MEASURING THE VALIDITY OF TWO CONTINUOUS PERFORMANCE TESTS: DIFFERENT PARAMETERS AND SCORING INDICES

A Dissertation

by

SUSAN RAE HOMACK

Submitted to the Office of Graduate Studies of
Texas A&M University
in partial fulfillment of the requirements for the degree of

DOCTOR OF PHILOSOPHY

August 2005

Major Subject: School Psychology

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Approved by:

Chair of Committee, Cynthia Riccio
Committee Members, Michael Ash
William Rae
Christine Stanley
Head of Department, Michael Benz

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ABSTRACT

Measuring the Validity of Two Continuous Performance Tests:

Different Parameters and Scoring Indices.

(August 2005)

Susan Rae Homack, B.A., Southwest Missouri State University;

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Chair of Advisory Committee: Dr. Cynthia Riccio

Today, there are numerous versions of the continuous performance test (CPT) used in clinical and research settings. Although CPTs may constitute a similar group of tasks with a common paradigm, they are very different in the parameters they measure (Conners, 1995). To learn more about the effects of different CPT versions as well as the numerous scoring indices, two very different CPTs, the Conners' Continuous Performance Test-Second Edition (CCPT-II) and the Gordon Diagnostic System (GDS), were compared with a population of children and adolescents exhibiting ADHD and normal controls. Major findings were as follows: (a) the CCPT-II and GDS measures were not able to separate children with ADHD from normal controls; (b) individual variables from neither the CCPT-II nor the GDS were able to adequately differentiate children with ADHD and normal controls; and (c) score profiles obtained from the overall group of children and adolescents did not successfully separate the ADHD group from normal controls using the CCPT-II and GDS.

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CHAPTER I

INTRODUCTION

Attention-Deficit/ Hyperactivity Disorder

Attention-Deficit/ Hyperactivity Disorder (ADHD) is a heterogeneous disorder of unknown etiology. Its impact on society is enormous in terms of financial cost, stress on families, interference with academic activities, as well as negative effects on self-esteem (Biederman, 1998). By definition, children with ADHD display difficulties with attention relative to normal children of the same age and sex. However, attention is a multidimensional construct that can refer to alertness, arousal, selective or focused attention, distractibility, and sustained attention, among others (Barkley, 1998; Mirsky, 1996). According to Douglas (1983), children with ADHD most likely have their greatest difficulties with sustaining attention to tasks, persistence to effort, and vigilance. These difficulties are sometimes apparent in free-play settings (Barkley & Ullman, 1975; Routh & Schroeder, 1976), but they are most evident in situations requiring sustained attention to dull, boring, repetitive tasks (Luk, 1985; Milich, Loney, & Landau, 1982; Ullman, Barkley, & Brown, 1978). In the area of impulsivity, children with ADHD have problems thinking before they act. According to Barkley (1981, 1997), it is a struggle for children with ADHD to follow rule-governed behavior due to their problems with separating experience from response, thought from emotion, and action from reaction.

This dissertation follows the style of *Archives of Clinical Neuropsychology.*

Due to their lack of inhibition, children with ADHD tend to be excessively restless and overactive. Also due to problems with inhibiting behavior, children with ADHD have difficulties working toward a long-term goal, and thus, often require brief, repeated payoffs rather than a long-term reward (Goldstein, 1999). Finally, due to their impulsivity, children with ADHD often appear to be on a roller-coaster ride of emotions throughout childhood. Research indicates that children with ADHD appear more vulnerable to personality problems, especially those related to antisocial difficulty, and are more prone to depression (Goldstein, 1999).

The presentation and symptoms of ADHD may change as a child develops. In the preschool age range, a child with ADHD generally may have temper tantrums and display argumentative and aggressive behavior. Fearless behavior also leads to frequent accidental injury and noisy behavior. Campbell (1990) found that noncompliance and sleep disturbance is often a major problem with preschool age children. In the elementary school-age child, cognitively effortful work is challenging. In addition, school-age children often exhibit impulsivity, hyperactivity, and inattention that leads to difficulty in peer relationships. In adolescence, the core symptoms may manifest as an internal sense of restlessness. Inattention and cognitive problems may lead to poorly organized approaches to school. Problems with work completion at school are common in the adolescent age range. Risky types of behavior such as more frequent auto and bicycle accidents may occur in adolescents with ADHD (Weiss & Hechtman, 1994).

Assessment Measures for Children with ADHD

Problems arising from symptoms of ADHD constitute the largest single source of referrals to mental health centers (Barkley, 1981); more recently, it has been suggested that children with ADHD may account for as many as 40% of referrals to child guidance clinics (Barkley, 1998). While a large number of children are referred to clinics, the development of a norm-referenced, psychometric assessment battery specifically designed for ADHD has been an elusive goal for clinicians (Goldstein, 1999). Computerized assessment of behaviors associated with ADHD represents an effort to incorporate reliable and objective assessment into evaluations for ADHD. These techniques were born of concern about the degree to which diagnostic decisions were founded upon subjective measures and clinical judgment. Currently, the diagnosis of ADHD is made primarily from anecdotal information from parents' and teachers' report. One problem with using scores from parents' and teachers' reports is the lack of congruence often found between these measures. As noted by Sattler (1990), a lack of reliability between these measures is primarily related to varying expectations and tolerance on the part of parents and teachers.

Computer-based measures allow the clinician to incorporate data into the assessment that is derived from a child's actual behavior. Unlike other clinical techniques, computer-based measures generate objective data about a child's ability to perform in situations tailored to assess the characteristic weaknesses of a child with ADHD (Gordon, 1986). One venue for a computerized measure includes the development of continuous performance tests (CPTs). The CPT is one group of paradigms used for the evaluation of attention and the response inhibition component of executive control. The CPTs are frequently used to obtain quantitative information regarding an individual's ability to sustain attention over time; the duration of the task varies, but is intended to be sufficient to measure sustained attention. The CPT involves selective attention or vigilance for an infrequently occurring target or relevant stimulus. The CPT paradigm is generally characterized by rapid presentation of continuously changing stimuli with a designated target stimulus or target pattern (Riccio, Reynolds, & Lowe, 2001).

Since the advent of the CPT paradigm, results regarding the validity of these tasks have been equivocal. As a group, children with ADHD exhibit more difficulties on CPT measures than relative controls (Barkley, DuPaul, & McMurray, 1990; Fischer, Barkley, Edelbrock, & Smallish, 1990; Halperin, et al., 1988; Klee & Garfinkel, 1983; Sykes, Douglas, & Morganstern, 1973). Although scores on the CPT appear to discriminate between children with ADHD and normal controls at a group level, the utility of these measures at assessing individual children is limited by several factors. While significant correlations between CPT scores and teacher ratings of inattention, impulsivity, and hyperactivity have been obtained (Halperin et al., 1988; Klee & Garfinkel, 1983; Shapiro & Garfinkel, 1986), several studies have failed to obtain significant correlations between criterion measures (e.g., teacher ratings, other laboratory measures of vigilance and impulsivity) and scores on various CPTs (DuPaul, Anastopoulos, Shelton, Guevremont, & Metevia, 1992; Halperin, Sharma, Greenblatt, & Schwartz, 1991; Lovejoy & Rasmussen, 1990). Second, when the effects of age, sex, and

IQ have been partialed out, scores on CPT measures have failed to discriminate among children with ADHD, conduct disorder, anxiety disorder, and normal controls (Werry, Elkind, & Reeves, 1987). Finally, even when the effects of age, sex, and IQ have not been partialed out, scores on CPT measures have not reliably discriminated among different groups of clinic-referred children (Campbell, 1974: Halperin et al., 1990; Riccio et al., 2001; Shapiro & Garfinkel, 1986).

The ability of laboratory tests to identify, define and determine the severity of symptoms of ADHD has been increasingly questioned (Barkley, 1991; Barkley & Grodzinsky, 1994). Although CPTs may demonstrate high positive predictive power (e.g., if a child fails such a task, it strongly confirms the presence of symptoms related to ADHD), Goldstein (1999) reported that they possess poor negative predictive power (e.g., if a child passes a task, conclusions cannot be drawn one way or the other concerning a diagnosis). Furthermore, Baren and Swanson (1996) noted that if a CPT test was used to screen a typical elementary school with about 500 students to identify those with ADHD (about 5% or 25 students), almost four times as many non-ADHD students (66.5) than ADHD students (18.25) would be expected to have abnormal scores. Matier-Sharma, Perachio, and Newcorn (1995) noted in an assessment of a clinical sample that CPT measures were only slightly better than chance at distinguishing ADHD from non-ADHD disorders. Nonetheless, many clinicians rely on such instruments to provide additional data as part of the diagnostic process, rather than specifically to confirm or disconfirm the diagnosis of ADHD (Conners, 1994).

Differences in administration, stimulus presentation, duration, and scoring of the various CPTs complicate score interpretation. Furthermore, few studies have investigated how different score types may impact the interpretation of CPT performance (Riccio et al., 2001). The Conners' Continuous Performance Test (CCPT-II; Conners, 1994) differs from traditional CPT in several ways. For example, the difference between the CCPT-II and the Gordon Diagnostic System (GDS; Gordon, 1983) raises several issues in interpreting scores. When using the CCPT-II, the individual is instructed to press the space bar after every letter presented on the computer screen except for the "X". In contrast, when using the GDS, the individual is instructed to press the space bar only when the number "9" follows the number "1". When interpreting scores, the "omission errors" are defined on both tasks as 'failure to press the key at the appropriate time.' On the GDS, omission errors (failure to press the key when the "9" appears) suggest an inability to detect the critical signal, presumably because of a lapse of sustained attention. In contrast, on the CCPT-II, omission errors (failure to press the key when any letter other than "X" appears) indicate a failure to maintain an ongoing, repetitive motor response. Similarly, "commission errors" on both tasks are defined as "pressing the key at an incorrect time." On the GDS, commission errors (pressing the key when "9" did not appear) suggest either impulsivity or sensory discrimination errors. In contrast, commission errors on the CCPT-II (pressing the key when "X" appears) suggest an inability to inhibit a habitual response (Ballard, 2001).

Today, there are numerous variations of the CPT used in clinical and research settings. The number of CPTs in use and the number of differences across CPTs suggest that although CPTs may constitute a similar group of tasks with a common paradigm, they are also very distinct in the parameters they measure (Conners, 1992, 1995). To learn more about the effects of different CPT versions as well as the numerous scoring indices, two very different CPTs, the Conners' Continuous Performance Test second edition (CCPT-II; Conners, 1994) and the Gordon Diagnostic System test (GDS; Gordon, 1983) were compared on a population of children and adolescents with ADHD and normal controls as part of a larger study.

Research Questions

The current study explored the effects of two different CPT versions, the CCPT-II and the GDS, with children exhibiting ADHD and normal controls. As part of a larger neuropsychological assessment battery, volunteers ages 9-17 completed the continuous performance tests. This study answered the following research questions:

1) Is there a significant difference in the CCPT-II scores for children with ADHD and children with No Diagnosis?

1A) If there is a significant difference, which CCPT-II score(s) best describe the differences in children with ADHD from children with No Diagnosis?

1B) How well do the CCPT-II variables differentiate children with ADHD from those with No Diagnosis?

1C) Are CCPT-II score profiles obtained from the overall group of children able to separate the ADHD group from the No Diagnosis group?

1D) If two groups emerge from the cluster analysis, to what extent do the CCPT-II profiles correctly classify children with ADHD and No Diagnosis?

2) Is there a significant difference in the GDS scores for children with ADHD and children with No Diagnosis?

2A) If there is a significant difference, which GDS score(s) (delay, vigilance, distractibility) best describe differences in children with ADHD from children with No Diagnosis?

2B) How well do the GDS variables differentiate children with ADHD from those with No Diagnosis?

2C) Are GDS score profiles obtained from the overall group of children able to separate the ADHD group from the No Diagnosis group?

2D) If two groups emerge from the cluster analysis, to what extent do the GDS profiles correctly classify children with ADHD and No Diagnosis?

These questions are important for multiple reasons. First, the effect of the permutations of the CPT tasks has not been investigated thoroughly. Different CPTs may place different demands on an individual's attention. It is important for researchers and clinicians to evaluate whether varying CPTs are measuring the same or different constructs. Furthermore, it is important to know which type of CPT is more sensitive in differentiating children and adolescents with ADHD from normal controls. Secondly, there are many scores available for measuring CPT performance. This raises the question of which CPT variables are most useful in distinguishing children with ADHD from normal controls. It is important to assess score profiles to determine whether patterns of scores may be more useful than individual scores in differentiating children and adolescents with ADHD from normal controls.

This chapter has provided a brief overview of Attention Deficit/ Hyperactivity Disorder in children. Furthermore, the importance of evaluating two permutations of CPT tasks, the CCPT-II and the GDS, using children with ADHD and controls to determine whether they are measuring the same or different constructs was discussed. In the next chapter a review of the literature related to theories of attention and CPT measurement issues are offered. In the third chapter an in-depth discussion of the methodology used in this study is presented. Chapter IV provides a thorough presentation of the results of the study. A discussion of the study, including its implications for future research, as well as practice by neuropsychologists and other clinicians, is presented in the final chapter.

CHAPTER II

LITERATURE REVIEW

Attention-Deficit/ Hyperactivity Disorder

For over 100 years, attention-deficit/ hyperactivity disorder (ADHD) as been viewed as comprising three primary symptoms, these being poor sustained attention, impulsivity, and hyperactivity (American Psychiatric Association [APA], 1980, 1987, 2000; Barkley, 1981; Douglas, 1972, 1983). At the present time, the diagnostic criteria for ADHD is set forth in the *Diagnostic and Statistical Manual- Fourth Edition* (DSM-IV) of the APA (1994, 2000). Six or more symptoms of inattention and/ or hyperactivity and impulsivity must be present and must have persisted for at least 6 months to a degree that is maladaptive (clinically significant impairment) and inconsistent with developmental level. In addition, some of the symptoms must have been present before age 7 years; some impairment from the symptoms must be present in two or more settings; and there must be evidence of clinically significant impairment in social, academic, or occupational functioning. Please see Appendix A for DSM-IV criteria for a diagnosis of ADHD.

Recent prevalence rates of ADHD indicate that somewhere between 2% and 10% of children exhibit this disorder (Barkley, 1997; Tannock, 1998). Furthermore, there is a boy to girl ratio of around 4:1; boys, however, are generally more aggressive than girls and are more often referred. Apparent differences in prevalence rates between the United States, England, and Australia may disappear if common DMS-IV criteria are used (Sachdev, 1999). According to Barkley (1997), ADHD persists through adolescence in

50-80% of childhood diagnoses, and to adulthood in 30-50%, though its manifestations may change.

Children and adolescents with ADHD are likely to have at least one coexisting condition including communication and language disorders, learning disorders, disruptive behavior, anxiety or mood disorders (Barkley, 1998). Although relatively few community-based samples have been used to examine comorbidity with ADHD, the available data suggest that ADHD co-occurs more than 50% with oppositional defiant disorder, 30-50% with conduct disorder, 15-20% with mood disorders, and 20-25% with anxiety disorders (American Academy of Child and Adolescent Psychiatry, 1997; Biederman, Newcorn, & Sprich, 1991; Biederman, Faraone, Mick, Moore, & Lelon, 1996; Livingston, Dykman, & Acherman, 1990). In another review of studies, Jenson, Martin, and Cantwell (1997) indicated that the comorbidity between ADHD and conduct disorder/ oppositional defiant disorder was between 42.7% to 93.0%. Furthermore, these researchers found that the comorbidity of ADHD and internalizing disorders was between 13.0% and 50.8%. Studies indicate that the comorbidity of ADHD and reading disorder is between 20% and 30% (Hinshaw, 1992; Semrud-Clikeman et al., 1992). The high rates of comorbidity between ADHD and other disorders have created several problems that include choosing valid assessment instruments, making accurate diagnoses, and selecting appropriate treatments. These problems are compounded by disorders that have similar behavioral symptoms. For example, ADHD and anxiety disorders share several characteristics including difficulty concentrating, irritability, and restlessness.

The etiology of ADHD is unknown. Family genetic factors have been implicated as etiological in ADHD for at least 25 years. Family aggregation studies show that ADHD runs in close family members (Biederman, Baldessarini, Wright, Knee, & Harmatz, 1989). Heritability is estimated to be between 0.55 and 0.92 (Cantwell, 1996). The concordance rate of ADHD was 51% in monozygotic twins and 33% in dizygotic twins in one study (Goodman & Stevenson, 1989). The siblings of children with ADHD are six times more likely as controls to develop ADHD, whereas the child of a parent with ADHD has a 50% chance of exhibiting the disorder. Conversely, 1 in 4 diagnosed children has a biological parent with ADHD (Goodman & Stevenson, 1989). Adoption studies further indicate that ADHD tends to be genetic rather than environmental (Barkley, 1990; Cantwell, 1975).

Environmental factors have been associated with ADHD. Complications of pregnancy or delivery, poor maternal health, increased maternal age, fetal prematurity, prolonged labor, fetal distress, and reduced birth weight are potential factors (Faraone & Biederman, 1998). Other factors may include maternal use of alcohol or tobacco, brain injuries, or exposure to lead in childhood (Barkley, 1998).

Pyschosocial factors are not thought to play a primary etiological role in ADHD. Various types of parent-child relationships and family dysfunction are found in families of children with ADHD. Younger children with ADHD are more likely to have interaction conflicts with their mothers than older children with ADHD. In the adolescent range, more noncompliant and negative verbalizations are reported in families that have children with ADHD. These psychosocial factors are thought to be primarily related to the development of oppositional defiant disorder and conduct disorder rather than the core symptoms of ADHD (Cantwell, 1996).

Historical Perspectives on ADHD

One of the first approaches to describing an attention disorder was provided by Crinella (1973). Crinella included a clinical description (hyperactive-aggressive behavior) of minimal brain damage from Wender (1971) and technical concepts of attention (alertness and kinetic mobility) previously used by Luria (1966). In his groundbreaking work, Douglas (1972) challenged Clements (1966) clinical descriptions that were based on informal diagnoses of hyperactivity and hyperkinesis. Douglas stimulated the modern description based on the concept of attention deficit, and used descriptions of cognitive processes ("inability to sustain attention and control impulsivity") based on the classic descriptions of attention dating back to James (1890). Sergeant (1981) used the clinical description ("hyperactive and/or distractible") based on reliable ratings from multiple sources and the technical descriptions of cognitive processes (encoding, comparison, response selection, and response execution) from Sternberg (1969).

Before 1980, the core feature of ADHD was considered to be hyperactivity, and the label used to describe the disorder was hyperkinetic reaction of childhood (DSM-II, 1968). With the revisions of DSM-III in 1980, the term attention deficit disorder (ADD) was introduced as the new diagnostic label for the disorder. In addition, two cognitive symptom domains (inattention and impulsivity), along with the motor system domain (hyperactivity), were introduced to define the two subtypes of the disorder with (ADDH) and without (ADD) hyperactivity. The revisions of DSM-III-R (1987) changed the label only slightly to attention-deficit hyperactivity disorder (A-DHD) but the criteria was changed significantly; the polytypic concept was rejected in favor of a monotypic concept based on merging the three domains of symptoms and defining a single type of the disorder. The revisions of DSM-IV (1994) re-established the use of multiple domains of systems and multiple subtypes of the disorder and changed the name to attention-deficit/ hyperactivity disorder (A-D/HD). Furthermore, two domains of symptoms were established by merging the DSM-III symptom domains of impulsivity and hyperactivity into one (hyperactivity/ impulsivity) and expanding the symptoms of inattention in a separate domain (inattention).

The multiple changes in the DSM were intended to refine the clinical definition of ADHD. Although the symptom domains and subtypes of ADHD have changed often, the specific domains and symptoms have remained the same, with a gradual increase in the number of symptoms in the inattention domain, a gradual decrease in the number of symptoms in the impulsivity domain, and the addition of symptoms of verbal activity in the hyperactivity domain (Swanson et al., 1998).

Assessment Measures for Children with ADHD

The continuous performance test is a paradigm used for the evaluation of attention and the response inhibition component of executive control that represents an effort to incorporate reliable and objective assessment into evaluations for ADHD. These techniques were born of concern about the degree to which diagnostic decisions were founded upon subjective measures and clinical judgment. Loeber, Green, Lahey, and

Stouthamer-Loeber (1989) identified discrepancies between parent and teacher reports of hyperactivity and inattention, suggesting that certain elements of this disorder may be situationally specific and thus difficult for informants who do not observe the child in that situation to precisely identify. Furthermore, clinical ratings may be susceptible to halo effects, in which other behavioral disturbances (e.g., defiant behaviors) inflate perceptions of ADHD symptomatology (Abikoff, Courtney, Pelham, & Koplewicz, 1993; Schachar, Sandberg, & Rutter, 1986). Additionally, inattention, impulsivity, and hyperactivity, although conceptually distinct, may be difficult to distinguish through behavioral observations (Hinshaw, 1987). Unlike other clinical techniques, computer-based measures generate objective data about a child's ability to perform in situations tailored to assess the characteristic weaknesses of a child with ADHD (Gordon, 1986).

The term "Continuous Performance Task" was first coined over 40 years ago to describe a vigilance task designed to study attention in epileptic subjects (Rosvold, Mirsky, Sarason, Bransome, & Beck, 1956). In the original task by Rosvold and colleagues (1956), letters were presented visually one at a time, at a fixed rate with 920 milliseconds between letter presentation. The subject was to respond by pressing a level whenever the letter "X" appeared and to inhibit responding when any other letter appeared (X-type CPT). Rosvold and colleagues (1956) found the X-type CPT correctly classified 84.2-89.5% of younger subjects with identified brain damage. Rosvold and colleagues also introduced a variation of this task in which the target was the letter "X" but only if the "X" was immediately preceded by the letter "A" (AX-type CPT). The ability to classify subjects accurately based on CPT performance increased with the increased difficulty level of the AX-type CPT.

Since 1956, researchers have devised multiple variations of the components of CPTs. For example, the target stimulus in the CPT may be the letter “X”, as in the original version, or a number (e.g., Gordon, 1983), a picture of an object or person (e.g., Anderson, Siegel, Fisch, & Wirt, 1969), or a word (e.g., Earle-Boyer, Serper, Davidson, & Harvey, 1991). The task may be the X-type CPT, an AX-type CPT, or a further modification of the AX such that the target must be preceded by itself (XX-type; e.g., Fitzpatrick, Klorman, Brumaghim, & Borgstedt, 1992) or where color and letter are critical features (e.g., an orange “T” followed by a blue “S”; Garfinkel & Klee, 1983) or such that two digits in a number series (or letters in a letter series) are the same in two consecutive stimuli (Identical Pairs or IP-type, Cornblatt, Lenzenweger, & Erlenmeyer-Kimling, 1989). Other variations involve changing the modality such that the presentation may be visual as in the initial version, or auditory (e.g., Earle-Boyer et al., 1991), or variable within the same task between auditory and visual stimuli (e.g., Sandford & Turner, 1995). In addition to the multiple variations of the CPT, these tasks allow the examiner freedom to modify the task for use with participants of varying ages and abilities. The target and distractor stimuli can be changed to meet the needs of the examiner and the characteristics of the participants. The interstimulus interval, duration of the task stimuli, and size of stimuli also can be manipulated to either increase or reduce the task demands for the intended population.

According to Riccio and colleagues (2001), the CPT paradigm is characterized by a rapid presentation of continuously changing stimuli with a designated target stimulus or target pattern. The CPT involves vigilance and selective attention for an infrequently occurring target or relevant stimulus. Presently, there are numerous variations of the CPT used in research and clinical settings. According to Conners (1992, 1995), the number of CPTs in use and the number of differences across CPTs suggest that although CPTs may constitute a similar group of tasks with a common paradigm, they are also very distinct in the parameters they measure.

Measurement Issues and Standards

When using CPTs to measure attention, the validity of the tests should be taken into account. Analyses of the relationship of test scores to variables external to the test provide an important source of validity evidence referred to as validity generalization (American Educational Research Association [AERA, 1999], American Psychological Association [APA, 1999], National Council on Measurement in Education, [NCME, 1999]. External variables may include some criteria the test is expected to predict, as well as relationships to other tests hypothesized to measure the same, related, or different constructs. Categorical variables, including group membership variables, become relevant when the theory underlying a proposed use of the test suggests that group differences should be present or absent (AERA, APA, NCME, 1999).

Research suggests that CPT performance is correlated with performance on several laboratory measures of attention and activity level (Allen, 1993; Kardell, 1994; Robbins, 1992; Slicker, 1991). Several studies have investigated the relationship between the Stroop Color and Word Test (Stroop, 1935) and CPT performance (Allen, 1993; Bock, 1982; Carter, Krener, Chanderjian, Northcutt, & Wolfe, 1995; List, 1985; Rasile, Burg, Burright, & Donovick, 1995). Moderate correlations were found between the Stroop and CPTs (Burg, Burright, & Donovick, 1995; Das, Snyder, & Mishra, 1992); only in one study was the correlation not found to be at least moderate (Rasile et al., 1995). Research also suggests that the CPT omission errors correlate moderately with the Wisconsin Card Sorting Test (Heaton, 1981) Total Errors and Total Categories scores (Allen, 1993; Kardell, 1994; Slicker, 1991).

Although scores on various CPTs appear to correlate with some neuropyschological measures of attention, the utility of CPTs at assessing individual children is limited by several factors. Modest correlations between commission and omission CPT scores and teacher ratings of inattention, impulsivity, and hyperactivity have been obtained in the .25 to .51 range (Barkley, 1991; Gordon, DiNiro, Mettelman, & Tallmadge, 1989; Halperin et al., 1988; Klee & Garfinkel, 1983; Pascaulvaca, Wolf, Healey, Tweedy, & Halperin, 1988). Furthermore, studies examining types of commission errors have found significant associations between certain patterns of errors and behavior ratings. For example, one study found a significant correlation between errors made to the first member of the target sequence and teacher ratings of impulsive and hyperactive behavior, whereas errors made in response to the second member of the target pair were associated with teacher ratings of inattentiveness (Halperin et al., 1988). However, several studies have failed to obtain significant correlations between criterion measures (e.g., teacher ratings, other laboratory measures of vigilance and impulsivity) and scores on various CPTs (DuPaul et al., 1992; Halperin et al., 1991; Lovejoy & Rasmussen, 1990; Thompson & Nichols, 1992). For example, Gordon, DiNiro, and Mettelman (1988) found that the diagnostic "hit rate" of CPT scores agreed with classifications based on parent and teacher ratings for approximately 50% of a large sample of clinic-referred children. Fisher, Newby, and Gordon (1993) pointed out that a high level of agreements between CPTs and behavior ratings are of uncertain value. They asserted that an agreement rate of 100% would make a CPT dispensable since it would not add anything unique to the clinical decision process.

Epstein and colleagues (2003) attempted to examine relations between various CPT variables and phenotypic behaviors using the Conners' CPT-II and a structured parent interview called the Child and Adolescent Psychiatric Assessment (CAPA: Angold & Costello, 1995, 2000). Using an epidemiological sample of 817 children, the researchers found that the overall measures of commission errors, mean hit RT standard error, sensitivity (d'), and response bias (β) appeared to be highly related to the entire constellation of ADHD symptoms across symptom domains. When examining performance on these measures over time, increased mean hit RT standard errors over time was highly associated with most ADHD symptoms. The only CPT performance measure that showed a specific relationship to a particular domain of symptoms was the relationship between slowed RTs over time and hyperactive-impulsivity symptomatology.

Results regarding the validity of CPT tasks are inconclusive. As a group, children with ADHD exhibit more difficulties on CPT measures than relative controls (Barry, Klinger, Lyman, Bush, & Hawkins, 2001; Barkley et al., 1990; Dickerson Mayes, Calhoun, & Crowell, 2001; Fischer et al., 1990; Halperin, et al., 1988; Klee & Garfinkel, 1983; Sykes, Douglas, & Morganstern, 1973). In order to determine the extent to which CPT performance can discriminate children with ADHD from controls, Corkum and Siegel (1993) conducted a narrative review of nine studies comparing overall performance between these groups. Four of the nine studies found significant differences in commission errors between children in ADHD and control groups. Of the five studies that employed measures derived from signal detection theory (i.e., sensitivity and bias), two found significant differences between children with ADHD and controls. A more recent meta-analytic review of CPT research (Losier, McGrath, & Klein, 1996), confirmed poorer CPT performance as measured by omission and commission error rates in ADHD children compared to normative controls.

The CPT measures lack specificity in discriminating among children with ADHD and other clinical groups. When the effects of age, sex, and IQ have been partialed out, scores on CPT measures have failed to discriminate among children with ADHD, conduct disorder, anxiety disorder and normal controls (Koriath, Gualtieri, Van Bourgondien, Quade, & Werry, 1985; Werry et al., 1987). Even when the effects of age, sex, and IQ have not been partialed out, scores on CPT measures have not reliably discriminated among different groups of clinic-referred children (Campbell, 1974: Halperin et al., 1990; Riccio et al., 2001; Shapiro & Garfinkel, 1986). Matier-Sharma and colleagues (1995) noted that CPT measures were only slightly better than chance at distinguishing ADHD from non-ADHD disorders in a clinical sample. In a recent study by McGee, Clark, and Symons (2000), children with ADHD did not have higher CPT scores than clinical controls; however, children with reading disorders did. Published practice parameters by the American Academy of Child and Adolescent Psychiatry (Dulcan and the Work Group on Quality Issues, 1997), stated that CPTs 'generally are not useful in diagnosis because they suffer from low specificity and sensitivity' (p. 87S). In a study assessing the ability of the GDS composite score in predicting ADHD and non-ADHD group membership, Dickerson Mayes and colleagues (2001) found the composite score to correctly classify 79% of children with ADHD and 52% of the children in the non-ADHD group. Carpenter (2002) found that the overall level of predictive accuracy of the GDS in predicting the presence of ADHD was 73%. In one study using either the Response Control Quotient or the Attention Control Quotient of the Integrated Visual and Auditory CPT (IVA; Sandford & Turner, 1995), only 71% of the ADHD group were correctly classified as ADHD and 36% of the non-ADHD group were classified as ADHD (Edwards, 1998). A diagnosis of more than 1 of 3 persons examined who do not actually have ADHD as having the disorder is an unacceptable error rate.

Methodological Limitations of Research Using CPTs

A review of the literature using CPTs with children with ADHD reveals discrepant findings. The reason for these equivocal findings is unknown. However, equivocal findings may be related in part to one or more of the following points, all of which have varied considerably in this literature (Seidel & Joschko, 1990).

Definition of ADHD Subjects

Studies relying on dissimilar types of data, such as rating scales, behavior observations and diagnostic classifications, may result in different populations being identified as ADHD (Barkley, 1981). Additionally, even when common categories are used (e.g., ADHD), the optimal cutoff points on CPTs for diagnostic classification vary substantially from one study to another. Furthermore, some studies have used ADHD subtypes (i.e., ADHD, Combined Type; ADHD, Predominantly Inattentive Type) while other studies have combined subtype groups. Finally, the multiple changes in DMS definitions of ADHD subtypes over time make it difficult to interpret literature findings.

Characteristics of the Groups Used

Differences in age ranges used, group size, and inconsistent matching procedures for ADHD and control groups on important dimensions (e.g., age, gender, and IQ) can lead to discrepant results. Furthermore, the inclusion of children and adolescents with comorbid diagnoses (e.g. ADHD with LD or psychiatric disorders) in some studies but not others may lead to a lack of agreement in results. Stimulant medication usage by children and adolescents with ADHD may decrease group differences on the CPT in research studies. Research by Losier and colleagues (1996) indicated that children with ADHD treated with methylphenidate exhibited statistically significant reductions in the rates of both omission and commission errors on the CPT.

Parameters used in the CPT

Task parameters such as length of task and stimuli characteristics (e.g., modality, complexity, and presentation rate) differ across studies. With respect to sensory modality, visual tasks tend to yield steeper decrements and lower overall accuracy than do auditory tasks (Buckner, Harabedian, & McGrath, 1968; Davies, Jones, & Taylor, 1984: Hatfield & Loeb, 1968; Warm & Jerison, 1984). Research indicates that the target letter or number typically makes up between 8% and 39% of the total stimulus set shown (e.g., Barkley et al., 1990; Bergman, Winters, & Cornblatt, 1990; Michael, Korlman, Salzman, Borgstedt, & Dainer, 1981). Performance is poorer when signals occur very infrequently (Baker, 1959a; McGrath, Harabedian, & Buckner, 1968; Matthews, Davies, & Lees, 1988; Warm & Jerison, 1984) and when there is a short interstimulus interval. The specific effects of changes in parameters has been largely unexplored in any systematic way despite evidence that changes in stimuli complexity (Nuechterlein, Parasuraman, & Jing, 1983) and presentation rate (Parasuraman & Davies, 1977) can affect performance on tasks of attention.

Testing Procedure Employed

Testing procedures on the CPT task might include the instructions given, the presence or absence of an examiner, and the presence or absence of feedback. Performance of subjects with ADHD on experimental tasks has been shown to be affected by instructional set (Sergeant & Scholten, 1985) and may set up expectancies that facilitate or degrade performance (Berch & Kanter, 1984; Dember, Galinsky, & Warm, 1992; Lucaccini, Freedy, & Lyman, 1968; McGrath et al., 1968). The use of feedback (Berch & Kanter, 1984; Mackworth, 1970; Parry & Douglas, 1983; Warm & Jerison, 1984), and the presence of an examiner (Draeger, Prior, and Sanson, 1986) can affect performance as well. Ballard (1996) indicated that environmental factors such as noise and temperature interact with testing procedures and affect performance. When comparing children with ADHD to normal controls, Rickman (2001) found that exposure to classroom-based distractions while completing a CPT resulted in more errors suggestive of inattention and impulsivity.

The ability of the CPT to provide additional data as part of a thorough assessment depends on whether the instrument measures constructs that are consistent with deficits evidenced in individuals with ADHD. Attention is not unitary; rather it is a nebulous and complex construct (Mirsky, Fantie, & Tatman, 1995). Zubin (1975) suggested that attention could be conceptualized as having multiple components or elements. As attention has been studied by researchers from different scientific perspectives, it is not hard to see why a variety of attentional models have emerged. Although not comprehensive, a review of several neurological and neuropsychological models of attention and ADHD is provided.

Neurological Basis of Attention and ADHD

Neuroanatomical Basis of ADHD

To learn more about the neuroanatomical basis of ADHD, several recent studies have employed modern magnetic resonance imaging technology to evaluate the structural volumes in various regions of the brain in children with ADHD. These studies have consistently demonstrated significantly reduced regions in the right prefrontal cortex, the striatum, the corpus collosum, and the right cerebellum in children having ADHD (Baumgardner et al., 1996; Castellanos et al., 1994, 1996; Hynd et al., 1993; Semrud-Clikeman et al., 1994). Filipek and colleagues (1999) reported smaller volumes in the following structures: the left total caudate and caudate head, right anterior superior (frontal) region and white matter, bilateral anterior inferior region, and bilateral retrocallosal (parietal-occipital) region white matter. These localized structural anomalies appear to be concordant with theoretical models of abnormal frontal-striatal and parietal function. Castellanos and colleagues (1996) provided supporting evidence for the hypothesized dysfunction of right-sided prefrontal-striatal systems in ADHD. They found that ADHD subjects had a 4.7% smaller total cerebral volume, a significant loss of normal right-greater-than-left asymmetry in the caudate, smaller right globus pallidus, a smaller right anterior frontal region, and reversal of normal lateral ventricular asymmetry. In addition, Hynd, Semrud-Clikeman, and Lorys (1991) reported a trend in ADHD cases to have smaller anterior (genu) and posterior (splenium) regions of the corpus callosum, which connect the frontal and parietal brain regions of the two hemispheres. Despite some inconsistencies in the specific region, other studies have confirmed this general pattern of reduced size of the corpus callosum. Reduced volume in the right prefrontal and striatal regions is associated with significantly greater difficulties in behavioral inhibition (Casey et al., 1997). Overall, these studies of brain anatomy suggest that children with ADHD have abnormalities (smaller than normal size) in three brain structures: the corpus callosum (orienting), basal ganglia (executive control, coordination, switching off automatic responses), and right frontal lobes (alerting, “editing” ones behavior, resisting distractions).

Physiological functioning in certain brain regions has been shown to be reduced significantly below normal levels in children with ADHD. Using SPECT measures of blood flow with children exhibiting ADHD has indicated a decreased cerebral blood flow (about 10% lower blood flow), particularly in the prefrontal and striatal regions (Lou, Henriksen & Bruhn, 1984; Sieg, Gaffney, Preston, & Hellings, 1995) and in the occipital brain areas. Decreased brain electrical activity, particularly in prefrontal regions (Hastings & Barkley, 1978; Klorman, 1992) also has been noted. Researchers have used PET measures of brain glucose metabolism in adults and adolescents with ADHD (Zametkin et al., 1990; Zametkin et al., 1993). Despite some inconsistencies (i.e., lower global glucose metabolism in adults with ADHD did not hold up in a subsequent study of adolescents with ADHD), there was consistency across this work when the ADHD and control groups were compared on the basis of normalized brain images; the prefrontal areas showed evidence of relative underactivity (i.e., about 10% lower glucose metabolism). In a recent MRI study, Yeo and colleagues (2003) assessed the concentration of neurometabolites in the right prefrontal region in children with ADHD, Combined Type. Results indicated that children with ADHD Combined Type exhibited smaller right dorsolateral volumes; however, they did not evidence any abnormalities in concentration of neurometabolites in the right prefrontal region. Overall, these brain-imaging studies of individuals with ADHD suggest an abnormality in the frontal lobes (implicating the alerting and executive control networks).

Neurochemical Basis of ADHD

Researchers have postulated that catecholamines (dopamine, norepinephrine) are implicated in ADHD and appear to affect attention, inhibition, and motivation (Clark, Geffen, & Geffen, 1987). An imbalance in the formation of dopamine or norepinephrine that results in decreased stimulation of the brainstem reticular activating system has been postulated (Mefford & Potter, 1989). Some support for this conceptualization is provided from the efficacy of treatment with psychostimulants in some children with ADHD. Stimulant medication is thought to achieve its pharmacologic effect by potentiating the effects of dopamine and possibly norepinephrine (Shekim, Javaid, DeKirmenjian, Chapel, & Davis, 1982). Tucker and Williamson (1984) suggested that attentional control involves two separate neural systems: (1) an activation system that is centered in the left hemisphere and controls sequential, routine, and analytic operations such as motor responses. This system is modulated by dopaminergic transmitters. (2) An arousal system that is centered in the right hemisphere and specializes in parallel, holistic, and novel cognitive functions, such as perceptual orienting responses. This system is modulated by norepinephrinergic neurotransmitters. Related to the dopaminergic models, Posner, Inhoff, and Fredrich (1987) posited that the parietal lobe is involved in covert shifting of visual attention, whereas the frontal lobe is the attentional command system, and that both work together to regulate attentional processes.

Neurophysiological Basis of Attention

A review of research pertaining to neuroanatomical specialization and biochemical models of attention suggest that a single model of attention is incomplete and that a conceptualization of a complex functional system would be more appropriate. Across studies, various models of attention consistently suggest the interaction of cortical (frontal, prefrontal, parietal) with subcortical (limbic system, reticular activating system (RAS), and basal ganglia) structures as well as pathways between the basal ganglia, thalamus, and frontal lobes to form a complex functioning system (Riccio, Reynolds, Lowe, & Moore, 2002).

Mesulam (1981, 1990) was one of the first to offer a model of an integrated attentional system. Based on data from brain-damaged patients and from neuroanatomical studies of nonhuman primates, Mesulam proposed a network model of attention in which several distinct cortical regions interact. These regions include the posterior parietal cortex, the cingulate cortex, and the frontal cortex, all of which are influenced by the reticular activating system. According to Mesulam's model (1990), a separate spatial coordinate system is represented within each of these brain regions. The parietal component provides an internal perceptual map of the external world; the cingulate component regulates the spatial distribution of motivational valence; the frontal component coordinates the motor programs for exploration, scanning, reaching, and fixating; and the reticular component (including noradrenergic, dopaminergic, and cholinergic ascending systems) provides the underlying level of arousal (Robbins & Everett, 1995). The cortical components within this network are believed to be heavily and reciprocally interconnected with each other (Barbas & Mesulam, 1985).

The Frontal-Diencephalic-Brainstem System (FDB) was proposed to Stuss and Benson (1984). This model included the reticular system and the frontal lobes consistent with Mesulam's model. However, Stuss and Benson placed additional emphasis on the role of the frontal-thalamic gating system and the afferent and efferent projections associated with the thalamus (e.g., to and from the reticular system and the frontal system). According to Stuss and Benson (1984), damage to the thalamic projection system may impair an individual's level of alertness while damage to the frontal-thalamic gating system is associated with impaired selective attention and the inability to self-monitor arousal and sustained attention over time.

Pribram and McGuiness (1975) and McGuiness and Pribram (1980) contended that there are three separate but interacting neural systems that operate to control attention. First, there is an arousal system regulating short-lived, physiological responses to novel input (i.e., the orienting response). Forebrain control over this arousal system (i.e., spinal cord, reticular formation, etc.) is regulated by two reciprocal circuits based in the amygdala and controlled neurochemically by norepinephrine and serotonin. According to this theory, the dorsolateral frontal cortex circuit is excitatory and the orbitofrontal cortex is inhibitory. The second activation system regulates tonic, long-lasting physiological readiness to respond. This system is based in the basal ganglia and mediated by dopamine and acetylcholine. The third system is responsible for the effort involved in the integration and coordination of the arousal and activation systems. This effortful control system is based in the hippocampus.

The model of attention proposed by Posner (1990, 1995) incorporated the same brain regions as that of Mesulam (1990), but the regions are organized into somewhat different functional networks that perform the presumably different cognitive functions of alerting, orienting, and executive control. The model consists of a posterior attention network, an anterior attention network, and a vigilance network. According to Posner (1990), the posterior network involves the parietal cortex, the pulvinar, and the superior colliculus. These areas work together to perform the operations need to bring attention to, or orient to, a location in space. According to Posner and Petersen (1990), the parietal cortex disengages the attention from the present target, the superior colliculus acts to move attention to the intended target, and the pulvinar is involved in the engagement of attention at the intended target. The anterior attention network involves the anterior cingulate cortex and supplementary motor areas in the frontal cortex, which work together in a wide variety of situations involving the detection of events and the preparation of appropriate responses. According to Posner (1990), it is the anterior attention network that is proposed to exercise executive control over voluntary behavior and thought processes. Finally, the vigilance network involves the locus coeruleus noradrenergic input to the cortex (Harley, 1987), that is crucial for maintaining a state of alertness.

In contrast to other models of attention, Quay (1997) viewed the hyperactive, impulsive variant of ADHD as due to an underactive behavioral inhibitory system (BIS). Quay placed the BIS in the septohippocampal complex together with the prefrontal (orbitofrontal) cortex, and under largely noradrenergic and serotonergic control. The BIS controls passive avoidance (inhibition of responding learned under threat and nonreward) and acts in opposition to a (dopaminergic) reward/ activation system, which responds to conditioned stimuli for reward and underlies active avoidance and escape. Damage to the BIS is thought to result in an inability to inhibit responding in the face of probable punishment or nonreward, and children with ADHD are likely to be less responsive to conditioned stimuli. According to Quay, the BIS in children with ADHD may be overactive. Quay noted that this model does not apply to the predominantly inattentive type of ADHD.

Weinberg and Harper (1993) described a primary disorder of vigilance. According to these researchers, vigilance is defined as steady-state alertness-wakefulness. In the psychological context, the authors indicated that vigilance involves tasks in which attention is directed to one or more sources of information over varying periods of time without interruption for the purpose of detecting small changes in the information being presented. Studies using evoked cortical potentials and EEG desynchronization have attempted to relate various cortical brain sites to different states of supported vigilance. Findings indicate that the right cerebral hemisphere, predominantly the right inferior parietal lobule and posterior parietal cortices, appear specialized for vigilance (Dimond, & Beaumont, 1973).

Current etiologic theories suggest that ADHD stems from abnormalities in the cortico-striato-thalamo-cortical network, believed to be critical for executive functions and the regulation of behavioral responses such as arousal, attention, and inhibition. In the 1990's, this circuit was the object of intense study in rodents, nonhuman primates, and humans (Albin, Young, & Penney, 1995; DeLong, 1990; Gerfen, 1992). This network is believed to be critical for the proper maintenance of prefrontal based executive functions and the regulation of behavioral responses (Barkley, 1997; McCracken, 1991). Researchers have proposed that dysfunction in this system leads to problems with arousal, behavioral inhibition, and, more generally, with attention processes (Barkley, 1997; Sergeant, Oosterlaan, & Van der Meere,1999; Swanson et al.,1998).

Neuropyschological Theories of Attention

Neuropsychologists, at the most basic level, conceptualize attention as involving selective processing and awareness of stimuli (Mesulam, 1985a, 1985b). The term "attention" may be used to refer to the following: (1) initiation or focusing of attention; (2) sustaining attention or vigilance; (3) inhibiting responses to irrelevant stimuli or selective attention; and (4) shifting attention (Denckla, 1996; Mirsky, 1989; Mirsky, Anthony, Duncan, Ahearn, & Kellam, 1991; Sohlberg & Mateer, 1989; Zubin, 1975). Mirsky (1987) proposed restricting the myriad of aspects of attention to the focusing of attention, sustaining of attention, and shifting of attention. According to Mirsky's model, the *focus* element represents the ability to select target information from an array for enhanced processing. The *sustain* component represents the capacity to maintain focus and alertness over time, or vigilance. The term *shift* represents the ability to change attentive focus in a flexible and adaptive manner.

Cooley and Morris (1990) reviewed evidence for three components of attention called selective, divided, and sustained attention. According to Cooley and Morris (1990), the concept of vigilance or sustained attention is based on paradigms that evaluate the ability of individuals to detect changes in stimulus events over relatively long time intervals. Theories of vigilance have sought to explain two main questions: (a) the vigilance decrement or decline in correct detection over time, and (b) determinants of the overall level of performance (Davies & Parasuraman, 1982; Frankmann & Adams, 1962; Warm, 1977).

According to Cooley and Morris (1990), the first explanation of the vigilance phenomenon, was offered by Mackworth and termed the Inhibition Theory (Mackworth, 1950). Vigilance was seen as an excitatory state that was opposed by an inhibitory state as the task progressed. Broadbent's (1958, 1971) Filter Theory, proposed that a decrement in correct detections occurs because the repeated presentation of a stimulus results in reduced novelty which then allows other aspects of the stimulating situation to assume higher priority. Another theory of vigilance was proposed by Deese (1955) and later elaborated by Baker (1959a, 1963). According to Baker (1959b, 1963), signal detection depends on an individual's expectancy or ability to predict the occurrence of a signal.

Norman and Shallice's (1986) model of attentional control incorporated a supervisory attentional system (SAS). The SAS assumes that two complementary processes operate in the selection of control of action. The basic mechanism is termed *contentional scheduling* which is thought to be able to control routine activities automatically, without conscious control or attentional resources. In nonroutine situations requiring novel actions, the contention scheduling mechanism is modulated by the deliberate, conscious control of the SAS (Norman & Shallice, 1986; Shallice, Burgess, Schon, & Baxter, 1989). The model is based on the operation of self-contained, well-learned action and thoughts called *schemata*. A schemata can be activated by well-learned triggers. The contention scheduling mechanism prevents schemata from competing for the same cognitive resources by means of a lateral inhibitory mechanism (Shallice & Burgess, 1991). However, conflict between schemata are inevitable and, therefore, a conflict resolution procedure is necessary. The SAS performs this procedure by modulating the activation level of the schemata, thus biasing their probability of being selected (Shallice & Burgess, 1991). Higher level processes of the SAS are implicated when the conscious control of action is required.

While many theorists propose models that include components of initiating, focusing and sustaining attention, Barkley's unified theory of ADHD (1997), based on the theoretical work of Bronowski (1967), proposed that the symptoms described in ADHD are most accurately explained by an impairment in response inhibition, that results in difficulty self-regulating responses to stimuli. According to Barkley (1997), inhibition is assessed by performance on cognitive or behavioral tasks that require withholding or delayed responding, cessation of ongoing responses, and resisting distraction by competing events. This impairment causes the symptoms seen in ADHD, such as hyperactivity, inattention, distractibility, and impulsivity. Barkley further postulated that this delayed responding is mediated by underfunctioning of the orbital frontal cortex and subsequent connections to the limbic system. The result is hyper-responsivity to stimuli producing hyperactivity primarily and, secondarily, inattentiveness (Barkley, 1997).

Barkley's model (1997) is arranged hierarchically and hypothesizes that deficiencies in behavioral inhibition, at the top of the hierarchy, set the occasion for difficulties in four executive neuropsychological functions that include: (a) working memory, (b) self-regulation of affect-motivation-arousal, (c) internalization of speech, and (d) reconstitution (behavioral analysis and synthesis). Barkley explained that behavior inhibition does not directly cause the four executive or self-directed actions to occur; behavioral inhibition provides the delay necessary for the four executive functions to occur. According to Barkley (1997), ADHD is believed to disrupt these executive functions because the first executive, self-regulatory act must be inhibition of responding.

Developmental Theories of Attention

Neurodevelopment is believed to follow an ontogenetic course, with primary cortical areas generally mature by birth (Luria, 1980). Secondary and tertiary areas continue to develop postnatally. These include the functional systems involved in attention as well as learning, memory, emotion, cognition and language. Researchers also suggest that the pathways that connect structures within these area and the primary areas are likely to change over time also (Rutter, 1981; Vygotsky, 1980). Mattes (1980) suggested that one explanation for hyperactivity may be a developmental delay in the myelination of the prefrontal area.

Luria (1973) suggested that the prefrontal regions of the brain in normal children do not begin to become prepared for action until the child is between the ages of 4 and 7, whereas Golden (1981) has suggested the frontal regions do not become functionally mature until adolescence. Empirical studies (Chelune & Baer, 1986; Welsh, Pennington, & Grossier, 1991) have demonstrated that behaviors presumed to be dependent on frontal lobe functioning develop rapidly from the ages of 6 and essentially reach adult levels of mastery between the ages of 10 and 12 in normal children.

Research indicates that there are marked developmental gains in the ability to inhibit responses throughout childhood that continue into early adulthood (Band, van der Molen, Overtoom, & Verbaten, 2000, Schachar & Logan, 1990). Normal children younger than 6 years old appear to be able to verbalize when a response should be inhibited but not necessarily make the related motor response (Bell & Livesey, 1985; Livesey & Morgan, 1991). Studies suggest that by the age of 7, typically developing children have the conceptual understanding of when to inhibit responses, but that this may not always translate into successful procedural behavioral performance (Dowsett & Livesey, 2000). In one study examining the performance of normal male and female children at four age levels between 6 and 12 years of age, Passler, Isaac, and Hynd (1985) found that the greatest period of frontal lobe development appeared to occur at the 6 and 8-yer-old levels. By the age of 10, the ability to inhibit attention to irrelevant stimuli was fairly complete with mastery evident by age 12. Researchers have speculated that improvements in inhibitory control correlate with increasing cognitive abilities and with the maturation of the prefrontal cortex (Carver, Livesey, & Charles, 2001; Passler et al., 1985). Reaction time improvements for both response execution and response inhibition are observed between the ages of 6 and 20 (Band et al., 2000, Williams, Ponesse, Schachar, Logan, & Tannock, 1999). Conners, Epstein, Angold, and Klaric (2003) administered the CCPT to a random sample of 816 normal 9-17 year olds. Systematic main effects of improved performance with older children were found for all variables. According to Passler and colleagues (1985), the issue of developmental change is particularly relevant for understanding the nature of ADHD since it is essentially defined as a disorder of maturation; that is, the cognitive and behavioral abilities of the child with ADHD are not seen as deviant, but rather as developmentally inappropriate for the child's chronological and mental age.

As is evident from a review of neurological and neuropsychological theories, attention is a complex functional system that can include various components. As such, assessment of attention and executive control must be multifaceted. The best any single measure, including the CPT, can provide is data on specific aspects of attention and executive control that can provide additional information to be used in the diagnostic decision-making process. To accurately interpret results of the CPT task, clinicians need to be knowledgeable of the many CPT scores and what they measure.

Scores Provided by the CPT

The effects of different CPT scores used in the interpretation process of the CPT warrants further investigation. When Rosvold and colleagues (1956) introduced the CPT, the focus was on correct hits (number or percent of correct responses to targets) as an indication of selective attention (e.g., Allen, 1993; Brumm, 1994; Carter et al., 1995). More recently, other types of scores have been used as measures of CPT performance. For example, both correct hits and omission errors (i.e., failure to respond to target) are interpreted as indicative of alertness as well as the individual's ability or inability to selectively attend to the target. The number of commission errors, or responses to stimuli other than the target, is frequently reported as a measure of response inhibition, impulsivity, or response control (Riccio et al., 2001). Halperin and colleagues (1991) suggested that there are subtypes of commission errors. Specifically, they identified a "fast reaction-time response" associated with impulsivity and hyperactivity and a "slow reaction-time response" or delayed response associated with attention. Finally, relative accuracy, defined as the percent of correct responses of total responses, or total errors (omission and commission errors) is often reported (e.g., Bock, 1982).

Reaction time or latency is believed to reflect the speed of processing and decision to respond. Reaction time may be reported directly as latency or reaction time or as a Speed Quotient (Sandford & Turner, 1995). The time lapse between presentation of the stimuli (interstimulus interval or ISI) has been varied with studies using a shorter interval or longer interval or variable interval (Girardi et al., 1995; Rueckert & Grafman, 1996). Variable intervals may be preset such that for some blocks of trials the ISI is at one rate while for other blocks the ISI is either longer or shorter and is "test generated" (Conners, 1995). Another method that has been used involves an "adaptive" variable rate such that the computer program automatically increases or decreases the ISI by 5% depending on the accuracy of the subject's last response (Brumm, 1994; Girardi et al., 1995; Rapoport et al, 1980; Weingartner, Rapoport, Buchsbaum et al., 1980). In those studies that employ an adaptive rate ISI, the ISI is subject driven and the mean ISI is believed to reflect the response latency associated with accuracy of responding (e.g., Girardi et al., 1995; Rapoport et al., 1980; Weingartner et al., 1980). Research implications for event rate indicate that there may be an optimal ISI of approximately two seconds for making more correct responses and fewer false alarms. Sykes, Douglas, Weiss, & Minde (1971) found that both hyperactive and control children made more correct responses and fewer false alarms when the ISI was longer (ISI = 1.5 seconds) than when it was shorter (ISI = 1.0). Chee, Logan, Schacher, Lindsay, & Wachsmuth (1989) manipulated the stimulus onset asynchrony (the length of time from the onset of one stimulus to the onset of the next stimulus). They found that all subjects, regardless of group membership, made fewer correct detections of targets when they were presented at one and four second intervals compared to two second intervals. The children missed the most targets and made the most false alarms when the event rate (ISI) was 4 seconds. Finally, some CPTs are designed such that the subject receives feedback on their performance following either correct or incorrect responses or both (Nuechterlein, 1983; O'Dougherty, Nuechterlein, & Drew, 1984).

Another variable of interest is the consistency or variability of the individual's performance over time. Some CPTs provide information on the standard deviation of the reaction time across blocks as a measure of consistency in responding and ability to sustain attention over time (Riccio et al., 2001). Some researchers use the standard error of the reaction time (Cohan, 1995), while others use the standard deviation of the standard error over time as an indication of consistency (Levin, Wilson, Rose, & McEvoy, 1996; Mahan, 1996). When the CPT includes a systematic variation of the ISI (e.g., Conners, 1992, 1995), an additional indicator of variability is the change in reaction time as a function of the change in the ISI as well as the standard error of this change. The vigilance decrement (Parasuraman, 1984a, 1984b) refers to the decline in performance (i.e., the extent to which the individual's accuracy declines over the course of the task) and may or may not include consideration of the decreased speed in responding over time. Some clinicians focus on comparisons of correct and incorrect responses over differing blocks of time within the same administration to determine if there is a significant vigilance decrement (e.g., Fleming, 1991). Some programs provide comparative information from blocks at the beginning of the task and at the end of the task (e.g., Conners, 1992, 1995).

Many clinicians incorporate signal detection theory (SDT) in generating performance indices (e.g., Keilp, Herrera, Stritzke, & Cornblatt, 1997; Klorman, Brumaghim, Fitzpatrik, & Borgstedt, 1990; Koelega, Brinkman, Hendriks, & Verbaten, 1989; Liu, Hwu, & Chen, 1997). SDT posits that the decision to respond is based on the subject's setting himself or herself a certain criterion for responding. Sensitivity (d' or d-prime) is derived from the mean distribution of responses to both signal and noise such that d' is equivalent to the difference between the sum of the distribution for both signal and noise, and noise alone. Sensitivity (d') is believed to represent the likelihood that the individual will detect the signal or respond to the target when the target is presented or the ability to discriminate targets from nontargets. Sensitivity (d') is believed to be dependent on the intensity of the stimulus and the sensitivity of the individual.

Response style or response bias (Beta) is the other index that is derived based on SDT. Beta is believed to reflect the extent to which the individual is being conservative or impulsive in responding. Response bias is presumed to relate to the strategy used in making the decision to respond and the individual's tendency toward risk taking as well (Riccio et al., 2001). Because of the way in which d' and Beta are computed (Swets, 1964, 1973, 1984), Beta is dependent on sensitivity. Based on signal detection theory, the sensitivity and bias indexes may be more sensitive to differences in performance on CPTs than omission or commission errors (Lam & Beale, 1991). Concerns have been raised by some, however, as to the applicability of signal detection theory to CPT performance (Jerison, 1967; Parasuraman, 1979). These concerns relate to the rate of presentation, target frequency, and stimulus duration. According to Riccio and colleagues (2001), the specifics of the CPT may dictate the appropriateness of using signal detection theory for interpretation of results.

The proceeding discussion of possible score types demonstrates the many methods available for measuring CPT performance. The large number of possibilities raises the question of which variables are most useful. Differences in administration, stimulus presentation, duration, and scoring of the various CPTs complicate score interpretation. Furthermore, few studies have investigated how different score types may impact the interpretation of CPT performance (Riccio et al., 2001). In a recent study, researchers (Epstein et al., 2003) attempted to examine the relations between various CCPT-II variables and phenotypic behaviors so as to better understand the various CCPT-II variables. An epidemiological sample of 817 children were administered the CCPT-II. Diagnostic interviews were conducted with parents to determine ADHD symptom profiles for all children. Children diagnosed with ADHD had more variable RTs, made more errors of commission and omission, and demonstrated poorer perceptual sensitivity than control children. Generalized estimating equations (GEE) and ANCOVAs were conducted to determine specific relationships between the eighteen

DSM-IV ADHD symptoms and 6 CPT parameters. The CCPT-II performance measures demonstrated significant relationships to ADHD symptoms but did not demonstrate symptom domain specificity. Overall performance on *d'* and *β* was highly related to all ADHD symptoms across symptom domains. Furthermore, increased variability in RTs over time was related to most ADHD symptoms. Finally, it appeared that one CCPT-II variable, mean hit RT, was minimally related to ADHD symptoms as a whole, but did demonstrate some specificity in its link with symptoms of hyperactivity (Epstein, et al., 2003).

In a recent study, Axelrod (2002) identified homogenous groups of children with ADHD and children with clinically elevated levels of anxiety. The two groups were compared on SDT measures of *d'* and *β* calculated from performance on the CCPT. Findings suggested that SDT measures were able to differentiate between children with ADHD and children with clinically elevated levels of anxiety when overall scores were compared. The two groups were not different with respect to performance over time on the SDT measures.

There are many differences between the CCPT-II and traditional CPTs. For example, the difference between the CCPT-II and the GDS raises several issues in interpreting scores. On the CCPT-II, the individual is instructed to press the space bar after every letter presented on the computer screen except for the "X". On the GDS, the individual is instructed to press the space bar only when the number "9" follows the number "1". The CCPT-II includes a higher proportion of "targets" to which the participant must respond by pressing a key (90% of stimuli), as well as a relatively high proportion of infrequently appearing “critical signals” (10% of stimuli) for which the participant maintains a vigil. According to Corkum and Siegel (1993), the percentage of targets is an important factor in determining a subject’s decision criteria. If the target is infrequent, the subject may choose to adopt a lax criterion and risk periods of inattention since the cost will be low (e.g., the number of targets that the subject will miss will be small). If the target is frequent, then the subject needs more continuous monitoring and the cost for inattention will be high (e.g., more targets will be missed).

When interpreting scores, the “omission errors” are defined on both tasks as ‘failure to press the key at the appropriate time.’ On the GDS, omission errors (failure to press the key when the “9” appears) suggest an inability to detect the critical signal, presumably because of a lapse of sustained attention. In contrast, on the CCPT-II, omission errors (failure to press the key when any letter other than “X” appears) indicate a failure to maintain an ongoing, repetitive motor response. Similarly, “commission errors” on both tasks are defined as “pressing the key at an incorrect time.” On the GDS, commission errors (pressing the key when “9” did not appear) suggest either impulsivity or sensory discrimination errors. In contrast, commission errors on the CCPT-II (pressing the key when “X” appears) suggest an inability to inhibit a habitual response (Ballard, 2001).

Because of differences in the conceptual basis for “omission” and “commission” errors, difficulties also arise for the interpretation of measures derived from omission and commission rates, such as *d’* (perceptual sensitivity) and β (response criterion). Hit reaction time measures are affected by the definition of “target stimulus.” On the GDS, hit reaction time is the latency between the onset of target stimuli ("9") and pressing the key. In contrast, hit reaction time on the CCPT-II is the latency between onset of any letter except "X" and pressing the key (Ballard, 2001). For the CCPT-II, it is not possible to compute an average reaction time for correct responses to the critical signal of "X" (not pressing the key).

Other differences between the CCPT-II and traditional CPTs are noteworthy. Task variables including type of stimulus discrimination, stimulus exposure duration, and ISI need to be studied in order to determine their affect on vigilance performance. On the CCPT-II, the subject responds every time a letter other than an X appears on the screen. On traditional successive discrimination CPT tasks such as the GDS, the subject responds to a designated target after the occurrence of a specific warning signal (e.g., subject responds to the 9 only if it was preceded by the number 1). Schacher, Logan, Wachsmuth, and Chajezyk (1988) hypothesized that differences in test versions may differentially affect attention. For example, in a successive discrimination task such as the GDS, the warning target can alert the subject to the possibility of an occurrence of the target stimulus. This could help mobilize the subject's attention and thereby allow the subject to process the target more quickly and accurately. On the other hand, successive discrimination tasks require a larger memory component than the CCPT-II task.

Stimulus exposure duration (display time) has been found to affect the subject's performance on vigilance tasks. In Corkum and Siegel's (1993) review of 13 studies that examined the differences between children with ADHD and normal children's performance on CPT tests, 11 reported stimulus exposure durations; these ranged from 40 milliseconds to 500 milliseconds. In a review of the literature on the effects of display time, Davies and Parasuraman (1981) concluded that increases in display time may reduce vigilance decrements while decreases in display time may increase the vigilance decrements and reduce overall efficiency.

The variability of interstimulus interval (ISI) differs for the CCPT-II and traditional CPTs. Traditional CPTs usually maintain a constant ISI across the task. For the CCPT-II, each block of trails is divided into three randomly ordered sub-blocks of one second, two second, and four second ISIs (Ballard, 2001). Equal numbers of critical signals ("X") and background events (other letters) are presented within each sub-block. Like most CPTs, ISI is confounded with task duration, so that sub-blocks with longer ISIs last for a greater proportion of total block time. Averaged across stimulus events, the mean ISI per block is about 2.3 seconds. Averaged across time, however, the participant spends more time per block observing a slower presentation rate. In summary, the CCPT-II produces a relatively slower event rate compared to traditional CPTs. In a recent study comparing the CCPT-II and a traditional CPT on normal adults, Ballard (2001) found significant differences between tasks in omission and commission error rates, reaction time, reaction time variability, and response to critical signals. In addition, traditional CPTs produced time-related performance decrements, but the CCPT-II produced improvement across initial blocks of trials. Ballard (2001) indicated that CCPT-II may measure "executive control" rather than sustained attention, and therefore may represent functions of different brain systems. Recent models of brain systems of attention suggest that the ability to inhibit habitual responses may depend on the anterior cingulate and its connections (Parasuraman, Warm, & See, 1998; Posner & Peterson, 1990), while the ability to sustain attention as measured by traditional CPTs may depend primarily on connections between brainstem nuclei and the right frontal cortex (Mirsky et al., 1991; Parsuraman et al., 1998).

Research Questions

A review of this literature regarding effects of different CPT versions, score interpretations, and theories of ADHD has led to the following research questions and hypotheses.

1) Is there a significant difference in the CCPT-II scores for children with ADHD and children with No Diagnosis? It is hypothesized that there will be a significant group difference in CCPT-II scores for children with ADHD and No Diagnosis. *Rationale:* As a group, children with ADHD have more difficulties on CPT measures relative to normal controls.

1A) If there is a significant difference, which CCPT-II score(s) best describe the differences in children with ADHD from children with No Diagnosis? It is hypothesized that the omission errors, commission errors, sensitivity (d') and hit reaction time CCPT-II variables will best describe the differences in children with ADHD from children with No Diagnoses. *Rationale:* There is limited research on individual CCPT-II scores with ADHD and normal controls; however, Epstein and colleagues (2003) found that children with ADHD had more variable RTs, made more errors of commission and omission, and demonstrated poorer perceptual sensitivity than control children.

1B) How well do the CCPT-II variables differentiate children with ADHD from those with No Diagnosis? When determining how well the CCPT-II variables differentiate children with ADHD from those with No Diagnosis, it is hypothesized that the omission errors, commission errors, sensitivity (d'), and hit reaction time variables will do a better job differentiating groups than the other variables. *Rationale:* Epstein and colleagues (2003) found that children with ADHD had more variable RTs, made more errors of commission and omission, and demonstrated poorer perceptual sensitivity than control children.

1C) Are CCPT-II score profiles obtained from the overall group of children able to separate the ADHD group from the No Diagnosis group? It is hypothesized that CCPT-II score profiles obtained from the overall group of children will be able to separate the ADHD group from the No Diagnosis group. *Rationale:* As a group, children with ADHD have more difficulties on CPT measures relative to normal controls.

1D) If two groups emerge from the cluster analysis, to what extent do the CCPT-II profiles correctly classify children with ADHD and No Diagnosis?

2) Is there a significant difference in the GDS scores for children with ADHD and children with No Diagnosis? It is hypothesized that there will be a significant group difference in GDS scores for children with ADHD and No Diagnosis.

Rationale: As a group, children with ADHD have more difficulties on CPT measures relative to normal controls.

2A) If there is a significant difference, which GDS score(s) (delay, vigilance, distractibility) best describe differences in children with ADHD from children with No Diagnosis? Because there is limited research using the 11 GDS scores with children with ADHD and normal controls, it is difficult to predict which GDS scores may best describe differences between groups.

2B) How well do the GDS variables differentiate children with ADHD from those with No Diagnosis? Based on a paucity of research with the 11 GDS variables, it is difficult to predict which GDS scores may best describe differences between the ADHD and No Diagnoses groups.

2C) Are GDS score profiles obtained from the overall group of children able to separate the ADHD group from the No Diagnosis group? It is hypothesized that GDS score profiles obtained from the overall group of children will be able to separate the ADHD group from the No Diagnosis group. *Rationale:* As a group, children with ADHD have more difficulties on CPT measures relative to normal controls.

2D) If two groups emerge from the cluster analysis, to what extent do the GDS profiles correctly classify children with ADHD and No Diagnosis?

In the next chapter, the methods that were used to address these questions are presented. This includes a review of the measures, participants, and statistical procedures used in the study. Results and discussion then are presented in chapters IV and V, respectively.

CHAPTER III

METHOD

This study compared two variations of continuous performance tests, the Conners' Continuous Performance Test-second edition and the Gordon Diagnostic System with children and adolescents with ADHD and normal controls. This chapter describes the participants, procedures, instruments, and materials used in the study. The research questions and the data analyses completed in an attempt to answer these questions are presented.

Participants

Participants were children and adolescents ages 9-17 who were consecutive referrals to a Memory, Attention, and Planning study (MAPS). Participants were recruited through the use of announcements distributed in the local community to physicians, local support groups for individuals with Attention Deficit Hyperactivity Disorder, a community-based counseling center, on local bulletin boards, and in the local newspaper. The announcements indicated that the research study focused on memory, attention, and planning/ problem-solving. Participation was voluntary with obtained consent from the parent of each child or adolescent and written assent of the child or adolescent. Parents received a comprehensive report of the results, along with recommendations if appropriate, following completion of the evaluation. For inclusion in this study, participants obtained an IQ$\geq$80 and spoke and read English. Prior diagnosis of schizophrenia or a history of severe head injury served as exclusionary criteria. Following the evaluations, research participants were assigned to one of the three groups: No Diagnosis, ADHD, or Other Clinical Diagnosis. For the purpose of this study, only participants included in the No Diagnosis or ADHD Diagnosis groups were included.

The participants in this study included 67 children and adolescents with a mean age of 11.97 (*SD*=2.38); age ranged from 9.00 years to 17.92 years. Of these, 53 (79.1%) were white non-Hispanic, 6 (9.0%) were African American, 7 (10.4%) were Hispanic English-speaking, and 1 (1.5%) was Asian American; ethnicity was based on parent report. For the total sample, 47 (70.1%) were male and 20 (29.9%) were female. Based on diagnostic considerations, 41 (61.2%) met criteria for ADHD and 26 (38.8%) did not meet criteria for any disorder. Of the children and adolescents diagnosed with ADHD, 26 (66.7%) had a previous diagnosis of ADHD and 21 (51.2%) had a current prescription for stimulant medication (e.g., Ritalin, Concerta, Aderall). Twenty-two (53.7%) of the children and adolescents in the ADHD group were diagnosed with an additional disorder (e.g., learning disability, depression, anxiety). Ten children and adolescents in the ADHD group (24.4%) had received special education support.

The sample was made up of elementary, middle school, and high school age participants with a mean education level as a sixth grade student (6.06 years: *SD*= 2.48). Information on parent education indicates that despite a range of 11 to 19, for the majority of the participants, at least one parent had completed some college (Mean Years= 15.02; *SD*= 2.35). The No Diagnosis and ADHD groups differed significantly with regard to Full Scale IQ [$F(1,66)= 5.68$, $p=0.02$] and Total Achievement from the Woodcock-Johnson Test of Achievement-Third Edition [$F(1,66)= 10.28$, $p= 0.002$].

With regard to Full Scale IQ, the No Diagnosis group (Mean Full Scale IQ of 108.69; *SD*= 14.86) performed significantly better than the ADHD group (Mean Full Scale IQ of 101.00; *SD*= 11.46). With regard to Total Achievement, the No Diagnosis group (Mean Total Achievement of 107.08, SD= 15.94) performed significantly better than the ADHD group (Mean Total Achievement of 95.83, *SD*=12.62). Groups did not differ on age, grade, or educational level of parent. Demographics and descriptive data on specific variables by group are provided in Table 1.

Procedures

Children and adolescents participated in a comprehensive assessment including assessment of cognition, achievement, language, memory, executive function, attention, behavioral, and emotional status as part of a larger study. Advanced doctoral students supervised by a licensed psychologist, or a licensed psychologist administered all measures consistent with standardization. Measures were administered in a random order; however, continuous performance tests were counterbalanced to control for order effects. Test sessions varied in length based on the individual being assessed. For those participants with a previous diagnosis of ADHD who were currently taking stimulant medication (n = 21), participants were asked to consult their physician regarding the possibility of omitting medication on the days they were evaluated. To facilitate this, evaluations were scheduled when children were on a medication ‘holiday’ (e.g., Saturdays, vacations). For individuals taking other types of medication (n = 6), they continued on the medication as prescribed without interruption.

Table 1

Demographics Among Groups of Children with ADHD and No Diagnosis

	Groups of Children	
	ADHD (n=41)	**No Diagnosis** (n=26)
Male	85.4%	46.2%
Female	14.6%	53.8%
White	80.5%	76.9%
African-American	9.8%	7.7%
Hispanic	9.8%	11.5%
Asian	0%	3.8%
Taking Psychotropic Medication	51.2%	0%
Previous Diagnosis of ADHD	66.7%	0%
Ever Received Special Education	24.4%	3.8%
Currently Receiving Special Education	10.0%	3.8%
	Mean (SD)	Mean (SD)
Full Scale IQ	101.00 (11.46)	108.69 (14.86)
Total Achievement	95.83 (12.62)	107.08 (15.94)
Education Level of Parents	14.78 (2.24)	15.60 (2.47)

Diagnostic Decision-making

At least two individuals (advanced doctoral student and at least one licensed psychologist) independently reviewed the participant history using the Behavior Assessment System for Children-Structured Developmental History (BASC-SDH), and the Diagnostic Interview for Children and Adolescents (DICA-IV). Parent and teacher rating scales were reviewed including the Behavior Assessment System for Children-Parent Rating Scale (BASC-PRS), Behavior Assessment System for Children-Teacher Rating Scales (BASC-TRS), Conners' Parent Rating Scale-Revised (Short form), and the Conners' Teacher Rating Scale-Revised (Short form). Self-report forms were reviewed including the Behavior Assessment System for Children Self-Report of Personality Scale (BASC-SRP), State-Trait Anxiety Inventory for Children (STAIC), the Children's Depression Inventory (CDI), and the Substance Abuse Subtle Screening Inventory-Third Edition (SASSI-3) when age appropriate.

Finally, intelligence test (Wechsler Intelligence Scale for Children-Third Edition; WISC-III,) vocabulary tests (Peabody Picture Vocabulary Test; PPVT-III, ExpressiveVocabulary Test; EVT) and achievement test (Woodcock Johnson Tests of Achievement-Third Edition; WJ-III) results were reviewed. The individuals providing diagnostic considerations were blind to the results of the dependent variables (e.g., GDS, CCPT-II). Any disagreements were resolved through discussion and further review of the pertinent information and diagnostic criteria from the DSM-IV (4^{th} edition; American Psychiatric Association, 1994). Other dependent measures of executive functioning were included as part of a larger study and will not be discussed here. Cohen's kappa for the sample was 0.86 and proportion of agreement observed was 0.9.

Instrumentation

Conners' Continuous Performance Test- Second Edition

On the Conners' Continuous Performance Test-II (CCPT-II), the stimuli are capital letters presented sequentially on a computer screen. The "target stimuli" for which the individual must press the computer key includes all the letters *except* X. The duration of the task is approximately 14 minutes. The examiner remains in the room while the individual completes the test.

Thirteen indices are generated from the CCPT-II for use in interpretation (Conners, 1992, 1995). The most commonly reported variables include correct hits, omission errors, commission errors, and mean reaction time. In addition, the CCPT-II provides the variables from SDT of sensitivity (d') and response bias (Beta). Several measures relate specifically to the consistency or variability of the individual's performance across blocks or subblocks. These include the reaction time standard error; the standard deviation of the standard error for each subblock; the change in response time during testing as measured by the slope; the change in response time standard errors during testing, as measured by the slope; the reaction time at the subblock changes for the 1000, 2000, and 4000 ms ISIs as measured by the slope: and the standard error of the subblock changes over the 1000, 2000, and 4000 ms ISI across the six blocks as measured by the slope. Finally, the CCPT-II provides an overall index for the individual's performance which is a weighed score derived from all of the other indices (Riccio et al., 2001).

The CCPT-II indices are presented in raw score, percentile, and T-score formats. The mean T-score for the comparison group is 50 (SD=10). The correct hits, reaction time, d', and Beta index scores have been transformed so that higher T-scores suggest that attention problems exist. T-scores of 60 or above and percentiles of 90 or above on two or more indices indicate potential attention problems (Conners, 1992, 1995). According to Riccio and colleagues (2001), this interpretation by Conners makes certain assumptions about the base rates of the disorders underling problematic CPT performance (i.e., they occur in 10% of the population).

The normative sample included 1920 individuals from the general population who were 6 years of age and older. Approximately 30 sites from geographically diverse locations were involved in a nation-wide standardization project. The multi-site, nonclinical youth data came from several elementary, middle, and high schools. Data from the CCPT-II norms were used to assess the applicability of the CCPT-II to some minority groups. The African American group made slightly fewer commission errors than the general population and showed slightly better discriminatory power as measured by *d* prime. Overall, the general population norms were reportedly highly applicable to minority groups (Conners, 1995).

For the epidemiological study, CCPT-II data was collected in the homes of children aged 9 to 18 (Conners, 1995). Originally, 17,117 children from four counties were randomly selected from a database by the North Carolina Department of Education.

This number was reduced to 5400 children by excluding children when a sibling had already been interviewed, when the family had moved somewhere outside of the four counties, when children were developmentally delayed, when household members spoke primarily a language other than English, when parents could not be contacted, or when parents refused to participate in the study. Children were then classified according to their scores on the Child Behavior Checklist (CBCL; Achenbach, 1991). Children were placed into 18 cohort groups. In 14 of the cohort groups, children were selected so that there was a proportionate representation of children within each CBCL score range placed in the cohort sample. According to the CCPT-II manual, the 14 cohorts contained the range and frequency of behavioral symptomatology expected in the general population. For cohorts 1 to 4, all the children selected had high CBCL scores (greater than 20). The process of selecting children for the cohort groups, in combination with some research attrition, resulted in the availability of 812 CPT respondents. The general population norms are likely slightly elevated as a result of the structure of the epidemiological data. As a result, a fairly low cutoff criteria (approximately 60 or higher) should be taken as a flag for poor performance on the CCPT-II (Conners, 1995).

The clinical data for the CCPT-II includes data from those identified with ADHD, neurological impairments, anxiety disorders, depression, conduct disorder, and oppositional defiant disorder. According to the CCPT-II manual (Conners, 1995), sufficient data were obtained to constitute norms for the ADHD group and for the adult neurologically impaired group. The ADHD clinical data came from a variety of the 30 data collection sites. All of the sites followed the criteria specified in the DSM-IV (APA, 1994). A battery of information was used to make the classification including rating scales, performance based tasks, interview findings, and behavioral and historical information.

Gordon Diagnostic System

One of the most widely used procedures is the Gordon Diagnostic Systems (GDS: Gordon, 1983). The GDS is a microprocessor-based portable unit that allows for the administration of multiple tasks. The internal microprocessor generates the tasks and records quantitative features of a child's performance both for the entire session as well as for the individual time blocks so that the patterns of a child's performance across the session can be analyzed (Gordon, 1986). Each task takes approximately nine minutes to complete. The examiner remains in the room when the individual is completing the test.

The Delay Task requires the child to inhibit responding in order to earn points. The child is instructed to press the button, wait awhile, and then press the button again. If he or she refrains from responding for at least six seconds, a light flashes and a reward counter records a point. If the child responds before the interval lapses then the timer resets and no reward points are recorded. The Delay Task is programmed using a differential reinforcement of low rate responding (DRL) six-second schedule, so that the child earns a point each time he or she waits a minimum of six seconds. The Delay Task yields three primary scores: the number of responses, the number of correct responses, and the Efficiency Ratio, which represents the percentage of correct responses (number of points earned divided by total number of responses). According to the GDS manual

(Gordon Systems, Inc., 1991), the efficiency ratio is the ‘best single Delay Task indicator of the level of impulsivity demonstrated by a subject’ (p. 54).

The Vigilance Task requires the child to respond under conditions that make demands for sustained attention. Numbers are presented on a display screen at the rate of one per second. The stimulus number remains for 800-milliseconds with a 200-millisecond delay between stimuli. The child is told to press the button every time a “1” is followed by a “9.” This target pair is presented a total of 45 times during the testing. The GDS records the number of responses, the number of time the child failed to respond to the “1/9” combination (i.e., Errors of Omission), and the number of extraneous button presses (i.e., Errors of Commission).

The Distractibility Task is another version of the GDS that assesses the impact of distraction on a child’s ability to sustain attention. The Distractibility Task is identical to the standard Vigilance Task except that random digits flash at random intervals on the outer positions of the electronic display. The child is still required to press the blue button when a “9” comes right after a “1.” The only difference is that numbers flash on either side of the center (i.e., relevant) digit.

For both the vigilance and distractibility tasks, measures of performance include correct responses, omission and commission errors, and latency (reaction time) for each block as well as the average latency for the entire task. The GDS is not programmed to separate commission errors by type (ie., target-related vs. random errors); however, error-tracking data are available for those who wish to do an error analysis. The GDS manual describes commission errors on the Vigilance Task and Distractibility Task that may be due to problems with inhibition, slow response, a disregard for salient targets, or a target-related error. When an examinee responds to a 1 before the 9 is presented, it may be due to problems with inhibiting a response. When an examinee responds to the digit following right after the 1-9 sequence, it may be due to slow responding. If an examinee makes random errors or responds to a 9 when not immediately preceded by a 1, it may be due to a disregard for the salient target or a target-related error, respectively. Finally, instructions for computing the slope of the reaction time across blocks (block variability) to assess consistency are provided (Gordon Systems, Inc., 1991).

Scoring indices include raw score, percentile, and threshold tables. The threshold tables were derived based on the percentile information with scores divided into three different ranges: abnormal (5^{th} percentile or lower), borderline (6^{th} to 25^{th} percentile), and normal (26^{th} percentile and above). While means and standard deviations are provided for the normative samples, standard errors of the mean are not included in the children's version. (Gordon Systems, Inc., 1991).

The standardization sample for the GDS is comprised of 1300 boys and girls from 4 to 16 years of age (Gordon & Mettleman, 1985). A series of validation studies has shown that the GDS tasks differentiated accurately between hyperactive and nonhyperactive children from both outpatient and day treatment setting (Gordon, 1979; McClure & Gordon, 1984). In a study by Gordon and McClure (1983) using a sample of school-referred children, the GDS distinguished children with ADD from those classified as reading disabled, overanxious, and normal.

Data Analyses

Several steps were taken to prepare the CCPT-II and GDS data for analyses. While the CCPT-II indices were recorded in t-score format, the GDS data were recorded in raw score format. In order to make comparisons between the measures, the GDS raw scores were transformed to t-scores. The GDS raw data of the two 17-year olds included in the present study was transformed to t-scores using means and standard deviations from the 12-16 year old age group normative data. To alleviate problems with skewness and kurtosis, the CCPT-II and GDS data was trimmed to four standard deviations. Because problems with kurtosis remained evident, a logarithmic data transformation was performed on both measures. Kline (1998) indicated that logarithmic data transformation uses a mathematical operation to convert original scores to new scores that are more normally distributed.

This chapter has detailed the methodology used for this study. In the next chapter, the results of these analyses will be presented. The final chapter will include a discussion of the findings and limitations of the study, as well as implications for further research and practice.

CHAPTER IV

RESULTS

Today, there are numerous versions of the CPT used in clinical and research settings. The number of CPTs in use and the number of differences across CPTs suggest that although CPTs may constitute a similar group of tasks with a common paradigm, they are very different in the parameters they measure (Conners, 1992, 1995). The current study explored the effects of two different CPT versions, the CCPT-II and the GDS, with children and adolescents exhibiting ADHD and normal controls. MANCOVAs were performed to determine whether there was a significant difference in the CCPT-II or GDS scores for children with ADHD and children with No Diagnosis. In order to determine how well the individual CCPT-II and GDS variables differentiated children with ADHD from those with No Diagnosis, ROC curves were calculated. To examine whether the CCPT-II or GDS scores profiles obtained from the overall group of children were able to separate the children into an ADHD group and a No Diagnosis group, cluster analysis was utilized. Chi-square analyses were calculated to determine to what extent the children's performance on the CCPT-II and GDS was able to predict ADHD and No Diagnosis group membership. For each of these analyses an alpha level of .05 was used to indicate statistical significance. Results are organized based on the five research questions for the CCPT-II and GDS.

Research Questions

To learn more about the effects of different CPT versions as well as the numerous scoring indices, two very different CPTs, the CCPT-II and the GDS, were compared on a population of children and adolescents with ADHD and No Diagnosis as part of a larger study. For research questions 1 through 1D, the following CCPT-II variables were included in the analyses: the clinical index, omission errors t-score, commission errors, hit reaction time, hit reaction time standard error, variability, sensitivity (d'), response style (beta), perseveration, hit rate block, hit standard error of block, hit rate ISI, and hit standard error ISI. For research questions 2 through 2D, the following GDS variables were included in analyses: delay total ER, delay ER block variability, delay ER slope, delay total responses, delay correct, vigilance total commissions, vigilance commission block variability, vigilance correct, distractibility total commissions, distractibility commission block variability, and distractibility correct. The following research questions were addressed:

1. Is there a significant difference in the CCPT-II scores for children with ADHD and children with No Diagnosis?

1A. If there is a significant difference, which CCPT-II score(s) best describe the differences in children with ADHD from children with No Diagnosis?

1B. How well do the CCPT-II variables differentiate children with ADHD from those with No Diagnosis?

1C. Are CCPT-II score profiles obtained from the overall group of children able to separate the ADHD group from the No Diagnosis group?

1D. If two groups emerge from the cluster analysis, to what extent do the CCPT-II profiles correctly classify children with ADHD and No Diagnosis?

2. Is there a significant difference in the GDS scores for children with ADHD and children with No Diagnosis?

2A. If there is a significant difference, which GDS score(s) (delay, vigilance, distractibility) best describe differences in children with ADHD from children with No Diagnosis?

2B. How well do the GDS variables differentiate children with ADHD from those with No Diagnosis?

2C. Are GDS score profiles obtained from the overall group of children able to separate the ADHD group from the No Diagnosis group?

2D. If two groups emerge from the cluster analysis, to what extent do the GDS profiles correctly classify children with ADHD and No Diagnosis?

For the CCPT-II, research question 1 asks: Is there a difference in the CCPT-II scores for children with ADHD and children with No Diagnosis? To answer this question, a multiple analysis of covariance (MANCOVA) was performed to determine whether there was a significant group difference on the CCPT-II for children and adolescents with ADHD as compared to those with No Diagnosis. The analysis was conducted covarying for Full Scale IQ and Total Achievement. There was not a statistically significant group difference on the CCPT-II for children with ADHD as compared to those with No Diagnosis using Wilks' Lambda [$F(2, 66) = 1.74$, $p = .08$]. This lack of group difference indicated that children and adolescents with ADHD and No Diagnosis performed similarly on the CCPT-II. The normalized means and standard deviations for children and adolescents with ADHD and No Diagnosis on CCPT-II variables are presented in Table 2.

Research question 1A asks: If there was a significant difference, which CCPT-II score(s) best describe differences in children with ADHD from children with No Diagnosis? Because there was not a significant group difference for children and adolescents with ADHD and No Diagnosis in research question 1, it was not necessary to run ANCOVA analyses to answer research question 1A.

Research question 1B asks: How well does the CCPT-II differentiate children with ADHD from those with No Diagnosis. To answer this question, a Receiver Operating Characteristic curve (or ROC curve) was calculated. The accuracy of the CCPT-II depended on how well the individual variables separated the overall group being tested into the ADHD group and No Diagnosis group. According to Fleiss (1981), the area under the curve is a measure of test accuracy. The area under the curve is the percentage of randomly drawn pairs for which the variable correctly classifies the two participants in the random pair. An ROC curve is a graphical representation of the trade off between the false negative and false positive rates for every possible cut off. The ROC curve is the representation of the tradeoffs between sensitivity and specificity. The closer the area is to 1.0 the better the test, and the closer the area is to 0.5, the less well the test classifies the participants.

Results of the ROC analysis indicated that three of the CCPT-II variables, the clinical index (0.69), omission errors t-score (0.61), and perseveration (0.61) did a

Table 2

Conners' Continuous Performance Test-II Variable Means for Children with ADHD and No Diagnosis

	ADHD (n= 41)		**No Diagnosis** (n=26)		
	mean	SD	mean	SD	F
CCPT-II variables					
clinical index	1.80	0.11	1.71	0.17	0.13
omission errors t-score	1.72	0.08	1.69	0.07	0.57
commission errors	1.68	0.12	1.68	0.12	0.60
hit reaction time	1.71	0.10	1.67	0.10	0.96
hit reaction time standard error	1.74	0.07	1.72	0.08	0.98
variability	1.73	0.07	1.71	0.09	0.76
sensitivity (d')	1.71	0.11	1.71	0.10	0.52
response style (beta)	1.73	0.08	1.72	0.09	0.91
perseveration	1.74	0.09	1.71	0.08	0.29
hit rate block	1.68	0.13	1.71	0.11	0.14
hit standard error of block	1.69	0.10	1.70	0.09	0.31
hit rate ISI	1.72	0.08	1.73	0.07	0.19
hit standard error ISI	1.71	0.07	1.70	0.08	0.64

Note. CCPT-II variable means were normalized using logarithmic data transformation; No differences were significant.

"poor" to "fair" job of separating the overall group into the ADHD and No Diagnosis groups while all other CCPT-II variables "failed" to separate the two groups. The area under the ROC curve for the CCPT-II variables that failed to separate the groups were as follows: commission errors (0.54); hit reaction time (0.56); hit reaction time standard error (0.57); variability (0.58); sensitivity or d' (0.50); response style or beta (0.52); hit rate block (0.46); hit standard error of block (0.52); hit rate ISI (0.46); and hit standard error ISI (0.53). The ROC area under the curve for each CCPT-II variable are presented in Figures 1-13.

Research question 1C asks: Are CCPT-II score profiles obtained from the overall group of children and adolescents able to separate the ADHD group from the No Diagnosis group?

Although it may not be possible to determine whether a child or adolescent exhibits attention problems by looking at a single variable on a CPT, inspection of individuals' *pattern* of scores on variables of attention may provide information about how their scores cluster together to form profiles. Cluster analysis classification is based upon the placing of variables into more or less homogeneous groups, in a manner such that the relationship between groups is revealed (Aldenderfer & Blashfield, 1984). A cluster analysis was therefore used to classify the heterogeneous pool of participants in the study into a smaller number of homogeneous children based on patterns of scores on variables of attention. If CCPT-II score profiles are able to separate children and adolescents with ADHD from those with No Diagnosis, two groups will emerge from

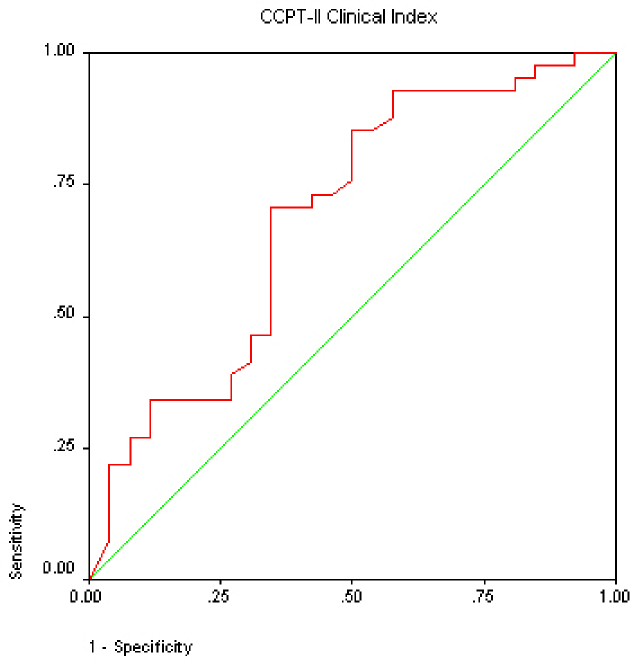

Figure 1. ROC area under the curve for the CCPT-II Clinical Index variable.

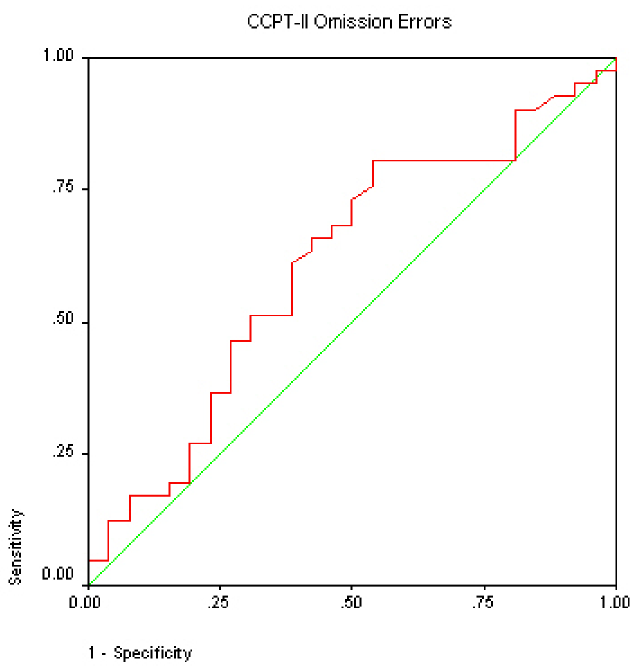

Figure 2. ROC area under the curve for CCPT-II Omission Errors variable.

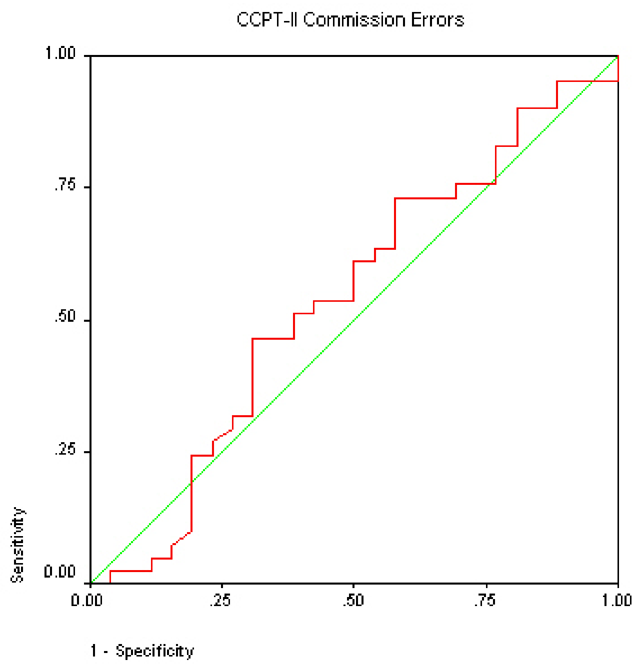

Figure 3. ROC area under the curve for the CCPT-II Commission Errors variable.

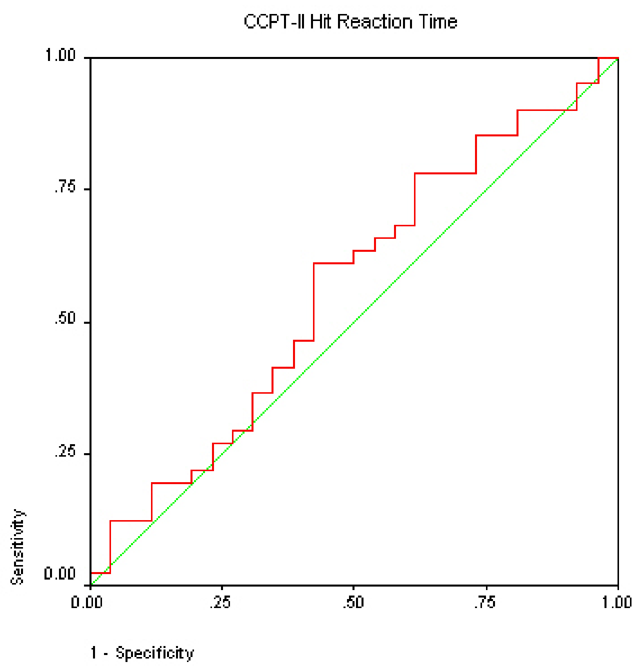

Figure 4. ROC area under the curve for the CCPT-II Hit Reaction Time variable.

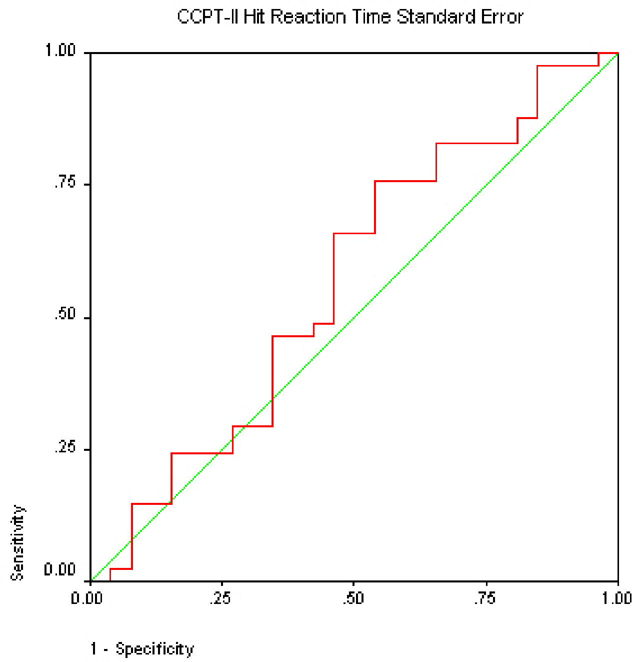

Figure 5. ROC area under the curve for the CCPT-II Hit Reaction Time Standard Error variable.

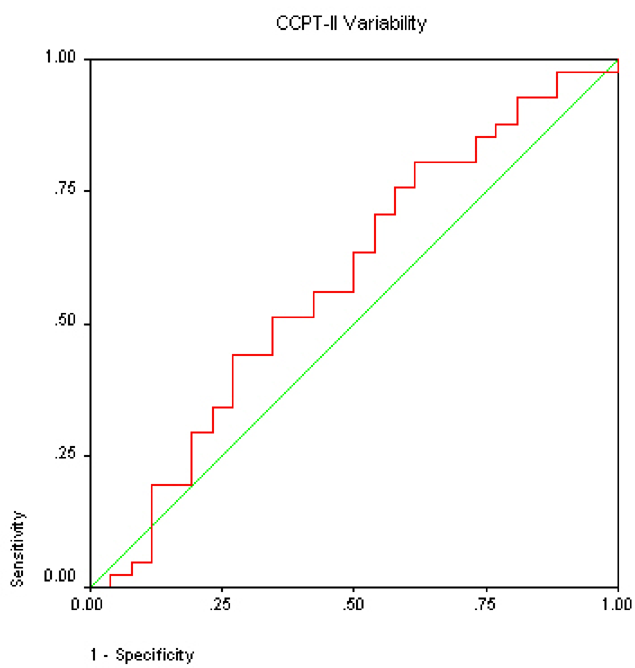

Figure 6. ROC area under the curve for the CCPT-II Variability variable.

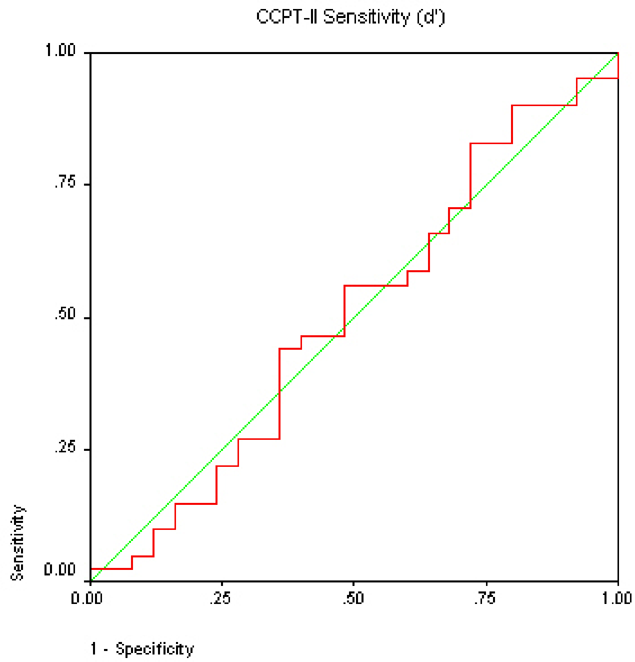

Figure 7. ROC area under the curve for the CCPT-II Sensitivity (d') variable.

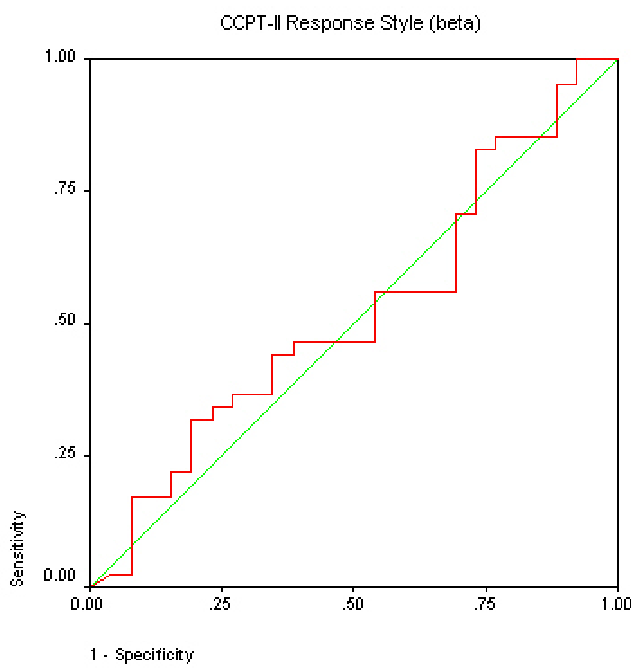

Figure 8. ROC area under the curve for the CCPT-II Response Style (beta) variable.

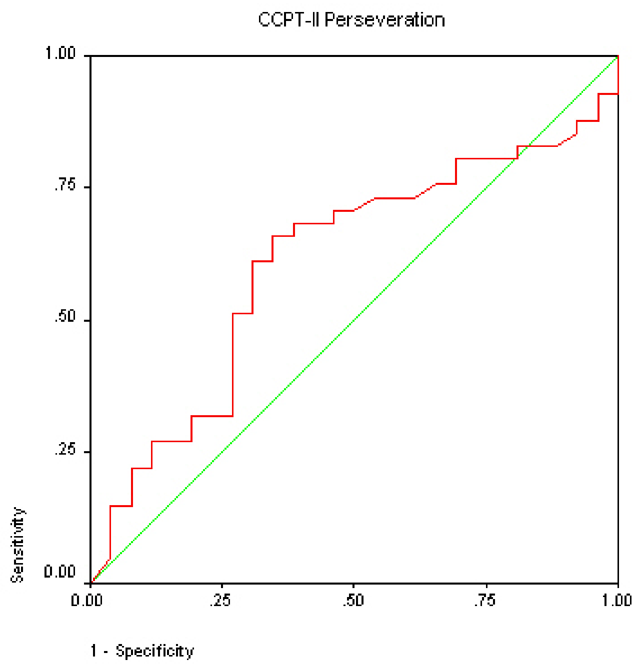

Figure 9. ROC area under the curve for the CCPT-II Perseveration variable.

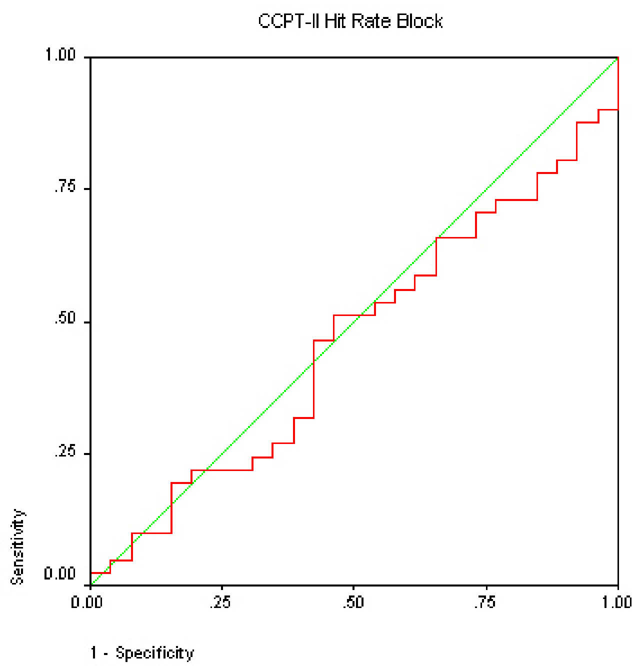

Figure 10. ROC area under the curve for the CCPT-II Hit Rate Block variable.

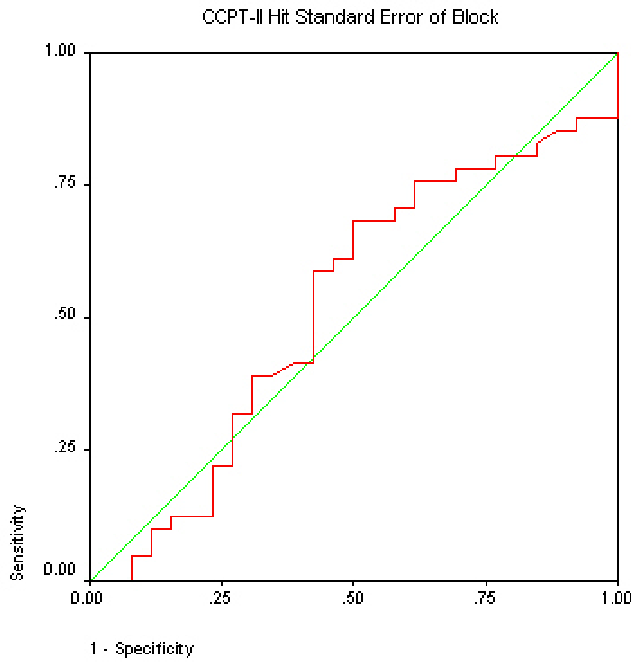

Figure 11. ROC area under the curve for the CCPT-II Hit Standard Error of Block variable.

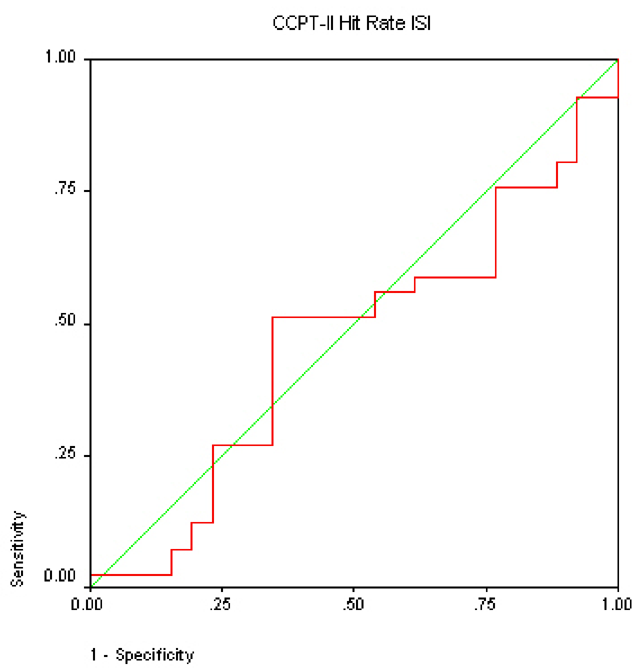

Figure 12. ROC area under the curve for the CCPT-II Hit Rate ISI variable.

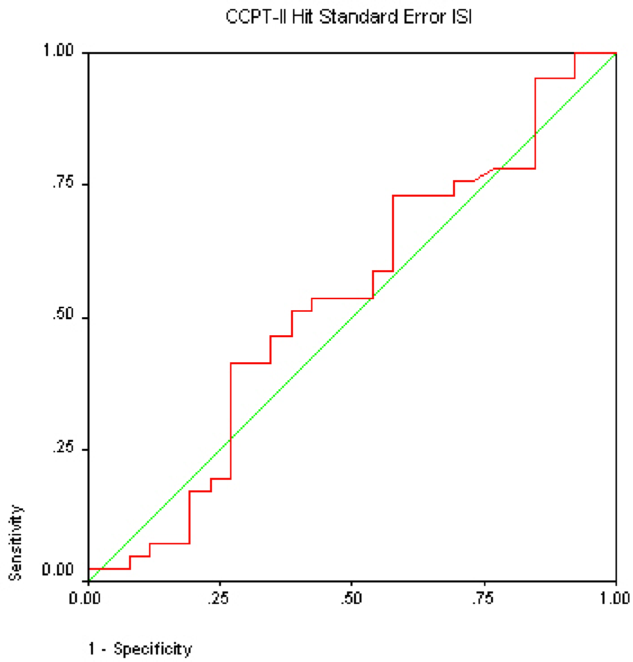

Figure 13. ROC area under the curve for the CCPT-II Hit Standard Error ISI variable.

the cluster. Squared Euclidean distance was selected as the similarity/ dissimilarity measure and Ward's hierarchical agglomeration method was used as the clustering method. Ward's method is variance-based with the group variance assessed to enable clustering. The group which sees the smallest increase in variance with the iterative inclusion of a case will receive the case. Milligan and Cooper (1987) concluded that Ward's method was generally the best hierarchical agglomerative clustering technique for recovering underlying structure. Because there are no completely satisfactory methods available for determining the number of clusters to retain, the decision in this case was based on hierarchical dendogram inspection and profile interpretability. Z scores are basic standard scores and were used in these analyses.

K-means cluster analysis was used to confirm the results of the hierarchical cluster analysis, using cluster means derived from the hierarchical cluster analysis as seeds for K-means groups. K-means cluster analysis is a nonhierarchical clustering method that requires an *a priori* choice of the number of clusters to be generated (Kaufman & Rousseeuw, 1990). It uses distance from the group centroid to decide which group an item will be assigned. The SPSS program starts with K random clusters, and then moves objects between those clusters with the goal to (1) minimize variability within clusters and (2) maximize variability between clusters.

The cluster analysis using CCPT-II variables yielded a 2-cluster solution that accounted for the 66 children and adolescents that had complete CCPT-II data. One child in the No Diagnosis group did not have complete CCPT-II data and was not included in this analysis. Of the 66 children, 41 were in the ADHD group and 25 were in the No Diagnosis group. The two clusters are described below.

(1) <u>Less Attention Problems Cluster.</u> Twenty-three of the 66 children and adolescents were in this cluster. These participants scored lower than average on the 13 CCPT-II variables. Of the 23 children and adolescents in this cluster, 11 were from the ADHD group and 12 were from the No Diagnosis Group. Following the logarithmic data transformation, normalized mean scores on CCPT-II variables ranged from 1.63 to 1.68 for this cluster.

(2) <u>More Attention Problems Cluster.</u> Forty-three of the 66 children and adolescents were in this cluster. These children exhibited more attention problems than children in the Less Attention Problems cluster on 13 CCPT-II variables. Of the 43

participants in this cluster, 30 were from the ADHD group and 13 were from the No Diagnosis group. Following the logarithmic data transformation, means scores on the CCPT-II variables ranged from 1.70 to 1.82 for this cluster. The normalized means scale scores on the 13 CCPT-II variables for the Less Attention Problems and More Attention Problems clusters are presented in Figure 14.

Research question 1D asks: If two groups emerge from the cluster analysis, to what extent do the CCPT-II profiles correctly classify children with ADHD and No Diagnosis? A Chi-square Test of Association was calculated. This technique was used to determine to what extent the children's performance on the CCPT-II was able to predict group membership.

The data used in this analysis included cluster membership according to CCPT-II performance (more attention problems, less attention problems) and group membership (ADHD or No Diagnosis group). Using Chi-Square analysis, an independent relationship between the two cluster solution and ADHD and No Diagnosis group membership was found ($\underline{X}^2 = 3.39$, $p = .07$). According to chance, 62% of children with ADHD should be included in each of the two clusters. Forty-eight percent of the children in the Less Attention Problems cluster were from the ADHD group while 70% of children in the More Attention Problems were from the ADHD group.

For the GDS, research question 2 asks: Is there a significant difference in the GDS scores for children with ADHD and children with No Diagnosis? A multiple analysis of covariance (MANCOVA) was computed to determine whether there was a

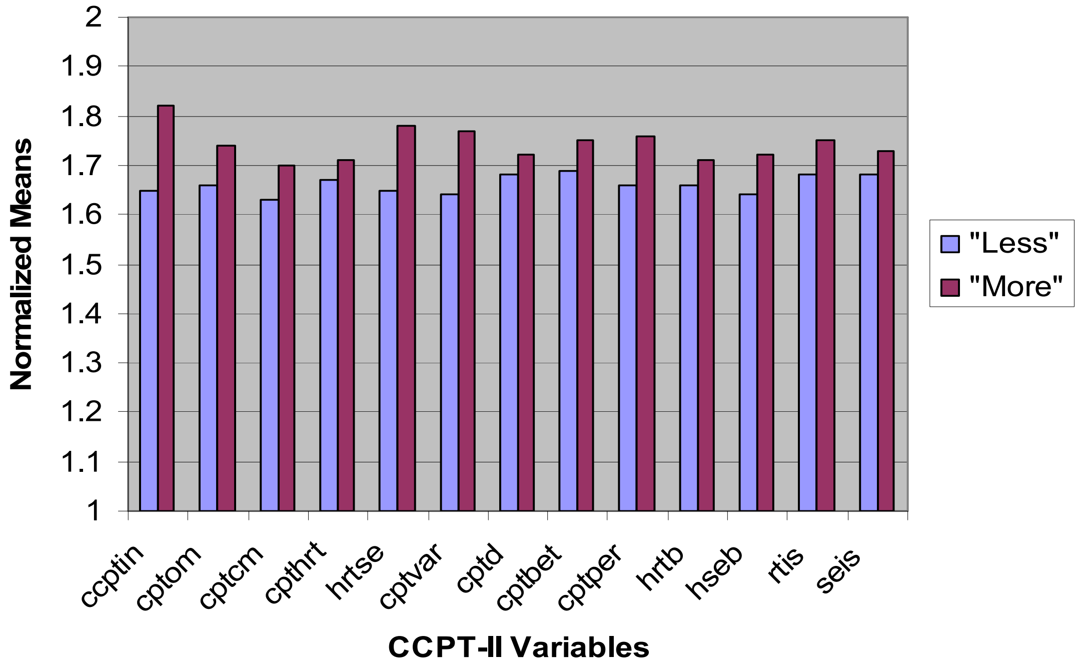

Figure 14. Normalized variable means for CCPT-II Less Attention Problems and More Attention Problems clusters.

Note. ccptin= ccpt clinical index; cptom= omission errors; cptcm= commission errors; cpthrt=hit reaction time; hrtse= hit reaction time standard error; cptvar= variability; cptd= sensitivity (d'); cptbet= response style (beta); cptper= perseveration; hrtb= hit rate block; hseb= hit standard error of block; rtisi= hit rate interstimulus interval; seis= hit standard error of interstimulus interval.

significant group difference on the GDS for children and adolescents with ADHD as compared to those with No Diagnosis. The analysis was conducted covarying for Full Scale IQ and Total Achievement. There was not a significant group difference on the GDS for children with ADHD as compared to those with No Diagnosis using Wilks' Lambda [F (2,66) = 1.08, p= 0.39]. This lack of significance indicated that children and adolescents with ADHD did not perform differently on the GDS than children with No

Diagnosis. The normalized means and standard deviations for children and adolescents with ADHD and No Diagnosis on GDS variables are presented in Table 3.

Research question 2A asks: If there is a significant difference, which GDS scores (delay, vigilance, distractibility) best describe differences in children with ADHD from children with No Diagnosis? Because there was not a significant group difference for children and adolescents with ADHD and those with No Diagnosis, it was not necessary to run ANCOVA analyses to answer research question 2A.

Research question 2B asks: How well does the GDS differentiate children with ADHD from those with No Diagnosis? An ROC curve was calculated to determine how well the individual GDS variables separated the overall group being tested into the ADHD group and No Diagnosis group. Results of the ROC analysis indicated that one GDS variable, the vigilance total commissions (0.61), did a “poor” job of separating the overall group into the ADHD and No Diagnosis groups while all other GDS variables “failed” to separate the two groups. The area under the ROC curve for the GDS variables that failed to separate the groups were as follows: delay total ER (0.33); delay ER block variability (0.50); delay ER slope (0.58); delay total responses (0.59); delay correct (0.39); vigilance commission block variability (0.56); vigilance correct (0.49); distractibility total commissions (0.55); distractibility commission block variability (0.53); and distractibility correct (0.52). The ROC area under the curve for GDS variables is presented in Figures 15-25.

Table 3

Gordon Diagnostic System Variable Means for Children with ADHD and No Diagnosis

	ADHD (n= 41)		**No Diagnosis** (n=26)		
	mean	SD	mean	SD	F
Delay task					
Total ER	1.61	0.17	1.69	0.09	0.08
ER block variability	1.73	0.11	1.73	0.13	0.87
ER slope	1.70	0.16	1.67	0.09	0.49
Total responses	1.72	0.10	1.66	0.16	0.03
Correct	1.67	0.09	1.68	0.16	0.50
Vigilance task					
Total commissions	1.78	0.11	1.76	0.12	0.40
Commission block variability	1.76	0.11	1.76	0.12	0.86
Correct	1.57	0.27	1.61	0.23	0.93
Distractibility task					
Total commissions	1.79	0.13	1.76	0.13	0.32
Commission block variability	1.76	0.12	1.75	0.12	0.70
Correct	1.64	0.18	1.59	0.22	0.08

Note. GDS variable means were normalized using logarithmic data transformation.

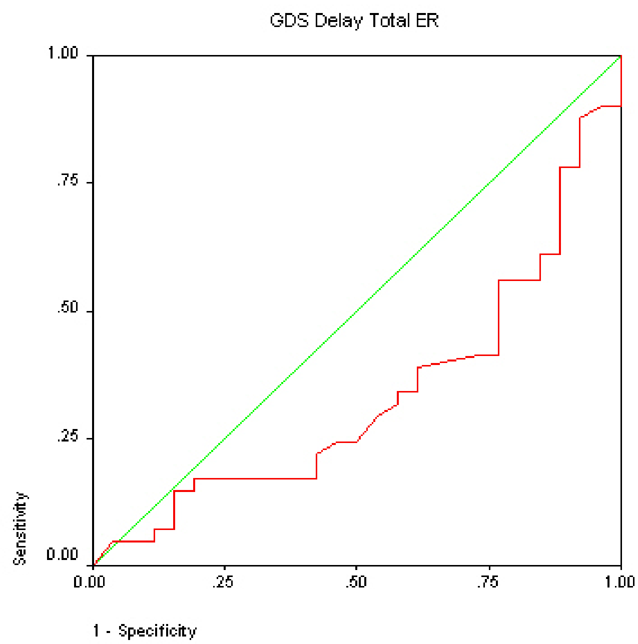

Figure 15. ROC area under the curve for the GDS Delay Total ER variable.

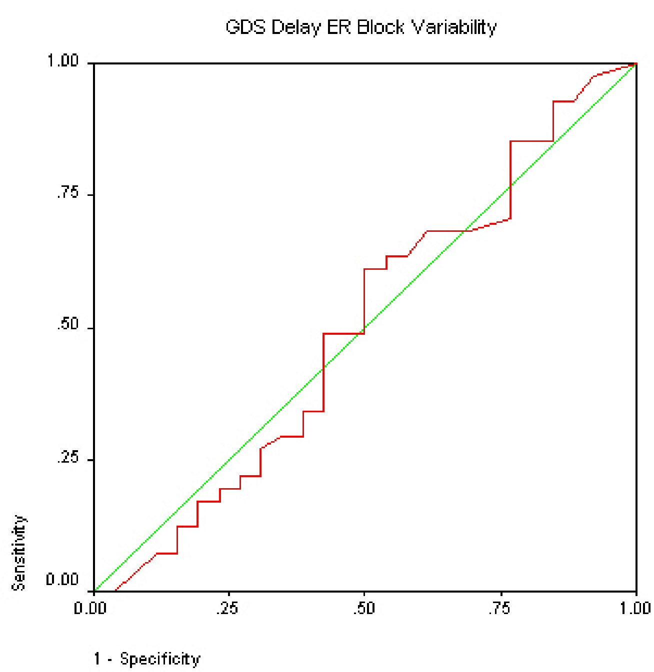

Figure 16. ROC area under the curve for the GDS Delay ER Block Variability variable.

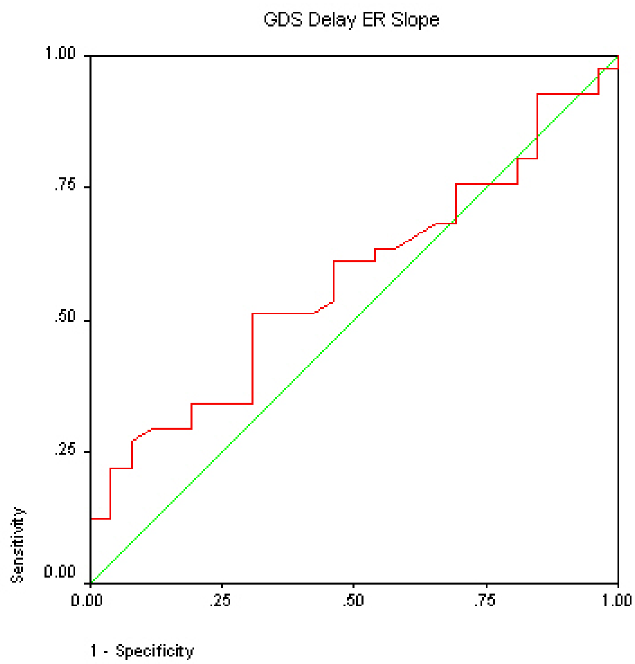

Figure 17. ROC area under the curve for the GDS Delay ER Slope variable.

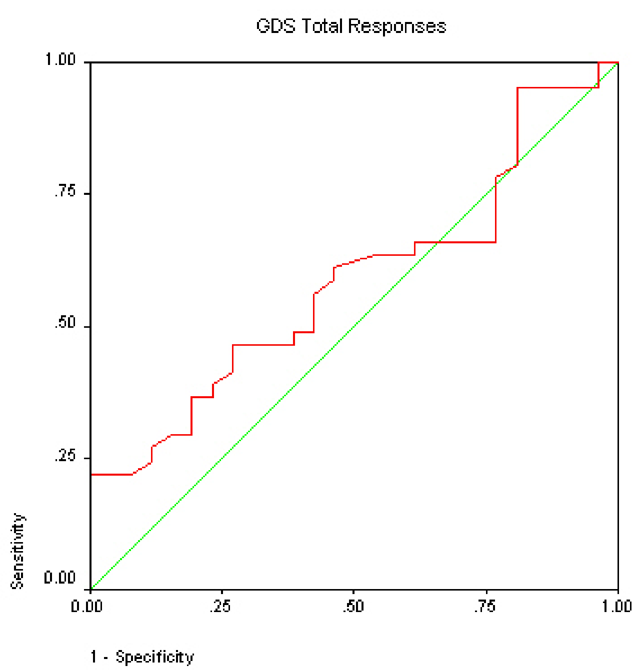

Figure 18. ROC area under the curve for the GDS Total Responses variable.

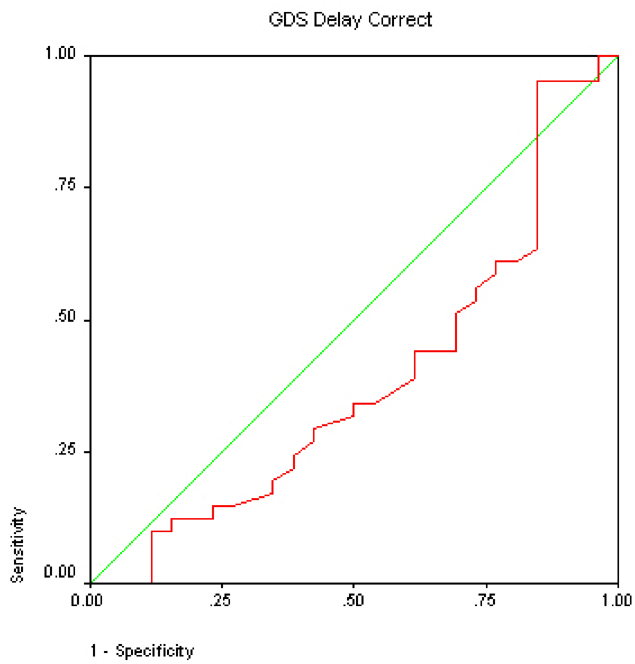

Figure 19. ROC area under the curve for the GDS Delay Correct variable.

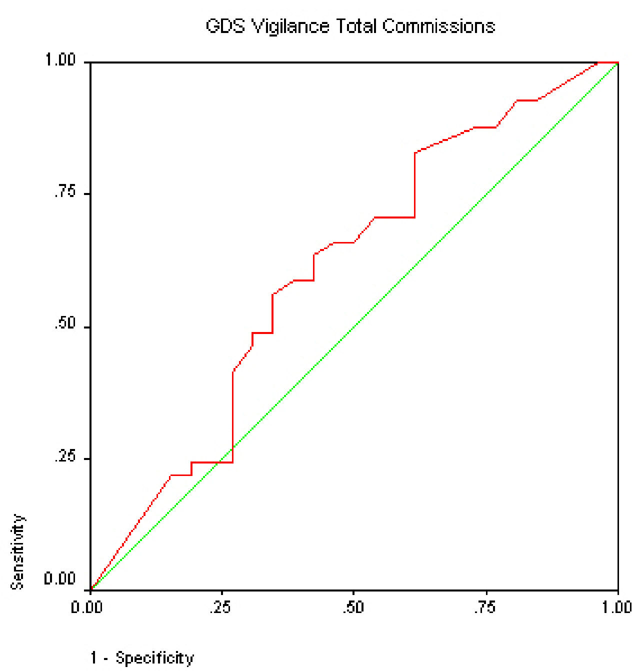

Figure 20. ROC area under the curve for the GDS Vigilance Total Commissions variable.

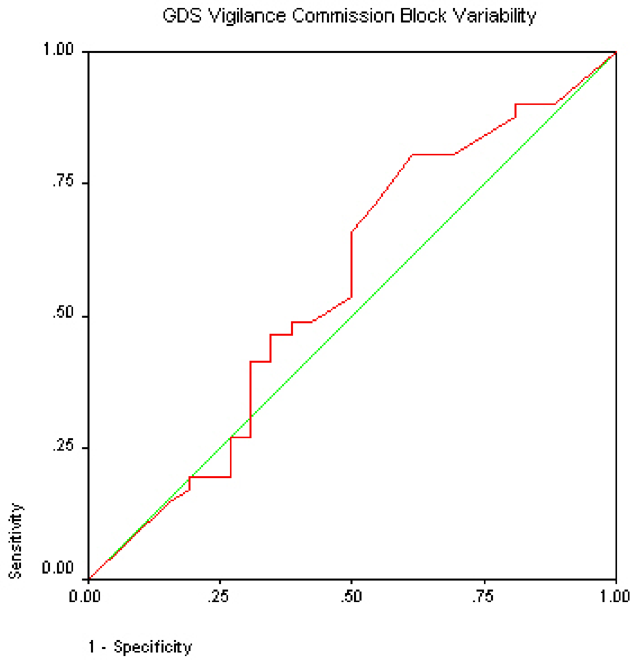

Figure 21. ROC area under the curve for the GDS Vigilance Commission Block Variability variable.

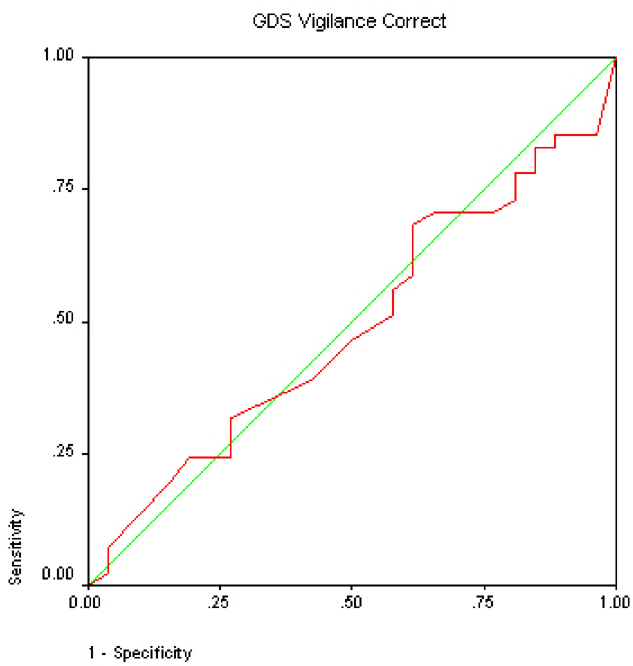

Figure 22. ROC area under the curve for the GDS Vigilance Correct variable.

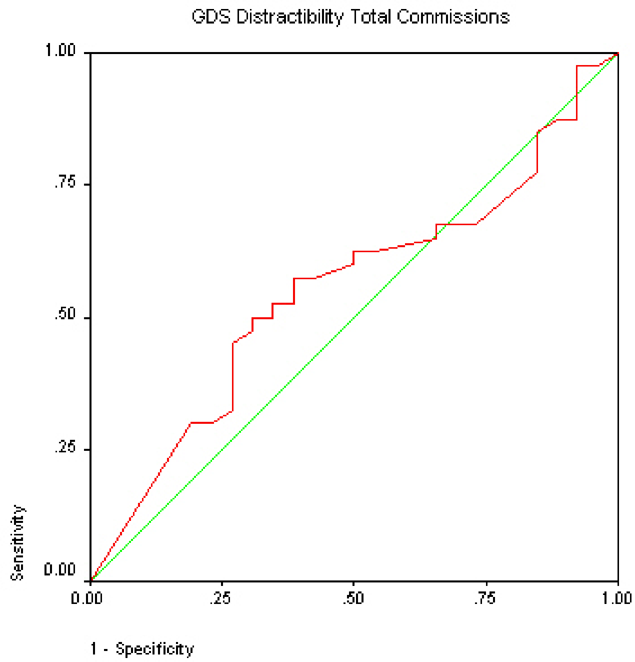

Figure 23. ROC area under the curve for the GDS Distractibility Total Commissions variable.

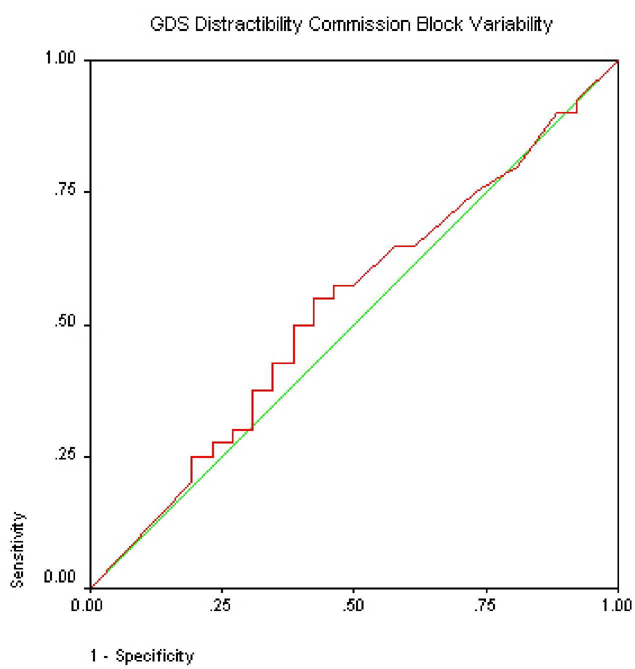

Figure 24. ROC area under the curve for the GDS Distractibility Commission Block Variability variable.

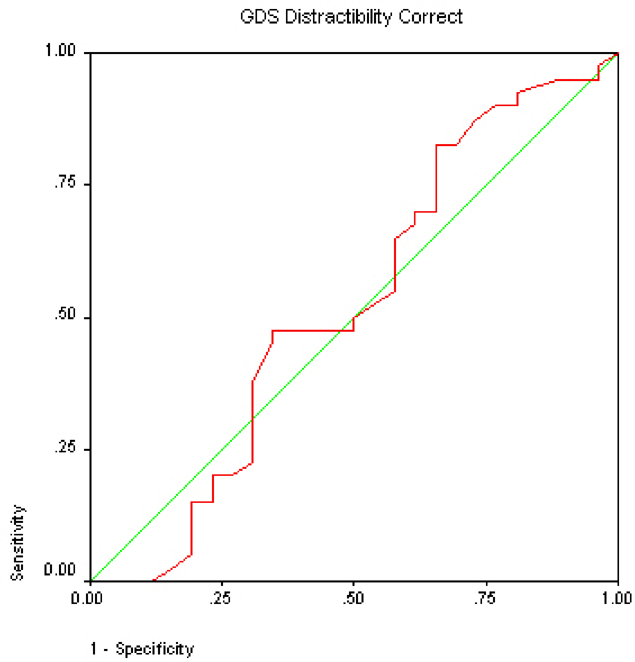

Figure 25. ROC area under the curve for the GDS Distractibility Correct variable.

Research question 2C asks: Are GDS score profiles obtained from the overall group of children and adolescents able to separate the ADHD group from the No Diagnosis group? As with the CCPT-II, cluster analysis was used to group GDS score profiles. The cluster analysis using the GDS variables yielded a 2-cluster solution that accounted for the 66 children and adolescents that had complete GDS data. One child in the ADHD group had incomplete data on the GDS distractibility task and was not included in this analysis. Of the 66 children, 40 were in the ADHD group and 26 were in the No Diagnosis group. While high scores on the delay ER block variability, delay ER slope, delay total responses, vigilance total commissions, vigilance commission block variability, distractibility total commissions, and distractibility commission block variability are indicative of attention problems, low scores on the delay total ER, delay correct, vigilance correct, and distractibility correct variables are indicative of attention problems. The two clusters are described below.

(1) Less Attention Problems Cluster. Fifty-six of the 66 children and adolescents were in this cluster. These participants scored within an average range of performance on the 11 variables. Of the 56 children and adolescents in this cluster, 33 were from the ADHD group and 23 were from the No Diagnosis group. Following the logarithmic data transformation, normalized means scores on the GDS variables ranged from 1.63 to 1.74 for this cluster.

(2) More Attention Problems Cluster. Ten of the 66 children and adolescents were in this cluster. Overall, these children exhibited more problems with GDS tasks than the children in the Less Attention Problems cluster. Of the 10 participants in this cluster, seven were from the ADHD group and three were from the No Diagnosis group. Following the logarithmic data transformation, normalized mean scores on the GDS variables indicated that children in the More Attention Problems cluster performed poorly on the vigilance total commissions, vigilance commission block variability, vigilance correct, distractibility total commissions, and distractibility commission block variability variables. The standardized mean scale scores on the 11 GDS variables for the Less Attention Problems and More Attention Problems clusters are presented in Figure 26.

Research question 2D asks: If two groups emerge from the cluster analysis, to what extent do the GDS profiles correctly classify children with ADHD and No

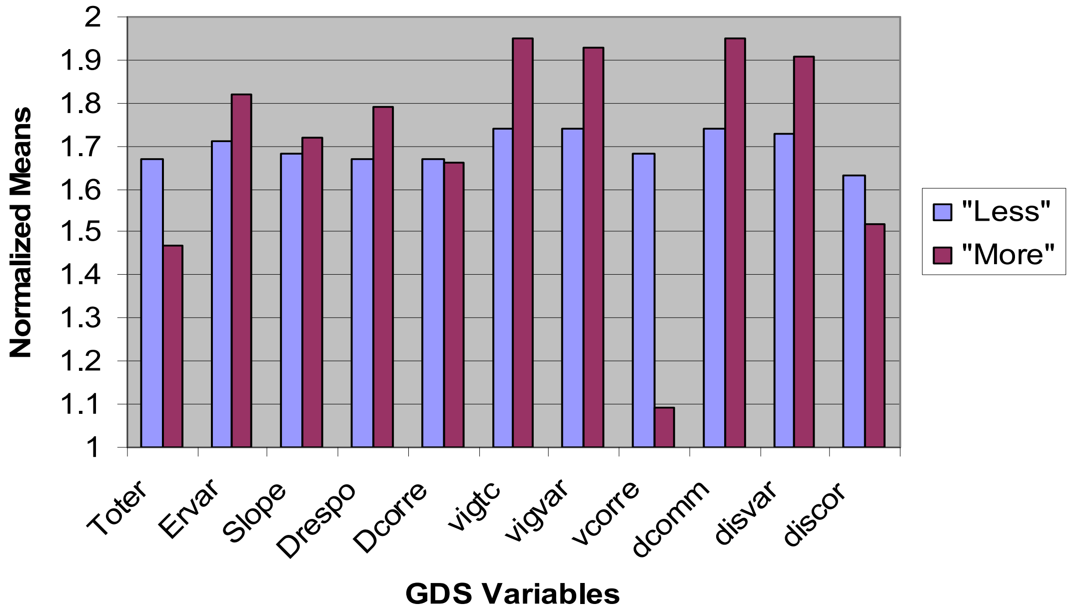

Figure 26. Normalized variable means for GDS Less Attention Problems and More Attention Problems clusters.

Note. Toter= delay total efficiency ratio; ervar= delay efficiency ration block variability; slope= delay efficiency ratio slope; drespo= delay total responses; dcorre= delay correct; vigtc= vigilance total commissions; vigvar= vigilance commission block variability; vcorre= vigilance correct; dcomm= distractibility total commissions, disvar= distractibility commission block variability; discor= distractibility correct.

Diagnosis? A Chi-square Test of Association was calculated. This technique was used to determine to what extent the children's performance on the GDS was able to predict group membership. The data used in this analysis included cluster membership according to GDS performance (more attention problems, less attention problems) and group membership (ADHD or No Diagnosis group). Using Chi-Square analysis, an independent relationship between the two cluster solution and ADHD and No Diagnosis group membership was found ($\underline{X}^2$= 3.36, *p*=.07). According to chance, 61% of children with ADHD should be included in each of the two clusters. Fifty-nine percent of the children in the Less Attention Problems cluster were from the ADHD group while 70% of children in the More Attention Problems were from the ADHD group.

Additional Analyses

Additional analyses were conducted to determine whether there were group differences between the 26 children with ADHD Combined Type only and the 26 normal controls using the CCPT-II and GDS measures. To determine whether the groups differed on the CCPT-II measure, a MANCOVA was performed. The analysis was conducted covarying for Full Scale IQ and Total Achievement. There was not a statistically significant group difference on the CCPT-II for children with ADHD Combined Type as compared to those with No Diagnosis using Wilks' Lambda [$F(2, 51) = 1.60$, $p = .13$]. A second MANCOVA was performed to determine whether the groups differed using the GDS measure. The analysis was conducted covarying for Full Scale IQ and Total Achievement. Results indicated that there was not a statistically significant group difference on the GDS for children with ADHD Combined Type as compared to those with No Diagnosis using Wilks' Lambda [$F(2, 51) = 1.41$, $p = .21$]. This lack of group difference indicated that when the ADHD group was restricted to a more homogenous group of children using those with ADHD Combined Type only, group comparisons with the No Diagnosis group were non-significant.

Several additional cluster analyses were conducted using the three separate GDS tasks (delay, vigilance, distractibility) to further understand the data. Because the three GDS tasks may place inherently different demands on examinees, three separate cluster analyses were computed to determine how well each of the tasks separated the ADHD and No Diagnoses groups.

The cluster analysis using the GDS delay variables (delay total ER, delay ER block variability, delay ER slope, delay total responses, and delay correct) yielded a 2-cluster solution that accounted for the 67 children and adolescents that had complete GDS data on the delay variables. Of the 67 children, 41 were in the ADHD group and 26 were in the No Diagnosis group. The normalized mean scale scores on the five GDS delay variables for the Less Attention Problems and More Attention Problems clusters are presented in Figure 27.

The two clusters are described below.

(1) Less Attention Problems Cluster. Seven of the 67 children and adolescents were in the Less Attention Problems cluster. These participants' scores were higher than average on the delay total ER, and lower than average on the delay ER block variability, ER slope variable, delay total responses, and delay correct variables. Of the seven children in this cluster, three were in the ADHD group and four were in the No Diagnosis group.

(2) More Attention Problems Cluster. Sixty of the 67 children and adolescents were in this cluster. Overall, these children exhibited more problems with the GDS delay tasks than the children in the Less Attention Problems cluster. These participants' scores were lower than average on the delay total ER variable and near average on the ER block variability, ER slope variable, delay total responses, and delay correct variables. Of the 60 children in this cluster, 38 were in the ADHD group and 22 were in the No

Diagnosis group. Overall, the children in the Less Attention Problems cluster made fewer, more efficient responses than the children in the More Attention Problems clusters. Less Attention Problems and More Attention Problems clusters are presented in Figure 27.

Using Chi-Square analysis, an independent relationship between the two cluster solution and ADHD and No Diagnosis group membership was found ($\underline{X}^2 = 3.36$, $p = .07$). According to chance, 61% of children with ADHD should be included in each of the two clusters. Forty-three percent of the children in the Less Attention Problems cluster were from the ADHD group while 63% of children in the More Attention Problems cluster were from the ADHD group.

The cluster analysis using the GDS vigilance variables yielded a 2-cluster solution that accounted for the 67 children and adolescents that had complete GDS data on vigilance variables. Of the 67 children, 41 were in the ADHD group and 26 were in the No Diagnosis group. The two clusters are described below.

(1) Less Attention Problems Cluster. Fifty-four of the 67 children and adolescents were in the Less Attention Problems cluster. These participants scored within the average range on the vigilance total commissions, vigilance commission block variability, and vigilance correct variables. Of the 54 children in this cluster, 32 were in the ADHD group and 22 were in the No Diagnosis group.

(2) More Attention Problems Cluster. Thirteen of the 67 children and adolescents were in this cluster. Overall, these children exhibited more problems with the GDS vigilance tasks than the children in the Less Attention Problems cluster. These

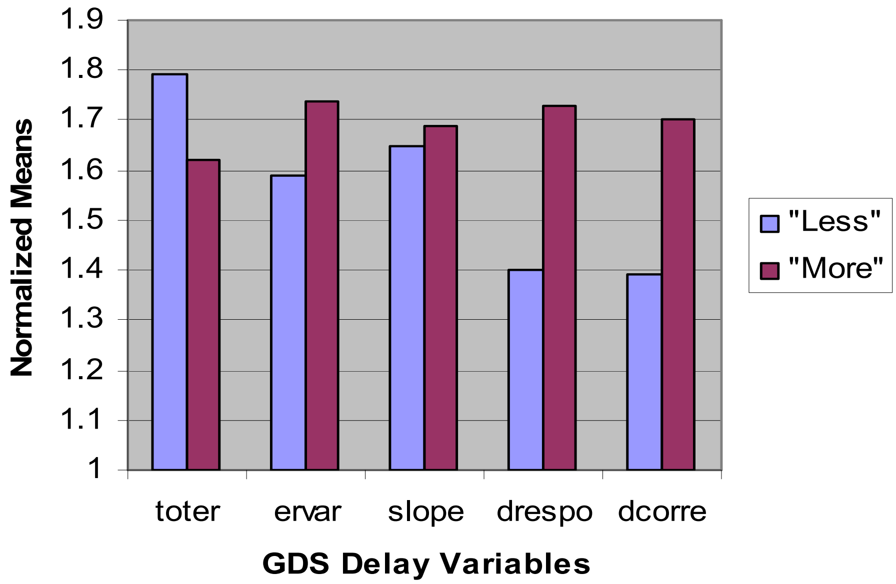

Figure 27. Normalized variable means for GDS Delay variables Less Attention Problems and More Attention Problems clusters.

Note. Toter= delay total efficiency ratio; ervar= delay efficiency ration block variability; slope= delay efficiency ratio slope; drespo= delay total responses; dcorre= delay correct.

participants scored higher than average on the vigilance total commissions and vigilance commission block variability variables and lower than expected on the vigilance correct variable. This pattern of performance indicated that these children and adolescents made more errors and responded correctly less often than their same age peers. Of the 13 children in this cluster, nine were in the ADHD group and four were in the No Diagnosis group. The normalized mean scale scores on the three GDS vigilance variables for the Less Attention Problems and More Attention Problems clusters are presented in Figure 28.

Using Chi-Square analysis, an independent relationship between the two cluster solution and ADHD and No Diagnosis group membership was found ($\underline{X}^2$= 3.36, *p*=.07). According to chance, 61% of children with ADHD should be included in each of the two clusters. Fifty-nine percent of the children in the Less Attention Problems cluster were from the ADHD group while 69% of children in the More Attention Problems were from the ADHD group.

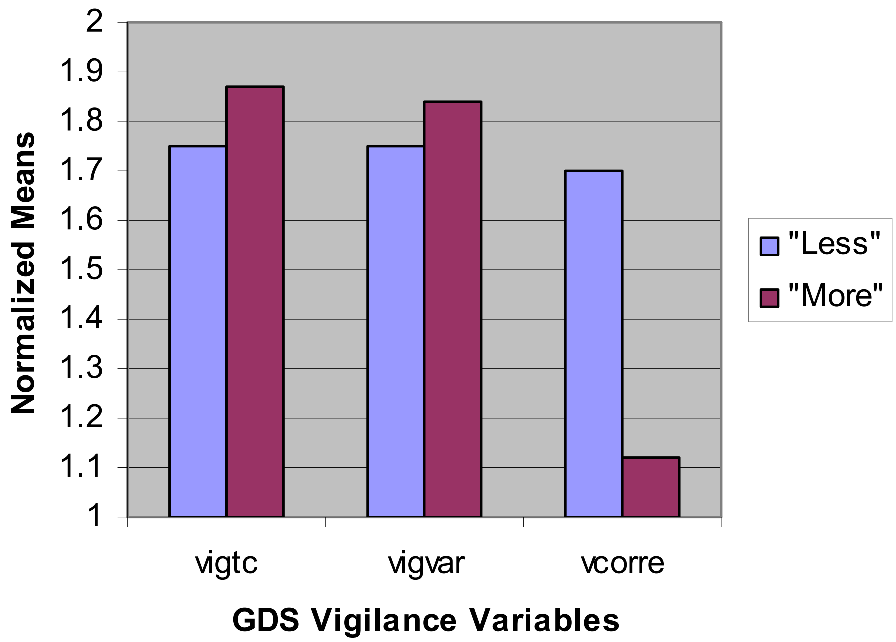

Figure 28. Normalized variable means for GDS Vigilance variables Less Attention Problems and More Attention Problems clusters.

Note. Vigtc= vigilance total commissions; vigvar= vigilance commission block variability; vcorre= vigilance correct.

The cluster analysis using the GDS distractibility variables yielded a 2-cluster solution that accounted for the 66 children and adolescents that had complete GDS data on distractibility variables. One child in the ADHD group had incomplete data on the

GDS distractibility task and was not included in this analysis. Of the 66 children, 40 were in the ADHD group and 26 were in the No Diagnosis group. The two clusters are described below.

(1) Less Attention Problems Cluster. Thirty-two of the 66 children and adolescents were in the Less Attention Problems cluster. These participants scored within the average range on the distractibility total commissions, distractibility commission block variability, and distractibility correct variables. Of the 32 children in this cluster, 17 were in the ADHD group and 15 were in the No Diagnosis group.

(2) More Attention Problems Cluster. Thirty-four of the 66 children and adolescents were in this cluster. These participants scored higher than average on the distractibility total commissions and distractibility commission block variability variables and lower than expected on the distractibility correct variable. This pattern of performance indicated that these children made more errors and responded correctly less often than their same age peers. Of the 34 children in this cluster, 23 were in the ADHD group and 11 were in the No Diagnosis group. The normalized mean scale scores on the three GDS distractibility variables for the Less Attention Problems and More Attention Problems clusters are presented in Figure 29.

Using Chi-Square analysis, an independent relationship between the two cluster solution and ADHD and No Diagnosis group membership was found (X^2= 3.36, *p*=.07). According to chance, 61% of children with ADHD should be included in each of the two clusters. Fifty-three percent of the children in the Less Attention Problems cluster were

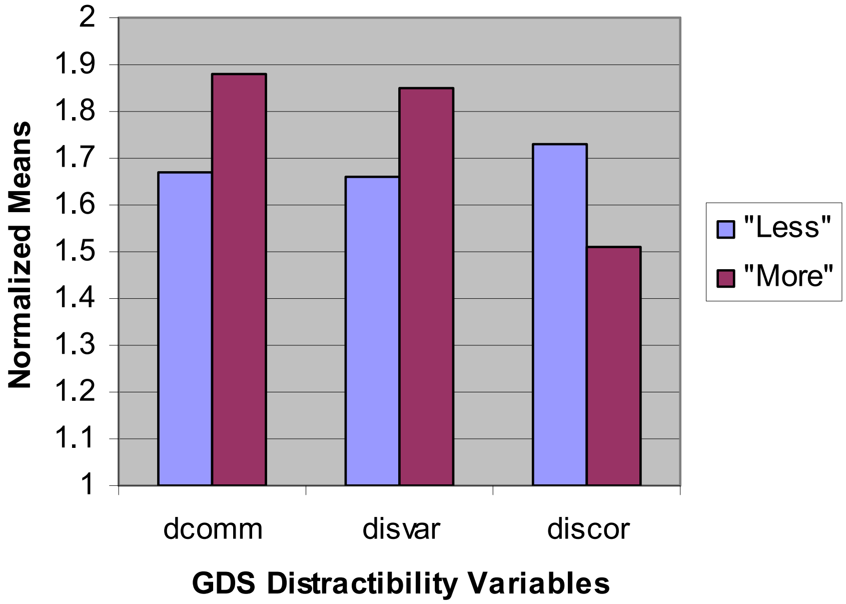

Figure 29. Normalized variable means for GDS Distractibility variables Less Attention Problems and More Attention Problems clusters.

Note. dcomm= distractibility total commissions, disvar= distractibility commission block variability; discor= distractibility correct.

from the ADHD group while 68% of children in the More Attention Problems were from the ADHD group.

This chapter has presented the analyses and results for the study. The next and final chapter includes a discussion of the results of the study as well as a presentation of the limitations of the study. Directions for future research and practice also are offered.

CHAPTER V

SUMMARY AND CONCLUSIONS

To learn more about the effects of different CPT versions as well as the numerous scoring indices, two very different CPTs, the CCPT-II and the GDS, were compared with a population of 41 children and adolescents who exhibited ADHD and 26 normal controls. Overall, the CCPT-II and GDS measures were not able to separate children with ADHD from normal controls. Furthermore, individual variables from neither the CCPT-II nor the GDS were able to adequately differentiate children with ADHD from those with No Diagnosis. Finally, score profiles obtained from the overall group of children and adolescents did not successfully separate the ADHD group from normal controls using the CCPT-II and GDS. In the section to follow, research findings are considered in detail as related to the research questions.

Group Differences

It was predicted that there would be significant differences in CCPT-II and GDS performance for children with ADHD as compared to children with No Diagnosis. There were not significant differences between the ADHD and No Diagnosis groups on the CCPT-II or GDS tasks. Results from this study are inconsistent with most studies to date that have found children with ADHD, as a group, to have more difficulties on CPT measures relative to normal controls.

When assessing individual task variables, it was predicted that the omission errors, commission errors, sensitivity (d’), and hit reaction time variables of the CCPT-II would do a better job differentiating ADHD and normal control groups than the other

CCPT-II variables. This hypothesis was based on Epstein and colleagues (2003) research findings with a group of children with ADHD and normal controls. Results from this study only partially supported Epstein and colleague's (2003) study. In the current study, only one of the variables previously reported by Epstein and colleagues (2003), the omissions variable, was able to separate children with ADHD from normal controls. The omission errors t-score did a "poor" job separating children and adolescents with ADHD from normal controls. The omission errors t-score was able to correctly classify randomly drawn pairs of participants 61% of the time. According to the CCPT-II manual (Conners, 1995), 'high omission error rates are a good indication that the respondent is literally not orienting and responding to the stimuli, or that he or she has a very slow and sluggish response' (p.29). The clinical index, a weighed score derived from all of the other indices, did a "fair" job of separating children with ADHD from normal controls. The clinical index was able to correctly classify randomly drawn pairs 69% of the time. The perseveration variable did a "poor" job of separating children with ADHD from normal controls and was able to correctly classify randomly drawn pairs 61% of the time. According to the CCPT-II manual (Conners, 1995), 'a perseveration is a response that occurs less than 100ms following a stimulus' (p.31). Conners' (1995) also indicated that, 'a large number of perseverations are likely the result of anticipatory responding, random responding, or frequent very slow responses to the preceding stimuli' (p.31). The remaining individual CCPT-II variables "failed" to differentiate children with ADHD from normal controls. The percentage of randomly drawn pairs for which each of the remaining 10 variables correctly classified the two participants ranged from 46% to 58%. Percentages were no better than chance for most variables.

Based on the paucity of research with the 11 GDS variables, it was difficult to predict which GDS scores may best describe differences between the ADHD and No Diagnosis groups. Results indicated that one of the GDS variables, the vigilance total commission, did a “poor” job at separating the groups. The vigilance total commission variable was able to correctly classify randomly drawn pairs of participants 61% of the time. According to the GDS manual (Gordon Systems, Inc., 1991), an elevated vigilance total commission score ‘provides evidence for an impulsive response style’ (p.41). It is possible that children with ADHD exhibited somewhat more difficulty with impulsivity as measured by the vigilance total commission variable than normal controls. The other ten GDS variables “failed” to separate children with ADHD from normal controls. For these nine variables, the percentage of randomly drawn pairs for which each variable correctly classified the two participants ranged from 33% to 59%.

There may be several reasons why the CCPT-II and GDS were not able to adequately separate children with ADHD from normal controls. Children with attentional deficits form a neuropsychologically heterogeneous group, and the complete correspondence between this behaviorally defined syndrome and cognitive measures of inattention and inhibition is hardly to be expected. Children with ADHD exhibit differing levels of attention difficulties. For example, while some children with ADHD exhibit problems with attention that are severe, other children with ADHD have less severe problems with attention. One would expect that children with more significant problems with attention would exhibit more problems on CPT measures.

Also, it seems likely that the CPT performance of children and adolescents diagnosed with each subtype of ADHD would be different. Barkley's (1997) theory asserts that the Combined Type differs from the Predominately Inattentive Type not only in that there is the presence of disinhibition, but that the types of attentional problems in the Combined Type differ from the attentional problems of the Predominately Inattentive Type. If Barkley's theory is correct and what are currently viewed as subtypes are significantly and qualitatively different, it would be expected that CPT performance would vary as a function of subtype. In the current study, however, results did not change when analyses were conducted using the ADHD Combined Type group only. Finally, there is a high rate of comorbidity between ADHD and other disorders that exhibit similar behavioral symptoms. For example, ADHD and anxiety disorders share several characteristics including difficulty concentrating, irritability, and restlessness. It is possible that children with ADHD who exhibit comorbid disorders such as anxiety disorders may exhibit more difficulty on CPT measures than children with ADHD only.

It is possible that the CCPT-II and GDS provide data on specific aspects of attention and executive control that are not central to ADHD. The CCPT-II and GDS Delay task may provide information specific to inhibition while the GDS Vigilance and Distractibility tasks may provide information specific to sustained attention. As Mirsky and colleagues (1995) pointed out, attention is not unitary; rather it is a nebulous and complex construct. Many theorists have proposed models that include several components of attention including initiating, focusing, and sustaining attention. The sheer complexity of the constructs of attention and executive control suggests that no single measure can provide information that is sufficiently comprehensive to address all components of attention.

Finally, there was a significant difference for children with ADHD and normal controls on Full Scale IQ on the WISC-IV and Total Achievement on the Woodcock-Johnson-Tests of Achievement. It is possible that covarying for these differences may have lessened the likelihood of finding differences across groups. A review of the literature indicated that when variables such as age, social economic status, and verbal ability were controlled (e.g., Koriath et al., 1985; Werry, Elkind, & Reeves, 1987), the likelihood of finding differences across groups subsided somewhat.

Cluster Analysis

It was predicted that CCPT-II score profiles and GDS score profiles obtained from the overall group of children would be able to separate the ADHD group from normal controls. Because children with ADHD, as a group, have more difficulties with CPT measures than normal controls, it would make sense that CPT patterns of scores would differ for the two groups of children. In this study, however, CCPT-II and GDS score profiles obtained from the overall group of children were no more useful than individual scores in differentiating children with ADHD from normal controls. While cluster analyses for the CCPT-II score profiles and GDS score profiles resulted in two-cluster solutions (more attention problems, less attention problems), chi-square analyses indicated that the CCPT-II and GDS score profiles were not able to correctly classify children with ADHD and No Diagnosis. To determine whether the three individual GDS tasks placed inherently different task demands on examinees, additional analyses were conducted using the three separate GDS tasks (delay, vigilance, distractibility). Results indicated that patterns of scores obtained for the overall group of children on the separate delay, vigilance, and distractibility GDS tasks were not useful in differentiating children with ADHD from normal controls.

Interestingly, results of the cluster analysis indicated that the CCPT-II may have placed more cognitive demands on most children in our study than the GDS. Specifically, 43 of the 66 children with complete data were in the "more attention problems" cluster using the CCPT-II score profiles while only 10 of the 66 children with complete data were in the "more attention problems" cluster using the GDS score profiles. Furthermore, cluster analysis results using only the GDS Delay task (a measure of inhibition) indicated that 60 of the 67 children were in the "more attention problems" cluster. These results may imply that most children, in general, exhibit more problems with inhibition than vigilance as measured by these instruments. If ADHD diagnoses were made based on patterns of children's performance on the CCPT-II or GDS Delay task, there would likely be a high rate of false positives. Conversely, if ADHD diagnoses were based on patterns of children's performance on the three combined GDS tasks (delay, vigilance and distractibility) or the GDS Vigilance task only, there would likely be a high rate of false negatives.

While it may be possible that the CCPT-II placed more cognitive demand on most children than the GDS, it is possible that differences in administration, stimulus presentation, duration, and scoring complicate interpretation. For example, differences in task demands such as interstimulus interval may explain why more children had difficulties on the CCPT-II than the GDS. While the interstimulus interval on the GDS is consistently one second, the interstimulus interval on the CCPT-II is varied within blocks at one second, two seconds, and four seconds. Averaged across time, participants spend more time observing a slower presentation rate on the CCPT-II. CPTs with longer interstimulus intervals make it more difficult for children to attend to the task. For example, O'Dougherty and colleagues (1984) found that children missed more targets and made the most false alarms when the interstimulus interval was four seconds.

Alternatively, the GDS is composed of three 9-minute tasks that are separated by directions and have different task demands, while the CCPT-II is a 14-minute test with one task demand. It is possible that children were more likely to have their attention captivated by the GDS than the CCPT-II because each of the GDS tasks was shorter and there were different task demands. It is well recognized that many children with ADHD can be attentive in "high-interest" situations such as watching television (Douglas, 1983; Ross & Pelham, 1981).

Limitations

The findings of this study must be considered in the context of several limitations. The current investigation was performed with a small sample size (n=67) of children. Participants were non-randomized volunteers from a small university-based community. A second limitation of the study is that there is no definitive means of diagnosing ADHD with guaranteed accuracy. According to Gordon and colleagues

(1996), ‘a gold standard for ADHD is far more a myth than a reality because there are significant and well-documented vulnerabilities associated with all diagnostic strategies’ (p.34). Therefore, there is no guarantee that all children and adolescents in the ADHD group indeed had ADHD and that all children and adolescents in the No Diagnosis group did not have ADHD. However, diagnostic accuracy was maximized by conducting comprehensive multi-method assessments. Furthermore, results were independently reviewed by two or more individuals (i.e., licensed psychologists or advanced doctoral students supervised by a licensed psychologist) for diagnostic consideration.

Another limitation in this study was that the DSM-IV subtypes of ADHD were combined to form the ADHD group. Based on the subtyping literature, the extent to which the symptoms of inattention and hyperactivity/ impulsivity co-occur varies by subtype, with different neuropsychological profiles for each subtype (Clure et al., 1999; Dykman & Ackerman, 1993; Ebert, 1995; Marks, Himelstein, Newcorn, & Halperin, 1999; Marshall, Hynd, Handwerk, & Hall, 1997; Morgan, Hynd, Riccio, & Hall, 1996). In the current study, small numbers of children meeting criteria for each of the subtypes prevented any between-group comparisons. Also of concern, 22 (53.7%) of the children and adolescents in the ADHD group were diagnosed with an additional disorder such as a learning disability, depression, or anxiety. According to a review of the literature, children with disorders other than ADHD (e.g., anxiety disorders, reading disorders) have performed poorly on CPTs. The high rates of comorbidity between ADHD and other disorders make it difficult to determine whether poor performance is due to ADHD, the comorbid disorder, or a combination of disorders.

The fact that this study only included two continuous performance tests is another limitation. Although the GDS and CCPT-II are representative of some of the most commonly used CPT measures included in neuropsychological assessments, there is an abundance of variation in task parameters (stimuli type, stimuli quality, ISI) as well as CPT demands (X-CPT, AX-CPT, not-X-CPT). The pattern of results indicated in this study may not hold true for other CPT versions with differing task parameters and demands. However, given the exploratory nature of this study, the CPTs chosen provide the framework for future research in this area.

Implications and Future Research

In a climate of rising health-care costs, it behooves clinicians to use a brief but efficient battery to optimize differential diagnoses. One of the main implications that this study highlights is the need for objective measures of attention, such as CPTs, that neuropsychologists can use as part of a multifaceted assessment battery. As such, test developers and publishers need to provide CPT manuals that include detailed instructions for standardized administration, instructions for scoring, and guidelines for interpretation. Technical data, including reliability of the test scores and validity of the interpretation of scores obtained should be provided as well (Riccio et al., 2001). Continuous performance test manuals that meet current standards for educational and psychological testing set forth by the AERA (1999) are needed.

Research findings to date on CPT measures with children are discrepant and may be related to one or more of the following points. Because of the multiple variations in CPT task parameters as well as CPT demands, making generalizations based on performance data continues to be a difficult task. Different CPTs may place different demands on an individual's attention, executive, and memory systems; one cannot be substituted for another (Riccio et al., 2001). Furthermore, it is difficult to draw conclusions regarding the validity of CPTs with children with ADHD when studies are of limited sample size and rely on different types of data to define ADHD in subjects. Differences in age ranges used and inconsistent matching procedures on age, gender, and IQ can lead to discrepant results. Further, some studies collapse children with different subtypes of ADHD into one group, while other studies compare children by subtype. The multiple changes in DSM definitions of ADHD subtypes serves as another area of concern. The inclusion of children with comorbid diagnoses in some studies but not others may lead to a lack of agreement in results. Although controlled for in this study, stimulant medication usage by children with ADHD may decrease group differences on the CPT in research.

In order to avoid contributing to the convoluted research described above, it is imperative that large-scale, geographically diverse studies of CPTs with children be conducted. Riccio and colleagues (2001) described such a study as including two or three of the most common CPTs paradigms. In addition to including visual CPTs, it may be interesting to include an auditory CPT. Such a study may include samples of children diagnosed with rigorous, common criteria. Diagnostic groups including children with ADHD (by subtype), anxiety disorders, depression, learning disorders, traumatic brain injury, and normal controls should be included. Such large-scale studies may make it possible to determine whether CPT paradigms, individual variables, or patterns of scores can discriminate among these groups of children. This type of research ultimately will provide the information necessary to help clinicians determine whether CPTs should be included in assessment batteries as an objective measure of attention with various populations of children.

REFERENCES

Abikoff, H., Courtney, M., Pelham, W., & Koplewicz, H. (1993). Teacher ratings of disruptive behaviors: The influence of halo effects. *Journal of Abnormal Child Psychology, 21*(5), 519-533.

Achenbach, T. M. (1991). *Manual for the child behavior checklist and 1991 child behavior profile*. Burlington: University of Vermont, Department of Psychiatry.

Albin, R. L., Young, A. B., & Penney, J. B. (1995). The functional anatomy of disorders of the basal ganglia. *Trends in Neuroscience, 18,* 63-64.

Aldenderfer, M., & Blashfield, R. (1984). *Cluster analysis: Quantitative applications in the social sciences*. Beverly Hills, California: Sage Publications, Inc.

Allen, L. F. (1993). *Developmental delay of frontal lobe functioning: A possible cause of attention deficits in children*. Unpublished doctoral dissertation, Texas A & M University, College Station.

American Academy of Child and Adolescent Psychiatry (1997). Practice parameters for the assessment and treatment of children, adolescents, and adults with attention-deficit/hyperactivity disorder. *Journal of the American Academy of Child and Adolescent Psychiatry, 36*(suppl): 85S-121S.

American Educational Research Association, American Psychological Association, & National Council on Measurement in Education (1999). *Standards for educational and psychological testing*. Washington, DC: Author.

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

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

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

American Psychiatric Association (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., Text Revision). Washington, DC: Author.

Anderson, V. E., Siegel, F. S., Fisch, R. O., & Wirt, D. (1969). Response of phenylketonuric children on a continuous performance test. *Journal of Abnormal Psychology, 74*, 358-362.

Angold, A., & Costello, E. J. (1995). A test-retest reliability study of child-reported psychiatric symptoms and diagnoses using the Child and Adolescent Psychiatric Assessment. (CAPA-C). *Psychological Medicine, 25,* 755-762.

Angold, A., & Costello, E. J. (2000). The Child and Adolescent Psychiatric Assessment (CAPA). *Journal of the American Academy of Child and Adolescent Psychiatry, 39*, 39-48.

Axelrod, M. (2002). Signal detection, perceptual sensitivity, and response bias: Assessing attention and impulsivity in children with attention deficit hyperactivity disorder and children with anxiety using a continuous performance test. *Dissertation Abstracts International: Section B: The Sciences & Engineering, 63*(5-B), 2572.

Baker, C. H. (1959a). Attention to visual displays during a vigilance task: II. Maintaining the level of vigilance. *British Journal of Psychology, 50*, 30-36.

Baker, C. H. (1959b). Towards a theory of vigilance. *Canadian Journal of Psychology, 13*, 35-42.

Baker, C. H. (1963). Further toward a theory of vigilance. In D. N. Buckner & J. J. McGrath (Eds.), *Vigilance: A symposium* (pp.127-170). New York: McGraw Hill.

Ballard, J. C. (1996). Computerized assessment of sustained attention: A review of factors affecting vigilance performance. *Journal of Clinical and Experimental Neuropsychology, 18*(6), 843-863.

Ballard, J. C. (2001). Assessing attention: Comparison of response inhibition and traditional continuous performance tests. *Journal of Clinical and Experimental Neuropsychology, 23*(3), 331-350.

Band, G., van der Molen, M., Overtoom, C., & Verbaten, M. (2000). The ability to activate and inhibit speeded responses: Separate developmental trends. *Journal of Experimental Child Psychology, 75*, 263-290.

Barbas, H. & Mesulam, M. (1985). Cortical afferent input to the principalis region in rhesus monkey. *Neuroscience, 15*, 619-637.

Baren, M., & Swanson, J. M. (1996). How not to diagnose ADHD. *Contemporary Pediatrics, 13,* 53-64.

Barkley, R. A. (1981). *Hyperactive children: A handbook for diagnosis and treatment.* New York: Guilford.

Barkley, R. A. (1990). *Attention deficit hyperactivity disorder: A handbook for diagnosis and treatment* (pp. 3-673). New York: Guilford Press.

Barkley, R. A. (1991). The ecological validity of laboratory and analogue assessment methods of ADHD symptoms. *Journal of Abnormal Child Psychology, 19,* 149-178.

Barkley, R. A. (1997). Behavioral inhibition, sustained attention, and executive functions: Constructing a unifying theory of ADHD. *Psychological Bulletin, 121*(1), 65-94.

Barkley, R. A. (1998). *Attention-deficit hyperactivity disorder: A handbook for diagnosis and treatment* (2nd ed.). New York: Guilford Press.

Barkley, R. A., DuPaul, G. J., & McMurray, M. B. (1990). A comprehensive evaluation of attention deficit disorder with and without hyperactivity as defined by research criteria. *Journal of Consulting and Clinical Psychology, 58*, 775-789.

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

Barkley, R. A., & Ullman, D. G. (1975). A comparison of objective measures of activity and distractibility in hyperactive and nonhyperactive children. *Journal of Abnormal Child Psychology, 3*, 231-244.

Barry, T. D., Klinger, L. G., Lyman, R. D., Bush, D., & Hawkins, L. (2001). Visual selective attention versus sustained attention in boys with attention-deficit/ hyperactivity disorder. *Journal of Attention Disorders, 4*(4), 193-202.

Baumgardner, T. L., Singer, H. S., Denckla, M. B., Rubin, M. A., Abrams, M. T., Colli, M. J., & Reiss, A. L. (1996). Corpus callosum morphology in children with Tourette syndrome and attention deficit hyperactivity disorder. *Neurology, 47*, 477-482.

Bell, J., & Livesey, P. (1985). Cue significance and response regulation in 3- to 6-year-old children's learning of multiple choice discrimination tasks. *Developmental Psychobiology, 18*, 229-245.

Berch, D. B., & Kanter, D. R. (1984). Individual differences. In J. S. Warm (Ed.), *Sustained attention in human performance* (pp. 143-178). Chichester, UK: John Wiley & Sons.

Bergman, A., Winters, L., & Cornblatt, B. (1990). Methylphenidate: Effects on sustained attention. In L. L. Greenhill & B. B. Osman (Eds.). *Ritalin: Theory and management* (pp. 223-231). New York: Mary Ann Liebert, Inc.

Biederman, J. (1998). Attention-deficit/ hyperactivity disorder: A Life-Span Perspective. *Journal of Clinical Psychiatry, 59*(7), 4-16.

Biederman, J., Baldessarini, R., Wright, V., Knee, D., & Harmatz, J. (1989). A double-blind placebo controlled study of desipramine in the treatment of ADD: I. Efficacy. *Journal of the American Academy of Child and Adolescent Psychiatry, 28*, 777-784.

Biederman, J., Faraone, S., Mick, E., Moore, P., & Leon, E. (1996). Child behavior checklist findings further support comorbidity between ADHD and major depression in a referred sample. *Journal of the American Academy of Child and Adolescent Psychiatry, 35*, 734-742.

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.

Bock, R. D. (1982). *The role of arousal in Tourette syndrome*. Unpublished doctoral dissertation, New York University, New York City.

Broadbent, D. E.(1958). *Perception and communication*. New York: Pergamon.

Broadbent, D. E. (1971). *Decision and stress*. London: Academic.

Bronowski, J. (1967). Human and animal languages. In *To honor Roman Jakobson* (Vol. 1). The Hague, Netherlands: Mouton.

Brumm, V.L. (1994). *Neuropsychological and psychological correlates of marital violence in a clinical sample*. Unpublished doctoral dissertation, University of Southern California, Los Angeles, CA.

Buckner, D. N., Harabedian, A., & McGrath, J. J. (1968). A study of individual differences in vigilance performance (Technical Report 2, January, 1960). In *Studies of human vigilance: An omnibus of technical reports* (pp. 109-144). Goleta, CA: Human Factors Research.

Burg, J. S., Burright, R. G., & Donovick, P. J. (1995). Performance data for traumatic brain injured subjects on the Gordon Diagnostic System (GDS) tests of attention. *Brain Injury, 9*, 395-403.

Campbell, S. B. (1974). Cognitive styles and behavior problems of clinic boys: A comparison of epileptic, hyperactive, learning-disabled, and normal groups. *Journal of Abnormal Child Psychology, 2*, 307-312.

Campbell, S. B. (1990). *Psychiatric disorder in preschool children*. New York: Guilford Publications.

Cantwell, D. P. (1975). The hyperactive child: Epidemiology, classification and diagnosis. In D. P. Cantwell (Ed.). *The hyperactive child: Diagnosis management, and current research*. New York: Spectrum Publications.

Cantwell, D. P. (1996). Attention deficit disorder: A review of the past 10 years. *Journal of the American Academy of Child and Adolescent Psychiatry, 35*(8), 978-987.

Carpenter, M. F. (2002). Predictive accuracy of the Gordon Diagnostic System and Wide Range Assessment of Memory and Learning in ADHD evaluation. *Dissertation Abstracts International: Section B: The Sciences & Engineering, 62*(12-B), 5955.

Carter, C. S., Krener, P., Chaderjian, M., Northcutt, C., & Wolfe, V. (1995). Abnormal processing of irrelevant information in attention deficit hyperactivity disorder. *Psychiatry Research, 56*, 59-70.

Carver, A. C., Livesey, D. J., & Charles, M. (2001). Age related changes in inhibitory control as measured by stop-signal task performance. *International Journal of Neuroscience, 107*, 43-61.

Casey, B. J., Castellanos, F. X., Giedd, J. N., Marsh, W. L., Hamburger, S. D., Schubert, A. B.,Vauss, Y. C., Vaituzis, A. C., Dickstein, D. P., Sarfatti, S. E., & Rapoport, J. L. (1997). Implications of right frontostriatal circuitry in response inhibition and attention-deficit/ hyperactivity disorder. *Journal of the American Academy of Child and Adolescent Psychiatry, 151*, 1791-1796.

Castellanos, F. X., Giedd, J. N. Eckburg, P., Marsh, W. L., Vaituzis, C., Kaysen, D., Hamburger, S. D., & Rapoport, J. L. (1994). Quantitative morphology of the caudate nucleus in attention deficit hyperactivity disorder. *American Journal of Psychiatry, 151*, 1791-1796.

Castellanos, F. X., Giedd, J. N., Marsh, W. L., Hamburger, S. D., Vaituzis, A. C., Dickstein, D.P., Sarfatti, S. E., Vauss, Y. C., Snell, J. W., Lange, N., Kaysen, D., Krain, A. L., Ritchie, G. F., Rajpakse, J. C., & Rapoport, J. L. (1996). Quantitative brain magnetic resonance imaging in attention-deficit hyperactivity disorder. *Archives of General Psychiatry, 53*, 607-616.

Chee, P., Logan, G., Schachar, R., Lindsay, P., & Wachsmuth, R. (1989). Effects of event rate and display time on sustained attention in hyperactive, normal, and control children. *Journal of Abnormal Child Psychology, 17*, 371-391.

Chelune, G., & Baer, R. (1986). Developmental norms for the Wisconsin Card Sorting Test. *Journal of Clinical and Experimental Neuropsychology, 17*, 219-228.

Clark, C. R., Geffen, G. M., & Geffen, L. B. (1987). Catecholamines and attention II: Pharmacological studies in normal humans. *Neuroscience and Biobehavioral Research, 11*, 353-364.

Clements, S. (1966). *Minimal brain dysfunction in children* (PHS Report No. 1415). Bethesda, MD: National Institute of Health.

Clure, C., Brady, K. T., Saladin, M. E., Johnson, D., Waid, R., & Rittenbury, M. (1999). Attention deficit/hyperactivity disorder and substance use: Symptom pattern and drug choice. *American Journal of Drug and Alcohol Abuse, 25*, 441-448.

Cohan, M. F. (1995). *Validity investigation of the Conners' Continuous Performance Test.* Unpublished doctoral dissertation, State University of New York, Albany.

Conners, C. K. (1992). *Conners' Continuous Performance Test user's manual.* Toronto Canada: Multi-Health Systems.

Conners, C. K. (1994). *Conners Continuous Performance Test* (Version 3.0): [Computer Software]. Toronto; Multi-Health Systems.

Conners, C. K. (1995). *Conners' Continuous Performance Test-II user's manual.* Toronto, Canada: Multi-Health Systems.

Conners, C. K., Epstein, J. N., Angold, A., & Klaric, J. (2003). Continuous performance test performance in a normative epidemiological sample. *Journal of Abnormal Child Psychology, 31*(5), 555-562.

Cooley, E. L., & Morris, R. D. (1990). Attention in children: A neuropsychologically based model for assessment. *Developmental Neuropsychology, 6*(3), 239-274.

Corkum, P. V., & Siegel, L. S. (1993). Is the continuous performance task a valuable research tool for use with children with attention-deficit-hyperactivity disorder? *Journal of Child Psychology and Psychiatry, 34*(7), 1217-1239.

Cornblatt, B. A., Lenzenweger, M. F., & Erlenmeyer-Kimling, L. (1989). The continuous performance test, identical pairs version (CPT-IP): II. Contrasting attention profiles in schizophrenia and depressed patients. *Psychiatry Research, 29*, 65-85.

Crinella, F. M. (1973). Identification of brain dysfunction syndromes in children through profile analysis. *Journal of Abnormal Psychology, 82,* 33-45.

Das, J. P., Snyder, J. J., & Mishra, R. K. (1992). Assessment of attention: Teachers' rating scales and measures of selective attention. *Journal of Psychoeducational Assessment, 10*, 37-46.

Davies, D. R., Jones, D. M., & Taylor, A. (1984). Selective and sustained-attention tasks: Individual and group differences. In R. Parasuraman & D.R. Davies (Eds.), *Varieties of attention* (pp. 395-447). Orlando: Academic Press.

Davies, D. R., & Parasuraman, R. (1981). *The psychology of vigilance.* Toronto: Academic Press.

Davies, D. R., & Parasuraman, R. (1982). *The psychology of vigilance*. London: Academic.

Deese, J. (1955). Some problems in the theory of vigilance. *Psychological Review, 62,* London: Academic.

DeLong, M. R. (1990). Primate models of movement disorders of basal ganglia origin. *Trends in Neuroscience, 13,* 281-285.

Dember, W. N., Galinsky, T. L., & Warm, J. S. (1992). The role of choice in vigilance performance. *Bulletin of the Psychonomic Society, 30*, 201-204.

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

Diagnostic and Statistical Manual, II (DSM-II), (1968), Washington DC: American Psychiatric Association.

Diagnostic and Statistical Manual, III (DSM-III), (1980), Washington DC: American Psychiatric Association.

Diagnostic and Statistical Manual, III-R (DSM-III-R), (1987), Washington DC: American Psychiatric Association.

Diagnostic and Statistical Manual, IV (DSM-IV), (1994), Washington DC: American PsychiatricAssociation.

Dickerson Mayes, S., Calhoun, S., & Crowell, E. (2001). Clinical validity and interpretation of the Gordon Diagnostic System in ADHD assessments. *Child Neuropsychology, 7*(1), 32-41.

Dimond, S. J., & Beaumont, J. G. (1973). Difference in the vigilance performance of the right and left hemispheres. *Cortex, 9*, 259-265.

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

Douglas, V. I. (1983). Attention and cognitive problems. In M. Rutter (Ed.), *Develop-mental neuropsychiatry* (pp. 280-329). New York: Guilford Press.

Dowsett, S. M., & Livesey, D. J. (2000). The development of inhibitory control in preschool children: Effects of "executive skills" training. *Developmental Psychobiology, 36*, 161-174.

Draeger, S., Prior, M., & Sanson, A (1986). Visual and auditory attention performance in hyperactive children: Competence or compliance. *Journal of Abnormal Child Psychology, 14*, 411-424.

Dulcan, M., and the Work Group on Quality Issues (1997). Practice parameters for the assessment and treatment of children, adolescents, and adults with attention-deficit/ hyperactivity disorder. *Journal of the American Academy of Child and Adolescent Psychiatry,* (10 Supplement), *36*, 85S-121S.

DuPaul, G. J., Anastopoulos, A. D., Shelton, T. L., Guevremont, D. C., & Metevia, L. (1992). Multimethod assessment of attention-deficit hyperactivity disorder: The diagnostic utility of clinic-based tests. *Journal of Clinical Child Psychology, 24*(4), 394-402.

Dykman, R. A., & Ackerman, P. T. (1993). Behavioral subtypes of attention deficit disorder. *Exceptional Children, 60*, 132-141.

Earle-Boyer, E. A., Serper, M. R., Davidson, M., & Harvey, P. D. (1991). Continuous performance tests in schizophrenic patients: Stimulus and medication effects on performance. *Psychiatry Research, 37*, 47-56.

Ebert, J. (1995). Review of attention-deficit/hyperactivity disorder: Implications for identification and management. *Journal of Psychological Practice, 1*, 81-98.

Edwards, G. (1998). Determining the role of a new continuous performance test in the diagnostic evaluation for ADHD. *ADHD Report, 6*(3), 11-13.

Epstein, J. N., Erkanli, A., Conners, C. K., Klaric, J., Constello, J. E., & Angold, A. (2003). Relations between continuous performance test performance measures and ADHD behaviors. *Journal of Abnormal Child Psychology, 31*(5), 543-554.

Faraone, S. V., & Biederman, J. (1998). Neurobiology of attention-deficit hyperactivity disorder. *Biological Psychiatry, 44,* 951-958.

Filipek, P. A., Semrud-Clikeman, M., Steingard, R. J., Renshaw, P. F., Kennedy, D. N, & Biederman, J. (1997). Volumetric MRI analysis comparing subjects having attention-deficit hyperactivity disorder with normal controls. *Neurology, 48*, 589-601.

Fischer, M. Barkley, R. A., Edelbrock, C. S., & Smallish, L. (1990). The adolescent outcome of hyperactive children diagnosed by research criteria: II. Academic, attentional, and neuropsychological status. *Journal of Consulting and Clinical Psychology, 58*, 580-588.

Fischer, M., Newby, R. F., & Gordon, M. (1995). Who are the false negatives on continuous performance tests? *Journal of Clinical Child Psychology, 24*, 427-433.

Fitzpatrick, P. A., Klorman, R., Brumaghim, J. T., & Borgstedt, A. D. (1992). Effects of sustained release and standard preparations of methylphenidate on attention deficit disorder. *Journal of the American Academy of Child and Adolescent Psychiatry, 31*, 226-234.

Fleiss, J. L. (1981). *Statistical methods for rates and proportions*, 2nd ed. John Wiley & Sons; Chichester, UK.

Frankmann, J. P., & Adams, J. A. (1962). Theories of vigilance. *Psychological Bulletin, 59,* 257-272.

Garfinkel, B. G., & Klee, S. H. (1983). A computerized assessment battery for attention deficits. *Psychiatry Hospitals, 14*, 163-166.

Gerfen, C. R. (1992). The neostriatal mosaic: Multiple levels of compartmental organization. *Trends in Neuroscience, 15,* 133-139.

Girardi, N. L., Shaywitz, S. E., Marchione, K., Fleischman, S. J., Jones, T. W., & Tamborlane, W. V. (1995). Blunted catecholamine responses after glucose ingestion in children with attention deficit disorder. *Pediatric Research, 38*, 53-542.

Golden, C. J. (1981). The Luria-Nebraska Children's Battery: Theory and formulation. In G. Hynd & J. Obrzut (Eds.), *Neuropsychological assessment of the school aged child*. New York: Grune & Stratton.

Goldstein, S. (1999). Attention deficit/ hyperactivity disorder. In S. Goldstein & C. R. Reynolds (Eds.), *Handbook of neurodevelopmental and genetic disorders in children* (pp.154-184). New York: Guilford Press.

Goodman, R., & Stevenson, J. (1989). A twin study of hyperactivity: II. The etiologic role of genes, family relationships, and perinatal adversity. *Journal of Child Psychology and Psychiatry, 30*, 691-709.

Gordon, M. (1979). The assessment of impulsivity and mediating behaviors in hyperactive and nonhyperactive children. *Journal of Abnormal Psychology, 7*, 317-326.

Gordon, M. (1983). *The Gordon Diagnostic System*. DeWitt, NY: Gordon Systems.

Gordon, M. (1986). How is a computerized attention test used in the diagnosis of attention deficit disorder? *Journal of Children in Contemporary Society, 19*, 53-64.

Gordon, M., DiNiro, D., & Mettelman, B. B. (1988). Effect upon outcome of nuances in selection criteria for ADHD/hyperactivity. *Psychological Reports, 62*, 539-544.

Gordon, M., DiNiro, D., Mettelman, B., & Tallmadge, J. (1989). Observations of test behavior, quantitative scores, and teacher ratings. *Journal of Psychoeducational Assessment, 7*, 141-147.

Gordon, M., McClure, F. D., & Aylward, G. P. (1996). *The Gordon Diagnostic System interpretive guide* (3rd ed.). DeWitt, NY: GSI Publications.

Gordon, M. & Mettleman, B. B. (1985). Special tables for the *Gordon Diagnostic System.* (Personal Collection).

Gordon Systems, Inc. (1991). *Administration manual for the Gordon Diagnostic System*. DeWitt, NY: Author.

Halperin, J. M., O'Brien, J. D., Newcorn, J. H., Healey, J. M., Pascualvaca, D. M., Wolfe, L. E., et al. (1990). Validation of hyperactive, aggressive, and mixed hyperactive/ aggressive childhood disorders. A research note. *Journal of Child Psychology and Psychiatry, 31*, 455-459.

Halperin, J. M., Sharma, V., Greenblatt, E., & Schwartz, S. T. (1991). Assessment of the continuous performance test: Reliability and validity in a nonreferred sample. *Psychological Assessment: A Journal of Consulting and Clinical Psychology, 3*, 603-608.

Halperin, J. M., Wolf, L. E., Pascaulvaca, D. M., Newcorn, J. H., Healey, J. M., O'Brien, J. D., Morganstein, A., & Young, G. (1988). Differential assessment of attention and impulsivity in children. *Journal of the American Academy of Child and Adolescent Psychiatry, 27*, 326-329.

Harley, C. W. (1987). A role for norepinephrine in arousal, emotion and learning: Limbic modulation by norepinephrine and the key hypothesis. *Progress in Neuro-psychopharmacology and Biological Psychiatry, 11*, 419-458.

Hastings, J. E., & Barkley, R. A. (1978). A review of psychophysiological research with hyperactive children. *Journal of Abnormal Child Psychology, 7*, 413-447.

Hatfield, J. L., & Loeb, M. (1968). Sense mode and coupling in a vigilance task. *Perception and Psychophysics, 4*, 29-36.

Heaton, R. K. (1981). *A manual for the Wisconsin Card Sorting Test.* Psychological Assessment Resources. Odessa, FL.

Hinshaw, S. P. (1987). On the distinction between attentional deficits/ hyperactivity and conduct problems/ aggression in child psychopathology. *Psychological Bulletin, 101,* 443-463.

Hinshaw, S. P. (1992). Externalizing behavior problems and academic underachievement in childhood and adolescence: Causal relationships and underlying mechanisms. *Psychological Bulletin, 111*, 127-155.

Hynd, G. W., Hern, K. L., Novey, E. S., Eliopulos, D., Marshall, R., Gonzalez, J. J., & Voeller, K. K. (1993). Attention-deficit hyperactivity disorder and asymmetry of the caudate nucleus. *Journal of Child Neurology, 8*, 339-347.

Hynd, G. W., Semrud-Clikeman, M., & Lorys, A. R. (1991). Corpus callosum morphology in attention-deficit hyperactivity disorder (ADHD): Morphometric analysis of MRI. *Journal of Learning Disabilities, 24*, 141-146.

James, W. (1890). *Principles of psychology*. New York: Dover.

Jenson, P. S., Martin, D., & Cantwell, D. P. (1997). Comorbidity in ADHD: Implications for research, practice and DSM-V. *Journal of the American Academy of Child and Adolescent Psychiatry, 36*, 1065-1079.

Jerison, H. J. (1967). Signal detection theory in the analysis of human vigilance. *Human Factors, 9*, 285-288.

Kardell, E. G. (1994). Evaluation of the WISC-III in the assessment of attention and concentration processes in children. Unpublished Ph.D. dissertation, Pace University, New York.

Kaufman, L., & Rousseeuw, P. J. (1990). *Finding groups in data: An introduction to cluster analysis.* New York: John Wiley & Sons, Inc.

Keilp, J. G., Herrera, J., Stritzke, P., & Cornblatt, B. A. (1997). The Continuous Performance Test, Identical Pairs version (CPT-IP): III. Brain functioning during performance of numbers and shapes subtasks. *Psychiatry Research*: *Neuroimaging Section, 74*, 35-45.

Klee, S. H., & Garfinkel, B. D. (1983). The computerized Continuous Performance Task: A new measure of inattention. *Journal of Abnormal Child Psychology, 11*, 487-496.

Kline, R.B. (1998). *Principles and practices of structural equation modeling.* New York: The Guilford Press.

Klorman, R. (1992). Cognitive event-related potentials in attention deficit disorder. In S. E. Shaywitz & B. A. Shaywitz (Eds.). *Attention deficit disorder comes of age: Toward the twenty-first century* (pp.221-244). Austin, TX: Pro-ED.

Klorman, R., Brumaghim, J. T., Fitzpatrick, P. A., & Borgstedt, A. D. (1990). Clinical effects of a controlled trial of methylphenidate on adolescents with attention deficit disorder. *Journal of the American Academy of Child and Adolescent Psychiatry, 29*, 701-709.

Koelega, H. S., Brinkman, J. A., Hendriks, L., & Verbaten, M. N. (1989). Processing demands, effort and individual differences in four vigilance tasks. *Human Factors, 31*, 45-62.

Koriath, U., Gualtieri, C. T., Van Bourgondien, M. E., Quade, D., & Werry, J. S. (1985). Construct validity of clinical diagnosis in pediatric psychology: Relationship among measures. *Journal of the American Academy of Child Psychiatry, 24*, 429-436.

Lam, C. M., & Beale, I. L. (1991). Relations among sustained attention, reading performance, and teachers' ratings of behavior problems. *Remedial and Special Education (RASE), 12*, 40-47.

Levin, E. D., Wilson, W., Rose, J. E., & McEvoy, J. (1996). Nicotine-haloperidol interactions and cognitive performance in schizophrenics. *Neuropsycho-pharmacology, 15,* 429-436.

List, M. A. (1985). A developmental study of distractibility in the school-aged child. Unpublished Ph. D. dissertation, University of Waterloo, Canada.

Liu, S. K., Hwu, H., & Chen, W. J. (1997). Clinical symptom dimensions and deficits on the continuous performance test in schizophrenia. *Schizophrenia Research, 25*, 211-219.

Livesey, D. J., & Morgan, G. A. (1991). The development of response inhibition in 4- and 5- year-old children. *Australian Journal of Psychology, 43*, 133-137.

Livingston, R., Dykman, R., & Ackerman, P. (1990). The frequency and significance of additional self-reported psychiatric diagnoses in children with attention deficit disorder. *Journal of Abnormal Child Psychology, 18*(5), 465-478.

Loeber, R., Green, S., Lahey, B., & Stouthamer-Loeber, M. (1989). Optimal informants on childhood disruptive behaviors. *Development and Psychopathology, 1*, 317-337.

Losier, B. J., McGrath, P. J., & Klein, R. M. (1996). Error patterns on the continuous performance test in non-medicated and medicated samples of children with and without ADHD: A meta-analytic review. *Journal of Child Psychology and Psychiatry, 37*(8), 971-987.

Lou, H. C., Henriksen, L., & Bruhn, P. (1984). Focal cerebral hypoperfusion in children with dysphasia and/or attention deficit disorder. *Archives of Neurology, 11*, 825-829.

Lovejoy, M. C., & Rasmussen, N. H. (1990). The validity of vigilance tasks in differential diagnosis of children referred for attention and learning problems. *Journal of Abnormal Child Psychology, 18*, 671-681.

Lucaccini, L., Freedy, A., & Lyman, J. (1968). Motivational factors in vigilance: Effects of instructions on performance in a complex vigilance task. *Perceptual and Motor Skills, 26*, 783-786.

Luk, S. (1985). Direct observations studies of hyperactive behaviors. *Journal of the American Academy of Child Psychiatry, 24*, 338-344.

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

Luria, A. R. (1973). *The working brain*. New York: Basic Books.

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

Mackworth, J. F. (1970). *Vigilance and attention: A signal detection approach*. Harmondsworth, England: Penguin Books.

Mackworth, N. H. (1950). Researchers on the measurement of human performance. *Medical Research Council Special Report,* No. 268. London: Medical Research Council.

Mahan, S. M. (1996). *Relationships among measures used in the assessment of attention deficit hyperactivity disorder.* Unpublished doctoral dissertation, Indiana State University, Terre Haute.

Marks, D. J., Himelstein, J., Newcorn, H. H., & Halperin, J. M. (1999). Identification of ADHD subtypes using laboratory-based measures: A cluster analysis. *Journal of Abnormal Child Psychology, 27*, 167-175.

Marshall, R. M., Hynd, G. W., Handwerk, M. J., & Hall, J. (1997). Academic underachievement in ADHD subtypes. *Journal of Learning Disabilities, 30*, 635-642.

Matier-Sharma, K., Perachio, N., & Newcorn, N. (1995). Differential diagnosis of ADHD. *Child neuropsychiatry, 1,* 118-127.

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

Matthews, G., Davies, D. R., & Lees, J. L. (1988). *Arousal, extraversion, and the deployment of resources in sustained attention*. Poster presented at the 96th Annual Convention of the American Psychological Association, Atlanta, GA.

McClure, F. D., & Gordon, M. (1984). The performance of disturbed hyperactive and nonhyperactive children on an objective measure of hyperactivity. *Journal of Abnormal Child Psychology, 12*(4), 561-572.

McCracken, J. T. (1991). A two-part model of stimulant action on attention-deficit hyperactivity disorder in children. *Journal of Neuropsychiatry and Clinical Neuroscience, 3,* 201-20.

McGee, R. A., Clark, S. E., & Symons, D. K. (2000). Does the Conners' continuous performance test aid in ADHD diagnosis? *Journal of Abnormal Child Psychology, 28*(5), 415-424.

McGrath, J. J., Harabedian, A., & Buckner, D. N. (1968). Review and critique of the literature on vigilance performance (Technical Report 206, December, 1959). In *Studies of human vigilance: An omnibus of technical reports* (pp. 1-108). Goleta, CA: Human Factors Research.

McGuinness, D., & Pribram, K. (1980). The neuropsychology of attention: Emotional and motivational controls. In M. C. Wittrock (Ed.), *The brain and psychology* (pp.95-139). New York: Academic.

Mefford, I. N., & Potter, W. Z. (1989). A neuroanatomical and biochemical basis for attention deficit disorder with hyperactivity in children: A defect in tonic adrenaline mediated inhibition of locus coeruleus stimulation. *Medical Hypotheses, 29*, 33-42.

Mesulam, M. M. (1981). A cortical network for directed attention and unilateral neglect. *Annuals of Neurology, 10*, 309-325.

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

Mesulam, M. M. (1985a). Attention, confusional states and neglect. In M. M. Mesulam (Ed.), *Principles of behavioral neurology* (pp. 125-168). Philadelphia: Davis.

Mesulam, M. M. (1985b). *Principles of behavioral neurology.* Philadelphia: Davis.

Michael, R. I., Korman, R., Salzman, L. F., Borgstedt, A. D., & Dainer, K. B. (1981). Normalizing effects of methylphenidate on hyperactive children's vigilance performance and evoked potentials. *Psychophysiology, 18*, 665-677.

Milich, R., Loney, J., & Landau, S. (1982). The independent dimensions of hyperactivity and aggression: A validation with playroom observation data. *Journal of Abnormal Psychology, 91*, 183-198.

Milligan, G. W., & Cooper, M. C. (1987). Methodology review: Clustering methods. *Applied Psychological Measurement, 11,* 329-354.

Mirsky, A. F. (1987). Behavioral and psychophysiological markers of disordered attention. *Environmental Health Perspectives, 74,*191-199.

Mirsky, A. F. (1989). The neuropsychology of attention: Elements of a complex behavior. In E. Perecman (Ed.), *Integrating theory and practice in neuro-psychology* (pp. 75-91). Hillsdale, NJ: Erlbaum.

Mirsky, A. F. (1996). Disorders of attention: A neuropsychological perspective. In R. G. Lyon & N. A. Krasnegor (Eds.), *Attention, memory, and executive function* (pp. 71-96). Baltimore: Paul H. Brookes.

Mirsky, A. F., Anthony, B. J., Duncan, C. C., Ahearn, M. B., & Kellam, S. G. (1991). Analysis of the elements of attention: A neuropsychological approach. *Neuro-Psychology Review, 2*, 109-145.

Mirsky, A. F., Fantie, B. D., & Tatman, J. E. (1995). Assessment of attention across the lifespan. In R. L. Mapou & J. Spector (Eds.), *Clinical neuropsychological assessment: A cognitive approach* (pp. 17-48). New York: Plenum Press.

Morgan, A. E., Hynd, G. W., Riccio, C. A., & Hall, J. (1996). Validity of DSM-IV ADHD predominantly inattentive and combined types: Relationship to previous DSM diagnoses/subtype differences. *Journal of the American Academy of Child and Adolescent Psychiatry, 35*, 325-333.

National Council on Measurement in Education. (1999). *Standards for educational and psychological testing.* Washington, DC: Author.

Norman, D. A., & Shallice, T. (1986). Attention to action: Willed and automatic control of behavior: In R. J. Davidson, G. E. Schwartz, & D. Shapiro (Eds.), *Consciousness and self-regulation* (Vol. 4; pp. 1-18). New York: Plenum.

Nuechterlein, K. H. (1983). Signal detection in vigilance tasks and behavioral attributes of schizophrenic mothers and among hyperactive children. *Journal of Abnormal Psychology, 92*, 4-28.

Nuechterlein, K. H., Parasuraman, R., & Jiang, Q. (1983). Visual sustained attention: Image degradation produces rapid sensitivity decrement over time. *Science, 220*, 327-329.

O'Dougherty, M., Nuechterlein, & Drew, B. (1984). Hyperactive and hypoxic children: Signal detection, sustained attention, and behavior. *Journal of Abnormal Psychology, 93*, 178-191.

Parasuraman, R. (1979). Memory load and event rate control sensitivity decrements in sustained attention. *Science, 205*, 924-927.

Parasuraman, R. (1984a). Sustained attention in detection and discrimination. In R. Parasuraman & D.R. Davies (Eds.), *Varieties of attention* (pp. 243-271). Orlando, FL: Academic Press.

Parasuraman, R. (1984b). The psychobiology of sustained attention. In J.S. Warm (Ed.), *Sustained attention in human performance* (pp. 61-100). New York: Wiley.

Parasuraman, R., & Davies, D. R. (1977). A taxonomic analysis of vigilance performance. In R. R. Mackie (Ed), *Vigilance: Theory, operational performance and physiological correlates* (pp. 559-574). New York: Plenum.

Parasuraman, R., Warm, J. S., & See, J. E. (1998). Brain systems of vigilance. In R. Parasuraman (Ed.). *The attentive brain* (pp. 221-256). Cambridge, MA: The MIT Press.

Parry, P. A., & Douglas, V. I. (1983). Effects of reinforcement on concept identification in hyperactive children. *Journal of Abnormal Child Psychology, 11*, 327-340.

Pascaulvaca, D. M., Wolf, L. E., Healey, J. M., Tweedy, J. R., & Halperin, J. M. (1988). *Sex differences in attention and behavior in school-age children*. Paper presented at the Meeting of the International Neuropsychological Society, New Orleans.

Passler, M., Isaac, W., & Hynd, G. (1985). Neuropsychological development of behavior attributed to frontal lobe functioning in children. *Developmental Neuropsychology, 1*, 349-370.

Posner, M. I. (1990). Hierarchical distributed networks in the neuropsychology of selective attention. In A. Caramazza (Ed.), *Cognitive neuropsychology and neurolinguistics* (pp. 187-210). Hillsdale, NJ: Lawrence Erlbaum.

Posner, M. I. (1995). Attention in cognitive neuroscience: An overview. In M. S. Gazzaniga (Ed.), *Handbook of cognitive neuroscience* (pp. 615-624). Cambridge, MA: MIT Press.

Posner, M. I., Inhoff, A. W., & Fredrich, F. S. (1987). Isolating attentional systems: A cognitive-anatomical analysis. *Psychobiology, 15*, 107-121.

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

Pribram, K. H., & McGuinness, D. (1975). Arousal, activation, and effort in the control of attention. *Psychological Review, 82*, 116-149.

Quay, H. C. (1997). Inhibition and attention deficit hyperactivity disorder. *Journal of Abnormal Child Psychology, 25*, 7-13.

Rapoport, J. L., Buchsbaum, M. S., Weingarten, H., Zahn, T. P., Ludlow, C., & Mikkelsen, E. J. (1980). Dextroamphetamine: Its cognitive and behavioral effects in normal and hyperactive boys and normal men. *Archives of General Psychiatry, 32*, 933-943.

Rasile, D., Burg, J., Burright, R. G., & Donovick, P. J. (1995). The relationship between performance on the Gordon Diagnostic System and other measures of attention. *International Journal of Psychology, 30*, 35-45.

Riccio, C. A., Reynolds, C. R., & Lowe, P. A. (2001). *Clinical applications of continuous performance tests: Measuring attention and impulsive responding in children and adults.* New York: John Riley & Sons, Inc.

Riccio, C. A., Reynolds, C. R., Lowe, P. A. & Moore, J. J. (2002). The continuous performance test: A window on the neural substrates for attention? *Archives of Clinical Neuropsychology, 17*(3), 235-272.

Rickman, D. L. (2001). The effect of classroom-based distraction on continous performance test scores of ADHD and non-ADHD children. *Dissertation Abstracts International: Section B: The Sciences & Engineering, 61*(10-B), 5578.

Robbins, P. M. (1992). A comparison of behavioral and attentional functioning in children diagnosed as hyperactive or learning disabled. *Journal of Abnormal Child Psychology, 20*, 65-82.

Robbins, T. W. & Everett, B. J. (1995). Arousal systems and attention. In *Handbook of cognitive neuroscience*, M. S. Gazzaniga (Ed.), pp. 703-720. Cambridge, MA: MIT Press.

Ross, A. O., & Pelham, W. E. (1981). Child psychopathology. *Annual Review of Psychology, 32*, 243-278.

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

Routh, D. K., & Schroeder, C. S. (1976). Standardized playroom measures as indices of hyperactivity. *Journal of Abnormal Child Psychology, 4*, 199-207.

Rueckert, L., & Grafman (1996). Sustained attention deficits in patients with right frontal lesions. *Neuropsychologia, 34*, 953-963.

Rutter, M. (1981). Psychological sequelae of brain damage in children. *American Journal of Clinical Neuropsychology, 138*, 1533-1544.

Sachdev, R. (1999). Attention deficit hyperactivity disorder in adults. *Psychological Medicine,29*, 507-514.

Sandford, J. A., & Turner, A. (1995). *Manual for the Integrated Visual and Auditory (IVA) Continuous Performance Test*. Richmond, VA: BrainTrain.

Sattler, J. M. (1990). *Assessment of children.* (3rd ed.) San Diego, CA: Jerome M. Sattler.

Schachar, R., & Logan, G. D. (1990). Impulsivity and inhibitory control in normal development and childhood psychopathology. *Developmental Psychology, 26*, 710-720.

Schachar, R., Logan, G., Wachsmuth, R., & Chajezyk, D. (1988). Attaining and maintaining preparation: A comparison of attention in hyperactive, normal, and disturbed control group. *Journal of Abnormal Child Psychology, 16*, 361-378.

Schachar, R., Sandberg, S., & Rutter, M. (1986). Agreement between teachers' ratings and observations of hyperactivity, inattentiveness, and defiance. *Journal of Abnormal Child Psychology, 14*(2), 331-345.

Seidel, W. T., & Joschko, M. (1990). Evidence of difficulties in sustained attention in children with ADHD. *Journal of Abnormal Child Psychology, 18*(2), 217-229.

Semrud-Clikeman, M., Biederman, J., Sprich, S., Krifcher, B., Norman, D., & Faraone, S. (1992). Comorbidity between ADHD and learning disability: A review and report in a clinically referred sample. *Journal of the American Academy of Child and Adolescent Psychiatry, 31*, 439-448.

Semrud-Clikeman, M., Filipek, P. A., Biederman, J., Steingard, R., Kennedy, D., Renshaw,P., & Bekken, K. (1994). Attention-deficit hyperactivity disorder: Magnetic resonance imaging morphometric analysis of the corpus callosum. *Journal of the American Academy of Child and Adolescent Psychiatry, 33*, 875-881.

Sergeant, J. A. (1981). *Attentional studies in hyperactivity*. Groningen, The Netherlands: Veenstra.

Sergeant, J. A., Oosterlaan, J., & Van der Meere, J. (1999). Hyperactivity: passed the point of no return? In: Quay, J. C., Hogans, A. E. *Handbook of disruptive behavior disorders.* New York: Kluwer Academic/Plenum Publishers.

Sergeant, J. A., & Scholten, C. A. (1985). On resource strategy limitations in hyperactivity: Cognitive impulsivity reconsidered. *Journal of Child Psychology and Psychiatry, 26*, 97-109.

Shallice, T., & Burgess, P. W. (1991). Higher order cognitive impairments and frontal lobe lesions in man. In H. S. Levin, H. M. Eisenberg, & A. L. Benton (Eds.), *Frontal lobe function and dysfunction* (pp.125-138). New York: Oxford University Press.

Shallice, T., Burgess, P. W., Schon, F., & Baxter, D. M. (1989). The origins of utilization behavior. *Brain, 112*, 1587-1598.

Shapiro, S. K., & Garfinkel, B. D. (1986). The occurrence of behavior disorders in children: The interdependence of attention deficit disorder and conduct disorder. *Journal of the American Academy of Child Psychiatry, 25*, 809-819.

Shekim, W. O., Javid, J., Dekirmenjian, H., Chapel, J. L., & Davis, J. M. (1982). Effects of D-amphetamine on urinary metabolites of dopamine and norepinephrine in hyperactive boys. *American Journal of Psychiatry, 139*, 485-488.

Sieg, K. G., Gaffney, G. R., Preston, D. F., & Hellings, J. A. (1995). SPECT brain imaging abnormalities in attention deficit hyperactivity disorder. *Clinical Nuclear Medicine, 20*, 55-60.

Slicker, E. K. (1991). Validation of an objective measure of perseveration in children with attention deficit hyperactivity disorder. Unpublished Ph.D. dissertation, Texas A & M University, College Station, TX.

Sohlberg, M. M., & Mateer, C. A. (1989). *Attention process training*. Puyallup, WA: Association for Neuropsychological Research and Development.

Sternberg, S. (1969). Discovery of processing stages: Extensions of Donders' method. In *Attention and performance II,* A. Sanders (Ed.), pp. 276-315. Amsterdam: Elsevier.

Stroop, J. R. (1935). Studies of interference in serial verbal reaction. *Journal of Experimental Psychology, 18*, 643-662.

Stuss, D. T., & Benson, D. F. (1984). Neuropsychological studies of the frontal lobes. *Psychological Bulletin, 95*, 3-28.

Swanson, J., Posner, M. I., Cantwell, D., Wigal, S., Crinella, F., Filipek, P. et al.(1998). Attention-deficit/hyperactivity disorder: Symptom domains, cognitive processes, and neural networks. In R. Parasuraman, (Ed.), *The attentive brain* (pp. 445-460). Cambridge, MA; MIT Press.

Swets, J. A. (1964). *Signal detection and recognition by human observers.* New York: Wiley.

Swets, J. A. (1973). The relative operating characteristic in psychology. *Science, 182*, 990-1000.

Swets, J. A. (1984). Mathematic models of attention. In R. Parasuraman & D. R. Davies (Eds.), *Varieties of attention* (pp. 183-242). New York: Academic Press.

Sykes, D. H., Douglas, V. I., & Morganstern, G. (1973). Sustained attention in hyper-active children and the effect of methylphenidate (Ritalin). *Journal of Child Psychology and Psychiatry, 14*, 213-220.

Sykes, D. H., Douglas, V. I., Weiss, G., & Minde, K. K. (1971). Attention in hyperactive children and the effect of Ritalin. *Journal of Child Psychology and Psychiatry, 95*, 341-345.

Tannock, R. (1998). Attention deficit hyperactivity disorder: Advances in cognitive, neurobiological, and genetic research. *Journal of Child Psychology and Psychiatry, 39*, 65-99.

Thompson, R. W., & Nichols, G. T. (1992). Correlations between scores on a continuous performance test and parents' rating of attention problems and impulsivity in children. *Psychological Reports, 70*, 739-742.

Tucker, D. M., & Williamson, P.A. (1984). Asymmetric neural control systems in human self-regulation. *Psychological Review, 91*, 185-215.

Ullman, D. G., Barkley, R. A., & Brown, H. W. (1978). The behavioral symptoms of hyperkinetic children who successfully respond to stimulant drug treatment. *American Journal of Orthopsychiatry, 48*, 425-437.

Vygotsky, L. S. (1980). *Mind in society: The development of higher psychological process.* Cambridge, MA: Harvard University Press.

Ward, J. H. (1963). Hierarchical grouping to optimize an objective function. *Journal of the American Statistical Association, 58,* 236-244.

Warm, J. S. (1977). Psychological processes in sustained attention. In R. R. Mackie (Ed.), *Vigilance: Theory, operational performance and physiological correlates* (pp. 624-644).

Warm, J. S., & Jerison, H. J. (1984). The psychophysics of vigilance. In J. S. Warm (Ed.), *Sustained attention in human performance* (pp. 15-59). Chichester, UK: John Wiley & Sons.

Weinberg, W. A., & Harper, C. R. (1993). Vigilance and its disorders. *Behavioral Neurology, 11*(1), 59-78.

Weingartner, H., Rapoport, J. L., Buchsbaum, M. S., Bunney, W. E., Jr., Ebert, M. H. Mikkelson, E. J., et al. (1980). Cognitive processes in normal and hyperactive children and their responses to amphetamine treatment. *Journal of Abnormal Psychology, 89*, 25-37.

Weiss, G., & Hechtman, L. (1994). *Hyperactive children grown up*. (2nd Ed.). New York:Guilford Press.

Welsh, M., Pennington, B., & Grossier, D. (1991). A normative developmental study of executive function: A window on the prefrontal function in children. *Developmental Neuropsychology, 7*, 131-139.

Wender, P. H. (1971). *Minimal brain dysfunction in children*. New York: Wiley.

Werry, J. S., Elkind, G. S., & Reeves, J. C. (1987). Attention deficit, conduct, oppositional, and anxiety disorders in children: III. Laboratory differences. *Journal of Abnormal Child Psychology, 15*, 409-428.

Williams, B. R., Ponesse, J. S., Schachar, R. J., Logan, G. D., & Tannock, R. (1999). Development of inhibitory control across the life span. *Developmental Psychology, 35*, 205-213.

Yeo, R. A., Hill, D. E., Campbell, R. A., Vigil, J., Petropoulos, H., Hart, B., Zamora, L., & Brooks, W. M. (2003). Proton magnetic resonance spectroscopy investigation of the right frontal lobe in children with attention-deficit/ hyperactivity disorder. *Journal of the American Academy of Child and Adolescent Psychiatry, 42*(3), 303-310.

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

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

Zubin, J. (1975). Problem of attention in schizophrenia. In M. L. Kietzman, S. Sutton, & J. Zubin (Eds.), *Experimental approaches to psychopathology* (pp. 139-166). New York: Academic Press.

APPENDIX A

Diagnostic criteria for Attention-Deficit/ Hyperactivity Disorder according to the *Diagnostic and statistical manual of mental disorders* (4th edition text revision).

A. Either (1) or (2):

(1) six (or more) of the following symptoms of **inattention** have persisted for at least 6 months to a degree that is maladaptive and inconsistent with developmental level:

Inattention

(a) often fails to give close attention to details or makes careless mistakes in schoolwork, work, or other activities
(b) often has difficulty sustaining attention in tasks or play activities
(c) often does not seem to listen when spoken to directly
(d) often does not follow through on instructions and fails to finish schoolwork, chores, or duties in the workplace (not due to oppositional behavior or failure to understand instructions)
(e) often has difficulty organizing tasks and activities
(f) often avoids, dislikes, or is reluctant to engage in tasks that require sustained mental effort (such as schoolwork or homework)
(g) often loses things necessary for tasks or activities (e.g., toys, school assignments, pencils, books, or tools)
(h) is often easily distracted by extraneous stimuli
(i) is often forgetful in daily activities

(2) six (or more) of the following symptoms of **hyperactivity-impulsivity** have persisted for at least 6 months to a degree that is maladaptive and inconsistent with developmental level:

Hyperactivity

(a) often fidgets with hands or feet or squirms in seat
(b) often leaves seat in classroom or in other situation in which remaining seated is expected
(c) often runs about or climbs excessively in situations in which it is inappropriate (in adolescents or adults, may be limited to subjective feelings of restlessness)
(d) often has difficulty playing or engaging in leisure activities quietly
(e) is often "on the go" or often acts as if "driven by a motor"
(f) often talks excessively

Impulsivity

(g) often blurts out answers before questions have been completed
(h) often has difficulty awaiting turn
(i) often interrupts or intrudes on others (e.g., butts in to conversations or games)

B. Some hyperactive-impulsive or inattentive symptoms that caused impairment were present before age 7 years.

C. Some impairment from the symptoms is present in two or more settings (e.g., at school [or work] and at home).

D. There must be clear evidence of clinically significant impairment in social, academic, or occupational functioning.

E. The symptoms do not occur exclusively during the course of a Pervasive Developmental Disorder, Schizophrenia, or other Psychotic Disorder and are not better accounted for by another mental disorder (e.g., Mood Disorder, Anxiety Disorder, Dissociative Disorder, or a Personality Disorder).

Code based on type:

314.01 Attention-Deficit/ Hyperactivity Disorder, Combined Type: if both Criteria A1 and A2 are met for the past 6 months
314.00 Attention-Deficit/ Hyperactivity Disorder, Predominantly Inattentive Type: if Criterion A1 is met but Criterion A2 is not met for the past 6 months
314.01 Attention-Deficit/ Hyperactivity Disorder, Predominantly Hyperactive Impulsive Type: if Criterion A2 is met but Criterion A1 is not met for the past 6 months

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