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**Finger Localization
as a test of
Posterior Callosal Functioning
in a
Clinically-Referred Sample
of
ADHD Adults**

by

Maria Tsiakas

A thesis submitted to the
Department of Psychology
in partial fulfillment of
the requirements for the degree of

Master of Arts
Carleton University
Ottawa

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The undersigned recommend to the
Faculty of Graduate Studies and Research

Acceptance of the thesis:

Finger localization as a Test of Posterior Callosal Functioning
in a Clinically-Referred Sample of ADHD Adults

submitted by

Maria Tsiakas

in Partial fulfillment of the requirements for
the degree of Master of Arts

A handwritten signature in black ink, appearing to be 'K. ...', written over a horizontal line.

Chair

A handwritten signature in black ink, appearing to be 'Shelly Parlor', written over a horizontal line.

Thesis Supervisor

Carleton University

October, 2000

Abstract

Attention deficit hyperactivity disorder (ADHD) is the most common neurobehavioural disorder diagnosed in childhood. One theory that has been proposed to account for ADHD involves callosal dysfunction. The corpus callosum (CC) interconnects regions of the brain hypothesized to be involved in ADHD and has also been implicated in attention, a core cognitive deficit in ADHD. Several magnetic resonance imaging (MRI) studies have demonstrated that anterior and posterior regions of the CC are smaller in ADHD patients in comparison with controls, supporting the theory of callosal dysfunction. In this study, the finger localization test (FLT) was utilized to assess posterior callosal functioning in a clinically-referred sample of male ADHD adults ($n = 27$) and in a normal control group of adult males ($n = 26$). Compared with the control participants, the ADHD group made significantly more errors in the crossed condition (which requires efficient functioning of the CC) of the FLT, but not the uncrossed condition. The results of this study provide further support for the notion of callosal dysfunction in ADHD.

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Attention Deficit Hyperactivity Disorder (ADHD) is the most common neurobehavioural disorder diagnosed in childhood (Epstein, Shaywitz, Shaywitz, & Woolston, 1991; Shaywitz, Fletcher, & Shaywitz, 1994; Castellanos, 1997) and accounts for just below 50% of all clinical referrals for children (Offord, Boyle, & Szatmari, 1987). It typically appears between 2 and 7 years of age and it can affect children throughout the life span from pre-school to elementary school, and into adulthood (Wender, 1995). The prevalence of ADHD in school-aged children is estimated to be between 3-5%. As many as 50 to 65% of children diagnosed with ADHD will continue to exhibit symptoms in adolescence and adulthood (Barkley, 1998) and up to 30% will continue to display the full syndrome (Klein & Mannuzza, 1991; Weiss & Hechtman, 1993). Reviews of the literature indicate that scientific research in the area of ADHD is rapidly proliferating as interest in the nature of this disorder increases (Epstein et al., 1991; Shaywitz et al., 1994).

Although the organic etiology of ADHD is unknown, there are a number of theories under investigation. One recent theory which has been proposed to account for ADHD posits callosal dysfunction (Hynd, Semrud-Clikeman, Lorys, Novey, Eliopoulos, & Lyytinen, 1991; Giedd, Castellanos, Casey, Kozuch, King, Hamburger, & Rapoport, 1994; Semrud-Clikeman, Filipek, Biederman, Steingard, Kennedy, Renshaw, & Bekken, 1994; Baumgardner, Singer, Denckla, Rubin, Abrams, Colli, & Reiss, 1996; Lyoo, Noam, Lee, Lee, Kennedy, & Renshaw, 1996) and will be the major focus of the proposed research. Specifically, this project will investigate callosal functioning in clinically-referred adults with ADHD using a behavioural measure known as the Finger Localization Test (Geffen, Nilsson, Quinn, & Teng, 1985). General issues central to the

ADHD literature and pertinent to the present thesis will be addressed prior to discussing in greater detail the putative role of the corpus callosum in ADHD. In particular, the definition, diagnosis, assessment, and treatment of ADHD will be reviewed as well as the most popular alternative theories of ADHD, cognitive deficits associated with the condition, co-morbidity, DSM-IV subtypes of ADHD, and gender differences. Although the present research focuses on adults with ADHD, most research on ADHD is based on children and consequently, the child literature will be selectively reviewed in order to clarify and better understand the considerably smaller literature on adults.

General Issues in the Study of ADHD

The Definition and Diagnosis of ADHD

Establishing a precise definition of ADHD and determining a concise set of symptoms for the diagnosis of ADHD has been problematic. A review of the nomenclature used to describe ADHD in the Diagnostic and Statistical Manual of Mental Disorders (DSM) from 1957 to the present, illustrates this lack of consensus on the definitive diagnosis of ADHD (Goodman & Poillion, 1992). For example, in the first DSM (American Psychiatric Association (APA), 1957), the clinical characteristics of ADHD were subsumed under Organic Brain Syndromes. DSM-II employed the term Hyperkinetic Reaction to Childhood (APA, 1968). In DSM-III, the term Attention Deficit Disorder (ADD) was adopted and two categories were used to delineate ADHD: Attention Deficit Disorder with Hyperactivity (ADD+H), and Attention Deficit Disorder without Hyperactivity (ADD-H) (APA, 1980). DSM-III-Revised (DSM-III-R) took a different approach and grouped the three categories of ADD of DSM-III into one all-encompassing category, ADHD (APA, 1987). A category of Undifferentiated Attention

Deficit Disorder was incorporated in DSM-III-R so as to include ADD-H children, but this diagnostic entity was vague and unclear, and therefore lacked clinical and research utility (Power & DuPaul, 1996).

The current scheme in DSM-IV has reintroduced the notion of subtypes and presently, three subtypes of ADHD are recognized: Predominantly Inattentive (ADHD-IA), Predominantly Hyperactive-Impulsive (ADHD-HI), and Combined (ADHD-C) (APA, 1994). This system identifies two symptom clusters (i.e., inattentive and hyperactive-impulsive) and there are nine behaviors in each cluster. A child must display a minimum of 6 of the 9 behaviors for at least 6 months to a degree inconsistent with their developmental level in order to be considered significantly inattentive and/or hyperactive-impulsive. The symptoms of ADHD must have appeared before the age of 7 and must be present across 2 or more settings (i.e., at school (or work) and at home). The symptoms must result in significant impairment in social, academic, and/or occupational functioning and the symptoms must not be better explained by another disorder, such as mood disorder, psychosis, or pervasive developmental disorder. The DSM-IV classification scheme will be used in the present paper unless otherwise specified.

Although the terms used to describe the clinical characteristics of ADHD have been changed three times by the American Psychiatric Association in 14 years (from DSM-III to DSM-IV), the features of inattention, hyperactivity, and impulsivity have remained consistent throughout (Hynd, Lorys, Semrud-Clikeman, Nieves, Huettner, & Lahey, 1991). As a result, Barkley and Grodzinsky (1994) appropriately define ADHD as a set of disorders comprised of developmentally inappropriate degrees of inattentiveness, hyperactivity, and impulsivity that arise in childhood (typically before the age of 7) and

are relatively chronic in nature (p. 121).

Inattention in children may manifest itself as difficulty sustaining attention in tasks or play activities, difficulty persisting on tasks until they are completed, frequently shifting from one uncompleted activity to another, failure to pay close attention to details or to make careless mistakes in schoolwork or other tasks, difficulty organizing tasks and activities, avoiding activities that require sustained mental effort or concentration (i.e., homework or paperwork), forgetfulness, and distractibility (APA, 1994). Hyperactivity may be manifest in children by squirming or fidgeting in their seat, excessive running or climbing in situations where it is inappropriate, difficulty playing quietly, talking excessively, appearing as though driven by a motor, and impatience. Impulsivity may manifest itself in children as a difficulty in delaying responses or blurting out answers before questions have been completed, difficulty in waiting their turn, and frequently interrupting others.

Previously, it was thought that the symptoms of ADHD would disappear with the onset of puberty (Munoz-Millan & Casteel, 1989). This belief most likely reflected the fact that motor hyperactivity (i.e., excessive running and climbing, difficulty remaining seated) is not as prominent in adults as in children (Downey, Stelson, Pomerleau, & Giordani, 1997; Spencer, Biederman, Wilens, & Faraone, 1998). Recently however, many clinical and epidemiological longitudinal follow-up studies have confirmed the notion that the majority of children with ADHD continue to exhibit symptoms, although somewhat modified, into adolescence and adulthood (Wender, 1995; Searight, Nahlik, & Campbell, 1995; Weynandt, Linterman, & Rice, 1995; Zuddas, Ancilleta, Muglia, & Cianchetti, 2000). Studies of adults retrospectively diagnosed as having had childhood

ADHD have also provided validity to the ADHD syndrome in adulthood (Goldman, Genel, Bezman, & Slanet, 1998; Biederman, Wilens, Mick, Faraone, & Spencer, 1998; Downey et al., 1997; Murphy & Barkley, 1996). Although systematic epidemiological data on the prevalence of ADHD in the adult population is lacking, it is estimated that 1-3% of adults may have ADHD (Biederman, 1998; Wilens, Biederman, Spencer, & Prince, 1995; Shaffer, 1994).

Achenbach, Howell, McConaughy, and Stanger (1994) have suggested that the manifestations of childhood ADHD symptoms change in adulthood, such that, for example, motor hyperactivity in childhood is translated in adulthood into irresponsibility and difficulty in making decisions. It has also been suggested that the symptoms of hyperactivity in childhood take the form in adulthood of feelings of restlessness and difficulty engaging in quiet, sedentary activities (APA, 1994). Although motor hyperactivity may be less prominent in adults, the manifestations of inattention and impulsivity often have serious implications for this age group as they may affect an individual's ability to function in higher education, at work, and in social relationships (Biederman, Faraone, Spencer, Wilens, Norman, Lapey, Mick, Lehman, & Doyle, 1993; Dulcan, Dunne, Ayres, Arnold, Benson, Bernet, Bukstein, Kinlan, Leonard, Licamele, McClellan, Sloan, & Miles, 1997).

The adult version of ADHD has not been formally recognized in DSM-IV however; the adoption of age-neutral wording in this latest version recognizes the potential for the lifelong persistence of the disorder into adulthood (Ranseen, Campbell, & Baer, 1998; Feifel, 1996). Furthermore, the term Attention-Deficit/Hyperactivity Disorder, In Partial Remission in DSM-IV may be applied to adolescents and adults who

had the full ADHD syndrome as children and although they have current symptoms that cause functional impairment, they no longer meet the full criteria (APA, 1994; Spencer et al., 1998). To qualify for the diagnosis of ADHD as an adult, ADHD symptoms must have been experienced as a child (APA, 1994). Due to the lack of formal diagnostic criteria in adults with ADHD and hence, the difficult task of retrospectively diagnosing childhood ADHD, the diagnosis of adult ADHD is complicated and controversial (Ranseen et al., 1998).

As with ADHD children, the common presenting symptoms for ADHD adults are marked inattention, poor concentration, easy distractibility, day dreaming, forgetfulness, and a frequent shift in activities. ADHD adults also report marked impulsivity, intrusiveness, low frustration tolerance, and extreme impatience. Although far less common in adults than in children with ADHD, hyperactivity, restlessness, and fidgeting may be present (Wilens et al, 1995).

In addition to and as a result of ADHD, children and adults with the disorder are often plagued with academic, occupational, and social difficulties. Due to their impaired concentration and attention, poor self-organization, poor discipline, procrastination, and difficulty structuring time and activities, children with ADHD are often viewed as lazy, unmotivated, selfish, thoughtless, immature, irresponsible, and reckless (Barkley, 1998; Searight et al., 1995). As a result, they experience harsh judgments, punishments, moral denigration, social rejection, and ostracism (Barkley, 1998). Not surprisingly, children with ADHD are often at a greater risk for low academic achievement, grade retention in school, and school suspensions and expulsions than non-ADHD children (APA, 1994; Abikoff, Gittleman, & Klein, 1980). In addition, children with ADHD often suffer

significant social and emotional difficulties that impair the formation and maintenance of relationships with classmates, peers, parents, and teachers (Abikoff et al., 1980; APA, 1994).

Similarly, adults with ADHD evidence greater self-reported psychological maladjustment, lower vocational and educational achievement, lower socioeconomic status, more frequent job changes, more changes of residence, more speeding violations and automobile accidents, more multiple marriages, and more difficulty maintaining close interpersonal relationships than normal adults (Ranseen et al., 1998; Downey et al., 1997; Eppright, Tevendale, & Kelley, 1997; Murphy & Barkley, 1996; Searight et al., 1995; Fargason & Ford, 1994; Biederman et al., 1993).

Methodological Issues/Concerns in the Study of ADHD

Before reviewing theories of ADHD, several methodological issues in the ADHD literature will be briefly discussed. In reviewing the literature on ADHD, it is important to note that most studies have been based on samples consisting of: (1) boys, (2) in pre-adolescence and more recently, adulthood (very few in adolescence or pre-school years), (3) with co-morbid Conduct Disorder or Oppositional Defiant Disorder, and (4) who are predominantly Caucasian and of Western ethnicity (Popper, 1997). In this paper, research in gender differences and co-morbidity in children and adults with ADHD will be reviewed. However, the present paper will not examine ADHD in pre-school children and adolescents, and in non-Caucasian individuals of non-Western ethnicity.

In general, most children with ADHD are cared for by primary care providers such as pediatricians and family practitioners (Goldman et al., 1998). Psychiatrists and neurologists typically see a more severely impaired and complex group of individuals

with ADHD (e.g., those with significant co-morbidity) than do primary care providers (Goldman et al., 1998). Research in ADHD is typically conducted using either epidemiological (population-based) or clinically-referred samples. Groups identified as ADHD in studies may vary as a reflection of the source of the sample (Lahey, Pelham, Schaughency, Atkins, Murphy, Hynd, Russo, Hartdagen, & Lorys-Vernon, 1988). For example, epidemiological studies minimize referral, help-seeking, and clinical setting biases typically seen in clinically-referred samples (August & Garfinkel, 1993). Further, ADHD children from clinically-referred samples tend to have a more severe form of ADHD and more co-morbid disorders than those ADHD children from epidemiological samples. Therefore, results from clinically-referred patient samples cannot always be applied to population-based samples, and vice versa (Ellenberg & Nelson, 1980).

Murphy and Barkley (1996) have noted that children with ADHD followed into adulthood may have a more severe form of the disorder than adult self-referrals seeking help for themselves. Adult self-referrals may also have higher intelligence, more education, higher socioeconomic status, and a greater self-awareness of the significance of their condition than those children with ADHD followed to adulthood.

Studies conducted with clinically-referred samples and epidemiological samples have yielded valuable information in the understanding of ADHD and how it affects individuals, and they are both a necessary part of research. It is important however, to understand the inherent biases of each sample type and to keep these in mind when interpreting results.

Theories of ADHD

Several theories have been suggested to explain the occurrence of ADHD. The

two main causes that are best supported by evidence are genetics and neurochemical dysfunction. The empirical support for these two theories will be briefly reviewed.

Although its precise etiology remains unknown, evidence from epidemiological and clinical family, adoption, and twin studies has indicated that genetic risk factors may be operant in ADHD (Faraone, Biederman, Chen, Krifcher, Keenan, Moore, Sprich, & Tsuang, 1992; Faraone & Biederman, 1994; Jiang, Xin, Wu, Lin, Qian, Ren, Tang, & Wang, 2000). Family studies derived from clinically referred samples have consistently supported the assertion that ADHD runs in families (Faraone & Biederman, 1994). These studies have demonstrated that compared to the relatives of non-ADHD children, the relatives of ADHD children are at a higher risk for ADHD and other co-morbid psychiatric disorders such as depression and anxiety disorders (Faraone, Biederman, Mennin, Gershon, & Tsuang, 1996). Twin studies have reported up to 92% concordance in monozygotic twins versus 33% in dizygotics. This data suggests that ADHD is highly heritable (Goodman & Stevenson, 1989; Gilger, Pennington, & DeFries, 1992; Gillis, Gilger, Pennington, & DeFries, 1992). Adoption studies have shown that the adoptive relatives of ADHD are less likely to have ADHD than biological relatives (Cantwell, 1975). The findings from these studies converge to suggest that there is a substantial genetic contribution to the etiology of ADHD and that transmission in families is mediated, in part, by genetic factors (Spencer et al., 1998; Muglia, Jain, Macciardi, & Kennedy, 2000).

Although the evidence is inconclusive, it is hypothesized that a decreased turnover of dopamine and norepinephrine plays a central role in the neurophysiology of ADHD - hence the theory of neurochemical dysfunction (Rostain, 1991). Dopamine and

norepinephrine are the predominant neurotransmitters in the connecting pathways vital for maintaining attention (Rostain, 1991). Stimulant medications are often effective in the treatment of ADHD. They are structurally similar to endogenous catecholamines (i.e., dopamine and norepinephrine) and serve to increase the release of catecholamines and allow the brain to inhibit unwanted behaviors resulting in increased attention (Ballard, Bolan, Burton, Snyder, Pasterczyk-Seabolt, & Martin, 1997; Castellanos, 1997; Pliszka, McCracken, & Maas, 1996). The fact that stimulant medications act as dopaminergic and noradrenergic agonists provides support for the hypothesis of neurochemical dysfunction in ADHD (Pliszka et al., 1996).

Other origins or causes of ADHD have also been suggested including birth complications such as low birth weight (Botting, Powls, & Cooke, 1997) and perinatal/prenatal insults to the brain (Barkley, DuPaul, & McMurray, 1991), environmental causes including poor diet, food additives, vitamin deficiencies, allergies, and toxicity (e.g., exposure to lead and/or toxic waste) (Goodman & Poillion, 1992; Rice, 2000), and maladjustment due to psychological adversity (Cantwell & Baker, 1992). Although these factors have been implicated in the etiology of the disorder, none has emerged as a necessary and sufficient cause that can account for most cases of ADHD (Faraone & Biederman, 1998).

Neuroimaging and neuropsychological studies confirm several brain areas that are affected in ADHD, notably in frontal and parietal brain regions. The empirical evidence for involvement of these two regions, as well as a third structure, the corpus callosum, will be discussed later in the paper.

Treatment of ADHD

The prescription of stimulant medication is the most common form of treatment for ADHD in children and adults (Goldman et al., 1998). Methylphenidate is the most commonly prescribed stimulant for ADHD and accounts for more than 90% of the stimulant use in the United States (Goldman et al., 1998).

The widespread clinical use of stimulant drugs for the treatment of ADHD stems from their demonstrated short-term efficacy, compared to placebo conditions, in dramatically reducing the core behavioural symptoms of hyperactivity, impulsivity, and inattention in both children and adults, in addition to improving associated cognitive and social difficulties (Goldman et al., 1998; Spencer, Biederman, Wilens, Harding, O'Donnell, & Griffin, 1996; Richters, Arnold, Jensen, Abikoff, Conners, Greenhill, Hechtman, Hinshaw, Pelham, & Swanson, 1995; Barkley, 1998). However, long-term efficiency of stimulant medication has not been demonstrated for any domain of functioning in ADHD (Jacobvitz, Stroufe, Stewart, & Leffert, 1990). Thus further study is required to determine if stimulant medications will be as effective longitudinally as they have been shown to be in the short-term.

For patients with ADHD who are intolerant or unresponsive to stimulants, tricyclic antidepressant medications have also proven useful in clinical practice (Spencer et al., 1996). However, the literature suggests that although tricyclic antidepressants are as effective as stimulants in treating the social and emotional difficulties associated with ADHD, they may be less effective in improving cognitive impairments (e.g., sustained attention) (Cyr & Brown, 1998).

The role of non-pharmacological, psychosocial interventions in the treatment of

ADHD remains equivocal (Cyr & Brown, 1998; Barkley, 1998). Psychosocial interventions that have been systematically explored include parent and teacher training in behaviour management techniques, social skills and cognitive training, intensive summer treatment programs, and parent, family and teacher counseling (Richters et al., 1995; Barkley, 1998; Cyr & Brown, 1998).

Multi-modal therapy is another form of treatment for ADHD, combining multiple forms of intervention. Typically, it involves integrating pharmacotherapy (e.g., stimulants or tricyclic antidepressants) with one or more psychosocial interventions tailored to the particular needs of the ADHD individual and their family (Satterfield, Satterfield, & Schell, 1987). For a comprehensive review of treatments for children and adults with ADHD, see Barkley's recent review (Barkley, 1998).

Assessment of ADHD

The DSM-IV diagnostic criteria for ADHD emphasize particular behavioural characteristics and the core symptoms of the disorder are defined on the basis of these characteristics (Shaywitz & Shaywitz, 1991). There are no confirmed laboratory measures that can diagnose ADHD (APA, 1994). There are no physical features or psychological tests that have been established as pathognomonic for ADHD (Biederman, 1998; Agerter & Rasmussen, 2000). Rather, the assessment and diagnosis of ADHD in children and adults is dependent upon a thorough developmental and psychiatric history obtained through multiple informants (parents, teachers, etc.) as well as a comprehensive review of functioning in the core areas of ADHD known to be deficient (Epstein, Conners, Sitarenios, & Erhardt, 1998; Dulcan et al., 1997; Hinshaw, 1994; Fargason & Ford, 1994).

Semi-structured interviews with the individual and their parents, teachers and/or significant others (i.e., persons who currently spend the most time with the ADHD individual) and behaviour rating scales and checklists are the most commonly used assessment tools (Goldman et al., 1998). Medical and school records (when available) are also a potential source of information, especially in the case of adults where a diagnosis of childhood ADHD must be established retrospectively (Eppright et al., 1997). The use of multiple informants is important as they help to provide a clearer picture of relevant symptomatology and to reduce the risk that the individual with ADHD will exaggerate or minimize their difficulties (Eppright et al., 1997; Agerter & Rasmussen, 2000).

Behavioural rating scales and checklists are useful for assessing both current and past behaviours as reported by the patient and other informants through the use of normative comparisons, and may assess the extent to which diagnostic criteria are met (Eppright et al., 1997; DuPaul, Guevremont, & Barkley, 1991). Numerous parent and teacher rating scales have been used to assess the presence, severity and course of ADHD in children. The most widely used questionnaires include the Child Behavior Checklist (CBCL; Achenbach, 1991), Conners Parent Rating Scale (CPRS; Conners, 1989) and Conners Teacher Rating Scale (CTRS; Conners, 1989). These three scales generally show good agreement among parent, teacher, and youth ratings (Biederman, Keenan, & Faraone, 1991) and have excellent psychometric properties (Conners, Sitarenios, Parker, & Epstein, 1998).

There are fewer scales for ADHD adults as the recognition of the adult form of the disorder is fairly recent. However, several do exist. These include the Wender Utah

Rating Scale (WURS; Ward, Wender, & Reimherr, 1993), the Brown Adult Attention Deficit Disorder Scale (BAADS; Brown, 1995), and most recently, the Conners' Adult ADHD Rating Scale (CAARS; Conners, Erhardt, & Sparrow, 1998). These scales and their psychometric properties will be discussed later in the paper.

Neuropsychological performance is not diagnostic of ADHD. However, objective measures such as neurological examinations (e.g., magnetic resonance imaging) and neuropsychological testing are useful in assessing the symptoms and specific deficits in ADHD, and may support the diagnosis (Biederman, 1998; Fargason & Ford, 1994). Furthermore, the study of neuropsychological function in ADHD has considerable importance both as an empirical study of performance and as a window into hypothesized alterations in brain function (Seidman, Biederman, Faraone, Milberger, Norman, Seiverd, Benedict, Guite, Mick, & Kiely, 1995).

Neuropsychological assessment is an important and very useful part of rigorous patient evaluation as it can help to determine the patient's specific cognitive strengths and weaknesses and aids in directing compensatory training activities and vocational counseling (Barkley, 1998; Feifel, 1996). Moreover, neuropsychological measures directly assess performance, are easy to administer and score, and can be given longitudinally to assess their stability over time (Seidman, Biederman, Faraone, Weber, Mennin, & Jones, 1997).

Furthermore, since other psychiatric and neurological disorders (i.e., traumatic brain injury, depression) may result in inattention or disturbances in activity level (Brown, Voigt, & Elksnin, 1996), it is important to ensure from the patient's history, neurological examination, and neuropsychological test performance that no other

condition is primarily responsible for producing the clinical picture (Goldman et al., 1998).

Cognitive Deficits in ADHD

Cognitive deficits, particularly impairments in attention and executive functioning are hypothesized to be a core part of ADHD (Barkley, 1998; Barkley, Grodzinsky, & DuPaul, 1992) and are believed to play a major role in the life difficulties of children and adults with the disorder (Seidman et al., 1995; Barkley et al., 1992; Barkley, 1998). Cognitive functioning has been extensively investigated in ADHD in an effort to better understand the nature of possible mechanisms underlying the clinical disorder (Shaywitz & Shaywitz, 1991). Some researchers have reported that inattention is the primary cognitive deficit in ADHD while others have contended that a more widespread deficit, executive dysfunction, is the core problem. These two areas of research will be discussed here.

Attention and ADHD

Many investigators have reported that children and adults with ADHD have attentional difficulties in comparison with age-matched controls (Swaab-Barneveld, de Sonnevile, Cohen-Kettenis, Gielen, Buitelaar, Van Engeland, 2000; Brandeis, van Leeuwen, Rubia, Vitacco, Steger, Pascual-Marqui, & Steinhausen, 1998; Jenkins, Cohen, Malloy, Salloway, Johnson, Penn, & Marcotte, 1998; Overtom, Verbaten, Kemner, Kenemans, van Engeland, Buitelaar, Camfferman, & Koelega, 1998; Ben-Artzy, Glicksohn, Soroker, Margalit, & Myslobodsky, 1996; Seidman et al., 1995). Attention is a multifactorial cognitive construct (Denckla, 1996) and neuropsychological testing helps to determine which aspects of attention are disturbed (Fargason & Ford, 1994). The main

attentional deficits that have been implicated in ADHD are sustained and selective attention.

Typically, when a task requires attentional performance over a relatively long period of time, it is said to require sustained attention (Cohen, 1993). The Continuous Performance Task (CPT) is a computer-based test that has been used to quantify the deficit in sustained attention in children and adults with ADHD (van der Meere, Shalev, Borger, & Gross-Tsur, 1995). The CPT requires an individual to sustain attention in order to respond (by pressing a response button on a keyboard) to an infrequently occurring stimulus (Eliason & Richman, 1987). Performance on the CPT is typically scored in terms of correct responses (or missed responses/omission errors) as an indication of inattention, and incorrect/false responses as an indication of impulsivity. Stimuli can be presented in different modalities; the majority of studies using the CPT paradigm with ADHD children have used visual stimuli (Riccio, Cohen, Hynd, & Keith, 1996). Some studies have not found evidence of a sustained attention deficit in ADHD children and adults with the visual CPT (Epstein et al., 1998; Holdnack, Moberg, Arnold, Gur, & Gur, 1995). However, other researchers have reported a deficit in sustained attention in child and adults with ADHD with the use of both visual and auditory CPT paradigms (Borger & van der Meere, 2000; Borger, van der Meere, Ronner, Alberts, Geuze, & Bogte, 1999; Semrud-Clikeman & Wical, 1999; Overtom et al., 1998; Seidman, Biederman, Weber, Hatch, & Faraone, 1998; Mariani & Barkley, 1997; van der Meere et al., 1995; Carter, Krener, Chaderjian, Northcutt, & Wolfe, 1995; Arcia & Gualtieri, 1994).

Selective attention can be conceptualized as a process by which some

informational elements are given priority over others (Cohen, 1993). With the use of various tasks that assess the ability to selectively allocate attention to specific locations in the visual field, several researchers have reported that children and adults with ADHD display deficits in selective attention (Sheppard, Bradshaw, Mattingley, & Lee, 1999; Jonkman, Kemner, Verbaten, Koelega, Camfferman, Gaag, Buitelaar, & van Engeland, 1997; Carter et al., 1995; Swanson, Posner, Potkins, Bonforte, Youpa, Fiore, Cantwell, & Crinella, 1991; Voeller & Heilman, 1988). Several investigators have also reported that ADHD individuals are slower and/or less accurate at auditory selective attention tasks as well (Jonkman et al., 1997; Satterfield et al., 1994; Satterfield, Schell, Nicholas, Satterfield, & Freese, 1990; Holcolmb, Ackerman, & Dykman, 1985).

Therefore, although there is evidence contrary to the notion of attention as a core cognitive deficit in ADHD, a large literature suggests that it is indeed present as a deficit in sustained and/or selective attention. Swanson et al. (1991) noted that the complex construct of attention might be dependent on several different elementary attentional operations functioning as a network. Each operation may be localized to specific brain areas and neural loci. In addition to several other regions of the brain, the parietal lobes, the frontal lobes, and the corpus callosum have been implicated in the neural network of attentional functioning (Williams, Stott, Goodyer, & Sahakian, 2000; Nigg, Swanson, & Hinshaw, 1997; Shelley-Tremblay & Rosen, 1996; Rueckert & Grafman, 1996; Malone et al., 1994; Grodzinsky & Diamond, 1992; Voeller, 1991; Heilman et al., 1991; Posner, Peterson, Fox, & Raichle, 1988), as will be discussed further in the paper.

Executive Function and ADHD

In contrast to the researchers who believe that attention is the primary deficit in

ADHD, some researchers attribute the core behavioral deficits that characterize ADHD to a general impairment of higher-order cognitive functions, collectively referred to as executive function (Barkley, 1997a & b; Barkley, Klopowitz, Anderson, & McMurray, 1997; Denckla, 1996; Castellanos, 1997; Shue & Douglas, 1992). Executive function (EF) is a term that refers to a domain of cognitive abilities that includes self-regulation, set maintenance, working memory, selective inhibition of responding, response preparation, cognitive flexibility, and organizing time and space (Schaughency & Hynd, 1989). These abilities are believed to be dependent on the full mature integrity of anterior subcortical structures (primarily the basal ganglia) and their interconnections with the frontal lobes, as well as the frontal lobes themselves (Denckla, 1993). A deficit in EF may account for the deficits in sustained and selective attention observed in ADHD and is also associated with difficulties in the inhibition of verbal and non-verbal responses, as well as problems in the areas of organization, planning, self-monitoring, goal-directed behaviour, verbal fluency, problem solving, strategic memorization, and the capacity to shift response sets during tasks (Denckla, 1993; Barkley et al., 1992; Goodyear & Hynd, 1992).

The various tests that have been employed to assess EF in ADHD patients are those that have been demonstrated to be sensitive to frontal lobe injuries in adults. They include for example, the Wisconsin Card Sorting Task, Stroop Color-Naming Task, Rey Osterrieth Complex Figure Task and various verbal fluency tasks (Holdnack et al., 1995; Mariani & Barkley, 1997; Seidman et al., 1998). A description of these tests is beyond the scope of the present paper. However, all of these frontal lobe tests are purported to assess one or more aspects of EF (e.g., response inhibition, cognitive flexibility, working

memory). Numerous studies have reported EF deficits in ADHD children and adults when compared with healthy age-matched controls (Ross, Harris, Olincy, & Radant, 2000; Seidman, Biederman, Monuteaux, Weber, & Faraone, 2000; Schachar, Mota, Logan, Tannock, & Klim, 2000; Castellanos, Marvasti, Ducharme, Walter, Israel, Krain, Pavlovsky, & Hommer, 2000; Vaidya, Austin, Kirkorian, Ridlehuber, Desmond, Glover, & Gabrielli, 1998; Seidman, Biederman, Faraone, Weber, & Ouellette, 1997; Mariani & Barkley, 1997; Carte, Nigg, & Hinshaw, 1996; Oosterlan & Sergeant, 1996; Reader, Harris, Schuerholz, & Denckla, 1994; Koziol & Stout, 1992).

In summary, it is apparent that two different viewpoints exist in the understanding of the primary cognitive deficit in ADHD. These alternative views are not incompatible however. The more recent theory of executive dysfunction expands the conceptualization of ADHD from one solely involving problems of attention to one which incorporates inattention as part of a broader, higher-order system of executive dysfunction (Mariani & Barkley, 1997; Carte et al., 1996; Shue & Douglas, 1992). The theory of executive dysfunction takes into account the other prominent behavioural symptoms and cognitive deficits observed in ADHD, namely impulsivity and disinhibition. In terms of this thesis, both views are important for understanding the effects of the postulated underlying neuroanatomical abnormalities. These will be discussed later in the paper.

Co-morbidity in ADHD

Co-morbidity is defined as the occurrence of two or more psychiatric disorders in the same individual (Shaywitz et al., 1994). In recent years, epidemiological and clinical studies have established that ADHD is frequently co-morbid with other disorders

(Biederman, Newcorn, & Sprich, 1991). In fact, it has been reported that as many as 65% of children with ADHD have or will have one or more co-occurring conditions (Dulcan et al., 1997; Biederman, Newcorn et al., 1991).

The presence of co-morbid disorders can complicate or obscure the accurate diagnosis and effective treatment of ADHD in children and adults (Hornig, 1998). The identification of co-morbid disorders is therefore important because a child with ADHD and a co-occurring disorder may have a different clinical presentation, life course, and response to treatment than a child with ADHD alone (Pliszka, 1998). In childhood, ADHD frequently co-occurs with two externalizing disorders (i.e., Conduct Disorder and Oppositional Defiant Disorder), two internalizing disorders (i.e., depression and anxiety disorders), Tourette's syndrome, and learning disabilities (Biederman, Newcorn et al., 1991).

A diagnosis of Conduct Disorder is based on the observation of a repetitive and persistent pattern of behaviour in which the basic rights of others or age-appropriate social norms or rules are violated. Aggressive conduct, destructiveness or vandalism, deceitfulness or theft, and serious rule violations are the main areas in which an individual with Conduct Disorder (usually under 18 years of age) must demonstrate persistent difficulties (Davison & Neale, 1994). A diagnosis of Oppositional Defiant Disorder requires a recurrent pattern of negative, hostile, and defiant behaviour toward authority figures demonstrated by frequent arguing, temper outbursts, overt non-compliance with rules, and blaming others for their behaviour (Searight et al., 1995). Clinical and epidemiological studies have reported that approximately 20% of ADHD children have co-morbid Conduct Disorder and 40% meet the criteria for Oppositional

Defiant Disorder (Richters et al., 1995; Biederman, Newcorn et al., 1991).

Major depression co-occurs with ADHD in 20 to 30% of ADHD children as reported in epidemiological (Szatmari, Boyle, & Offord, 1989) and clinical studies (Biederman & Newcorn, 1990). Studies with clinical and epidemiological samples (Lahey et al., 1988; Pliszka, 1989) have reported that anxiety disorders co-occur in 25% of children with ADHD. Tourette's syndrome is a tic disorder which usually first appears in childhood and is characterized by chronic motor and vocal tics that wax and wane in severity, frequency and complexity over time (Sherman, Shepard, Joschko, & Freeman, 1998; Baumgardner et al., 1996). Tourette's syndrome co-occurs in 7% of individuals with ADHD (Comings, 1997). The incidence of learning disabilities is also increased in individuals with ADHD (Shaywitz et al., 1994). Approximately 25% of children with ADHD also have a learning disability in language, reading, writing, spelling or math (Castellanos, 1997; Richters et al., 1995). ADHD can generally be distinguished from these co-morbid disorders by the presence of persistent attentional deficits, impulsivity, and hyperactivity (Wender, Reimherr, Wood, & Ward, 1985).

Faraone, Biederman, Keenan, and Tsuang (1991) argue that methods from genetic epidemiology can help to clarify the relationships among co-morbid disorders. A detailed analysis of the genetic studies of ADHD and its co-morbid disorders is beyond the scope of this paper. However, recent genetic data from family, twin and adoption studies suggest that ADHD and depressive disorders share common familial vulnerabilities (Biederman, Mick, & Faraone, 1998), ADHD and anxiety disorders may be transmitted independently (Biederman, Newcorn, & Sprich, 1991), ADHD and learning disabilities are genetically independent conditions (Faraone, Biederman,

Lehamn, Keenan, Norman, Seidman, Kolodny, Kraus, Perrin, & Chen, 1993), and ADHD and Conduct Disorder may comprise a distinct subtype (Pliszka, 1998; Faraone, Biederman, Jetton, & Tsuang, 1997). Further, while some investigators have reported a genetic relationship between Tourette's syndrome and ADHD (Knell & Comings, 1993), others have disputed this notion (Pauls, Hurst, & Kruger, 1986).

Depressive and anxiety disorders and learning disabilities are common in adults with ADHD (Shaywitz & Shaywitz, 1991; Biederman et al., 1993). In addition, adults with ADHD have been reported as having a high degree of co-morbidity with substance use disorders and Antisocial Personality Disorder (Feifel, 1996; Murphy & Barkley, 1996; Biederman et al., 1993). Antisocial Personality Disorder involves a pervasive pattern of disregard for and violation of the rights of others that begins in childhood or early adolescence and continues into adulthood (APA, 1994). To receive the diagnosis, an individual must be at least 18 years of age and must have had a history of some symptoms of Conduct Disorder before age 15 years of age.

In a recent clinically-referred sample of 78 adults with ADHD described by Downey et al. (1997), 37% had a current depressive disorder, 33% had histories of alcohol abuse/dependence, 20.5% had histories of drug abuse/dependence, and 12.8% had Antisocial Personality Disorder. Longitudinal studies suggest that 18% to 32% of ADHD children grow up to have Antisocial Personality Disorder and that 10% to 16% develop substance use disorders (Weiss & Hechtman, 1993; Klein & Mannuzza, 1991). While ADHD by itself conveys some risk for antisocial behaviour, it is when the ADHD child has co-morbid Conduct Disorder that the risk of adult Antisocial Personality Disorder rises sharply (Mannuzza, Klein, & Bonagura, 1991). In support of this notion,

most studies have found that antisocial behaviour is rare in later life without early conduct problems (Dulcan et al., 1997).

Relative to ADHD without co-morbidity, the presence of ADHD with co-morbid disorders further increases the risk of behavioural and emotional problems at subsequent stages of life (Goldman et al., 1998). The detection of co-morbid conditions is critical, because many cause symptoms that overlap with those of ADHD, but require distinct treatment (Feifel, 1996). However, several studies have suggested that the cognitive deficits in children with ADHD are the result of ADHD and not the associated co-morbid disorders (Biederman, Faraone, Milberger, Guite, Mick, Chen, Mennin, Marrs, Ouellette, Moore, Spencer, Norman, Wilens, Kraus, & Perrin, 1996; Seidman, Biederman, Faraone, Weber, Mennin et al., 1997; Seidman et al., 1995). A discussion of the treatment of ADHD with co-morbid conditions is beyond the scope of the present paper (for a recent review, see Wilens & Biederman, 1992).

In sum, although the high level of co-morbidity in ADHD may lead to problems with differential diagnosis, Biederman, Newcorn et al. (1991) have suggested that these difficulties do not invalidate the diagnosis of ADHD. Rather, further examination of the patterns of co-morbidity in ADHD could help to revise and improve existing methods of classification.

DSM-IV Subtypes of ADHD

The validity of subtypes of ADHD has been a constant subject of debate as evidenced by the changes in the classification of the disorder in the DSM. The majority of research conducted thus far has utilized subjects with diagnoses based on DSM-III-R ADHD that did not recognize subtypes. Currently, there is much interest in examining

the differences between the DSM-IV subtypes in both children and adults (Denckla, 1996).

Research on subtypes of ADHD collected prior to the publication of DSM-IV primarily focused on the comparison of ADD subjects with and without hyperactivity (based on DSM-III). It has been argued that there exists a strong association between this distinction and that between the inattentive and combined-types from DSM-IV (McBurnett, Pfiffner, Willcutt, Tamm, Lerner, Ottolini, & Furman, 1999; Dulcan et al., 1997). As such, in this review, in order to discuss the DSM-III literature and relate it to the present nosology, ADD+H and ADD-H will be considered to be roughly equivalent to ADHD-C and ADHD-IA, respectively.

Goodyear and Hynd (1992) reviewed 21 behavioral studies that compared ADD+H and ADD-H. They concluded that ADD+H children were less popular, more self-destructive, more likely to have a co-diagnosis of Conduct Disorder, and generally had more behavioral problems than children with ADD-H. In contrast, ADD-H children tended to be more socially withdrawn and self-conscious, and had a slower cognitive tempo and a higher incidence of learning disabilities. Similarly, Cantwell and Baker (1992) found systematic differences between ADD+H and ADD-H children. Hyperactive participants ($n = 221$) had higher rates of Conduct Disorder and were more likely to be impulsive, distractible, and aggressive. In contrast, non-hyperactive participants ($n = 42$) were more likely to have diagnoses of anxiety and depression, and higher rates of learning disabilities. Both groups showed social dysfunction, but hyperactive children tended to show higher rates of peer rejection and unpopularity, whereas non-hyperactive subjects showed increased social withdrawal.

More recently, in a school-based population, Gaub and Carlson (1997a) compared the behavioural characteristics of ADHD children (subdivided into the three DSM-IV subtypes) with those of normal children. They reported that ADHD-C children presented with the most pervasive pattern of difficulties with more severe reported impairments in attention, social problems, anxiety, and depression. Children in the ADHD-IA subtype displayed fewer externalizing behaviors and behaved more appropriately than ADHD-C or ADHD-HI children. However, they were still impaired in these domains compared to normal controls. Children with ADHD-HI were no different from controls in learning and internalizing measures although they had significantly poorer ratings in the social functioning, externalizing, and attention domains.

Several other investigators have found that children with ADHD-C exhibit higher rates of Conduct Disorder and Oppositional Defiant Disorder and other externalizing and internalizing behaviour problems than ADHD-IA children (Lalonde, Turgay, & Hudson, 1998; Wolraich, Hannah, Baumgaertel, & Feurer, 1998; Morgan, Hynd, Riccio, & Hall, 1996; Baumgaertel, Wolraich, & Dietrich, 1995), and higher lifetime rates of language and tic disorder and higher rates of counseling and treatment than ADHD-IA and ADHD-HI children.

ADHD-IA children have been noted by several researchers to have fewer externalizing disorders (Lalonde et al., 1998; Morgan et al., 1996; Baumgaertel et al., 1995), more academic problems and difficulties in school (i.e., repeating a grade) (Baumgaertel et al., 1995; Faraone, Biederman, Weber, & Russell, 1998; McBurnett et al., 1999), and a higher proportion of females to males than the other two subtypes (Fargason & Ford, 1994; Wolraich et al., 1998; Lalonde et al., 1998; Arnold, 1996;

Goodyear & Hynd, 1992). Children with ADHD-HI have been reported as having a higher prevalence of Conduct Disorder than the other two subtypes (Lalonde et al., 1998) and in general, to show more externalizing behaviour than ADHD-IA children (Baumgaertel et al., 1995).

In summary, ADHD-C children can be characterized as hyperactive and impulsive with conduct problems and aggressive behaviour, likely to experience social rejection. ADHD-IA children are more likely to be characterized as sluggish, distractible, and are more likely to daydream, repeat a grade, exhibit depressed mood and symptoms of anxiety, and to suffer social neglect. The data on ADHD-HI children suggests that they may have a clinical profile of high rates of externalizing problems, low rates of internalizing problems, and low rates of academic problems (Gaub & Carlson, 1997a; Wolraich et al., 1998).

In non-referred populations, ADHD-IA appears to be the most prevalent subtype followed by ADHD-C and ADHD-HI. In clinical samples, ADHD-C appears to be the most prevalent followed by ADHD-IA and ADHD-HI (Faraone et al., 1998; Lalonde et al., 1998; Gaub & Carlson, 1997a; Wolraich et al., 1998; Baumgaertel et al., 1995). This discrepancy may be due to less frequent referrals to clinics among those with ADHD-IA compared to those who demonstrate more hyperactive-impulsive and externalizing behaviours. Although ADHD-HI individuals demonstrate hyperactive-impulsive symptoms and externalizing behaviours, they may be referred less frequently because they have fewer associated difficulties (e.g., academic problems) than ADHD-C individuals.

Potentially different neural regulatory-control systems may be implicated in the

DSM-IV subtypes. The attempt to distinguish between the subtypes with neuropsychological tests however has not proven unequivocally successful (Barkley et al., 1992; Goodyear & Hynd, 1992; Barkley & Grodzinsky, 1994). While several studies have reported that ADD+H and ADD-H do not differ on measures of attention (Lahey, Schaughency, Hynd, Carlson, & Nieves, 1987; Holcolmb et al., 1985), other investigators have reported differences. For example, several researchers have reported that predominantly hyperactive-impulsive symptoms are associated with sustained attention deficits and executive dysfunction, characterized by impairments in inhibitory control, while children with predominantly inattentive symptoms may be characterized more by deficits in selective attention and a more sluggish cognitive processing tempo (Barkley, 1998; Barkley & Grodzinsky, 1994; Malone, Kershner, & Swanson, 1994; Goodyear & Hynd, 1992; Voeller, 1991; Barkley et al., 1991; Heilman, Voeller, & Nadeau, 1991; Sergeant & Scholten, 1985; Berry, Shaywitz, & Shaywitz, 1985). Barkley et al. (1991) and Pennington (1991) have also reported that there are fewer stimulant responders in ADHD-IA in comparison with ADHD-HI children.

There has only been one study on DSM-IV subtypes in adults with ADHD. Hudziak, Heath, Madden, Reich, Bucholz, Slutske, Bierut, Neuman, and Todd (1998) examined DSM-IV subtypes of ADHD in a group of adolescent female twins ($N = 1,629$ pairs). They found that ADHD symptomatology was best conceptualized in their sample as existing in three separate domains of inattention, hyperactivity-impulsivity, and combined type problems corresponding to the DSM-IV subtypes. From this one study, it would appear that the DSM-IV scheme may be applicable to adults with ADHD. However, further research in this area is required to verify the validity of the DSM-IV

subtypes in adults with ADHD.

Gender Differences in ADHD

Although ADHD has been widely studied and it is widely accepted that more boys are diagnosed with ADHD than girls (Arnold, 1996), there has been comparatively little research on gender differences. In addition, very few studies have systematically examined ADHD solely in females (Hinshaw, 1994; Seidman, Biederman, Faraone, Weber, Mennin et al., 1997; Arnold, 1996; Arcia & Conners, 1998). This limited literature however, clearly indicates that ADHD females share with their male counterparts the prototypical features of the disorder (inattention, impulsivity, and hyperactivity) in addition to high rates of school failure, and co-morbidity with learning disabilities and depressive and anxiety disorders. However, rates of co-morbid Conduct Disorder and other externalizing symptoms are far less prevalent in females than in males (Barkley, 1998).

Recently, Gaub and Carlson (1997b) conducted a meta-analysis of 18 studies of gender differences in ADHD children. In general, it was reported that girls with ADHD displayed lower levels of hyperactivity, lower rates of Conduct Disorder and other externalizing behaviours, but greater intellectual impairment than boys with ADHD. Boys and girls did not differ on measures of impulsivity, academic performance, social functioning, fine motor skills, parental education or parental depression. Additional recent studies have confirmed that girls typically display less motor hyperactivity and exhibit less disruptive behavior in classroom settings in comparison with their male ADHD counterparts (de Haas, 1986; de Haas & Young, 1984; James & Taylor, 1990).

In the only study specifically examining cognition in ADHD girls, Seidman,

Biederman, Faraone, Weber, Mennin et al. (1997) compared 43 referred ADHD girls with 36 non-ADHD girls on various neuropsychological tasks. They reported that in concordance with studies with ADHD boys, ADHD girls were significantly more impaired on tasks of attention, intellectual performance, and academic achievement. However, ADHD girls did not perform worse than non-ADHD girls on measures of EF including response inhibition and cognitive flexibility. This latter finding is in contrast to what has been observed in studies with ADHD boys (e.g., Mariani & Barkley, 1997) who typically do display deficits in EF. Therefore, it is important for future researchers to replicate this study to see if similar findings would emerge.

In children with ADHD, male-to-female ratios typically range from 9:1 to 6:1 in clinical studies to 2:1 in population-based studies (Sharp, Walter, Marsh, Ritchie, Hamburger, & Castellanos, 1999; Arnold, 1996). This discrepancy between clinical and epidemiological rates is likely a function of boys being referred more frequently due to their higher rates of disruptive externalizing behaviours (Jensen, Martin, & Cantwell, 1997; Hinshaw, 1994). This also suggests that ADHD may be under-identified in girls (Biederman, 1998) and indicates that fewer girls are referred to clinics. Since fewer girls are referred, it is conceivable that the girls seen in treatment clinics represent the most severely affected girls. In their meta-analysis, Gaub and Carlson (1997b) provided support for this notion as they observed that girls with ADHD were less impaired than boys in the general population, but equally impaired in clinic-referred populations. Specifically, in non-referred populations, ADHD boys displayed higher levels of inattention, internalizing and externalizing disorders, aggression, and were less liked by peers than were ADHD girls. In contrast, in clinical samples, ADHD boys and girls had

similar impairment on all of these variables, with the exception of a non-significant trend for more severe attentional deficits among girls ($p < .056$).

The male preponderance in pediatric clinical samples of ADHD subjects appears to be less characteristic of adult ADHD samples (Murphy & Barkley, 1996). In fact, several investigators have reported that by adulthood, the ratio of men to women with ADHD is about equal (Feifel, 1996; Biederman, 1998). It has been suggested that differing developmental patterns may exist between boys and girls with ADHD resulting in a more equal ratio of ADHD cases between males and females in adulthood. For example, Brown, Madan-Swain and Baldwin (1991) found that with increasing age, girls with ADHD evidenced more severe problems in cognitive functioning, academic achievement and problems with peers compared to their male counterparts. Similarly, Berry et al. (1985) reported that as ADHD children grow older, the social problems of girls tend to increase relative to those of boys, for whom social status remains stable. However, in a follow-up study of ADHD subjects first observed in childhood, it was suggested that there was a better prognosis for ADHD females in adulthood such that they had lower rates of Antisocial Personality Disorder and substance abuse than their adult male counterparts (Klein, 1994 as cited in Arnold, 1996). Alternatively, it has been suggested that the more even sex ratio in adulthood is a result of more female self-referral (Arcia & Conners, 1998; Biederman, Faraone, Spencer, Wilens, & Mick, 1994).

Therefore, there appear to be no large discrepancies in the clinical manifestation of ADHD between boys and girls. To the extent that differences exist, ADHD boys may be characterized by more co-morbid aggressive externalizing disorders, while ADHD girls seem to be more severely cognitively affected with more attentional deficits

(Hinshaw, 1994). Although there is a preponderance of ADHD cases among males, there is little doubt in the literature that ADHD affects both genders in children and adults (Biederman, 1998).

The Neurobiology of ADHD

There are three major theories of the neurobiology of ADHD: frontal or fronto-striatal dysfunction, parietal dysfunction, and callosal dysfunction. Although the notion of dysfunction in the anterior regions of the brain has received the most support, each of these theories will be reviewed below. Particular emphasis will be placed on callosal dysfunction as it is the focus of the present research.

Frontal Dysfunction in ADHD

As noted, the pattern of behavioural and cognitive deficits in children and adults with ADHD resembles that which has been observed among adults with acquired frontal lobe damage (Arcia & Gualtieri, 1994; Benson, 1991). This has led many investigators to hypothesize that ADHD may be a neurological disorder primarily affecting the frontal cortex or the subcortical structures projecting to the frontal cortex (Mariani & Barkley, 1997; Shue & Douglas, 1992; Barkley et al., 1992).

Evidence of frontal and frontal-striatal dysfunction in ADHD comes from a variety of techniques including neuropsychological and cognitive testing, magnetic resonance imaging (MRI), single photon emission computed tomography (SPECT), positron emission tomography (PET), and functional magnetic resonance imaging (fMRI). These studies will be discussed in detail below.

The frontal lobes receive neural connections from various cortical and subcortical regions and also have efferent connections to various sensory, motor, and subcortical

areas (Nauta, 1971). Because of the complexity of frontal circuitry, whether the frontal dysfunction in ADHD is due to lesions of the frontal cortex or to brain areas with frontal projections has not yet been made clear. As such, the term fronto-striatal has been utilized by many investigators as a neuropsychological description of ADHD since it denotes cognitive or behavioural dysfunction that looks frontal, but may be influenced by abnormal subcortical (primarily the basal ganglia) projections (Shue & Douglas, 1992).

Neuropsychological and Cognitive Testing

Individuals with ADHD display deficits in attention and executive functioning. As noted earlier, in addition to the parietal lobes, the corpus callosum, and several other structures in the brain, the frontal lobes and anterior subcortical structures have been implicated in attentional functioning (Williams et al., 2000; Nigg et al., 1997; Shelley-Tremblay & Rosen, 1996; Rueckert & Grafman, 1996; Malone et al., 1994; Grodzinsky & Diamond, 1992; Voeller, 1991; Heilman et al., 1991; Posner et al., 1988). Specifically, it has been reported that both sustained and selective attention are subserved by the frontal lobes (Lewin, Friedman, Wu, Miller, Thompson, Klein, Wise, Hedera, Buckley, Meltzer, Friedland, & Duerk, 1996; Coull, Frith, Fackowiak, & Grasby, 1996; Posner & Dehaene, 1994; Koziol & Stout, 1992; Swanson et al., 1991; Posner & Peterson, 1990). The caudate nucleus, part of the basal ganglia, is also associated with attentional functioning in addition to behavioural inhibition (Mendez, Adams, & Lewandowski, 1986).

As mentioned earlier, although no precise neural substrate has been identified, EF is heavily dependent on the frontal lobes and anterior subcortical structures, and their interconnections with the frontal lobes. In support of this, patients with frontal lobes

lesions typically display deficits in EF (Benson, 1991; Heilman et al., 1991). As reviewed earlier, individuals with ADHD evidence EF deficits, providing further evidence for the notion of anterior dysfunction in ADHD.

MRI Studies

The majority of investigations of the neuroanatomy of ADHD have utilized MRI. MRI is a superior technique for the structural analysis of brain anatomy because exceptional anatomical detail can be achieved, with excellent grey and white matter differentiation, and without invasive radiation (Weight & Bigler, 1998; Kertesz, 1994). MRI studies find reduced volumes in a number of brain regions in ADHD individuals, including the frontal lobes and basal ganglia (Aylward, Reiss, Reader, Singer, Brown, & Denckla, 1996; Castellanos, Giedd, Eckbury, Marsh, Vaituzis, Kaysen, Hamburger, & Rapoport, 1994; Castellanos, Giedd, Marsh, Hamburger, Vaituzis, Dickstein, Sarfatti, Vauss, Snell, Lange, Kaysen, Krain, Ritchie, Rajapakse, & Rapoport, 1996; Filipek, Semrud-Clikeman, Steingard, Renshaw, Kennedy, & Biederman, 1997; Hynd, Hern, Novey, Eliopoulos, Marshall, Gonzalez, & Voeller, 1993; Mataro, Garcia-Sanchez, Junque, Estevez-Gonzales, & Pujol, 1997).

Small ($n = 7-11$) and larger samples ($n = 50-57$) of ADHD boys have been observed to have smaller anterior frontal cortices, particularly on the right side (Hynd, Semrud-Clikeman, Lorys, Novey, & Eliopoulos, 1990; Castellanos et al., 1994; Castellanos et al., 1996). Furthermore, Castellanos et al. (1994) and Castellanos et al. (1996) reported that relative to age-matched healthy controls, ADHD boys had a significantly smaller caudate nucleus and globus pallidus. The differences observed in these nuclei of the basal ganglia were predominantly in the right hemisphere. Similarly,

Filipek et al. (1997) and Casey, Castellanos, Giedd, Marsh, Hamburger, Schubert, Vauss, Vaituzis, Dickstein, Sarfatti, and Rapoport (1997) observed that ADHD children had smaller cerebral hemispheres in frontal regions as well as in basal ganglia structures compared to matched healthy controls, predominantly in the right hemisphere.

SPECT and PET Studies

SPECT and PET are techniques for imaging local metabolic and physiological brain functions of the brain and are used to construct tomographic representations of functional brain areas (Loring, 1999). Functional brain images are produced by using radioisotope tracers and measuring their perfusion in the brain (Weight & Bigler, 1998). They are both valuable research tools for the investigation of functional changes in the brain associated with cognitive activation tasks (e.g., sustained attention) (Loring, 1999).

Although the spatial resolution of SPECT and PET images is not as exquisite or as detailed as that obtained with MRI, these techniques allow for the measurement of metabolic activity in different regional brain areas through the detection of injected radiopharmaceuticals and their subsequent perfusion. The difference between PET and SPECT is that PET imaging allows for measures of temporal changes in addition to resting changes in brain structure, while SPECT is more widely used to measure steady states of the brain rather than activation of function (Weight & Bigler, 1998; Kertesz, 1994). Two studies, one using SPECT (Sieg, Gaffney, Preston, & Hellings, 1995) and one using PET (Amen & Carmicheal, 1997) have confirmed reduced metabolism in frontal and basal ganglia areas in ADHD individuals.

Functional brain studies of ADHD assessing either regional cerebral blood flow or glucose metabolism with PET are consistent with MRI studies implicating fronto-

striatal system pathophysiology in ADHD. However, not all agree on the locus of lateralization of the observed impairments (Faraone & Biederman, 1998). For example, Zametkin, Nordahl, Gross, King, Semple, Rumsey, Hamburger, and Cohen (1990) found that extensive frontal areas of the right hemisphere showed reduced metabolic activation during an auditory CPT in ADHD adults relative to normal controls. However, in a subsequent study, Zametkin, Liebenauer, Fitzgerald, King, Minkunas, Herscovitch, Yamada, and Cohen (1993) observed that ADHD children showed reduced glucose metabolism in left frontal areas during the performance of an auditory CPT.

Using xenon inhalation to study cerebral blood flow, Lou, Henriksen, Bruhn, Berner, and Nielsen (1989) and Lou, Henriksen, and Bruhn (1984) reported that low right frontal-striatal activity characterized ADHD children in comparison with controls. Both investigations reported increased flow in these areas after the administration of methylphenidate.

fMRI Studies

Functional MRI allows for structural and functional images by measuring focal changes in blood flow during cerebral activity. By comparing the brain images produced during periods of rest and activity (i.e., the execution of a task), increases in blood flow can be inferred (Weight & Bigler, 1998). Vaidya et al. (1998) found that relative to age-matched healthy controls, ADHD children had atypical frontal-striatal activation during the performance of response inhibition tasks (greater frontal activation on one task and reduced striatal activation on another). The authors reported that this abnormal activation in both areas perhaps reflected a greater inhibitory effect in the ADHD individuals. Similarly, abnormal activation in frontal areas has been observed in ADHD

individuals compared to normal controls during the performance of a task of EF requiring response inhibition (Rubia, 1997 as cited in Zametkin & Liotta, 1998).

In conclusion, there is considerable neuroradiological evidence using a variety of techniques that implicates frontal-striatal dysfunction and anterior structural brain abnormalities in children and adults with ADHD (Popper, 1997; Jenkins et al., 1998). Many of the findings suggest an unusual asymmetry of cerebral function in this population, with greater dysfunction generally noted in the right frontal lobe (Castellanos et al., 1996; Lou et al., 1989; Heilman et al., 1991) and one study finding greater left hemisphere dysfunction (Zametkin et al., 1993). The nature of this asymmetry remains equivocal at this time (Oades, 1998). However, it has been reported that the right frontal cortex plays an important role in response inhibition (Casey et al., 1997). Furthermore, although the frontal-striatal circuit is bilateral, it has been suggested that the right hemisphere circuit may be dominant in directing attention (Pardo, Fox, & Raichle, 1991; Corbetta, Miczin, Shulman, & Petersen, 1993).

Parietal Dysfunction in ADHD

Although the preponderance of existing evidence is consistent with frontal dysfunction, there are findings that suggest a parietal contribution to ADHD as well (Lou, Henriksen, & Bruhn, 1990). The parietal lobe theory of ADHD has been introduced more recently than the frontal lobe theory. Unlike the frontal lobe theory, however, the parietal lobe theory has received little attention. Nonetheless, evidence from a variety of techniques exists to support this theory.

Neuropsychological and Cognitive Testing

As mentioned earlier, the parietal lobes have been implicated in a putative neural

network subserving attention. Posner and colleagues (Posner & Dehaene, 1994; Posner & Petersen, 1990; Posner et al., 1988) and Weinberger and Harper (1993) reviewed evidence implicating neural systems in the right hemisphere, especially in the parietal lobe, in the capacity to sustain attention. Several other researches have reported that the two parietal lobes may play a significant role in both sustained and selective attention (Coull et al., 1996; Lewin et al., 1996; Mennemeier, Chatterjee, Watson, Wertman, Carter, & Heilman, 1994; Shaughency & Hynd, 1989). Accordingly, the attentional deficits in individuals with ADHD have been attributed to dysfunction in the parietal lobes (Brandeis et al., 1998; Overtom et al., 1998; Pliszka et al., 1996; Malone et al., 1994; Loge, Staton, & Beatty, 1990; Voeller & Heilman, 1988; Voeller, 1986; 1991; Mesulam, 1981).

Further evidence implicating the right parietal lobe in ADHD comes from poor performance on tasks known to be sensitive to parietal lobe damage. For example, on letter cancellation tasks, ADHD children have been found to make significantly more errors of omission and left-sided errors than non-ADHD children (Voeller & Heilman, 1988). Additionally, children with ADHD have demonstrated significantly poorer performance on mental rotation tasks (Snow, 1990), resembling the performance of patients with right parietal lobe damage (e.g., Dittunno & Mann, 1990).

MRI Studies

Abnormal findings on neuroanatomic studies of ADHD have not been limited to frontal-striatal regions of the brain. For example, Filipek et al. (1997) reported that compared to matched healthy controls, ADHD children had significantly smaller cerebral hemisphere volumes in parietal regions of the brain in addition to frontal regions. In

addition, a medication response was noted with ADHD children non-responsive to stimulants having the smallest measurements for these parietal regions.

PET Studies

Zametkin et al. (1990) found that compared with normal controls, extensive posterior parietal areas of the right hemisphere in ADHD adults showed reduced metabolic activation during a CPT, in addition to the right frontal area. Similarly, in addition to low activation in right frontal-striatal areas, Lou and colleagues (Lou et al., 1989; Lou et al., 1990) reported low bilateral posterior parietal activation in ADHD children relative to controls in their xenon inhalation studies.

FMRI Studies

In addition to frontal areas, abnormal activation in both parietal lobes in ADHD patients during the performance of a task requiring response inhibition has been reported with fMRI (Rubia, 1997 as cited in Zametkin & Liotta, 1998).

In conclusion, there appears to exist both structural and functional evidence for parietal lobe dysfunction in ADHD. More research is required however, to understand the nature of this dysfunction and to replicate the findings.

Therefore, both frontal dysfunction and/or posterior parietal dysfunction have been observed in ADHD. Several researchers have observed that the DSM-IV subtypes of ADHD are characterized by different cognitive deficits. It has been speculated that these cognitive differences are based on an anterior-posterior distinction between the subtypes (Matzow & Hynd, 1992). Specifically, as discussed in the section on DSM-IV subtypes, the deficits displayed in ADHD-IA appear to be in the domain of focused or selective attention and sluggish cognitive arousal whereas the reported problems of

attention in the ADHD subtypes with hyperactive-impulsive features (ADHD-HI and ADHD-C) are most likely due to poor behavioural inhibition (Barkley, 1997a; 1997b; Quay, 1997). Therefore, the inattention displayed in ADHD-HI and ADHD-C has been viewed as a secondary deficit resulting from poor behavioural inhibition which is linked to anterior dysfunction, while the deficits demonstrated in ADHD-IA seem to be more reflective of posterior dysfunction (Barkley, 1998; Filipek et al., 1997; Voeller, 1991; Lou et al., 1989; Schaughency & Hynd, 1989; Lou et al., 1984).

Unfortunately, no structural or functional imaging studies to date have looked at the DSM-IV subtypes in ADHD. It can be speculated that it is this failure to account for the subtypes that discrepancies in the imaging literature exist, with some finding evidence of frontal abnormalities and some parietal, and others reporting both frontal and parietal abnormalities. Further research is required to disentangle this issue.

Callosal Dysfunction in ADHD

In addition to the notion of frontal and parietal dysfunction, it has recently been suggested that there may exist structural abnormalities of the corpus callosum in ADHD. Before discussing this literature, general information on the structure and function of the corpus callosum will be reviewed.

The Corpus Callosum: Structure

The corpus callosum (CC) is the major white matter fiber tract connecting the left and right cerebral hemispheres of the brain (Burke & Yeo, 1994). It is composed of axons that have their cell bodies in the cortical layers of one or the other hemisphere (Gladstone & Best, 1985). Using MRI, the CC is clearly recognizable on the midsagittal (cross-sectional) plane (Rajapske, Giedd, Rumsey, Vaituzis, Hamburger, & Rapoport,

1996). Figure 1 shows an MRI of the CC in the midsagittal plane in the living human brain.

In the average human adult, the CC is composed of approximately 200 million interhemispheric fibers, which represents approximately 2-3% of all cortical fibers (LaMantia & Rakic, 1990). Callosal fibers contain both myelinated and unmyelinated axons that establish primarily homotopic connections along an anterior-posterior gradient (Driesen & Raz, 1995) paralleling that of the cerebral cortex (Pandya & Seltzer, 1986; de Lacoste, Kirkpatrick, & Ross, 1985). Homotopic connections interconnect homologous areas of the two cerebral hemispheres via the fibers of the CC. For example, the left frontal cortex projects axons through the anterior portion of the CC to the corresponding point in the right frontal cortex. Heterotopic connections also exist. These callosal neurons have axon collaterals in both hemispheres as do homotopic connections, but the target in the contralateral hemisphere is different from the site of origin (Guyton, 1991; Geffen et al., 1985).

For descriptive purposes and in order to examine the structure of the CC more precisely, researchers divide the CC into subregions. As there are no natural subdivisions of the CC, these subregions are arbitrary. Further, the number of subregions identified varies between studies. The CC, as viewed post-mortem or by MRI, is usually measured in the midsagittal view and is partitioned into 4, 5, 6 or 7 regions. Dividing the CC into seven subregions is a common method and is based on studies of the topography of callosal fibers in relation to cortical regions in rhesus monkeys (Pandya & Seltzer, 1986) and humans (Witelson, 1989; de Lacoste et al., 1985). Based on the data from Witelson (1989), Table 1 provides a summary of these subregions in rostral to caudal order, and

the cortical areas they interconnect. Figure 2 depicts the information contained in this table pictorially with an illustration of the brain.

The Corpus Callosum: Function

The majority of fibers in the CC appear to connect association areas rather than primary cortical regions or subcortical structures (Aboitiz, Scheibel, Fisher, & Zaidel, 1992a; La Mantia & Rakic, 1990). Although few primary sensory or motor fibers cross the CC (Kertesz, Polk, Howell, & Black, 1987), the CC is critical for the interhemispheric transfer of information and the facilitation of associative information between the hemispheres in healthy subjects (Hoptman & Davidson, 1994). In fact, the CC is the most important structure for the control of interhemispheric communication of sensory, motor and higher-order information between the left and right cerebral hemispheres (Clarke, McCann, & Zaidel, 1998; Hellige et al., 1998; Levander & Sonnesson, 1998).

The Corpus Callosum and Attention

In addition to the CC's well-established information transmitting role, another important function which has been ascribed to the CC and which is relevant to the present paper is the regulation of attention (Kinsbourne, 1973; Kreuter, Kinsbourne, & Trevarthen, 1972; Levy, 1985).

Several investigators have hypothesized and have demonstrated a privileged role of the CC in attentional mechanisms. In various clinical populations with callosal dysfunction or abnormalities, attentional deficits have been observed. One such population is the so-called "split-brain" patients whom have had the entire CC severed, usually as an elective surgery to treat epilepsy (see review by Kolb & Wishaw, 1990).

Dimond (1976; 1979) observed that relative to partial commissurotomy patients (i.e., those with in which the anterior two-thirds of the CC was sectioned), split-brain patients demonstrated a decline in performance on a task similar to a CPT that required sustained attention for a long period of time with little external stimulation. Similarly, Ellenberg and Sperry (1979) observed that split-brain patients demonstrated a weakened ability to sustain attention on a CPT-like task whereas normals were able to maintain their performance throughout the duration of the task. However, Ellenberg and Sperry (1979) observed that split-brain patients were able to sustain prolonged concentration and alertness on a sorting task for up to an hour. It has been suggested that continual proprioceptive and external stimulation may be necessary to maintain arousal and increase concentration and that with little external input over a protracted period of time, subjects must internally sustain their own attention (Rueckert, Sorensen, & Levy, 1994). It has been argued that the CC is one of several cortical mechanisms involved in mediating the focusing of attention in the absence of external reinforcement. A markedly weakened ability to sustain attention in the absence of external stimulation is typically observed in patients who have had their entire CC severed (Ellenberg & Sperry, 1979; Dimond 1976; 1979).

Wale and Geffen (1989) observed that split-brain patients performed less efficiently than healthy age-matched controls on tasks of both divided and focused attention. The divided attention task required the subjects to attend and respond to verbal targets (word pairs) presented orally with one word of each pair being spoken in a female voice and the other in a male voice. The focused attention task required the subjects to respond to targets (the same specified word pairs) in only one of the two voices. The

split-brain patients demonstrated poorer target detection and discrimination, and slower response to targets in both tasks. It was concluded that the results implicated the CC and hence, the unified functioning of both hemispheres as being critical for efficient attentional skills.

Rueckert and Grafman (1996) provided further evidence that adults with callosal lesions have attentional dysfunction. Compared with healthy controls, patients with anterior section of the CC showed a relative increase in the number of targets missed on a visual CPT between the first and second half of the task.

Therefore, it would appear from the above split-brain studies that sustained attention measured in a traditional way (e.g., a change in performance over the course of a block of trials, or between the first and second block) and sustained attention gauged as the ability to sustain attention over shorter periods of time with minimal external stimulation may be both dependent on an intact CC (Banich, 1998; Rueckert & Levy, 1996).

However, it must be noted that the split-brain studies suffer from several methodological limitations. Namely, very small samples of split-brain patients (i.e., $n < 10$) were obtained and essentially the same split-brain subjects have been employed in multiple studies by different authors. Furthermore, these split-brain individuals had not only undergone complete section of the CC, but of the hippocampal and anterior commissures as well, for the relief of severe and progressive epileptic attacks. It was noted in the Dimond studies (Dimond 1976; 1979) that extracallosal damage not associated with the commissurotomy was likely present premorbidly in all split-brain cases. Therefore, although the evidence supports the notion of a role for the CC in

attention, the results must be interpreted with caution.

Another clinical population, patients with Multiple Sclerosis, also evidence callosal abnormalities. These patients suffer from a reduction in myelination that affects the cerebrum and in particular, the CC, as indexed on autopsy and MRI. Attentional deficits are frequently observed in Multiple Sclerosis patients (Banich, 1998). Rao, Bernadin, Leo, Ellington, Ryan, and Bury (1989) observed that the ability to sustain attention (as assessed by multiple cognitive measures) was correlated with the size of the CC in their patients with Multiple Sclerosis as measured by MRI. Further, this correlation was significant even when the total amount of brain demyelination was considered.

Additional support for the notion that attention is mediated, in part, by the CC comes from studies with healthy individuals. Several researchers have demonstrated a relationship between the CC and attention in healthy individuals with intact CCs. For example, Rueckert et al. (1994) examined the relationship between callosal functioning and sustained attention in normal children. Based on their performance on a tachitoscopic task that involved comparing two visual stimuli when they were presented very briefly and simultaneously to both hemispheres, children were placed in one of two groups. Those with better performance on this task (i.e., more correct comparisons) were considered to have better callosal functioning and those with poor performance were considered to have poor callosal functioning. Those children with better callosal functioning performed a task of sustained attention with minimal external input more efficiently than those with poorer callosal functioning.

In an attempt to replicate the findings of Rueckert et al. (1994), Rueckert and

Levy (1996) conducted a study in which normal children were tested on a vigilance task to assess callosal involvement in sustained attention. The children were placed in either a high interhemispheric disruption (ID) group if they demonstrated a significant amount of disruption when they were required to perform various bimanual tasks that required efficient functioning of the CC, or a low ID group if they did not demonstrate a significant amount of disruption on this task. The bimanual tasks measured the child's ability to draw two different shapes (e.g., a circle and a line) with the right and left hand, simultaneously. Subjects in the high ID group performed less efficiently on the CPT-like vigilance task than those in the low ID group. This suggests that low ID subjects with more efficient callosal functioning had sufficient self-regulation to sustain attention in the absence of meaningful external output. The results of Rueckert et al. (1994) and Rueckert and Levy (1996) add to those of previous studies in clinical populations suggesting a relationship between the CC and attention.

The mechanism behind callosal involvement in attention is not clear (Rueckert & Levy, 1996). Banich (1998) has argued that the interactions between the hemispheres can influence attentional functioning because they modulate the processing capacity of the brain. These dynamic interactions between the hemispheres that affect attentional processing are proposed to occur through the CC. Similarly, Kinsbourne (1970; 1973; 1974) has hypothesized that the CC plays a role in the equilibrium of the attentional balance between the two hemispheres. Rueckert and Levy (1996) have suggested that the CC's involvement in attention is accomplished by either crosstalk between the two hemispheres facilitating the maintenance of arousal in the absence of external input, or possibly that one hemisphere is differentially involved in sustaining attention in the other.

From this review, it can only be concluded that the CC likely forms an integral part of a neural system that regulates attentional capacity in an as of yet, unknown way (Gladstone & Best, 1985; Banich, 1998).

The Corpus Callosum and Executive Function

There are no studies (to the author's knowledge) that have examined the role of the CC in EF. However, since the anterior portions of the CC have fibers which originate in the frontal and prefrontal areas and these areas form the largest lobe, it seems reasonable to postulate that the CC may be involved in the higher-order functions (i.e., EF) subserved by these cortical areas. Further research is required in this area.

Individual Differences in the CC: Gender, Age, and Handedness

There is great variability in the size of the CC in normal individuals (Burke & Yeo, 1994). Specifically, it has been reported that the size and/or shape of the CC varies as a function of sex, handedness and age. However, as will be reviewed, the literature in this area is fraught with a bewildering array of contradictory observations.

Sex differences in the size and shape of the CC were first observed by de Lacoste-Utamsing and Holloway (1982). They reported from an autopsy sample of nine males and five females that the splenium was both larger and more bulbous in females. Since this landmark study, many researchers have attempted to replicate this finding, the majority using MRI with healthy adult subjects. Several studies (Clarke, Kraftsik, Van der Loos, & Innocenti, 1989; Salat, Ward, Kaye, & Janowsky, 1997; Davatzikos & Resnick, 1998) have replicated the findings of de Lacoste-Utamsing and Holloway (1982) while others (Witelson, 1989; Steinmetz, Jancke, Kleinschmidt, Schlaug, Volkmann, & Huang, 1992; Clarke & Zaidel, 1994) have reported sex differences

favouring females in various other subregions of the CC (e.g., the isthmus). However, an almost equal number of studies have failed to find support for the notion of sex differences in the CC (Kertesz et al., 1987; Parashos, Wilkinson, & Coffey, 1995; Constant & Ruther, 1996) or have reported a smaller size of the CC (or its various subregions) in females relative to males (Denenberg, Kertesz, & Cowell, 1991; Rauch & Jenkins, 1994).

Several researchers have examined the effect of handedness on CC size. As with sex differences, the results have been conflicting. For example, the brains of left-handers have been observed post-mortem and using MRI, and appear to have larger CCs than those of the brains of right-handers (Witelson, 1989; Witelson & Goldsmith, 1991; Habib, Gayraud, Oliva, Regis, Salamon, & Khalil, 1991). Other researchers however, have found no variation in CC size due to handedness (Kertesz et al., 1987; Steinmetz et al., 1992; O'Kusky, Strauss, Kosaka, Wada, Li, Druhan, & Petrie, 1988; Clarke, Lufkin, & Zaidel, 1993).

Although the findings of both sex and handedness effects have been conflicting, there have been more consistent findings of sex and handedness interactions. For example, studies which have reported a larger CC in non-consistent right-handers versus consistent right-handers (Clarke & Zaidel, 1994; Witelson, 1989; Denenberg et al., 1991; Witelson & Goldsmith, 1991; Habib et al., 1991) have found this effect to be larger in males than in females (Aboitiz, 1992; Hoptman & Davidson, 1994). Non-consistent right-handers are defined as those who prefer to use their left hand to perform some tasks and are therefore not as strongly right-handed as consistent right-handers, who prefer to use their right hands for all tasks (Witelson, 1989).

Variations in callosal size have been reported as a function of age.

Developmentally, the CC increases in overall size from birth throughout childhood (Ratinckx, Brysbaert, & d'Ydewalle, 1997). There is a period of rapid growth until the age of 3 followed by a much slower rate of growth after this age until adolescence (Giedd, Rumsey, Castellanos, Rajapakse, Kaysen, Vaituzis, Vauss, Hamburger, & Rapoport, 1996; Pujol, Vendrell, Junque, Marti-Vilalta, & Capdevilla, 1993). This growth is due entirely to a myelination process that begins at birth when virtually all axons are unmyelinated, until adulthood where approximately 40% of axons are myelinated (Barkovich, Lyon, & Evrard, 1992). However, myelination of callosal axons is nearly complete by the age of 10 as has been shown in both autopsy and MRI studies (Hayakawa, Konishi, Matsuda, Kuriyama, Konishi, Yamashita, Okumura, & Hamanaka, 1989; Yakovlev & Lecours, 1967). With increasing age after that (between the ages of 20 and 80 years), the total area or length of the CC actually decreases as has been reported in several MRI studies with adult samples (Driesen & Raz, 1995; Parashos et al., 1995). From this we can infer that the CC is constantly undergoing changes in size and/or shape throughout the lifespan.

As with handedness, a relationship between age and sex and CC size has been noted by several researchers. Specifically, in an MRI study with healthy elderly subjects (older than 65 years of age), Salat et al. (1997) observed that women, but not men, showed an age-related atrophy of the anterior and middle sectors of the CC, but not the posterior sector. No age-related changes in CC size and/or shape were observed in men. Conversely, with post-mortem samples, Witelson (1989; 1991) reported that callosal size decreased with age in males, but not in females. Similarly, using MRI, Burke and Yeo

(1994) observed a greater decline in CC area with age among males than among females.

In sum, a review of individual differences in the CC due to sex, handedness and age yields a confusing picture with many contradictory findings. One reason for these divergent findings is the fact that when examining one variable (i.e., sex or handedness or age), a large majority of studies fail to account for the other two variables (Driesen & Raz, 1995; Bishop & Wahlsten, 1997). From the preceding review, this omission would quite likely lead to discrepant findings among studies.

Another factor in producing divergent findings is the methodological differences between studies, particularly those using MRI. For example, there are several different techniques for orienting the MRI image or brain specimen (Constant & Ruther, 1996). In addition, measurements of the CC are performed in a variety of ways in different studies (Parashos et al., 1995). As mentioned earlier, different researchers identify different numbers of subregions of the CC, and these subregions are based on arbitrary measurements or definitions. For example, for the splenium, some researchers define it as the posterior fifth or the posterior fourth, or they define it as a percentage of the total area of the CC. Further, in demarcating the subregions, there are different techniques including the straight line, curved line, bent line, and radial gravity methods (for a review of these methods, please see Bishop & Wahlsten, 1997).

Another area of debate concerns the use of absolute versus relative measures of the CC. Some researchers have found a correlation between brain size and CC size (Parashos et al., 1995; Witelson, 1989), while others have not (Kertesz et al., 1987). In comparing CC size between individuals, some researchers state that it is necessary to control for individual differences in brain size (area or volume or weight), while others

argue that it is necessary to have a correlation between some index of brain size and CC size before one is justified in using relative values. Similarly, some investigators use regional measurements of the CC relative to the total CC (area or length), while others use absolute measures of the subregions (Clarke & Zaidel, 1994).

Therefore, in conducting and interpreting MRI and autopsy studies of the morphology of the CC, these issues must be taken into account. In general however, the evidence suggests that differences in CC size and/or shape may be observed as a function of sex, handedness and age. Baumgardner et al. (1996) have suggested that size differences in the CC reflect differences in the size of the axons (which is related to the extent of myelination) or the number of axons. They have also indicated that differences in the size of the CC may be a function of the number of cortical neurons within homologous regions. Although it is unclear at this point whether a decreased CC area is caused by fewer axons or decreased axonal diameter related to deficient myelination (Riley, Mattson, Sowell, Jernigan, Sobel, & Jones, 1995), recent histological findings from two postmortem studies of human brains (Aboitiz et al., 1992a; Aboitiz, Scheibel, Fisher, & Zaidel, 1992b) have concluded that differences in callosal area among individuals or groups reflect decreasing numbers of fibers. Thus, according to these authors, a smaller CC is likely the result of less fibers and not smaller diameter fibers due to deficient myelination.

The Corpus Callosum and ADHD

Theoretically, structural abnormalities in the CC may result from primary damage to the tract itself or secondary damage from abnormalities in the regions of the brain which it connects (i.e., where the cell bodies lie), both of which may subsequently result

in difficulties on tasks of interhemispheric communication (Giedd et al., 1994; Hines, McAdams, Bentler, Chiu, & Lipcamon, 1992). It seems reasonable to postulate that since the frontal and parietal regions of the brain have been implicated in ADHD, perhaps there exist abnormalities in anterior and posterior regions of the CC that interconnect these regions between the two hemispheres. Furthermore, callosal and ADHD patients suffer from attentional deficits, and as reviewed earlier, the CC has been implicated in attention. Six recent MRI studies have examined the structure of the CC in ADHD children and adolescents. These studies will be reviewed in chronological order. Table 2 provides a summary of the pertinent methodological details of the studies to be reviewed.

As reviewed earlier, Hynd et al. (1990) reported that ADHD children had significantly smaller right frontal width measurements in comparison with normal controls. Based on this finding, in a subsequent study, Hynd, Semrud-Clikeman et al. (1991) hypothesized that the smaller right frontal width measurement was related to CC size, particularly in the anterior regions of the CC. They compared 7 outpatients diagnosed with DSM-III ADD-H to 10 non-disabled healthy controls they obtained from the community on MRI measurements of the CC. From 7.5 mm midsagittal slices of the CC, they divided the CC into 5 regions by: (1) drawing two vertical lines down from the anteriormost tip of the genu and down from the posteriormost tip of the splenium; (2) connecting these two vertical lines with a horizontal line and dividing this horizontal line into five equal segments which intersected the CC and; (3) drawing radial tangents from the five points to the middle point of the horizontal line. Regional measurements of the CC were adjusted relative to the total length of the CC for each individual. The

investigators reported that ADD+H children had significantly smaller area measurements of the genu (area 1), splenium (area 5) and region just anterior to the splenium (area 4) in comparison with controls.

Giedd et al. (1994) compared 18 children recruited from a day treatment program diagnosed with DSM-III-R ADHD to 18 normal controls recruited from the community on MRI measurements of the CC. The researchers obtained 1.5 mm midsagittal slices and divided the CC into seven regions using Witelson's (1989) technique. Briefly, they established the maximum length of the CC by joining the anterior and posteriormost points of the CC with a horizontal line. They then divided the CC into seven regions by drawing perpendicular lines from the horizontal line. First, they drew a perpendicular line to divide the CC into anterior and posterior halves, and then they drew two other perpendicular lines to separate the CC into thirds. Another line was drawn to mark the posterior fifth as the splenium, and the last perpendicular line was drawn through the anteriormost point of the inner convexity of the anterior callosum. The authors found that the rostrum and rostral body of the CC was significantly smaller in the ADHD group. Furthermore, these CC areas were still significantly smaller when considered as a ratio of the region to total CC area, and as a ratio of the region to total brain volume. Additionally, they reported that the size of the rostral body in all subjects was inversely correlated with hyperactivity/impulsivity on the Conners' Parent Rating Scale and Conners' Teacher Rating Scale; a smaller area was associated with a higher rating. No differences were found in any other subregions of the CC.

In view of the fact that posterior parietal regions of the brain have been implicated in ADHD, Semrud-Clikeman et al. (1994) hypothesized that the posterior

region of the CC would be compromised in children with ADHD. They compared MRI images of 15 outpatient children with DSM-III ADD+H to those of 15 normal controls obtained from the community. They obtained 1.00 mm thick midsagittal slices and sectioned the CC using Witelson's (1989) technique, as described above. The CC and its seven subregions were measured relative to brain size in each subject. The authors observed that ADD+H children had smaller spleniums compared with normal controls. Further, those ADD+H children who failed to respond to stimulants ($n=5$) had the smallest area measurements of the splenium when compared to the stimulant responders and normal controls. They reported no differences in any other subregions of the CC between the ADHD and control participants.

Confirmation of posterior callosal involvement in ADHD was provided by Lyoo et al. (1996). They obtained MRIs from 51 inpatient ADHD children and compared them with MRIs from 28 non-ADHD controls obtained from the hospital community. The ADHD children were evaluated either by chart review or the Diagnostic Interview Schedule for Children (DISC-C) as having DSM-III-R ADHD. Regional MRI measurements of the CC were obtained from 6mm thick midsagittal slices and the CC was partitioned for examination according to Witelson's (1989) technique. The investigators reported that ADHD children (according to both chart review and DISC-C diagnosis) had significantly smaller spleniums than non-ADHD controls. Additionally, those ADHD children diagnosed according to DISC-C diagnosis only, had a significantly smaller isthmus region than non-ADHD controls. This region is believed to interconnect between the two superior temporal cortices and the two posterior parietal cortices (Witelson, 1989; Hellige et al., 1998). No other subregions of the CC were found to be

different between the ADHD and non-ADHD participants.

Baumgardner et al. (1996) compared MRI images of the CC between four groups of children: 16 children with Tourette's syndrome recruited from a specialty clinic for Tourette's syndrome, 21 children with Tourette's syndrome recruited from the same clinic, but with a co-morbid diagnosis of ADHD by DSM-III-R criteria, 27 healthy control subjects, and 13 ADHD outpatients diagnosed with DSM-III-R criteria. The investigators obtained 3 mm thick midsagittal slices of the CC and the outline of the CC was circumscribed manually using a trackball device. A line was drawn through the center of the CC throughout the length of the CC and this line was divided into five regions. Based on a segmentation procedure, properties such as area of each subregion, bending angle of CC, and centerline length were computed. How these properties were computed however, was not described in the study. The authors found that the ADHD group had significantly smaller rostral bodies compared with the other three groups with no differences in any other CC subregions.

Castellanos et al. (1996) compared MRI images of the brains of 55 healthy control children and adolescents recruited from the community to those of 57 children and adolescents with DSM-III-R ADHD recruited from a day treatment program. In addition to several other brain structures, MRI images of the CC were compared between the two groups. Midsagittal 1.5 mm slices were used to divide the CC into seven subregions using Witelson's (1989) technique. No significant differences were observed between the two groups for total CC area or for any CC subregion relative to total cerebral volume. Post-hoc comparisons of CC size between a subset of the ADHD group without co-morbid learning disabilities ($n = 48$) and controls, and between a subset of the

ADHD group without co-morbid Conduct Disorder ($n = 28$) and controls did not reveal any significant differences between the aforementioned groups.

In sum, there appears to be a great deal of evidence to suggest that callosal abnormalities are present in ADHD. Specifically, the results of five of the six studies suggest that the anterior and/or posterior regions are smaller in ADHD children and adolescents compared with normal children and adolescents. Two of the six studies reported smaller rostral bodies and one of these additionally reported a smaller rostrum. Three of the six studies reported smaller spleniums and two of these additionally reported a smaller isthmus (or what would correspond to the isthmus in Hynd, Semrud-Clikeman et al., 1991). One study reported a smaller genu in addition to a smaller splenium and isthmus. Only one of the studies did not report any evidence of callosal abnormalities in ADHD.

Therefore, it appears as though the evidence is equally strong for anterior and posterior callosal dysfunction in ADHD. Methodological difficulties within these studies include small sample sizes, different age and gender compositions of the study populations, and the inclusion of ADHD individuals with extensive co-morbidity in several studies. Further, several studies utilized different techniques for measuring the size of the CC. All of these factors may have contributed to the differences observed between the studies. Further, all of the studies used children and adolescents so it is difficult to ascertain how these results might generalize to adults.

The subregions found to be smaller in ADHD are known to interconnect the anterior and posterior regions of the brain (as reviewed earlier). Therefore, although differences between the studies did emerge, the results from them converge to add

support to the notion of frontal and posterior parietal dysfunction in ADHD.

Thesis Foci

Despite interest in individual differences in the size of the CC and its subregions, the functional significance of aberrations from normal is presently unclear. There have been relatively few investigations that have attempted to determine how variations in CC anatomy relate to cognitive functioning (Beaton, 1997). However, several studies have demonstrated that a smaller CC as observed on MRI in various clinical populations is indicative of poorer callosal function because these patients demonstrate poor performance on tasks requiring interhemispheric communication via the CC (Pelletier, Habib, Lyon-Caen, Salamon, Poncet, & Khalil, 1993; Quinn & Geffen, 1986). For example, Pelletier et al. (1993) observed a correlation in patients with Multiple Sclerosis between the degree of CC atrophy on MRI and the severity of functional impairment of interhemispheric transfer as assessed by several behavioural tests of the CC.

Several studies have documented functional specialization of the CC subregions (de Lacoste et al., 1985; La Mantia & Rakic, 1990). For example, from studies of patients with callosal lesions, it has been observed that visual disconnection results from lesions to the posterior splenium, auditory disconnection from lesions to the isthmus, and tactile disconnection for non-verbal stimuli from lesions to the mid-body of the CC (Clarke & Zaidel, 1994). However, several studies have suggested a more posterior location for the transfer of tactile information (Lakmache, Lassonde, Gauthier, Frigon, & Lepore, 1998; Moore, Brown, Markee, Theberge, & Zvi, 1996; Pelletier et al., 1993; Geffen et al., 1985). Lastly, bimanual coordination deficits have been reported to result from lesions to the genu (Preilowski, 1972; Wolff, Cohen, & Drake, 1984).

Reduced size in several anterior and posterior CC subregions has been observed in children with ADHD using MRI. These differences in size may imply a reduced capacity for the interhemispheric transfer of information in these particular subregions. Therefore, there is a need to examine ADHD subjects on behavioral tests of the CC that are known to tap these specific areas to determine if they will exhibit performance decrements. Research of this sort would converge with the MRI findings and provide additional support for the notion of callosal dysfunction in ADHD.

The present study assessed posterior callosal involvement in ADHD. As discussed earlier, the notion of a posterior parietal involvement in ADHD is more recent than the theory of frontal-striatal dysfunction. However, there is considerable structural and functional evidence to suggest that abnormalities in posterior parietal cortex are present in ADHD. Study of this brain area in ADHD has been neglected and more research is clearly required to understand the nature of this dysfunction. Examining posterior callosal involvement in ADHD would therefore contribute to the limited knowledge in this important, but under-researched area. Furthermore, of the five MRI studies reporting size differences in the CC of ADHD individuals relative to controls, three of these observed a smaller splenium. While three of the five MRI studies also reported smaller anterior CC sizes in ADHD individuals relative to controls, the specific CC sub-regions were not consistent with a smaller genu (Hynd, Semrud-Clikeman et al., 1991), smaller rostrum (Giedd et al., 1994), or a smaller rostral body (Baumgardner et al., 1996; Giedd et al., 1994) being reported. Therefore, as the splenium was found most consistently to be smaller among ADHD children and adolescents compared to controls, it is of paramount interest to examine its functioning in an ADHD sample.

Selection of a Test Instrument

The callosal test that was selected for the present study was the Finger Localization Test (Geffen et al., 1985). Geffen et al. (1985) designed this test for the purpose of comparing tactile localization performance of patients with complete and partial section of the CC and healthy control participants with an intact CC, to determine whether the extent of the cross-localization deficit was correlated with the extent of the CC section. Additionally, they wanted to determine if specific areas of CC being sectioned would differentially affect cross-localization ability. The Finger Localization Test (FLT) was selected for the present study because it is functionally specific to the posterior portion of the CC, including the splenium. The splenium has been observed to be smaller in ADHD children. Therefore, the FLT is an appropriate test because it is linked with the posterior region found to be abnormal in ADHD. Further, it is linked with functioning in the posterior parietal cortex that is known to have fibers projecting through, among other regions, the splenium.

Although other experimental paradigms (a detailed description of these paradigms is beyond the scope of this paper) have been used that are functionally specific to the splenium in the literature, the majority of these tests involve visual stimuli (Lakmache et al., 1998). To the author's knowledge, none of the tasks involving visual stimuli have been used as extensively in the literature or with such a broad range of patients as the FLT. For example, the FLT has been used in the literature to test callosal functioning in normal children (Geffen et al., 1985; Quinn & Geffen, 1986; Pipe, 1991; Summerfield & Michie, 1993) and normal adults (including university students) (Geffen et al., 1985; Piccirilli, Finali, & Sciarma, 1989; Moore et al., 1996; Lakmache et al.,

1998) in addition to clinical samples of split-brain adults (Geffen et al., 1985; Satomi et al., 1991), adults with multiple sclerosis (Pelletier et al., 1993), adults with Alzheimer's disease (Lakmache et al., 1998), adults with alexithymia (Zeitlin et al., 1989), adults with dyslexia (Moore et al., 1996), and reading-disabled children (Summerfield & Michie, 1993). Therefore, in order to have results generalizable with rest of the literature, the FLT was chosen.

The procedure for the FLT will be described in detail further in the paper. In general however, to perform the FLT, a person must, with vision occluded, identify the correct fingers for example, on the left hand, that have been stimulated by the experimenter with a pencil tip, with their thumb of the left hand (uncrossed condition) or they must identify the corresponding correct fingers on the right hand with their thumb of the right hand (crossed condition). The crossed condition requires interhemispheric communication via the CC (Geffen et al., 1985). The FLT limits both stimulus and response to the somatosensory/motor system and allows the investigation of interhemispheric communication of tactile information and motor sequencing abilities (Moore et al., 1996). Altering the number and/or sequence of fingers touched can vary the difficulty of the task.

The validity of the FLT as a measure of interhemispheric transfer is founded on anatomical evidence regarding sensory and motor connections of the hands. The somatosensory cortex of each hemisphere is connected with the homologous region of the contralateral hemisphere via the CC. Although the somatosensory connections from the hands reach both the ipsilateral and contralateral hemispheres, it has been shown that the ipsilateral connections convey only gross information (Gazzaniga & LeDoux, 1978).

Fine motor control of the fingers is controlled exclusively by the contralateral hemisphere (Brinkman & Kuypers, 1973). However, there are no direct connections between the somatosensory or motor cortices of the two hemispheres that receive information from or project to the distal extremities of the limbs (i.e., hands and fingers) (Corkin, 1978; Jones & Powell, 1981). Therefore, it has been hypothesized that the ability to indicate with the right hand, which finger was touched on the left hand, requires that information initially received in the primary somatosensory area (SI) of the right parietal cortex is transmitted to the somatosensory association areas of the right posterior parietal cortex in order to be transferred across the CC to the posterior parietal association area of the opposite hemisphere, where it can then traverse a pathway to the motor cortex for a response to be initiated (Quinn & Geffen, 1986).

Tests of callosal function require two types of trials: within-hemisphere trials (uncrossed) and across-hemisphere (crossed) trials (Banich & Shenker, 1994) in order to take advantage of Teuber's double dissociation principle (Gladstone & Best, 1985). This principle ensures that results confirm one hypothesis while disconfirming another. The FLT conforms to this criterion. For example, if it can be shown that ADHD patients have difficulty in the crossed, but not the uncrossed conditions of the FLT, it is possible to find support for callosal dysfunction while simultaneously refuting an explanation positing a failure of left or right hemisphere dysfunction. In addition, it is possible to examine the relative involvement of the two systems, that is, even in the presence of unilateral dysfunction, it is possible to look for a disproportionate deficit in the crossed condition.

Empirical evidence that the FLT requires the efficient functioning of the CC is based on the poor performance observed in the ability of adult split-brain and callosal

agenesis patients (i.e., individuals born without a CC) to cross-localize tactile stimuli (Dennis, 1976; Geffen et al., 1985; Lasseonde, Sauerwein, McCabe, Laurencelle, & Geoffrey, 1988). For example, Geffen et al. (1985) employed the FLT to study 10 cases of cerebral commissurotomy in which the whole or a portion of the CC had been sectioned. They found that the patient's ability to perform the FLT was significantly determined by the region of the CC that was surgically severed. The cross-localization deficit was defined as the difference between same- and other-hand (or uncrossed and crossed) scores. For example, complete section of the CC was found to produce a mean deficit of 82% with a single finger stimulated. Partial section involving the trunk of the CC produced a mean deficit of 28%. With only the splenium spared, the deficit was 31%, and thus did not increase significantly. Therefore, the splenium appears to be capable of the extra 50% of intermanual transfer. Geffen et al. also reported that the greater the extent of the CC section, the more severe the loss of cross-localization accuracy. Additionally, they observed that increasing the numbers of fingers to be touched exacerbated cross-localization difficulties as well as making within-hand performance less accurate.

The results from Geffen et al. (1985) implicate the functional specificity of the trunk of the CC for the interhemispheric transfer of simple tactile information. However, the functional specificity is not absolute since a considerably reduced deficit (by 50%) was found with the splenium spared. Similarly, in their study of the relationship between callosal atrophy and callosal dysfunction in MS patients, Pelletier et al. (1993) determined that the performance of the crossed portion of the FLT was correlated with the size of several subregions of the CC. Specifically, the authors took MRI

measurements of the CC in 90 patients with Multiple Sclerosis and 25 matched normal control participants. They then had participants perform several tests that explored interhemispheric transfer of auditory, sensory, and motor information. The FLT was the sensory transfer test and performance on this task was found to correlate with MRI measurements of the mid-anterior as well as the posterior regions of the CC. The authors speculated that the relationship between the FLT performance and the mid-anterior region of the CC was likely due to the motor component inherent in the task.

Finger localization in normal adults with an intact CC has not been systematically investigated. Rather, such subjects have served as controls rather than their performance being of primary interest. In their control group of normal adults, Geffen et al. (1985) found cross-localization to be 7% poorer than within-hand finger localization. This finding may be due to the indirect nature of the anatomical route that information must travel for a response to be made with the other hand. Quinn and Geffen (1986) showed that the tactile transfer of information in children increased with age, consistent with the sequence of myelination of the CC during the first decade in life (Moore et al., 1996). Summerfield and Michie (1993) replicated the results of Quinn and Geffen (1986) and found developmental improvement in cross-localization accuracy from 7 to 9 years of age in normal children.

As in previous studies using the FLT (Geffen et al., 1985; Summerfield & Michie, 1993; Quinn & Geffen, 1996; Moore et al., 1996) and in order to ensure generalizability with these studies, participants in the present study were required to touch the same fingers stimulated by the experimenter, in the same sequence.

The Sample

In order to further aid in the understanding of the nature of ADHD as it affects adults, finger localization was assessed in an ADHD sample and a control group (all participants over 18 years of age) in the present study. Furthermore, the evidence that suggests that the CC does not fully myelinate until adolescence would possibly create potential confounds in this study if children had been used as participants.

ADHD is a heterogeneous disorder that may affect males and females differentially in terms of cognitive deficits and associated co-morbidity. Therefore, for the present study, only male subjects were used in an attempt to make the sample more homogeneous. Moreover, because gender differences have also been reported in CC size and/or shape, it is important to control for this factor. Similarly, because differences in CC size have been noted for right versus left-handers and this may influence performance on the FLT, only right-handed adult males were used since the majority of the population is right-handed. The Edinburgh Handedness Inventory is commonly used in the literature to assess the strength and degree of handedness (Oldfield, 1971) and only those that scored zero or above (as an indication of right-handedness) were included in the sample. This questionnaire will be described later in the paper.

According to the DSM-IV, children with ADHD tend to score somewhat lower on IQ tests than non-ADHD children (APA, 1994). However, from the author's review of the literature, IQ scores have generally not been reported for ADHD adults. In an attempt to ensure that the ADHD and control groups would score above 80, and to explore if the two groups were comparable on cognitive variables, the Quick Test was administered as an index of IQ in the present study. This test will be described later in the paper.

Although not as extensively studied in adults, co-morbidity in ADHD is frequent

and must be addressed to increase generalizability. Participants with mood disorders such as depression and anxiety were therefore included in the sample. These individuals were included for practical purposes as it has been noted that individuals with ADHD, particularly adults, have significant co-morbidity with depression and anxiety disorders. Furthermore, to the author's knowledge, there has been no known association with callosal size or abnormality with either depression or anxiety in the literature and are therefore not confounded. Participants with Tourette's syndrome and schizophrenia however, were excluded from the sample because differential performance might be predicted on the FLT based on a literature review suggesting neurological differences between these disorders and ADHD. Specifically, individuals with Tourette's syndrome have been shown on MRI imaging to have significantly larger callosal regions in the following areas: rostral body, mid-body, isthmus, and splenium than ADHD subjects (Baumgardner et al., 1996); and schizophrenic patients have been observed to have ventricular enlargement, temporal and frontal lobe abnormalities, and various structural CC abnormalities (Calev, 1999). Finally, individuals with current substance abuse/dependence were excluded from the analyses as their substance disorder may hinder or bias their ability to perform the required tests for the present study.

Stimulants improve fine motor control, reduce distractibility, and improve sustained attention, especially to boring tasks (Dulcan et al., 1997). These factors are all necessary for the FLT. Therefore, those ADHD patients taking stimulants were required to discontinue their stimulant medication for the day prior to and the day of testing. To ensure that this procedure did not compromise participants in any way, a psychiatrist pre-screened candidates and selected only those for whom he judged this would not present a

problem.

All ADHD subjects in the present study were categorized into the DSM-IV subtypes (ADHD-IA, ADHD-HI, and ADHD-C) because the literature review indicated that differing cognitive deficits and neuropsychological performance may exist between the subtypes and hence, they may have differing neurological anomalies.

There are few scales that assess current DSM-IV symptomatology in adults. These include the Brown Attention-Deficit Disorder Scale (BAADS; Brown, 1995) and the Conners' Adult ADHD Rating Scale (CAARS; Conners, Erhardt et al., 1998). The Brown Attention-Deficit Disorder Scale does not adequately assess hyperactivity and impulsivity (Conners, Erhardt et al., 1998), which renders the scale less useful in determining subtypes. Furthermore, the small standardization sample of 150 subjects, the absence of empirically-derived and cross-validated subscales, in addition to constructs not tied to DSM-IV makes this scale conceptually and empirically unsatisfactory (Conners, Erhardt et al., 1998; Conners, Erhardt, Epstein, Parker, & Sitarenios, unpublished data).

The Conners' Adult ADHD Rating Scale was recently developed and published by Conners, Erhardt et al. (1998) and is intended as a quantitative measure of current DSM-IV ADHD symptomatology and includes a scheme to delineate the three DSM-IV subtypes. Although it is a very new scale, the technical manual provides evidence for the CAARS' internal reliability and discriminant validity which will be discussed in detail later in the paper. The CAARS was used in the present study to validate the presence of ADHD in the ADHD group and determine to which DSM-IV subtype they belonged. It was also used to ensure that the control group did not exhibit ADHD symptoms.

A childhood history of ADHD is a necessary component of adult ADHD diagnosis. The Wender Utah Rating Scale (WURS; Ward et al., 1993) was therefore included in the present study to assess childhood characteristics of ADHD in the ADHD and control group. The WURS is commonly used in the literature and has been shown to have adequate reliability and validity as will be discussed in detail later in the paper.

Control groups commonly utilized in the ADHD literature include normal age-matched controls and psychiatric controls, for example, those with learning disabilities or Tourette's syndrome. In this study, healthy (not diagnosed or referred) individuals comprised the control group and were matched with the ADHD group on gender and handedness. They were screened for ADHD and other co-morbid conditions in the same manner as the ADHD group. In addition, an attempt was made to match the control group with the ADHD group on age, IQ, and socioeconomic status so that the two groups would be comparable on these demographic and cognitive variables. A General Background Questionnaire was used to obtain the information on co-morbidity and socioeconomic status, as will be discussed later in the paper.

Hypotheses

Based on the preceding review of the literature, two main hypotheses were tested in this study.

Hypothesis 1

It was predicted that ADHD subjects (ADHD-C, ADHD-IA, and ADHD-HI) would make significantly more errors in identifying the correct fingers stimulated in the correct order than normal controls on the crossed, but not the uncrossed portion of the FLT. This would support the notion of callosal dysfunction in ADHD.

Hypothesis 2

The literature suggested that ADHD-IA may be more reflective of posterior dysfunction while ADHD with hyperactive-impulsive features (ADHD-HI and ADHD-C) may reflect fronto-striatal dysfunction. It was therefore predicted that ADHD-IA subjects would perform significantly worse on the crossed portion of the FLT compared with ADHD-HI and ADHD-C subjects. This would provide support the theory that there is an anterior-posterior distinction between the DSM-IV subtypes.

Method

Participants

A total of 58 participants volunteered to participate in this study. Twenty-eight participants with ADHD were recruited from a local psychiatric clinic for adults with ADHD and mood disorders based on a referral by a qualified psychiatrist. All candidates approached agreed to participate in the study. All participants with ADHD had previously been diagnosed with adult ADHD by the same psychiatrist. Participants with ADHD were paid a small fee (\$10.00) for voluntarily participating in the study. Thirty Carleton University students also volunteered to participate in this study for an experimental credit for a first year psychology course, and were tested as possible candidates for the control group. However, in order to be included in the final analyses, all participants had to meet the following criteria: (a) male, (b) over 18 years of age, (c) right-handed, as confirmed by self-report and as confirmed by the Edinburgh Handedness Inventory (EHI), (d) not previously diagnosed with Tourette's syndrome, (d) not previously diagnosed with schizophrenia, (e) IQ score of 80 or above on the Quick Test, and (f) no current substance abuse. In order to meet criterion (f), participants were

screened for alcohol abuse and drug dependence in the past six months. No participant in either the ADHD or control group consumed more than four ounces of liquor or four beers more than four times per week. No participant in either the ADHD or control group reported a dependence on recreational drugs in the past 6 months.

ADHD Participants

Of the 28 participants with ADHD, 1 was excluded from the sample because he was found to have co-morbid Tourette's syndrome, as determined by self-report. This resulted in a final sample of 27 ADHD participants. Seventy percent ($n = 19$) of the ADHD individuals were strongly right-handed ($\text{EHI} \geq 70$) while the remainder were moderately right-handed ($\text{EHI} \geq 30$). The median score was 76; the mode was 100. ADHD participants ranged from 21 to 55 years in age ($M \pm SD = 37 \pm 8.7$ years).

Sixty-six percent ($n = 18$) of the ADHD participants reported that a qualified medical professional had previously diagnosed them with depression. Of these 18 individuals, 4 reported an additional co-morbid diagnosis of anxiety, 2 a co-morbid diagnosis of learning disabilities, 4 with additional co-morbid diagnoses of anxiety and bipolar disorder, and 4 with co-morbid diagnoses of anxiety and learning disabilities. Two participants reported that they had previously been diagnosed with an anxiety disorder, but not depression or a learning disability, and three participants reported a co-morbid learning disability, but not anxiety or depression. Only four participants (14.8%) did not report any co-morbid diagnoses. As well, six individuals (22.2%) in the ADHD group reported having sustained one or more previous head injuries. Two of these had sustained multiple head injuries. One ADHD individual reported epilepsy secondary to a head injury and this subject had lost consciousness for 2 hours. Excluding this one

individual, by self-report, loss of consciousness averaged 5.5 minutes (range = 0-45 minutes) for the remaining five.

Sixty-three percent ($n = 17$) of the ADHD participants were currently taking stimulant medication. All agreed to discontinue for the day prior to and the day of testing. Sixty-three percent ($n = 17$) were taking anti-depressant and/or anti-obsessional agents and seven percent ($n = 2$) were also taking medication for bipolar disorder. Participants were asked to discontinue stimulant medications, but to continue taking other prescribed medications.

Control Participants

Of the 30 control participants, 4 were excluded from the final analyses. One participant was excluded because a qualified professional had previously diagnosed him with schizophrenia, as determined by self-report, one because he was left-handed ($\text{EHI} < 0$), one because a qualified professional had diagnosed him with ADD as a child, but not as an adult, and one for having an IQ score < 80 , likely due to English being his second language and therefore being unable to confirm a normal IQ. Thus, a total of 26 control participants were included in the sample. Of the remaining 26 controls, none had been diagnosed with ADHD as an adult or child. Fifty percent ($n = 13$) of the controls were strongly right-handed ($\text{EHI} \geq 70$) while 11 (42%) were moderately right-handed ($\text{EHI} \geq 30$) and 2 (8%) were slightly right-handed ($\text{EHI} \geq 10$). The median score was 69; the mode was 100. Control participants ranged from 18 to 45 years in age ($M \pm SD = 23 \pm 6.16$ years).

In the control group, three (11.5%) participants reported that they had previously been diagnosed with a learning disability. Additionally, one participant reported that he

had previously been diagnosed with depression, anxiety, and bipolar disorder. Four control participants (15.4%) reported having sustained head injuries. Two of these had sustained multiple head injuries. No participant sustained a loss of consciousness of more than 15 minutes and using self-report data, loss of consciousness in the controls averaged 2 minutes (range = 0-15 minutes). None of the controls were taking stimulant medication, but one control participant was taking both an anti-depressant and a medication for bipolar disorder.

Multitple one-way analyses of variance were used to confirm that ADHD and control participants were matched on gender, handedness, and socioeconomic status. However, the ADHD and control groups differed significantly on age and IQ, as estimated on the Quick Test. ADHD participants were significantly older than the controls, $F(1, 51) = 44.05, p < .001$. Although scores on the Quick Test for the ADHD group ($M \pm SD = 109.37 \pm 11.37$) and the control group ($M \pm SD = 101.23 \pm 9.56$) were in the normal range of intelligence, the ADHD group scored significantly higher on the Quick Test than controls, $F(1, 51) = 7.92, p < .01$.

Apparatus

For the FLT, a 50 cm x 42 cm wooden board with wooden side supports (so that it would stand upright on a flat surface) was constructed. A 40 cm x 13 cm hole was cut out at the bottom of the wooden board and centered so that 5 cm remained on each side of the hole. A curtain made out of felt was glued on the board and placed over the hole. The felt was cut into strips that fell vertically over the hole in the board. Participants rested their hand(s) on a cushion while performing the FLT.

Procedure

Participants were tested individually for one session lasting approximately 45-60 minutes. Testing of ADHD participants was conducted at the clinic in a quiet room free of distraction. Participants completed the testing session after meeting with their psychiatrist for a regularly scheduled appointment. Testing of control participants was conducted at Carleton University in a quiet room free of distraction. The principal investigator tested all participants. After an informed consent was read and signed (see Appendix), the session began with the Edinburgh Handedness Inventory.

Following this, eight practice trials of the FLT were performed to ensure that the participant understood the task. This procedure was adapted from the work of Moore et al. (1996) and Geffen et al. (1985). See the Appendix for the FLT protocol used in the present study. Firstly, to ensure that participants could not see their hands, they were asked to extend both of their arms through the curtain of the wooden board, and rest their elbows on a table in front of them. No visual feedback was permitted for any trials (practice or experimental). Their hands, palms down, were placed in a relaxed position, fingers spread, on a cushion. They were told that one or more of their fingers would be touched with a sharpened pencil tip and that they would be required to indicate which fingers were stimulated by touching them with their thumb of the same or opposite hand. Fingers were stimulated with the tip of a sharpened pencil tip (two millimeters maximum depression of the skin) for approximately 0.25 seconds.

Hand Order was counter-balanced and participants were randomly assigned to two groups such that approximately half started with their right hand being stimulated first and half with their left hand. The practice trials were then performed with the first

hand. These included: two practice trials where the participant touched one finger stimulated with the thumb of the same hand, and two practice trials where the participant touched two fingers stimulated with the thumb of the same hand (uncrossed conditions). The next four practice trials were identical to the first four with the exception that different finger sequences were used (see Appendix) and the subject was required to indicate which finger(s) was stimulated by touching the corresponding finger(s) on the opposite hand with the thumb of the opposite hand.

Participants performed the experimental trials in exactly the same manner as the practice trials except that three and four-finger sequences were used instead of one-and two-finger sequences. The experimental trials consisted of four blocks of 16 trials each, for a total of 64 trials. Every block consisted of eight three-finger sequences and eight four-finger sequences. Participants were given eight trials in each of eight possible combinations (i.e., three or four finger sequence, right hand or left hand stimulated first, and same hand or opposite hand response). Whether a participant would be asked to respond with either the same (uncrossed) or opposite (crossed) hand was also counterbalanced (by random assignment) so that approximately half of the participants responded with the same hand as that which was stimulated and half responded with the opposite hand to that which was stimulated for each of the 4 blocks of 16 trials.

The first two blocks were performed with the first hand, the last two blocks with the second hand. Finger sequences were randomized such that each finger was represented the same number of times in each position of each trial for 16 three-finger and 16 four-finger trials for the first hand as recommended by Moore et al. (1996). The second hand trials consisted of the same finger sequences presented in a different order.

The first block of trials was followed by the administration of the General Background Questionnaire. The second block of trials was then completed, followed by the administration of the CAARS. The third block of trials was completed after the CAARS, followed by the Quick Test. The fourth block of trials was then completed, followed by the WURS. A detailed description of the tests and questionnaires used in the present study follows.

Conners' Adult ADHD Rating Scale. The Conners' Adult ADHD Rating Scale (CAARS; Conners, Erhardt et al., 1998; Appendix) is a 66-item self-report measure of current adult ADHD symptoms. This scale assesses the DSM-IV subtypes and yields three DSM-IV ADHD symptom subscales: a 9-item Inattentive Symptoms subscale, a 9-item Hyperactive-Impulsive Symptoms scale, and a Total ADHD Symptom scale (the sum of the two previous subscales). An ADHD Index of 12 items is also obtained and represents a measure of the overall level of ADHD-related symptoms. According to the authors, this index can be used as an indicator of those adults that are most likely to be diagnosed with ADHD. All items are answered on a 4-point Likert scale (ranging from "0" for "not at all true" to "3" for "very much true"). Each of the four points on the Likert scale also include temporal descriptions (e.g., very often) in order to simplify responding and interpretation. Psychometric properties of the scale are reported in the CAARS' manual (Conners, Erhardt, & Sparrow, 1999). Cronbach's alpha coefficient was calculated to estimate internal reliability. This is an overall summary coefficient that varies between 0.00 (poor reliability) and 1.00 (perfect reliability). These scores ranged from .64 to .82 for 18-29 year old males, from .64 to .84 for 30-39 year old males, and from .70 to .85 for 40-49 year old males for the ADHD Index and three DSM-IV

Symptom subscales. Discriminant validity was calculated for the ADHD Index and an overall correct classification rate of 73% was reported. The authors concluded that the ADHD Index is useful as a screening tool to help identify those adults who would benefit from further assessment (pg. 70).

Based on the manual, a *T*-score of 65 or above on either the Inattentive Symptoms subscale or the Hyperactive-Impulsive Symptoms scale resulted in assignment to the ADHD-IA or ADHD-HI subtypes, and a *T*-score of 65 or above obtained on the Total DSM-IV Symptoms scale resulted in assignment to the ADHD-C subtype. A *T*-score of 65 or above on the ADHD index resulted in confirmation of the ADHD diagnosis, if obtained in a pre-diagnosed ADHD subject.

Wender Utah Rating Scale. The Wender Utah Rating Scale Short Form (WURS; Ward et al., 1993; Appendix) Short-Form version is retrospective self-report measure designed to assess childhood characteristics of ADHD in adults. Participants rated each symptom on a 5-point Likert scale indicating the severity of each symptom (ranging from “0” for “not at all or very slightly” to “4” for “very much”). The respondent was instructed to rate each item on the basis of “as a child I was (or had)” or “as a child in school”. Scores were derived by summing the patient’s ratings on the items. A cutoff score of 46 has been used to identify ADHD in childhood for the WURS Short-Form (Ward et al., 1993). The WURS has adequate reliability (Rossini & O’Conner, 1995) and a factor structure consistent with that derived from ratings of children’s behavior (Stein, Sandoval, Szurnowski, Roizen, Reinecke, Blondis, & Klein, 1995). All participants completed the full WURS questionnaire (61 items). However, only the 25 items that comprise the short form were scored.

General Background Questionnaire

This questionnaire (Appendix) was devised by Parlow (personal communication) and modified by the author. In its present form, it consisted of 17 questions about the participant's personal and medical history. Personal history included questions concerning the individual's socioeconomic status and education. As an index of socioeconomic status, parental education was assessed on a Likert-type scale ranging from "0" for "grade 8 or less" to "7" for a "post-graduate degree" (i.e., M.A., M.D., or Ph.D.). Medical history included questions about any prior head injuries or injuries to fingers in addition to questions concerning medications being taken and the presence of other disorders such as learning disabilities, depression, anxiety, substance abuse/dependence, Tourette's syndrome, and schizophrenia.

Edinburgh Handedness Inventory

The Edinburgh Handedness Inventory (EHI; Oldfield, 1971; Appendix A) is a 10-item questionnaire that measures hand preferences for everyday activities. A left or right hand preference is indicated by a (+) sign in the appropriate column and a strong preference is indicated by a (++) sign. If both hands are equally preferred, a (+) sign is placed in both columns. The EHI yields a handedness laterality quotient that ranges from -100 for extreme left-handedness to +100 for extreme for extreme right-handedness. A score of 0 or greater indicates right handedness (Oldfield, 1971). The laterality quotient is obtained by determining the difference between the tallied right- and left-hand scores divided by the aggregate of both scores.

Quick Test

The Quick Test (QT; Ammons & Ammons, 1962; Appendix A) is a popular

instrument used to get an index of IQ in a short amount of time. Participants looked at a word and listened to the word read aloud by the experimenter and they were required to match it to one of four pictures. The number of correct matches were summed and this score was transformed into an IQ score as per the manual.

Results

Preliminary Analyses

Descriptive statistics were run to verify the assumptions of Analysis of Variance (ANOVA). There were no outliers and based on Levene's test for homogeneity of variance, this assumption was not violated for any of the variables of interest. To ensure that the following variables were controlled, multiple one-way analyses of variance were conducted to assess Group differences (ADHD versus Controls) based on: age, mother's education (as an index of SES), father's education (as an index of SES), frequency of alcohol consumption and amount of alcohol consumed (as index of alcohol abuse), EHI, CAARS, WURS, and the Quick Test. With the exception of the CAARS and the WURS, the results of these ANOVAs were discussed earlier in the paper in the Participants section. Table 3 shows the scores obtained on the CAARS and the WURS questionnaires for both the ADHD and Control groups.

For all sub-scales of the CAARS, the ADHD group scored significantly higher than the controls, indicating more ADHD symptomatology than controls: CAARS-IA, $F(1, 51) = 35.51, p < .001$; CAARS-HI, $F(1, 51) = 15.59, p < .001$; CAARS-TOT, $F(1, 51) = 32.72, p < .001$; and CAARS-IND, $F(1, 51) = 43.50, p < .001$. Seventy-four percent ($n = 20$) of ADHD participants obtained a T-score of 65 or above on the CAARS-IA sub-scale, thus meeting the criteria for ADHD-IA. Forty-eight percent ($n = 13$) met the

criteria for ADHD-HI (T-score ≥ 65 on CAARS-HI sub-scale). Seventy-eight percent ($n = 21$) scored 65 or above on the CAARS-TOT sub-scale, meeting the criteria for ADHD-C. No ADHD participant met the criteria for ADHD-HI alone, and only one met the criteria for ADHD-IA alone. Five ADHD participants did not score significantly on any sub-scale of the CAARS. Therefore, of the 27 ADHD participants, 21 were classified as ADHD-C, 1 was classified as ADHD-IA, and 5 did not meet the clinical cut-off for any DSM-IV symptom subscale on the CAARS. Consequently, subtypes could not be used in the analyses that followed. The mean score of these five individuals on the CAARS-HI was 52 ($SD = 9.09$), on the CAARS-IA ($M \pm SD = 54 \pm 8.29$), and on the CAARS-TOT ($M \pm SD = 55 \pm 8.61$). In the control group, five participants scored 65 or above on the CAARS-IA, three scored 65 or above on the ADHD-HI, and five scored 65 or above on the CAARS-TOT. Therefore, of the 26 controls, 19 did not meet the cutoff score of 65 on any sub-scale of the CAARS, 5 could be classified as ADHD-C, and 2 as ADHD-IA.

None of the control participants scored above 65 on the CAARS-IND score. However, 52% ($n = 14$) of the participants with ADHD obtained a T-score of 65 or less. Therefore, the CAARS-IND failed to identify 52% of the ADHD participants. As such, the CAARS-IND was useful only in identifying controls successfully confirming that none of the controls had ADHD. This measure underestimated the presence of ADHD in the ADHD group.

On the WURS, 21 (78%) of the participants with ADHD scored above the cutoff of 46. The remaining six participants scored between 29 and 45. None of the controls scored above 46. The ADHD group scored significantly higher on the WURS, $F(1, 51) = 82.74$, $p < .001$, reporting significantly more symptoms of ADHD in childhood. For the

means and standard deviations of the two groups, please see Table 3.

Because the two groups differed on age and IQ, these two variables were investigated to determine if they would be appropriate covariates. Age and performance on the FLT were positively correlated ($r = .34$; $p < .05$). Age was therefore included in the subsequent analyses as a covariate. IQ and performance on the FLT however, were not found to be significantly correlated, $r = .08$, $p > .05$. Therefore, it was decided not to treat IQ as a covariate and the effect of this factor was pooled into the error term.

Subsequently, an Analysis of Covariance was conducted with age as a covariate to examine the extent to which scores on the FLT differed between ADHD subjects and controls across of each of the two variables (Hand Stimulated and Condition). A preliminary analysis was conducted to examine the effect of Hand Order. A $2 \times 2 \times (2 \times 2)$ analysis of covariance (ANCOVA) was performed with Group (ADHD versus Controls) and Hand Order (Left First versus Right First) treated as between-subjects variables. Within-subject variables were Condition (Crossed versus Uncrossed) and Hand Stimulated (Left versus Right). The dependent variable was the number of errors made in identifying the fingers stimulated in the correct order.

A significant interaction between Hand Order $\times$ Hand Stimulated was observed, $F(1, 49) = 7.60$, $p < .01$. In addition, a significant main effect for Hand Order was reported, $F(1, 48) = 6.82$, $p < .05$, indicating that those participants that began with their left hand being stimulated made fewer errors than those that began with the right hand. Post-hoc comparisons were conducted for the Hand Order $\times$ Hand Stimulated interaction and it was revealed that significantly more errors were made with the right hand when the right hand was stimulated first, than the errors made by the right hand when the left hand

was stimulated first, $F(1, 48) = 11.89, p < .01$. In addition, significantly more errors were made with the left hand than the right hand when the left hand was stimulated first, $F(1, 49) = 4.67, p < .05$. Figure 3 shows this interaction graphically.

The effect of Hand Order and its interaction with Hand Stimulated appeared to be an evenly distributed practice effect, and because there was no interaction of Hand Order with either of the variables of interest (Condition or Group), Hand Order was pooled and the ANCOVA was repeated.

Main Analyses

A second $2 \times (2 \times 2)$ ANCOVA with age as a covariate was conducted for Group (ADHD versus Controls). The within-subject variables were Condition (Crossed versus Uncrossed) and Hand Stimulated (Left versus Right). Age-adjusted means and standard deviations within each condition for the ADHD group and Controls can be found in Table 4.

Only one significant interaction was found in this analysis: Group $\times$ Condition, $F(1, 51) = 5.64, p < .03$. Post hoc comparisons revealed that while the ADHD and control participants did not differ significantly in the number of errors made in the uncrossed condition, $F(1, 50) = 1.094, p > .05$, ADHD subjects made significantly more errors than controls in the crossed condition, $F(1, 50) = 4.26, p < .025$. Thus, when the task required interhemispheric communication, the ADHD participants performed significantly more poorly. Figure 4 depicts this interaction graphically. A significant main effect for Condition was also observed, as more errors were made in the crossed versus the uncrossed condition in both groups, $F(1, 51) = 129.37, p < .001$. There were no other significant main effects. The cross-localization deficit for the ADHD participants was

calculated and it was found that 18% more errors were made in the crossed condition than the uncrossed condition. Conversely, the cross-localization deficit in controls was 13%.

As the CAARS questionnaire showed that the majority (78%) of ADHD subjects could be classified as ADHD-C, while none could be classified as ADHD-HI and only one (4%) as ADHD-IA, the second hypothesis comparing the scores of the finger localization task between the DSM-IV ADHD subtypes was not tested.

Discussion

The present study investigated the adequacy of posterior callosal functioning in a sample of clinically-referred ADHD adult males and a comparison group of non-ADHD males. To the author's knowledge, this is the first study that has examined callosal functioning in an adult ADHD sample with a behavioural test of the CC. There were two hypotheses postulated at the outset of the study. However, only the first was tested as the composition of the sample did not permit the investigation of differences between the ADHD subtypes of DSM-IV. The first hypothesis predicted that ADHD adults would perform significantly more poorly on the crossed, but not the uncrossed portion of the FLT based on the findings of the MRI studies in the literature that have reported anatomical abnormalities of the CC in ADHD children and adolescents compared with controls.

In accordance with this hypothesis, the data revealed, after adjusting for the age difference, that clinically-referred adult males with ADHD made more errors on the crossed interhemispheric condition of the FLT than did non-ADHD males. However, there were no differences in the number of errors made by the ADHD and control groups

on the uncrossed intrahemispheric condition of the FLT. Therefore, it would appear that the ADHD adults in the present study were able to perform the FLT as accurately as normal controls when the tactile information was confined to one hand/hemisphere. However, when tactile information had to cross over the CC, the ADHD group performed significantly more poorly. Given this finding and in accordance with Teuber's principle of double dissociation (Gladstone & Best, 1985), the difficulties experienced by the ADHD group in the crossed condition appears to be compatible with callosal dysfunction and is not due to either left or right hemisphere dysfunction.

As a result of the fact that none of the ADHD individuals could be classified as having ADHD-HI and only one with ADHD-IA, the second hypothesis could not be tested. This hypothesis had predicted that, based on an anterior-posterior distinction that has been postulated between the subtypes of ADHD (Matzow & Hynd, 1992), significantly more errors would be made on the FLT in individuals with ADHD-IA than the ADHD-HI.

However, the absence of the ADHD-HI and ADHD-IA subtypes in this sample is in of itself an interesting finding and can be interpreted in several ways. Firstly, perhaps it is only those individuals within the ADHD-C subtype, whom have been noted as having the most severe ADHD symptoms and related difficulties (Gaub and Carlson, 1997a; Lalonde et al., 1998; Wolraich et al., 1998), that continue to exhibit ADHD symptoms worthy of clinical diagnosis in adulthood. Alternatively, as discussed earlier, the DSM-IV criteria are not specific to the diagnosis of ADHD in adults since the disorder is subsumed under 'Disorders usually First Diagnosed in Infancy, Childhood, or Adolescence' in the DSM-IV (APA, 1994), and there are no formal diagnostic criteria for

adults with ADHD (Ranseen et al., 1998; Feifel, 1996). Therefore, the DSM-IV criteria may not be appropriate or capable of distinguishing between the subtypes in ADHD adults. Lastly, the lack of DSM-IV subtypes may be an artifact of this particular sample population.

The ADHD participants in the present study were clinically-referred adult males who had been diagnosed with ADHD in adulthood. The fact that this is a clinically-referred sample means that the results may not be generalizable to the whole ADHD population since, as discussed earlier, it has been demonstrated previously that epidemiological samples may have a less severe form of the disorder. However, generalizability was improved by the fact that as expected, the ADHD participants had significant co-morbidity with depression and anxiety disorders, and several had previously been diagnosed with learning disabilities. This finding is compatible with what has been previously reported in other research with clinically-referred ADHD adults.

The ADHD participants in this study were significantly older than the controls. However, they did not differ in socioeconomic status as indicated by self-reported parental education. Interestingly, the ADHD group achieved a higher IQ score than the control group. This finding suggests that although this group had ADHD and a significant number of co-morbid diagnoses, they were quite intelligent. It is difficult to determine if this finding is abnormal in the literature since, as mentioned earlier, intelligence scores have not often been reported in the literature on ADHD in adults. All of the ADHD participants had been assessed and diagnosed with ADHD in adulthood. By self-report, only four had been previously diagnosed with ADHD as a child as well.

Therefore, with the exception of these four individuals, the fact that this ADHD sample appeared to have higher intelligence and similar socioeconomic status (relative to the controls) suggests that Murphy and Barkley (1996) were correct in suggesting that adult self-referrals may be different from ADHD children followed into adulthood and are more likely to include individuals with higher intelligence, more education, and higher socioeconomic status.

Comparison of the present study results with MRI literature

The FLT is purported to be a measure of the posterior CC, including the splenium. In the present study, ADHD adults performed the crossed portion of the FLT (which requires interhemispheric interaction) more poorly than normal control subjects. This finding provides support for the five MRI studies that reported callosal abnormalities in the anterior and/or posterior subregions of the CC (Hynd, Semrud-Clikeman et al., 1991; Giedd et al., 1994; Semrud-Clikeman et al., 1994; Baumgardner et al., 1996; Lyoo et al., 1996). The most consistent subregion found to be smaller in ADHD individuals was the splenium. The present behavioural findings converge with the observed anatomical reductions in splenial size in the MRI literature to suggest that posterior callosal functioning may be compromised in ADHD.

However, as MRI scans were not conducted as part of the present study, it cannot be determined if performance on the FLT was indeed correlated with the size of the CC, or any specific subregion of the CC such as the splenium. Inasmuch as the FLT is believed to assess posterior callosal functioning, it can only be inferred that the results of the present study support the notion of posterior callosal dysfunction in ADHD. In one study discussed earlier, it was observed that performance on the FLT in patients with

Multiple Sclerosis correlated with the mid-anterior (anterior midbody) as well as posterior subregions of the CC (Pelletier et al., 1993). As such, it cannot be determined if the cross-localization deficits observed in the present study in the ADHD group were solely a result of posterior callosal dysfunction. More anterior regions of the CC could quite possibly have been involved as well as posterior regions, especially since there is a motor aspect implicated in the performing the FLT (i.e., moving the fingers) and fibers cross more anteriorly between the motor regions of the two hemispheres (see Table 1). The present study is the first to suggest evidence of callosal dysfunction in ADHD.

Comparison of the present study results with Finger Localization Test studies

The present study is the first to have utilized the FLT to examine callosal functioning in ADHD adults. However, several other studies using different sample populations have utilized the FLT. Control participants were able to identify the correct fingers in the correct order on average, 89% of the time in the uncrossed condition, and 76% of the time in the crossed condition. In comparison, in Moore et al. (1996) reported that a similar sample of university students identified the correct fingers in the correct order on average, 85% of the time in the uncrossed condition, and 73% of the time in the crossed condition. Similarly, in Geffen et al. (1985), the control group identified on average, 87% of the stimulated fingers correctly in the uncrossed condition, and 77% correctly in the crossed condition. Therefore, it would appear that the present study findings are comparable with other studies that have used similar control participants.

It is interesting to note that the partial commissurotomy patients (i.e., those with the trunk of the CC sectioned) in Geffen et al. (1985) demonstrated a deficit in interhemispheric transfer of 28% while controls had a deficit of 10%. Responses that

required cross-localization were on average, 82% worse than same-hand responses in full commissurotomy patients. The comparable interhemispheric transfer deficits observed for the present study were 13% for controls versus 18% for ADHD individuals. These results suggest that the deficit in interhemispheric transfer in these ADHD individuals may fall somewhere midway between that of normal individuals and partial commissurotomy patients, reflecting a much less severe deficit than that reported for split-brain patients with the entire CC sectioned.

Implications of the present study

The CC is the largest “bridge” in the central nervous system that connects the two cerebral hemispheres (Levander & Sonnesson, 1998). Attempts to relate human behaviour to callosal morphology have been difficult due to the wide variability in the size and shape of the CC that exists in adults (Parashos et al., 1995). However, as discussed earlier, several researchers have concluded that a smaller CC is indicative of poorer callosal functioning (Pelletier et al., 1993; Quinn & Geffen, 1986). The findings of reduced callosal size in several subregions of the CC in ADHD individuals combined with the present findings of poor performance on the FLT provide additional support to the notion that anomalies in callosal morphology may indeed be linked to differences in the performance of tasks of interhemispheric interaction. If reduced size of the CC or any of its subregions is indeed indicative of dysfunction, it would be expected that ADHD individuals would perform tasks requiring efficient callosal functioning with difficulty. Accordingly, in the present study, ADHD individuals demonstrated a reduced ability to perform the crossed portion of the FLT, which is purported to require efficient functioning of the CC. Therefore, this study provides further evidence that in individuals

with reported anatomical reductions in CC size, tasks that require interhemispheric interaction are performed less efficiently.

Although the CC is a relatively large structure in the brain, we have a limited understanding of its role in higher cognitive functioning (Clarke & Zaidel, 1994). In spite of this limited understanding, there is little argument in the literature on the main functions of the CC. A great deal of research has demonstrated that the CC plays a dynamic role in the brain in controlling the flow of sensory, motor, and higher order information between the hemispheres (Burke & Yeo, 1994; Levander & Sonesson, 1998; Hellige et al., 1998). This dynamic role not only allows for the coordination of processing between the hemispheres, but it has been argued that it may have a profound influence on attentional functioning. As discussed earlier, numerous researchers from different laboratories have demonstrated that the CC appears to have a role in maintaining attentional unity between the two hemispheres in the intact brain (Wale & Geffen, 1989; Rueckert et al., 1994; Rueckert & Levy, 1996).

A deficit in attention has been demonstrated in ADHD (Barkley, 1998; Barkley et al., 1992), either as the core problem or as part of larger network of difficulties known as executive dysfunction. Given that attention has been demonstrated to be subserved by, among other regions, the CC, it can be speculated that the reduced callosal subregions observed in ADHD may result in some of the deficits observed in ADHD, and specifically, inattention. In support of this notion, Rao et al. (1989) observed that decreased performance on tasks of attention correlated with a smaller CC size in Multiple Sclerosis patients. With the present study results, in conjunction with the MRI literature in ADHD, it can be speculated that perhaps reduced size of the CC is

responsible for at least some of the inattention observed in ADHD (as evidenced on cognitive tasks such as the CPT) and further results in difficulties on tasks of interhemispheric interaction.

Although functioning of the anterior CC was not specifically examined in the present study, it is likely that the anterior CC is also required to perform the FLT efficiently due to the inherent motor aspect of the task. Future research should consider utilizing behavioural tasks of the CC that tap all the subregions of the CC, from the anteriormost tip of the genu to the posteriormost tip of the splenium in order to determine if functioning in subregions of the CC may be compromised selectively. Given the large body of research that suggests frontal lobe or frontostriatal dysfunction in ADHD, it would be of particular interest to examine callosal functioning in ADHD individuals on behavioural tasks of the anterior CC. For example, bimanual tapping tasks are purported to require efficient functioning of the anterior CC (Wolff, Cohen, & Drake, 1984) and could be used with this population.

It must be noted that the present study results are limited in that it cannot be determined whether the same pattern of results would have been obtained with an un-referred population-based sample. Further, these results cannot be generalized to ADHD women or to ADHD children of both sexes. As was to be expected, the ADHD group had a significant number of co-morbid disorders including depression, anxiety, and learning disabilities. It would be prudent to replicate the present research with a psychiatric control group, in addition to a group of normal participants without any psychiatric disorders. For example, if it could be found that a group of depressed and/or anxious individuals without ADHD did not evidence poor performance on the crossed

condition of the FLT as compared with normal controls, but that the ADHD group did, this would provide stronger support for the notion of callosal dysfunction in ADHD in that it could be concluded that the poor performance was a result of ADHD alone, and not due to the co-morbid conditions.

Ideally, future research should conduct selected behavioural tests of the CC in conjunction with neuroimaging techniques such as MRI, fMRI, etc. This would provide important confirmation of the link between behavioural evidence of callosal dysfunction in ADHD and neuroanatomical abnormality in this population. Furthermore, as was attempted in the present study, it would be of interest to examine anterior and posterior callosal functioning in the DSM-IV ADHD subtypes to test the prediction that ADHD-IA is linked more with posterior dysfunction and ADHD-C and ADHD-HI with anterior dysfunction. Sample limitations precluded assessing this variable in the present study.

Finally, cognitive differences have been observed between males and females with ADHD (Hinshaw, 1994) and anatomical differences in CC size and/or shape have also been observed between males and females in general (Salat et al., 1997; Davatzikos & Resnick, 1998). Therefore, a comparison of ADHD males and females would be of interest to see if any significant gender differences would emerge on tests of callosal functioning. As ADHD is a lifelong disorder with the symptoms of ADHD in childhood appearing to change somewhat in adulthood (Achenbach et al., 1994), age differences might be anticipated. Given that the CC undergoes changes in structure and function from childhood to adulthood (Ratinckx et al., 1997; Giedd et al., 1996), examining the performance of ADHD children on callosal tasks and comparing it to that of ADHD adults may be of particular interest.

In conclusion, the present study has provided behavioural evidence in support of the notion of callosal dysfunction in ADHD. In conjunction with anatomical studies of ADHD using MRI and other techniques reported in the literature, the present behavioural study may provide the impetus for further study of interhemispheric interaction in individuals with ADHD, and suggest that the CC is one of several neuroanatomical areas of dysfunction associated with this disorder. More research is required to replicate the findings with additional behavioural tests of the CC, as well as in conjunction with brain imaging.

Figure 1¹

¹From Corpus callosum morphology in children with Tourette's syndrome and attention deficit hyperactivity disorder by T.L. Baumgardner, H.S. Singer, M.B. Denckla, M.A. Rubin, M.T. Abrams, M.J. Colli and A.L. Reiss, 1996, *Neurology*, 478.

Figure 2²

²From Neuropsychology: the neural bases of mental function, by M.T. Banich, 1997, Boston, New York: Houghton Mifflin Company, 110.

Figure 3

Figure 4

Table 1

Subregions of the Corpus Callosum and the Cortical Regions they Interconnect

Callosal Subregion	Cortical Region
Rostrum	Caudal/orbital, Frontal, Prefrontal, Inferior premotor areas
Genu	Prefrontal areas
Rostral body	Premotor, Supplementary motor areas
Anterior midbody	Motor areas
Posterior midbody	Somaesthetic, Posterior parietal areas
Isthmus	Superior temporal, Posterior parietal areas
Splenium	Occipital, Inferior temporal, *Superior temporal, *Posterior parietal areas

Note. The items marked * are cortical regions that have been recently suggested by several prominent researchers in this field (Clarke, 1998; Hellige, Taylor, Lesmes, & Peterson, 1998).

Table 2

MRI Studies of the Corpus Callosum in ADHD (1991 and 2000)

Study	Subjects	Inclusion Criteria	Exclusion Criteria	Co-morbidity
Hynd et al. (1991)	(1) 7 ADD/H (5M, 2F) age=9; range=7-10 (2) 10 healthy C (8M, 2F) age=11; range=not given	(1) IQ> 85, Caucasian, free of other neurological conditions. RH (2) Caucasian, free of DSM-III-R diagnoses. RH		(1) 3 CD, 1 SAD, 1 DEP, 2 ODD (2) None
Griedd et al. (1994)	(1) 18 ADD/H (18M) age=11; range=6-15 (2) 18 healthy C (18M) age=10; range=6-15	(1) RH (2) Matched for age, weight, height, Tanner stage and handedness	(1) IQ < 80, LD, evidence of medical or neurological disease, TS or other Axis I psychiatric disorder (2) Physical or neurological disorder, lifetime history of psychiatric abnormalities family history of ADHD, or 1 st degree relatives with major psychiatric disorders	(1) 2CD, 16 ODD (2) None
Semrud-Clikeman et al. (1994)	(1) 15 ADD/H (15M) age=13; range=8-18 5 stimulant non-responders (2) 15 healthy C(15M) age=14; range=8-19	For both groups: RH, RE, RF, result of normal pregnancy & uneventful birth history	For both groups: no co-morbid psychiatric diagnoses (including DEP, ANX, CD or LD)	For both groups: None
Lyoo et al. (1996)	(1) 51 ADHD (45M, 6F) age=11 (2) 28 non-ADHD (16M, 12F) age=12	For both groups: RH	For both groups: MR, autism, PDD, psychiatric disorder, EEG abnormality, seizure disorder	(1) 37 CD, 17 DYS (2) 22 CD, 6 DYS
Baumgardner et al. (1996)	(1) 16 TS (13M, 3F) age=12±2.2 (2) 21 TS+ADHD (19M, 2F) age=11±1.6 (3) 13 ADHD (13M) age=11±1.4 (4) 27 C (21M, 6F) age=10±2.6	(1) 3 LH, 13 RH (2) 2 LH, 19 RH (3) All RH (4) 2 LH, 25 RH	For all groups: Does not meet either clinical or psychometric criteria for normal intellectual ability	Not addressed in this study
Castellanos et al. (1996)	(1) 57 ADHD (57M) age=11; range=5-17 (2) 55 healthy C (55M) age=12; range=5-17	(1) 81% RH (2) 87% RH	(1) IQ < 80, medical or neurological disorders, TS, any psychiatric disorder (2) Physical, neurological or lifetime history of psychiatric abnormalities, 1 st or > 20% 2 nd degree relatives with psychiatric disorders	(1) 9 CD, 20 ODD, 2 ANX, 9 LD (RD) (2) None

Note. M = male; F = female; C = controls; RH = right-handed; RE = right-eared; RF = right-footed; LH = left handed;

CD = conduct disorder; SAD = separation anxiety disorder; DEP = depression; ODD = oppositional defiant disorder; LD

= learning disability; TS = Tourette's syndrome; ANX = anxiety disorder; RD = reading disorder; MR = mental

retardation; PDD = pervasive developmental disorder; DYS = dyslexia.

Table 3

Comparison of scores on the Conners' Adult ADHD Rating Scale (CAARS) and the Wender Utah Rating Scale (WURS) for the ADHD and Control groups

Questionnaire	ADHD (n=27) Mean (SD)	Controls (n=26) Mean (SD)
CAARS-IA	75.44 (13.53)*	55.58 (10.49)
CAARS-HI	64.26 (12.19)*	52.19 (9.90)
CAARS-TOT	73.56 (12.49)*	55.38 (10.51)
CAARS-IND	63.81 (11.71)*	46.96 (5.81)
WURS	62.44 (16.61)*	28.15 (9.85)

Note. CAARS-IA = Conners' Adult ADHD Rating Scale DSM-IV Predominantly Inattentive Symptoms subscale; CAARS-HI = Conners' Adult ADHD Rating Scale DSM-IV Predominantly Hyperactive-Impulsive Symptoms subscale; CAARS-TOT = Conners' Adult ADHD Rating Scale DSM-IV Total Symptoms subscale; CAARS-IND = Conners' Adult ADHD Rating Scale ADHD Index; WURS = Wender Utah Rating Scale; SD = standard deviation.

* $p < .001$

Table 4

Mean Number of Errors (adjusted for age) made on the Finger Localization Task by

Group (ADHD versus Controls)

Hand Stimulated and Condition	ADHD (n=27) Mean (SD)	Controls (n=26) Mean (SD)
Left Hand, Uncrossed	9.06 (1.28)	5.94 (1.31)
Left Hand, Crossed	17.28 (1.86)	13.21 (1.86)
Right Hand, Uncrossed	6.52 (1.26)	5.99 (1.29)
Right Hand, Crossed	17.52 (2.08)	14.38 (2.13)

Note. SD = standard deviation.

Figure Captions

Figure 1. MRI T₁ -weighted midsagittal image with boundaries delineating intracranial area and the five area segments of the corpus callosum.

Figure 2. A. Diagram of the corpus callosum. The numbers shown in each section indicate the brain region depicted in B. (top, left hemisphere, lateral view; bottom, left hemisphere, midsagittal view) that are connected through that section of the corpus callosum. The connections occur in a topographic manner: anterior sections (I) of the corpus callosum connect anterior sections of the brain (Region 1), middle sections (II and III) connect brain regions that are more central (Region 3 and 7), and posterior sections (IV and V) connect posterior brain regions (Regions 4, 5, and 6). Region 2 has little, if any, corpus callosum connections.

Figure 3. Line graph of FLT errors as a function of Hand Order by Hand Stimulated.

Figure 4. Line graph of FLT errors as a function of Group by Condition.

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APPENDIX

Study Protocol: Tests and Questionnaires

Finger Localization as a Test of Posterior Callosal Functioning in a Clinically-Referred Sample of ADHD Adults

Name: _____
Number: _____

Date: _____

(1) Informed Consent

Please read this over carefully.

(2) Edinburgh Handedness Inventory

Please indicate your preference in the use of hands for the following activities listed on this sheet by putting a plus sign in the appropriate column. If the preference is so strong that you never use the other hand unless absolutely forced to, put ++. If you are really indifferent, put a plus sign in both columns.

(3) Finger Localization Test

For this task, I am going to touch the tip of one or more of your fingers. Immediately after I touch them and say go, you will have to touch them with your thumb in the same order as I touched them.

You will either have to touch them with the thumb of the same hand or you will have to touch the corresponding fingers on the opposite hand with the thumb of that hand. Like this. Demonstrate the task for both uncrossed and crossed conditions.

Do you have any questions?

Practice

First Hand Stimulated: Right Hand ____ Left Hand ____

We'll practice for a little bit now. Please place your (left or right) hand through this screen, palm down on this pillow. I would like you to touch the fingers I touch with the thumb of the same hand. Remember to wait until I say go before you touch them.

Uncrossed

Trial 1: 1___ Trial 3: 2___ 4___
Trial 2: 3___ Trial 4: 4___ 1___

Now, place both of your hands through the screen. I would like you to indicate the fingers I touch by touching the corresponding fingers on the opposite hand with the thumb of that hand.

Crossed

Trial 5: 2___ Trial 7: 3___ 2___
Trial 6: 4___ Trial 8: 1___ 4___

Block 1

First Hand Stimulated: Right Hand ___ Left Hand ___
Condition: Crossed ___ Uncrossed ___

Okay. Now that you know how to do it, we'll start. For this first set of items, I will only be touching your (left or right depending on Hand Order) hand. First, I want you to (depending on Condition):

(i)touch the fingers I touch with the thumb of the same hand. So please place only your (left or right) hand through the screen.

(ii)indicate which fingers I touch by touching the corresponding fingers of the opposite hand with the thumb of that hand. So please place both of your hands through the screen.

Trial 1: 1___ 3___ 4___	Trial 9: 3___ 4___ 2___ 1___
Trial 2: 2___ 4___ 1___	Trial 10: 1___ 2___ 4___ 3___
Trial 3: 4___ 2___ 3___	Trial 11: 2___ 1___ 3___ 4___
Trial 4: 3___ 1___ 2___	Trial 12: 4___ 2___ 1___ 3___
Trial 5: 1___ 2___ 4___	Trial 13: 2___ 3___ 4___ 1___
Trial 6: 3___ 4___ 2___	Trial 14: 1___ 4___ 3___ 2___
Trial 7: 4___ 3___ 1___	Trial 15: 3___ 1___ 2___ 4___
Trial 8: 2___ 1___ 3___	Trial 16: 4___ 3___ 1___ 2___

(4) General Background Questionnaire

Please answer the following questions as precisely as you can.

Block 2

First Hand Stimulated: Right Hand ____ Left Hand ____
Condition: Crossed ____ Uncrossed ____

For this next set of items, once again, I will only be touching your (left or right depending on Hand Order) hand.

This time, I would like for you to (depending on Condition):

(i) touch the fingers I touch with the thumb of the same hand. So please place only your (left or right) hand through the screen.

(ii) indicate which fingers I touch by touching the corresponding fingers of the opposite hand with the thumb of that hand. So please place both of your hands through the screen.

Trial 1: 2____ 3____ 1____

Trial 2: 3____ 2____ 4____

Trial 3: 1____ 4____ 2____

Trial 4: 2____ 3____ 4____

Trial 5: 4____ 1____ 2____

Trial 6: 3____ 4____ 1____

Trial 7: 1____ 2____ 3____

Trial 8: 4____ 1____ 3____

Trial 9: 1____ 3____ 4____ 2____

Trial 10: 1____ 4____ 2____ 3____

Trial 11: 2____ 4____ 3____ 1____

Trial 12: 2____ 3____ 1____ 4____

Trial 13: 3____ 2____ 1____ 4____

Trial 14: 3____ 1____ 4____ 2____

Trial 15: 4____ 1____ 2____ 3____

Trial 16: 4____ 2____ 3____ 1____

(5) Conners' Adult ADHD Rating Scale

This questionnaire consists of 66 statements. Please read each statement carefully and then select the best answer from the following choices: 0 for not at all or never, 1 for just a little or once in a while, 2 for pretty much or often, and 3 for very much or very frequently.

Practice before Block 3 & 4

Check list for hand order (half start with right hand and half with left hand):

Second Hand Stimulated: Right Hand ____ Left Hand ____

Okay. Now we'll go back to this task again. This time however, I will only be touching your (left or right depending on Hand Order) hand. We'll practice for a little bit first. Please place your (left or right) hand through the screen. I would like you to touch the fingers I touch with the thumb of the same hand.

Uncrossed

Trial 1: 4____ Trial 3: 3____ 2____

Trial 2: 1____ Trial 4: 2____ 4____

Now I would like for you to indicate the fingers I touch by touching the corresponding fingers on the opposite hand with the thumb of that hand.

Crossed

Trial 5: 3____ Trial 3: 1____ 4____

Trial 6: 2____ Trial 4: 3____ 1____

Block 3

Second Hand Stimulated: Right Hand ____ Left Hand ____

Condition: Crossed ____ Uncrossed ____

O.K. For the first set of items, I want you to (depending on Condition):

(i) touch the fingers I touch with the thumb of the same hand. So please place only your (left or right) hand through the screen.

(ii) indicate which fingers I touch by touching the corresponding fingers of the opposite hand with the thumb of that hand. So please place both of your hands through the screen.

Trial 1: 4___ 2___ 3___

Trial 2: 3___ 1___ 2___

Trial 3: 2___ 1___ 3___

Trial 4: 1___ 3___ 4___

Trial 5: 3___ 4___ 2___

Trial 6: 2___ 4___ 1___

Trial 7: 1___ 2___ 4___

Trial 8: 4___ 3___ 1___

Trial 9: 2___ 3___ 4___ 1___

Trial 10: 4___ 3___ 1___ 2___

Trial 11: 1___ 2___ 4___ 3___

Trial 12: 3___ 1___ 2___ 4___

Trial 13: 3___ 4___ 2___ 1___

Trial 14: 4___ 2___ 1___ 3___

Trial 15: 1___ 4___ 3___ 2___

Trial 16: 2___ 1___ 3___ 4___

(6) Quick Test

This is kind of a picture game. I am going to show you some pictures and read some words. You point to the best pictures for the words. Some of the words will be very easy and some of the words will be hard. You may not know all of the words. If I read a word that you don't know, just tell me that you don't know, and I will go on to another word.

Give 2 or 3 easy and 2 or 3 hard words to practice so that they understand not to guess.

Administer Form 1 to all subjects.

Test until there have been six consecutive passes and six consecutive fails.

Mark a correct response with a + sign placed next to the word on the left. Mark an incorrect response with a minus sign in the same position.

Block 4

Second Hand Stimulated: Right Hand ___ Left Hand ___

Condition: Crossed ___ Uncrossed ___

For this next set of items, once again, I will only be touching your (left or right depending on Hand Order) hand. This time however, I want you to (depending on Condition):

(i)touch the fingers I touch with the thumb of the same hand. So please place only your (left or right) hand through the screen.

(ii)indicate which fingers I touch by touching the corresponding fingers of the opposite hand with the thumb of that hand. So please place both of your hands through the screen.

Trial 1: 3___ 2___ 4___

Trial 2: 1___ 2___ 3___

Trial 3: 2___ 3___ 4___

Trial 4: 3___ 4___ 1___

Trial 5: 4___ 1___ 3___

Trial 6: 2___ 3___ 1___

Trial 7: 4___ 1___ 2___

Trial 8: 1___ 4___ 2___

Trial 9: 3___ 2___ 1___ 4___

Trial 10: 1___ 3___ 4___ 2___

Trial 11: 4___ 2___ 3___ 1___

Trial 12: 2___ 3___ 1___ 4___

Trial 13: 1___ 4___ 2___ 3___

Trial 14: 2___ 4___ 3___ 1___

Trial 15: 3___ 1___ 4___ 2___

Trial 16: 4___ 1___ 2___ 3___

(7) Wender Utah Rating Scale

This questionnaire consists of 61 statements. After reading each statement carefully, put an X in the space which best rates the statement. The statement can either be rated as not at all or very slightly, mildly, moderately, quite a bit, or very much.

The first 42 statements concern behaviours or feelings that must be rated for their presence in childhood. For example, the first item would be "as a child I was active, restless, always on the go".

The next 7 items concern medical problems as a child, and the last 11 items concern behaviours or feelings that must be rated for the presence when you were a child in school. Do you have any questions?

EDINBURGH HANDEDNESS INVENTORY

Name: _____

Date (D/M/Y): ____ / ____ / ____

Date of Birth (D/M/Y): ____ / ____ / ____

Telephone #: _____

Please indicate your preferences in the use of hands in the following activities by putting + in the appropriate column. Where the preference is so strong that you would never try to use the other hand unless absolutely forced to, put ++. If in any case you are really indifferent, put + in both columns.

Some of the activities require both hands. In these cases, the part of the task, or object, for which preference is wanted is indicate in brackets.

Please try to answer all the questions, and only leave a blank if you have no experience at all of the object or task.

		LEFT	RIGHT
1	Writing		
2	Drawing		
3	Throwing		
4	Scissors		
5	Toothbrush		
6	Knife (without fork)		
7	Spoon		
8	Broom (upper hand)		
9	Striking Match (match)		
10	Opening Box (lid)		
i	Which foot do you prefer to kick with?		
ii	Which eye do you use when using only one?		

L.Q.	
------	--

Leave these spaces blank

DECILE	
--------	--

General Background Questionnaire

Name: _____

Date (D/M/Y): ____ / ____ / ____

Date of Birth (D/M/Y): ____ / ____ / ____

Telephone #: _____

1. Do you consider yourself (circle the best answer):

- a. right-handed
- b. left-handed
- c. ambidextrous

2. (a) Mother's principle occupation when you were a child: _____

(b) Mother's education (Put a check mark beside highest level achieved):

Some high school ____
Graduated high school ____
Some trade school or college ____
Diploma from trade school or college ____
Some university ____
Bachelor's Degree from university ____
Post-graduate Degree (ie. M.D., M.A., Ph.D.) ____

3. (a) Father's principle occupation when you were a child: _____

(b) Father's education (Put a check mark beside highest level achieved):

Some high school ____
Graduated high school ____
Some trade school or college ____
Diploma from trade school or college ____
Some university ____
Bachelor's Degree from university ____
Post-graduate Degree (e.g., M.D., M.A., Ph.D.) ____

4. Your approximate total current household income (Put a check mark beside the best answer):

Less than or equal to \$14,999 ____

\$15,000 - \$24,999 ____

\$25,000 - \$34,999 ____

\$35,000 - \$44,999 ____

\$45,000 - \$54,999 ____

\$55,000 - \$64,999 ____

\$65,000 - \$74,999 ____

\$75,000 or more ____

5. Have you even been diagnosed with Attention Deficit Hyperactivity Disorder (ADHD)?

- a. No
- b. Yes, by a Medical Doctor
- c. Yes, by a School Psychologist
- d. Yes, by a Clinical Psychologist outside of the school system
- e. Yes, by _____
- f. If you answered yes, at what age? _____

6. Do you believe that you have or had in the past a learning disability? If so, in what area? Circle as many answers as are appropriate.

- a. No
- b. Yes, in Reading
- c. Yes, in Math
- d. Yes, in Spelling
- e. Yes, in _____

7. Have you even been identified by a qualified professional as having a learning disability?

- a. No
- b. Yes, by a School Psychologist
- c. Yes, by a Clinical Psychologist outside of the school system
- d. Yes, by a Medical Doctor
- e. Yes, by _____
- f. If you answered yes, at what age? _____

8. Have you even been identified by a qualified professional as having Tourette's Syndrome?
- a. No
 - b. Yes
9. Have you even been identified by a qualified professional as having schizophrenia?
- a. No
 - b. Yes
10. Have you even been identified by a qualified professional as having depression?
- a. No
 - b. Yes
11. Have you even been identified by a qualified professional as having an anxiety disorder?
- a. No
 - b. Yes
12. Have you ever been identified by a qualified professional as having bipolar disorder?
- a. No
 - b. Yes
13. In the past 6 months, how often have you drank alcohol?
- a. seldom or never
 - b. once a month
 - c. 2-3 times / week
 - d. 4 or more times / week
14. If you drank alcohol in the past 6 months, how much did you usually consume at one time?
- a. an ounce of liquor or one beer
 - b. 2-4 ounces of liquor or 2-4 beers
 - c. more than 4 ounces of liquor or more than 4 beers

CAARS-Self-Report: Long Version (CAARS-S:L)

by C. K. Conners, Ph.D., D. Erhardt, Ph.D., & E. P. Sparrow, M.A.

Name: _____ Gender: M F
(Circle One)

Birthdate: ____/____/____ Age: ____ Today's Date: ____/____/____
Month Day Year Month Day Year

Instructions: Listed below are items concerning behaviors or problems sometimes experienced by adults. Read each item carefully and decide how much or how frequently each item describes you recently. Indicate your response for each item by circling the number that corresponds to your choice. Use the following scale: 0 = Not at all, never; 1 = Just a little, once in a while; 2 = Pretty much, often; and 3 = Very much, very frequently.

	Not at all, never	Just a little, once in a while	Pretty much, often	Very much, very frequently
1. I like to be doing active things.	0	1	2	3
2. I lose things necessary for tasks or activities (e.g., to-do lists, pencils, books, or tools).	0	1	2	3
3. I don't plan ahead.	0	1	2	3
4. I blurt out things.	0	1	2	3
5. I am a risk taker or a daredevil.	0	1	2	3
6. I get down on myself.	0	1	2	3
7. I don't finish things I start.	0	1	2	3
8. I am easily frustrated.	0	1	2	3
9. I talk too much.	0	1	2	3
10. I am always on the go, as if driven by a motor.	0	1	2	3
11. I'm disorganized.	0	1	2	3
12. I say things without thinking.	0	1	2	3
13. It's hard for me to stay in the place I'm supposed to.	0	1	2	3
14. I have trouble doing leisure activities quietly.	0	1	2	3
15. I'm not sure of myself.	0	1	2	3
16. It's hard for me to keep track of several things at once.	0	1	2	3
17. I'm always moving even when I should be still.	0	1	2	3
18. I forget to remember things.	0	1	2	3
19. I have a short fuse/hot temper.	0	1	2	3
20. I'm bored easily.	0	1	2	3
21. I leave my seat when I am not supposed to.	0	1	2	3
22. I have trouble waiting in line or taking turns with others.	0	1	2	3
23. I still have tantrums.	0	1	2	3
24. I have trouble keeping my attention focused when working.	0	1	2	3
25. I seek out fast-paced, exciting activities.	0	1	2	3
26. I avoid new challenges because I lack faith in my abilities.	0	1	2	3
27. I feel restless, even if I am sitting still.	0	1	2	3
28. Things I hear or see distract me from what I'm doing.	0	1	2	3
29. I am forgetful in my daily activities.	0	1	2	3
30. Many things set me off easily.	0	1	2	3
31. I dislike time-intensive activities.	0	1	2	3
32. I lose things that I need.	0	1	2	3
33. I have trouble listening to what other people are saying.	0	1	2	3

Items continued on back page...

CAARS-Self-Report: Long Version (CAARS-S:L)

by C. K. Conners, Ph.D., D. Erhardt, Ph.D., & E. P. Sparrow, M.A.

	Not at all, never	Just a little, once in a while	Pretty much, often	Very much, very frequently
34. I am an underachiever.	0	1	2	3
35. I interrupt others when talking.	0	1	2	3
36. I change plans/jobs in midstream.	0	1	2	3
37. I act okay on the outside, but inside I'm unsure of myself.	0	1	2	3
38. I am always on the go.	0	1	2	3
39. I make comments/remarks that I wish I could take back.	0	1	2	3
40. I can't get things done unless there's an absolute deadline.	0	1	2	3
41. I fidget (with my hands or feet) or squirm in my seat.	0	1	2	3
42. I make careless mistakes or have trouble paying close attention to detail.	0	1	2	3
43. I step on people's toes without meaning to.	0	1	2	3
44. I have trouble getting started on a task.	0	1	2	3
45. I intrude on others' activities.	0	1	2	3
46. It takes a great deal of effort for me to sit still.	0	1	2	3
47. My moods are unpredictable.	0	1	2	3
48. I don't like homework or job activities where I have to think a lot.	0	1	2	3
49. I'm absent-minded in daily activities.	0	1	2	3
50. I am restless or overactive.	0	1	2	3
51. I depend on others to keep my life in order and attend to the details.	0	1	2	3
52. I annoy other people without meaning to.	0	1	2	3
53. Sometimes my attention narrows so much that I'm oblivious to everything else; other times it's so broad that everything distracts me.	0	1	2	3
54. I tend to squirm or fidget.	0	1	2	3
55. I can't keep my mind on something unless it's really interesting.	0	1	2	3
56. I wish I had greater confidence in my abilities.	0	1	2	3
57. I can't sit still for very long.	0	1	2	3
58. I give answers to questions before the questions have been completed.	0	1	2	3
59. I like to be up and on the go rather than being in one place.	0	1	2	3
60. I have trouble finishing job tasks or schoolwork.	0	1	2	3
61. I am irritable.	0	1	2	3
62. I interrupt others when they are working or playing.	0	1	2	3
63. My past failures make it hard for me to believe in myself.	0	1	2	3
64. I am distracted when things are going on around me.	0	1	2	3
65. I have problems organizing my tasks and activities.	0	1	2	3
66. I misjudge how long it takes to do something or go somewhere.	0	1	2	3

1	2	3
belt dancing traffic whistle fence	cans chewing falling dinner cow	sheet exercise machine burners audience
drink wreck music medicine gun	groceries hat sitting country danger	dish drying food fork crowd
pepper racing salt woman sugar	plate river tasting shelves sky	slice washing tears fighting kitchen
track school partner couples rail	table carelessness manners adding injury	tasty windy pitiful contest sorrow
respectful betting daring stadium pedestrian	merchandise waitress horizon retail irrigation	loser heartbreak struggle rotary opponents
graceful fluid solution discipline bleachers	unaware current fertile descending spacious	grief utensils lever portion edible
crystallized turntable saccharin immature cordiality	proprietor inattentive indulging precipitation freshest	exhibition soothed caress combatant forlorn
velocity decisive laceration foliage imperative	transom consumption aquatic perilous terrain	nutrient solace pacify contorted jets
intimacy concoction conviviality chevrons condiment	imminent foresight condensation satiation visceral	doleful tines disconsolate sustenance maudlin
cacophony miscible imbibe amicable pungent	bovine replete prehension ingress celerity	gustatory poignant bellicose comestible despondency

WENDER UTAH RATING SCALE (WURS)

Patient Name: _____

Date: _____

Patient Number: _____

	Not at all or very slightly	Mildly	Moderately	Quite a bit	Very much
As a child I was (or had):					
1. Active, restless, always on the go					
2. Afraid of things					
3. Concentration problems, easily distracted					
4. Anxious, worrying					
5. Nervous, fidgety					
6. Inattentive, daydreaming					
7. Hot- or short-tempered, low boiling point					
8. Shy, sensitive					
9. Temper outbursts, tantrums					
10. Trouble with stick-to-it-tiveness, not following through, failing to finish things started					
11. Stubborn, strong-willed					
12. Sad or blue, depressed, unhappy					
13. Incautious, dare-develish, involved in pranks					
14. Not getting a kick out of things, dissatisfied with life					
15. Disobedient with parents, rebellious, sassy					
16. Low opinion of myself					
17. Irritable					
18. Outgoing, friendly, enjoyed company of people					
19. Sloppy, disorganized					
20. Moody, ups and downs					
21. Angry					
22. Friends, popular					
23. Well-organized, tidy, neat					
24. Acting without thinking, impulsive					
25. Tendency to be immature					
26. Guilty feelings, regretful					
27. Losing control of myself					
28. Tendency to be or act irrational					
29. Unpopular with other children, didn't keep friends for long, didn't get along with other children					
30. Poorly coordinated, did not participate in sports					
31. Afraid of losing control of self					
32. Well-coordinated, picked first in games					
33. Tomboyish (for women only)					
34. Running away from home					
35. Getting into fights					
36. Teasing other children					
37. Leader, bossy					

	Not at all or very slightly	Mildly	Moderately	Quite a bit	Very much
38. Difficulty getting awake					
39. Follower, led around too much					
40. Trouble seeing things from someone else's point of view					
41. Trouble with authorities, trouble with school, visits to principal's office					
42. Trouble with police, booked, convicted					
Medical problems as a child:					
43. Headaches					
44. Stomachaches					
45. Constipation					
46. Diarrhea					
47. Food allergies					
48. Other allergies					
49. Bedwetting					
As a child in school, I was (or had):					
50. Overall a good student, fast					
51. Overall a poor student, slow learner					
52. Slow in <i>learning</i> to read					
53. Slow reader					
54. Trouble reversing letters					
55. Problems with spelling					
56. Trouble with mathematics or numbers					
57. Bad handwriting					
58. Able to read pretty well but never really enjoyed reading					
59. Not achieving up to potential					
60. Repeating grades (which grades? _____)					
61. Suspended or expelled (which grades? _____)					