The Dissertation Committee for Brianne Janeé Butcher Certifies that this is the approved version of the following dissertation:

Attention in Children and Adolescents with Nonverbal Learning Disabilities

Committee:

Timothy Keith, Co-Supervisor

Nancy Nussbaum, or Co-Supervisor

Cindy Carlson

Barbara Dodd

Margaret Semrud-Clikeman

Attention in Children and Adolescents with Nonverbal Learning Disabilities

by

Brianne Janeé Butcher, B.A., M.A.

Dissertation

Presented to the Faculty of the Graduate School of

The University of Texas at Austin

in Partial Fulfillment

of the Requirements

for the Degree of

Doctor of Philosophy

The University of Texas at Austin

August, 2009

Dedication

To my mom and dad for their love and support, to my sister for always making me laugh, and to my frontal lobes for the vigilance to complete a dissertation.

Acknowledgements

I sincerely thank my committee members, Drs. Timothy Keith, Nancy Nussbaum, Margaret Semrud-Clikeman, Cindy Carlson, and Barbara Dodd, for their insight and guidance throughout my graduate education. I also extend my appreciation to Dr. Nussbaum and Austin Neuropsychology, PLLS for graciously allowing me to use years of data, without which this research project could not have been possible.

Attention in Children and Adolescents with Nonverbal Learning Disabilities

Publication No._____________

Brianne Janeé Butcher, Ph.D.
The University of Texas at Austin, 2009

Supervisors: Timothy Keith and Nancy Nussbaum

Nonverbal Learning Disability (NVLD) is a syndrome characterized by impaired social perception, visual-spatial skills, fine motor coordination, and mathematics abilities. Researchers have found that children with NVLD often have significant symptoms of inattention, and there is evidence that the majority of children with NVLD also meet clinical criteria for Attention Deficit/Hyperactivity Disorder, Predominantly Inattentive Subtype (ADHD:PI) (Brown, 2000; Gross-Tsur & Shalev, 1995; Voeller, 1996). Although significant overlap is observed between NVLD and behavioral symptoms of ADHD, little research has focused on the specific attention problems of children with NVLD.

Given the high incidence of co-morbid attention problems with NVLD (Brown, 2000), many researchers have proposed that overlapping neural regions are responsible for the similarity in attention impairments observed in both NVLD and ADHD:PI (Denckla, 2000; Stefanatos, 2001). Other researchers suggest that there are distinct neurological impairments in children with NVLD and both subtypes of ADHD that result in attention problems. Specifically, Rourke (1995) suggested a developmental sequence that results in generally intact auditory attention with impaired attention for visual stimuli in children with NVLD.

This study sought to reconcile the discrepancy between conceptualizations of attention problems in children with NVLD. It was hypothesized that children with NVLD would exhibit distinct profiles of strengths and weaknesses on neuropsychological measures of attention compared to children with ADHD, Predominantly Inattentive Subtype (ADHD:PI) and ADHD, Combined Subtype (ADHD:C). Specifically, it was expected that the three diagnostic groups would differ on the neuropsychological measures depending on the attention modality (auditory vs. visual).

Extant neuropsychological data from 88 children between the ages of 9 and 15 years of age with diagnoses of NVLD, ADHD:PI, and ADHD:C were analyzed. Neuropsychological measures of processing speed, working memory, vigilance, and inhibition were examined to compare specific domains of attention functioning in the three groups. Evidence from the current study supported the model in which NVLD and the two ADHD subtypes represent a continuum of dysfunction dependant on overlapping neural regions. Moreover, specific attention strengths and weaknesses in children with NVLD compared to children with ADHD:PI, ADHD:C, and normative data were identified in order to inform clinical diagnosis and intervention.

Table of Contents

List of Tables

List of Figures

CHAPTER I: INTRODUCTION

Nonverbal Learning Disability (NVLD) is a developmental disorder that affects a child's academic achievement and ability to socialize effectively with others. The syndrome is characterized by impaired social perception, visual-spatial skills, fine motor coordination, and mathematics abilities. Children with NVLD are reported to comprise approximately 5-10% of the learning disabled population, although this may be an underestimation since NVLD continues to be refined in terms of clinical features and diagnostic criteria (Pennington, 1991; Rourke, 1989). While the prevalence of NVLD is relatively low compared to other learning disabilities such as dyslexia, the impact of the disorder on the child's daily social, emotional, and academic functioning can be immense (Fuerst, Fisk, & Rourke, 1990; Rourke & Fisk, 1981; Rourke, 1989).

In addition to the primary impairments associated with NVLD, including deficits in tactile and visual perception, psychomotor coordination, and adaptability, researchers have found that children with NVLD often have significant symptoms of inattention (Brown, 2000). There is evidence that the majority of children with NVLD also meet clinical criteria for Attention Deficit/Hyperactivity Disorder, Predominantly Inattentive Subtype (ADHD:PI) (Brown, 2000; Gross-Tsur, Shalev, & Amir, 1995; Voeller, 1986). Attention Deficit Hyperactivity Disorder (ADHD) is a childhood onset disorder defined by persistent and developmentally inappropriate symptoms of inattention or impulsivity/hyperactivity (or both) which cause significant impairment in academic, social and occupational functioning (American Psychiatric Association [APA], 2000). ADHD is often conceptualized as impairment in executive functions including working memory, response inhibition, vigilance, planning, organizing, set-maintenance, and cognitive flexibility (Barkley, 1997; Pennington & Ozonoff, 1996; Willicutt, Doyle, Nigg, Faraone, & Pennington 2005). Although significant overlap is observed between NVLD and *behavioral* symptoms of ADHD, little research has focused on the specific attention problems of children with NVLD.

Given the high incidence of attention problems with NVLD, many researchers have proposed that overlapping neural regions are responsible for the attention impairments observed in both NVLD and ADHD:PI (Denckla, 2000; Stefanatos & Wasserstein, 2001). Rourke (1995) conceptualized NVLD as developmental right hemisphere syndrome that manifests to the extent that white matter in the brain is underdeveloped, damaged, or dysfunctional. Similarly, research indicates that ADHD results from disturbances in right hemisphere neural regions and white matter, specifically the frontal and parietal areas of the right hemisphere and their connections to subcortical structures including the striatum, limbic system and diencephalic nuclei (Stefanatos & Wasserstein, 2001).

Other researchers suggest that there are distinct neurological impairments in children with NVLD and ADHD that result in attention problems. Landau, Auerback, Gross-Tsur, and Shalev (2003) found differences in patterns of vigilance and processing speed in children with NVLD and ADHD. Additionally, Rourke (1995) speculated that inattention to visual and tactile stimuli in children with NVLD stems from primary deficits in visual and tactile perception rather than a primary failure in the attention system.

Although the term comorbidity abounds in discussing NVLD and ADHD, research has yet to present an accurate description of the relation between these two disorders. Are NVLD and ADHD two unrelated disorders with distinct etiologies that co-occur in children? Are the disorder related and originate from overlapping neurological substrates? Or do the behavioral symptoms of NVLD mimic those of ADHD while the underlying deficits are quite different?

This study sought to reconcile the discrepancy between conceptualizations of attention problems in children with NVLD. It was hypothesized that children with NVLD would exhibit distinct profiles on neuropsychological measures of attention from children with ADHD, Predominantly Inattentive Subtype (ADHD:PI) and ADHD, Combined Subtype (ADHD:C). Neuropsychological measures of processing speed, working memory, vigilance, and inhibition were employed to compare specific domains of attention functioning in the three groups. It was expected that the three diagnostic groups would exhibit different patterns of functioning across the domains. The diagnostic groups were also expected to differ on neuropsychological measures depending on the attention modality (auditory vs. visual). The children in the NVLD group were expected to be significantly impaired compared to the two ADHD groups on visual attention measures. The NVLD group was to perform significantly better than the ADHD groups on measures of auditory attention.

By examining the neuropsychological constructs of attention, this study clarified the relations between attention problems in children with NVLD, ADHD:PI, and ADHD:C. Knowledge regarding the specific attention deficits of children with NVLD can contribute to the formulation of valid diagnostic criteria and assist in the implementation of precise and effective intervention strategies.

The following literature review will discuss prominent theories and research on NVLD and ADHD. The disorders will be presented within a neuropsychological framework, integrating information from psychology and neuroscience in order to present a more comprehensive understanding of the cognitive, behavioral, and neurological conceptualizations of the disorders. A summary of the current understanding of each disorder will be followed by research regarding the theories of underlying neural dysfunction. Finally, the review will focus on the issue of attention problems in children with NVLD and the relationship between NVLD and ADHD.

CHAPTER II: LITERATURE REVIEW

Learning Disabilities

Learning Disability (LD) is a term that applies to a group of disorders that manifest as significant difficulties in acquiring and using age-appropriate skills in listening, speaking, reading, writing, reasoning or mathematics despite normal capacity to learn and appropriate opportunity (Hammill, Leigh, McNutt, & Larsen 1981; Tannock & Brown, 2000). These difficulties cannot be the direct result of mental retardation, sensory impairment, emotional disorders, or environmental influences (Hammill et al., 1981). According to the definition by the National Joint Committee for Learning Disabilities (NJCLD), Learning Disabilities are "intrinsic to the individual and presumed to be due to central nervous dysfunction" (Hammill, et al., 1981, p. 336).

Early investigators treated Learning Disability as a unitary entity, and LD groups were compared to control groups in order to establish patterns of functioning. This method assumed that the LD group was a homogenous group and afflicted individuals would share similar characteristics (Rourke, 1999). It soon became clear the group was heterogeneous and different constellations of abilities emerged (Fuerst & Rourke, 1993; Porter & Rourke, 1985). Through a neuropsychological approach to understanding learning disabilities in children, researchers have identified a number of LD subtypes based on patterns of neuropsychological, academic, and socioemotional assets and deficits. Subtyping helps to "establish reliable and valid characterizations of individuals such that with respect to the variables under consideration, they have very much more in common with others within their subtype than they have with members of other subtypes" (Rourke, 1999). Parceling the heterogeneous group of LD into more homogeneous LD subtypes has proven useful in the understanding of LD subtype etiology and neurological underpinnings, generalizability and predictive validity of study results, and development of effective interventions. This study will focus on understanding attention in one subtype of LD, specifically the subtype of Nonverbal Learning Disabilities (NVLD).

Nonverbal Learning Disabilities

DEFINITION AND SCOPE

Nonverbal Learning Disabilities were first described by Johnson and Myklebust (1971). Their account of NVLD focused primarily on "social imperception," or impairment in the ability to apply meaning to nonverbal aspects of daily living and understand one's social environment (Semrud-Clikeman & Hynd, 1990). Although the NVLD subtype continues to be refined, it is currently characterized by three broad areas of dysfunction including motoric skills, visual/spatial organizational skills, and social abilities (Thompson, 1997).

NVLD is estimated to occur in 5 to 10% of students with learning disabilities and in approximately one percent of the general population (Pennington, 1991; Rourke, 1989). Because NVLD is not as prevalent as verbal learning disabilities, such as dyslexia, less research has been devoted to this population (Pennington, 1991). Although NVLD is less common than verbal learning disabilities, the pervasive and persistent nature of the difficulties associated with NVLD may effect more spheres of daily living (Myklebust; 1975). It is thought that the impact of the pattern of deficits in children with NVLD contribute to extensive, long-term negative effects on interpersonal relationships, academic achievement, and emotional adjustment (Fuerst et al., 1990; Rourke & Fisk, 1981; Rourke, 1989).

NVLD PHENOTYPE

Byron Rourke (1989) presents a conceptualization of the Nonverbal Learning Disability phenotype in terms of brain-behavior relationships within a developmental context. The model delineates a causative relation between a pattern of primary, secondary, tertiary, and linguistic assets and deficits. These patterns are described in the neuropsychological, academic and socio-emotional/adaptational domains of functioning. Appendix A depicts a detailed model of Rourke's conceptualization of the developmental pattern of assets and deficits in NVLD.

The primary deficits encompass dimensions of tactile perception, psychomotor coordination, visual-spatial perception, and adaptability. Rourke (1995) predicts bilateral tactile perception and psychomotor coordination deficits that are more pronounced on the left side of the body. The few studies investigating this hypothesis generally support the deficit in psychomotor and tactile perception abilities but results are mixed regarding whether the deficits are bilateral or predominantly left side deficits (Harnadek & Rourke, 1994; Rourke & Strange, 1978). Several studies corroborate the presence of visual-spatial deficits in children with NVLD compared to children with verbal learning disabilities, children with ADHD, and children without disabilities. Children with NVLD were significantly impaired on measures of visual-spatial organization (Woods, Weinborn, Ball, Tiller-Nevin, & Pickett, 2000). They also demonstrated significant weaknesses on spatial reasoning tasks and visual-motor integration tasks (Wilkinson, Schafer, & Semrud-Clikeman, 2002; Worling, Humphries, & Tannock, 1999).

The primary deficits lead to secondary deficits including problems with tactile and visual attention and limited exploratory behavior. Rourke posited that since children with NVLD have primary deficits in tactile and visual perception, it would take more effort and resources to deploy attention to these modalities with fewer payoffs in regards to understanding their environment. These primary and secondary deficits cause tertiary deficits in tactile memory and visual memory. Because children with NVLD have difficulty perceiving and attending to tactile and visual stimuli, they fail to adequately encode this information resulting in poor tactile and visual memory. Empirical evidence supports Rourke's hypothesis that visual memory is more impaired in children with NVLD than in children without disabilities and within the NVLD population, visual memory is significantly more impaired than verbal memory (Liddell, 2005; Cornoldi, Rigoni, Tressoldi, & Vio, 1999). Rourke also suggested that the primary and secondary deficits in adaptability and dealing with novel situations results in deprived exploratory behavior resulting in difficulties with concept-formation, problem-solving, and hypothesis-testing skills.

Children with NVLD also show a pattern of relative strengths including auditory perception, auditory attention, and verbal memory. They often have intact simple motor skills, verbal associations, and verbal output. This pattern of neurological assets and deficits constitutes the nonverbal learning disorder phenotype that manifests in a specific pattern of academic and social functioning.

Academically these children show relative strengths in single-word reading, spelling, and rote verbal abilities. They often have significant difficulties in mathematics, science, and verbal academic skills that have a strong pragmatic component such as reading comprehension and writing (Gross-Tsur et al., 1995; Pennington, 1991; Rourke, 1989; Rourke, Ahmad, Collins, Hayman-Abello, & Warriner, 2002; Semrud-Clikeman & Hynd, 1990). While children with verbal learning disabilities can also show weaknesses in mathematics, the underlying problems that children with NVLD exhibit are distinct. Children with NVLD often can adequately memorize math facts, perform simple calculations and answer word problems. They have more difficulty performing math problems involving visual-spatial skills such as those involving graphs, charts, measurement, fractions and geometry (Johnson, 1987; Roman, 1998).

In addition to academic difficulties, Children with NVLD struggle considerably with social competence as a function of extreme difficulty with social perception, judgment, and interaction skills. Social competence consists of the "child's capacity to integrate behavioral, cognitive, and affective skills to adapt flexibly to diverse social contexts and demands" (Bierman & Welsh, 2000, p. 536). Specific examples of social problems often noted in children with NVLD include: misunderstanding or misuse of nonverbal cues, difficulty adapting to novel social situations, misinterpreting humor or other pragmatic aspects of speech, and verbosity. Rather than attributing a causal relation to LD and social functioning, Rourke hypothesized that both the learning and social deficits act as dependent variables and stem from neurological dysfunction in visual-spatial processing and difficulty integrating novel information (Pelletier, Ahmad, & Rourke 2001; Rourke, 1989). Gerrard-Morris (2006) found that visual-spatial skills did not predict social perception skills and hypothesized that the two skills sets may originate in proximal neuroanatomical areas of the brain but not have a causal relation. Nevertheless, social competence in the NVLD population has been shown to suffer as a result of poor social perception and adaptability. Children with NVLD were significantly less accurate at interpreting adult facial expressions and gestures than children with a verbal learning disability or no learning disability (Petti, Voelker, Shore, and Hayman-Abello, 2003). It is thought that dysfunction in intermodal integration, especially within the right hemisphere, obviates generation of new descriptive systems. Deficits in generating new descriptive systems has extremely deleterious effects on social functioning when right-hemisphere functions are arrested early in development (Semrud-Clikeman & Hynd, 1990). At this prelinguistic stage, infants rely on nonverbal cues in social interactions including facial expressions and voice tone. Failure to integrate and respond to emotional cues because of right-hemisphere processing deficits may disrupt the mother-infant bond, exploratory behavior, and subsequent social-emotional functioning of the child (Semrud-Clikeman & Hynd, 1990).

Furthermore, these social perception deficits compound as the child is involved in more complex interactions. During social interactions, children must perceive novel information and integrate material from a variety of sources including verbal and nonverbal stimuli (Frith & Frith, 2004). While people generally think of communication in terms of verbal communication, nonverbal communication actually plays a very significant role in social competence. Successful social interactions largely depend upon the process of monitoring others' reactions and emotions through adept perception and evaluation of nonverbal social cues including facial expressions, voice intonation, and body language (Dimitrovsky, Spector, Levy-Shiff, & Vakil, 1998; Frith & Frith, 2004). As Charles Darwin eloquently stated, “The movements of expression give vividness and energy to our spoken words. They reveal the thoughts and intentions of others more truly than do words, which may be falsified.”(Darwin, 1872) Current studies support the importance of understanding nonverbal cues in communication. Research suggests that these nonverbal cues are a more valid indicator of emotional states than verbal cues, and the ability to encode and evaluate these cues is a vital component of social competence (Nowicki & Duke, 1994; Rothman & Nowicki, 2004). Additionally, approximately 65% of the communication in an average conversation is nonverbal (Rothenberg, 1998).

Children with NVLD may have had difficulty acquiring basic social knowledge in infancy because of perceptual difficulties and delayed attachment, and continue to have difficulty integrating information and analyzing novel material in childhood. They revert to overlearned descriptive systems and apply strategies which may not be socially appropriate. This accumulation of deficits clarifies the negative developmental trajectory of NVLD as these children manifest increasingly severe social problems with age (Rourke et al., 2002).

ASSESSMENT AND DIAGNOSTIC CRITERIA OF NVLD

Currently there are no formal diagnostic criteria or a consensus in the field for the diagnosis of NVLD. Generally, it is suggested that children suspected of having NVLD undergo a complete neuropsychological assessment, and clinical judgment is used to diagnose the disorder. Many investigations on NVLD exclusively used the criteria of a PIQ that is significantly less than VIQ on cognitive tests. In a study validating classification rules of NVLD, Pelletier and colleagues (2001) found that using the PIQ<VIQ criteria alone in their samples would likely miss 72.7% of the "definite" and "probable" NVLD population. Rourke and colleagues (2002) outlined the following diagnostic criteria for research samples:

1. Bilateral deficits in tactile perception
2. Bilateral deficits in psychomotor coordination
3. Significant impairments in visual-spatial-organizational abilities
4. Significant difficulty in dealing with novel or complex situations with a tendency to rely on rote, over-learned responses.
5. Impairments in nonverbal problem-solving, concept-formation, and hypothesis testing.
6. Distorted sense of time.
7. Well developed rote verbal abilities in context of impaired reading comprehension.
8. Verbosity and deficits in pragmatic aspects of language.
9. Deficits in mechanical arithmetic
10. Deficits in social perception, judgment, and interaction, often leading to social withdrawal.

While these criteria closely adhere to the phenotype of NVLD suggested by Rourke (1989), there is little empirical evidence using neuropsychological measures that validate the reliability and specificity of these criteria in diagnosing NVLD. Although Pelletier and colleagues (2001) provided some evidence of the specificity of these criteria in distinguishing between children with NVLD and a verbal learning disability in a clinically referred sample, more research is needed in order to clearly define the NVLD diagnosis.

WHITE MATTER MODEL OF NVLD

Rourke suggested the "white matter model" as a neurological explanation for the NVLD phenotype. The primary premise of this model is that the NVLD phenotype will manifest to the extent that white matter in the brain is underdeveloped, damaged, or dysfunctional (Rourke, 1995; Tsatsanis & Rourke, 1995). White matter is made up of long myelinated axons that are grouped into bundles that integrate different parts of the brain. The myelin plays an important role in facilitating neuronal impulse propagation which is necessary for the efficiency of the nervous system. These bundles of myelinated axons connect the right and left hemispheres, cortical regions, and subcortical structures with one another and the rest of the brain. There is a higher ratio of white matter to grey matter in the right hemisphere largely because it contains longer axons (Goldberg & Costa 1981; Ellis & Gunter, 1999). This finding suggests that the *inter alia*, or intermodal associative areas are larger in the right hemisphere, with more developed inter-regional integration. Left hemisphere systems are more encapsulated within the three major opercula and utilize short association fibers for communication. Damage to the white matter would therefore be more detrimental to the functioning of the right hemisphere (Rourke, 1995). Additionally, white matter damage would impede interhemispheric communication (Gunter, Ghaziuddin, & Ellis, 2002).

These differences in the composition and organization of the left and right hemispheres of the brain have significant implications for children with NVLD who are hypothesized to have white matter damage. Although most tasks require activation of more than one area of the brain and often initiate complex circuits, one hemisphere is usually dominant for a particular task (Teeter & Semrud-Clikeman, 1997). Research has implicated right-hemisphere dominance in processing novel information, visual-spatial tasks, and processing nonverbal social cues.

Because the right hemisphere is more dependent on intermodal integration, it appears to be better able to process simultaneous and complex information than the left hemisphere. Thus, it is adapted for tasks requiring novel responses or integration of complex material (Goldberg & Costa, 1981). Damage to white matter, and thus more significant damage to the right hemisphere, would therefore make it difficult for a child to integrate complex, novel material and they would revert to over-learned, rote responses.

The right hemisphere of the brain is also thought to be dominant in the visuoperceptive, visuospatial, and visuoconstructive processes (Heilman, Watson & Valenstein, 2003). Even in infancy, people show right hemisphere lateralization in visual-spatial tasks such as registering flashing lights and recognizing simple patterns (Teeter & Semrud-Clikeman, 1997). Impairment in visual-spatial perception usually assessed by perception of line orientation strongly depends on the parietal cortex with pronounced asymmetry in favor of right-hemisphere involvement (Farah, 2003). Additionally constructional apraxia, characterized by detailed yet disorganized constructions, is associated with right hemisphere lesions (Farah, 2003).

Finally, the right hemisphere of the brain is responsible for processing many of the nonverbal social cues necessary for social competence. These nonverbal cues include prosody, recognizing faces, and interpreting the emotional state of others from these cues. Facial recognition has been found to be lateralized to the right hemisphere in children as young as four years of age (Kolb & Fantie, 1989). Studies with stroke patients, split-brain patients, and patients undergoing intracarotid sodium amytal procedures consistently demonstrate right hemisphere dominance for facial affect recognition and comprehension of emotional prosody (Heilman, Blonder, Bowers, & Valenstien, 2003). The right hemisphere contributes to comprehending affect from nonverbal cues above and beyond that of basic visual-perceptual abilities or emotional knowledge (Hielman et al., 2003). It is suspected that the right hemisphere contains representations of species-typical affective facial and prosodic expressions (Heilman et al., 2003). Right hemisphere dysfunction could thus impair comprehension and discrimination of affective nonverbal cues.

In summary, the impairment of functions including deficits in processing novel information, visual-spatial skills, and interpreting nonverbal cues in children with NVLD has contributed to the formulation of the white matter/right hemisphere model of NVLD (Rourke, 1995).

EMPIRICAL SUPPORT OF THE WHITE MATTER MODEL

Although Rourke's description of the NVLD phenotype is widely accepted, many aspects of his neuropsychological model have yet to be confirmed. Rourke and colleagues (2002) provided tentative support for this hypothesis by comparing NVLD to phenotypically similar pediatric disorders including callosal agenesis and multiple sclerosis that have more extensive empirical support for white matter disease. Furthermore, White and Krengal (1997) outlined common features of multiple sclerosis, a demyelinating disorder that significantly damages white matter in the brain, and the NVLD phenotype. Individuals with multiple sclerosis show significant problems with motor coordination, visual-spatial processing, and cognitive flexibility. While social problems are often observed in multiple sclerosis, the authors highlighted that other features of the disorder including significant affective symptoms may also contribute to social problems above and beyond that seen in NVLD.

Structural and functional neuroimaging now allows researchers to obtain information about brain structures and activity on a variety of tasks across various psychological disorders (Semrud-Clikeman & Pliszka, 2005). Neuroimaging on populations with white matter or right hemisphere damage lends indirect support to the concept that there is a white matter or right-hemisphere deficit in children with NVLD but few studies have directly investigated the neurological underpinnings of NVLD. The findings from the few imaging studies exploring the NVLD phenotype are complicated by heterogeneous populations with brain damage or genetic disease that affect white matter. Several studies investigated the relation between cognitive functions and white matter hyperintensities (WMHI), patches of diffuse or hypodense white matter, on MRI scans. A negative relation was found between the extent of WMHI and performance on tests of executive function in elderly subjects with WMHI (Tupler, Coffey, & Logue, 1992). Other studies with children that had neurofibromatosis had mixed results regarding the effects of WMHI on cognitive functions. Several studies demonstrated no significant differences in cognitive functions based on WMHI (Dunn & Ruoos, 1989; Legius et al. 1995), while others demonstrated the number of brain locations with WMHI correlated significantly with decreased intelligence and visual-spatial abilities (Hofman, Harris, Bryan, & Denckla 1994). One of the few imaging studies using a developmental NVLD, a population without genetic disorder or brain injury, found that PET scans revealed a hypometabolism in the right parietal lobe and bilateral superior temporal areas (DeLuca et al., 1997). In summary, a great deal more research is need to directly investigate children with NVLD through the use of imaging technology for there to be conclusive statements regarding the neurological underpinnings of the disorder.

Attention Deficit/ Hyperactivity Disorder

DEFINITION AND SCOPE

Attention Deficit Hyperactivity Disorder (ADHD) is a childhood onset disorder defined by persistent and developmentally inappropriate symptoms of inattention or impulsivity/hyperactivity or both which cause significant impairment in academic, social and/or occupational functioning (APA, 2000). ADHD is one of the most common childhood disorders with prevalence rates generally estimated to be between 3 and 7% of the school-aged population (APA, 2000; Barkley, 1998), although some estimates range as high as 8% to 12% of children (Faroane, Sergeant, Gillberg, & Biederman., 2003). The disorder is thought to constitute 30 to 40% of all referrals to child guidance clinics. Boys are diagnosed significantly more than girls with ADHD by an average ratio of approximately 3:1 (Barkley, 2003; Brown, Madan-Swain, & Baldwin, 1991). While many clinical and epidemiological studies confirm this gender ratio (American Psychiatric Association [APA], 1994; Lahey, Applegate, & McBurnett, 1994), some researchers question the validity and sensitivity of the current DSM-IV-TR criteria for diagnosing females. The grounds for the controversy are that the criteria was developed based on primarily male samples and may not be generalizable to girls (Staller & Faraone, 2006). Additionally, there may be referral bias due to females generally displaying less disruptive behaviors than males (Gaub & Carlson, 1997).

EVOLVING CONCEPTUALIZATION AND DIAGNOSTIC CRITERIA

Attention Disorders have been referred to by various and sometimes stigmatizing labels throughout history including Morbid Defect of Moral Control, Post Encephalitic Behavior Disorder, Minimal Brain Dysfunction/Damage, and Hyperkinesis (McGough & McCracken, 2006; Pliska, 1991). Even since attention-related disorders have been included in the Diagnostic and Statistical Manual for Mental Disorders (DSM), the conceptualization and criteria have undergone significant changes (Milich, Balentine, & Lyman, 2001). In the DSM-II, the disorder that is now recognized as ADHD was termed, "the hyperkinetic reaction of childhood." The definitive symptoms were associated with motor excess and there were implications that the behavioral disturbance was a reaction to family environment (Carlson, Shin, & Booth, 1999; McGough & McCracken, 2006). In the DSM-III, the description of the disorder referred to as Attention Deficit Disorder (ADD) emphasized attention and was regarded as multidimensional. It could be divided into subtypes of Attention Deficit Disorder with or without Hyperactivity. The subtypes were revoked in the DSM-III-R and the uni-dimensional disorder termed Attention Deficit Hyperactivity Disorder (ADHD) was defined by 8 or more of the possible 14 symptoms of inattention, hyperactivity, or impulsivity (DSM-III-R; APA, 1987). Currently, the DSM-IV-TR refers to the disorder as Attention Deficit Hyperactivity Disorder (ADHD) but provides criteria for three subtypes differentiated by the presence of predominantly inattentive symptoms (ADHD:PI), predominantly hyperactive/impulsive symptoms (ADHD:HI) , or combined symptoms (ADHD:C) (APA; 2000).

Clinical evidence and factor analytic studies generally support the DSM-IV subtypes (Morgan et al., 1996). A growing body of research suggests that the ADHD:PI subtype significantly differs from the ADHD:C subtype and the subtypes manifest different patterns of behavioral profiles, comorbidities, impairments in executive functioning, underlying neurological problems, and treatment outcomes (Carlson et al., 1999; Carlson & Mann, 2000; Diamond, 2005; Milich et al. 2001). In addition, factor analysis and cluster analysis support a distinction between impairments in children exhibiting inattention with hyperactivity and inattention without hyperactivity (Milich et al., 2001).

There continues to be controversy concerning the validity of the ADHD:HI because this subtype is predominantly seen in early childhood. As such, the demands of attention are considerably less in early childhood, and some researchers believe that the ADHD:HI subtype is actually a transient phase that represents a precursor to the combined subtype (Lahey et al., 1998).

NEUROANATOMY

While family, twin, and adoption studies have consistently pointed towards a biological basis for ADHD, until recently the neuroanatomical underpinnings of the disorder have not been known (Biederman et al., 1992; Beiderman, 2005; Thapar, Hervas, & McGuffin, 1995; Giedd, Blumenthal, Molloy, & Castellanos., 2001). With advances in neuroimaging technology, an expanding research base in the fields of neuroimaging and neurobiology strongly implicate the involvement of particular neurological substrates in ADHD. Specifically, symptoms of ADHD appear to be related to a dissociation or impairment in a distributed right-side fronto-striatal network (Stefanatos & Wasserstein, 2001). Additionally, several studies have found underdevelopment of white matter regions in children with ADHD, especially in the corpus collosum (Filipek et al., 1997; Semrud-Clikeman et al., 1994). See Appendix B for a summary of the structural and functional magnetic resonance imaging (MRI) studies involving children and adults with ADHD reviewed in this section.

Structural brain imaging studies have found differences in particular anatomical regions associated with the frontostriatal network including the prefrontal cortex, caudate, globus pallidus and thalamus. In developmentally normal populations, the right prefrontal cortex is larger than the left prefrontal cortex. MRI studies have shown significant decrease in this normal asymmetry in children with ADHD (Castellanos et al., 1996; Hynd, Semrud-Clikeman, Lorys, Novey, & Eliopulos, 1990). Casey and colleagues (1997) found that lower performance on a response inhibition task significantly correlated with smaller right prefrontal cortex and the basal ganglia. Boys with ADHD were also found to have decreased right frontal white matter (Filipek et al., 1997). Structural imaging studies have suggested differences in the corpus collosum of individuals with ADHD. The corpus collosum is the largest interhemispheric commissure in the brain consisting of mostly myelinated fibers (white matter) and facilitates lateralization of functions in the brain (Giedd et al., 2001; Gazzaniga; 2000). The rostral body of the corpus collosum and splenium were found to be smaller in the ADHD groups (Hynd et al. 1991; Semrud-Clikeman et al., 1994).

Volumetric studies have also suggested that smaller cerebellum size, particularly the posterior inferior vermis, is related to ADHD (Valera, Faraone, Murray, & Seidman, 2007). Castellanos et al. (2000) reported that severity of ADHD symptomology in girls correlated significantly with smaller cerebellum volume. Similarly, Berquin, Giedd, & Jacobsen (1998) reported that regions of the cerebellum were significantly smaller in boys with ADHD than controls and hypothesized that cerebello-thalamo-prefrontal circuit dysfunction contributes to executive function and motor control deficits in ADHD. Giedd and colleagues (2001) noted that smaller cerebellum volumes were also reported in childhood-onset schizophrenia and in multiple-episode adult bipolar disorder patients, highlighting the non-specificity of many anatomic deviations.

Functional neuroimaging studies further implicate the right fronto-striatal network in ADHD symptoms, particularly for tasks involving inhibition. Individuals with ADHD were found to exhibit widespread activation, especially in occipital regions on tasks of inhibition, whereas controls primarily activate frontal and temporal regions (Schweitzer et al., 2000). This indicates that the ADHD subjects may use a compensatory strategy that is less efficient and possibly more visually-mediated than controls. Additionally, adolescents with ADHD demonstrate less activation in the right prefrontal cortex and left caudate during an inhibition and planned motor response tasks (Rubia et al., 1999). While this functional imaging research is promising in elucidating the neuroanatomical underpinnings of ADHD, there are few studies to date, and many of them have not controlled for factors including ADHD subtype or medication history (Semrud-Clikeman & Pliszka, 2005).

NEUROPSYCHOLOGICAL THEORIES OF ADHD

In 1890, William James matter-of-factly stated, "Everyone knows what attention is." He went on to explain, "It is the taking possession of the mind, in clear and vivid form, of one out of what seem several simultaneously possible objects or trains of thought. Focalization, concentration of consciousness are of its essence. It implies withdrawal from some things in order to deal effectively with others" (1890, p. 403). While his conceptualization of attention beautifully captured several important facets of attention, researchers have discovered that attention is actually an extremely complex and multidimensional construct. In order to explain attention and how it relates to children with ADHD, numerous theories informed by a variety of theoretical orientations have been proposed. This review will focus on theories of ADHD that stem from the neuropsychological orientation and look at ADHD in terms of brain-behavior relationships.

Posner's Model

Posner's (1994) model of normal attention delineates three attention networks including: (1) executive-control network, (2) alerting network, and (3) orienting/shifting network. The executive control network is responsible for detecting stimuli and bringing it into conscious awareness. In the brain, this network is hypothesized to include midline frontal areas and parts of the basal ganglia. The alerting network is necessary for vigilance and permits maintenance of alertness. This network is thought to involve the right superior frontal regions and right parietal regions of the brain. Finally, the orienting/shifting network functions to disengage, orient, and engage attention to new stimuli, and is thought to involve the parietal lobes, thalamus, and midbrain. Berger and Posner (2000) proposed examining deficits in ADHD from this network approach to attention and described how theories of ADHD can be conceptualized in terms of this these networks.

Barkley's Model

A leading theory for ADHD is Barkley's (1997) theory of disinhibition. Many researchers suggest that the symptoms of hyperactivity, impulsivity, and inattention in children with ADHD are best conceptualized as impairments in executive functions (Barkley, 1997; Barkley, 2003; Nigg, 2001; Willicutt et al., 2005). The term executive function refers to a broad set of higher order functions that regulate goal-directed behavior. These processes include working memory, response inhibition, vigilance, planning, organizing, set-maintenance, and cognitive flexibility (Barkley, 1997; Pennington & Ozonoff, 1996; Willicut et al., 2005). Barkley (1997) provided a unifying theory in which he conceptualized ADHD as a developmental disorder of self-control. He proposed that the impairments observed in children with ADHD were a result of disinhibition of higher order executive functions. In this manner ADHD is not a disorder marked by inattention to stimuli, but rather problems with inhibiting responses. Berger and Posner (2000) suggested that Barkley's ideas concerning disinhibition of executive functions can be tied to deficits in the executive network of his attention model. Thus, it would follow, that problems with inhibiting behavior would be mediated by the frontal lobes and basal ganglia.

Sergeant's Model

While Barkley's theory focuses on disinhibition, Sergeant et al. (1999) proposed a different model to explain attention problems, the cognitive-energetic model. This model suggested a state regulation or energetic impairment in ADHD, in which children with ADHD have deficits in cortical arousal, activation, and effort. It should be noted that effort in this model is defined as, "the necessary energy to meet the demands of a task." These primary deficits in energetic maintenance and allocation of resources lead to secondary problems with disinhibited behavior (Seargent, 1999; Berger & Posner, 2000). On sustained attention tasks, it was found that children with children with ADHD made more commission errors on the fast and slow rates of stimuli presentation than on the medium rate suggesting disinhibition in ADHD is mediated by difficulties adjusting their energetic state (Meerel, Stemerdink, & Gunning, 1995; Seargent, 2000). Other research has shown that individuals with ADHD have difficulty maintaining the steady-state alertness required for good performance on vigilance measures (Seidman, Biederman, Weber, Hatch, & Faraone, 1998). Seargent's cognitive-energetic theory of ADHD can also be explained within the framework of Posner's model of normal attention. Deficits in activation on executive function tasks may be explained by dysfunction in the executive-control networks mediated by frontal areas and basal ganglia (Berger & Posner, 2000; Sergeant, 2000). The deficits associated with arousal and effort may be more closely related to the alerting networks mediated by the right frontal and right parietal regions of the brain, although the executive-control network may also be involved (Berger & Posner, 2000).

Though there are many theories of ADHD, two prominent theories were presented here in the context of Posner's unifying model of normal attention. The two models of ADHD stress different cognitive aspects of the disorder and may be useful as heuristic frameworks for understanding the ADHD subtypes. Barkley (1997) noted that his theory of behavioral disinhibition is more applicable to ADHD:C and ADHD:HI subtypes than the ADHD:PI subtype, which he suggested may represent a different disorder entirely. Sergeant's (1999) cognitive-energetic model is more concerned with cognitive arousal in children with ADHD and appears to be more closely related to the manifestation of symptoms in the ADHD:PI subtype. These theories draw from multiple areas of research including psychology, biology, and neuroscience. Tools that are being used to validate these models range from behavioral and cognitive psychological measures to EEG and fMRI data. It is hoped that by integrating knowledge from multiple disciplines, that a more comprehensive and unifying conceptualization of attention can be achieved.

Attention in NVLD

While Learning Disabilities and ADHD are generally regarded as highly comorbid, there continues to be confusion and inconsistencies in the literature regarding the specific relationship between NVLD and ADHD. Conservative estimates suggest that 20% to 25% of children with ADHD also have a learning disability (Tannock & Brown 2000, Hinshaw & Zalecki, 2001; Semrud-Clikeman et al., 1992). The inattentive subtype of ADHD, rather than the hyperactive/impulsive or combined subtype, is more often associated with all types of learning disabilities (Barkley, 1997; Hynd et al., 1991). Alternately, it has been reported that approximately 20 to 50% of children with reading disabilities also have comorbid ADHD (Shaywitz & Shaywitz, 1988; Pliszka, Carlson, & Swanson, 1999; Semrud-Clikeman, 2005). ADHD has been identified in higher rates than the normal population in children with writing disabilities, mathematics disabilities, and social-emotional learning disabilities as well (Barkley, DuPaul, & McMurray, 1990; Denckla, 2000; Semrud-Clikeman, 2005). Although there have been no large scale prevalence studies, NVLD appears to co-occur with ADHD significantly more than other learning disabilities (Brown, 2000; Pliszka et al., 1999). Several studies with small clinical samples have reported almost 100% overlap between NVLD and behavioral symptoms of ADHD (Gross-Tsur et al., 1995; Voeller, 1986).

Though significant overlap is observed between NVLD and behavioral symptoms of ADHD, there continues to be controversy regarding the exact nature of this association. Children with NVLD generally exhibit symptoms of inattention unaccompanied by hyperactivity or impulsivity (Rourke, 1995; Voeller, 1986). These findings suggest that children with NVLD are behaviorally more similar to children with ADHD, Predominantly Inattentive subtype according to DSM-IV-TR criteria (APA, 2000). Many researchers have hypothesized that overlapping neural regions account for the similar behavioral symptoms of ADHD:PI and NVLD in children (Denckla, 2000; Hynd et al., 1991; Stefanatos & Wasserstein, 2001). It has been suggested that both disorders stem from neurological dysfunction in the right hemisphere and are associated with white matter dysfunction. As discussed in detail in the "Neuroanatomy of ADHD" section of this literature review, structural and functional MRI studies implicate dysfunction in the right-side fronto-striatal networks and white matter regions, including the corpus collosum in children with ADHD. Similarly, Rourke (1995) theorized that right-hemisphere and white matter dysfunction explain the pattern of assets and deficits observed in the NVLD syndrome. Denckla (2000) described a "cognitive overlap zone" of executive functions in NVLD and ADHD due to overlapping neural regions. The author suggested that the two disorders may not be distinct but rather may represent a continuum of impairment based on the degree of severity, with ADHD representing the less severe end of the continuum and NVLD representing the more severe end.

Rourke (1995) posited that while the behavioral symptoms of inattention may mimic those of ADHD, the underlying deficits are quite different. Unlike children with ADHD, children with NVLD demonstrate average to above average attention to verbal stimuli, especially to that presented in the auditory modality. His explanation of this phenomenon is derived from the developmental sequence of the NVLD syndrome. He suggested that there is a cause and effect sequence of attention development in which good sensory-perceptual abilities lead to good attention deployment and poor sensory-perceptual abilities lead to impaired attention deployment. The author explained that all biological organisms have a propensity to practice what they do well and avoid activities that are hard for them. Children with NVLD have a primary strength in auditory perception and weakness in visual-perception so attending to auditory stimuli requires fewer cognitive resources for them. They are expected to prefer hearing their environment rather than seeing it. Since auditory attention is the preferred modality early in development for these children, the networks for auditory attention are more fully developed and reinforced in the brain, while the visual attention networks are neglected.

Rourke (1995) suggested that this developmental sequence results in generally intact auditory attention with impaired attention for visual stimuli in children with NVLD.

Although Rourke clearly articulates his notion that there are differences in the etiology of the behavioral symptoms of inattention in children with NVLD and ADHD, less than a handful of studies have attempted to quantitatively measure these neuropsychological differences. Since attention is a multidimensional construct, it is generally accepted that a unitary measure is not sufficient for assessing attention difficulties. Neuropsychological measures are used to determine the relation between specific attention subcomponents and neurocognitive functioning. Although there continues to be much debate over what constitutes attention, current research suggests that the major components of attention can be measured using neuropsychological tests of working memory, processing speed, vigilance, and inhibition (Willicut et al., 2005).

Emerging research suggests that there are differences on neuropsychological measures of attention in children with ADHD:C, ADHD:PI, and normally developing children. Willicut and colleagues (2005) found that children with ADHD were significantly impaired on the majority of executive functioning measures compared to controls. Moderate to large impairments in working memory were found in children with ADHD. Children with ADHD with hyperactivity also have been found to demonstrate significant impairment on measures of response inhibition compared to children with ADHD:PI and controls (Barkley, 1997; Nigg, 2001; Pennington & Ozonoff, 1996). Processing speed impairments are also evident in children with ADHD, although results are mixed as to whether there are differences in processing speed abilities between ADHD subtypes. Barkley and colleagues (1990) found that children with ADHD without hyperactivity demonstrated slower processing speed compared to controls while children with ADHD with hyperactivity did not. Other studies have found that both subtypes are significantly impaired in processing speed (Chhabildas, 2001; Nigg, Blaskey, Huang-Pollock, Rappley, 2002).

While many studies have examined the differences in neuropsychological functioning on measures of attention in children with ADHD, there is a paucity of research in this area involving children with NVLD. Gross-Tsur and colleagues (1995) found that children with NVLD were significantly impaired on measures of vigilance compared to controls. Landau, Gross-Tsur, Auerbach, Van der Meere, and Shalev (1999) found that children with ADHD and NVLD were both significantly slower than controls on a computerized measure of sustained attention; however, the children with ADHD showed significantly higher variability than the children with NVLD. The authors proposed that while the distinctions between the disorders are not evident when comparing maladaptive behaviors, the neuropsychological bases for the behaviors are distinct.

In summary, two opposing hypotheses regarding attention problems in children with NVLD can be gleaned from current literature. The first hypothesis is that the attention problems in children with NVLD and ADHD have similar etiologies founded on dysfunction in white matter and right hemisphere neural regions. An alternate viewpoint is that the relationship between the behavioral symptoms of inattention in ADHD and NVLD more accurately represent a case of equifinality. Specifically, the two disorders have different underlying attention problems but manifest similar behavioral profiles.

Statement of the Problem

NVLD continues to be refined in terms of clinical features and diagnostic criteria. Although researchers have found that many children with NVLD also meet clinical criteria for ADHD (Gross-Tsur & Shalev, 1995; Voeller, 1996), little research has focused on the specific attention problems of children with NVLD.

Given the high incidence of comorbidity of NVLD and ADHD:PI (Brown, 2000), many researchers have proposed that overlapping neural regions are responsible for the attention impairments observed in both NVLD and ADHD:PI (Denckla, 2000; Stefanatos & Wasserstein, 2001). Rourke conceptualized NVLD as developmental syndrome that manifests to the extent that white matter in the brain is underdeveloped, damaged, or dysfunctional (Rourke, 1995a). Because of the higher ratio of white matter to grey matter in the right hemisphere of the brain, damage to the white matter would therefore be more detrimental to the functioning of the right hemisphere (Rourke, 1995), thus NVLD is often referred to as a developmental right hemisphere disorder. Similarly, research indicates that ADHD results from disturbances in right hemisphere neural regions, specifically the frontal and parietal areas of the right hemisphere and their connections to subcortical structures including the striatum, limbic system, and diencephalic nuclei (Stefanatos & Wasserstein, 2001).

Other researchers have suggested that there are distinct neurological impairments in children with NVLD and ADHD that result in attention problems. Landau and colleagues (2003) found differences in patterns of vigilance and processing speed in children with NVLD and ADHD. Additionally, Rourke (1995) speculated that inattention to visual and tactile stimuli in children with NVLD stems from primary deficits in visual and tactile perception rather than a primary failure in the attention system. Finding significant differences between visual and auditory attention in children with NVLD and both subtypes of ADHD would have important implications for clinical recommendations and intervention. For instance, it is often recommended that children with ADHD receive more visual stimuli to enhance attention during instruction (Dupaul & Power, 2000). Although this approach may be appropriate for children with ADHD:C and ADHD:PI, it may actually be detrimental to children with NVLD if it is found that they demonstrate significant deficits compared to the two ADHD groups on measures of visual attention.

This study attempted to reconcile the discrepancy between conceptualizations of attention problems in children with NVLD. By examining the neuropsychological constructs of attention including working memory, processing speed, vigilance, and inhibition, this study clarifies the relation between attention problems in children with NVLD, ADHD:PI, and ADHD:C. Furthermore, evaluating attention in children with NVLD compared to the two subtypes of ADHD separately permits more precise conclusions regarding the neurological underpinnings of NVLD. It was hypothesized that children with NVLD, ADHD:PI, and ADHD:C differ on neuropsychological measures of attention depending on the modality (visual vs. auditory), and the three disorders result in distinct neuropsychological profiles of attention-related strengths and weaknesses.

Proposed Study

RESEARCH QUESTION 1

Do children with NVLD, ADHD:PI, and ADHD:C significantly differ on neuropsychological measures of attention and do those differences depend on the attention modality (auditory vs. visual)?

Hypothesis 1

It is hypothesized that significant group differences are present. The NVLD group is expected to differ significantly from the ADHD:PI group, ADHD:C group, or both on neuropsychological measures of attention, including measures of working memory, processing speed, vigilance, and inhibition. Specifically, the children in the NVLD group will be significantly impaired compared to the two ADHD groups on visual attention measures. The NVLD group will perform significantly better than the ADHD groups on measures of auditory attention.

Rationale

Although children with NVLD exhibit many of the behavioral manifestations of ADHD, it is unclear whether the neuropsychological impairments are similar. Rourke (1995) posited that while children with NVLD and ADHD may both appear to have attention problems, the underlying deficits may be different. According to Rourke's Model of NVLD, inattention to visual stimuli is a secondary deficit in children with NVLD that stems from a primary deficit in visual perception (Rourke, 1995). Research has shown that children with NVLD also perform worse than children with ADHD on visual-spatial tasks (Wilkinson et al, 2002). Conversely, children with NVLD demonstrate neuropsychological assets in most areas of auditory perception, auditory attention, and auditory memory, especially for verbal material (Rourke, 2002). Given this pattern of assets and deficits, it is expected that children with NVLD will perform adequately on neuropsychological measures of auditory attention, while demonstrating significant impairments on measures of visual attention.

It is consistently evidenced that children with ADHD often have impairments in central auditory functioning (Brown, 2000). Furthermore, Diamond (2005) suggested that visual attention is often preserved in children with ADHD while auditory attention and working memory are impaired. These finding suggest that children with ADHD will perform adequately on neuropsychological measures of visual attention and will demonstrate significant impairments on measures of auditory attention.

Additionally, recent research has found differences between the neuropsychological profiles of the two ADHD subtypes (Lockwood et al., 2001). Comparing NVLD to the ADHD subtypes separately may provide a better framework for understanding the attention problems in NVLD as well as contribute to a better understanding of the neuropsychological profiles of the ADHD subtypes. Since behavioral manifestations of attention problems in children with NVLD are more similar to the ADHD:PI subtype (Voeller, 1994), group differences will likely be smaller between these groups than between the NVLD and ADHD:C groups.

RESEARCH QUESTION 2

Do children with NVLD, ADHD:PI, or ADHD:C demonstrate statistically and clinically significant impairment on neuropsychological measures of attention compared with published normative data?

Hypothesis 2

Impairments compared to normative data are expected to differ depending on the group and task. The NVLD group, ADHD:PI group, and ADHD:C group are expected to demonstrate impairments on measures of working memory and vigilance. The NVLD group may not demonstrate significant impairments on measures of verbal working memory, but they will likely be impaired compared to the normative data on the measure of visual working memory. The ADHD:PI and NVLD groups are expected to demonstrate impairments compared to normative data on measures of processing speed. The ADHD:C group will demonstrate impairments on measures of inhibition compared to normative data.

Rationale

Although research examining differences in executive functioning constructs (i.e., working memory, inhibition, and vigilance) between subtypes of ADHD is inconsistent, Willicutt et al.'s (2005) expansive meta-analysis showed that both subtypes of ADHD differed significantly from controls on the majority of executive functioning tasks. Marinussen (2005) found moderate to large impairments in working memory for children with ADHD. There is no current research that examines executive functions in NVLD, although Rourke (2002) hypothesized that visual working memory will be impaired compared to the normal population. Additionally, Gross-Tsur and colleagues (1995) found that vigilance was significantly impaired in children with NVLD.

Barkley, DuPaul, and McMurray (1990) found that children with ADHD without hyperactivity performed significantly worse than controls on measures of processing speed but that the ADHD:C children did not significantly differ; however, research is inconsistent as to whether the ADHD:C group also has difficulties with processing speed compared to controls (Chhabildas et al., 2001; Nigg et al., 2002). Gross-Tsur and colleagues (1995) found that children with NVLD were significantly slower than controls on measures of processing speed. Stefanatos and Wasserstein (2001) posited that slow processing speed in NVLD and ADHD are mediated by right hemisphere dysfunction.

Finally, it is generally thought that children with ADHD:C demonstrate impairments in response inhibition (Barkley, 1997; Nigg et al., 2001; Pennington & Ozonoff, 1996) but that children without hyperactivity/impulsivity do not have significant impairment in response inhibition (Diamond, 2005). There is no current data suggesting response inhibition problems in children with NVLD and since behavioral manifestations are similar to the ADHD:PI subtype (Voeller, 1994), it is hypothesized that they will not be impaired compared to normative data.

RESEARCH QUESTION 3

Does performance on neuropsychological measures of attention reliably discriminate between children with NVLD, ADHD:PI, and ADHD:C?

Hypothesis 3

Performance on the six neuropsychological measures of attention will predict two functions based on attention modality and inhibition. These functions will reliably separate the NVLD, ADHD:PI, and ADHD:C groups from each other.

Rationale

Though question 4 is more exploratory in nature, it is hoped that the neuropsychological measures of attention can reliably predict diagnostic groups and thus be used to inform the design of a neuropsychological battery of measures that has more diagnostic specificity. It is thought that a discriminant function analysis will yield two functions: one that can be thought of as Attention Modality and one that can be thought of as Inhibition. It is hypothesized that the Attention Modality function will discriminate between the NVLD and ADHD groups. The Inhibition Function will discriminate between the ADHD:PI and ADHD:C groups.

CHAPTER III: METHOD

Project Approval

This study complies with the ethical issues and standards of research delineated by the American Psychological Association (2002) and the Procedures Governing Research with Human Subjects at Austin Neuropsychology, PLLC. The Departmental Review Committee in the Department of Educational Psychology and the Institutional Review Board of The University of Texas at Austin approved this study (Appendix C).

Procedure

Comprehensive neuropsychological assessments were conducted for each participant between 2004 and 2007 at Austin Neuropsychology, PLLC (Austin Neurological Clinic prior to January 2007). A licensed neuropsychologist conducted a clinical interview with each participant and his/her parent(s) or guardian(s). During the interview, the neuropsychologist gathered developmental and background information pertaining to the presenting complaint, as well as a more detailed account of the child's current problems. This information, in conjunction with a structured developmental and family history form (Appendix D), was used by the neuropsychologist to develop an appropriate assessment battery specific to the child's needs. The neuropsychologist also conducted a clinical motor exam with the child. The assessments were conducted by professional psychometrists or graduate students in educational and clinical psychology programs trained in individual assessment. All graduate students had completed doctoral level courses in psycho-educational, psycho-emotional, and neuropsychological assessments. Additionally, all graduate students and psychometrists also completed a training in administration and scoring for each measure in the neuropsychological assessment battery followed by observations by senior level graduate students or psychometrists. Graduate students received approximately one hour of supervision from the licensed neuropsychologist for each case. In order to ensure reliability and accuracy of the neuropsychological assessments, files completed by psychometrists and graduate students were randomly chosen for review.

Although each neuropsychological assessment was individualized to best address the referral question, the test battery generally measured motor functioning, auditory functioning, memory, attention, cognitive functioning, academic achievement, and social-emotional functioning. The testing was conducted at Austin Neuropsychology in a single or multiple sessions lasting approximately 6 to 8 hours with breaks provided as needed. Parents were given parent and teacher checklists/rating scales including, but not limited to, the SNAP-IV and the Achenbach Child Behavior Checklist during the clinical interview or through the mail which were returned at the time of the testing appointment. Children and parents were made aware that release of records for research purposes was voluntary and that the participant could withdraw at any time without repercussions. Participant files for this study were chosen based on inclusionary and exclusionary criteria defined in the next section.

Participants

Participants were referred to Austin Neuropsychology for comprehensive neuropsychological evaluations. Prior to testing, written parental consent was obtained in order to use the child's testing records for research purposes (Appendix E). The participants were 88 right-handed children between the ages of 9 and 15. All participants had a measured Full Scale Intelligence Quotient (FSIQ) of 85 or higher on the Wechsler Intelligence Scale for Children-4th Edition (WISC-IV, The Psychological Corporation, 2003). All participants had reported hearing and vision within normal range. Only children with English as the primary language were included. Additionally, children were excluded from this study if they had a positive history for epilepsy, seizures, head injury, or progressive neurological disorder. Children with evidence of verbal learning disabilities, thought disorders, and bipolar disorder were also excluded. Participants who met criteria for one of the three diagnostic groups including NVLD, ADHD:PI, and ADHD:C were included in the sample. The groups were defined as follows.

NVLD GROUP

Since there are no current DSM-IV-TR criteria for diagnosis of NVLD, children were diagnosed based on criteria informed by classification rules for children between the ages of 9 to 15 suggested by Pelletier, Ahmad, and Rourke (2001), Rourke and colleagues' (2002) most current description of the NVLD phenotype, and clinical judgment. Children were diagnosed with NVLD based on the presence of criteria (A) and at least two of criteria (B) through (E). The criteria are outlined below.

A. Presence of a clinically significant impairment in social skills as measured by a significant elevation (≥93 percentile) on the Social Problems Scale from the Achenbach Child Behavior Checklist, or a significant elevation (scaled score of ≥ 6) on the Social Scale of the Gilliam's Asperger Disorder Scale, or presence of clinically significant impairments in nonverbal communication skills as measured by a significant elevation ($\geq$20 points per subscale) on at least two subscales of the Emory Dyssemia Index Revised (EDI-R).

B. Presence of a clinically significant impairment (at least one *SD* below the mean) in fine motor performance for the dominant or non-dominant hand measured by the Grooved Pegboard (GPT; Knights, & Moule, 1968).

C. Presence of a clinically significant impairment (at least one *SD* below the mean) in visual-motor integration abilities measured by the Test of Visual-Motor Integration (VMI; Beery & Buktenica, 1996) or a clinically significant impairment (scaled score of $\leq$7) on the copy portion of the Rey-Osterrieth, or a significant weakness on the WISC-IV Block Design subtest compared to the overall mean scaled score of the ten core subtests.

D. Impaired performance in math calculation or math reasoning as measured by the Kaufman Test of Educational Achievement, Second Edition (KTEA-II) or the Woodcock-Johnson Tests of Achievement, Third Edition (WJ-III) that is at least 16 points below the individual's Full Scale IQ on the WISC-IV.

E. Presence of a nonverbal or visual-perceptual deficit in the presence of adequate verbal functioning: PRI < VCI by at least 15 points measured by WISC-IV.

ADHD:PI GROUP

Participants in the Attention Deficit Hyperactivity Disorder, Predominantly Inattentive Group (ADHD:PI) met Diagnostic and Statistical Manual of Mental Disorder, 4^{th} Edition-Revised (DSM-IV-TR) criteria for a diagnosis of ADHD:PI (APA, 2000).

These criteria were assessed by the Swanson, Nolan, and Pelham Questionnaire Rating Scale (SNAP-IV) (Swanson, 1992) and data from the clinical parent interview. Specifically, a diagnosis of ADHD:PI was assigned based on an endorsement of at least 6 significant inattentive items and less than 4 of the hyperactivity/impulsivity items on the rating scale. On the measure, ratings of 2 or 3 are considered significant. Subjects in this group were excluded based on the outlined exclusionary criteria or if they meet diagnostic criteria for a Learning Disorder in Mathematics or a Pervasive Developmental Disorder, including Asperger's Disorder, Autism, or Pervasive Developmental Disorder-Not Otherwise Specified. Additionally, they were excluded from this group if they met the criteria for NVLD outlined in the NVLD Group criteria of this study.

ADHD-C GROUP

Participants in the Attention Deficit Hyperactivity Disorder, Combined Group (ADHD:C) met Diagnostic and Statistical Manual of Mental Disorder, 4th Edition-Revised (DSM-IV-TR) criteria for a diagnosis of ADHD:C (APA, 2000). These criteria were assessed by the Swanson, Nolan, and Pelham Questionnaire Rating Scale (Swanson, 1992) and data from the clinical parent interview. Specifically, a diagnosis of ADHD:C was assigned based on an endorsement of at least 6 significant inattentive items and at least 6 of the hyperactivity/impulsivity items on the rating scale. Subjects in this group were excluded based on the outlined exclusionary criteria or if they met diagnostic criteria for a Learning Disorder in Mathematics or a Pervasive Developmental Disorder, including Asperger's Disorder, Autism, or Pervasive Developmental Disorder-Not

Otherwise Specified. Additionally, they were excluded from this group if they met the criteria for NVLD outlined in the NVLD Group criteria of this study.

Instrumentation

INDEPENDENT MEASURES

The following measures were used as part of the comprehensive neuropsychological assessment that each child received in order to determine group membership.

Wechsler's Intelligence Scale for Children-Fourth edition (WISC-IV; Wechsler, 2003)

The WISC-IV is an individually administered intelligence test that provides a comprehensive measure of general cognitive functioning for children 6-16 years of age. The test yields a Full Scale Intelligence Quotient (FSIQ) which estimates general intelligence. Additionally it provides four index scores which are more consistent with CHC theory than previous versions of the Wechsler tests (Straus, Sherman, & Spreen, 2006). These indices are Verbal Comprehension (VCI), Perceptual Reasoning (PRI), Working Memory (WMI), and Processing Speed (PSI). FSIQ reliability estimates for the standardization sample were excellent with internal consistency coefficients ranging from 0.96-0.97 and test-retest reliability coefficients greater than or equal to 0.91 for each age group (Wechsler, 2003). Additionally, the four indices also showed impressive reliability estimates in the standardization sample. Internal consistency coefficients for VCI, PRI, and WMI were greater than 0.9 and the PSI coefficient was greater than 0.8. The test-retest reliability for all indices was greater than 0.8. The WISC-IV and WISC-III were found to be highly correlated ($r = .89$) with an average FSIQ decrease of 2.5 points

(Wechsler, 2003). The FSIQ was used to determine participants' cognitive functioning and eligibility for the study. The PRI and VCI were used in criterion (E) for NVLD group assignment because children with NVLD often exhibit at least 10 point higher VCI scores than PRI scores (Pelletier, 2001). Additionally the Block Design subtest is found to measure visual-motor coordination (Wechsler, 2003) and is often significantly lower (p<.05) than the mean subtest scaled score in children with NVLD (Pelletier et al., 2001). The Block Design subtest was utilized in this study for NVLD group criterion (C).

Kaufman Test of Education Achievement, Second Edition (KTEA-II; Kaufman & Kaufman, 2004)

The KTEA-II is an individually administered measure of academic achievement for ages 4.5 to 25 years. The test consists of 14 academically-related subtests that comprise 4 broad areas of achievement. For this study, the two subtests which make up the Math Composite, Math Concepts & Application and Math Computation, will be used to determine an academic weakness in math for the NVLD group criterion (D). Internal consistency and alternate form reliabilities for the Math composite, and subtests which make up that composite, were high with coefficients between .8 to .9. Confirmatory factor analysis indicated that the Math Concepts & Application and the Math Computation subtests both loaded significantly on the "Math Factor" with loadings of coefficients of .90 and .82, respectively. Adjusted correlation coefficients that were corrected for variability in the norm group indicate that the Math Concepts & Application and the Math Computation subtests were moderately correlated with the Math Calculation Cluster from the WJ-III with coefficients of .79 and.83, respectively (Kaufman & Kaufman, 2004).

Woodcock-Johnson III Test of Achievement (WJ-III ACH; Woodcock, McGrew, & Mather, 2001)

The Woodcock-Johnson III Test of Achievement is individually administered achievement battery designed to measure academic performance in reading, mathematics, written language, knowledge, and oral language. The national standardization sample consisted of 1,818 individuals between the ages of 24 months and 95+ years (Mather, Wendling, Woodcock, 2001). The standard battery consists of 12 subtests with all standard battery subtest internal reliabilities found to be in the range of .76 to .91 for the standardization sample. Content validity for the WJ-III is based on adherence to the Cattell-Horn-Carroll (CHC) theory of cognitive abilities. To ensure that all items in a test measured the same narrow ability or trait, stringent fit criteria based on the Rasch model were employed during item selection (Straus et al., 2006). Additionally, cluster content was aligned with core curricular areas and domains specified in federal legislation to increase applicability of the battery (Straus et al., 2006). The test is highly correlated with other achievement batteries for normal and clinical populations. The Total Achievement score correlated .79 with the KTEA Battery Composite in the standardization sample (Woodcock et al., 2001). In this study, the WJ-III Achievement test was used to evaluate math weakness for the NLVD group criterion (D).

Swanson, Nolan, and Pelham Questionnaire- Revised for DSM-IV Rating Scale (SNAP-IV; Swanson, 1992)

The SNAP-IV rating scale is a diagnostic checklist for ADHD and ODD (Swanson, 1992). The 26-item checklist consists of questions that directly assess DSM-IV diagnostic symptoms for both ADHD and ODD. The SNAP-IV is composed of three subscales: Inattention (9 items), Hyperactivity/Impulsivity (9 items), and ODD (8 items).

Each item is rated 0 (“not at all”), 1 (“just a little”), 2 (“quite a bit”), or 3 (“very much”). Ratings were completed by a parent and/or a teacher. Respondents were asked to circle the number that best describes the child. Ratings of 2 or 3 were considered significant for each item. Although there is no published reliability or validity data for this measure, it is used extensively in ADHD research and, since the ADHD and ODD items from the SNAP-IV closely resemble the diagnostic criteria in the DSM-IV, it is thought to accurately and reliably evaluate ADHD symptoms in children (Collett et al., 2003; MTA Cooperate Group, 1999). The SNAP-IV was used in group assignment for ADHD:PI and ADHD:C criterion (A).

Child Behavior Checklist (CBCL; Achenbach & Rescorla, 2001)

The CBCL is an empirically based rating scale of childhood behavior and emotional functioning. The CBCL is a 113 item rating scale completed by parents/guardians. The parents rate their child for how true each item is now or within the past 6 months using the following scale: 0 = not true; 1 = somewhat or sometimes true; 2 = very true or often true. The Parent report form yields *T*-scores and percentiles for eight clinical scales: Anxious/Depressed, Withdrawn/Depressed, Somatic Complaints, Social Problems, Thought Problems, Attention Problems, Rule Breaking Behavior, and Aggressive behavior. Internal consistency estimates ranged from .71 to .97 on the scales in the standardization sample. Test-retest reliability ranged from .88 to .90 (Achenbach & Rescorla, 2001). The Social Problems scale was used in criterion (A) for NVLD group assignment.

Gilliam's Asperger Disorder Scale -Social Interaction Subscale (GADS; Gilliam, 2001)

The GADS is a behavioral rating scale used in the field of psychology to identify persons with Asperger's Disorder. It has 32 items divided into four subscales, including Social Interaction, Restricted Patterns of Behavior, Cognitive Patterns, and Pragmatic Skills subscales. For this study, the Social Interaction Scale was utilized for the NVLD group criterion (A). This subscale describes social interactive behaviors, expression of communicative intent, and emotional behaviors that are characteristic of Asperger's Disorder. These social problems are consistent with those observed in children with NVLD and researchers have found an overwhelming concordance between the neurological profiles of individuals with AS and NVLD (Klin, Volkmar, Sparrow, Cicchetti, & Rourke, 1995). The GADS was normed on a sample of 371 individuals between the ages of 3 to 22 years who were previously diagnosed with Asperger's Disorder. The sample was very diverse with subjects from 46 states in the United States and more than 5 countries. The Social Interaction Scale yields a standard score and percentile rank. Since the normative sample consisted of individuals with Asperger's Disorder, an average score (*SS* =10) indicates an average probability that the individuals Social Interactions are similar to that of a person with Asperger's Disorder. For the Social Interaction subscale, internal consistency reliability coefficient was high with a coefficient alpha of .90 and test-retest reliability was moderate (r = .76) in the standardization sample. The median item discrimination coefficient for the Social Interaction subscale was .60 (p<.01), providing evidence for content validity (Gilliam, 2001).

Emory Dyssemia Index-Revised (EDI-2; Duke & Norwicki, 2005)

The Emory Dyssemia Index-2 was developed in order to quantify teachers' and parents' observations of a child's nonverbal communication skills. The EDI-2 consists of 42 items that are divided into seven subscales, each of which pertains to a different area of nonverbal communication. The subscales include Gaze and Eye Contact, Space and Touch, Paralanguage, Facial expression, Objectics, Social rules/norms, and Nonverbal receptivity. Items are scored on a 5-point scale with 1 indicating that the behavior is "never" observed and 5 indicating that it is "very often" observed. For each section a raw score of 20 or more is considered clinically significant. The normative sample was 200 school-aged children (Duke & Norwicki, 2005). Validity and reliability data are currently being collected on this measure but preliminary data suggest significant correlations between unstructured social interactions of children, structured laboratory measures of nonverbal communication including the DANVA, and EDI-R scores (Duke & Norwicki, 2005). The EDI-R was used in criterion (A) for NVLD group assignment.

Lafayette Grooved Pegboard (GPT; Knights & Moule, 1968)

The Grooved Pegboard is a timed, individually administered measure of motor speed and dexterity. Subjects place 25 ridged pegs into holes on a 4-inch square pegboard with each hand separately. A time is recorded as the raw score for each hand. The dominant hand is generally 10% faster than the non-dominant hand according to published norms (Knights& Moule, 1968). For children ages 9-15, test-retest reliability coefficients were 0.80 for the dominant hand and 0.81 for the nondominant hand in the standardization sample (Knights& Moule, 1968). Validity studies indicated that the GPT discriminated between children with neurological dysfunction and normal controls

(Trites, 1989). Normative data for children is based on the results of 184 children between the ages of 5 to 15 years old (Knights, 1968). The GPT was used in criterion (B) for NVLD group assignment.

Rey-Osterrieth Complex Figure Test (ROCF; Denman, 1987)

The ROCF is an individually administered test used to measure visual-spatial constructional ability and visual memory. The test is administered in three parts with an initial copy where the subject is asked to copy a drawing of a complex figure, the immediate recall, and the delayed recall after 45 minutes (Denman, 1987). Although there are various scoring methods, this study will utilize the Denman scoring method, a more objective system that is reported to have better inter-rater reliability (Denman, 1987). For each drawing, there are 24 scoring criteria, each scored on a 3-point scale. The raw scores can be converted to standard scores and percentiles. The standardization sample consisted of more than 500 normal and neurologically impaired individuals between the ages of 9 and 69 years. Analysis of correlation with other psychological tests indicate that the copy portion of the ROCF moderately correlates (r = 0.38-0.48) with the Block Design subtest from the Wechsler intelligence tests (Poutlin & Moffit, 1995). For this study, the Copy score was used to measure visual-spatial constructional ability for criterion (C) of the NVLD group.

The Beery-Buktenica Developmental Test of Visual-Motor Integration (Beery VMI; Beery& Buktenica, 2004)

The Beery VMI is a test of visual-motor integration that can be used for individuals ages 2-18 years. It is thought to be one of the most valid tests of visual-motor integration, and has been standardized five times since the initial publication in 1967 with more than 11,000 children. On the 30-item Beery VMI, children or adolescents are asked to copy geometric figures using pencil and paper. The figures follow a developmental sequence of increasing difficulty. Items are scored as correct or incorrect based on explicit criteria. Interrater reliability is high with a reported coefficients ranging from .92 to .99 (Beery, 2004). The average of inter-scorer, internal consistency, and test-retest reliability was .92 in the standardization sample for the Beery VMI indicating high reliability. Factor analytic studies have found that the Beery VMI fits well into the visuospatial-motor factor (Williams & Dykman, 1994). For this study, the Beery VMI scaled score was utilized for the NVLD group criterion (C).

DEPENDENT MEASURES

The following measures were used to examine neuropsychological aspects of attention functioning in the three diagnostic groups. See Appendix F for a summary of the construct(s) of attention measured by each neuropsychological test in this study.

Connor's Continuous Performance Task- Second Edition (CPT-II; Conners & MHS Staff, 2000).

The Conner's Continuous Performance Test II is a computerized measure of sustained attention and response inhibition. The respondent is instructed to press the space bar when any letter appears on the computer screen except the target letter (X). The letters appear on the computer screen for fourteen minutes at varying intervals of one, two, and four seconds with a display time of 250 milliseconds. Normative data is provided for ages 6 to 65 collected from a sample of 1920 individuals from a research study and epidemiological sample. The computer program provides 14 scores related to inattention, impulsivity, and vigilance. All scores are presented as *T*-scores and the authors suggest a lower than normal cut-off for clinical significance of 60 or above (Conners & MHS, 2000). Test-retest correlation coefficients for the 14 scores range from .55 to .84. Validity evidence is strong given that research has demonstrated that the CPT accurately discriminates between children with ADHD and those without ADHD (Barkley et al., 2001). Two scores from this test were analyzed in the current study. The Variability score is a measure of response time consistency calculated as the standard deviation for the standard error values for each sub-block and was used as a measure of vigilance. The Commission score reflects the number of time the respondent erroneously responded to the target letter (X). In a factor analysis of 101 adolescence with ADHD, Barkley et al. (2001) found that the Commission score contributed significantly to the inhibition factor and was used in this study as a measure of response inhibition.

Trails Making Test (TMT)

The Trail Making Test is an individually administered measure of attention, speed, and mental flexibility. The Intermediate Version has normative data for ages 9 to14 years of age and the Adult Version has normative data for ages 15 to 89 years of age. This is a widely used neuropsychological test with at least 46 normative studies (Straus, 2006). The test consists of two parts, with a sample practice for each part. For Part A, the subject is instructed to connect a random array of numbered circles with pencil lines in number order as quickly as possible. For Part B, the subject is instructed to connect a random array of numbers and letters in order alternating between a number and a letter. Findeis and Weight (1995) presented meta-norms for the childhood neuropsychological tests from the Halstead-Reitan batteries to reflect a continuum of abilities rather than the previous cut-off scores which suggested dichotomous brain functioning. The test-retest reliability varies with the age range and population but is generally adequate, especially for Part B with a correlation coefficient of approximately .75 (Straus, 2003).

Coding, Digit Span Backward, and Letter/Number Sequencing subtests from the Wechsler's Intelligence Scale for Children- Fourth edition (WISC-IV)

As described in the independent measures from this study, the WISC-IV is an intelligence test that provides a comprehensive measure of general cognitive functioning for children 6-16 years of age. Selected subtests from this measure are of particular interest to this study as measures of attention and executive functioning. The Coding subtest is a core Processing Speed subtest. The child is instructed to copy symbols that are paired with numbers. In addition to processing speed, the subtest measures visual scanning ability, visual-motor coordination, cognitive flexibility and attention (Wechsler, 2003). The internal consistency for the Coding subtest is .85 and the test-retest reliability coefficient was .84 in the standardization sample. Digit Span is a Working Memory subtest composed of two parts, Digit Span Forward and Digit Span Backward. While Digit Span Forward requires the child to repeat verbally presented strings of numbers, Digit Span Backward requires the child to repeat the string in reverse order. Digit Span Backward has a stronger working memory component because the information must be manipulated and is more related to executive functioning processes (Hale, Hoeppner, & Fiorello, 2002). Internal consistency reliability was estimated to be .87 and the test-retest reliability estimate was .71 in the standardization sample (Wechsler, 2003). The Letter/Number Sequencing subtest is also a core working memory subtest. The child is verbally presented with a sequence of numbers and letters and is instructed to recall them in ascending number order and then ascending letter order. This test involves mental manipulation and attention (Crow, 2000). The internal reliability was estimated to be .90 and the test-retest reliability was estimated to be .83 in the standardization sample (Wechsler, 2003).

CHAPTER IV: RESULTS

This chapter reports the results of analyses conducted to answer the research questions proposed in Chapter II. All analyses were conducted using the Statistical Package for the Social Sciences (SPSS) for Windows, Version 13.0. An alpha level of .05 was set prior to analysis. Preliminary analyses were conducted to identify differences among the three diagnostic groups on independent variables including gender, race/ethnicity, age, and FSIQ. The results of the preliminary analysis and the results of the three research hypotheses are presented below.

Preliminary Analysis

Differences between groups in age, FSIQ, and parents' highest level of education were considered to determine potential covariates in subsequent analyses of the research hypotheses. Separate Analyses of Variance (ANOVAs) were conducted to determine whether significant group differences existed on these variables in the sample. Means and standard deviations for age and FSIQ are presented in Table 1. The ANOVAs indicated that groups did not vary significantly by age, F (2, 85) = .646, p = .527 or by FSIQ, F (2, 85) = .719, p = .490.

Table 1: Means and Standard Deviations for Age in Months and FSIQ

	NVLD (n=24)		ADHD:PI (n=37)		ADHD:C (n=27)	
Variables	*M*	*SD*	*M*	*SD*	*M*	*SD*
Age	139.42	23.086	144.14	22.271	138.11	22.011
FSIQ	102.83	12.107	104.22	8.025	106.30	11.721

Parents' highest level of education in years was also analyzed to determine the presence of group differences. Of the 88 participants, data for the highest level of education achieved for at least one parent was available for 71 participants. Given the missing data on parent education, the following statistics should be interpreted with some degree of caution. Means and standard deviations for parents' highest level of education are presented in Table 2. The three groups did not vary significantly by parent education, $F(2, 68) = 1.545$, $p = .221$.

Table 2: Means and Standard Deviations for Parent Education in Years

	NVLD	ADHD:PI	ADHD:C
Mean	16.00	16.87	15.91
Standard Deviation	2.828	2.0809	1.571

Additionally, differences between groups in ethnicity and gender were considered as possible confounds of the results of the research hypotheses. Table 3 and Table 4 delineate the frequency counts of Gender and Ethnicity/Race for each of the diagnostic groups.

Table 3: Gender Frequency Across Groups

	NVLD	ADHD:PI	ADHD:C
Male	19	25	22
Female	5	12	5

Table 4: Ethnicity/Race Frequency Across Groups

	NVLD	ADHD:PI	ADHD:C
White-Not of Hispanic origin	20	32	22
Hispanic	1	4	2
African American	1	1	1
Asian/Pacific Islander	1	0	1
Multiethnic/Multiracial	1	0	1

A chi-square analysis of gender was conducted to detect statistically significant differences between groups on this categorical demographic variable. The results of the chi square test for gender is presented in Table 5. No statistically significant differences were found among groups on this variable in the study sample. It should be noted that limited representation in the study sample of minority groups and females limits generalizability of the analysis of these demographic variables to the general population.

Table 5: Chi-Square Analysis for Gender by Group

Variable	*n*	*Df*	χ^2	*p*
Gender	88	2	1.917	.383

Given no significant group differences were found on Age, FSIQ, Parent Education, or Gender in the study sample, no covariates were incorporated into subsequent analyses.

Primary Analysis

ANALYSIS OF RESEARCH QUESTION 1

Research question one examined whether children with NVLD, ADHD:PI, and ADHD:C differed on neuropsychological measures of attention. It was hypothesized that there would be significant differences on scores among diagnostic groups on the neuropsychological measures of attention. Furthermore, it was expected that groups would differ depending upon the attention modality (auditory vs. visual). Six neuropsychological measures of attention were included in this study: WISC-IV Digit Span Backward, WISC-IV Letter/Number Sequencing, WISC-IV Coding, Trails B, CPT-Variability, and CPT-Commission. Means and standard deviations for these measures in the study sample are presented in Table 6. Data were prescreened for multivariate outliers using a Mahalanobis Chi Square test. The accepted criterion for outliers is a value which is significant at the $p \leq .001$, determined by comparing the chi square critical value to the Mahalanobis distance value (Mertler and Vannatta, 2005). The test revealed no statistically significant multivariate outliers with 6 *df* at $p \leq .001$. The Box's test revealed that homogeneity of variance-covariance can be assumed at the $p < .05$ level.

A multivariate analysis of variance (MANOVA) was conducted to determine diagnostic group differences on the attention measure scores. Results of the overall MANOVA are presented in Table 7. Results indicated a statistically significant difference among groups, *Wilks'* $\lambda = .631$, $F(12, 160) = 3.456$, $p < .001$. Twenty-one percent of the multivariate variance was associated with diagnostic group membership ($\eta_p^2 = .206$). Univariate ANOVAs were conducted on each dependent variable as a follow-up test to

MANOVA. Results indicated that scores on the WISC-IV Coding, $F(2, 85) = 5.185$, $p = .008$; Trails B, $F(2, 85) = 3.393$, $p = .038$; CPT Variability, $F(2, 85) = 3.462$, $p = .036$; and CPT Commissions, $F(2, 85) = 7.474$, $p = .001$; were all significantly related to diagnostic group. Effect sizes indicate that moderate proportions of variance were accounted for by Coding, Trails B, and CPT Variability, while a substantial proportion of variance was accounted for by CPT Commissions. Scores on the WISC-IV Digit Span Backwards, $F(2, 85) = .094$, $p = .910$, and WISC-IV Letter/Number Sequencing $F(2, 85) = 1.035$, $p = .360$, were not significantly related to diagnostic group. It is notable that groups did not significantly differ on any of the measures presented in the auditory modality, while significant group differences were found on all the visual measures of attention.

Table 6: Means, Standard Deviations, and Partial Eta-Squared Coefficients for Neuropsychological Measures

	NVLD (n=24)		ADHD:PI (n=37)		ADHD:C (n=27)			
Variables	*M*	*SD*	*M*	*SD*	*M*	*SD*	*F*	η_p^2
WISC-IV DSB (scaled score)	9.88	1.963	9.59	2.544	9.67	2.828	.940	.002
WISC-IV LNS (scaled score)	10.71	1.334	10.73	2.143	9.89	3.545	1.035	.024
WISC-IV Coding (scaled score)	7.00	1.865	8.11	2.612	9.30	2.946	5.185*	.109
Trails B (Z-score)	-1.21	1.469	-.57	1.639	-.048	1.619	3.393*	.074
CPT-Variability (percentile rank)	50.95	31.88	33.98	26.23	31.26	30.13	3.462*	.075
CPT- Commissions (percentile rank)	55.92	26.01	49.55	30.40	27.91	25.25	7.474*	.150

* p < .05

Post-hoc tests were conducted to assess differences among the groups on the WISC-IV Coding, Trails B, CPT Variability, and CPT Commissions. Gabriel's procedure for group comparisons was utilized to control error because of the unequal group sizes. Table 7 summarizes the results of the post-hoc analyses. On the WISC-IV Coding subtest, the NVLD group performed significantly lower than the ADHD:C group (p = .006) . No significant group differences were found on WISC-IV Coding scores between the NVLD and the ADHD:PI groups (p = .266) or between the two ADHD groups (p = .189). A similar pattern was observed on the Trails B test. The NVLD group performed significantly lower than the ADHD:C group (p = .032), and no significant group differences were found between the NVLD and the ADHD:PI groups (p = .333) or between the two ADHD groups (p = .476).

Table 7: Summary of Post-Hoc Assessing Differences Among Groups on Measures

Measure	Diagnoses	Mean Difference	*p*-value
WISC-IV Coding			
	NVLD – ADHD:PI	-1.108	.266
	NVLD – ADHD:C	-2.296*	.006
	ADHD:PI – ADHD:C	-1.188	.189
Trails B			
	NVLD – ADHD:PI	-.638	.333
	NVLD – ADHD:C	-1.161*	.032
	ADHD:PI – ADHD:C	-.523	.476
CPT-Variability			
	NVLD – ADHD:PI	16.974	.080
	NVLD – ADHD:C	19.691	.052
	ADHD:PI – ADHD:C	2.717	.976
CPT-Commissions			
	NVLD – ADHD:PI	-6.371	.760
	NVLD – ADHD:C	28.008*	.002
	ADHD:PI – ADHD:C	21.636*	.008

* $p < .05$

Post-hoc analysis using Gabriel's procedure revealed no significant group differences on CPT Variability. This result could be related to the stringent Gabriel test statistic, and given the non-significant trends, group differences may have been observed with a balanced design. Although not statistically significant, these trends imply that the NVLD group scored higher than the ADHD:PI group ($p = .080$) and the ADHD:C group ($p = .052$), while the two ADHD groups performed similarly (.976).

As expected on the CPT Commissions score, the NVLD and ADHD:PI groups did not significantly differ from each other (.760), but both the NVLD and the ADHD:PI groups performed significantly higher than the ADHD:C group ($p = .002$; $p = .008$, respectively).

Analysis of Research Question 2

Research question 2 examined whether children with NVLD, ADHD:PI, or ADHD:C demonstrate significant impairment on neuropsychological measures of attention compared with published normative data. Given the inconsistencies in published findings on the neuropsychological measures of interest, it is unclear whether the children in the three diagnostic groups exhibit statistically significant and/or clinically significant impairments on the attention measures. This research question was addressed by comparing sample data from the three diagnostic groups to published normative data on the six neuropsychological measures of interest. *Z*-scores were calculated for the individual child on each of the six scores obtained from the neuropsychological measures. One sample *t*-tests were conducted for each diagnostic group on the six attention measures to determine whether the sample groups statistically differed from the normative sample at the *p*<.05 level. Additionally, mean *z*-score differences for the groups were examined to determine whether a clinically significant difference of more than one standard deviation from the normative sample mean was present. See Table 8 for a summary of the results.

Table 8: Mean *z*-score Differences (MzD) and t values by Diagnostic Group

	NVLD		ADHD:PI		ADHD:C	
Variables	*MzD*	t	*MzD*	t	*MzD*	t
WISC-IV DSB	-.04	.312	-.14	.969	-.11	.612
WISC-IV LNS	.23	2.600*	.24	2.071*	-.04	.163
WISC-IV Coding	-1.01**	7.880*	-.63	4.406*	-.23	1.241
Trails B	-1.21**	4.030*	-.57	2.118*	-.05	.153
CPT-Variability	-.09	.401	-.53	3.648*	-.66	3.323*
CPT- Commissions	.25	1.354	-.03	.149	-.68	3.715*

* statistically significant at p<.05
**clinically significant at Mean z-score difference > 1 standard deviation

Children with NVLD in the current sample performed significantly lower than the normative sample on the WISC-IV Coding ($t(23) = -7.88$, $p < .05$) and Trails B ($t(23) = -4.03$, $p < .05$) tests. Furthermore, the NVLD group showed a clinically significant mean z-score difference on both WISC-IV Coding and Trails B. The NVLD group performed significantly higher than the normative sample on the WISC-IV Letter Number Sequencing test ($t(23) = 2.60$, $p < .05$); however, the difference was not clinically significant. No statistically or clinically significant differences compared to normative means were found on the WISC-IV DSB, CPT Variability, or CPT Commissions.

Children with ADHD:PI in the current sample performed significantly lower than the normative sample on the WISC-IV Coding ($t(23) = -4.406$, $p < .001$), Trails B ($t(23) = -2.118$, $p < .05$), and CPT Variability ($t(23) = -3.648$, $p = .001$). Similar to the NVLD group, the ADHD:PI group performed significantly higher than the normative sample on the WISC-IV LNS ($t(23) = 2.071$, $p < .05$). While statistical significance occurred on these four measures, clinically significant differences in performance were not observed on any of the measures. No statistically or clinically significant differences compared to the normative data were found on the WISC-IV DSB or CPT Commissions.

Children with ADHD:C in the current sample performed significantly lower than the normative sample on the CPT Variability ($t(23) = -3.323$, $p = .003$) and CPT Commissions ($t(23) = -3.715$, $p = .001$). A mean difference of greater than one standard deviation was not observed on either of these measures indicating that the ADHD:C group did not exhibit a clinically significant weakness on either measure. No statistically or clinically significant differences compared to the normative data were found on the WISC-IV Coding, WISC-IV Digit Span Backwards, WISC-IV Letter/Number Sequencing, or Trails B.

ANALYSIS OF RESEARCH QUESTION 3

Finally, research question three explored whether the neuropsychological measures of attention in this study are useful and accurate in discriminating between children with NVLD, ADHD:PI, and ADHD:C. It was hypothesized that the measures would yield two functions, and that these functions would discriminate between the three groups. The hypothesized functions included one that represented the Attention Modality (visual vs. auditory) and one that represented Inhibition style.

Results from the Univariate ANOVAs and post hoc tests in the analysis of research question one indicated that groups significantly differed on WISC-IV Coding, Trails B, CPT-Variability, and CPT-Commission scores. Since the groups did not significantly differ on WISC-IV DSB and WISC-IV LNS scores, these measures were not included in further analysis (Field, 2005). It should be noted that these two measures represented the auditory attention modality, eliminating the possibility of a visual versus auditory attention modality function.

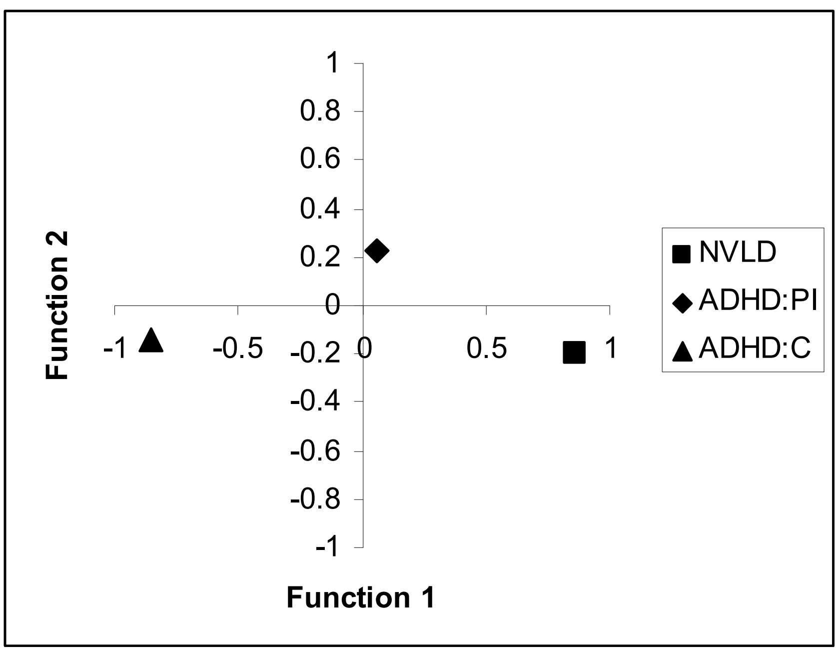
The four variables (WISC-IV Coding, Trails B, CPT-Variability, and CPT-Commission) were submitted to a discriminant analysis. The analysis generated two functions; however, only one function was significant, (Function 1: Wilks' $\Lambda = .67$, $\chi^2(8) = 33.86$, $p < .001$; Function 2: Wilks' $\Lambda = .96$, $\chi^2(3) = 9.30$, $p = .36$). The significant function accounted for 30.69% of the explained variance. The standard function coefficients in Table 9 represent the degree to which each measure contributes to the significant function. Examination of the Standardized Function Coefficients suggests that all four variables are related to the function, with CPT Commissions exhibiting the highest loading. Function 1 is interpreted as an Inhibition and Visual Processing Speed Function.

Table 9: Standardized Function Coefficients for the Significant Function

Measures	*Standardized Function Coefficients*
WISC-IV Coding	-.482
Trails B	-.500
CPT Variability	.429
CPT Commission	.621

The relation between the functions and the groups is illustrated in Figure 1, where the group centroids are plotted in discriminant space. The horizontal distance between centroids clearly shows that Function 1 discriminates between the NVLD and ADHD:C group.

Figure 1: Group Centroids plotted in Discriminant Space



By examining the signs of the group centroids, it appears that the first function also discriminates between ADHD:PI and ADHD:C, but to a considerably lesser extent than between the NVLD and ADHD:C groups. The non-significant Function 2 did not differentiate between groups as shown by the very small vertical group centroid separation on this variate.

Classification results revealed that only 59.1% of the original groups were classified correctly. While the overall classification results were unimpressive, the percent correctly classified varied greatly by group. Of the original group, 75% of the NLVD group were correctly classified, 40.5% of the ADHD:PI group were correctly classified, and 70.4 percent of the ADHD:C group were correctly classified. These results suggest that the significant function effectively separates the children with NVLD from ADHD:C. See Table 10 for detailed classification results for each group.

Table 10: Classification Categories Relative to the Original Diagnosis

Original Diagnosis	Predicted Group Membership		
	NVLD	ADHD:PI	ADHD:C
NVLD	75.0	16.7	8.3
ADHD:PI	21.6	40.5	37.8
ADHD:C	18.5	11.1	70.4

Note. Values in table are percentages.

The results suggest that the neuropsychological measures of attention may be most useful for discriminating between children with NVLD and ADHD:C. Furthermore, children with higher Inhibition and lower Visual Processing Speed are more likely to be classified as having NVLD or ADHD:PI. Inversely, children with lower Inhibition and higher Visual Processing Speed are likely to be classified as having ADHD:C.

CHAPTER V: DISCUSSION

Summary and Integration of Findings

This study aimed to reconcile the discrepancy between conceptualizations of attention problems in children with NVLD. While it is widely recognized that many children with NVLD also meet clinical criteria for ADHD (Gross-Tsur & Shalev, 1995; Voeller, 1996), little research has focused on the specific attention problems of children with NVLD. Research suggests that children with NVLD are behaviorally more similar to children with ADHD, Predominantly Inattentive subtype according to DSM-IV-TR criteria (APA, 2000; Rourke, 1995; Voeller, 1986). One hypothesis explains these behavioral similarities between children with NVLD and ADHD:PI through overlapping neural regions (Denckla, 2000; Stefanatos & Wasserstein, 2001). It has been suggested that ADHD:PI and NVLD stem from neurological dysfunction in the right hemisphere and are associated with white matter dysfunction. According to this model, NVLD and the two ADHD subtypes represent a continuum of dysfunction dependant on overlapping neural regions. An opposing view posited by Rourke (1995) speculates that while the behavioral symptoms of inattention in children with NVLD may mimic those of ADHD, the underlying deficits are distinct. Rourke (1995) suggested that a developmental sequence of strengths and deficits in children with NVLD results in generally intact auditory attention with impaired attention for visual stimuli.

Given these opposing views of attention problems in children with NVLD, the current study examined neuropsychological constructs of attention including working memory, processing speed, vigilance, and inhibition. Performance on attention measures was explored in the visual and auditory modality separately in order to address Rourke's modality-specific attention model of NVLD. Furthermore, the current study evaluated performance of children with NVLD in comparison to children with ADHD:PI and ADHD:C. Although considerably more research is needed in order to determine the exact neurological underpinnings of ADHD:PI and ADHD:C, there is evidence that symptoms of ADHD:C are more closely related to dysfunction of midline frontal areas and parts of the basal ganglia, while symptoms of ADHD:PI are related to right superior frontal regions and right parietal regions of the brain (Berger & Posner, 2000; Posner, 2000; Seargent, 1999). By separating the ADHD group into the two subtypes, more detailed hypotheses of underlying neurological dysfunction could be posed for the NVLD group depending on similarities and differences between groups on the neuropsychological measures of attention.

While two opposing models of attention problems in children with NVLD were examined from a neuropsychological perspective, evidence from the current study supported the model in which NVLD and the two ADHD subtypes represent a continuum of dysfunction dependant on overlapping neural regions (Denckla, 2000; Stefanatos & Wasserstein, 2001). Although considerably more research is needed to determine exact anatomical correlates of neuropsychological measures, these findings from the current study suggest a continuum of dysfunction from right posterior to bilateral anterior brain regions. Thus, the children with NVLD would be expected to have greater dysfunction in parietal and right hemisphere functioning, children with ADHD:C would exhibit dysfunction in bilateral prefrontal regions, and ADHD:PI would exhibit overlap between these two disorders. The findings from this study are not definitive proof of this anatomical continuum, but may lend neuropsychological support for future research in this area. Moreover, a specific profile of attention strengths and deficits in children with NVLD compared to children with ADHD:PI, ADHD:C, and normative data were identified in order to inform clinical diagnosis and intervention.

SAMPLE CHARACTERISTICS

The sample utilized in the current study consisted of 88 children between the ages of 9 and 15 years old. The sample was comprised of twenty-four children with NVLD, thirty-seven children with ADHD:PI, and twenty-seven children with ADHD:C. Within the NVLD group, two of the children met criteria for ADHD:C and twenty met criteria for ADHD:PI. The three groups were found to be well-matched on age, IQ, parent education, and gender, as these variables were not found to differ significantly across diagnostic groups. While gender did not differ significantly across groups, it should be noted that overall the sample consisted of 3:1 ratio of boys to girls. Most participants were from families where at least one parent is college-educated. Eighty-four percent of the sample was composed of white children of non-Hispanic decent.

DIAGNOSTIC GROUP DIFFERENCES

The first research question examined the differences among the NVLD, ADHD:PI, and ADHD:C diagnostic groups on neuropsychological measures of attention. This study assumed Rourke's (1995) modality-specific perspective that children with NVLD have primary deficits in visual perception that result in secondary visual attention deficits, in the absence of auditory attention deficits. Thus, it was hypothesized that children with NVLD would perform better than children in both ADHD groups on auditory attention measures and similarly, or even more poorly, than children with ADHD on visual attention measures. While differences in visual and verbal attention were noted, the results are more accurately explained by the underlying attention constructs because the directionality of strengths and weaknesses was not consistently associated with the attention modality. Specifically, the three diagnostic groups differed only on visual attention measures, but the NVLD group did not consistently perform more poorly on the visually mediated measures as hypothesized by Rourke's (1995) modality-specific model.

The three groups did not significantly differ on either the WISC-IV Digit Span Backwards or the WISC-IV Letter/Number Sequencing. These measures represented the auditory attention modality indicating that the NVLD group did not perform better than the two ADHD groups as expected. These measures also had the strongest working memory component. These findings suggest that children with NVLD, ADHD:PI, and ADHD:C do not significantly differ on auditory working memory abilities.

The groups significantly differed on the visual attention measures, but the differences were not consistently in the expected direction according to the modality-specific model. As expected, the NLVD performed more poorly on the visually mediated processing speed measures, WISC-IV Coding and Trails B, than the ADHD:C group. The NVLD group, however, did not perform significantly worse than the ADHD:PI group. The ADHD:PI group mean was in between the NVLD and ADHD:C groups, and did not significantly differ from either group. These results are more consistent with the overlapping neural regions model (Denckla, 2000; Stefanatos & Wasserstein, 2001). As described in detail in the Literature Review, Denckla (2000) described a "cognitive overlap zone" of executive functions in NVLD and ADHD due to overlapping neural regions, resulting in a continuum of impairment. The overlapping neural regions model suggests that NVLD, ADHD:PI, and ADHD:C represent a continuum of cognitive impairment dependant on the degree and location of white matter dysfunction in the brain. The findings from the current study suggest that for brain regions responsible for visual processing speed, NVLD represents one end of the continuum and ADHD:C represent the other, with ADHD:PI in the middle.

On the CPT-Variability, a measure of visual vigilance, the NVLD group trended toward better performance than both ADHD groups; however, no group differences were statistically significant. These findings are not consistent with the modality-specific model which states that children with NVLD should perform worse than children with ADHD on visual attention measures. The findings may be more consistent with the overlapping neural regions model in that regions responsible for vigilance may exhibit more overlap in these three disorders. According to Posner's (1994) model of attention networks in the brain, the alerting network is necessary for vigilance and permits maintenance of alertness. This network is thought to involve the right superior frontal regions, and possibly right hemispheric parietal regions.

Finally, on the CPT Commissions, a measure of visual impulsivity, the ADHD:C group performed significantly lower than both the NVLD and ADHD:PI groups. The NVLD and ADHD:PI groups did not significantly differ from each other on this measure. Again, these findings are inconsistent with the modality-specific model; however, precisely represent expected results in the overlapping neural regions model. According to this model, children with ADHD:C are thought to have more difficulty with midline frontal areas and show significant impulsivity, whereas children with NVLD and ADHD:PI have more posterior deficits unrelated to impulsive behavior. It follows then that children with ADHD:C would perform significantly worse than the NVLD and ADHD:PI groups on CPT Commissions which measures impulsivity. Overall, the findings regarding group differences support the overlapping neural regions model rather than the modality-specific model of attention problems in children with NVLD.

Furthermore, results suggest a continuum of dysfunction with NLVD more closely related to ADHD:PI than ADHD:C.

It should also be noted that even though the findings do not support the model based on auditory versus visual attention modality, there is evidence that visual-spatial perception deficits negatively affect performance on visual attention measures in children with NVLD. Children with NVLD performed significantly worse than children with ADHD:C on the two measures that required the highest visual-spatial component, Coding and Trails B. While the CPT is visually mediated, the visual-spatial perception difficulty is low compared to the visual-spatial component of the Coding subtest and Trails B.

DIAGNOSTIC GROUPS VERSUS NORMATIVE DATA

Research question 2 explored whether the measures utilized in this study, and commonly used in clinical practice, are statistically and clinically useful in separating the three diagnostic groups from the normal population using normative data. Furthermore, the pattern of strengths and weaknesses compared to normative data were examined to further explore the discrepancy between the attention-modality model versus the overlapping neural regions model of attention problems in children with NVLD. Specifically, the current study examined whether children with NVLD, ADHD:PI, or ADHD:C demonstrate significant impairment on neuropsychological measures of attention compared with published normative data. Previous research suggests (Chhabildas et al., 2001; Marinussen, 2005; Nigg et al., 2002; Willicutt et al., 2005) children with ADHD differed significantly from controls on the majority of executive functioning tasks, but results are inconsistent on whether these results are clinically significant. There are few, if any, conclusive findings on the statistical and clinical significance of attention functions in children with NVLD.

Statistically all the attention measures except WISC-IV DSB were able to separate one or more of the diagnostic groups from the published normative data. The NVLD and ADHD:PI group exhibited statistically significant impairment on WISC-IV Coding and Trails B and a statistically significant strength compared to normative data on WISC-IV LNS. These findings indicate children with NVLD and ADHD:PI exhibit deficits in visual processing speed, while they exhibit strengths in verbal working memory. Additionally, the ADHD:PI group exhibited a statistically significant weakness on CPT Variability indicating poor vigilance. Children with ADHD:C exhibit statistically significant impairment on both the CPT Variability and CPT Commissions indicating weaknesses in vigilance and impulsivity. The overlap in dysfunction between the NVLD and ADHD:PI group on measures of visual working memory, in the presence of a relative strength in verbal processing speed further supports the overlapping neural regions model. The results also support the modality-specific model for NVLD, but further indicate that children with ADHD:PI may exhibit similar modality-specific attention problems. Thus, since the ADHD:PI group exhibits similar modality-specific impairments as the NVLD group, the overlapping neural regions model is more salient than Rourke's (1995) model, which states that children with NVLD and ADHD have distinct impairments.

While many of the measures statistically separated groups, the clinical significance was not as compelling. Only the WISC-IV Coding and Trails B measures were clinically relevant in separating the NVLD group from normally developing children according to published normative data. Neither ADHD group exhibited clinically significant strengths or weaknesses on the attention measures.

Again, these results are consistent with the overlapping neural region model proposed by Denckla (2000) that conceptualizes the disorders of NVLD and ADHD as a continuum of brain dysfunction with NVLD at the more severe end. While the measures were not sensitive enough to clinically separate the two ADHD groups from the normal population using normative data, the data partially supports Denckla’s model since clinically significant weaknesses were observed in the NVLD group which is supposed to be the most severe end of the continuum.

A contributing explanation for this dearth of clinically significant differences between diagnostic groups and normative data on measures of attention is that the measures, in isolation, are not sensitive enough to determine attention problems. In practice, clinicians compile data from a variety of sources including behavioral, observational, and test data in order to diagnose children with attention disorders. Because attention is such a multivariate construct, a pattern of strengths and weaknesses must be observed to separate children with attention disorders from the normal population. Using measures in combination to separate children with attention disorders and the normal population will be explored further in the discussion of the results from discriminate function analysis in Hypothesis 3.

DISCRIMINATIVE POWER OF NEUROPSYCHOLOGICAL MEASURES

Research question three explored whether the neuropsychological measures of attention in this study are useful and accurate in discriminating between children with

NVLD, ADHD:PI, and ADHD:C. Because performance on auditory modality measures did not significantly differ between groups, the expected dual function model (visual versus auditory) was not supported. The four visual attention measures resulted in a single function that clearly discriminated between the NVLD and ADHD:C diagnostic groups. Of the original group, 75 percent of the NLVD group were correctly classified and 70.4 percent of the ADHD:C group were correctly classified using the four attention measures. Less than half, 40.5 percent, of the ADHD:PI group were accurately classified using the four attention measures. These results lend even greater support to the overlapping neural regions model which conceptualizes the three disorders as a continuum of dysfunction. Higher scores on visual processing speed measures, with lower scores on inhibition resulted in a classification as ADHD:C. In contrast, lower scores on visual processing speed measures with higher scores on the inhibition measure resulted in classification as NVLD. Children with ADHD:PI exhibited performance between these two extremes on the continuum resulting in poor accuracy of classification.

Clinical Implications

DIFFERENTIAL DIAGNOSIS

There are currently no formal diagnostic criteria for NVLD. Many professionals in the field use a single criterion, namely the Verbal versus Visual Intelligence discrepancy, for diagnosing children with NLVD. Research suggests that this criterion only applies to approximately 42% of children with NVLD (Pelletier, 2001). This study looked at NVLD as a syndrome and included children in the NVLD group that displayed at least three areas of impairment consistent with the most recent data on the NVLD-related deficits. While the primary aim of the current study was to examine two opposing theoretical models of the NVLD syndrome, the study was also designed to have practical applications for diagnosis in a clinical setting.

Taken together, the results from the current study confirm that a battery of measures is more effective in discriminating between attention problems in children with NVLD, ADHD:PI, and ADHD:C than any single measure. No single neuropsychological measure of attention utilized in this study was able to discriminate the three groups from one another; however, by using a battery of attention measures including measures of processing speed, working memory, vigilance, and impulsivity, group differences emerged. Evidence from the discriminate function analysis showed that approximately 75 percent of the NVLD group and 70 percent of the ADHD:C group were correctly classified using a battery of attention measures including WISC-IV Coding, Trails B, CPT-Variability, and CPT-Commission. Furthermore, the second analysis showed that WISC-IV Coding and Trails B were the most sensitive to attention problems in children with NVLD. The visual processing speed and visual working memory measures not only detected statistically significant impairments compared to normative data, but also clinically significant impairments described as performance that was less than one standard deviation below the mean of the normative sample.

INTERVENTION

As literature on the clinical profiles of children with NVLD increases, interventions have begun to emerge targeting this group. Efficacious interventions specifically address deficits while drawing on strengths. Attention problems are a salient feature of this syndrome since literature suggests problems with attention can contribute to social, emotional, and academic problems. This study contributed to the understanding of specific attention problems and attention-related strengths in children with NVLD. Children with NVLD exhibited clinically significant attention deficits in visual processing speed and set-shifting. Thus, intervention and accommodations designed for children with NVLD should target these attention deficits specifically. For instance, children with NVLD exhibited clinically significant deficits on the WISC-IV Coding subtest compared to normative data. This subtest draws on similar skills as note-taking in school. Children with NVLD may need targeted instruction on note-taking strategies. Accommodations could include providing the student with outlines or a peer note-taker that can make copies of notes for the child with NVLD. Additionally, since children with NVLD showed many similar neuropsychological attention deficits as children with ADHD:PI, behavioral interventions that are shown to be effective for children with ADHD:PI could be applied to children with NVLD. It should be noted that these interventions targeting attention problems would need to be augmented for children with NVLD to address other problems associated with the disorder such as impairment in social competence.

In addition to behavioral intervention implications, this study also has tentative implications for pharmacological interventions. Although more extensive research would be necessary to determine the exact nature of the neural overlap between ADHD:PI and NVLD, this study showed that there is considerable overlap in terms of neuropsychological functioning. Medications that effectively treat symptoms of ADHD:PI may also be effective on attention problems in children with NVLD since the attention problems may represent similar neural dysfunction on the same continuum. This hypothesis warrants a great deal more research since the effects of medications used for ADHD on the other areas of impairment involved in the NVLD syndrome are unknown.

Limitations

While the sample size consisted of 88 children, exceeding the estimated 75 participants necessary for adequate statistical power, the group sizes varied considerably. The groups consisted of twenty-four children with NVLD, twenty-seven children with ADHD:C, and thirty-seven children with ADHD:PI. Given the unbalanced design, more stringent test statistics were used to limit Type I error rate. The conservative statistics may have limited the statistical power and resulted in a higher Type II error rate. This means that it is possible that group differences were not detected when these differences actually exist.

Secondly, the sample was drawn from a clinically referred population. Because these children were identified as exhibiting significant problems by teachers and/or parents resulting in a referral to the clinic, they may differ from children in the general population. It is likely that the clinically referred children represented the more severe end of the diagnostic spectrum and thus are more easily identified as needing assessment and intervention. Furthermore, since the sample was collected from extant data from this clinically referred population, there was not data collected for a control group. While this study primarily examined differences between ADHD:PI, ADHD:C, and NVLD, a control group would have been ideal to determine how groups differed from normally developing children. This question was partially answered by utilizing normative data; however, the current sample may have differed from the normative sample.

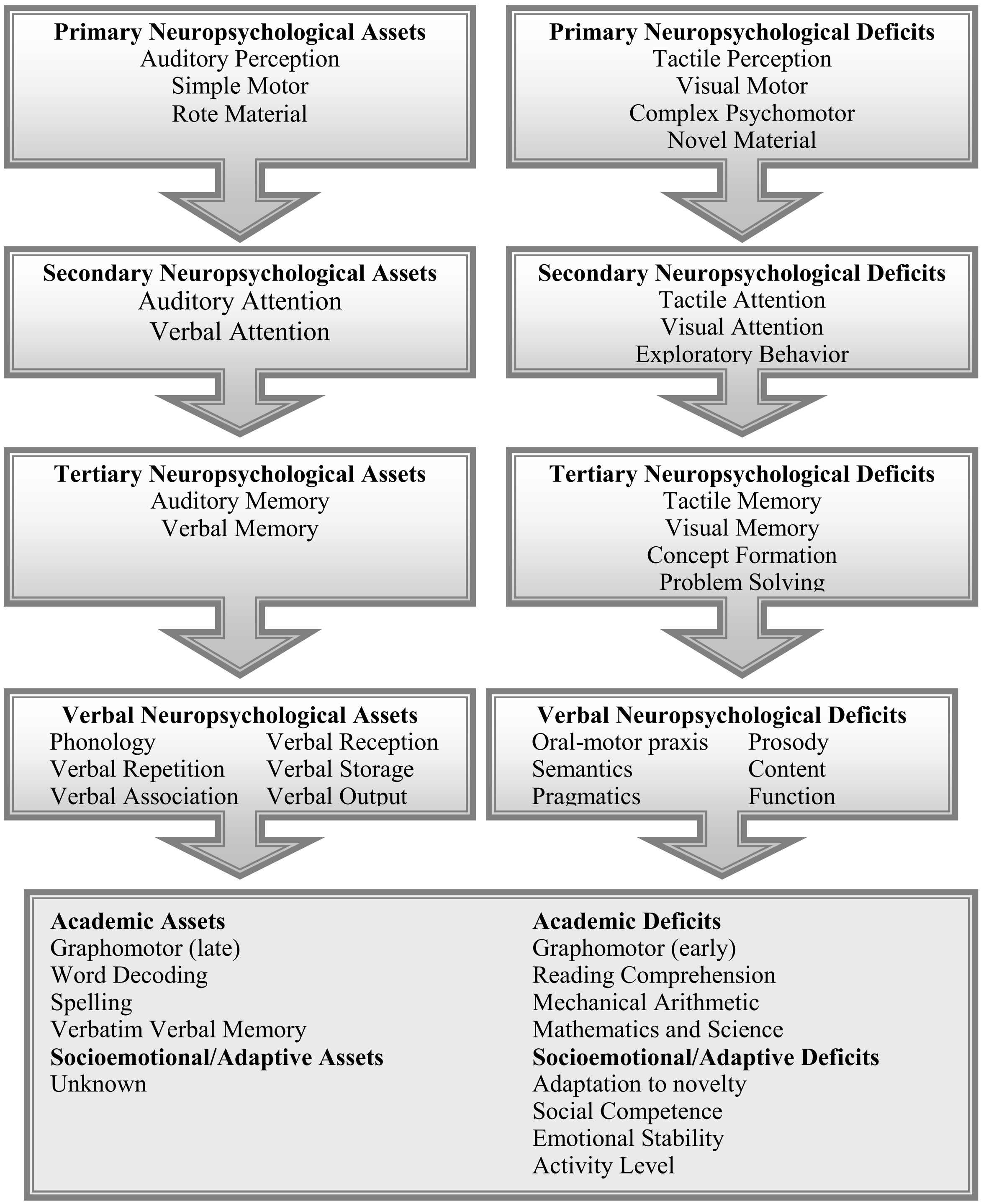
Another important limitation of this study was the homogenous sample in terms of race/ethnicity, gender, and parent education. There was a restricted representation of ethnic/racial groups since eighty-four percent of the sample was white-not of Hispanic origin. The variation in parent education also was limited, with most participants having at least one parent with a college education or higher. Additionally, the sample was seventy-five percent male. Thus, these sample demographics limit generalizability of finding to a many ethnic groups, females, and children of parents with lower education levels.

Directions for Future Research

This study represented one small part of what needs to be a much larger endeavor to understand the profile of children with NVLD. NVLD is a syndrome characterized by impairments ranging from social, motor, and academic problems to the attention deficits addressed in the present study. While researchers have begun to examine the profile of NVLD, extensive research is needed to definitively determine diagnostic criteria for the disorder. Furthermore, studies examining children with NVLD have generally been conducted from solely a neuropsychological perspective. As evidenced by the gains in understanding of ADHD, it is important to coordinate research from a variety of disciplines. For instance, findings in previous studies and the current study suggest overlapping neural regions in NVLD and ADHD:PI. There are suspected right hemisphere neuropsychological impairments and white matter deficits both disorders. By incorporating tools such as Diffusion Tensor Imaging (DTI), which examines white matter tracts in the brain, or Functional Magnetic Resonance Imaging (fMRI), which aids in localization of cognitive functions, these neuropsychological hypotheses could be compared to neurological findings. Additionally, NVLD is hypothesized to be a developmental disorder. During development, myelination in various fiber systems in the brain occurs at different speeds at different points in time. This process proceeds rapidly until two years of age but continues into adulthood (Rourke, 1995). Longitudinal studies including imaging and neuropsychological evaluations from early childhood through adulthood would be beneficial in determining the exact developmental trajectory of impairments including attention problems in children with NVLD.

In sum, NVLD is a syndrome of deficits and strengths that requires a comprehensive battery of neuropsychological measures, observations, and clinical judgment for accurate diagnosis. Evidence from the current study supported the model in which NVLD and the two ADHD subtypes represent a continuum of dysfunction dependant on overlapping neural networks involving right posterior to bilateral anterior brain regions.

Appendix A: Developmental Dynamics of Assets and Deficits in NVLD



Appendix B: Imaging Studies of Attention Deficit/Hyperactivity Disorder

Study	ADHD/ Controls	Imaging Type	Results
Castellanos et al., 1996	57/55	Structural	Right prefrontal volume smaller in ADHD
Hynd et al., 1990	10/10	Structural	Normal R>L prefrontal volume reversed in ADHD
Casey et al., 1997	26/26	Structural	Smaller right prefrontal brain regions in ADHD boys which correlated with poorer performance on response inhibition task
Filipek et al., 1997	15/15	Structural	Smaller caudate and decreased right anterior superior white matter volumes in ADHD; Decreased posterior white matter volumes in ADHD stimulant non-responders
Hynd et al., 1991	7/10	Structural	Smaller corpus collosum in ADHD
Semrud-Clikeman et al., 1994	15/15	Structural	Smaller posterior corpus collosum (splenium) in ADHD
Castellanos et al., 2000	50/50	Structural	Smaller cerebellum volume correlated with greater ADHD symptomotology
Berquin et al., 1998	46/47	Structural	Smaller posterior inferior cerebellar vermal volume in ADHD
Schweitzer et al., 2000	6/6	PET	Men with ADHD showed widespread activation and activation in occipital area, whereas men without ADHD showed activation in the frontal and temporal regions during a response inhibition task
Rubia et al., 1999	7/9	Functional MRI	ADHD subjects showed lower activation in the right mesial prefrontal cortex, right inferior prefrontal cortex and left caudate during a motor inhibition task.

Appendix C: Institutional Review Board Approval

OFFICE OF RESEARCH SUPPORT

THE UNIVERSITY OF TEXAS AT AUSTIN

P.O. Box 7426, Austin, Texas 78713 (512) 471-8871 - FAX (512) 471-8873
North Office Building A, Suite 5.200 (Mail code A3200)

Title: Attention in Children and Adolescents with Nonverbal Learning Disabilities
APPROVAL – IRB Protocol # 2007-11-0127

Dear: Tim and Brianne

FWA # 00002030

Date: 01/27/2009

PI(s): Keith, Butcher Department & Mail Code: Educational Psychology

In accordance with Federal Regulations for review of research protocols, the research study listed above has been re-approved for the following period of time: **01/27/2009-01/26/2012**

RESPONSIBILITIES OF PRINCIPAL INVESTIGATOR FOR ONGOING PROTOCOLS:

(1) Report **immediately** to the IRB any unanticipated problems.

(2) **File an amendment application for changes to this project that will involve increased risk to participants.** Such changes cannot be initiated without IRB review and approval. Changes to the protocol that will not raise the level of risk to participants may be initiated without filing an amendment application for IRB review. For a description of the types of modifications that DO require an amendment application, please refer to the ORS webpage: http://www.utexas.edu/research/rsc/humansubjects/policies/section6.html#635b

(3) Report any significant findings that become known in the course of the research that might affect the willingness of subjects to continue to take part.

(4) Use **only** a currently approved consent form.

(5) Follow the approved protocol in regard to the privacy and confidentiality of all persons and identifiable data and train your staff and collaborators on policies and procedures for ensuring privacy and confidentiality.

(6) Submit a **continuing review application** prior to the approval end date if you wish to extend the approval period. Please note that data collection is not allowed beyond the approval cessation date.

(7) Notify the IRB when the study has been completed and complete a **closure report** form.

Thank you for your assistance in this matter. Please include the above protocol number on all future correspondence relating to this protocol.

Sincerely,

Jody L. Jensen, Ph.D.
Professor
Chair, Institutional Review Board

Appendix D: Developmental and Family History Form

FAMILY DATA
(To be filled out by parents)

Date: ____________________

Child's Name: ________________________________ Adopted? _____ Birthdate: __________ Age ___
Home Address: ________________________________ Home Phone: ______________
School: ____________________ Grade: ________ School Phone: ______________
Father's Name: ____________________ Age: ________ Education: ______________
Employment: ____
Mother's Name: ____________________ Age: ________ Education: ______________
Employment: ____
Other Children in the Home:
Name & Age: ____________________ Name & Age: ____________________
Name & Age: ____________________ Name & Age: ____________________
Other relatives or persons living in the home:

____

____

UNDERLINE THE APPROPRIATE ITEMS BELOW:

(Our, My) reason for bringing child in today is (routine physical check-up, physical problems, speech problems, poor school work, behavior problems, not doing well at home or school).

Problem has been going on for (weeks, months, year or more).

FAMILY (underline correct answers)

Child lives with (both his own parents, step-father, step-mother)

(Father, mother, neither, both) had similar troubles.

Child (disrupts, gets along with) family.

Child has mostly been a source of (pride, worry, friction) for family.

Parents (agree, disagree) on how to discipline child.

Discipline has been (strict, lenient, inconsistent, all of these).

Marital troubles are (mild, moderate, severe, none).

Parents have problems of (alcoholism, chronic disease, mental illness, none).

Other children in the home have problems with (school behavior, grades, illness, emotional adjustment, none).

PREGNANCY HISTORY - Mother

While you were pregnant with this child, were you under a doctor's care? YES___ NO___

During this pregnancy did you have:	Yes	No	When	Describe
Anemia				
Elevated blood pressure				
Toxemia				
Swollen ankles				
Kidney disease				
Heart disease				
Bleeding				
Measles				
German measles				
Flu				
Other virus				
Other illness				
Vomiting				
Injury				
Medication during pregnancy				
Threatened miscarriage or early contractions				
Alcohol consumption			Amount:	
Drug abuse			Type/Amount:	
Smoking			Amount:	

BIRTH HISTORY

How many hours from first contractions to birth?___

Were you given medications? YES___ NO___ What kind? ___

Did you have natural childbirth? YES___ NO___

Were you under anesthesia during childbirth? YES___ NO___ Don't know___

Was labor induced? YES___ NO___ Was induced labor planned? YES___ NO___

Was this a breech (feet first) delivery? YES___ NO___

Was the delivery unusual in any way? YES___(How?___ NO ___

Did you have a cesarean? YES___ NO___Complications?___

Name any medical problems.___

Duration of pregnancy. ___ What did the baby weigh?___

Did you have twins? YES___ NO___ Which born first?___

Did this baby have: breathing problems? YES___ NO___ Don't know___
cord around neck? YES___ NO___ Don't know___

Did this baby cry quickly? YES___ NO___ Don't know___

Was the baby's color normal? YES___ NO___ Blue?___Yellow?___Don't know___

Was oxygen used for the baby? YES___ NO___ If so, how much?___

Did you take the baby home with you from the hospital? YES___ NO___ Describe:___

Did you have problems with feeding? YES___ NO___ Describe:___

Was the baby normally active? YES___ NO___Describe:___

DEVELOPMENTAL HISTORY* (As best you remember)

Age held head up____________, age turned over____________, age smiled at parents____________,
age crawled____________, age sit____________, age pull up at crib____________,
age walk with help____________, age walk alone____________, bottle fed?____________,
breast?____________, age weaned____________, age say 4-10 words____________,
age use sentences____________, speech problems?____________, did he/she hold out arms and want to picked up?____________, age say "No, no" to everything____________,
shy or timid____________, liked attention?____________, friendly baby?____________,
affectionate?____________, wanted to be left alone____________, more interested in things than in people?____________, stubborn?____________, ate well?____________,
feed self, age____________, temper tantrums?____________, breath holding?____________,
tears up toys more than normal?____________, much too active?____________,
bowel trained, age____________, dry at what age?____________, age helped with dressing____________,
age dressed alone____________, right or left handed____________, age this settled?____________,
well coordinated?____________, clumsy?____________, good with hands?____________,
blank spells?____________, falling spells?____________, dare-devil behavior?____________,
impulsiveness?____________, unusual fears?____________, sleep problems?____________,
rocking?____________, head bumping?____________.

SCHOOL HISTORY (underline correct answers)

According to school the child's I.Q. is (average, below average, above average, I don't know).

Since first grade, school personnel have reported (no serious problems, problems with behavior, speech, reading, writing, spelling, math).

Child has (been in Special Education Class, failed a grade, been tutored, made satisfactory grades).

School personnel have reported through the years that the child (adjusts to other children, doesn't adjust).

Child (likes school, hates school, is indifferent).

* - If additional space is needed to answer any questions, use above space.

MEDICAL HISTORY OF CHILD

Has your child had:	Yes	No	Age	Describe
Measles				
German measles				
Chicken pox				
Whooping cough				
Diphtheria				
Flu				
Meningitis				
Encephalitis				
High fever				
Abscessed ears				
Allergy				
Convulsions				
Injuries to head				
Other injuries				
Other illnesses				
Hospitalizations				
Operations				

FAMILY HISTORY

Is there family history of the following:	Yes	No	If yes, what relation
Seizures			
Cerebral Palsy			
Mental Retardation			
Psychiatric or behavior problems			
Movement disorder			
Headaches			
Learning disability			
Hyperactivity			
Tumors			
Other neurologic conditions			

Signature of Parent(s), Guardian, etc.______________________________

Appendix E: Consent for Research

Austin Neuropsychology, PLLC

Patient Name:______________________________ Patient #_______________

Research Participation: In our effort to learn more about how people function and how to help our patients, we sometimes carry out research studies using past test data. In this research, ***all identifying information is removed and confidentiality is protected.*** Also, occasionally we contact past patients to see if they are interested in participating in new research studies. Your decision to participate or decline to participate in research will in no way affect your relationship with Austin Neuropsychology or our commitment to your clinical care. If you agree to research participation, please indicate your consent by initialing below:

I _____ give (or) _____ do NOT give permission for you to contact me about research participation.

I _____ give (or) _____do NOT give permission for you to use the patient's testing data for research / teaching purposes.

____________________________________ ____________________

(Signature of Patient or Parent/Legal Guardian) Date

____

Name of Parent /Legal Guardian (if applicable)

Email address:____

For Office Use Only

Clinic Number ____________________

UT_______

Appendix F:

ATTENTION CONSTRUCT(S) MEASURED BY THE NEUROPSYCHOLOGICAL TESTS

Neuropsychological Test	Attention Construct(s) Measured
WISC-IV Digit Span Backward	Verbal working memory
WISC-IV Letter/Number Sequencing	Verbal working memory
Trails B	Visual working memory and processing speed
WISC-IV Coding	Visual processing speed
CPT-Variability	Vigilance
CPT-Commissions	Inhibition

References

Achenbach, T. M., & Rescorla, L. A. (2001). Manual for ASEBA School-Age Forms & Profiles. Burlington, VT: University of Vermont, Research Center for Children, Youth, & Families.

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

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

American Psychological Association (2002). *Ethical Principles of Psychologists and Code of Conduct.* Washington, D.C.: American Psychological Association.

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(6)*, 775-789.

Barkley, R.A. (1997). *ADHD and the Nature of Self Control.* Guilford Press: New York.

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. (1998). *Attention Deficit Hyperactivity Disorder: A Handbook for Diagnosis and Treatment.* New York: Guildford.

Barkley, R. A. (2003). Attention-Deficit/Hyperactivity Disorder. In E. J. Mash & R. A. Barkley (Eds.) *Child Psychopathology, 2nd edition* (pp. 75-143). New York: The Guilford Press.

Barkley, R.A., Edwards, G., Laneri, M., Fletcher, K., & Metevia, L. (2001). Executive functioning, temporal discounting, and sense of time in adolescents with attention deficit hyperactivity disorder (ADHD) and oppositional defiant disorder (ODD). *Journal of Abnormal Child Psychology, 29*(6), 541-556.

Beery, K. E., & Buktenica, N. A. (1996). *Beery-Buktenica Developmental Test of Visual Motor Integration,* (4th Ed.). Wilmington, DE: Wide Range.

Berger, A. & Posner, M.I. (2000). Pathologies of brain attentional networks. *Neuroscience and Biobehavioral Reviews, 24,* 3-5

Beiderman, J. (2005). Attention-Deficit/Hyperactivity Disorder: A Selective Overview. *Biological Psychiatry, 57,* 1215-1220.

Beiderman J., Faraone S.V., Keenan K., Benjamin J., Krifcher B., & Moore C. (1992). Further evidence for family-genetic risk factors in attention deficit hyperactivity disorder. Patterns of comorbidity in probands and relatives in psychiatrically and pediatrically referred samples. *Archives of General Psychiatry*, *49,* 728-738.

Berquin, P. C., Giedd, J. N., & Jacobsen, L. K. (1998). Cerebellum in attention-deficit hyperactivity disorder: A morphometric MRI study. *Neurology 50(4),*1087-1093.

Bierman, K. L., & Welsh, J.A. (2000). Assessing Social Dysfunction: The Contributions of Laboratory and Performance-Based Measures. *Journal of Clinical Child Psychology, 29(4),* 526-539.

Brown, T. E. (2000). Emerging understandings of attention-deficit disorders and comorbidities. In T. E. Brown (Ed.), *Attention-deficit disorders and comorbidities in children, adolescents, and adults.* Washington, D.C.: American Psychiatric Press, Inc.

Brown, R. T., Madan-Swain, A., & Baldwin, K. (1991). Gender differences in a clinic-referred sample of attention-deficit-disordered children. *Child Psychiatry and Human Development, 22(2),* 111-128.

Carlson, C.L., Shin, M., & Booth, J. (1999). The Case for DSM-IV Subtypes in ADHD. *Mental Retardation and Developmental Disabilities Research Reviews, 5,* 199-206.

Casey, B.J., Castellanos, X.F., Giedd, J.N., Marsh, W.L., Hamburger, S.D., Schubert, A.B., et al. (1997). Implication of right frontostriatal circuitry in response inhibition and attention-deficit/hyperactivity disorder. *American Academy of Child and Adolescent Psychiatry, 36***,** 374–383.

Castellanos, F.X., Giedd, J.N., Marsh, W.L., Hamburger, S.D., Vaituzis, A.C., Dickstein, D.P., et al. (1996). Quantitative brain magnetic resonance imaging in attention-deficit hyperactivity disorder. *Archives of General Psychiatry*, *53*(7), 607–616.

Castellanos, F.X., Marvasti, F.F., Ducharme, J.L., Walter, J.M., Israel, M.E., Krain, A., et al. (2000). Executive function oculomotor tasks in girls with ADHD. *Journal of the American Academy of Child and Adolescent Psychiatry, 39,* 644–650.

Chhabildas, N., Pennington, B.F., Willicut, E.G. (2001). A Comparison of Neuropsychological Profiles of the DSM-IV Subtypes of ADHD. *Journal of Abnormal Child Psychology, 29,* 529-540.

Collett, B.R., Ohan, J.L, & Myers, K.M. (2003). Ten-Year Review of Rating Scales Assessing Attention-Deficit/Hyperactivity Disorder. *Journal of the American Academy of Child and Adolescent Psychiatry, 42*(9), 1015-1037.

Conners, C.K., & MHS Staff. (2000). *Conners' continuous performance test (CPT-II) computer program for Windows technical guide and software manual.* North Tonawanda, NY: Multi-Health Systems, Inc.

Cornoldi, C., Rigoni, R., Tressoldi, P. E., & Vio, C. (1999). Imagery deficits in nonverbal learning disabilities. *Journal of Learning Disabilities, 32,* 18–57.

Darwin. C. (1872). *The Expression of the Emotions in Man and Animals.* In Darwin, C. (1998) 3rd ed.with Introduction, Afterword and Commentary by Paul Ekman: London: Harper Collins; New York: Oxford University Press.

DeLuca, J.W., Moore, G.J., Mueller, R.A., Slovis, T.L., and Chugani, H.T. (1997). Right Hemisphere learning disability syndrome: differential findings from PET and MRS. *Journal of Neuropsychiatry and Clinical Neurosciences., 9(1),* 131.

Denckla, M.B. (2000). Learning Disabilities and Attention-Deficit/Hyperactivity Disorder in Adults: Overlap with Executive Dysfunction. In T. E. Brown (Ed.), *Attention-deficit disorders and comorbidities in children, adolescents, and adults.* Washington, D.C.: American Psychiatric Press, Inc.

Denman, S. (1984). *Denman Neuropsychology Memory Scale Manual*. Charleston, S.C.

Diamond, A. (2005). Attention-deficit disorder (attention-deficit/hyperactivity disorder without hyperactivity): A neurobiologically distinct disorder from attention-deficit/hyperactivity disorder (with hyperactivity). *Development and Psychopathology, 17,* 807-825.

Dimitrovsky, L., Spector, H., Levy-Shiff, R. & Vakil, E. (1998). Interpretation of facial expressions of affect in children with learning disabilities with verbal or nonverbal deficits. *Journal of Learning Disabilities, 31,* 286-292.

Duke, M & Norwicki, S. (2005) The Emory Dyssemia Index. In Manusov, V. (Ed) Beyond Words: A Sourcebook of Methods for Measuring nonverbal Cues . New York: Erlbaum Associates. pp 35-46.

Dunn, D.W. & Roos, K.L. (1989). MRI evaluation of learning difficulties and in-coordination in children with neurofibromatosis type1. *Neurofibromatosis, 2,* 1-5.

Ellis, H.D. & Gunter, H.L. (1999). Asperger Syndrome: a simple matter of white matter? *Trends in Cognitive Science, 3(5),* 192-200.

Farah, M. (2003). Disorders of visual-spatial perception and cognition. In K.M Heilman & E. Valenstien (Eds.), *Clinical Neuropsychology, 4th Edition,* New York: Oxford Press.

Faraone S.V.,Sergeant J., Gillberg C., & Biederman J. (2003).The worldwide prevalence of ADHD: is it an American condition? *World Psychiatry, 2*,104–113.

Field, Andy. (2005). *Discovering Statistics Using SPSS, Second Edition.* Thousand Oaks, California: Sage Publications.

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

Frith, U. & Frith, C. (2004). Development and neurophysiology of mentalizing. In C. Frith & D. Wolpert (Eds.), *The neuroscience of social interaction: decoding, imitating, and influencing the actions of others.* New York: Oxford University Press.

Fuerst, D.R., Fisk, J.L., & Rourke, B.P. (1990). Psychosocial functioning of learning disabled children: Relations between WISC Verbal IQ-Performance IQ discrepancies and personality subtypes. *Journal of Consulting and Clinical Psychology, 58,* 657-660.

Fuerst, D.R., & Rourke, B.P.(1993). Psychosocial functioning of children: Relations between personality subtypes and academic achievement. *Journal of Abnormal Child Pscyhology, 21,* 597-608.

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

Gazzaniga, M.S. (2000). Cerebral specialization and interhemispheric communication: Does the corpus callosum enable the human condition? *Brain, 123(*7), 1293-1326.

Gerrard-Morris. A.E.(2006). *Internalized Socioemotional Functioning of Children and Adolescents with Nonverbal Learning Disability and Asperger's Syndrome*. Unpublished doctoral dissertation, University of Texas, Austin.

Giedd, J.N., Blumenthal, J., Molloy, E., & Castellanos, F.X. (2001) Brain Imaging of Attention Deficit/Hyperactivity Disorder. *Annals of the New York Academy of Sciences, 931* (1), 33–49.

Gilliam, J. (2001) *Gilliam Asperger's Disorder Scale Examiner's Manual*. Austin, Tx: Proed Publishing.

Goldberg, E., & Costa, L.D. (1981). Hemisphere differences in the acquisition and use of descriptive systems. *Brain and Language, 14,* 144-173.

Gross-Tsur, V., Shalev, R. S., Manor, O., & Amir, N. (1995). Developmental Right Hemisphere Syndrome: Clinical Spectrum of the Nonverbal Learning Disability. *Journal of Learning Disabilities, 28(2),* 80-87.

Gunter, H.L., Ghaziuddin, M., & Ellis, H.D. (2002). Asperger Syndrome: Tests of Right Hemisphere Functioning and Interhemispheric Communication. *Journal of Autism and Developmental Disorders, 32(4),* 263-281.

Hale, J.B., Hoeppner, J.B., & Fiorello, C.A. (2002). Analyzing digit span components for assessment of attention processes. *Journal of Psychoeducational Assessment*, 20, 128-143.

Hammill, D.D., Leigh, J.E., McNutt, G., Larsen, S.C. (1981). A New Definition of Learning Disabilities. *Learning Disability Quarterly*, *4 (4)*, 336-342.

Harnadek, M.C.S., & Rourke, B.P. (1994). Principal identifying features of the syndrome of nonverbal learning disabilities in children. *Journal of Learning Disabilities, 27*(3), 144-154.

Heilman, K.M., Blonder, L.X., Bowers, D., & Valenstein, E. (2003). In K.M Heilman & E. Valenstien (Eds.), *Clinical Neuropsychology, 4th Edition,* (pp. 447-478). New York: Oxford Press.

Heilman, K.M., Watson, R.T., & Valenstein, E. (2003). Neglect and Related Disorders. In K.M Heilman & E. Valenstien (Eds.), *Clinical Neuropsychology, 4th Edition,* (pp. 296-346). New York: Oxford Press.

Hinshaw, S.P., & Zalecki, C. (2001). Attention-Deficit Hyperactivity Disorder. In H. Orvaschel, J. Faust, & M. Hersen (Eds.) *Handbook of Conceptualization and Treatment of Child Psychopathology* (pp. 77-106). New York: Pergamon.

Hofman, K.J., Harris, E.L., Bryan, R.N., & Denckla, M.B. (1994). Neurofibromatosis type 1: The cognitive phenotype. *Journal of Pediatrics, 124,* S1-S8.

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

Hynd, G.W., Semrud-Clikeman, M., Lorys, A.R., Novery, E.S., & Eliopulos, D. (1991). Corpus callosum morphology in attention deficit-hyperactivity disorder: Morphometric analysis of MRI. *Journal of Learning Disabilities, 24*(3), 141-146.

James, W. (1890). *The Principles of Psychology.* New York: Henry Holt, pp. 403-404.

Johnson, D.J. (1987). Nonverbal learning disabilities. *Pediatric Annals, 16*(2), 133-141.

Johnson, D. J., & Myklebust, H. R. (1971). *Learning disabilities. New* York: Grune & Stratton.

Klin, A., Volkmar, F. R., Sparrow, S. S., Cicchetti, D. V., & Rourke, B. P. (1995). Validity and neuropsychological characterization of Asperger syndrome: Convergence with Nonverbal Learning Disabilities syndrome. *Journal of Child Psychology and Psychiatry, 36,* 1127-1140.

Knights, R.M. & Moule, A.D. (1968). Normative data on the motor steadiness battery for children. *Perceptual and Motor Skills, 26,* 643-650.

Kolb, B. & Fantie, B. (1989). Development of the child's brain and behavior. In C.R. Reynolds & E.F. Janzen (Eds.), *Handbook of clinical child neuropsychology* (3rd ed.). San Francisco: Freeman.

Lahey, B., Applegate, B., McBurnett, K., Biederman, J., Greenhill, L., Hynd, G., et al. (1994). DMS-IV field trials for attention deficit hyperactivity disorder in children and adolescents. *American Journal of Psychiatry,* 151, 1673-1685.

Lahey B.B., Pelham W.E., & Stein M.A.(1998) Validity of attention-deficit/hyperactivity disorder for younger children. *Journal of the American Academy for Child and Adolescent Psychiatry, 37*, 695–702.

Landau, Y.E., Auerback, J.G., Gross-Tsur, V., Shalev, R.S. (2003). Speed of Performance of Children with Developmental Right Hemisphere Syndrome and with Attention-Deficit Hyperactivity Disorder. *Journal of Child Neurology, 18,*264-268.

Landau, Y.E., Gross-Tsur, V., Auerbach, J.G., Van der Meere, J., & Shalev, R.S., (1999). Attention-Deficit Hyperactivity Disorder and Developmental Right-Hemisphere Syndrome: Congruence and incongruence of cognitive and behavioral aspects of attention. *Journal of Child Neurology, 14,* 299-303.

Leguis, E. , Descheemaeker, M.J., Steyaert, J., Spaepen, A., Vlietinck, R., Casaer, P., Demaerel, P., & Fryns, J.P. (1995). Neurofibromatosis type 1 in childhood: Correlation of MRI finding with intelligence. *Journal of Neurology, Neurosurgery, and Psychiatry, 59,* 638-640.

Liddell, G.A. & Rasmussen, C. (2005). Memory Profile of Children with Nonverbal Learning Disability. *Learning Disabilities Research & Practice, 20*(3), 137-141.

Lockwood, K.A., Marcotte, A.C., & Stern, C. (2001). Differentiation of Attention-Deficit/Hyperactivity Disorder Subtypes: Application of a Neuropsychological

Model of Attention. *Journal of Clinical and Experimental Neuropsychology, 23*, 317-330.

Lunneborg, C. E. & Lunneborg, P. W. (1978). Improved counseling information through rotation of discriminant functions. *Educational and Psychological Measurement, 38*, 737-753.

McGough, J.J. & McCracken, J.T. (2006) Adult Attention Deficit Hyperactivity Disorder: Moving Beyond DSM-IV. *American Journal of* Psychiatry*, 163*, 1673-1675.

Meere J.J., Stemerdink N., Gunning B.(1995). Effects of presentation rate of stimuli on response inhibition in ADHD Children with and without tics. *Perceptual and Motor Skills*, *81*,259–62.

Mertler, C.A. & Vannatta, R.A. (2005). *Advanced and Multivariate Statistical Methods, Third Edition.* (pp. 253-310). Glendale, CA: Pyrczak Publishing.

Milich, R., Balentine, A. C., & Lyman, D. R. (2001). ADHD combined type and ADHD predominantly inattentive type are distinct and unrelated disorders. *Clinical Psychology: Science and Practice*, *8(4)*, 463-488.

MTA Cooperative Group. (1999). A 14-month randomized clinical trial of treatment strategies for attention-deficit/hyperactivity disorder. *Archives of General Psychiatry, 56*, 1073-1086.

Myklebust, H.R. (1975). Nonverbal Learning Disabilities.: Assessment and intervention. In H.R. Myklebust (Ed.), *Progress in learning disabilities.* (pp. 85-121). New York: Grune & Stratton.

Nigg, J.T. (2001). Is ADHD a Disinhibitory Disorder? *Psychological Bulletin, 127,* 571-598.

Nigg J.T., Blaskey L., Huang-Pollock C., Rappley M.D. (2002). Neuropsychological executive functions and ADHD DSM-IV subtypes. *Journal of the American Academy of Child and Adolescent Psychiatry, 41*,59–66.

Nowicki, S. Jr., & Duke, M. P. (1994). Individual differences in the nonverbal communication of affect: The Diagnostic Analysis of Nonverbal Accuracy Scale. Special Issue: Development of nonverbal behavior: II. Social development and nonverbal behavior. *Journal of Nonverbal Behavior*, *18*, 9–35.

Pennington, B.F. (1991). Right hemisphere learning disorders. In *Diagnosing learning disabilities: A neuropsychological framework* ((pp.111-134). New York: Guilford Press.

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

Pelletier, P. M., Ahmad S. A., & Rourke, B. P. (2001). Classification rules for basic phonological processing disabilities and nonverbal learning disabilities: formulation and external validity. *Child Neuropsychology, 7*(2), 84-98.

Petti, V.L., Voelker, S.L., Shore, D.L., & Hayman-Abello, S.E. (2003). Perception of Nonverbal Emotion Cues by Children With Nonverbal Learning Disabilities. *Journal of Developmental and Physical Disabilities, 15, 23-36.*

Pliszka, S. R. (1991). Attention-deficit hyperactivity disorder: a clinical review. *American Family Physician, 43(4)*, 1267-1276.

Pliszka, S. R., Carlson, C. L., & Swanson, J. M. (1999). *ADHD with comorbid disorders:*

Clinical assessment and management. New York: The Guilford Press.

Porter, J., & Rourke, B.P. (1985). Socio-emotional functioning of learning disabled children: A subtypal analysis of personality patterns. In B.P. Rourke (Ed.), *Neuropsychology of learning disabilities: Essentials of subtype analysis* (pp. 257-280). New York: Guilford Press.

Posner, M. (1994). Attention: The mechanisms of consciousness. *Proceedings of the National Academy of Science, 91,* 7398–7403.

Roman, M. A. (1998). The Syndrome of Nonverbal Learning Disabilities: Clinical Description and Applied Aspects. *Current Issues in Education* [On-line], *1*, *(7)*.

Rothenberg, S. (1998). Nonverbal learning disabilities and social functioning. From NLD on the Web. www.nldontheweb.org/Rothenberg-1.htm.

Rothman, A. D., Nowicki Jr., S. (2004)A Measure of the Ability to Identify Emotion in Children's Tone of Voice. *Journal of Nonverbal Behavior*, *28(2)*, 67-92.

Rourke, B. P. (1989). *Nonverbal learning disabilities: The syndrome and the model.* New York: Guilford Press.

Rourke, B. P. (1995). The NLD syndrome and the white matter model. In B. P. Rourke (Ed.), *Syndrome of nonverbal learning disabilities: Neurodevelopmental manifestations,* (pp. 1-26). New York: Guilford.

Rourke, B.P. (1999). Neuropsychological and Psychosocial Subtyping: A Review of Investigations within the University of Windsor Laboratory. *Canadian Psychology/Psychologic Canadienne, 41(1)*, 34-51.

Rourke, B. P., Ahmad, S. A., Collins, D. W., Hayman-Abello, B. A., & Warriner, E. M. (2002). Child clinical/pediatric neuropsychology: Some recent advances. *Annual*

Review of Psychology, 53, 309-339.

Rourke, B. P, & Fisk, J. L. (1981). Socio-emotional disturbances of learning disabled children: The role of central processing deficits. *Bulletin of the Orton Society, 31,* 77-88.

Rourke, B. E, & Strang, J. D. (1978). Neuropsychological significance of variations in patterns of academic performance: Motor, psychomotor, and tactile perception abilities. *Journal of Pediatric Psychology, 3*, 62-66.

Rubia, K., Overmeyer, S., Taylor, E., Brammer, M., Williams, S. C., Simmons, A., & Bullmore, E. T. (1999). Hypofrontality in attention deficit hyperactivity disorder during higher-order motor control: a study with functional MRI. *American Journal of Psychiatry*, *156*(6), 891–896.

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

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

Sergeant, J. (2000). The cognitive-energetic model: an empirical approach to Attention-Deficit Hyperactivity Disorder. *Neuroscience & Biobehavioral Reviews, 24,* 7-12.

Semrud-Clikeman, M. (2005). Neuropsychological Aspects for Evaluating Learning Disabilities. *Communication Disorders Quarterly, 26*(4), 242-247.

Semrud-Clikeman, M., Biederman, J., Sprinch-Buckminster, S., Lehman, B.K., Faraone, S.V., & Norman, D. (1992). Comorbidity between ADHD and Learning Disability: A review and report in a clinically referred sample. *Journal of the American Academy of Child and Adolescent Psychiatry, 31*(3), 439-448.

Semrud-Clikeman, M., Filipek, P.A., Biederman, J., Steingard, R., Kennedy, D., Renshaw, P., & Bekken, K. (1994). Attention-deficit hyperactivity disorder: Magnetic resonance imaging morphometric analysis of the corpus callosum. *Journal of the American Academy of Child & Adolescent Psychiatry, 33*(6), 875-881.

Semrud-Clikeman, M., & Hynd, G. W. (1990). Right hemispheric dysfunction in nonverbal learning disabilities: social, academic, and adaptive functioning in adults and children. *Psychological Bulletin, 107*(2), 196-209.

Semrud-Clikeman, M. & Pliszka, S.R. (2005). Neuroimaging and Psychopharmacology. *School Psychology Quarterly, 20,* 176-186.

Shaywitz, S.E., & Shaywitz, B.E. (1988). Attention deficit disorder: Current perspectives. In J.F. Kavanaugh & T.J. Truss (Eds.), *Learning disabilities: Proceedings of the national conference* (pp.369-523). Parkton, MD: York Press.

Staller, J. Faraone, S.V. (2006). Attention-Deficit Hyperactivity Disorder in Girls. *CNS Drugs, 20*(2), 107-123.

Stefanatos, G.A. and Wasserstein, J. (2001) Attention Deficit Hyperactivity Disorder as a right hemisphere syndrome: Selective review and detailed neuropsychological case studies. *Annals of the New York Academy of Sciences, 931*, 172-195.

Straus, E., Sherman, E.M., & Spreen, O. (2006). *A Compendium of Neuropsychological Tests, Third Edition.* New York: Oxford University Press.

Swanson, J. (1992). *School Based Assessments and Interventions for ADD Students*. Irvine, CA: KC.

Tannock, R. & Brown, T.E. (2000). Attention-Deficit Disorders with Learning Disorders in Children and Adolescents. In T. E. Brown (Ed.), *Attention-deficit disorders and comorbidities in children, adolescents, and adults.* Washington, D.C.: American Psychiatric Press, Inc.

Teeter, P.A., & Semrud-Clikeman, M. (1997). *Child Neuropsychology: Assessment and interventions for neurodevelopmental disorders.* Boston: Allyn & Bacon.

Thapar, A., Hervas A., & McGuffin, P. (1995). Childhood hyperactivity scores are highly heritable and show sibling competition effects: twin study evidence. *Behavioral Genet*ics, *25*, 537–544.

Thompson, S. (1997). Nonverbal learning disorders revisited. Retrieved July, 30, 2005, from http://www.ldonline.org/ld_indepth/general_info/nld_revisited.html

Trites, R. (1989). *Grooved Pegboard Test Manual.* Lafayette, IN: Lafayette Instruments.

Tsatsanis, K.D., & Rourke, B.P. (1995). Conclusions and future directions. In B. P. Rourke (Ed.), *Syndrome of nonverbal learning disabilities: Neurodevelopmental manifestations,* (pp. 476-496). New York: Guilford.

Tupler, L.A., Coffey, C.E., & Logue, P.E. (1992) Neuropsychological importance of subcortical white matter hyperintensity. *Archives of Neurology, 49,* 1248-52.

Valera, E.M., Faraone, S.V., Murray, K.E. & Seidman, L.J. (2007). Meta-Analysis of Structural Imaging Findings in Attention-Deficit/Hyperactivity Disorder. *Biological Psychiatry, 61,* 1361-1369.

Voeller, K.K.S. (1986). Right-hemisphere deficit syndrome in children. *American Journal of Psychiatry, 142*, 1004-1009.

Voeller, K. K. S. (1994). Techniques for measuring social competence in children. In G. R. Lyon (Ed.), *Frames of reference for the assessment of learning disabilities* (pp. 523-554). Baltimore, MD: Paul H. Brookes Publishing Co., Inc.

Wechsler, D. (2003). *Wechsler Intelligence Scale for Children, Fourth Edition: Technical and Interpretive Manual.* San Antonio, Tx: The Psychological Corporation.

White, R.F., & Krengal, M. (1995). Multiple Sclerosis. In B. P. Rourke (Ed.), *Syndrome of nonverbal learning disabilities: Neurodevelopmental manifestations,* (pp. 407-432). New York: Guilford.

Wilkinson, A.D., Schafer, V.A., & Semrud-Clikeman, M. (2002). *Visual-motor and visual-perceptual abilities of children and adolescents with NVLD and ADHD.* Paper presented at the annual convention of the American Psychological Association, Chicago, IL.

Williams, J., & Dykman, R.A. (1994). Nonverbal factors derived from children's performance on neuropsychological test instruments. *Developmental Neuropsychology,* 10, 1, 19-26.

Willicutt, E. G., Doyle, A. E., Nigg, J. T., Faraone, S. V., & Pennington, B. F. (2005). Validity of the executive function theory of attention-deficit/hyperactivity disorder: A meta-analytic review. *Biological Psychiatry, 57,* 1336-1346.

Woodcock, R.W., McGrew, K.S., & Mather, N. (2001). *Woodcock-Johnson III Tests of Achievement.* Itasca, IL: Riverside Publishing.

Woods, S.P., Weinborn, M., Ball, J.D., Tiller-Nevin, S. & Pickett, T.C. (2000). Periventricular leukomalacia (PVL): An identical twin case study illustration of white matter dysfunction and nonverbal learning disability (NLD). *Child Neuropsychology, 6*(4), 274-285.

Worling, D.E., Humphries, T., & Tannock, R. (1999). Spatial and emotional aspects of language inferencing in nonverbal learning disabilities. *Brain and Language, 70,* 220-239.

Vita

Brianne Janeé Butcher was born in Austin, Texas on November 25, 1980 to Regina and Michael Butcher. Brianne graduated from Lake Travis High School in Austin, Texas in May 1999. She attended the University of Texas at Austin, where she graduated with a Bachelor of Arts degree in Biology with college honors in May 2003. Brianne entered the Graduate School of Educational Psychology at the University of Texas at Austin in the fall of 2003, and earned her Master of Arts degree in May 2007. Brianne is currently completing her pre-doctoral internship at Miami Children's Hospital.

Permanent address: 11008 River Plantation Dr., Austin, Tx 78747.

This dissertation was typed by the author.