COGNITIVE IMPULSIVENESS IN AFRIKAANS SPEAKING PRIMARY SCHOOL CHILDREN WITH ATTENTION DEFICIT HYPERACTIVITY DISORDER

by

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DECLARATION

I, Dirk Cornelius Badenhorst, declare that the dissertation hereby submitted to the University of the North as partial fulfillment for the degree of Masters 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: 16/7/03

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ABSTRACT

This study explored the neuropsychological performance of Afrikaans speaking primary school children screened for ADHD symptoms. Children with ADHD (n=31) were compared with normals (n=31) in terms of cognitive impulsiveness. A battery of neuropsychological tests used to measure executive functions was employed. The battery consisted of the SSAIS-R, TOL, WCST and Stroop. The results of the tests were analysed in relation to the different age, gender and subtype (inattentive, hyperactive/impulsive or combined) groups. In the majority of the tests the clinical groups performed worse than the control groups. The younger girls with ADHD appeared to be particularly severely impaired. The combined diagnoses subtypes had consistently lower scores than the other clinical groups. The results indicate that children with ADHD are more cognitively impulsive than controls.

Chapter 1

GENERAL INTRODUCTION AND BACKGROUND

1.1. Introduction

ADHD is the most common childhood psychiatric disorder (Castellanos, 1997; Hazelwood, Bovingdon, & Tiemens, 2002). The disorder is also among the most prevalent chronic health conditions affecting school-age children (American Academy of Pediatrics, 2000). A diagnosis of ADHD predisposes the individual to an increased vulnerability for anxiety and mood disorders, drug abuse, anti-social behaviour and thus lower quality of life. It has a debilitating effect on the child's social, personal and academic development, and some degree of impairment often remains in adulthood (Brown, 2000; Pary, Lewis, Matuschka, & Lippmann, 2002a). Early identification, diagnosis and treatment of the disorder can decrease this vulnerability.

At present the aetiology of the disorder is not well understood, but recent research efforts have been focussing on a neurobiological cause. Possible neurobiological contributions that are being considered are: genetic contributions; the role of neurotransmitters; differences in neuro-anatomy and neurochemistry (Barkley, 1998b; Levy & Swanson, 2001) (Rapoport et al., 2001; Schrimsher, Billingsley, Jackson, & Moore, III, 2002; Smalley et al., 2002; Swanson et al., 2000a; Wickens & Tripp, 1999).

It is unlikely that a sole causal factor will be identified. It is more likely that various neurological systems are dysfunctional in the disorder.

1.2. Objective of the study

The main goal of the research is to submit the neuropsychological theory of Attention-Deficit Hyperactivity Disorder (Sagvolden, 1999; Sagvolden & Sergeant, 1998a; Johansen, Aase, Meyer, & Sagvolden, 2002) to systematic and experimental testing among Afrikaans speaking primary school children screened for ADHD.

According to this theory, ADHD symptoms are caused by dysfunctioning dopamine systems impairing the conduction of non-dopaminergic inputs to the frontal cortex and the ventral and dorsal striatum. These dysfunctioning dopamine systems lead to observable and measurable behavioural and cognitive deficits. One of these deficits – cognitive impulsiveness – was assessed in this study through various neuropsychological tests.

1.3. Purpose and significance of the study

This study forms part of an international research project on the neurological basis of ADHD, with branches in Norway, the U.K., the Netherlands and the USA. The identification of a neurological cause for ADHD will have a significant impact on the understanding of the aetiology of the disorder and the development of new treatment strategies.

The anticipated outcome will be a confirmation of the neuropsychological theory of ADHD. This means that assessment methods can be based on neuropsychological deficits and will be culture-free and unbiased. Children at risk for aggressive and possible antisocial behaviour, due to cognitive impulsiveness, can be identified at an early age by means of an accurate, scientific measurement battery suitable for all cultures and language groups.

A number of treatment implications result from this model of ADHD. Methylphenidate and other dopamine agonists can be used as a temporary corrective treatment. The use of stimulant medication however, needs to be accompanied by treatment methods that address the underlying neuropsychological deficiencies.

Although there are well-documented deficits in executive functions in children with ADHD, the term executive functions denotes a variety of psychological functions. In contrast, the term cognitive impulsiveness is both theoretically grounded (in the theory this study is based on) and specific (as it pertains to the ability or inability to inhibit cognitive impulses). It is expected that this clarity in conceptualisation and theoretical grounding will be an aid to further research, and will enable the identification of other specific deficits.

1.4. Delineation of the study

A general introduction to ADHD is provided in Chapter 2. This chapter gives a description of the historical background of the disorder, diagnostic criteria, symptoms, prevalence and gender differences. It also describes co-morbid disorders, secondary deficits, aetiologies, the developmental course of the disorder and treatment options.

In Chapter 3 the neurobiological basis of ADHD is discussed. The chapter provides an overview of the neuropsychological theory this study is based on. It also provides information on the domains of neuro-anatomy, genetics, neurochemistry, pharmacology and neuropsychology and their impact on the disorder.

The process of diagnosis and assessment is discussed in Chapter 4. In this chapter the different screening and assessment methods that are often used in the diagnosis and treatment planning of patients with ADHD are explained.

Chapter 5 comprises of the problem statement and provides the research hypotheses, and the methodology of the study is discussed in Chapter 6. Chapter 7 gives a representation of the data results of the study.

Chapter 8 comprises of a discussion of the results of the study, description of the limitations, and outlines possible areas for future research.

GENERAL INTRODUCTION TO ATTENTION DEFICIT / HYPERACTIVITY DISORDER

2.1. Historical background

The precursors of the disorder that is today known as Attention-Deficit/Hyperactivity Disorder, was already recognised in the 1800's. A German physician, Heinrich Hoffmann (1845), described hyperactive symptoms in a children's story titled "Fidgety Phil" (De Armas, 2001; Penrice, 1996; Zametkin, 1995).

It would go through various permutations in description and name before becoming recognisable as the disorder presently known as ADHD. At various times in history, the disorder was called Encephalitis Lethargica, Minimal Brain Damage, Minimal Brain Dysfunction, Attention Deficit Disorder (ADD) and Attention Deficit Hyperactivity Disorder (ADHD) Rafalovich, 2001. These changes were brought forth in no small part through massive research efforts, which are still continuing today.

The association of Attention-Deficit/Hyperactivity Disorder with some type of underlying neurological condition has its origin early in the 20th century (Barkley, 1998b; Ravalovich, 2001). This is an association that continues to this day, and makes Attention-Deficit/Hyperactivity Disorder a disorder that is studied by a variety of disciplines, including neurology, psychology, psychiatry, pharmacology and physiology.

George Frederic Still, a British paediatrician, is the first author credited for seriously focussing on Attention-Deficit Hyperactivity/Disorder (or at least an early precursor of the disorder) in a scientific manner (Barkley, 1998b; De Armas, 2001; Swanson et al.,

1998b). Still (1902) presented three lectures to the Royal College of Physicians, in which he described 43 children he had seen in his practice. These children displayed a cluster of symptoms that is today often associated with the disruptive behaviour disorders, of which Attention-Deficit/Hyperactivity Disorder is one. The children were often aggressive, defiant, resistant to discipline, and excessively emotional or passionate. 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).

In the early 1900's, Psychoanalytic theories had a stronghold on psychiatry (Castellanos, 1997; Jordaan & Jordaan, 1992; Kaplan & Sadock, 1998b). Thus the idea that hyperactive symptoms could have an organic cause rather than a psychosocial one was a radical departure from accepted thought (Cates, 2002). Still's observations were published in the journal Lancet in 1902, which gave some scientific credibility to his work (Cates, 2002; Rafalovich, 2001; Zametkin, 1995).

An outbreak of encephalitis (Encephalitis Lethargica) in 1917 – 1918 led to interest in Attention-Deficit/Hyperactivity Disorder in North America. The disease was also known as "sleepy sickness" and reached epidemic proportions to the end of World War I (Rafalovich, 2001). Many children survived the epidemic, but were left with residual behavioural and cognitive sequelae (Barkley, 1998b; Cates, 2002; De Armas, 2001). "Such children were described as being impaired in attention, regulation of activity, and impulsiveness, as well as other cognitive capabilities, including memory, and were often noted to be socially disruptive" (Barkley, 1998b, p5).

These behavioural sequelae were termed Postencephalitic Behaviour Disorder (Cates, 2002). This event was important in focussing attention on brain-damage in children, and the behavioural and cognitive effects thereof. In many ways it can be seen as the

precursor for the neurological understanding of Attention-Deficit/Hyperactivity Disorder that is prevalent today.

The management of hyperactive children with stimulant medication (which today is the first-line treatment for ADHD) originated in the late 1930's. In 1937 hyperactive children were treated with the stimulant benzedrine (now known as dextroamphetamine) at the Bradley Hospital in Providence, Rhode Island. Charles Bradley and Maurice Loffer recognised that amphetamines were useful in managing hyperactive and impulsive behaviour (Bradley, 1937). However, even with this knowledge, it was only in the 1960's that stimulant medication became commonly used in the management of ADHD-symptoms (Penrice, 1996).

Continued research efforts lead to the questioning of the concept of "a unitary syndrome of brain damage in children" (Barkley, 1998b, p.8). Researchers started considering whether ADHD could be caused by environmental factors, such as poor parenting, food additives or environmental toxins (Barkley, 1998b; De Armas, 2001; Martin, 2002).

Stella Chess (1960) and her associates were the first to describe hyperactivity. They used the term "Hyperactive Child Syndrome" It became the generally accepted name for the disorder in the psychological community (Cates, 2002; De Armas, 2001). Although there was a continuing focus on environmental factors, Chess was of the opinion that the disorder had a biological cause.

As explained previously, the association of hyperactivity with brain damage led to the conclusion that children who were hyperactive necessarily also had brain damage. The term minimal brain dysfunction became a widely used general term to describe these children (Cates, 2002; Gilles-Thomas, 1989, Rafalovich, 2001). However, by the late

sixties dissatisfaction with the term Minimal Brain Dysfunction led to the development of the concept of a Hyperactive Child Syndrome (Barkley, 1998b).

> The concept of MBD would die a slow death as it eventually became recognized as vague, overinclusive, of little or no prescriptive value, and without much neurological evidence. Its value remained in its emphasis on neurological mechanisms over the often excessive, pedantic, and convoluted environmental mechanisms proposed at that time, particularly those etiological hypotheses stemming from psychoanalytic theory, which blamed parental and family factors entirely for these problems (Barkley, 1998b, p8)

Hyperactivity was included in the Diagnostic and Statistical Manual of Mental Disorders, for the first time in the second edition, published in 1962 (American Psychiatric Association, 1962).

There was an explosion of research interest into hyperactivity in the 1970's (Barkley, 1998b). Research focus shifted to the attentional aspects of the disorder, mostly due to the groundbreaking work of a Canadian psychologist, Dr Virginia Douglas.

Douglas was the first to focus on "cognitive impulsiveness", then described as daydreaming and lack of attention. She described four major characteristics of the disorder: 1. Deficits in attention. 2. Impulsiveness. 3. Problems regulating arousal levels 4. A need for immediate reinforcement. Thus from her work what is today seen as the three core symptoms of ADHD (hyperactivity, impulsivity and inattention) could already be defined (Douglas, 1972).

Douglas made a Presidential address to the Canadian Psychological Association in 1972, in which she presented her theory that deficits in the sustained attention and impulse control were more likely to account for the difficulties of these children than hyperactivity (Douglas, 1972; Martin, 2002). Thus the shift in research focus from

hyperactivity to the attentional aspects of the disorder had begun. Douglas is credited as being influential in the fact that the American Psychiatric Association created a new diagnostic category, Attention Deficit Disorder, with or without hyperactivity (in 1980) (De Armas, 2001; Gilles-Thomas, 1989).

The third edition of the DSM was published as the decade of the 1980's opened. It contained a radical reconceptualisation of hyperactivity, changing from Hyperkinetic Reaction in Children (DSM-II) (American Psychiatric Association, 1962) to Attention-Deficit Disorder (American Psychiatric Association, 1972). Throughout the decade, there seemed to be some controversy surrounding the sub-diagnosis of hyperactivity or no hyperactivity. When the DSM was revised, in 1987, Attention-Deficit- Disorder became Attention-Deficit/Hyperactivity Disorder (American Psychiatric Association, 1987).

According to Barkley (1998b) the decade closed with the following prevailing view on Attention-Deficit/Hyperactivity Disorder:

> ....it was a developmentally handicapping condition generally chronic in nature, having a strong biological or hereditary predisposition, and having a significant impact on academic and social outcomes for many Attention-Deficit/Hyperactivity Disorder children. However, its severity, comorbidity, and outcome were viewed as significantly affected by environmental, particularly familial, factors. (Barkley, 1998b, p8)

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, 1998b; Martin, 2002).

In addition, research by the academic community, mental health care practitioners and pharmaceutical companies continues at an expanding pace. ADHD is now recognised as a disorder with a neurological base, although the severity of the symptoms can be influenced by environmental factors (Barkley, 1998b; De Armas, 2001; Penrice, 1996).

It is clear from this historical perspective that there has always been some neurological involvement suspected in the aetiology of Attention-Deficit/ Hyperactivity Disorder. As neuro-imaging developed and knowledge about the brain expanded, the evidence for some sort of neurological involvement became stronger, to the point where there is almost an exclusive focus on the neurobiology of this disorder in research efforts.

2.2. Diagnostic criteria, primary symptoms, prevalence and gender differences

2.2.1 Diagnostic criteria and primary symptoms

The developments in the diagnostic process of Attention-Deficit/Hyperactivity Disorder have been mentioned in the previous section. Currently, there are two manuals used most often in diagnosis of mental disorders, the fourth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) (American Psychiatric Association, 1994) and the International Classification of Diseases (World Health Organization, 1993). The specific diagnostic criteria for Attention-Deficit/Hyperactivity Disorder are in the DSM-IV and those for HKD (Hyperkinetic Disorder) are in the ICD-10.

Swanson and Sergeant (1998) have stated that a perplexing series of changes in labels and definitions produced confusion since the 1960's, when criteria for childhood psychiatric disorders were first included in diagnostic manuals. Differences arose between countries about the diagnosis and subsequently about the national differences in the epidemiology of these disorders.

Various methods of assessment should be used to diagnose ADHD (Barkley, 1998b; Brown, 2000; Castellanos, 1997; Pary , Lewis, Matuschka, & Lippmann, 2002). Each method has its strong and weak points, and thus an approach utilising multiple assessment methods can compensate for these weaknesses (Brown, 2000). Usually, assessment instruments in the process includes clinical interviews, behaviour rating scales and behavioural observation (Brown, 2000; Pary et al., 2002a)

There are no laboratory correlates that can be used in the diagnostic process, although advances in this area are being made (Castellanos, 1997; Kaplan & Sadock, 1998). Neuropsychological tests may be helpful, but are not sufficient basis on which to make a diagnosis (Pary et al., 2002a).

According to Swanson et al, (1998b), after decades of different operational definitions, DSM and ICD manuals in their most recent versions now recognise the same problem behaviours as the basis of the diagnosis, in almost identical sets of 18 symptoms.

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, 1994) allows for 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.

314.01. 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 (American Psychiatric Association, 1994).

314.00 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.

314.01. 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.

Coding note: For individuals (especially adolescents and adults) who currently have symptoms that no longer meet full criteria, "in partial remission" should be specified.

Differential Diagnosis

Kaplan and Sadock (1998) state that the following conditions should also be considered before the diagnosis of Attention-Deficit/Hyperactivity Disorder is made: (1) a temperamental constellation consisting of high activity level and short attention span, but in the normal range of expression for the child's age, (2) Anxiety, (3) Depression, (4) Mania, (5) Conduct disorder. It is important to note that any of these conditions may be co-morbid to the Attention-Deficit/Hyperactivity Disorder, thus clouding the clinical picture.

Recently, various researchers have stated that inattention may be a less important diagnostic criteria then impulsivity/hyperactivity (Johansen, Aase, Meyer, & Sagvolden, 2002; Sagvolden & Sergeant, 1998). These researchers state that inattention is a feature of a variety of psychiatric disorders, such as depression, and thus may not be a defining feature of Attention-Deficit/Hyperactivity Disorder. The research seems to indicate that impulsiveness is a much more relevant symptom when it comes to the diagnostic process (Johansen et al., 2002).

The diagnostic criteria can be summarised as follows:

Table 2.1: ICD-10 and DSM-IV symptom domain criteria for Attention-Deficit/Hyperactivity Disorder/HKD

ICD-10	Inattention (6 or more)	Impulsiveness (1 or more)	Hyperactivity (3 or more)
DSM-IV	Inattention (6 or more)	Hyperactivity-	Impulsiveness (6 or more
	1. Fails to attend to details 2. Has difficulty sustaining attention 3. Does not seem to listen 4. Fails to finish 5. Has difficulty organizing tasks 6. Avoids sustained effort 7. Loses things 8. Is distracted by extraneous stimuli 9. Is forgetful	1. Blurts out answers 2. Difficulty waiting turn 3. Interrupts or intrudes 4. Talks excessively (in ICD-10)	4. Talks excessively (in DSM-IV) 5. Fidgets with hands or feet 6. Leaves seat in classroom 7. Runs about or climbs 8. Difficulty playing quietly 9. Motor excess

Table from Swanson et al. (1998b)

However, there are still three major differences in decision rules between DSM-IV and ICD-10, according to Swanson et al. (1998b). 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 (Attention-Deficit/Hyperactivity Disorder) does not, but instead specifies partial subtypes if symptoms are from only one domain.

ICD-10 (World Health Organization, 1993) 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. DSM-IV (American Psychiatric Association, 1994) does not make any special provision for conduct disorder as a comorbid condition but allows its diagnosis.

From the preceding it should be clear that children with Attention-Deficit/Hyperactivity Disorder vary in the number and relative severity of the following behaviours:

Inattention: easily distracted, flits from task to task, best with one to one supervision, poor short-term memory, forgets instructions, loses focus, slow completing work, hard to reach and teach. Some are distant, dreamy and "spaced-out".

Impulsiveness: speaks and acts without thinking, interrupts, low frustration tolerance, short fuse. Problems with behaviour inhibition, seems aggressive.

Overactivity: restless, fidgets, fiddles, has to touch. Appears as if "driven by a motor".

2.2.2 Prevalence and gender differences

Attention-Deficit/Hyperactivity Disorder is the most common childhood mental disorder, with prevalence of 3% to 5%, (American Psychiatric Association, 1994).

In childhood, the disorder is more common in boys than in girls (Taylor, 1998), but during adolescence and young adulthood, the proportion of females affected increases (Biederman et al., 1994). Swanson et al. (1998b), estimate the ratio of boys to girls with Attention-Deficit/Hyperactivity Disorder to be between 3:1 to 9:1. They ascribe the difference between the sexes in part to referral bias related to symptoms of disruptive behaviour since boys have more hyperactive/impulsive symptoms and more conduct and oppositional symptoms than girls.

Attention-Deficit/Hyperactivity Disorder is usually observed before the age of seven, and is observable in a variety of settings (e.g. at home and at school) (American Psychiatric Association, 1994; Barkley, 1998b; Kaplan & Sadock, 1998). The combination of inattentive, hyperactive and impulsive behaviours in children is recognised as a disorder when these behaviours are severe, developmentally inappropriate, and impair function at home and school (Swanson et al., 1998b)

Attention-Deficit/Hyperactivity Disorder is not a culture-bound syndrome, as it presents in a various cultures (Alarcon, Westermeyer, Foulks, & Ruiz, 1999; Leung et al., 1996; Meyer & Aase, 2003a; Meyer, 1998; Taylor, 1998). It is difficult to establish this diagnosis in children younger than 4 or 5 years, because their behaviour is much more variable than that of older children, and may include features that are similar to symptoms of Attention-Deficit/Hyperactivity Disorder (American Psychiatric Association, 1994; Johansen et al., 2002). The symptoms of inattention in toddlers or preschool children are often not readily observed because young children typically experience few demands for sustained attention (American Psychiatric Association, 1994). Young children with Attention-Deficit/Hyperactivity Disorder move excessively and are difficult to contain (Sonuga-Barke, Daley, Thompson, Laver-Bradbury, & Weeks, 2001).

By late adulthood and early adolescence, signs of excessive gross motor activity are less common, and hyperactivity symptoms may be confined to fidgetiness or an inner feeling of jitteriness or restlessness (American Psychiatric Association, 1994).

2.3. Comorbid disorders and secondary deficits

Attention-Deficit/Hyperactivity Disorder forms part of a cluster of disorders termed the Disruptive Behaviour Disorders (American Psychiatric Association, 1994). As its name implies, these disorders are characterised by a disruption in developmentally appropriate behaviour. The other disorders that complete the trilogy of disruptive disorders are Oppositional Defiant Disorder (ODD) and Conduct Disorder (CD) (Pliszka, Carlson, & Swanson, 1999). There may be a neurobiological or genetic link between these disorders (Pliszka et al., 1999). Children with ADHD are also at risk of developing other psychiatric disorders, such as depression or anxiety (Brown, 2000; Kaplan & Sadock, 1998).

In addition to the three primary symptoms of inattention, hyperactivity and impulsiveness, there are a number of secondary behaviour dysfunctions which may appear in individuals with Attention-Deficit/Hyperactivity Disorder. The disorder has a pervasive impact on various domains of functioning, including academics, peer relations, familial relationships and self-esteem (Hazelwood, Bovingdon & Tiemens, 2002).

Poor academic performance may lead to conflict at home and at school, as well as contributing to low self-esteem. According to the DSM-IV, inadequate self-application to tasks is often interpreted by others as indicating laziness, a poor sense of responsibility and oppositional behaviour. The family relationships of individuals with Attention-Deficit/Hyperactivity Disorder is often characterised by resentment and antagonism, especially if parents believe the troublesome behaviour to be wilful (Barkley, 1998b)

Learning disorders are often co-morbid with ADHD. These include disorders in reading, math, and written or spoken language (Castellanos, 1997; Zametkin, 1995).

2.4. Aetiologies

A variety of causal factors have been identified in the development of ADHD, including neurological, genetic (hereditary), environmental factors and psychosocial factors (Brown, 2000; Wagner, 2000; Zappitelli, Pinto, & Grizenko, 2001).

As stated in the section on the historical background, most researchers now acknowledge that aetiological theories based on neurobiological factors are best supported by research results. – "although multiple aetiologies may lead to Attention-Deficit/Hyperactivity Disorder, evidence points to neurological and genetic factors as the greatest contributor to this disorder" (Barkley, 1998b, p164). However, other factors, such as environmental toxins, side-effects of medication and psychosocial factors may have modulatory influences on the presentation of symptoms of the disorder.

The neurobiological factors (which include neurological and genetic factors) will be discussed in detail in Chapter 3, and will therefore not be discussed in this section.

2.4.1. Environmental factors

Although environmental factors have been shown to influence the development of Attention-Deficit/Hyperactivity Disorder, the main influence of the environment is on the biology of the individual. Thus it is environmental factors' influence on the biological elements of a person's functioning and not the factors in themselves which contribute to the development of Attention-Deficit/Hyperactivity Disorder. One could thus speak of environmental factors as secondary causative factors (Barkley, 1998b; Wagner, 2000).

Environmental factors that may be implicated in Attention-Deficit/Hyperactivity Disorder include pre-, peri-, and postnatal complications and malnutrition, certain diseases, trauma, environmental toxins and other neurologically compromising events that may occur during the development of the nervous system before and after birth (Barkley, 1998b).

2.4.2. Psychosocial factors

Few theories on a psychosocial basis for the development of Attention-Deficit/Hyperactivity Disorder are taken seriously anymore (Barkley, 1998b). It seems as if psychosocial factors may influence the actual severity of the symptoms, the continuity of those symptoms over development, the types of secondary symptoms, and the outcome of the disorder (Barkley, 1998b).

As there is a genetic contribution to the development of Attention-Deficit/Hyperactivity Disorder (discussed in Chapter 3), it can be difficult to separate psychosocial and genetic contributions when the role of parenting styles is considered. In other words, many parents of children with Attention-Deficit/Hyperactivity Disorder also suffer from the syndrome, making it difficult to identify if it is genetics or the parents' parenting style that lead to the development of the disorder (Conrad & Potter, 2000; Serfontein, 1994).

2.5. Developmental course and adult outcome

Contrary to what was previously believed, it is now accepted that Attention-Deficit/Hyperactivity Disorder can (and often does) continue in adolescence and adulthood if it is left untreated (Hazelwood et al., 2002; Pary et al., 2002). It is estimated that up to 60% of children with ADHD will have residual symptoms as adults (Serfontein, 1994, Zametkin, 1995).

Adult Attention-Deficit/Hyperactivity Disorder is characterised by disorganization, impulsiveness and poor work skills. These individuals tend to be disorderly and impatient and may display any of the following characteristics: poor concentration, impulsiveness, easy distractibility and problems with speech, co-ordination, short-term memory and associated behavioural difficulties (Pary et al., 2002, Serfontein, 1994).

The course of Attention-Deficit/Hyperactivity Disorder cannot be easily predicted. Symptoms are inconsistent between children and adults, though decreased attention span and problems with impulse behaviour commonly persist (Pary et al., 2002).

DSM-IV recognises the adult type of ADHD in its category, ADHD residual type (American Psychiatric Association, 1994).

2.6. Treatment

2.6.1. Pharmacological agents

Stimulant medication has been the treatment of choice for a number of years (Brown, 2000; Castellanos, 1997; Gitlin, 1996; Hardman, Limbird, Molinoff, Goodman & Gillman, 1996; Zametkin, 1995).

The stimulants most often used are dextroamphetamine, methylphenidate (Ritalin®) and pemoline (Kaplan & Sadock, 1998). Treatment may start with a 5mg dose of methylphenidate in the morning and at lunch; the dose is increased gradually over a period of weeks depending on the response judged by parents, teachers and the physician. The total daily dose generally should not exceed 60mg, because of its short duration of action; most children require two or three doses of methylphenidate daily. The timing of doses is adjusted individually in accordance with rapidity of onset of effect

and duration of action. Some children may not respond, and the drug should be discontinued after one month of dosage adjustment (Hardman et al., 1996).

Stimulant medication has repeatedly shown to deliver good results, with limited side-effects if it is administered correctly (Elia, Ambrosini, & Rapoport, 1999; Greenhill, Halperin, & Abikoff, 1999). Robust data is available regarding the safety and efficacy of the drug in the short term and in the treatment in children between five and fourteen. Less is known, however, about the implications of prolonged use or about methylphenidate's long-term effects (Buitelaar, Van der Gaag, & Swaab-Barneveld, 1996; Johnston & Fine, 1996).

The improvements in impulsive and overactive behaviour are more pronounced than in inattentiveness (Swanson, Pfiffner, Wigal, & McBurnett, 1993). The pharmacological action of stimulant medication will be focussed on in Chapter 3.

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

Antidepressants have been shown to deliver some success, especially in the circumstances where Attention-Deficit/Hyperactivity Disorder is co-morbid with anxiety or depressive disorders (Metha, Sahakian, & Robbins, 2001). As was shown in the section on diagnostic issues, these conditions often do co-exist with Attention-Deficit/Hyperactivity Disorder.

It can be useful to explore the meaning of taking medication, as the psychodynamics of this may hamper an intervention, i.e. lead to non-compliance (Gitlin, 1996).

According to Kaplan and Sadock (1998), medication alone is often not enough to satisfy the comprehensive therapeutic needs of children with the disorder and is usually only one facet of a multimodal intervention.

2.6.2. Psychosocial interventions

Non-pharmaceutical therapies are recommended in addition to medications (Pary et al., 2002). These therapies may include individual psychotherapy, behaviour modification, parenting classes, parent support groups, school involvement, and education about Attention-Deficit/Hyperactivity Disorder. When applied in preschool children, intensive parent management training may be as effective as medical treatment in reducing symptoms of ADHD, in addition to reducing depression in mothers and increasing parents' experience of themselves as successful (Sonuga-Barke et al., 2001).

Parent-training is also often a part of a psycho-social intervention. This would focus on empowering parents to create a structured environment in which appropriate behavioural responses are duly rewarded. Kaplan and Sadock (1998) state that children with ADHD do not benefit from being exempted from the requirements, expectations, and planning applicable to other children.

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 (1997c) and Barkley and 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, 1997c).

The treatment of the patient with Attention-Deficit/Hyperactivity Disorder remains challenging. The stimulant medications are the mainstay of treatment, even though the non-pharmaceutical treatments make sense intuitively. Unfortunately, their efficacy is often questioned (Hazelwood et al., 2002; Pary et al., 2002).

Where behavioural therapy does not have the desired result, medication can enhance the efficacy of the treatment programme considerably (Klein et al., 1997). In many cases, the greatest effect on the core symptoms of ADHD during the treatment period is to be achieved by medication combined with a carefully planned programme of counselling, supervision, and monitoring. In other domains, such as social interaction, psychosocial well-being, and academic performance, there is little difference between the two modes of treatment, other than that the effects of behavioural therapy are slightly longer lasting (MTA-Cooperative Group, 1999). Where medication is given in combination with behavioural therapy, the drug can be administered in lower doses (Horn et al., 1991).

The most appropriate form of treatment should be selected for each case individually following thorough diagnostic assessment and taking account of the capacities of the parents, educational system, and culture of the child.

Chapter 3

NEUROBIOLOGY OF ATTENTION-DEFICIT/HYPERACTIVITY DISORDER

3.1. Background

From the review of the history of ADHD in chapter 2, it should be clear that an underlying neurobiological dysfunction has always been suspected in this disorder. As explained before, the hypotheses of neurological dysfunction has a long history, originating with observed similarities between brain-damaged children and those with ADHD.

This aetiological hypothesis is reflected in the different names the disorder has had, including Minimal Brain Dysfunction (MBD), Minimal Brain Damage, Encephalitis Lethargia etc., all implying that ADHD-children are in some way mentally disabled (Barkley, 1998a, De Armas, 2001; Martin, 2002; Rafalovich, 2001).

Since the 1970's neurobiological theories of ADHD have gained greater prominence. The advances made in neuro-imaging techniques, and the explosion of knowledge about the functioning of the brain, have contributed greatly to the increased research focus on the neurobiological aetiological factors (Barkley, 1998a; De Armas, 2001, Rapoport et al., 2001;).

However, just as a single aetiological factor responsible for the development of the disorder cannot be identified, neither can one neuro-biological factor be singled out as the cause of ADHD. As in the general aetiological meta-theories of ADHD, in the

neurobiological realm, many neurobiological factors play an important role (Barkley, 1998a; Gilles-Thomas, 1989; Wagner, 2000).

Swanson, Castellanos, Murias, LaHoste, and Kennedy (1998a), provide a useful conceptualisation of the different aspects of neurobiological functioning involved when considering the aetiology of ADHD. These include neuroanatomy, genetics, neurochemistry, neuropharmacology and neuropsychology. This structural division will be used in this chapter. Preceding these sections, the neurobiological theory that underlies this project will be discussed.

3.2. Neurobiological theory underlying this project

The neurobiological theory underlying this research project is based on the model of Sagvolden and co-workers (Johansen et al., 2002; Sagvolden, 1999; Sagvolden & Sergeant, 1998b). This model focuses on dysfunctioning dopamine projection branches as aetiological factors in the observed ADHD symptoms.

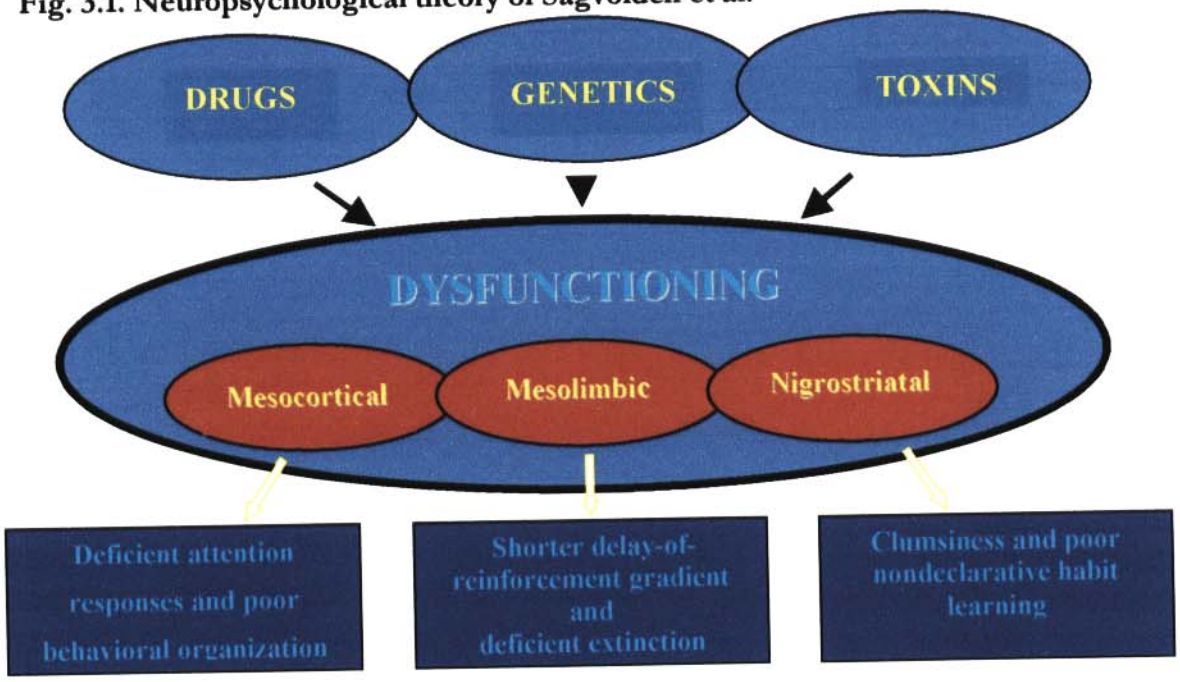
According to this model, altered dopamine functioning causes ADHD. These authors argue that ADHD symptoms are, to a large extent, caused by dysfunctioning dopamine branches impairing non-dopaminergic signal transmission.

The dopamine projection branches that are focussed on in this theory are: the meso-cortical, meso-limbic and nigrostriatal branches.

Various factors may contribute to a dysfunctioning dopamine system. These include genetic factors, drugs, and environmental factors such as pre-, peri-, and postnatal trauma (Sagvolden, 2000; Zappitelli et al., 2001).

Family and twin studies provide evidence for a genetic component in ADHD (Pary et al., 2002). Some environmental pollutants may also alter the functioning of dopamine, as may certain drugs (Solanto, 1998).

Fig. 3.1. Neuropsychological theory of Sagvolden et al.



The specific focus in this study is on the dysfunctioning of the mesocortical branch of the dopaminergic system, which according to the theoretical perspective leads to cognitive impulsiveness.

Importantly, not all the dopamine pathways may be equally dysfunctional in all individuals with ADHD. This also presupposes different treatment strategies for different subtypes, based on the dopamine pathway hypotheses.

As the dysfunctioning of dopamine plays an important role in this theory, the exact functioning of dopamine and it's functioning in Attention-Deficit Hyperactivity / Disorder will be discussed in more detail under the section on neurochemistry.

The different dopamine branches are discussed in the neuroanatomy section, while the observable symptoms will be discussed in the section on neuropsychology.

3.3. Neuroanatomy

Before the advances and developments in brain imaging techniques, localisation of brain areas implicated in ADHD was highly dependant on observed similarities between patients with localised traumatic brain injuries and deductive reasoning from neuropsychological tests (Rapoport et al., 2001).

The similarities found between children with ADHD and adults with frontal lobe damage was an early research finding (Shue & Douglas, 1992). As early as 1936 these similarities were noted, with both the ADHD group (then termed 'minimal brain dysfunction') and the frontal-lobe damaged group displaying behaviour such as excessive restlessness, poor ability to sustain interest in activities, aimless wandering, and excessive appetite (Barkley, 1998b).

Frontal lobe dependant performances, termed "executive functions" have consistently shown impairment in ADHD groups (Grodzinsky & Diamond, 1992; Shue & Douglas, 1992).

The advances in non-invasive brain-imaging techniques have helped greatly not only to confirm the involvement of the frontal lobes, but also to identify other structures involved in the disorder (Rapoport et al., 2001).

The advances in brain imaging included the development of CT, MRI, PET and SPECT scans. An overview of these techniques is in order to facilitate the understanding of the meaning of research results based on these techniques.

Computerised Tomography (CT) scans are computer-generated images of tissue density, produced by tomographically measuring the attenuation of tissue to X-rays passed through the body at different angles (Neuroimaging Tutorials, 2003).

Magnetic Resonance Imaging (MRI) depends on the action of spinning nuclei when exposed to magnetic fields and radio waves (Neuroimaging Tutorials, 2003). Every tissue in the body can be characterised by its normal relaxation values. When tissue is altered by disease, the relaxation values may change. Swanson et al. (1998a) point to the contribution of the convergence of findings from (MRI) studies. These studies have indicated two specific, but rather coarsely defined brain areas, namely the frontal lobes and basal ganglia, that are smaller in Attention-Deficit / Hyperactivity Disorder compared to control groups.

It is important to note that (Barkley, 1998b) has stated that a smaller brain volume does not necessarily imply brain damage. He is of the opinion that differences in brain structure can be accounted for by abnormalities in brain development within the particular regions, caused probably by genetic reasons.

Positron Emission Tomography (PET) is a nuclear medicine technique that produces an image of the distribution of radioactivity in the human body, resulting from the administration of a substance containing radioactive atoms. PET has enhanced our understanding of the biochemical basis of normal and abnormal functions within the body, and permitted biochemical examination of patients as part of their clinical car. These capabilities are important because:

1. The basis of tissue is chemical
2. Diseases result from errors introduced into its chemical systems by viruses, bacteria, genetic abnormalities, drugs, environmental factors, aging and behaviour.
3. The most selective, specific, and appropriate therapy is one chosen from diagnostic measure of basic chemical abnormality.
4. Detection of chemical abnormalities provides the earliest identification of diseases, even in the presymptomatic stages before the disease process has

exhausted the chemical reserves or overridden the compensatory mechanisms of the brain.

5. Assessment of restoration of chemical function provides an objective means for determining the efficacy of therapeutic intervention in the individual patient.

6. The best way to judge whether tissue is normal is by determining its biochemical function (Neuroimaging Tutorials, 2003)

According to Rapoport et al. (2001), the availability of non-invasive brain imaging permits the study of normal and abnormal brain development in childhood and adolescence. They reviewed the pattern and temporal characteristics of anatomic brain magnetic resonance imaging (MRI), in ADHD and Childhood Onset Schizophrenia. They found "consistent, diagnostically specific" patterns of brain abnormality for both of these childhood disorders. ADHD was characterised by a slightly smaller (4%) brain volume (both white and grey matter), less consistent abnormalities of the basal ganglia and a striking (15%) decrease in posterior inferior cerebellar vermal volume.

They came to the conclusion that in ADHD, the developmental pattern suggests an early non-progressive 'lesion' involving neurotrophic factors controlling overall brain growth and selected dopamine circuits (Rapoport et al., 2001). The authors are of the opinion that the findings from their study support other biological models of Attention-Deficit / Hyperactivity Disorder implicating frontal-basal ganglia and dopaminergic circuits (such as the one this study is based on).

The authors point to the fact that the volume reduction in the posterior inferior lobule of cerebellar vermis is important, as this region of the cellular vermis is highly dopaminergic and appears, like most brain volumetric measures to be highly heritable. This, of course, strengthens the dopamine-hypotheses of ADHD, discussed in this chapter under the section neurochemistry.

Other research findings from imaging studies show the involvement of the prefrontal cortex, the striatum (specifically its anterior part, the caudate nucleus) and the basal ganglia (Rapoport et al., 2001; Schrimsher, Billingsley, Jackson & Moore, 2002). Indications are that the prefrontal cortex specifically seems to be involved in the development of symptoms of ADHD (Barkley, 1998b). The research is the most consistent in finding smaller right prefrontal cortical regions. Research indicates that there is a smaller caudate volume, though there are different results as to whether the right or left sides are smaller. Other studies have indicated a different volume in other areas of the basal ganglia (Swanson et al., 1998a).

According to Lezak (1995), the cortex and underlying white matter of the frontal lobes is the site of interconnections and feedback loops between the major sensory and the major motor systems, linking and integrating all components of behaviour at the highest level. "Pathways carrying information about the external environment from the posterior cortex and information about internal states from the limbic system converge in the anterior portions of the frontal lobes, the prefrontal cortex" (Lezak, 1995, p89).

The prefrontal cortex can be subdivided into three functional areas, mostly based on behavioural deficits associated with lesions in these areas. The three areas have projections to different thalamic nuclei, as well as interconnections with other cortical and subcortical structures. These connections are both to and from the prefrontal cortex, thus they can be viewed as bi-directional (Lezak, 1995).

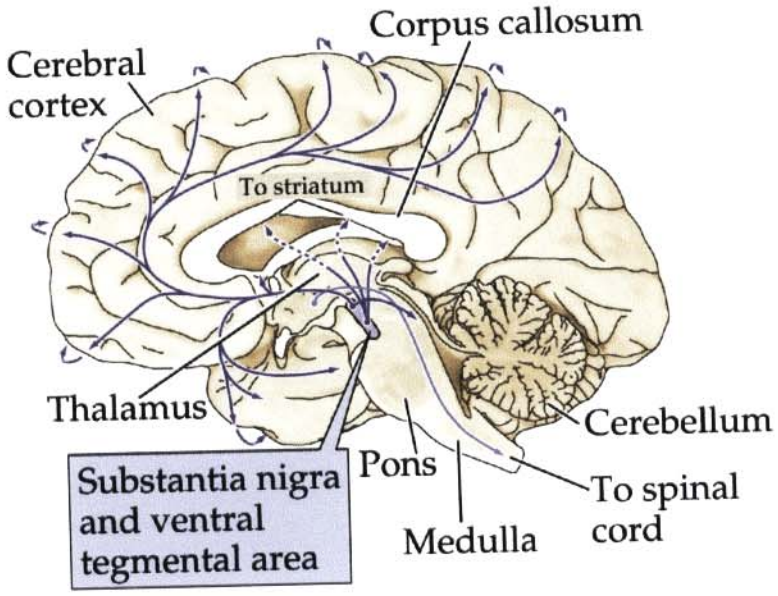
Despite being the subject of frequent study, research on the pathophysiological underpinnings of ADHD (and its specific subtypes) has produced inconsistent results (Lockwood, Marcotte, & Stern, 2001). These authors state that the inconsistencies in neuroimaging studies are perhaps due to the fact that any single brain region may involve

a wide range of neurotransmitter systems. As such, multiple neurochemical theories may be needed to explain different aspects of the disorder.

According to Lockwood et al. (2001), ultimately, the integration of neuroanatomical and biochemical models will be necessary to comprehensively explain the aetiology of ADHD subtypes. The model underlying this project is one attempt at such an aetiological explanation.

Figure 3.2 illustrates the various dopamine projection areas and neuro-anatomical brain structures involved in ADHD.

Fig. 3.2. Dopamine projection areas and neuro-anatomical brain structures involved in ADHD



3.4. Genetics

Heredity plays a distinct role in the aetiology of ADHD (Pary et al., 2002). Monozygotic twins have a higher concordance rate for Attention-Deficit Hyperactivity/Disorder than dizygotic twins and children of a parent with ADHD have a 50% likelihood of having ADHD (Faraone et al., 1995). 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, 1998a). The biological families of ADHD children have high rates of alcoholism, mood disorders, and antisocial personality disorders (Kaplan & Sadock, 1998).

More than one gene may contribute to the development of the disorder. It appears as if genes encoding the dopamine transporters and receptors are implicated. Currently, research seems to be focussing on the DAT1 transporter gene and the D4 receptor gene.

A gene known as DRD4 controls the structure of the D4 DA receptor. The receptor's ability to bind to DA is determined by it's structure, which then influences the impact of synaptic DA on postsynaptic cell activity (Denney & Rapport, 2001).

> Mutations in the dopamine receptor gene can render receptors less sensitive to dopamine. Conversely, mutations in the dopamine transporter gene can yield overly effective transporters that scavenge secreted dopamine before it has a chance to bind to dopamine receptors on a neighboring neuron (Barkley, 1998a, p68).

Genetic polymorphisms in the dopamine receptors may reduce dopamine activity and alter normal development of the meso-cortical and nigrastriatal dopamine systems, which modulate activity in these networks (Castellanos, 1997). Swanson and his co-workers

(Swanson et al., 1998c; Swanson et al., 2000b) have pointed to the association of the dopamine receptor D4 (DRD4) gene located on chromosome 11p15.5 with ADHD.

Recently, UCLA Neuropsychiatric Institute researchers have localized a region on chromosome 16 that is likely to contain a risk gene for ADHD (Fisher et al., 2002; Smalley et al., 2002). The study suggests that the suspected risk gene may contribute as much as 30% of the underlying genetic cause of ADHD and may also be involved in a separate childhood-onset disorder, autism.

If a gene with a major role in ADHD can be identified, it will help researchers and clinicians not only to better understand the biology of this disorder, but also to design and implement more effective treatment strategies. It will also promote understanding in the general population the ADHD is a disorder with a biological base, and that it runs in families.

3.5.Neurochemistry

Various neurotransmitter systems may be dysfunctional in ADHD (Denney, 2001; Pary et al., 2002). The theoretical model underlying this model focuses on the role of dysfunctioning dopamine systems.

ADHD is associated with changes in dopamine functioning (Johansen et al., 2002). One of the aetiological factors proposed in the development of ADHD symptoms is dysfunctioning dopamine branches impairing non-dopaminergic signal transmission (Cohen, Braver, & Brown, 2002; Johansen et al., 2002).

When the dopamine theory of ADHD was published (Levy, 1991), the predominant theory of ADHD was noradrenergic. The effectiveness of stimulants, along with animal models, point to cathecolamine dysregulation as at least one source of ADHD brain dysfunction (Levy et al., 2001). Despite the plethora of scientific knowledge garnered by

such an inductive approach, some authors have pointed to the necessity for deductive models that could provide a meta-organisation of the functioning of neurotransmitter systems (Rapport, Chung, Shore, & Isaacs, 2001).

According to Swanson et al. (1998a), since the 1970's the pre-eminent theory of ADHD has been based on the catecholamine hypothesis. However, after 25 years of research its clinical significance remains unclear. Recently refinements of this theory have focussed specifically on the primary roles of dopamine and norepinephrine

From the theory this study is based on (Johansen et al., 2002; Sagvolden, 1999; Sagvolden & Sergeant, 1998b), it follows that certain brain structures are involved in ADHD, particularly as these relate to the (dys)functioning of dopamine. These are based on the different projection areas. The projection areas are:

Meso-limbic branch

The meso-limbic dopamine pathway projects from the midbrain ventral tegmental area to the nucleus accumbens, a part of the limbic system of the brain. Studies on animal models of ADHD have given morphometric evidence of a hyperfunctioning mesolimbic system (Viggiano, Grammatikopoulos, & Sadile, 2002).

Meso-cortical branch

The meso-cortical pathway is related to the meso-limbic pathway. It also projects from the midbrain ventral-tegmental area, but it sends its axons to the limbic cortex (Stahl, 1996).

Nigro-striatal pathway

The nigro-striatal pathway projects from the substantia nigra to the basal ganglia, and is thought to control movement (Stahl, 1996).

The connections between the neuroanatomical and neurochemical dopamine systems are complex. In addition, the dopamine system in itself is complex and not well understood. For example there are 5 known Dopamine receptors (D1-D5), divided into two types: D1-like (D1 and D5) and D2-like (D2,D3D4) (Stahl, 1996; Swanson et al., 1998c). These receptors are situated in different areas in the brain, depending on the type of neuron, e.g. presynaptic or postsynaptic location. Also genetic variants (polymorhisms) further complicate the understanding of Dopamine systems. With the complexity of the system in mind, any model is necessarily a simplification to enhance understanding.

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

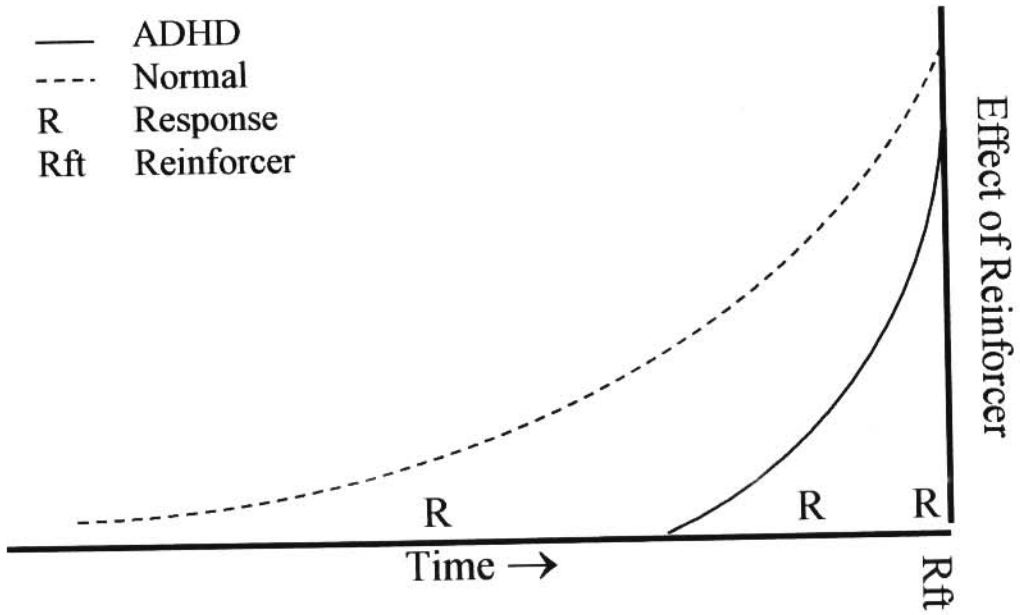
The model used by Johansen et al. (2002) is based on the observation that the behaviour of children with ADHD and normal children is differently affected by reinforcement contingencies. The key diagnostic features of the disorder, impulsiveness, hyperactivity and inattention, may all be due to altered reinforcement mechanisms and a shorter delay-of-reinforcement gradient.

An understanding of reinforcement and extinction is dependent on knowledge of some basic concepts from the behaviourist school of psychology.

A stimulus is a positive reinforcer if its presentation increases the probability of future occurrence of the response that produced it. The reinforcement contingencies are the conditions under which a response produces a reinforcer. (Jordaan & Jordaan, 1992; Kaplan & Sadock, 1998)

Therefore, reinforcers act on responses that have already taken place by increasing the probability of future responding. Reinforcers may vary along several dimensions like density (frequency), the temporal response-reinforcer relationship (continuity), predictability and value (attractiveness). The reinforcing effect is largest when the reinforcer is delivered immediately after the occurrence of the response and wanes as a function of the delay in the delivery of the reinforcer. This relation between the effect of the reinforcer and the time interval between response and reinforcer is commonly known as the "delay of reinforcement gradient" or simply as the "delay gradient" (Johansen et al., 2002). It is important to note that a reinforcer acts not only on the response that produced it, but also, albeit to a lesser degree, on responses emitted earlier (Johansen et al., 2002, Jordaan & Jordaan, 1992; Kaplan & Sadock, 1998).

The delay of reinforcement gradient is illustrated in figure 3.3.



Discontinuation of reinforcer deliveries (i.e. an extinction procedure) starts an extinction process. This process has traditionally been understood as part of the process generated

by reinforcement: responding is maintained as long as reinforcers are delivered contingent on the responses, and stops, or is reduced to the level previous to the reinforcement, when this contingency is discontinued (Johansen et al., 2002, Jordaan & Jordaan, 1992; Kaplan & Sadock, 1998)

3.5.1. The role of dopamine in this process of reinforcement and extinction:

Through electrophysiological measurements it has been shown that dopaminergic neurons can fire in either tonic or phasic modes, and that phasic firing accompanies the anticipatory phases of reward (Schultz, 2002; Schweighofer & Doya, 2003; Wightman & Robinson, 2002).

Extracellular dopamine levels are characterised by low, tonic background activity and short-lasting phasic activity. Reinforcement is associated with dopamine release in the nucleus accumbens shell (Johansen et al., 2002). Dopamine is released when reinforcers start controlling behaviour (acquisition) and is associated with reinforcer unpredictability. Dopamine is released by unpredicted reinforcers, reinforcers with higher values or earlier occurrences than predicted (Johansen et al., 2002; Schultz, 2002; Schweighofer & Doya, 2003; Wightman & Robinson, 2002).

Omissions of predicted reinforcers (extinction) and reinforcers with a lower than predicted reinforcer value is signalled by a short lasting phasic decrease in tonic dopamine activity. The phasic decrease in tonic dopamine activity has also been observed in animals failing to obtain a reinforcer due to erroneous behaviour. Thus, the extinction signal depends on the tonic dopamine activation constituting the background for a phasic depression of dopamine activity (Johansen et al., 2002).

3.5.2. Altered reinforcement processes

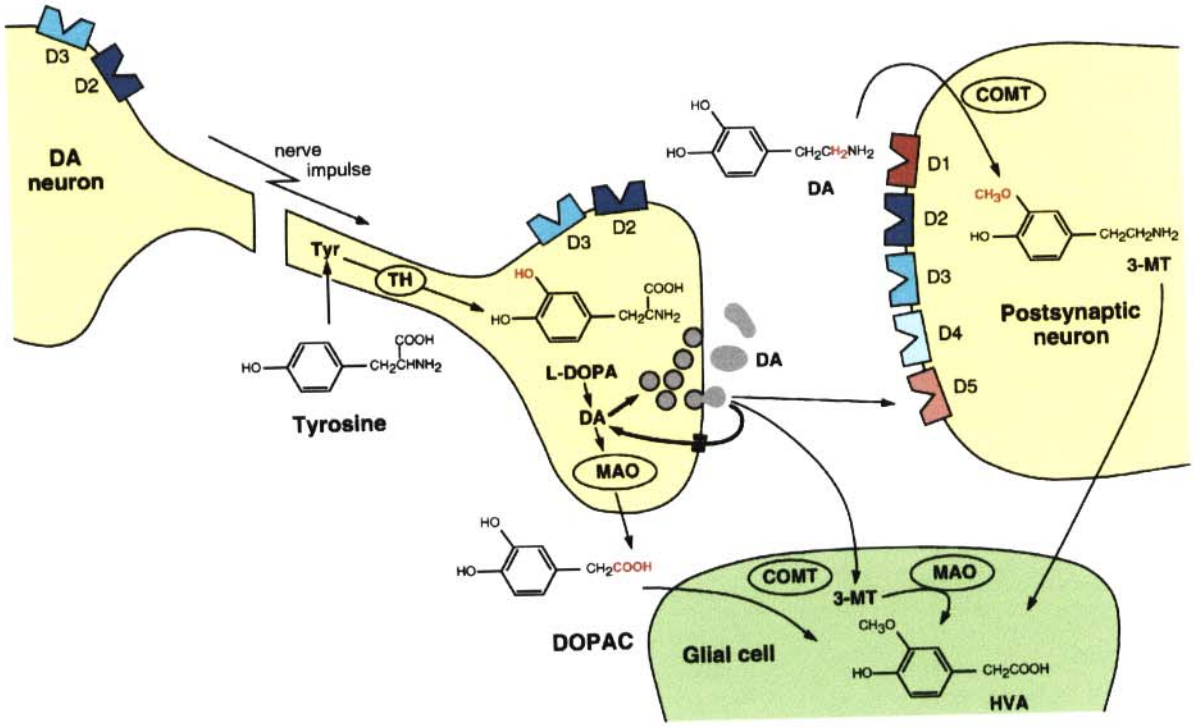
Johansen et al. (2002) have argued that the delay of reinforcement gradient is shorter in ADHD than in normals, implying that only responses in close proximity to the delivery of the reinforcer will be effective in ADHD.

> Assuming that the same phasic extracellular dopamine level is needed in children with ADHD as in normals for reinforcement to take place, several interesting implications will follow. Compared to normals, a reduced tonic dopamine level in children with ADHD will require an increased release of dopamine during the 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. They are also less sensitive to changes in reinforcement contingencies (Johansen et al., 2002, p 41).

The importance of reinforcement is supported by the fact that children with ADHD are not always cognitively impulsive as they temporarily do manage to plan ahead, organise themselves and remember important things, if this behaviour is maintained by potent and frequent reinforcers (Johansen et al., 2002).

The synthesis of dopamine at the synapse is shown in Figure 3.3.

Figure 3.4. Dopamine synthesis



3.6. Pharmacology

Psychotropic medication is commonly used in the treatment of the symptoms of inattention and hyperactivity. However, although it can thus be said to decrease inappropriate behaviour, it does not necessarily increase appropriate behaviour without the psychosocial interventions (Brown, 2000; Whalen, 2001).

Table 3.1. Efficacy ratings of Psychopharmacological agents in the treatments of

Medication	*Efficacy rating*
First line agents	
Stimulants:	
Methylphenidate (Ritalin)	*+++*
d-amphetamine (Dexedrine)	*+++*
Pemoline (Cylert)	*+++*
Second line agents:	
Antidepressants	
Tricyclics	*++*
Bupoprion	*++*
Third line agents	
MAO inhibitors	*+*
Antipsychotics	*+*
+++ = definite efficacy	
++ = probable efficacy	
+ = possible efficacy	

From Gitlin (1996)

As can be seen from the above table, the primary treatment of ADHD is with stimulant medication (e.g. methylphenidate, dextroamphetamines and pemoline). Stimulants have been shown to be effective in the management of ADHD in the majority of patients, although up to 30% may not respond to stimulant treatment (Brown, 2000, Jadad et al., 1999; Weiss & Murray, 2003). After a dramatic increase in the prescription of stimulant medication over the past several years in USA, it is estimated that about 6% of boys and 2% of girls in the USA are now receiving this pharmacological treatment (Kaplan & Sadock, 1998).

Other psychotropic medications used in the treatment of ADHD include tricyclic antidepressants, and alpha adrenergic agonists, but there is limited data available on their effectivity (Guevara, Lozano, Wickizer, Mell, & Gephart, 2002).

Although the topic of ADHD is controversial in itself (some see it as a myth), this controversy is heightened when the psychopharmacological treatment of the disorder is discussed. As the diagnosis of the disorder is time-consuming and complicated, there appears to be fears surrounding over-prescription of stimulant medication. In addition, the controversy seems to centre around possible side effects and fears that it may have long-lasting negative effects. However, to date, researchers could not identify long-term detrimental effects.

Children differ in their responses to stimulant medication, as they do in response to most medications. (Gitlin, 1996) splits improvements into primary and secondary classes. Primary improvements may include attention, distractibility, hyperactivity, and increased mood stability. Secondary improvements are those that may be observed in improved school performance, family relations, and peer relationships.

Methylphenidate is by far the most often prescribed medication for children with ADHD (Gitlin, 1996; Kaplan & Sadock, 1998). Some response is seen very quickly once treatment is started. Gitlin (1996) points to the difficulty in balancing dosing, so as to reach an optimum balance between gaining positive effects without suffering from side effects.

When prescribing for children, methylphenidate is started at 5mg in the morning and at noon. Increments of 5 to 10 mg can be added to obtain the lowest effective dose (<60mg per day). A regimen of two to three doses per day is required because of the short duration of action (Hardman et al., 1996; Pary et al., 2002). Long acting methylphenidate (Ritalin LA® and Concerta®) is a recent development

Side effects from 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 (Hardman et al., 1996; Pary et al., 2002).

Gitlin (1996) identifies the important issue of "drug holidays" – the discontinuation of the medication over weekends and holidays. He points to some positive effects (diminished side effects and continual reassessment of the necessity for the drug). However, he also states that it is important to realise that a significant number of ADHD patients will need to continue the medication into adolescence and beyond.

If a patient does not respond to stimulant medication, the other classes of drugs may be utilised.

It is important to be aware of differences in treatment strategies in Europe and the United States. In Europe, a more conservative approach is adopted, where stimulant medication is prescribed only when a course of psychosocial interventions have proved to be ineffective. The reverse is true in the United States. The early neuropharmacological investigations of ADHD were based on the guiding assumption that the primary neurotransmitter system affected by neuroleptic and stimulant medications was the same (i.e. dopamine) but that the site of action was different (blocking of dopamine receptors by neuroleptics versus stimulating release and blocking re-uptake of dopamine by stimulants). According to Swanson et al. (1998a) investigations have clearly identified the site of action of methylphenidate, which blocks the dopamine transporter and thus increases the temporal and spatial presence of dopamine at the synapse where it is released.

3.7. Neuropsychology

Deficits in various test performances of neuropsychological functioning have been shown in ADHD children. These include impaired performance on tests such as the

Tower of London, Stroop, Wisconsin Card Sorting Test, Pegboard, Trail Making Test, verbal and practical subtests of children's intelligence tests and a plethora of other tests (Lockwood et al., 2001; Pineda et al., 1998; Schmitz et al., 2002a; Smith, Taylor, Rogers, Newman, & Rubia, 2002).

Studies attempting to identify neuropsychological tests that could discriminate ADHD children from normals have rendered mixed results (Rapport, Chung, Shore, Denney, & Isaacs, 2000). Many of these studies are plagued with problems of small experimental groups, methodological errors or deficits in theoretical grounding.

A relatively consistent research finding is deficits in "executive functions" (Pineda et al., 1998; Reeve & Schandler, 2001; Schmitz et al., 2002; Shue & Douglas, 1992). "Executive functions" is an umbrella term applied to a wide variety of complex functions of the prefrontal cortex, including working memory, internalisation of self-directed speech, controlling emotions, motivation and state of arousal and reconstitution (Barkley, 1997a; Bayliss & Roodenrys, 2000).

Thus, executive functions denote an assembly or a summary of psychological processes involved in organisation and planning of behaviour, and do not refer to any basic cognitive or neuropsychological mechanisms underlying these capacities (Tannock, 1998).

It could be said that the term 'executive functions' describes a broad range of behaviours that may be influenced by various mental disorders. These include response inhibition, nonverbal working memory, questioning and reading comprehension and self-regulation of emotion and motivation (Lesaca, 2001). Executive functions are thus a collection of varying abilities that involve regulatory control over thought and behaviour in the service

of goal-directed or intentional action, problem solving, and flexible shifting of actions to meet demands.

Besides formal neuropsychological testing, clinical data about executive function can be obtained by observing an individual's ability to problem solve in the natural environment and assessing how flexible a person is when faced with a changing routine (Lezak, 1995).

From the preceding discussion it should be clear that executive functions is a broad term that is not well defined. Therefore a more specific description, depending on the theoretical orientation adopted, could be more useful in explaining specific frontal lobe-dependent deficits (Bayliss & Roodenrys, 2000).

This is the rationale behind using the term "cognitive impulsiveness" in this study (see sections on theoretical background and the role of dopamine). In this model, the purpose is to disentangle "executive functions" into motor and cognitive impulsiveness, and deficient motor control (Johansen et al., 2002).

Carrillo-de-la-Pena, Otero, & Romero (1993) have argued that impulsiveness is a multidimensional construct. They also indicated that there was more convergence on rating scales with regard to the cognitive element of impulsiveness. This is another part of the rationale behind the replacement of the term "executive functions" with "cognitive impulsiveness" in this study, as thus renaming the function highlights the cognitive aspects of impulsivity.

In general terms, impulsiveness means acting without reflecting and failure to plan ahead (Reber & Reber, 2001). Thus the concept of impulsiveness has both a motor and a cognitive part. Motor impulsiveness is presently defined as bursts of responses with short inter-response times. "cognitive impulsiveness" implies that private events like thoughts and plans are dealt with for short sequences of time with rapid shifts, resulting in

problems generating and following plans, problems organising own behaviour, and forgetfulness and inefficient use of time (Johansen et al., 2002; Sagvolden, Aase, Zeiner, & Berger, 1998).

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

The focus of this study is the dopamine branches projecting to the frontal cortex. Impairment in this area leads to impaired 'executive functions' (Lezak, 1995).(Lazar & Frank, 1998; Sagvolden & Sergeant, 1998; Seidman, Biederman, Monuteaux, Doyle, & Faraone, 2001;)

The frontal cortex receives a major dopamine input from the ventral tegmental area (Stahl, 1996). There are well-documented changes in the structure and function of the right frontal cortex in ADHD. 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-limbic and a dysfunctional meso-cortical dopamine branch will contribute in the development of cognitive impulsiveness.

The importance of learning is supported by the fact that children with ADHD are not always cognitively impulsive as they do manage to plan ahead, organise themselves and remember important things, if this behaviour is maintained by potent and frequent reinforcers. The prediction is therefore, that the symptoms of cognitive impulsiveness as manifested by lack of planning ahead, behavioural disinhibition, poor time management and impairments in working memory, are mediated by a shorter than normal delay-of-reinforcement gradient (Johansen et al., 2002). In this study the fundamental underlying

behavioural changes were subjected to behavioural analysis in Afrikaans speaking primary school children.

3.8. Summary and Conclusion

A variety of factors are important when considering the neurobiological background of ADHD. These include neuoranatomy, genetics, neurochemistry, neuropharmacology and neuropsychology. It is helpful to have a neuropsychological theory to provide an organisation on a meta-level to what is a huge amount of often disparate information.

ASSESSMENT: SCREENING; INTERVIEWS AND TESTS

4.1. Introduction

In most psychiatric disorders, proper assessment underlies the process of diagnosis and treatment. In fact, the assessment process can be seen as the basis from which the diagnostic and treatment procedures spring forth. Hence, inaccurate assessments will have an impact on diagnosis and treatment planning. This is particularly important in ADHD as different subtypes of the disorder may require different interventions to be effective (Hazelwood et al., 2002).

Due to the multi-dimensional nature of ADHD, the process of assessment can arguably be said to be even more complicated than in most other psychiatric disorders. Multiple aetiologies, various clinical presentations and a multitude of co-morbid conditions can impact on results gained from an assessment.

The assessment of children with Attention-Deficit Hyperactivity Disorder is best done in a multi-faceted approach, consisting of (amongst others) the clinical interview, medical tests and behaviour rating scales. A multi-faceted approach has the advantage that limitations of a single assessment method are overcome (Barkley, 1998b; Brown, 2000).

Barkley (1998b) identifies the following goals to be kept in mind in assessment:

1. To determine the presence or absence of Attention-Deficit Hyperactivity Disorder and differentiate it from other childhood psychiatric disorders.

2. Delineate the types of interventions needed to address the disorder

3. Determine the conditions that are co-morbid with Attention-Deficit Hyperactivity Disorder.

4. Identify the pattern of the child's psychological strengths and weaknesses.

Making an accurate diagnosis is difficult in children, and even harder in adults, since no specific test is diagnostic of ADHD (Brown, 2000; Pary et al., 2002). According to these authors, matters are further complicated when other disorders co-exist. Symptoms may overlap with other psychiatric disorders, confounding precise diagnosis. They are of the opinion that a thorough history and evaluation of the clinical presentation are critical, and reports from parents, teachers and others involved may help substantiate patient information.

Thus it seems as if a thorough clinical interview, medical tests and behaviour rating scales are imperative in the diagnostic process. Meyer and Aase (2003) state that the diagnosis of the disruptive behaviour disorders (of which Attention-Deficit Hyperactivity Disorder is one) requires both medical and psychosocial expertise.

4.2. Culturally-sensitive assessment

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).

Hyperactivity (and hyperactive behaviours) is a construct of western societies of America, Australia, and Europe (Leung et al., 1996). As culture may thus 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).

Assessment instruments in common use in one society could be misleading or invalid when used with culturally different students (Reid et al., 1998).

Important factors in psychometric assessments are the validity and reliability of the instruments used. The validity of an assessment instrument relates to its property of measuring that which it is supposed to measure (Reber & Reber, 2001). Thus an intelligence test should measure intelligence, and not something else.

Reliability is a generic term for all aspects of the dependability of a measurement device or test (Reber & Reber, 2001). A test is reliable if it can be shown to be consistently valid i.e. it measures what it is supposed to measure repeatedly, over time.

These two factors, validity and reliability, can be influenced by a variety of factors, one of which is culture. Thus assessment tools should in all cases (including that of ADHD) be valid and reliable in the cultural context within which it is being administered. In testing, standardisation refers to the process of establishing a set of procedures for the administration of a particular test, the scoring techniques to be used and the methods of evaluation and interpretation of obtained scores (Reber & Reber, 2001). Obviously, a test can only be said to be standardised when it is both reliable and valid.

> 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 Attention-Deficit Hyperactivity Disorder is just a matter of cultural definition. Attention-Deficit Hyperactivity Disorder is defined as a neurologically based, genetically transferred, developmental disorder. As such, Attention-Deficit Hyperactivity Disorder 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 Attention-Deficit Hyperactivity Disorder (Meyer & Aase, 2003, p 173).

4.3. The clinical interview

Parent and child interviews are recommended in making the diagnosis of ADHD (Barkley, 1998b; Brown, 2000; Kaplan & Sadock, 1998). Although often controversial because of reliability issues, they do provide a wealth of information. After all, the parent is presumably generally the person who knows his child's history most intimately.

There are standardised interviews for the assessment of child and adolescent psychopathology. The most current, standardised 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). 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, ODD or CD. Their disadvantage is that they are time consuming and cumbersome.

The diagnostic Interview for Children and Adolescents (DICA) (Boyle et al., 1993) is a structured interview that provides categorical information on 185 symptoms reflecting the DSM-IV diagnostic criteria. Versions for parent and child and for adolescents are available.

Clinical interviews may provide a rich source of information, but may not systematically cover every clinical area. To increase clinical information, the clinician may use semi-structured or structured interviews such as the Diagnostic Interview Schedule for Children Revised (DISC-R), Barkley and Murphy's (1998) semi-structured interview and rating scales (Kaplan & Sadock, 1998).

4.4. The Medical Examination

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

4.4.1. The Medical Interview

In many areas the structure and content of the medical interview overlaps with that of the psychological interview, the difference between the two being that 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, 1998b).

Three major purposes of the medical interview are (1) differentiation between the diagnosis of Attention-Deficit Hyperactivity Disorder from other medical conditions, (2) a thorough evaluation of any coexisting conditions that may require medical management, (3) to determine whether physical conditions that are contra-indications for treatment with medications (Barkley, 1998b).

4.4.2. Physical Examination

Although there are no results from a physical examination that can be used in isolation to diagnose Attention-Deficit Hyperactivity Disorder, a thorough physical examination may be useful in providing a detailed clinical picture. 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, 1998b; Kaplan & Sadock, 1998).

Because of the considerably greater distress Attention-Deficit Hyperactivity Disorder children present to their care-givers, 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, 1998b).

4.4.3. Laboratory tests

Although some research reports have found a variety of physical, physiological and psychophysiological measure to assess potential differences between Attention-Deficit Hyperactivity Disorder and other clinical or control groups of children, none of these laboratory measures are of value in the diagnostic process as yet (Barkley, 1998b; Brown, 2000; Kaplan & Sadock, 1998).

> Parents, teachers, or even other mental health professionals are sometimes mislead by reports of such findings or by the conclusion that Attention-Deficit Hyperactivity Disorder is a biologically based disorder, and they frequently ask for their children to be tested medically to confirm the diagnosis. At this moment, not such test exists. Consequently laboratory studies, such as blood work, urinanalysis, chromosome studies, electroencephalograms, averaged evoked responses, magnetic resonance imaging, or computerised axial tomograms should not be used routinely in the evaluation of Attention-Deficit Hyperactivity Disorder children. Only when the medical and developmental history or physical exam suggests 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, 1998b, p288).

4.5. Behaviour Rating Scales

Rating scales are important tools in the assessment and diagnosis of children with behaviour problems. They provide a wealth of information from people who have spent years with the patient being assessed.

There are several advantages of using rating scales in the assessment of Attention-Deficit Hyperactivity Disorder. They are easy to use, they provide a great deal of information about different behaviours across different environments and informants, they can determine the amount of "deviance" from "normal" child behaviour, they allow for normative comparisons, and they can be used to determine treatment effectiveness over time (Barkley, 1997c; Reid et al., 1998)

There are several scales with excellent psychometric properties available for the assessment of Attention-Deficit Hyperactivity Disorder 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. Some common parent rating scales are: Disuptive Behaviour Rating Scale (DBD) (Pelham, Jr., Gnagy, Greenslade, & Milich, 1992; Pillow, Pelham, Jr., Hoza, Molina, & Stultz, 1998); Child behaviour Checklist (CBCL) (Achenbach, 1991a; Achenbach, 1991b), the Conners rating scales (Conners, 1998, Conners, Sitarenios, Parker, & Epstein, 1998b; Conners, Sitarenios, Parker, & Epstein, 1998a) and the Home Situation Questionnaire (Barkley, 1997c)

The effect that culture can have on an assessment instrument's validity and reliability has already been discussed. Suffice it to say that this may also be the case in behaviour rating scales. Clinicians should be cautious in administering and interpreting rating scales on populations for which it has not been standardised.

4.6. Psychological and Psycho-educational assessment

As stated previously, most psychometric tests are not considered essential to making the diagnosis of ADHD. The diagnostic process consists of an integration of data from

various sources, and reliance on one specific source (such as data from a psychometric evaluation) is considered insufficient (Barkley, 1998b; Brown, 2000; Pary et al., 2002a).

A psychometric evaluation is indicated when data from different sources appears to be disparate, when a clinician wants to differentiate between possible co-morbid disorders, or when the level of impairment in function needs to be ascertained (Barkley et al., 1998).

The reliability of psychometric instruments in the assessment of ADHD has been questioned by some, but as explained above, they do have some value. "Even if the design of highly reliable instruments cannot be immediately realised, they may play a complementary role in improving the sensitivity and specificity of diagnostic decisions while providing information concerning individual differences in treatment response" (Rapport et al., 2000, p.555).

Psychometric instruments may be useful in giving an indication of the level of impairment present. For example, some researchers see the continuous performance tests, which are standardised measures of attention and impulsivity, as useful in assessing distractibility and inattention, whereas others disagree (Brown, 2000; Kaplan & Sadock, 1998; Rapport et al., 2000).

A variety of psychometric instruments have been used in the assessment of children with ADHD, including intelligence tests, general neuropsychological batteries, individual neuropsychological instruments and projective tests (Barkley, 1998b; Brown, 2000; Rapport et al., 2000).

In a review of the use of psychometric instruments, Barkley (1998b) found that none were indicated as a basis on which to make a diagnosis. They can be usefully applied in the contexts explained above, such as differentiating between co-morbid disorders or to indicate the severity of functional impairment.

Where a clinician suspects the presence of a co-morbid learning disorder, psycho-educational assessment instruments can be of particular value in discerning whether impairments are due to core ADHD symptoms or the learning disorder (Brown, 2000). This is especially important when it is taken into consideration that learning disorders often co-exist with ADHD (Kaplan & Sadock, 1998).

In research studies, various neuropsychological tests, such as the Wisconsin Card Sorting Test (WCST), the Tower of London (TOL), the Stroop word-color test, the Go-no-go test, the stop task and the TOVA. 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, 1998b; Reeve & Schandler, 2001).

PROBLEM STATEMENT

5.1. Introduction

There are a variety of cognitive deficits observed in people with ADHD. These include deficits in the functions of self-regulation, self-initiation and inhibition, strategic planning, cognitive flexibility, and impulse control. These functions are commonly termed 'executive functions' (Bayliss & Roodenrys, 2000). However, 'executive functions' is an umbrella term, describing a variety of neuropsychological functions (Barkley, 1997a; Bayliss & Roodenrys, 2000). A criticism of studies of executive functions is that they are not specified theoretically (Pennington & Ozonoff, 1996). In this study an attempt is made to overcome this shortcoming by using the term 'cognitive impulsiveness' (which derives from the neuropsychological theory underlying this project) as a substitute for the term executive functions.

5.2. The problem: Assessing ADHD according to a Neuropsychological model

5.2.1. Problem statement

The main focus of this study is on cognitive impulsiveness in children with ADHD. Cognitive impulsiveness is a more specific term than executive functions, which indicates a variety of cognitive deficits, or neurological "soft signs", often observed in ADHD-children (Barkley, 1998b; Bayliss & Roodenrys, 2000). For a further discussion on cognitive impulsiveness see the section on neuropsychology in Chapter 3.

Impaired executive functions are usually associated with frontal lobe dysfunction (Pineda et al., 1998; Reeve & Schandler, 2001; Schmitz et al., 2002b; Shue & Douglas, 1992). The

frontal cortex receives a major dopamine input from the ventral tegmental area (Cohen, Braver & Brown, 2002; Stahl, 1996). There are well-documented changes in the structure and function of the right-frontal cortex in ADHD (Oades, 1998; Solanto, 1998).

According to the model of Johansen et al. (2002), cognitive impulsiveness is viewed as a product of neurobiological learning and growth.

In the process of development, learning mediated by the meso-cortical branch will be an important input for the acquisition of components necessary for planning and organisation of behaviour. Thus, both a dysfunctioning meso-limbic and a dysfunctional meso-cortical dopamine branch will contribute to the development of cognitive impulsiveness.

The importance of learning is supported by the fact that children with Attention-Deficit Hyperactivity Disorder are not always cognitively impulsive, as they do manage to plan ahead, organise themselves and remember important things, if this behaviour is maintained by potent and frequent reinforcers (Douglas, 1999).

The prediction is therefore, that the symptoms of cognitive impulsiveness as manifested by lack of planning ahead, behavioural disinhibition, poor time management and impairments in working memory, are mediated by a shorter than normal delay-of-reinforcement gradient.

5.2.2. Aim of the study

The main aim of the research is to submit the neuropsychological theory of ADHD (Johansen et al., 2002; Sagvolden, 1999; Sagvolden, 2000; Sagvolden & Sergeant, 1998) to systematic and experimental testing among Afrikaans speaking primary school children screened for symptoms of ADHD. This theory predicts that ADHD symptoms are to a

large extent caused by dysfunctioning dopamine systems impairing non-dopaminergic signal transmission.

5.2.3. Hypotheses

Research hypothesis 1:

Children with ADHD will have lower performances on tests that measure the mesocortical dopamine branch, than children who do not have ADHD. There will be differences in scores between the genders.

Null hypotheses 1:

Children with ADHD will not have lower performances on tests that measure the meso-cortical dopamine branch, than children who do not have ADHD. There will be no differences between the genders.

Specific null hypotheses derived from Research hypothesis 1:

1. Children with ADHD will not have lower scores than a control group on the Numbers subtest of the SSAIS-R. There will be no differences between the genders.

2. Children with ADHD will not have lower scores than a control group on the Story Memory subtest of the SSAIS-R There will not be differences in performance between the genders.

3. Children with ADHD will not have lower scores than a control group on the Pattern Completion subtest of the SSAIS-R. There will not be differences in performance between the genders.

4. Children with ADHD will not have lower scores than a control group on the Blocks Completion subtest of the SSAIS-R. There will not be differences in performance between the genders.

5. Children with ADHD will not have higher perseverative error scores on the Wisconsin Card Sorting Test than children without ADHD. There will not be differences in performance between the genders.
6. Children with ADHD will not make more non-perseverative errors on the Wisconsin Card Sorting Test than children without ADHD. There will not be differences between the genders.
7. Children with ADHD will not achieve less categories on the Wisconsin Card Sorting Test than children without ADHD. There will not be differences in performance between the genders.
8. Children with ADHD will not have lower scores on the Tower of London than children without ADHD. There will not be differences in performance between the genders.
9. Children with ADHD will not have higher error scores than a control group on phase 1 of the Stroop test. There will not be differences in performance between the genders.
10. Children with ADHD will not have higher error scores than a control group on phase 2 of the Stroop test. There will not be differences in performance between the genders
11. Children with ADHD will not have higher errors scores than a control group on phase 3 of the Stroop test. There will not be differences in performance between the genders.

Research hypothesis 2:

There will be differences between the scores of the subtypes. The groups with ADHD will have lower scores than the group without ADHD.

Null hypotheses 2:

There will not be differences between the scores of the subtypes. The groups with ADHD will not have lower scores than the group without ADHD.

Specific null hypotheses derived from Research hypothesis 2:

1. The groups of children with the hyperactive/impulsive, inattentive and combined form of ADHD will not have lower scores on the Numbers subtest of the SSAIS-R than a control group.
2. The groups of children with the hyperactive/impulsive, inattentive and combined form of ADHD will not have lower scores on the Story Memory subtest of the SSAIS-R than a control group
3. The groups of children with the hyperactive/impulsive, inattentive and combined form of ADHD will not have lower scores on the Blocks subtest of the SSAIS-R than a control group.
4. The groups of children with the hyperactive/impulsive, inattentive and combined form of ADHD will not have lower scores on the Pattern Completion subtest of the SSAIS-R than a control group.
5. The groups of children with the hyperactive/impulsive, inattentive and combined form of ADHD will not make more perseverative errors on the Wisconsin Card Sorting Test than a control group.
6. The groups of children with the hyperactive/impulsive, inattentive and combined form of ADHD will not make more non-perseverative errors on the Wisconsin Card Sorting Test than a control group.
7. The groups of children with the hyperactive/impulsive, inattentive and combined form of ADHD will have lower categories achieved on the Wisconsin Card Sorting Test than a control group.

8. The groups of children with the hyperactive/impulsive, inattentive and combined form of ADHD will not have lower scores on the Tower of London test than a control group .
9. The groups of children with the hyperactive/impulsive, inattentive and combined form of ADHD will not have higher error scores on Phase 1 of the Stroop test than a control group .
10. The groups of children with the hyperactive/impulsive, inattentive and combined form of ADHD will not have higher error scores on Phase 2 of the Stroop test than a control group.
11. The groups of children with the hyperactive/impulsive, inattentive and combined form of ADHD will not have higher error scores on Phase 3 of the Stroop test than a control group

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

METHODOLOGY

6.1. Research Design

This is a quantitative study and the quasi-experimental research design was used, as the subjects could not be randomly assigned to the conditions for the independent variable because they already exhibit the variable. Children screened positively for ADHD (n=31) were compared with normal children (n=31). The children were compared on cognitive impulsiveness (dysfunctional meso-cortical dopamine system).

6.2. Sample

The sample was drawn from Afrikaans speaking primary school children aged 6 – 12 who were screened for ADHD using the Disruptive Behaviour Disorders Rating Scale (DBD) (Meyer, Eilertsen, Sundet, Tshifularo, & Sagvolden, in press; Pelham et al., 1992b; Pillow et al., 1998). The control group was matched with the experimental group for age, sex and socio-economic status (SES).

Table 6.1 gives an overview of the demographic characteristics of the sample when compared according to age.

Table 6.1 Demographic characteristics (age groups)

	Age (age groups)		
Group	**N**	**Means (in months)**	**Std. Dev.**
Clinical male	23	127.30	20.33
Clinical female	8	119.25	24.94
Control male	23	127.04	17.58
Control female	8	120.12	13.90
All groups	62	125.24	19.10

The differences in age between the clinical and control groups were statistically not significant.

Table 6.2. gives an overview of the demographic characteristics of the sample when organised according to subtype.

Table 6.2. Demographic characteristics (subtype).

	Age (subtypes)		
Group	**N**	**Means (in months)**	**Std. Dev.**
Combined	9	116.44	23.85
Hyperactive/Impulsive	13	127.54	18.66
Inattentive	9	131.00	22.46
Normal	31	125.16	16.78

The differences in age between the subtypes were statistically not significant.

6.3. Measurement instruments

6.3.1. Screening device

The Disruptive Behaviour Disorders rating scale (DBD) (Pelham, Jr., Gnagy, Greenslade, & Milich, 1992; Pillow, Pelham, Jr., Hoza, Molina, & Stultz, 1998) was used for screening the children who formed the two groups: ADHD and non-ADHD children. This instrument has been translated, standardised and norms been established for all the language groups in the Limpopo Province (Meyer, Eilertsen, Sundet, Tshifularo, & Sagvolden, 2002).

The DBD assesses the presence and degree of ADHD-related symptoms (inattention and hyperactive/impulsive); Oppositional Defiant Disorder (ODD) and Conduct Disorder (CD) as formulated in the DSM IV. There are 18 items on the scale that measure ADHD-related symptoms. Respondents are asked to rate the behaviour on a four point scale comprising of the following options: not at all (0); just a little (1); pretty much (2); and very much (3).

The total score is added up, and compared to the cut-off point of the 95th percentile, which has previously been identified as clinically significant (Barkley, 1997c; Barkley & Murphy, 1998). The group scoring below the 85th percentile was taken as the control group.

Table 6.3 Illustrates the results of the DBD screening procedure.

Table 6.3 DBD scores (clinical and control groups)

Group	N	Inattention scale		Hyperactive/Impulsive scale	
		Mean	Std. Dev	Mean	Std. Dev
boys 6-9 comb	3	22.33	0.58	22.333	3.79
boys 6-9 h/i	5	15.00	2.00	19.200	1.78
boys 6-9 inatt	2	21.50	0.71	12.000	7.07
boys 6-9 control	10	2.20	2.62	2.300	3.09
girls 6-9 comb	3	22.00	1.73	20.667	4.62
girls 6-9 h/i	1	16.00	0.00	19.000	0.00
girls 6-9 inatt	1	22.00	0.00	15.000	0.00
girls 6-9 control	5	3.200	3.03	3.8000	5.31
boys10-13comb	3	23.00	1.73	22.33	3.77
boys 10-13 h/i	5	17.20	2.39	21.60	5.41
boys 10-13 inatt	5	22.00	1.23	13.40	3.68
boys 10-13 control	13	3.76	4.94	4.92	4.49
girls 10-13 comb	0				
girls 10-13 h/i	2	19.50	.7071	19.50	2.12
girls 10-13 inatt	1	22.00	0.000	14.00	0.00
girls 10-13 control	3	3.33	3.055	4.333	5.86

The differences in scores on both the inattention and hyperactive/impulsive scales of the DBD were statistically significant between the clinical groups and normal controls.

6.3.2. SSAIS-R

The Senior South African Individual Scale (Revised) (SSAIS-R) is an instrument used to measure general intelligence. It consists of verbal and practical sections, each containing five subtests. The instrument has been standardised for South African pupils between the

ages of 7 years 0 months and 16 years 11 months with Afrikaans or English as mother tongue and the relevant norms are available.

> The SSAIS-R is used to obtain a differential picture of certain cognitive abilities. Firstly the level of general intelligence is determined, for instance to predict scholastic achievement. Secondly relative strengths and weaknesses in certain important facets of intelligence are evaluated to obtain diagnostic and prognostic information (Van Eeden, 1997, p. 3).

Four subtests (two from the verbal section and two from the non-verbal section) of the SSAIS-R were administered.

Test 1: Number Problems

Description:

The test consists of 20 verbally formulated arithmetic problems. 11 items are set verbally, while the other nine are also set on cards. Items 1-11 are set without any further instructions. Before item 12 is set, the following instruction is given (in the testee's own language - Afrikaans):

> *Now I want you to read this question with me. Give me your answer as soon as you have finished. You are not allowed to use a pencil or paper, but you may read the question again if you want.*

The test is stopped after five consecutive zero scores (wrong responses).

Aim

The test measures numerical reasoning ability and underlying that abstract thinking. Productive concentration ability is also important to succeed in this test.

Rationale

The test is based on the assumptions that the ability to solve number problems is an indication of general intelligence. The basic numerical computations, namely addition, subtraction, multiplication and division, are learned through practical experience and in formal education.

According to the theory this study is based on, a dysfunctional meso-cortical dopamine branch will lead to cognitive impulsiveness, i.e. the inability to inhibit impulses on a cognitive level (in the same manner as motor impulsiveness cannot be inhibited). This forms the rationale behind selecting this particular subtest, as the ability to perform mental arithmetic will presumably be highly dependent on the ability to inhibit initial responses. As this test provides no external structure (in the form of pen and paper), which could compensate for deficiencies in cognitive abilities, it is seen as a good measure of cognitive impulsiveness

Test 2: Story memory

Description

The test consists of a short story of three paragraphs that is read to the testee. The testee is then immediately asked to verbally repeat the facts that he is able to remember. There are 43 facts/items in the story.

The testee is given the following instruction:

> *I am now going to read a short story to you. You have to listen very carefully, as I am going to ask you to repeat everything you can remember afterwards. Try to remember everything I am going to read.*

Aim

The test measures short-term auditory memory. Meaningful verbal learning matter is used to measure the testee's ability to pay attention in a relatively simple situation.

Rationale

The test is based on the assumption that logical memory (the ability to repeat, not necessarily verbatim, essential content/meaning) is one of the abilities of which a certain minimum is required at every level of intellectual functioning. The test is probably a good measure of general intelligence, especially at the lower levels of intelligence, and may also have diagnostic value.

In terms of the specific theoretical background of this study, a person who is cognitive impulsive is likely to have a lower performance on this test due to two factors. Firstly, cognitive impulsiveness will inhibit the ability to pay attention to the story being read. Secondly the testee is unlikely to consider possible correct answers when repeating the story.

Test 3: Pattern completion.

Description

Partially completed patterns have to be completed. Each item consists of three figures from which the testee has to deduce a pattern in order to draw the fourth figure. Free responses are required of the testee. The test consists of four practice examples which are taken into account in the scoring, and a further 15 items. There is a time limit of 40 seconds on items 1-6, and a limit of 60 seconds on items 7-15. The test is discontinued after six consecutive wrong responses.

The testee is given the following instruction:

> *Have a look at this drawing. It is a pattern or design that is partially completed. I want you to complete the design in the space provided.*

Aim

The test is a non-verbal measure of the processes underlying logical thinking. Accurate visual perception, concrete reasoning with the help of figures, concept formation and concentration are important to succeed in this test. The mental manipulation of the pattern parts comprises mainly synthesis in the easier items, whereas the more difficult items possibly require verbalising the observed relations.

Rationale

The test is based on the assumption that reasoning by means of analogies is an indication of general intelligence. It also requires the testee to perform certain cognitive calculations (although pen and paper is provided on which to draw the correct designs, the testee is not allowed to use paper to work out the correct answer, i.e. the process of identifying the correct pattern has to be performed mentally). A person who is cognitively impulsive is thus unlikely to inhibit initial responses and will thus have a greater number of wrong responses.

Test 4: Block Designs

Description

Plastic cubes are used to copy patterns from an example. A model is presented for items 1 and 2 and design cards are used for the other items. The test consists of 15 items of which the first three are also used as practice examples. Four cubes/blocks are used for the first seven items and nine blocks for the remaining items. There is a time limit on each item. The test is discontinued after four consecutive wrong scores.

Aim

The test measures non-verbal intelligence and non-verbal problem-solving skills. The testee has to solve problems in spatial relations by using logical reasoning. Shapes have to

be observed and analysed by reducing a whole (design) into its component parts and then reassembling them in an identical design.

The test therefore measures the testee's non-verbal concept formation, including perceptual organisation, spatial visualisation and orientation, and abstract conceptualisation. Concentration and visual-motor co-ordination are also important.

Rationale

The test is based on the assumption that the ability to analyse, synthesise and copy an abstract two-dimensional geometric pattern is a valid criterion of general intelligence. This test requires a certain amount of perseverance and dedication, as the tests become progressively more difficult. It has also been used as an assessment of neuropsychological dysfunction. As a measure of cognitive impulsiveness, it could identify the person who is unable to persevere long enough to complete the design correctly.

6.3.3. Wisconsin Card Sorting Test (WCST)

The WCST is a neuropsychological test that assesses the ability to form abstract concepts, to sustain attention, and to shift cognitive set flexibly in response to changing rules while inhibiting inappropriate responses. It assesses organisational capacity, attention shifting, and sustained attention (Schmitz et al., 2002b),

According to Lezak (1995) the WCST is one of the most commonly administered psychometric measures of frontal lobe dysfunction.

The computerised version (Ormond Software Enterprises, 1998)was used for testing and scoring. The subject has to sort cards according to the colour, shape and number of stimuli that are depicted. After every sort the subject receives accuracy feedback that

s/he must use to figure out what the correct sorting rule is. The sorting rule changes after every 10 consecutive responses, cycling through colour, form and number.

In this study the testees' performances were evaluated according to the following criteria:

a) Accuracy and Set Shifting

Correct sorts: A correct response is one that matches the sorting rule.

Incorrect sorts: An incorrect response is one that does not match the sorting rule.

Categories shifted: The sorting rule shifts after every 10 correct consecutive responses. The rule changes through colour, form and number. The number changes is denoted as categories achieved.

b) Perseveration

Perseveration response: A perseverative response is defined as one that matches the perseverated-to 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.

Perseverative errors: A perseverative error is an incorrect perseverative response.

Non-perseverative errors: A non-perseverative error is one that is incorrect but not perseverative.

6.3.4. Tower of London (TOL)

The TOL has been used in normal and neuropsychological populations as an assessment instrument of planning ability (Phillips, Wynn, McPherson, & Gilhooly, 2001). This task places heavy emphasis on working memory, especially non-verbal working memory (Barkley, 1998). The test requires the subject to construct a design using coloured pegs of

different sizes and three upright pegs, employing the least moves possible and with several constraints. Forethought and planning are felt to be instrumental to performance in this task. It requires that individuals be able to mentally represent and test out various ways of removing and replacing beads on a set of pegs or spindles to match the design presented by the examiner. This task involves substantial mental planning that must occur before and while undertaking actual motor execution or arrangement.

The TOL was initially developed by Shallice (1982) in order to study impairments in planning. It was based on the Tower of Hanoi problem. In the TOL, coloured beads must be moved one by one from an initial state to match a goal state. Instructions are given to plan the whole sequence of moves that must be carried out mentally, before executing the sequence.

Successful completion of this test requires mentally planning the moves to be made and thus inhibiting the impulse to move discs without a strategy. Thus a person who is cognitively impulsive will struggle to mentally plan the strategy and to inhibit the impulse to start moving discs.

6.3.5. Stroop Word-Color Test

The Stroop is a timed test measuring the ability to suppress or inhibit automatic responses. It measures the facility with which the individual can shift his perceptual set to conform to changing demands and suppress a habitual response in favour of an unusual one. (Lezak, 1995). There are numerous versions of the Stroop. The key comparison is between a condition in which responses are habitual (e.g., indicate the colour of the letters) and a condition in which responses are unusual (i.e. indicate the colour of the letters despite the fact that they spell a different colour word). The examinee must read the names of colours although the names are printed in a different coloured ink from the colour specified in the name (e.g. the word "red" is printed in blue ink). The task requires

the examinee to inhibit the cognitive impulse to read and pronounce the word, and substitute this impulse with naming the colour.

A computerised version of the Stroop was used (Ormond Software Enterprises, 1998). It consists of three phases. In the first phase the testee is presented with rows of words on a monochrome screen. The testee has to read the words and every error is recorder.

In the second phase the testee is represented with rows of coloured blocks. The testee has to name the colours and every error (e.g. a wrong colour is assigned to a block) is recorded.

In the last phase the testee is presented with names of colours presented in different colour letters than the word they represent (e.g. the word blue will be written in red). The testee is required to name the colour of the letters, i.e. inhibit the impulse to read the word. An individual who is cognitively impulsive will experience difficulty in inhibiting the impulse to read the word, rather than naming the colour.

6.4. Procedure

Written permission was obtained from the Limpopo Province of Education and the Principals of the schools involved, to conduct this study. Parents (of the clinical and control groups) were given a letter describing the study and were asked to complete a consent form. The Ethics Committee of the University of the North approved the study.

The sample was drawn from two Afrikaans-language primary schools within a 60 kilometre radius of one another. Teachers and parents were asked to complete the Disruptive Behaviour Disorders checklist (DBD).

A biographical questionnaire was completed. The children on ADHD medication were asked not to take any for at least 16 hours prior to assessment. Children with an IQ < 80,

a history of neurological trauma, psychosis or other severe psychiatric disorders were not included in this study.

The complete battery was administered in one sitting. The children were tested during school time. The researcher and two assistants conducted the testing. The researcher is registered with the Health Professions Council of South Africa as a psychometrist and has extensive experience in the administration of psychometric tests, especially the SSAIS-R. The assistants were involved with the administration of the other tests on various other study groups (in the same project), before commencing with this process of administration and were also well-versed in the assessment instruments. Before the actual neuropsychological testing commenced, the children were acquainted with a computer and the use of a mouse. The battery consisting of the four subtests of the SSAIS-R, WCST, Stroop and TOL multiple schedule task were administered to the experimental and control groups.

6.5. Method of Data Analysis

The computer programmes SPSS 11 (SPSS, 1999) and STATISTICA 5 (StatSoft Inc., 2002) were employed.

ANOVA models were used to investigate possible between-group differences (groups were divided according to age, gender and subtype) in raw scores. Duncan's multiple range test was used to perform multiple comparison procedures.

Chapter 7

RESULTS

7.1. Introduction

The aim of this study was to establish whether there are significant differences on the measures of cognitive impulsiveness between children screened for symptoms of ADHD and a control group. A battery of neuropsychological tests was used in this assessment. The battery included the Senior South African Individual Scale (Revised), Wisconsin Card Sorting Test (WCST), Tower of London (TOL) and the Stroop Word/Color interference test. Scores obtained from the administration of these measures were compared for significant differences between clinical and control group, gender, age and subtype.

The results are presented in the following format: firstly the descriptive statistics are provided for each individual test. The results for all the parts of the test are first given in table format. Thereafter it is given in graph format in order to provide a graphical representation. This has a double function: it makes it easier to interpret individual group scores as well as enabling easier between-group comparisons.

The statistical analysis (Analysis of Variance and Post-hoc Duncan tests) for each individual forms the second part of the chapter.

7.2. Results of the study

7.2.1. Descriptive statistics

SSAIS-R

Table 7.1 illustrates the descriptive statistics for the subtests of the SSAIS-R according to the age groups.

Table 7.1: SSAIS-R descriptive statistics (age groups)

		Number problems		Story memory		Patterns		Blocks	
	N	Mean	SD	Mean	SD	Mean	SD	Mean	SD
ADHD Boys 6-9	10	10.4	2.8	6.4	1.4	10.7	3.8	10.5	2.9
Control Boys 6-9	10	12.7	3.4	9.5	2.5	12.1	4.4	10.3	2.9
ADHD Boys 10-13	13	7.3	2.3	5.9	2.7	9.9	2.1	10.0	3.5
Control Boys 10-13	13	11.3	2.5	8.7	2.7	11.5	2.9	12.0	2.4
ADHD Girls 6-9	5	7.0	1.7	3.8	1.5	6.6	3.8	7.6	2.4
Control Girls 6-9	5	11	2.0	9	1.6	13.4	2.4	11.6	2.2
ADHD Girls 10-13	3	9.3	1.5	7.7	0.6	10.7	0.6	12.7	4.0
Control Girls 10-13	3	12.7	4.6	10.7	2.8	14.3	4.9	11.7	2.1

Figure 7.1. illustrates the results of the numbers problems subtest of the SSAIS-R

Fig 7.1

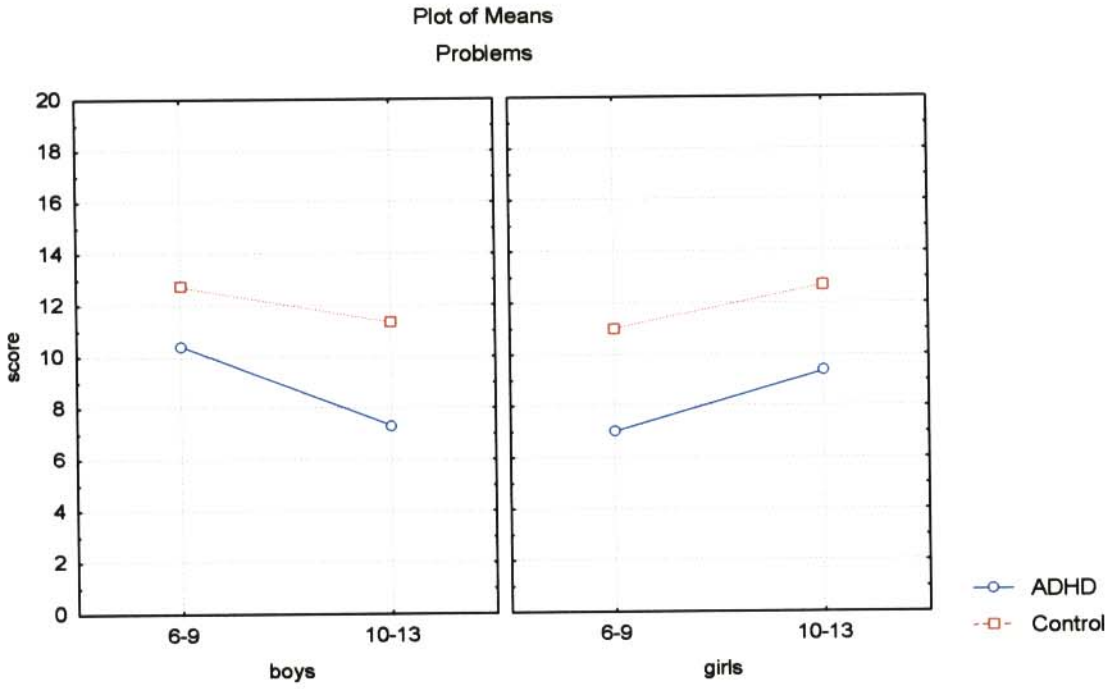


Figure 7.2. is an illustration of the results of the Story Memory subtest of the SSAIS-R

Fig. 7.2

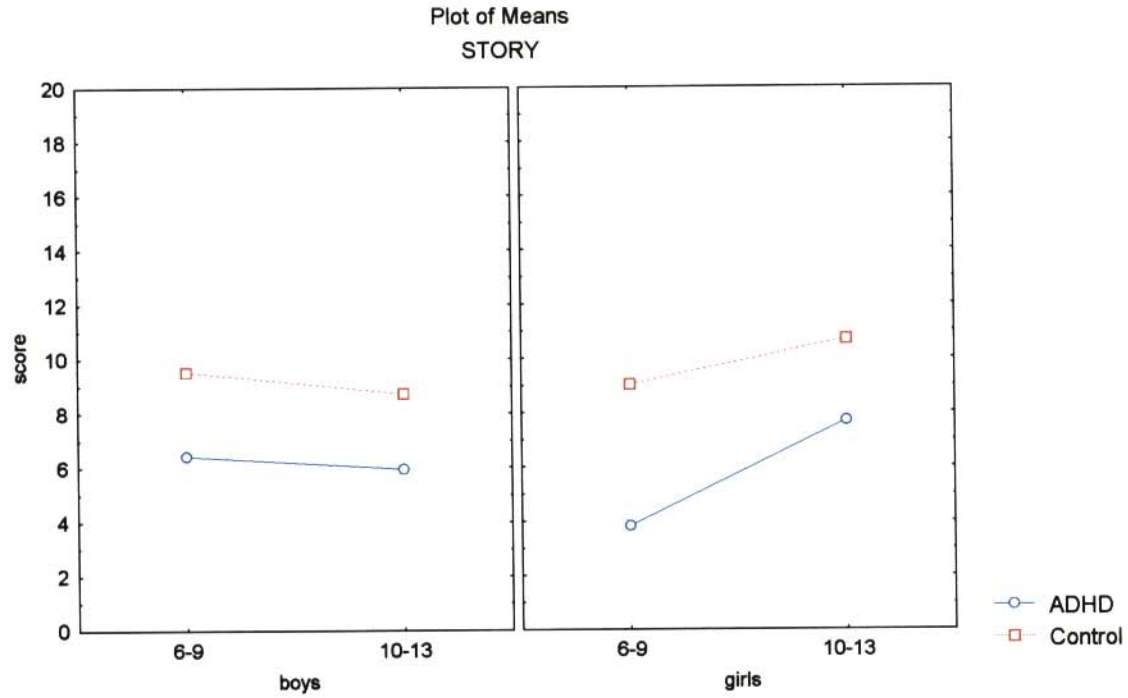
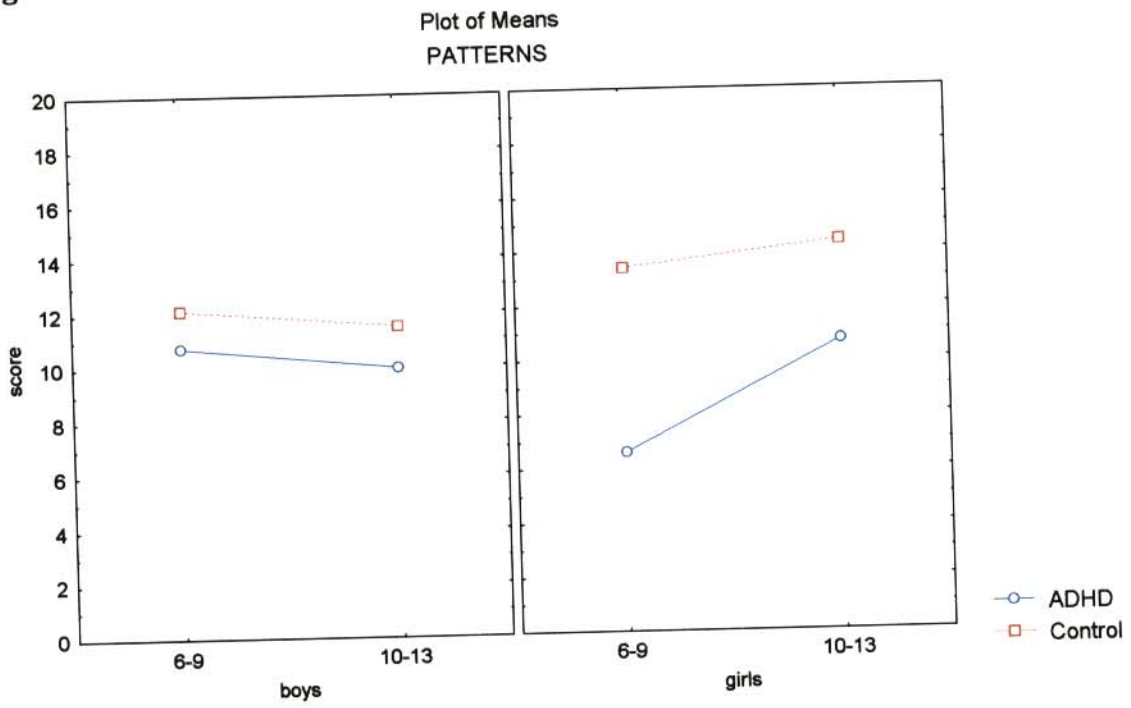


Figure 7.3 provides an illustration of the Pattern Completion subtest of the SSAIS-R

Fig. 7.3



The results of the Block Designs subtest are shown in Figure 7.4.

Fig 7.4

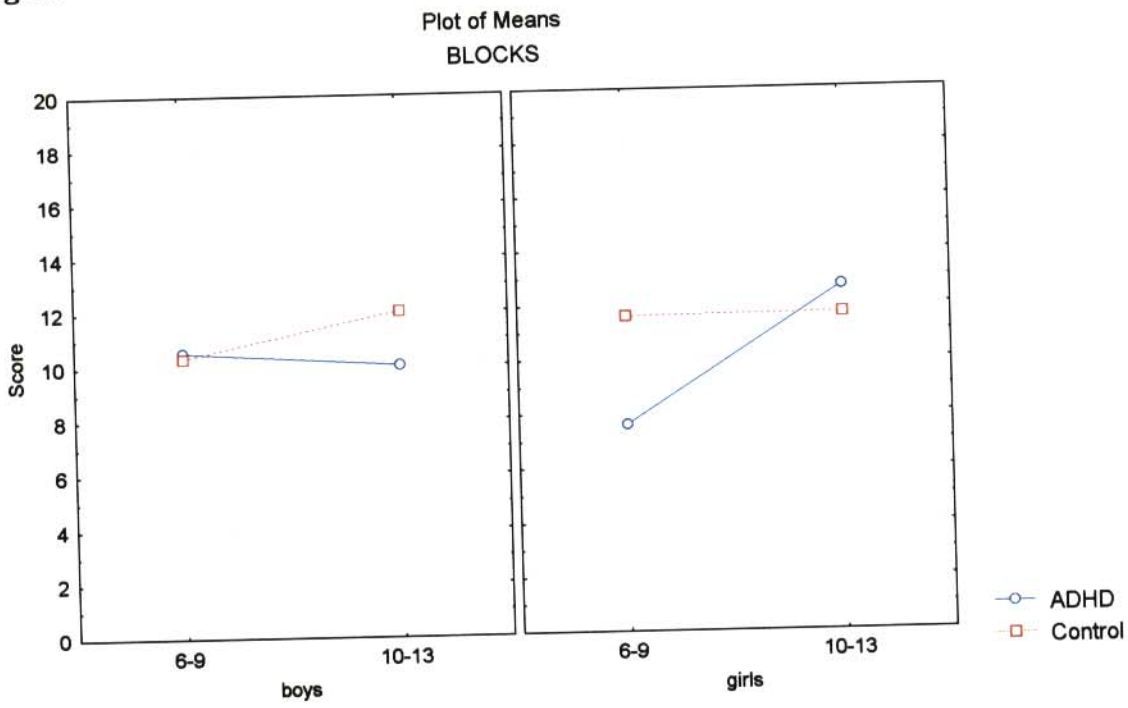


Table 7.2 provides the descriptive statistics for the subtests of the SSAIS-R according to the subtypes.

Table 7.2 SSAIS-R descriptive statistics (subtypes)

		Number Problems		Story memory		Patterns		Blocks	
	N	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Combined	9	7.9	2.5	5.2	1.8	8.4	3.6	9.3	1.8
H/I	13	9.5	2.5	6.5	2.3	10.8	3.2	10.6	3.9
Inattentive	9	7.4	3.	5.9	2.7	8.9	2.5	10.1	3.8
Normal	31	11.9	2.8	9.1	2.4	12.4	3.4	11.3	2.6

Figure 7.5 is a graphic illustration of the results of the Number Problems subtest

Fig.7.5

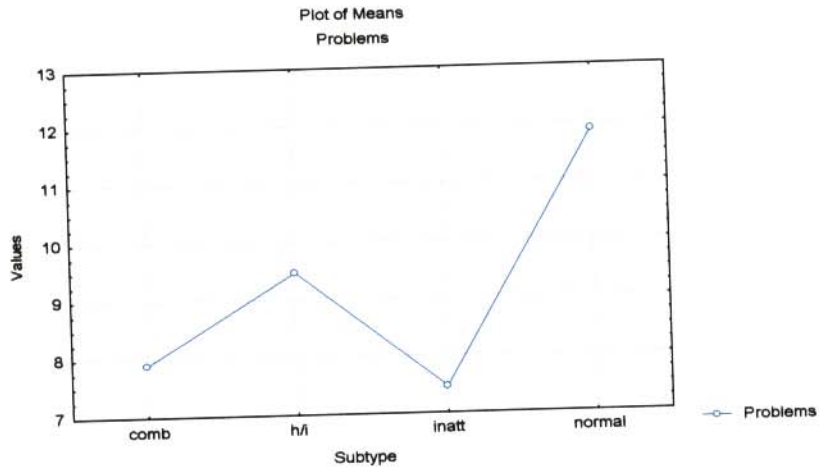
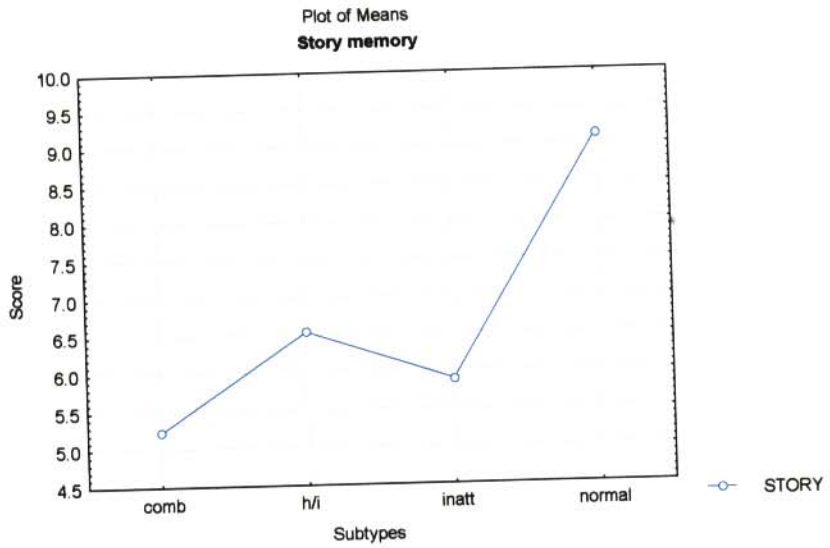


Fig. 7.6 illustrates the results of the Story Memory subtest

Fig 7.6



The results of the Pattern Completion subtest is shown in Figure 7.7.

Fig 7.7

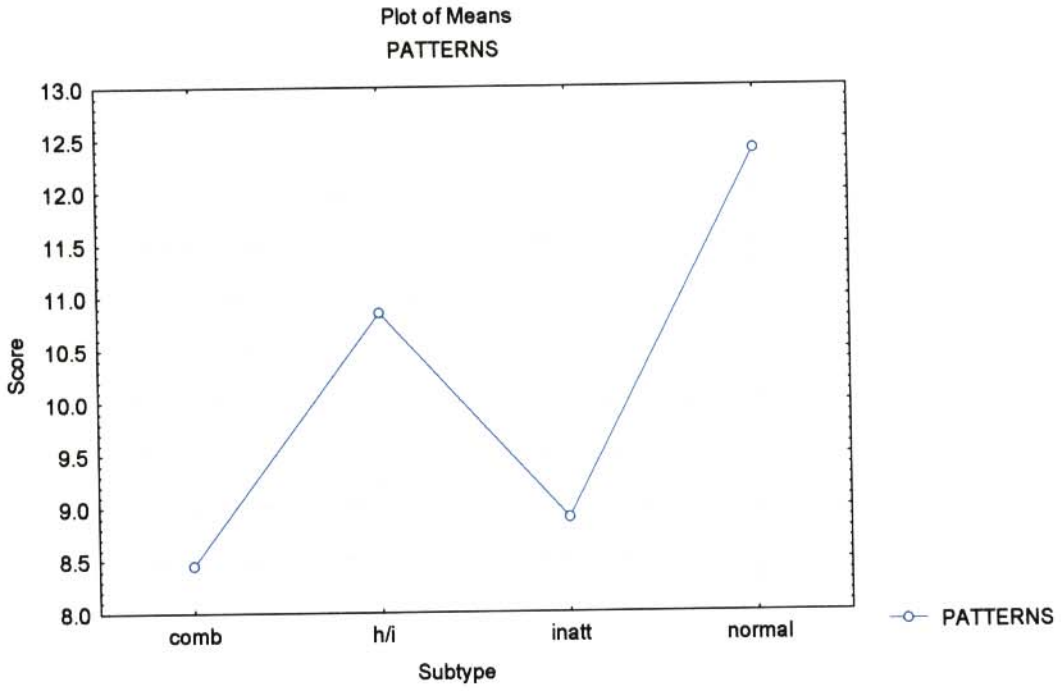
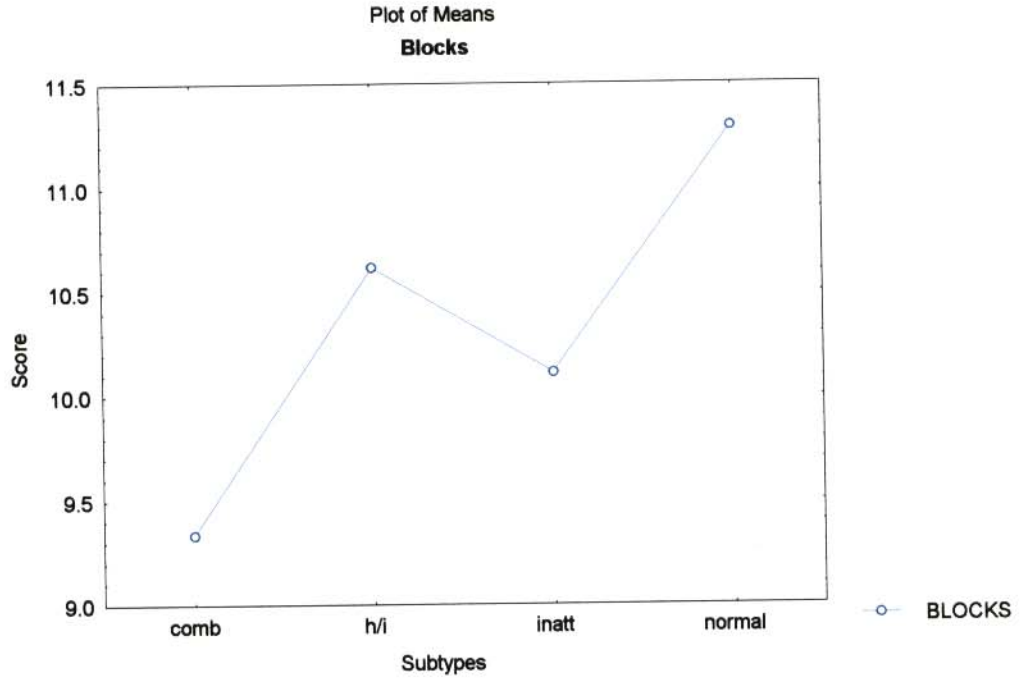


Figure 7.8 illustrates the results of the Block Design subtest

Fig 7.8



WCST and TOL

The descriptive statistics of the results of the WCST and TOL according to age groups is illustrated by Table 7.3.

Table 7.3 WCST and TOL descriptive statistics (age groups)

		WCST						TOL	
	N	Perseverative Errors		Non-perseverative errors		Categories achieved			
		Mean	SD	Mean	SD	Mean	SD	Mean	SD
ADHD Boys 6-9	10	9.0	6.2	33.9	17.6	6.0	2.7	24.2	5.0
Control Boys 6-9	10	7.2	3.6	27.0	16.8	6.2	2.2	28.6	3.8
ADHD Boys 10-13	13	13.0	8.8	26.9	11.9	5.2	2.0	26.9	4.7
Control Boys 10-13	13	6.3	4.9	25.5	10.3	6.38	1.90	31.2	2.8
ADHD Girls 6-9	5	11.2	9.2	32.0	12.1	5.6	1.1	24.6	2.3
Control Girls 6-9	5	3.0	1.2	19.4	7.5	7.8	1.1	30.6	1.5
ADHD Girls 10-13	3	9.3	6.1	21.0	11.5	7.7	2.5	25.7	4.7
Control Girls 10-13	3	20.3	22.3	24.7	12.0	5.0	3.0	22.7	2.5

Figure 7.9 illustrates the perseverative error scores on the WCST

Fig 7.9

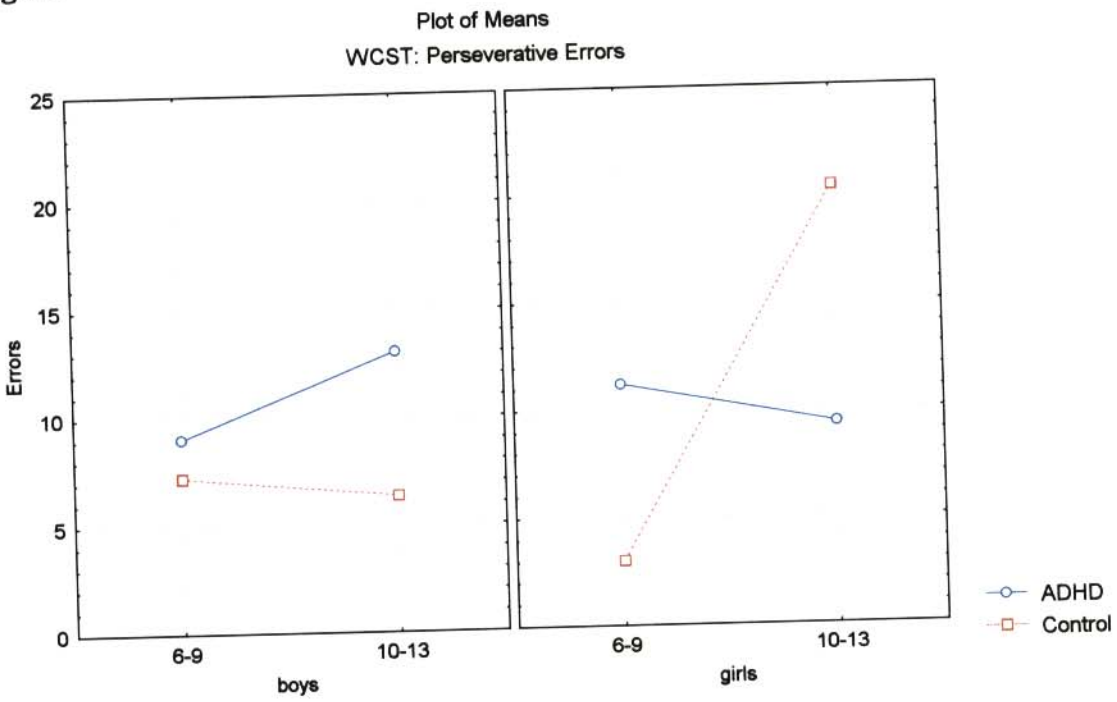
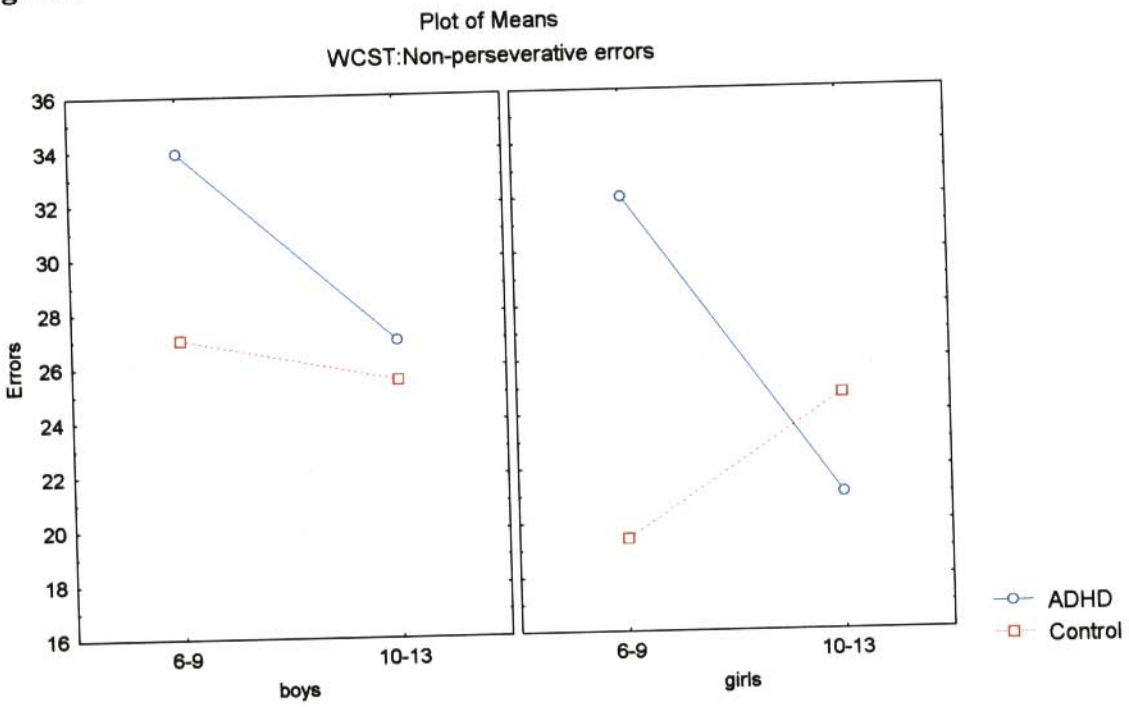


Figure 7.10 is an illustration of the non-perseverative errors on the WSCT

Fig. 7.10



The categories achieved on the WCST is shown by Figure 7.11

Fig. 7.11

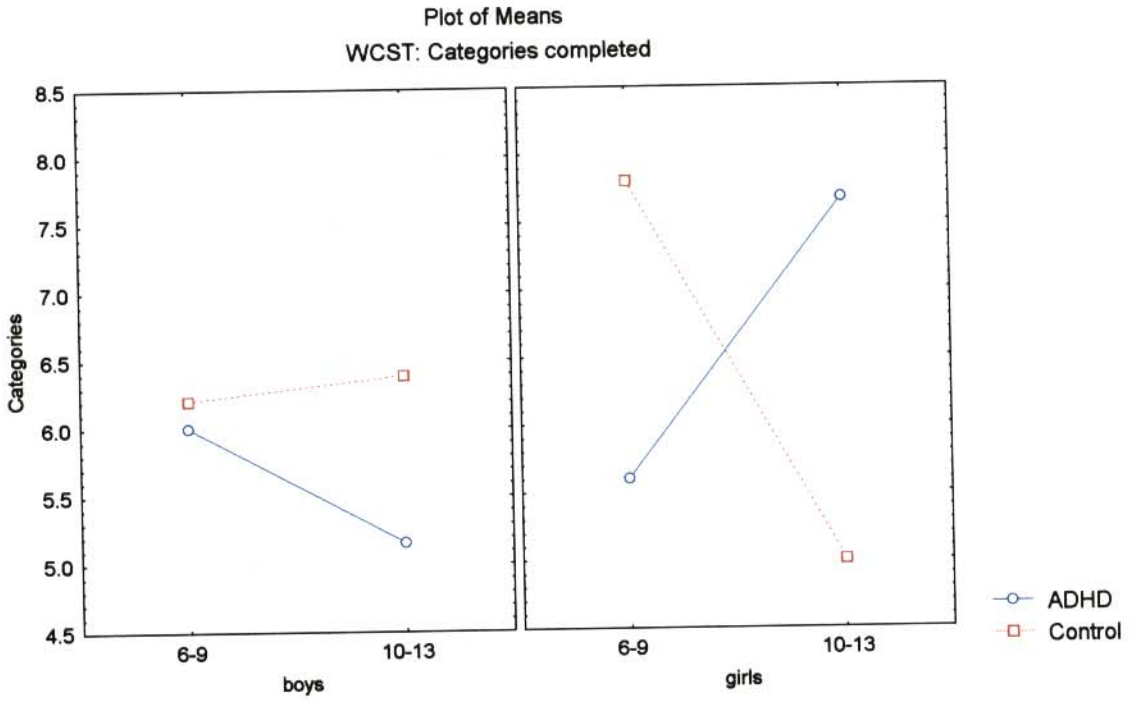


Figure 7.12 shows the scores on the TOL

Fig. 7.12

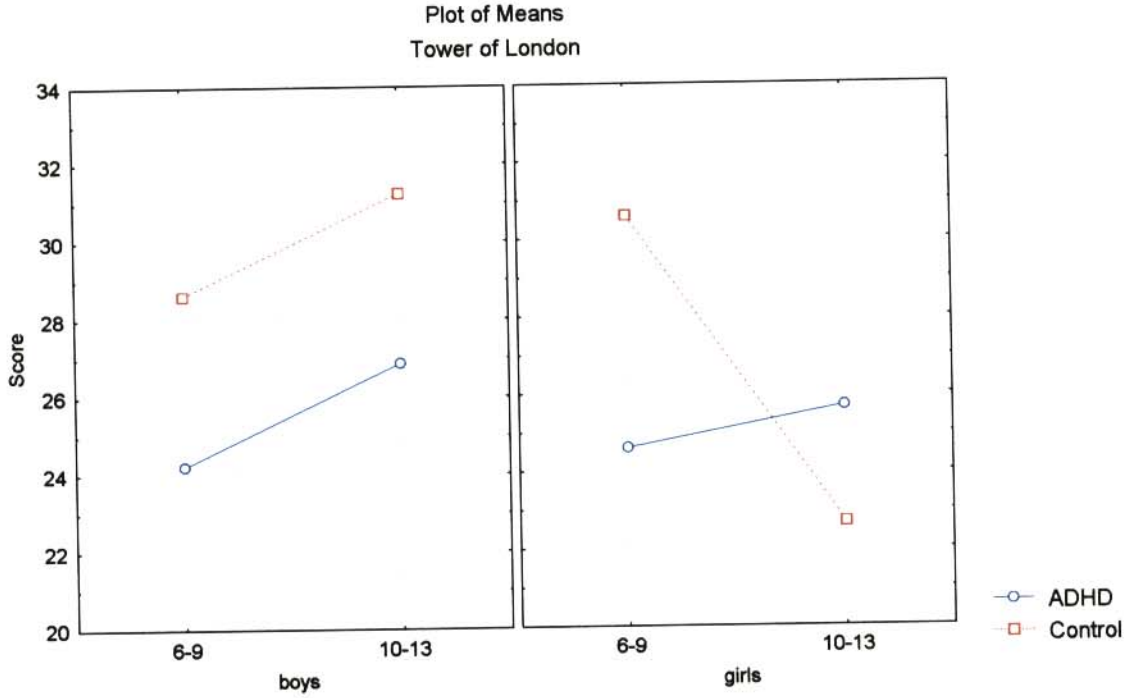


Table 7.4 illustrates the descriptive statistics for the WCST and TOL according to the subtypes.

Table 7.4 Descriptive statistics for the WCST & TOL: subtypes

		WCST						TOL	
		Perseverative errors		Non-perseverative errors		Categories achieved			
	N	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Combined	9	11.6	7.1	39.4	18.4	4.9	2.5	25.1	5.0
Hyperactive /Impulsive	13	9.2	6.7	24.7	8.7	6.3	1.8	25.8	4.8
Inattentive	9	13.6	9.4	28.7	13.0	5.4	2.6	26.7	4.2
Normal	31	7.3	8.3	24.2	11.6	6.5	1.9	29.1	4.0

Figure 7.13 provides an illustration of the Perseverative Errors on the WCST

Fig. 7.13

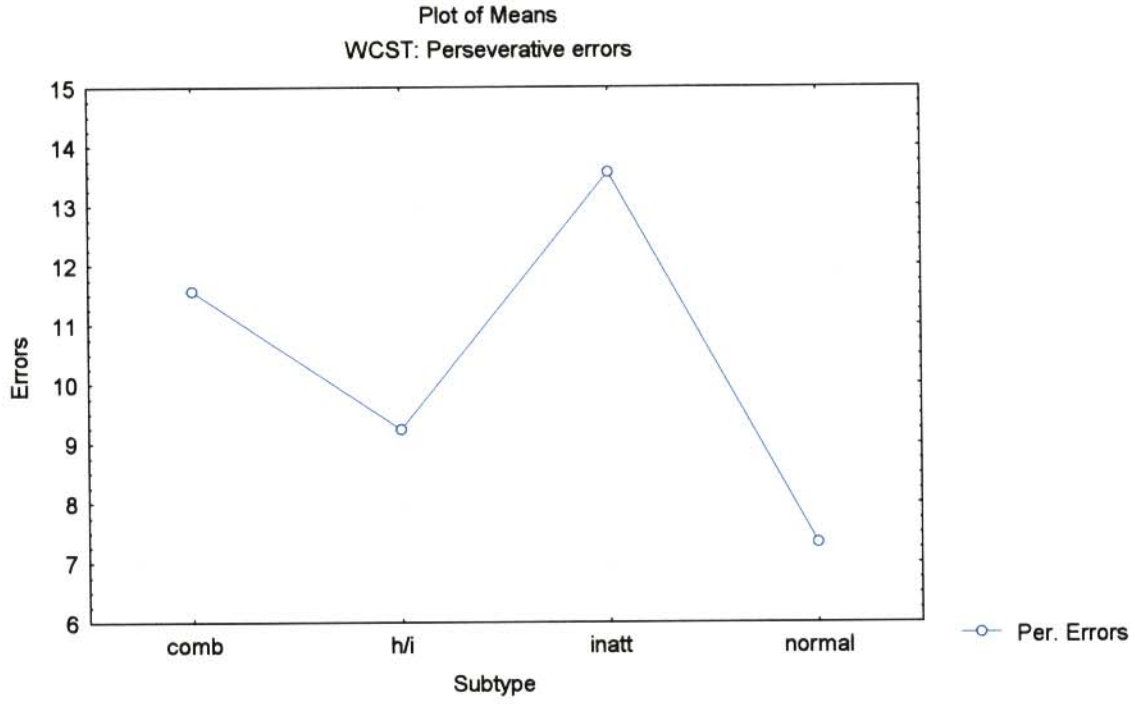


Figure 7.14 illustrates the Non-Perserverative Errors on the WCST

Fig. 7.14

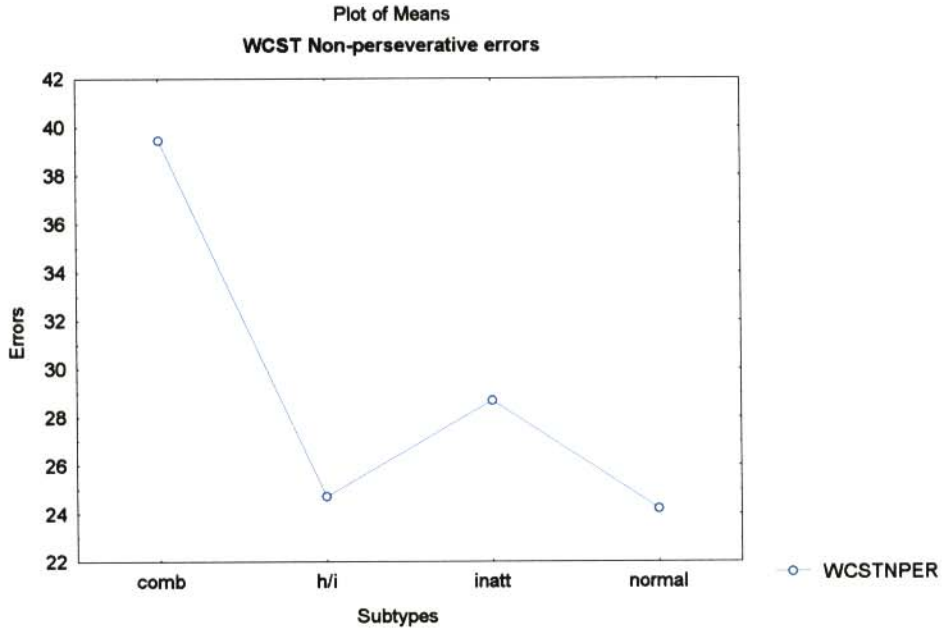


Figure 7.15 illustrates the categories achieved on the WCST

Fig 7.15

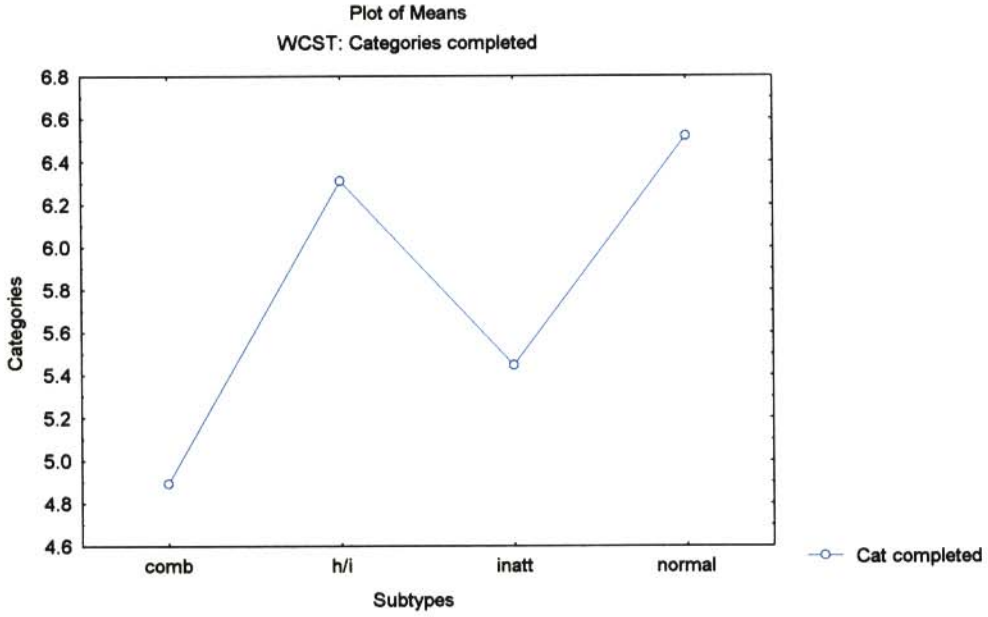
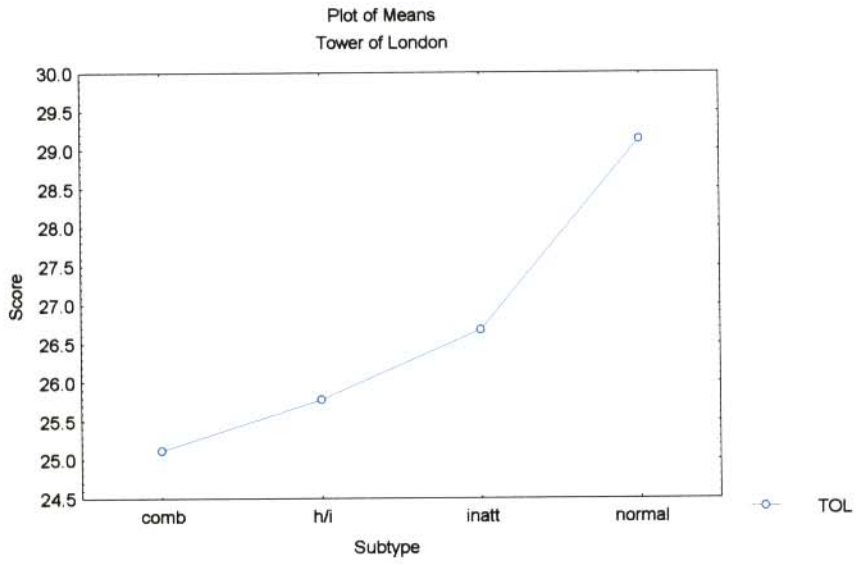


Figure 7.16 illustrates the scores on the TOL

Fig. 7.16



STROOP

Table 7.5. represents the descriptive statistics for the three phases of the Stroop test.

Table 7.5. Descriptive statistics (age groups)

		Phase 1		Phase 2		Phase 3	
	N	Mean	SD	Mean	SD	Mean	SD
ADHD Boys 6-9	10	0.80	0.78	2.90	2.28	11.10	7.99
Control Boys 6-9	10	0.40	0.69	1.70	1.70	7.9	7.46
ADHD Boys 10-13	13	0.69	0.94	3.84	3.33	12.23	4.39
Control Boys 10-13	13	0.15	0.37	0.92	1.25	3.92	2.62
ADHD Girls 6-9	5	1.80	1.64	3.00	2.00	15.00	7.42
Control Girls 6-9	5	0.20	0.45	0.40	0.89	4.00	2.82
ADHD Girls 10-13	3	1.00	1.73	1.66	0.57	8.33	4.04
Control Girls 10-13	3	0.33	0.58	1.00	1.00	8.33	5.50

The number of errors in Phase 1 of the Stroop is shown in figure 7.17.

Fig. 7.17

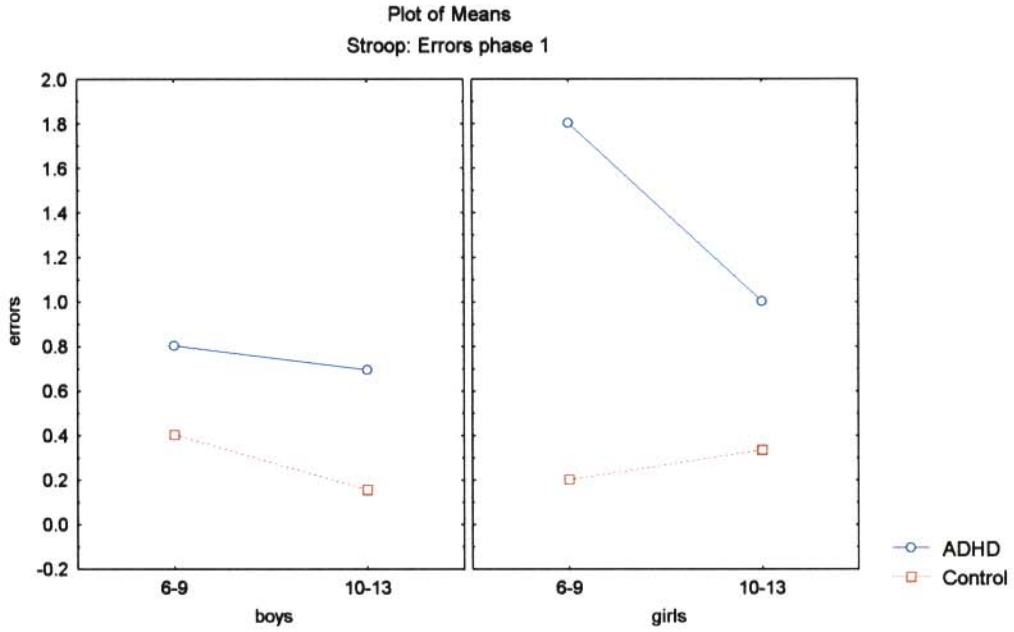


Figure 7.18 illustrates the number of errors on Phase 2 of the Stroop test

Fig 7.18

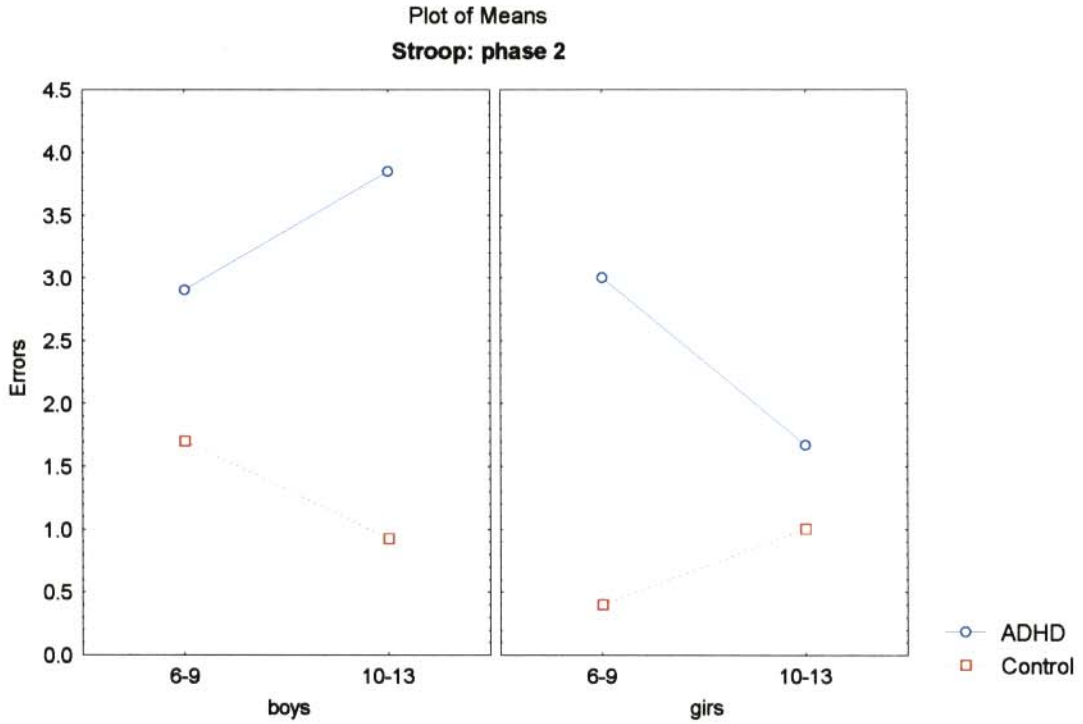


Figure 7.19 illustrates the errors made in phase 3 of the Stroop

Fig. 7.19

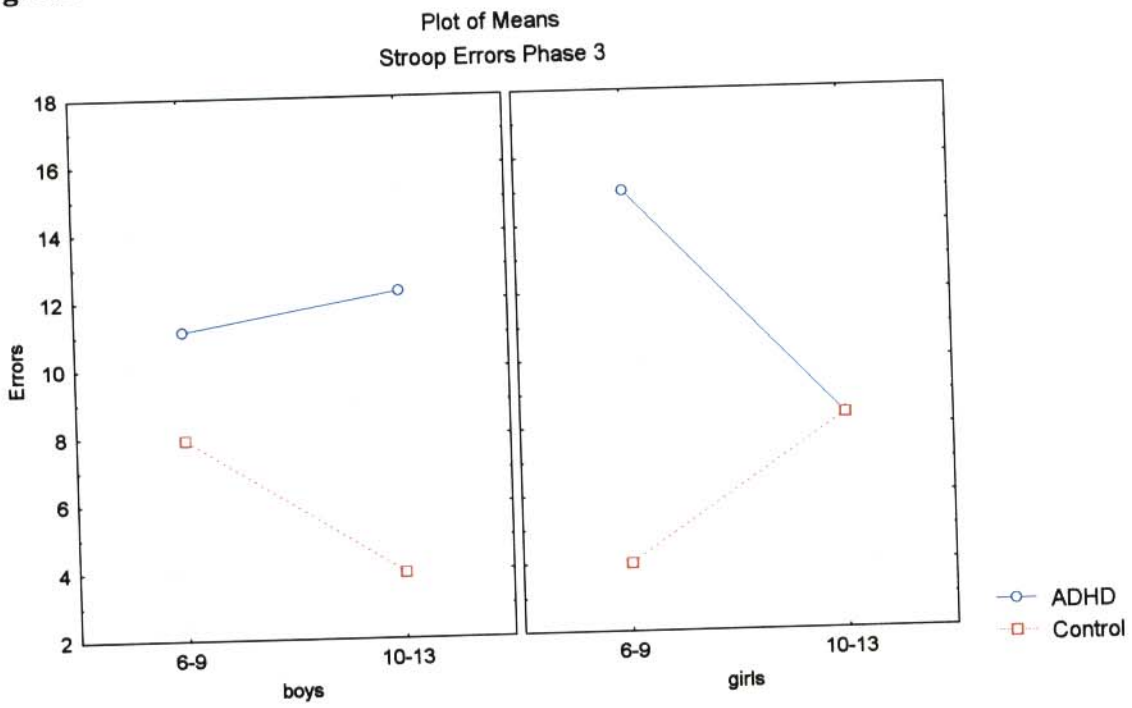


Table 7.6. provides the descriptive statistics for the three phases of the Stroop test according to subtypes

Table 7.6 Stroop test descriptive statistics: subtypes

		Phase 1		Phase 2		Phase 3	
	N	Mean	SD	Mean	SD	Mean	SD
Combined	9	1.77	1.30	3.00	2.39	13.33	8.54
Hyperactive/Impulsive	13	0.69	0.94	2.69	1.84	11.76	6.09
Inattentive	9	0.55	0.72	4.22	3.70	9.44	3.46
Normal	31	0.22	0.49	1.06	1.34	6.03	5.48

Figure 7.20 shows the number of errors made in Phase 1 of the Stroop

Fig. 7.20

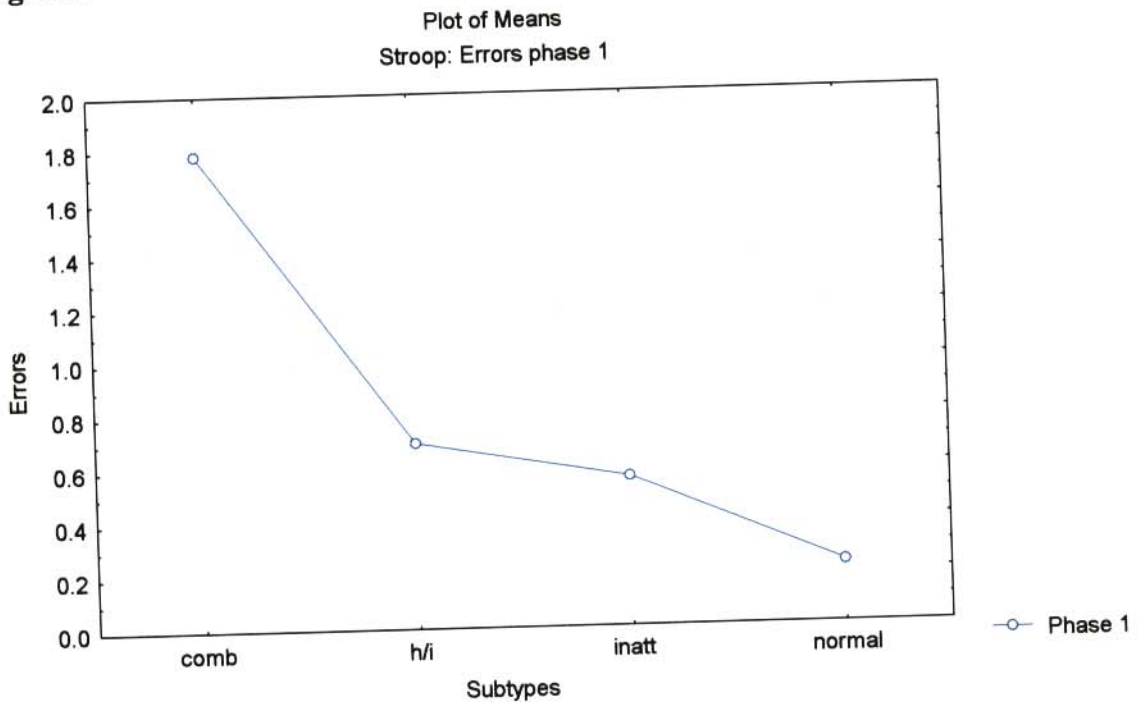
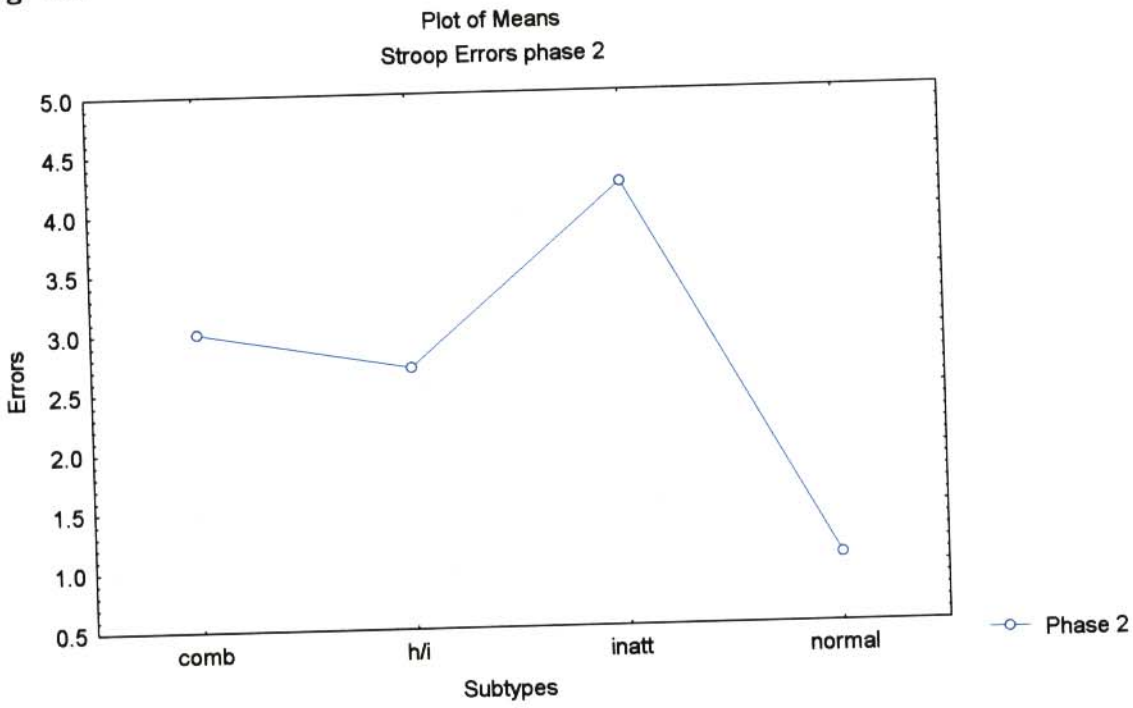


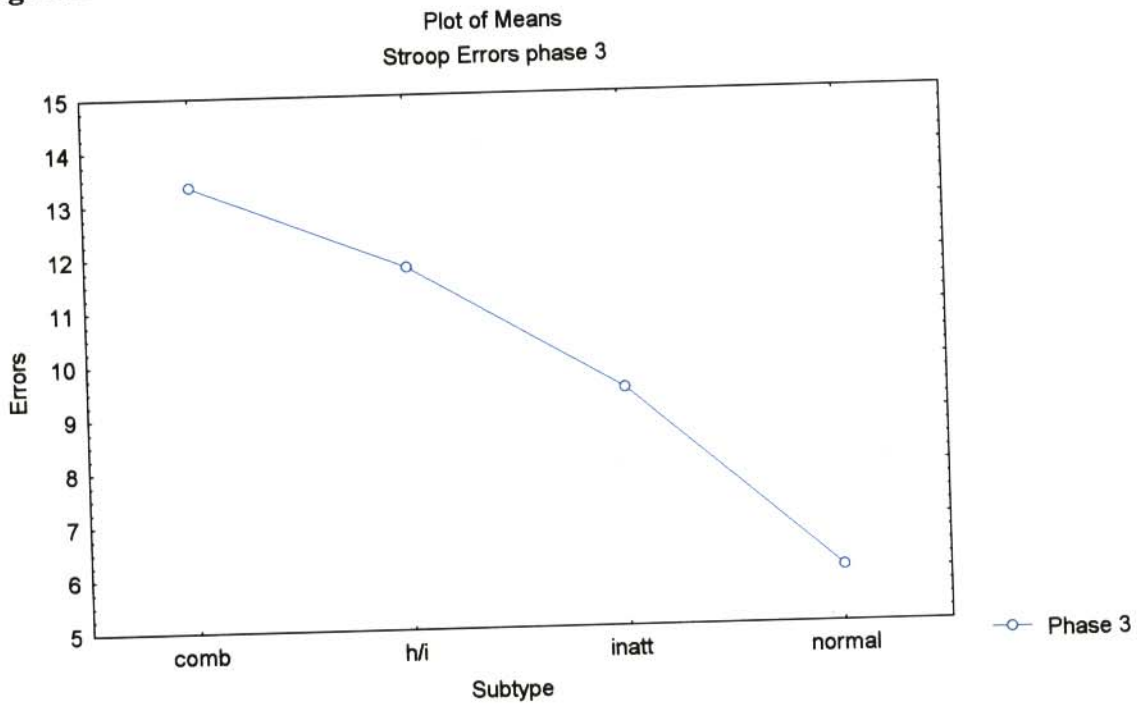
Figure 7.21 illustrates the number of errors made in Phase 2 of the Stroop

Fig. 7.21



The number of errors made in Phase 3 of the Stroop are shown in Figure 7.22.

Fig. 7.22



7.3. Statistical analysis

7.3.1 SSAIS-R

Analysis of variance

Table 7.7 illustrates the analysis of variance for the SSAIS-R subtests

Table 7.7 Analysis of variance: SSAIS-R

	Marked effects are significant at p<0.05					
	Age groups			**Subtype**		
Variable	**df**	**F**	***p***	**df**	**F**	***P***
Number problems score	7	5, 19	0.000*	3	9,18	0.000*
Story memory score	7	5, 12	0.000*	3	9, 76	0.000*
Pattern completion score	7	2, 47	0.028*	3	4, 91	0.004*
Block Designs score	7	1, 62	0.147	3	1, 13	0.341

Post-Hoc (Duncan)

The results of the post-hoc test performed on the subtests of the SSAIS-R is illustrated in Table 7.8.

No post-hoc analysis was performed on the Blocks subtest as there was no statistically significant analysis of variance.

Table 7.8 Post-hoc (Duncan) test: SSAIS-R.

	Numbers	Story memory	Patterns
Group	***p***	***p***	***p***
Boys 6-9	0.208	0.043*	0.509
Boys 10-13	0.027*	0.064	0.485
Girls 6-9	0.027*	0.001*	0.003*
Girls 10-13	0.067	0.050*	0.109
Combined	0.001*	0.000*	0.007*
H/I	0.036*	0.009*	0.253
Inattentive	0.000*	0.002*	0.015*

*$p \leq 0,05$

7.3.2.WCST and TOL

Analysis of variance

Table 7.9 gives an illustration of the analysis of variance for the WCST and TOL

Table 7.9 Analysis of variance: WCST & TOL

	Marked effects are significant at p<0.05					
	Age groups			Subtype		
Variable	**df**	**F**	***p***	**df**	**F**	***P***
TOL score	7, 54	4.57	0.001*	3, 58	3.12	0.032*
WCST perseverative errors	7, 54	2.29	0.040*	3, 58	1.73	0.170
WCST non-perseverative Errors	7, 54	0.83	0.566	3, 58	3.71	0.016*
Categories shifted	7,54	1.26	0.285	3, 58	1.72	0.172

Post-Hoc (Duncan) tests

The Post-Hoc (Duncan) analyses performed on the WCST and TOL are illustrated by Table 7.10.

Table 7.10. Post-Hoc: WCST & TOL

	TOL	WCST Perseverative errors	WCST Non-Perseverative errors
Group	***p***	***p***	***p***
Boys 6-9	0.093	0.692	
Boys 10-13	0.087	0.202	
Girls 6-9	0.022*	0.117	
Girls 10-13	0.242	0.028*	
Combined	0.041*		0.007*
H/I	0.078		0.922
Inattentive	0.171		0.411

*$p \leq 0.05$

7.3.3.Stroop

Analysis of variance

Table 7.11 is an illustration of the analysis of variance for the three phases of the Stroop test

Table 7.11 Analysis of variance: Stroop

	Marked effects are significant at p<0.05					
	AGE			Subtype		
Variable	**df**	**F**	***p***	**df**	**F**	***P***
Stroop phase 1	7	2, 30	0.039*	3	9, 11	0.000*
Stroop phase 2	7	2, 74	0.016*	3	6, 60	0.000*
Stroop phase 3	7	3, 69	0.002*	3	5, 15	0.003*

Post-Hoc (Duncan)

The post-hoc test results of the phases of the Stroop test are given in table 7.12.

Table 7.12 Post-hoc test: Stroop

	Stroop phase 1	Stroop Phase 2	Stroop Phase 3
Group	***p***	***p***	***p***
Boys 6-9	0.472	0.351	0.396
Boys 10-13	0.007*	0.050*	0.035*
Girls 6-9	0.008*	0.082	0.005*
Girls 10-13	0.260	0.603	1.000
Combined	0.000*	0.033*	0.006*
H/I	0.175	0.059	0.027*
Inattentive	0.308	0.001*	0.162

$p \leq 0,05$

7.4 Hypotheses testing

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

Hypotheses 1 must be partially rejected, as there is a difference between the clinical and control groups and between the genders, although not in all the tests.

Null hypothesis 1.1 must be partially rejected, as the clinical groups had lower scores on the Numbers subtest of the SSAIS-R, except for the younger boys and the older girls group, where the differences were not statistically significant.

Null hypothesis 1.2 must be partially rejected, as there was a difference in scores between the clinical and control groups, and the gender groups, on the Story Memory subtest of the SSAIS-R. The clinical groups performed worse than the control groups, except for the older boys group, where the difference was not statistically significant.

Null hypothesis 1.3 must be partially accepted, as the only statistically significant difference between the clinical and control groups on the Pattern Completion subtest of the SSAIS-R occurred in the younger girls group.

Null hypothesis 1.4 must be accepted, as the difference in scores between the clinical and control groups, and the gender groups, on the Block Design subtest of the SSAIS-R, was not statistically significant.

Null hypothesis 1.5 must be partially accepted, as the only instance where the clinical group made more perseverative errors on the WCST than the control group, and where the difference was statistically significant, was in the older girls group.

Null hypothesis 1.6 must be accepted, as the differences in scores between the clinical and control groups, in terms of non-perseverative errors on the WCST, was not statistically significant.

Null hypothesis 1.7 must be accepted, as there was no statistically significant difference in categories achieved on the WCST between the clinical and control groups.

Null hypotheses 1.8 must be partially accepted. The only significant difference between the clinical and control groups, in terms of scores on the TOL, was in the younger girls group.

Null Hypotheses 1.9 must be partially accepted, as the only instances where the control groups performed better than the clinical groups in Phase 1 of the Stroop test, were in the older boys and younger girls groups.

Null Hypothesis 1.10 must be partially accepted. Only in the older boys groups was there a statistically significant difference in performance between the clinical and control groups on Phase 2 of the Stroop test.

Null Hypothesis 1.11 must be partially rejected. The difference between the scores of the clinical and control groups of the younger boys and older girls in Phase 3 of the Stroop test was not statistically significant.

Hypothesis 2 must be partially rejected. The control group performed better than the subtype-groups except for certain tests.

Null Hypothesis 2.1 must be rejected, as the control group had the best performance of all the groups on the Numbers subtest of the SSAIS-R, and the differences in scores between groups was statistically significant.

Null Hypothesis 2.2 must be rejected. The control group had the highest score on the Story Memory subtest of the SSAIS-R.

Null Hypothesis 2.3 must be partially rejected. The subtype-groups had lower scores than the control group on the Pattern Completion subtest of the SSAIS-R, but the differences in the hyperactive/impulsive group was not statistically significant.

Null Hypothesis 2.4 must be accepted. There was a difference in scores between the subtype-groups and the control group on the Block Design subtest of the SSAIS-R. The control group had the best performance, but the difference in scores was not statistically significant.

Null Hypothesis 2.5 must be accepted, as the differences in number of perseverative errors on the WCST between the subtype groups was not statistically significant.

Null Hypothesis 2.6 must be partially accepted, as the control group made the least number of non-perseverative errors on the WCST, but the differences statistically significant only for the combined group.

Null Hypothesis 2.7 must be partially accepted, as the control group achieved the highest number of categories in the WCST, but the difference was only significant between the control and combined groups.

Null Hypothesis 2.8 must be partially accepted. The control group had the highest score on the TOL, but the difference was significant only between the control and combined groups.

Null Hypothesis 2.9 must be partially accepted. Although the subtype-groups made more errors than the control group on Phase 1 of the Stroop test, the difference in scores was only significant for the combined group.

Null Hypothesis 2.10 must be partially rejected. The control group had the least number of errors on Phase 2 of the Stroop test. The difference in scores between the hyperactive/impulsive group and the control group was not statistically significant.

Null Hypothesis 2.11 must be partially rejected. The control group had a lower error score than the subtype-groups, on Phase 3 of the Stroop test. The difference in scores between the inattentive group and the control group was not statistically significant.

A discussion of the results follows in the next chapter.

DISCUSSION OF RESULTS

8.1 Introduction

The aim of this study was to apply the neuropsychological theory of Sagvolden and co-workers (Sagvolden, 1999; Sagvolden & Sergeant, 1998a; Johansen, Aase, Meyer, & Sagvolden, 2002) to the neuropsychological testing of children screened for ADHD. A further aim was to assess the children on measures of cognitive impulsiveness, as a substitute for the general term of executive functions. The results of the test scores were analysed in relation to the different genders and age and subtype groups.

In accordance with expectations, in most of the tests the control groups performed better than the ADHD groups. This correlates well with other studies that found impairment in 'executive functions' in the neuropsychological testing of children with ADHD (Bayliss & Roodenrys, 2000; Castellanos, 1997; Reeve et al., 2001).

The exceptions to this were the following tests:

a) The blocks-subtest of the SSAIS-R, where the ADHD boys in the younger age group performed better than the control group. On the same subtest, the older ADHD girls performed better than the control group.

b) In phase 3 of the Stroop test, the older girls in both the clinical and control groups had an equal performance.

c) On the Tower of London, the ADHD girls in the older age group performed better than the control group.

d) This was also observed on the Wisconsin Card Sorting test, in terms of perseverative errors, non-perseverative errors and categories completed.

The data results can be briefly summarised as follows:

1. Girls in the 6-9 year age group appear to be severely affected by ADHD.

2. Older boys appear to be more severely affected by the disorder than their younger counterparts.

3. A diagnosis of the combined form of ADHD predisposes an individual to be more severely affected by the cognitive deficits that are typical of ADHD

4. The Stroop, Tower of London and Wisconsin Card Sorting Tests appear to be good measures of cognitive impulsiveness.

5. The numbers, story memory and pattern completion subtests of the SSAIS-R appear to be good measures of cognitive impulsiveness.

6. The blocks-subtest of the SSAIS-R does not appear to be a good test of cognitive impulsiveness.

8.2. Discussion of results

Although it was not a primary aim of this study to analyse the differences in neuropsychological performance between genders, when the data was analysed a pattern emerged that was so striking that it merits further examination.

In four tests the older girls' results rendered a different result to the one that was expected, with the clinical groups performing as well as or *better* than the control group. As the number of participants in these groups was small, it is difficult to generalise from these findings. For example, one child in the control group that was impaired in some way on the day of testing could have influenced the results to give such a distribution.

Nevertheless, as this particular distribution was found among a number of tests, it does deserve further scrutiny. It could mean that girls' impairment diminishes as they get older, to such an extent that they are not functionally impaired on the functions measured by these particular tests. Some theorists have indeed argued that although certain symptoms remain as children with ADHD grow older, the symptom severity seems to decrease with age, even if the condition is left untreated (Castellanos, 1997).

A different distribution was that the younger girls with ADHD were the group with the worst performance on the number problems, story memory, pattern completion, blocks, Stroop phase 2, and perseverative errors, and non-persevaritive errors on the Wisconsin tests. Not only did they perform worse than the older girls with ADHD, they also performed much worse than the boys with ADHD in the same age group. Once again it is difficult to generalise from these findings due to the small sample size, but these results also deserve further scrutiny.

It is also interesting that as discussed earlier, the older girls in the clinical group performed better than their younger counterparts. Even taking into consideration the small number of participants, it could be important to do further studies on whether ADHD-related impairments do in fact remit in older girls, or are expressed differently as they get older. This is especially important as longitudinal studies on the effects of ADHD on girls are poorly researched (Heptinstall & Taylor, 2002).

The findings of this study are in contrast to some others, which found that girls with ADHD did not perform less well on tests of executive function than controls (Seidman, Biederman, Faraone, Weber, & Ouellette, 1997).

As ADHD is more prevalent in boys, at least according to current diagnostic and referral criteria, research efforts have traditionally been focused on boys (Reid, Riccio, Kessler, DuPaul, Power, Anastoupoulos, Rogers-Adkinson & Noll, 2000; Robison, Skaer, Sclar, & Galin, 2002). Thus there is a dearth of data on ADHD in girls. There are some studies that have suggested that girls tended to be more severely affected, though in different ways than boys. Whereas boys tend to have greater problems with externalising behaviour, girls are more severely affected in terms of cognitive impairment. Unfortunately, most of the research results on ADHD in girls are contradictory or insufficient (Heptinstall & Taylor, 2002).

In a reversal of the pattern observed with the girls, the older boys tended to perform *worse* than the younger boys in the clinical group. This observation was true in all of the tests performed. Not only was there a consistent pattern within the clinical groups, the comparison between the control groups and clinical groups shows that in all the tests the clinical group performed worse than the control group, a pattern that was expected. The only test where this was not the case was in the Blocks subtest. The test did not show statistically significant differences between the other groups, and thus appears not to be a useful measure of cognitive impulsiveness in children with ADHD.

The difference in scores between boys and girls may suggest that in contrast to girls, in boys the symptoms of ADHD does not readily remit, or that they remit at age (eg later in adolescence). It is well known that boys develop slower than girls, both cognitively and physically (Louw, 1996). This would be in accordance with the theoretical perspective of ADHD as a neuro-developmental disorder. This study also focussed on the functions of

the frontal lobes, which are not fully developed until adulthood. This could be another possible explanation for the differences in performance between the genders.

When the data was analysed according to subtypes, a distribution developed that was observed in almost all the tests. The group with combined hyperactivity/impulsiveness and inattention consistently had the lowest scores or the highest number of errors. The only tests where this was not the case were the number problems subtest, phase two of the Stroop test, and the perseverative errors on the Wisconsin card sorting test, where interestingly, the inattentive group had the worse performance.

This would seem to indicate that the ability to pay attention (or not be cognitively impulsive) would be more important in these tests than the influence of hyperactivity. It would thus appear as if children with the combined form of the disorder are the most severely impaired.

This has also been found in other studies (Faraone, Biederman, Weber, & Russell, 1998).

The hyperactive/impulsive group was the one section of the clinical population that consistently had the best performance. The only tests that were the exception to this rule were the Tower of London, and phase one and three of the Stroop. On these tests impulsiveness may be more detrimental on the score than on the other tests. Thus the Stroop and the TOL appear to be good measures of cognitive impulsiveness, at least in terms of differentiating between subtypes. This finding could also indicate that hyperactivity and impulsiveness may be less impairing than inattention, when applied to these specific tests. This is in accordance with other studies which found the children with the combined diagnosis to be more severely impaired than the predominantly hyperactive/impulsive and inattentive groups (Schmitz et al., 2002a).

A weakness of many studies on neuropsycholgical performance in ADHD is that the different subtypes are clustered together. This makes it difficult to generalise results, especially if the discussion about the aetiological difference of the Inattentive type is taken into consideration. The present study is one attempt to overcome this weakness and analyse the data in terms of the subtypes.

The differences in the scores between the clinical and control groups indicate that children with ADHD tend to be more cognitively impulsive than children without ADHD. This affirms the neuropsychological theory underlying this study, which states that a dysfunctioning meso-cortical dopamine branch leads to cognitive impulsiveness.

8.3 Limitations of the study

One of the limitations of this study was the small number of participants, particularly among the girls. This limitation is a characteristic of many studies of ADHD as girls are underrepresented in the disorder and thus it is difficult to find large numbers of them for clinical population groups.

The fact that the children in the clinical group were only screened for ADHD, rather than diagnosed, poses another possible limitation. Although the DBD used for screening purposes are an important part in the diagnostic process, (American Academy of Pediatrics, 2000) they cannot be used separate of other procedures to diagnose ADHD.

Because a number of tests were administrated, there is an increased chance that a Type 1 error occurred. However, the differences between the control and clinical groups that were significant, tended to follow a similar trend.

8.4. Possibilities for further research

Due to the differences observed between the genders (and between ages in the same gender groups), it may be useful to investigate the differences in cognitive deficits between the genders. This may be a particularly important research area, as up to now it has been believed that girls are underrepresented in the disorder, but an epidemiological study has shown that ADHD is increasing in girls (Robison et al., 2002). If this is true, there is a case to be made for research studying the aetiology and treatment of ADHD in terms of gender.

Gender differences in childhood psychiatric disorders has indicated that boys are more likely to be referred for treatment than girls. However, when girls are referred, they tend to have a more severe disorder (Nolan, Gadow, & Sprafkin, 1999). This suggests that there may be a population of girls with less severe, but still debilitating symptoms, who are overlooked and thus not treated.

The data suggests that though they may be more severely affected than their male counterparts, the cognitive dysfunctions may be in different domains (inattention in the girls versus impulsiveness in the males). If girls are being overlooked or misdiagnosed, it follows that they are not being treated. Thus identification and diagnoses of girls with ADHD may be enhanced by research efforts that focus specifically on them.

It has been stated that the inattention aspect of ADHD may have a separate aetiology, and involve different neurological systems, than the hyperactive and impulsive symptoms (Barkley, 1997b; Johansen et al., 2002). This is reflected in the DSM-IV nosology, with its emphasis on teasing apart inattention from hyperactivity/impulsiveness.

The difficulty with the term executive functions (of which the ability to pay attention is a part) has already been explained. As such a possible further research area is the study of

cognitive impulsiveness specifically in hyperactive/impulsive groups. In other words children with a predominantly inattentive type of ADHD will be excluded from this study. This will build on the present study and provide a clearer theoretical basis from which to study cognitive deficits in general, and cognitive impulsiveness specifically. It will provide another opportunity to test the neuropsychological theory the present study is based on, but without the confounding effects of inattention subtype.

8.5. Concluding remarks

The neurobiological theories of ADHD provide a sound scientific base from which to investigate the symptom domains of ADHD. This is especially true in the domain of cognitive deficits, which often are the most debilitating effects of the disorder. The advances made in the neurosciences (for example the human genome project) has broadened the knowledge base about the functioning of the brain in no small way. These advances continue at an ever-expanding rate, thereby heightening the need for sound theories to explain the neurobiological data.

Although the process of relating neurobiological facts to neuropsychological theories of cognitive functioning can at times be daunting, the results it yields in terms development of theories and identifying new research areas are undeniable.

The theory of cognitive impulsiveness forms part of one such effort of integrating theory and facts. As the present study has shown, it provides an opportunity to investigate neuropsychological functions in a manner that is specific and theoretically grounded.

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Appendix A: Letter to school principals

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PSYCHOLOGY

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Die Hoof

Laerskool .....

Geagte Meneer/Mevtou:

Attention Deficit / Hyperactivity Disorder (AD/HD) is 'n afwyking wat tussen 2% en 5% van laerskool kinders affekteer. Die kind sukkel om aandag te skenk, om sy of haar aktiwiteite te beheer en is impulsief. Dit is egter baie meer as net 'n kwessie van aandagtekort en ooraktiwiteit. Die kind sukkel om sy of haar skoolwerk baas te raak en kom moontlik nie goed oor die weg met ander kinders nie. Hulle is ook nie in staat om take te voltooi sonder toesig nie en kan ontwrigting in die familie veroorsaak.

Die probleme kan veroorsaak dat die kind nie kan aanpas by die normale vereistes van die alledaagse lewe nie. Dit word nie ontgroei nie en kan in die toekoms probleme veroorsaak met roekelose gedrag, 'n moontlike risiko vir wetsoortredings en dwelmmisbruik.

Die oorsake van AD/HD is onbekend, maar navorsing dui daarop dat dit oorerflik kan wees. Besoedelende stowwe en ongebalanseerde voeding kan ook 'n rol speel. Dit word nie veroorsaak deur 'n onvermoë om die kind te dissiplineer of beheer nie.

Dit is baie belangrik dat hierdie kinders op 'n vroeë ouderdom gediagnoseer en behandel word, om sodoende probleme by die huis en skool te voorkom en die risiko vir toekomstige probleemgedrag uit te skakel.

Die diagnose van AD/HD was nog altyd problematies. Tot op hede was die enigste instrumente vir die diagnose van AD/HD vraelyste wat voltooi is deur ouers en onderwysers. Hierdie vraelyste is egter baie subjektief en die betekenis van sekere konsepte kan verskil tussen kulture en taalgroepe. Daarom beskou ons hierdie vraelyste as onvoldoende vir diagnose.

Die departement van Fisiologie, Universiteit van Oslo, Noorweë, het 'n kultuurvrye toets ontwikkel, sensitief vir die simptome van AD/HD. Ons wil graag hierdie toets op die veskillende kultuur en taalgroepe in die Noordelike Provinsie toepas. As hierdie instrument kinders met AD/HD suksesvol kan identifiseer, kan dit aansienlike probleme vir ouers en kinders voorkom. Terselftertyd probeer ons ook die rol van voeding en industriële besoedelende stowwe bepaal.

Hierdie is 'n goedgekeurde departementele projek van die Universiteit van die Noorde, in samewerking met die Universiteit van Oslo, Noorweë. Toestemming is verkry van die Superintendent van Onderwys vir die Noordelike Provinsie.

Metode:
Die navorsingspan sal die deelnemende skool besoek en sal die kinders met ADHD identifiseer. Die Disruptive Behaviours Disorder Rating Scale (DBD) sal gebruik word. Hierdie skaal (wat gestandardiseer is vir al die populasies in die Limpopo Provinsie, sal deur die kinders se klasonderwysers ingevul word. Die kinders wat op die basis van die DBD gekies word sal dan verder getoets word. Die volgende toetse sal geadministreer word:

- Biografiese data vraelys
- Senior Suid-Afrikaanse Intelligensieskaal (SSAIS-R) (4 subtoetse)
- Wisconsin Card Sorting Test (WCST)
- Tower of London (TOL)
- Stroop word/colour toets

Die data sal alleenlik vir statistiese analise gebruik word en onder geen omstandighede sal die identiteit van die kind of die skool bekend gemaak word nie.

U goedkeuring vir hierdie baie belangrike studie sal bydra tot die ontwikkeling van 'n geldige diagnostiese metode, wat dit sal moontlik maak om kinders te idenitfiseer wat 'n risikogroep vorm vir opvoedkundige, sosiale en emosionele probleme.

Die uwe

Prof. Anneke Meyer
Projekleier

Appendix B: Letter to parents and parental consent form

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Geagte Ouers

Attention Deficit / Hyperactivity Disorder (AD/HD) is 'n afwyking wat tussen 2% en 5% van laerskool kinders affekteer. Die kind sukkel om aandag te skenk, om sy of haar aktiwiteite te beheer en is impulsief. Dit is egter baie meer as net 'n kwessie van aandagtekort en ooraktiwiteit. Die kind sukkel om sy of haar skoolwerk baas te raak en kom moontlik nie goed oor die weg met ander kinders nie. Hulle is ook nie in staat om take te voltooi sonder toesig nie en kan ontwrigting in die familie veroorsaak.

Die probleme kan veroorsaak dat die kind nie kan aanpas by die normale vereistes van die alledaagse lewe nie. Dit word nie ontgroei nie en kan in die toekoms probleme veroorsaak met roekelose gedrag, 'n moontlike risiko vir wetsoortredings en dwelmmisbruik.

Die oorsake van AD/HD is onbekend, maar navorsing dui daarop dat dit oorerflik kan wees. Besoedelende stowwe en ongebalanseerde voeding kan ook 'n rol speel. Dit word nie veroorsaak deur 'n onvermoë om die kind te dissiplineer of beheer nie.

Dit is baie belangrik dat hierdie kinders op 'n vroeë ouderdom gediagnoseer en behandel word, om sodoende probleme by die huis en skool te voorkom en die risiko vir toekomstige probleemgedrag uit te skakel.

Die diagnose van AD/HD was nog altyd problematies. Tot op hede was die enigste instrumente vir die diagnose van AD/HD vraelyste wat voltooi is deur ouers en onderwysers. Hierdie vraelyste is egter

baie subjektief en die betekenis van sekere konsepte kan verskil tussen kulture en taalgroepe. Daarom beskou ons hierdie vraelyste as onvoldoende vir diagnose.

Die departement van Fisiologie, Universiteit van Oslo, Noorweë, het 'n kultuurvrye toets ontwikkel, sensitief vir die simptome van AD/HD. Ons wil graag hierdie toets op die verskillende kultuur en taalgroepe in die Noordelike Provinsie toepas. As hierdie instrument kinders met AD/HD suksesvol kan identifiseer, kan dit aansienlike probleme vir ouers en kinders voorkom. Terselftertyd probeer ons ook die rol van voeding en industriële besoedelende stowwe bepaal.

Hierdie is 'n goedgekeurde departementele projek van die Universiteit van die Noorde, in samewerking met die Universiteit van Oslo, Noorweë. Toestemming is verkry van die Superintendent van Onderwys vir die Noordelike Provinsie.

Ons vra eerstens dat u die aangehegde vraelys invul deur af te merk wat die meeste van toepassing is op u kind se gedrag.

As u sou instem dat u kind deelneem aan hierdie projek, sal die navorsingspan u kind se skool besoek en verskeie kinders (seuns en dogters) vanuit drie ouderdomsgroepe (7-8, 9-10, 11-12) kies. Behalwe vir die toets (in die vorm van rekenaarspeletjies) sal ons ook toets vir spierbeheer (AD/HD kinders het dikwels probleme met handskrif en ander motor-vaardighede), lengte en gewigsmates neem en indien u toestem, 'n bloedmonster neem. Die bloedmonster sal geanalaliseer word vir industriële besoedelende stowwe en ander aanduidings wat ons mag help om die oorsake van AD/HD te vind.

Die inligiting sal gebruik word vir statistiese analise en onder geen omstandighede sal die identiteit van die kind of skool geopenbaar word nie. Die feit dat u kind gekies word vir die toetse beteken nie dat hy of sy aan AD/HD ly nie, maar dit is nodig dat al die kinders in die Noordelike Provinsie verteenwoordig word in hierdie ondersoek. Indien u ons egter wil konsulteer oor enige probleme wat u kind ervaar, kan die Universiteit sielkundiges voorligting verskaf.

U en u kind se deelname wat hierdie belangrike studie betref sal bydra tot die vestiging van 'n geldige diagnostiese metode, wat kundiges in staat sal stel om kinders te identifiseer wat 'n risiko loop vor skolastiese, sosiale en emosionele probleme.

Dankie by voorbaat

Prof. A. Meyer
PROJEKLEIER

Appendix C: Parental consent form

Ek, die moeder/vader/voog van _____

gee hiermee toestemming dat my kind deur die sielkunde span van die Universiteit van die Noorde getoets word.

JA _____ NEE _____

Vir 'n bloedmonster om geneem te word deur 'n geregistreerde verpleegkundige.

JA _____ NEE _____

GETEKEN _____ DATUM _____

Appendix D Biographical questionnaire

BIOGRAFIESE DATA

Kind No.: _____

Getoets deur _____ Datum: _____/_____/200__

Skool: _____ Graad: _____ Geslag: M/V

Taal: _____

Medikasie (indien enige) _____

Dominante hand L / R

Dominante voet L / R

Rekenaarvaardig JA / NEE

Kind se naam: _____

Geboortedatum: _____ Ouderdom (maande) _____

Adres: _____
_____ Kode: _____

Pa se naam: _____ Ouderdom _____
Opleidingsvlak: _____ Beroep: _____

Pa se inkomste: Minder as R1000 ☐

R1000-R5000 ☐

Meer as R5000 ☐

Ma se naam: _____ Ouderdom _____
Opleidingsvlak: _____ Beroep: _____

Ma se inkomste: Minder as R1000 ☐

R1000-R5000 ☐

Meer as R5000 ☐

	JA	NEE
Besit die gesin "n TV?	☐	☐
"n Motor?	☐	☐
Elektrisiteit in die huis?	☐	☐
Water in die huis?	☐	☐
Is die kind aangeneem?	☐	☐
Is die ouers getroud?	☐	☐
Vervreem?	☐	☐
Geskei?	☐	☐

Is daar enige ander kinders in die gesin?

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

Appendix E: DBD (Afrikaans)

Onderwyser/Ouer DBD Vraelys

Naam van kind: _____ Vorm voltooi deur: _____

Geslag: M / V Ouderdom: _____ Skool: _____

Graad: _____ Datum voltooi: _____

Huistaal: Afrikaans/Engels/Sepedi/Xitsonga/Tshivenda/ Ander:_____

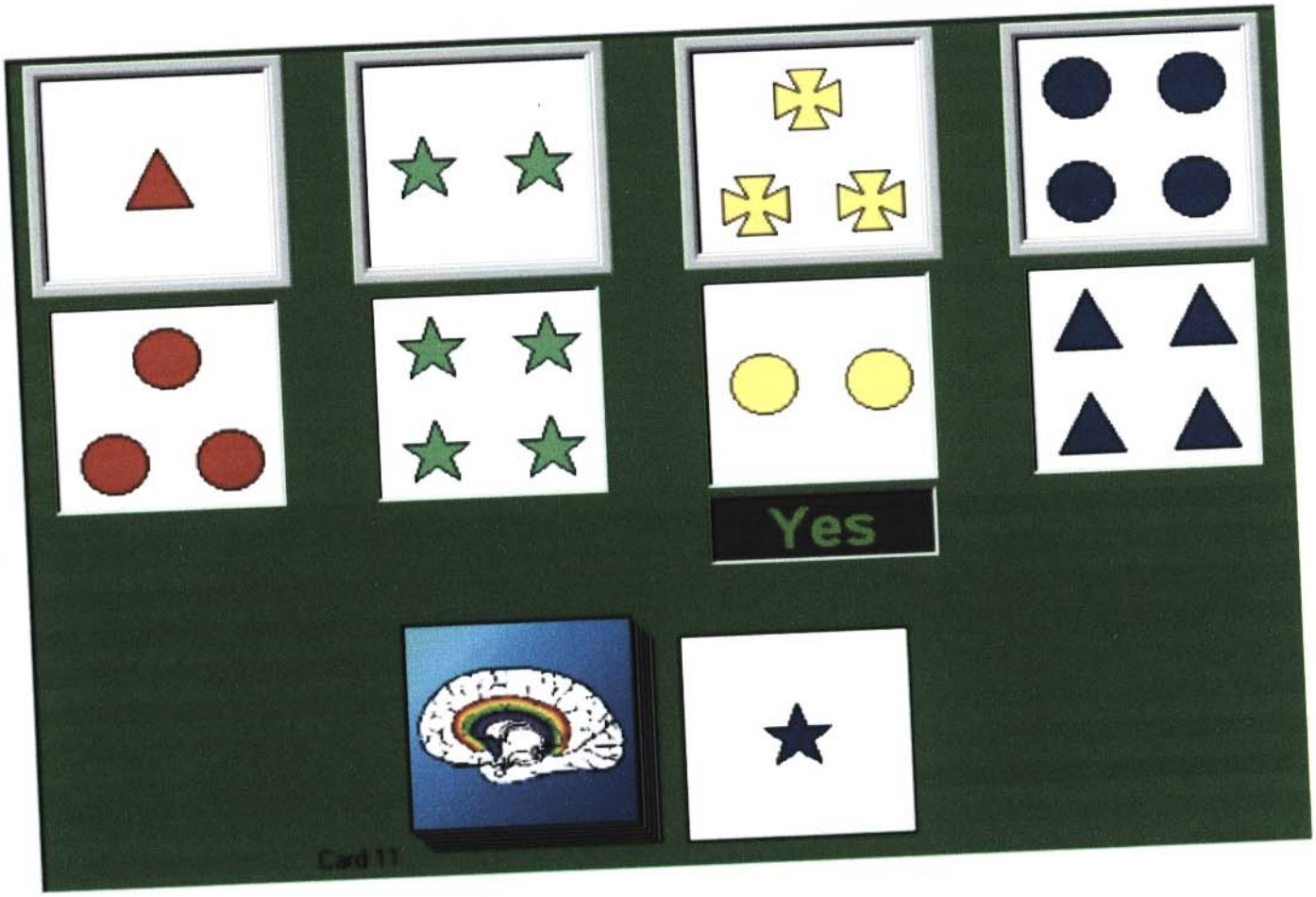
Maak 'n merkie in die kolom wat hierdie/u kind die beste beskryf. Plaas 'n vraagteken na die item waarvan u die antwoord nie weet nie.

	Nooit	Soms	Dikwels	Altyd
1. Onderbreek of steur ander persone dikwels (bv. val in die rede tydens gesprekke of meng in gedurende spel).				
2. Het minstens twee keer van die huis of plek van versorging weggeloop en oornag weggebly (of een keer vir 'n lang tydperk).				
3. Argumenteer dikwels met volwassenes.				
4. Vertel dikwels leuens om artikels of gunste te verkry of om verpligtinge te ontduik (lei ander om die bos).				
5. Inisieer dikwels fisieke bakleiery met ander lede van die huisgesin.				
6. Het persone liggaamlik mishandel.				
7. Praat dikwels onophoudelik.				
8. Het artikels van geringe waarde gesteel sonder om die slagoffer fisies te bedreig (bv. winkeldiefstal, vervalsing).				
9. Word dikwels maklik afgelei deur uitwendige prikkels.				
10. Draai dikwels stokkies, beginnende voor 13 jaar oud.				
11. Vroetel dikwels met hande en voete, kan nie stilsit nie.				
12. Is dikwels boosaardig en wraaksugtig				
13. Blameer ander vir sy of haar foute of wangedrag				
14. Het ander se eiendom moedswillig vernietig (nie deur brandstigting nie)				
15. Verontagsaam dikwels opsetlik volwassenes se versoeke of reëls				
16. Lyk of hy/sy nie luister wanneer hy/sy direk aangespreek word.				
17. Roep dikwels die antwoord uit voordat 'n vraag voltooi is				
18. Inisieer fisieke bakleiery met ander wat nie deel van sy huishouding is nie (maats op skool of in sy omgewing)				
19. Ondervind probleme om ontspanningsaktiwiteite op 'n rustige en bedaarde wyse te doen				
20. Ondervind moeite met aandag skenk aan fyn besonderhede of maak agtelosige foute in skoolwerk of ander opdragte				

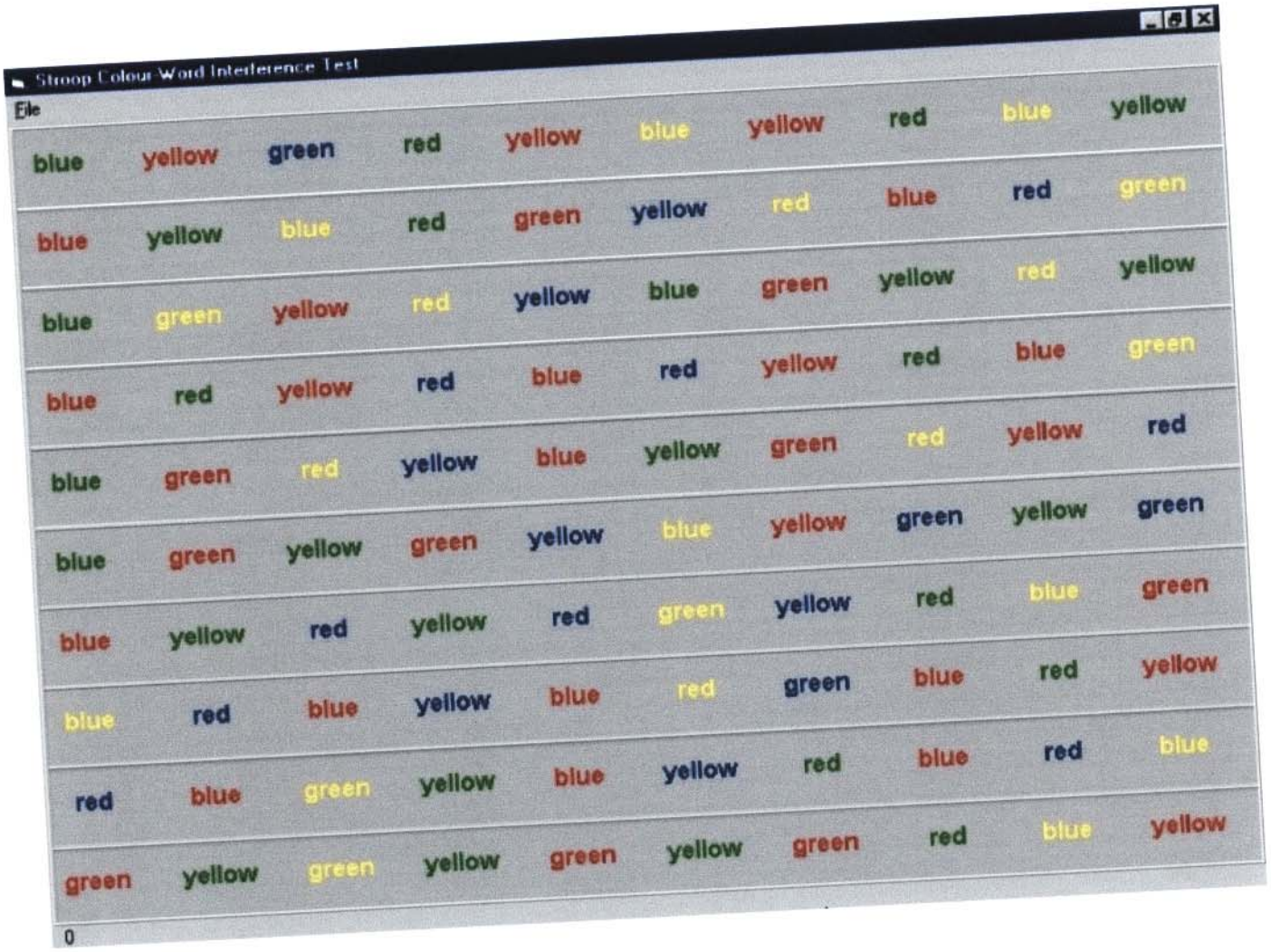
Vertaal met toestemming van Dr. WE Pelham, State University New York, Buffalo, USA

	Nooit	Soms	Dikwels	Altyd
21. Is dikwels kwaad en verontwaardig.				
22. Verlaat dikwels sitplek in die klas of in ander situasies waar daar van hom/haar verwag word om te bly sit..				
23. Is dikwels liggeraak of vererg hom/haar maklik vir ander.				
24. Het dikwels probleme om instruksies te volg en voltooi nie skoolwerk, take en opdragte nie (nie a.g.v. opposisionele gedrag of taalonvermoë nie).				
25. Verloor dikwels sy/haar humeur.				
26. Het dikwels probleme om aandag te behou gedurende take of spelaktiwiteite.				
27. Vind dit dikwels moeilik om vir sy/haar beurt te wag.				
28. Het iemand gedwing tot seksuele aktiwiteite.				
29. Terroriseer, bedreig of intimideer dikwels ander.				
30. Is voortdurend aan die gang, of tree op asof aangejaag.				
31. Verloor dikwels voorwerpe wat noodsaaklik is vir take of aktiwiteite (bv.potlode, boeke, sporttoerusting, gereedskap).				
32. Hardloop of klim en klouter dikwels oormatig in situasies waar dit onvanpas is (by adolossente en volwassenes mag dit beperk wees tot subjektiewe gevoelens van rusteloosheid).				
33. Het diere mishandel.				
34. Vermy dikwels, hou nie van, of is onwillig om take uit te voer wat volgehoue verstandelike inspanning vereis (bv. skoolwerk of huiswerk).				
35. Bly dikwels in die nag weg van die huis, ondanks ouerlike verbod, voor die ouderdom van 13 jaar.				
36. Vererg ander persone dikwels opsetlik.				
37. Het gesteel terwyl hy/sy 'n slagoffer gekonfronteer het (d.m.v. geweld of dwang).				
38. Het opsetlik brand gestig met die doel om ernstige skade te berokken.				
39. Het dikwels probleme om take en aktiwiteite te struktureer en te organiseer.				
40. Het by iemand anders se huis, motor of gebou ingebreek.				
41. Is dikwels vergeetagtig tydens daaglikse aktiwiteite.				
42. Het 'n wapen gebruik wat ernstige beserings aan ander kan veroorsaak (bv. 'n knuppel, baksteen, gebreekte bottel, mes, vuurwapen).				

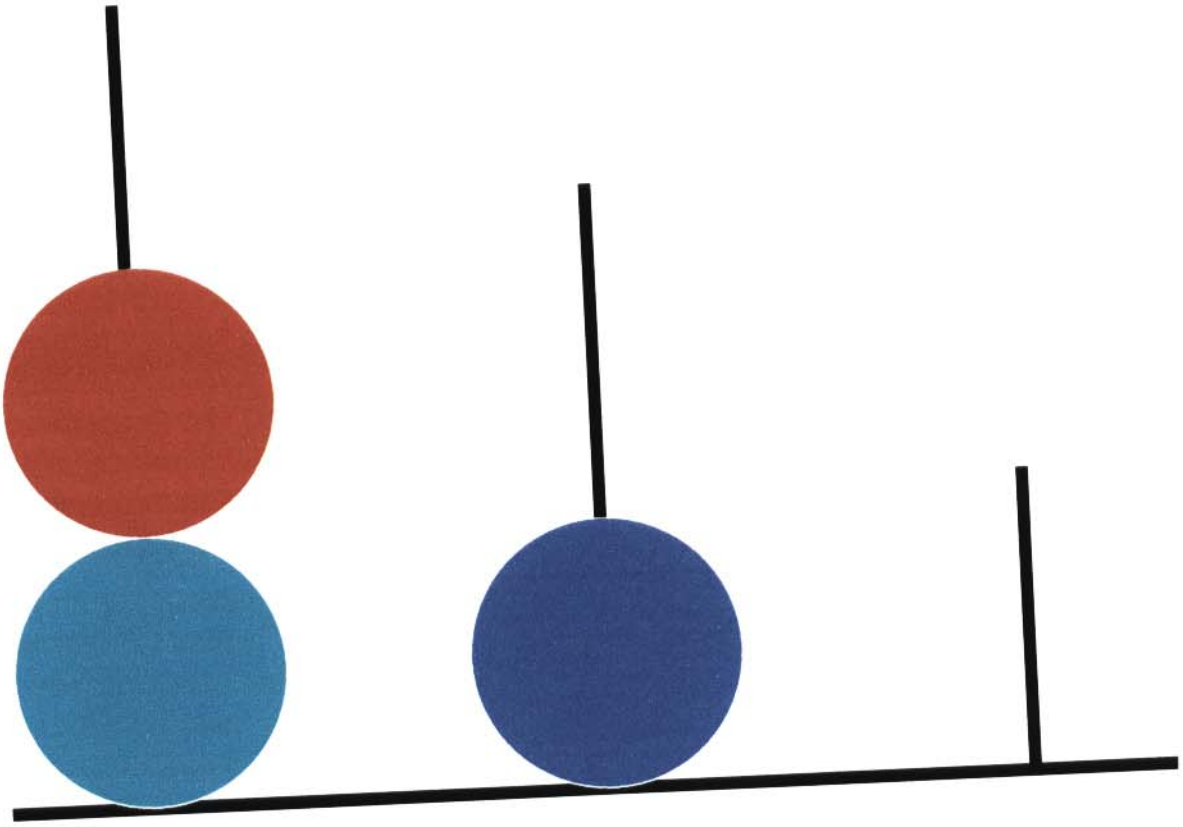
Appendix F: WCST illustration



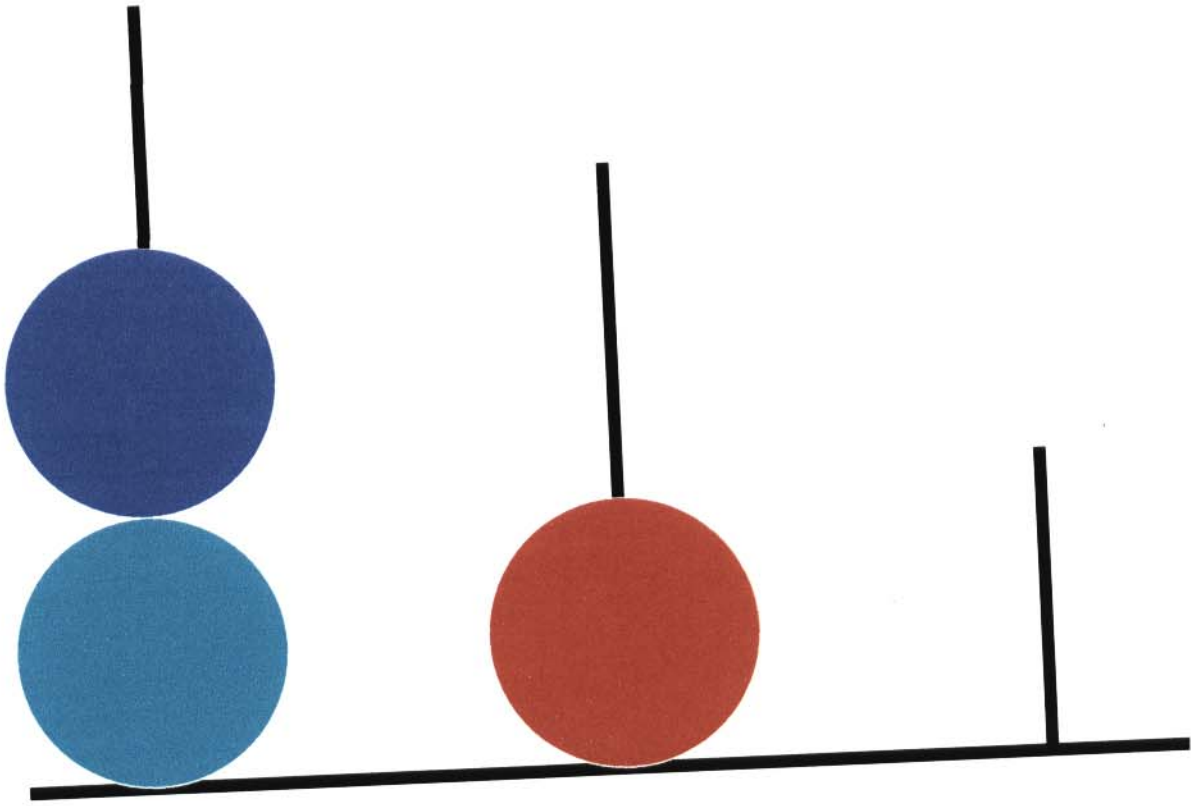
Appendix G: Stroop test illustration



Appendix H: TOL



Start



Task 3: 3 moves