of the electrical signal, locus of the phenomena, waveform morphology, interhemispheric symmetries (symmetry of amplitude, frequency, wave shapes), character of waveform occurrence, regulation of amplitude and frequency, and reactivity (Cantor, 1999).

Besides power and or amplitude QEEG spectral measures, other QEEG variables of interest have included coherence, phase, amplitude asymmetries, and amplitude ratios

(Thatcher, 1999). EEG coherence has been shown to be analogous to a cross-correlation coefficient in the frequency domain. It has shown considerable clinical utility and can directly reflect neural connectivity and neural network dynamics (Nunez, 1995; Thatcher, 1998, 1999). QEEG phase is defined by the amount of time shift of one time series with respect to another. QEEG phase can be used to estimate axonal conduction velocities and is related to the underlying corticocortical connectivity. It also has clinical utility but has been shown to be more variable and must be evaluated with more caution than QEEG coherence (Thatcher, 1999). Amplitude differentiation between electrode sites is related to the functional differentiation in the brain (Thatcher, 1999). Besides amplitude differences, other ratios such as theta/beta ratios, alpha/beta ratios, or theta/alpha ratios have been shown to be of clinical use (Lubar, 1991, 1995; Mann, Lubar, Zimmerman, Miller, & Muenchen, 1992; Monastra, Lubar, & Linden, 2001; Monastra et al., 1999; Thatcher, 1999).

The coupling of brain cell function to the vascular system has been the basis for several methods of functional neuroimaging (Villringer & Dirnagl, 1995). These methods have included PET, fMRI, SPECT, and Near-infrared Spectroscopy (NIRS) (Villringer & Dirnagl, 1995). These methods map localized brain activation through vascular responses, such as an increase in regional cerebral blood flow (rCBF) or a change in blood oxygenation (Villringer & Dirnagl, 1995).

Computerized X-ray tomography (CT). CT scans became available in the early 1970s (Posner & Raichle, 1997). These scans depicted brain structures based on images produced from successive pictures of "slices", or tomes, of structures (Fitzhugh-Bell,

1997). CT did not achieve widespread use until the mid to late 1970s (Bigler, Lowry, & Porter, 1997). It wasn't until the modern advances in computer technology in the early to mid 1980s that CT images became closer to current standards (Bigler et al., 1997).

Magnetic resonance imaging (MRI). About the same time as CT scans were being developed, nuclear magnetic resonance (NMR), or magnetic resonance imaging (MRI) was introduced (Bigler et al., 1997). MRI exploited the fact that many atoms in the presence of a magnetic field behave like bar magnets or compass needles (Posner & Raichle, 1997). By manipulating the hydrogen atoms via radio waves, physical and environmental characteristics could be reconstructed yielding high-detail images of organ anatomy (Posner & Raichle, 1997).

Positron emission tomography (PET). PET scans followed CT. With roots in autoradiography, this technique was developed by using the injection of radioisotopes and x-rays to electronically reconstruct body organs (Posner & Raichle, 1997). These radioisotopes are used in the nanomolar or Pico molar range, allowing PET to assay biological systems without producing significant mass disturbance on the biological system being investigated (Cherry & Phelps, 1996). Since it is an *in vivo* analog of autoradiography, it provides unique information about brain function that is complementary to anatomical information gained by structural imaging techniques such as CT and magnetic resonance imaging (MRI) (Cherry & Phelps, 1996).

Near-infrared Spectroscopy (NIRS). NIRS is also a relatively new non-invasive optical technique used to measure the absorbance of light in brain tissue, which in turn is used to estimate metabolic functioning of the area being assessed. Since near-infrared

light penetrates biological tissues and bone, measurements can be performed through the intact skull (Villringer, Planck, Hock, Schleinkofer, & Dirnagl, 1993). By using light in the near-infrared range, transcranial measurements of reflected light can be performed in NIRS (Villringer & Dirnagl, 1995). The light is produced by a pulsed laser source, or optode, placed on the individual's head. The light is strongly scattered by brain tissue and a second optode fixed to the individual's head several centimeters apart is able to detect some of the reflected light (Obrig et al., 1996).

Despite improvements in anatomical imaging, there has remained a quest for advanced functional or metabolic imaging techniques. Computerized electroencephalography (EEG) or quantitative EEG (QEEG) has enabled physiological mapping of brain activity. PET and single photon emission tomography (SPECT) have also been front-runners for examining temporal changes in brain. Functional MRI (fMRI) is a relatively new technology and has taken the front spot in terms of spatial resolution but studies are still needed to correlate structural and functional observations (Bigler et al., 1997).

Neurochemical and Neurophysiological Approaches to ADHD

These direct measures have contributed to the study of correlative behavior, especially in pathology. There have been studies examining specific neurochemical and neurophysiological activity in the brains of those diagnosed with ADHD. ADHD symptomatology is one of the neurodevelopmental profiles observed in children with generalized resistance to thyroid hormone (GRTH) (Weiss et al., 1993). Neuropsychiatric manifestations of this condition include hyperactivity, behavioral

problems, and cognitive defects (Elia, Gulotta, Rose, Marin, & Rapoport, 1994). Cook et al. (1995) reported that previous studies found ADHD more common in individuals with GRTH when compared to control participants. Weiss et al. (1993) found that the prevalence of thyroid abnormalities is higher in children with ADHD than in the normal population although without a higher incidence of specific GRTH. This research suggests that the prevalence of GRTH in ADHD symptomatology may be rare, but further investigation seems warranted (Weiss et al., 1993). Elia et al. (1994) also found that there may be a higher rate of ADHD symptoms in individuals with GRTH but not all individuals with GRTH are ADHD. Overall, the level of physiological activity appears to be related to metabolic rate.

Research has shown that children with ADHD have decreased metabolic activity in prefrontal lobes and increased activity in primary sensory and sensorimotor cortex (Lou, Henriksen, & Bruhn, 1984; Lou, Henriksen, Bruhn, Borner, & Nielsen, 1989). Lower metabolism in the left anterior frontal lobe has been found to be inversely correlated with the severity of ADHD symptomatology in teenagers (Zametkin et al., 1993). Research utilizing Positron Emission Tomographic (PET) studies have suggested that adults with ADHD have decreased glucose metabolism in particular areas of the brain (Jensen et al., 1993; Weiss et al., 1993).

In contrast to the aforementioned research, some studies on cerebral glucose metabolism have found no differences between normal adolescents and adolescents with ADHD (Ernst et al., 1994). However, adolescent females with a diagnosis of ADHD were reported to have lower glucose metabolism levels than a comparison group (no

ADHD) (Ernst et al., 1994; Zametkin et al., 1993). These findings implicate the possibility of a gender-related role in the pattern of brain metabolism in ADHD (Ernst et al., 1994).

Given the responses of ADHD individuals to differing drugs, it has been suggested that possible neurotransmitter dysfunction or imbalances are associated with ADHD (Barkley, 1998c). Researchers have implicated dopamine and norepinephrine in ADHD and have suggested that they are involved in a span of behaviors, including attention, inhibition and response of the motor system, and motivation (Riccio et al., 1993). Overall, deficiencies in the availability of dopamine and norepinephrine appear to be suggested but cannot be considered conclusive (Barkley, 1998c).

Previous research has supported a prefrontal rCBF increase or a metabolic increase during the performance of frontal cognitive tasks such as the Continuous Performance Test, the Tower of London, and the WCST (Catafau et al., 1998). Where attention is a function required for the performance of frontal cognitive tests (Catafau et al., 1998; Heaton et al., 1993). Recently, SPECT was used to evaluate the role of the frontal cortex and cingulate gyrus during the WCST in a study of 13 non-clinical volunteers. The researchers investigated the cingulate gyrus separately from the prefrontal cortex for an independent evaluation of each structure. A brain SPECT was carried out for both a baseline and the WCST task condition. Results of the Wilcoxon Test indicated a significant rCBF increase during the WSCT ($p < .033$) for the left inferior cingulate and the left posterior frontal regions. Although not statistically significant, it was observed that the rCBF ratios corresponding to left and right prefrontal

regions and to right inferior cingulate were also higher during the WCST in nine out of 13 participants (Catafau et al., 1998).

NIRS has also been used to detect metabolic changes during the performance of cognitive tasks (Fallgatter & Strik, 1997; Obrig & Villringer, 1997). Yamashita et al., 1996, found changes in the level of oxygen, reflected in oxy- and deoxy-hemoglobin concentrations in the left fronto-central region as a function of motor stimulation. Obrig et al., 1996, also found increases in oxy-hemoglobin during functional stimulation and a decrease in deoxy-hemoglobin during motor stimulation. In both paradigms, concentration levels returned to baseline some seconds after terminating the stimulus. Obrig et al. also noted that the changes in response to contralateral stimulation were more pronounced than ipsilateral stimulation. Interestingly, they reported a different pattern for somatosensory stimulation. There were slight decreases in both oxy- and deoxy-hemoglobin concentration during stimulation. Differences between modalities (cognitive, auditory, and visual) were also slight.

In a study of healthy individuals, Fallgatter and Strik (1997) found that there were significant differences of deoxy-Hb between left and right hemispheres in frontal brain areas, as measured by NIRS, during the administration of the continuous performance task. In post-hoc t-tests, the concentration of deoxy-Hb in the left hemisphere was significantly higher than the right frontal hemisphere ($t = 3.32$, $p < .01$) and this difference was lost with return to poststimulus-baseline ($t = 1.92$, not significant) (Fallgatter & Strik, 1997). Thus, the activation was specific for the test condition.

NIRS has also been used in conjunction with other neuroimaging methods. Villringer et al., 1997, examined NIRS accompanied by PET. Changes in oxy-Hb, deoxy-Hb, and total-Hb were measured simultaneously during rest and during performance of a calculation task and a Stroop task. They found a significant correlation between changes in rCBF and changes in total-Hb thus validating NIRS measurements in human adults. Overall, It has been concluded that NIRS measurements demonstrating and increase in oxy-Hb and a decrease in deoxy-Hb reflect cortical activation in the corresponding area of cortex assessed (Obrig & Villringer, 1997). The implication for assessment has been in the measures that may directly tap differential cortical function for ADHD.

Electroencephalographic (EEG) and Neurological Correlates of ADHD

EEG research has demonstrated CPT activation associated with increased frontal, frontotemporal, and temporal beta with increased beta more evident in the right frontal and frontotemporal areas (Rasey, 1998; Riccio et al., 1998). Concurrently, there has been a decrease in alpha and theta in posterior regions of the brain. Increases in theta in the frontal and frontotemporal areas were associated with increased omission errors (Rasey, 1998; Riccio et al., 1998). Overall, in their review of the electrophysiology research with CPTs, Riccio and colleagues (1998) found that better CPT performance was also associated with a greater anterior to posterior gradient.

EEG research has been inconclusive in documenting both the prevalence and or underlying neurophysiological dysfunction in ADD/ADHD children (Chabot, Merkin, Wood, Davenport, & Serfontein, 1996). However, there has been support for

maturational lag and cortical hypoarousal models to clarify the etiology of some of the behavioral problems associated with ADHD (Lazzaro et al., 1998; Mann et al., 1992; Monastra et al., 1999). In a study of 25 right-handed males, 9-12 years of age, with ADHD, topographic brain mapping revealed increased theta (4-7.75 Hz) and decreased beta 1 (12.75-21 Hz) when compared with 27 age and grade level matched controls (Mann et al., 1992). The differences were greater during task conditions than the baseline. Principal component analysis was used to combine variables into two components, which accounted for 82% of the total variance. These two components were comprised of increased theta being more prominent in frontal regions whereas decreased beta was more evident in temporal regions. The results of the study provided for 80% predictability for ADHD group membership and 74% for membership in the control group (Mann et al., 1992).

Chabot & Serfontein (1996) performed QEEG analyses on data from 407 children having attention deficit disorder and compared them with 310 normal children. Discriminant analysis resulted in a specificity of 88% and a sensitivity of 93.7% for distinguishing normal children from those with attention deficit disorder. Two major neurophysiological subtypes were evident within the 92.6% abnormal QEEG profiles encountered. The first subtype showed varying degrees of QEEG slowing, especially in frontal regions, whereas the second showed an increase in QEEG activity, especially in frontal regions. In conjunction with recent magnetic resonance imaging, positron emission tomography, and regional cerebral blood flow studies, these results have indicated neurophysiological dysfunction within the cortical and subcortical structures

that serve the frontal striatal system. Models suggesting both hypo- or hyperarousal of these structures were supported (Chabot et al., 1996).

Most ADHD studies have focused on children; however, behavioral characteristics of ADHD, such as impulsivity and attentional deficits, are known to continue into adolescence and adulthood (Lazzaro et al., 1998). In a sample of 26 unmedicated adolescent males (mean age = 13.4 years), Lazarro et al. (1998) found that absolute theta activity was increased in ADHD adolescents, especially in the anterior regions (F (1, 49) = 5.79, p = .02), as compared to controls. There were no differences found between groups in any other QEEG bands in the anterior region. The ADHD adolescents also showed significantly reduced relative beta activity (F (1, 49) = 10.75, p < .005) in the posterior region as compared to controls. The authors noted that a possible maturational lag, as indexed by increased anterior absolute theta activity and reduced posterior relative beta activity, may be evident for adolescents with ADHD. The results replicated and further supported earlier research by Mann et al. (1992). Recognition of the involvement of frontal regions in adolescent ADHD that was similar to previous results in childhood ADHD suggests that the pathophysiology is maintained in frontal areas (Lazzaro et al., 1998). It was also noted that this facet needs to be addressed in an adult population.

At present, there have been few published studies examining the use of QEEG assessment with adults with ADHD. However, those reviewed to date have indicated significant maturational effects in cortical arousal in the prefrontal cortex, as well as evidence of cortical slowing overall for samples including ADHD adults (Monastra et al.,

2001; Monastra et al., 1999). In a study of 40 undergraduate males (ages 18-25; mean = 21.25 years), Rasey (1998) examined absolute power (magnitude) differences in theta, alpha, and beta bandpasses during an eyes-open baseline, eyes-closed baseline, continuous performance test (CPT) with visual stimuli only and CPT with auditory stimuli only. The CPT used in this study was the Integrated Visual and Auditory (IVA) CPT. Screening measures found that individuals with ADHD were significantly different than controls on the Wender Utah Rating Scale (ADHD: $M = 49.7$, $SD = 7.96$; Control: $M = 26.2$, $SD = 4.32$) (t (29.132) = 11.601, $p < .001$), the diagnostic criteria from the *DSM-IV* (ADHD: $M = 6.75$, $SD = 1.65$; Control: $M = 2.25$, $SD = 1.35$) (t (38) = 9.171, $p < .001$), and the Brown Attention Deficit Disorder Scale for Adults (ADHD: $M = 49.5$, $SD = 5.92$; Control: $M = 34.55$, $SD = 6.07$) (t (38) = 7.885, $p < .001$) (Rasey, 1998). Results reflected a correlative relationship between QEEG and behavioral measures.

For the task conditions, Rasey (1998) found that individuals in the ADHD group ($n = 20$) ($M = 5.1547$, $SD = .3104$), had significantly higher theta activity (t (38) = 2.840, $p = .0035$) than control participants ($n = 20$) ($M = 4.8936$, $SD = .2696$) during the CPT visual stimuli only condition. During the eyes-open baseline, individuals in the ADHD group ($M = 4.3528$, $SD = .2345$) had significantly lower beta activity (t (36.008) = -2.998, $p = .0025$) than controls ($M = 4.6070$, $SD = .2981$). Finally, during the eyes-closed baseline, individuals in the ADHD group ($M = 4.2015$, $SD = .3616$) had significantly lower beta activity (t (33.139) = -2.788, $p = .0045$) than controls ($M = 4.4725$, $SD = .2415$). The results were consistent with the primary hypotheses that adults

with ADHD would demonstrate higher theta activity and lower beta activity during the study conditions (Rasey, 1998).

Monastra et al. (1999) examined a group of 482 individuals between the ages of six and thirty years old in order to evaluate the hypothesis that cortical slowing in the prefrontal region could serve as a basis for differentiating individuals with ADHD from non-clinical control groups. Through examining QEEG activity at the vertex (Cz), in the theta (4-8 Hz) and beta (13-21 Hz) bands, the researchers found a significant association between age and the theta/beta ratio. Scores on this neurometric indicator of cortical slowing were significantly higher in patients with attention deficit disorders than non-clinical controls for ages 6 though 30 (Monastra et al., 1999). Critical values obtained from the theta/beta ratios of the non-clinical controls were shown as being able to serve as basis for accurate classification of the participants of the study in 88% of cases, with no evidence that the degree of cortical slowing was related to participant gender. (Monastra et al., 1999).

Summary

Current diagnosis of ADHD is based on subjective reports of developmentally inappropriate behaviors across the three symptom domains of inattention, impulsivity, and hyperactivity (Swanson, Castellanos, Murias., LaHoste, & Kennedy, 1998). Despite attempts to identify the utility of neuropsychological tests in the evaluation of ADHD (Katz et al., 1998), objective psychological or physiological tests with adequate sensitivity and specificity have not been established to replace a thorough clinical history as the basis for diagnosis (Swanson et al., 1998).

Statement of the Problem

The purpose of the present study is to examine the performance of college-aged adults with ADHD on several neuropsychological measures (PASAT, WCST, IVA) purported to measure various aspects of brain functions that have been implicated in ADHD. Furthermore, as a means to further examine neurophysiological markers in adult ADHD, electroencephalographic recordings will be taken during each of the measures.

Research Hypotheses

The following hypotheses, stated in the null, will be tested in this study:

$H_0$1: For the PASAT, WCST, and IVA, there will be no difference between scores obtained for the ADHD group when compared with the non-ADHD controls.

$H_0$2: During the PASAT, WCST, and IVA, there will be no significant difference in theta, alpha, and beta activity in adults with ADHD when compared with non-ADHD controls.

$H_0$3: There will be no difference in QEEG ratio measures across these instruments in adults with ADHD when compared with non-ADHD controls.

$H_0$4: There will be no significant relationship between QEEG ratio measures and scores from each of the task conditions in adults with ADHD when compared with non-ADHD controls.

$H_0$5: For the PASAT, WCST, and IVA, there will be no discriminant predictors differentiating membership in the ADHD group when compared with the non-ADHD controls.

CHAPTER TWO

Methodology

Chapter Two presents a description of the participants, an overview of the methodology, and specific information about the procedures used in this study. Following recommendations for neuropsychological testing in ADHD, standard measures of working memory and sustained attention were used. Although no standard battery of neuropsychological tests has been discerned for diagnosing behavioral components of criterion-referenced ADHD (Katz et al., 1998), the present investigation has included some of the most reliable indices; furthermore, this study has addressed problems associated with immaturity in frontal lobe functioning.

Continuous performance tasks and tests of set shifting and reversal, working memory, and cognitive processing were employed because they have been shown to discriminate ADHD from controls (Biederman et al., 1996; Biggs, 1995; Horton, 1996; Katz et al., 1998; Kovner et al., 1998; Seidman, Biederman, Faraone, Weber, Mennin et al., 1997). By selecting adult participants, differences between those with ADHD and controls were believed to be more exclusively due to frontal functions having reached maturity (Grodzinsky & Diamond, 1992; Lovejoy et al., 1999). The issue of frontal lobe maturity was further examined in refining the sampling procedure through age-dependent analyses and grouping.

Participants

Participants were recruited via information flyers and class announcements in undergraduate and graduate courses in the College of Arts & Sciences, the College of

Education and through the University of Tennessee's Office of Disability Services. Before becoming involved in any aspect of the study, participants were informed as to the purpose and requirements for participation. All participants provided informed consent via a signed permission form. All aspects of this study were reviewed and approved by the University of Tennessee, Office of Compliances, Institutional Review Board.

For both clinical and control groups, incentives for participation included extra course credit, when applicable, upon completion of participation in the study; this is part of standard procedure established by the Department of Psychology's research participant pool. Participants were free to terminate their involvement in the study at any time and were fully informed that they would not receive course extra credit unless they participated fully as per the department's *a priori* requirements. All participants participated fully in the study. Students with ADHD may have had an additional, intrinsic incentive to contribute to the knowledge of accurate diagnosis of adult ADHD. Participant selection did not discriminate on gender, racial, and/or ethnic background.

Participants for the clinical group were recruited via an informational letter that was mailed by the Office of Disability Services with whom they were registered. In order to protect students' confidentiality, no student information was provided directly to the researcher; interested individuals were asked to respond to the researcher to initiate their desired participation. The letter was mailed to those students who were currently registered with the Office of Disability Services and were recognized as having ADHD. Those registered required that a physician or psychologist had made the diagnosis of ADD/ADHD with supporting documentation not more than 3 years old. Inclusion

criteria for the clinical group required that participants be formally registered with the Office of Disability Services with a diagnosis of ADD or ADHD. Clinical participants were also required to demonstrate clear characteristics of the disorder as a part of data collection to validate symptom integrity and increase internal homogeneity of the clinical group. Measures included a self-report, *DSM-IV* symptom checklist for ADHD and were employed to decrease threat to internal validity of the investigation. The diagnostic criteria from the *DSM-IV* (American Psychiatric Association, 1994) required each individual clinically identified to experience symptoms of at least one of the subtypes of ADHD for a minimum of the past six months. Inclusion criteria were that characteristics be defined by personal endorsement of at least six of nine hyperactive-impulsive items or at least six of nine inattentive symptoms.

Participants for the control group were recruited from undergraduate and graduate classes in the Department of Psychology and the Department of Counseling, Deafness, and Human Services via posted flyers and class announcements. Inclusion in the control group required individuals to be free of current or past history of ADHD symptomatology as assessed though the personal interview, self-report, and the *DSM-IV* symptom checklist for ADHD. Inclusion criteria for participants in the control group required that each individual endorse three or fewer ADHD symptoms of either inattentive or hyperactive-impulsive subtyping.

Exclusion criteria for both groups were three: (1) an obtained standard score less than 85 on the Peabody Picture Vocabulary Test – 3rd Ed. (PPVT-III), (2) a history of neurological disorder, head injury, or substance abuse, and (3) previous diagnosis of

specific learning disabilities, as assessed though the health history questionnaire and personal interview. The PPVT-III has been validated as an appropriate screening instrument for general intellectual functioning (Lezak, 1995; Spreen & Strauss, 1998) and has been used in concert with neuropsychological assessment in several earlier studies (Hutchens, 1989). Neurological disorders, head injury, and substance abuse can alter the EEG and were thus negatively indicated in the present study. Individuals with a comorbid learning disability would decrease the homogeneity of the study group and were also excluded.

The use of medication for both groups was also addressed and controlled to increase within-group homogeneity and decrease threats to internal validity. To control for medication effects, no member of the control group was evaluated while using any type of medication. For those ADHD participants being treated with stimulant medication, testing was conducted after a medication-free period of at least 12 hours. Given that the half-life of Ritalin, Pemoline, and other short-acting stimulants necessitate at least twice-daily dosing (Wilens, Spencer, & Biederman, 1998), individuals who were currently taking medication for ADHD were asked to schedule their appointments before their first dose of medication for the day and that at least 12 hours had elapsed since their last dose. Given the clinical action of these medications and their short half-life, the clinical group was considered to be medication free as well. For both control and clinical groups, the only exception to these rules was for females taking oral contraceptives.

Instruments

Paced Auditory Serial Addition Task (PASAT)

The PASAT is a neuropsychological assessment instrument validated in the measure of attention and information processing (Brittain et al., 1991; Gronwall, 1977; Gronwall & Sampson, 1974). The version used in this study was the adaptation by Levin et al. (1987), adopted for use due to established norms and procedures. Administration consisted of participants listening to a pre-recorded tape, which delivered a series of 50 single digits in different random sequences across each of four trials. Following standardized administration, participants were instructed to add pairs of numbers such that each number was added to the one that immediately preceded it (Spreen & Strauss, 1998). The number of correct responses for each trial and the total number of correct responses across the four separate trials were used to quantify performance as per standardized scoring.

Wisconsin Card Sorting Test (WCST)

The WCST, a neuropsychological instrument, has been validated in the assessment of abstract reasoning and ability to shift cognitive strategies in response to changing environmental contingencies (Berg, 1948; Grant & Berg, 1948; Heaton et al., 1993; Heaton et al., 1999). The version used in the present study was the Wisconsin Card Sorting Test: Computer Version 3 for Windows Research Edition (WCST: CV3) and has been used as part of a neuropsychological evaluation of Adult ADHD in previous study (Seidman, Breiter et al., 1998). The following subscores assessed performance: Categories completed, Failure to maintain set, Perseverative errors, and Nonperseverative

errors. These variables have been previously described as offering information about executive functions and may yield specific information about executive function deficits (Heaton et al., 1993).

Integrated Visual and Auditory Continuous Performance Test (IVA)

The IVA is an integrated, 13-minute auditory and visual continuous performance test designed to assess response control and attention (Sandford, 1995). Using computerized administration, the test involved responding or inhibiting a response to counter-balanced auditory and visual stimuli for 500 trials, 1.5 seconds in length, thus demanding constant, sustained attention for adequate performance (Sandford, 1995). The IVA was administered according to standardized procedure.

Electroencephalograph (EEG) Recording

The EEG, a measure of the brain's electrical activity, was recorded from scalp electrodes. The quantitative referential EEG was recorded from 19 electrodes in an array following the International 10-20 Placement System (Andreassi, 1995; Jasper, 1958), with linked earlobe references. Standardized preparation procedures involved all electrode impedances being maintained at or below 5 KOhms. The raw EEG was collected using a Lexicor NeuroSearch-24 Electroencephalograph with a sampling rate of 128 samples per second. Frequency band passes were theta (4-8 Hz), low-alpha (8-10Hz), high-alpha (10 - 13 Hz), low-beta (13-21 Hz), and high-beta (21-32 Hz).

Procedure

Interested participants reported to the Brain Research and Neuropsychology Laboratory in order to obtain informed consent, completion of the screening measures,

and EEG recordings with all tasks completed in a single, two-hour appointment. All data collection was performed between the hours of 8 A.M. and 1 P.M.

Valid administration of the PASAT, WCST, & IVA required that participants have normal or corrected vision and hearing sufficient to adequately comprehend the test instructions and be able to visually and aurally discriminate the stimulus parameters. These requirements were assessed with the Health History Questionnaire. No volunteers were excluded due to these criteria.

Following a personal interview reviewing and elaborating on the participant's responses on the Health History questionnaire, all participants completed the self-report symptom checklist developed from *DSM-IV* criteria for ADHD (American Psychiatric Association, 1994). Participants were then assessed for general intellectual ability using the PPVT-III. All participants, ADHD and controls, had a PPVT-III standard score greater than or equivalent to 85.

EEG Collection

All task conditions and simultaneous EEG recordings took place in a sound and light attenuated experimental room (60 square feet). Participants were seated in a standard office chair, accompanied by the examiner. They were instructed to remain as motionless as possible and to limit the total number of eye-blinks, vertical eye movements, and horizontal eye movements during the recording session.

EEG Recordings were made using a fitted electrode cap (Electro Cap Inc.) to insure standardized electrode placement. Participants had the electrode cap with 19 electrodes in standard positions placed on their heads. Conductive gel was applied to each

electrode by a small tube inserted through the electrode; the gel was used to form a conductive pathway between the electrode and the scalp. There was no significant discomfort with this procedure either in the preparation or the wearing of the cap during the testing. Two ear clip electrodes were placed on the earlobes after a light cleaning with OmniPrep solution, which removed skin oil and allowed for optimum electrode contact. All creams and gels used were hypoallergenic.

There were additional electrodes placed at the outer canthus of each eye to monitor horizontal eye movement and an electrode above and below the left eye to monitor vertical eye movement. These were used to determine flutter-type eye blinks, which can mimic alpha activity in the EEG record and slow horizontal eye movements may produce what appears to be excessive slow activity (EEG delta) overlying the frontal lobe. Electrodes were also placed at the base of the mentalis muscle on the chin with a reference to the cheek to monitor EMG activity while speaking, as was required during the PASAT. Use of these "guard" electrodes has been shown to facilitate artifact recognition at time of recording, during artifact rejection, and in differentiating artifact from cerebral phenomena during statistical analysis (Duffy, 1994). Accordingly, data from these guard channels were used to aid in the elimination of epochs containing artifactual data stemming from muscle activity.

All participants completed five quantitative electroencephalographic (QEEG) recordings during two baselines and the three different test conditions. The conditions included a 3-minute eyes-closed QEEG baseline, 3-minute eyes-open QEEG baseline,

administration of the PASAT, administration of the WCST: CV3, and administration of the IVA. The order of administration remained standard for all participants.

The eyes-closed baseline required the participant to sit for 3 minutes, while remaining as motionless as possible. During the 3-minute eyes-open baseline and administration of the PASAT, participants were asked to gaze at a 17-inch computer monitor, adjusted to eye-level, approximately three feet in front of them, and were again asked to refrain from any extraneous body or eye movement.

During administration of the PASAT, the EEG collection began with the first stimulus item and continued until the completion of the assessment. Standardized administration of the PASAT allows for the examiner to stop the audio stimulus to correct re-occurring errors. For the purpose of the present study, the audiotape was not paused in order to establish a standardized length of administration. No participants missed a sufficient number of items that would have discontinued the task; provisions were established *a priori* had such occurred. Scoring of the PASAT yielded a total number of correct responses for each time trial and a total score across all four time trials. The WCST: CV3 and IVA were administered on the same computer; participants viewed these instruments on the same 17-inch computer monitor as was used in the baseline, also adjusted to eye-level, approximately three feet in front of them. The participants responded by using a computer mouse on an adjoining, adjustable stand; it offered minimal movement and muscle strain for its operation to minimize muscle artifact on the EEG recording. For both the WCST: CV3 and IVA, all participants were allowed to become familiar, comfortable, and competent with the computer and use of the mouse.

During the IVA, EEG recordings were only taken during the actual CPT portion of the IVA test, not during the warm-up, practice, or cool-down portions.

Data Analysis

The data from the EEG was initially inspected off-line with a Pentium 200 processor using Lexicor NeuroLex software (Lexicor Medical Technology, 1992). With a sampling rate of 128 samples per second, standardized application of NeuroLex allowed for visual examination of 2-second epochs with the option of accepting or rejecting the epoch for analysis. Data from the artifact channels was carried throughout the visual analysis to aid in differentiating phenomena attributed to the cerebral EEG and phenomena of non-cerebral origin.

Following artifact rejection, a Fast Fourier Transform (FFT) analysis of the artifact-free 2-second epochs was used to determine absolute EEG activity in the theta, low-alpha, high-alpha, low-beta, and high-beta bands using the Exporter Quantitative EEG Export Program (Lexicor Medical Technology, 1996). Magnitude, the energy within a particular band's frequency range, was reported in millivolts (uV). The exported magnitude values were placed into an Excel spreadsheet and were squared, resulting in absolute power measures. The absolute power data was then imported into SPSS 10.0 for Windows. To obtain normality of distribution and homogeneity of variance, log transformations of absolute power scores were generated before submitting the data to statistical analysis.

Statistical analyses examined absolute power data during the two baseline and three task conditions, for 5 (theta, low-alpha, high-alpha, low-beta, high-beta) frequency

bands, for the 9 central (F3, FZ, F4, C3, CZ, C4, P3, PZ, P4) QEEG channels corresponding to the International 10-20 System of electrode placement (see Figure 1). The nine central sites were chosen due to them being less susceptible to the influence of EMG activity and as a safeguard against the active nature of the neuropsychological tasks.

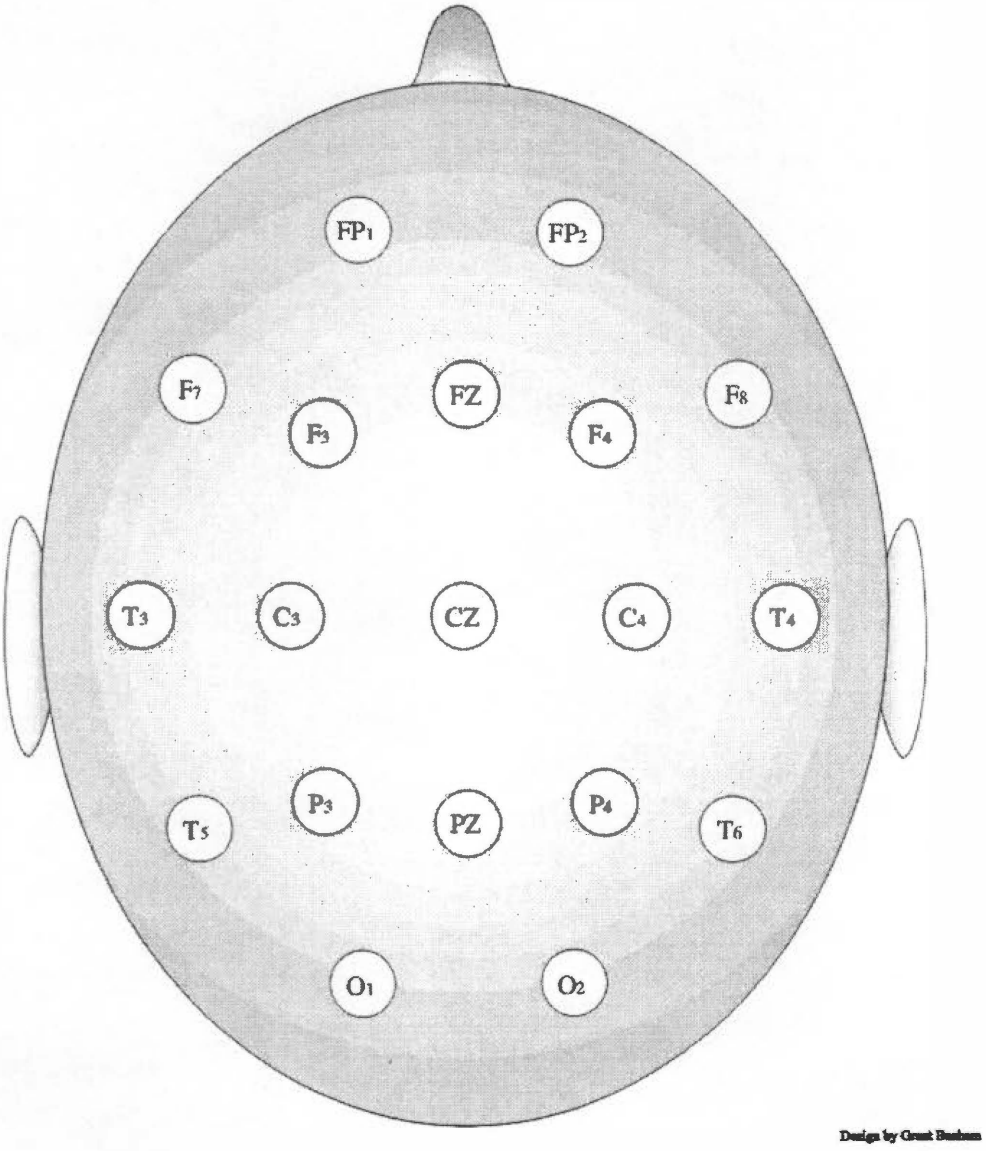


Figure1. International 10-20 System of EEG Electrode Placement.

CHAPTER THREE

Results

Chapter Three provides the results of the statistical analyses used to evaluate the hypotheses of this dissertation and the results of the statistical analyses of exploratory hypotheses generated during the course of the present investigation.

Analysis of Demographic Variables

Thirty-three individuals were screened using the previously identified, criteria-based questionnaires, *DSM-IV* symptom checklist, intellectual assessment, neuropsychological measures, and QEEG recordings. Sixteen of the participants were female, 17 were male. All participants were adults, aged 18 years or older (M = 25.64, SD = 8.93) and were currently enrolled as undergraduate or graduate students at The University of Tennessee.

Initial Participant Groups

Control Sample. The control sample consisted of 23 adults aged 18 – 55 years (M = 24, SD = 9.16). One participant reported having a head injury with loss of consciousness and was excluded from the control group and subsequent statistical analysis. The remaining participants comprising the non-clinical sample were aged 18 – 44 years (M = 22.59 years, SD = 6.32). This sample yielded an aggregate estimate of intelligence in the average range (85 – 115) as obtained by the PPVT-III (M = 108.91, SD = 10.77, Range = 88 - 132). Symptom endorsement on the *DSM-IV* checklist did not support the behavioral characteristics or pathology of ADHD (see Table 1).

Table 1: *Demographic Variable Means: Initial Comparison*

Demographic Variable	Group	*M*	*SD*	*t*	*df*	*p*
Age	Control[a]	22.59*	6.32	-2.670	30	.012
	ADHD[b]	29.40	7.47			
Grade	Control[a]	3.41	3.63	-.489	30	.628
	ADHD[b]	4.00	1.63			
PPVT-III	Control[a]	108.91	10.77	.421	30	.677
	ADHD[b]	107.20	10.33			
Inattentive Symptoms	Control[a]	0.91***	1.19	-15.377	30	<.001
	ADHD[b]	7.80	1.14			
Hyperactive Symptoms	Control[a]	1.32***	1.13	-9.995	12.922	<.001
	ADHD[b]	7.10	1.66			

Note. Grade refers to years of college education.

[a]$n = 22$. [b]$n = 10$.

*$p < .05$. **$p < .01$. ***$p < .001$.

ADHD Sample. The clinical sample consisted of 10 adults aged 21 - 47 years (*M* = 29.40, *SD* = 7.47). This sample yielded an aggregate estimate of intelligence in the average range (85 – 115) as obtained by the PPVT-III (*M* = 107.20, *SD* = 10.33, Range = 88 - 125). Symptom endorsement on the *DSM-IV* checklist supported a diagnosis of ADHD. The clinical group was not segregated by ADHD subtype. According to the self-report checklist, three participants endorsed symptoms supporting predominantly inattentive and the remaining seven adhered to classification of combined inattentive and hyperactive-impulsive.

Initial Comparison. Overall, the preliminary analyses compared the demographic data (age, grade, PPVT) for each of the two groups. Independent *t*-tests revealed significant age differences between the two groups, t (30) = -2.670, p = .012. On the average, controls (M = 22.59, SD = 6.32) were younger than the ADHD participants (M = 29.40, SD = 7.47). No differences were found for PPVT, t (30) = .421, p = .677 or years of education, t (30) = -.489, p = .628 (see Table 1).

Realignment of Control Sample

The significant difference in age between the two diagnosis groups prompted a realignment of the non-clinical sample, with those individuals younger than 21 years of age placed into a "younger" group (M = 19.08, SD = .67) and individuals 21 years old or older being placed into a "older" group (M = 26.8, SD = 7.52). The cut off age of 21 was chosen in an effort to better match the clinical group in which the youngest member was 21 years old. The two sub-groups of control participants were then compared.

The junior and senior control members differed with regard to age, t (9.118) = -3.232, p = .01, and years of college education, t (9.588) = -2.277, p = .047. The control sub-groups did not differ on reported symptoms of inattentiveness but differed on the number of reported hyperactive-impulsive symptoms, t (16.589) = 2.25, p = .038. Younger controls (M = 1.75, SD = 1.29) reported on average more hyperactive-impulsive symptoms than older controls (M = .80, SD = .63) (see Table 2).

Given the apparent equivalence in age of the "older" control group as compared to the ADHD group, the older grouping of non-clinical individuals were chosen for comparison and were used in all statistical comparisons against the clinical sample. All subsequent referrals to the control group apply to the "senior" sub-sample (age ≥ 21) of the non-clinical participants.

Comparison of Realigned Controls to ADHD Sample

The comparison control group was comprised of 5 females and 5 males. Nine controls were right-handed; all were Caucasian. The ADHD sample was comprised of 4 females and 6 males; nine were Caucasian and one was Hispanic. Of the clinical sample, nine members were right-handed; one reported being ambidextrous but used their right hand for writing and using a computer mouse.

Independent samples t-tests were conducted to evaluate the null hypotheses that the control and clinical groups did not differ on demographic variables or on self-reported symptoms of ADHD on a *DSM-IV* checklist. The results were not significant for the demographic variables of age, grade, and IQ estimates from the PPVT-III, thus supporting the null hypothesis and verifying demographic equivalence.

Table 2: *Demographic Variable Means of Control Subgroups*

Demographic Variable	Group	*M*	*SD*	*t*	*df*	*p*
Age	Younger[a]	19.08**	0.67	3.232	9.118	.010
	Older[b]	26.80	7.52			
Grade	Younger[a]	1.83*	0.94	2.277	9.588	.047
	Older[b]	5.30	4.74			
PPVT-III	Younger[a]	109.75	9.64	.393	20	.699
	Older[b]	107.90	12.46			
Inattentive Symptoms	Younger[a]	1.17	1.34	1.117	20	.277
	Older[b]	0.60	0.97			
Hyperactive Symptoms	Younger[a]	1.75*	1.29	2.250	16.589	.038
	Older[b]	0.80	0.63			

Note. Grade refers to years of college education.

[a]$n = 12$. [b]$n = 10$.

*$p < .05$. **$p < .01$. ***$p < .001$.

On the *DSM-IV* checklist, the results of the *t*-tests were significant for the number of self-reported Inattentive symptoms, t (18) = -15.274, $p < .001$, and the number of self-reported Hyperactive symptoms, t (11.549) = -11.195, $p < .001$. Adults with ADHD (M = 7.80, SD = 1.14) reported a higher average of inattentive symptoms than those adults without ADHD (M = .60, SD = .97). Furthermore, adults with ADHD (M = 7.10, SD = 1.66) reported a higher average of hyperactive-impulsive symptoms than those adults without ADHD (M = .80, SD = .63). Overall, these results suggest that the two groups were demographically equivalent except for their self-reported behavioral symptoms of ADHD (see Table 3).

Analysis of Research Hypotheses

Comparison of non-ADHD and ADHD adults on neuropsychological measures

Independent *t*- tests were calculated to examine the null hypothesis that for the Paced Auditory Serial Addition Task (PASAT), Wisconsin Card Sorting Test (WCST), and Integrated Visual and Auditory Continuous Performance Test (IVA), there would be no difference between scores obtained for the ADHD group when compared with the non-ADHD controls.

Paced Auditory Serial Addition Task (PASAT). Following a Bonferroni correction (.05/5 = .01) to control for Type I errors, the results of the *t*-tests were significant for all trials ($ps < .002$) and total score of the PASAT, t (18) = 5.460, $p < .001$. Overall, adults with ADHD had consistently lower scores on each time trial and the subsequent total score on the PASAT (see Table 4). These results suggest that adults

Table 3: *Demographic Variable Means: Final Comparison Groups for Study*

Demographic Variable	Group	*M*	*SD*	Range	*t*	*df*	*p*
Age	Control[a]	26.80	7.52	21 - 44	-.775	18	.448
	ADHD[b]	29.40	7.47	21 - 47			
Grade	Control[a]	5.30	4.74	1 – 13	.820	11.108	.429
	ADHD[b]	4.00	1.63	2 – 7			
PPVT-III	Control[a]	107.90	12.46	88 – 132	.137	18	.893
	ADHD[b]	107.20	10.33	88 – 125			
Inattentive Symptoms	Control[a]	0.60***	0.97	0 – 3	15.274	18	<.001
	ADHD[b]	7.80	1.14	6 – 9			
Hyperactive Symptoms	Control[a]	0.80***	0.63	0 – 2	11.195	11.549	<.001
	ADHD[b]	7.10	1.66	5 – 9			

Note. Grade refers to years of college education.

[a]$n = 10$. [b]$n = 10$.

*$p < .05$. **$p < .01$. ***$p < .001$.

Table 4: *Neuropsychological Performance of Control and ADHD Participants*

	Control[a]		ADHD[b]				
	Mean	*SD*	*Mean*	*SD*	*t*	*df*	*p*
Paced Auditory Serial Addition Task							
Trial 1	44.50**[†]	5.52	32.80	8.44	3.667	18	.002
Trial 2	41.90***[†]	5.47	24.80	8.56	5.324	18	<.001
Trial 3	37.40***[†]	6.98	21.60	7.35	4.929	18	<.001
Trial 4	33.90***[†]	8.52	15.70	6.22	5.457	18	<.001
Total	157.70***[†]	21.26	94.90	29.51	5.460	18	<.001
Wisconsin Card Sorting Test							
Categories	5.20	1.87	5.80	0.42	-.988	18	.336
Failure to maintain set	0.30	0.67	0.80	1.40	-1.018	18	.322
Perseverative errors	100.90	34.05	100.10	10.99	.071	10.86	.945
Nonperseverative errors	98.40	15.94	99.40	9.50	-.170	18	.867
Integrated Visual and Auditory CPT							
Full Scale RCQ	107.30	7.57	102.60	14.62	.903	18	.378
Auditory RCQ	106.00	7.50	101.40	10.92	1.098	18	.286
Visual RCQ	107.00	8.50	103.40	17.17	.594	18	.560
Full Scale Attention Quotient	90.80*	9.05	71.30	24.20	2.386	11.47	.035
Auditory AQ	85.90*	12.34	61.80	28.92	2.423	12.17	.032
Visual AQ	96.60	9.09	87.70	16.91	1.466	18	.160

Note. CPT = Continuous Performance Test; RCQ = Response Control Quotient; AQ = Attention Quotient.

[a]$n = 10$. [b]$n = 10$.

*$p < .05$. **$p < .01$. ***$p < .001$.

[†] Significant after Bonferroni correction for multiple comparisons

with ADHD perform at lower levels on tasks involving working memory that require efficient information processing.

Wisconsin Card Sorting Test (WCST). Using a Bonferroni correction (.05/4 = .0125) to control for Type I errors, the results of the *t*-tests were not significant for Categories Completed, (t (18) = -.988, p = .336), Failure to Maintain Set, (t (18) = -1.018, p = .322), number of Perseverative Errors, (t (10.855) = .071, p = .945), or number of Nonperseverative Errors, (t (18) = -.170, p = .867) (see Table 4). On the WCST, non-clinical controls and ADHD adults demonstrated relatively equivalent performance on a task requiring abstract reasoning and ability to shift cognitive strategies in response to changing environmental contingencies.

Integrated Visual and Auditory Continuous Performance Test (IVA). Following a Bonferroni correction (.05/6 = .008), the results of the *t*-tests revealed that there were no significant differences between the groups as measured by the IVA Full Scale Response Control Quotient (t (18) = .903, p = .378), the IVA Auditory Response Control Quotient (t (18) = 1.098, p = .286), the IVA Visual Response Control Quotient (t (18) = .594, p = .560), and the IVA Visual Attention Quotient (t (18) = 1.466, p = .160). The IVA Full Scale Attention Quotient, (t (11.470) = 2.386, p = .035), and IVA Auditory Attention Quotient, (t (12.172) = 2.423, p = .032) were significant at the .05 level but did not meet criteria after the Bonferroni correction (see Table 4). Overall, both groups demonstrated equivalent test performance in terms of impulsivity or response control. They also performed at comparable levels during parts of the task requiring visual attention. However, in general, ADHD adults

on average had lower overall attentional performance as compared to controls with specific deficits in auditory attention.

In sum, the null hypothesis of equivalent neuropsychological task performance between groups was not supported. The obtained results indicated that the two groups' neuropsychological performance differed on specific tasks. Adults with ADHD performed significantly lower on tasks of working memory and speeded information processing. ADHD adults also had lower overall attentional performance during a task requiring sustained attention, especially during parts requiring auditory attention. However, these differences on the continuous performance test did not meet statistical significance after correction for multiple comparisons. Lastly, when compared on a task requiring abstract reasoning and cognitive set shifting, the groups performed at equivalent levels.

QEEG Band Power during Neuropsychological Task Performance

A repeated-measures multivariate analysis of variance (MANOVA) was conducted to examine the null hypothesis that adults with ADHD would not differ from non-ADHD adults in specific bandpasses of QEEG activity during the PASAT, WCST, and IVA. The single between-subjects factor was diagnosis; the two within-subjects variables were task (PASAT, WCST, IVA) and QEEG bandpass (theta, low-alpha, high-alpha, low-beta, high-beta) activity at Fz, the standardized electrode location in the center of the frontal region of the scalp. Although QEEG lacks the spatial resolution of other neuroimaging methods, it is generally accepted that those cortical areas underlying a given electrode contribute maximally to the activity recorded at that location. By

examining QEEG activity at Fz (see Figure 1, p. 52), activity of the frontal lobes in general was examined. Furthermore, the frontal lobes have been implicated in the successful performance of all three tasks.

A significant interaction was found among diagnosis, condition, & band pass, Wilks' $\lambda = .28$, $F\ (8, 11) = 3.62$, $p = .026$. The multivariate μ^2 based on Wilks' λ was .73. Additional repeated-measures MANOVA's were then conducted as follow-up tests to the original MANOVA, subtracting one factor at a time in a stepwise fashion and examining the multivariate *F* to determine which bandpasses were contributing the most to the multivariate effect (Weinfurt, 1995; Wilkinson, 1975).

Removal of the high-beta factor (data from theta, low-alpha, high-alpha, low-beta included) resulted in a loss of the significant interaction. Low-beta was then removed and a significant interaction was found among diagnosis, condition, and bandpass (theta, low-alpha, high-alpha included) Wilks' $\lambda = .53$, $F\ (4, 15) = 3.31$, $p = .039$. When examined without theta, low-alpha and high-alpha yielded a significant main effect for condition, Wilks' $\lambda = .40$, $F\ (2, 17) = 12.64$, $p < .001$, but without a significant interaction. The significant interaction re-emerged when theta and high-alpha were analyzed concomitantly, Wilks' $\lambda = .62$, $F\ (2, 17) = 5.20$, $p = .017$. This was the largest multivariate *F*, suggesting that the majority of the multivariate variance was accounted for activity in the theta and high-alpha bands. See Figure 2 for a graph of theta and high-alpha activity at Fz across all three tasks.

A 2 x 3 repeated-measures ANOVA was then conducted with condition as the factor and the activity in the respective bandpass at Fz as the dependent variable.

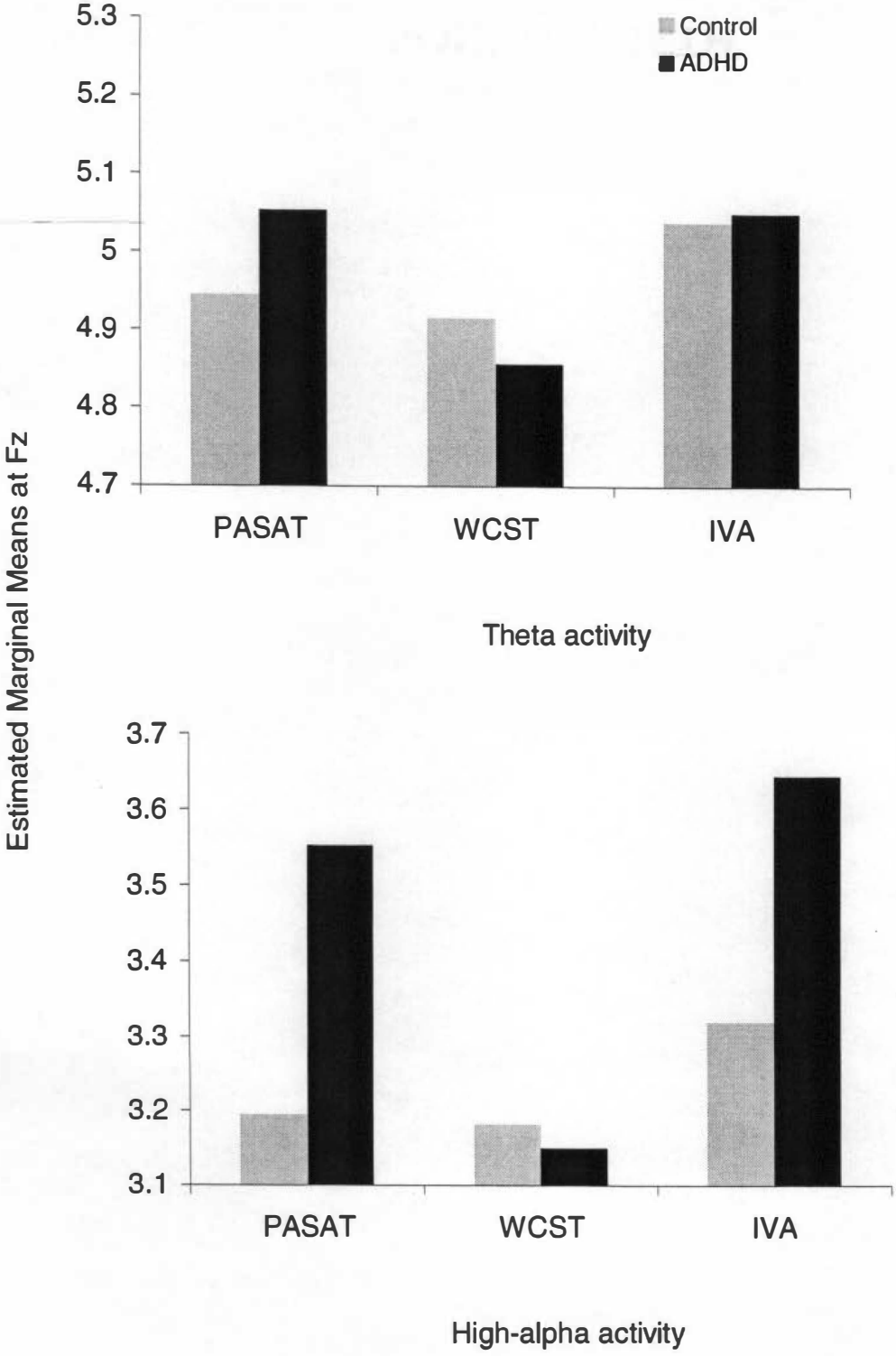


Figure 2. Estimated Marginal Means of Theta and High-alpha Activity.

Diagnostic group membership was the single between subjects factor. The results of the ANOVA for theta indicated a significant main effect for condition, Wilks' $\lambda = .61$, F (2, 17) = 5.34, $p = .016$, μ^2 based on Wilks' λ was .39. A paired-samples t-test was then conducted to evaluate which conditions differed significantly from each other in relation to theta activity at Fz. Using a Bonferroni correction (.05/3 = .017), the results indicated that, across the entire sample of ADHD and controls, the mean theta activity at Fz during the WCST ($M = 4.89$, $SD = .41$) was significantly lower than the mean theta activity at Fz during the IVA ($M = 5.05$, $SD = .49$), t (19) = -2.83, $p = .011$.

Results of the ANOVA for high-alpha indicated a significant interaction between diagnosis and condition, Wilks' $\lambda = .70$, F (2, 17) = 3.74, $p = .045$, μ^2 based on Wilks' λ was .31. A paired-samples t-test was then conducted for controls and ADHD's to examine the conditions that differed significantly from each other in relation to high-alpha activity at Fz. Only one of the six pairwise comparisons was significant while controlling for familywise error rate across the six tests at the .05 level, using the Holm's sequential Bonferroni procedure (see Table 5). The results indicated that for the ADHD group, the mean high-alpha activity at Fz during the WCST ($M = 3.15$, $SD = .45$) was significantly lower than the mean high-alpha activity at Fz during the IVA ($M = 3.65$, $SD = .71$), t (9) = -4.24, $p = .002$.

In sum, from the initial analysis we can reject the null hypothesis that during the PASAT, WCST, and IVA, there are no significant differences in theta, alpha, and beta activity at Fz between adults with and without ADHD. Furthermore, the follow-up analyses suggest that the majority of the variance in this multivariate effect is accounted

Table 5: *Paired-sample t-tests for High-alpha activity at Fz*

Paired Conditions: Controls[a]	Condition	*M*	*SD*	*t*	*df*	*p*
Pair 1	PASAT	.692	.058	-.196	9	.849
	WCST	.696	.052			
Pair 2	PASAT	.692	.058	-.695	9	.505
	IVA	.703	.063			
Pair 3	WCST	.696	.052	-.495	9	.633
	IVA	.703	.063			

Paired Conditions: ADHD[b]	Condition	*M*	*SD*	*t*	*df*	*p*
Pair 1	PASAT	.742*	.119	2.635	9	.027
	WCST	.683	.088			
Pair 2	PASAT	.742	.119	-.277	9	.788
	IVA	.747	.113			
Pair 3	WCST	.683**[†]	.088	-4.224	9	.002
	IVA	.747	.113			

[a] $n = 10$. [b] $n = 10$.

*$p < .05$. **$p < .01$. ***$p < .001$.

[†] Significant after Bonferroni correction for multiple comparisons

for by cortical activity in the theta and high-alpha bands, and this cortical activity varied according to diagnosis and task. On average, both non-clinical controls and ADHD participants produced less fronto-central theta during the WCST as compared to the IVA. When activity in the high-alpha band was evaluated, ADHD participants produce significantly lower levels of high-alpha at Fz during the WCST as compared to the IVA.

QEEG Power Ratio Measures during Neuropsychological Task Performance

In addition to QEEG band power measures, the ratio of specific band passes in relation to other band passes has been used to indicate degree of cortical involvement during active tasks. For example, the theta/beta ratio is a measurement of slow wave activity as compared to fast wave activity. Historically, the theta/beta ratio has been used in the ADHD literature (e.g., Mann et al. 1992; Monastra et al. 1999) but possible relationships of activity in the low-alpha and high-alpha bands were also of interest, thus, those ratio measures were calculated as well. The statistical strategy used to evaluate QEEG band power activity was also used to evaluate the QEEG ratio measures as related to task performance. Again, activity at Fz, the standardized electrode location in the center of the frontal region of the scalp was used for comparisons.

A repeated-measures MANOVA was conducted to evaluate the null hypothesis that adults with ADHD would not differ from non-ADHD adults in specific ratio measures of QEEG activity at Fz. The single between-subjects factor was diagnosis group; the two within-subjects variables were task (PASAT, WCST, IVA) and QEEG ratio measure (theta/low-beta, low-alpha/low-beta, high-alpha/low-beta).

A significant interaction was found among diagnosis, condition, and ratio, Wilks' $\lambda = .50$, F (4, 15) = 3.82, $p = .025$. The multivariate μ^2 based on Wilks' λ was .51. Subsequent MANOVA's were conducted, removing one ratio measure factor at a time, to determine which measures were contributing the most to the overall variance. Removal of the high-alpha/low-beta ratio factor resulted in a significant interaction between condition and ratio (theta/low-beta and low-alpha/low-beta), Wilks' $\lambda = .54$, F (2, 17) = 7.16, $p = .006$. The multivariate μ^2 based on Wilks' λ was .46. Diagnosis was not a significant contributor to this relationship.

The high-alpha/low-beta ratio was reintroduced and low-alpha/low-beta was removed resulting in a significant multivariate interaction between condition, ratio (theta/low-beta & high-alpha/low-beta), and diagnosis, Wilks' $\lambda = .61$, F (2, 17) = 5.34, $p = .016$. The multivariate μ^2 based on Wilks' λ was .39.

A 2 x 3 repeated-measures ANOVA was then conducted with condition as the factor and the dependent variable being activity as represented by the respective ratio measure at Fz. Diagnosis group was the single between subjects factor. The results of the ANOVA for theta/low-beta ratio resulted in no significant relationships for either condition or the diagnosis x condition. The results of the ANOVA for high-alpha/low-beta ratio indicated a significant main effect for condition, Wilks' $\lambda = .61$, F (2, 17) = 5.36, $p = .016$, μ^2 based on Wilks' λ was .39. A paired-samples t-test was then conducted across controls and ADHD's to examine the conditions that differed significantly from each other in relation to activity at Fz as measured by the high-alpha/low-beta ratio measure. One of the three pairwise comparisons was significant,

controlling for familywise error rate across the three tests at the .05 level, using the Holm's sequential Bonferroni procedure. The results indicated that both for both ADHD's and controls, the mean high-alpha/low-beta activity at Fz during the WCST (M = .69, SD = .007) was significantly lower than the mean high-alpha/low-beta activity at Fz during the IVA (M = .73, SD = .009), t (19) = -2.91, p = .009.

Overall, from the initial analysis the null hypothesis was not supported. As measured by the theta/low-beta, low-alpha/low-beta, and high-alpha/low-beta ratio measures at Fz, there was a significant difference in cortical activity between adults with and without ADHD during the PASAT, WCST, and IVA. The follow-up analyses and examination of the multivariate F suggest that the majority of the variance in this multivariate effect was accounted for by activity as measured by the theta/low-beta and high-alpha/low-beta ratio measures. When examined further, mean activity at Fz, as measured by the high-alpha/low-beta ratio measure, is significantly lower during the WCST than the mean activity during the IVA for both adults with and without ADHD.

Correlation of QEEG Power Ratios to Neuropsychological Task Performance

Given that the theta/low-beta and high-alpha/low-beta ratio measures accounted for the majority of the variance in the previous analysis, these two ratio measures were then used in comparison against scores from the neuropsychological tasks that the two groups were found to score differently on, the PASAT and the IVA. The ratio measure as collected at Fz was used for the correlational analysis.

Correlation coefficients were computed among the two ratio measures (theta/low-beta, high-alpha/low-beta) measured at Fz for a given task and scores from the respective

task. The scores used were PASAT Total, IVA Full Scale Attention Quotient, and IVA Auditory Attention Quotient. Using the Bonferroni approach to control for Type I error across the 12 correlations, a p-value of less than .004 (.05/12 = .004) was required for significance. The results of the correlational analyses for each of the task conditions presented in Table 6 indicate that only 1 out of the 12 correlations was statistically significant following correction for multiple comparisons.

For the ADHD group, the correlation between activity measured by the theta/low-beta ratio and the IVA Auditory Attention Quotient was significant, r (8) = -.82, p = .004. Also for the ADHD group, the correlation between the theta/low-beta ratio and the IVA Full Scale Attention Quotient approached statistical significance but did not meet the Bonferroni adjusted criteria, r (8) = -.78, p = .007. Overall, for the ADHD group higher theta/low-beta ratio values at Fz were significantly correlated with lower IVA Auditory Attention Quotient scores.

Prediction of Group Membership from Neuropsychological Task Scores

The ADHD and control samples performed significantly differently on the PASAT and IVA. Accordingly, scores from those two measures were evaluated in their ability to differentiate membership in either the ADHD group or control group.

A discriminant analysis was conducted to determine whether two predictors – PASAT total score and IVA Full Scale Attention Quotient – could predict ADHD diagnosis. The overall Wilks' lambda was significant, $\lambda = .37$, $\chi^2(2, N = 20) = 16.83$, $p <$ .001, indicating that overall, the predictors differentiated between the two participant groups. Accordingly, 63% of the variability of the scores for the discriminant function is

Table 6: *Correlation of QEEG Ratios at Fz with Neuropsychological Test Scores*

Test Score	Group	*TB*		*HAB*	
Paced Auditory Serial Addition Test		r^c	*p*	r^c	*p*
Total Score	Control[a]	0.338	.339	-0.628	.052
	ADHD[b]	-0.292	.413	0.152	.674
Integrated Visual and Auditory CPT					
Full Scale Attention Quotient	Control[a]	-0.163	.653	0.434	.210
	ADHD[b]	-0.784	.007	-0.249	.489
Integrated Visual and Auditory CPT					
Auditory Attention Quotient	Control[a]	-0.176	.626	0.207	.565
	ADHD[b]	-0.821*	.004	-0.290	.417

Note. TB = Theta/low-beta ratio; HAB = High-alpha/low-beta ratio.

[a]$n = 10$. [b]$n = 10$. [c]$df = 8$

* Significant after Bonferroni correction for multiple comparisons, .05/12 = .004

accounted for by the differences between the participant groups on the PASAT and IVA. The control group (M = 1.23) had higher mean scores on the overall discriminant function, while the ADHD group (M = -1.23) had lower scores.

When prediction of diagnostic group membership was attempted, 95% of the individuals in the sample were classified correctly. In order to take into account chance agreement, a kappa coefficient was computed which resulted in a value of .90, indicating very strong predictive value. Finally, to assess how well the classification procedure would predict in a new sample, the percent of individuals accurately classified was estimated by using the leave-one-out technique, which correctly classified 95% of the cases.

In sum, the null hypothesis that there are no discriminant predictors from the PASAT and IVA for predicting ADHD group membership can be rejected. By using the PASAT Total score and the IVA Full Scale Attention Quotient, the diagnosis of ADHD can be successfully predicted within 95% probability in this sample.

Exploratory Analyses

The following exploratory hypotheses were also tested in this research and stem from observations of the raw EEG during data collection and the results of the previous hypotheses. In additional to activity at Fz, the exploratory analyses investigate the relationship of QEEG information from the nine central electrode sites; F3, Fz, F4, C3, Cz, C4, P3, Pz, P4 (see Figure 1, p. 52). The nine central sites were chosen due to their relatively lower risk of artifactual contamination by muscular activity as compared to the peripheral recording sites. The bandpasses used for these exploratory analyses were theta

and high-alpha, subsequent to their contributions to the majority of the variance in the previous analyses.

Comparison of Theta and High-alpha Activity Averaged Across Nine Central Sites During the Neuropsychological Tasks

The null hypothesis, that there would be no significant difference in theta and high-alpha activity averaged across the nine central electrode sites between adults with ADHD and non-ADHD controls, was evaluated through two multiple regression analyses. The results of the first analysis were not significant (R^2 = .05, F (3, 16) = .27, p = .85) and suggested that averaged theta activity and task condition did not account for a significant amount of the diagnostic variability. Results of the second multiple regression analysis revealed that high-alpha activity and task condition also did not account for a significant amount of the diagnosis variability, R^2 = .25, F (3, 16) = 1.77, p = .19. Accordingly, the null hypothesis was supported in that there were no significant differences in averaged theta and averaged high-alpha activity, individually averaged across the central nine electrode sites, in adults with ADHD as compared to non-ADHD controls during completion of the PASAT, WCST, and IVA.

Comparison of Adults with ADHD and Non-clinical Controls on QEEG "Index Value" across Baseline and Task Conditions

Previous studies have calculated an "index value" which consists of the average of activity across several different tasks, as measured at Cz (Monastra et al., 2001; Monastra et al., 1999). The Cz electrode position is located at the vertex, in the middle of the scalp, on top of the head (see Figure 1, p. 52).

A multiple regression analysis was conducted to evaluate how well the theta/low-beta and high-alpha/low-beta ratio measures averaged across all conditions at Cz predicted ADHD diagnosis. The linear combination of QEEG ratio measures was not significantly related to the diagnostic category, F (2, 17) = 3.00, p = .077. Accordingly, the null hypothesis was supported. Adults with ADHD did not differ significantly from non-clinical controls in regard to theta/low-beta and high-alpha/low-beta ratio measures at Cz, when averaged across all task conditions.

Separation of ADHD Participants into Subgroups

Despite the significant interaction between activity in the theta and high-alpha bands during the neuropsychological tasks, the statistical analyses did not reflect a significant level of difference between the two groups as was suggested by observation and visual review of the QEEG data. This prompted a return to the objective measures to evaluate possible differences in the ADHD group. In fact, with a criteria of 1.5 standard deviations from the mean, five of the ten ADHD participants evidenced significantly impaired quotient scores for at least two of the three Attention quotient scores on the IVA.

Comparison of QEEG Ratios in ADHD Subgroups

Subsequent to this observation, the ADHD group was separated according to the 1.5 SD criteria, resulting in two subgroups of 5. An ANOVA was conducted to evaluate the null hypothesis that the two subgroups differenced in cortical activity at Fz as measured by the theta/low-beta ratio measure during each neuropsychological task condition. All three of the comparisons were significant while controlling for familywise

error rate across the three tests at the .05 level, using the Holm's sequential Bonferroni procedure.

The results indicated that the ADHD participants who scored at least 1.5 standard deviations below the mean on at least two of the three IVA Attention Quotient Scales, the "impaired" group, displayed higher average theta/low-beta ratio values during the IVA (M = 1.11, SD = .004) as compared to the other ADHD participants (M = .98, SD = .004), t (9) = 29.55, p = .001. The "impaired" group also had higher theta/low-beta ratios during the WCST (M = 1.13, SD = .007) as compared to the other ADHD subgroup (M = 1, SD = .003), t (9) = 12.76, p = .007) and during the PASAT (M = 1.13, SD = .008) as compared to the other ADHD subgroup (M = 1.02, SD = .005), t (9) = 7.03, p = .029 (See Figure 3). The two ADHD subgroups did not differ according to low-alpha/low-beta or high-alpha/low-beta ratio measures (see Table 7).

In sum, for this group of ADHD adults, 50% demonstrated more impaired functioning on the IVA. These adults also demonstrated higher theta/low-beta ratios at Fz across all three neuropsychological tasks as compared to their ADHD colleagues.

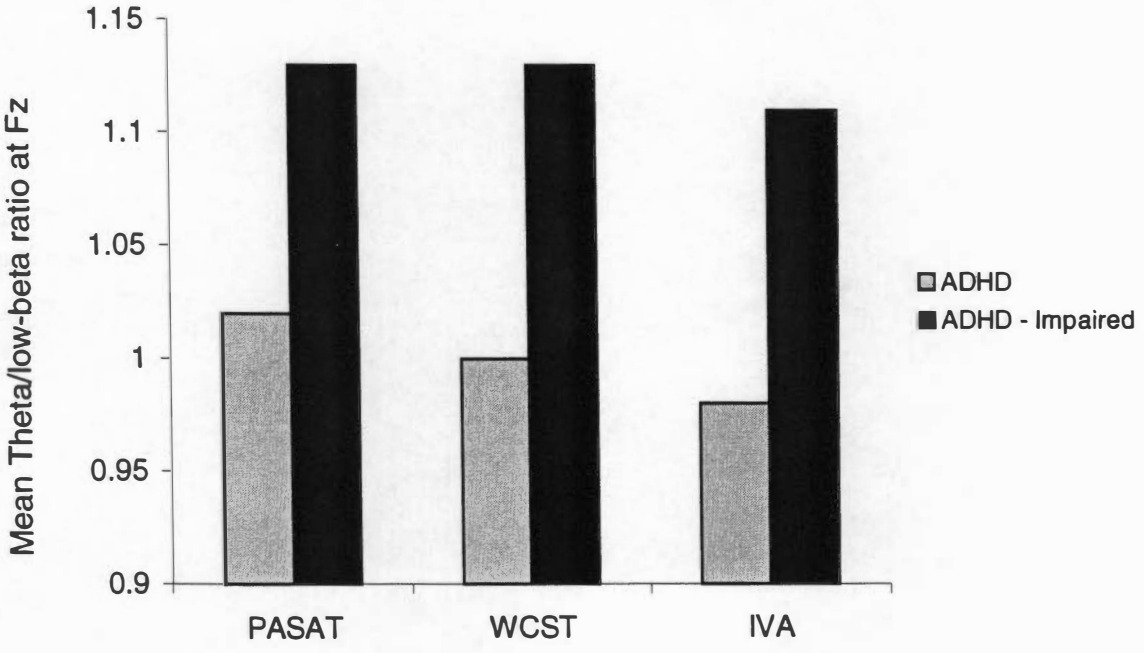


Figure 3. ADHD subgroups' mean theta/low-beta ratios at Fz across tasks

Table 7: *ADHD Subgroups' QEEG Ratios at Fz During Task Performance*

Theta/low-beta Ratio	Subgroup[a]	*M*	*SD*	*F*	*p*
PASAT	ADHD	1.016	0.050	7.027	0.029[†]
	ADHD+	1.126	0.078		
WCST	ADHD	1.000	0.029	12.760	0.007[†]
	ADHD+	1.128	0.075		
IVA	ADHD	0.984	0.036	29.545	0.001[†]
	ADHD+	1.114	0.040		

Low-alpha/low-beta Ratio		*M*	*SD*	*F*	*p*
PASAT	ADHD	0.690	0.199	0.382	0.554
	ADHD+	0.772	0.220		
WCST	ADHD	0.630	0.123	0.732	0.417
	ADHD+	0.684	0.068		
IVA	ADHD	0.692	0.176	0.296	0.601
	ADHD+	0.752	0.173		

High-alpha/low-beta Ratio		*M*	*SD*	*F*	*p*
PASAT	ADHD	0.682	0.089	3.219	0.111
	ADHD+	0.803	0.121		
WCST	ADHD	0.654	0.094	1.096	0.326
	ADHD+	0.712	0.080		
IVA	ADHD	0.699	0.126	1.935	0.202
	ADHD+	0.794	0.086		

Note: ADHD+ refers to subgroup of ADHD subjects who scored at least 1.5 SD below the mean on at least two of three Attention Quotient Scales of the IVA.

[a]each subgroup $n = 5$.

[†] Significant after correction for multiple comparisons using Holm's sequential Bonferroni procedure, each ratio evaluated separately.

CHAPTER FOUR

Discussion

Chapter Four provides a discussion of the results obtained from the statistical analyses used to evaluate the hypotheses of this study. Limitations of the present investigation, potential implications for clinical application and directions for additional research are also discussed.

Background

The purpose of the present study was to determine if significant differences existed between adults with and without Attention Deficit Hyperactivity Disorder (ADHD) when assessed on self-report questionnaires of diagnostic symptomatology, neuropsychological tests of working memory, attention, and set shifting; and cortical brain activity as measured by quantitative electroencephalography (QEEG). The primary hypothesis, as stated in the null, was that there would be no discernable differences between the two groups.

Previous studies have demonstrated differential performance on neuropsychological measures with ADHD individuals having specific patterns of cognitive deficits. Furthermore, research has supported measured cortical differences, primarily regional slowing or hypoactivation, as indicated by QEEG. However, the QEEG literature to date has been sparse with regard to adults with ADHD and few studies have examined neuropsychological performance with concurrent QEEG measurement. If in fact discernable differences are found between adults with and

without ADHD in neuropsychological performance and cortical electrical activity, these differences may prove useful in the accurate diagnosis of ADHD in adults.

Initial Comparison and Realignment of Control Sample

The initial comparison of the control (n = 22) and ADHD (n = 10) participants revealed that on average, the control subjects were significantly younger than the participants with ADHD ($p < .05$). Given the results of previous studies that have indicated that hyperactive symptoms decrease with age (Biederman, Mick, & Faraone, 2000), age equivalence between the comparison groups was a highly desirable component of this study.

Subsequent to the discovery of significant age differences between groups and the direct implication of age on the developmental qualities of attention and working memory (Laursen, 1997; Swanson, 1999), the control group was divided with those individuals less than twenty-one years of age placed into a "younger" subgroup and those 21 or older placed into an "older" subgroup. The subsequent analyses revealed that these two subgroups of controls differed in regard to age ($p < .01$), number of years of college education ($p < .05$), and number of hyperactive symptoms ($p < .05$) reported on the *DSM-IV* checklist. The "older" controls had more years of college education and on average, reported less hyperactive symptoms. Following these results, the "older" subgroup of control participants was chosen for all subsequent comparisons against the ADHD group.

Comparison of Realigned Adult Controls to ADHD Sample

The analysis of the older control group subset (n = 10) to the ADHD group (n = 10) revealed that the groups did not differ in regard to age, number of years of college

education, or estimate of general intelligence from the Peabody Picture Vocabulary Test – 3rd ED. (PPVT-III). Furthermore, as compared to the subgroup of control participants of equivalent age, educational level, and general level of intellectual functioning, adults with ADHD endorsed significantly more inattentive ($p < .001$) and hyperactive-impulsive ($p < .001$) symptoms on the self-report measure of *DSM-IV* criteria.

These results are consistent with current literature that indicate that adults may accurately self-report their own behavioral symptoms of ADHD (Murphy & Schachar, 2000). Previously, there has been concern about the reliability and validity of self-report measures in adult ADHD (Arcia & Conners, 1998; Feifel, 1996). Given that the current diagnostic criteria is based on subjective recall of symptoms prior to the age of 7 and an accurate account of current behavior, any indication that adults may serve as their own referees should assist clinicians in the diagnostic process.

Neuropsychological Performance

The neuropsychological tasks used in the current investigation were putative measures of working memory and information processing, cognitive strategy and set shifting, as well as sustained attention or vigilance. These areas correspond to the theoretical neuropsychological underpinnings of ADHD. Specifically, the cognitive components of working memory, development of cognitive strategy, and ability to sustain attention fall under the rubric of "executive functions." Of the various neuropsychological domains, deficits in these executive functions are those most commonly observed in ADHD.

In the present study, the Paced Auditory Serial Addition Test (PASAT) was used to measure working memory and information processing. The Wisconsin Card Sorting Test – Computer Version 3 (WCST) was used to measure abstract reasoning and ability to shift cognitive sets in response to contingent feedback. Lastly, the Integrated Visual and Auditory Continuous Performance Test (IVA) was used to assess sustained attention or vigilance. By using these three instruments, the core neuropsychological domains that are most often impacted in ADHD were examined in an objective and reproducible manner.

Paced Auditory Serial Addition Task (PASAT). As reflected by the PASAT total score, adults with ADHD performed significantly less well on this task as compared to control subjects ($p < .001$). They also scored significantly lower on each of the four time trials of the PASAT (range from $p < .001$ to $p < .002$) (see Table 4). The results support a decreased capacity and rate of information processing in the clinical sample. Overall, the adults with ADHD demonstrated less efficient information processing.

In an exploratory factor analysis of attentional tasks, the PASAT was found to load heavily on a factor termed auditory-verbal working memory (Barkley, 1997a; Robertson, Ward, Ridgeway, & Nimmo-Smith, 1996). Furthermore, functional neuroimaging studies with the PASAT have found cortical activations, especially in frontal regions, consistent with models of working memory (Schweitzer et al., 2000). In comparison, results of the same study illustrated that adults with ADHD not only demonstrated lower performance on the PASAT but, also exhibited a more diffuse pattern of cortical activation; findings also revealed a relative lack of task-related frontal

activation demonstrated by the non-clinical controls (Schweitzer et al., 2000). The present results offer additional support for such a lack of information processing efficiency in adults with ADHD.

Both adult males and females with ADHD scored significantly lower on all four trials and subsequent total score of the PASAT as compared to their non-ADHD counterparts. Previous studies of PASAT performance in ADHD have consisted primarily of male participants. Schweitzer (2000) found that six males with ADHD performed significantly lower than six males without ADHD on a version of the PASAT. The present findings lend support to the utility of the PASAT in the evaluation of adult ADHD in both genders.

Wisconsin Card Sorting Test (WCST). In general, the WCST measures an individual's ability to identify relevant stimuli dimensions and to employ response contingencies to successfully develop concepts. As such, it is used as a measure of executive function, a cognitive domain implicated in ADHD. In the present investigation, adults with ADHD did not differ significantly from non-ADHD adults on the WCST. As measured by the WCST variables of Number of Categories Completed, Failure to Maintain Set, Number of Perseverative Errors, and Number of Nonperseverative Errors, the two groups displayed equivalent performance. These variables have been previously described as offering information about executive functions (Heaton et al., 1993).

The adults with ADHD in this study performed at a level equivalent to their non-clinical counterparts. Schweitzer (2000) suggested that individuals with ADHD may

develop compensatory mental and neural strategies to compensate for disrupted abilities such as inhibiting attention to non-relevant stimuli. This is a required skill to maintain the correct response set during the WCST. It is also possible that in this sample of relatively high-functioning adults, the WCST was not challenging enough to exacerbate any potential deficits.

Overall, the present finding of equivalent performance between adults with and without ADHD is congruent with some previous investigations. Whereas some investigations with older adolescents and young adults offer support for the WCST in classifying ADHD (Seidman, Biederman, Faraone, Weber, & Ouellette, 1997), others have not found this support with adults (Gansler et al., 1998; Seidman, Biederman et al., 1998). Despite these inconsistencies, the WCST has been one of the most frequently used measures in ADHD research (Grodzinsky & Barkley, 1999).

Integrated Visual and Auditory Continuous Performance Test (IVA). During this test of sustained attention, ADHD subjects in the present study generated lower total scores as measured by the IVA's Full Scale Attention Quotient (FSAQ) ($p = .035$) and Auditory Attention Quotient (AAQ) ($p = .032$). However, although the results were significant at the .05 level, the obtained differences for the FSAQ and AAQ failed to meet criteria after correction for multiple comparisons (.05/6 = .008). Response Control Quotient scores and Visual Attention Quotient scores were not significantly impacted.

As a general rule, the hyperactive and impulsive symptoms of ADHD tend to decline with age at a higher rate than inattention symptoms (Biederman et al., 2000). Hyperactivity and impulsivity are also the symptoms of ADHD that are reflected in the

IVA Response Control Quotient scores. Thus, the present results are consistent with the general finding of decreased hyperactivity-impulsivity as individuals with ADHD grow older. In sum, despite the apparent lack of statistical significance, the results of the IVA continuous performance test are congruent with previous research and reported observations of behavior in adult ADHD. Adults with ADHD were impacted on measures reflecting attentional demands and not on measures of response control.

Summary of Neuropsychological Measures

The present results found that the two groups differed on their ability to perform on the PASAT and the IVA. Subsequently, general scores from these two measures were evaluated in their ability to accurately classify the participants into their respective groups. In contrast, the WCST was not found to discriminate the performance of adults with ADHD from that of non-clinical controls, a finding previously reported in the literature (Gansler et al., 1998; Seidman, Biederman et al., 1998). Overall, these results are congruent with previous investigations of neuropsychological performance in adult ADHD (Epstein et al., 1998; Schweitzer et al., 2000; Walker et al., 2000) in that tasks involving working memory and attention are likely impacted whereas those involving categorical abstract thinking may not be affected.

The PASAT Total score and the IVA Full Scale Attention Quotient significantly differentiated the two participant groups ($p < .001$) with controls averaging higher performance. When prediction of group membership was attempted, 95% of the individuals were correctly classified with a kappa value of .90, indicating that these two

measures had very strong predictive value. When the classification procedure was assessed for a new sample, 95% of cases were correctly classified.

Whereas all four trials and subsequent total score on the PASAT were markedly different between groups, differences in IVA Attention Quotient scores were less pronounced. Although apparently disparate, these findings can be accounted for by the different cognitive domains assessed by each instrument. Whereas the PASAT is a demanding test of working memory and divided attention, the IVA loads heavier on an individual's ability to remain engaged in a mildly boring repetitive task. In fact, the PASAT has been recommended when subtle attentional deficits are being measured (Lezak, 1995), and would be expected to be more sensitive to subtle levels of impairment as seen in adults with ADHD.

Quantitative Electroencephalography (QEEG) Analyses

Quantitative electroencephalography (QEEG) was incorporated as an adjunctive measure with the neuropsychological instruments used to evaluate the performance of adults with and without ADHD. QEEG, as an indicator of the electrical activity of the cerebral cortex as measured by scalp electrodes, offers a relatively inexpensive and noninvasive means of measuring the activity of the brain during completion of cognitive tasks. In the current investigation, the QEEG was collected during the concurrent completion of the aforementioned neuropsychological tasks. Thus, the QEEG obtained during each of the task conditions has been shown to be one indicator of the specific neural processes required for the task demands.

QEEG activity is generally described in relation to specific frequency bands of activity and expressed in hertz (hz) or cycles per second. Magnitude, the energy within a particular band's frequency range, is reported in millivolts (uV); magnitude squared results in power values. The bands used in the present study were theta (4 – 7 hz), low-alpha (8 – 10 hz), high-alpha (10 – 13 hz), low-beta (13 – 21 hz), and high-beta (21 – 32 hz). In general, slow wave activity, theta and low-alpha, is considered to be inversely related to the degree of cortical involvement in a given task. In contrast, faster activity in the high-alpha and beta range is assumed to be directly associated with functional cortical activation.

In general, hypofunctioning of the frontal lobes has been implicated in ADHD. The frontal lobes are also typically recruited in the successful performance of the aforementioned neuropsychological tasks. By examining QEEG activity at Fz, the standardized electrode location in the center of the frontal scalp region, activity of the frontal lobes in general was examined. Furthermore, by comparing the QEEG measures obtained from adults with ADHD to the QEEG measures obtained from non-clinical controls, the neural processes unique to ADHD might be discerned.

Topographic Power

In the examination of potential QEEG power differences at Fz, results indicated that there was a significant interaction between diagnosis, task, and QEEG bandpass (p = .026). Follow-up analyses revealed that the majority of the variance between groups and task conditions was due to activity in the theta and high-alpha bands (p = .017). These results suggest that the most discriminating QEEG bandpasses were either inversely

associated with cortical activation, namely theta, or directly associated with cortical activation, specifically high-alpha. In turn, the degree of activation found varied according to condition and the neuropsychological task being performed.

Theta Activity. Both adults with and without ADHD had increases in slow wave theta activity during the IVA as compared to the WCST ($p = .011$). Accepting the general assumption that increases in slow wave activity are inversely related to the degree of cortical involvement, this finding suggests that the WCST required more active frontal cortical activation for it's successful completion than did the IVA. Inherently, for non-clinical subjects, the WCST is generally accepted to be more cognitively demanding than a continuous performance test such as the IVA.

Theoretically, when the demands of a task become less engaging, one would expect theta levels to increase as the subject become less engaged or more inattentive (Lubar, 1995). The overall increase in frontal theta for both groups during the IVA, a measure of vigilance and sustained attention, as compared to the WCST, a test of abstract reasoning and set shifting, is congruent with this theoretical relationship between theta activity and the task demands of an inherently boring sustained attention task such as the IVA.

An absence of significant differences was found in fronto-central theta activity between the two groups on the IVA. There was also no difference between groups during the WCST and the PASAT, a test of working memory and divided attention. These findings are in contrast to previous studies that have revealed significant differences in QEEG activity between adults with and without ADHD; these differences were most

prominent during active tasks (Mann et al., 1992; Monastra et al., 2001; Monastra et al., 1999).

The lack of significant difference between groups in fronto-central theta activity during the IVA may be attributed to the relatively small differences in test performance between the two groups. The performance levels of the ADHD subjects in this study may have been too high to detect discernable differences in QEEG activity that lower levels of vigilance may have revealed. However, this explanation can not account for the lack of differences in theta activity between groups in light of markedly different performance during the PASAT. For this working memory task, it is possible that both groups increased their frontal theta in response to the demands of the test. Although this appears to be in direct contrast to previously accepted roles of theta activity in ADHD, recent studies have suggested that working memory tasks are often associated with increases in frontal-midline theta activity (Gevins & Smith, 2000; Nunez & Silberstein, 2000).

This is an apparent paradox. A possible explanation may lie in a recent investigation of activity patterns in the medial prefrontal cortex (MPFC), a portion of the frontal lobes directly underlying the electrode location at Fz. The MPFC is among those brain areas having the highest baseline metabolic activity at rest and one that decreases from baseline across goal-directed behaviors in functional imaging studies (Gusnard, Akbudak, Shulman, & Raichle, 2001) as shown by functional magnetic resonance imaging (fMRI).

Gusnard et al. (2001) used fMRI to examine activity in the MPFC during externally focused and self-referential tasks. The level of activity in the MPFC varied

according to the nature of the task being performed. It was suggested that reductions from baseline activity in ventral MPFC are consistent with attention-demanding tasks (Gusnard et al., 2001). In contrast, the presence of self-referential or introspectively oriented mental activity appears to be associated with increases from baseline in regions of MPFC (Gusnard et al., 2001).

Although the exact relationship between QEEG power measures and the functional activity demonstrated in fMRI experiments has yet to be defined, the aforementioned study may offer some insight into the apparently discordant findings of increased theta activity during working memory tasks. If increases in theta are in fact inversely related to functional cortical activity such as in the MPFC, the decreases in ventral MPFC activity during tasks requiring sustained focused attention would be accompanied by increases in theta activity. Extending this hypothesis further, the typical observation of increased fronto-central theta in ADHD might be explained by an individual's hyperfocus to internal states. Instead of being inattentive, individuals with ADHD might have a predisposition to be internally focused to the detriment of attention to external cues or the specific task at hand.

High-alpha Activity. Adults with ADHD also showed increases in higher frequency activity, specifically high-alpha, during the IVA as compared to the WCST (p = .002). The finding of elevated high-alpha suggests that the ADHD adults utilized increased frontal cortical activation to perform the IVA. As noted in the summary of neuropsychological task performance, ADHD adults performed at equivalent levels on the WCST and tended to perform at slightly lower levels on the IVA.

The increased frontal activation in high-alpha during the IVA may be indicative that the ADHD adults had to work harder to maintain task performance as compared to the non-clinical comparison group. As such, this increased activity, as measured over their frontal lobes, may suggest decreased efficiency as compared to those individuals without ADHD. This interpretation is consistent with hypofunctioning of the frontal lobes in ADHD (Castellanos, 2001). Gevins and Smith (2000) reported that when initially confronted with a novel spatial working memory tasks, non-clinical participants with relatively better performance initially made use of frontal regions and shifted activity to parietal regions as the task progressed. In contrast, non-clinical participants with relatively lower performance relied more exclusively on frontal regions (Gevins & Smith, 2000).

QEEG Ratio Measures

Traditionally, specific QEEG ratio measures have been effective in differentiating individuals with ADHD versus those without ADHD. By and large, the ratio of theta activity to beta activity has been higher in individuals with ADHD as compared to controls. Results of the present study reveal that the theta/low-beta ratio and the high-alpha/low-beta ratio measures varied across specific neuropsychological tasks (e.g. the WCST and the IVA) and also between groups ($p = .016$). However, when examined further, adults with ADHD and non-clinical controls were not found to have significantly different theta/low-beta ratios ($p > .05$), either between groups or during the different task conditions. This finding is in contrast to previous research supporting elevated theta/low-

beta ratios in individuals with ADHD (Mann et al., 1992; Monastra et al., 2001; Monastra et al., 1999).

Alternative contemporary observations may account for this finding. Monastra et al. (1999) found that theta/low-beta ratios values tend to decrease with age and are less prominent in ADHD adults as compared to non-clinical controls of equivalent age. The existence of the aforementioned frontal midline theta phenomenon may also impact the use of ratio measures to diagnose adult ADHD. If frontal midline theta increases during tasks requiring working memory and there is not an accompanying increase in fast wave activity, the use of the theta/low-beta ratio would be negated as an indicator of inattention.

The high-alpha/low-beta ratio was found to vary significantly across tasks for both groups (p = .016) but univariate between-group differences only approached statistical significance (p = .056). As such, the high-alpha/ low-beta ratio might offer another neurometric for the evaluation of ADHD, at least for adult subjects. In the present investigation, adults with ADHD tended to display elevated high-alpha/low-beta ratios as compared to controls.

Relation of QEEG Ratio Measures to Task Performance

Despite the apparent differences in activity in the theta and high-alpha bands, both between groups and tasks, only one ratio measure was significantly related to specific task scores after correcting for multiple comparisons. Using the Bonferroni approach to control for Type I errors across the 12 correlations, a p-value of less than .004 (.05 / 12 = .004) was required for significance.

The ADHD group obtained lower IVA Auditory Attention Quotient scores as their theta/low-beta ratios at Fz increased, r (8) = -0.82, p = .004. This finding appears to support increases in theta activity, as related to beta activity, were associated with poorer attentional performance during a continuous performance test. The ADHD group also gravitated toward lower Full Scale Attention Quotient scores as their theta/low-beta ratios at Fz increased, r (8) = -0.784, p = .007, but the value did not meet criteria after correction for multiple comparisons.

In sum, changes in QEEG ratio measures may be related to neuropsychological task performance but they appear to vary differentially between ADHD and control subjects. Given that adults with ADHD may incorporate different strategies for task completion as compared to the non-clinical controls, direction and magnitude of QEEG ratio change may be unique to each condition.

Summary of QEEG Measures

In summary, the results of the present examination suggest a significant difference in levels of cortical activity between adults with ADHD and adults without ADHD during the three neuropsychological tasks. These differences varied according to task and QEEG bandpass. No between group differences were found in theta, and both groups evidenced increased theta during the IVA as compared to the WCST. With regard to high-alpha activity, the ADHD group demonstrated significantly different levels of activation for the WCST and IVA.

In all, QEEG ratio measures were suggestive of differential levels of activation dependent upon neuropsychological task. The apparent lack of support for the previously

established measure of theta to low-beta activity is likely related to reduced theta/low-beta ratios as individuals grow older (Monastra et al., 1999) or the choice of electrode location used to measure cortical activity. However, the use of an alternative QEEG ratio measure such as the high-alpha/low-beta ratio may prove useful in differentiating adults with ADHD from non-clinical controls during active tasks.

When the relationship between QEEG ratio measures and neuropsychological performance was examined, an indistinct pattern of activity was observed. Theta/low-beta ratios did not differentiate performance between the two groups but increases in this ratio were associated with poorer attentional performance on the IVA for the ADHD group. No significant relationships between task performance and high-alpha were revealed.

Exploratory Investigations

The results of the current study speak uniquely to QEEG activity at a fronto-central location. Although statistical concerns necessitated a reduction in the number of variables examined, it is not assumed that if, in fact, significant differences in QEEG activity exist, they would only be observable in this one location. As such, activity averaged across the nine central sites was explored with incorporation of QEEG activity during the baseline conditions. Subsequent to the findings of theta and high-alpha varying the most between groups, average activity in each bandpass, across the nine central electrode locations, was calculated for each task. No discernable differences were found for either task or condition.

Also based on previous investigations, an exploratory analysis of QEEG activity at Cz, the central electrode location in the middle of the scalp, was performed. The cortical activity as measured at Cz was averaged across both baseline and active tasks. This measure, or index value, was calculated for both the theta/low-beta and high-alpha/low-beta ratios. The linear combination of QEEG ratio measures was not significantly related to diagnosis.

In general, the investigation of activity average across the central scalp locations or during the baseline conditions did not offer further insight into significant differences between these two groups. These findings suggested that there might have been a level of variance that was not previously accounted for in the study. Accordingly, the clinical sample was revisited to examine the objective measures for some characteristic that might be contributing to these observations. In fact, a difference was found for the adults with ADHD in their performance on the IVA.

Examination of ADHD Subgroups

Even with significant performance differences between groups on the PASAT and IVA, along with the significant differences in theta and high-alpha activity across task conditions, the degree of QEEG differentiation that was expected during data collection was not found. This prompted a closer evaluation of the ADHD group's objective test data. This examination revealed performance differences on the IVA. Using a criterion-reference of obtaining a quotient score at least 1.5 standard deviations from the mean, five of the ten ADHD participants demonstrated greater impairment than their colleagues as evidenced by at least two of the three Attention Quotient scores on the IVA.

Upon post hoc analysis, it was discovered that even in the present sample, 50% of the ADHD participants demonstrated significantly higher theta/low-beta ratios at Fz as compared to their ADHD comparators during the PASAT, $p = .029$, the WCST, $p = .007$, and the IVA, $p = .001$. Overall, these ADHD individuals demonstrated higher theta/low-beta ratios across all three tasks and concurrently demonstrated greater attentional impairment on the IVA. No differences were found between the ADHD subgroups for the other ratio measures. This finding speaks to potential diversity in the manifestation of this disorder in relatively high functioning adults and the need for the examination of possible behavioral subtypes within this diagnostic category.

Study Limitations and Areas of Future Investigation

Limitations

Limitations of the present study may reduce the application of this sample's data to the general population of adults with ADHD. Foremost, this investigation was restricted by the limited number of subjects who participated. The sample was comprised of volunteers with no recruitment incentive, resulting in a small number of participants. The small sample size resulted in decreased power for detecting significant differences, especially in light of relatively small clinically significant effects. This was further compounded by attempts to minimize Type I errors by using Bonferroni corrections for multiple comparisons, often yielding a very conservative p value for each of the respective analyses. Concurrently, as the minimally accepted p value was decreased, power was also decreased. For example, by moving the minimally accepted p value from

.05 toward .001, the likelihood of Type I errors was reduced but the likelihood of a Type II error was increased. A larger sample size would help resolve these issues.

The nature of the QEEG data itself also poses potential difficulty for statistical analysis. In QEEG research, it is common to transform the raw data in order to meet the assumptions of parametric statistics. However, an alternative approach for future investigations would be in the use of nonparametric analyses, embracing the non-Gaussian distribution of the data. Furthermore, by using resampling techniques such as randomization analysis, the problem of Type I errors from multiple comparisons might be better controlled (Frederick, Timmerman, & Lubar, 2001).

Also of issue is the potential introduction of muscle artifact during the active nature of the neuropsychological tasks used. Although measures were taken to monitor the introduction of electrical activity not attributable to cortical sources, artifact contamination resulted in a large amount of unusable data. Furthermore, only specific bandpasses of QEEG activity were examined due to the possibility of artifact contamination. Tasks that do not require a physical response would lend themselves to less likelihood of artifact contamination and data loss.

The present study benefited from a gender-mixed composition. However, ethnic diversity was virtually nonexistent as was the apparent restricted range of socioeconomic status levels represented. Although comparable to the ratio of multiracial and multiethnic pool from which this sample was obtained, future studies would benefit from a broader sample across gender, ethnicity, SES, education, and geographical or cultural representation.

Directions for Clinical Application

Given the observation of apparent differences in neuropsychological task performance and QEEG activity within the ADHD sample, future studies would benefit from a larger number of ADHD subjects to examine for possible subtypes within this highly heterogeneous disorder. The exploratory analyses of the two ADHD subgroups in the present project were a step in that direction. A larger sample size would also allow for examination of QEEG activity from additional anatomical regions and in more discrete QEEG bandpasses. Furthermore, a larger sample would lend itself to incorporating such techniques as factor analysis to help discern possible patterns of task-related activity for both ADHD and non-clinical controls.

In general, both the neuropsychological and QEEG findings of the present study need to be replicated with a broader sample of adults, with a wider range of educational backgrounds. Additional psychiatric groups are also needed to discern if the current observations are unique to individuals with ADHD.

Other cognitive challenge tasks need to be explored. Of the neuropsychological tasks used for concurrent QEEG collection, the PASAT and the IVA proved to lend themselves to less muscle artifact contamination in the QEEG than did the WCST. Interestingly, the same tasks also had the greatest diagnostic quality for the present sample. Other neuropsychological tasks lending themselves to concurrent QEEG collection need to be identified.

Directions for Future Research

Future areas of inquiry might include the examination of QEEG changes during task progression or during distinct stimulus presentation. QEEG techniques benefit from potentially superb temporal resolution, which could be used in future studies. Given the relatively recent focus on oscillatory phenomena in the EEG (Basar, 1999; Basar, Basar-Eroglu, Karakas, & Schürmann, 1999), and such procedures as wavelet analysis (Basar, Demiralp, Schürmann, Basar-Eroglu, & Ademoglu, 1999) and event-related desynchronization (Lopes da Silva & Pfurtscheller, 1999), there are likely a plethora of additional hypotheses and avenues available for further inquiry.

Dynamic QEEG measures such as coherence and phase also offer new lines of investigation. These measures have been used previously in the examination of the effects of methylphenidate on the QEEG in a group of boys with ADHD (Lubar, White, Swartwood, & Swartwood, 1999). The use of these techniques with adults during cognitive challenge tasks may offer novel ways of conceptualizing cognitive processes in adult ADHD. They may also offer insight into the specific relationships between cortical regions and the speed of information transfer between these areas.

Another particularly exciting avenue of future inquiry involves low-resolution electromagnetic tomography (LORETA) (Pascual-Marqui et al., 1999; Pascual-Marqui, Michel, & Lehmann, 1994). LORETA is one of a class of minimum-norm algorithms used to obtain solutions to the inverse problem in electroencephalography. These solutions provide an estimate of the three dimensional distribution of the source-current density inside the brain underlying the electrical activity measured on the scalp. As such,

LORETA provides a method of determining where in the cerebral cortex a specific QEEG field is being generated.

Finally, studies evaluating both normal and clinical populations need to be performed on clearly defined and experimentally controlled tasks of specific cognitive or neuropsychological function. For example, examining the QEEG during successful task completion during the PASAT as compared to trials characterized by errors. Through greater experimental control, the unique electroencephalographic characteristics of the cognitive task may be better ascertained. These characteristics may then be used in operationally defining such disorders as ADHD. In turn, these can provide more objective measures of criterion-referenced behaviors and improve diagnostic decisions.

Summary and Conclusions

This study attempted to illuminate potential differences in neuropsychological task performance between adults with and without Attention Deficit Hyperactivity Disorder (ADHD). The results suggest that the groups differ significantly on neuropsychological tasks that involve working memory, processing speed, and sustained attention. Accordingly, the Paced Auditory Serial Addition Test (PASAT) and the Integrated Visual and Auditory Continuous Performance Test (IVA) offer potential worth in the differential diagnosis of ADHD in adults. Further investigation is needed.

Furthermore, as a means to examine neurophysiological processes in adult ADHD, electroencephalographic recordings were taken and analyzed both within and between groups on the neuropsychological tasks. The results indicate that adult ADHD

appears to be characterized by similar neurophysiological markers as childhood ADHD but the relationships between task and cortical activity are more complex.

Finally, additional research is necessary with a larger sample so that additional variables of interest and regions of cortical activity can be examined. By increasing the degree of inquiry into this potentially disabling disorder, methods of diagnosis offering increased objectivity and reliability may be generated. By having access to such tools, clinicians may then be better able to accurately diagnosis and subsequently offer effective treatment paradigms to aid in the resolution of symptoms of adult ADHD.

REFERENCES

American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: American Psychiatric Association.

American Psychiatric Association. (1987). *Diagnostic and statistical manual of mental disorders* (3rd, revised ed.). Washington, DC: American Psychiatric Association.

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

Andreassi, J. L. (1995). *Psychophysiology: Human Behavior & Physiological Response* (3rd ed.). New Jersey: Lawrence Erlbaum Associates, Inc.

Arcia, E., & Conners, C. K. (1998). Gender differences in ADHD? *Journal of Developmental and Behavioral Pediatrics, 19*(2), 77-83.

Artiloa i Fortuny, L., & Heaton, R. K. (1996). Standard versus computerized administration of the Wisconsin Card Sorting Test. *The Clinical Neuropsychologist, 10*, 419-424.

Auronowitz, B., Liebowitz, M., Hollander, E., Fazzini, E., Dulach-Misteli, C., Frenkel, M., Mosovich, S., Garfinkel, R., Saoud, J., Delbene, D., Cohen, L., Jaeger, A., & Runin, A. L. (1994). Neuropsychiatric and neuropsychological findings in conduct disorder and attention-deficit hyperactivity disorder. *Journal of Neuropsychiatry, 6*(3), 245-249.

Baerwald, J. P., & Tryon, W. W. (1999a, August). *Modal attention asymmetry in patients with Schizophrenia and Bipolar Disorder.* Paper presented ant the annual meeting of the American Psychological Association, Boston.

Baerwald, J. P., & Tryon, W. W. (1999b, August). *Response control and attentional differentials in patients with schizophrenia and bipolar disorder.* Poster session presented at the annual meeting of the American Psychological Association, Boston.

Banich, M. T. (1997). *Neuropsychology: The neural bases of mental function.* Boston: Houghton Mifflin Company.

Barbarin, O. A., & Soler, R. E. (1993). Behavioral, emotional, and academic adjustment in a national probability sample of african american children: Effects of age, gender, and family structure. *Journal of Black Psychology, 19*(4), 423-446.

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

Barkley, R. A. (1997a). *ADHD and the nature of self-control.* New York: Guilford Press.

Barkley, R. A. (1997b). Attention-deficit/hyperactivity disorder. In E. J. Mash & L. G. Terdal (Eds.), *Assessment of childhood disorders* (pp. 71-129). New York: The Guilford Press.

Barkley, R. A. (1998a). Attention-deficit hyperactivity disorder. *Scientific American, 279*(3), 66-71.

Barkley, R. A. (1998b). Developmental course, adult outcome, and clinic-referred ADHD adults. In R. A. Barkley (Ed.), *Attention-deficit hyperactivity disorder: A handbook for diagnosis and treatment* (2nd ed., pp. 186-224). New York: The Guilford Press.

Barkley, R. A. (1998c). Etiologies. In R. A. Barkley (Ed.), *Attention-deficit hyperactivity disorder: A handbook for diagnosis and treatment* (2nd ed., pp. 164-185). New York: The Guilford Press.

Barkley, R. A. (1998d). Symptoms, diagnosis, prevalence, and gender differences. In R. A. Barkley (Ed.), *Attention-deficit hyperactivity disorder: A handbook for diagnosis and treatment* (2nd ed., pp. 56-96). New York: The Guilford Press.

Barkley, R. A., & Grodzinsky, G. M. (1994). Are tests of frontal lobe functions useful in the diagnosis of attention deficit disorders? *Clinical Neuropsychologist, 8*(2), 121-139.

Barkley, R. A., & Murphy, K. R. (1998). *Attention-deficit hyperactivity disorder: A clinical workbook* (2nd ed.). New York: The Guilford Press.

Basar, E. (Ed.). (1999). *Brain function and oscillations* (Vol. II. Integrative brain function. Neurophysiology and cognitive processes). Berlin: Springer-Verlag.

Basar, E., Basar-Eroglu, C., Karakas, S., & Schürmann, M. (1999). Are cognitive processes manifested in event-related gamma, alpha, theta and delta oscillations in the EEG? *Neuroscience Letters, 259*(3), 165-168.

Basar, E., Demiralp, T., Schürmann, M., Basar-Eroglu, C., & Ademoglu, A. (1999). Oscillatory brain dynamics, wavelet analysis, and cognition. *Brain And Language, 66*(1), 146-183.

Berg, E. A. (1948). A simple objective test for measuring flexibility in thinking. *Journal of General Psychology, 39*, 15-22.

Biederman, J., Faraone, S. V., Spencer, T., Wilens, T., Norman, D., Lapey, K. A., Mick, E., Lehman, B., & Doyle, A. (1993). Patterns of psychiatric comorbidity, cognition, and psychosocial functioning in adults with attention-deficit hyperactivity disorder. *American Journal of Psychiatry, 150*(12), 1792-1798.

Biederman, J., Mick, E., & Faraone, S. V. (2000). Age-dependent decline of symptoms of attention deficit hyperactivity disorder: impact of remission definition and symptom type. *American Journal of Psychiatry, 157*(5), 816-818.

Biederman, J., Newcorn, J., & Sprich, S. (1991). Comorbidity of attention deficit hyperactivity disorder with conduct, depressive, anxiety, and other disorders. *American Journal of Psychiatry, 148*, 564-577.

Biederman, J., Wilens, T. E., Spencer, T. J., Faraone, S., Mick, E., Ablon, J. S., & Kiely, K. (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: The Guilford Press.

Biggs, S. H. (1995). Neuropsychological and psychoeducational testing in the evaluation of the ADD adult. In K. G. Nadeau (Ed.), *A comprehensive guide to attention deficit disorder in adults* (pp. 109-131). New York: Brunner/Mazel.

Bigler, E. D., Lowry, C. M., & Porter, S. S. (1997). Neuroimaging in clinical neuropsychology. In A. M. Horton & D. Wedding & J. Webster (Eds.), *The neuropsychology handbook - Volume 1L Foundations and assessment* (pp. 171-219). New York, NY: Springer Publishing Company.

Bleier, R. (1986). Sex differences research: Science or belief. In R. Bleier (Ed.), *Feminist approaches to science* (pp. 147-164). New York: Pergamon Press Inc.

Boone, K. B. (1999). Neuropsychological assessment of executive functions: Impact of age, education, gender, intellectual level, and vascular status on executive test scores. In B. L. Miller & J. L. Cummings (Eds.), *The human frontal lobes: Functions and disorders* (pp. 247-260). New York: The Guilford Press.

Brittain, J. L., La Marche, J. A., Reeder, K. P., Roth, D. L., & Boll, T. J. (1991). Effects of age and IQ on Paced Auditory Serial Addition Task (PASAT) performance. *The Clinical Neuropsychologist, 5*(2), 163-175.

Cantor, D. S. (1999). An overview of quantitative EEG and its applications to neurofeedback. In J. R. Evans & A. Abarbanel (Eds.), *Introduction to quantitative EEG and neurofeedback* (pp. 3-27). San Diego: Academic Press.

Carlson, C. L., Tamm, L., & Gaub, M. (1997). Gender differences in children with ADHD, ODD, and co-occurring ADHD/ODD identified in a school population. *Journal of the American Academy of Child and Adolescent Psychiatry, 36*(12), 1706-1714.

Carter, R. (1998). *Mapping the mind.* London: University of California Press, Ltd.

Casini, L., & Ivry, R. B. (1999). Effects of divided attention on temporal processing in patients with lesions of the cerebellum or frontal lobe. *Neuropsychology, 13*(1), 10-21.

Castellanos, F. X. (2001). Neuroimaging studies of ADHD. In M. V. Solanto & A. F. T. Arnsten & F. X. Castellanos (Eds.), *Stimulant Drugs and ADHD: Basic and Clinical Neuroscience*. New York: Oxford University Press.

Catafau, A. M., Parellada, E., Lomena, F., Bernardo, M., Setoain, J., Catarineu, S., Pavia, J., & Herranz, R. (1998). Role of the cingulate gyrus during the Wisconsin Card Sorting Test: a single photon emission computed tomography study in normal volunteers. *Psychiatry Research: Neuroimaging Section, 83*, 67-74.

Chabot, R. J., Merkin, H., Wood, L. M., Davenport, T. L., & Serfontein, G. (1996). Sensitivity and specificity of QEEG in children with attention deficit or specific developmental learning disorders. *Clinical Electroencephalography, 27*(1), 26-34.

Cherry, S. R., & Phelps, M. E. (1996). Imaging brain function with positron emission tomography. In A. W. Toga & J. C. Mazziotta (Eds.), *Brain mapping: The methods* (pp. 191-221). San Diego: Academic Press.

Cohen, M. J., Morgan, A. M., Vaughn, M., Riccio, C. A., & Hall, J. (1999). Verbal fluency in children: Developmental issues and differential validity in distinguishing children with attention-deficit hyperactivity disorder and two subtypes of dyslexia. *Archives of Clinical Neuropsychology, 14*(5), 433-443.

Cohen, P., Cohen, J., & Brook, J. (1993). An epidemiological study of disorders in late childhood and adolescence - II. Persistence of disorders. *Journal of Child Psychology and Psychiatry, 34*(6), 869-877.

Cook, E. H., Jr., Stein, M. A., Krasowski, M. D., Cox, N. J., Olkon, D. M., Kieffer, J. E., & Leventhal, B. L. (1995). Association of attention-deficit disorder and the dopamine transporter gene. *The American Journal of Human Genetics, 56*, 993-998.

Corbett, B., & Stanczak, D. E. (1999). Neuropsychological performance of adults evidencing attention-deficit hyperactivity disorder. *Archives of Clinical Neuropsychology, 14*(4), 373-387.

de Mesquita, P. B., & Gilliam, W. S. (1994). Differential diagnosis of childhood depression: Using comorbidity and symptom overlap to generate hypotheses. *Child Psychiatry and Human Development, 24*(3), 157-172.

Diehr, M. C., Heaton, R. K., Miller, W., & Grant, I. (1998). The Paced Auditory Serial Addition Task (PASAT): Norms for age, education, and ethnicity. *Assessment, 5*(4), 375-387.

Duffy, F. H. (1994). The role of quantified electroencephalography in psychological research. In G. Dawson & K. W. Fischer (Eds.), *Human behavior and the developing brain* (pp. 93-133). New York: The Guilford Press.

Duffy, F. H., Iyer, V. G., & Surwillo, W. W. (1989). *Clinical electroencephalography and topographic brain mapping*. New York: Springer-Verlag.

Edwards-Lee, T. A., & Saul, R. E. (1999). Neuropsychiatry of the right frontal lobe. In B. L. Miller & J. L. Cummings (Eds.), *The human frontal lobes: Functions and disorders* (pp. 304-320). New York: The Guilford Press.

Elia, J., Gulotta, C., Rose, S. R., Marin, G., & Rapoport, J. L. (1994). Thyroid function and attention-deficit hyperactivity disorder. *Journal of the American Academy of Child and Adolescent Psychiatry, 33*(2), 1169-1172.

Epstein, J. N., Conners, C. K., Sitarenios, G., & Erhardt, D. (1998). Continuous performance test results of adults with Attention Deficit Hyperactivity Disorder. *The Clinical Neuropsychologist, 12*(2), 155-168.

Ernst, M., Liebenauer, L. L., King, C., Fitzgerald, G. A., Cohen, R. M., & Zametkin, A. J. (1994). Reduced brain metabolism in hyperactive girls. *Journal of the American Academy of Child and Adolescent Psychiatry, 33*(6), 858-868.

Eslinger, P. J., Biddle, K. R., & Grattan, L. M. (1997). Cognitive and social development in children with prefrontal cortex lesions. In N. A. Krasegnor & G. R. Lyon & P. S. Goldman-Rakic (Eds.), *Development of the prefrontal cortex: Evolution, neurobiology, and behavior* (pp. 295-335). Baltimore: Paul H. Brookes Publishing Company.

Fallgatter, A. J., & Strik, W. K. (1997). Right frontal activation during the continuous performance test assessed with near-infrared spectroscopy in healthy subjects. *Neuroscience Letters, 223*(2), 89-92.

Feifel, D. (1996). Attention-deficit hyperactivity disorder in adults. *Postgraduate Medicine, 100*(3), 207-211, 215-208.

Fergusson, D. M., Horwood, L. J., & Lynskey, M. T. (1993). Prevalence and comorbidity of DSM-III-R diagnoses in a birth cohort of 15 year olds. *Journal of the American Academy of Child and Adolescent Psychiatry, 32*(6), 1127-1134.

Fisher, B. C. (1997). *Attention deficit disorder misdiagnosis: Approaching ADD from a brain-behavior/neuropsychological perspective for assessment and treatment.* Boca Raton, FL: St. Lucie Press.

Fitzhugh-Bell, K. B. (1997). Historical antecedents of clinical neuropsychology. In A. M. Horton & D. Wedding & J. Webster (Eds.), *The neuropsychology handbook - Volume 1: Foundations and assessment* (pp. 67-90). New York, NY: Springer Publishing Company.

Forbes, G. B. (1998). Clinical utility of the Test of Variables of Attention (TOVA) in the diagnosis of Attention-Deficit/Hyperactivity Disorder. *Journal of Clinical Psychology, 54*(4), 461-476.

Frederick, J. A., Timmerman, D. L., & Lubar, J. F. (2001). *EEG coherence effects of audio-visual stimulation (AVS) at dominant alpha frequency and twice dominant alpha frequency, Manuscript submitted for publication.*

Fuster, J. M. (1997). *The prefrontal cortex: Anatomy, physiology, and neuropsychology of the frontal lobe* (3rd ed.). Philadelphia: Lippincott-Raven.

Fuster, J. M. (1999). Cognitive functions of the frontal lobes. In B. L. Miller & J. L. Cummings (Eds.), *The human frontal lobes: Functions and disorders* (pp. 187-195). New York: The Guilford Press.

Gansler, D. A., Fucetola, R., Krengel, M., Stetson, S., Zimering, R., & Makary, C. (1998). Are there cognitive subtypes in adult attention deficit/hyperactivity disorder? *Journal of Nervous and Mental Disease, 186*(12), 776-781.

Gaub, M., & Carlson, C. L. (1997). Gender differences in ADHD: A meta-analysis and critical review. *Journal of the American Academy of Child and Adolescent Psychiatry, 36*(8), 1036-1045.

Gevins, A., & Smith, M. E. (2000). Neurophysiological measures of working memory and individual differences in cognitive ability and cognitive style. *Cerebral Cortex, 10*(9), 829-839.

Gilman, S., & Newman, S. W. (1996). *Manter and Gatz's essentials of clinical neuroanatomy and neurophysiology* (9th ed.). Philadelphia: F. A. Davis Company.

Gingerich, K. J., Turnock, P., Litfin, J. K., & Rosen, L. A. (1998). Diversity and attention deficit hyperactivity disorder. *Journal of Clinical Psychology, 54*(4), 415-426.

Grant, D. A., & Berg, E. A. (1948). A behavioral analysis of degree of reinforcement and ease of shifting to new responses in a Weigl-type card sorting problem. *Journal of Experimental Psychology, 34*, 404-411.

Greenberg, L. M., & Kindschi, C. L. (1996). *T.O.V.A. Test of variables of attention: Clinical guide.* St. Paul, Minnesota: Universal Attention Disorders.

Greve, K. W., Williams, M. C., Haas, W. G., Littell, R. R., & Reinoso, C. (1996). The role of attention in Wisconsin Card Sorting Test performance. *Archives of Clinical Neuropsychology, 11*(3), 215-222.

Grodzinsky, G. M., & Barkley, R. A. (1999). Predictive power of frontal lobe tests in the diagnosis of Attention Deficit Hyperactivity Disorder. *The Clinical Neuropsychologist, 13*(1), 12-21.

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

Gronwall, D. M. A. (1977). Paced Auditory Serial Addition Task: A measure of recovery from concussion. *Perceptual and Motor Skills, 44*, 367-373.

Gronwall, D. M. A., & Sampson, H. (1974). *The psychological effects of concussion.* Auckland, N. Z.: Auckland University Press.

Gusnard, D. A., Akbudak, E., Shulman, G. L., & Raichle, M. E. (2001). Medial prefrontal cortex and self-referential mental activity: Relation to a default mode of brain function. *Proceedings Of The National Academy Of Sciences Of The United States Of America, 98*(7), 4259-4264.

Halperin, J. M., Newcorn, J. H., Matier, K., Sharma, V., McKay, K. E., & Schwartz, S. (1993). Discriminant validity of attention-deficit hyperactivity disorder. *Journal of the American Academy of Child and Adolescent Psychiatry, 32*(5), 1038-1043.

Halstead, W. C. (1947). *Brain and intelligence: A quantitative study of the frontal lobes.* Chicago: University of Chicago Press.

Heaton, R. K., Chelune, G. J., Talley, J. L., Kay, G. G., & Curtiss, G. (1993). *Wisconsin Card Sorting Test manual: Revised and expanded.* Odessa, FL: Psychological Assessment Resources, Inc.

Heaton, R. K., Ryan, L., Grant, I., & Matthews, C. G. (1996). Demographic influences on neuropsychological test performance. In I. Grant & K. M. Adams (Eds.), *Neuropsychological assessment of neuropsychiatric disorders* (pp. 141-163). New York: Oxford University Press.

Heaton, R. K., Thompson, J. A., & Gomez, E. (1999). *Wisconsin Card Sorting Test: Computer Version 3 for Windows Research Edition manual.* Odessa, FL: Psychological Assessment Resources, Inc.

Hellman, S. G., Green, M. F., Kern, R. S., & Christenson, C. D. (1992). Comparison of card and computer versions of the Wisconsin Card Sorting Test for psychotic patients. *International Journal of Methods in Psychiatric Research, 2*, 151-155.

Horton, A. M., Jr. (1996). Neuropsychological findings in adult attention deficit disorder: A pilot study. *Applied Neuropsychology, 3*(4), 181-185.

Hughes, J. R., & John, E. R. (1999). Conventional and quantitative electroencephalography in psychiatry. *The Journal of Neuropsychiatry and Clinical Neurosciences, 11*, 190-208.

Hutchens, T. A. (1989). *Automaticity, naming, and reading in learning disabled and normally achieving college students.* Unpublished doctoral dissertation, University of Georgia., Athens.

Hutchens, T. A., & White, J. N., Jr. (1998). Metabolic disorders in childhood and adolescence. In L. Phelps (Ed.), *Health-related disorders in children and adolescents: A guidebook for understanding and educating* (pp. 426-433). Washington, D.C.: American Psychological Association.

Jasper, H. H. (1958). The ten twenty electrode system of the international federation. *Electroencephalography and clinical neurophysiology, 10*, 371-375.

Jensen, P. S., Koretz, D., Locke, B. Z., Schneider, S., Radke-Yarrow, M., Richters, J. E., & Rumsey, J. M. (1993). Child and adolescent psychopathology research: Problems and prospects for the 1990s. *Journal of Abnormal Child Psychology, 21*(5), 551-580.

Katz, L. J., Wood, D. S., Goldstein, G., Auchenbach, R. C., & Geckle, M. (1998). The utility of neuropsychological tests in evaluation of attention-deficit/hyperactivity disorder (ADHD) versus depression in adults. *Assessment, 5*(1), 45-51.

Kolb, B., & Whishaw, I. Q. (1996). *Fundamentals of human neuropsychology* (4th ed.). New York: W. H. Freeman and Company.

Kovner, R., Budman, C., Frank, Y., Sison, C., Lesser, M., & Halperin, J. (1998). Neuropsychological testing in adult attention deficit hyperactivity disorder: A pilot study. *International Journal of Neuroscience, 96*, 225-235.

Krauss, G. L., & Webber, W. R. S. (1999). Digital EEG. In E. Niedermeyer & F. Lopes da Silva (Eds.), *Electroencephalography: Basic principles, clinical applications, and related fields* (4th ed., pp. 781-796). Baltimore: Williams & Wilkins.

Laursen, P. (1997). The impact of aging on cognitive functions. An 11 year follow-up study of four age cohorts. *Acta Neurologica Scandinavica. Supplementum, 172*, 7-86.

Lazzaro, I., Gordon, E., Whitmont, S., Plahn, M., Li, W., Clarke, S., Dosen, A., & Meares, R. (1998). Quantified EEG activity in adolescent attention deficit hyperactivity disorder. *Clinical Electroencephalography, 29*(1), 37-42.

Levin, H., & Krauss, M. F. (1994). The frontal lobes and traumatic brain injury. *Journal of Neuropsychiatry, 6*, 443-454.

Levin, H. S., Mattis, S., Ruff, R., Eisenberg, H. M., Marshall, L. F., Tabaddor, K., High, W. M., Jr., & Frankowski, R. F. (1987). Neurobehavioral outcome following minor head injury: a three-center study. *Journal of Neurosurgery, 66*(2), 234-243.

Lexicor Medical Technology. (1992). NeuroLex™ Software for EEG - Data Acquisition & Analysis [Computer software]. Denver, CO: Lexicor Medical Technology.

Lexicor Medical Technology. (1996). Exporter Quantitative EEG Export Program [Computer software] (Version 1.2). Denver, CO: Lexicor Medical Technology.

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

Loeber, R., & Keenan, K. (1994). Interaction between conduct disorder and its comorbid conditions: Effects of age and gender. *Clinical Psychology Review, 14*(6), 497-523.

Lopes da Silva, F. (1999). Computer-assisted EEG diagnosis: Pattern recognition and brain mapping. In E. Niedermeyer & d. S. Lopes (Eds.), *Electroencephalography: Basic principles, clinical applications, and related fields* (4th ed., pp. 1164-1189). Baltimore: Williams & Wilkins.

Lopes da Silva, F. H., & Pfurtscheller, G. (1999). Basic concepts on EEG synchronization and desynchronization. In G. Pfurtscheller & F. H. Lopes da Silva (Eds.), *Handbook of electroencephalography and clinical neurophysiology* (Vol. 6: Event-related desynchronization, pp. 3-11). Amsterdam: Elsevier.

Lou, H. C., Henriksen, L., & Bruhn, P. (1984). Focal cerebral hypoperfusion in children with dysphasia and/or attention deficit disorder. *Archives of Neurology, 41*(8), 825-829.

Lou, H. C., Henriksen, L., Bruhn, P., Borner, H., & Nielsen, J. B. (1989). Striatal dysfunction in attention deficit and hyperkinetic disorder. *Archives of Neurology, 46*(1), 48-52.

Lovejoy, D. W., Ball, J. D., Keats, M., Stutts, M. L., Spain, E. H., Janda, L., & Janusz, J. (1999). Neuropsychological performance of adults with attention deficit hyperactivity disorder (ADHD): Diagnostic classification estimates for measures of frontal lobe/executive functioning. *Journal of the International Neuropsychological Society, 5*, 222-233.

Lowe, P. A., Reynolds, C. R., Riccio, C. A., & Moore, J. J. (1998, November). *Addressing the psychometric properties of the Continuous Performance Test (CPT): Will the real CPT please stand up?* Paper presented at the annual meeting of the National Academy of Neuropsychology, Washington, DC.

Lubar, J. F. (1991). Discourse on the development of EEG diagnostics and biofeedback for attention-deficit/hyperactivity disorders. *Biofeedback & Self Regulation, 16*(3), 201-225.

Lubar, J. F. (1995). Neurofeedback for the management of attention-deficit/hyperactivity disorders. In M. S. Schwartz (Ed.), *Biofeedback: A practitioner's guide* (pp. 493-522): Guilford Press, New York, NY, US.

Lubar, J. F., Swartwood, M. O., Swartwood, J. N., & O'Donnell, P. H. (1995). Evaluation of the effectiveness of EEG neurofeedback training for ADHD in a clinical setting as measured by changes in T.O.V.A. scores, behavioral ratings, and WISC--R performance. *Biofeedback & Self Regulation, 20*(1), 83-99.

Lubar, J. F., White, J. N. J., Swartwood, M. O., & Swartwood, J. N. (1999). Methylphenidate effects on global and complex measures of EEG. *Pediatric Neurology, 21*(3), 633-637.

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

Luria, A. R. (1973b). *The working brain: An introduction to neuropsychology.* New York: Basic Books Inc.

Malone, M. A., Kershner, J. R., & Swanson, J. M. (1994). Hemispheric processing and methylphenidate effects in attention-deficit hyperactivity disorder. *Journal of Child Neurology, 9*(2), 181-189.

Mann, C. A., Lubar, J. F., Zimmerman, A. W., Miller, C. A., & Muenchen, R. A. (1992). Quantitative analysis of EEG in boys with attention-deficit-hyperactivity disorder: controlled study with clinical implications. *Pediatric Neurology, 8*(1), 30-36.

Miller, D. K., & Blum, K. (1996). *Overload: Attention deficit disorder and the addictive brain.* Kansas City: Andrews and McMeel.

Milner, B. (1963). Effects of different brain lesions on card sorting. *Archives of Neurology, 9*(July), 100-110.

Mirsky, A. F. (1996). Disorders of attention: A neuropsychological perspective. In G. R. Lyon & N. A. Krasegnor (Eds.), *Attention, memory, and executive function.* Baltimore: Paul H. Brookes Publishing Co.

Monastra, V. J., Lubar, J. F., & Linden, M. (2001). The development of a quantitative electroencephalographic scanning process for Attention Deficit-Hyperactivity Disorder: Reliability and validity studies. *Neuropsychology, 15*(1), 136-144.

Monastra, V. J., Lubar, J. F., Linden, M., VanDeusen, P., Green, G., Wing, W., Phillips, A., & Fenger, T. N. (1999). Assessing attention deficit hyperactivity disorder via quantitative electroencephalography: an initial validation study. *Neuropsychology, 13*(3), 424-433.

Murphy, P., & Schachar, R. (2000). Use of self-ratings in the assessment of symptoms of attention deficit hyperactivity disorder in adults. *American Journal of Psychiatry, 157*(7), 1156-1159.

Newcorn, J. H., Halperin, J. M., Schwartz, S., Pascualvaca, D., Wolf, L., Schmeidler, J., & Sharma, V. (1994). Parent and teacher ratings of attention-deficit hyperactivity disorder: Implications for case identification. *Developmental and Behavioral Pediatrics, 15*(2), 86-91.

Niedermeyer, E. (1999). Historical aspects. In E. Niedermeyer & F. Lopes da Silva (Eds.), *Electroencephalography: Basic principles, clinical applications, and related fields* (4th ed., pp. 1-14). Baltimore: Williams & Wilkins.

Nunez, P. L. (1995). *Neocortical dynamics and human EEG rhythms*. New York: Oxford University Press.

Nunez, P. L., & Silberstein, R. B. (2000). On the relationship of synaptic activity to macroscopic measurements: Does co-registration of EEG with fMRI make sense? *Brain Topography, 13*(2), 79-96.

Obrig, H., & Villringer, A. (1997). Near-infrared spectroscopy in functional activation studies: Can NIRS demonstrate cortical activation? In A. Villringer & U. Dirnagl (Eds.), *Optical imaging of brain function and metabolism 2: Physiological basis and comparison to other functional neuroimaging methods* (pp. 113-127). New York: Plenum Press.

Obrig, H., Wolf, T., Doge, C., Hulsing, J. J., Dirnagl, U., & Villringer, A. (1996). Cerebral oxygenation changes during motor and somatosensory stimulation in humans, as measured by near-infrared spectroscopy. In Ince et al. (Eds.), *Oxygen transport to tissue XVII* (pp. 219-224). New York: Plenum Press.

Pascual-Marqui, R. D., Lehmann, D., Koenig, T., Kochi, K., Merlo, M. C., Hell, D., & Koukkou, M. (1999). Low resolution brain electromagnetic tomography (LORETA) functional imaging in acute, neuroleptic-naive, first-episode, productive schizophrenia. *Psychiatry Research, 90*(3), 169-179.

Pascual-Marqui, R. D., Michel, C. M., & Lehmann, D. (1994). Low resolution electromagnetic tomography: a new method for localizing electrical activity in the brain. *International Journal of Psychophysiology, 18*(1), 49-65.

Pascualvaca, D. M., Anthony, B. J., Arnold, L. E., Rebok, G. W., Ahearn, M. B., Kellam, S. G., & Mirsky, A. F. (1997). Attention performance in an epidemiological sample of urban children: The role of gender and verbal intelligence. *Child Neuropsychology, 3*(1), 13-27.

Posner, M. I., & Petersen, S. E. (1990). The attention system of the brain. *Annual Review of Neuroscience, 13*, 25-42.

Posner, M. I., & Raichle, M. E. (1997). *Images of mind*. New York: Scientific American Library.

Power, T. J., & DuPaul, G. J. (1996). Attention-deficit hyperactivity disorder: The reemergence of subtypes. *School Psychology Review, 25*(3), 284-296.

Rasey, H. W. (1998). *Quantitative EEG differences during the administration of a continuous performance task among adults with and without attention deficit hyperactivity disorder.* Unpublished doctoral dissertation, The University of Tennessee, Knoxville.

Reid, R., & Katsiyannis, A. (1995). Attention-deficit/hyperactivity disorder and Section 504. *Remedial and Special Education, 16*(1), 44-52.

Reynolds, C. R., Lowe, P. A., Moore, J., & Riccio, C. A. (1998, November). *Sensitivity and specificity of CPT in the diagnosis of ADHD: Much of one and none of the other.* Paper presented at the annual meeting of the National Academy of Neuropsychology, Washington, DC.

Riccio, C. A., Hynd, G. W., Cohen, M. J., Gonzalez, J. J., & et al. (1993). Neurological basis of attention deficit hyperactivity disorder. *Exceptional Children, 60*(2), 118-124.

Riccio, C. A., Moore, J. J., Lowe, P., & Reynolds, C. R. (1998, November). *The continuous performance test: A window on the neural substrates for attention?* Paper presented at the annual meeting of the National Academy of Neuropsychology, Washington, DC.

Riordan, H. J., Flashman, L. A., Saykin, A. J., Frutiger, S. A., Carroll, K. E., & Huey, L. (1999). Neuropsychological correlates of methylphenidate treatment in adult ADHD with and without depression. *Archives of Clinical Neuropsychology, 14*(2), 217-233.

Robertson, I. H., Ward, T., Ridgeway, V., & Nimmo-Smith, I. (1996). The structure of normal human attention: The Test of Everyday Attention. *Journal of the International Neuropsychological Society, 2*(6), 525-534.

Rosvold, H. E., Mirsky, A. F., Sarason, I., Bransome, E. D., Jr., & Beck, L. H. (1956). A continuous performance test of brain damage. *Journal of Consulting Psychology, 20*(5), 343-350.

Sabatino, D. A., & Vance, H. B. (1994). Is the diagnosis of attention deficit/hyperactivity disorders meaningful? *Psychology in the Schools, 31*(188-196).

Samango-Sprouse, C. (1999). Frontal lobe development in children. In B. L. Miller & J. L. Cummings (Eds.), *The human frontal lobes: Functions and disorders* (pp. 584-603). New York: The Guilford Press.

Sandford, J. A. (1995). *Intermediate Visual and Auditory continuous performance test interpretation manual* (Version 1.2 ed.). Richmond, VA: Sandford, Joseph A.

Scheibel, R. S., & Levin, H. S. (1997). Frontal lobe dysfunction following closed head injury in children: Findings form neuropsychology and brain imaging. In N. A. Krasegnor & G. R. Lyon & P. S. Goldman-Rakic (Eds.), *Development of the prefrontal cortex: Evolution, neurobiology, and behavior* (pp. 241-263). Baltimore: Paul H. Brookes Publishing Co.

Schweitzer, J. B., Faber, T. L., Grafton, S. T., Tune, L. E., Hoffman, J. M., & Kilts, C. D. (2000). Alterations in the functional anatomy of working memory in adult attention deficit hyperactivity disorder. *American Journal of Psychiatry, 157*(2), 278-280.

Seidman, L. J., Biederman, J., Faraone, S. V., Weber, W., Mennin, D., & Jones, J. (1997). A pilot study of neuropsychological function in girls with ADHD. *Journal of the American Academy of Child and Adolescent Psychiatry, 36*(3), 366-373.

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*(1), 150-160.

Seidman, L. J., Biederman, J., Weber, W., Hatch, M., & Faraone, S. V. (1998). Neuropsychological function in adults with attention-deficit hyperactivity disorder. *Biological Psychiatry, 44*(4), 260-268.

Seidman, L. J., Breiter, H. C., Goodman, J. M., Goldstein, J. M., Woodruff, P. W., O'Craven, K., Savoy, R., Tsuang, M. T., & Rosen, B. R. (1998). A functional magnetic resonance imaging study of auditory vigilance with low and high information processing demands. *Neuropsychology, 12*(4), 505-518.

Semrud-Clikeman, M., Hynd, G. W., Lorys, A. R., & Lahey, B. B. (1993). Differential diagnosis of children with ADHD and ADHD/with co-occurring conduct disorder. *School Psychology International, 14*(4), 361-370.

Shallice, T. (1988). *From neuropsychology to mental structure.* Cambridge: Cambridge University Press.

Shaywitz, S. E., Fletcher, J. M., & Shaywitz, B. A. (1994). Issues in the definition and classification of attention deficit disorder. *Topics in Language Disorders, 14*(4), 1-25.

Shealey, A. H. (1994). Attention-deficit hyperactivity disorder - Etiology, diagnosis, and management. *Journal of Child and Adolescent Psychiatric Nursing, 7*(2), 24-36.

Spreen, O., & Strauss, E. (1998). *A compendium of neuropsychological tests: Administration, norms, and commentary* (2nd ed.). New York: Oxford University Press.

Stuss, D. T., & Benson, D. F. (1986). *The frontal lobes.* New York: Raven Press.

Swanson, H. L. (1999). What develops in working memory? A life span perspective. *Developmental Psychology, 35*(4), 986-1000.

Swanson, J., Castellanos, F. X., Murias., M., LaHoste, G., & Kennedy, J. (1998). Cognitive neuroscience of attention deficit hyperactivity disorder and hyperkinetic disorder. *Current Opinion in Neurobiology, 8*, 263-271.

Thatcher, R. W. (1998). Normative EEG databases and EEG biofeedback. *Journal of Neurotherapy, 2*(4), 8-39.

Thatcher, R. W. (1999). EEG database-guided neurotherapy. In J. R. Evans & A. Abarbanel (Eds.), *Introduction to quantitative EEG and neurofeedback* (pp. 29-64). San Diego: Academic Press.

van Zomeren, A. H., & Brouwer, W. H. (1994). *Clinical neuropsychology of attention*. New York: Oxford University Press.

Villringer, A., & Dimagl, U. (1995). Coupling of brain activity and cerebral blood flow: Basis of functional neuroimaging. *Cerebrovascular and Brain Metabolism Reviews, 7*(240-276).

Villringer, A., Planck, J., Hock, C., Schleinkofer, L., & Dimagl, U. (1993). Near infrared spectroscopy (NIRS): a new tool to study hemodynamic changes during activation of brain function in human adults. *Neuroscience Letters, 154*(1-2), 101-104.

Walker, A. J., Shores, E. A., Trollor, J. N., Lee, T., & Sachdev, P. S. (2000). Neuropsychological functioning of adults with attention deficit hyperactivity disorder. *Journal of Clinical and Experimental Neuropsychology, 22*(1), 115-124.

Waxman, S. G. (1996). *Correlative neuroanatomy* (23rd ed.). Stamford, CT: Appleton & Lange.

Weinfurt, K. P. (1995). Multivariate analysis of variance. In L. G. Grimm & P. R. Yarnold (Eds.), *Reading and understanding multivariate statistics* (pp. 245-276). Washington, DC: American Psychological Association.

Weiss, R. E., Stein, M. A., Trommer, B., & Refetoff, S. (1993). Attention-deficit hyperactivity disorder and thyroid function. *The Journal of Pediatrics, 123*(4), 539-545.

Wender, P. H. (1995). *Attention-deficit hyperactivity disorder in adults.* New York: Oxford University Press.

Wilens, T. E., Spencer, T. J., & Biederman, J. (1998). Pharmacotherapy of adult ADHD. In R. A. Barkley (Ed.), *Attention-deficit hyperactivity disorder: A handbook for diagnosis and treatment* (2nd ed., pp. 592-606). New York: The Guilford Press.

Wilkinson, L. (1975). Response variable hypothesis in the multivariate analysis of variance. *Psychological Bulletin, 82*, 408-412.

Zametkin, A. J., Liebenauer, L. L., Fitzgerald, G. A., King, A. C., Minkunas, D. V., Herscovitch, P., Yamada, E., & Cohen, R. M. (1993). Brain metabolism in teenagers with attention-deficit hyperactivity disorder. *Archives of General Psychiatry, 50*(5), 333-340.

VITA

Noland White was born in Atlanta, Georgia, on September 25, 1967. He attended schools in Macon, Georgia and graduated high school from Windsor Academy in June of 1985. After high school, he initially gained an Associate of Arts degree in Psychology from Macon Junior College in 1988. He then entered Georgia College, presently Georgia College and State University, in September of 1988 and obtained his Bachelor of Science degree in 1990. After working with Project Adventure, located in Covington, GA for a year, he returned to Georgia College to pursue a graduate degree and obtained his Master of Science in Psychology in 1992. Noland then worked for three years in the Developmental Disabilities Division of Central State Hospital in Milledgeville, GA before entering the Counseling Psychology program at The University of Tennessee in 1995. After completing his predoctoral internship at the Malcom Randall VAMC in Gainesville, FL, he will receive his doctoral degree in August, 2001.