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COVERT ORIENTING IN CHILDREN WITH ADHD

By

Cynthia Leigh Huang

A THESIS

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ABSTRACT

COVERT ORIENTING IN CHILDREN WITH ADHD

By

Cynthia Leigh Huang

Childhood attention deficit hyperactivity disorder (ADHD) is a serious, common, and chronic behavioral syndrome characterized by impaired attention, impulsivity, and excessive motor activity. Although psychosocial influences doubtlessly affect childhood behaviors, there is strong evidence to support the existence of neuro-cognitive mechanisms in ADHD which may differ between the subtypes. Further defining these mechanisms would offer valuable clues to the etiology of ADHD and suggest better treatment methods for affected children. Therefore, Posner's covert orienting task was used to specify underlying neuro-cognitive mechanisms in ADHD and its subtypes. Diagnostic groups included (1) unimpaired control children (2) children with the combined subtype of ADHD, and (3) children with the inattentive subtype of ADHD.

Contrary to previous findings, results did not support the theory of a right hemisphere dysfunction in children with ADHD, of an anterior attention system deficit in children with the combined subtype of ADHD, or of a posterior attention system deficit in children with the inattentive subtype of ADHD. However, children with ADHD as a whole did exhibit greater overall variability in reaction time than controls, a pattern consistent with a deficit in arousal.

For my grandfather

1913-1998

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Introduction

Attention deficit hyperactivity disorder (ADHD) is a serious, common, and chronic behavioral syndrome characterized by impaired attention, impulsivity, and excessive motor activity (APA, 1994). As many as three to seven percent of school-aged children are affected, with three times as many boys as girls likely to be identified in non-referred populations (Barkley, 1997), and nine times as many in clinic-referred children (Barkley, 1996). ADHD is among the most common reasons for referral to child mental health services (Offord et al., 1987), and is also a risk factor for poor academic functioning, social/emotional maladjustment, behavioral disorders, later substance abuse, and other medical problems (e.g. accident proneness, sleep disturbances, and chronic health problems; Barkley, 1996). Recent research estimates that 30-50% of affected children continue to exhibit symptoms into adulthood (Barkley, 1996; Denckla, 1991; Pelham, 1993), with 10% displaying disabling symptomology (Mannuzza, Klein, Bessler, et al., 1993). Stimulants (e.g. methylphenidate) and a number of antidepressants are effective in treating 70-80% of children (Comings et al., 1991; McCracken, 1991; Zametkin, 1989), and such pharmacologic treatment is the most common form of intervention (Pelham, 1993).

In contrast to the DSM-III R, which did not distinguish between subtypes, the DSM-IV currently recognizes three: primarily hyperactive/impulsive (ADD+H), primarily inattentive (ADD-H), and combined (APA, 1994). Approximately 85% of children with ADHD are diagnosed with the primarily hyperactive/impulsive subtype, and although ADD-H becomes more common in adolescence, it is still less common than ADD+H (Barkley, 1996). More girls with ADHD cluster into the ADD-H as opposed to ADD+H

subtype (Gaub & Carlson, 1997), but boys are still more prevalent in either subtype. The ADD-H subtype is poorly understood, and research on girls is limited in part by the low prevalence rate of ADHD in girls. Other than the presence of subtypes, another complicating aspect of ADHD is the frequent co-occurrence of other psychiatric disorders (e.g. oppositional defiant disorder (ODD), conduct disorder (CD), anxiety disorders, and learning disabilities (LD)) (Barkley, 1996), the presence of which may mask or account for any observed effects.

Although psychosocial influences doubtlessly affect childhood behaviors, there is strong evidence to support the existence of neuro-cognitive mechanisms in ADHD (Barkley, 1997; Logan, Schachar, & Tannock, 1997). However, whether these mechanisms differ between subtypes, or change when comorbid conditions are present, is unknown. Answers to these questions would ultimately be used to improve outcomes for affected individuals through the creation of more accurate diagnostic criteria and the development of treatment and prevention programs designed to remediate specific areas of cognitive dysfunction.

Thus, the following discussion will note key etiologic theories, review a prominent theory of attention, and discuss how such a theory may be relevant to ADHD. The discussion will then focus on a paradigm based on the visuospatial attention system which may prove useful towards the understanding of ADHD.

Theories of Etiology

Some researchers have suggested that toxic reactions (e.g. ingestion of fine sugar, food dyes, food allergies, lead poisoning, anticonvulsant medication (in epileptic children), and maternal toxic exposure to nicotine or alcohol) can play a role in the development of ADHD (Anastopoulos & Barkley, 1988). Although lead poisoning, anticonvulsant medication, and maternal toxic exposure have been shown to give rise to or to exacerbate symptoms of ADHD, these risk factors do not account for the majority of children with ADHD symptoms (Anastopoulos & Barkley, 1988). It has likewise been suggested that pre and perinatal problems (e.g. bleeding during pregnancy or anoxia) may lead to problems such as ADHD or learning disabilities. However, like toxic exposure, such occurrences are probably not major factors in the development of ADHD for the majority of affected children (Anastopoulos & Barkley, 1988).

At present, heredity is one of the best substantiated etiologic theories in ADHD research (Goodman & Stevenson, 1989; Stevenson, 1992). Although no direct evidence of abnormal chromosome structure exists in most cases of ADHD, parents and relatives of both male and female probands exhibit increased incidences of psychopathology (e.g., retrospectively diagnosed ADHD, alcoholism, affective disorders, and conduct problems; Faraone, Biederman, Keenan, & Tsuang, 1991). Furthermore, adoption and twin studies not only point to a higher incidence of ADHD in the biological parents of adopted children with ADHD, but also indicate a higher concordance rate among monozygotic than dizygotic twins (Goodman & Stevenson, 1989; Stevenson, 1992). Although the presence of heritable factors is most likely involved in the etiology of ADHD, it is still unclear why boys are more often diagnosed than girls.

It is most likely that ADHD etiology is polygenic and multifactorial, but the apparent efficacy of medications which increase the release of dopamine and decrease the release of norepinephrine (McCracken, 1991), has lead to the examination of candidate genes involved in catecholamine regulation, particularly those involved with dopaminergic functioning (e.g. the dopamine D₂ receptor gene (D₂A1) (Comings et al., 1991) and the dopamine transporter gene (DAT1) (Cook et al., 1993; Waldman, in review). A central problem for genetic research in the psychological sciences is the lack of a homogenous phenotype used for diagnosis (Deutsch & Kinsborne, 1990). Without such specifications, patient groupings remain heterogeneous, and the search for genetic markers is severely hampered. Finding and using objective measures of cognitive operations is one method which might improve the homogeneity within groupings (Nigg & Goldsmith, 1998).

Because catecholamines are heavily involved with CNS functioning, and because preliminary evidence suggests the presence of neurologic abnormalities in individuals with ADHD, recent literature has focused on uncovering possible neurologic dysfunctions in individuals with ADHD, and correlating those findings with cognitive or behavioral symptoms. Although this neurologic approach to understanding ADHD has become more prevalent, it would be premature to fully adopt such a view. Indeed, others argue that psychosocial influences (e.g. the effect of blended families, social disadvantage, marital conflicts, inconsistent parenting style, etc.) also affect childhood behaviors, and theories which take such factors into account may allow a fuller understanding of the disorder (Sandberg, 1996). With this precaution in mind, the focus herein will be potential neurologic mechanisms of ADHD.

Neurologic Mechanisms

Right Hemisphere Dysfunction

The hypothesis that individuals with ADHD suffer from a subtle neurological dysfunction has existed throughout the history of modern clinical observations, beginning from Still's (1902) hypothesis that a "perversion of function [exists] in the higher nervous centers," (Still, 1902, p. 1166). One prominent theory is that ADHD may result from a right hemisphere deficit. Part of what makes the right hemisphere theory so appealing is the proposal that it is dominant in the regulation of attention, arousal, and motor activation (Tucker & Williamson, 1984). However, the generalizability and findings related to this theory are somewhat limited because many studies do not sufficiently control for comorbidity.

Both behavioral observations and imaging techniques have provided evidence for a right hemisphere deficit. A study on developmental right hemisphere syndrome (or non-verbal learning disability) found that of the 20 children studied, all were comorbid for ADHD and 13 exhibited soft neurological signs on the left side of their body (Gross-Tsur, Shalev, Manor, & Amir, 1995). These signs consisted of asymmetric upper left extremity posturing while maintaining arm extension or during forced gait maneuvers, slow alternate movements on the left side, and hyperflexia of the left limbs (Gross-Tsur et al., 1995). Soft neurologic signs (although not specified) were also found in a similar study using children with comorbid conduct disorder (CD) and ADHD (Aronowitz et al., 1994).

Neuroimaging techniques have provided further physical evidence of a right hemisphere deficit. For instance, using MRI techniques, Castellanos et al. (1996) found a lack of the normally observed right larger than left caudate asymmetry, smaller right

globus pallidus, and smaller right anterior frontal region in ADHD as compared to normal controls. Furthermore, examination of rCBF distribution found that in a group of ADHD children, striatal regions, specifically the right striatum, were hypoperfused in comparison to normal controls (Lou, Henriksen, Bruhn, Børner, & Nielsen, 1989).

Right hemisphere abnormalities have also been correlated with poor performance (mean accuracy and response times) on tasks requiring inhibitory control (Casey et al., 1997). In this study, performance on sensory and response selection tasks were positively correlated with right caudate nucleus volume in the ADHD, but not control, group. On the inhibitory trials of the sensory selection task, right prefrontal cortex volume was positively correlated with mean accuracy for control, but not ADHD, group. The authors concluded that these results supported a theory of right frontal striatal dysfunction in ADHD, a dysfunction which correlated with poor performance on tasks requiring inhibitory control (Casey et al., 1997).

That hemispheric asymmetry could have an effect on attentional mechanisms and result in performance deficits has been shown in attentional cueing paradigms with collosotomy patients. Because lesions to the right hemisphere typically have more dramatic and long lasting effects on patients than lesions to the left, researchers believe that the two hemispheres differ in their control of spatial attention (Mangun et al., 1994). In one study, collosotomy patients were given valid (which accurately predicted the target location), invalid (which inaccurately predicted target location), and bilateral or diffuse (control conditions) cues to the location of a target on a spatial cueing task (Mangun et al., 1994). All cue conditions, except for the invalid (which increased reaction time), decreased time to detection when targets were presented to the right hemisphere (left

visual field). No effect for cueing was shown for the left hemisphere. Therefore, the right hemisphere attentional system can attend either the right or left visual field at any time, but the left hemisphere attends the right visual field at all times. These results help explain clinical observations that if the right hemisphere is damaged in some manner, neglect of the left visual field is often observed. But, if damage occurs to the left hemisphere, neglect does not occur as often, presumably because the right hemisphere is still capable of orienting to the right visual field (Mangun et al., 1994).

Based on such findings, some researchers have predicted that if children with ADHD suffer from a right hemisphere dysfunction, then the attentional mechanisms dependent upon right hemisphere functioning may be impaired, leading to subclinical neglect of the left visual field. Whereas left visual field neglect was once believed to be a perceptual dysfunction, it is now known to be an attentional disorder due to a hypersensitivity for information in the right visual field (Robertson, 1992). Patients suffering from hemispatial neglect retain the ability to selectively focus attention, but they are unable to disengage their attention from the ipsilateral visual field. Using a letter cancellation task, Voeller and Heilman (1988) found that in comparison to controls, children with ADHD not only made more total errors, but made significantly more left sided ones, a pattern similar to that of adults with right hemisphere damage. However, using the same letter cancellation task, Malone, Couitis, Kershner, & Logan (1994) found that although children with ADHD did indeed detect fewer targets on the left, the effect was accounted for mainly by children who had a comorbid learning disability. When these comorbid cases were removed from analysis, the effect was no longer significant (Malone et al., 1994), highlighting the need to control for such confounds. A third study

on children with ADHD used a line bisection and visual target cancellation test and likewise found no evidence for hemi-neglect (Ben-Artsy, Glicksohn, Soroker, Margalit, & Myslobodsky, 1996). Given these replication failures, the existence of a left visual field neglect resulting from a dysfunctional right hemisphere-based attention system, is questionable at best.

A conceptualization of attention in which attentional processing is represented by a distributed network rather than localized to a specific area, has recently come to the forefront. Although many models of attention exist (Mirsky, 1996), the present study will adopt the model developed by Posner & Petersen (1990). Posner and Petersen (1990) propose three systems which together are responsible for the control of attention: the anterior attention system, the posterior attention system, and the vigilance network.

Anterior Attention System

The anterior attention system (AAS) or executive attention network (Posner & Raichle, 1994) is a supervisory system based on the anterior cingulate gyrus, the supplementary motor cortex, and other areas of the midprefrontal cortex (Jackson, Marrocco, & Posner, 1994; Mirsky, 1996; Posner & Petersen, 1990). The AAS is responsible for exercising conscious control over information processing (Posner & Raichle, 1994) such as the inhibition of prepotent responses, planning, decision making, target detection, and the voluntary shifting of attention to locations in space (Jackson et al., 1994; Jonides, 1981; Pennington & Ozonoff, 1996; Posner & Raichle, 1994). According to Posner's model, these functions are collectively known as executive functions. However, as a concept, the term "executive function" remains underspecified, becoming an umbrella term under which a number of complex tasks reside (Jackson, et

al., 1994; Pennington & Ozonoff, 1996; Posner & Petersen, 1990). Furthermore, Posner's conceptualization of executive functions is neither the only conceptualization, nor is it the most widely accepted. There is a wide diversity of opinion regarding the definition of executive functions, some of which stress emotional regulation, social behavior, modulation of behavior based on task demands, and working memory, rather than inhibitory and attentional processes (Lyon & Krasnegor, 1996).

In spite of these limitations, there is evidence that the concept of executive functions possesses both convergent and divergent validity (Pennington & Ozonoff, 1996). Performance on neuropsychological tests of executive functions distinguishes between ADHD and control groups. Likewise, children with ADHD exhibit fewer deficits on non-executive function tasks (Pennington & Ozonoff, 1996). In a recent integrative theoretical proposal, Barkley (1997) proposed that a primary deficit in inhibitory processes leads to the disruption of the development of five neuropsychological abilities (the first four of which are considered executive functions): working memory, self-regulation of affect/motivation/arousal, internalization of speech, reconstitution, and motor control/fluency/syntax. Examples of commonly used neuropsychological measures include the Wisconsin Card Sorting Task, Trail Making, Stroop, Matching Familiar Figures Test, Tower of Hanoi, Word Fluency, and the Rey-Osterreith Complex Figure. Although some overlap exists between Posner's conceptualization of executive function and other researchers', the following findings are not necessarily based on Posner's model.

Whereas many studies have examined executive dysfunction in boys with ADHD (e.g. Hamlett, Pellegrini, & Conners, 1987; Reader, Harris, Schuerholz, & Denckla, 1994;

Weyandt & Willis, 1994), only one study has examined executive dysfunctions in girls. Although girls with ADHD performed more poorly than normal control girls on most of the tasks, the groups were not significantly different following age correction (Seidman et al., 1997). However, all but seven of the girls were medicated at the time of testing, a strong confounding factor because methylphenidate improves performance on such tasks in boys (Pelham, Walker, Sturges, & Hoza, 1989). Visuospatial orienting paradigms have also been used to behaviorally examine AAS functioning in children with ADHD, but these studies will be discussed in a subsequent section following the present review of Posner & Petersen's (1990) three attentional network model.

The proposal that children with ADHD possess deficits in the anterior attention system, specifically in the prefrontal cortices, was initially inspired by the similarity of performance on measures of executive functions between individuals with ADHD and patients with documented frontal lobe lesions. The theory has been extensively refined since its inception and has been buttressed by positive neuropsychological findings using tests which purportedly measure executive functions. In adults, lesions in the frontal lobes, or in areas with close connections to them, can lead to poorer performance on tasks requiring set shifting, planning, working memory, contextual memory, inhibition, and fluency (Pennington & Ozonoff, 1996). Although frontal lesions in childhood are rare, there is evidence that such lesions can have long lasting effects on cognition similar to those seen in adults (Benton, 1991; Scheibel & Levin, 1997).

In addition to these behavioral similarities, physiologic measures have also supported the theory of generalized frontal lobe dysfunction. In normal controls, the right prefrontal cortex is involved with response inhibition, and children with ADHD not only

possess smaller right frontal cortical structures, but perform significantly worse than normal control children on tasks requiring such inhibition (Casey et al., 1996). Likewise, Lou, Henriksen, & Bruhn (1984) found evidence of decreased blood flow to the frontal lobes in children with ADHD which was corrected following administration of methylphenidate. A later study found that the striatal regions, particularly on the right side, were hypoperfused in children with ADHD (Lou et al., 1989). Regarding girls with ADHD, a study on cerebral glucose metabolism (CMRglu) found that global CMRglu was significantly decreased in ADHD girls in comparison to ADHD boys as well as normal girls (Ernst et al., 1994). Furthermore, in comparison to normal girls, girls with ADHD exhibited reduced regional absolute CMRglu in the premotor and orbital frontal cortex and the temporal cortex (Ernst et al., 1994). However, they were unable to find global or regional differences in CMRglu which could reliably distinguish between ADHD and normal control groups as a whole (Ernst et al., 1994). These findings imply that like boys with ADHD, girls may also exhibit frontal lobe dysfunction.

Although it is a more indirect measure of frontal lobe abnormalities, examining corpus callosum morphology has provided some support for a frontal dysfunction. Because the callosum receives fibers from areas of the cortex and retains the topographic structure of these areas, it is possible that abnormalities detected in the callosum reflect abnormalities in the structures from which the fibers originated. Using MRI, Giedd et al. (1994) determined that the rostrum and rostral body were significantly smaller in ADHD children. Furthermore, there was a negative correlation between callosum area and ratings of impulsivity/hyperactivity on the Conner's teacher and parent questionnaires. In partial agreement with these findings, Hynd et al. (1991) found that children with ADHD had

smaller corpus callosums, specifically in the genu, splenum, and the area just anterior to the splenum. In addition to supporting the frontal lobe theory, these results also suggest an alternative to the theory of right frontal dysfunction, namely, that there may be abnormalities in interhemispheric transmission of information.

In summary, initial findings suggest that children with the combined subtype of ADHD exhibit deficits in the anterior attention system, particularly within the prefrontal cortices. However, in addition to the anterior attention system, Posner's model proposes the existence of two other attention systems, the posterior attention system and the vigilance network, both of which are closely connected with the AAS, and could also be the site of dysfunction in ADHD.

Posterior Attention System

In Posner & Petersen's (1990) model of attention, attention is not only voluntarily directed through the AAS, but, depending upon the instructional set, it can also be automatically directed through the posterior attention system (PAS). The PAS, which includes the superior parietal cortex, pulvinar, and superior colliculus, is responsible for the automatic orientation of attention, and receives extensive norepinephrine-rich projections from the locus coeruleus (Posner & Raichle, 1994; Rothbart, Posner, & Rosicky, 1994). Studies of patients with lesions in these areas have determined that these structures are responsible for the disengagement, re-engagement, and shifting of attention in space, respectively (Posner & Raichle, 1994).

Through norepinephrine inputs, the PAS works to orient to novel stimuli (Pliszka, McCracken, & Maas, 1996). Low baseline levels of catecholamine release followed by higher acute release during periods of stress result in good performance on focused

selective attention tasks (Pliszka et al., 1996). Focused selective attention is often described as a "filter mechanism" used to not only discriminate between relevant and irrelevant stimuli but also to bring the relevant stimuli into conscious awareness. It is hypothesized that children with ADHD have increased basal levels of norepinephrine, so that during periods of stress, the relative increase is not as great as in non-ADHD children. In corroboration, stimulant medications work in part by lowering the basal level of norepinephrine so that the PAS can respond more robustly during the presentation of novel stimuli or during periods of stress (Pliszka et al., 1996).

As enticing as this hypothesis may be, a number of studies have been unable to categorically show a deficit in focused selective attention in children with ADHD (Douglas, 1983; Halperin, 1991). Children with ADHD are also comparable to normal controls on levels of distraction (as measured by performance on a number of school-related tasks) under conditions of classroom noise, and do not show greater improvement than controls in settings without auditory distraction (Steinkamp, 1980). Although children with ADHD exhibit deficits on the Freedom from Distractibility scale (composed of Arithmetic, Digit Span, and Coding/Digit Symbol subtests) on the WAIS-R and WISC-III (for review, see Kaufman, 1994), children with other disorders (e.g. ODD, CD, PTSD, GAD, and LD) also show deficits on this scale.

Using a version of the continuous performance task, in which distracter numbers were flashed either to the right or left of a target, one study found that adolescents with ADHD, while committing more errors of omission and commission than controls, did not differ significantly from them (Barkley, Anastopoulos, Guevremont, & Fletcher, 1991). In another study, both ADHD and control children showed large effects for distracters on

tasks of visual discrimination, but no significant interaction was found between groups, indicating that letter noise distracters affected search processes for ADHD and control children to an equivalent degree (McIntyre, Blackwell, & Denton, 1978). However, these studies failed to control for common comorbid disorders (e.g. reading disability, oppositional defiant, and conduct disorder) or for subtypes, the presence of which may have masked possible effects.

Furthermore, the lack of support for a focused selective attention deficit may be restricted to boys with the ADHD primarily hyperactive or combined subtype. In comparison to boys, girls tend to cluster in the inattentive subtype (Gaub & Carlson, 1997). Teachers often describe children with ADD-H as more daydreamy, lethargic, confused, and lost in thought, than children with hyperactive ADHD (Barkley, DuPaul, & McMurray, 1990). Based on these symptoms, it is hypothesized that as opposed to the more frontal combined subtype, ADD-H may reflect a posterior attention dysfunction (Barkley et al., 1990; Goodyear & Hynd, 1992). Because poor performance on focused selective attention tasks is more closely related to reading disability than ADHD (Halperin, 1996), it has been proposed that the ADHD primarily inattentive subtype is more closely associated with reading disability than ADHD combined type. In support, James & Taylor (1990) reported that girls who met ICD-9 criteria for hyperkinetic syndrome of childhood suffer from more severe language and cognitive problems than boys with the same diagnosis.

Although the AAS and the PAS are able to perform independently of each other, strong neural links exist between the two (Rothbart et al., 1994), and both systems usually work together in order to produce attentional shifts (Colby, 1991). Therefore, damage

anywhere along this chain can lead to deficits in attentional processing (Colby, 1991; Jackson et al., 1994). For example, Malone, Kershner, & Swanson (1992) propose that a deficit in right frontal inhibitory control may lead to the disinhibition of the right posterior areas, resulting in the distractibility often observed in ADHD. Few studies have operationally separated the two systems.

The Vigilance Network

Vigilance (often referred to as sustained attention and not to be confused with phasic alertness, or arousal) is defined as the positive ability to maintain a steady state of alertness and wakefulness during prolonged and sustained mental activity (Weinberg & Harper, 1993). The reticular activation system (RAS) has numerous interconnections with the locus coeruleus (LC), and together, these two structures maintain inhibitory actions and regulatory roles in sleep, arousal, and other autonomic functions (Rothbart et al., 1994). Like the PAS, this third attentional network has close connections with the anterior cingulate and other components of the AAS (Jackson et al., 1994). Recently, many studies have put forth an argument to equate vigilance tasks to phasic arousal tasks. However, in contrast to vigilance tasks, phasic arousal tasks are typically warned reaction time tasks in which the duration is not of minutes or hours, but rather of seconds and milliseconds (Parasuraman, Warm, & See, 1998). A review of vigilance and arousal studies indicated that the factors which increase or decrease a participant's arousal levels are positively correlated to the participant's overall degree of vigilance (Parasuraman et al., 1998). However, while phasic arousal and vigilance are associated concepts and are both part of the larger attentional network, there is little support to accept or reject the

argument that brief, warned reaction timed tasks are measures of vigilance (Parasuraman et al., 1998).

Experimentally, poor performance (as measured by reaction time and number of errors) at the outset of a task, is believed to reflect deficits in arousal (Parsuraman et al., 1998; Van der Meere, Wekking, & Sergeant, 1991) because the arousal system is responsible for the initial allocation of attention. In contrast, a decrement in performance over time is believed to reflect deficits in sustained attention (vigilance) (Parsuraman et al., 1998; Halperin, 1991). Although a number of studies have not found a sustained attention deficit in children with ADHD, patterns of performance on the CPT have supported an arousal deficit (Sergeant & Van der Meere, 1990; Van der Meere et al., 1991). That is, children with ADHD are slower, commit more errors, and are more variable in their performance than controls (Sergeant, 1989). However, their performance is poor from the outset, and does not decrease over time to a greater degree than controls, a pattern indicative of a deficit in arousal. That is, if the arousal system does not function optimally, varying states of hyper or hypoarousal would be predicted, resulting in variable performance. Therefore, an examination of error rates and performance variance (i.e. standard deviation) is necessary to discriminate vigilance from arousal deficits.

Norepinephrine, because it is released in situations which require rapid response or allocation of attention (Servan-Schreiber & Cohen, 1992), has been implicated in the maintenance of arousal within this network (Rothbart et al., 1994). Medications which have the greatest therapeutic benefit in ADHD not only increase the release of dopamine but also increase adrenergic-mediated inhibition of norepinephrine in the locus coeruleus (McCracken, 1991). Thus, reducing norepinephrine activity through α_2 -adrenergic

inhibition (through which most drugs used in the treatment of ADHD work) can have a positive effect on ADHD symptomology (McCracken, 1991). Experimentally, improvements on the CPT can be seen following administration of methylphenidate, a NE agonist (Nigg, Hinshaw, & Halperin, 1996). However, neither MAO-A inhibitors (clorgyline) nor mixed MAO-A/MAO-B inhibitors (tranylcypromine), which, like methylphenidate, are also NE agonists, improve performance on the CPT (Levy, 1991). If this system has been correctly understood phenomenologically, the ineffectiveness of some NE agonists, but not others, to improve CPT performance somewhat weakens the argument for a dysfunction in this attentional system.

However, the argument for a dysfunctional arousal system does not only rest on pharmacologic evidence and performance on the CPT. Physiologic measures (e.g. EEG, auditory evoked responses, and skin conductance measures) reveal a negative correlation between state of arousal and degree of hyperactivity (Weinberg & Harper, 1993). A deficit in arousal results in an inability to maintain a wakeful state if prevented from fidgeting, moving, or daydreaming, during continuous mental processing (Weinberg & Harper, 1993). Weinberg & Harper (1993) believe that children with ADHD maintain excessive motor behavior in order to stimulate or maximize the functioning of their arousal network. Based on this reasoning girls with ADHD and the inattentive subtype would be expected to display less severe arousal deficits because they tend to exhibit lower levels of hyperactivity and fewer externalizing behaviors than boys with ADHD (Gaub & Carlson, 1997).

Because of the intricate connections between the AAS and the vigilance network (Jackson et al., 1994; Posner & Raichle, 1994), and the associations between vigilance and

arousal systems (Parasuraman et al., 1998), it is possible that a dysfunction within the right lateral frontal lobe, which is part of the AAS, could lead to deficits in the maintenance of arousal (Rothlind, Posner, & Schaughency, 1991). This hypothesis is particularly inviting because lowered arousal leads to less inhibition, which may account for the impulsivity observed in children with ADHD. In addition, individuals diagnosed with primary disorders of vigilance (e.g. narcolepsy, brain lesions of the midbrain and right cerebral hemisphere, depression, etc.) typically report trouble with concentration, daydreaming, difficulty focusing attention, disorganization, fidgeting, and talking excessively, which are also typical symptoms of ADHD (Weinberg & Harper, 1993).

The numerous interactions among these three networks exemplify the brain's ability to achieve a balance between reflexive, or data-driven processes, and controlled, or goal-oriented processes. This distributed view of attentional processes emphasizes the fact that no one structure operates independently, and that damage to one part of the attentional system leads to dysfunction in another. All three systems are therefore potentially relevant to understanding the core mechanism of dysfunction in children with ADHD. Based on an understanding of these neural networks, an attentional orienting paradigm was created to examine visuospatial orienting in brain-lesioned individuals. The next section examines the use of this paradigm in children with ADHD, and how such a paradigm may be useful in the study of ADHD.

Visuospatial Orientation of Attention

One laboratory paradigm that measures the orientation of visuospatial attention to the right or left visual field (Posner, 1987), has not only been used to study ADHD (Carter, Krener, Chaderjian, Northcutt, & Wolfe, 1995; Nigg, Swanson, & Hinshaw,

1997; Novak, Solanto, Abikoff, 1995; Pearson, Yaffee, Loveland, & Norton, 1995; Swanson, Posner, Potkin, Bonforte, Youpa, & Fiore, 1991; Tomporowski, Tinsley, & Hager, 1994), but also Alzheimer's (Parasuraman, Greenwood, & Alexander, 1995), Parkinson's (Flowers & Robertson, 1995), brain injury, and collosotomy patients (Cremona-Meteyard & Geffen 1994; Mangun et al., 1994). The benefit of this using this paradigm to study children with ADHD is it can distinguish between dysfunctions of the AAS, PAS, or vigilance network, as well as determine if any lateral effects are present.

In this paradigm, participants fixate on a crosshair located at the center of a computer screen. Because they are required to maintain fixation and refrain from making eye movements, this task is intended to measure the covert, as opposed to the overt, orientation of attention. A box is located on either side of the fixation point within which a target (usually an asterisk) appears at variable intervals following an exogenous cue (a "brightening" of one of the boxes). The exogenous cue validly predicts target location 50% of the time, with invalid (brightening of the box in which the cue does not appear) and null (double brightening) cues used for comparison (Posner & Raichle, 1994). In contrast, the endogenous cue (a central arrow) probabilistically determines the location of the target (80% valid, 20% invalid), so participants can utilize information provided by the cue in a voluntary manner to control the location of their attention in space. Both cueing methods facilitate detection at the cued location with an accompanying increase in reaction time costs at the non-cued position. The child's task is to press the spacebar as fast as possible following detection of the target, with the dependent variable being time to keypress.

There are many differences between orienting via an exogenous or endogenous cue. Because exogenous orientation is automatic, the processing of peripheral cues does not draw upon attentional capacity, is more difficult to suppress than endogenous cues, is not dependent upon a set of expectations (i.e. the probability of a peripheral signal), and is more effective than endogenous cues in drawing attention (Jonides, 1981). It is also believed that exogenous cues are predominantly processed within the PAS, in contrast to the endogenous cues which are predominantly processed within the AAS. Furthermore, in contrast to the exogenous cue, endogenous cueing results in a longer period of facilitation and produces a longer delay period before increased reaction time costs are observed at the contralateral position (Rafal & Henik, 1994).

The cue-target delay, or stimulus onset asynchrony (SOA), can also be adjusted to examine the voluntary or involuntary allocation of attention to areas in space. A 100 ms SOA taps the automatic allocation of attention because such a delay is too rapid for attention to be moved voluntarily. On the other hand, when cue-target intervals exceed 300 ms during exogenous cueing conditions, a phenomenon known as the inhibition of return occurs. Inhibition of return is a pattern of response in which reaction times for validly cued targets are longer than those for invalidly cued targets. The effect lasts for one to two seconds following reorientation at a new location and is hypothesized to reflect a bias towards orienting to novel locations during visual scanning (Clohessy, Posner, Rothbart, & Vecera, 1991; Posner, Rafal, Choate, & Vaughan, 1985). Because the appearance of the target is delayed, following an initial facilitation at the cued location, it is in the subject's best interest to call upon the AAS to actively maintain attention at fixation, or to spread it diffusely across the display. When the target finally

appears at the cued location, the bias towards orienting towards novel locations in space results in slower detection at a validly cued location (Rafal & Henik, 1994).

Developmentally, Lane & Pearson (1983) demonstrated that children as young as five years of age were able to covertly orient attention when given peripheral cues. Younger children also exhibited more costs associated with invalid cueing than did older children, and when cues probabilistically determined target location, only adults utilized this information to aide their performance (Lane & Pearson, 1983). The relative paucity of developmental data on children with respect to covert orienting processes make it difficult to draw firm conclusions about these processes in normal children, much less children with developmental disorders such as ADHD. For instance, although robust cueing effects, including inhibition of return, are observed in adults with ADHD, it is unclear whether the effects would be as robust in normal children.

Covert orienting in children with ADHD

Several studies have used variations of this paradigm to study children with ADHD. Summaries of the findings are presented in Table 1.

In the first of these studies, Swanson et al. (1991) hypothesized that ADHD combined subtype boys possess a dysfunction in the right parietal lobe which would manifest itself in slower reaction time to left visual field targets following a 100 ms SOA. Although the results of the study did not support this hypothesis, it was unclear if the same could be said of girls with ADHD (only 3 of the 28 ADHD children were girls) or of the primarily inattentive subtype. The three girls with ADHD exhibited significant right visual field deficits in all six cue x delay conditions, but boys with ADHD only exhibited right visual field deficits in the un-cued and invalid cueing conditions at the

Table 1. Summary of results found for studies examining covert attention in children with ADHD. Standard deviations appear in parentheses when available. LVF= left visual field, RVF = right visual field.

Source	Sample	Procedure	Results	Implications
Swanson et al. (1991)	ADHD: n=28, Girls: Boys = 3:25 Mean age (yrs) = 9.0 IQ= not obtained Control: n=27, Girls: Boys=15:12 Mean age (yrs.) =9.2 IQ= not obtained	1) Used valid, invalid, and “no cue” probabilistic exogenous cues 2) No observation of eye movement 3) Did not control for ODD or CD	1) ADHD children exhibited decreased costs to detection of invalidly/null cued LVF targets at an 800 ms SOA or 2) Increased costs to invalidly/null cued right visual field targets at an 800 ms SOA 3) Number of omission, but not anticipation, errors greater in ADHD children.	1) A left hemispheric deficit in sustained attention or 2) A right hemisphere deficit in the disengagement or movement of attention

Table 1 (cont'd).

Tompson et al. (1994)	ADHD: n=17, All boys Mean age (yrs) = 10.67 IQ= not obtained Control: n=18, All boys Mean age (yrs) = 9.58 IQ= not obtained	1) Used valid, invalid, and “no cue” endogenous cues. 2) Forced choice response requiring the use of two hands 3) No observation of eye movement 4) Excluded children who were comorbid for ODD or CD	1) ADHD children had slower mean reaction times than normal control children or adults 2) No group differences in the cost or benefit of cueing 3) Benefits of valid cueing first seen in adults, then normal controls, then ADHD children 4) No group differences in anticipation errors (responses occurring prior to target onset) 5) No visual field data provided	A deficit in the controlled orientation of attention
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Table 1 (cont'd).

Pearson et al. (1995)	ADHD: n=20, Girls: Boys = 2:18 Mean age (yrs) =10.7 IQ (PPVT-R) =99 Control: n=20; F:M=2:18 Mean age (yrs) =10.7 IQ (PPVT-R)=106	1) Used probabilistic valid, invalid, and null exogenous and endogenous cues. 2) Forced choice response task requiring the use of two hands 3) No observation of eye movement 4) Did not control for LD, ODD, or CD	1) No group difference in the mean of the median reaction times 2) No group differences in reaction time to exogenous or endogenous cues. 3) ADHD children slower to respond to targets during exogenous cueing at an 800 ms SOA 4) Waxing and waning pattern of responses with increasing cue- target intervals 5) No visual field data provided and no analysis of anticipation or omission error data performed	1) Possible AAS dysfunction 2) Children with ADHD possess less flexible orienting abilities
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Table 1 (cont'd).

Novak et al. (1995)	ADHD: n=16, Girls: Boys = 1:15 Mean age (yrs) =11.1 (1.42) IQ (WISC-R FS)= 110 (13.8) Control: n=10, Girls: Boys = 2:8 Mean age (yrs) =11.5 (1.83) IQ (PPVT)= 104 (14.4)	1) Used simultaneously appearing non-predictive exogenous and endogenous cues at a single 500 ms cue-target delay. 3) No observation of eye movement 4) Controlled for LD, CD, and other psychiatric conditions other than ODD	1) No group differences in median reaction times or visual field 2) ADHD children on methylphenidate (0.3 mg/kg) exhibited decreased costs to invalidly cued R VF targets. 3) Children with ADHD committed more anticipation errors ($rt \leq 100$ ms) than controls	1) No evidence for a visual field deficit in children with ADHD. 2) Although methylphenidate treatment improved performance for R VF targets, the significance was only slight and due primarily to the placebo condition 3) No overall evidence of an orienting deficit
Carter et al. (1995)	ADHD: n=20, F:M=5:15 Mean age (yrs) = 10.5 (1.2) IQ=94.7 (12.5) Control: n=20, F:M=5:15 Mean age (yrs) =10.7 (1.2) IQ=101.9 (13.0)	1) Used separate endogenous and exogenous cueing procedures with 100 and 800 ms cue-target delays 2) Observed eye movements 3) Excluded children with comorbid anxiety/mood disorders, and CD	1) No group differences in reaction times for exogenous cues. 2) Loss of costs to invalidly cued L VF targets in the endogenous condition at 800 ms SOA 3) ADHD children committed more anticipation errors ($rt \leq 150$ ms) during endogenous cueing conditions than control.	1) A right hemisphere deficit in controlled orienting of attention

Table 1 (cont'd).

Nigg et al. (1997)	ADHD: n=27, All boys Mean age (yrs) =9.26 (2.16) IQ (WISC-R Verbal) =109.96 (14.2) Control: n=17, all boys Mean age (yrs) = 9.88 (1.86) IQ (WISC-R Verbal) = 122.06 (14.8)	1) Used probabilistic exogenous cues at 100 and 800 ms cue-target delay. Used valid, invalid, and "no cue" conditions 2) Did not observe eye movements 3) Did not control for comorbid conditions	1) ADHD children exhibited increased reaction times to LVF targets following exogenously presented "no cue" conditions at 100 ms SOA 2) Methylphenidate treatment improved reaction times to LVF targets at moderate doses (0.6 mg/kg). RVF improved at low doses (0.3 mg/kg) 3) No group differences in the number of anticipation errors	A right hemisphere deficit in arousal
Aman et al. (1998)	ADHD: n=22, All boys Mean age (yrs) =12.14 (1.21) IQ (WISC-R estimate) = 110.50 (8.18) Control: n=22, All boys Mean age (yrs) = 12.13 (1.21) IQ (WISC-R estimate) = 110.00 (7.34)	1) Used probabilistic exogenous cues. Used valid, invalid, and null cue conditions at 100 and 500 ms cue-target delay 2) Did not observe eye movements 3) Controlled for conduct disorder and reading disability	1) No group differences in median reaction times. 2) No main effect for visual field 3) No main effect for delay. 4) No analysis of anticipation or error data performed	Results do not support a parietal lobe deficit in children with ADHD

800 ms delay (Swanson et al, 1991). Although the control group exhibited a small right visual field advantage ($RVF-LVF = -12$ ms) when reaction times were averaged across cue and delay conditions, the ADHD group exhibited increased right visual field costs ($RVF-LVF = 21$ ms). Specifically, the ADHD children were faster to detect invalidly or null cued left (as opposed to right) visual field targets at the 800 ms SOA. Swanson et al. (1991) interpreted the decreased left visual field costs as a sustained attention deficit of the left hemisphere. In other words, when invalidly cued to the right visual field, children with ADHD were unable to sustain attention at that location for the duration of the 800 ms prior to target onset, so when the target appeared in the left visual field, ADHD boys oriented to the left more rapidly than controls (Swanson, 1991). To note, Swanson's "sustained attention" processes as measured by the 800 ms SOA, is not the same as the sustained attention measured by the CPT, which lasts for several minutes. Therefore, comparing or generalizing the performance between these two tasks is not necessarily valid (Swanson et al., 1991). Swanson et al. (1991) also noted that the patterns of performance could also indicate increased costs for invalidly and null cued right visual field targets, suggesting a right hemisphere deficit in the disengagement or movement of attention from the left to right visual field. That is, instead of a facilitated detection of invalidly cued left visual field targets at the 800 ms SOA, the data could also indicate increased costs for invalidly and null cued right visual field targets (Swanson et al., 1991). The latter interpretation is supported by comparing reaction times between the 100 and 800 ms SOA. Although both groups exhibited a reduction in reaction time for invalidly and null cued left visual field targets at the 800 ms SOA, only the control group exhibited a significant reduction for invalidly and null cued right visual field targets

(Swanson et al., 1991). So, rather than interpreting the reduction in reaction time for the left invalid and null cue in the ADHD group as a facilitation, it may be more accurate to view lack of such a reduction in the right visual field as an increase in cost. This interpretation therefore suggests a right hemisphere deficit in the disengagement or movement of attention from the left to right visual field. Swanson et al. (1991) further found that in comparison to controls, the number of omission (responses made 3000 ms after target onset), but not anticipation (responses made within 100 ms of target onset), errors was significantly greater in children with ADHD. Therefore, it might be argued that based on this measure of impulsivity/inattention, children with ADHD did not appear to be more impulsive in their responses, but did exhibit increased inattention.

However, neither the control nor ADHD group exhibited inhibition of return. Because inhibition of return develops between the third and six month of life (Clohessy et al., 1991; Harman, Posner, Rothbart, & Thomas-Thrapp, 1994), it should be visible with the nine year olds used in the study. The absence might be explained by Swanson et al.'s (1991) use of predictive exogenous cues in combination with a long (800 ms) cue-target delay. Although Jonides (1981) found that probabilistic expectations did not affect performance during exogenous cueing, it is possible that predictive cueing, in combination with a long cue-target delay (which allows enough time for voluntary attentional orientation to occur), worked together to invoke attention in more of an endogenous, than an exogenous, manner.

The next study used forced-choice endogenous cueing conditions and five cue-target intervals (50, 150, 300, 500, and 1000 ms) to compare performance between 18 adults, 18 non-ADHD children, and 17 ADHD children (Tomprowski, et al., 1994).

Although ADHD children had the slowest mean reaction times in all conditions, the non-ADHD and adult groups were not significantly different from one another, and, unlike Swanson et al.'s (1991) findings, there were no significant differences between the three groups in terms of the costs and benefits of cueing (Tomprowski et al., 1994). However, the benefits of valid cueing were first seen at the 150 ms SOA with adults, at the 300 ms SOA with non-ADHD children, and at the 500 ms SOA with ADHD children. The differences in the age at which participants were able to take advantage of the predictive nature of the cues may be indicative of neuro-maturational changes which allow progressively greater control over the voluntary allocation of attention. That children with ADHD exhibited this facilitory effect later than non-ADHD children could indicate a developmental delay in the AAS, leading to a deficit in the voluntary allocation of attention. Tomporowski et al. (1994) did not find group differences in the number of anticipation errors (responses occurring prior to target onset), supporting Swanson et al.'s (1991) findings, and indicating that on this measure of impulsivity, children with ADHD do not differ from controls. The number of omission errors was not reported. And, unfortunately for the laterality model of ADHD, Tomporowski et al. (1994) did not report right/left visual field data.

A third study used forced choice responses to endogenous and predictive exogenous cueing conditions at two visual angles (2.8° and 8.2°) (Pearson et al., 1995). Based on the means of the median reaction times, Pearson et al. (1995) found that although children with ADHD responded more slowly than normal controls, the difference was not significant. There were also no group differences in reaction time based on visual angle, indicating that the time required to move attention did not differ

between ADHD and controls (Pearson, 1995). Furthermore, reaction times to central and peripheral cues did not differ between children with ADHD and normal controls, indicating that both groups were equally able to direct their attention in an automatic or voluntary manner. These results contradict Tomporowski et al. (1994) who found that ADHD children were significantly slower than controls when endogenously cued. There was, however, a small but significant increase in reaction time at the 800 ms delay in the ADHD group, indicating that children with ADHD may not be as efficient in utilizing the extra time to voluntarily orient their attention, thus implying the presence of an AAS dysfunction (Pearson et al., 1995). These results partially support Swanson et al. (1991), who found increased costs for invalidly cued right visual field targets at the 800 ms cue-target delay. When Pearson et al. (1995) collapsed data from the endogenous and exogenous cueing conditions, they observed that children with ADHD exhibited a "waxing and waning" pattern of performance at longer cue-target intervals, in which reaction times to invalid and neutral cues varied. To Pearson et al. (1995), these data implied that children with ADHD possess less flexible orienting capabilities which later lead to deficits in higher attentional functioning as development progresses. However, closer examination of the data reveal that the waxing and waning pattern was mainly observed during the exogenous cueing condition, and may simply reflect a carryover of the inhibition of return from trial to trial. Unfortunately, the inter-trial interval was not reported, preventing confirmation of this hypothesis. In addition, although the ADHD group exhibited a significant facilitory effect of valid cueing, there were only small differences in reaction times between neutral (mean = 933 ms) and invalid cues (mean = 955 ms), indicating a lack of cost for invalid cues. This is in contrast to the control group,

which showed significant costs associated with invalid cueing. This finding partially supports Swanson et al.'s (1991) first interpretation that children with ADHD exhibit decreased costs to invalidly cued left visual field targets at the 800 ms SOA. No analysis of commission or omission errors was undertaken by Pearson et al. (1995), and like Tomporowski et al. (1994), no visual field data were presented.

The fourth study on covert orienting in children with ADHD simultaneously cued subjects with predictive endogenous and exogenous cues followed 500 ms later by the appearance of a target (Novak et al., 1995). As in the Pearson et al. (1995) study, no difference in median reaction times was found between the ADHD versus non-ADHD group. These findings were in contrast to Tomporowski et al.'s (1994) findings, and may be the result of sampling bias, the simultaneous use of both endogenous and exogenous cueing procedures, or an older sample of children (mean age = 11.5 years in Novak et al. (1995), 10.7 years in Pearson et al. (1995)). This may also have occurred because both Pearson et al. (1995) and Novak et al. (1995) used median rather than mean reaction times for analysis. When performance was assessed during methylphenidate treatment (0.3 mg/kg), children with ADHD exhibited a decrease in reaction time to invalidly cued right visual field targets (Novak et al., 1995). That is, methylphenidate preferentially improved right hemisphere attentional orienting. This finding supports Swanson et al.'s (1991) secondary interpretation of greater costs associated with right visual field target detection. Novak et al. (1995) also found that children with ADHD made significantly more anticipation errors (responses occurring within 100 ms following target onset) than normal controls (4.3% and 1.9%, respectively), contradicting Swanson et al. (1991)'s and Tomporowski et al. (1994)'s null findings in this respect.

The fifth study of covert orienting in children with ADHD, Carter et al. (1995), is the only study which adequately differentiated between endogenous and exogenous cueing procedures. Children with ADHD made significantly more anticipation errors (responses less than 150 ms) than controls during endogenous, but not exogenous, cueing procedures, which may, along with Novak's findings, further indicate an anterior system dysfunction manifesting as increased impulsivity, particularly when attention must be effortfully controlled (Carter et al., 1995). Carter et al. (1995) found no significant group differences in the exogenous cueing procedures and observed normal inhibition of return in both ADHD and control groups. However, children with ADHD did exhibit a lack of costs towards invalidly cued left visual field targets at the 800 ms cue-target delay for endogenous cues. While Swanson et al. (1991), interpreted their findings of decreased costs to exogenously and invalidly cued left visual field targets as a left hemisphere deficit in sustained attention, Carter et al. (1995) proposed that because only the target appeared in a peripheral location (the endogenous cue being foveal), any lateral asymmetry observed must reflect the function of the hemisphere processing detection (the right hemisphere). That is, when invalidly cued to the right visual field, Carter et al. (1995) proposed that right hemisphere frontal inhibitory mechanisms were unable to adequately inhibit detection of objects in the left visual field, so that when the target appeared in the left visual field, reaction time to detection was facilitated. Although such an explanation is inviting, it is unclear which hemisphere is responsible for the voluntary orientation of visual spatial attention. No analysis of error rate and visual field was performed, but if children with ADHD did possess a right hemisphere inhibitory deficit, they might also have committed more anticipation errors for left as opposed to right

visual field targets. The similarity in findings between Swanson's exogenous and Carter's endogenous cueing effects further bolsters the hypothesis that Swanson et al.'s (1991) use of predictive exogenous cues facilitated the voluntary movement of attention. Carter et al. (1995) hypothesized that the right hemisphere dysfunction was the result of diminished right frontal catecholamine activity. Such an interpretation would be consistent with Novak et al.'s (1995) finding that methylphenidate preferentially improves right, but not left, hemisphere functioning.

That covert orienting can be disrupted following catecholamine depletion was observed in an endogenous cueing task following the administration of droperidol or clonidine (both of which are central dopamine and noradrenergic inhibitors) in normal male adults (Clark, Geffen, & Geffen, 1989). In comparison to controls, participants in the experimental group exhibited reduced costs to invalidly cued targets, indicating facilitated attentional disengagement or movement (Clark et al., 1989). These results are also consistent with Pearson et al.'s (1995) and Swanson et al.'s (1995) report of decreased costs to invalid cueing in children with ADHD, and provide support for a catecholamine imbalance in ADHD. No visual field data were analyzed.

In the first direct attempt to replicate any findings for covert orienting in children with ADHD, Nigg et al. (1997) used predictive exogenous cues and found that boys with ADHD exhibited slower overall reaction times to target detection than non-ADHD boys. ADHD boys, and their biologic, but not adoptive, parents further exhibited slower reaction times to un-cued left visual field targets. Biologic parents also exhibited an increased time to detection of invalidly cued right visual field targets at the 100, but not 800, ms cue-target delay. Performance following administration of a low (0.3 mg/kg) or

moderate (0.6 mg/kg) dose of methylphenidate improved for the left visual field, but only low doses improved functioning of the right visual field (Nigg et al., 1997). This improved performance for targets in the right visual field was similar to Novak et al.'s (1995) findings of improved performance for invalidly cued right visual field targets which used the same low dosage. Nigg et al. (1997) interpreted their overall findings as evidence of a noradrenergic dysfunction of the initial activation of attention, implying a right lateralized dysfunction in the vigilance network. Of the six studies reviewed, only Nigg et al. (1997) found a deficit at the 100 ms cue-target delay, and, like Swanson et al. (1991), Nigg et al. (1997) failed to observe the inhibition of return at the 800 ms cue-target delay. Along with Swanson et al. (1991) and Tomporowski et al. (1994), Nigg et al. (1997) did not find group differences in the number of anticipation errors.

The most recent study examining covert orienting in children with ADHD used predictive exogenous cues at 100 and 500 ms cue-target delays and did not find overall differences between ADHD boys and controls in median times to target detection (Aman, Roberts, & Pennington, 1988). In addition, no effect of delay or visual field was observed. Given consistent previous findings in both normal and ADHD populations of expected delay effects, this lack of a delay effect is striking. It may be that the 500 ms delay did not allow enough time to produce facilitation of target detection. Furthermore, there were no group differences in the validity effect, indicating that both groups were equally facilitated and inhibited by valid and invalid cues, respectively. These findings contradict Swanson et al. (1991), Carter et al. (1995), and Nigg et al. (1997), who found interactions between cue type and group. Although mean reaction times were not reported, Aman et al. (1998) reported longer reaction times to invalidly versus validly

cued trials. Based on this report, it is likely that Aman et al. (1998) also failed to observe inhibition of return at the 500 ms cue-target delay.

The prior seven studies have examined the covert, as opposed to overt, orientation of attention. The distinction is an important one because eye movements cannot occur prior to the covert allocation of attention to a target location (Hoffman & Subramaniam, 1995). Furthermore, although people may be capable of monitoring several areas of input at once, once a target is detected, the probability of detecting others is decreased (Jackson et al., 1994). Monitoring eye movements is therefore an issue to consider in designing such studies. Of the studies which looked at covert orienting in children with ADHD, only Carter et al. (1995) monitored eye movements and they reported that during endogenous cueing, control and ADHD children moved their eyes on 14% and 17% of the trials, respectively. Eye movements were even greater during exogenous cueing; 21% of controls and 27% of ADHD children made eye movements. However, the data were not reanalyzed to exclude trials in which movements were made. Although it takes at least 200 ms for a saccade to occur, in previous studies, the range of cue-target intervals has fallen between 100 to 1000 ms.

Although not directly related to the study of visuospatial attention in children with ADHD, commonly associated problems such as Oppositional Defiant Disorder (ODD), Conduct Disorder (CD), and Learning Disabilities (LD), can have a substantial effect on findings. Because children with such disorders often possess similar deficits to those with ADHD, the presence of these disorders may conceal or even fully account for results which might otherwise be attributed to ADHD alone (Barkley, 1996; Hechtman, 1994). For instance, Brannan & Williams (1987) found that children who were poor readers ($n =$

6, scoring one standard deviation below a diagnostic reading scale), in comparison to children who were good readers and adults, were less accurate on a forced choice cueing paradigm when presented with probabilistic exogenous cues. Poor readers were less able to utilize the cueing information to direct their attention to locations in space.

Of the seven studies on covert orienting in children with ADHD, only four (Aman et al., 1998, Carter et al., 1995, Novak et al., 1995, and Tomporowski et al. 1994), specifically excluded children with comorbid conditions (e.g. anxiety/mood disorders, CD, ODD, and LDs) from their sample. Although no consistent differences between these three studies and the remaining three are clearly evident, it is difficult to determine how much of the variability in findings is due to wide methodological differences in paradigm specifics, and how much it is due to uncontrolled factors such as comorbidity.

Exclusionary precautions are a good first step in controlling for the possible effects of comorbidity, but it does not allow for a dimensional analysis of subclinical problems.

This type of analysis is important in clinical research because cutoffs for diagnoses are somewhat arbitrary. Furthermore, children with ADHD often have elevated symptoms of aggression, defiance, and lower reading and IQ scores, even when comorbid disorders are excluded (Nigg, Hinshaw, Carte, & Treuting, 1998). For instance, all participants in the Novak et al. (1995) study possessed scores on the reading, spelling, and mathematics subtests of the Kaufman Test of Educational Achievement (KTEA) ≥ 80 and within 15 points of their WISC-R full scale IQ. However, had a child scored 85 on the KTEA and an 86 on the WISC-R, s/he would not have been excluded, even though such scores may be indicative of a subclinical learning disability. Regression or covariance analysis would

allow for dimensional analysis of the data which would statistically remove effects related to comorbid behavioral problems (Nigg et al., 1998).

Given the variety of cueing methods, it is difficult to make comparisons regarding the nature of the orienting deficit (i.e. anterior, posterior, right, or left hemisphere dysfunction), but it appears that children with ADHD have more difficulty with the voluntary orientation of attention as opposed to the automatic, and more right as opposed to left hemisphere deficits. That is, Carter et al. (1995), Pearson et al. (1995), Swanson et al. (1991), and Tomporowski et al. (1994) all found poorer performance for the ADHD group at either longer cue-target delays or with endogenous cues. However, Nigg et al. (1997) found increased reaction times for exogenously presented null cues at a 100 ms SOA, but Novak et al. (1995) and Aman et al. (1998) did not find significant group x delay differences. If the rate of anticipation and omission data can be interpreted as evidence for frontal as opposed to parietal deficits, then two studies (Novak et al., 1995; Carter et al., 1995) found more anticipation errors in children with ADHD, and three did not (Swanson et al., 1991; Tomporowski et al., 1994; Nigg et al., 1997).

With respect to lateral differences, four studies found evidence for right hemisphere deficits (either in terms of reaction time differences or improvements with the administration of methylphenidate; Carter et al., 1995; Nigg et al., 1997; Novak et al., 1995; Swanson et al., 1991) but one found no effect of visual field on performance (Aman et al., 1998). None of the studies examined possible subtype effects on performance. Clarification regarding the effects of subtypes of ADHD and comorbidity on performance is sorely required.

Rationale for Current Study

Finding laboratory measures to make ADHD diagnoses might aid in reducing the heterogeneity and reliability in diagnoses as well as help in deciding among competing etiologic theories. For example, if the theories of a right hemisphere or frontal lobe deficit are upheld, certain patterns of performance on Posner's covert attention task could be used to subtype ADHD with greater validity than observer reports. However, studies utilizing this paradigm with ADHD children have yielded inconsistent results with only one direct replication attempt. To clarify the situation, in addition to further replication studies, needed changes in procedure include: (1) separation of endogenous and exogenous cueing conditions (Carter et al., 1995), (2) examination of ADHD subtypes, (3) controlling for common comorbid diagnoses and problems, specifically learning disability and conduct disorder, (4) examination of girls (separately from boys), and (5) assessment of eye movements. Although examination of all these issues was beyond the scope of this initial study, the first three issues were chosen for the present study and the fourth was partially considered.

In an attempt to replicate and extend Carter et al.'s (1995) findings, the study examined the allocation of visuospatial attention to the right or left visual field at 100 and 800 ms cue-target delays following an exogenous cue in boys and girls with ADHD and ADD-H and normal controls while controlling for comorbid conditions.

Hypotheses were as follows:

Hypothesis 1: All children with ADHD, regardless of subtype, have a right hemisphere deficit. They will therefore exhibit: (1) slower time to detection of targets appearing in the left versus right visual field following a null cue, (2) smaller reaction

time benefits observed for validly cued left versus right visual field, (3) smaller reaction time costs associated with invalid cueing to the left versus right visual field (Carter et al., 1995). Therefore, a significant group x visual field interaction will be observed in the directions stated.

Hypothesis 2: If children with the combined subtype of ADHD primarily suffer from an AAS dysfunction. Their reaction time to target detection at the 800 ms SOA will therefore more reliably distinguish them from controls or the inattentive subtype, regardless of sex. Likewise, if children with the inattentive subtype of ADHD primarily suffer from a PAS dysfunction, their reaction time to target detection at the 100 ms SOA will more reliably distinguish them from controls or the combined subtype, regardless of sex. If this hypothesis is supported, a group x delay interaction is predicted in the directions stated.

Hypothesis 3: If ADHD is primarily a result of a dysfunction of the arousal network, in comparison to controls, an increased overall variability in reaction time will be observed in all children with ADHD regardless of subtype (i.e. a main effect of overall standard deviation between groups).

Hypothesis 4: If children with the combined subtype of ADHD possess more frontal than parietal dysfunction (manifesting as increased impulsivity), the total number of commission errors (i.e. responses faster than 100 ms) will be greater in children with ADHD than in children with ADD-H or controls. If children with the inattentive subtype of ADHD possess more parietal than frontal dysfunction (manifesting as decreased sensitivity to novel stimuli). The total number of omission errors (i.e. responses slower than 1500 ms) will be greater in ADD-H as opposed to ADHD or controls.

Method

Participants

Sixty-three children (41 boys and 22 girls), between the ages of 6 and 13 (mean age = 120.25 ± 17.91 months) from a wide range of socio-economic strata participated. Families were recruited through local schools and clinic referrals in the greater Lansing area. Ethnic makeup of child participants was: 80.4% White, 8.9% Black, 8.9% Hispanic, and 1.8% Asian American. Children were excluded if they had a primary sensorimotor handicap, frank neurological disorder, psychosis, autism (or other pervasive developmental disorder), an estimated Full Scale IQ below 80, or if English was not their first language. Children were screened in as possible ADHD if (1) they had a prior diagnosis of ADHD by a pediatrician who had examined rating scales from both parents and teachers, or (2) they exceeded screening cutoffs on at least one normative parent and one teacher rating of ADHD (i.e. if the CBCL Attention Problems Scale Rating was $T > 60$, if the Conner's Hyperactivity Index was $T > 65$, or if at least 4 symptoms of ADHD were endorsed on the Parent and Teacher SNAP-IV).

Diagnostic groups included (1) unimpaired control children ($n = 23$, 9 girls and 14 boys), (2) children with ADHD ($n = 26$, 8 girls and 18 boys), and (3) children with ADD-H ($n = 14$, 5 girls and 9 boys). Child diagnoses were based on the Diagnostic and Statistical Manual of Mental Disorders, 4th Ed. (DSM-IV; APA, 1994). Positive diagnoses for ADHD required that children met stringent criteria (e.g. symptom-related impairment in two settings, home and school, an onset prior to 7 years of age, and symptoms present for at least 6 months) on the DISC-IV. If impairment criteria were met, and if by parent report, children were one or two symptoms shy of full diagnosis on the

DISC-IV, then additional teacher-reported symptoms were “substituted” to bring children to diagnostic threshold for ADHD. The latter procedure is similar to that employed by the NIMH multi-site study of ADHD treatment outcome (Hinshaw et al., 1997). Children were excluded from the study if they did not meet diagnostic threshold on the DISC-IV. Children who met criteria in only one setting (e.g. parent, but not school, ratings exceeded criteria, or vice versa) were considered to have situational problems and were placed in the control group (n = 2).

The presence or absence of comorbid anxiety disorder, depression, mania, post traumatic stress disorder, obsessive compulsive disorder, tic disorder, oppositional defiant disorder, and conduct disorder were screened in the same fashion using the Conners', CBCL, and SNAP-IV. The DISC-IV was used to establish final diagnoses. Learning disability was diagnosed when (1) the mean of the WIAT Reading and Spelling standard scores was less than or equal to 85, and (2) there was a 15 point (-1 SD) discrepancy between the WISC-III Full Scale IQ and the mean of the WIAT Reading and Spelling standard scores. Of those participating, 6 children (5 ADHD, 1 ADD-H) had a comorbid learning disability and 3 (all ADHD) had comorbid conduct disorder (See Table 2).

Medications

Children previously diagnosed with ADHD were free of psychostimulant medication 24 hours prior to the day of testing. The short half-life of stimulants (Pelham, 1993) and the 24 hour washout period suggests that medication effects on performance were minimal.

Table 2. Primary and comorbid diagnostic data for ADHD, ADD, and Control children

	ADHD	ADD-H	Control	Total
<u>n</u> Girls	8	5	9	22
<u>n</u> Boys	18	9	14	41
Comorbid LD Present	5	1	0	6
Comorbid CD Present	3	0	0	3
Total	26	14	23	63

Procedures and Measures

Parents responded to an initial mailing through local schools and pediatric clinics. Participants thus represented both clinic-referred and non-referred children. Control children for clinic-referred ADHD children were recruited through a neighboring general pediatric clinic in the same medical center. Control children for community-identified ADHD children were recruited from a community-wide mailing. Efforts were made to match control children on age and sex as much as possible. After screening and prior to testing, parents provided written informed consent to the procedures and children provided verbal assent. Families then completed two on-campus visits, during which the children completed IQ, achievement, and the computer-generated attentional tests. Parents completed child rating scales and a diagnostic interview during the visits (SNAP-IV, Conners, CBCL, and DISC-IV).

WISC-III (5 subtests). Full scale IQs were estimated from a five subtest short form from the WISC-III (Information, Similarities, Vocabulary, Picture Completion, and Block Design; Wechsler, 1991). Information, Similarities, and Vocabulary on the WISC-III tap verbal reasoning abilities. Picture Completion and Block Design tap non-verbal

reasoning abilities. Standardization data for the WISC-III are based on a nationally representative stratified random sample of 2,200 children. Test-retest reliability for the Full Scale IQ estimate from this short form is $r = 0.95$. Validity of the short form in relation to the full battery is $r = 0.90$ (Sattler, 1992).

WIAT Reading and Spelling. In Reading, children are read a list of words of increasing complexity. The words are read aloud, followed by a sentence using the word, and then the word is repeated. Normed on a subset of the same sample as the WISC-III, age-based reliabilities (6-13 yrs.) for spelling range between 0.88 and 0.93, and for reading, between 0.91 and 0.95. Test-retest reliability across grades 1, 3, 5, 8, and 10 for both spelling and reading is 0.94 (Wechsler, 1992). Respective scaled scores were the outcome variables of interest.

Covert Orienting. The exogenous visual orienting task (lasting approximately 15 minutes) was presented on a Dell Pentium or 486 PC using the MEL programming language. During the procedure, children had their heads centered and stabilized with a chin rest 14.05 in. from the monitor. Ambient light was eliminated and the examiner remained present.

An initial instruction screen with a diagram of the experimental procedure was read aloud. Children were told to always keep their eyes on the central fixation cross, that sometimes one of the boxes would light up to let them know where the asterisk would appear, sometimes the wrong box would light up, and sometimes both boxes would light up. They were told to press the spacebar as fast as they could when they saw an asterisk in one of the peripheral boxes. The experimenter then turned off the lights, sat directly behind the child, and started the task.

Two dimly lit boxes appeared throughout the procedure 15° to the right and left of the central fixation point. Although this angle is larger than those used in previous studies, if children with ADHD possess an attentional orienting deficit, the larger visual angle could be expected to exacerbate this weakness and increase performance differences between groups. A second, larger box drawn around the first box resulted in a “brightening” effect and served as the exogenous cue. Left, right, and null (double box) cues occurred randomly and with equal probability (i.e. one third of the trials each). Single sided cues validly predicted target location 50% of the time. The target and cue, once initiated, remained visible throughout the duration of the trial. The inter-trial interval was set at 1500 ms and the cue-target delay randomly varied at either 100 or 800 ms (See Figure 1).

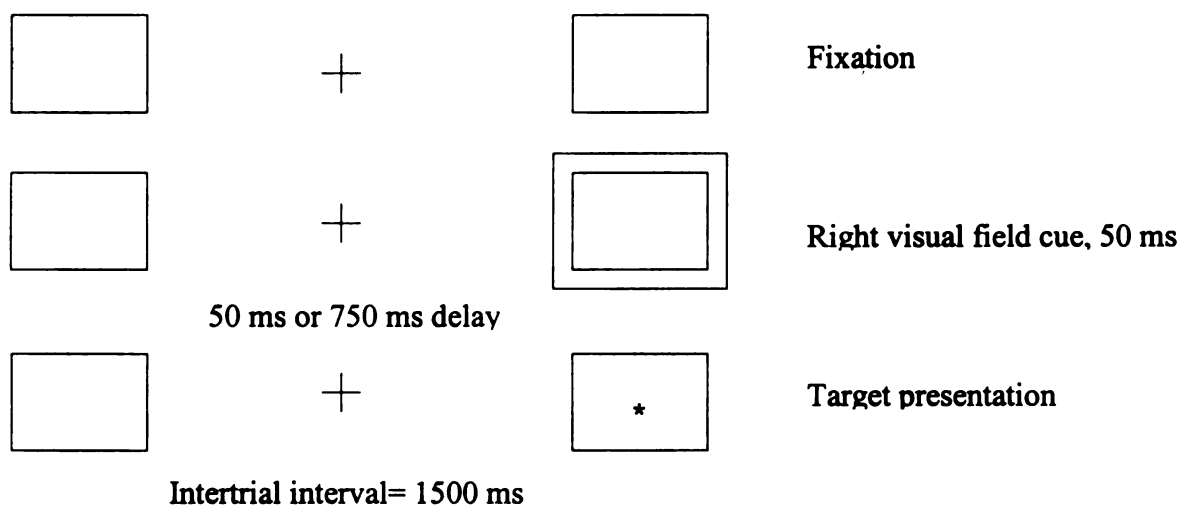


Figure 1. Overview of stimulus presentation for a validly cued right visual field target. Not to scale.

A total of five blocks of 48 trials were presented, with rest periods offered between each

block. Dependent variables included individual mean and standard deviation in reaction time, as well as the number of commission (i.e. responses occurring within 0-99 ms of target onset) and omission errors (i.e. responses occurring within 1501-3000 ms of target onset).

Swanson, Nolan, and Pelham, DSM-IV symptom checklist (SNAP-IV). The SNAP-IV (Swanson et al., 1982) is a face-valid DSM-IV checklist of symptoms for ADHD and a range of associated conditions (e.g. ODD, CD, anxiety disorders, depression, tic disorders, obsessive compulsive disorder, and post-traumatic stress disorder) which is widely used for diagnostic screening within research settings.

Conners' Abbreviated Symptom Questionnaire (Conners'). Normed on a sample of 2,426 children from over 95% of the states in North America, the Conners' norming sample is composed of 84% Caucasians, 4.3% African-Americans, 3.8% Hispanic, 2.1% Asian, 1% Native American, and 4.7% Other. Test-retest reliability scores (in a 6-8 week interval) are 0.62, 0.73, 0.85, and 0.72 for the oppositional, cognitive problems, hyperactivity, and ADHD subscales, respectively (Conners, 1997). Internal reliability estimates range from 0.86 to 0.94. Parent and teacher forms possess similar reliabilities.

Child Behavior Checklist and Teacher Report Form (CBCL/TRF). The CBCL/TRF and is a standardized checklist which examines both internalizing and externalizing behavior problems. Parent and teacher forms are parallel and demonstrate similar reliabilities. It is perhaps the most widely used measure of child behavior in clinical and research settings. The checklist was normed on a national sample of non-handicapped children between the ages of 4 and 18. These children were representative of the 48 contiguous states in terms of SES, ethnicity, region, and urban/rural residence. At a

seven day interval, the CBCL has a test-retest reliability score of 0.87 for the competence scales, 0.89 for problem scales, and 0.90 for the attention scales. Over one year, test-retest reliability is 0.62, 0.75, and 0.77 for the competence, problem, and attention scales, respectively. In terms of content validity, almost all the CBCL items can discriminate between demographically matched referred and non-referred children (Achenbach, 1991). The screening cut-off score of $T = 60$ on the attention problem scale has the best empirical support for ruling out ADHD (Chen et al., 1994).

Diagnostic Interview Schedule for Children for DSM-IV (DISC-IV). The DISC-IV is a computer-assisted structured interview developed by NIMH (NIMH, 1993). It was administered by trained interviewers to the primary caregiver (in most cases, the mother) who answered questions regarding their child's behavior within the last year, last six months, last month, and whole life. Depending upon parental report of behavior on the SNAP-IV, CBCL, Conner's, and a brief clinical interview, specific modules in the DISC-IV (i.e. ADHD, ODD, CD, generalized anxiety disorder, mania, major depression, tic disorders, and obsessive compulsive disorder) were administered to formally confirm or rule out the presence of comorbid disorders. Information regarding the severity of problem behaviors, remission, and age of onset, is obtained. DSM-IV diagnoses are made by an algorithm based on the overall problem score, age of onset, duration, and level of impairment for each module. Satisfactory reliability and validity data have been previously reported in the literature (Shaffer et al., 1993).

Sample Size and Power Analysis

Previous studies using the visuospatial attentional task indicate “medium” to “large” effect sizes ($f = \frac{1}{2}d$) (Cohen, 1988) for simple main effects ($f = 0.25-0.40$) as well

as two and three way interactions ($f = 0.33$) (Nigg et al., 1997). Assuming medium effect sizes ($f = 0.25$), given the repeated measures design, power for simple effects and interactions of intercept exceeded 0.80 for three group ANOVAs. However, power was greater to detect 2-group differences between ADHD and controls than it was for ADD-H versus controls due to the smaller ADD-H group. Power to detect whether group sex effects were not satisfactory, so sex effects were checked in a preliminary analysis across groups.

Data Reduction and Analysis

Dependent variables. The dependent variables were reaction time to target detection (using only those responses which occurred between 100-1500 ms following target onset), within subject standard deviation in reaction time, and the total number of omission (i.e. responses occurring between 1501-3000 ms following target onset) and commission (i.e. responses occurring between 0-99 ms following target onset) errors.

Analysis. The experiment generated a five-factor design with three within-subject factors (cue validity, visual field, and delay) and one between-subject factors (group), which was analyzed using a mixed factorial ANCOVA. Initial analyses indicated no significant sex effects, therefore, boys and girls were combined for the remainder of the analyses. Children with comorbid Conduct Disorder (CD) and Learning Disorder (LD) were included in all initial analyses. Composite CD and LD scores were then created to dimensionally control for subclinical CD or LD in secondary and tertiary analyses. Four composite disruptive behavior scores were generated to cover the range of associated behavior children display, by averaging the ODD and CD symptoms on the parent and teacher SNAP-IV, and by averaging the aggressive and delinquent symptoms endorsed on

the CBCL and TRF. A composite reading score was generated by averaging the WIAT Reading and Spelling scaled scores. In secondary analyses, children with comorbid LD or CD were included and composite ODD/CD/LD scores were covaried. Then, children with comorbid LD/CD were removed ($n = 9$) with remaining subclinical CD or LD scores covaried. Children with other comorbid disorders (e.g. post traumatic stress disorder, generalized anxiety disorder, or depression) were also comorbid for either learning disability or conduct disorder. Thus, when children with LD/CD were removed from analysis, all comorbid disorders were removed. These procedures lead to no changes in any findings, as noted later. Reaction times of less than 100 ms (commission errors) or more than 1500 ms (omission errors) were excluded from analyses.

Hypothesis 1 was designed to replicate Swanson et al.'s (1991), Carter et al.'s (1995), and Nigg et al.'s (1997) findings of hemispheric differences in attentional control between ADHD and controls at different cue-target delays.

Hypothesis 1 (Lateral effects). If supported, regardless of subtype, a repeated measures ANCOVA would find a significant group (2) x visual field (2) x cue type (3) interaction.

Hypothesis 2 (Subtype differences). If supported, a repeated measures ANCOVA would find a significant subgroup (3) x delay (2) interaction.

Hypothesis 3 (Arousal): If supported, regardless of subtype, a one-way ANOVA would find a main effect of group on standard deviation.

Hypothesis 4 (Errors): If supported, a one-way ANOVA would find a main effect of group on the number of commission (i.e. responses less than 100 ms) and omission (i.e. responses greater than 1501 ms) errors.

Results

Preliminary Description. Diagnostic groups (ADHD, ADD-H, and controls) did not differ in age or Reading scores and the proportion of boys and girls did not differ significantly, $p > 0.05$. Differences in IQ approached significance, $F(2,56) = 2.72$, $p = 0.07$ due to marginally lower scores in the ADHD group. Differences in WIAT Spelling scores were significantly different, $F(2, 59) = 7.05$, $p < 0.01$ (See Table 3). Post-hoc analyses indicated that WIAT Spelling scores were significantly better in controls than ADHD ($p < 0.01$) and ADD-H children ($p \leq 0.05$). This was not surprising given that all of the LD children were in the ADHD and ADD-H groups.

Table 3. Demographic data for ADHD, ADD, and Control children

	ADHD Mean (SD)	ADD-H Mean (SD)	Control Mean (SD)
Mean age (years)	9.81 (1.83)	10.54 (1.17)	9.94 (1.20)
Mean IQ	101.33 (15.43)	108.43 (16.65)	111.95 (14.84)
Mean WIAT Reading standard score	97.35 (15.94)	100.64 (15.40)	109.14 (27.82)
Mean WIAT Spelling standard score	93.92 (13.59)	96.57 (14.92)	109.41 (15.95)

Between-group comparisons of inattentive and hyperactive symptoms endorsed by parents and teachers on the SNAP-IV and CBCL generally supported the diagnostic groupings (See Table 4). Parents and teachers rated ADHD and ADD-H children as significantly more inattentive than controls, and although parents rated ADHD children as more inattentive than ADD-H children, teachers did not. Parents and teachers rated children with ADHD as significantly more hyperactive than children with ADD-H or

Table 4. Symptom endorsements on the SNAP-IV and CBCL for ADHD, ADD, and Control children.

	ADHD Mean (SD)	ADD-H Mean (SD)	Control Mean (SD)
SNAP Attention —Dad	1.73 (0.71)	1.24 (0.70)	0.47 (0.40)
SNAP Attention —Mom	2.02 (0.72)	1.45 (0.56)	0.61 (0.49)
SNAP Attention —Teacher	1.96 (0.62)	1.5 (0.85)	0.34 (0.42)
CBCL Attention Problems —Dad	70.50 (7.81)	59.23 (7.41)	52.63 (5.96)
CBCL Attention Problems—Mom	73.50 (8.63)	57.69 (8.57)	54.43 (7.03)
CBCL Attention Problems—Teacher	66.82 (10.23)	61.18 (9.84)	51.83 (2.66)
SNAP Hyperactivity —Dad	1.61 (0.76)	0.66 (0.49)	0.34 (0.45)
SNAP Hyperactivity —Mom	1.91 (0.65)	0.62 (0.48)	0.43 (0.47)
SNAP Hyperactivity —Teacher	1.14 (0.64)	0.29 (0.38)	0.33 (0.60)

controls, but neither parents nor teachers rated ADD-H children as more hyperactive than controls.

With regards to the orienting paradigm, significant main effects were observed for cue type, $F(2, 124) = 27.83$, $p < 0.01$ and delay, $F(1, 62) = 50.40$, $p < 0.01$, but not visual field $F(1, 62) = 0.06$, $p = 0.81$ (See Figure 2). Such effects were expected based on the nature of the design. Overall, reaction times following invalid cues were significantly slower than those following valid and null cues. However, reaction times following valid cues were not significantly faster than null cues. As expected, reaction times were faster following an 800 than 100 ms cue-target delay because an 800 ms SOA provides time for attention to fully orient prior to target onset. A significant cue x delay interaction, $F(2, 124) = 19.54$, $\eta^2 = 0.24$, $p < 0.01$, was also found due to the presence of expected cueing

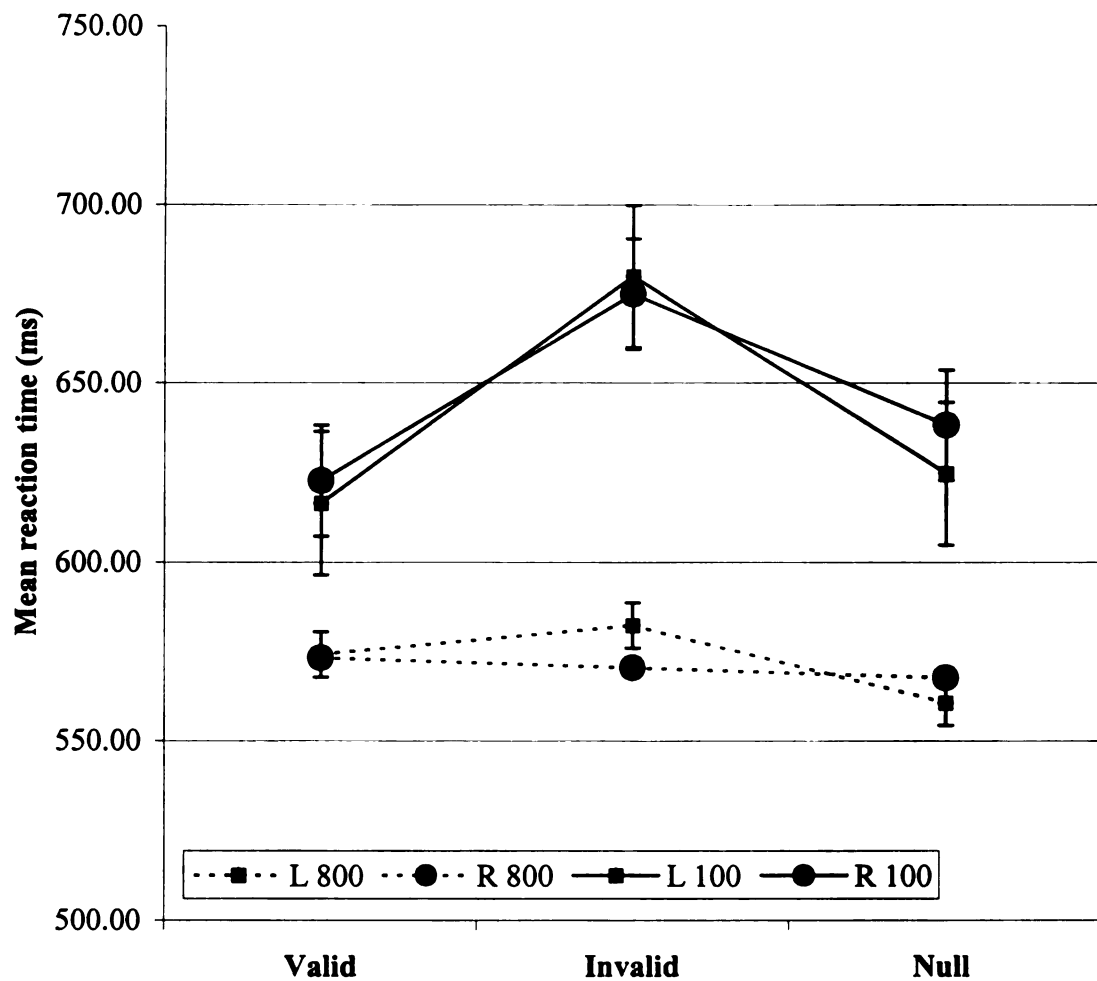


Figure 2. Mean reaction time to target detection across groups.
 L = Left visual field target, R = Right visual field target, 100 = 100 ms SOA, 800 = 800 ms SOA

effects at the 100 but not 800 ms SOA. Inhibition of return, in which reaction times to invalid cues are faster than those following valid cues at longer cue-target delays, was not observed, $F(1, 62) = 0.22$, $\eta^2 < 0.01$, $p = 0.64$. Sex main effects for reaction time approached but did not exceed significance, $F(1, 61) = 3.42$, $\eta^2 < 0.05$, $p = 0.07$. The group x sex interaction was not significant, however, $F(2, 57) = 0.06$, $\eta^2 < 0.01$, $p = 0.94$ (See Figure 2).

Because the main effect of sex was not significant and because power was not satisfactory to examine group sex effects, sexes were combined for the remainder of analyses.

Hypothesis 1: When diagnoses were collapsed across subtypes (i.e. when ADHD and ADD-H children were combined), a mixed factorial ANCOVA found a marginally significant visual field x cue x group interaction, $F(2, 122) = 2.55$, $\eta^2 = 0.04$, $p = 0.08$ (See Figure 3). Extremely small and non-significant group x visual field [$F(1, 61) = 0.002$, $\eta^2 < 0.01$, $p = 0.97$] and group x cue interactions [$F(1, 122) = 0.32$, $\eta^2 < 0.01$, $p = 0.73$], were also found.

Effects never approached significance ($p > 0.05$) when compared between ADHD subtypes, when ADD-H children were removed from analysis, when composite CD and LD symptoms were covaried, or when children with CD and LD were removed from analysis with remaining composite CD and LD symptoms covaried.

A one-way ANOVA examining visual field differences between groups in the cost of invalid cueing (invalid-null), benefit of valid cueing (valid-null), and the validity effect (invalid-valid) was performed as a secondary analysis because of prior reports concerning them (Swanson et al., 1991). No between group differences in the overall

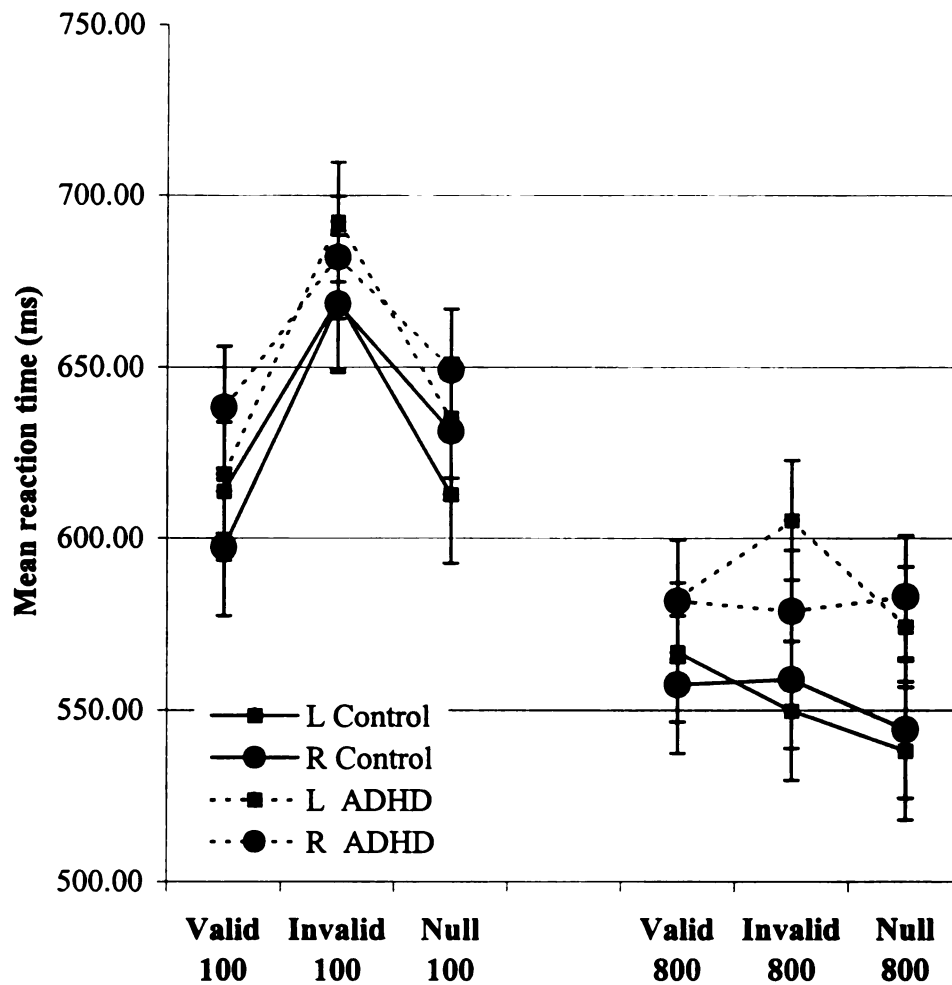


Figure 3. Mean reaction time for target detection for ADHD (collapsed across subtypes) and Control children.
L = Left visual field target, R = Right visual field target

validity effect, costs, or benefits of cueing were observed, (all $p > 0.05$), although the validity effect for left visual field targets at an 800 ms SOA approached significance, $F(1, 61) = 3.17$, $\eta^2 = 0.05$, $p = 0.08$. When children with comorbid LD or CD were removed from analysis, benefits of valid cueing for right visual field targets following a 100 ms SOA also approached significance, $F(1, 52) = 2.98$, $\eta^2 = 0.05$, $p = 0.09$.

Overall, results did not support theories theory of a lateral attentional dysfunction in children with ADHD or ADD-H.

Hypothesis 2: The subtype x delay interaction was non-significant, $F(2,60) = 0.20$, $\eta^2 < 0.01$, $p = 0.82$ (See Figure 4 and Table 5). That is, children with the combined or inattentive subtype of ADHD did not respond differently from controls to either the 100 or 800 ms cue-target delay. Results remained non-significant when controls were removed from analysis ($F(1,38) = 0.08$, $p = 0.78$), when the composite CD and LD symptoms were covaried ($F(2,54) = 0.29$, $p = 0.75$), or when children with CD and LD were removed from analysis with remaining composite CD and LD symptoms covaried ($F(2,46) = 0.96$, $p = 0.91$).

This pattern of results does not support the theory of a PAS dysfunction in children with the inattentive subtype of ADHD, or an AAS dysfunction in children with the combined subtype of ADHD.

Hypothesis 3: Children with ADHD, regardless of subtype, exhibited greater overall standard deviations in reaction time than controls, $F(1, 60) = 11.56$, $\eta^2 = 0.16$, $p < 0.01$ (See Table 5). When broken down by subtypes, ADHD, but not ADD-H, children exhibited greater variability than controls, $F(2,59) = 5.85$, $\eta^2 = 0.17$, $p < 0.01$.

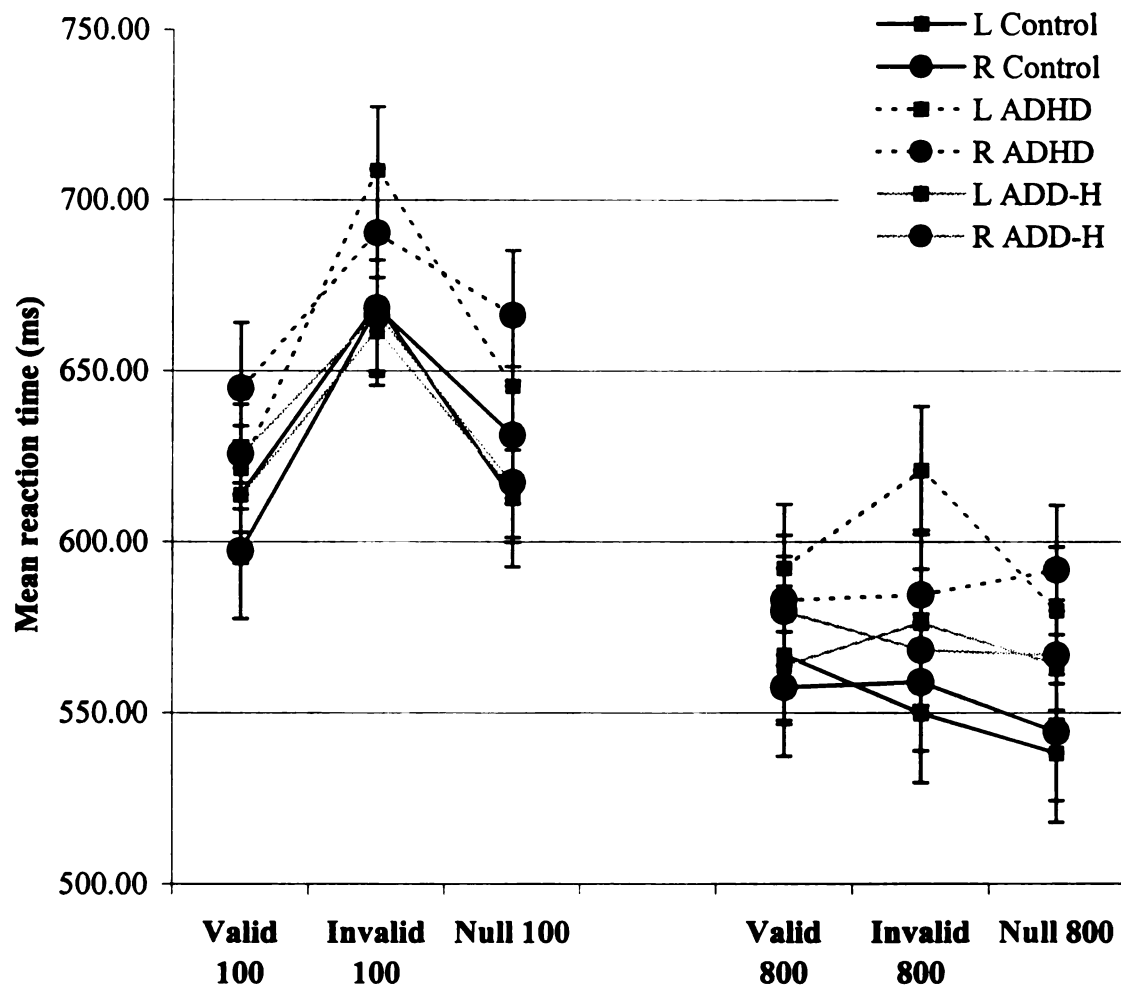


Figure 4. Mean reaction time to target detection for ADHD, ADD-H, and Control children. L = Left visual field, R = Right visual field

Table 5. Reaction time to target detection for ADHD, ADD-H, and Control children

Delay	Visual Field	Cue	ADHD Mean (SD)	ADD-H Mean (SD)	Controls Mean (SD)
100	Left	Valid	621.49 (89.80)	613.83 (104.71)	613.74 (162.22)
100	Left	Invalid	708.76 (121.10)	661.43 (114.05)	669.48 (167.73)
100	Left	Null	645.6 (109.83)	615.57 (141.35)	612.86 (171.71)
100	Right	Valid	644.93 (106.74)	625.73 (131.67)	597.3 (156.41)
100	Right	Invalid	690.29 (111.84)	666.13 (122.74)	668.33 (170.31)
100	Right	Null	666.1 (108.42)	617.32 (106.76)	631.22 (175.88)
800	Left	Valid	592.28 (128.97)	563.47 (114.46)	566.75 (143.74)
800	Left	Invalid	620.93 (129.95)	576.26 (84.89)	549.81 (143.78)
800	Left	Null	579.78 (124.73)	563.86 (94.49)	538.17 (137.78)
800	Right	Valid	582.81 (98.19)	579.63 (109.75)	557.36 (134.48)
800	Right	Invalid	584.37 (112.25)	568.18 (82.99)	558.84 (148.62)
800	Right	Null	591.73 (107.61)	566.72 (83.04)	544.33 (126.05)

Results remained significant when composite CD and LD symptoms were covaried [$F(2,54) = 5.00, \eta^2 = 0.16, p = 0.01$] and when children with CD and LD were removed from analysis with remaining composite CD and LD symptoms were covaried, $F(2,46) = 5.32, \eta^2 = 0.19, p < 0.01$.

When comparing standard deviations across time, a mixed factorial ANCOVA (with ADHD and ADD-H children combined), found no significant group x block interaction, $F(4, 240) = 0.15, \eta^2 < 0.01, p = 0.96$ (See Figure 5). Results remained non-significant when subtypes were separated, when composite CD and LD symptoms were covaried ($F(4, 220) = 0.60, p = 0.67$) and when children with CD and LD were removed from analysis with remaining composite CD and LD symptoms covaried ($F(4,188) = 0.87, p = 0.49$).

Overall, the results supported an arousal dysfunction in the combined, but not inattentive, subtype of ADHD.

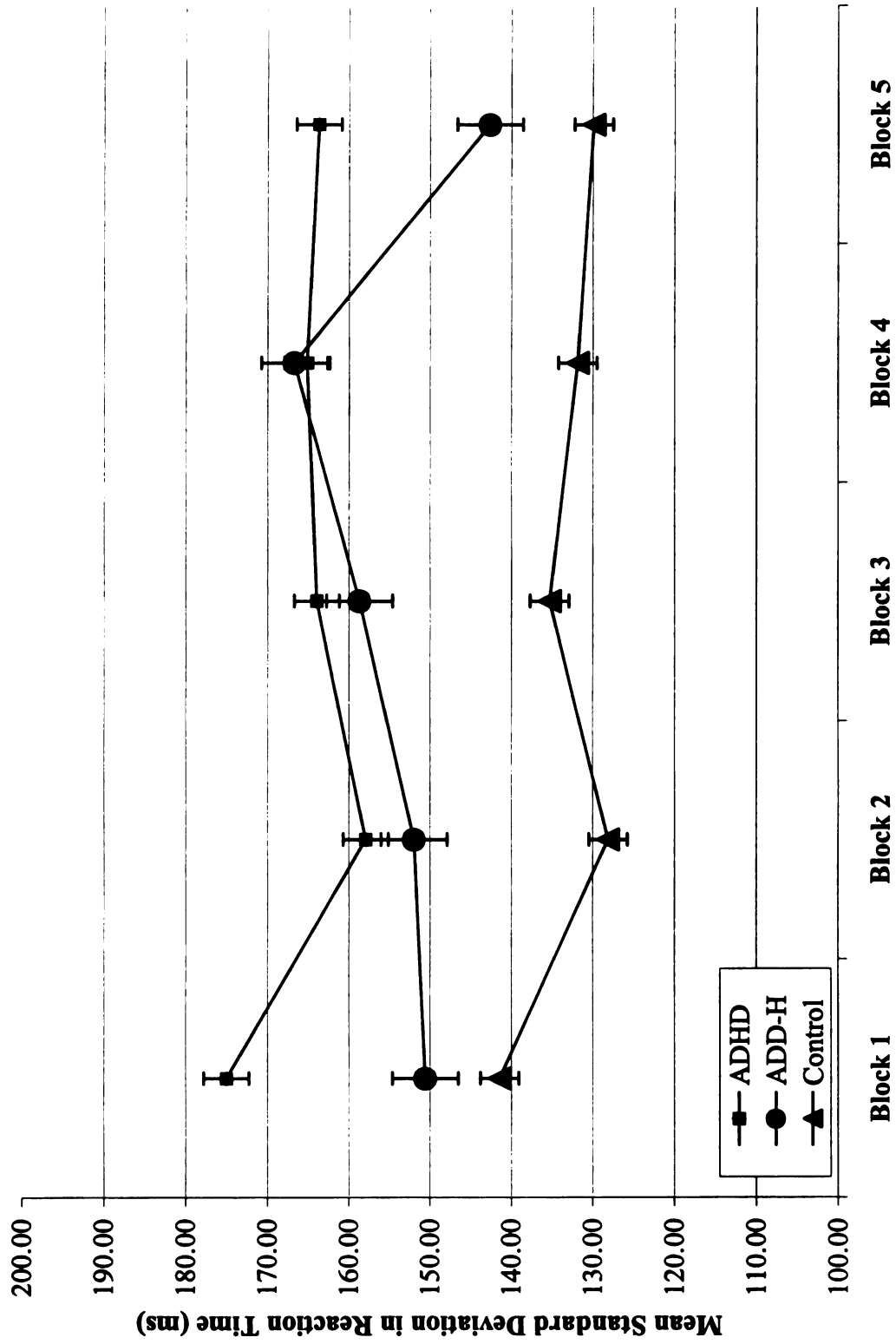


Figure 5. Mean standard deviation in reaction time across blocks of trials for ADHD, ADD-H, and Control children.

Hypothesis 4: A one-way ANOVA found no significant main effect of group on the number of commission errors (i.e. responses faster than 100 ms), $F(2,63) = 0.03$, $\eta^2 = 0.17$, $p = 0.97$ (See Figure 6). The mean percent of commission errors for the combined, inattentive, and control children were 4.08%, 3.24%, and 1.80%, respectively. The number of omission errors (i.e. responses slower than 1500 ms) between subtypes approached but did not exceed significance, $F(2,63) = 2.53$, $\eta^2 = 0.74$, $p = 0.09$ (See Figure 6). The mean percent of omission errors for combined, inattentive, and control children were 1.74%, 1.88%, and 1.98%, respectively. Results remained non-significant when children with comorbid LD or CD were removed from analysis.

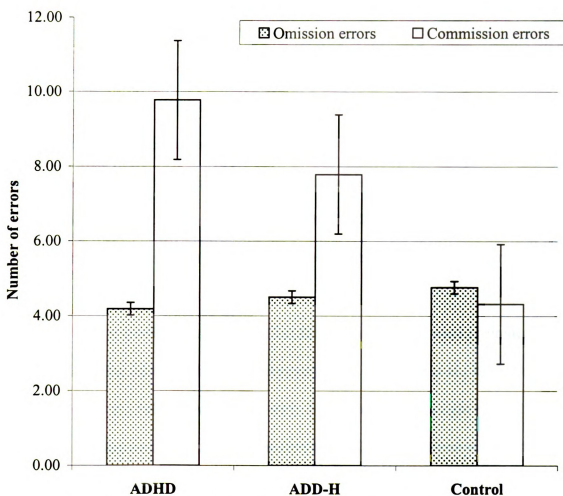


Figure 6. Number of omission (responses occurring 1501-3000 ms after target onset) and commission errors (responses occurring 0-99 ms after target onset)

Discussion

The purpose of this study was to test competing hypotheses about attentional mechanisms in ADHD by examining performance on a version of Posner's covert attention task. Although replication attempts have been few and of only limited success, prior studies found some evidence of a right hemisphere dysfunction and of an anterior, as opposed to posterior, dysfunction in attentional orientation. The inconsistent findings across prior studies may have been in part due to methodological differences, although the present results suggest that the effects observed in previous studies do not replicate well.

The current study found no main effect for sex, so the sexes were combined for the remainder of the analyses. Although previous studies (Swanson et al., 1991; Carter et al., 1995; Nigg et al., 1997; Novak et al., 1995) found evidence for a right hemispheric dysfunction in attentional orientation in children with ADHD the current study did not. And, although Carter et al. (1995) and Swanson et al. (1991) found group differences in the costs and benefits of invalid and valid cueing, respectively, the current study did not.

The current study also did not find support for an anterior attention system dysfunction in children with the combined subtype of ADHD, or for a posterior attention system dysfunction in children with the inattentive subtype of ADHD. Like Aman et al.'s (1998) lack of significant findings, these null results contradict other studies (Carter et al., 1995; Pearson, 1995; Swanson et al., 1991; Tomporowski, 1994) which have argued for the presence of a frontal, as opposed to parietal, lobe dysfunction in children with ADHD.

Children with ADHD, regardless of subtype, did exhibit greater overall variability in reaction time than controls, a pattern that might be interpreted as consistent with an

arousal dysfunction. This finding contradicts Novak et al. (1995) who did not find between group differences in reaction time standard deviation. However, the groups did not differ in reaction time to target detection over successive blocks, supporting previous arguments that children with ADHD do not possess a sustained attention, or vigilance system dysfunction (Van der Meere, 1996). This finding somewhat contradicts the Pearson et al. (1995) study which found that when endogenous and exogenous cues were collapsed, in comparison to controls, children with ADHD exhibited greater inconsistencies in the benefits and costs of valid and invalid cueing over time.

In comparison to controls, the number of omission errors children with the inattentive subtype of ADHD committed approached, but did not exceed, significance. Commission errors did not differ between groups. And, as with Hypothesis 2 (subtype differences in delay effects), this pattern of results does not support predictions that children with the combined subtype of ADHD suffer from an AAS dysfunction manifesting as increased impulsivity in responses, or that children with the inattentive subtype of ADHD suffer from a PAS dysfunction manifesting as increased lack of responses. In previous studies, Carter et al. (1995) and Novak et al. (1995), but not Nigg et al. (1997), Swanson et al. (1991), or Tomporowski et al. (1994), found that in comparison to controls, children with ADHD made significantly more anticipation errors (i.e. responses faster than 100 ms). Notably, Carter et al. (1995) only found this result in the endogenous, but not exogenous cueing condition. In similarity with the current study, however, Swanson et al. (1991) did find that the number of omission errors (responses over 3000 ms) was significantly higher in ADHD as opposed to control children.

It is important to note that the lack of results is unlikely to be due to insufficient power to detect effects. Previous studies using this task indicated “medium” to “large” effect sizes, and power for simple effects and interactions in the present study exceeded 0.80 to detect medium effect sizes. Furthermore, effect sizes for key hypothesized effects in the present study were very small, failing to replicate the effect sizes reported elsewhere. No results were due to the presence of comorbid disorders; no difference in results was observed when such children were removed from analysis, or when they were removed from analyses and the remaining subclinical symptoms were covaried.

One methodological difference which may account for the lack of significant results is that 15° visual angle may have been too wide to generate reliable cueing effects or to allow normal orienting processes to occur. Carter et al. (1995), Nigg et al. (1996), Novak et al. (1995), and Swanson et al. (1991) used a 5° visual angle, while Aman et al. (1998) used a 7.5° visual angle, Tomporowski et al. (1994) used a 2° visual angle, and Pearson et al. (1995) used angles ranging from 2°-8°. The original logic behind the use of such a large angle was that if children with ADHD possessed difficulties in attentional orientation, the difficulties may be exacerbated and the effects magnified by increasing the length which attention had to travel. However, it may be that by making the angles so large, children were unable to be effectively cued.

The large visual angle is probably not the only explanation for the lack of significant results for Hypothesis 1 or 2. It may also be that although the paradigm produces robust effects in adults, it is not as reliable in children. Difficulty obtaining the expected cueing effects has been found in each of the previous studies as well. For example, Swanson et al. (1991) found that valid cues significantly decreased reaction

time to target detection in comparison to invalid or null cues, but that reaction times following invalid or null cues did not significantly differ. Similarly, Carter et al. (1995), Tomporowski et al. (1994), and the present study found that reaction times following invalidly cued targets at a 100 ms SOA were significantly slower than those following valid or null cues, but that reaction times to valid or null cues were no different from one another. When Pearson et al. (1995) collapsed the ADHD and control groups, they found significant differences across all cueing conditions (i.e. valid < neutral < invalid), but noted that children with ADHD showed virtually no added cost of invalid as compared to null cues. And, although Novak et al. (1995), Nigg et al (1997), and Aman et al. (1998) found faster reaction times to valid as opposed to invalid cues, they did not directly compare these to reaction times following null cues (either double brightening of the boxes or no alerting cue at all) to determine if those reaction times were significantly different than those following valid or invalid cues.

Aside from the expected benefits, costs, and neutral effects of valid, invalid, and null cues, respectively, another effect normally observed in adults is the inhibition of return. Inhibition of return refers to a pattern of response in which the time to detection of invalidly cued targets is faster than to validly cued targets at long cue-target delays (e.g. 800 ms). This occurs because the visuospatial attention system is primed towards novelty. That is, if attention is automatically drawn to a location in which the target does not immediately appear, it is less likely to return to that position. Inhibition of return is not observed when attention is voluntarily moved. Of all the studies examining covert visuospatial attention in children with ADHD, only Carter et al. (1995) has been able to produce this effect, and their success may be due to the use of non-predictive exogenous

cues. With the exception of Carter et al. (1995), each of the previous studies used predictive exogenous cues, and at the longer cue-target intervals, these types of cues encourage the voluntary movement of attention. It is therefore most likely that in previous studies, inhibition of return was not observed because participants were voluntarily moving their attention in response to the predictive nature of the cues. The cues in the current study were non-predictive, so in this case, the absence of inhibition of return may be because the visual angle between the central fixation point and target was too wide. That is, the resulting distance attention had to travel in order to detect a target in the invalidly cued position was so great that any reaction time benefit for invalid cueing at this cue-target interval was neutralized.

Another reason why expected cueing effects were not observed may be because children were not able to maintain fixation during the task. Covert shifts of attention are believed to “program” future eye movements, so it is necessary to control for such movements to ensure that children have not shifted their attention to an area where the cues are less effective. The difficulty in generating the normal and expected cueing responses in this and previous studies is of concern given that the logic behind the paradigm lies in the assumption that the cues are capable of effectively orienting attention. If attention is not oriented in the expected manner in healthy controls, or if the paradigm is not as dependable in children as in adults, then any conclusions regarding attentional processes in clinical populations, such as ADHD children, are questionable.

With this in mind, the overall results of this study do not indicate that children with ADHD or ADD-H differ from each other or from controls in visuospatial attentional orientation. This in turn further suggests that the inattentive and combined subtype of

ADHD do not possess neurologic differences, at least as far as the underlying mechanisms involved in attentional orientation are concerned. And, although children with the inattentive and combined subtypes can be validly distinguished behaviorally, the theory proposing parietal deficits in ADD-H children and frontal deficits in ADHD children, was not supported. In addition, behavioral symptoms can be affected by a multitude of non-neurologic or biologic factors, and given that the vast majority of psychological disorders have no clear-cut etiology and that DSM-IV diagnostic criteria are based on behavioral symptoms, it may be erroneous to assume that the inattentive and combined subtypes of ADHD are distinct simply because behavioral ratings differ between groups. However, since it has also proven difficult to replicate results using this paradigm, it may also be that differences in performance do exist between groups, but that they are too sensitive to minor changes in methodology to be observed consistently.

Conclusions, Limitations, and Suggestions for Future Studies

The current study found no significant differences in performance between groups with respect to the visual field of target presentation, the validity of the cue, or the cue-target delay, but did find support for an arousal dysfunction in children with ADHD. Such lack of evidence for an overall attentional orienting dysfunction in children with ADHD must be weighed along with the knowledge that expected cueing effects in the control group were not observed. This may have occurred because the visual angle was too large, because children were not able to maintain fixation, because the effects are extremely sensitive to slight changes in methodology, or because performance truly does not differ between groups. However, it is unlikely that these results are due to low power, comorbid disorders, or sex effects.

In future studies using Posner's covert orienting task, it is suggested that a smaller visual angle be used and eye movements be monitored. However, given the inability of previous studies to replicate their results, overall, the evidence for group differences on this particular task is not encouraging, and may indicate that a change of course is required. Future studies attempting to separate ADHD subtypes might consider examining performance on other controlled attention tasks to tap into AAS functioning. And, given the strong evidence for a heritable etiology, studies linking the presence of particular alleles responsible for catecholamine regulation to performance on such cognitive tasks, may be used improve diagnostic accuracy.

BIBLIOGRAPHY

BIBLIOGRAPHY

Achenbach, T. (1991). Manual for the CBCL/4-18 and 1991 Profile. Burlington, VT: University of Vermont Department of Psychiatry.

Aman, C. J., Roberts, R. J., & Pennington, B. F. (1998). A neuropsychological examination of the underlying deficit in ADHD: The frontal lobe versus right parietal lobe theories. Developmental Psychology, 34, 956-969.

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

Anastopoulos, A.D. & Barkley, R.A. (1988). Biological factors in attention deficit-hyperactivity disorder. The Behavior Therapist, 11 (3), 47-53.

Aronowitz, B., Liebowitz, M., Hollander, E., Fazzini, E., Durlach-Misteli, C., & Frenkel, M. (1994). Neuropsychiatric and neuropsychological findings in conduct disorder and attention-deficit hyperactivity disorder. The Journal of Neuropsychiatry and Clinical Neuroscience, 6, 245-249.

Barkley, R.A. (1996). Attention deficit/hyperactivity disorder. In Mash, E.J. & Barkley, R.A. (Eds.), Child Psychopathology (pp. 63-112). New York: Guilford Press.

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

Barkley, R.A., Anastopoulos, A.D., Guevremont, D.C., & Fletcher, K.E. (1991). Adolescents with ADHD: Patterns of behavioral adjustment, academic functioning, and treatment utilization. Journal of the American Academy of Child and Adolescent Psychiatry, 30, 752-76.

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

Ben-Artzy, A., Glicksohn, J., Soroker, N., Margalit, M., & Myslobodsky, M. (1996). An assessment of hemineglect in children with attention-deficit hyperactivity disorder. Developmental Psychopathology, 12 (3), 271-281.

Benton, A. (1991). Prefrontal injury and behavior in children. Developmental Neuropsychology, 7, 275-281.

Brannan, J. & Williams, M. (1987). Allocation of visual attention in good and poor readers. Perception and Psychophysics, 41, 23-28.

Carter, C., Krener, P., Chaderjian, M., Northcutt, C., & Wolfe, V. (1995). Asymmetrical visual-spatial attentional performance in ADHD: Evidence for a right hemisphere deficit. Biological Psychiatry, 37, 789-797.

Casey, B., Castellanos, F., Giedd, J., Marsh, W., Hamburger, S., & Schubert, A. (1997). Implication of right frontostriatal circuitry in response inhibition and attention-deficit/hyperactivity disorder. Journal of the American Academy of Child and Adolescent Psychiatry, 36 (3), 374-383.

Castellanos, F., Giedd, J., Marsh, W., Hamburger, S., Vaituzis, A., & Dickstein, D. (1996). Quantitative brain magnetic resonance imaging in attention-deficit hyperactivity disorder. Archives of general psychiatry, 53, 607-616.

Chen, W.J., Faraone, S.V., Biederman, J., & Tsuang, M.T. (1994). Diagnostic accuracy of the child behavior checklist scales for attention-deficit hyperactivity disorder: A receiver-operating characteristics analysis. Journal of Consulting and Clinical Psychology, 62, 1017-1025.

Clark, C., Geffen, G., & Geffen, L. (1989). Catecholamines and the covert orientation of attention in humans. Neuropsychologia, 27 (2), 131-139.

Clohessy, A., Posner, M., Rothbart, M., & Vecera, S. (1991) The development of inhibition of return in early infancy. Journal of Cognitive Neuroscience, 3 (4), 345-350.

Cohen, J. (1988). Statistical Power Analysis for the Behavioral Sciences—2nd Ed. Hillsdale, NJ: Erlbaum Associates.

Colby, C. (1991). The neuroanatomy and neurophysiology of attention. Journal of Child Neurology, 6, S90-S118.

Comings, D., Comings, B., Muhleman, D., Dietz, G., Shahbahrani, B., & Tost D. (1991). The dopamine D2 receptor locus as a modifying gene in neuropsychiatric disorders. JAMA, 266, 1793-1800.

Conners, Keith (1997). Conners' Rating Scales-Revised Technical Manual. NY: Multi Health Systems.

Cook, E., Stein, M., Krasowski, M., Cox, N., Olkon, D., & Kieffer, J. (1994). Association of attention-deficit disorder and the dopamine transporter gene. American Journal of Human Genetics, 56, 993-998.

Cremona-Meteyard, S. & Geffen, G. (1994). Persistent visuospatial attention deficits following mild head injury in Australian rules football players. Neuropsychologia, 32, 649-662.

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

Deutsch, C. & Kinsbourne, M. (1990). Genetics and biochemistry in attention deficit disorder. In M. Lewis & S. Miller (Eds.) Handbook of Developmental Psychopathology (pp. 93-107) NY: Plenum Press.

Douglas, V. I. (1983). Attentional and cognitive problems. In M. Rutter (Ed.) Developmental Neuropsychiatry (pp. 280-329), NY: Guilford.

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

Faraone, S., Biederman, J., Keenan, K., & Tsuang, M. (1991). A family-genetic study of girls with DSM-III attention deficit disorder. American Journal of Psychiatry, 148, 112-117.

Flowers, K.A., & Robertson, C (1995). Perceptual abnormalities in Parkinson's disease: top-down or bottom-up processes? Perception, 24, 1201-1221.

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

Giedd, J., Castellanos, F., Casey, B., Kozuch, P., King, A., & Hamburger, S. (1994). Quantitative morphology of the corpus callosum in attention deficit hyperactivity disorder. American Journal of Psychiatry, 151 (5), 665-669.

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

Goodyear, P. & Hynd, G. (1992). Attention deficit disorder with (ADD/H) and without (ADD/VO) hyperactivity: Behavioral and Neuropsychological differentiation. Journal of Clinical Child Psychology, 21, 273-305.

Gross-Tsur, V., Shalev, R., Manor, O., & Amir, N. (1995). Developmental right-hemisphere syndrome: clinical spectrum of the nonverbal learning disability. Journal of Learning Disabilities, 28 (2), 80-86.

Halperin, J. (1991). The clinical assessment of attention. International Journal of Neuroscience, 58, 171-182.

Halperin, J. (1996). Conceptualizing, describing, and measuring components of attention: A summary. In G.R. Lyon & N. K. Yirasagorth (Eds.), Attention, memory, and executive functions. (pp. 119-136) Baltimore: Paul Brooks.

Hamlett, K., Pellegrini, D., & Conners, C. (1987). An investigation of executive processes in problem-solving of attention-deficit disorder-hyperactive children. Journal of Pediatric Psychology, 12 (2), 227-241.

Harman, C., Posner, M., Rothbart, M., & Thomas-Thrapp, L. (1994). Development of orienting to locations and objects in human infants. Canadian Journal of Experimental Psychology, 48, 301-318.

Hechtman, L. (1994). Genetic and neurobiological aspects of attention deficit hyperactive disorder: A review. Journal of Psychiatric Neuroscience, 19 (3), 193-201.

Hinshaw, S.P., March, J.S., Abikoff, H., Arnold, L.E., Cantwell, D.P., & Conners, C.K. (1997). Comprehensive assessment of childhood attention-deficit hyperactivity disorder in the context of a multisite, multimodal clinical trial. Journal of Attention Disorders, 1, 217-234.

Hoffman, J. & Subramaniam, B. (1995). The role of visual attention in saccadic eye movements. Perception and Psychophysics, 57 (6), 787-795.

Hynd, G., Semrud-Clikeman, Lorys, A., Novey, E., Eliopoulos, D., & Lyytinen, H. (1991). Corpus callosum morphology in attention deficit-hyperactivity disorder: Morphometric analysis of MRI. Journal of Learning Disabilities, 24 (3), 141-146.

Jackson, S., Marrocco, R., & Posner, M. (1994). Networks of anatomical areas controlling visuospatial attention. Neural Networks, 7 (6/7), 925-944.

James, A. & Taylor, E. (1990). Sex differences in the hyperkinetic syndrome of childhood. Journal of Child Psychology and Psychiatry, 31, 437-446.

Jonides, J. (1981). Voluntary versus automatic control over the mind's eye movement. In J. Long & A. Baddeley (Eds.) Attention and Performance IX (pp.187-203). Hillsdale, NJ: Lawrence Erlbaum Associates.

Kaufman, A. (1994). Intelligence testing with the WISC-III. NY: John Wiley & Sons.

Lane, D. & Pearson, D. (1983). Attending to spatial locations: A developmental study. Child Development, 54, 98-104.

Levy, F. (1991). The dopamine theory of attention deficit hyperactivity disorder (ADHD). Australian and New Zealand Journal of Psychiatry, 25, 277-283.

Logan, G., Schachar, R., & Tannock, R. (1997). Impulsivity and inhibitory control. Psychological Science, 8 (1), 60-64.

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

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

Lyon, G. & Krasnegor, N. (Eds.).(1996). Attention, Memory, and Executive Function. Baltimore: Paul H. Brooks.

Malone, M., Couitis, J., Kershner, J., & Logan, W. (1994). Right hemisphere dysfunction and methylphenidate effects in children with attention-deficit/hyperactivity disorder. Journal of Child and Adolescent Psychopharmacology, 4 (4), 245-253.

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

Mangun, G., Luck, S., Plager, R., Loftus, W., Hillyard, S., & Handy, T. (1994). Monitoring the visual world: Hemispheric asymmetries and subcortical processes in attention. Journal of Cognitive Neuroscience, 6 (3), 267-275.

Mannuzza, S., Klein, R., Bessler, A., Malloy, P., LaPadula, M. (1993) Adult outcome of hyperactive boys: educational achievement, occupational rank, and psychiatric status. Archives of General Psychiatry, 50, 565-576.

McCracken, J.T. (1991). A two-part model of stimulant action on attention deficit hyperactivity disorder in children. Journal of Neuropsychiatry, 3 (2), 201-208.

McGee, R. & Feehan, M. (1991). Are girls with problems of attention underrecognized? Journal of Psychopathology and Behavioral Assessments, 13, 187-198).

McIntyre, C., Blackwell, S., & Denton, C. (1978). Effect of noise distractibility on the spans of apprehension of hyperactive boys. Journal of Abnormal Child Psychology, 6 (4), 483-492.

Mirsky, A. (1996). Disorders of attention: A neuropsychological perspective. In G.R. Lyon & N.A. Krasnegor (Eds.), Attention, Memory, and Executive Function (pp. 71-95). Baltimore: Paul H. Brooks.

National Institute of Mental Health (NIMH) (1993). Diagnostic Interview Schedule for Children. Ontario, Canada: Author.

Nigg, J. & Goldsmith, H. (1998). Developmental psychopathology, personality, and temperament: Reflections on recent behavior genetic research. Human Biology, 70, 387-412.

Nigg, J., Hinshaw, S., Carte, E., & Treuting, J. (1998). Neuropsychological correlates of antisocial behavior and comorbid disruptive behavior disorders in children with ADHD. Journal of Abnormal Psychology, 107, 468-480.

Nigg, J., Hinshaw, S., & Halperin J. (1996). The continuous performance task in boys with ADHD: Methylphenidate dose response and relations to observed behaviors. Journal of Clinical Child Psychology, 25, 330-340.

Nigg, J., Swanson, J., & Hinshaw, S. (1997). Covert visual attention in boys with attention deficit hyperactivity disorder: lateral effects, methylphenidate response, and results for parents. Neuropsychologia, 35 (2), 165-176.

Novak, G., Solanto, M., Abikoff, H. (1995). Spatial orienting and focused attention in attention deficit hyperactivity disorder. Psychophysiology, 32, 546-559.

Offord, D., Boyle, M., Szatmari, P., Rae-Grant, N., Links, P., & Cadman, D. (1987). Ontario Child Health Study, II: Six month prevalence of disorder and rates of service utilization. Archives of General Psychiatry, 44, 832-836.

Parasuraman, R., Greenwood, P., & Alexander, G. (1995). Selective impairment of spatial attention during visual search in Alzheimer's disease. Neuroreport, 6, 1861-1864.

Parsuraman, R., Warm, J. & See, J. (1998). Brain systems of vigilance. In R. Parsuraman (Ed.), The Attentive Brain (pp.221-256). Cambridge: MIT press.

Pearson, D., Yaffee, L., Loveland, K., & Norton, M (1995). Covert visual attention in children with attention deficit hyperactivity disorder: Evidence for developmental immaturity? Development and Psychopathology, 7, 351-367.

Pelham, W., Walker, J., Sturges, J., & Hoza, J. (1989). Comparative effects of methylphenidate on ADD girls and ADD boys. Journal of the American Academy of Child and Adolescent Psychiatry, 28, 773-776.

Pelham, W. (1993). Pharmacotherapy for children with attention-deficit hyperactivity disorder. School Psychology Review, 22 (2), 199-27.

Pennington, B. & Ozonoff, S. (1996). Executive functions and developmental psychopathology. Journal of Child Psychology and Psychiatry, 37 (1), 51-87.

Pliszka, S., McCracken, J., & Maas, J. (1996). Catecholamines in attention deficit hyperactivity disorder: Current perspectives. Journal of the American Academy of Child and Adolescent Psychiatry, 35, 264-272.

Posner, M. (1987). How do the parietal lobes direct covert attention? Neuropsychologia, 25, 135-145.

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

Posner, M., Rafal, R., Choate, L., & Vaughan, J. (1985). Inhibition of return: Neural basis and function. Cognitive neuropsychology, 2 (3), 211-228.

Posner, M. & Raichle, M. (1994). Networks of attention. In Images of Mind (pp. 152-179) New York: Scientific American Library.

Rafal, R. & Henik, A. (1994). Neurology of inhibition: Integrating controlled and automatic processes. In Dagenbach & Carr (Eds.), Inhibitory processes in attention, memory, and language (pp. 1-51). San Diego: Academic Press.

Reader, M., Harris, E., Schuerholz, L., Denckla, M. (1994). Attention deficit hyperactivity disorder and executive dysfunction. Developmental Neuropsychology, 10 (4), 493-512.

Robertson, L. (1992). Perceptual organization and attentional search in cognitive deficits. In Margolin, I. (Ed.), Cognitive Neuropsychology in Clinical Practice (pp.70-95). New York: Oxford University Press.

Rothbart, M. , Posner, M., & Rosicky, J. (1994). Orienting in normal and pathological development. Development and Psychopathology, 6, 635-652.

Rothlind, J., Posner, M., & Schaughency, E. (1991). Lateralized control of eye movements in attention deficit hyperactivity disorder. Journal of Cognitive Neuroscience, 3 (4), 377-381.

Sandberg, S. (1996). Hyperkinetic or attention deficit disorder. British Journal of Psychiatry, 169, 10-17.

Sattler, J. (1992). Assessment of Children (3rd Ed.). San Diego: Jerome M. Sattler, Publisher, Inc.

Scheibel, R. & Levin, S. (1997). Frontal lobe dysfunction following closed head injury in children. In N.A. Krasnegor, G.R. Lyon, & P.S. Goldman-Rakic (Eds.), Development of the prefrontal cortex (pp.241-260), Baltimore: Paul H. Brookes Publishing Co.

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

Sergeant, L. (1989). In search of processing deficits of attention in ADD-H children. In L.M. Bloomingdale & J.M. Swanson (Eds.), Attention Deficits Disorder, pp. (255-272). Amsterdam: Elsevier.

Sergeant, J. & Van der Meere, J. (1990). Convergence of approaches in localizing the hyperactivity deficit.

Servan-Schreiber, D., & Cohen, J. (1992). A neural network model of catecholamine modulation of behavior. Psychiatric Annals, 22 (3), 125-130.

Shaffer, D., Schwab-Stone, M., Fisher, P., Cohen, P., Piacentini, J., & Davies, M. (1993). The Diagnostic Interview Schedule for Children--Revised Version (DISC-R): I. Preparation, field testing, interrater reliability, and acceptability. Journal of the American Academy of Child and Adolescent Psychiatry, 32, 643-650.

Steinkamp, M. (1980). Relationships between environmental distractions and task performance of hyperactive and normal children. Journal of Learning Disabilities, 13 (4), 40-45.

Stevenson, J. (1992). Evidence of a genetic etiology in hyperactivity in children. Behavior Genetics, 22, 337-344.

Still, G.F. (1902). Some abnormal psychical conditions in children. Lancet, 1, 1163-1168.

Swanson, J., Nolan, W., Pelham, W. (1982). The SNAP-IV rating scale. Resources in Education.

Swanson, J., Posner, M., Potkin, S., Bonforte, S., Youpa, D., & Fiore, C. (1991). Activating tasks for the study of visual-spatial attention in ADHD children: A cognitive anatomic approach. Journal of Child Neurology, 6, S119-S127.

Tomprowski, P., Tinsley, V., & Hager, L. (1994). Visuospatial attentional shifts and choice responses of adults and ADHD and non-ADHD children. Perceptual and Motor Skills, 79, 1479-1490.

Tucker, D. & Williamson, P. (1984). Asymmetric neural control systems in human self-regulation. Psychological Review, 9 (2), 185-215.

Van der Meere, J. (1996). The role of attention. In S. Sandberg (Ed.) Hyperactive Disorders of Childhood (pp.111-148). Cambridge: Cambridge University Press.

Van der Meere, J., Wekking, E., & Sergeant, J. (1991). Sustained attention and pervasive hyperactivity. Journal of Child Psychology and Psychiatry, 32, 275-284.

Voeller, K. & Heilman, K. (1988). Attention deficit disorder in children: A neglect syndrome? Neurology, 38, 806-808.

Waldman, I., Rowe, D., Abramowitz, A., Kozel, S., Mohr, J., Sherman, S., Cleveland, H., Sanders, M., & Stever, C. (1998). Association and linkage of the dopamine transported gene (DAT1) and attention deficit hyperactivity disorder in children. American Journal of Human Genetics, 63 (6), 1767-1776.

Wechsler, D. (1992). Wechsler Individual Achievement Test Screener. San Antonio: Harcourt Brace and Company.

Wechsler, D. (1991). Wechsler Intelligence Scale for Children--3rd. Edition Manual. San Antonio: Harcourt Brace and Company.

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

Weyandt, L. & Willis, W. (1994). Executive function in school-aged children: Potential efficacy of tasks in discriminating clinical groups. Developmental Neuropsychology, 10 (1), 27-38.

Zametkin, A.J. (1989). The neurobiology of attention deficit hyperactivity disorder: A synopsis. Psychiatric Annals, 19 (11), 584-586.

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