

NEUROPSYCHOLOGICAL DEFICITS IN TSHIVENDA  
SPEAKING CHILDREN WITH ATTENTION-  
DEFICIT/HYPERACTIVITY DISORDER

by

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## DECLARATION

I, Mudzunga Mathivha, declare that the dissertation hereby submitted to the University of Limpopo as partial fulfilment for the degree of Master of Arts in Clinical Psychology, has not previously been submitted by me for a degree at any other university, that it is my own work in design and execution, and that all the material contained therein has been duly acknowledged.

Signature .....

Date .....

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## ABSTRACT

The aim of this study was to establish whether children with Attention-Deficit/Hyperactivity show deficits in cognitive impulsiveness and motor functions, caused by a hypofunctioning dopamine system. A group of 84 primary school children, 42 classified as ADHD and 42 controls, matched for age, gender and SES, with children without ADHD symptomatology, were compared on their performance on neuropsychological tests which test the functions of the cortical areas supplied by two dopamine branches, the mesocortical and nigrostriatal branches. The battery consisted of the Wisconsin Card Sorting Test, the Grooved Pegboard, and the Maze Coordination Task. The results of the tests were analysed as a function of gender and ADHD-subtypes. In the majority of tests the clinical groups performed worse than the control groups. This was the case for both genders. The Hyperactive/Impulsive and Combined subtypes consistently performed poorer than the other groups. The results indicate that children with ADHD are more impulsive (deficient executive functions) and have poorer motor control than their control counterparts, which may be an indication of dopamine dysfunction.

## *Chapter 1*

### INTRODUCTION

#### **1.1 Introduction**

Attention-Deficit/Hyperactivity disorder (ADHD) is a psychiatric disorder characterized by difficulties sustaining attention and difficulties with impulse control. Evidence suggests that genetic factors are important in its pathogenesis (Barkley, 1998; Swanson et al., 1998c) and that its pathophysiology involves alterations in central dopaminergic and noradrenergic systems (Swanson & Castellanos, 1998).

The disorder begins early in life and has been studied primarily in children, among whom its prevalence is 3-7% (American Psychiatric Association, 2000). As children mature, symptoms can become less problematic, most likely through the developmental changes of the brain, by learning to compensate for deficits with adaptive behaviour, however, the disorder often persists into adulthood (Biederman, Faraone, & Lapey, 1992) and can cause impairments in all aspects of life (Barkley, 1997a).

#### **1.2 Background**

Attention-Deficit/Hyperactivity Disorder (ADHD) is a persistent pattern of inattention and/or hyperactivity-impulsiveness that is more severe than is typically observed in individuals at a comparable level of development (American

Psychiatric Association, 2000). ADHD children present with significant problems of impulse control, Inattention and overactivity that is not consistent with the child's developmental level. This disorder is associated with a number of complicating features, including neurodevelopmental impairments, impaired intellectual development, scholastic and vocational underachievement and impaired social adjustment (Barkley, 1997a).

According to the DSM IV (American Psychiatric Association, 2000), most children with ADHD are likely to have other cognitive, developmental, emotional academic and medical difficulties. About one third of children diagnosed as having ADHD in childhood will still meet the criteria in adulthood, and 20% of those ADHD diagnosed children are observed as having antisocial personality disorders in adulthood (Swanson et al., 1998c). ADHD is a major risk factor for later delinquency, substance abuse, and other psychiatric disorders (Taylor, 1998b). It therefore, constitutes one of the strongest risk factors known for mental illness in early adulthood.

Researchers stress a high genetic contribution, which gives rise to dopamine hypofunctioning that may be the cause of the behavioural functioning. Other neurobiological imbalances may also be involved (Castellanos, 1997a; Johansen, Aase, Meyer, & Sagvolden, 2002; Sagvolden & Sergeant, 1998; Swanson et al., 1998d) This will be discussed in details in subsequent chapters.

### **1.3 Objectives of the study**

Current instruments used for the diagnosis of ADHD are usually culturally biased and have to be translated in many languages.

The aim of the study is to establish whether children with ADHD are having executive function- and motor deficits caused by a hypofunctioning dopamine system. Tests devised for deficiencies in the areas supplied by the three-dopaminergic branches should be able to show these deficits. The objective of the study therefore, is to submit the neuropsychological theory of ADHD as postulated by Sagvolden et al. (Johansen et al., 2002; Sagvolden, 1999; Sagvolden & Sergeant, 1998) to systematic and experimental testing among Venda primary school children, classified as having ADHD. Hopefully, this may lead to the development of a culture-free test battery sensitive to impulsiveness, inattention and motor deficiencies.

### **1.4 Delineation of the study**

Chapter 2 will focus on the nature of ADHD, its symptoms, the diagnostic criteria, the prevalence, gender differences, aetiologies and treatment. Chapter 3 will deal with the neurobiology of ADHD and will also describe a neuropsychological model of the disorder. Assessment methods, which include screening, interviews and tests, will be described in Chapter 4. Chapter 5 will discuss the problem statement and the research hypotheses. Chapter 6 will describe the methodology used in this study. The research results will be

presented in Chapter 7 and Chapter 8 will discuss the results obtained, limitations of the study, possibilities for further research and suggestions for clinical applications.

## *Chapter 2*

### ATTENTION DEFICIT/ HYPERACTIVITY DISORDER

#### **2.1 Historical background**

Attention-Deficit/Hyperactivity Disorder (ADHD) has known a variety of names during the 20<sup>th</sup> century. Some of these include Encephalitis Lethargica (sequelae thereof), Minimal Brain Damage, Minimal Cerebral Palsy, Mild Retardation, Minimal Brain Dysfunction, Hyperkinesis, Atypical Ego Development, Attention Deficit Disorder (ADD) and Attention-Deficit/Hyperactivity Disorder as it is called today (Ravalovich, 2001).

Strauss and Lehtinen (1947) termed it ‘minimal brain damage dysfunction’ (MBD) syndrome. This was followed by ‘hyperkinetic impulsive disorder’, with symptoms of hyperactivity, short attention span, variability, impulsiveness, irritability, explosive anger fits, and poor school work (Laufer & Denhoff, 1957). Clements (1966) re-introduced the MBD terminology, broadly defined to include specific learning deficits, perceptual-motor deficits, general coordination deficits, hyperkinesis, impulsiveness, emotional liability, short attention span, and/or distractibility, and equivocal neurological signs.

The first clear systematic accounts of hyperactivity are attributed to Sir George Frederic Still, a paediatrician, and the first professor of children’s diseases at King’s College Hospital, London. In 1902, he presented the Cloustonian

lectures to the Royal College of Physicians describing the case histories of 20 children, whose presentation was similar to what at present would be called hyperactivity (Still, 1902). In his descriptions, features such as extreme restlessness and almost choreic movements were frequently mentioned. Another common character was that of ‘an abnormal incapacity for sustained attention’ causing school failure even despite normal intellect. In this behaviour, many of the children were mischievous, destructive and violent, and appeared not to respond to punishment. According to Still, this pattern occurred more often in boys than in girls. Still was of the opinion that this behaviour was not the result of poor parenting or moral weakness, but had a biological cause – either inherited or caused by perinatal injury (Still, 1902).

The disorder was first listed in the second edition of the Diagnostic and Statistical Manual of Mental Disorders, DSM-II (American Psychiatric Association, 1968) as hyperkinetic reaction of childhood, emphasizing inattention, impulsiveness and motor activity. DSM III (American Psychiatric Association, 1980) renamed it ‘Attention Deficit Disorder’ (ADD), shifting emphasis from motor activity to attention, with two types: with hyperactivity (ADHD) and without hyperactivity (ADDWO). In the DSM III-R (American Psychiatric Association, 1987) it was renamed ‘Attention Deficit Hyperactivity Disorder’ (ADHD), and considered undimensional in nature, with a single diagnostic checklist. A second category, ‘Undifferentiated Attention Deficit

Disorder' (U-ADD), featured marked inattention without specific diagnostic criteria. Subsequent studies suggested that the disorder was multidimensional, characterized by inattention and hyperactivity/impulsiveness (Barkley, 1997a). In the DSM-IV (American Psychiatric Association, 1994), the criteria changed to reflect these findings, with 3 main types: Attention-Deficit/Hyperactivity Disorder, Predominantly Inattentive Type; Attention-Deficit/Hyperactivity Disorder, predominantly Hyperactive-Impulsive Type, and Attention-Deficit/Hyperactivity Disorder Combined Type. It remained unchanged in the DSM-IV-TR (American Psychiatric Association, 2000)

The advances made in neuroimaging (especially in the 1990's) have played a great role in the understanding of the disorder. Neuroimaging has confirmed what numerous scientists, researchers and academics have long suspected – that Attention-Deficit/Hyperactivity Disorder is associated in some way with abnormalities or developmental delays in brain functioning (Barkley, 1998).

ADHD is now recognized as a disorder with a neurological base, although the severity of the symptoms can be influenced by environmental factors (Barkley, 1998; De Arnas, 2001; Penrice, 1996).

## **2.2 Primary symptoms of Attention Deficit Hyperactivity Disorder (ADHD).**

The primary diagnostic symptoms of ADHD as described in DSM-IV-TR (American Psychiatric Association, 2000) are a developmentally inappropriate

degree of inattention, impulsiveness, and motor activity (Barkley, 1998; Johansen, Aase, Meyer, & Sagvolden, 2002). The salient features of ADHD can be expected to interfere with the child's learning in school (Johansen et al., 2002). They usually occur during the pre-school years when hyperactivity may be prominent or during the early school years when problems in learning become apparent (Barkley, 1998).

### **2.2.1 Inattention**

By definition, children who have ADHD display difficulties with attention relative to children without ADHD of the same age and gender (Barkley, 1998). A child suffering from inattention; flits from task to task, does not follow instructions easily, makes careless mistakes, cannot concentrate easily, battles to pay attention to detail, has difficulty in organizing tasks and activities, and is forgetful and easily distracted (Barkley, 1998).

The problem with sustained attention occurs in situations where stimuli are widely spaced in time (van der Meere, 1996). It might be that the attention problems are associated with motivation as they might evident "only when the ability to concentrate stressed by the task being unwelcome or uninteresting" (Johansen et al., 2002c; Taylor, 1998a). Johansen et al. (2002) also argued that the apparent deficient sustained attention may be an effect of a shorter than normal delay of reinforcement gradient.

### **2.2.2 Hyperactivity**

The second primary characteristic of children with ADHD is their excessive or developmentally inappropriate levels of activity, whether motor or vocal (Barkley, 1998). Characteristics of hyperactivity include: fidgeting, running about, talking excessively, not being able to sit still or play quietly, blurting out answers to questions, interrupting others or talking out of turn and excessive restlessness (Barkley, 1998).

Although hyperactivity is seen in some situations like in the classroom, it might not be present in other situations like in play. It seems that the ADHD hyperactivity is absent in novel situations (Sagvolden & Sergeant 1998; Sleator & Ullman, 1981).

### **2.2.3 Impulsiveness / Behavioural Disinhibition**

In general terms, impulsiveness means acting without reflecting and a failure to plan ahead (Kuhle, Hoch, Rautzenberg, & Jansen, 2001). The problem of impulsiveness is sometimes scientifically defined as a pattern of rapid, inaccurate responding to a task. Clinically, children with ADHD are often noted to respond quickly to situations without waiting for instructions to be completed or adequately appreciating what is required in the setting. They may also fail to think of the potentially negative, destructive, or even dangerous results that maybe associated with particular situations or behaviour (Barkley, 1998).

The concept impulsiveness may be divided into two general subcategories: “Motor impulsiveness” and “cognitive impulsiveness” (Johansen et al., 2002).

Motor impulsiveness is presently defined as bursts of responses with short inter-response times. This is typical of ADHD behaviour and can be explained by altered reinforcement processes. This peculiar ADHD style of brief, short sequence of activity on tasks and rapid change is primarily seen in the premature responding, over-rapid responsiveness and excessive attraction to immediate reward (Sagvolden & Sergeant, 1998).

Cognitive impulsiveness is primarily seen in planning deficits, forgetfulness, poor use of time, recklessness and impetuous behaviour associated with ADHD. This behaviour has frequently been explained as problems with "executive functions", usually associated with prefrontal cortical function (Denckla, 1996b). The cognitive impulsiveness seen in ADHD has also inhibition, a concept that is not well defined (Johansen et al., 2002; Sagvolden & Sergeant, 1998).

### **2.3 Diagnostic criteria and primary symptoms**

The specific diagnostic criteria for ADHD are published in the American Psychiatric Association’s Diagnostic and Statistical Manual (Text Revision) (DSM-IV-TR) (American Psychiatric Association, 2000) and those for HKD (Hyperkinetic Disorder) in the International Classification of Diseases (ICD-10) (World Health Organization, 1993).

Various methods of assessment should be used to diagnose ADHD (Barkley, 1998; Castellanos, 1997b). An approach utilizing multiple assessment methods is required to bring reliable results. Neuropsychological tests may be helpful, but are not sufficient basis on which to make a diagnosis (Pary, Lewis, Matuschka, & Lippmann, 2002). Usually, assessment instruments in the process include clinical interviews, behaviour rating scales and behavioural observation. This will be further discussed in chapter 4.

The DSM-IV criteria for Attention-Deficit/Hyperactivity Disorder are as follows:

A. Either (1) or (2):

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

**Inattention**

- (a) often fails to give close attention to details or makes careless mistakes in schoolwork, work, or other activities.
- (b) often has difficulty sustaining attention in tasks or play activities.
- (c) often does not seem to listen when spoken to directly.

- (d) often does not follow through on instructions and fails to finish schoolwork, chores, or duties in the workplace (not due to oppositional behaviour or failure to understand instructions).
  - (e) often has difficulty organizing tasks and activities.
  - (f) often avoids, dislikes, or is reluctant to engage in tasks that require sustained mental effort (such as schoolwork or homework).
  - (g) often loses things necessary for tasks or activities(e.g., toys, school assignments, pencils, books, or tools).
  - (h) is often easily distracted by extraneous stimuli.
  - (i) is often forgetful in daily activities.
- (2) six (or more) of the following symptoms of hyperactivity-impulsiveness have persisted for at least 6 months to a degree that is maladaptive and inconsistent with developmental level:

### **Hyperactivity**

- (a) often fidgets with hands or squirms in seat.
- (b) often leaves seat in classroom or in other situations in which remaining seated is expected.
- (c) often runs about or climbs excessively in situations in which it is inappropriate (in adolescents or adults, may be limited to subjective feelings of restlessness).
- (d) often has difficulty playing or engaging in leisure activities quietly.

(e) is often “on the go” or often acts as if “driven by a motor”.

(f) often talks excessively.

### **Impulsiveness**

(g) often blurts out answers before questions have been completed.

(h) often has difficulty waiting turn.

(i) often interrupts or intrudes on others (e.g. butts into conversations or games).

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

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

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

E. The symptoms do not occur exclusively during the course of a Pervasive Developmental Disorder, Schizophrenia, or other Psychotic Disorder and are not better accounted for by another mental disorder (e.g., Mood Disorder, Anxiety Disorder, Dissociative Disorder, or a Personality Disorder (American Psychiatric Association, 1994).

### ***Subtypes***

DSM-IV (American Psychiatric Association, 2000) allows four subtypes of the disorder. Sometimes one or other pattern (either hyperactivity/impulsiveness

or inattention is predominant, even though most individuals have symptoms of both inattention and hyperactivity. The appropriate subtype should be indicated based on the predominant symptom pattern for the past 6 months.

Attention-Deficit/Hyperactivity Disorder, combined type: this subtype should be used if six (or more) symptoms of inattention and six (or more) symptoms of hyperactivity-impulsiveness have persisted for at least 6 months. Most children and adolescents with the disorder have the Combined Type. It is not known whether the same is true of adults with the disorder.

Attention-Deficit/Hyperactivity Disorder, predominantly inattentive type: this subtype should be used if six (or more) symptoms of inattention (but fewer than six symptoms of hyperactivity-impulsiveness) have persisted for at least 6 months.

Attention-Deficit/Hyperactivity Disorder, predominantly Hyperactive-Impulsive Type: this subtype should be used if six (or more) symptoms of hyperactivity-impulsiveness (but fewer than six symptoms of inattention) have persisted for at least 6 months. Inattention may often still be a significant clinical feature in such cases (American Psychiatric Association, 2000).

### ***Differential Diagnosis***

There are several disorders that have symptoms similar to those of the core symptoms of ADHD.

Anxiety may have prominent symptoms of restlessness and inattention. Depression in childhood may produce inattention and psychomotor agitation, which may be confused with symptoms of ADHD. Medical or neurological disorders can also be manifested in problems with attention, impulsiveness, or overactivity (Pary et al., 2002). A thorough developmental and medical history may signal potential medical concerns that need additional investigation by a physician. A detailed history of onset and duration of symptoms will often point to an alternative explanation of the child's behaviour. Direct observations may be helpful in discriminating ADHD from other related psychiatric or developmental disorders (Coles, Platzman, Smith, James, & Falek, 1992); Sagvolden & Sergeant, 1998).

### ***ICD-10 Diagnostic Criteria for Hyperkinetic Disorder (HKD)***

The research diagnosis of hyperkinetic disorder (HKD) requires the definite presence of abnormal levels of inattention, hyperactivity, and restlessness that are pervasive across situations and persistent over time and that are not caused by other disorders such as autism or affective disorders.

(3) G1. *Inattention*: At least six of the following symptoms of inattention have persisted for at least 6 months to a degree that is maladaptive and inconsistent with the developmental level of the child:

1. often fails to give close attention to details or makes careless mistakes in schoolwork, work, or other activities.

2. often fails to sustaining attention in tasks or play activities.
3. often appears not to listen to what is being said to him or her.
4. often fails to follow through on instructions or fails to finish schoolwork, chores, or duties in the workplace (not because of oppositional behaviour or failure to understand instructions).
5. is often impaired in organizing tasks and activities.
6. often avoids, or strongly dislikes tasks such as schoolwork or homework that require sustained mental effort.
7. often loses things necessary for certain tasks or activities such as, school assignments, pencils, books, toys or tools.
8. is often easily distracted by extraneous stimuli.
9. is often forgetful in the course of daily activities.

G2. *Hyperactivity*: At least three of the following symptoms of hyperactivity have persisted for at least 6 months to a degree that is maladaptive and inconsistent with the developmental level:

1. often fidgets with hands or squirms in seat.
2. leaves seat in classroom or in other situations in which remaining seated is expected.
3. often runs about or climbs excessively in situations in which it is inappropriate (in adolescents or adults, only feelings of restlessness may be present).

4. is often unduly noisy in playing or has difficulty in engaging quietly in leisure activities.
5. exhibits a persistent pattern of excessive motor activity that is not substantially modified by social contexts or demands.

G3. *Impulsiveness*. At least one of the following symptoms of impulsiveness has persisted *for* at least 6 months to a degree that is maladaptive and inconsistent with the developmental level:

1. often blurts out answers before questions have been completed.
2. often fails to wait in lines or awaits turns in games or group situations.
3. often interrupts or intrudes on others (e.g. butts into other's conversations or games).
4. often talks excessively without appropriate response to social constraints.

G4. *Onset* of the disorder is no later than the age of seven years.

G5. *Pervasiveness*. The criteria should be met for more than a single situation, e.g., the combination of inattention and hyperactivity should be present both at home and at school, or at both school and another setting where children are observed.

G6. The symptoms in G1-G3 cause clinically significant *distress or impairment* in social, academic, or occupational functioning.

G7. The disorder does not meet the criteria for pervasive development disorders, manic episode, depressive episode, or anxiety disorder (World Health Organization, 1993).

There are still major differences in decision rules between DSM-IV and ICD-10, according to Swanson et al. (1998c). In the symptoms domain groups (inattention, hyperactivity and impulsiveness) the ICD-10 diagnosis of HKD needs some symptoms in all three groups, whereas DSM-IV does not, but instead specifies partial subtypes if symptoms are from only one domain.

ICD-10 makes special provision for a combined diagnosis category if a conduct disorder is present and, because of the high frequency of this combination, uses the presence or absence of a conduct disorder as the basis for the main subdivision of HKD (World Health Organization, 1993).

DSM-IV-TR (American Psychiatric Association, 2000) does not make any special provision for conduct disorder as a comorbid condition but allows its diagnosis.

## **2.4 Prevalence and gender differences**

Attention-Deficit/Hyperactivity Disorder is the most common childhood mental disorder, with prevalence of 3% to 5%, (American Psychiatric Association, 2000). It is the most common heritable and behavioural disorder of childhood (Brown, Freeman, & Perrin, 2001; Curran et al., 2003; Rietveld, Hudziak, Bartels, van Beijsterveldt, & Boomsma, 2003). Epidemiological studies suggests that the syndrome is three to four times higher in males than in females but during adolescence and young adulthood, the proportion of females affected

increases (Biederman et al., 1994; Gaub & Carlson, 1997; Taylor, 1998) and occurs approximately in 3-5 % of school children in Western countries (Buitelaar, 2002) or 5-10 % world wide (Brown et al., 2001; Swanson et al., 1998c). The observed male to female difference is not due to an X-linked gene but considered due to familial factors (Smalley et al., 2000).

Swanson et al. (1998c), estimate the ratio of boys to girls with Attention Deficit/Hyperactivity Disorder to be 3:1 to 9:1, largely as a result of referral bias. Many girls with ADHD may not reach the attention of mental health services because they are less likely to have the more externalizing, aggressive behaviours seen in boys (Leung et al., 1996; Meyer & Aase, 2003; Meyer, 1998; Taylor, 1998).

Although there are no exact figures in South Africa, Meyer (Meyer, 1998; Meyer, Eilertsen, Sundet, Tshifularo, & Sagvolden, 2004) studied prevalence in primary school children of the Limpopo province. She found the prevalence rates of 5.1 % in the total primary school population, with the prevalence among boys 6.9 % and among girls 3.8 %.

## **2.5 Aetiology**

The risk factors for ADHD appear to be primarily biological but environmental, psychosocial factors, side effects of medications are apparent in many cases. Historically, brain damage has been linked to ADHD, but few persons with ADHD display neurological abnormalities (Erickson, 1998).

### **2.5.1 Pre-natal, Peri-natal and Postnatal Physical Factors.**

Complications of pregnancy, labour, and delivery appear to be at least weakly linked to ADHD. Maternal alcohol consumption during pregnancy is associated with increased activity levels and attention problems that persist through the childhood years (Taylor et al., 1998a). Children with ADHD show a higher than average rate of minor physical abnormalities whose origins have been placed in the first trimester of pregnancy (Erickson, 1998). Some environmental pollutants such as polychlorinated biphenyl's (PCBs) may cause dopamine dysfunction. The lipophilic nature of PCBs makes organs like the brain particularly vulnerable. Intake of these pollutants by pregnant women may cause developmental abnormalities in humans including low birth weight, disruptive behaviour and overactivity in their children (Johansen et al., 2002).

### **2.5.2 Biological / Genetic Factors**

In the early 1990s a variety of functional imaging techniques showed that patients with ADHD had less active and smaller frontal basal-ganglia neural network areas than unaffected individuals. The Magnetic Resonance Imaging (MRI) studies of brain anatomy have also reported consistent abnormalities in children with ADHD (Swanson, Castellanos, Murias, LaHoste, & Kennedy, 1998b). The brain imaging studies implicate the frontal basal-ganglia neural network, and the molecular genetic studies implicate the dopamine pathways that modulate and integrate neural activity of these networks (Swanson et al., 1998b).

These specific neuroanatomical and biological abnormalities provide ADHD firm ground to build and understanding of a biological basis.

For over 25 years, a genetic component to ADHD has been found in adoption, family, and twin studies. Acquired and inherited factors such as foetal distress due to environmental factors may selectively damage striatal neurons and affect developing frontal lobe basal ganglia neural networks. Genetic polymorphisms in the dopamine receptor may reduce dopamine activity and alter normal development of the meso-cortical and nigro-striatal dopamine branches, which moderate activity in these network (Swanson et al., 1998a; Kuhle et al., 2001).

It is likely that dopamine dysfunction plays a pivotal role in the neurobiology of ADHD. Therefore, ADHD like symptoms may be produced not only by genetic factors, but also by other agents altering dopaminergic functioning. Chronic intake of the dopamine agonists like cocaine, crack and amphetamines will produce a down-regulation of dopamine synthesis (Johansen et al., 2002; Sagvolden, Aase, Zeiner, & Berger, 1998)

### **2.5.3 Psychosocial Factors.**

A number of studies have shown ADHD to be related to marital discord, family dysfunction, and abuse of the child. More severe ADHD symptoms have been associated with increased family problems (Erickson, 1998).

It seems as if psychosocial factors may influence the severity of the symptoms, the types of the secondary symptoms and the outcome of the disorder (Barkley, 1998).

Barkley (1996) in his family studies of ADHD, recorded socioeconomic status and family integrity. He found that families with an ADHD parent to be less cohesive and have more conflicts than families without an ADHD parent.

In this study, there was no effect of socioeconomic status on pattern of familial transmission.

#### **2.5.4 Environmental pollutants**

In general, environmental factors have not been systematically addressed although a wide variety of factors have been implicated, such as lead toxins, early institutional care, psychosocial stress, pregnancy and delivery complications, and prenatal alcohol exposure (Meyer, 1998; Taylor et al., 1998).

Polychlorinated biphenyls (PCBs) constitute a group of halogenated aromatic hydrocarbons that are lipophilic and bioaccumulating. The lipophilic nature of PCBs makes organs like brains, vulnerable. Intake of these pollutants causes developmental abnormalities in humans, including low birth weight, disruptive behaviour and overactivity (Johansen et al., 2002). A series of studies of effects of PCBs exposure on behaviour and brain chemistry showed that normal male rats exposed to subtoxic doses of PCB congener 153 through

mother milk when pups, were hyperactive and impulsive when they grow up. PCB 153 produces hyperactivity and impulsiveness via the monoaminergic pathways (Holene, Nafstad, Skaare, & Sagvolden, 1998).

Studies of monozygotic twins with careful assessment of environmental variables are needed to clarify the nonshared environmental familial factors. Any differences between identical twins, which have identical genotype and live in the same household, are assumed to originate from environmental factors (psychosocial and biological) such as parental attitude, classrooms, peer relations, or medical problems different for each twin (Faraone, Doyle, Mick, & Biederman, 2001).

## **2.6 Comorbid Disorders**

Almost one third of children with ADHD have one or more psychological and/or developmental disorder. Even if the clinician may not always be in a position to make a precise diagnosis of coexisting conditions, consideration and examination for such a co-existing condition should be an integral part of the evaluation (Barkley, 2002)

Disruptive behavioural disorders and internalising disorders are the most common comorbid disorders in ADHD (Barkley, 1998; Halperin et al., 1990; Pliszka, Carlson, & Swanson, 1999; Taylor, Sandberg, Thorley, & Giles, 1991). The disruptive behavioural disorders, Oppositional Defiant Disorder (ODD) and Conduct disorder (CD) coexist with ADHD in 35% of the children. The DSM-

IV-TR (American Psychiatric Association, 2000) defines Conduct Disorder as a repetitive and persistent pattern of behaviour in which the basic rights of others or major age-appropriate societal norms are violated. This disorder is more common in hyperactive (ADHD) children. Many researchers regard Conduct Disorder as a more severe version of Oppositional Defiant Disorder. Children with CD are often truant from school and may run away from home. Vandalism with deliberate destruction e.g. setting fires, smashing windows, early alcohol and drug abuse and precocious sexual activity is common (Ledingham, 1999).

Internalising disorders like mood disorder, and anxiety disorders coexists with ADHD in 25% and 18%, respectively (American Psychiatric Association, 2000; American Academy of Pediatrics, 2000). Finally, children with late onset hyperactivity are more likely to have behaviour problems confined to school and to show specific learning difficulties (e.g. reading disorder, dyslexia, dyscalculia, problems with writing) are common in boys with ADHD (Biederman, Faraone, & Monuteaux, 2002; Brown, 2000; Castellanos, 1997a; Seidman, Biederman, Monuteaux, Doyle, & Faraone, 2001a; Taylor, 1998).

The incidence of comorbid disorders is more likely for children whose parents have a history of psychopathology or whose families are disorganized or dysfunctional (Livingston, 1999).

Children who receive treatment for ADHD tend to have lower rates of comorbid disorders (Hinshaw, Heller, & McHale, 1992).

## **2.7. Treatment**

There is a widespread agreement that intervention for the child or adolescent with ADHD should be multimodal (Pary et al., 2002).

Effective interventions for children with ADHD fall into 3 broad categories that can, and often should, be used together: behaviour modification, educational modifications, and stimulant drug therapy (Meyer & Aase, 2003).

### **2.7.1 Pharmacological treatment**

#### *2.7.1.1 Stimulant medication*

The dopaminergic and adrenergic effects of stimulants such as Methylphenidate (Ritalin®, Methylin®, Metadate®); d-amphetamine (Dexedrine®, Dextrostat®) D, L-amphetamine (Adderall®) and Pemoline (Cylert®, PemADD®) symptomatically improve ADHD core symptoms as well as associated features including aggression, social interaction, and academic productivity. These medications require multiple daily doses to maintain efficacy (Swanson, 2003). The effects are similar in children and adults without ADHD, and are therefore neither diagnostic nor specific. The improvements in impulsive and overactive behaviour are more pronounced than inattentiveness (Swanson, Pfiffner, Wigal, & McBurnett, 1993). Compliance with medication has been found to yield good results.

The mechanism of action of psychostimulants is thought to be re-uptake blockade of catecholamines into the presynaptic nerve endings, thereby preventing their degradation by monoamine oxidase (Stahl, 1996).

A new generation of long-acting stimulant drugs in the treatment of ADHD employs novel delivery systems to overcome tachyphylaxis, or acute tolerance. Concerta® uses an osmotic pump mechanism that creates an ascending profile of methylphenidate in the blood to provide effective extended treatment for 10-12 hours. Concerta® is available in 18, 36, and 54 mg to approximately 3 times daily dosing of 5, 10, and 15 mg immediate-release methylphenidate.

Ritalin LA® contains a mixture of immediate and delayed- release beads to provide effective methylphenidate treatment for 8-9 hours. Ritalin LA is available in 20-, 30-, and 40- mg capsules to approximately 10, 15, and 20 mg twice daily dosing of immediate –release of methylphenidate (MTA-Cooperative Group, 1999).

#### *Side effects of stimulant medication*

Side effects of stimulants are usually transient and commonly include anorexia, abdominal discomfort, insomnia, headaches, and irritability. Difficulties with sleep are usually temporary and minimized by avoiding dosing late in the day (Pary et al., 2002).

Long term stimulant treatment (2-4 years) with average doses of 40 mg of methylphenidate per day results in a reduction in the rate of weight gain and lesser extent height gain in treated children (Spencer et al., 1996). Stimulants are eliminated more rapidly in children than in adults because of relatively less hepatic capacity, more glomerular clearance, and less body fats. In children, when stimulant levels decline, hyperactivity often returns (Pliszka, Browne, Olvera, & Wynne, 2000)

Alternative medications, dietary modifications, and neurobehavioral techniques are much less researched. As with other chronic conditions, such as asthma or diabetes mellitus, pharmacological treatment may be necessary as the cornerstone of adequate management, but it should be highly interactive with the management of the home, school, and extracurricular environment. Treatment decisions should be guided by what is in the best interest of the child and what is acceptable to the family (MTA-Cooperative Group, 1999).

Other types of medication have also been used in the treatment of Attention-Deficit/Hyperactivity Disorder, with various rates of success. These second line agents include tricyclic antidepressants, selective serotonin reuptake inhibitors (SSRI's) and bupropion; third line agents would include MAO inhibitors and Antipsychotics (Kaplan & Sadock, 1998).

### *2.7.1.2 Tricyclic Antidepressants (TCAs)*

TCAs modulate various brain neurotransmitters, especially norepinephrine and serotonin, by blocking re-uptake presynaptically. Imipramine and desipramine are the most frequently studied TCAs. Advantages of this class of drugs include their relatively long half-life (approximately 12 hours), absence of abuse potential, and putative positive effects on mood, anxiety, sleep, and tics. Studies of TCAs have uniformly reported considerable improvement in ADHD symptoms in ADHD subjects with comorbid disorders (Wilens, Biederman, Mick, & Spencer, 1995).

### *2.7.1.3 Bupropion*

Bupropion appears to possess both indirect dopamine agonist and noradrenergic effects. Bupropion was shown to be effective for ADHD in children in a large, controlled multisite study (Conners et al., 1996).

### *2.6.1.4 Selective serotonin reuptake inhibitors (SSRI's)*

Available SSRI's include fluoxetine, paroxetine, fluvoxamine, citalopram, and sertraline. The use of these serotonergic compounds is not supported; however these compounds are frequently combined with effective anti-ADHD agents such as cholinergic agents which has been seen to enhance dopaminergic neurotransmission (National Institute of Mental Health, 2002).

#### *2.7.1.5 MAO inhibitors*

The MAOIs inhibit the intracellular catabolic enzyme monoamine oxidase. Both MAO-A and MAO-B, metabolise tyramine and dopamine. Zametkin, Rapoport, Murphy, Linnoila, & Ismond (1985) reported a significant reduction in ADHD symptoms with minimal adverse effects in children.

#### *2.7.1.6 Antipsychotics*

In the early literature, typically antipsychotics for ADHD were found to be mildly efficacious for behavioural symptoms in hyperactive children. However the lack of cognitive enhancement and the risk of long-term adverse effects (such as tardive dyskinesia) greatly limit their usefulness in the treatment of ADHD (Gitlin, 1996).

Table 2.1 represents some of these medications in order of preference

**Table 2.1. Efficacy of Psychopharmacological agents in the treatment of ADHD**

<b>Medication</b>	<b>Efficacy rating</b>
<i>First line agents</i>	
Stimulants:	
Methylphenidate (Ritalin)	+++
d-amphetamine (Dexedrine)	+++
Pemoline (Cylert)	+++
<i>Second line agents:</i>	
Antidepressants	
Tricyclics	++
Bupropion	++
<i>Third line agents</i>	
MAO inhibitors	+
Antipsychotics	+
+++ = Definite efficacy ++ = Probable efficacy + = Possible efficacy	

From (Gitlin, 1996)

### **2.7.2 Psychosocial interventions**

Non-pharmaceutical therapies are recommended in addition to medication. Individual psychotherapy, behaviour modification, parenting classes, parent support groups, school involvement, and education about Attention-Deficit/Hyperactivity Disorder can enhance results (Pary et al., 2002).

According to Meyer and Aase (2003) behavioural therapy programmes for children with Disruptive Behaviour Disorders have been increasingly well

developed during the past two decades. These programmes aim to teach parents and teachers behaviour management skills based on sound behavioural learning principles, particularly the systematic use of reinforcement contingencies. Among the most comprehensively described are the parent and teacher training programmes by Barkley (1997b) ; Barkley & Murphy (1998). The goals of the programmes are: (1) to improve parental (and teacher) management skills and competence in dealing with child behaviour problems, (2) to increase parent and teacher knowledge of the cause of childhood defiant behaviour, (3) to improve child compliance with commands, directives, and rules, given by parents and teachers and (4) to increase family harmony through the improvement of parental and teacher use of positive attention, the provision of clear guidelines and rules, the application of swift, fair, and just discipline for inappropriate child behaviour and general reliance on principle-guided behaviour (Barkley, 1997b).

#### *2.7.2.1 Behavioural and Educational Management*

Behavioural and educational management is directed at improving behavioural control and cognitive efficiency and requires intense teacher and parent involvement. This mode of management complements drug treatment and allows for minimum medication dosage. Behavioural interventions frequently require a trained specialist who can help parents or teachers to develop, implement, and maintain an effective program. Most of the programs entail a structured system that targets appropriate and inappropriate behaviours and

provides contingent and immediate rewards and punishments for the behaviours. Reinforcement usually takes the form of a point or token system where children can earn points/tokens toward a reward. Removal of points for inappropriate behaviour is also frequently part of the program.

In addition to behavioural interventions, another significant aspect of the treatment plan is adapting the teaching/learning process and academic goals to the child's cognitive and behavioural needs. Many different types of cognitive behavioural treatments have been applied to children with ADHD, including verbal self- instructions, problem solving strategies, cognitive modelling, self-monitoring, self- evaluation, and self-reinforcement (Abikoff, 1991; Pary et al., 2002).

### **2.7.3 Combined Interventions**

Combinations of pharmacological and psychosocial treatments for ADHD are generally recommended on the basis that multiple areas of impairment require multiple modalities of treatment. The underlying theme of these subtypes of treatments is the promotion of self-controlled behaviour through the enhancement of problem-solving strategies (Klein et al., 1997)

## **2.8 Cultural Influences**

Geographical variations in the diagnosis of hyperactivity are very strong. Variations in prevalence rates between countries depend on a number of factors,

including the diagnostic criteria used, age and gender of the population, socio-economic status and urban living (Barkley, 1996). Studies comparing the UK and Chinese populations argue with much more strength that the standards of the recognition of disorder can be very different, and that deviance is socially constructed. That, which is diagnosed in one culture may not be so in another, therefore it is important for clinicians to realize that any neuropsychological deviations found in a different country will not necessarily apply to the people whom they are studying (Taylor, 1998). Very little is known about ADHD in the African continent. Recent research indicates that ADHD is the most prevalent child psychiatric disorder also in South Africa (Meyer, 1998). A study by Meyer et al. (2004) attested to a cross-cultural agreement on classifications of the kinds of behaviour problems assessed by the Disruptive Behavior Disorders Rating Scale (DBD). It seems that even though various cultures may have different perceptions and interpretations of behaviour, they discriminate between different kinds of behaviour patterns in a similar way as implied in the Western diagnostic system of ADHD. Another survey of 25% of all children in a small village close to Khartoum, Sudan, found that 12% of the children suffered severe symptoms frequently including overactivity, temper tantrums, fights, and repeating grades at school, which may be related to ADHD (Baasher & Ibrahim, 1976).

## **2.9 Prognosis and Adult outcome**

It is estimated that 3% to 5 % of the general population is affected by ADHD. Five million adults are affected by deficits in attention, (West, 1999). Follow-up studies in the USA have confirmed a poor prognosis for children with ADHD. Attention-Deficit/Hyperactivity Disorder can (and often does) continue in adolescence and adulthood if it is left untreated (Klein & Mannuzza, 1991; Mannuzza, Klein, Bessler, Malloy, & LaPadula, 1993; Mannuzza, Klein, Bessler, Malloy, & Hynes, 1997; Mannuzza, Klein, Bessler, Malloy, & LaPadula, 1998; Mannuzza, Klein, & Moulton, III, 2002a; Mannuzza & Klein, 1992; Mannuzza, Klein, & Moulton, III, 2002b; Taylor, Chadwick, Heptinstall, & Danckaerts, 1996)

The course of ADHD is unpredictable. Symptoms are inconsistent between children and adults, though decreased attention span and problems with impulse control commonly persist. Difficulties occur in all areas of life: family and social relationships, education and career or employment. Some people may experience partial remission but may be still vulnerable to antisocial personality disorder, chemical dependence, and mood disorders (Kaplan & Sadock, 1998).

DSM-IV (American Psychiatric Association, 2000) recognizes the adult type of ADHD in its category, ADHD residual type (American Psychiatric Association, 2000).

## **2.10 Conclusion**

ADHD is the most prevalent psychiatric condition of childhood. Heritability studies suggest that approximately 80% of symptoms of ADHD are due to genetic factors (Faraone, Doyle, Mick, & Biederman, 2001; Levy & Swanson, 2001; Swanson et al., 2000).

Identification of environmental factors, studying additive gene effects and understanding the environmental and genetic interaction may provide a better insight about ADHD aetiology that may be directed toward a better method for its intervention.

Education of ADHD patients and their families is considered the first step in treatment. Pharmacotherapy and non-pharmacologic interventions are used. Collaborative Multisite Multimodal Treatment Study of children with ADHD (MTA) document improvement of ADHD with medication, behavioural therapy, and a combination of two (MTA-Cooperative Group, 1999).

## *Chapter 3*

### NEUROBIOLOGY OF ATTENTION-DEFICIT/HYPERACTIVITY DISORDER

#### **3.1 Introduction**

Attention-Deficit/Hyperactivity Disorder (ADHD) is characterised by symptoms of inattention, impulsiveness, and overactivity that arise in childhood and persists into adulthood. Psychostimulant medications are effective in alleviating these cardinal symptoms of ADHD and are widely used to treat the disorder. The robust response of individuals with ADHD to these medications has stimulated considerable research into the possible neurobiological basis of the disorder. Yet, the specific nature of the pathophysiology of ADHD remains elusive. Attempts to distinguish individuals with ADHD from controls on measures of cognitive function, neuroanatomy, neurotransmitter activity, and genetic factors have generally failed to yield replicable results. However, pharmacological treatment studied, as well as basic research in animals and humans have provided some clues to the possible pathophysiology substrates of ADHD (Faraone et al., 2001).

#### **3.2 Neuroanatomy**

Neuroimaging studies provided the first in vivo evidence of structural and functional brain abnormalities associated with ADHD (Swanson, Castellanos, Murias, LaHoste, & Kennedy, 1998). Morphological studies using

magnetic resonance imaging (MRI) have identified subtle anomalies in the prefrontal cortex (PFC) and the striatum that may be unique to ADHD. The PFC as well as the interconnected caudate nucleus and globus pallidus in the basal ganglia in children with ADHD have been found to be smaller relative to controls (Barkley, 1998). The volumes of the cerebellar hemispheres have been found to be significantly smaller in children with ADHD (Castellanos et al., 2002) .

Recently there is also interest centred on functional neuroimaging (fMRI) that allows examination of the working brain in vivo since structural abnormalities do not necessarily imply functional impairments. Recent studies with functional MRI have tentatively linked the executive function deficits in ADHD with abnormal neural activity in the striatum and the PFC (Faraone & Doyle, 2000; Rubia et al., 1999).

Although the findings are not entirely dependable, neuroimaging studies have revealed a pattern of structural and functional abnormalities in the PFC and the basal ganglia that likely play a central role in ADHD (Faraone et al., 2000; Giedd, Blumenthal, Molloy, & Castellanos, 2001).

Research using single photon emission computerised tomography (SPECT) and positron emission tomography (PET) have yielded contradictory evidence of reduced resting brain activity in ADHD. SPECT studies have reported hypoperfusion in the striatum and PFC of children with ADHD. In

contrast, PET studies have found diminished glucose metabolism in the superior PFC of adults and not adolescents with ADHD (Faraone et al. 2000).

Fig 3.1 illustrates the various dopamine projection areas and neuroanatomical brain structures involved in ADHD.

**Fig 3.1 Dopamine projection areas and neuroanatomical brain structures involved in ADHD (Purves et al., 2001)**

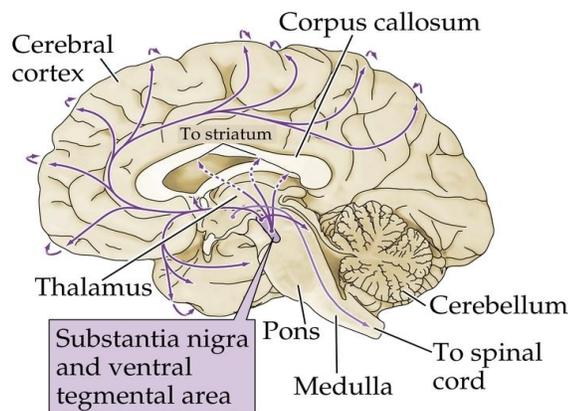


Table 3.1 summarizes the regions of the brain which are involved in ADHD.

**Table 3.1. Neuroanatomical structures relevant to ADHD**

Basal ganglia	Forebrain of the neocortex, includes the caudate nucleus, putamen, and globus pallidus – sequencing and inhibiting of motor movements
Limbic system	Subcortical area – regulates emotional expression
Frontal lobes	Part of the cortex in front of the central sulcus – higher cognitive reasoning (e.g., organising, interpreting, prioritising information)
Temporal lobe	Inferior to the lateral sulcus, posteriorly terminate in the angular gyrus- central to auditory and visual perceptions and effective tone for sensory input  -primary site for language comprehension and time recognition.  -assist with organization and categorisation of events
Reticular formation	Diffuse, interconnected neuronal network occupying the core of the brain stem  Central role in sleep and arousal, Attention, muscle tone, movement, and several vital reflexes  Receives sensory information from areas throughout the brain and projects impulses to the cerebral cortex, thalamus and spinal cord
Cingulated gyrus	Area of cortex just above the corpus callosum, acts as an alternate route for sensory information.
Corpus callosum	Pathways of fibres connecting right and left corresponding structures and associated structures  Primary route for hemispheric communication

From (Swanson et al., 1998a)

### 3.3 Neurochemistry of ADHD

The neuroanatomical networks associated with ADHD have led to the search for specific biochemical abnormalities that may be linked to its neuroanatomy. The pre-eminent biochemical theory of ADHD has been based on a catecholamines hypothesis since the 1970's (Swanson, 1998), but recent refinements of this theory focuses on the role of dysfunctioning dopamine systems (Johansen et al., 2002; Pary et al., 2002b).

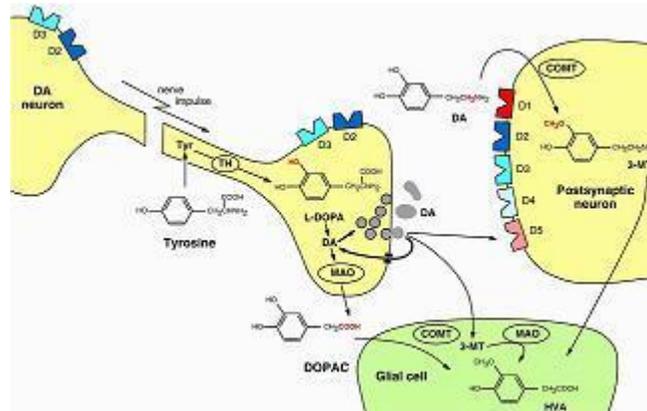
Pliszka, McCracken, and Maas (1996) focused on the unitary dopamine theory of ADHD, based on the proposal that different abnormalities might exist in two dopamine regions: underactivity in a cortical region (i.e. anterior cingulate) which results in cognitive deficits, and overactivity in a subcortical region (i.e. caudate nucleus), which results in motor excesses (Castellanos, 1997b). Arnsten, Steere, and Hunt (1996), however, modified the noradrenergic theory of ADHD based on the proposal that different abnormalities may exist in two noradrenergic regions: underactivity in a cortical region (i.e. dorsolateral prefrontal) which result in primary memory deficits and overactivity in a subcortical region (i.e. locus coeruleus), which result in overarousal. ADHD is associated with dysfunctioning dopamine branches, (Johansen et al., 2002; Levy & Hobbes, 1988; Levy, 1992; Levy et al., 2001).

There are five distinct dopaminergic receptors coded by five different genes (D1-D5) grouped into two families: D1/D5 and D2/D3/D4 (Carey et al., 1998). ADHD seems to be having genetic components associated with genes coding for receptors in the dopamine D-2 family and the membrane dopamine transporter (DAT) protein. The dopaminergic systems consists of two major branches i.e., the nigra striatal system originating in the substantia nigra projecting mainly to the neostriatum and the meso-cortico-limbic system originating in the ventral tegmental area and projecting to the prefrontal cortex, the nucleus accumbens septi and the olfactory tubercle. It is therefore quite possible that frontal dopamine dysfunctioning may contribute to the observed cognitive impulsiveness (planning deficits, forgetfulness, poor use of time, recklessness and impetuous behaviour) of ADHD children (Swanson et al., 1998c; Taylor, 1998).

### **3.3.1 The role of dopamine in ADHD**

Dopamine is a neurotransmitter that travels across the synapses in the brain (fig3.2). It was found that high levels cerebrospinal fluid leads to high levels of hyperactivity which may lead to the believe that dopamine hypofunctioning is associated with ADHD (Barkley, 1998; Castellanos, 1997b; Sagvolden & Sergeant, 1998). Dopamine is secreted by neurons, as indicated in Figure 3.1 in specific parts of the brain in order to inhibit or enhance the activity of other neurons, particularly those in emotion and behaviour (Barkley, 1998).

Fig 3.2 A dopamine synapse (Waters, 1995)



### 3.4 Genetics of Attention Deficit Hyperactivity Disorder

Data from prior family-, twin-, and molecular genetic studies suggest that genetic factors are involved in ADHD (Faraone, 2000). Siblings, parents, and second-degree relatives of ADHD probands have been shown to increase frequency of ADHD. In twin studies, monozygote twins have a 45% to 100% concordance rate, but dizygote twins have only a 4% to 20% concordance rate in ADHD. Large twin studies of ADHD has shown that ADHD has a heritability of almost 80%- thus 80% of the differences in attention, hyperactivity and impulsivity between people with and without the disorder can be explained by genetic factors (Barkley, 1998). Several distinct neuropsychiatric disorders that run in families, probably genetically related to

ADHD and with relatively high co-morbidities with ADHD, are indicative of some shared genes. These neuropsychiatric disorders include depression, anxiety, tic disorders, learning disorders, substance abuse, and conduct disorders (Comings, 2001; Faraone et al., 2001; Wilens et al., 2002).

Studies aimed at delineating the specific genetic component of ADHD have focused primarily on dopamine (DA) system genes (Faraone, 2000); (LaHoste et al., 1996). Consistent with evidence of altered DA activity in children with ADHD, molecular studies have identified a number of DA genes as potential candidate genes in the disorder. These include the D2 (DRD2), D4 (DRD4) and D5 (DRD5) receptor genes; the dopamine b-hydroxylase gene (DBH), and the DA transporter gene (DAT1). However, the strongest evidence links ADHD with variable number tandem repeat (VNTR) polymorphisms of DAT1 and DRD4 (Faraone et al., 2001; LaHoste et al., 1996).

Studies have also found an association between ADHD and both the 7-repeat allele of DRD4 which mediates an intercellular response to DA and the 10-repeat allele of DAT1, which is linked to elevated DA reuptake (Barr et al., 2002; Holmes et al., 2002; Manor et al., 2002; Faraone et al., 2001; Swanson & Volkow, 2002).

Cook et al. (1995) demonstrated an association between ADHD and the 480-bp allele of the dopamine transporter gene (480-bp DAT1 allele) using

a family-based association study. This finding was replicated in a population-based study that found the 480-bp allele to be associated with ADHD and Conduct Disorder. These findings suggest that the 480-bp allele may be a risk factor for several disorders. Using a "knockout" mouse model, Giros, Jaber, Jones, Wightman, and Caron (1996) showed that disrupting the mouse DAT gene leads to a hyperdopaminergic phenotype that includes spontaneous hyperlocomotion. This may provide a simple animal model of hyperactivity and is consistent with the idea that abnormalities in the DAT gene could be a risk factor for ADHD. LaHoste et al. (1996) noted several reasons why the DRD4 7-repeat allele has functional implications that are relevant for ADHD. This variant of DRD4 mediates a blunted response to dopamine. Moreover, the distribution of DRD4 mRNA in the brain suggests it plays a role in cognitive and emotional functioning. A population study found higher rates of the 7-repeat allele among ADHD children compared with control children who were carefully matched for ethnicity and gender (Faraone, 2000). Swanson et al. (2000) argue that although the presence of the DRD4 7-repeat allele may be associated with a modestly increased risk for ADHD, it is not a necessary condition, as about half of the ADHD children do not have a 7-repeat allele.

### **3.5 Neurobiological models of ADHD**

The change in the nosology from hyperactivity early in the 20<sup>th</sup> century to ADHD by the early 1980s paralleled the shifting from the belief that this disorder was primarily bad behaviour to the hypothesis that the disorder represents a cognitive brain problem that results in associated maladaptive behaviour.

Several researchers have proposed neurobiological models of ADHD that attempt to account for the deficits in attention and inhibitory control that are frequently seen in the disorder. Heilman, Voeller, and Nadeau (1991) and Sagvolden (2000) proposed that reduced dopaminergic (DA) tone in the prefrontal-striatal circuits that gate sensory inputs into motor systems produces the inattention, impulsiveness, and hyperactivity of ADHD. According to this model, the Prefrontal Cortex (PFC) receives sensory input from association cortices and, in turn, influences motor function through connections with the supplementary motor area (SMA). The PFC also projects to the caudate nucleus, which in turn innervates the substantia nigra in the midbrain. The substantia nigra provides feedback to the PFC via the ventral anterior thalamus. This feedback influences the activity of the PFC projections to the SMA, thereby gating sensory input to this motor area. The gating function of the PFC is modulated by the input from the mesocortical DA, system, which originates in the midbrain ventral tegmental area (VTA). Therefore, diminished DA tone in the PFC might impair

the gating system and disrupt the function of the SMA, thereby producing the deficits in attention and executive functions characteristic of ADHD.

### **3.5.1 Barkley's model of Executive Dysfunctions**

Barkley proposed a model of executive functions located in the prefrontal cortex that explains the cognitive and behavioural deficits associated with ADHD. This model comprises 5 major executive functions that enable individuals to recognise and control their actions to achieve a goal (Barkley, 1997a).

#### *Behavioural Inhibition*

In Barkley's model behavioural inhibition is seen as having three roles, it delays and interrupts responses and controls interference to allow individuals to control verbal and motor impulses. Deficits in inhibition interrupt thoughtful behaviours.

#### *Nonverbal Working memory*

Nonverbal Working memory enables the person to have a sense of the past and future and a cognitive awareness of self.

#### *Internalisation of speech*

Internalisation of speech is the self-talk used to evaluate and direct behaviour of the self. It allows a person to reflect before acting and to select behaviour or action that is going to help them reach their goal.

### *Self-regulation of affect*

Self-regulation of affect allows people to consider emotional responses before responding and helps to modify inappropriate responses. It promotes self-motivation and also helps people to carry out boring and repetitive tasks that have no motivating factors. This executive function also helps a person to be more objective and realistic in their self-evaluation.

### *Reconstitution*

Reconstitution is a form of play that allows people to analyse the experiences in their working memories to synthesize new responses, which they accept or reject based on the likelihood that the response can help them to achieve their goal. It also promotes effective and flexible problem solving where alternative plans or actions need to be generated (Barkley, 1997a).

Barkley has proposed that, of these 5 executive functions, response inhibition is the most obviously deficient in individuals with ADHD and that this deficit may lead to the impairments observed in the psychological and social abilities associated with the other 4 executive functions (Barkley, 1997a; Barkley, 1997b).

### **3.5.2 Cognitive-energetic model**

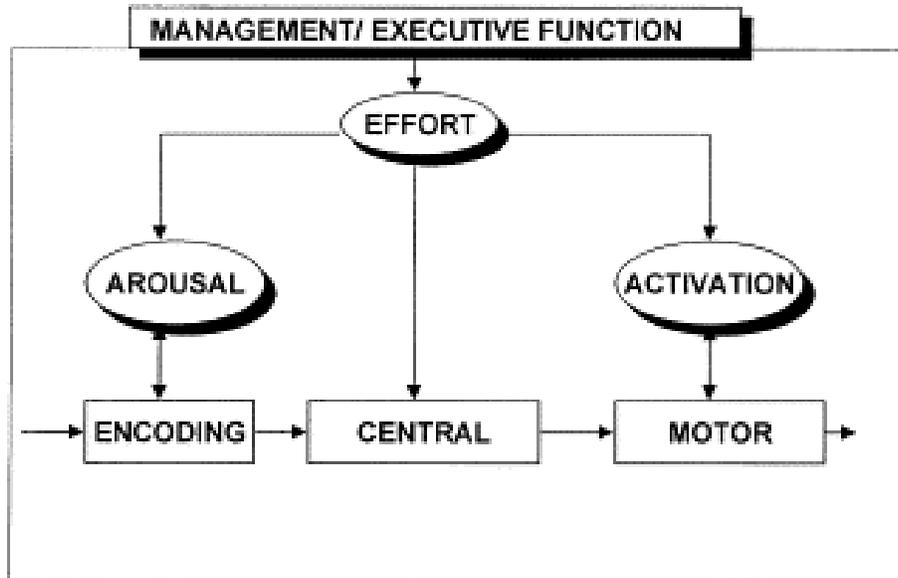
The cognitive-energetic model draws attention to the fact that ADHD has effects at three levels: cognitive mechanisms such as response output, energetic mechanisms and control systems of executive functions (Sergeant, Oosterlaan, &

Van der Meere, 1999). The model suggest that disruptive disorders have common deficiencies in EF control systems and may be possibly differentiated either at an energetic level or at specific elementary cognitive stages (Sergeant, 1998).

Information processing in this model is said to be determined by computational and state factors (such as effort, arousal and activation). Computational mechanisms of attention include four general stages: encoding, search, decision and motor organisation. These stages of information processing are associated with experimental task variables.

The second level of the model has three energetic pools. They are effort, which is the necessary energy to meet the demands of a task. Effort can be affected by factors such as cognitive load. The second pool arousal is defined as phasic responding, which is time, locked to stimulus processing. Tonic changes of physiological activity by contrast, were thought to represent the operation of the activation pool. The activation pool was identified with the basal ganglia and the striatum (Pribram & McGuinness, 1973). The third level of this model includes management or evaluation mechanism, which is associated with planning, monitoring, detection of their errors and corrections, this is currently associated with the concept of executive functioning (EF) (Sergeant, 1998; Sergeant et al., 1999). Fig 3.3 illustrates the Cognitive-energetic model.

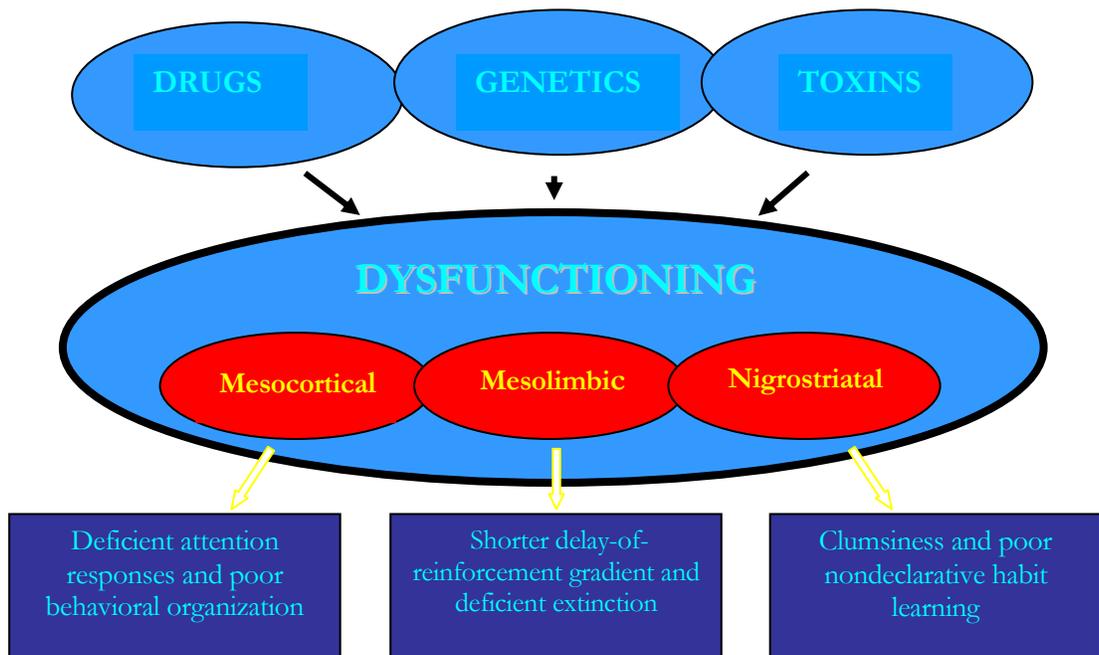
Fig. 3.3 The Cognitive-energetic model (Sergeant, 1998)



### 3.6 Neurobiological theory

This research project is based on the neurobiological model of Sagvolden et al. (Johansen et al., 2002; Sagvolden, 1999; Sagvolden & Sergeant, 1998). This model focuses on the dysfunctioning dopamine projection branches as aetiological factors in the observed ADHD symptoms. These authors argue that at neurobiological level ADHD symptoms are to a large extent caused by a dysfunctioning dopamine system (see figure 3.4).

Figure 3.4. Neuropsychological theory of Sagvolden et al. (from Johansen et al., 2002)



ADHD symptoms may to a large extent be caused by dopamine dysfunction.

A dysfunctioning *mesocortical dopamine branch* will cause deficient attention responses and poor behavioural organization (deficient ‘executive functions’).

A dysfunctioning *mesolimbic dopamine branch* will contribute to a shorter delay-of-reinforcement gradient and deficient extinction (premature responding).

A dysfunctioning *nigro-striatal dopamine branch* will cause impaired timing and force regulation of muscle groups (poor motor control).

Dopamine dysfunction may be caused by various factors, which may include environmental factors like drug of abuse or pollutants, which may also explain geographical differences in prevalence rates, genetic factors such as pre-, peri-, and postnatal trauma. (Johansen et al., 2002).

Johansen et al. (2002) proposed a theory of ADHD behaviour explained by dysfunctioning dopamine systems causing dysfunctioning reinforcement and extinction process.

### **3.6.1 Reinforcement and extinction and the role of dopamine**

*The meso-limbo-cortical dopamine branch:*

Phasic and tonic dopamine activities seem to play significant roles in reinforcement and extinction. A dysfunctioning meso-limbo cortical dopamine branch in ADHD may alter reinforcement and extinction signals hence the neurobiological foundation of the altered reinforcement processes is repeatedly suggested as one factor in ADHD symptomatology (Sagvolden, 1999)

### **3.6.2 Altered reinforcement processes:**

It is argued that the delay of reinforcement is shorter in ADHD than in controls without ADHD symptoms, implying that only responses in close proximity to the delivery of the reinforcer will be effective in ADHD. Compared to non-ADHD children, a reduced tonic dopamine level in children with ADHD

will require an increased release of dopamine during phasic activation to affect a sufficient number of dopamine receptor-associated ion channels for reinforcement to take place. Also, the phasic dopamine activation as a prediction error signal will require a relatively greater error (e.g. reinforcer value contrast) to release sufficient dopamine for a correction to take place. These arguments are in accordance with the clinical observation that children with ADHD have a motivation problem: stronger and more salient reinforcers are needed to control their behaviour (Johansen et al., 2002; Sagvolden, 1999; Sagvolden & Sergeant, 1998).

*Motor impulsiveness explained as due to altered reinforcement process*

Not only single responses, but also relations between responses (interresponse times, IRTs) are conditioned and maintained by reinforcers. In contrast to the normal delay gradient, only short IRTs may be reinforced and maintained by a short delay gradient. This explains why motor impulsiveness, responses emitted with short interresponse times, is not present in a novel situation, but develops gradually as more reinforcers modify the behaviour (Johansen et al., 2002; Sagvolden, 1999; Sagvolden & Sergeant, 1998).

*Cognitive impulsiveness- due to altered reinforcement process*

Impaired “executive functions” are usually associated with frontal dysfunction. There are well-documented changes in the structure and function of the right frontal cortex in ADHD. Not only dopamine, but also other

neuromodulators affect frontal lobe activity. Although an imbalance in one or more of these systems will have an impact on problems with organising own behaviour, the emphasis is on the role of dopamine system through its close connection to reinforcement and extinction. The importance of reinforcement is supported by the fact that children with ADHD are not always cognitively impulsive as they temporarily manage to plan ahead, organise themselves, and remember important things, if potent and frequent reinforcers maintained this behaviour (Johansen et al., 2002; Sagvolden, 1999; Sagvolden & Sergeant, 1998).

*Impaired sustained attention as explained due to altered reinforcement processes*

Normally, effects of reinforcers will bridge the time interval between two reinforcers. This will elicit associations between discriminative stimuli present throughout this interval, responses emitted, and reinforcer (i.e. the three-term contingency). An abnormally steep and short delay gradient will result in an apparently less consistent relation between the factors in the three-term contingency, causing poor stimulus control and an impaired “sustained attention” in ADHD (Johansen et al., 2002; Sagvolden, 1999; Sagvolden & Sergeant, 1998).

*Altered extinction process*

Procedurally and behaviourally, extinction is defined in relation to reinforcement. Neurobiologically, however, reinforcement and extinction may be separate processes associated with different aspects of dopamine activity. Dopamine dysfunction may lead to reduce tonic dopamine activity in ADHD.

Omission of a predicted reinforcer (i.e. extinction) is normally signalled by a depression in tonic dopamine activity. An abnormally low tonic dopamine activity may thus cause a failing extinction signal due to a “floor” effect. In this perspective, acquired responses are not subjected to extinction, but accumulate as a function of exposure to different reinforcement contingencies. This view is consistent with studies finding excessive responding during extinction in children with ADHD. It is also consistent with studies showing that children with ADHD are not hyperactive in novel situations (Johansen et al., 2002; Sagvolden, 1999; Sagvolden & Sergeant, 1998).

*Overactivity explained as due to failing extinction.*

Altered reinforcement processes characterized by a shorter delay gradient in ADHD will not itself generate the gradually developing overactivity. It is hypothesised that ADHD overactivity is acquired and maintained by a combination of scheduled and unscheduled (“intended” and “unintended”) reinforcers and failing extinction increasing the frequency of acquired responses. The deficient extinction process will lead to an accumulation of responses, which may be seen as excess motor activity where no reinforcer can be identified (Johansen et al., 2002; Sagvolden, 1999; Sagvolden & Sergeant, 1998).

*The Nigro-striatal dopamine branch*

*Impaired timing and force regulation of muscle groups:* Children with ADHD may show several motor problems: longer and more variable reaction times,

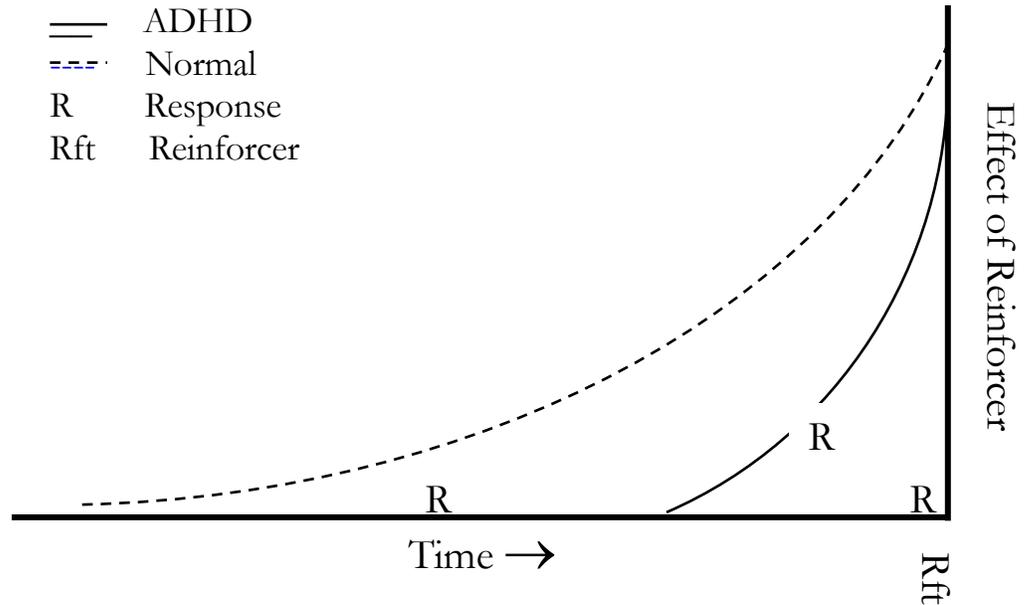
increased variability in speed and less accurate responses re-engagement, and impaired orienting responses an increase number of responses with very long reaction times. ADHD children with a pervasive problem are more likely to show language and motor delays and to have an onset in the first two years of life. A dysfunctioning nigro-striatal dopamine branch will cause several “extrapyramidal” symptoms (neurological “soft signs”) associated with ADHD in the form of impaired timing and force regulation of muscle groups: poor motor control (clumsiness), longer reaction times, poor response timing, abnormal control of eye saccades, poor handwriting, poor correlation of the activity of different body parts, etc. Thus, findings previously attributed to response disinhibition due to frontal – lobe dysfunction may rather be due to impaired motor control associated with dopamine dysfunction of the neostriatum.

Compared to children without ADHD, children with ADHD will:

- Be more impulsive, seen in a higher frequency of short inter-response time
- Have more problems in learning, especially when the reinforcement schedule is lean
- Respond more
- Show more variable behaviour
- Have poor motor control

(Johansen et al., 2002; Sagvolden, 1999; Sagvolden & Sergeant, 1998).

**Fig. 3.5 Delay of reinforcement gradient (Graph from (Johansen, Aase, Meyer, & Sagvolden, 2002))**



### 3.7 Neuropsychology

Neuropsychological approaches have been employed in an attempt to isolate the cognitive deficit or deficits underlying ADHD as to improve the diagnosis and implement more targeted treatments.

ADHD is associated with a myriad of deficits on neuropsychological measures, most notably those assessing executive functions. Denckla (1996b), defines executive functions as control processes involving inhibition and responding, allowing the individual to initiate, sustain, stop and shift. Also associated with executive functions are the abilities to prioritize, organize, and strategize (Denckla, 1989). The self-directed actions occurring during the delay in

the response constitute the executive functions, which are more covert, privatised forms of behaviour (Denckla, 1996b; Sagvolden & Sergeant, 1998; Taylor et al., 1998)

Executive functioning is also defined as the ability to maintain an appropriate problem-solving set for the attainment of a future goal. This includes such functions as: intention to inhibit a response, defer to respond to a future moment, strategic planning and mental representation of a task. Executive functions are a collection of varying abilities that involve regulatory control over thought and behaviour (Denckla, 1996b).

Clear deficits on performance of some neuropsychological tests have been reported for children with ADHD. (Barkley, 1997a; Swanson et al., 1998c). These include impaired performance on tests such as the Wisconsin Card Sorting Test, the Tower of London, the Stroop Interference Test, and several motor tests, (Schmitz et al., 2002; Smith, Taylor, Rogers, Newman, & Rubia, 2002).

Clinical data about executive functioning can be obtained by observing an individual 's ability in solving natural problems and assessing their flexibility when face with a changing routine (Lezak, Howieson, & Loring, 2004).

In this study executive functioning would refer to cognitive and motor impulsiveness (Johansen et al., 2002), as the term EF is very broad.

In the model of Johansen et al. (2002) problems like speed variability, and increased reaction times have been described as evidence of impaired executive functions.

The focus of this study is the dopamine branches projecting to the frontal cortex and the dorsal striatal structures. Impairment in these areas lead to impaired 'executive functions and poor motor control (Sagvolden & Sergeant 1998; Seidman, Biederman, Monuteaux, Doyle, & Faraone, 2001).

The frontal cortex receives a major dopamine input from the ventral tegmental area. During development, learning mediated by the meso-limbic dopamine branch will be an important input for the acquisition of components necessary for planning and organisation of behaviour (Carrey, 2001). Thus, both a dysfunctional meso-cortical dopamine and a dysfunctional meso-limbic branch will contribute in the development of cognitive impulsiveness.

Frequent and potent reinforcement helps children with ADHD to plan ahead, remember important things and organise themselves. This model therefore, predicts that symptoms of cognitive impulsiveness as manifested by lack of behavioural inhibition, poor time management, planning ahead, and impairment in working memory, are mediated by a shorter than normal delay of reinforcement gradient (Johansen et al., 2002). Continuous release of dopamine reinforces previous behaviour (Johansen et al., 2002; Sagvolden, 1999). For proper motor control, especially fine motor functions, a optimal levels of

dopamine are also required. In this study the fundamental underlying behavioural changes were subjected to behavioural analysis in Tshivenda speaking primary school children.

### **3.8 Summary and Conclusion**

A variety of factors are important when considering the neurobiological background of ADHD. These include neuroanatomy, neurochemistry, genetics, and neuropsychology. Recent investigations provide converging evidence that phenotype of ADHD is characterised by neuropsychological deficits in executive functions and reduced size in neuroanatomical regions in the frontal lobes and basal ganglia. Molecular genetic studies have shown that diagnosis of ADHD is associated with polymorphisms in some dopamine genes (the dopamine D4 receptor gene and the dopamine transporter gene).

The assessment of ADHD Screening, Interviews and Tests will be discussed in the next chapter.

## *Chapter 4*

### ASSESSMENT: SCREENING, INTERVIEWS AND TESTS

#### **4.1. Introduction**

The identification and assessment of children with ADHD can present a challenge to clinicians for several reasons. Firstly, there is no single objective measure of ADHD that has acceptable diagnostic validity. ADHD is defined behaviourally and clinicians rely primarily on the subjective report of the caregivers and direct observations of the child. Second, the behaviours associated with ADHD are seen in many children. Some studies have shown that 30 to 48% of boys and 14 to 42% of girls in the general population displayed behaviours consistent with the primary features of ADHD by parents or teachers report (i.e., overactivity, restlessness, short attention span, and distractibility (Barkley, 1997a; Swanson et al., 1998c; Werry & Quay, 1971). However, the mere presence of ADHD symptoms is not sufficient for the diagnosis, the DSM-IV-TR (American Psychiatric Association, 2000) criteria for ADHD should be met. Thirdly, the situational specificity of children's behaviour and the degree of inconsistency of behaviour displayed by children with ADHD across settings will likely result in a low level agreement among informants (i.e., parents, teachers, clinicians, observers, and child) (Barkley, 1998). Lastly, diagnosing ADHD is difficult due to

the possibility that some other primary problem is producing the ADHD symptoms (Brown, 2000; Pary, Lewis, Matuschka, & Lippmann, 2002).

To meet these challenges, standards of practice in the assessment of ADHD recommend a comprehensive approach, involving multiple methods, informants, and discipline (Barkley, 1998; Schaughency & Rothlind, 1991; Brown, 2000).

In each of these standards, the clinical interview, the medical examination, and objective testing and scoring of the behaviour rating scales are recommended (Meyer & Aase, 2003).

A multi-faceted approach has the advantage that limitations of a single assessment method are overcome (Barkley, 1998; Brown, 2000).

#### **4.2. Culturally-sensitive assessment**

Currently most assessment methods and treatment interventions are developed, tested and applied in Western countries. Research is just on the verge of investigating the applicability of both assessment methods and interventions in other parts of the world (Meyer & Aase, 2003).

The recognition of culture and ethnicity are extremely important in the development of Attention-Deficit/Hyperactivity Disorder, as culture shapes the environment in which behaviour is defined as inattentive, impulsive or hyperactive. This is not to say that ADHD is just a matter of cultural definition.

ADHD is defined as a neurologically based, genetically transferred, developmental disorder. As such, ADHD is expected to be present all over the world, but cultural norms and rules will modify how the disorder is manifested. It is therefore essential that the ethnic, cultural and language factors be taken into account in considering the development, manifestation, diagnosis and treatment of ADHD (Meyer & Aase, 2003).

Important factors in psychometric assessments are the validity and reliability of the instruments used. Assessment tools should in all cases be valid and reliable in the cultural context within which it is being administered (Anastasi & Urbina, 1997).

#### **4.3 The clinical interview**

The clinical interview is the mostly widely used method in the assessment process for ADHD. Structured interviews such as the Diagnostic Interview Schedule for Children (DISC) (Shaffer et al., 1993; Shaffer, Fisher, Lucas, Dulcan, & Schwab-Stone, 2000) and the Semi-structured Clinical Interview Schedule for Children and Adolescents (McConaughy & Achenbach, 1994) are constructed around specific diagnostic criteria and have relatively high reliabilities (Edwards, 2000).

The maternal and child interview, although often criticized for its unreliability and subjectivity, is an important part of the evaluation of the child

since parents provide the most ecologically valid information concerning the child's difficulties (Barkley, 1998), while children, particularly over the age of 10, can reliably report on their behaviour (Edwards, 2000).

There are standardized interviews for the assessment of child and adolescent psychopathology. The most current, standardized interview is the Diagnostic Interview Schedule for Children, which has a parent version (DISC-P) and a child version (DISC-C) (Shaffer et al., 1993; Shaffer et al., 2000). These have the advantages of being based directly on the DSM-IV and permit the clinician to make an accurate diagnosis of Attention-Deficit Hyperactivity Disorder, Oppositional Defiance Disorder or Conduct Disorder. Their disadvantage is that they are time consuming and cumbersome.

Teacher interviews provide important information about the child's behavioural symptoms, social behaviour and academic performance (Barkley & Murphy, 1998; DuPaul & Stoner, 1994; Sattler, 1992).

#### **4.4. The Medical Examination**

The medical examination should ideally comprise of the medical interview, a physical examination and laboratory tests (Barkley, 1998).

##### **4.4.1. The Medical Interview**

A more detailed focus is made on a thorough review of the child's genetic background, pre- and peri-natal events, and developmental and medical history as

well as the child's current health, nutritional status, and gross sensory-motor development (Barkley, 1998).

There are three major purposes of the medical interview (1) it focuses on differential diagnosis of Attention-Deficit Hyperactivity Disorder from other medical conditions, (2) it thoroughly evaluate any coexisting conditions that may require medical management, and (3) it determines whether there are physical conditions that are contra-indications for treatment with medications (Barkley, 1998).

#### **4.4.2. Physical Examination**

Routine physical examinations of ADHD children are frequently normal though they are of little help in diagnosing the condition or suggesting its management. However, the physician needs to rule out the rare possibility of visual or hearing deficits, which may give, rise to ADHD like symptoms. The neurological examination is often used to look for signs of previous central nervous system insult or a progressive neurological condition, abnormalities of muscle tone, and a difference in strength, tone, or deep tendon reflex response between the two sides of the body (Barkley, 1998).

Given the considerably greater distress ADHD children present to their caregivers, their risk of being physically abused would seem to be higher than normal. Thus greater attention by physicians to physical or other signs of abuse during clinical examination is therefore required (Barkley, 1998).

#### **4.4.3. Laboratory tests**

Some research studies have used a variety of physical, physiological and psychophysiological measures to assess potential differences between ADHD and other clinical or control groups of children, although some of these studies have demonstrated such differences, as in increased cerebral blood flow to the striatum or diminished orienting galvanic skin responses, none of these laboratory measures are of value in the diagnostic process as yet (Barkley, 1998; Kaplan & Saddock, 1998).

Parents, teachers, or even other mental health professionals are sometimes misled by reports of such findings or by the conclusion that ADHD is a biologically based disorder and they frequently ask for their children to be tested medically to confirm the diagnosis. At this moment, no such tests exist. Consequently laboratory studies, such as blood work, urine analysis, chromosome studies, electroencephalograms, averaged evoked responses, magnetic resonance imaging, or computerised axial tomograms should not be used routinely in the evaluation of ADHD children. Only when the medical and developmental history or physical examination suggest that a treatable medical problem exists, such as a seizure disorder, or that a genetic syndrome is a possibility, would these laboratory procedures be recommended, and yet these cases are quite rare (Barkley, 1998).

#### **4.5. Behaviour Rating Scales**

Rating scales can also provide more reliable and objective data about the child. There are several scales with excellent psychometric properties available for the assessment of ADHD symptoms (e.g. hyperactivity/impulsivity and inattention), general competence in social situations, common forms of child psychopathology, and behaviour problems specific to home or school environments (Brown, 2000; Rapport, Chung, Shore, Denney, & Isaacs, 2000).

##### **4.5.1 Teachers and Parents Rating Scales**

Because ADHD symptoms are well developed by school age and are often most clearly and consistently observed in the school and classroom environment, the teacher observations are believed to be an essential resource in the clinical evaluation of ADHD. Teacher rating scales were found to be valuable research tools (American Academy of Pediatrics, 2000; Wolraich, Hannah, Pinnock, Baumgaertel, & Brown, 1996).

In the teacher form, the teacher evaluates and describes the behaviour of the child. The teacher should also mention how the behaviour of that particular child interferes with the activities, during the past six months (Barkley & Murphy, 1998).

In the parent form, the parent evaluates and describes the behaviour of the child. The parent should also mention how the child's behaviour interferes

with the child's ability to function in life activities, during the past six months (Barkley & Murphy 1998).

Common rating scales are:

#### *4.5.1.1 Parent/Teacher Disruptive Behaviour Disorder Rating Scale*

The Disruptive Behaviour Rating Scale (DBD) (Pelham, Jr., Gnagy, Greenslade, & Milich, 1992; Pillow, Pelham, Jr., Hoza, Molina, & Stultz, 1998) which is used in this study is used for screening children with ADHD. This scale was translated into Tshivenda, Northern Sotho, Afrikaans, English and Tswana. Internal consistency measures and norms for these language groups have been established (Meyer et al., 2004). The DBD will be discussed in detail in chapter 6 under measuring instruments.

#### *4.5.1.2 The Child Behaviour Checklist (CBCL)*

Achenbach & Edelbrock, (1983); Achenbach, Howell, Quay, & Conners (1991) Achenbach (1991) developed the CBCL. The CBCL comprises of 138 items, 20 assess social competence, and 118 items comprise the Behaviour Problem Scale. The Social Competence Scale generates three scores: Social (friendship, organisations, etc), School (performance, problems etc), Activities (sports, hobbies, etc) it takes 15-20 minutes to complete. The CBCL is used to assess social competence and psychopathology in children. The attention and aggressive scales of the instruments are applicable to children with ADHD, CD,

& ODD. The CBCL has an inter-rater, test-retest reliability, as well as internal consistency (Achenbach & Edelbrock, 1983).

#### *4.5.1.3 The Conners rating scales*

##### *4.5.1.3.1 The Conners Teacher Rating Scale (CTRS)*

The Conners Teacher Rating Scale (CTRS) was developed by Keith Conners (Conners, 1969). It consists of 28 items with several questions that collect demographic information from the respondent. It uses the following 4-point rating scale: *not at all present; just a little present; pretty much present; and very much present*. Both the parent and the child's teacher complete the CTRS. A discrepancy score determined from the two questionnaires and a determination should be made whether the symptoms showed by the child proportionate with ADHD (Conners & Jett, 1999).

##### *4.5.1.3.2 Conners Abbreviated symptoms Questionnaire (ASQ)*

The Conners Abbreviated symptoms Questionnaire (ASQ) is used to diagnose children with hyperactivity and to assess changes in hyperactivity and conduct problems particularly on children who are on stimulant drug therapy. It consists of 10-item rating scale for screening purpose to identify hyperactive children. The ASQ has been found to over identify normal children and disproportionately identifies children who are aggressive and hyperactive, and under-identifies distractible children when rated by teachers (Conners, Sitarenios,

Parker, & Epstein, 1998a; Conners, Sitarenios, Parker, & Epstein, 1998b; Conners, 1998).

#### **4.5.2 Adaptive behaviour scales and inventories**

Roizen, Blondis, Irwin, and Stein (1994) developed the Vineland Adaptive Behaviour Inventory. It includes (1) self-help skills, such as bathing, dressing, feeding and toilet requirements, as well as telling and using time and understanding and using money; (2) interpersonal skills, such as sharing, cooperation and trust; (3) motor skills, such as fine motor (buttoning, drawing, printing, etc) (4) communication skills; and (5) social responsibility, such as degree of freedom permitted within and outside the home, running the errands and performing chores (Roizen et al., 1994).

#### **4.5.3 The Home and School Questionnaires**

The Home situations questionnaire developed by Barkley (1997c) and Barkley & Murphy (1998) in a form given to parents to evaluate the pervasiveness and the severity of children's behaviour problems across 16 different home and public situations, whereas the school situations questionnaire is a form given to teachers to evaluate the pervasiveness and the severity of the children's behaviour problems across 12 different situations.

#### **4.5.4 Advantages of behaviour rating scales**

Behaviour rating scales have the capacity of gathering information from informers with many years of experience with the child across diverse settings

and circumstances. Behaviour Rating Scales permit the collection of the data on behaviours that occur extremely infrequently and are likely to be missed by in vivo measures. These kinds of rating scales are inexpensive to administer and are not time consuming to complete. They may have normative data for establishing the statistical deviance of child behaviour rating scales. Behaviour rating scales exist in a variety of forms focusing on a diversity of dimensions of child psychopathology, incorporate different opinions of the important people in the child's natural environment who are responsible for the care, management, and the treatments the child receive (Barkley, 1997a; Barkley, 1998; Barkley & Murphy 1998).

#### **4.6. Psychological tests**

Researchers have made a great progress over the past decade by developing objective tools for assessing ADHD symptoms in children (Barkley, 1998).

Psychological tests can be used in a clinical setting but not to replace other sources of information but to supplement it.

In research studies, various neuropsychological tests, such as the Wisconsin Card Sorting Test (WCST), the Tower of London (ToL), the Go-no-go test, the Stop task, the TOVA, Gordon Diagnostic System (GDS), and the Stroop Word Colour Test were able to differentiate between ADHD and control

groups, although such differentiation is still not sufficient evidence on which to base a diagnostic process (Barkley, 1998; Lezak et al., 2004).

#### **4.6.1 Task for interference control**

The Stroop Word Colour Test (Stroop, 1935) is a speed test measuring the ability to suppress or inhibit habitual responses in the presence of salient conflicting information. The subject first reads a repeating list of colour names (“red, blue, green”) printed in black; then names of the colour of the repeating series of blocks of “X”s printed in those colours; and finally, the colour of the ink must be named when it conflicts with the colour name printed. This last condition, interference task, is assumed to be sensitive to failure to inhibit habitual responses (i.e., to read the colour name) and to maintain task focus.

#### **4.6.2 Task of response inhibition/ impulsivity**

The Gordon Diagnostic System (GDS) vigilance Task (Gordon & Mettelman, 1988) is a continuous performance test used to assess sustained attention and ability to inhibit inappropriate responses. The vigilant sub-test requires continuous attention to a computer screen for 9 minutes during which 45 targets are shown. Stimuli appear at a rate of one per second. The child is told to respond by pressing a button whenever the number 9 follows number 1. Three scores are computed: the number of correct responses, the number of omission errors (failing to respond to correct stimuli), and the numbers of commission

errors (responding to incorrect stimuli). It is predicted that DHD children will generate more errors than the non-ADHD children (Douglas, 1983).

#### **4.6.3 The test of Variable of Attention (TOVA)**

The Test of Variable of Attention (TOVA) was developed by Greenberg and Kindschi (Greenberg & Kindschi, 1996). The TOVA is a computerised measure of sustained attention and destructibility. It offers the opportunity to observe the patient cope with a task that requires sustained attention and impulse control (Barkley, 1998).

#### **4.6.7 Tasks of Cognitive Flexibility**

##### *The Multiple VIVI Test*

The VI/VI test was developed by Hall, Sonuga-Barke, and Sagvolden, (1997). This test measures abilities, which are deficient in ADHD. This test is used to measure shorter delay-of reinforcement gradient and deficient extinction in the mesolimbic branch of the dopamine system. The child should solve a task by pointing at one of two targets presented. No instructions are given. Correct solutions are rewarded, on the average every 15 seconds in one presentation (blue background) and on the average every 45 seconds in another presentation (yellow background) by the presentation of cartoons on the screen. Children with a short delay of reinforcement gradient will manifest this only when the reinforcement of a correct response is delayed.

#### *4.6.8 Tower of London (ToL)*

Krikorian, Bartok, and Gay, (1994) developed the ToL. The ability to plan is a component of executive problem solving thought to be central construct assessed by the TOL, while other constructs for example, procedural memory are believed to have a lesser effect on the ToL performance (Culbertson & Zillmer, 1998a; Culbertson & Zillmer, 1998b). This task requires the subject to plan ahead to determine the order of moves necessary to rearrange three coloured rings or beads from the initial position on two of three upright sticks to a new set of predetermined positions on one or more of the sticks. Level of the difficulty of the test items depends on number and complexity of sub goals required to achieve the desired arrangement.

#### *4.6.9 Wisconsin Card Sorting Test*

The WCST (Berg, 1948; Grant & Berg, 1948) is a psychological test that measures frontal lobe (executive) functioning, originally designed to study “abstract behaviour” and “shift of set”. The test has proved to be useful to neuropsychologists as it has a high sensitivity to frontal lobe dysfunction (Lezak et al., 2004). This test will be described in details in the chapter on the methodology, chapter 6.

### **4.7 Conclusion**

Assessment is an integral part in the diagnosis of ADHD. ADHD rating scales accurately distinguish between children with ADHD and children without

ADHD even without a clinical diagnosis of ADHD when given either to parents or teachers (American Academy of Pediatrics, 2000).

In research studies, various neuropsychological tests such as the Wisconsin Card Sorting test, Tower of London, TOVA, etc. were able to differentiate between ADHD and control groups (Barkley, 1998).

As culture may have an influence on the validity of diagnostic criteria, clinicians from non-western societies should be careful of prematurely copying western assessment methods in their own society (Leung et al., 1996; Meyer & Aase, 2003). It seems that even though various cultures may have different perceptions and interpretations of behaviour, rating scales such as the DBD have been found to discriminate between different kinds of behaviour patterns in a similar way in South Africa as implied in the western diagnostic system of ADHD (Meyer, 1998; Meyer et al., 2004).

## *Chapter 5*

### PROBLEM DELIANATION

#### **5.1 Introduction**

Attention-Deficit/Hyperactivity Disorder is a physiological condition with severe psychological impairments (Barkley, 1997a; Bayliss & Roodenrys, 2000; Meyer, 1998). ADHD is a persistent and severe impairment of psychological development, resulting from a high level of impulsive behaviour, inattentiveness, and overactivity. ADHD affects 3-5 % of primary school children; two to three percent more boys than girls are affected (Barkley, 1998; Swanson et al., 1998c).

Should the symptoms of ADHD not been detected or diagnosed at an early age, ADHD can develop into Oppositional Defiant Disorder and/or Conduct Disorder, at a later stage. These disorders, which place the child at risk for school failure and dropout, substance abuse and sexual promiscuity and as a result HIV/AIDS, juvenile delinquency and criminality (Biederman, Wilens, Mick, Spencer, & Faraone, 1999; Taylor, 1998) are extremely costly, both to society and to the afflicted individuals and their families. An understanding of the biological and psychosocial aetiologies of ADHD, the assessment methods and instruments and diagnosis, will lead to early detection or diagnosis of ADHD symptoms and will help in the development of the most effective intervention

methods, early treatment and better management strategies of ADHD children.

According to the Neuropsychological theory of ADHD (Johansen et al., 2002; Sagvolden, 1999; Sagvolden & Sergeant 1998) ADHD may be to a large extent caused by a dysfunctioning dopamine system. A dysfunctioning mesocortical dopamine branch will cause deficient attention and poor behavioural organisation. The Wisconsin Card Sorting test was used to measure deficit attention responses and poor behavioural organisation. A dysfunctioning nigro-striatal dopamine branch will cause poor fine motor control, the Maze Coordination task and the Grooved Pegboard tests were used to measure poor response timing and poor motor coordination in the nigro-striatal dopamine branch of the dopamine system.

## **5.2 The Problem: assessing ADHD according to a neuropsychological model**

### **5.2.1 Problem Statement**

Current instruments used for the diagnosis of ADHD are culturally biased and have to be translated in many languages. These rating scales are often inaccurate because of the subjectivity of the rater.

A culture free, non-verbal assessment instrument should be able to identify children with ADHD, who are at risk for aggressive and antisocial behaviour, delinquency and possible substance abuse as well as learning problems.

Identifying the children at an early age, and giving early intervention, might prevent later problems.

According to the neuropsychological model of ADHD (Johansen et al., 2002; Sagvolden, 1999) , the mesocortical branch of the dopamine system is responsible for attention responses and behavioural organisation. Damage to this branch due to insufficient dopamine function leads to deficient attention responses and poor behavioural organisation (Barkley, 1997a). Dysfunction in the nigro-striatal dopamine system will result in “extra pyramidal” symptoms such as poor motor control, poor response timing etc, (Johansen et al., 2002).

### **5.2.2 Aim of the study**

The aim of the study is to establish whether children with ADHD are having executive function- and motor deficits caused by a hypofunctioning dopamine system. Tests devised for deficiencies in the areas supplied by the three-dopaminergic branches should be able to show these deficits. The objective of the study therefore, is to submit the neuropsychological theory of ADHD as postulated by Sagvolden et al. (Johansen et al., 2002; Sagvolden, 1999a; Sagvolden & Sergeant 1998) to systematic and experimental testing among Venda primary school children classified as having ADHD. Hopefully, this may lead to the development of a culture-free test battery sensitive to impulsiveness, inattention and motor deficiencies.

## **5.3 Research Hypotheses**

### **5.3.1 Research Hypothesis 1**

ADHD children will perform poorer as compared to their control counterparts on the Wisconsin Card Sorting test that measures deficit attention responses and poor behavioural organisation. The mesocortical dopamine branch supplies the cortical areas implicated. There will be differences in scores between genders. Children with ADHD will perform poorer on the Maze Coordination Test and the Grooved Pegboard, which measures clumsiness and poor motor coordination in the nigro-striatal dopamine branch of the dopamine system. There will be difference in scores between genders.

#### *5.3.1.1 Null Hypothesis 1*

ADHD children will not perform poorer on the Wisconsin Card Sorting test that measures deficit attention responses and poor behavioural organisation. The mesocortical dopamine branch supplies the cortical areas implicated. There will be no differences in scores between genders. Children with ADHD will not perform poorer on the Maze Coordination Test and the Grooved Pegboard, which measures clumsiness and poor motor coordination in the nigro-striatal dopamine branch of the dopamine system. There will be no differences in scores between genders.

*Specific null hypothesis derived from null hypothesis 1*

1. Children with symptoms of ADHD will not have higher perseverative error scores on the Wisconsin Card Sorting Test than children without ADHD. There will be no differences between the genders.
2. Children with ADHD will not make more non- perseverative error scores on the Wisconsin Card Sorting Test than children without ADHD. There will be no differences between the genders.
3. Children with ADHD will not achieve fewer categories on the Wisconsin Card Sorting Test than children without ADHD. There will be no differences between the genders.
4. Children with ADHD will take not longer to finish the Grooved Pegboard than children without ADHD. There will be no differences in performance between genders.
5. Children with ADHD will not have more touches in the Maze Coordination Task than children without ADHD. There will be no differences in performance between genders

**5.3.2 Research hypothesis 2**

There will be significant differences between the scores of the Hyperactive/Impulsive, Inattention and the Combined subtypes of ADHD and the control group without ADHD symptoms on the Wisconsin Card Sorting test that measures deficit attention responses and poor behavioural organisation. The mesocortical dopamine branch is responsible for attention responses and

behavioural organisation. The ADHD subtypes (Inattentive, Hyperactive/Impulsive, and Combined) will also perform poorer on the motor test, the Grooved Pegboard and the Maze coordination Task than a control group without ADHD symptomatology. The nigrostriatal dopamine branch supplies the area that is involved in motor coordination and a hypofunctioning system may cause motor clumsiness.

#### *5.3.2.1 Null hypothesis 2*

There will be no significant differences between the scores of the Hyperactive/Impulsive, Inattention and the Combined subtypes of ADHD and the control group on the Wisconsin Card Sorting test that measures deficit attention responses and poor behavioural organisation. The mesocortical dopamine branch is responsible for attention responses and behavioural organisation. There will be no significant differences between the scores of the no Hyperactive/Impulsive, Inattention and the combined subtypes of ADHD and the control on the Maze Coordination Test and the Grooved Pegboard test that measures clumsiness and poor motor coordination in the nigro-striatal dopamine branch. The nigro-striatal dopamine branch is responsible for fine motor coordination.

#### *Specific null hypothesis derived from null hypothesis 2*

1. The groups of children with the hyperactive/ impulsive, inattentive, and combined subtypes of ADHD will not have higher perseverative error

scores on the Wisconsin Card Sorting Test than the control group without ADHD.

2. The groups of children with the hyperactive/ impulsive, inattentive, and combined subtypes of ADHD will not make more non- perseverative error scores on the Wisconsin Card Sorting Test than the control group without ADHD.
3. The groups of children with hyperactive/ impulsive, inattentive, and combined subtypes of ADHD will not achieve fewer categories on the Wisconsin Card Sorting Test than children without ADHD.
4. The groups of children with the hyperactive/ impulsive, inattentive, and combined subtypes of ADHD will not take longer to finish the Grooved Pegboard than the control group.
5. The groups of children with the hyperactive/ impulsive, inattentive, and combined subtypes of ADHD will have more touches in the Maze Coordination Task than the control group.

A description of the statistical tests employed to accept or reject the hypothesis formulated will be supplied in the next chapter.

RESEARCH METHODOLOGY

**6.1 Introduction**

The neuropsychological theory of ADHD of Sagvolden et al (Johansen et al., 2002; Sagvolden, 1999b; Sagvolden & Sergeant, 1998) will be submitted to a systematic and experimental testing among Tshivenda speaking primary school children. According to this theory, ADHD symptoms are caused by dysfunction in three main dopamine systems 1. The meso-cortical dopamine system, dysfunction in this system will result in cognitive impulsiveness (attention responses and poor behavioural organisation). 2. The nigro-striatal dopamine system, dysfunction will result in “extra pyramidal” symptoms such as poor motor control, poor response timing etc, (Johansen et al., 2002b). Neuropsychological assessments were used to assess the functions of the areas supplied by the above dopamine systems. The Wisconsin Card Sorting test (Berg, 1948) was used to measure deficit attention responses and poor behavioural organisation in the mesocortical dopamine branch. The Maze Coordination Test and the Grooved Pegboard (Matthews & Kløve, 1964) were used to measure poor response timing and poor motor coordination in the nigro-striatal dopamine branch of the dopamine system.

## **6.2 Research Design**

As the subjects could not be randomly assigned to the conditions for the independent variable because they already exhibit the variable, a quasi-experimental research design was used. Children with ADHD (n = 42) were compared with the control group without ADHD symptomatology (n = 42). The children were compared on cognitive impulsiveness and poor motor coordination (dysfunctional meso-cortical and, nigro-striatal dopamine systems respectively).

## **6.3 Sample**

The sample for this study was drawn from Tshivenda speaking primary school children from the former Republic of Venda. They came from rural, semi-rural, and township areas in the Thohoyandou district, with ages ranging from 6 – 13. They were screened for ADHD symptoms by using the Disruptive Behaviour Disorders rating scale (DBD). The DBD was developed by Pelham et al. (Pelham, Gnay, Greenslade, & Milich, 1992; Pillow et al., 1998) and translated and standardized by Meyer et al. (2004) for the different language groups in the Limpopo Province. 900 questionnaires were handed out, 850 were received back and 84 of these formed the sample. Teachers filled in the questionnaires since most parents are frequently difficult to get hold of, as many people in rural areas lack postal addresses and telephones, children are often left in the care of relatives, and not all parents and relatives are literate. School-based completion

of Questionnaire is, therefore, the only feasible method for screening for psychiatric disorders like ADHD (American Academy of Pediatrics, 2004; Wolraich et al., 2003). The 42 children who fulfilled the criteria for inclusion in the ADHD sample were matched with the experimental group for age, sex, and SES. None of the children were receiving psychotropic medications at the time.

Table 6.1 represents the demographic characteristics according to age and gender of the sample.

**Table 6.1. Demographic characteristics (age groups)**

<i>Gender groups</i>	<b>N</b>	<b>Age (in months) Mean</b>
<b>ADHD boys</b>	32	117.53 ± 21.29
<b>girls</b>	10	104.80 ± 14.39
<b>Control boy</b>	32	119.84 ± 22.04
<b>girl</b>	10	116.40 ± 4.33
<b>All groups</b>	84	116.76 ± 19.91

There were no significant differences between the ages of the ADHD and Control groups ( $p = 0.22$ )

Table 6.2 represents the demographic characteristics of the sample when organised according to the ADHD subtypes.

**Table 6.2. Demographic characteristics: age of the subtypes**

<i>Subtype</i>	<i>N</i>	<i>Age (in months)</i> <i>Mean</i>
<b>Hyperactive/Impulsive</b>	10	109.40 ± 24.27
<b>Inattentive</b>	19	115.26 ± 16.00
<b>Combined</b>	13	117.31 ± 23.91
<b>Non-ADHD</b>	42	119.02 ± 19.33

There were no significant differences in age between the ADHD subtypes and the Non-ADHD control group ( $p= 0.57$ ).

Table 6.3 represents the DBD scores according to the gender groups of the sample.

**Table 6.3 DBD scores according to gender groups**

<i>Group</i>	<i>N</i>	<i>Hyperactive/Impulsive</i> <i>Scale</i> <i>Mean</i>	<i>Inattention Scale</i> <i>Mean</i>
<b>ADHD boys</b>	32	17.16 ± 4.95	22.59 ± 3.29
<b>ADHD girls</b>	10	16.10 ± 6.84	21.00 ± 4.92
<b>Control boys</b>	32	5.28 ± 3.49	8.78 ± 4.39
<b>Control girls</b>	10	3.20 ± 2.48	3.60 ± 3.57

The differences in DBD scores for both scales between ADHD- and the control groups were statistically significant ( $p=0.00$ ).

Table 6.4 represents the DBD scores according to subtypes of the sample.

**Table 6.4 Results of the DBD Scores (subtypes)**

<i>Subtype</i>	<i>N</i>	<i>H/I Mean</i>	<i>Inatt. Mean</i>
<b>Hyperactive/impulsive</b>	10	21.10 ± 2.13	16.40 ± 2.41
<b>Inattentive</b>	19	12.26 ± 4.17	24.37 ± 1.57
<b>Combined</b>	13	20.46 ± 2.63	23.54 ± 1.51
<b>Non-ADHD</b>	42	4.79 ± 3.38	7.55 ± 4.73

There were significant differences in the DBD scores between the Hyperactive/Impulsive, Inattentive, and Combined subtypes and the Non-ADHD comparison group on both the Hyperactive/Impulsive ( $p=0.00$ ) and Inattentive scales ( $p=0.00$ ).

## **6.4 Measuring Instruments**

The following measurement instruments were used in this study:

### *6.4.1 Screening device*

The Disruptive Behaviour Disorder rating scale (DBD) developed by Pelham et al. (Pelham, et al., 1992; Pillow, et al., 1998) was used for screening the children, for the presence of and degree of ADHD related symptoms, thus establishing an experimental ADHD group and a control group.

This instrument has been translated, standardized and norms have been established for all the language groups in the Limpopo Province (Tshivenda, Tswana, Tsonga, Northern Sotho, English and Afrikaans) (Meyer et al., 2004).

The DBD consists of 42 items based on the DSM-IV diagnostic criteria (American, Psychiatric Association, 2000), of which 18 measure ADHD. Separate scores are calculated for four sub-scales: two ADHD (Inattention and Hyperactivity/Impulsiveness), ODD (Oppositional Defiant Disorder) and CD (Conduct Disorder). The behaviour is rated on a four point scale: *not at all* (0), *just a little* (1), *pretty much* (2), and *very much* (3). Children with a score of three (“very much”) on at least six items on a sub-scale are considered disordered (Pelham, et al., 1992; Pillow et al., 1998). To score this rating scale, the total points are added up and compared to the cut off points of the 95<sup>th</sup> percentile, which has been identified as clinically significant (Barkley, 1997d; Barkley & Murphy, 1998). The group matched with the ADHD group, who were scoring below the 85<sup>th</sup> percentile was taken as the control group. For the purpose of this study, only the scores on the two ADHD scales were taken into account.

#### *6.4.2 Neuropsychological tests*

Tests were selected according to the model of dopamine dysfunction theory of ADHD (Johansen et al., 2002; Sagvolden, 1999b) According to this model, the mesocortical branch of the dopamine system is involved in attention responses and behavioural organisation. Damage to this branch due to

insufficient dopamine function leads to deficient attention responses and poor behavioural organisation (Barkley, 1997a). The Wisconsin Card Sorting Test is thus used to measure deficits in attention responses and poor behavioural organisations.

The nigro-striatal dopamine branch is associated with motor coordination. Dysfunctioning of this branch will cause poor motor coordination.

#### *6.4.2.1 Frontal lobe functioning (mesocortical dopamine branch)*

##### *Wisconsin Card Sorting Test*

This is a computerized version of the test (Ormond Software Enterprises, 1998), developed by Grant et al. (1948) used to test frontal lobe functions, deficit attention responses and poor behavioural organisation, in which the child is asked to place 128 different cards under four stimulus cards, according to a principle that the child must deduce from the computer feedback given to the child's placement of the cards (Heaton & Pendleton, 1981). The test works on this principle: the correct solution is first the colour, once the subject has figured out this solution, the correct solution then changes without warning, according to form. Thus, the subject must inhibit classifying the cards on the basis of colour and shift to form. Once the subject masters to select by form, the correct solution again changes without warning, this time the principle requires the number of elements. It will later become colour again and so forth (Kolb & Whishaw, 1996; Lezak, Howieson, & Loring, 2004). Measures are done in terms of the number of

trials to first category, the number of categories achieved, and failure to maintain set, frequency of perseverative errors, and frequency of non-perseverative errors (Grodzinsky & Diamond, 1992).

Hodges (1994) and Lezak et al. (2004) state that the number of categories achieved and numbers of perseverative errors are the most widely used scores. For this study a measure of the non-perseverative errors was also calculated as a measurement of inattentiveness.

*Category shifted:*

The sorting rule shifts after every 10 consecutive responses. The rule cycles through colour, form and then number. The number of changes is denoted as categories achieved and most normal subjects should achieve at least 4 shifts of the sorting rule.

*Perseverative responses*

A perseverative response is defined as one that matches the perseverated principle, i.e. a response that would have been correct in the previous stage, e.g. sorting according to colour when the current rule is form. Not all perseverative responses are errors. (Twenty or more perseverative responses indicate impairment). A perseverative error is an incorrect perseverative response. Score above 13 are predictive of brain damage and above 16 of frontal lobe involvement.

### *Non-perseverative errors*

Non-perseverative errors are responses that are incorrect but not perseverative.

The instructions of the Wisconsin Card Sorting test are as follows:

*When the test starts, you will see the screen as shown below. Please sort out the deck of cards, using the mouse, by moving each card to the placeholder below the key card you think it matches, once you have moved a card, you cannot move it again. After each sort move, you will receive a feedback, informing you if the sort was correct or not. Please try to sort the cards correctly. Good*

### *6.4.2.2 Motor Tests (nigro-striatal dopamine branch)*

#### *Grooved Pegboard*

This test consists of a small metal board (10 x 10 cm) that contains a 5 x 5 set of slotted holes angled in different directions. Each peg has a ridge along one side requiring it to be rotated into position for correct insertion (Matthews & Kløve 1964). The child has two trials, the first using the dominant hand and the second using the non-dominant hand, hand dominancy may be determined by requesting the child to hit a nail with a hammer with one hand. Starting with the dominant hand the child will insert each of the 25 pegs into each one of the 25 holes and after that, the same with the non-dominant hand. For every trial, the time is taken and the number of dropped pegs noted (Lezak et al., 2004).

The instructions of the Grooved Pegboard test are as follows:

*“You are now going to put each of these pegs into the holes of this board (show). You can only use one hand. Pick up one peg at a time. Notice that the pegs neither the holes in the board are round. In order to insert it you will have to rotate the pegs so that it fits exactly (show two pegs, let the child try the three next, then remove all five pegs). When I tell you to start, you shall start over here (point to the upper left hole if the child is using the right hand and to the upper right hole if the child is using the left hand), fill this upper row, continue on the next, and so on until the pegs are inserted. Try to be quick. Use only your (dominant/non-dominant) hand”*

#### *The Maze Coordination Test*

This is a test from the Kløve-Matthews Motor Steadiness Battery, developed by Kløve and Matthews (1964). The child has to follow a trail without touching the sides, using a stylus. The stylus is connected to an electric clock and a counter, which records the number of contacts the stylus is making with the side. There is no speed requirement. The main aim of the test is to move the stylus through the maze without touching the sides (Matthews & Kløve, 1964). The child has two trials with each hand, for every trial the touches are recorded. For the first trial the child uses the dominant hand and for the second trial the child uses the non-dominant hand. The total sum of touches and cumulative time

of contact of two trials with the same-side hand are the final scores (Lezak et al., 2004).

The instructions for the Maze Coordination test are as follows:

*“Take this stylus and move it through the maze all the way to here (point). Try to avoid touching the sides (show). Do this with about this speed. (Show by moving the stylus through about ¼ of the maze). You do not have to rush, if you move too quickly you will make more errors. Try to be accurate. Start with your (dominant) hand. Do not rest your arm against anything”*

#### **6.5 Procedure:**

The Provincial Department of Education granted written permission to conduct the research among the primary school children. A letter obtained from the Ethics committee of the University of the North outlining the aim and the purpose of the study was presented to the respective school principals who in turn presented the research project to the teachers. Written consent was obtained from the parents.

Masters students in Psychology who were acquainted with the testing procedures assisted the researcher.

The testing procedure was as follows:

A biographical questionnaire was completed. Children with a history of neurological trauma, psychosis or other severe psychiatric disorders were not included in this study.

Teachers were asked to complete the Disruptive Behaviour Disorder checklist. The ADHD and Control groups were selected. Children were assessed during school time as arranged with the school principal and class teachers. Children came to the testing room in groups of 3-4 and they were assessed on a battery of tests including: the Wisconsin Card Sorting Test, Grooved Pegboard and the Maze Coordination Task in the same order. Before the actual neuropsychological testing started, the children were acquainted with a computer and the use of a mouse.

The total time children spent in the testing room was approximately 1 hour including time for snacks and other activities like mouse-training. Children were tested individually. The instructions for all the tests were given in the child's own language (Tshivenda).

## **6.6 Data Analysis**

The Data was analysed quantitatively by using the statistical computer programme SPSS 11 (SPSS, 2003) and STATISTICA 6 (Statsoft Inc., 2003). Analysis of Variance (ANOVA) was used to investigate possible group differences in raw scores as a function of gender and subtypes. Post-hoc tests

Least Significant Difference (LSD) were used to establish were the differences occurred.

## **6.7 Conclusion**

Research methodologies including the research design, sampling, measuring instruments, procedure and method of data analysis followed in this study were discussed in this chapter. The next chapter will present the results in the form of tables and graphs.

## *Chapter 7*

### RESULTS

#### **7.1 Introduction**

The aim of the study was to establish whether children with ADHD are having executive function- and motor deficits caused by a hypofunctioning dopamine system. Tests devised for deficiencies in the areas supplied by the three-dopaminergic branches should be able to show these deficits. The objective of the study therefore, is to submit the neuropsychological theory of ADHD as postulated by Sagvolden et al. (Johansen et al., 2002b; Sagvolden, 1999; Sagvolden & Sergeant, 1998) to systematic and experimental testing among Venda primary school children classified as having ADHD. Hopefully, this may lead to the development of a culture-free test battery sensitive to impulsiveness, inattention and motor deficiencies.

Identifying the children at an early age, and giving early intervention, might prevent later problems. A battery of neuropsychological tests was used in this assessment. According to the neuropsychological model of ADHD (Johansen et al., 2002; Sagvolden, 1999b) , the mesocortical branch of the dopamine system is responsible for attention responses and behavioural organisation. Damage to this branch due to insufficient dopamine function leads to deficient attention responses and poor behavioural organisation (Barkley, 1997a). The Wisconsin

Card Sorting Test was used to measure deficit attention responses and poor behavioural organisation. Dysfunction in the nigro-striatal dopamine system will result in “extra pyramidal” symptoms such as poor motor control, and poor response timing (Johansen et al., 2002). The Grooved Pegboard and the Maze Co-ordination were used to measure poor motor control and poor response timing.

Test Scores obtained from the administration of these tests were compared for significance difference on performance between the ADHD clinical group and control group, on the bases of gender and subtypes.

The results for each individual test are presented first in a table form then a graphical representation followed by Analysis of Variance and Least Significant Difference (LSD) *post- hoc* follows respectively.

## **7.2 Results of the study**

### **7.2.1 Tests for Cognitive impulsiveness (Meso-cortical dopamine branch): *Wisconsin Card Sorting Test (WCST)***

#### *Descriptive statistics*

Table 7.1 illustrates the descriptive statistics of the results obtained on the WCST according to gender groups.

**Table 7.1 Results of the WCST according to (gender)**

<i><b>Groups</b></i>	<i><b>N</b></i>	<i><b>Perseverative Errors</b></i>	<i><b>Non-Perseverative Errors</b></i>	<i><b>Categories Achieved</b></i>
<b>ADHD boys</b>	32	23.00 ± 16.95	25.66 ± 15.84	3.03 ± 1.53
<b>ADHD girls</b>	10	46.70 ± 30.95	18.70 ± 13.74	1.80 ± 1.23
<b>Control boys</b>	32	10.94 ± 8.72	26.38 ± 10.03	2.86 ± 1.52
<b>Control girls</b>	10	12.80 ± 8.19	26.00 ± 3.77	3.00 ± 1.41

Figure 7.1 illustrates the results on the WCST (perseverative error scores and the non-perseverative error scores) for the genders (ADHD group and Control group).

Figure 7.1

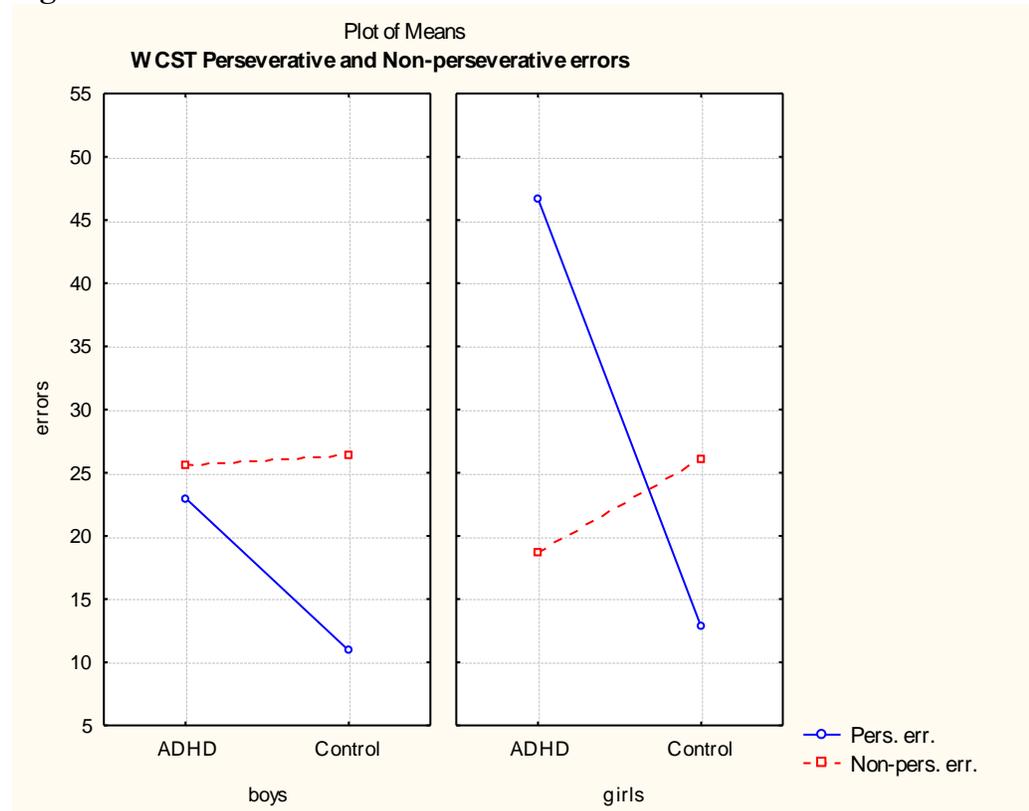
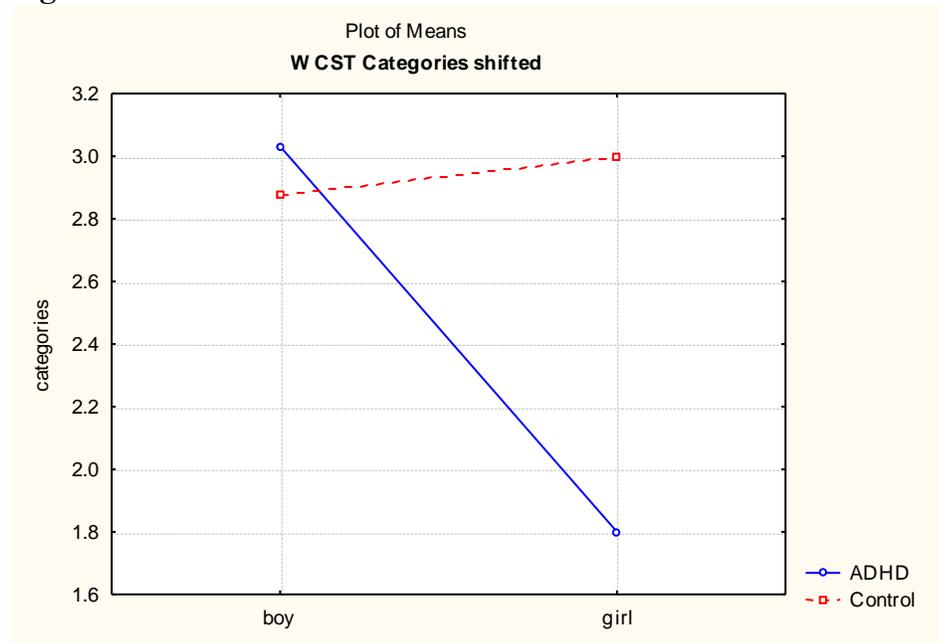


Figure 7.2 illustrates the results on the WCST (Categories Shifted) for gender (ADHD group and Control group)

**Figure 7.2**



*Analysis of Variance*

Table 7.2 gives an illustration of an analysis of variance (ANOVA) for the WCST (Perseverative errors, non perseverative errors and Categories Shifted).

**Table 7.2 Results of the Analysis of variance (ANOVA): WCST**

Variable	df	F	p
Perseverative. Errors	3, 80	13.75	0.00*
Non-perseverative Errors	3, 80	1.01	0.39
Categories Shifted	3, .80	1.86	0.14

\*p ≤ 0.05

*Post-hoc tests*

*Post-hoc* tests (LSD) test were used to establish where the differences occurred on the perseverative errors. There were no *post-hoc* test on the non-perseverative errors because the differences were statistically not significant ( $p=0.39$ ;  $p=0.14$ ).

Table 7.3 represents the *post-hoc* Least Significant Difference test WCST (perseverative errors) for the ADHD groups, according to gender.

**Table 7.3: Results of the *post-hoc* (LSD) test: WCST (Perseverative errors)**

Comparison	$p$
ADHD boys versus control boys	0.00*
ADHD girls versus control girls	0.00*

\* $p \leq 0.05$

The difference in performance between the ADHD group and the non-ADHD control was statistically significant for both boys and girls ( $p=0.00$ ).

**Subtypes:**

*Descriptive Statistics*

Table 7.4 illustrates the descriptive statistics for WCST (perseverative errors, non-perseverative errors and categories shifted) according to subtypes

**Table 7.4: Results of the WCST according to subtypes**

Subtype	N	Pers.Errors	Non-Pers.Errors	Categories Shifted
Hyp/imp	10	42.10 ± 37.35	22.00 ± 20.47	2.00 ± 1.56
Inattentive	19	23.95 ± 13.94	26.47 ± 13.78	2.79 ± 1.44
Combined	13	25.15 ± 16.75	21.92 ± 14.27	3.23 ± 1.59
Normal	42	11.38 ± 8.53	26.26 ± 8.90	2.90 ± 1.48

Figure 7.3 illustrates the results on the WCST (perseverative error and the non-perseverative error scores according to Subtypes.

Figure 7.3

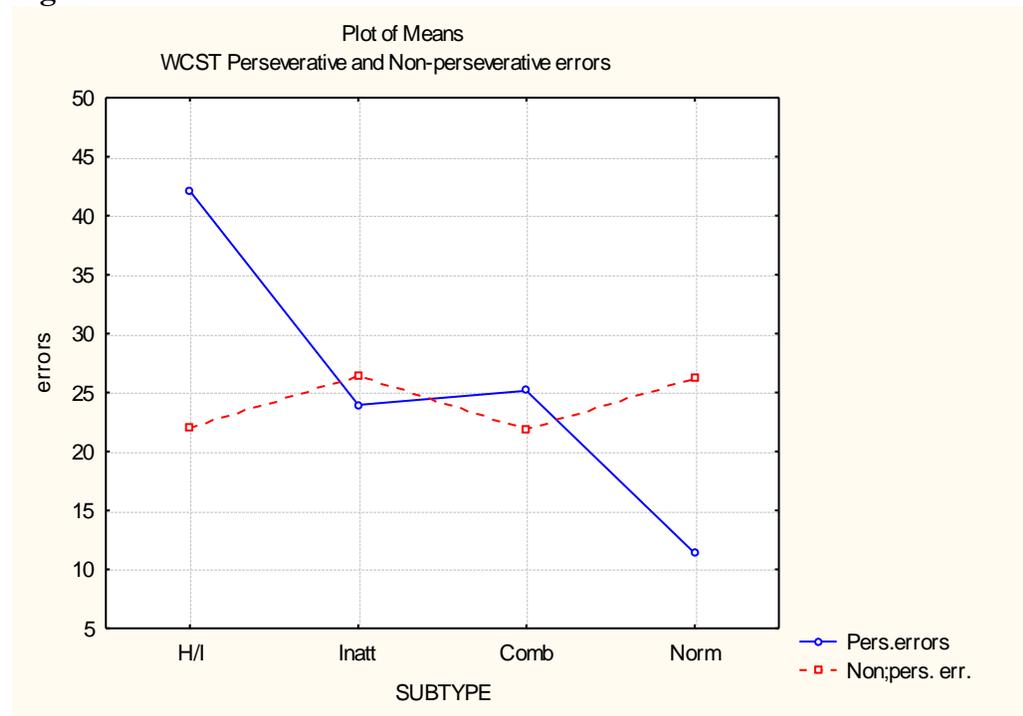


Figure 7.4 is an illustration of the results for Categories Shifted on the WCST for the different subtypes

**Figure 7.4**

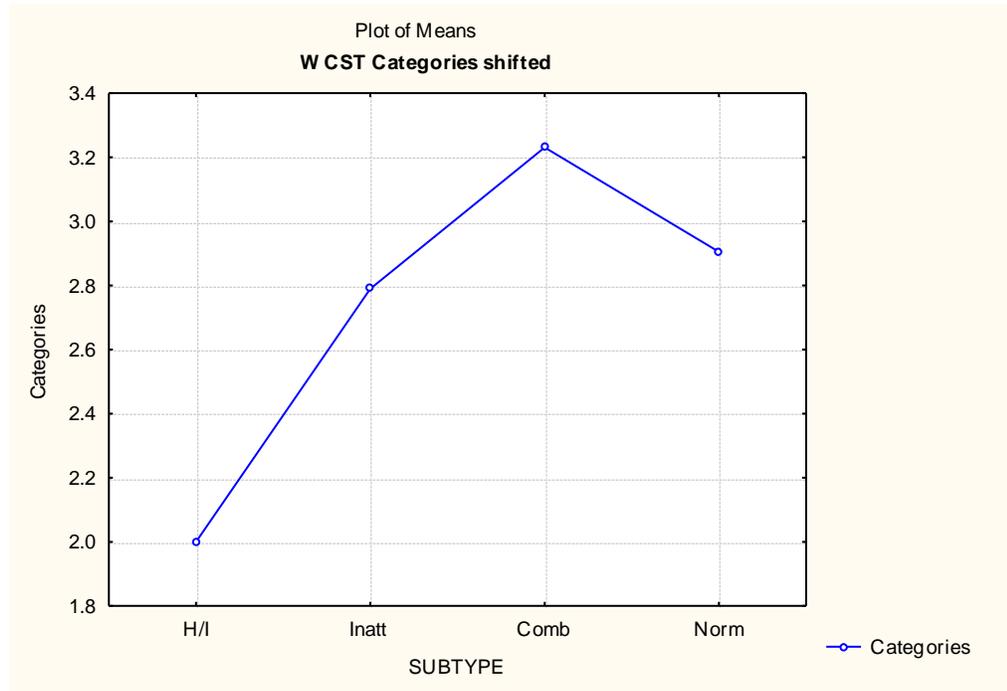


Table 7.5 gives an illustration of the analysis of variance (ANOVA) on the WCST (Perseverative errors, Non-Perseverative Errors and Categories Shifted) according to subtypes

**Table 7.5 ANOVA results of the WCST according to subtypes**

<b>Variables</b>	<b>df</b>	<b>F</b>	<b><i>p</i></b>
<b>Pers. Errors</b>	3, 80	10,29	0,00*
<b>Non. Pers.Errors</b>	3, 80	0,66	0,57
<b>Cat. Achieved</b>	3, 80	1.38	0.26

\* $p \leq 0.05$

Table 7.6 represents the results of the *post-hoc* (LSD) test on the WCST (Perseverative Errors) according to subtypes.

**Table 7.6 Results of the *post-hoc* (LSD) test: WCST (Perseverative Errors)**

Comparison	p
Hyperactive/Impulsive versus controls	0.00*
Inattentive versus controls	0.00*
Combined versus controls	0.01*

\* $p \leq 0.05$

The difference in performance was statistically significant for the perseverative errors when the Hyperactive/ Impulsive and the Control groups were compared ( $p=0.00$ ).

The difference in performance was statistically significant for the perseverative errors when the Inattention and the Control groups were compared ( $p=0.00$ ).

The difference in performance was statistically significant for the perseverative errors when the Combined and the Control groups were compared ( $p=0.01$ ).

In this study most the ADHD subjects were found to be deficient only on the perseverative errors of the WCST. No statistically significance was found on the other two variables, non-perseverative errors and categories achieved.

### 7.2.2 Motor tests (Nigro-striatal dopamine branch)

#### *The Grooved Pegboard Test*

##### *Descriptive Statistics*

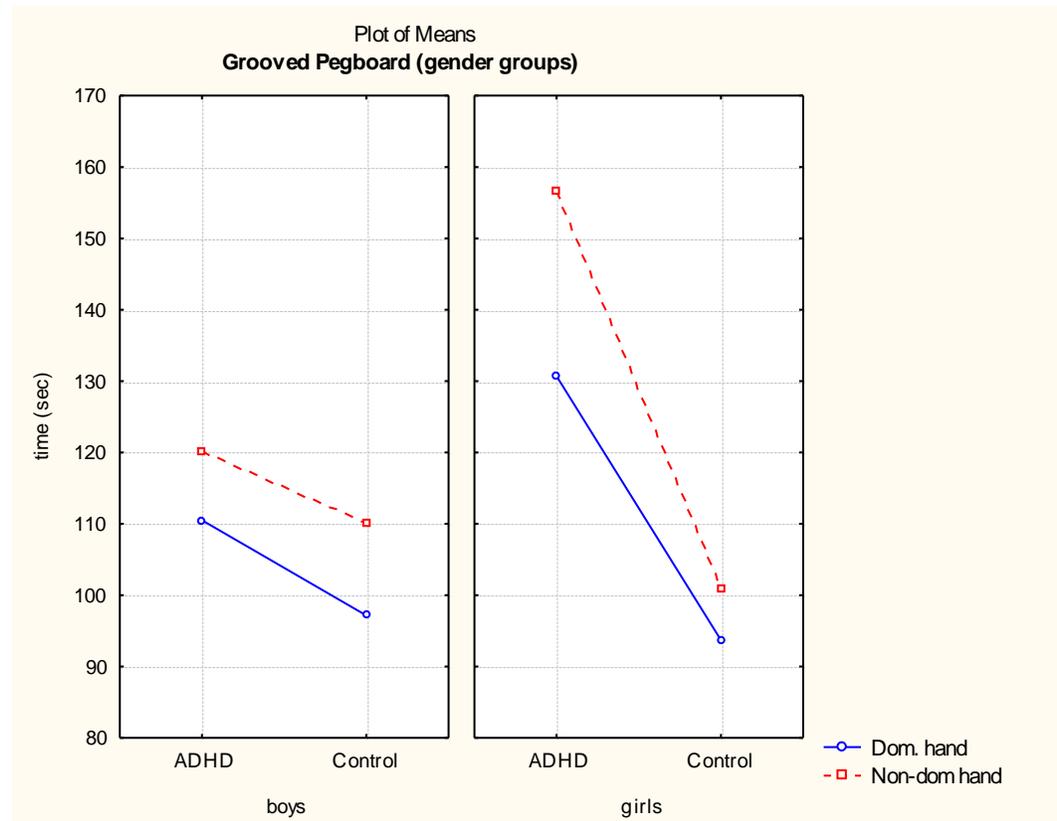
Table 7.7 represents the descriptive statistics for the Grooved Pegboard test (dominant hand and non-dominant hand) according to gender.

**Table 7.7 Results of the Grooved Pegboard Test (gender groups).**

<b>Gender groups</b>	<b>N</b>	<b>Dom. hand</b>	<b>Non-dom. hand</b>
<b>ADHD boys</b>	32	110.50 ± 45.15	120.23 ± 43.92
<b>ADHD girls</b>	10	130.70 ± 73.45	156.60 ± 78.18
<b>Control boys</b>	32	97.16 ± 29.90	110.00 ± 45.27
<b>Control girls</b>	10	93.70 ± 18.52	100.90 ± 21.38

Figure 7.5 illustrates the results of the Grooved Pegboard, for the dominant hand and non-dominant hand for the gender groups.

Figure 7.5



ANOVA:

Table 7.8 represents the analysis of variance (ANOVA) for the Grooved pegboard: dominant and non-dominant hand for the gender groups.

Table 7.8 ANOVA results for the Grooved Pegboard (gender groups)

	df	F	<i>p</i>
Dominant Hand	3, 78	2.01	0.11
Non-Dominant hand	3, 77	2.90	0.04*

\* $p \leq 0.05$

The results show that there was statistical significance for the non-dominant hand only ( $p = 0.04$ ).

*Post-hoc test (LSD):*

Table 7.9 represents the *post-hoc* (LSD) test for the grooved peg-board test according to gender group for the non-dominant hand.

**Table 7.9 *Post- hoc* (LSD) test for the Grooved Pegboard (gender groups)**

<b>Comparison</b>	<b><i>p</i></b>
ADHD boys v. Control boys	0.41
ADHD girls v. Control girls	0.01*

\* $p \leq 0.05$

The difference was statistically significant for the ADHD girls only at  $p = 0.01$ , for the non-dominant hand.

*Subtypes:*

*Descriptive Statistics*

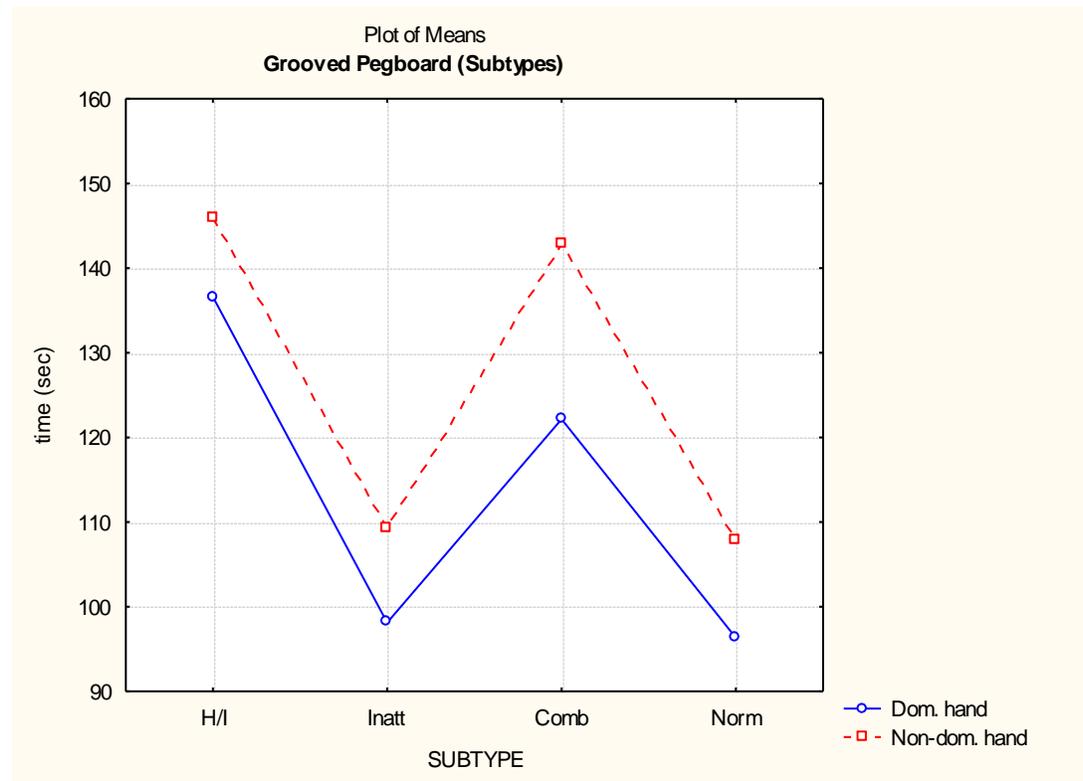
Table 7.10 represents the descriptive statistics of the grooved pegboard test results for the dominant and non- dominant hand according to subtypes.

**Table 7.10 Results of the Grooved pegboard Test (Subtypes)**

Subtypes	N	Dominant hand	Non-dom. hand
H/I	10	136.60 ± 73.78	145.90 ± 79.03
Inatt	17	98.12 ± 37.71	109.24 ± 40.36
Comb	13	122.15 ± 49.01	142.85 ± 47.08
Normal	42	96.33 ± 27.45	107.78 ± 40.69

Fig 7.6 illustrates the plot of the means of the results obtained on the grooved pegboard test for both hands for the different ADHD subtypes.

**Figure 7.6**



ANOVA:

Table 7.11 represents the results of the analysis of variance (ANOVA) for the grooved pegboard test for both the dominant- and non-dominant hands for the ADHD subtypes.

**Table 7.11 ANOVA results for the Grooved Pegboard test (Subtypes)**

	<b>df</b>	<b>F</b>	<b><i>p</i></b>
<b>Dom. hand</b>	3, 78	2.01	0.02*
<b>Non-dom. hand</b>	3, 77	2.90	0.04*

\* $p \leq 0.05$

There were statistically significant differences for the performances on the dominant hand ( $p = 0.02$ ) and the non-dominant hand ( $p = 0.04$ ) among the ADHD subtypes.

*Post-hoc test (LSD):*

Table 7.12 represents the *post-hoc* test (LSD) on the grooved pegboard test (dominant hand and non dominant hand) according to subtypes:

**Table 7.12 results of the Grooved Pegboard *post-hoc* (LSD) subtypes**

<b>Comparison</b>	<i>Dom. hand</i>	<i>Non-dom.hand</i>
<b>H/I v. Control</b>	0.02*	0.03*
<b>Inattentive v. Control</b>	0.88	0.90
<b>Combined v. Control</b>	0.05*	0.02*

\* $p \leq 0.05$

The difference in performance was significant for both hands when the Hyperactive/Impulsive and Control groups were compared ( $p=0.02$ ;  $p = 0.03$ ). The differences in performance between the Combined group and the Control group were also significant for both hands ( $p = 0.05$ ;  $p = 0.02$ ), while there were no significant differences between the Inattentive and the Control groups.

***The Maze Coordination Test***

*Descriptive statistics:*

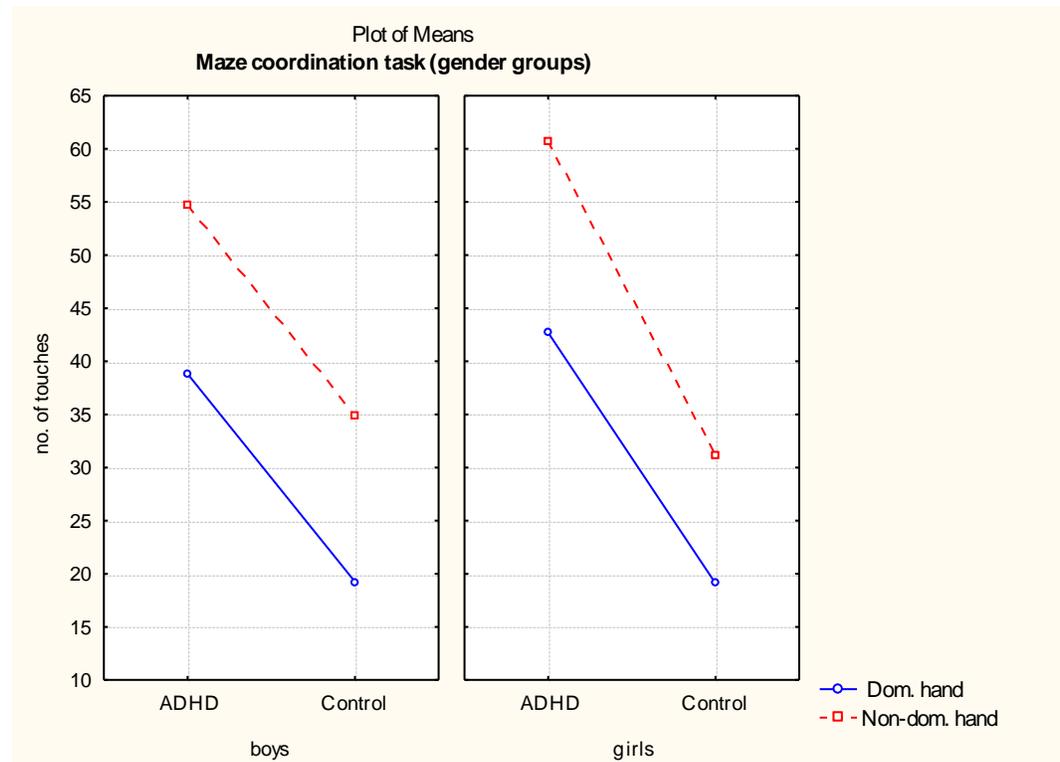
Table 7.13 represents the results obtained on the Maze coordination Test when the ADHD groups were compared with the control groups for both genders.

**Table 7.13 Results of the Maze Coordination Test (Gender)**

<b>Group</b>	<b>N</b>	<b>Dominant hand Means</b>	<b>Non-dominant hand Means</b>
ADHD boys	32	110.50 ± 45.15	120.23 ± 43.92
girls	10	130.70 ± 73.45	156.60 ± 78.18
Control boys	32	97.16 ± 29.90	110.00 ± 45.27
girls	10	93.70 ± 18.52	100.90 ± 21.38

Fig 7.7 illustrates the plot of means for the number of touches on the Maze Coordination Test, for the ADHD groups, according to gender.

Figure 7.7



*ANOVA:*

Table 7.14 represents the results of the ANOVA for the number of touches on the Maze Coordination Test for the ADHD and Control groups, according to gender groups.

**Table 7.14 ANOVA results for the Maze Coordination Test (gender groups)**

	<b>df</b>	<b>F</b>	<b>p</b>
<b>Dominant hand</b>	3, 80	4.47	0.01*
<b>Non-dominant hand</b>	3, 80	4.03	0.01*

\* $p \leq 0.05$

The results show that there was a statistically significant difference in performance for both hands ( $p = 0.01$ ).

*Post-hoc (LSD) test:*

Table 7.15 represents the results of the *post-hoc* test performed on the Maze Coordination Test for the number of touches for both the dominant and non-dominant hand according to the gender groups.

**Table 7.15 Post-hoc (LSD) test for the Maze Coordination Test (gender groups)**

<b>Comparison</b>	<b>Dominant hand</b>	<b>Non-dom.hand</b>
<b>ADHD boys v. Control boys</b>	0.00*	0.01*
<b>ADHD girls v. Control girls</b>	0.04*	0.03*

\* $p \leq 0.05$

There is statistical difference between the ADHD boys and the control group for the dominant hand ( $p = 0.00$ ) and the non-dominant hand. ( $p=0.01$ )

There is also a statistical difference between the ADHD girls and the control group for both the dominant hand (0.04), and the non-dominant hand. (0.03).

*Subtypes*

*Descriptive Statistics*

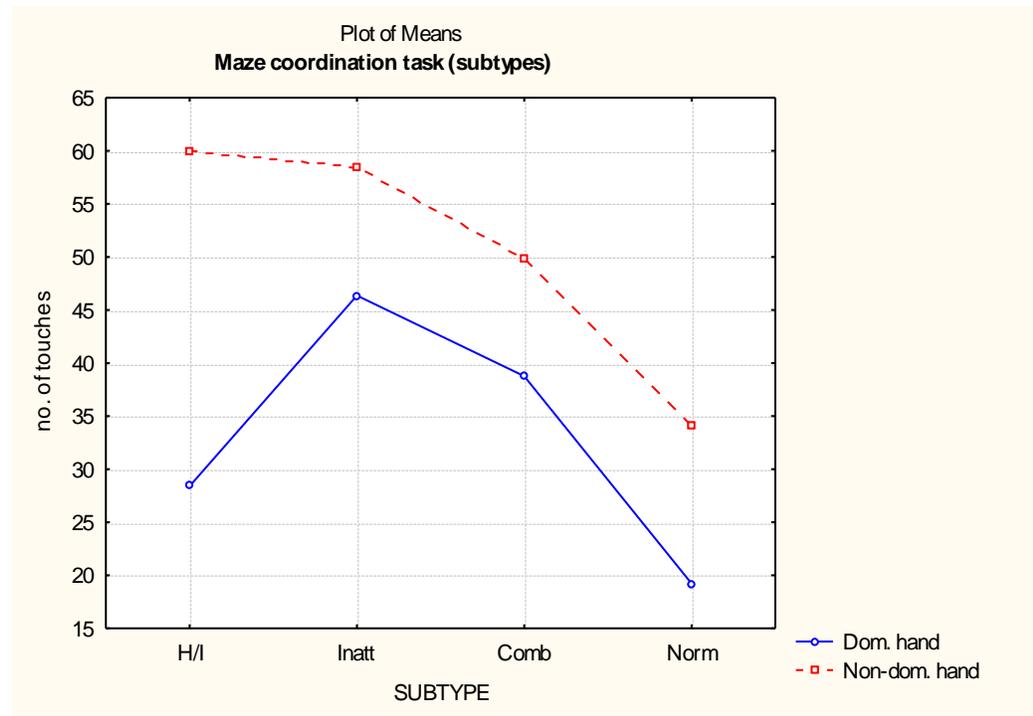
Table 7.16 represents the results obtained on the maze coordination test for the different ADHD subtypes

**Table 7.16 Results of the Maze Coordination Test (subtypes).**

<b>Subtype</b>	<b>N</b>	<b>Dominant hand</b>	<b>Non-dominant hand</b>
<b>H/I</b>	10	28.40 ± 19.77	60.00 ± 20.77
<b>Inattentive</b>	19	46.37 ± 39.81	58.37 ± 44.13
<b>Combined</b>	13	38.77 ± 30.81	49.92 ± 32.95
<b>Normal</b>	42	19.21 ± 13.10	34.05 ± 20.10

Figure 7.8 illustrates the plot of means for the number of touches on the Maze coordination with both the dominant- and non-dominant hand for the ADHD subtypes.

Figure 7.8



ANOVA:

Table 7.17 represents the results of the ANOVA performed on the number of touches on the Maze Coordination test.

Table 7.17 ANOVA results for the Maze Coordination Test (subtypes)

	df	F	<i>p</i>
<b>Dom. hand</b>	3, 80	5.69	0.00*
<b>Non-dom. hand</b>	3, 80	4.19	0.01*

\* $p \leq 0.05$

The ANOVA results show that there were statistically significant differences in performance on the Maze Coordination Task between the groups for both the dominant ( $p = 0.00$ ) and non-dominant hand ( $p=0.01$ ).

*Post-hoc test (LSD):*

Table 7.18 represents the results of the *post hoc* test (LSD) for the Maze coordination test for the subtypes for both the dominant and non-dominant hand.

**Table 7.18 Results of the *post hoc* (LSD) for the Maze Coordination Test (subtypes)**

Subtypes	Dom. hand p	N-Dom. hand p
H/I v. Normal	0.31	0.01*
Inattentive v. Normal	0.00*	0.00*
Combined v. Normal	0.02*	0.09

\* $p \leq 0.05$

The difference in performance was significant for the non-dominant hand when the Hyperactive/Impulsive and Control groups were compared ( $p=0.01$ ), while there were no significant differences between the Hyperactive/Impulsive and controls for the dominant hand.

The differences in performance between the Inattentive group and the control group were also statistically significant for both hands ( $p=0.00$ ).

The difference in performance was also significant for the dominant hand when the Combined group and the Control groups were compared ( $p=0.02$ ), while there were no significant differences between the Combined subtype and the Controls for the non-dominant hand.

#### **7.4 Hypotheses testing**

##### **Research hypothesis 1**

Based on the research results, the following conclusions about the research hypothesis can be made:

Null Hypothesis 1.1 must be partially rejected: children with ADHD made more perseverative errors than non-ADHD children; however, there was no difference between the genders.

Null Hypothesis 1.2 must be accepted, as the differences in scores between the clinical and control groups, in terms of non-perseverative errors on the WCST, were statistically not significant.

Null Hypothesis 1.3 must be accepted, as the differences in scores between the clinical and control groups, in terms of categories achieved on the WCST was not statistically significant.

Null Hypothesis 1.4 must be partially rejected, children with symptoms of ADHD took longer to finish the grooved pegboard with their non-dominant hand only and only in the case of the girls.

Null Hypothesis 1.5 must be partially rejected; children with ADHD had more touches with their dominant and non-dominant hands than controls, however, there was no difference in performance between the gender groups..

### **Research Hypothesis 2**

Null Hypothesis 2.1 must be rejected, the ADHD subtypes made more perseverative errors than the controls. The difference in number of perseverative errors on the WCST between the subtypes was statistically significant.

Null Hypothesis 2.2 must be accepted, as the differences in the number of non-perseverative errors on the WCST scores between the clinical and the control groups were statistically not significant.

Null Hypothesis 2.3 must be accepted, as the differences in the number of categories achieved on the WCST scores between the clinical and the control groups were statistically not significant.

Null Hypothesis 2.4 must be partially rejected; the Hyperactive/Impulsive and the combined subtypes did take longer to finish the grooved pegboard with both hands; the difference was statistically significant between the Hyperactive/Impulsive subtype versus the control group, and the combined subtype versus the control group. There was no significant difference in performance between the Inattentive subtype and the control group.

Null Hypothesis 2.5 must be partially rejected; children with ADHD subtypes did have more touches in the Maze Coordination Task than the controls. The only exceptions were the Hyperactive/Impulsive subtype with their dominant hand and the combined subtype with the non-dominant hand, where there was no statistically significant difference in performance when compared with the control group.

A discussion of the results will follow in the next chapter.

## DISCUSSION OF RESULTS

### **8.1 Introduction**

Research studies have shown that children with ADHD perform more poorly than children without ADHD on neuropsychological tasks that measure cognitive impulsiveness and motor inhibition (Grodzinsky & Barkley, 1999; Rasmussen & Gillberg, 2000).

The aim of this study was to establish whether or not children classified as ADHD do have cognitive impulsiveness and motor deficits caused by a hypofunctioning dopamine system, as compared to children without ADHD symptomatology. Tests devised for neuropsychological deficiencies in the areas supplied by the three-dopaminergic branches, namely: the meso-cortical dopamine branch (cognitive impulsiveness - attention responses and poor behavioural organisation), as measured by the Wisconsin Card Sorting test; the nigro-striatal dopamine branch (“extra pyramidal” symptoms such as poor motor control, poor response timing) as measured by both the Grooved Pegboard and the Maze Coordination test should all be able to show these deficits (Johansen et al., 2002). The objective of the study therefore, was to subject the neuropsychological theory of ADHD as postulated by Sagvolden et al. (Johansen

et al., 2002; Sagvolden, 1999; Sagvolden & Sergeant, 1998), to systematic and experimental testing among Tshivenda speaking primary school children classified as having ADHD.

## 8.2 Summary of results

The results of the test scores were analysed in relation to the differences between children with ADHD and children without ADHD according to gender and subtype groups.

Most results supported the theory that children with ADHD will perform poorer on tasks measuring both cognitive impulsiveness and poor motor coordination as compared to the control group (Castellanos, 1997a; Rasmussen et al., 2000; Reeve & Schandler, 2001).

Table 8.1 illustrates the summary of the results according to gender

**Table 8.1 Summary of significant results on the WCST and motor tests**

<b>WCST</b>	<b>ANOVA</b>	<b>Post-hoc: boys</b>	<b>Post-hoc: girls</b>
	<i>p</i>	<i>p</i>	<i>p</i>
<i>Pers. Errors</i>	0.00*	0.00*	0.00*
<b>Motor tests</b>			
<i>Pegboard non-dom hand</i>	0.04*	n/s	0.01*
<i>Maze dom. hand</i>	0.01*	0.00*	0.04*
<i>Maze non-dom. hand</i>	0.01*	0.03*	0.01*

\* $p < 0.05$

There were no post-hoc analyses on non-perseverative errors variable, and categories shifted of the WCST and on the dominant hand of the Grooved Pegboard, because the differences were statistically not significant.

Table 8.2 illustrates the summary of the significant ANOVA results and the subsequent post-hoc test according to subtypes

**Table 8.2 Summary of significant results when the subtypes are compared with the non-ADHD control group**

Measure	ANOVA	<i>post-hoc</i> LSD (subtypes)		
	<i>p</i>	<i>p</i>		
WCST		Hyp/Impulsive	Inattentive	Combined
<i>Pers. Errors</i>	0,00*	0.00*	0.00*	0.01*
<b>Motor tests</b>				
<i>Pegs dom. Hand</i>	0,02*	0.02*	n/s	0.05*
<i>Pegs non-dom. hand</i>	0,04*	0.03*	n/s	0.02*
<i>Maze dom. Hand</i>	0.00*	n/s	0.00*	0.02*
<i>Non-dom. Hand</i>	0.01*	0.01*	0.00*	n/s

\* $p < 0.05$

The following results are unexpected and difficult to explain:

1. On the Wisconsin Card Sorting Test, the ADHD girls made fewer non-perseverative errors than the control group without ADHD

symptoms. However, the difference in performance was statistically not significant.

2. In the WCST (Categories shifted), the combined ADHD subtype achieved more categories than the controls without ADHD symptoms, but the difference was statistically not significant.

The results of the study rejected most of the null and most alternate hypotheses were accepted. The exceptions were the hypotheses regarding the non-persistent errors and categories achieved of the WCST, which were accepted.

### **8.3 Discussion of results**

This study found that children with ADHD of both genders were significantly more likely to experience both cognitive impulsiveness and poor motor coordination in comparison with their non-ADHD counterparts, as indicated on the Wisconsin Card Sorting Test, the Grooved Pegboard and the Maze Coordination Task.

#### **8.3.1 Cognitive Impulsiveness (Executive functions - meso-cortical dopamine branch)**

##### *8.3.1.1 The Wisconsin Card Sorting Test*

The Wisconsin Card Sorting test (WCST) variables include perseverative errors, non-persistent errors and categories shifted.

The ADHD children tended to make more perseverative errors in the WCST than their compatriots without ADHD. These findings support the studies of Reeve et al. (2001); Seidman et al., (1997); and Tsuchiya, Oki, Yahara, & Fujieda, (2005). Various other studies (Romine et al., 2004; Romine & Reynolds, 2004; Sergeant, Geurts, Huijbregts, Scheres, & Oosterlaan, 2003; and Scheres et al., 2003), however found no differences between children with ADHD and a group of non-ADHD controls. The ADHD girls made more of these errors than the ADHD boys. This means that in this study, girls with ADHD could not learn from the feedback on the mistakes made, hence they had difficulty changing their behaviour.

Children with ADHD made more non-perseverative errors, although the difference was not statistically significant. Tsuchiya et al. (2005) also found that children with ADHD made more of these errors.

With the categories shifted variable, the results for the ADHD girls tended towards being significant ( $p=0.07$ ) indicating also problems in this area. Seidman et al., (1997) also found differences in impairment in the categories achieved.

The findings are in agreement with studies reviewed by Sergeant, Geurts, & Oosterlaan (2002) that the WCST could distinguish between children with ADHD from the controls without ADHD symptoms, but the findings depend on the used variables.

All the ADHD subtypes performed poorly in the WCST perseverative error variable: the Hyperactive/Impulsive versus controls ( $p=0.00$ ); Inattentive versus controls ( $p=0.00$ ); and Combined versus controls ( $p=0.01$ ). These findings support those of Faraone, Biederman, Weber, & Russell, (1998), but they differ from the study by Geurts, Verte, Oosterlaan, Roeyers, & Sergeant, (2004); and Gadow et al., (2000), who failed to find deficits in children with ADHD on cognitive flexibility as measured by the WCST when compared to their control counterparts.

The differences in non-perseverative errors were statistically not significant in all ADHD subtypes.

The Hyperactive/Impulsive subtype also achieved fewer categories on the WCST, as compared to other subtypes and the control group, which may be due to their poor planning deficit and their reckless manner of responding (Denckla, 1996a).

Children with ADHD subtypes have been reported to perform poorly on the WCST, as compared to their controls (Seidman, Biederman, Faraone, Weber, & Ouellette, 1997). As a measuring instrument cognitive impulsiveness, and deficient planning behaviour, which are functions of the areas supplied by the mesocortical dopamine branch,, the WCST was able to distinguish between children with ADHD and the control subjects in this study.

### **8.3.2 Motor functions (Nigro-striatal dopamine branch)**

#### *8.3.2.1 The Grooved Pegboard*

This study found that children with ADHD from both genders were significantly more likely to experience motor control problems, in comparison with their non-ADHD counterparts, as indicated by the results of both the Grooved Pegboard and Maze Coordination Task.

In the Grooved pegboard, ADHD children took a longer time to complete the test with both hands, as compared to their control counterparts.

The difference was observed as being statistically significant with the ADHD girl's non-dominant hand ( $p= 0.01$ ), which may be due to the fact that ADHD is associated with difficulties in movement skills (Harvey & Reid, 2003). This is also in accordance with Sagvolden and Sergeant's (1998) postulation that children with ADHD have poor motor control; poor control of eye saccades; longer reaction timing; and poor handwriting, as compared to children of the same age. These behavioural patterns are associated with the dysfunction of the nigro-striatal dopamine branch, with resulting symptoms in the form of both impaired timing and force muscle groups (Johansen et al., 2002; Sagvolden and Sergeant, 1998).

The ADHD subtypes (Hyperactive/Impulsive and the Combined) demonstrated slower psychomotor speed in both dominant and non-dominant

hand, whereas the Inattentive did not demonstrate differences in psychomotor speed for both hands, when compared to the control group. Difficulty in controlling their fine motor skills when compared to their control counterparts has been demonstrated by other studies (Blondis, 1999; Rasmussen & Gillberg, 2000).

#### *8.3.2.2 Maze coordination task*

Children with ADHD showed deficits in psychomotor coordination as indicated by their poor performance (number of touches on the maze alley sides), when compared to the controls. The difference was statistically significant for both dominant hand ( $p=0.00$ ) and non-dominant hand ( $p = 0.01$ ) for boys; and for girls' dominant hand ( $p=0.04$ ) and non-dominant hand ( $p=0.03$ ), when compared to their control counterparts. The poor performance by the ADHD children may be due to impaired psychomotor functions that result in poor motor control, which in turn is associated with dysfunctioning nigro-striatal dopamine branch (Johansen et al., 2002).

There were no statistical differences between the Hyperactive/Impulsive subtype and the control group for the dominant hand and between the Combined subtype and the control group for the non-dominant hand, although there was a tendency towards significance for the latter ( $p = 0.09$ ).

Both the Inattentive and the Combined subtypes seem to be having considerable difficulty in psychomotor integration, hence their performance on

the Maze Coordination Task when compared to the controls. The Hyperactive/Impulsive subtype seems to be less affected.

*The findings can be briefly summarized as follows:*

This study demonstrated a trend among ADHD boys and girls towards poorer performances than their non-ADHD counterparts on ADHD-sensitive instruments, namely the Wisconsin Card Sorting Test, Grooved Pegboard and Maze Coordination Task.

The ADHD children in this study tended to be more vulnerable to hyperactive/impulsive and inattentive symptoms, as reflected in poorer scores on the measure on frontal lobe functioning (perseverative errors on the WCST). As the mesocortical dopamine branch supplies the prefrontal area, this may point to a deficiency in dopamine functioning. This would support the theory of dopamine functioning as postulated by Sagvolden et al. (Johansen et al., 2002; Sagvolden, 1999; Sagvolden & Sergeant, 1998).

This study also found that ADHD children of both genders and all subtypes were significantly more likely to experience motor control problems in comparison to their non-ADHD counterparts, as indicated in the Grooved Pegboard and Maze Coordination results. Studies by Blondis (1999), Rasmussen and Gillberg (2000) and Piek et al. (2004) confirm these findings. The latter authors also found a relationship between motor coordination, executive functioning, and attention in school aged children (Piek et al., 2004). Impaired

motor control among the ADHD groups may indicate a dysfunctioning nigrostriatal dopamine branch (Johansen et al., 2002; Sagvolden, 1999; Sagvolden & Sergeant, 1998) and will therefore confirm the hypothesis of Sagvolden et al. The Maze Coordination Task seemed to be more sensitive to inattention than the Grooved Pegboard.

#### **8.4 Limitations of the study**

The clinical group consisted of children who were selected by means of screening, making use of the DBD rating scale and not by means of a clinical interview.

Future research should preferably also include parents' ratings because presently the information was only obtained from teachers, so that there is no information omitted about how the children behave in other settings (Wolraich et al., 2003). The comparison of the DBD scored by both the teachers and the parents would possibly provide more reliable information about the children (American Academy of Pediatrics, 2004).

Another limitation of this study is the number of ADHD girls, which was smaller than the ADHD boys. Studies have reported that girls are under represented in this disorder, which makes it difficult to have a large clinical group.

The motor tests used in this study are all simple, inexpensive and easy to be used in cross-cultural studies but were developed and standardized in Western countries (Meyer & Aase, 2003).

Some children were computer illiterate and this would likely have an effect on the time taken to complete the tasks, thus having some impact on the results, although a training program was used.

Given a high comorbidity of CD, ODD etc, with suggested differential etiologies, future research exploring ADHD should control for the presence of comorbid and possibly confounding symptomatology.

No refined method was used to determine SES. Children were categorized into three groups, namely low, middle and high SES. The categorization was only estimation on basic information.

### **8.5 Possibilities for further research**

From the study, it was clear that certain factors, when considered, would improve the findings of this study in future research.

Semi-structured interviewing is recommended to complement the DBD rating scale in cases where parents are illiterate.

Differences in cognitive functioning may occur according to developmental stages; hence studies with homogeneous age samples are needed

## **8.6. Clinical implementation of the findings**

The findings show that problems with behavioural organization and motor clumsiness are linked to a diagnosis of ADHD. Testing of deficiencies in these areas should be part of the diagnostic process in order to develop remedial procedures and early intervention methods.

## **8.7 Concluding remarks**

The neuropsychological model of Johansen et al. (2002) enhances the understanding of the deficits that affect ADHD children and provides a scientific approach to the investigation of the three main symptoms of ADHD, i.e., hyperactivity, impulsiveness, and inattention. The findings in this study indicate that children with ADHD have deficiencies in cognitive impulsiveness as well as in motor control, when compared to their non-ADHD counterparts. These deficiencies most probably compromise a child's performance at school, i.e., more grade retention, higher dropout rate and greater expulsion rate. Social-emotional impairments are increased with more parent-child conflict, peer relationship problems and poor emotional control, especially in ADHD children with ODD/CD as comorbid disorders.

Early intervention can help to prevent the devastating effects of underachievement, poor self-image and, in the long term, possible delinquent behaviour.

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**Appendix A: Letter to school principals**



**UNIVERSITY OF THE NORTH**

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13 August 2002

The Principal  
.....Primary School

Dear Sir/ Madam:

## RESEARCH PROJECT: ATTENTION DEFICIT/ HYPERACTIVITY IN THE LIMPOPO PROVINCE

Attention Deficit Hyperactivity Disorder (ADHD) is a developmental disorder, which affects between 2% and 5% of primary school children. It consists of problems with impulse control, attention span, and activity level. However, it is much more than a matter of being inattentive and overactive. The disorder is an obstacle to benefit from normal educational methods and to form acceptable social relations. It is not temporary state that will be outgrown, for most of the children will still be suffering from the disorder as adolescents and adults.

The child usually is disorganized, has problems with planning his/her activities and may be very forgetful. There are severe problems with sustained attention, especially in the classroom situation. The child has also problems with sitting still, is overactive and fidgety. Problems with gross and fine motor co-ordination are frequent.

The cause of ADHD is not known yet, but research suggests a genetic origin. Pollutants and poor nutrition may also play a role. It is not caused by a failure to discipline or control the child.

ADHD children not diagnosed and treated at an early age at risk for the future delinquent behaviour, psychiatric problems and substance abuse. The financial costs for the society will be considerable. The families of these children experience undue stress and it has severe impact on academic activities at schools.

Diagnosis of ADHD has always caused a problem. Up to now, all instruments, which are used for the diagnosis of ADHD, are rating scales completed by teachers and /or parents and usually are culturally biased and have to be translated into all the official languages. These rating scales are mostly inaccurate because of the subjectivity of the rater. Especially in South Africa, with its many culture and language groups, the rating methods are often invalid.

The Department of Physiology, University of Oslo, Norway has therefore developed a culture-free, non-verbal test sensitive to impulsiveness, inattention and motor activity, the three major symptoms of ADHD. Together with tests for planning deficiencies and fine motor co-ordination, we are hoping to have been implicated to play a role in the disorder. This project is funded by the Norwegian Programme for Development related Research and Education (NUFU).

Postgraduate students from both the University of the North and the University of Oslo also form part of the research team.

**Method:**

The research team will visit the participating school and will screen the pupils for ADHD. The Disruptive Behaviour Disorder Rating Scale- DBD- (Pelham, Gnagy, Greenslade, and Milich, 1992) will be used. This scale, which is standardized for use with all the population groups of the Province (Meyer, Eilertsen, Sundet, Tshifularo, and Sagvolden; 2000) will be filled in by the child's class teacher. The screened children, who comply with the ADHD criteria, will then undergo further testing. The following tests will be administered:

- Biographical data questionnaire
- Test for fine motor co-ordination
- Test for planning abilities
- Test for overactivity, impulsiveness and impaired sustained attention

The data will be used for statistical analysis only and in no circumstances will the identity of the child and the school be revealed.

Your approval of this very important study will contribute to the establishment of a valid diagnostic method, which will enable professionals to identify children at risk for educational, social and emotional problems.

Yours Sincerely

-----  
Prof. Anneke Meyer  
Project Leader

## Appendix B: Letter to parents



25 August 2002

Dear Parents

Attention Deficit/Hyperactivity Disorder or AD/HD is a disorder, which affects between 2% and 5% of primary school children. The child has difficulty paying attention, controlling his or her activity and is impulsive. However, it is much more than a matter of being inattentive and overactive. The child has problems in coping with his or her schoolwork and may not be getting along well with other children. They are also unable to complete assigned tasks without supervision and cause disruptions in the family.

The problems may cause that the child is unable to adjust to the normal requirements of ordinary life. They are not likely to be outgrown and could cause future problems with reckless behaviour, possible risk of law- breaking and drug abuse. The disorder is more common in boys than in girls.

The cause of AD/HD is not known yet, but research suggests that it may be an inherited condition. Pollutants and poor nutrition may also play a role. It is not caused by failure to discipline or control the child. The children benefit from medication.

It is extremely important that these children are diagnosed and treated at an early age so that suffering at home and at school can be prevented and the child may not be at risk for future problem behaviour.

The diagnosis of AD/HD has always caused a problem. Up to now, all instruments, which are used for the diagnosis of AD/HD, are questionnaires, completed by teachers and/or parents. However, these questionnaires are very subjective and the meaning of certain concepts may differ among the various cultures and language groups of our society. We therefore, consider them to be inadequate as a method of diagnosis.

The Department of Physiology, University of Oslo, Norway, has developed a culture-free test sensitive to the major symptoms of AD/HD, which we would like to try out on the different culture- and language groups of our province. If this instrument proves to be able to successfully identify children who are suffering from AD/HD, it will prevent considerable suffering for children and their parents.

At the same time we will try to establish the role that nutrition and industrial pollutants play in the disorder. This is an approved departmental research project of the University of the North in collaboration with the University of Oslo, Norway. The Superintendent General of Education of the Northern Province has granted permission.

If you should agree in letting your child participate in this project, the research team will visit your child's school and select several children (both boys and girls) from three age groups: 7-8, 9-10 and 11-12. Not more than 10 children from each age group per school will be selected. In addition to the test (which is in the form of computer games), we will also test for muscle control (AD/HD children have frequently problems with handwriting and other motor skills), take height and weight measures, and should you

agree to it, take a blood sample. The blood will be analysed for industrial pollutants and other indicators that may help us to find a cause for this condition.

The information will be used for statistical analysis only and in no circumstances will the identity of the child and the school be revealed. The fact that your child is selected for the tests does not mean that he or she is suffering from AD/HD, because we need all the children in the Northern Province represented in this investigation. However, would you like to consult us on any problems your child is experiencing; the University psychologists will provide counseling.

Your and your child's participation in this very important study will contribute to the establishment of a valid diagnostic method, which will enable professionals to identify children at risk for educational, social and emotional problems.

Prof. Anneke Meyer  
Project leader

**Appendix C: Parental Consent form**

I, mother/father/guardian of \_\_\_\_\_ hereby give my consent for my child to be tested by the Psychology team of The University of the North.

Signed: \_\_\_\_\_

Date: \_\_\_\_\_

**Appendix D: Biographical data questionnaire**

*Biographical Data*

Tested by: \_\_\_\_\_ Date: \_\_\_\_\_  
/ \_\_\_\_\_ 2002  
School: \_\_\_\_\_ Grade: \_\_\_\_\_ Sex: M  
/ F  
Language: \_\_\_\_\_  
Height: \_\_\_\_\_ Weight: \_\_\_\_\_  
Medication (if any) \_\_\_\_\_

Dominant hand L / R  
Dominant hand foot L / R  
Computer experience: Y / N

Child's Name: \_\_\_\_\_  
Birth Date: \_\_\_\_\_ Age in months: \_\_\_\_\_  
\_\_\_\_\_  
Address: \_\_\_\_\_  
\_\_\_\_\_  
\_\_\_\_\_ Code: \_\_\_\_\_

Father's name: \_\_\_\_\_ Age: \_\_\_\_\_  
Years of education: \_\_\_\_\_  
Occupation: \_\_\_\_\_

Father's salary per month:    Less than R 1000    \_\_\_\_\_  
   R 1000 – R5000    \_\_\_\_\_  
   More than R5000    \_\_\_\_\_

Mother's name: \_\_\_\_\_ Age: \_\_\_\_\_  
Years of education: \_\_\_\_\_  
Occupation: \_\_\_\_\_  
Mother's salary per month: Less than R 1000    \_\_\_\_\_  
   R 1000 – R5000    \_\_\_\_\_  
   More than R5000    \_\_\_\_\_

Does the family have a TV?

A Car?

Electricity in the house?

Water in the house?

Is the child adopted?

Are the parents married:

Separated?

Divorced?

Please list all the other children in the family

1.	
2.	
3.	
4.	
5.	
6.	
7.	
8.	

## The Motor Test Form

Language: \_\_\_\_\_ Sex: M / F

Tested by: \_\_\_\_\_ Date: \_\_\_\_\_ / \_\_\_\_\_ 2002

*Dominant Hand L/ R*

## Grooved Pegboard Test

*Dominant Hand*

<b>Time</b>	
<b>Pegs dropped</b>	

*Non-Dominant Hand*

<b>Time</b>	
<b>Pegs dropped</b>	

## Maze Co-ordination Test

*Dominant hand*

	<b>Touches</b> (counter)	<b>Time</b> (timer)
1 <sup>st</sup> trial		
2 <sup>nd</sup> trial		
Total		

*Non-Dominant hand*

	<b>Touches</b> (counter)	<b>Time</b> (timer)
1 <sup>st</sup> trial		
2 <sup>nd</sup> trial		
Total		



## Appendix E: DBD (Tshivenda)

### Parent/Teacher DBD Rating Scale

Dzina la nwana.....  
 Duvha le fomo ya dadzwa ngalo.....  
 Minwaha.....  
 Mbeu.....

Mudadzi wa fomo.....  
 Luambo lwa hayani.....  
 Murole.....

Kha vha nange tshidanga tshine tsha sumbedza vhu vha ha nwana wavho. Kha vha nwale DK tsini na tshidanga u sumbedza phindulo ine vha si vhe na vhutanzi nayo.

	Na khatihi	Zwituku	Lunzhi	Nga maanda
1. u anzela u dzhenelela musi vhathu vha tshi amba kana mitamboni				
2. no vhuya a edela nnda nga nnda ha thendelo lu no fhira luvhili musi a tshi khou dzula na vhabebi vhawe (luthihi o				
3. tuwa lwa shifhinga tshilapfu)				
4. u anzela u nanwisana na vhahulwane				
5. u anzela u shumisa mazwifhi musi a tshi humbela tshithu kana a tshi shavha u ita mushumo				
6. u anzela u thoma nndwa na vhanwe vhana henefha hayani				
7. ndi muthu wa tshituhu				
8. u a ambesa				
9. o vhuya a tswa thundu i sina ndeme a sa vhonwi				
10. u anzela u vhonala sa muthu o xelaho muhumbuloni (zwi tshi sumbedza u itiswa nga zwithu zwine an si kone u zwilaula				
11. u anzela u tamba mitambo ine ya vha na khomno a sa di londi				
12. o thoma u lova tshikolo na musi a sa				

athu u swikisa minwaha ya 13				
13. u anzela u vhonala a muthu asa tokomelwi				
14. u anzela u sasaladza (ndi muthu asa khodi tsha munwe)				
15. u dzulela u ana na u semana				
16. u anzela u pomoka vhanwe kha vhukhaki hawe				
17. o no di vhuya a tshinya thundu ya vhathu nga khole				
18. u na lunyadzo na hone u hana u rumiwa nga vhahulwane khae				
19. tshifhinga tshinzhi u sumbedza u vha a muthu a sa thetshelesi musi a tshiambiwa naye				
20. u gidimele u fhindula mbudziso naho muvhudzisi a sa athu u fhedza u vhudzisa				
21. u anzela u levhela na u tokonya nndwa na vhathu vhane vha sive mashaka naye				
21. u anzela u sia mishumo yawe I gake				
22. anzela u vhonala a tshi balelwa u di dzhenisa kha mitambo, naho hu udi netulusa zwaho				
23. tshifhinga tshinzhi u sumbedza u sa thetshelesa kana u ita vhukhaki hu sa divhalei kha mushumo wawe wa tshikolo				
24. u anzela u vhonala e muthu ano dzula o sinyuwa na u dala vengo				
25. u dzulela u sia madzulo awe na nga tshifhinga tshine a lavhelelwa uri a vhe o dzula				
26. ndi muthu wa u fara-fara vhathu mara ene ha lengi u sinyuwa				
27. u anzela u sa tevhedza milayo a dovha a balelwa u ita mushumo wa tshikolo kana mushumo munwe na munwe ( tshifhinga tshinzhi zwi sa itiswi ngauri u na khani kana ha ngo pfa ndacla)				

28. u anzela u sinyuwa nga u tavhanya				
29. u anzela u vha na thaidzo kha udi dzhenisa mishumoni kana mitamboni				
30. u anzela u sa kona u lindela				
31. o no vhuya a kombetshedza uri a edele na musidzana/muthannga				
32. u anzela u shushedza vhanwe na u di ita mboho				
33. u anzela u vha muthu asa tokomelwi				
34. u anzela u xedza zwishumiswa zwa ndeme khau ita mishumo ya tshikolo, zwithu zwi nonga bugu, kana zwi tambiswa zwawe				
35. u anzela u gidima-gidima kana u gonya miri naho minwaha yawe I sa tsha mutendela u ita izwo zwithu				
36. u tambudza na u rwa zwifuwa				
37. u litsha, kana u teledza u ita mushumao wa tshikolo une wa toda uri a dzhie tshifhinga tshilapfu e khawo (sa mishumo wa tshikilo kana tshunwa haya)				
38. u vhuya hayani vhusiku naho vhabebi vho zwi hanela(zwo thoma a sa a thu uvha na minwaha ya 13)				
39. u dina vhathu nga khole				
40. o no tswa a tshi khou tshuwisa mu dzhielwa( u dzia bege, ka na u dzhiela wo fara zwithavhane)				
41. u thoma mulilo hu u itela u tshinyadza				
42. ha koni u dzudzanya mishumo yawe zwa vhudi				
43. o no vhuya a dzhena nduni, kana goloini ya muthu nga nda ha thendelo				
44. u anzela u hangwa mishumo yawe ya duvha				
45. o no shumisa tshithavhane tshine tsha nga huvhadza muthu (sa tshidina, bodelo, lufhanga kana tshigidi)				

Appendix F: DBD (English)

**Teacher / Parent DBD Rating Scale**

Child's name: \_\_\_\_\_ Form completed by: \_\_\_\_\_

Sex: M / F Age: \_\_\_\_\_ School: \_\_\_\_\_

Grade: \_\_\_\_\_

Home language: English /Afrikaans/ N-Sotho/ Xitsonga/ Tshivenda/  
Other: \_\_\_\_\_

**Check the column that best describes this child. Please put a question mark next to any item for which you don't know the answer.**

	Not at All	Just a Little	Pretty Much	Very Much
1. often interrupts or intrudes on others (e.g, butts into conversations or games)				
2. has run away from home overnight at least twice while living in parental or parental surrogate home (or once without returning for a lengthy period)				
3. often argues with adults				
4. often lies to obtain goods or favours to avoid obligations (i.e., "cons others)				
5. often initiates physical fights with other members of his or her household				
6. has been physically cruel to people				
7. often talks excessively				
8. has stolen items of nontrivial value without confronting a victim (e.g. shoplifting, but without breaking and entering; forgery)				
9. is often easily distracted by extraneous stimuli				

10. often engages in physically dangerous activities without considering possible consequences (not for the purpose of thrill-seeking), e.g. runs into the street without looking				
11. often truant from school, beginning before age 13 years				
12. often fidgets with hands or feet or squirms in seat				
13. is often spiteful or vindictive				
14. often swears or uses obscene language				
15. often blames others for his or her mistakes or misbehaviour				
16. has deliberately destroyed others' property (other than by fire setting)				
17. often actively defies or refuses to comply with adults' request or rules				
18. often does not seem to listen when spoken to directly				
19. often blurts out answers before questions have been concluded				
20. often initiates physical fights with others who do not live in his or her household (e.g. peers at school or in the neighbourhood)				
21. often shifts from one uncompleted activity to another				
22. often has difficulty playing or engaging in leisure activities quietly				
23. often fails to give close attention to details or makes careless mistakes in schoolwork, work or other activities)				
24. is often angry and resentful				
25. often leaves seat in classroom or in other situations in which remaining seated is expected				
26. is often touchy or easily annoyed by others				
27. often does not follow through on instructions and fails to finish schoolwork, chores, or duties in				
the workplace (not due to oppositional behaviour or failure to understand instructions)				

28.often loses temper				
29.often has difficulty sustaining attention in tasks or play activities				
30.often has difficulty awaiting turn				
31.has forced someone into sexual activity				
32.often bullies, threatens, or intimidates others				
33.is often “on the go” or often acts as “if driven by a motor”				
34.often loses things necessary for tasks or activities (e.g toys, school assignments, pencils, books, or tools)				
35.often runs about or climbs excessively in situations in which it is inappropriate				
36.has been physically cruel to animals				
37.often avoids, dislikes, or is reluctant to engage in tasks that require sustained mental effort (such as schoolwork or homework)				
38.often stays out at night despite parental prohibitions, beginning before age 13 years				
39.often deliberately annoys people				
40.has stolen while confronting a victim (e.g., mugging, purse snatching, extortion, armed robbery)				
41.has deliberately engaged in fire setting with the intention of causing serious damage				
42.often has difficulty organising tasks and activities				
43.has broken into someone else’s house, building, or car				
44.is often forgetful in daily activities				
45.has used a weapon that can cause serious physical harm to others (e.g. a bat, brick, gun.				