An ecosystemic approach to addressing attentional difficulties and heightened motor activity

Keywords

AD/HD

Attention

Motor Activity

Hyperactivity

Academic Performance

Ecosystemic

Sound Therapy

EEG Neurotherapy

Neurodevelopmental

Nutrition

Alternative Approach

Neurophysiological

Neuro anatomincal

Neurochemical

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General Orientation

1. Introduction

This chapter comprises a general exploration of the research problem, and the questions that arose in the process of outlining the problem. These questions together inform the aim of the research. Chapters 2 to 7 report on the in-depth literature study and the experimental research study that were undertaken to answer these questions. The final chapter summarises the findings of the literature study and the results of the experimental research study, suggests some answers to the research questions, explores some insights offered through the research process, and makes recommendations for possible future use.

2. Research Questions

Whilst dealing with a large number of children presenting with attentional difficulties and heightened motor activity in my private practice as an educational psychologist, a specific problem started to present itself. Traditionally, children with attentional difficulties and heightened motor activity were medicated, and some form of therapeutic intervention was provided, usually behavioural modification. Despite these interventions, most children persisted with attentional difficulties and heightened motor activity to some degree. The usual methods did not seem to alleviate or remediate the behaviour in the long term. I began to search for more information as to how children presenting with attentional difficulties and heightened motor activity could be successfully assisted.

Many practitioners and theorists in the field of AD/HD believe that attentional difficulties and heightened motor activity can and should be viewed from a number of perspectives deviating from the usual 'medical model'. Their rationale is that possible underlying causes of AD/HD symptoms may arise in different areas. Symptoms should be seen as indicators of underlying processes rather than as

problems in themselves. Symptoms do not explain their presence, nor do they present possible solutions. With this in mind, and some personal experience of success when using alternative approaches, this thesis aims to explore non-traditional interventions with learners showing attentional difficulties and heightened motor activity. Even though this thesis is presented within the epistemology of educational psychology, the traditional practices of a psycho-educational approach are not used as a frame of reference

I investigated less conventional kinds of interventions and eventually, the need arose for experimental research into possible alternative theories and intervention techniques. Experimental research and academic publications in specifically alternative methods are limited. This lack proved to be a challenge in terms of reference material for this thesis.

3. Aim of Research

This thesis aims to address the need for experimental research into non-traditional theories and methods to successfully address attentional difficulties and heightened motor activity. The theories were also tested against academic outcomes as a result of appropriate attention and modulated motor activity.

4. Hypotheses

The following hypotheses were formulated as directive questions to the research:

Hypothesis 1:

H0: Disturbances in **attention** will not improve after an ecosystemic intervention.

H1: Disturbances in attention will improve after an ecosystemic intervention.

Hypothesis 2:

H0: Disturbances in **motor activity** will not improve after an ecosystemic intervention.

H1: Disturbances in **motor activity** will improve after an intervention based on an ecosystemic intervention.

Hypothesis 3:

H0: **Academic performance** will not improve after an ecosystemic intervention.

H1: Academic performance will improve after an ecosystemic intervention.

5. Research Methodology

Triangulation was used for the research design. The following components formed the cornerstones of the triangulation:

5.1 Literature Study

An in-depth literature study was conducted into the specific cluster of behaviours that are used as criteria to classify children with attentional difficulties and heightened motor activity into diagnostic categories. Both traditional and non-traditional sources were included in this literature study. As stated previously, experimental research and academic publications in specifically alternative methods are limited. This lack proved to be a challenge in terms of reference material for this thesis.

5.2 Quantitative Research

The quantitative research component of this thesis comprises true experimental research as well as a quasi-experimental design. This research has a strong quantitative component, as a number of standardised measuring tools were included in the pre-, mid- and post-testing phases.

5.3 Qualitative Research

As some of the aspects of human behaviour cannot be quantified, a qualitative research component and subjective observation were also included in the investigation.

6. Thesis Outline

The thesis presents as follows:

Chapter 1:	General orientation					
Chapter 2:	Traditional	conceptualisation	and	treatment	of	attention
	difficulties a	nd heightened moto	r activi	tv		

- Chapter 3: Theoretical conceptualisation of attention and motor activity
- Chapter 4: An ecosystemic conceptualisation of attentional difficulties and heightened motor activity
- Chapter 5: Intervention design
- Chapter 6: Research methodology
- Chapter 7: Research results
- Chapter 8: Conclusion and recommendations

7. Terminology

Clarification of terminology

Even though this thesis does not attempt to diagnose, label or categorise, the term AD/HD has been used when directly quoted from a source, or when referring to the specific traditional practice of diagnosing AD/HD. The terms *attention difficulties* and *heightened motor activity* were used as the core terminologies in this thesis, referring to the condition of AD/HD but without the context of diagnosing or labelling.

AD/HD

Although many different possible definitions are found in the literature, AD/HD as a cluster of symptoms that manifests in 3-5% of the population is referred to in the manner used in the *Diagnostic and Statistical Manual of Mental Disorders* as "a persistent pattern of inattentive and/or hyperactivity/impulsivity that is more frequent and severe than is typically observed in individuals at a comparable level of development" (American Psychiatric Association [APA] 1994:78).

Attention

Attention is described in the Collins English Dictionary (Sinclair 1992:97) as "concentrated direction of the mind, especially to a problem or task" as well as "the act of concentrating on any one of a set of objects or thoughts."

Attentive behaviour

This has been defined by Warner-Rogers, Taylor and Sandberg (2000:520) as the ability to "systematically sample and extract relevant information from the environment and to regulate responsiveness accordingly."

Inattention

The DSM-IV (APA 1994:78) describes individuals with **inattention** as people who may fail to give close attention to details or may make careless mistakes in schoolwork or other tasks. Work is often messy and performed carelessly and without considered thought. Individuals often have difficulty sustaining attention in tasks or play activities and find it hard to persist with tasks until completion.

Hyperactive

Hyperactivity is described in the Collins English Dictionary (Sinclair 1992:764) as "abnormally active."

The DSM-IV (APA 1994:78) describes **hyperactivity** as a condition that may be manifested by fidgetiness or squirming in one's seat, by not remaining seated when expected to do so, by excessive running or climbing in situations where it is inappropriate, by having difficulty playing or engaging quietly in leisure activities, by appearing to be often "on the go" or as if "driven by a motor", or by talking excessively.

Concentration

Concentration refers to "intense mental application; complete attention or the act or process of concentrating" (Sinclair 1992:333).

Neural development

The study of **neural development** draws on both **neuroscience** and **developmental biology** to describe the cellular and molecular mechanisms by which **complex nervous systems** emerge during **embryonic development** and throughout life. Developmental biology is the study of the process by which the organism grows and develops. Modern developmental biology studies the **genetic control of cell growth**, **differentiation** and **morphogenesis**, which is the process that gives rise to tissues, organs and anatomy (http://en.wikipedia.org/wiki /Neurodevelopment).

Neurodevelopmental delay

Neurodevelopmental delay refers to any inconsistency in the normal development as set out in the previous definition, and which is outside of the normal developmental sequence of age-appropriate activities.

Neurodevelopmental programme

A neurodevelopmental programme generally refers to a programme (usually movement based) to address identified neurodevelopmental delays, as discussed in the previous definition.

<u>Movement</u>

On a neurophysiological level body movement occurs when muscles contract across joints (Marieb 2003:176) and a chemical is released that stimulates neural activity (Changeux & Conic 1987:264).

Neurofeedback

Neurofeedback is a form of biofeedback. Biofeedback is the use of instrumentation to mirror psychophysiological processes of which the individual is not normally aware and which may be brought under voluntary control (Thompson & Thompson 2003:2). In neurofeedback the psychophysiological process that is brought under the individual's attention is that of brainwave patterns or the person's EEG.



Nutrition

The Collins English Dictionary (Sinclair 1992:1074) refers to nutrition as a process involving the intake of nutrient materials and their subsequent assimilation into the tissues.

Sound therapy

Sound therapy can best be described as a programme of sound stimulation to develop and improve listening (Madaule 1994:19).

Ecosystemic

The term "systemic" is defined in the Collins English Dictionary (Sinclair 1992:1565) as "affecting the entire body." Ecosystem generally refers to environmental aspects. In this study the term *ecosystemic* will refer to both the internal and external environment of the human organism.

8. Conclusion

This chapter outlined the aim of the study and the research hypotheses under investigation. Key terms were explained, and a brief overview of the chosen research methodology was given. The following chapter (chapter 2) begins with a presentation of the traditional views on AD/HD, the diagnostic criteria currently used and the procedures used in making diagnoses.

Traditional Conceptualisation and Treatment of Attentional Difficulties and Heightened Motor Activity

1. Introduction

Since the early 1900s, attention difficulties and heightened motor activity, as an affliction of children, has been perceived from various perspectives. In the 20th century, the term AD/HD was coined to describe a cluster of behaviours observed in children. Usually, once this label has been assigned, the label and the symptoms associated with the label are treated. Some researchers suggest that AD/HD is a physiological problem of the brain, a malfunction in the central nervous system; others believe it to be a reaction due to an allergy. Yet others claim it exists largely in the eye of the beholder (Davison 2001:231).

This thesis does not focus on the label AD/HD as a diagnosis, but rather examines attentional difficulties and heightened motor activity. This thesis will thus look beyond the label at the underlying causes of the behaviours listed as symptoms. However, for the sake of brevity, this chapter will use the term AD/HD to refer to attentional difficulties and heightened motor activity.

One of the goals of this thesis is to investigate, as widely as possible, existing perceptions of AD/HD and to determine the underlying causes of so-called AD/HD. This could possibly lead to a new understanding of this phenomenon and new methods of intervention that would improve attention and motor activity, with a positive outcome in academic performance. This chapter provides an overview of the traditional and historical conceptualisation of AD/HD, the history of the diagnostic criteria over the years and current methodology concerning diagnosis and treatment.

2. The History of AD/HD

Dysfunction in attention has been described in the medical literature for about one hundred years. Some researchers (Singh 2002; Barkley 1997) claim that the British physician George Still first described AD/HD in a series of lectures in *The Lancet* in the early 1900s. According to Amen (2001:xvi), Still unfortunately did not understand that AD/HD is a medical disorder and labeled these children as "morally defective." This disorder was thought to be inherited by some children, or was the result of brain injury in others. It is now generally agreed that Still's young patients, while presenting with some **hyperactive** and **inattentive** behaviours, meet more of the current diagnostic criteria for oppositional-defiant disorder or conduct disorder (Palmer & Finger as cited in Singh 2002:361; Young-Loveridge 1997:2).

The association between behaviour problems and biological factors was reinforced by an epidemic of encephalitis during and after World War I (Young-Loveridge 1997:2). The idea that some type of brain disturbance could be responsible for abnormalities in attention and hyperactivity in children arose from the pathological findings and physiological theories related to this epidemic that spread through the city of Vienna in the early years of the 20th century. This infection of the brain, which was called *encephalitis lethargia*, left a strange disruption of behaviour in its victims. A Viennese physician named von Economo (1918) distinguished himself by his classic study on the brain damage that this disorder inflicted on its patients. He identified two symptomatic patterns associated with two areas of inflammatory injury to the rostral brain stem and the hypothalamus. In one of these patterns patients were inattentive and lethargic, often becoming comatose; in the other, they showed the opposite behaviour, being unable to fall asleep and displaying impulsive and often uncontrolled physical behaviour. Von Economo concluded that the two parts of the brain affected were interactive, and collectively responsible for the coordination of mental and physical arousal (Sterman 2000:1).

In the 1940s researchers working with mentally retarded children studied the psychological effects of brain injury and noted the **distractibility** and **hyperactivity** of these children. Drawing upon these observations, neurologists who later encountered a non-lethal pattern of mental lethargy combined with motor

hyperactivity in children proposed that elements of this same regulatory system were damaged in some complex way by infection or brain trauma. The term **Brain Damage Syndrome** was adopted. By mid-century, however, the lack of evidence for gross neurological damage or deficits associated with this syndrome led to a change in accepted terminology and the adoption of labels such as **Minimal Cerebral Dysfunction** (MCD) or **Minimal Brain Damage** (MBD) (Young-Loveridge 1997:2), and **Childhood Behaviour Disorder** to describe a syndrome that included hyperactive behaviour, short attention span, frequent mood shifts, and various minor perceptual disturbances (Stewart, Steffler, Lemoine & Leps 2001:103).

In 1968, the term **Hyperkinetic Reaction of Childhood** was officially recognised and named as a disorder by the American Psychiatric Association (APA). The use of the word **reaction** here is significant, because the APA makes a distinction between a disorder and a reaction, the latter suggesting a milder, possibly less chronic condition (Levingston 1997:10). In the 1970s, the focus turned from **behavioural problems** to **attentional problems**, and, in 1980, the term **Attention Deficit Disorder** (ADD) became the official term used by the APA (Young-Loveridge 1997:2).

The label effectively shifted diagnostic emphasis from hyperactivity to attention as the core problem of the disorder. Now children with or without hyperactivity could be diagnosed with ADD. Several years later, the DSM-III-R (APA 1987) reflected this shift in thinking about the core of the disorder when ADD was changed to the present-day AD/HD – Attention Deficit Disorder with or without Hyperactivity. These are not merely changes in nomenclature; as a diagnostic category AD/HD has widened significantly over the years. Baumgaertel, Wolraich and Dietrich (1995) and other have shown that changes in diagnostic criteria for AD/HD between the 1987 version of DSM-III-R to the 1994 version of DSM-IV support a 57% increase in children meeting AD/HD criteria (Singh 2002:361). The table below summarises the changes in terminology used to define this condition over time. Singh (2002:361) makes it clear that the history of AD/HD is characterised mainly by the pursuit of a clinical definition.

Table 1

A historical overview of Attention-Deficit / Hyperactivity Disorder terminology

1900s	Still's Disorder: A disorder of defective moral control
1920s	Post-Encephalitic Disorder
1940s	Brain Damage Syndrome
1960s	Minimal Brain Dysfunction (MBD)
1968	Hyperkinetic Reaction of Childhood (DSM-II)
1980	Attention Deficit Disorder with Hyperactivity (ADD-H; DSM - III)
	Attention Deficit Disorder without Hyperactivity (ADD/noH; DSM – III)
1987	Attention Deficit / Hyperactivity Disorder (AD/HD; DSM – III – R)
1991	Attention Deficit Disorder (ADD; US Department of Education Policy
	Memo)
1994	Attention Deficit / Hyperactivity Disorder; 3 subtypes (AD/HD; DSM – IV)

(Mather & Goldstein 2001:50)

3. Current Clinical Diagnostic Criteria

Diagnosing AD/HD has always presented a challenge. The behavioural criteria that have been specified, namely inattention, low frustration tolerance, impulsivity, poor organisation of behaviour, distractibility, and hyperactivity are all exhibited to some extent by all children at one time or another. In early childhood, it may be difficult to distinguish symptoms of Attention Deficit/Hyperactivity Disorder from age-appropriate behaviors in active children (e.g., running around or being noisy) (APA 1997).

Diagnosis is further complicated by the fact that these symptoms frequently appear in other disorders as well, such as learning disabilities, Tourette's Syndrome, conduct disorders, phobic and anxiety disorders, lead poisoning, fetal alcohol syndrome, retardation, depression, mania, substance abuse, and even some seizure disorders. These facts have raised significant problems for both professionals and parents alike, and have led to the suggestion that AD/HD is being over-diagnosed, particularly in the United States (Sterman 2000:3). It is likely that this trend is duplicated in South Africa.

The tenth revision of the *International Classification of Diseases* (ICD-10; World Health Organisation 1993) and the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV; APA 1994) list similar criteria for this disorder characterised by **inattention**, **impulsivity**, and **hyperactivity**. Whereas the DSM-IV recognises the existence of subtypes, allowing a diagnosis based on symptoms either from a combined hyperactive-impulsive dimension or from inattentive behaviours, the ICD-10 requires symptoms to be present from each of the three categories of behaviour. This difference has had a significant impact on the reported prevalence of AD/HD and on the selection of which children should undergo treatment (Clarke, Barry, McCarthy, Selikowitz & Brown 2002:276). An outline of the two resources is given below. For the purpose of accuracy and completeness, the sections from the ICD-10 manual and the DSM-IV-R are quoted directly without changes or interpretation. These sections are presented here in this manner to allow a full representation of current literature on AD/HD and to form a comprehensive backdrop against which other findings will be viewed.

3.1 International Classification of Disorders (ICD-10)

The table below gives a direct transcription of the ICD-10 classification of AD/HD as it is currently in use as a diagnostic tool.

Table 2 ICD-10 classification

Behavioural and emotional disorders with onset usually occurring in childhood and adolescence (F90-F98)

F90 Hyperkinetic disorders

A group of disorders characterized by an early onset (usually in the first five years of life), lack of persistence in activities that require cognitive involvement, and a tendency

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to move from one activity to another without completing any one, together with disorganized, ill-regulated, and excessive activity. Several other abnormalities may be associated. Hyperkinetic children are often reckless and impulsive, prone to accidents, and find themselves in disciplinary trouble because of unthinking breaches of rules rather than deliberate defiance. Their relationships with adults are often socially disinhibited, with a lack of normal caution and reserve. They are unpopular with other children and may become isolated. Impairment of cognitive functions is common, and specific delays in motor and language development are disproportionately frequent. Secondary complications include dissocial behaviour and low selfesteem.

Excludes: anxiety disorders (F41.-) mood [affective] disorders (F30 – F39) pervasive developmental disorders (F84. -) schizophrenia (F20. -)

F90.0 Disturbance and activity level

Attention deficit:

- Disorder with hyperactivity
- Hyperactivity disorder
- Syndrome with hyperactivity

Excludes: hyperkinetic disorder associated with conduct disorder (F90.1)

F90.8 Other hyperactivity disorders

F90.0 Hyperkinetic disorder, unspecified

Hyperkinetic reaction of childhood or adolescence NOS Hyperkinetic syndrome NOS

F90.1 Hyperkinetic conduct disorder

Hyperkinetic disorder associated with conduct disorder
F90.8 Other hyperactivity disorders

F90.0 Hyperkinetic disorder, unspecified

Hyperkinetic reaction of childhood or adolescence NOS Hyperkinetic syndrome NOS

Typical ADD falls under the code:

F98 Other behavioural and emotional disorders with onset usually occurring in childhood and adolescence A heterogeneous group of disorders that share the characteristics of an onset in childhood but otherwise differ in many respects. Some of the conditions represent well-defined syndromes but others are no more than symptoms complexes that need inclusion because of their frequency and association with psychosocial problems, and because they cannot be incorporated into other syndromes.

F98.8 Other specific behavioural and emotional disorders with onset usually occurring in childhood and adolescence.

(WHO 2006: http://www.who.int/classifications/apps/icd/icd10online/)

3.2 Diagnostic and Statistical Manual of Mental Disorders

The *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Test Revision* (APA 2000), defines the range of childhood developmental, emotional, and behavioural problems that appear to set children apart from their peers and cause impairments in daily functioning.

The DSM-IV diagnostic criteria, presented in table 3, make an effort to correct the previously mistaken course that AD/HD represents a disorder of only one type (APA 1994:83-85). The field studies of the AD/HD diagnosis were more comprehensive



and better structured than previous efforts. Note that using the current criteria, three subtypes of AD/HD is identified: 1) AD/HD, combined type; 2) AD/HD, predominantly inattention type; 3) AD/HD, predominantly hyperactive-impulsive type (Mather & Goldstein 2001:50).

Table 3

DSM-IV Diagnostic criteria for attention deficit / hyperactivity disorder

Diagnostic criteria for attention deficit/hyperactivity disorder (cautionary statement)

A. Either (1) or (2):

(1) *inattention:* 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 developmental level:

(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 behavior 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)

(h) is often easily distracted by extraneous stimuli

(i) is often forgetful in daily activities

(2) *hyperactivity-impulsivity:* six (or more) of the following symptoms of hyperactivity-impulsivity 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 feet 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 adolescent 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

Impulsivity

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

(h) often has difficulty awaiting 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 age 7 years.

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

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



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E. The symptoms do not occur exclusively during the course of a <u>pervasive developmental disorder</u>, <u>schizophrenia</u>, or other psychotic disorder and are not better accounted for by another mental disorder (e.g., <u>mood disorder</u>, <u>anxiety disorder</u>, <u>dissociative disorder</u>, or a <u>personality disorder</u>).

Code based on type:

314.01 Attention Deficit/Hyperactivity Disorder, Combined
Type: if both Criteria A1 and A2 are met for the past 6 months
314.00 Attention Deficit/Hyperactivity Disorder,
Predominantly Inattentive Type: if Criterion A1 is met but
Criterion A2 is not met for the past 6 months
314.01 Attention Deficit/Hyperactivity Disorder,
Predominantly Hyperactivity-Impulsivity Type: if Criterion
A2 is met but Criterion A1 is not met for the past 6 months

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

(APA 1994:83-85)

3.2.1 Diagnostic features according to the DSM-IV (1994)

The essential feature of Attention Deficit/Hyperactivity Disorder is a **persistent pattern of inattentive and/or hyperactivity/impulsivity that is more frequent and severe than is typically observed in individuals at a comparable level of development** (criterion A). Some hyperactive-impulsive or inattentive symptoms that cause impairment must have been present before the age of seven, although many individuals are diagnosed after the symptoms have been present for a number of years (criterion B). Some impairment from the symptoms must be present in at least two settings (e.g., at home and at school or work) (criterion C). There must be clear evidence of interference with developmentally appropriate social, academic, or occupational functioning (criterion D). The disturbance does not occur exclusively during the course of a pervasive developmental disorder, schizophrenia, or other

psychotic disorder and is not better accounted for by another mental disorder (e.g., a mood disorder, anxiety disorder, dissociative disorder, or personality disorder) (criterion E) (APA 1994:78).

3.2.1.1 Inattention

Inattention may be manifest in academic, occupational, or social situations. Individuals with this disorder may fail to give close attention to details or may make careless mistakes in schoolwork or other tasks (criterion A1a). Work is often messy and performed carelessly and without considered thought. Individuals often have difficulty sustaining attention in tasks or play activities and find it hard to persist with tasks until completion (criterion A1b). They often appear as if their mind is elsewhere or as if they are not listening or did not hear what has just been said (criterion A1c). There may be frequent shifts from one uncompleted activity to another. Individuals diagnosed with this disorder may begin a task, move on to another, and then turn to vet something else, prior to completing any one task. They often do not follow through on requests or instructions and fail to complete schoolwork, chores, or other duties (criterion A1d). Failure to complete tasks should be considered in making this diagnosis only if it is due to inattention as opposed to other possible reasons (e.g., a failure to understand instructions). These individuals often have difficulty organizing tasks and activities (criterion A1e). Tasks that require sustained mental effort are experienced as unpleasant and markedly aversive. As a result, these individuals typically avoid or have a strong dislike for activities that demand sustained selfapplication and mental effort or that require organizational demands or close concentration (e.g., homework or paperwork) (criterion A1f). This avoidance must be due to the person's difficulties with inattention and not due to a primary oppositional attitude, although secondary oppositionalism may also occur. Work habits are often disorganized and the materials necessary for doing the task are often scattered, lost, or carelessly handled and damaged (criterion A1g). Individuals with this disorder are easily distracted by irrelevant stimuli and frequently interrupt ongoing tasks to attend to trivial noises or events that are usually and easily ignored by others (e.g., a car hooting, a background conversation) (criterion A1h). They are often forgetful in daily activities (e.g., missing appointments, forgetting to bring lunch) (criterion A1i). In social situations, inattention may be expressed as frequent shifts in conversation, not listening to others, not keeping one's mind on conversations, and not following details or rules of games or activities (APA 1994:78-79)

3.2.1.2 Hyperactivity

Hyperactivity may be manifested by fidgetiness or squirming in one's seat (criterion A2a), by not remaining seated when expected to do so (criterion A2b), by excessive running or climbing in situations where it is inappropriate (criterion A2c), by having difficulty playing or engaging quietly in leisure activities (criterion A2d), by appearing to be often "on the go" or as if "driven by a motor" (criterion A2e), or by talking excessively (criterion A2f). Hyperactivity may vary with the individual's age and developmental level, and the diagnosis should be made cautiously in young children. Toddlers and preschoolers with this disorder differ from normally active young children by being constantly on the go and into everything; they dart back and forth, are "out of the door before their coat is on," jump or climb on furniture, run through the house, and have difficulty participating in sedentary group activities in preschool classes (e.g., listening to a story). School-age children display similar behaviours but usually with less frequency or intensity than toddlers and preschoolers. They have difficulty remaining seated, get up frequently, and squirm in, or hang on to the edge of their seat. They fidget with objects, tap their hands, and shake their feet or legs excessively. They often get up from the table during meals, while watching television, or while doing homework; they talk excessively; and they make excessive noise during quiet activities. In adolescents and adults, symptoms of hyperactivity take the form of feelings of restlessness and difficulty engaging in quiet sedentary activities (APA 1994:79).

3.2.1.3 Impulsivity

This manifests itself as impatience, difficulty in delaying responses, blurting out answers before questions have been completed (criterion A2g), difficulty awaiting one's turn (criterion A2h), and frequently interrupting or intruding on others to the point of causing difficulties in social, academic, or occupational settings (criterion A2i). Others may complain that they cannot get a word in edgewise. Individuals with this disorder typically make comments out of turn, fail to listen to directions, initiate conversations at inappropriate times, interrupt others excessively, intrude on others, grab objects from others, touch things they are not supposed to touch, and clown around. Impulsivity may lead to accidents (e.g. knocking over objects, banging into people, grabbing a hot pan) and to engagement in potentially dangerous activities without consideration of possible consequences (e.g., riding a skateboard over extremely rough terrain) (APA, 1994: 79).

Behavioral manifestations usually appear in multiple contexts, including home, school, work, and social situations. To make the diagnosis, some impairment must be present in at least two settings (criterion C). It is very unusual for an individual to display the same level of dysfunction in all settings or within the same setting at all times. Symptoms typically worsen in situations that require sustained attention or mental effort or that lack intrinsic appeal to novelty (e.g., listening to classroom teachers, doing class assignments, listening to or reading lengthy materials, or working on monotonous, repetitive tasks). Signs of the disorder may be minimal or absent when the person is under very strict control, is in a novel setting, is engaged in especially interesting activities, is in a one-to-one situation (e.g., the clinician's office), or while the person experiences frequent rewards for appropriate behaviour. The symptoms are more likely to occur in group situations (e.g., in playgroups, classrooms, or work environments). The clinician should therefore inquire about the individual's behaviour in a variety of situations within each setting (APA 1994:79-80).

Subtypes

Although most individuals have symptoms of both inattention and hyperactivityimpulsivity, there are some individuals in whom one or the other pattern is predominant. The appropriate subtype (for a current diagnosis) should be indicated based on the predominant symptom pattern for the past 6 months.

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

Attention Deficit/Hyperactivity Disorder, Predominantly Hyperactive-Impulsive **Type.** This subtype should be used if six (or more) symptoms of hyperactivityimpulsivity (but fewer than six symptoms of inattention) have persisted for at least 6 months. Inattention may often still be significant clinical feature in such cases (APA 1994:80).

The essential feature of ADD is a persistent pattern of **inattention** and/or **hyperactivity** that is more frequent or severe than is typically observed in individuals at a comparable level of development (criterion A).

When problems with <u>inattention</u>, hyperactivity, and impulsiveness develop in childhood and persist, in some cases into adulthood, this <u>mental disorder</u> may be diagnosed (APA 1994:80).

3.3 Conclusion

The issue of which dimension represents the most distinguishing impairment of the disorder is not resolved. The general consensus is that symptoms of AD/HD fall into two broad dimensions: those related to the behavioural manifestation of faulty **attention** and those related to **impulsivity and hyperactivity**. Symptoms of impulsivity and hyperactivity appear to co-occur at such a high frequency that on a statistical basis it is difficult to separate them. With regard to predicating future functioning, however, the level of impulsivity appears to correlate positively with impaired classroom performance and life outcomes: the greater the degree of reported impulsive behaviour, the more problems in the classroom and later in life. Thus, it has been increasingly hypothesised that the core impairment in AD/HD represents faulty inhibition or self-control, leading to a constellation of related symptoms (Mather & Goldstein 2001:49).

4. Prevalence of AD/HD

According to Wender (1975:47), the childhood cognitive and behaviour problem characterised by disorders of attention, impulse control, and hyperactivity has long constituted the most chronic behaviour disorder, and since the early 1980s, these children have comprised the largest single source of referrals to child mental health centers (Mather & Goldstein 2001:45). The inherent ambiguity of AD/HD symptoms makes estimating the actual percentage of children in the population with AD/HD a difficult and controversial enterprise. Most children will at some point or the other present with some of the symptoms for a period of time. As previously noted, the mere changes in the DSM criteria over time contributed to a 57% increase in diagnoses between 1987 and 1994.

4.1 Estimates

Cooper (2001) notes that prevalence rates in the US and UK are similar, but estimates that the prevalence of AD/HD in American children range widely. According to Stewart et al. (2001:103) the prevalence of Attention Deficit/Hyperactivity Disorder was estimated at 3%-5% in school-age children. Data on prevalence in adolescence and adulthood are limited. Since 2001, the rate of incidence has doubled every four to seven years. Barkley (1990:61) points out that the 3-5% prevalence figure hinges on how one chooses to define AD/HD, the population studies, the geographic location of the survey, and the degree of agreement required among parents, teachers, and professionals on the symptoms displayed. Thus, estimates vary between 1% and 20% (Barkley 1990:61). Some research (e.g., Davison 2001; Du Paul, Guevrenot & Barkley 1990; Walters & Barrett 1993) set estimates at 3% to 5%, while Biederman (1996:26) suggests that 10% of all children meet the diagnostic criteria. Estimates from Sears and Thompson (1998:7) fall within this category as they state that two million school-aged children in the US (at least 5%) are thought to have AD/HD. Mather and Goldstein (2001:46) as well as Young-Loveridge (1997:5) estimates that the figure is as high as 40%, while Clarke (2002b:) cites Cantwell (1996) in stating that this group constitutes 50% of child psychiatric populations. Alarmingly, Davison (2001:230) also reports that in some classrooms more than 40% of the children were medicated for AD/HD. Diller

(1998, in Mather & Goldstein 2001:47) and McNamara (1972) believe that as the tempo of society increases, a greater incidence of AD/HD may occur.

The incidence of AD/HD varies from country to country depending on the criteria used for diagnosis. In Australia the rate of incidence ranks between 8% and 12% (Serfontein 1994:9). A study conducted by the then Hyperactive Children's Support Group of South Africa (Ryan 1995) found that hyperactivity and inattention is believed to affect 10% of the South African population in every ethnic and socioeconomic group. The Attention Deficit Hyperactivity Disorder Association of South Africa for Children and Adults (ADHASA) does not have any recent research data on the number of children diagnosed or treated with AD/HD. Novartis, the company that distributes methylphenidate (Ritalin) in South Africa is unclear about the actual number of children taking Ritalin as this Schedule 7 drug is also prescribed for people presenting with narcolepsy. A recent study by Kokot in Tshwane (equal distribution in all socioeconomical strata) indicates that 51% of children experienced some problem in Grade 1, with the most indicated difficulties pointing to **concentration**, **focusing** and task completion (Kokot 2006: in press).

Even though a number of research studies indicate an increase in the prevalence in diagnosis of AD/HD, not all are convinced that this is indeed the case. A article in the *Journal of the American Medical Association* concludes that there is no evidence that AD/HD is overdiagnosed in our society (Goldman, Genel, Bezman & Slanetz 1998:1100), and child psychiatrist Peter Jensen from the National Institute of Health in the USA finds that less than one in eight children who meet the diagnostic criteria for AD/HD are taking medication (National Institute of Health [NIH], 1998).

According to Goldstein (1995), Spring, Yellin and Greenberg (1976), and Wechsler (1991), normative data from standard educational assessment tools do not support the hypothesis that children are increasingly less attentive or more impulsive. In fact, as a result of children's early exposure to the media, their capacity for sustained attention may have increased in the younger age groups. A more likely explanation for the increase in diagnoses reflects increasing community, professional, and parental awareness of the symptoms of AD/HD, leading to more children being referred, correctly identified, and offered appropriate treatments.

4.2 Gender

The prevalence of AD/HD in terms of gender distribution has also received a great deal of attention. According to the DSM-IV (APA 1994:81) and researchers such as Armstrong (1995), Barkley (1997), and Green and Chee (1994), the disorder is much more frequent in males than in females, with male-to-female ratios ranging from 4:1 to 9:1, depending on the setting (i.e., general population or clinics). Sears and Thompson (1998:7) generally support this ratio with the statistic that boys diagnosed with AD/HD outnumber girls by about 3:1, but in ADD without hyperactivity, the overall incidence is similar in both sexes. In younger children, however, both ADD and AD/HD are diagnoses (Sears & Thompson 1998:7). Lubar (1991:104) state that symptoms are between four and six times more prevalent in males than in females.

However, Barabasz and Barabasz (2000:25) hypothesise that this apparent differential prevalence rate may be erroneous. They concluded that, when males and females are compared to their same sex normative groups and appropriate controls are added for symptoms of hyperactivity and antisocial behaviours, the occurrence of AD/HD in males and females might be equal. Females with AD/HD present with problems in mood, affect and emotion, while showing considerably less difficulty with aggression than boys. Girls are more socially withdrawn than boys and show more internalising symptoms, such as depression and anxiety. The majority of children with AD/HD are referred to mental health clinics because of aggression and other forms of misbehaviour, which are more common in boys. Because of these biases and the earlier DSM-III R (APA 1987) diagnostic criteria favouring hyperactivity, girls may have been denied needed treatment more frequently than boys. Clearly, more than mere reliance on behavioural observation is needed if diagnosis of the disorder is to become more accurate (Barabasz & Barabasz 2000:28). Armstrong (1995:32) supports this notion and suggests that AD/HD might simply reflect normal gender differences. He argues that boys may be at a disadvantage compared with girls, because their behaviour clashes with "the expectations of a highly verbal, highly schedule-oriented, and usually female-dominated classroom environment" (Armstrong 1995:32).

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4.3 Familial Patterns

The patterns of prevalence of AD/HD in families are also well documented. Sears and Thompson (1998) report that AD/HD is more common (25%) in the first-degree biological relatives of children with AD/HD. If one identical twin has AD/HD, there is an 80% to 90% chance that the other twin will also have AD/HD (Sears & Thompson 1998:7). The genetic component seems to far outweigh the environmental component in AD/HD. Sears and Thompson add that inheritance will produce the AD/HD traits and that environmental influences may only affect how severe and persistent these traits are.

5. Recording and Diagnosing Procedures

This cursory glance at the historical search for a true definition of AD/HD, its comorbid factors, the ambiguity and the correlation between AD/HD symptoms and temperament illustrate the difficulties inherent in the accurate and responsible diagnosis of AD/HD. The fact that there is no laboratory or true clinical tests to diagnose AD/HD compounds this challenge. These issues alone add to the burden of accurately diagnosing AD/HD within a clinical setting.

It is noteworthy that the definition provided for this disorder, while continually being updated, has been purely behavioural; that is, professionals rely entirely on a defined constellation of subjective observed behavioural characteristics in the home, at school, and in social situations in order to diagnose the disorder. The people most affected by the person presenting with the disorder make these behavioural observations. The DSM-IV states clearly that no laboratory tests or specific physical features have been established as diagnostic criteria for the assessment of AD/HD (Sterman 2000:2). The DSM-IV notes that "tests that require effortful mental processing have been noted to be abnormal in groups of individuals with Attention-Deficit/Hyperactivity Disorder compared with control subjects, but are not yet entirely clear what fundamental cognitive deficit is responsible for this" (APA 1997:81). The only other references to non-behavioural diagnostic criteria are occasional minor physical anomalies (e.g., hypertelorism, highly arched palate, and low-set ears) that are found more often than in the general population, and a slightly higher rate of physical injury (APA 1997:81).

Officially, very specific and stringent recommendations exist about how to correctly and responsibly diagnose AD/HD. After reviewing a decade's research on AD/HD, Cantwell (1996) published the following recommendations in the *Journal of the American Academy of Child and Adolescent Psychiatry:*

- a) The diagnosis should begin with thorough interviews with anyone who acts as parents to the child. The goal is to establish, in detail, under what circumstances the presenting symptoms occur and to take a complete developmental, medical, and family history.
- b) The clinician should interview the child in order to elicit the child's view of the problem.
- c) The interview should include screening for other problems that might be the real source of difficulty, including other mental disorders.
- d) The child should be given a thorough medical examination to rule out neurological or sensory problems (poor hearing or eyesight) as the cause of the symptoms.
- e) The child should be given tests of intelligence and achievement.
- f) The clinician should evaluate questionnaires completed by the parents and teacher on observed behaviour.

In reality, few physicians report anything like this level of scrutiny before prescribing treatment (Levingston 1997:12). The research literature suggests that it is behavioural ratings by teachers and parents that are most often used to assign the AD/HD classification. The problem, of course, is that the decision about where to draw the line on inappropriate behaviour is a highly subjective decision.

According to Sears and Thompson (1998:57-60), any good assessment should contain the following multimodal approach:

• History

The core of any assessment is a detailed history. In order for the psychologist and other professionals to understand the person they see before them, it is necessary to hear what this person was like as an infant, toddler, and preschooler, and how they behaved in the foundational school phase. Indeed, it is even helpful to go back further and enquire about the pregnancy and birth



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and to gather information about the parents and grandparents. AD/HD symptoms that are currently present should be identified as well as the degree of the symptoms. The history should also cover risk factors such as depression or alcoholism in extended family members. The child's medical history is important. More frequently in the AD/HD population are birth difficulties (including not just prematurity but also post-maturity), allergies, head injuries, and frequent early ear infections that required antibiotics and may have led to the insertion of tubes.

Questionnaires

There are a number of ways to supplement the history. The most common is to use questionnaires, and these come in a wide variety for both parents and teachers to complete. Questionnaires nearly always rely on subjective observations made by others and thus tend to focus on the symptoms that are disturbing to parents and teachers and disruptive in social and academic situations.

Psychological testing

In addition to questionnaires, psychological testing (psycho-educational) is employed to assist in diagnosis. This testing includes individual IQ testing and academic testing. Sometimes it also includes tests that identify learning disabilities. The psychological testing usually does not include personality testing or projective techniques.

IQ Tests

An IQ test cannot be used to diagnose AD/HD because there is no single pattern that is unique to children with AD/HD. However, certain subtests are particularly affected by the ability to sustain attention. On the WISC-R (revised), children with attentional problems were more likely to show the ACID pattern (Sears & Thompson 1998:59). This acronym stands for the subtests Arithmetic, Coding, Information, and Digit Span. Children with AD/HD tend to obtain lower scores in these areas.

 Arithmetic involves mental problem solving, and these children do poorly because of their difficulty with working memory, in this case, keeping numbers in mind while they do calculations in their heads.

- Digit Span also involves attention and auditory memory. The child repeats strings of digits of increasing length both forwards and backwards. Repeating digits backwards strains the working memory.
- Coding involves copying a digit-symbol code for two minutes and requires sustained attention skills.
- The Information subtest involves questions that sample knowledge of subjects such as science, geography, and history, and it is the only subtest directly related to school learning. Information scores tend to be low in children with AD/HD because they do not pay attention to things they find boring.

On the WISC-III (currently in use), psychologists can calculate four index scores in addition to the verbal, performance, and full-scale scores. Of particular interest is the Freedom from Distractibility Index, which is calculated from the Arithmetic and Digit Span subtests. As noted above, these require good listening skills and working memory, so children with AD/HD may find them difficult. Processing Speed Index is also weak in some of these children. This index is calculated from scores on Coding and on a subtest new to the WISC-III called Symbol Search (Sears & Thompson 1998:60).

Academic tests

Tests for reading, mathematics, spelling and writing are usually done to supplement the information given by the teacher. Scores on standardised academic tests help determine whether children are underachieving in relation to their ability as measured by the intellectual tests (Sears & Thompson 1998:60).

Tests for learning disabilities

Children with AD/HD frequently also have specific learning disabilities, separate from AD/HD. Depending on which study is being cited, the figure for children with AD/HD having coexisting language disabilities is anywhere from 30 percent to 80 percent (Sears & Thompson 1998:60).

Comment

The above literature clearly states that there is no single test or laboratory procedure to reliably diagnose AD/HD, although many factors are indicated in the cluster of behaviours that are symptoms of AD/HD. For this reason a multidimensional approach may be more appropriate in assisting children struggling with attentional difficulties and heightened motor activity.

6. Associated Features and Comorbid Disorders

As previously stated, children with AD/HD seldom present with attentional difficulties, impulsivity and hyperactivity in isolation. Associated features vary depending on age and developmental stage and may include demoralisation, low frustration tolerance, mood lability, temper outbursts, bossiness, dysphoria, stubbornness, excessive and frequent insistence that their requests be met, rejection by peers, and poor selfesteem. Inadequate self-application to tasks that require sustained effort is often interpreted by others as indicative of a poor sense of responsibility, lasiness, and oppositional behaviour. Family relationships are often characterised by resentment and antagonism, especially because variability in the individual's symptomatic status often leads parents to believe that all the troublesome behaviour is willful. Individuals with AH/HD may obtain less schooling than their peers and have poorer vocational achievement. Academic achievement is often impaired and devalued, typically leading to conflict with the family and school authorities. Children may be placed in academic settings that are inappropriate for their intellectual ability. These behaviours must be distinguished from similar signs in children with AD/HD. In children with mental retardation, an additional diagnosis of AD/HD should be made only if the symptoms of inattention or hyperactivity are excessive for the child's mental age. Inattention in the classroom may also occur when children with high intelligence are placed in academically understimulating environments.

AD/HD must also be distinguished from difficulty in goal-directed behaviour experienced by children from inadequate, disorganised, or chaotic environments (APA 1997:82-83). In its severe form, the disorder significantly impairs social, familial, and scholastic adjustment. A substantial proportion of children referred to clinics with

AD/HD also have Oppositional Defiant Disorder or Conduct Disorder. There may be a higher prevalence of learning disorders, anxiety disorders, mood disorders, and communication disorders in children with AD/HD. This disorder is not infrequent among individuals with Tourette's Disorder; when the two disorders coexist, the onset of AD/HD often precedes the onset of the Tourette's Disorder. There may be a history of child abuse or neglect, multiple foster placements, neurotoxin exposure (e.g., lead poisoning), infections (e.g., encephalitis), drug exposure in utero, low birth weight, and mental retardation (APA 1997:80-81). A discussion on some of the comorbid factors follows.

6.1 AD/HD and Behavioural Problems

AD/HD is not diagnosed if the symptoms are better accounted for by **another mental disorder** (e.g., a mood disorder, anxiety disorder, dissociative disorder, personality disorder, personality change due to a general medical condition, or a substance-related disorder). In all these disorders, the symptoms of inattention typically have an onset after age seven, and the childhood history of school adjustment generally is not characterised by disruptive behaviour or teacher complaints concerning inattentive, hyperactive, or impulsive behaviour. When a mood disorder or anxiety disorder co-occurs with AD/HD, each should be diagnosed individually. AD/HD is not diagnosed if the symptoms of inattention and hyperactivity occur exclusively during the course of a pervasive developmental disorder or a psychotic disorder (APA 1997:81).

Attention deficit disorder without hyperactivity is sometimes referred to as ADD (Barkley cited in Lubar & Lubar 1999:103). This subtype tends to overlap more with anxiety disorders and learning disabilities. Attention deficit disorder with hyperactivity, ADD+, also overlaps with learning disabilities, but is more likely to co-occur with oppositional defiant disorder (ODD) and conduct disorder (CD) (Barkley 1990:103-104).

ODD is diagnosed behaviourally when the individual, often a child, purposely does not follow rules, does not see the significance of them, and therefore does not believe they are important. If this is not corrected or treated, it can evolve into a conduct disorder. Conduct disorder is more severe and tends to involve more premeditative or purposefully destructive behaviours than ODD. If the conduct disorder is not brought under control, especially in late adolescence and early adulthood, it can evolve into a much more serious disorder known as antisocial personality disorder. Many individuals who have antisocial personality disorder spend part of their lives in prison, as do some with conduct disorder. When only oppositional defiant disorder is present, individuals usually do not reach the point in the legal system at which they are jailed (Barkley 1990:104). At least 80% of adolescents with AD/HD continue to manifest symptoms consistent with AD/HD into adulthood, and 60% develop at least one additional disruptive disorder. Between 20% and 60% of adolescents with AD/HD are involved in delinguent behaviour, as compared with the typical occurrence of 3% to 4% of adolescents without AD/HD. At least 50% to 70% of these adolescents develop oppositional defiant disorder, often prior to adolescence, and a significant number develop a conduct disorder (Barkley, Fischer, Edelbrock & Smallish 1990:546). For some individuals, the primary symptoms of AD/HD may diminish in intensity by adolescence; however, most adolescents with AD/HD continue to experience significant problems.

There is a high degree of overlap between AD/HD and depression. Some children with AD/HD have movement or tic disorders ranging from benign tics to the more severe Tourette's syndrome. Occasionally they may have a seizure disorder, and there is a higher incidence of substance abuse in the AD/HD population than in the non-AD/HD population (Barkley 1990:104).

Apart from comorbid diagnoses on the mood or behavioural spectrums, comorbidity with academic problems is also often found with the typical AD/HD child. Depending on the diagnostic criteria, approximately 20% to 30% of children with AD/HD also suffer from a concomitant, often language-based, learning or reading disability (Mather & Goldstein 2001:57).

6.2 AD/HD and Language Disorders

Research suggests that the comorbidity of language disorders with AD/HD merits routine screening of children suspected of AD/HD and language disorders, especially during their younger years. Children with both these disorders appear to have a

much poorer prognosis than those with AD/HD alone (Mather & Goldstein 2001:57). One may surmise that the language skills of children with AD/HD may be underdeveloped because they do not attend to their environment effectively. Another hypothesis may be that these children exhibit AD/HD symptoms because they lack efficient language skills necessary for behavioural self-control. Several researchers have addressed this issue and concluded that the symptoms of AD/HD and language disability develop and are maintained independently. Nonetheless, children who show impulsivity combined with a language disorder are more likely to exhibit behavioural problems (Mather & Goldstein 2001:57).

6.3 AD/HD and Learning Disabilities

The overlap of both AD/HD and specific **learning disabilities** (LDs) is as high as 70% (Hynd, Marshall & Gonzales 1991:283-296). The Lubars also report on the fact that there are supposedly more than 250 different kinds of LDs; and in some cases, AD/HD and LD overlap with **conduct** problems.

Although AD/HD may prevent children from achieving their academic potential, the presence of a LD may make some children appear more inattentive than others. Students with AD/HD often experience specific weaknesses within their processing areas that contribute to poor school performance. Both auditory and visual memory problems are common among these students, whereas weaknesses in motor memory are relatively rare (Goldstein & Goldstein cited in Mather & Goldstein 2001:57).

Barkley (1981, 1990, 1995), a well known researcher in the field of AH/HD, argues that although there was a tendency in the mid-1970s to described children with AD/HD as intellectually less competent than their peers, poor performance on intellectual tasks may rather result from the impact of impulsivity and inattention on task-taking behaviour. In addition, due to the fact that many children with AD/HD also have a LD, they often under-perform or underachieve during the primary school years. During high school, at least 80% of these children fall behind in a basic academic subjects requiring repetition and attention for competence, such as basic

mathematical knowledge, spelling, or written expression (Mather & Goldstein 2001:57).

6.4 AD/HD and Social Difficulties

Sociometric and play studies suggest that children with AD/HD are not chosen as often as peers to be partners in activities or best friends. An awareness of their difficulties may precipitate lower self-esteem (Mather & Goldstein 2001:57). They appear to experience either high incidence-low impact problems that result in poor social acceptance or low incidence-high impact problems that result in social rejection. Children with AD/HD also have difficulty adapting their behaviour to different situational demands. The impulsive behavioural patterns of children with AD/HD are more responsible for their social difficulty, rendering those with concomitant hyperactive-impulsive problems at an even greater risk of developing social difficulties (Pelham & Bender 1982:365).

From an educational perspective, the concept of **attention** as an executive or foundational skill required for classroom success has increasingly gained popularity. Sustained mental effort, planning, execution, self-regulation, and task maintenance are considered measures of executive functioning as reported by Mather and Goldstein (2001:47). Ability to **adequately pay attention** and **inhibit motor activity** may ultimately result in better academic achievement and pleasing social behaviour.

Studies also suggest that there is a higher prevalence of mood and anxiety disorders, learning disorder, substance-related disorders, and antisocial personality disorder in family members of individuals with attention-deficit/hyperactivity disorder (APA 1997:82).

7. Possible Causes of AD/HD

7.1 Biology and Environment

Although some have argued that the problems of AD/HD may in part represent a cultural phenomenon (Block 1997:236), others suggest that AD/HD is a disorder in

which the severity of the child's problems reflects the interaction of temperamental traits and the environmental demands placed on the child. The symptoms of AD/HD may reflect an interaction of biology and environment (nature and nurture). Biology appears to determine the risk for problems. The type of classroom environment, however, is likely to affect the number of teacher complaints and the severity of reported problems. In addition, although culture makes a difference in terms of the expectations and tasks placed on children in educational settings, children worldwide with AD/HD demonstrate a fairly homogeneous set of symptoms (Mather & Goldstein 2001:47).

The DSM-IV states that AD/HD is known to occur in various cultures, with variation in reported prevalence among Western countries probably arising more from different diagnostic practices than from differences in clinical presentation (APA 1994:81).

7.2 Genes

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A number of genes and types of gene code for brain chemistry appear to place individuals at risk for developing AD/HD. The hypothesis of genetics as a contributor to AD/HD is powerfully reinforced through the findings of concordance for the disorder among identical twins (Edelbrock, Rede, Plomin & Thompson 1995:776; Zahn-Waxler, Schmitz, Fulker, Robinson & Ende 1996:103). Evidence from Kenneth Blum and his colleagues (Miller & Blum 1995; Noble, Blum, Ritchie, Montgomery & Sheridan 1991) suggests there is a strong genetic component in a whole constellation of problems located on chromosome 11. These researchers identified the series of alleles associated with AD/HD that lie close to another set associated with alcoholism, other addictions, and Tourette's syndrome. They refer to the whole constellation of problems as the "reward deficiency syndrome". The researchers postulate that this may be the reason why these disorders often occur together as comorbidities. In their evaluation of children with AD/HD, they found that manifestations of this disorder were commonly traceable back to between one and three generations (Lubar 1991:204).

This theory is summarised by Mather and Goldstein (2001) and Ingersoll and Goldstein (1995) when they state that genetics may set the stage for a possible risk

and that life experience can determines whether an individual ultimately receives a diagnosis of AD/HD. McCandless (2003:20) summarises the situation succinctly in her assertion that genetics loads the proverbial gun and that environment pulls the trigger.

7.3 The Brain

Researchers have identified areas in the prefrontal cortex, basal ganglia, and cerebellum that are important in helping individuals to self-regulate. These structures have been referred to as the brain's "braking systems". A large body of research demonstrates **structural (anatomy)**, **biochemical**, and **physiological** differences in the brains of individuals with AD/HD. Although results from these group studies have helped to develop an understanding of the causes of AD/HD, these types of findings to date have not been helpful in making the diagnosis of AD/HD in individual children (Mather & Goldstein 2001:53). This subject will be explored in detail in chapter 3.

7.4 Other General Causes

A disease, illness, or other disorder may also cause AD/HD. Genetic disorders, such as Fragile X syndrome, neurofibromatosis, Turner syndrome, Noonan syndrome, and Williams's syndrome, are all chromosomal and genetic disorders in which AD/HD has been reported (Hagerman 1991:4). Disorders resulting from fetal alcohol syndrome (FAS), cocaine exposure in utero, lead poisoning, vapour abuse, perinatal complications, certain medical problems (e.g., hypothyroidism, encephalitis), and radiation therapy secondary to leukemia have all been reported as responsible for creating problems with **inattention** and **impulsivity**. There are reported cases of children experiencing specific medical conditions, such as pinworm or thyroid problems, who demonstrate a sudden onset of inattention and hyperactive classroom behaviour. These aspects will also be explored in detail in chapter 3.

8. Developmental Course of AD/HD

An increasing body of scientific data has been generated on the development course and adult outcome of children with AD/HD. AD/HD in isolation appears to best predict school struggles, difficulty meeting expectations in the home, and possible mild substance abuse in adulthood. It does not predict, however, the significant negative emotional, behavioural, and personality outcomes that have been reported (Mather & Goldstein 2001:53).

8.1 Early Symptoms

Infants who demonstrate difficult temperament traits do not handle changes in routines well and exhibit a low frustration threshold and a high intensity of response. In follow-up studies of such infants as many as 70% develop school problems. These infants also appear to be at greater risk than others for receiving a diagnosis of AD/HD and show more negative relationships with caregivers – a relationship that is critical in predicting a child's life outcome (Mather & Goldstein 2001:53).

Although early symptoms of AD/HD may be viewed as transient problems experienced by most children, research data suggest that ignoring these early signs could result in the loss of valuable treatment time. At least 60% to 70% of children later diagnosed with AD/HD showed clear symptoms during the preschool years. Young children manifesting symptoms of AD/HD are more likely to have language problems and develop a wide range of behavioural problems than are children who do not have these symptoms (Mather & Goldstein 2001:56).

The DSM-IV however warns that although the symptoms are clearly identifiable at an early age, caution should be taken in making this diagnosis in early years (APA 1994:82).

8.2 Adolescents and Adulthood

In the majority of cases seen in clinical settings, the disorder remains relatively stable through early adolescence. In most individuals, symptoms attenuate during late adolescence and adulthood, although a minority experience the full complement of symptoms of AD/HD into mid-adulthood. Other adults may retain only some of the symptoms, in which case the diagnosis of AD/HD, in partial remission, should be used. This diagnosis applies to individuals who no longer have the full disorder but

still retain some symptoms that cause functional impairment (APA 1997:82). The question arises of whether these individuals "outgrow" their symptoms or merely learn to cope with them.

The developmental course and prognosis of AD/HD is influenced by the extent to which it affects a person's life. The following effects have been identified (Amen 2001:xvii; Sears & Thompson 1998:7):

- 35% of children diagnosed with AD/HD never finish school (25% repeat at least one grade)
- 52% of untreated teens and adults abuse drugs or alcohol
- 19% smoke cigarettes compared to 10% of the general population
- 43% of untreated hyperactive boys will be arrested for a felony by age 16
- 50% of inmates in a number of studies have been found to have ADD (75% in one study)
- 75% have interpersonal problems
- untreated ADD sufferers have a higher percentage of motor vehicle accidents, speeding tickets, citations for driving without a license, and suspended or revoked licenses
- they have more medical and emergency room visits than people without AD/HD
- parents of ADD children divorce three times more often than the general population

9. Traditional Treatment

Attentional difficulties and hyperactivity are traditionally treated with prescription drugs, usually psychostimulants such as **methylphenidate** (Ritalin), and behavioural modification; but while these medications and therapeutic interventions may suppress symptoms, they do not address possible underlying causes. The dramatic increase in the use of Ritalin is a concern. Since 1990, the use of Ritalin has risen two and a half times. In the USA, more than 1,3 million children take Ritalin regularly (Stevens 2000:20). Currently, there are two other newly introduced medications on the market, namely, **Concerta** and **Strattera**. As they have only very recently been

introduced into South Africa, a consideration of these medications will not be included in this thesis.

9.1 Psychostimulants

In the 1930s, Charles Bradley (1937), director of the Emma Pendleton Bradley Home in Rhode Island, demonstrated the effectiveness of the stimulant Benzedrine in improving problematic behaviours in children with minimal brain dysfunction (MBD) (Bradley 1937). In 1957, Maurice Lauffer, second director of the Bradley Home, suggested a new name for MBD, focusing on an aspect of this broad diagnosis that responded very well to medication: 'hyperkinetic disorder'. Lauffer claimed that "a favourable response to amphetamine is supportive evidence for a diagnosis of the hyperkinetic syndrome" (Lauffer & Denhoff 1957:473). This statement has huge implications and caused great controversy surrounding the use of amphetamine such as Ritalin. As clearly stated by Garber, Garber and Spizman (1996:27): "The fact that your performance improved when you take Ritalin doesn't mean that you are AD/HD. The fact that you do not respond to Ritalin doesn't mean that you are not AD/HD." Despite this warning, many physicians use the drug as a diagnostic tool - in other words, if amphetamines (Ritalin) seem to improve attention, the patient is assumed to have AD/HD. Studies conducted during the mid-seventies and early eighties by Judith Rapaport of the National Institute of Mental Health clearly show that stimulant drugs improve the performance of most people on tasks requiring good attention, regardless of whether they have a diagnosis of AD/HD (Levingston 1997:3).

9.1.1 Psychostimulant classification

Ritalin is classified as psychostimulant medication. Amphetamine was synthesised in the late 1920s and was introduced into medical practice in 1936. Dextroamphetamine is the major member of the class, although many other amphetamine (Methedrine, "speed"), phenmetrazine (Preludin), and methylphenidate (Ritalin), were subsequently introduced (Katzung 1989:387). Psychostimulants fall under the classification of centrally acting sympathomimetics. The *South African Medicines Formulary* (SAMF) (2003:449) states "this group includes the amphetamines (prohibited in South Africa), methylphenidate and pemoline". Pemoline has been

withdrawn in several countries because of numerous reports of acute, sometime fatal, liver failure. This publication also states that this class of medication was used primarily in the past for its altering and euphoriant effects, and these agents have no place nowadays in medical practice, except for treating narcolepsy and AD/HD in children. As they are dependence forming, they should not be used for the relief of fatigue or in the treatment of depression (SAMF 2003:449).

9.1.2 Chemical working of stimulant medication

With every thought or action produced by the brain, messages travel from one nerve to another, telling the brain what to do. The messages are carried by neurotransmitters, chemicals such as norepinephrine, dopamine and serotonin, which are secreted at the junction between brain cells (synapse) to facilitate transmission of messages. Stimulant drugs are thought to increase or stimulate the secretion of specific neurotransmitters or to prevent the re-uptake of these neurotransmitters to make them available for a longer period of time. Barkley writes, "By increasing how much of these chemicals are available in the brain, the stimulant increases the action of these brain cells, which seem to be those most responsible for inhibiting behaviour and helping us stick to something we are doing" (cited in Sears & Thompson 1998:230).

Many highly addictive drugs are stimulant drugs, producing excitement, alertness, elevated mood, decreased fatigue, and sometimes increased motor activity. Stimulant drugs increase activity at dopamine receptors, including those in the nucleus accumbens. Receptor types D2, D3 and D4 are apparently more important for the reinforcing effects, whereas type D1 is more important for the increased motor activity (Xu Hu, Cooper, White & Tonegawa 1996:70).

Methylphenidate (Ritalin) and cocaine both block the reuptake of dopamine in receptors in the brain, but the reaction time in the two substances differs. When people take a methylphenidate pill, its concentration in the brain increases gradually over an hour and then declines with a half-life of more than an hour and a half, in contrast to cocaine, which produces a four to five times faster rise and fall of effects. Therefore, methylphenidate pills do not produce the sudden rush of excitement, the

strong withdrawal effects, the cravings, or the addiction that are common with cocaine. In addition to its effects on dopamine, methylphenidate increases serotonin release. The benefits for people with AD/HD probably include improved attention due to the dopamine effects and a calming of activity due to serotonin effects (Gainetdinov, Wetsel, Jones, Levin, Jaber & Caron, 1999:397).

Stimulant drugs are known primarily for their short-term effects, but repeated use of high doses may produce long-term, even permanent disruption of brain functioning. Cocaine users suffer lasting changes in brain metabolism and blood flow, thereby increasing their risk of stroke, epilepsy, and memory impairments (Strickland, Miller, Kowell, & Stein 1998:1). The drug methylenedioxy-methamphetamine (MDMA or "ecstasy") stimulates the release of dopamine at low doses. At higher doses it also stimulates serotonin synapses, producing hallucinogenic effects similar to those of lysergic acid diethylamide (LSD). Unfortunately MDMA not only stimulates axons that release dopamine and serotonin, it also destroys them (McCann, Lowe & Ricaurte 1997:399), as shown in figure 1 (at least it does so in rats and monkeys; and it may only be assumed that it does the same in humans).

In monkeys the toxic effects of the chronic amphetamine use include damage to cerebral blood vessels, neuronal loss, and microhemorrhages (Breggin 2001:63). Other animal studies indicate that amphetamines can cause constriction of brain vessels, increase the likehood of convulsive seizures, and influence carbohydrate metabolism in an unknown way. They also alter the output of growth hormones, and affect the sensitive biochemical balance in the brain and the central nervous system. Some researchers in the field (e.g. Walker 1999) are of the opinion that stimulant drugs may well produce greater harm in the long term than the hyperactive symptoms they are meant to control (Breggin 2001:63).



Figure 1 Brain damage produced by MDMA

These slices through monkey brains have been stained with a chemical that makes axons containing serotonin white. Photos in the top row are from a normal monkey; those below are from a monkey that was exposed to MSMA ("ecstasy") a year and a half earlier. Notice the decreased density of serotonin axons in the photos above (McCann, Lowe & Ricaurte 1997)

Cocaine and LSD are discussed here because these substances belong to the same family as Ritalin. These substances differ in only one or two strains in the generic make-up, making them very closely related to each other.

Because dopamine is mostly an inhibitory neurotransmitter, drugs that increase activity at dopamine synapses decrease the activity in much of the brain. Figure 2 shows the results of a PET scan, which measures relative amounts of activity in various brain areas (London et al. 1990:567). The question might be asked as to how drugs that decrease brain activity lead to behavioural arousal. One hypothesis is that

high dopamine activity mostly decreases "background noise" in the brain and therefore increases the signal-to-noise ratio (Mattay et al. 1996:4816). Amphetamines are thought to excite neurotransmitters, which stimulate the areas that are underactive in AD/HD. There are numerous studies that demonstrate performance improvements both in persons who have AD/HD and in normal controls (Solanto, Arnsten & Castellanos 2001:303-331). Stimulant medications have been shown to increase blood flow in the dorsolateral prefrontal cortex and posterior parietal cortex during tests of spatial working memory. Complex working memory tasks and sustained attention may also improve with stimulant medication. Tests of cognitive flexibility in normal volunteers have shown that methylphenidate (40mg) may facilitate the shifting of attention toward newly relevant stimuli, although they may increase mean response latencies (Thompson & Thompson 2003:117). Stimulant medications may also reduce the rate of both omission and commission errors on continuous performance tests (Thompson & Thompson 2003:117).



Figure 2 Effects of cocaine on the brain

These PET scans show that the brain has a lower metabolism and lower overall activity under the influence of cocaine than it has ordinarily. Red indicates higher activity, followed by yellow, green and blue. A and B represent brain activity under normal conditions; H and I show activity after a cocaine injection (London et al. cited in Kalat 2001:71).

9.1.3 Comment

It can thus be concluded that stimulant medication has a twofold effect, namely, to increase the secretion of certain neurotransmitters (dopamine, norepinephrine and serotonin), as well as to prohibit the reuptake of these neurotransmitters, thus making them available for longer.

Some researchers believe that movement produces dopamine in much the same way as it increases serotonin and other endorphins in the brain (http://en.wikipedia.org/wiki/Endorphin). However, this possibility has not typically been capitalised upon in therapeutic programmes.

9.1.4 Safety and common side effects

9.1.4.1 Safety

The U.S. Drug Enforcement Administration (DEA) carefully regulates the prescription of Ritalin and other stimulant medication and lists the drug as a schedule two (the same as morphine). In South Africa, Ritalin is listed as a schedule seven drug (highly regulated). Generally the drug is considered at high risk for abuse. Doctors in the USA who prescribe it are required to obtain an extensive narcotic license, renew it every two years, and write the prescription on special triplicate prescription pads provided by the DEA. The doctor, the pharmacy and the DEA each retain a copy. To further avoid 'prescribing abuse', the doctor is limited by law to prescribing a one-month supply only, and children must be re-evaluated every month (Sears & Thompson 1998:232) in order to re-establish the need for medication.

In 1987, Copeland and her colleagues reported in *Developmental and Behavioural Pediatrics* that most paediatricians do not adequately monitor the medication of their AD/HD patients once they have prescribed the medication (Levingston 1997:12).

The controversy surrounding the safety of the use of stimulant medication was propelled into the limelight during the 1980s, when several lawsuits were filed by parents contending that Ritalin had harmed their children (Garber, Garber & Spizman 1996:9). This controversy is clearly not one of the past, as on 16 February 2006, an FDA advisory panel advised that attention deficit hyperactivity disorder drugs should

carry "black box" warnings about an increased risk of sudden death and serious cardiovascular problems. The panel recommended warning labels for methylphenidate drugs -- sold as Ritalin, Concerta, Methylin and Metadate, as well as the amphetamines Adderall and Adderall XR. The panel's recommendation comes after the public release of a 2004 FDA report stating 25 people taking ADHD drugs died suddenly between 1999 and 2003, and 43 people taking the drugs experienced serious cardiovascular events such as strokes, cardiac arrest and heart palpitations. Children accounted for 19 of the deaths and 26 of the nonfatal cardiovascular conditions

(http://www.medicalnewstoday.com/medicalnews.php?newsid=37631&nfid=rss feeds).

The following section will provide an outline of the indications and contraindications for medication, specifically Ritalin, as well as possible side effects:

Indications

- Attention deficit hyperactivity disorders in children (no general agreement about its role in adolescents and adults)
- Narcolepsy

Contraindications

- Absolute history of schizophrenia, drug dependency or personality disorders; patients with glaucoma, thyrotoxicosis, anxiety, tension or ischaemic heart disease
- Relative hypertension, vocal or motor tics, epilepsy
- *Paediatrics*: Not recommended for children under six years of age or in any children without thorough neuropsychiatry assessment
- Adverse effects: Most commonly nervousness and insomnia. Weight loss and growth retardation may occur (particularly in children receiving > 30mg/day for prolonged periods); changes in blood pressure and pulse rate, nausea, drowsiness, dyskinesia, tremor, skin rash and dependency, especially in predisposed individuals (SAMF 2003:450).

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Sears and Thompson (1998:233) listed the following contrainductions in a practical list:

- 1. Any significant cardiac (heart) problem
- 2. Hypertension (high blood pressure)
- 3. Any family history of psychosis
- 4. Any family history of tics or Tourette syndrome
- 5. Significant symptoms of anxiety
- 6. Significant symptoms of depression
- 7. Any history of substance abuse
- 8. A seizure disorders
- 9. Simultaneous intake of the drug clonidine (Catapres)

9.1.4.2 Special prescriber's points

- Growth in children should be monitored (both height and weight gain), especially where doses exceed 30 mg/day for prolonged periods.
- Medication-free periods (e.g. over weekends or during school holidays) are recommended to determine the need for continued therapy.
- Owing to the stimulatory effects on the central nervous system, the drug should usually not be taken after 13h00 to reduce the risk of insomnia, although afternoon doses may sometimes be required (SAMF 2003:450).

9.1.4.3 Common side effects

The most common side effects associated with Ritalin are (Whalen & Henker 1991:126):

- 1. Lowered self-esteem due to taunting by peers
- 2. Stunting of growth
- 3. Insomnia
- 4. Poor appetite leading to malnutrition
- 5. Tics
- 6. Cardiovascular problems
- 7. Potential development of Tourette's syndrome
- 8. Cognitive impairment with high dosages
- 9. A small, but significant, number of cases show negative physiological side effects that do not diminish over time despite cessation of the medication

9.1.5 Limitations of psychostimulant medication

Psychoactive drugs used to treat AD/HD have many potential side effects, and 25% to 40% of those treated with methylphenidate are unresponsive (Swanson et al. 1993:154). In children younger than six and in adults, the response to stimulant medication is positive in only about 50% of cases. In school-aged children there is about a 75% positive response rate in trials of a stimulant drug (Thompson & Thomson 2003:117). Another limitation of stimulant medication is that children on stimulants become more responsive to punishment and less responsive to reward (Anderson, Barabasz, Barabasz & Werner 2000:52) - a situation that lays a weak foundation for adaptive learning and well-adapted social behaviour.

Stimulant medication has been shown to be effective for short-term but not long-term improvement of symptoms (Sears & Thompson 1998:230). In an extensive review of the long-term effects of stimulant medication on AD/HD children, James Swanson's team at the University of California, Irvine, reported that stimulant medications show "a short-term benefit for the management of behavioural symptoms of inattention, impulsivity, and hyperactivity; and a lack of demonstrated long-term effects on learning, achievement, or social adjustment" (cited in Sears & Thompson 1998:230). One problem with symptom management interventions is that when the medication and/or behaviour modification is stopped, AD/HD pre-treatment or baseline symptoms and level of dysfunction return (Barabasz & Barabasz 1995:1).

Because the medical profession views AD/HD as a non-curable disorder, the use of medication is necessarily long-term. The goal is to pharmacologically alter the distribution of neurotransmitters, especially dopamine and norepinephrine in the brain, and thus maintain the appropriate behaviours over a long period of time. Unfortunately, pharmacological treatments do not seem to have a long-term carryover effect. If a child has been on Ritalin for 10 years and an attempt is made to reduce and ultimately discontinue the medication, it is likely that the improved behaviours will continue to be maintained for approximately three months only (Whalen & Henker 1991:126).



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9.2 Behavioural Modification

Whereas Ritalin is the drug of choice of medical professionals, behavioural modification is the therapeutic intervention of choice by psychologists when dealing with AD/HD children. Behavioural modification may be effective when both parents and teachers work together consistently and continuously. Unfortunately, trained behaviours do not generalise to new situations, nor do they generalise to non-trained behaviours (Anderson et al. 2000:51).

A feature of this old but widely used approach is the collaborative involvement of both parents and teacher. Therapists train parents to use tokens, positive attention to appropriate behaviours, and time out or other punishment for non-compliant behaviour (Barabasz & Barabasz 2000:30). Teachers can use classroom contingency management, verbal praise and other rewards for appropriate behaviour, and withdrawal of privileges or punishment for undesirable behaviour.

Limitations of behavioural modification include the following:

- a) Not all children respond to the treatment.
- b) There is no carryover to the classroom of behaviour learned only with parents, and vice versa.
- c) Post-treatment behaviours rapidly return to baseline/pre-treatment levels upon cessation of the interventions.
- d) Fifty percent of patients discontinue it because of its complexity.
- e) Success is dependent on the cooperation of both parents and teacher. (Barabasz & Barabasz 2000:30-31).

9.3 Cognitive-behavioural Therapy

In 1971, Meichenbaum and Goodman published an article in the *Journal of Abnormal Psychology* entitled "Training impulsive children to talk to themselves: a means of developing self-control." In this and other articles that followed, the method of **cognitive-behavioural therapy** was discussed as first changing what a person thinks and feels, which in turn leads to changes in overt behaviour. These are commonly referred to as **stop**, **look** and **listen** techniques (Garber et al. 1996:146).

Cognitive-behavioural therapy has greater flexibility than behaviour modification and /or medication but, so far, has failed to demonstrate any lasting effects with children with AD/HD (Conte 1991:60). However, it may still hold promise because the addition of hypnosis might make it more viable. Kirsch, Montgomery and Sapirstein's (1995:214) meta-analysis showed that the addition of hypnosis to cognitive-behaviour therapy greatly improved treatment outcomes and long-term effects for a variety of disorders. However, the addition of hypnosis to cognitive-behavioural therapy for AD/HD has yet to be fully investigated.

9.4 Feingold Diet

In 1975 Benjamin Feingold suggested that the additives in food influenced children's behaviour. He specifically referred to hyperactivity, inattentiveness and other symptoms of AD/HD. This approach was extended over time to include all artificial flavorings, food colourings, and preservatives. Garber, Garber and Spizman (1996:146) state that there is, unfortunately, little research evidence to support Feingold's initial claims. The contribution of nutritional factors to attentional difficulties and heightened motor activity is discussed in more detail in chapter 4.

10. Conclusion

AD/HD is a condition that has received much attention. Huge amounts of money have been used to research the subject and thousands of articles and books have been published on the topic, yet it remains a heated subject with many unanswered questions. Many researchers believe that AD/HD is not curable, with others trying to prove that the symptoms are not conclusive evidence of a mental disorder.

Medication and behavioural modification strategies have largely aimed at alleviating the symptoms of AD/HD rather than treating the underlying cause. The following chapters will focus on the possible underlying causes of the symptoms of AD/HD and will document the findings within an ecosystemic approach.

Chapter 3

Theoretical Conceptualisation of Attention and Motor Activity

1. Introduction

From the previous chapter it emerged that AD/HD may possibly be illusive. Does it exist only because of the fact that we can compare behaviour to the criteria as set by the DSM-IV? Is there enough evidence to suggest that there is a definite neurological dysfunction? When is attention ineffective and motor activity inappropriate?

To be able to answer these and other questions surrounding the topic, it is necessary to explore the fundamental conceptualisation of attention and motor activity. A rational point of departure in answering these questions is the central nervous systems (CNS) and its involvement in attention and motor activity, and more specifically, the brain's role. This framework may assist in formulating possible solutions and methods of intervention as the development of the CNS and specifically the brain follows a basic pattern through the use of movement. The CNS also requires specific substances (nutrients) to assist in its optimal development and maintain optimal performance. When deviations in this fundamental developmental process are detected and the result points to attentional difficulties and heightened motor activity, a possible solution presents itself. Could a neurodevelopmental programme based on movement address attentional difficulties and heightened motor activity? Could nutritional supplementation assist attention and motor activity? This chapter investigates these issues to form a framework for understanding the constructs that form the cornerstones of the diagnosis of AD/HD, namely attention and motor activity.
2. Historical Background

The history of the efforts to unravel the source of human consciousness is well documented in Robbins' (2000:9-28) book: *A Symphony in the Brain*. This history goes back thousands of years and a brief summary is presented here.

Hundreds of ancient skulls with carefully drilled holes have been found in a variety of places around the world. Even though humankind explored the content of the cranium cavity, the Egyptians reserved the **heart** as the dwelling place of the human soul (for most of the human history, a cardiocentric view has dominated). Writings between 460 and 379 B.C. indicate that Hippocrates might have been the first persuasive proponent of the idea that the **brain** is the source of human intelligence. Building on the work of two of his teachers, Alcmaeon and Anaxagoras, he had the notion that epilepsy was the result of a disturbance in the brain. He believed that the grey matter was also the source of many other human experiences:

Men ought to know that from nothing else but the brain come joys, delights, laughter and sports and sorrows and grief, despondency, and lamentations. And by this, in an especial manner, we acquire wisdom and knowledge, and see and hear and know what are foul and what are fair, what are bad and what are good, and what are sweet and what are unsavoury. And by the same organ we become mad and delirious, and fears and terrors assail us. All these things we endure from the brain, when it is not healthy. In these ways I am of the opinion that the brain exercises the greatest power in the man. This is the interpreter to us of those things, which emanate from the air, when the brain happens to be in a sound state.

(Cited in Robbins 2000:11)

Hippocrate's view, however, was unique among his peers, too far ahead of its time to be taken seriously. Even Aristotle, who came along several decades later, was a primary proponent of the heart-centred human.

Galen, a physician to Roman gladiators and emperors in the second century, played a major role in the evolution of early thought about the brain. His **cell doctrine** reigned for fifteen hundred years, largely because, from the fourth through to the fourteenth century, the church banned study of the human body. In the seventeenth century, Thomas Willis, an English physician, published a thorough text on the anatomy of the brain in which he claimed that the brain itself, and not the ventricles, controlled memory and volition. His work sparked a new way of thinking and would later convince researchers to abandon the cell doctrine. Yet the cell doctrine survived for years after Willis's findings. René Descartes, the influential seventeenth-century French philosopher, is one of the most dominant early figures in the study of human behaviour. Descartes promoted the idea of **dualism**, the idea that mind and body are separate. Descartes chose the pineal gland as the place where the spirit entered the body, because it occupied a central place in the brain, because it was near the senses, and because it was surrounded by cerebrospinal fluid, which was still believed to be the liquid version of the animal spirits that allowed the body to move. Descartes' interpretation was the first attempt to assign a **specific task to a specific part of the brain**.

After the invention of the microscope, brain research developed rapidly. Chemical dyes, created for the textile industry, were used to dye slices of brain tissue for study under the newly invented instrument. The microscope lent itself to the next evolutionary step in thinking about the brain, the school of **localisation**. Researchers looking at cross sections of brain tissue noticed that different parts of the brain had different types and numbers of cells and asked whether this difference in structure – the cytoarchitecture – pointed to a difference in function.

Localisation gained substantial scientific support in 1861 as a result of the research of the respected French physician Paul Broca. Broca worked with a stroke patient who seemed to hear clearly but could only answer any question asked with a single word: "tan". After the patient died, Broca removed his brain and found a large lesion on the part of the organ called the posterior frontal cortex, on the left side of the head near the temple. Broca hypothesised that this small region of the brain – now called **Broca's area** – enabled humans to speak. His research rocked the medical world and kicked off a search for functions across the whole brain.

Not long after, a German neurologist named Carl Wernicke discovered another area of the brain involved in speech, further to the rear of the brain than Broca's area, and named it **Wernicke's area**. Wernicke also came up with a model of how speech is assembled by **networks** in the brain, a model that still holds up and provides some understanding into the complex nature and interrelated functions of the brain. Two Englishmen, Charles Beevor and Victor Horsley, mainly conducted the research that followed and provided a detailed map of the functions of every section of the brain.

In the 1880s an Italian anatomist named Camillo Golgi developed a new stain that made nerve cells much easier to study under the microscope. This was a fundamental development, for it had previously been impossible to enhance the microscopic cell without killing it. Using the new stain, a Spanish anatomist named Santiago Ramony Cajal revolutionalised the world of neuroscience when he discovered the brain cell, the **neuron**. These discoveries earned Golgi and Cajal the Nobel Prize in 1906. Until then the human brain had been thought of as just a grey mass. Cajal also described the way cells pass on impulses, by reaching out to the group of dendrites of an adjacent cell with a kind of cable called an axon. He went on to make several other major discoveries about brain cells, including the fact that nerve cells morph or change. This is particularly significant in the light of our current knowledge of the brain's plasticity, including that movement and stimulation may affect the plasticity positively. (This fact has huge implications for therapy and will be explored in further chapters of this study.) After the discovery of the neuron, Hans Berger, a physicist, expanded our knowledge on the physiology of the brain by adding the concept of electrical impulses.

The field of neuroscience continues to actively explore the brain and its capacities. The systems that govern the human brain are the most complex and compact on earth, and even though more has been learned about the brain in the last twenty years than in all of human history, science has not come close to understanding how all the pieces fit together to create human consciousness.

In this chapter, the traditional concepts of attention and motor activity will be discussed against this backdrop. This will lay the groundwork for a comprehensive understanding of attentional difficulties and heightened motor activity, the two most prevalent aspects of the AD/HD diagnosis.

3. General Conceptualisation of Attention

3.1 Introduction

There are a number of different theories and findings on **attention**. All attempt to delineate the areas of the brain (school of localisation) that are primarily involved in attention, as well as explain the function of these areas to produce and sustain attention. Needless to say, the theories do not always correspond. This section will thus present a few different perspectives.

3.2 Defining Attention

According to Johnston and Dark (cited in Lezak 1995:19), a clear and universally accepted definition of **attention** has not yet appeared in the literature. They postulate that attention can rather be conceptualised as "several different capacities or processes that are related aspects of how the organism becomes receptive to stimuli and how it may begin processing incoming or attended-to excitation [whether internal or external]" (Lezak 1995:19). The notion of exactly how widely divergent definitions of attention are may be best illustrated by Mirsky's (cited in Lezak 1995:20) placement of attention within the broader category of information processing, and Gazzaniga's (1987) conclusion that "the attention system … functions independently of information processing activities and [not as] … an emergent property of an ongoing processing system" (Lezak 1995:21).

Some researchers have, however, attempted to define attention more specifically. According to William James, attention could be defined as "the taking possession of the mind, in clear and vivid form, of one out of what seem simultaneously possible objects or trains of thought ... It implies withdrawal from some things in order to deal effectively with others" (cited in Sternberg 1999:68).

Sternberg (1999:68) himself defines attention as the means by which we actively process a **limited amount of information** from the **enormous amount of**

information available through our senses, our stored memories, and our other cognitive processes. It includes both **conscious** and **unconscious** processes. The content of attention may reside either within or outside of awareness.

He continues to say that, by **dimming the lights on many stimuli from outside** (sensation) and **inside** (thoughts and memories), we may **highlight the stimuli** that interest us. This heightened focus increases the likelihood that we may respond speedily and accurately to interesting stimuli. Heightened attention also paves the way for memory processes so that we are more likely to remember information that we paid attention to than to information we have ignored.

Many researchers emphasise one or more of the characteristics that William James (cited in Lezak 1995:22) ascribes to attention. These include

- the capacity for disengagement to shift focus
- the capacity to be responsive to either sensory or semantic stimuli characteristics

Additional characteristics of attention have been described as follows:

- sustained tonic attention as it occurs in vigilance, where tonic refers to the type of cognitive processing which requires slow, serial effort and which is affected by the under-production of dopamine
- phasic attention, which orients the organism to changing stimuli, where phasic attention refers to processing that is fast and simultaneous (Lezak 1995:23).

Lezak (1995:27) concludes that most researchers conceive attention as a **system in which processing** occurs **sequentially** in a series of stages within different brain systems involved. This system appears to be organised in a **hierarchical** manner in which the earliest entries are **modality specific** (i.e., visual, auditory, tactile, etc.) while late-stage processing – for example, at the level of awareness – is **supramodal** (cortical). Disorders of attention may arise from lesions involving any point in the system.

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Another salient characteristic of the attentional system is its **limited capacity** (Lezak 1995:29). Only so much processing activity may take place at any one time, so that engagement of the system in processing one attentional task requiring controlled attention may interfere with a second task with similar processing requirements.

Attentional capacities vary not only between individuals but also within each person at different times, under different conditions. Depression or fatigue, for example, may temporarily reduce it in intact adults; old age and brain damage may reduce attentional capacity more lastingly. Simple immediate span of attention, with the capacity for information than may be grasped at once, is a relatively effortless process that tends to be resistant to the effects of ageing and of many brain disorders. Immediate attention span may thus be considered a form of working memory, although it is an integral component of attentional functioning as a whole (Lezak 1995:30-31).

3.3 Models of Attention and Concentration

Different models of attention exist. Within these models researchers attempt to explain attention in terms of the mechanisms that are involved. A few of these are explored in the following section.

3.3.1 The attention circuit

Winston (2004:152) briefly explains the attention circuit as a process of focusing on information before we can use it, and involves several brain areas locked in a circuit. The thalamus relays sensory information to the appropriate part of the brain's cortex for processing. The information then enters short-term memory and is stored in the prefrontal cortex. The parietal lobes are also involved. The ability to shift attention is as important as the ability to focus, as without it a person will be unreceptive to new sensory input and so unable to adapt quickly to new situations.

3.3.2 Three-element theory of attention (Thompson and Thompson)

Thompson and Thompson (2003:114-115) give an overview of the three-element theory of attention in their book: *The Neurofeedback Book.* A short summary of this model is presented here:

i. Arousal

Arousal involves reticular activating system (RAS) activity. The nerve fibres from this system stretch as far as the frontal cortex. These fibres control consciousness, sleep/wake cycles, and the general level of activity in the brain. The axons activating the prefrontal lobe release **dopamine** and **noradrenaline**, which is followed by beta brainwave activity. Beta is the brainwave bandwidth that facilitates attention (see chapter 5 section 2.3 for an explanation of these concepts).

ii. Orientation

Orientation involves the superior colliculus (the area of the brain that stimulates the muscles that turn the eyes) and parietal neuronal activity to disengage attention from the current stimulus. To some extent the eyes direct attention to new stimuli and the brain inhibits attention to previous stimuli in order to free itself for new incoming stimuli.

iii. Focus

The lateral pulvinar nucleus in the thalamus operates like a spotlight shining on the stimulus, locking onto it and sending information about the target to the frontal lobes, which then lock on and maintain attention, together with the eyes (as in the orientation phase).

Comments

An important part that has not been included in this theory by Thompson and Thompson is the role of the cerebellum and the interaction between the cerebellum and the RAS. Their model does, however, include the involvement of other brain areas in attention such as the superior colliculus and the thalamus. The role of neurotransmitters and brainwave activity are also included in the model.

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3.3.3 A model of concentration (Levinson)

Levinson describes his view of attention in his book *Total concentration* (1990:46-56). He states that in our daily lives, we are constantly bombarded by more information that we can possibly handle, comprehend, absorb, or process. As a result, we are forced to select information and activities out of all the stimulation that surrounds us. This often occurs on a moment-to-moment basis. Two basic functions allow us the opportunity to be able to cope with the demand for attention. These are **selective attention** and **selective intention**.

(i) Selective attention

He states that "although many researchers have attempted to define the process of selective attention, I prefer to describe it as the **dual ability to focus on what's important and to simultaneously filter out what's not**" Levinson (1990:56). Levinson believes selective attention comprises eight specific steps that are analogous to the act of turning on and watching a television set. Non-clinical individuals without a concentration dysfunction report that the process feels automatic and that they are unaware of the fact that they are moving through these steps. When the process of selective attention is intact, individual is capable of paying attention and can accomplish tasks that require attention. When, however, the process of selective attention becomes impaired, individuals are not able to pay attention adequately. Individuals are then forced to compensate, consciously and deliberately, by attempting to re-learn the missing steps. Typically this process requires training in modification intervention sessions.

According to Levinson, there are two parallel but interrelated mechanisms responsible for modulating and implementing the process of selective attention:

- The reflexive, automatic determining mechanism (unconscious). This mechanism is inner ear-based (vestibular) and is also responsible for regulating all sensory-motor-related academic processes.
- The second mechanism is conscious and deliberate (intentional) and is determined by higher order cerebral or thinking functions.

Combined, these two concepts form the basis of functional concentration. Under normal circumstances, these two neuropsychological determinants of selective attention are completely interconnected and interwoven via feedback circuits, and thus act as one indivisible whole. In the presence of attentional difficulties, the functional unity of these two determining mechanisms becomes detached and thus clinically apparent. Compensatory processes (intervention) focusing on and enhancing either the vestibular and/or cerebral mechanisms facilitate a reunification of the whole process. A dysfunction in selective attention is characterised by 'improper tuning', distractibility, wandering focus, and reduced response to feedback, making self-correction difficult.

(ii) Selective intention

Even before the individual begins to create something, a certain amount of planning is required. This activity, called selective intention, assists in anticipating the outcome of the action and in selecting the best ways to accomplish a task.

According to Levinson (1990:65), these two processes (selective attention and selective intention) need to be fully functional in themselves, and when combined are fundamental to the ability to concentrate completely.

Comments

Levinson's emphasis on the role of the vestibular system in attention again suggests that movement may be implicated in this process since the vestibular system develops through movement, and movements are co-orinated by the vestibular system. Levinson, like Thompson and Thompson, does not mention the vestibular-cerebellum connection. It is well documented that these two structures are fundamentally linked (Ito 1984, 1987; Goddard 1996). Levinson does include the involvement of the frontal lobe in attention in his model, thereby indicating that attention is an unconscious as well as a conscious process.

3.3.4 Conscious attention (Sternberg)

According to Sternberg (1999:80-84), there are three main functions of conscious attention:

(a) **signal detection,** including vigilance and search, in which we must detect the appearance of a particular stimulus

(b) **selective attention,** in which we choose to attend to some stimuli and to ignore others

(c) **divided attention,** in which we prudently allocate our available attentional resources to coordinate our performance of more than one task at a time

(i) Signal detection

Signal detection theory was one of the first theories to suggest an interaction between the physical sensation of a stimuli and cognitive processes such as decision making. According to **signal-detection theory (SDT)**, there are four possible outcomes of an attempt to detect a signal or a target stimulus.

- correct positive (hit), in which the subject correctly identifies the presence of a target
- false positive (false alarm), in which the subject incorrectly identifies the presence of a target that is actually absent
- false negatives (misses), in which the subject incorrectly fail to observe the presence of a target
- 4) **correct negative** (correct rejection), in which the subject correctly identify the absence of a target

Usually, the presence of a target is difficult to detect, so the individual makes decisions based on inconclusive information, with some criteria for target detection.

(ii) Vigilance

Vigilance refers to a person's ability to attend to a **field of stimulation** over a prolonged period of time, in which the person **seeks** to detect a particular target stimulus. When being vigilant, the individual watchfully waits to detect a signal stimulus that may appear at any time. Typically, vigilance is needed in settings where a given stimulus occurs only rarely but requires immediate attention as soon as it does. Vigilance may thus be seen as a form of sustained attention.

(iii) Search

The difference between vigilance and search can mainly be compared in terms of the level of activity required from the subject. Whereas vigilance involves passively waiting for a signal stimulus to appear, search involves actively seeking out a target. Search refers specifically to scanning the environment for particular features; in other words, actively looking for something without being sure of where it will appear. In both instances, the subject may, whilst searching for something, respond by making false alarms. In the case of a search, false alarms usually arise when the subject encounters **distractions** (non-target stimuli that divert the attention away from the target stimulus) while searching for the target stimulus. The number of targets and distracters affects the difficulty of the task.

(iv) Selective attention

According to Sternberg, people use selective attention to track one message and simultaneously ignore others. Auditory selective attention may be observed by asking a participant to shadow information presented dichotically (both ears at same time, different messages). In essence, the subject is required to pay attention to one message only. Visual selective attention may be observed in tasks involving the *Stroop* effect.

(vii) Divided attention

Specific attentional processes are also involved during divided attention, when people attempt to handle more than one task at a time. Generally, the simultaneous performance of more than one automatised task (like driving a car) is easier to handle than the simultaneous performance of more than one controlled task that requires cognitive or cortical input (like placing names in alphabetic order while having to remember the names at the same time).

Comment

Sternberg focuses mainly on the mechanisms of attention rather than its possible neurological foundations. This implies that he considers the aspects of attention and its mechanisms on a higher cerebral (cognitive) level of operations, without considering possible involvement of lower brain levels or functions for support. Sternberg's theory has been applied in many measuring instruments of attention.

Many of these instruments are computer-based continuous performance tests such as the Integrated Visual and Auditory Test (IVA). Other tests such as the Cognitive Control Battery are also based on his theory. Since Sternberg's model is a wellknown model, these two tests were included in the test battery applied in this study. In addition, the aspects included in his model are quantitatively testable, as there are certain outcomes to observe with every aspect.

3.3.5 Lezak's model of attention

Lezak (1995:32-36) mentions four aspects of attention that are considered fragile and are thus often of greater clinical concern:

(i) Focused or selective attention

Lezak believes that this concept is probably the most studied aspect of attention, and the one people usually have in mind when referring to attention. It is the capacity to highlight the one or two important stimuli or ideas being dealt with while suppressing awareness of competing distractions. It is commonly referred to as concentration.

(ii) Sustained attention or vigilance

This refers to the capacity to maintain an attentional activity over a prolonged period of time.

(iii) Divided attention

This involves the ability to respond to more than one task at a time or to multiple elements or operations within a task, as in a complex mental task where more than one operations needs to be performed. It is thus very sensitive to any condition that reduces attentional capacity.

(iv) Alternative attention

This process allows for shifts in focus and tasks.

While the abovementioned aspects of attention may be demonstrated and evaluated by different examination techniques, even discrete damage involving any part of the attentional system may create alterations that can affect more than one aspect of attention. Underlying many patients' attentional disorders is slowed processing, which may have broad-ranging effects on general attentional activities (Lezak 1995:37).

Although attention, concentration, and tracking can be differentiated theoretically, in practice they are difficult to separate. Pure attention deficits appear as distractibility or impaired ability for focused behaviour, regardless of the patient's intention. Intact attention is a necessary precondition of both concentration and mental tracking activities. Concentration problems may be due to a simple attentional disturbance, or to an inability to maintain a purposeful attentional focus, or as is often the case, to both. At the next level of complexity, conceptual tracking may be prevented or interrupted by attention or concentration problems and also by a diminished ability to maintain focused attention on one's mental contents while solving problems or following a sequence of ideas. These concepts seem to have a hierarchical map as well as an interrelated and interdependent effect on one another.

Clarifying the nature of an attentional problem depends on observations of the patient's general behaviour, as well as his or her performance on tests involving concentration and tracking. It is only by comparing these various observations that the examiner may begin to distinguish the simpler global defects of attention from the more discrete, task-specific problems of concentration and tracking. Furthermore, impaired attention is not always a global disability but may involve one receptive or expressive modality more than others (Lezak 1995:352).

Comment

Lezak describes similar mechanisms to those mentioned by Sternberg. These mechanisms also seem to be an outcome of the functions described, in other words, they are higher order mechanisms. One possible reason for the fact that these concepts are usually included in standardised measurements is the fact that they are quantitatively measurable, as there is a set outcome within a specific time frame. In contrast to this, aspects such as those mentioned by Levinson (vestibular functioning) and Thompson and Thompson (RAS functioning) are more difficult to measure and are thus not standardisable. These aspects are generally known as

lower order functions. Both levels (higher and lower) were included in the research design.

3.4 Differential Perspectives on Attention

As stated before (see the introduction to chapter 3), a rational point of departure to understand attention is the functioning of the CNS. For this reason the model described by Riccio, Hynd, Cohen and Gonzalez (1993) and Mather and Goldstein (2001) has been chosen as a framework to support a possible new conceptualisation of attention, as it seems to provide a more holistic view (although still scientifically well-researched) on all the factors involved in attention. This model includes the related functional importance of different brain structures (neuroanatomy) (higher and lower levels), the chemicals working in these structures (neurotransmitters) as well as the way in which these relate to brain functions (neurophysiology).

Riccio et al. (1993:118) and Mather and Goldstein (2001:53) state that one may study attentional mechanisms from a **neuroanatomical**, **neurochemical**, or **neurophysiological** perspective. The **neuroanatomical** approach focuses on the location of brain areas that subserve those systems thought to mediate the regulation of attention and inhibit motor activity. The **neurochemical** approach addresses the role of specific neurotransmitters that facilitate communication among the neuronal circuits implicated in this disorder. The **neurophysiological** perspective attempts to explain the dynamic interaction between the neurochemical and neuroanatomical components that together form a functional system.

The illustration (figure 3) shows the cell body or nucleus, axons, dendrites as well as the myelin that surrounds the axons.



Figure 3 The neuron (Winston 2004:117)

3.4.1 Neuroanatomical Perspective

3.4.1.1 Hemispheric categorisation

The left hemisphere is involved in **focused**, **selective attention** (Thompson & Thompson 2003: 111). The right hemisphere is said to be involved in the **general maintenance of attention and arousal.** It regulates **information processing** as well as **rapid shifts in attention** (Thompson & Thompson 2003:112). The question may be asked as to whether, in the AD/HD inattentive type, there may be right frontal dysfunction. Children struggling with attention and motor control may demonstrate a tendency for hemi-spatial neglect of the left side; this condition is found with brain lesions on the right. Further, there is the possibility that this right frontal dysfunction might be responsible for a decreased inhibition of the right posterior areas, which would explain the high distractibility to external stimuli in these individuals (Thompson & Thompson 2003:112).

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Various methods of investigation and examination (such as neuro-imaging studies, MRI, PET) point to the right hemisphere's involvement in attention. Whether the research focused on the frontal lobes or the basal ganglia, results implicate the structures in question located in the **right hemisphere**. Riccio et al. (1993:119-120) argue that the right hemisphere is specialised for attentional processes in adults. Adult patients with right central-posterior lesions have shown less arousal when compared with patients with left hemisphere lesions. Further, other researchers have found a higher incidence of attentional deficits, problems in vigilance, distractibility, and difficulty performing intentional motor activities in children and adults with documented right hemisphere lesions (Riccio et al. 1993:119-120).

Imaging studies demonstrate decreased activity in the right hemisphere in the following areas:

- anterior cingulate (fixing attention on a given stimulus)
- **prefrontal cortex** (planning actions and controlling impulses, which also involves the orbito-frontal cortex) (Arnsten 2001:186)
- **upper auditory cortex** (integration of stimuli from several different sources) (Castellanos 2001:251)

According to Thompson and Thompson (2003:116), we may hypothesise that a lack of activity in these areas prevents children from grasping the whole picture and, instead, they view the world in fragments with one stimulus after another vying for attention.

Magnetic resonance imaging (MRI) has also demonstrated differences between children who have AD/HD and controls (Hill et al. 2003:496) in terms of smaller volume in the right prefrontal brain regions. Specifically, the normal 'right greater than left' asymmetry of the caudate nucleus, globus pallidus and a subregion of the cerebellar vermis (figure 10) appears to be absent in children with AD/HD compared to normal children (Castellanos 2001:251).

Further, inspection of MRI scans showed that children with AD/HD had a smaller corpus callosum in the region of the genu and the splenium and in the area just anterior to the splenium. Though subtle, the anatomical differences in these children

may ultimately affect both the cooperative and individual functioning of the hemispheres. This is because interhemispheric fibres (pathways) in these regions interconnect the two hemispheres of the brain, including the left and right frontal, occipital, parietal, and posterior temporal regions (Riccio et al. 1993:120) (see section on commissures and figure 22).

Research conducted on the neurobehavioral characteristics of children with evidence of right hemisphere dysfunction, using neurological or neuropsychological measures, provides additional support for the involvement of the right hemisphere in AD/HD (Riccio et al. 1993:120). In examining the behavioural, neurological, and neuropsychological characteristics of children with evidence of right-hemisphere dysfunction, Voeler (cited in Riccio et al. 1993:120) found that 93% of the children met the criteria for AD/HD.

Comment

Interpreting the research findings discussed above may elicit a chicken-egg dilemma. Are attentional difficulties caused by right hemisphere malfunctioning, or do the symptoms of AD/HD impair the functioning of the right hemisphere, causing these phenomena to appear in individuals presenting with AD/HD? Most researchers agree on the greater involvement of the right hemisphere in attentional difficulties; however, there is less concesus about the primary areas in the right hemisphere that are involved, as well as the cause for these dysfunctions.

3.4.1.2 Brain structures

Typically, neuroanatomical hypotheses on brain structure involvement in attention propose the involvement of cortical (frontal) and subcortical structures (brainstem, reticular activating system, thalamus, hypothalamus, and basal ganglia.) Some of these are identified in figure 4.



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Figure 4 Basic brain structures

(Sternberg 1999:50)

i) Orbito-frontal cortex

Together with the prefrontal cortex, this area is said to be involved in **inhibiting inappropriate action**. Decreased functional activity seems to be greatest in the left frontal area in AD/HD during tests of intellectual or attentional functions (Thompson & Thompson 2003:115; Amen et al. 1997). Amen found that that 65% of his AD/HD subjects showed decreased blood flow to the prefrontal cortex during a continuous performance task compared to 5% of the controls (Thompson & Thompson 2003:115).

ii) Frontal and prefrontal cortex

The frontal lobes have long been designated as the 'central executive' of all higher nervous system functions. Capabilities such as **directed attention**, **concentration**, critical thinking, activity level, and **impulse control** are all known to be regulated by this critical region (Sterman 2000:9). The frontal cortex is specifically involved with reason, planning, writing and reading, and a host of other cognitive functions. The cortex is what makes us human; it distinguishes us from animals by mitigating our basic instincts.

Parallels have been drawn between frontal lobe dysfunction and AD/HD, with the identification of the prefrontal region, in particular. One possible explanation for this includes a developmental delay in the myelination of the prefrontal area (Riccio et al. 1993:119-120; Thompson & Thompson 2003:115). The following chart, provided by Goddard (1995:2), illustrates this delay in myelination.



Figure 5



(Goddard 1996:2)

Positron emission tomographic (PET) scan studies support the involvement of the prefrontal lobe in reduced whole **brain glucose utilisation**, particularly in the right frontal and posterior-medial orbital areas (Riccio et al. 1993:119-120).

In conjunction with the frontal lobe, the caudate nucleus (within the basal ganglia, and specifically in the right hemisphere) has also been implicated in the neurological basis of AD/HD, partly because of the resemblance of AD/HD to the spectrum of behaviours associated with dysfunction in the caudate-frontal axis. This system in the brain is known to be important in motor regulation and behavioural inhibition (Riccio et al. 1993:119-120).

Magnetic resonance imaging (MRI) studies have also shown decreased tissue volume in the frontal, premotor, and sensorimotor cortex and in the caudate of AD/HD subjects (Thompson & Thompson 2003:115; Hill, Yeo, Campbell, Hart, Vigil & Brooks 2003:496).

Although enough evidence has been presented to support the notion of frontal lobe involvement, Welsh and Pennington (1988:199) caution against interpreting developmental alterations in prefrontal function as evidence of AD/HD. Not all studies focusing on the neurocognitive functioning of children with AD/HD have demonstrated significant frontal dysfunction for the group (Loge, Staton & Beatty 1990). Further, Benson (1991:S9) has argued that the neurological basis of AD/HD cannot be limited to frontal immaturity or anomaly, and states that AD/HD appears to be related to more widespread dysfunction. Thus, while the frontal/prefrontal area has been consistently implicated, there continue to be dissenting opinions.

iii) Anterior cingulate cortex (ACC)

This cortex focuses **attention** and facilitates tuning inwards to one's own thoughts. Increased blood flow on the **right** side of the ACC suggests that attention is focused on **internal events** as the ACC **distinguishes** between **internal and external** events. Problems here may be the cause of internal distractibility (Thompson & Thompson 2003:115).

iv) Dorso-lateral prefrontal cortex

This area is involved when subjects **hold** a thought in mind, **select** thoughts and perceptions to attend to, **inhibit** other thoughts and perceptions, **bind** the perceptions into a unified whole, endow them with **meaning**, **conceptualise** a plan and make **choices** (Thompson & Thompson 2003:115).

v) Reticular formation

The reticular activating system (RAS) is only fully myelinated after puberty. This developmental delay in myelination may partially explain why younger children have a short attention span. The RAS plays a major role in maintaining attention. A threat causes activation (a rush of adrenaline), which closes down all unnecessary activity resulting in an alerted brain, which appears on a brain scan as quiet. It also inhibits body activity. Breathing is shallow and guiet and heart rate is slow. In this state, however, activity remains in the superior colliculus and the lateral pulvinar nucleus of the thalamus and in the parietal cortex. These areas are involved in orienting and focusing (Thompson & Thompson 2003:116). This in part supports Kaneko's (Kaneko, Hoshino, Hashimoto, Okano & Kumashiro 1993:59) theory on the hypothalamic-pituitary-adrenal axis (HPA) that is involved in emotions, learning and attention. Tsigo and Chrousos (2002:865) mention the role of this system in stress. Some researchers (e.g., Gainetdinov, Wetsel, Jones, Levin, Jaber & Caron 1999) have confirmed that metabolic disturbances of serotonin in the brain might play an important role in the pathophysiology of AH/HD. Therefore, it seems appropriate to consider that the negative feedback mechanism of the HPA axis may be disturbed in some types of AH/HD children due to a disorder in regulation by serotonin metabolism.

Comment

As in the previous section, the issue of the direction of causality arises. Does a malfunction in the RAS lead to a metabolic dysfunction, or does the metabolic dysfunction cause the observed suboptimal functioning of the RAS?

vi) Hippocampus-septal nuclei-prefrontal cortex circuit

The hippocampus is important in **selective inhibition of attention** to things that might distract one's focus from the central problem. It also is a part of the brain's system of **arousal**, **alertness**, **awareness**, and **orientation** in the process of **focusing** attention on one set of environmental signals while excluding others. The hippocampus is also involved in laying down a conscious memory of events (Thompson & Thompson 2003:116).

vii) Cerebellum

The cerebellum is a relatively large portion of the brain and is located near the brainstem. It is primarily responsible for coordinated motor movements. There is also some recent evidence (http://www.autism.org/cerebel.html) that the cerebellum is partially responsible for speech, learning, emotions, and attention. In the late 1980s, Courchesne used magnetic resonance imaging (MRI) to examine specific areas in the cerebellum, lobules VI and VII, and the relationship between these areas and attention (http://www.autism.org/cerebel.html). He concluded that they might be responsible for shifting attention (figure 10).

Other researchers such as Justus and Ivry (2001:276) argue that cerebellar contributions to cognition are computationally plausible, given the cerebellum's reciprocal connectivity with the cerebral cortex. They suggest that this function of the cerebellum may be an example of an evolutionary process by which mechanisms originally evolved for one function (in this case, motor control) are adapted to other functions such as cognition and attention. Krain and Castellanos (2006) support this notion when they state that neuropsychological findings suggest that these behaviours (inattention, hyperactivity, and impulsivity) result from underlying deficits in response inhibition, delay aversion and executive functioning which, in turn, are presumed to be linked to dysfunction of frontal-striatal-cerebellar circuits.

Comments

Studies have implicated many areas and structures (from the prefrontal cortex right through to the base and the RAS and cerebellum) in attentional difficulties. Both hemispheres seem to be involved (although the right somewhat more than left). The relationship between these neuroanatomical and behavioral subtypes, however, has not been borne out by a study of group comparisons.

3.4.2 Neurochemical perspective

3.4.2.1 Neurotransmitters

Marieb (2003:211) defines neurotransmitters as "chemicals released by neurons that may, upon binding to receptors of neurons, or effector cells, stimulate or inhibit them". Researchers have generally accepted that the catecholamines (**dopamine**, **norepinephrine**) appear to affect a wide variety of behaviours, including **attention**, **inhibition**, **response of the motor system**, and motivation. Related to the focus on neurotransmitters, Mefford and Potter (1989:33-42) postulate that an imbalance in the formation of dopamine or norepinephrine results in the decreased stimulation of the locus coeruleus. Abarbanel (1999:317) supports this notion. As noted in the previous section, the RAS is involved with orientation and focusing, so a decrease in chemical activity could indicate a disturbance in these functions. Some support for this conceptualisation is provided by the efficacy of treatment with **clonodine** and **psychostimulants** (e.g. Ritalin) in some children with AD/HD (Riccio et al. 1993:121) as these agents have a direct influence on the blocking and reuptake of neurotransmitters. Figure 6 highlights the most important neurotransmitters.



Figure 6 Neurotransmitters

(Holford 2006-17)

i) Dopamine

The principal neurotransmitter for **sustained attentional** activity and information processing in the left hemisphere is **dopamine**, specifically in the **fronto-mesolimbic system** (Thompson & Thompson 2003:113). The type of cognitive processing which

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is negatively affected by the under-production of dopamine is that which requires **slow, serial effort**. This type of processing is called **tonic**. The left hemisphere is biased in the direction of carrying out routine and repetitive activities. It is this kind of processing that may be improved with stimulant medication. In a restatement of dopaminergic explanations of attentional difficulties, Levy (cited in Riccio et al. 1993:121) suggested that the underlying dysfunction in AD/HD is a disorder of dopaminenergic circuits between the **prefrontal** and **striatal centres** (basal ganglia.) The reason for reduced dopaminergic activity may be due to genetic differences with respect to dopamine receptors. The impact includes disorders of planning and automatic instinctual motor programming (Thompson & Thompson 2003:113; Riccio et al. 1993:121).

Related to the dopaminergic models, Posner, Inhoff and Fredrich (1987:107-121) posit that the **parietal lobe** is involved in covert shifting of visual attention, whereas the frontal lobe is the attentional command system, and that both work together to regulate attentional processes as a complex functional system. Both these functions and brain areas are reliant on dopamine to perform those tasks.

Theoretically, many of the foregoing symptoms may be due to under-activation of dopaminergic activity in the left hemisphere. Dopaminergic over-activity, on the other hand, is thought to be associated with blunting of affect, excessive intellectualisation and introversion. Pathologically, it may underlie other disorders such as paranoid states, anxiety, obsessive-compulsive disorder (OCD) and schizophrenia (Thompson & Thompson 2003:112).

Comment

The literature on brain structures and the chemical workings of these structures blurs the picture further by suggesting even more possible causes of the dysfunction that is described as AD/HD. An examination of brain structures implicates right hemispheric dysfunction; yet the above section pinpoints underactive dopamine levels as a possible contributor.

ii) Noradrenergic system (norepinephrine)

The right hemisphere is involved in the **general maintenance of attention** and **arousal.** It **regulates information processing**, which requires peripheral vision, spatial location, and rapid shifts in attention. These aspects of attention appear to involve the **noradrenergic (norepinephrine) system**. The noradrenergic system is thus more involved in initial attention plus arousal, wakefulness, alertness and the ability to respond to change and to new stimuli (Thompson & Thompson 2003:112).

Comment

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The different perspectives presented here invite the question of whether the location of dysfunction could determine whether or not symptoms respond to stimulant medication. For example, 40% of children do not respond to treatment with Ritalin. Possibly, those children who do respond positively to Ritalin do so when the source of dysfunction is incorrect dopamananic levels (specifically in the left hemisphere), while those children who do not respond to Ritalin do so because the cause is not a dopamine deficiency but rather a dysfunction in the right hemisphere. If this is the case, it is imperative that medical practitioners who prescribe medication determine the underlying cause of the symptoms.

Another possible cause of attentional difficulties is **excessive locus coeruleus norepinephrine** production, leading to extreme noradrenergic stimulation of the right cerebral hemisphere. This represents minor brain stem involvement in arousal (usually arousal is discussed in terms of the involvement of the reticular activation system) (Thompson & Thompson 2003:112; Abarbanel 1999:317). Thompson and Thompson (2003:112) further state that people with AD/HD seem to use automatic processing. This type of processing is fast and simultaneous and is called **phasic** attentional ability. This style of attention is biased towards novelty and change. It has also been noted that overactivation of the noradrenergic system in the right hemisphere is associated with extroversion, histrionic behaviour, impulsivity and manic behaviours and may account for external distractibility (Thompson & Thompson 2003:112).

Comment

The theories presented here are a reminder of the complexity of the attentional system and bring into question the methods used to address attentional dysfunction. Would Ritalin effectively address a norepinephrine irregularity in the brain if the pharmaceutical action of Ritalin specifically inhibits the reuptake of dopamine?

iii) Brief summary of dopaminergic and noradrenergic theory

Despite the divergent theories discussed here, researchers seem to agree that attentional control mainly involves two separate neural systems: (a) an activation system that is centred in the left hemisphere and specialises in analytic, sequential, and routinised cognitive operations such as motor responses; and which is modulated by dopaminergic transmitters, and (b) an arousal and maintenance system that is centred in the right hemisphere and is responsible for holistic, parallel, and novel cognitive functions such as perceptual orienting responses; this is modulated by norepinephrinergic neurotransmitters (Thompson & Thompson 2003:112). Schematically this may be represented as follows in figure 7.



Figure 7 Attention control

This theoretical framework is supported, in part, by the observed actions of stimulant medications. These may be listed as follows (Malone, Kershner & Swansen 1994:181-189; Abarbanel 1999:317).

- Stimulants in animal studies block uptake of norepinephrine (NEP) and dopamine in the striatum, hypothalamus and the cortex, thus allowing these substances to be available longer.
- Stimulants facilitate the release of dopamine (but not NEP) from the striatum (which includes the caudate). It has been observed that the left caudate is small in individuals with AD/HD and that stimulants increase activity in the left striatum, thereby increasing blood flow in this area.
- Stimulants dampen activity in the locus coeruleus.
- Stimulants increase left hemisphere processing speed.
- Stimulants decrease right hemisphere processing speed.

Comment

It is interesting to note that researchers seldom ask why an imbalance occurs in the amount of available dopamine and norepinephrine in the first place. In answering this, the effect of amino acids and essential amino acids as precursors of neurotransmitters (illustrated in figure 6) should be considered. The question could then be asked what role nutrition, as a supplier of amino acids, plays in attention and the maintenance of attention.

3.4.3 Neurophysiological perspective

A neurophysiological perspective combines neuroanatomy as well as neurochemical working in the specific brain structures. The functional outcome of the interplay between anatomy and chemistry may be viewed in terms of metabolism and bloodflow as well as brainwave activity.

3.4.3.1 Metabolism and bloodflow

Through regional cerebral blood flow comparisons of children with AD/HD and control children, Kim, Lee, Shin, Cho and Lee (2002:219) found that the AD/HD children showed decreased cerebral blood flow in right lateral prefrontal cortex, right middle temporal cortex, both orbital prefrontal cortex and both cerebral cortices (Kim et al. 2002:219). Studies on metabolic activity in the frontal lobes and basal ganglia indicate increased metabolic activity in the primary sensory and sensorimotor regions



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in individuals who struggle to pay attention (Lou, Henriksen & Bruhn 1984:825-829; Lou, Henriksen, Bruhn, Borner & Nielsen 1989:48-52).

3.4.3.2 Brainwave activity

Brainwave activity measured by an electroencephalogram (EEG) measures the electrical activity in a specific area that is facilitated by the availability of neurotransmitters.

The human brain normally operates within a range of 1Hz to about 40Hz. The frequencies are lumped into categories that denote their characteristics (bandwidths).



Figure 8 EEG bandwidths

http://www.addneurofeedback.com/imag es/neurofeedback/brainwaves.gif

The 1-to-4 hertz range is called a **delta** brainwave pattern, and occurs during sleep and some comas. **Theta** is represented between 4-to-8 hertz. It is called the hypnogogic state and is a kind of conscious twilight that occurs between being deeply relaxed and sleeping. Berger's 10-hertz, the first brainwave to have been measured, falls within what is called the **alpha** range, which is a relaxed but awake state, from 8-to-12 hertz. He also named the **beta** range, from 13 to around 30 hertz, which is the range of normal waking consciousness. The bandwidths differ in terms of the number of cycles per second as well as the morphology. Each bandwidth corresponds to a specific mental state. These bandwidths and their associated mental states are presented in table 4. Brainwaves and the generation of these electrical impulses is discussed in detail in chapter 5, section 2.3.

Table 4

	50 Hz	Electrical interference
	44 + Hz	Reflects the effect of muscle activity on the EEG
HIGH BETA	38 – 42 Hz Sheer (Gamma)	Cognitive activity – related to attention. Increasing it may help to improve learning disability. It is also referred to as a 'binding' rhythm. It may also be seen at the moment of correcting balance
	~ 27 Hz (Elevated in mid 20's)	May correlate with family history of addiction
	24 - 36 Hz	Can correlate with ruminating which is most often negative
	19 – 23 Hz	This may correlate with emotional intensity including anxiety
		Correlates with active problem-solving cognitive activity
BETA	16 - 20 Hz	More beta is required when learning a task than when it has been mastered
	17 Hz	Analysing information and specific problem solving
Slow Beta SMR	13 – 15 Hz	When this corresponds to sensory motor rhythm (only over the central cortex: C3, Cz, C4), it may correlate with decreased motor and sensory activity combined with a mental state that maintains alertness and focus Appears to correlate with a calm state, decreased anxiety and impulsivity It may also correlate with a decrease in involuntary motor activity
High Alpha	12 (11-13) Hz	Open awareness Can correlate with a very broad awareness state. This may be a readiness state seen especially in high-level athletes Persons with high intelligence often demonstrate a higher peak alpha frequency
Low Alpha	8-10 (or 11) Hz	Internally oriented and may be observed in some types of meditation It is possible but rare to have a dissociative experience when totally in this state Adults (eyes closed) have alpha as the dominant frequency

Correlations of bandwidths to mental states

	7 Hz	Visualisation
THETA	6 - 7 Hz	Internal orientation Important in memory recall Can be very creative but may not recall ideas from this mental state Not focused on external learning stimuli such as reading or listening The dominant frequency in children
	3 – 5 Hz	Tuned out Sleepy
DELTA	0.5 – 4 Hz	Sleep Brain damage Learning problems Dominant frequency in babies Movement or eye blink artefact

(Adapted from Thompson & Thompson 2003:10)

Quantitative analysis of EEGs in boys with AD/HD revealed **increased slow wave activity (Theta**), predominantly in the frontal regions, and **decreased beta activity** in the temporal regions, compared to controls matched for age and sex (Riccio et al. 1993:121; Clarke, Barry, McCarthy, Selikowitz & Brown 2002a:1036-1044). This indicates possible **decreased cortical arousal** in AD/HD children in those areas of the brain frequently associated with executive control and language as well as the **ability to pay attention**. Hynd, Senrud-Clikeman, Lorys, Nobey and Eliopulos (1990:917) used MRI to image the frontal lobes of children with AD/HD, dyslexia, and controls. Findings revealed decreased right frontal width measurements in children with AD/HD relative to children without AD/HD, providing additional brainwave morphological evidence of frontal lobe involvement in AD/HD (Thompson & Thompson 2003:115).

Sterman (2000:3-4) published a number of abnormal EEG patterns in AD/HD populations. A summary of these symptoms includes:

- 1. A localised excess of slow wave 4-8Hz theta activities in prefrontal, frontal, and sensorimotor cortex
- 2. A generalised excess of theta or slower alpha activity in all cortical areas during all tests states, often exaggerated during task engagement

- A significant excess of normal alpha rhythm activity mostly in anterior cortical areas
- A significant reduction of normal 12-20Hz rhythmic activity across sensorimotor area (known as the sensorimotor rhythm or SMR) associated with increased faster activity
- EEG hyper-coherence between left and right frontal recordings and between frontal/temporal regions within each hemisphere, as well as interhemispheric power asymmetry in left and right posterior temporal and parietal regions

The effect of slow wave activity in the prefrontal and frontal region is a kind of conscious twilight that occurs between being deeply relaxed and sleeping. This is found in an area that should be alert. During test states beta activity should be strong in order to maintain attention, but Sterman (2000:3) noted excess theta and slower alpha activity in the areas usually associated with cognitive abilities.

3.5 Attention in Conclusion

Riccio et al. (1993:121) write that although there seems to be evidence that supports the role of both neurochemical and neuroanatomical perspectives of attentional difficulties, neither theory, taken individually, fully accounts for the myriad behaviours associated with AD/HD. Researchers have proposed that certain ascending/arousal and descending/inhibitory pathways (e.g., the loops that connect the frontal lobes, basal ganglia, and thalamus) constitute a system that activates/inactivates other brain regions. When this functional system is disrupted along the ascending/arousal loop, this component is no longer able to maintain an adequate level of arousal in the specifically targeted brain regions at the cortical level. Consequently, when this (neurophysiological) functional system is disrupted along the descending/inhibitory loop, an adequate level of inhibition/selective attention cannot be maintained. Thus, as other researchers have hypothesised, interference at any level (anatomical, chemical or physical, or any combination) of this loop may lead to a cluster of clinically similar signs with diversity, depending on which level(s) are affected. Further, involvement of the subcortical limbic system (e.g. amygdala), along with the frontal lobes, might results in behaviour disorders that occur comorbidly with AD/HD. Riccio et al. (1993) therefore provide an explanation for "top-down" arousal that also accounts for the variability of behaviours and characteristics associated with children with AD/HD.

Specifically, on a neuroanatomical level, researchers have found that parietal, frontal, and limbic pathways terminate in the caudate. A similarly organised system exists between the somatosensory and premotor cortices to another component of the basal ganglia, the putamen (Riccio et al. 1993:121). Thus, the basal ganglia has emerged as a hub of influence over the thalamus and motor structures because of the number of crossing pathways that lead to and from the cortex.

In essence, the model published by Riccio et al. (1993) incorporates the **neurochemical** as well as the **neuroanatomical** perspectives, resulting in a **neurophysiological response**, without necessarily implicating or denying the importance of either hemisphere in particular. Voeller (1991:S2) suggests that this model provides the best explanation of the various clusters of behaviour associated with AD/HD. However, Voeller goes on to state that although this theory has appeal for understanding the diverse characteristics associated with AD/HD, it still lacks empirical support.

The above information suggests various possible neurological explinations for the behaviours that are interpreted as symptoms of AD/HD, according to the DSM-IV-R. The diversity of these findings (anatomical, chemical, physiological, and a combination of all three) supports the notion that no true single scientific model and explanation for AD/HD exists. As Lyall Watson states, "If the brain were so simple that we could understand it, we would be too simple to do so" (cited in Robbins 2000:29).

4. General Conceptualisation of Movement

4.1 Introduction

Eaton, McKeen and Campbell (2001:205) state that movement epitomises childhood, from the first fluttering in the womb to the preschooler on the move; the playful child

and the adolescent who sleeps until noon. Kalat (2001:224) asks the question: "Why do we need brains?" Plants grow, multiply and thrive without brains. But then again, plants do not need to move. Kalat answers his question by stating that humans need brains in order to control behaviour, and that our behaviours are mostly movements. He states that no matter how powerful our internal processing is, it would be absolutely worthless without execution in the form of movement. This resonates with previously stated information that movement plays an important role in the brain's functioning. Movement structures the brain and movement is also expression on the level of structure. It is then significant that most psychologists pay little attention to movement. The study of movement seems somehow less psychological than the study of visual processing, perception, learning, social interaction, attention or emotions.

The entire surface of the body is connected to the brain through different pathways, each with their own neurons. Sensitive areas such as the hands and fingers have more neural connections. They are allocated much more brain space than, for example, the feet. As actions are repeated, the cell groups that have been used team up to reinforce specific pathways. This is done through a chemical change that ensures that a trigger to one cell will fire strongly to the next. This is called Hibbing learning and explains why habitual movement can, after some initial practice, be performed almost automatically without thought. As these pathways are established, the unused dendrites are discarded. This process is known as "pruning of the dendritic arbor" (Macintyre & McVitty 2004:6).

4.2 The Importance of Movement

As the first part of this chapter focused mainly on attention, the following section will investigate movement to yield a comprehensive understanding of this basic human function. Pheloung (2003), Goddard (1996), Hanneford (1995), Bluestone (1994, 2000, 2004) and Fredericks, Kokot and Krog (2006) make a strong argument for the importance of movement and the recognition of the elaborate interplay between brain and body. These authors cite brain research showing that the brain is plastic in that it adapts continuously. The structure of the brain can be changed through certain kinds of stimulation, including movement. Fredericks, Kokot and Krog (2006:29)

state that the body is a sensory-motor response system that causes the brain to organise itself. A number of other theorists also support the importance of movement and brain function as a unified concept (De Jager 2001:8; Pica 1998:18). Hannaford (1995), a prominent author on the subject of movement, states that movement must be seen as an integral part of all mental processing, from the anatomic movement in cells (cellular movement) to the thought made manifest in action.

Piaget observed that movement is essential to the formation of intellect (Fredericks, Kokot & Krog 2006:29). He called the first stage of intellectual development the sensorimotor stage, because both sensory and motor activities are used as a source of information. This forms the foundation on which the subsequent hierarchy of all intelligence is built. Between birth and five or six years of age, children's bodies, as much as their minds, are the organ of intelligence (Fredericks, Kokot & Krog 2006:29).

4.3 Historical Background

The study of motor activity and children's movement in particular has a long empirical history. Early investigators looked at the relationship between measures of motor activity levels and other variables such as age, gender, and situation. The early Freudians viewed motor activity as part of an expressive process, which could not be separated from a person's unintentional reactions. Restraints in motor discharge were thought to be provided by the ego process of thought, and individual differences could be conceptualised in terms of intrapsychic dynamics. Learning theories and developmental psychology largely supplanted the psychoanalytic approach in the 1950s and 1960s, and eschewed interpretations of motor behaviour that involved internal motivation and drive states (Eaton, McKeen & Campbell 2001:206).

Fredericks, Kokot and Krog (2006:30) note that the importance placed on physical movement is also supported by other theorists such as Kephart (1975) (a specialist who applied perceptual-motor matching in the field of learning difficulties), Ayres (1994, 1979, 1982), who focused on the need for sensory integration through movement in order to learn successfully, Cratty (1972, 1973) and Delacato (1959, 1974) (who used movement patterning in order to improve neurological organisation).

These beliefs have been strengthened by brain research. Physiologists and neuroscientists have established movement as a form of stimulation that creates the neural pathways making up the structure of the brain (Berthoz 2000). Examples include the work on nerve growth factor for which Rita Levi-Montcalcini won the Nobel prize in 1986 (Gold 1997:140), Changeux and Conic (1987), whose work provided evidence that movement is necessary for neural growth, and Ito (1984, 1987), who found that stimulation of the vestibulo-ocular reflex arc resulted in changes to the structure of the cerebellum (Gold, 1997:141). Research by Thompson (1996) and Gold (1997) shows conclusively that the brain is plastic; and that structures may be changed by stimulation and specifically by movement (Kokot 2003b:11-24; Fredericks, Krog & Kokot 2006:30).

4.4 Movement

On a neuroanatomical level, every one of our 600-odd skeletal muscles is attached to bone, or to other connective tissue structures, at no less than two points. On a neurophysiological level, body movement occurs when muscles contract across joints (Marieb 2003:176). Movement is essential for survival: our initial ability to suck and feed ensures nourishment for growth. Internal movement ensures that the nourishment reaches its target, all the way to the electrical movement between neurons. Movement also ensures that the organism can defend itself or move away from physical danger. As Kalat (2001:224) points out, no thought process would be significant without execution. The mere fact that we can move, as well as the effects of movement, illustrate the undeniably critical link between the act of movement and the brain. A double loop exists between the body and the brain. Not only does movement structure the brain, movement can also reflect the neural structure (organisation) of the brain. This critical concept will be explored in sections to follow and is investigated as a possible therapeutic intervention for children struggling with inattention and heightened motor activity.

Movement facilitates physical development and enables children to develop muscle strength, kinaesthetic awareness and motor skills. Infants learn to crawl and walk, and these major milestones provide them with an early means for exploration. This exploratory activity continues as preschoolers refine their motor skills. From a

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developmental perspective, movement plays a paramount role in children's exploratory behaviour, and ultimately their cognitive development. Both Piaget (1952) and Rousseau (1948) emphasised the need for children to physically interact with their environment as a part of normal cognitive development (Eaton, McKeen & Campbell 2001:216). Although these well-known researchers highlighted the need for and role of movement in infancy and early childhood, a study by Eaton, McKeen and Campbell (2001) revealed that motor activity levels peaks at age seven to nine years, during Piaget's concrete operational stage. A maturational approach to movement provides an alternative perspective on AD/HD. Children's high levels of movement are typically accompanied by rapid shifts of attention, an important marker of AD/HD. Eaton et al. (2001) point out that the observed peak in motor activity corresponds with the peak in diagnosis of AD/HD at seven to nine years of age. These data were drawn from nonclinical, ostensibly normal children, suggesting that children progress through a natural development of age-related movement. In the end, though, Eaton et al. (2001:219) conclude that children do not move because it is fundamental to their development. They move because they enjoy it. Their enthusiasm reflects the developmental importance of physical movement in childhood.

4.5 A Model of Movement

There seems to be general consensus on the basic structures involved in motor activity. This makes the design of a general model of movement easier than the construction of a model of attention, which involves more brain structures and different theories of the role of the different areas in the brain.

4.5.1 Three kinds of motor control

According to Stafford and Webb (2005:157), there are three kinds of motor control systems used to modulate movement while they occur. These are used in situations from needing to move an arm more (modulating an initial action), to catching a ball, to a change in the walking pattern. This section discusses this model in terms of functionality. This is followed by a focus on its anatomy in the discussion of the brain structures involved in movement, and in particular, the section on the cerebellum.
i) Feedback

All neural systems include some 'noise', so even if movements are planned correctly and accurately (calculating the right amount of force to apply, etc), the brain needs to monitor the neural systems to check if these systems are going off course, and to reset them if they are.

ii) Feedforward

A feedback system can work in isolation, detecting errors and compensating for their effects. In comparison, feedforward systems use information from a component that may introduce error to anticipate the effort itself. This system sends information ahead to whatever has to deal with the potential difficulty so that it can prepare to compensate for or accommodate it. For example, the vestibular-ocular reflex translates head velocity into compensatory eye velocity. Head movement introduce distortions into vision, so the feedforward mechanism notices the head motion and triggers eye movements to cancel out any motion blur before it even occurs.

iii) Forward modeling

Some movements need correcting during their execution at a rate quicker than is possible with simple feedback. One way of doing this is to predict the effect of a signal from the brain to a muscle (this is why we cannot tickle ourselves). The prediction may then be used as pseudo-feedback to control movements at a speed faster than would be possible with actual sensory feedback (Stafford & Webb 2005:157). This principle was already described in 1890 by William James (Berthoz 2000:10-11).

4.5.2 Brain-based activity that impacts on movement

Every time a movement is made, the brain generates an efferent copy of the actual motor command in parallel. The efferent copy is just like a carbon copy or duplicate of the real motor command, and is used to make a prediction about the effect of the action (Stafford & Webb 2005:211). The predicted sensory effect of the efferent copy and the actual sensory effect of the motor command are compared. If there is a mismatch, the sensation is labeled as externally generated (Stafford & Webb 2005:211).



Mirror neurons, a type of nerve cell found in the brain, are one of the most exciting discoveries of recent years. A particular mirror neuron is activated when we carry out an action; and exactly the same nerve cells respond when we see someone else carrying out that same action. We use mirror neurons to map other people's actions directly onto our own bodies. This innate programming vastly facilitates learning by imitation – we do not first have to learn how to imitate (Winston 2004:155).

Neural pathways are neural tracts connecting one part of the nervous system with another. They usually consist of bundles of elongated, myelin-insultated neurons known collectively as white matter. Neural pathways connect relatively distant areas of the brain or nervous system, compared to the local communication of grey matter. Most neural pathways are made up of axons. Some neurons are responsible for conveying information over long distances. A good example is motor neurons, which travel from the spinal cord to the muscle. These pathways could have axons up to a metre in length in human

(http://en.wikipedia.org/wiki/Neural_pathways).

4.6 Differential Perspective on Movement and Motor Control

Much like attention, movement can be studied from a **neuroanatomical**, **neurochemical** or **neurophysiological** perspective. The *neuroanatomical* approach focuses on the location of brain areas that underlie those systems thought to mediate the regulation of movement and inhibit motor activity. The *neurochemical* approach addresses the role of specific neurotransmitters that facilitate communication among the neuronal circuits. The *neurophysiological* perspective attempts to explain the dynamic interaction and interplay between the neurochemical and anatomical components that together form a functional system.

The nervous system (NS), along with the endocrine system, provides the control functions of the body. In general the NS controls the rapid activities of the body, such as muscular contractions, rapid-changing visceral events, and even rates of secretion of some endocrine glands. The nervous system is unique in the vast complexity of the control actions that it can perform. It can receive literally thousands of bits of

information from different sensory organs and then integrate these to determine the response to be made by the body (Guyton 1972:5).

4.6.1 Neuroanatomical perspective

In order to understand heightened motor activity from a neuroanatomical perspective it is important to first obtain an overview of the three major levels of the central nervous system (CNS) and how it controls motor movement.

4.6.1.1 Hierarchical levels

The human CNS has inherited specific characteristics from each stage of evolutionary development. From this heritage, there remain three major levels of the nervous system that have special functional significance: (1) the spinal cord level, (2) the lower brain level, and (3) the higher brain or cortical level. The responses to nervous system responses may be involuntary or voluntary. This can best be illustrated in figure 9. Autonomic nervous system responses are automatic; they control the body's internal environment and help to regulate vital functions such as blood pressure. The two types of autonomic nervous system, sympathetic and parasympathetic, have opposite effects. The autonomic nervous system and its involvement in stress will be discussed in detail in the next chapter.



Figure 9 Hierarchical level of response

(Winston 2004:114)

Figure 9 clearly illustrates the different levels at which an organism may respond. The first figure illustrates an **autonomic response**. Information collected by internal receptors travels along sensory nerves to the spinal cord and the brain stem. Sympathetic and parasympathetic response signals have separate pathways. The second figure shows a **voluntary response**. Here sensory impulses that are responsible for triggering voluntary responses are dealt with in many areas of the brain, and they travel through complex nerve pathways. The third figure shows a **reflex**. Although some reflexes are processed in the brain, most are processed in the spine. This is the simplest nerve pathway, as the sensory and motor neurons are linked in the spinal cord (Winston 2004:114). Reflexes are discussed in detail in section 4.6.3.1 of this chapter.

i. The spinal cord level

The spinal cord of a human being still retains many functions of the multi-segmental animal. Sensory signals are transmitted through the spinal nerves into each segment of the spinal cord, and these signals may cause localised motor responses either in the segment of the body from which the sensory information is received, or in adjacent segments. Essentially, all spinal cord motor responses are automatic and occur almost instantaneously in response to the sensory signal. In addition, they occur in specific patterns of response called **reflexes** (Guyton 1972:7).

Reflexes mainly affect muscles normally under voluntary control, but are involuntary responses to stimuli (Winston et al. 2004:114).

ii. The lower brain level

Many of what we call subconscious activities of the body are caused by the lower areas of the brain – the medulla, pons, mesencephalon (midbrain), hypothalamus, thalamus, and basal ganglia. Subconscious control of arterial blood pressure and respiration is achieved primarily in the reticular substance of the medulla and pons (Guyton 1972:9).

The reticular formation is also known as the reticular activating system (RAS), and as the reticular alarm system. It occupies the central most parts of the brain stem, and extends downwards into the spinal cord to the sacrum (Upledger 1996:169). This

system is a network of intertwined and interconnecting nerve cell bodies and fibres that connect with all the major neural tracts going to and from the brain. This system communicates particularly with the nuclei of the motor nerves (cranial nerves III and IV) to the eye, and with the substrantia-nigra (Upledger 1996:169). The reticular formation is not a single functional unit (very non-specific in its functioning), but contains many nerve centres or nuclei (several million clusters of functionally-related nerve cells). The RAS receives input from the proprioceptive areas of all the muscles and tendons, from the sensors of balance and equilibrium, from the eyes, the ears, the touch receptors, the smell receptors, the taste receptors, and any other receptors in the body.

These nerve centres mediate important and complex postural reflexes, contributing to the smoothness of muscle activities, and maintain muscle tone. The RAS is the part of the network that regulates wakefulness and alerting mechanisms that ready the individual to react (Lezak 1995:48). Not only does it receive information from all the receptors, it also receives particular processed information from the cerebellum, the hypothalamus, the basal ganglia and the cerebral cortex (Upledger 1996:130).

iii. The higher brain or cortical level

The cerebral cortex is primarily a vast information storage area. The brain is a wonder of processing. Information comes into the brain from the external world through the senses and is converted to an extremely complex mix of electrical and chemical energy. The four lobes of the brain, as well as myriad areas across the surface of the cortex, communicate with one another constantly, and the brain accomplished this by means of a vast assembly of tiny electrical devices: the neurons (Robbins 2000:29).

Approximately three quarters of all the neuronal cell bodies of the entire NS are located in the cerebral cortex. It is here that most of the memories of past experiences are stored, and it is here that many of the **patterns of motor responses** are kept, and from where information may be called forth at will to **control motor functions** of the body. The cerebral cortex is actually an outgrowth of the lower regions of the brain, particularly of the thalamus. For each area of the cerebral cortex there is a corresponding and connecting area of the thalamus, and activation of the

minute portion of the thalamus activates the corresponding and much larger portion of the cerebral cortex. In this way, it is presumed that the thalamus may call forth cortical activities at will (Guyton 1997:10).

Some areas of the cerebral cortex are not directly concerned with either sensory or motor functions of the CNS – for example, the prefrontal lobe and large portions of the temporal and parietal lobe. These areas are set aside for the more abstract processes of thought, but even they have direct nerve connections with the lower regions of the brain.

Cells within relatively well-defined cortical areas have a significant number of components outside the local cortical centre. Of the cortical cells underlying voluntary movement (the primary motor cells), less than 40% are situated in the primary motor cortex, whereas up to 40% are situated in the parietal lobes, mostly within the primary somatosensory area (Lezak 1995:56) as shown in figure 27.

4.6.1.2 Brain structures involved in motor activity

In the outline of the CNS above, it is clear how many areas of the brain house motor patterns, motor responses and motor control. The spinal cord initiates localised motor responses, called reflexes, which are usually automatic and instantaneous.

Within the lower brain, coordinated motor control is made possible through the cerebellum and reticular substance of the medulla, pons and mesencephalon. The pons and cerebellum also regulate muscle movement. On the higher brain level we find planned motor activity in the form of volumetric movement.

Some researchers have documented the relationship between the hyperactivity/impulsive aspects of AD/HD and anterior or frontal lobe functioning, based on neurocognitive tasks, whereas others have explored the relationship between the inattentional aspects of AD/HD and right hemisphere parietal (posterior) lobe functioning. In view of these results, Schaughency and Hynd (1989) posit that an anterior (frontal)-posterior (parietal) gradient may well exist in the differential effects (attention deficit disorder with hyperactivity vs. attention deficit disorder

without hyperactivity) of these separate yet correlated systems (Riccio et al. 1993:122).

i. Basil ganglia

Situated at the base of each cerebral hemisphere are a number of nuclear masses known as the **basil ganglia**. The largest is the **corpus striatum** consisting of several complex **motor correlation centres** (the caudate and the putamen) that modulate both voluntary movement and automatic reaction. These motor nuclei have many interactions with the frontal cortex contributing to what is called a "primitive motor programme … basic blueprints providing the underlying structure for all movements" (Lezak 1995:54). Hyperactivity has appeared to follow basal ganglia infarcts.

Evidence to support possible dysfunction of the **caudate** includes reduced **cerebral blood flow**, particularly in the right caudate (Thompson & Thompson 2003:115; Riccio et al. 1993:121-122), which influences motor regulation and behaviour inhibition. However, the studies are correlational and do not indicate causality. This statement could be interpreted in many ways and the question could be asked: Do we see less cerebral blood flow in the caudate because of the inability to regulate motor activity, or do we see lower cerebral blood flow due to a dysfunction in the caudate?

ii. Cerebellum

The role of the cerebellum is extremely important for motor control. This has been illustrated by researchers such as Ito (1984, 1987) and Nicolson and Fawcett (2005). Insight into the relationship between the cerebellum and motor activity has been successfully ulitised in therapy by practitioners such as Ayres (1974, 1979, 1982), Bluestone (1994, 2000, 2004), Goddard (1996), Hannaford (1995), Kokot (2003b:11-24), Fredericks, Kokot and Krog (2006), Levinson (1990), and Pheloung (2003).

Control of equilibrium (balance) is a combined function of the older portions of the cerebellum and the reticular substance of the medulla, pons and mesencephalon (midbrain). The **cerebellum** (a Latin word that literally translates to "little brain") is a region of the brain that plays an important role in the integration of sensory

perception and motor output. Many neural pathways link the cerebellum with the motor cortex. Pathways send information to the muscles, resulting in movement. This action is also known as the spinocerebellar tract and provides feedback on the position of the body in space (proprioception). The cerebellum integrates these pathways, using the constant feedback on body position to fine-tune motor movements (http://en.wikipedia.org/wiki/Cerebellum).

The pons and cerebellum (which are both situated high in the hindbrain) correlate posture and kinaesthetic (muscle movement sense) information, refining and regulating motor impulses relayed from the cerebrum at the top of the brainstem. The coordinated turning movements of the head, the body, and the eyes are controlled by specific centres located in the mesencephalon (midbrain), paleocerebellum, and lower basal ganglia (Guyton 1972:9). Cerebellum damage is commonly reflected in problems with fine motor control, coordination and postural regulation. Dizziness (vertigo), the inability to balance, and jerky eye movements may also accompany cerebellar damage (Lezak 1995:49; Scheiman 2002:13).

The cerebellum has more neurons than the rest of the brain combined, and some of those neurons are broad branching with an enormous number of connections. So although the cerebellum is physically small compared to the rest of the brain, it is enormous in its potential for processing information (Kalat 2001:237). Due to the number of neurons, its broad connectivity base and incredible processing speed, the cerebellum is multi-functional. It is thus a complex structure, and different areas may perform different functions. Ito (1984) proposes that one key role is to establish new motor programmes that enable people to execute a sequence of actions as a whole instead of waiting for feedback. The cerebellum thus appears to be involved in habit formation, timing, and certain aspects of **attention** and **balance** (Kalat 2001:238).

Due to the importance of the cerebellum in the ability to move, basic structures and their functions are reviewed below. The structures are illustrated in figure 10 and are discussed as published on Wikipedia (http://en.wikipedia.org/wiki/Cerebellum).

The archicerebellum is associated with the flocculonodular lobe and is mainly involved in vestibular and eye movement functions via the cranial nerves. It receives input from the inferior and medial vestibular nuclei and sends fibres back to the vestibular nuclei, creating a feedback loop that allows for the constant maintenance of equilibrium (balance). The paleocerebellum controls proprioception related to muscle tone (constant, partial muscle contraction that is important for the maintenance of posture). The paleocerebellum receives its inputs from the dorsal and ventral spinocerebellar tracts, which carry information about the position and forces acting on the legs. The paleocerebellum then sends axonal projections to the deep cerebellar nuclei. The neocerebellum receives input from the pontocerebellar tract and projects to the deep cerebellar nuclei. The pontocerebellar tract originates at the pontine nuclei, which receive their input from the cerebral motor cortex. Thus, the neocerebellum is associated with motor control, in particular, the coordination of fine finger movements such as those required by writing and manipulation. When a patient presents with a balance and movement disorder, there is often a problem with integration of the visual, vestibular, and somatosensory systems. Scheiman (2002:193) states that somatosensory and vestibular integration, rather than the visual system, should control balance.



Figure 10

The functional organization of the cerebellum

Schematic representation of the major anatomical subdivisions of the cerebellum. Superior view of an "unrolled" cerebellum, placing the vermis in one plane (http://en.wiki/Cerebellum)



The vermis receives its inputs mainly from the spinocerebellar tracts that are situated in the trunk of the body. These tracts carry information to the vermis on the position and balance of the torso. The vermis sends projections to the fastigial nucleus of the cerebellum, which then sends output to the vestibular nuclei. The vestibular nuclei are structures that are important for the maintenance of balance. The intermediate zone (or paravermis) input receives from the corticopontocerebellar fibres that originate in the motor cortex. These fibres carry a duplicate of the information that was sent from the motor cortex to the spine in order to effect a movement. The intermediate zone also receives sensory feedback from the muscles. These two streams of information are integrated by the intermediate zone, allowing for the feedback comparison of what the muscles are supposed to be doing with what they are actually doing. This feedback loop is crucial as this allows the individual to modulate motor activity to the desired activity if necessary.

The **lateral zone** receives input from the parietal cortex via pontocerebellar mossy fibres regarding the location of the body in the space. The large numbers of feedback circuits allow for the integration of the body's orientation with indications of muscle position, strength, and speed. The four **deep cerebellar nuclei** are in the centre of the cerebellum, embedded in the white matter. These nuclei receive **inhibitory** inputs from Purkinje cells (Ito 1984, 1987, pioneered research on these cells) in the cerebellar cortex and **excitatory** inputs from mossy fibre pathways. Most output fibres of the cerebellum originate from these nuclei. One exception is that fibres from the flocculonodular lobe synapse **directly on vestibular nuclei** without first passing through the deep cerebellar nuclei. The vestibular nuclei in the brainstem are analogous structures to the deep nuclei, since they receive both mossy fiber and Purkinje cell inputs.

Comment

It seems as though most of the areas of the cerebellum are involved in motor activity and control. The cerebellum and the vestibular system have a direct neural link. This link will prove important in the work to follow and will be explored in later sections, with the option to use this critical link as a therapeutic tool.

iii. Premotor cortex

Situated just anterior to the precentral area, the premotor cortex or secondary motor association area, has been identified as the site where **motor skills** and learned action sequences are integrated. Lesions in this area do not result in a loss of the ability to move, but rather disrupt the **integration** of the motor components of complex acts, producing discontinuous or uncoordinated movements and impaired motor skills (Lezak 1995:88).

iv. The prefrontal cortex

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. Stuss and Benson (cited in Lezak 1995:39) state that "the human prefrontal cortex attends, integrates, formulates, executes, monitors, modifies, and judges all nervous system activities"; while Lezak (1995:48) refers to it as the "seat of consciousness."

4.6.2 Neurochemical perspective

Skeletal muscle cells must be stimulated by nerve impulses in order to contract. One motor neuron (nerve cell) may stimulate a few muscle cells or hundreds of them, depending on the particular muscle and the work it does. One neuron and all the skeletal muscle cells it stimulates form a motor unit. When a long threadlike extension of the neuron, called the nerve fibre or axon, reaches the muscle, it branches into a number of axonal terminals, each of which forms junctions with the sarcolemma of a different muscle cell. These junctions are called neuromuscular junctions. Within the synaptic cleft (opening between dendrite terminals) a neurotransmitter is released. The specific neurotransmitter that stimulates skeletal muscle cell is acetylcholine. Acetylcholine diffuses across the synaptic cleft and attaches to receptors that are part of the sarcolemma. If enough acetylcholine is released, the sarcolemma at the point becomes temporarily permeable to sodium ions, which then rush into the muscle cell. This sudden inward rush of sodium ions gives the cell interior an excess of positive ions, which upset and charge the electrical conditions of the sarcolemma. This "upset" generates an electrical current called an action potential. Once begun, the action potential is unstoppable; it travels



over the entire surface of the sarcolemma, conducting the electrical impulse from one end of the cell to the other. The result is a contraction of the muscle cell (Marieb 2003:169).

In the previous chapter the link between catecholamines (dopamine, norepineprine) and their connection to inhibition and the response of the motor system were discussed. Dopaminergic activity may thus impact on disorders of planned and automatic activity (Thompson & Thompson 2003:113; Riccio et al. 1993:121).

Comments

The information presented thus far addresses several fundamental aspects of human functioning. Obviously movement plays an important role. Thought without execution is worthless. Accurate execution implies modulation of activity. However, modulation and movement are all aspects that rely on chemical actions between neurons. It becomes clear why chemical action is said to lie at the core of human functioning. In addition, the structure and location of neurons play an equally important role. The development of brain structures and neural growth are dependent on and driven by neurotransmitters and the chemical reaction of these neurotransmitters. Brainwave activity (as measured by an EEG) is driven by neurotransmitters, as is the activity of brain structures. Figure 6 illustrated the precursor essential amino acids necessary for the manufacturing of neurotransmitters. Most amino acids are proteins and therefore readily available in good food sources. Could an intervention programme based on nutrition (supporting the production of neurotransmitters) and movement (that stimulates the release of neurotransmitters) potentially form the core of a natural approach to addressing attentional difficulties and heightened motor activity? A possible new approach is starting to emerge from the literature presented. This possibility is explored and tested in the chapters to follow.

4.6.3 Neurophysiological perspective

Neurophysiological perspectives on movement and motor control involve the combined effect of anatomical and chemical actions. Obviously, when any one of these actions malfunctions, it impacts on the physiological aspect.

4.6.3.1 Reflex system

As physiology is the functional outcome of anatomy and chemistry, this section focuses mainly on specific functional movement patterns (reflexes). Here we examine the development and maturation of reflexes and the whole reflex system.

There are certain activities that map general neurodevelopment. One group of activities that has been used for this purpose is reflexes. A short description follows of how reflexes can serve as markers for general developmental delay. The concept of neurodevelopmental delay is specifically mentioned here as a map of the functional outcome of the combination of anatomy and chemistry. The different cortical areas involved in attention and motor activity have been clearly mapped. If these cortical areas show any sign of immaturity, the ability to pay attention and control motor movement will obviously be affected.

Essentially, three body units – neck, arms, and legs – are linked through reflexes, so that movement in one area **automatically** produces a change in the muscular tension of the other two areas (O'Dell & Cook 1997:1). In the beginning, all essential movements made by the newborn child are reflexes. Motor development occurs as the child gains cortical control over the movement of their bodies. Children are not born coordinated; coordination develops through three basic levels (O'Dell & Cook 1997:14):

- 1. Reflex motor (automatic movement)
- 2. Gross motor (large muscle movement)
- 3. Fine motor (small muscle movement)

The reflex level provides the basis, or foundation, for all motor development. Reflex development comes first, followed in order by gross and fine motor development. Within this system, as with the rest of brain development and maturation, a hierarchical structure exists. If any one of these levels is not properly developed, there will be some form of disturbance or malfunctioning in the development of the levels above. Proper reflex development, therefore, is basic and necessary to complete motor development (O'Dell & Cook 1997:14). As previously mentioned, the brain instructs movement; the execution of these specific movement patterns in turn

structures the brain, and thus prepares the brain to mature to the next level. The process of reflex development may be explained as follows.

The newborn infant is essentially a reflexive organism. He survives because he breathes, roots, sucks, swallows, coughs, sneezes, cries and eliminates waste reflexively. To survive, the child is equipped with a set of **primitive (or primary) reflexes** designed to ensure immediate responses to his new environment and to his changing needs. He moves his arms, legs, and head in reflex responses to external and internal stimuli. Each time the infant responds reflexively by movement, feedback from the movement and the body position reaches the brain by way of the proprioceptive system. This sensory system provides a continuous stream of information about the movement and position of the body and its parts in relation to one another and gravity. As the infant continues to integrate primitive reflex patterns and recombine their parts into voluntary movement and patterns of coordinated movement, the infant begins to override and suppress the more primitive of these reflexes. As the child achieves some skill in a more efficient method of information processing, he is free to abandon his least efficient method (Bender 1976:12), in this case the set primitive reflex.

Figure 11 shows the different ages of emergence and suppression of some of the primitive reflexes.



Figure 11 Primitive reflex profile

(Goddard 1996:3)



Figure 12 Reflex response

Primitive reflexes are automatic, stereotyped movements (we know exactly how they look and thus know how to detect them) that are directed from the brain stem (primitive brain areas – lower level functioning) and executed without cortical involvement (no input from higher level functioning) conscious awareness is possible only when the cortex becomes involved in the event (Goddard 1996:1; Rose 2005:119).

From the hierarchical model presented earlier, it is clear that the lower structures of the brain are fully functional at birth. These lower structures are sometimes referred to as the 'primitive brain' because they are responsible for primitive activities that do not require cortical involvement. For this reason, the cortex is not evenly

developed at birth. Rose (2005:120) states that the one month-old infant is still a subcortical organism.

The following section, taken from Goddard (1996:1-3), outlines the importance of the suppression of these reflexes. If primitive reflexes remain active beyond six to twelve months of life, they are said to be **aberrant**, and they are evidence of a structural weakness or immaturity within the central nervous system (CNS). Prolonged primitive reflexes may also actively prevent the development of the succeeding postural reflexes, which should emerge to enable the maturing child to interact effectively with his or her environment. If retained beyond six months of age, primitive reflexes may result in immature patterns of behaviour or may cause immature systems to remain prevalent, despite the acquisition of later skills.

Depending on the degree of aberrant reflex activity, this poor organisation of nerve fibres may affect one or all areas of functioning: not only gross and fine muscle coordination, but also sensory perception (registering of sensory information in the brain), cognition (interpretation and understanding of that information) and avenues of expression. When primitive reflexes remain, fundamental equipment essential for learning will be faulty or ineffective despite adequate intellectual ability. It is as if later skills remain tethered to an earlier stage of development and instead of becoming automatic, may only be mastered through continuous conscious effort.

The inhibition or suppression of reflexes occurs when the first function ceases through the development of another function. The first function becomes integrated in the second, and the suppression of a reflex frequently correlates with the acquisition of a new skill. Thus, knowledge of reflex chronology and normal child development may be combined to predict which later skills may have been impaired as a direct result of retained primitive reflexes. Thus the individual's aberrant reflexes may provide clues as to what is actively hindering the mastery of later skills.

If a cluster of aberrant reflexes is present, **neurodevelopmental delay** is said to exist. The knowledge and interpretation of aberrant primitive reflexes does not only map the neurodevelopmental delay, but also provides valuable knowledge into a possible therapeutic intervention programme to address the neurodevelopmental delay. This is possible since a baby engages in specific movement patterns to suppress the primitive reflexes.

If it is the primitive reflexes that lay the foundations for all later functioning, it is the **postural (or secondary) reflexes** that form the framework within which other systems may operate effectively. The transition from primitive reflex reaction to postural control is not an automatic one. There are no set times at which the later reflexes assert control over the earlier ones, but it is a gradual process of interplay and integration during which both kinds of reflexes operate together for a short period of time. This period of growth, change and elaboration, operates rather like an interweaving spiral, through which nature ensures that primitive survival patterns are still accessible until such time as more mature postural reactions are becoming automatic (Goddard 1996:23). Postural reflexes are mediated from the level of the midbrain, and their development thus signifies the active involvement of higher brain structures over brain stem activity. As such, the development of postural reflexes is a sign of increased CNS maturity.

Thelan (1979:699) observed that all human babies make a series of stereotyped movements during their first year of life. These movements contain within them a

natural reflex inhibitor, and if a child never makes these movements in the correct sequence, the primitive reflexes may remain active as a result. Again, knowledge of this neural inhibitor may prove to be valuable, as presenting the exact stereotypical movement may actually trigger the natural inhibitor and suppress the reflex. By the application of stylised sequential movements, practised daily, it is thus possible to give the brain a "second chance" to register the reflex inhibitory movement patterns which should have been made at the appropriate stage in development. Goddard (1996:3) believes that as aberrant reflex activities are corrected, many of the physical, academic and emotional problems of the child will disappear, as general CNS functioning matures.

The stages of cortical growth and the types of movements are inextricably linked. Continuous cortical development is facilitated through these movement sequences, which lay down efficient neural pathways. Draper (cited in Goddard 1996:40) states that practice and repetition of movement patters result in the pathways being absorbed into the individual's repertoire of skills, probably also resulting in physical changes taking place in the neurons concerned, so that the flow of impulses along a specific pathway is facilitated. Repetition enhances facilitation and adeptness.

This section discussed how the reflex system may affect performance at the levels of processing and response, illustrating how brain stem reactions direct responses without higher brain level involvement. Equally, distorted sensory input may awaken a new reflex activity, which would otherwise be inhibited, and thus a vicious circle of distorted sensation and inappropriate response is established (Goddard 1996:41). For the purposes of this study, the focus will be on the profile of primitive reflexes because a cluster of these reflexes is indicative of neurodevelopmental delay.

4.6.3.2 EEG brainwave activity

In section 3.4.3.2 various bandwidths were discussed, and the effect of these bandwidths on consciousness and attention was illustrated in table 4. Apart from the effect that these EEG bandwidths have on attention, they also affect motor activity and movement.

Thompson and Thompson (2003:39) write about a specific bandwidth called **sensor-motor rhythm (SMR)**, which measures 13-15Hz when it is measured across the sensor-motor strip. Sterman named this range in 1967 after he conducted research with cats and epilepsy (Robbins 2000:39). It has a very specific spindle-like waveform produced in the ventral-basal nucleus of the thalamus. According to Sterman (in Robbins 2000:39), the person producing SMR is mentally alert without muscles being tense. SMR therefore appears to be associated with a calm mental state with increased reflecting-before-acting activity. Children presenting with impulsivity and hyperactivity or heightened motor activity lack high amplitude SMR across the sensor-motor strip.

As with neurodevelopment, understanding the processes involved in normal processes can actually guide the professional to render appropriate therapeutic interventions by applying exactly what should have been there in normal development and functioning in the first place. The same is true in the field of EEG neurofeedback. When lower amplitude SMR is measured across the sensory motor strip, a general protocol would be to increase or stimulate SMR across this strip with the help of EEG neurofeedback equipment (Robbins 2000:39) to facilitate the physical activity level associated with increased SMR over the sensory-motor strip. This spindle-like waveform is more apparent on the right hemisphere across the sensory-motor strip and the therapy would thus then focus on the right hemisphere in the protocol. EEG neurofherapy is discussed in detail in chapter 5, section 2.3.

Comment

Once again, as with the maintenance of attention discussed in section 3.4.1.2, the right hemisphere is implicated in movement more than the left hemisphere.

5. Conclusion

A full account of attention and movement has been presented in this chapter. The information illustrates the need to view so-called AD/HD within a broader framework. The undeniable connection between the body and the brain (body/brain) presents itself in the interaction of the effect of movement on the brain (releasing neurotransmitters, growth of pathways through movement, plasticity) and the effect of

the brain on movement (orchestrating movement). One cannot help to think about the effect of the seventeenth century philosopher, Rene Descartes' notion of the idea of **dualism**, the idea that mind and body are separated. Would research have looked different today if this notion had not dominated research for so long?

Chapter 4

An Ecosystemic Conceptualisation of Attentional Difficulties and Heightened Motor Activity

1. Introduction

In the previous chapter the discussion of the literature revealed that attention and motor movement are governed in both hemispheres (albeit more in the right) in most of the brain structures in the brain and on all levels of the CNS. Information was also presented that describes attention and movement in terms of neuroanatomy, neurochemical and neurophysiological activity. Dopaminergic and noradrenergic influences were investigated as well as the effect of these chemicals in specific cerebral locations in terms of EEG activity. This has great implications in terms of the diagnostic procedures recommended by the DSM-IV-R. If a diagnosis were made according to the DSM-IV-R, what then would be the correct procedure of treating this disorder in view of the information presented in chapter 3? The focus on treatment to date has been chiefly neurochemical (use of medication), ignoring to a great extent the other possible aspects involved as well as the interconnectiveness and interrelatedness of this disorder on all three levels (anatomical, chemical and physiological). Since no clinically approved laboratory test is available to determine that the neurochemical approach is always appropriate, it raises the question of why this approach is the principal approach followed if the literature clearly provides enough evidence that there is more to attentional difficulties and heightened motor activity than an imbalance of neurotransmitters. One may also ask why there seems to be an imbalance of these neurotransmitters in the first place? Clearly a more responsible approach, such as a full investigation into the possible underlying causes of inattention and heightened motor activity, is warranted.

In such an alternative approach, another way of looking at the presenting symptoms may be more appropriate. A more responsible approach may be to view the relationship between the cause of disorders and symptoms, which could be illustrated as a river delta (figure 13).



Illustrated causal effect

(Adapted from Tansley 1997:87)

The outlets of the river can be regarded as symptoms, each of which may be seen as a manifestation of the organism's endeavors to compensate for a dysfunction further up the river. Treatment of a symptom alone often results in a worsening of other symptoms or the production of new ones (Tansley 1997:87). A new approach may involve taking a presenting symptom and tracing the underlying causal pattern. The most immediate cause for symptom 2 is cause F and the most immediate cause for F is E and so on until cause A is reached, which is the underlying cause of all the symptoms. Unfortunately, in view of the scope of this thesis, the investigation did not travel all the way up the river. However, it does venture into some branches and finds interesting correlations to some of the symptoms described in the DSM-IV-R. This chapter focuses on these alternative brances to try and move closer to point A.



2. A New Conceptual Framework: An Ecosystemic Approach

From the literature study, a new conceptual framework is starting to emerge in order to explain and define inattention and heightened motor activity. This new framework will attempt to incorporate the presented information as well as search for new information outside the traditional framework, to create a new understanding of AD/HD and to suggest possible new methods of treatment and intervention.

From the information presented, it became apparent that the neuroanatomical, neurochemical and neurophysiological aspects are interrelated, interdependent and ultimately integrated as a unified whole. Accordingly to the researcher, these three aspects address the core essence of human functioning. In this study, the term 'ecosystemic' incorporates all three these aspects, as well as any combination of the three aspects. Ecosystemic refers to both the internal and external ecosystems of the human organism. The internal ecosystem refers to the internal anatomical, chemical and physiological aspects that contribute to biological and emotional aspects of behaviour, while external ecosystem implies the social aspects and external surroundings (physical, chemical, etc.) of an individual. An attempt to illustrate this model is presented in figure 14.



Figure 14

The three main aspects included in this approach form an interlocking system. One cannot function without the other. Chemical working functions affect anatomy (initial development of structures); anatomy and chemical activity produce physiological outputs. Physiological output in turn results in the release of neurotransmitters and firing of neurons at specific locations, resulting in structural changes in the brain. All three aspects (and the various combinations of the aspects) affect attention and movement (motor activity). Since this study aims to find possible alternative interventions to address inattention and heightened motor activity, the focus is directed by the following question: How can the information presented assist in designing a functional intervention programme? How can the chemical functioning of the brain be addressed naturally? How can the structural aspects of the brain be altered naturally to produce more effective production of attention and motor activity? How can overall physiological output (to be able to pay attention and to control motor activity) be appropriately modulated? Could our knowledge of basic neurodevelopment and effective nutrition provide an answer; and if so, how can this be applied effectively? These questions are explored in the following chapter.

2.1 Neurodevelopment

Levine (2002, 2005) describes a model of neurodevelopment that includes systems such as attention control, temporal-sequential ordering, spatial ordering, memory, language, neuromotor functions, social thinking and higher order cognition. Although these components do relate to cognitive functions and in essense neurodevelopment, this study mostly refers to neurodevelopment as a lower order developmental system and not the higher order functional outcomes as included in Levine's model van neurodevelopment.

The study of **neural development** draws on both **neuroscience** and **developmental biology** to describe the cellular and molecular mechanisms by which **complex nervous systems** emerge during **embryonic development** and throughout life. Developmental biology is the study of the process by which the organism grows and develops. Modern developmental biology studies the **genetic control of cell growth**, **differentiation** and **morphogenesis**, which is the process that gives rise to tissues, organs and anatomy. Some landmarks of embryonic neural development include the birth and differentiation of neurons from stem cell precursors, the migration of immature neurons from their birthplaces in the embryo to their final positions, the outgrowth of axons from neurons and guidance of the motile growth cone through the embryo towards postsynaptic partners, the generation of synapses between these axons and their postsynaptic partners, and finally the lifelong changes in synapses which are thought to underlie learning and memory, and form the basis of plasticity. Typically, these neurodevelopmental processes may be broadly divided into two classes: **activity-independent mechanisms** and **activity-dependent mechanisms** (http://en.wikipedia.org/wiki/ Neurodevelopment).

Activity-independent mechanisms are generally believed to occur as hardwired processes determined by genetic programmes and played out within individual neurons. These include differentiation, migration and axon guidance to their initial target areas. These processes seem to be independent of neural activity and sensory experience. Once axons reach their target areas, activity-dependent mechanisms come into play. Neural activity and sensory experience mediate the formation of new synapses, and are responsible for synaptic plasticity, which refine the nascent neural circuits (http://en.wikipedia.org/wiki/Neurodevelopment). Schematically, these concepts may be plotted as follows (figure 15).



Basic mechanisms involved in neurodevelopment

Within the context of neurodevelopment, Rose (2005:23) asks two questions:

- How does the dynamics of development account for the seeming invariance of the human brain, progressing from egg to embryo to fetus to child with such extraordinary precision?
- 2. How can it explain the differences between brains, which have so developed?

Neurodevelopment follows a **precise**, **specific sequence**, and yet, no two brains are alike even if they followed this sequence at exactly the same time and in exactly the same environment. Both questions are immanent in the process of **autopoiesis** (developmental systems theory). Rose (2005:63) explains his understanding of autopoieses as invariant development within a fluctuating environment, which is termed **specificity**. The variations that develop as adaptations to environment contingencies are referred to as **plasticity**.



Much of what needs to be understood about the brain is embraced by these two intertwined processes, a so-called developmental double helix. Without specificity, the brain would not be able to become accurately wired. However, without plasticity, the developing nervous system would not be able to repair itself following damage, or to mould its responses to changing aspects of the outside world. In so doing, the brain creates a model or representation of the outside world, and formulates a plan of how to act upon it. In other words, without plasticity, learning will not take place. It is **specificity** and **plasticity** rather than nature and nurture alone that provide the dialectic within which development occurs. Both processes are dependent on both **genes** (nature) and **environment** (nurture) (Rose 2005:64). Combined, it becomes clear that activity could be seen as **plasticity**.

The following section focuses on both these concepts within the notion of autopoiesis. Specificity is discussed in terms of the specific, sequential neurodevelopmental processes followed. Plasticity is discussed with regard to the

neurodevelopmental moulding process. Two additional concepts are added to fully understand neurodevelopment, namely, **stress**, and more specifically (in this context) **physiological stress** and **information processing systems**.

2.1.1 Specificity: The neurodevelopmental process

The brain is a highly ordered structure; neurons have to know their place, to recognise to whom they are supposed to be talking, and ensure that dendrites and axons make proper connections (Rose 2005:65). The packing of neurons (migration) – their interactions, their interconnectedness – determines who talks to whom. The effectiveness of a synapse (independent of how much neurotransmitter it releases) depends on its address and geometry – in other words, the neurons must be at the right location. Because neuronal geometry determines connectivity, the morphology of the dendrites themselves, with their many branches, adds to the complexity of the computations they make of the synaptic voices reaching them, even before their summation at the axon hillock (Rose 2005:146).

The specificity aspect of neurodevelopment entails the developmental sequence (maturation) within a preset time frame. Maturation and time are both linear processes, and in developmental terms are always linked with one another. Figure 17 demonstrates the timeline during the first 40 days after conception, and the relationship of the specific development to the ultimate adult version of the brain. A short description of the neurodevelopmental process is presented here, with specific reference to the time frames in which development occurs.

Because the basic elements of the nervous system are formed during the first month of embryonic development, any maternal infection early in pregnancy may have extremely harmful effects on the foetal nervous system (Marieb 2003:243). **Neurulation** (the earliest brain tissue that forms) begins as early as 19 days after fertilisation (Eliot 1999:15).





http://soma.npa.uiuc.edu/courses/bio303/Ch3.html

The antogeny/phylogeny relationship explains why complete development of the nervous system takes so long, especially when compared to the development of the organs. This relationship also helps explain the regional development of the nervous system. The fact that brain structures that control more rudimentary functions, such as breathing and feeding, mature earlier than regions that control more sophisticated functions like language and reasoning. At five weeks the initial three brain structures enlarge and further subdivide to make five structures. The front-most part or telencephalon (anterior part of the forebrain) begins to divide along the midline to form distinct left and right hemispheres. By six weeks, the swelling has begun differentiating into all the major brain structures, creating the rudiments of the pons, medulla, cerebellum, thalamus, basal ganglia, limbic system, and cerebral cortex. At this point the twelve cranial nerves that will transmit sensory and motor information between the brain and eyes, ears, nose, face, mouth, and other body structures have made their first appearance, although they are not yet connected to the face and other target organs (Eliot 1999:17). An illustration of the twelve nerves is given in figure 18.



Figure 18 Cranial nerves

(Winston 2004:113)

In the first eight weeks of pregnancy, a structure called the embryonic disc forms from the fertilised egg. The disc develops a series of folds that yield head and limb buds, and a stalk that eventually becomes the umbilical cord. A thin sheet of cells in the chest starts to flutter irregularly, to later become the heart. At eight weeks, the internal organs are formed. Although immature, they are developing rapidly, and the embryo becomes a foetus. By 12 weeks, the foetus measures around six centimetres from the head to the buttocks (Winston 2004:140).



At the beginning of the foetal period (nine weeks), the spinal cord is well formed and is even beginning to function. By 13 weeks a prominent thalamus (critical sensory relay station) is clearly identifiable behind two small, thin cortical hemispheres at the front. Below the thalamus is the cerebellum. By three months, fairly well-developed mid- and hindbrain structures have developed. The specific sequential development

of the brain also maps the establishment of a brain hierarchy, which relates to the antogeny/phylogeny relationship.

Two specific sequences within the neurodevelopmental process are important in understanding neurodevelopment. These sequences are **proximodistal**, or the development from the centre outwards and **cephalocaudal**, or development from the head downwards. These concepts are illustrated in figure 19 where the development takes place from the neural tube (the centre of the organism) towards the head, then towards the arms, and lastly towards the legs and feet. This information could be productively utilised in a therapeutic process that honours natural growth and development.

The developmental sequence of the embryo is illustrated in figure 20. **Cephalocaudal** development starts at the neural tube and progresses upward as well as downwards (though downwards development takes longer due to the higher priority of upwards growth). **Proximodistal** development begins at the neural tube and spreads outwards.

From this point of view the process accounts for the fact that the brain develops and matures from the spinal cord, and that the establishment of brain **hierarchy** starts at the brainstem level, and slowly moves forward towards the frontal regions where attention and impulse control resides (figure 20). Within this structural hierarchy other systems are included. One such system is the sensory/motor system and all its subsystems, which also follows a developmental hierarchy. Touch develops first, closely followed by taste and smell, and then the all-important vestibular system, and so on. The sensory-motor system and the developmental hierarchy of this system are discussed in chapter 4, section 4.





2.1.1.1 Developmental hierarchy

Yet another sequence that deserves mention is the sequence of **differentiation**, **lateralisation** and ultimately **interhemispheric integration**. This sequence is also developmental (maturational) in nature as time plays a role here. The young baby has no cortical influence over movement and planned action. As the toddler inhibits primitive reflexes and thus gains more cortical control, additional brain structures develop and the functioning of various areas in the brain improves (reflexes were discussed in chapter 3 section 4.6.3.1). A child's ability to differentiate different parts of the body leads to lateralisation, or the awareness of the two sides of the body. Being able to control and synchronise the two sides of the body together requires being able to cross the physical midline. Once this becomes automatic, the brain is able to share information over the 'neurological-midline', namely, the corpus

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collossum. This allows for interhemispheric integration. A short description of each component follows:





i. Differentiation

Heiniger and Randolph (1981:162) describe differentiation as the sorting out or separation of body parts from each other in a cephalocaudal and proximodistal direction. Conjugate movement of the eyes initiates the differentiation process by separating the eye movement from the head movement. Normal eye movement is dependant on neck stability. Neck stability is essential to control the fluctuating tone from vestibular input. Once the neck is stable, it must then be differentiated from the trunk to afford free and independent head orientation. As the differentiation progresses, the arms and legs are separated from the trunk as a unit.

In the previous chapter, reflexes were discussed. In this discussion, specific mention is made of the link between the neck, limbs and arms. Subsequently, each joint of the extremity is isolated in a proximal to distal direction. The highest level or last step in differentiation is the separation of the thumb from the palm of the hand and the isolation of each finger. Each step of differentiation is followed by a recombining of

the elements into generalised movement patterns (Heiniger & Randolph 1981:162-163). Joint receptors (proprioceptors) are critical in this differentiation process. They are responsible for the conscious awareness of the internal 'roadmap' of the different parts and how they link with one another.

Differentiation of response also includes the inhibition of primitive reflexes. This is the ability to direct one part of the body to move according to plan (cortical involvement) while all other parts remain still. It is the precursor to the development of lateralisation, and helps the brain establish specialised centres (Bluestone 1994).

ii. Lateralisation

Laterality is the normal awareness of the two sides of the body. It is an internal awareness (Heiniger & Randolph 1981:158) that the two sides of the body work together and in opposition. This awareness necessitates a good body awareness, which is made up of body concept, body image, and body scheme (Heiniger & Randolph 1981:228).

Laterality is our map of internal space. Directionality is our map of external directionality. Laterality is the basis of the individual's spatial concepts. Unless a right-left gradient has been established in reference to the line of gravity through the body, there is no basis for directionality (Heiniger & Randolph 1981:230-231). When the perception of laterality is projected externally, it becomes directionality. Directionality is therefore a learned process where lateralisation is a perceived process. The process must develop from the body outward to objects before it can develop between objects. Left-right orientation is delayed if children experience difficulty in crossing the midline of the body because of problems in laterality. The process of shifting from a right-left gradient in laterality to a left-right orientation in directionality is paramount. Without this shift from laterality to directionality, children appear lost in their spatial concepts (Heiniger & Randolph 1981:230-231).

Lateralisation also refers to the development of lateral dominance (right and left eye, ear, hand, leg) and the development of specialised centres and functions in the left and right cerebral cortex. Differentiation is the precursor to the development of

lateralisation. The ability to cross one's midline is also a necessary component for mature lateralisation and ultimately interhemispheric integration.

iii. Interhemispheric integration

Central to the notion of interhemispheric integration is the functioning of the corpus collosum. The corpus collosum is a single structure that connects the two brain hemispheres. Commissures (cross-over structures) are nerve fibre tracts that cross over the midline of the brain and functionally connect the right and left hemispheres. These nerve fibres develop during the third month after conception (Upledger 1996:83-84). The corpus callosum is, however, the larger and most prominent commissure that connects much of the brain (excluding olfaction). Optimal integration of the two hemispheres relies on optimal development of differentiation and lateralisation. The main aspect of interhemispheric integration is the communication between the two brain hemispheres. The following figure illustrates the different commissures in the brain.



Figure 22 Commissures in the brain

(Upledger 1996:83).

2.1.1.2 Developmental sequence of brain structures

The brain comprises many separate entities, which are all interlinked and dependent upon each other. At birth, connections to the superficial layers of the cortex are only tenuously made, and the neonate is a brain stem-dominated creature (Goddard 1996:32)

The brain stem is situated at the head of the spinal column and houses the nerve pathways, which carry impulses between the brain and the body. It is part of the CNS and responsible for the neurons which control heart beat, blood pressure, breathing and elimination, and also the signals to swallow, laugh, and cough. The brain stem also contains the point at which the nerve tracts between the brain and body cross over and change course to the opposite side (Goddard 1996:32), creating contralateral control.

The brain stem includes the medulla oblongata and pons. It is also closely linked to the vital reticular formation, which is responsible for maintaining consciousness and arousal. Forming the bridge between the brain stem and the cortex are the pons and the midbrain. These centres, together with the thalamus, basal ganglia, hypothalamus, and the cerebellum, interact to form the organisation centres of the sensory, motor and autonomic systems (Goddard 1996:32).

The thalamus is a major relay station that carries impulses from cerebellum, reticular system and neural ganglia to the cortex. All senses (except smell) are filtered through the thalamus before reaching specialised regions in the cortex. This system is important for the interpretation of sensory stimuli (Goddard 1996:33).

The hypothalamus is situated slightly below the thalamus and acts as the synthesiser of hormones involved in temperature control, water balance, hunger, and sexual behaviour. These hormones are then funnelled into the pituitary gland to be stored for later use. These two centres together are labelled the limbic system. From the limbic system all sensation (passion, drives, fear, anger) are generated. The midbrain and limbic system represents instinct, controls of metabolism and metabolic reaction to outside world (Goddard 1996:33). Goddard further suggests that some behaviour results directly from reflexive or spontaneous stimulation of the hypothalamus and related structures, combined with inhibitory learning processes emerging from the cerebral cortex.

The basal ganglia are responsible for the organisation of involuntary and semivoluntary activities upon which conscious, planned movements are superimposed. They maintain a balance between inhibitory and facilitating influences. Activities that need practice should eventually be absorbed into the automatic repertoire of the basal ganglia to form kinaesthesia (Goddard 1996:34).

Connected to the brain stem, but not part of it, is the cerebellum. Its name literally means "the little brain" because of its two hemispheres, and therefore resembles the larger cortex (Goddard 1996:34). While it is the cerebral cortex that enables us to perform all the higher functions unique to mankind, it is the cerebellum that governs man's every movement. Although it can initiate nothing by itself, the cerebellum monitors impulses from the motor centre in the brain and from the nerve endings in the muscle proprioceptors. Incoming impulses outnumber efferent impulses at a ration of 3:1; and it is the cerebellum's function to sift out and pass on relevant information. Impulses to the cerebellum are directed from the vestibular system (VS), the eyes, and the muscle joints of the lower limbs and trunk. Ultimately, the cerebellum is responsible for regulating the postural reflexes and muscle tone, thus maintaining body equilibrium (Goddard 1996:34).

The greatest period of growth and maturation occurs in the cerebellum between birth and 15 months of age, just at the period when the adjustment from primitive reflexes to postural control is being made. Maturation continues at a slow rate until age seven or eight, when the final linking takes place between the VS, the cerebellum and the corpus collosum.

It is through the process of primitive reflex inhibition, and then postural reflex development, that the infant recapitulates evolution. These stages of the brain growth and the movements they make are inextricably linked. Continuous brain development is facilitated through these movement sequences, which lay down efficient neural pathways (Goddard 1996:40).
Finally, at the top of the brain pyramid is the cerebral cortex, comprising two hemispheres, which are linked together by the corpus callosum as well as the other commisures. Although both hemispheres share some tasks, the left and right side of the cerebral cortex have specialised functions to perform. However, they are dependant upon each other for the optimal execution of those tasks, hence the significance of the commisures between them. The corpus callosum contains millions of nerve fibres, which facilitate communication and instantaneous feedback from one side of the brain to the other (Goddard 1996:35). It is in the cortex that information passed on from all the other brain centres becomes conscious, and on the basis of cortical analysis that decisions for action are made. The cortex is the seat of intellect, of decision making and of controlled response, but it can only do its job easily and effectively if the reflexive action of the lower centres are integrated at the correct time, in a hierarchical sequence (Goddard 1996:36).

The corpus callosum also seems to act as a screening device, at times shielding information between the two sides of the cortex. It is capable of both transmitting and inhibiting the exchange of information (Goddard 1996:36).

2.1.2 Neural plasticity

The concept of neuroplasticity – the idea that the brain is not static but malleable, if given the right stimulation, has been widely accepted as scientific fact and has been proven as early as the 1800s when Cajal (in Robbins 2000) made a major discovery that **nerve cells morph or change**. This concept has fomented a revolution in thinking about the brain and a growing number of studies show that the brain is indeed capable of great change. Plasticity is the ability of the brain to adapt or change in response to demands from the environment (Cheatum & Hammond 2000:34). Some of the more interesting work took place in a convent in Mankato, Minnesota, called the School Sisters of Notre Dame. Scientists wondered why these nuns were free of problems such as senility and Alzheimer's disease well into their eighties and nineties. These nuns occupied their days with intellectual work. Autopsies after their death revealed that the connections between the cells were very robust and dense; in other words, they had strong neural pathways. The explanation was that exerting the brain in new ways through the course of life creates new

neuronal pathways, more synaptic connections, and significantly more cortex. Thus, stimulating the brain builds a bigger and better brain (Robbins 2000:26).

Another study with a very solid design seemed to end for good the notion of a static brain. In November of 1998 the journal *Nature Medicine* published the results of a study conducted by a team of Swedes and Americans. Five terminally ill cancer patients at a Sweden hospital were injected with a fluorescent green chemical dye called bromodeoxyuridine. After the death of the patients, the hippocampus was removed and studied. In all the patients green areas lit up, proving that cell growth took place after the injection, towards the end of the patient's life. Undifferentiated cells were continuing to divide, and to produce new, fully functional neurons, right up until the death of each of the five subjects (Robbins 2000:28).

Not only does the brain have the ability to create new neural networks, but existing networks are in constant change, including mature neurons that have formed a relative stable, non-dividing cell population. These neurons are in constant flux. Under time-lapse videos the dendrites can be seen to grow and retract, to protrude spines and then to withdraw them again, to make and break synaptic contact. To prove this, one research study studied mouse brains and the area that encodes information from their whiskers. Fifty per cent of the dendritic spines persisted for only a few days, the rest all changed. Rose (2005:147) states that if this is brain architecture, it is a living, dynamic architecture in which the present forms and patterns can only be understood as a transient moment between past and future. He continues by stating that the present state of any neuronal connection and any synapse, both depends on its history and shapes its future. A representation of this concept is given in figure 23.



Figure 23 Dendrite density development

(Eliot 1999:28)

The brain, like all features of living systems, is both being and becoming; its apparent stability is a stability of process, not of fixed architecture. Today's brain in not yesterday's and will not be tomorrow's (Rose 2005:147). Rose continues this thought by comparing the process to "building a plane in mid-flight".

2.2 Physiological Stress

Walter B. Cannon was one of the first physiologists to study the powerful and longlasting effect of stress on the human body in the early part of the twentieth century. (*Stress* comes from the Latin word *strictus*, which means tight and narrow, which is exactly what occurs in the blood vessels when the organism is stressed.) Robbins (2000:56) reports that Cannon fed a cat food that was laced with a radioactive element called barium, so he could observe the cat's stomach with an x-ray. As long as the cat was content the stomach muscles digested the food in wavelike motions. When Cannon aggravated the animal to anger or frustration the stomach immediately halted its digestive motions and froze. These stressed-like states persisted for an hour or more after the stimulus was removed. The cat's body stopped nonessential

activity to prepare to defend itself or to escape. Cannon dubbed the response "fight or flight response" (Robbins 2000:56).

Cannon also studied the profound effect that the hormone adrenaline has on the human body. Just a couple of drops dissolved in 100,000 parts of water and injected into the cat caused it to arch its back, bare its claws, and dilate its eyes. Heart rate and respiration rate, the amount of glucose in the blood, and blood pressure all increased. Subsequent studies have shown that even being asked to solve a complicated math problem may evoke a milder version of the fight-or-flight response (Robbins 2000:57).



Figure 24 Divisions of the autonomic nervous system

http://www.ez2bsaved.com/brainbased_learning/

Heiniger and Randolph (1981:3) report that Hess coined the terms ergotropic and trophotropic in the 1930s. All of the stress reactions that initiate a fight-or-flight reaction may be considered **ergotropic** (Greek *ergos* – work). The opposite

reactons toward deep relaxation and vegetative functions may be considered **trophotropic** (Greek *trophos* – nutrition).

The three ergotropic-trophotropic elements include **autonomic nervous system**, **somatic muscle**, and **cortical activity**. The autonomic nervous system appears to be the dominant element and is used as the pivot for the concept.

The autonomic nervous system is divided into two separate but interdependent systems – **sympathetic** and **parasympathetic** (see figure 24). The sympathetic system may act selectively; however, it usually acts as a total unit with the parasympathetic system, exciting neural and glandular function. The fight-or-flight reaction is the body's most comprehensive reaction to extreme stress. The parasympathetic system is relatively specific and selective in its activation of organs. The parasympathetic system is primarily concerned with the vegetative functions of the body (Heiniger & Randolph 1981:9). The two systems have the opposite effect on organs. A diagram of the effects is presented in table 5.

Structure	Sympathetic Activity	Parasympathetic Activity
Iris	Pupil dilation	Pupil constriction
Salivary glands	Saliva production reduced	Saliva production increased
Oral/Nasal mucous	Mucous production reduced	Mucous production increased
Heart	Heart rate and force increased	Heart rate and force decreased
Lung	Bronchial muscle relaxed	Bronchial muscle contracted
Stomach	Peristalsis reduced	Gastric juice secreted; motility increased
Small intestine	Motility reduced	Digestion increased
Large intestine	Motility reduced	Secretion and motility increased
Liver	Increased conversion of glycogen to glucose	Stimulates gallbladder
Kidney	Decreased urine secretion	Increased urine secretion
Adrenal medulla	Norepinephrine and epinephrine secretion	

Table 5 Autonomic nervous system



Bladder	Wall relaxed	Wall contracted
	Sphincter closed	Sphincter relaxed

(The Quantum Centre of Excellence 2005:14)

The symptoms in the table mirror complaints often reported by children. Some often present with stomachaches and pains. Other children may present with enuresis or encopresis. Headaches are also common among children and may be due to the decrease of bloodflow and oxygen to the brain as the supply is redirected to muscles for fight-or-flight activity.

The fight-or-flight response consists of three parts. The first is a physical or verbal attack. The second is a physical flight in which people remove themselves from the situation. The third part is frequently overlooked because it is an emotional or psychological flight or withdrawal. Heiniger and Randolph (1981:10) believe that children who frequently daydream in class use the third part of the fight-or-flight response. When the physical characteristics of the sympathetic nervous system are evaluated, they may be found to prevail in all these individuals. This indicates that the individual has passed his or her stress threshold on the ergotropic-trophotropic continuum (Heiniger & Randolph 1981:10).



Ergotropic-trophotropic continuum

These two concepts are discussed in the following section and illustrated in figure 25.

i. Ergotropic system

The term ergotropic combines the triad of

- 1. increased sympathetic activity
- 2. increased somatic action
- 3. cortical desynchronisation or alpha rhythm (an alert or aroused cortex)

This triad is an integrated whole. Any increase or decrease in discharge of one element is paralleled in the other two elements.

The anatomical integrative "hub" of the ergotropic system includes the posterior hypothalamus (sympathetic), the cingulated gyrus, and the recticular facilitory area of the pons to thalamus. There are other brain structures that contribute to the arousal associated with the ergotropic syndrome such as parts of the neocortex (sensorimotor, temporal, and occipital) as well as association areas. Stimulation of the anterior lobe of the cerebellum also elicits an ergotropic response (Heiniger & Randolph 1981:3).

ii. Trophotropic system

The term trophotropic combines the triad of

- 1. increased parasympathetic activity
- 2. somatic muscle relaxation
- 3. cortical synchronisation or beta rhythm (sleep)

Just like the ergotropic triad, this triad is an integrated whole. Any shift in discharge of one element is paralleled in the other two elements.

The anatomical integrative "hub" of the trophotropic system consists of the anterior hypothalamus (parasympathetic), the hippocampus, and the reticular inhibitory area of the medulla oblongata and the caudal pons. Stimulation of the posterior lobe of the cerebellum elicits the trophotropic response. The intensity and frequency of cutaneous stimuli are the critical factors in determining which system responds. If the stimuli are of low intensity and frequency, the trophotropic system responds. However, same stimuli applied at a high frequency or intensity may produce an ergotropic response (Heiniger & Randolph 1981:4).

It may be helpful to place these two systems at each end of a continuum. Each person functions at some point on that continuum. Depending on what happens in a given situation, the person may move toward the ergotropic end or in the opposite direction toward the trophotropic end.

There is a very important reciprocal relationship between the two systems. Any change in one element of the triad is paralleled by a change in the other two elements; therefore any change in the trophotropic system is paralleled by a change in the ergotropic system. There is constant shifting in discharges from individual elements, which affects the balance of the entire system.

According to Gellhorn (cited in Heiniger & Randolph 1981:4), tonic activity of the nervous system constantly controls these two systems in a balance. Phasic activity modifies the tonic state in a specific organ or function. The combination of phasic and tonic activity provides an enormous variety of ergotropic-trophotropic patters to deal with different local demands at any given state of central autonomic balance. The combination of phasic and tonic activity ensures smooth transition from ergotropic to trophotropic responses. The reciprocal balancing also ensures minute gradations of each system's responses at every level of excitation.

The critical point on this continuum is the stress threshold. This is the point at which stressors are interpreted as potentially dangerous, and a protective fight-or-flight response is triggered. Messages are sent throughout the neuro-endocrine system, causing significant changes in the individual's biochemistry. Once the neurophysiological stress response subsides, the body should rebound into a deep relaxation or trophotropic state and ultimately seek its homeostasis. When a stress response is prolonged and unabated, the biochemical changes may become detrimental to the individual's health (Heiniger & Randolph 1981:5).

2.3 Psychophysiology of Stress

One can assume that every individual operates at a level of tolerable nonpathogenic stress. This stress actually heightens awareness, improves performance and plays

an important role in self-preservation. This healthy equilibrium may be upset by a wide variety of psychological, physical and environmental stressors.

According to Hans Selye (a researcher at McGill University in Montreal) (1956, 1974), stress could be seen as the common denominator of all adaptive reactions in the body (The Quantum Centre of Excellence 2005:14). Since he believed that this statement was simple and true but too vague for scientific analysis, he developed an operational definition: stress is the state manifested by a specific syndrome which consists of all the nonspecifically induced changes within a biological system. Stress has its very own form and composition but no particular cause. There are definite visible changes that are characteristic of its form regardless of the cause (Heiniger & Randolph 1981:6).

After defining stress, Selye found it necessary to coin a name for his syndrome – the general adaptation syndrome (GAS). The syndrome evolves in time through three stages:

- a) The **alarm reaction**, the first stage, affects the body as a whole. In his early work he found the following triad produced:
 - 1. considerable enlargement of the adrenal cortex
 - 2. an intense shrinking or atrophy of the thymus, spleen, lymph nodes and all other lymphatic structures
 - 3. deep bleeding ulcers in the stomach and duodenum

He continued that no living organism could survive a continuous alarm reaction.

- b) The second stage of the GAS is the stage of resistance. During this stage many of the alarm reaction elements are reversed, seemingly in an attempt to adapt to the situation and increase resistance (Heiniger & Randolph 1981:7). No symptoms present during this stage.
- c) The **third stage** is exhaustion. During this stage the adaptation energy appears to be exhausted and an alarm reaction reappears (Heiniger & Randolph 1981:9).

Comment

It is unclear how many of the children who present with symptoms of attention deficit with or without hyperactivity also present with stress. Could stress result in inattention and heightened motor activity? Certainly stress must play a role in the performance of children when they struggle to perform academically or when they are unable to cope with academic demands. Could prolonged stress be the reason why so many children present with chronic low immunity, allergies, colds, and other psychosomatic diseases?

2.4 Information Processing / Functional Organisation

Goddard (1996:1) and de Jager (2001:21) states that a neurodevelopmental approach concentrates upon the functioning of three systems:

- The **reception** of information through the sensory system (afferent system)
- Processing of information in the brain
- The repertoire of responses available to individuals with which to express themselves: motorically, linguistically and academically (efferent system). The three systems could be presented as follow:



Figure 26 Information processing

Sternberg (1999:38) and Lezak (1995:20) call this process "functional organisation." A general design of the nervous system according to Guyton (1972:6-7) and Sternberg (1999:38-41) provides a more detailed explanation of the information processing system or functional organisation.

2.4.1 The sensory division – sensory receptors

Most of the activities of the nervous system are originated by sensory experience emanating from **sensory receptors**, whether these be visual, auditory, or tactile receptors on the surface of the body, balance, or other kinds of receptors (we have more than five senses). This sensory experience may cause an immediate reaction, or its memory can be stored in the brain for longer periods for later use. The **somatic sensory** system transmits sensory information from the receptors of the entire surface of the body as well as deep brain structures. This information enters the nervous system through the spinal nerves and is conducted into (a) the spinal cord at all levels, (b) the reticular substance of the medulla, pons and mesencephalon (midbrain), (c) the cerebellum, (d) the thalamus, and (e) the somesthetic areas of the cerebral cortex. In addition to these primary sensory areas, signals are then relayed to essentially all other segments of the nervous system. The main region in the cortex receiving sensory information is the **somatosensory area**.

The somatosensory area receives perceives sensory input in a strip running across the parietal lobes of the brain. The somatosensory strip lies posterior to the motor strip. It is closer the brain_stem than the motor strip as the reception of sensory input is the first step in the information processing process, which is then passed onto the motor strip for response. The surface of the entire body is mapped across this strip. This map is called the **homunculus** (representing the body) (see figure 27). More sensitive areas (like the lips and fingertips and especially the thumb) take up a disproportionately large share of the cortical space, whereas large parts of the body like the back and legs cover comparatively little cortical ground due to less sensitivity. The mouth and thumb thus play a disproportionally large role in the brain's processing of sensory input. These sensitive areas are also responsible for early learning experiences.



Figure 27 Human homunculus

(Sternberg 1999:59)

2.4.2 Processing of information

The brain is a wonder of information processing. Information comes into the brain from the external world through the senses and is converted into an extremely complex mix of electrical and chemical energy. The nervous system would not be at all effective in controlling bodily functions if each bit of sensory information caused some motor reaction. Therefore, one of the major functions of the nervous system is to process incoming information in such a way that **appropriate motor** responses occur. Indeed, more that 99% of all sensory information is continually discarded by the brain as unimportant. For instance, most people are ordinarily unaware of the parts of their body that are in contact with clothes, and do not take note of the contact between their body and a chair, for example. Likewise, attention is only occasionally drawn to objects in their field of vision, and even the perpetual noise of our

surroundings is usually relegated to the background. After the important information has been selected, it must be channeled into the proper motor regions of the brain to produce the desired response (Guyton 1972:7; Robbins 2000:29). In some instances, the spinal cord directly connects to receptor nerves with effectors nerves, without routing either one through the brain until after the body has responded to the sensory information. The directly-connected responses are reflexes. The ability to pay attention and control motor activity is of major significance in the information processing system and specifically during the processing stage. This is discussed under the section of attentional priorities (section 6.3).

2.4.3 The motor division – the effectors

The most important role of the nervous system is the control of bodily activities. Achieving this requires, among others, the inhibition of responses. This is achieved by (a) contraction of skeletal muscles throughout the body, (b) contractions of smooth muscles in the internal organs, and (c) secretion by both exocrine and endocrine glands in many parts of the body. These activities are collectively called motor functions of the nervous system, and the muscles and glands are called effectors because they perform the functions dictated by the nerve signals. The portion of the nervous system directly concerned with transmitting signals to the muscles and glands is called the motor division of the nervous system. Another system that works parallel to the motor axis and controls the smooth muscles and glands, is the autonomic nervous system. The skeletal muscles may be controlled from many different levels of the nervous system, including (a) the spinal cord, (b) the substance of the medulla, pons, and mesencephalon, (c) the basal ganglia, and (d) the motor cortex. Each of these different areas plays its own specific role in the control of body movement, the lower regions being concerned primarily with automatic, instantaneous responses of the body to sensory stimuli, and the higher regions with deliberate movement controlled by the thought processes of the cerebrum.

In sum, the nervous system is extremely well organised, with lower levels in the hierarchy of command capable of responding without intervention of the brain when the immediate need arises, but with higher levels in the hierarchy essential for full

physical interaction with and perception of the world around us, including academic performance.

So far the natural development of the human brain has been discussed. In order to understand dysfunction in attention and motor activity (movement), we need to see what could interfere with or interrupt in the process. The following section deals specifically with possible delays.

3. Neurodevelopmental Delay

The discussion of neurodevelopment in terms of time and maturation is important as knowledge of these concepts and the sequence they follow allows a professional to gain insight into a child's development, and allows identification of a possible neurodevelopmental delay. The time factor has different aspects in terms of **chronological age** (actual time lapsed since birth) and **developmental age** (age according to set stages of development). The ability to determine developmental age stems from knowledge of the specificity of developmental stages, and knowledge of when this should take place. The discrepancy between chronological age and developmental age maps the **developmental delay**.

Goddard (1996:86) states that neurodevelopmental delay encompasses a variety of symptoms. These symptoms emanate from an arrested or omitted stage of development during either the foetal or the infantile period. This author goes on to state that although subsequent development could proceed normally thereafter, an underlying weakness or immaturity remains within the central nervous system (CNS), which may cause other systems in the body to misfire under certain conditions due to the initial weakness. Omitted stages could be primitive reflexes that did not transform into postural reflexes. These aberrant reflexes provide signposts of central nervous system maturity.

The presence of a primitive reflex, or the lack of a postural reflex at key stages in development, may be seen as evidence of continuing subcortical control over neuromuscular functions. Voluntary control of movement directly reflects the degree of cortical control in the individual. The cortex represents planned behaviour whereas

subcortical behavior is limited and stereotyped. Subcortical systems may remain dominant for a number of reasons:

- 1. Lack of use at an early stage in development
- 2. Lack of inhibition
- 3. Metabolic or pathological conditions
- 4. Direct injury

Any of the above may interfere with cortical functioning at a later stage. Detection and analysis of primitive and postural reflexes may therefore be used as a valuable tool in assessing the level of developmental delay and remediation required by a child, as it indicates the developmental stage a child has reached (Goddard 1996:86).

During normal development, the primitive reflexes should emerge, strengthen, fulfill a function, and then be inhibited during the first year of life. There should be a strict chronology, sequence, and rhythm to this reflex structure (specificity), so that by a certain age specific milestones have been achieved. Should the sequence be interrupted in any way, the early reflexes will remain present in the system, so that the emergence of subsequent (postural) reflexes is disturbed, and further central nervous system development is built upon eccentric foundations (Goddard 1996:101). The process of reflex inhibition and transformation aids in opening up neural pathways. Motor behaviour should be the product of a system in which brain and body work together to form a communicating system of response, action and expression (Goddard 1996:108).

Messages should be transmitted with equal efficiency from brain to body and back again, via the efferent and afferent systems. If this is disrupted in any way, then subsequent motor and sensory functions may be affected. The result of this is that the transmission of messages from one system in the body to another is altered; perceptions are distorted; and the translation of sensory experience into thought, language, and emotion – and even the ability to deal with that sensory experience – is disrupted (Goddard 1996:109). This has the potential to negatively affect academic performance.



Goddard (1996:109) believes that when the system receives distorted messages and images on an unregulated basis, the processing or filtering system in the brain cannot cope with the demand of information, and therefore "cuts out" at a very crude level. It is as if it is bombarded by conflicting sensory stimuli, which it cannot categorise immediately, and it becomes overloaded far too quickly.

The above suggests that misfiring in the brain stem as a direct result of aberrant reflexes may result in either extreme over-excitation (hyperactivity), or understimulation (blocking one or more sensory channels until eventual total unconsciousness occurs). In other words, the filtering mechanism is not performing properly. Instead of sifting and dumping irrelevant information and stimuli, all incoming information passes through to higher centres in the brain. This either heightens arousal (fight) to an abnormal level, or it blocks incoming information entirely, so that the system "shuts down" (flight) (Goddard 1996:114) and a physiological stress response occurs. If this continues, this process will result in psycho-physiological stress.

The result is an inadequate interaction between the vestibular apparatus, reticular activating system and cerebellum, with subsequent impairment to the arousal mechanisms (Goddard 1996:114). Other health difficulties, as mentioned by Selye could also arise.

4. Underlying Neurological (Sensory-motor) Subsystems

The sensory-motor subsystems are the components that comprise the neurological system. Most people are familiar with the five senses of hearing, seeing, taste, smell and touch. These are sometimes called the "far senses" because they respond to external stimuli that come from outside the body. Less familiar senses are the so-called "near senses," sometimes called the hidden senses (Kranowitz 1998:40-41), because a person is generally unaware of them and cannot control them. The following section focuses on these senses, as they are the underlying subsystems that form the neurological system.

Although the subsystems are discussed individually and in isolation, it should be remembered that none of the senses develop or operate in isolation. Each one is reinforced, modified and influenced by information from the others. Hearing has been described as a "specialised sense of touch", we "taste with our nose", "feast with our eyes", "see with our fingers," and according to Tomatis (1991), we "read with our ears". The neurological system requires balance between the different inputs. The sense of balance is perhaps the forgotten sixth sense of the twentieth century. Goddard (1996:42) states that an understanding of the senses and how they complement one another is essential if we are to understand and help the child who cannot make sense of the world, and who therefore has difficulty learning through the accepted channels of education.

4.1 Tactile System

Unlike any other sense, the sense of touch functions all over the surface of the body as well as deep inside. Touch encompasses mainly four different sensory abilities, each with its own direct neural pathways. These are the obvious sense of touch (cutaneous sensation), temperature (cold, hot), pain and proprioception (sense of body in space) or pressure (Eliot 1999:9123-124; Winston 2004:128).

i) Developmental stages

In the development of the **somatosensory system** (see section 2.4.1), the sense of touch is one of the newborn's most advanced abilities at birth even though it is not fully developed yet. Touch is perceived through the skin (the largest organ of the body), which contains millions of sensory receptors throughout and below the dermis (deep within different layers of skin tissue).

Tactility is thus the ability to discern touch in all of its nuances (smooth, rough, hard, cold, deep, painful). With all these receptors, the skin becomes one of the primary organs for early environmental learning as well as the establishment of a sense of security. This sense is developed very early in utero and is practised before birth. The practice in utero takes place in a wet and protected environment. After birth, the infant must adapt to a dry and unprotected environment. The adaptation of the central nervous system to this new environment supports Rose's (2005:63) theory of

autopoiesis, and specifically plasticity, where the developing nervous system moulds itself in response to changing aspects of the outside world.

Although the vestibular system is the first to be fully developed and myelinated, it is the sense of touch that provides us with our first source of contract with the outer world. The first observed response to tactile stimulation occurs at approximately five weeks after conception with the emergence of the mass cutaneous withdrawal reflexes. Gentle stimulation of the foetus's upper lip results in immediate withdrawal from the source of contact by the whole organism. The area of sensitivity rapidly spreads over the next four weeks to encompass the oral region of the face, the palms of the hands and the soles of the feet, until eventually the whole body surface is responsive to touch. In section 2.4.1 mention was made of the homunculus and the different sensitivity levels, specifically the oral and finger areas. However, the earliest primitive realisation of touch is a defensive one characterised by withdrawal (Goddard 1996:45).

During the second and third trimesters of pregnancy, tactile awareness should mature to allow the grasping reflexes to develop (palmer, plantar, rooting, suck, Moro, etc), so that by the time the baby is born, touch is associated with security, with feeding, comfort and eventually exploration. A diagram (figure 11) of the emergence of some of the reflexes is presented in chapter 3. Touch precedes both hearing and vision as the primary channel of learning (Goddard 1996:45) as survival of the organism precedes learning.

ii) Functionality

External environmental objects activate receptors in the skin that are endpoints of touch-sensitive sensory neurons. These special receptors translate mechanical pressure into long-distance electrical signals – action potentials – that propagate along the sensory neuron's tiny axons all the way into the spinal cord, and then up to the brain stem. When they finally reach the brain stem, these primary-touch neurons synapse on their first set of relay cells, neurons whose axons cross to the other side of the brain stem and terminate in the opposite (to body part touched) **thalamus** (the thalamus is the relay centre for almost all the sensory information). When action potentials in these relay neurons reach the thalamus, they activate a third leg of the

relay, namely, touch-communicating neurons, whose axons reach the somatosensory region of the cerebral cortex. It is here in the somatosensory cortex that neurons, once activated by the long-distance relay, allow the person to perceive the touch stimulus, note its intensity, and interpret the object touched (Eliot 1999:125). Temperature, pain, pressure and proprioceptor receptors follow the same route (but on different pathways) and merge at the "touch centre" in the cortex where interpretation (perception) occurs. Even the base of each hair follicle has touch receptors. The vestibular apparatus functions through the movement of fluid over hair cells. The sense of touch is essential for the functioning of balance, orientation and motion (Goddard 1996:45). The cilia in the cochlea also contain hairs that translate sound waves into electrical signals. The ear and it's "sense of touch" is thus also an organ that allows the body to orientate itself.



Figure 28 The anatomy of touch

Neural pathways of touch. The cold teething ring activates both touch and temperature receptors in the fingers, which in turn send electrical excitation through the spinal cord, brain stem, and thalamus to terminate in the somatosensory cortex, where the teething ring is consciously perceived. Gaps in the pathway indicate the location of synapses (Eliot 1000-124)

iii) Modulation

Ayres (1982) divided the tactile system into protective and discriminative subsystems:

- Protective receptors are located around the hair follicles and respond to subtle stimulation such as sound and air waves (vibration) moving across the body. They tell the organism where the body ends and where space (external environment) begins.
- Discriminative receptors are located in the dermis (deeper skin tissue) and respond either actively or passively when the person comes into contact with something.

These systems should be mutually exclusive, meaning that one shuts down as the other comes into action. The protective system stays in operation until the person is touched, and unless that contact is threatening, the discriminative system comes into play as soon as contact takes place (Goddard 1996:46).

The human being has a distinct need for touch. For example, specific forms of touch stimulate the production of specific neurotransmitters that regulate our response to anxiety (Levi-Montalcini & Calissano 1979:44). Just the act of being touched increases production of a specific hormone within the brain, the so-called nerve growth factors (NGF), activate the greater nervous system and, specifically, nerve net development (Hannaford 1995:39). NGF stimulates sensory neurons during embryonic development. For this reason, natural birth and the associated tactile input and pressure activate NGF and stimulate, amongst other areas, the cranial nerves. Touch right after birth stimulates growth of the body's sensory neurons will not operate fully.

Tactile input is filtered through the thalamus. The thalamus, as a relay centre, allows one third of incoming information through to the cortex. If the thalamus malfunctions, too much or too little information is filtered, resulting in defective sensory integration.

As early as 1972, Ayers recognised that the **gate control theory** of Melzack and Wall (1965) unified various historical perspectives on the duality of the tactile system. She proposed that the gate control theory provided a conceptual model for **tactile**

defensiveness. Briefly stated, this theory suggested that "gate neurons" present in the dorsal horn of the spinal cord controlled the passage of impulses to the CNS. Control of these gate neurons is influenced by both incoming tactile inputs and by cortical influences (Royeen & Lane 2002:116). **Tactile defensiveness** is an adverse response to tactile stimuli. Primitive protective survival responses are elicited instead of integrative discriminative responses. Behaviours such as **distractibility** and **hyperactivity** may be more evident that the defensiveness.

Some people may be hypersensitive to touch (allowing too much information into the brain), or hyposensitive (allowing too little information into the brain). Some individuals experience a mix of hyper- and hyposensitivity to different tactile sensations or on particular parts of their body (Bluestone 1994). The child who has an overactive protective system will be tactile defensive and may still have uninhibited cutaneous withdrawal reflexes (first tactile response in the uterus), which continue to influence the central nervous system. If this is the case, then touch may be neither an instant source of comfort nor a purveyor of information; for the reflex response will elicit withdrawal from the source of contact, and the child cannot adequately utilise his or her tactile discrimination skills to learn from the contact. The "hyper-tactile" child may have abnormal perception in all input to the area of the cortex that registers touch. Such children may have poor tolerance for or adaptive mechanisms to heat and cold. They may have a low pain threshold, particularly to pain associated with piercing the skin; but, paradoxically, a high tolerance to internal pain (Goddard 1996:46). At this point, it is also important to once again discuss the homunculus. As discussed, the mouth and the thumb have proportionally larger areas of representation in the cortex, suggesting the sensitivity of these areas. Hypersensitivity is thus more likely to occur in these areas, resulting in tactile sensitivity to food textures and a possible inability to display a correct pencil grip.

4.2 Olfactory System

Olfaction is the sense of smell. The sense of smell influences the sense of taste. Smell receptors are located at the top of the nasal cavity. There are hundred of different receptors, each sensitive to a different chemical substance. All the odours and tastes that people recognise are compiled in the brain from the **combinations** of information received from the mouth and nose (Winston 2004:128). Both smell and taste are known as chemical senses because both begin with neural excitation in response to specific molecules in the environment and both are known as **phylogenetically primitive senses** (the use of chemicals used for survival) (Eliot 1999:158). When people or animals are afraid, they secrete pheromones that may easily be picked up by sensitive (animal) noses. Like dogs, babies or young children may be able to pick up the sense of danger and fear felt within the immediate environment, and act to protect themselves (Hannaford 1995:39).

Substances that have an odour release physical odour molecules into the air. When we breathe in odour molecules they come in contact with two regions of fatty tissue at the top of each nostril, behind the bridge of the nose. These regions, called the olfactory regions, contain about five million nerve cells. The cells carry cilia (tiny hairs) that project into the mucous layer of the nose and are stimulated by the specific odour molecule. When triggered by an odour molecule, the cells send electrical signals to a site at the base of the brain, the olfactory bulb. The bulb in turn sends signals to different areas of the primary olfactory cortex (located at the bottom, innermost bulges of the temporal lobe). Signals are also sent to the limbic system (responsible for emotions, drives and memory) (Eliot 1999:158; Winston 2004:128). This information is thus not processed through the reticular activity system and the thalamus, as are all the other senses prior to reaching the cerebral cortex.

The sense of smell is quite acute at birth, and even before birth. Infants may distinguish the scent of their mother's breast from that of a stranger as early as three days after birth (MacFarlane 1975:103). Smell is a major contributor to the parent-infant bond. The sense of smell is relatively primitive, both in the way that it is processed and in the fact that is has such direct access to the neural circuitry that controls our memory, drives and emotions (Eliot 1999:159), without a filtering system such as the thalamus.



Figure 29 Olfactory pathway of the brain

Odour molecules bind to olfactory epithelial cells in the nasal cavity, triggering a chain of electrical excitation those progresses from the olfactory bulb to several cortical and subcortical areas. One such target is the entorhinal cortex, a part of the limbic system that participates in odour memory. Another target, the orbitofrontal cortex, is responsible for our conscious olfactory perception (Eliot 1999:160).

i) Development

The olfactory system begin forming early in the embryonic period. At just five weeks after fertilisation a nasal ridge appears in the primitive face, gradually deepening and dividing to form true nostrils by seven weeks. At this point the olfactory epithelial cells begin to form, taking their place along the lining of the nasal cavity. By eleven weeks the olfactory epithelia are abundant and outwardly mature, but will not fully function before months later. At 13 weeks the olfactory bulb becomes walled off from the nasal cavity. The olfactory bulb appears fully mature by mid-gestation, but does not begin to function fully until the third trimester. The ability to smell begins at about twenty-eight weeks of gestation, thus already assisting in the bonding process (Eliot 1999:162) as the child recognises the chemical molecules of its mother's scent.



The fact that the olfactory system is directly linked to the limbic system has an unconscious effect on emotions and responses, since the limbic system is the seat of emotional responses. Consequently, if a person has a dulled sense of smell, the person may have dulled emotions or even flat affect. Many individuals with multiple chemical sensitivities have an irregular sense of olfaction.

Because there is no relay or filtering system, smell can instantaneously summon multisensory images. It can also stimulate the production of hormones involved in the control of appetite, temperature and sexuality in the hypothalamus – the same brain region influenced by the Moro reflex (Goddard 1996:62) For example, the smell of school can easily become associated with stress, the smell of a hospital with pain.

A child that is sensitive to smell may easily be distracted by the many odours in a classroom, for example, the different body odours of the many individuals in a closed, confined space, or the chemicals found in art equipment. Strong chemicals usually used in a germ-rich area such as a school could interfere with concentration. A hyper-allergic reaction to these chemicals could also lead to hyperactivity. Since the sense of smell is a primitive sense that is beneath the level of consciousness, the reaction to sensitivity is uncontrollable.

4.3 Gustatory System

Along with touch, smell and the vestibular system (VS), the ability to taste emerges early in development. This makes sense, given the importance of nutrition in survival. The sense of taste (gestation) first becomes functional during the third trimester of gestation, and gets a considerable amount of practice in the womb. Like smell, taste is known as a 'chemical sense' in which the nervous system detects specific physical molecules in the environment and converts them into distinct electrical signals. Compared to the sense of smell, taste is relatively simple. It detects four basic categories (sweet, sour, bitter, salty) in different parts of the mouth. Full flavour appreciation therefore involves considerable interaction between taste and smell (Eliot 1999:173).

The organs of taste are the taste buds, of which there are around 10,000 in the mouth. Taste buds are located mainly on the surface of the tongue, but some are scattered on the palate, throat and tonsils. Each bud is a cluster of about 50 taste-detecting nerve cells contained within a pore (Winston 2004:128). As the various molecules wash over the taste buds, they activate taste receptor cells – special elongated epithelial cells that line the pore of each pit-like bud. It is within the receptor cells that the chemical information is translated into electrical information, where a change in the taste cell's voltage triggers its release of neurotransmitters, exciting little dendrite buds in the first neurons in the gustatory pathway. These primary neurons then transmit their action potentials along axons that run through the base of the skull into the medulla (figure 31). Taste input to the medulla triggers several brain-stem reflexes necessary for feeding. The medulla also relays information to the pons, the thalamus and the limbic system. The thalamus relays information to the cortex (Eliot 1999:174).



Taste bud

A taste bud (orange area) on the tongue's surface is embedded in spiky papillae (nodules) (Winston 2004:128).

Taste buds emerge eight weeks after conception and by thirteen weeks have formed throughout the mouth. There is good evidence that babies can taste even before birth (in the last two months of gestation) and that they respond to different tastes in amniotic fluid (Eliot 1999:176).

The significance of taste and smell for learning is perhaps largely concentrated in the earliest years of childhood, when children's mouths (a relative sensitive area) are their primary source of information, exploration, expression and satisfaction (Goddard 1996:62).



Figure 31 Neural pathway of taste perception

(Eliot 1999:175)

4.4 Vestibular System

The vestibular system is very old in evolutionary terms, since all earthy organisms have had to orient themselves with respect to gravity and their own motion.

Accordingly, this system emerges early during embryonic development. Unlike the other senses, we are generally unaware of the senses of balance and motion as the vestibular system largely functions below the level of the cerebral cortex. For this reason it is also described as a "near sense".

i) Anatomy

Several structures in the inner ear, referred to collectively as the **vestibular system**, help the human being to stand upright and move without losing balance. This apparatus includes three fluid-filled loops set at right angles to each other – the three semicircular canals – at the base of which sensory hair cells lie in small structures called cristae. The three sacs and the three semicircular canals contain endolymph, a fluid high in potassium. Within the endolymph are calcium carbonate crystals or otoliths, which sweep past the cilia (hairlike nerves) to send a message to the brain from the inner ear regarding the pull of gravity according to the tilt of the head.



Figure 32 Inner ear and position of the vestibular system (Winston 2004:126-127)

The other structures that contribute to balance are two chambers (the utricle and saccule) within a fluid-filled area called the vestibule (this fluid is perilymph, which is high in sodium). The chambers both contain a macula, a sensory structure that is also full of hair cells. From the semicircular canals and the maculae, information about the speed and direction of the head's movement is transmitted by the hair cells to the brain in the form of nerve impulses (Winston 2004:127).



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The otolith organs detect linear movement, head tilts and the body's position with respect to gravity (Eliot 1999:146). Linear acceleration and changes in the position of the head in relation to gravity are detected by hair cells in two maculae within the system. Rotational movement is detected by the sensory receptors, cristae, in at least one of the fluid-filled semicircular canals. When the head turns, the fluid in the ear structures bends the sensory hair cells, stimulating them to produce nerve impulses (Winston 2004:127).



Figure 34 Movement in the vestibular system

(Winston 2004:127)

ii) Development

While the vestibular and auditory systems start their development together, the vestibular system progresses more rapidly than the sense of hearing. This may be because the early onset of vestibular abilities is so critical for the proper development of other parts of the nervous system, as well as the fact that it is mainly the vestibular system that orientates the foetus in turning upside down to be born head first (Eliot 1999:146).

The differentiation of the vestibular system and the auditory system is apparent just five weeks after conception, when three ridges that will become the semi-circular canals fold up and out of the otocyst. By seven weeks they have formed into proper canals. Between seven and fourteen weeks all the hair cells develop, luring the neurons of the vestibular nerve to grow towards them so they can synapse onto them. The vestibular nerve is the first fibre track in the entire brain to begin myelinating (see figure 49), in about the last week of the first trimester. By the fifth month of gestation, the vestibular apparatus has reached its full size and shape, vestibular pathways to the eyes and spinal cord have begun to myelinate, and the entire vestibular system functions in a remarkably mature way, receiving stimulation from the mother's movement (Eliot 1999:146).



Development of the vestibular system

Emergence and development of the vestibular apparatus during the embryonic period. The picture below illustrates the location of vestibular apparatus in an 8-week foetus (Eliot 1999:150).

By the sixth month of gestation, projections from the vestibular system to other parts of the brain and sensory organs are well developed and myelinated. One of these projections is the vestibule-cerebellar projection. Through this connection the inner ear communicates directly with the brain and influences the autonomic nervous system. This explains why individuals may have problems breathing or may develop nausea or an irregular heart rate as a result of an overwhelmed vestibular system.

The fact that the vestibular system is mature so early provides the foetus with a sense of direction and orientation inside the womb. It is in place to help cope with the problem of gravity, which infants encounter in its full force for the first time when they are born (Goddard 1996:42).

iii) Function

Hair cells form synapses onto the first neurons in the vestibular pathway. The axons of approximately 20 000 cells extend from the ear into the brain stem, forming the vestibular nerve. Signals from the vestibular system then pass along the vestibular nerve to the cerebellum (Eliot 1999:147). The cerebellum has been called "the moderator between sensation and brain level response" (Levinson 1990:122) as it coordinates information from the inner ear with other parts of the body. It monitors where we are in space and what position we are in (i.e., standing, sitting, running, climbing, somersaulting, etc). If information from the vestibular system is out of alignment with information from the other senses, motion sickness results. Astronauts experience a unique form of this when placed in a gravity-free environment where their sense of "centre" is lost, and they rely heavily on tactile and visual stimuli to retain a sense of location (Goddard 1996:43).

In the brain stem these vestibular nerve fibres synapse on several groups of neurons that serve as the hub of vestibular traffic, shuttling information about balance and motion to many places, including:

- the eyes, which automatically move in compensation for a change in head position. Visual tracking occurs via neuronal processing of the vestibular system, and processing of the effects of the vestibular nuclear complex and the cerebellum on brain stem functions.
- motor neurons down in the spinal cord that control the body's overall posture and the position of arms and legs (motor planning). Thus muscle tone, equilibrium, and proprioception, are governed through an interconnected processing loop.
- the cerebellum, which integrates vestibular information with vision and touch (proprioceptive) input; thereby coordinating a sense of balance.
- the vestibular-cochlear nerve (8th cranial nerve), thus governing auditory functions. (Bluestone 1994; Goddard 1996:43)

The vestibular system is always directly or indirectly involved with the information processing system. The vestibular system is responsible for some of our sensory functions nearly all the time, and all of our motor functions. Smell, taste, and touch are the three modalities of human processing that are relatively unaffected by vestibular functions, mainly because these senses are so primitive, and feed the brainstem below the area served by the vestibular system. However, tactility impacts on the vestibular system due to the receptors at the base of all hair cells (also those found in the three semicircular canals).



Figure 36 Vestibular pathway

Balance and motion are perceived through tiny movements of hair cells located in the three organs of the vestibular apparatus: the semicircular canals, the utricle, and the saccule. The hair cells excite primary vestibular neurons, which in turn send electrical information to various parts of the midbrain, brain stem, and spinal cord, coordinating our many vestibular reflexes (Eliot 1999:148).

In general, the processing system process remains below the level of consciousness as long as everything is working smoothly. However, if a problem arises, such as an ear infection, the person may become acutely aware of the body's sense of balance and motion. As with the sense of touch, some vestibular fibres travel from the brain stem to the thalamus and from there, via relay cells, to the cerebral cortex, where perception of bodily motion and position becomes conscious (Eliot 1999:147).

iv) Early vulnerability

Because of its rapid development, the vestibular system is especially vulnerable during the prenatal period. The following could have an effect on the development, maturation and functioning of the vestibular system (Bluestone 1994):

- Aminoglycosides, streptomycin (antibiotics) and toxins
- Lack of movement (mother's before birth as well as the baby after birth)
- Explosive birth and caesarean section as the birth process and the tactile input and pressure associated with normal birth sends messages to the RAS to activate the cranial nerves to start functioning
- Invasive procedures such as insertion of grommets
- Ear infections (bacteria can affect the middle ear, or the bone surrounding the inner ear, and can produce toxins that inflame the cochlea or the vestibular system or both. Chronic untreated otitis media is the more common type of inner ear infection)
- Bacterial meningitis in the inner ear. Bacteria can also enter the labyrinth in the membranes that separate the middle from the inner ear, which are ruptured by a disease like otitis media or by an injury, for example, perilymph fistula

The importance of the vestibular system in establishing a sense of balance

Balance is the core function of all beings living on earth. Goddard (1996:42) states that every living creature shares one relationship: a relationship with gravity. It is gravity that provides us with our centre, whether it be in space, in time, motion, depth or sense of self Goddard (1996:42) states that balance is the nucleus from which all operations become possible. Problems in the balance system have repercussions for all other areas of functioning. Such problems affect the sensory systems because all sensation passes through the vestibular mechanism at brain stem level before being transmitted elsewhere for analysis (Goddard 1996:42).



Many developmental specialists (e.g., Ayres 1974, 1979, 1982; Delecarto 1959, 1974; Kephart 1975; Levinson 1990; Perloung 1997, 2003; Bluestone 1994, 2000; Kokot 2003a, 2003b; Fredericks, Krog & Kokot 2006; Hannaford 1995) have focused on the vestibular system as playing a fundamental role in development. Gravity and its constant demand on the vestibular system attests to the importance of this tiny system.

Levinson (1990:10) states that "90% or more of all people who seek help for impaired concentration and related symptoms of ADD suffer from a physiological problem within the inner_ear." He continues by stating that since nearly 90% of dyslexic or LD patients had problems related to concentration, distractibility, hyperactivity, and impulsivity, and as 90% of ADD patients had symptoms of related dyslexia or LD, it is clear that all are suffering from the same underlying disorder, regardless of the terms used (Levinson 1990:49). All these so-called separate disorders are in fact symptoms of a single disorder with overlapping features in the majority of patients.

Levinson (1990:52) studied over a thousand dyslectic profiles and noted that over 50% showed distinct evidence of impaired balance and/or coordination (only 1% of the same 1000 patients exhibited evidence that suggested to possible cortical dysfunction). Of the 1000 patients, the vast majority reported similar histories: delayed ability to sit, crawl, walk and/or talk, difficulty skipping, hopping, running and participating in sports; problems learning to ride a bike or walk a balance beam; tendency to trip or fall often; prone to accidents; poor fine motor coordination in such areas as tying shoelaces, buttoning, holding and writing with a pen, drawing within guidelines, using a knife and fork. Additional difficulties reported including: visual fixation, and difficulty tracking letters and words; dyscoordination and dysrhythmic speech functions such as stammering, articulations impairments, shuttering, difficulty with cross-balance and coordination.

Levinson concluded that all had an inner ear-related problem, adding that "these findings were confirmed in repeated studies, one of the latest consisting of the examination of four thousand learning disabled children, adolescents and adults" (Levinson 1990:52). After treating more that 20 000 people, Levinson (1990:52)

confirmed that AD/HD has an underlying common denominator, and that this neurophysiological or chemical malfunction lies primarily within the inner ear system.

In chapter 3 (section 3.3.3), whilst discussing the cerebellum, information was presented about the specific and direct link between the cerebellum and the vestibular system. The link is that the vestibular system orientates the body, which informs the cerebellum on how to modulate movement. Levinson (1990:22-25) suggests the following metaphors to describe the vestibular system and emphasise its role and function:

- The VS is like a guided missile computer system, guiding our eyes, hands, feet and various mental and physical functions in time and space. Misguidance results in poor and unrecognisable handwriting, eyes that cannot fixate, legs that run in an uncoordinated way.
- 2) This inner ear system fine-tunes all motor (voluntary and involuntary) responses leaving the brain and all sensory responses coming into the brain. If voluntary motor responses leave the brain without being fully tuned, the person's motor actions become uncoordinated and imbalanced. If involuntary motor responses leave the brain untuned, then toilet training delays may arise, as well as soiling. If sensory information enters the brain untuned, then the information is scrambled. The brain will thus struggle to interpret it. Some sensory information may then be received clearly while others may be received incorrectly or not at all.
- 3) The inner ear is also a compass system. It reflectively tells us spatial relations such as left and right, up and down, front and back. This compass system directs all bodily functions, including sensory, motor, speech, thought, even biological patterns.
- 4) The inner ear also acts as a timing mechanism, setting rhythms to motor tasks. A disturbance within this mechanism may result in difficulty in learning to tell time and sensing time.

Levinson (1990:163) treats his patients with anti-motion sickness medication, but laments: "Do these medications cure an inner ear disturbance? Unfortunately, the answer is no - at present, there are no medications or surgical procedures that are



capable of accomplishing that feat." He does mention, however, that movement can strengthen the vestibular system.

If repetitive motor tasks indeed improve underlying inner ear mechanisms, and if the assumption is made that this improvement extends to, or is transferred to, neighbouring inner ear circuits or channels, then we can readily explain the generalised improvements that occur when specific circuits are strengthened by repetition, practice, or coordination. But this does not always occur. Levinson (1990:27) noted that when astronauts were prepared to enter space, they were spun in various directions. An interesting observation was made. If they were spun only in a counter-clockwise pattern, they were better able to interpret counter-clockwise movements and not necessarily clockwise movements. This points to the specifics that the brain needs in terms of treatment.

4.5 Kinaesthetic System

Kinesthesia can best be described as muscle and joint memory. Generally, the kinaesthetic process can be described as the result of the process whereby children separate or differentiate the body parts into isolated movements, and then begin to recombine them into schema. As children master movements and recombine them, they attend to the sensation of movement (kinaesthesia), and, in the process, develop kinaesthetic figure-ground. As kinaesthetic figure-ground becomes established, the newly developed schema becomes automatic. The body can now operate without constant attention to movement (Heiniger & Randolph 1981:164). Without kinaesthesia, infants, children and adults are dominated by their primitive, protected responses (Heiniger & Randolph 1981:215). Kinaesthesia is interdependent on tactility, differentiation, proprioception and vestibular function as well as other subsystems. It is kinaesthetic memory that allows us to walk without thinking about the process (Reeves & Cermak 2002:71).

4.6 Muscle Tone System

Muscle tone is the amount of tension (or degree of readiness to respond) normally present when our muscles are in a resting state (muscles never relax completely
unless the organism is unconscious (Kranowitz 1998:110). Muscle tone is developed through movement with the tightening and relaxing of muscles, particularly in our efforts to maintain equilibrium. It is regulated by the vestibular system (Bluestone 1994; Kranowitz, 1998:109).

The vestibular system affects tone by regulating neurological information from the brain to the muscles, telling them exactly how much to contract, so that the person can resist gravity to perform skilled tasks. Usually muscle tone is perfect (not too loose and not too tight) so that the person do not have to use much effort to move and keep upright (Kranowitz 1998:111). A child with vestibular dysfunction may have a loose and floppy body, or low muscle tone. No structural dysfunctions are present in the physical muscles of the person presenting with low muscle tone. The brain is simply not sending out sufficient messages to control the tension in the muscles (Kranowitz 1998:111).

Vestibulo-proprioceptive input determines muscle tone. As the muscle tone in the extensor muscles develops it can be used to achieve co-contraction. The midline stability from co-contraction provides a stable base from which the extremities can move. This internal awareness of the difference between the muscle tone of the stable truck (ground) and the changing tone of the extremities (figure), including the neck, constitutes kinaesthetic figure-ground (Heiniger & Randolph 1981:162).

Muscles may be **hypotonic** or **hypertonic**, and muscle tone may exist in a reciprocal pattern of imbalance or a co-contraction pattern of balance (Heiniger & Randolph 1981:132). Since muscle tone is the readiness to respond, individuals with diminished muscle tone may have a delayed response time, and may mistakenly be thought to have diminished intelligence (Bluestone 1994). Alternatively, individuals with overly tense muscle tone may respond so rapidly that they may not have heard and processed the complete instruction given before they move to act on it (Bluestone 1994). Such people may be considered impulsive.

Another link has been made between low muscle tone and high motor activity. This principle is based on the relationship between the vestibular system and muscle tone. As long as the vestibular system generates adequate muscle tone, children do

not have to use much effort or concentration to maintain their position against gravity. If the vestibular system is disorganised, the muscles have low tone and the person may get tired easily. The vestibular system also helps balance the level of arousal of the nervous system. An underactive vestibular system may contribute to hyperactivity and distractibility because of the lack of its ability to modulate (Ayres 1982:72-74).

4.7 Proprioceptive System

Closely allied to the other senses and interdependent upon them is the compound sense of proprioception. While proprioception is the result of multisensory information, it also forms an information channel of its own. Proprioception and kinesthesis tell us where parts of our body are at any time, and allow us to make the appropriate postural adjustments. It is an internal sense (one of the 'near senses') of physical self, which allows us to carry out detailed manoeuvers without conscious awareness and in the absence of other sensory cues (Goddard 1996:61). Proprioception thus refers to the brain's unconscious sense of body-in-space.

Proprioception differs from kinaesthesia in that kinaesthesia is the sense of relative muscle, joint and tendon position in specific situations. Kinaesthetic memory involves learning these positions and the sequence of shifts in these positions for rote, repeated movements (such as gymnastics). Proprioception is a dynamic sense, allowing continuous accommodations and adaptations to a shifting environment (such as in dance, or moving through a crowded room) (Bluestone 1994).

Proprioceptors are located throughout the body in the tendons, joints, and muscles. Their input is processed primarily through the vestibular system, but also coordinates with information from all other sensory sources to influence body movements and direct adjustments for fine muscle coordination. Needless to say, distorted information coming from any one of these sources will also affect proprioception (Goddard 1996:61).

While proprioception and kinaesthesis are often used interchangeably, the term 'proprioception' encompasses all sensations involving body position, either at rest or in motion; while kinaesthesia refers only to sensations arising when active muscle

contraction becomes involved. Thus, some children who have little proprioceptive input when they sit still may constantly have to move because they rely on information from the movement of muscle to provide proprioceptive input.

Essentially the body uses five systems to determine its position relative to the external environment and to determine where various parts of the bodies are in relation to one another (Bluestone 1994):

- the information received by the brain from the vestibular system regarding the position of the heads, the pull of gravity, the speed and acceleration of our movement
- 2. the interpretation of messages received by our eyes about both the external space and the body's position and posture in this space
- 3. the assorted information received by our brain from tactile, kinaesthetic and proprioceptive sited located throughout the body
- 4. the messages received by the brain through smell, a sense on which the person unconsciously rely to discern direction and distance from objects and events in his/her environment
- 5. the interpretation of the messages from the auditory system, which also helps to orient ourselves to specific objects and events in the environment

If any of these functions is irregular, the person has either a diminished sense of body-in-space, or relies more heavily on another system (such as sound or vision) to compensate for this. In turn, this may mean the individual uses these systems ineffectively for broader or higher level functions than were originally meant (Bluestone 1994; Goddard 1996:61).

Goddard (1996:61) also believes that many children with poor individual sensory perception attempt to use proprioception for learning instead of the appropriate primary channels. Goddard compares this to using a huge net in an attempt to catch one individual fish. Learning can only be consistently successful if eyes, ears and balance system also provide accurate and consistent information about changing circumstances in the environment. Children who attempt to use proprioception to compensate for a weakness in another channel may require extra practice to master a skill. The quality in performance of such children is also often inconsistent.

4.8 Oral Motor System

Sucking is a powerful organiser of oral motor, facial, and sphincter muscle functioning. The trigeminal nerve, arising from the fifth cranial nerve, is a sensory nerve that branches to the eyes, sinuses, tongue, every tooth, nose, face and middle ear by way of the optic ganglion. Stimulation of one area of the nerve stimulates all areas served by the nerve. Information sent to the cerebellum by way of the trigeminal nerve pathway generates messages sent to the same areas by way of the motoric facial nerve. Use of the sphincter muscles surrounding the mouth to suck stimulates other sphincter muscles in the body, such as the pupils, oesophageal sphincter, and the muscles for bowel and bladder control (Bluestone 1994). The oral motor system is specifically important in nutrition, communication, speech, sound, bladder control, facial expressions and dentition.

4.9 Visual System

Compared to the other senses discussed so far, the sense of vision is still primitive at the time of birth. Due to the rapid wiring of neurons in the visual cortex, however, this sense improves dramatically within a few short months after birth. By six months of age, all the primary visual abilities have emerged, such as fine acuity, depth perception, colour vision, and well-controlled eye movements (ocular motility). By one year the visual system is almost fully functional, allowing the individual to engage in and benefit from the rich visual universe in all of its colourful, three-dimensional glory (Eliot 1999:196). Seiderman and Marais (1989:4) state that humans are born with sight, but vision is learned. In the first days after birth, the baby can see, but cannot do so in any meaningful sense because the brain has not yet learned how to see (perception).

The retina (a very thin membrane at the back of the eyeball) is where **sight** and **vision** meet. Sight can be explained as the light that enters the eye and falls on the retina. Sight is the process that occurs in the eye itself. Light is transformed into electrical messages on the retina and is sent to the part of the brain that deals with the visual images (occipital lobe). Vision is the process behind the eye, the interaction between the eye and the brain (perception) (Seiderman & Marais 1989:4).

i) Development

Eliot (1999:197-204) describes the neurodevelopmental aspects of the sense of sight: The physical apparatus for this sense starts developing in the fourth week of embryonic life, with the initial formation of the eye. It then proceeds in a sequence from outside in: neurons and synapses form first in the retina, followed by subcortical visual areas, then the primary visual cortex, and lastly, the higher visual centres in the temporal and parietal lobes. In spite of its early initial development, the visual system is only optimally functional in middle childhood when the pathways are firmly stabilised.

The first optic tissue emerges a mere twenty-two days after fertilisation, with the formation of two large bubbles at the front of the neural tube. By five weeks, these bubbles have collapsed into two cup-shaped structures and differentiated to include both the retina and the lens. Each 'eye cup' is attached to the brain by a short, broad stalk and takes up a large proportion of the space inside the primitive head. From the outside, however, they appear as just two small spots, facing sideways like a bird's or lizard's eyes. By eight weeks, the spots have migrated to face forward and now look like human eyes.

The retina itself consists entirely of neural ectoderm and consequently develops like a mini-brain in itself. Its neurons divide and migrate to successive layers, where they take up functions distinct to their layer. The first layer of neurons to emerge is the ganglion cells, all of which are formed between about six and twenty weeks of gestation. Ganglion cells immediately sprout their axons, and as early as eight weeks of gestation, young fibres can be found emerging out of the eye stalk, where they begin forming the optic nerves. The nerve thus develops from the eye to the brain.

The next visual relay station, the **lateral geniculate nucleus** (LGN), forms later than those in the retina, but the whole process is much quicker and by just eleven weeks of gestation, all the LGN neurons have formed. By the end of the first trimester, they receive their first synapses from retinal ganglion cells. Synapses continue forming in the LGN until early postnatal life, but these later connections come from the cerebral

cortex, and are responsible for establishing cortical control over lower-brain visual functions.

The second trimester marks a period of massive growth in the visual cortex. All of the 100 million neurons in the primary visual cortex are formed between just fourteen and twenty-eight weeks of gestation. In the fifth month, synapses begin forming in the primary visual cortex, but it is only the beginning of a process that continues for nearly another full year, at the astonishing rate of some ten billion new synapses per day.

Synaptic density reaches its peak in the primary visual cortex at around eight months after birth. Then, around age two, it begins a slow decline that lasts until late childhood. This long period of synaptic pruning, in which about 40% of visual cortex synapses are eliminated and the remaining circuits grow progressively more efficient, coincides with the gradual refinement of many visual skills. It also corresponds to the outer limit of the critical period for visual development.

Just as the emergence of cells and synapses progresses inward (from the eye, to the LGN, to the primary visual cortex to higher cortical areas), the myelination of visual axons proceeds along a similar gradient. The optic nerves begin to myelinate two months before birth, continuing until seven months postnatally. LGN neurons, however, do not begin to myelinate until seven weeks after birth, and they continue until about eight months of age. Within the primary visual cortex, cells from different layers myelinate in the same sequence in which their synapses formed. Finally, higher visual areas myelinate even later that the primary visual cortex, some of them continuing until mid-childhood.

The processing that makes vision seem effortless is actually enormously complex. The brain devotes more of its territory to vision that to all the other senses combined. It is due to the massive processing space, and the special ways in which its intricate components are wired together, that the brain can rapidly perform the complicated computations that make everyday visual tasks seem so easy.

ii) Functioning

Sight begins in the eye, when light passes through the **cornea** (the transparent outer coating), is focused by the **lens** and then strikes the **retina** (a three-layered blanket of neurons that covers the entire back surface of the eyeball). At this point the process turns into **vision** as the journey through the nervous system begins. During this process light is converted into electrical signals that map out colour and intensity at each point in the visual field.

The extraction of visual information thus begins at the very first level of visual processing, within the retina. When the human organism looks at an object, light reflects from the objects, striking the two different types of **photoreceptors** in the retina, **rods** and **cones**. Photoreceptors are specialised nerve cells that contain pigment molecules capable of capturing a single light particle or photon, and convert its energy into a chemical reaction (these pigment molecules are derived from vitamin A). The resulting chemical cascade in turn produces an electrical signal that begins the process of neural transmission in the visual system (Eliot 1999:198).

Like most sensory and motor abilities, visual perception is divided in half. The left side of the brain receives input from the right half of the visual field, and vice versa. This division actually requires some tricky routing on the part of retinal ganglion cells,



Figure 37 Visual pathway

This scan shows the optic nerve (yellow) that runs from the retina of each eye to the visual cortex at the back of the brain (Winston 2004:125) because each eye takes in some light from both the left and right halves of our visual field. Ganglion cell axons sort themselves out when they reach the crossover point of the two optic nerves, the **optic chiasm** (see figure 22). Regardless of the eye in which they originate, axons that "see" the right side of the visual field terminate in the left LGN and vice versa. Left LGN neurons then project exclusively to the left visual cortex. Thus, each side of the visual cortex contains a map of the visual field on the opposite side of the body. Figure 37 illustrates the neural pathway of the visual system.

The two parallel routes carry visual information from the occipital lobe to the prefrontal lobe and the frontal eye fields. Fibres from these two routes distribute



fibres to many other areas along each route before terminating in the prefrontal cortex and the frontal eye fields. The first route is the superior route via the parietal and frontal lobes. The other route is the inferior route that runs via the temporal and frontal lobes (Scheiman 2002:14).

The flip side of this routing is that each side of the brain receives input from both eyes. Despite the mixing of left and right ganglion cell axons at the optic chiasm, their synaptic terminals remain carefully segregated. All visual information originating from the right field travels in the left optic nerve tract, and all of the visual information originating from the left field travels in the right optic tract (Scheiman 2002:13) The LGN is composed of several layers of cells, and each layer receives input from only one eye. Similarly, in the primary visual cortex, whole columns of neurons are devoted exclusively to the input from one or the other eye. Every neuron in the LGN and in the first stage of the cerebral cortex is thus said to be **monocular**, while in the next stage of cortical processing, neurons receive input from both left and right monocular neurons and are known as **binocular**. The initial segregation of left and right eye inputs, as well as their successful reconvergence into binocular neurons, is essential for depth perception and other abilities. Such binocular abilities, however, are not present at birth, and their development is strongly influenced by a child's early visual experience (Eliot 1999:200), eye muscles and myelination of the nerves.

Sight is measured by determining at what distance a person can see clearly (20/20 is usually an indication of normal sight). Typically, a visual assessment focuses on visual acuity. If a problem is detected in any or both of the lenses, the person will receive a prescription in the form of a corrective lens. This is actually only the beginning of the physiological aspect of sight and vision. Humans are binocular beings because they have two forward-facing eyes that feed information to the brain at the same time that the brain must make sense of the information presented to it.

"Seeing" actually refers to vision, as the eye does not know and understand the information coming through it. The brain scans the messages sent by the eye to estimate size, shape and colour, comparing the results with images stored in visual memory, and concluding that what is seen is known as the object being looked at (Seiderman & Marais 1989:3-6). The volume of information collected by the eyes is

astonishing. Each eye sends the brain a billion messages during every waking second. Together the eyes send twice as many messages as the entire rest of the body, including all the other senses.

Levinson (1990:120) states that the vestibular and visual systems often work together to provide balance and proper posture. When the brain receives messages from the neurons in the inner ear, it can instruct the eyes to change their position in relation to the objects in the visual field while the body is in motion. Because of this working partnership, the individual is able to keep a fixed gaze on the object that is being focused on. While the individual moves towards the object, other systems also operate. These are proprioception, kinaesthesia, audition, and muscle tone, to name but a few.

Vision is obviously essential for academic learning and thus has a direct impact on higher cognitive functions. The skills of reading, writing, spelling and arithmetic are all dependent upon the ability to see written symbols. When learning difficulties arise, vision is often the first area to be checked. If the child passes a simple eye test, which only assesses acuity vision, further investigation into visual problems is seldom pursued. Acuity is only one component in the complex sense of sight. How the person sees, the way that the eyes are used and the perception of the world through sight, is the result of a complex series of connections and neural developments which should have taken place in the early formative years, and which are dependent upon adequate maturation of the central nervous system (CNS) (Goddard 1996:55). Oculo-motor, visual-perceptual and visual-motor integration skills are just as vital for learning as is good acuity sight. These functions will be discussed in the following section.





Figure 38 Eye muscles

The eye muscles work in pais to roll the eyes in various directions; the movements

are tiny but precisely controlled. Looking in and out: The lateral and medial rectus muscles move the eye to the mose and to the side. Looking up and down: The superiour and inferiour rectus muscles move the eye upwards and downwards. Looking up, down, and out: The superiour and inferiour oblique muscles roll the eye upwards, downwards, and to the sides (Winston 2004:124).

Ocular motility (frequently called visual tracking or scanning) is the ability of the eyes to move smoothly over all planes. Ocular motility includes fixation, saccades and pursuits (Scheiman 2002:62). Visual scanning is one of the primary means by which the central nervous system obtains visual information from the environment. Scanning involves both eye movement and fixation. When there are problems with ocular motility, specialists in the field usually refer to the three-component model of eye movement that includes **fixation**, **saccades** and **pursuits** (Scheiman 2002:62). This ability is dependent on the three sets of muscles (figure 38) and the cranial nerves (figure 18) that serve the eyes, as well as on the vestibular system, which provides information and regulation. This system also relies on lateralisation and the ability tocross the visual midline. Tracking is vital for the orderly progression from one word to another, and for finding the way from line to line without losing the place.





The right and left eyes send separate signals to the right and left visual cortex. The brain integrates the signals into a composite image (Winston 2004:125).

Anatomically each eye has three sets of muscles for controlled eye movement. Each of the six muscles has one position of gaze in which it exerts the main influence on eye position. Three cranial nerves supply innervation to the six extraocular muscles. The third cranial nerve innervates the superior, inferior, medial rectal, and the inferior oblique muscles. The fourth cranial nerve supplies innervation to the superior oblique muscle, and the sixth cranial nerve innervates the lateral rectus (Scheiman 2002:12).

Binocularity

Since humans are binocular beings, both eyes must work as a team so that each eye is directed to the same fixation point on the object. **Binocular vision**, the product of the two eyes working together, is what enables the individual to see in three dimensions and is essential to almost everything (Seiderman & Marais 1989:3-6). The most commonly observed aspects of binocular functions include **convergence** (the ability of the two eyes to team and focus on the same object), and **accommodation** (the ability of the eyes to shift their focus from near point to far point

or vice versa). Difficulties with either accommodation or convergence can affect the other function (Goddard 1996:56).

Some common problems of binocular functions include weak convergence, amblyopia (lazy or wandering eyes), over-convergence, alternating suppression, and light sensitivity, to name but a few. A number of activities that are part of normal development promote the development of binocular vision. The **sucking reflex** is one of the most influential of these. Kinaesthetic movements and muscle tone, as well as differentiation and interhemispheric integration, are also instrumental in the development of binocular vision (Bluestone 1994).

Directional awareness is as much a visual as a vestibular-based skill. The vestibular system acts like an internal compass providing a sense of "centre" from which the person can automatically judge up from down, left from right, start from finish. Neurodevelopmental delay could have an adverse effect on vision. Goddard (1996:57) describes how aberrant reflex activity can effect oculomotor functioning. The asymmetrical tonic neck reflex (ATNR) can have an adverse effect upon tracking, the tonic labyrinthine reflex (TLR) upon convergence, the Moro reflex upon fixation and the symmetrical tonic neck reflex STNR upon the readjustment of binocular vision from one distance to another.

Interaction between the components of this loop determines the efficiency of the visual system in later life, in other words, the rapid exchange of information between the vestibular apparatus, the eyes, and the level of reflex response to incoming stimuli. Any defect in one of these elements affects the smooth operation of the whole (Goddard 1996:60).

4.10 Auditory System

Like touch, smell, taste and the vestibular senses, the neural structures underlying the auditory system form early in utero and begin functioning well before the end of gestation. By birth, babies already have about twelve weeks' worth of actual listening experience and they have even become somewhat discriminating about what they

like to hear. In contrast to vision, where newborns show a distinct preference for similar stimuli, their hearing preferences tilt toward the more complex, and music or highly intonated speech is preferred to pure tones or other simple sounds (Eliot 1999:234).

The development of hearing also contrasts with vision in other ways as well. Whereas vision emerges late and matures quickly, hearing begins early but matures gradually. In human babies, in particular, hearing is much better at birth than in most young mammals, but their auditory skills continue to improve over a very long period up to school-going age (Eliot 1999:234). The gradual mastery of language parallels the development of the auditory system.

One feature that listening does share with vision is it ability to be modified by experience. The act of hearing itself influences the quality of auditory development, and all the listening that children do, from the third trimester on, importantly shapes the way their brains become wired to process and understand different sounds. This stimulation does not only affect the auditory system. Children's early experience with speech and music are tremendously important in shaping many higher aspects of brain function, including emotion, language and other cognitive abilities (Eliot 1999:228). Madaule (1994:xii) states that like any other skill, listening can be maximised through exposure and experience.

i) Neurodevelopmental process

The human ear is a compound developmental organ that forms during the second half of the embryonic period (4 – 8 weeks in utero). After just four weeks in the womb, two fireplug-shaped structures emerged on either side of the embryonic head, the primordial otocysts. These structures evolve into both the cochlea and vestibular organs (see figures 32 and 33). Between five and ten weeks in utero, while the vestibular canals are branching out of the top of the otocysts, two cochlear tubes elongate from the bottom, coiling as they grow. By eleven weeks, the cochleas have completed all of their turns and look much like the snail shells they resemble in adults, although they continue to grow in girth until mid-gestation. Between ten and twenty weeks of development, all of the roughly 16 000 hair cells (in each cochlea)

mature, sprouting their characteristic cilia and forming synapses with the first neurons in the auditory system (Eliot 1999:234; Goddard 1996:48).

Hair cells do not emerge simultaneously along the cochlea but in a gradient, beginning at the base and finishing at the apex, or coiled end. This gradient presents somewhat of a paradox because foetuses can first hear only in the low-frequency range, the part of the sound spectrum that is normally sensed by hair cells at the apex. But the paradox is explained by the fact that individual hair cells actually change their frequency response during development. Cells near the base, which mature first, initially respond to lower frequencies, but as the properties of the basilar membrane change, these cells become sensitive to higher and higher tones, while hair cells farther out in the cochlea, which are just beginning to mature, take over the function of sensing low frequencies. This signifies a gradual shift in the tonotopic map during development, with lower tones moving out to progressively more distant cochlear locations (Eliot 1999:234).

Well before the cochleas are fully formed, auditory neurons emerge in the embryonic brain stem. The earliest auditory neurons appear at just three weeks' gestation. By six weeks, the auditory nerve, the cochlear nuclei, and the superior olive are all clearly distinguishable. Higher brain stem auditory centres are apparent by thirteen weeks, and the **medical geniculate nucleus** (MGN) begins to show its adult-like subdivisions at just seventeen weeks. Cortical neurons emerge somewhat later, but in general, the auditory cortex matures earlier than any other part of the cerebral cortex, with the exception of areas involved in touch perception. In all parts of the auditory system, synapses form shortly after growing axons reach their targets, but there is a prolonged period of synaptic refinement during which the tonotopic maps become more sharply defined.

The pace of auditory development is perhaps best revealed by its pattern of myelination (see figure 49). Like other tracts in the brain stem, the first several projections in the auditory system start to myelinate quite early in development. By twenty-four weeks of gestation, all of the relays between the ear and the inferior colliculus have begun myelinating, and by birth, they are nearly fully insulated. By contrast, the higher relay tracts of the auditory system myelinate only gradually and

are not fully myelinated until at least two years of age (Eliot 1999:234; Goddard 1996:48). During the first three years of life, children must learn to use their ears to "tune in" to the specific frequencies and sound structures of the home language, in much the same way that a radio is adjusted to select specific stations. It is as this time that the child has the potential to learn any language if it is exposed to the sounds of that language continuously over a period of time, no matter what language his mother speaks. After the age of three years, when these fine-tuning adjustments have been made, it becomes far more difficult to assimilate a new language (Goddard 1996:48).



Neural pathway of hearing

Auditory information is shuttled from the cochlea through several relays in the brain stem, midbrain, and thalamus, before reaching the cerebral cortex (Eliot 1999:233).

Like vestibular sensation and touch, hearing is the reception and transmission of energy through motion and vibration. Sound waves are produced whenever a physically vibrating source, such as a violin string, a person's vocal cords, or a jet engine, creates an alternating pressure change in the surrounding medium (air or water). A sound wave is characterised by its amplitude (the height of its peaks), and its frequency (the number of times the wave crests per second) (Eliot 1999:229). The human being can detect sounds between 20 and 20,000Hz.

The cochlear hair cells synapse onto primary auditory neurons, cells whose axons travel from the inner ear, through the auditory nerve, to reach the **cochlear nucleus** in the lower brain stem. Then, from the cochlear nucleus, some auditory fibres travel up to a higher brain stem site, the **superior olive**, which plays an important role in localising sounds in space. The next relay station is in the midbrain, an area called the **inferiour colliculus**, which is located, as its name implies, just below the visual system's superior colliculus. The last stop before the cortex is the thalamus, in a region called the **medical geniculate nucleus** or MGN (adjacent to the visual system's lateral geniculate nucleus). Finally, fibres from the MGN travel to the primary auditory region of the cerebral cortex, which is located on the upper ridge of the temporal lobe. Higher order auditory areas surround this primary auditory cortex, but they are also all located within the upper part of the temporal lobe (Eliot 1999:232).

ii) Functionality

Madaule, 1994:xi) lists all the functions of the ear. He states that "we usually tend to associate the ear with hearing alone. Yes, we do hear with our ears, but that doesn't mean that hearing is the ears' sole function." A major part of the sensory energy received by the brain comes thought the ears. They control balance, body movement and coordination; they permit language, they make us speak eloquently and sing in tune, they even control our eyes when we read and our arm, hand and fingers when we write. And if that is not enough, they even protect us from what we do not want to hear, starting with the sounds of our own body. Interconnected with several different levels of the brain, the ears act as a double antenna receiving messages from both the body and the environment. They are a link between the world within and the

world without. Listening – the ability and the desire to use our ears – brings about the harmony both within us and in our relationship with others (Madaule, 1994:xii).



transmitted as vibrations through the eardrum and the middle ear to the cochlea in the inner ear (Winston 2004:126).

When listening does not develop correctly, the harmony is broken and optimal communication is cut off. Problems such as speech and language impairments, hyperactivity, depression, autism, feeling overwhelmed or lacking a direction in life are some of the possible consequences of auditory dysfunction. Reading problems such as dyslexia and other learning disabilities have seldom been considered and treated as listening problems (Madaule 1994:xii). Listening is thus the ability to tune in to sound messages and to tune out at will. A listening problem is the inability to perform this function well, if at all (Madaule 1994:33).





Figure 42 Listening profile

The difference between hearing and listening is best illustrated by the evaluation methods used by auditory integration practitioners, first introduced by Tomatis (1991). Previously, hearing had always been thought of in **quantitative terms:** whether one could or could not hear, and if so, to what extent. For the first time, the **qualitative aspect** (how one could hear, or a way of hearing) was introduced into the notion of audition (Madaule 1994:35). An illustration of the qualitative method of evaluating audition is presented in figure 42.

The illustrated **listening profile** (figure 42) shows the 11 frequencies usually tested during an auditory screening test. Normal hearing falls between 0 – 20Hz. This is usually the main focus of an audiologist's assessment. Any value obtained below 20Hz is considered a mild to profound hearing loss, depending on the quantitative placement. However, the qualitative interpretation is different. Even in a person with normal hearing, sounds at 4000Hz would be extremely difficult to hear if this frequency were presented at 10dB, like most other sounds. A word containing a 4000Hz sound could then be distorted. A distorted perception of the world affects the person's understanding of the world.

Tomatis, the originator of this viewpoint, also introduced another novel concept: the concept of an "active ear" as opposed to a "passive ear." The process is explained by Madaule (1994:38-39) in his book *When Listening Comes Alive*. Two mechanisms operate in vision. One permits visual perception, allowing us to see. The other makes

focusing possible, due to the mobility of ocular globes and the adaptation of the pupils. This allows us to look, or not look, at a particular object. Auditory phenomena that entail "focusing" qualities such as auditory processing, auditory discrimination or attention span, are usually explained at the level of brain processing. It is assumed that the ear passes all the information on to the brain and that it is the brain that makes the selection. Although the brain is responsible for ultimately selecting and processing information, why would it not involve the ear in the selection process, just as it does with the converging ocular globes in vision? In other words, why would the brain not actively involve the ear in focusing, or more appropriately, in attuning to sound?

The traditional description of hearing explains the passive part of sound perception (what corresponds to seeing in vision) and assigns an active role to the ear in exceptional situations only. When exposed to extremely loud sounds, the ear protects itself with the help of two tiny muscles located in the middle ear. This is known as the "acoustic reflex". The general consensus on the involvement of the middle ear muscles in protecting the cochlea has not gone unchallenged in recent years. The delay between the sudden onset of the sound and the protective action of the muscle would allow damage to the cochlea to take place. Also, the fact that sounds of dangerously high intensity are extremely rare in nature, brings into question the assignment of an exclusively protective role to these muscles. They are the hammer muscle (tensor tympani), and the stirrup muscle (stapedius). When sounds are dangerously loud, the hammer muscle attenuates the vibration of the incoming sound vibration.

The role of these middle ear muscles has been limited to such extreme situations, although recent findings suggest that they are much more important than previously thought. In particular, some audiologists are recognising that the stirrup muscle facilitates sound discrimination. These findings support Tomatis's theory.

Suggesting that the middle ear muscles play a role in attunement does not exclude their traditional role of protection against loud noises. To attune to the sounds the



individual wants to focus on, automatically implies cutting off the unwanted sounds. That is what happens when the person tries to converse in a noisy environment.

The vestibular apparatus is also a filtering point for sound. The ear acts as a collecting organ not only for sound stimuli, but also for those stimuli that are responsible for coordinating the vestibular portion of the labyrinth. If, as a result of aberrant reflexes, the vestibular system deals with conflicting messages from other sensory channels, it may impair its ability to process and relay sound messages to the language processing centres in the cortex (Goddard 1996:103).

Gomez and Condon (1999:150) compared central auditory processing deficit (CAPD) in children diagnosed with AD/HD and children without such a diagnosis. The study demonstrated a clear and significant difference in the two populations, with AD/HD children presenting with more CAPD. There seem to be two theories surrounding CAPD: the audio-logical model, which claims that CAPD is a perceptual disorder involving problems of attention, analysis and comprehension of relative auditory stimulation; and the speech-language model, which considers dysfunction to be due to poor linguistic, semantic and cognitive processing (Gomez & Condon 1999:150).

5. Integration of the Basic Neurological Subsystems

Goddard (1996:120) states that our eyes operate from the vestibular circuit in the brain. Our ears share the same cranial nerve, and the sense of touch is integrally linked to the vestibular through the movement across hair cells whose receptors are located in the dermis of the skin. If motion is children's first language, then sensation is their second. Only when both motion and sensation are integrated can the higher language skills of speech, reading and writing develop fluently.

All learning takes place in the brain, but it is the body that acts as the vehicle by which knowledge is acquired. Both brain and body work together, through the CNS, but both is dependent on the senses for all information about the outer world (Goddard 1996:41). Even though the senses are individually discussed in the previous section, none of the senses actually work or function in isolation. All the

senses transmit into the brain at the same time. There needs to be a balance between the different sensory channels so that cross-sensory reference may occur. This provides individuals with multi-sensory information about their environment, to which they may then adapt their responses. This is what is referred to as sensory integration (Goddard 1996:41). In a study by Dunn and Bennett (2002:7), the researchers found a significant correlation between children presenting with AD/HD and items on the Sensory Profile, indicating that these children generally present more sensory difficulties than children without AD/HD.

Integration is the neurological process of organising the information received from the body through the senses from the world. The process occurs in the central nervous system, which consists of countless neurons, the spine and the brain (Kranowitz 1998:42). The main task of the central nervous system is to integrate the senses. According to Ayres (cited in Kranowitz 1998:42), "Over 80 percent of the nervous system is involved in processing or organising sensory input, and thus the brain is primarily a sensory processing machine." When the brain efficiently processes sensory information, responses are appropriate and automatic. This process is possibly because the brain is equipped to modulate sensory messages. Modulation is the term used to describe the brain's regulation of its own activity and, therefore, of the individual's activity level (Kranowitz 1998:42). Activity level can be high, low or in between. Modulation balances the flow of sensory information coming into the central nervous system. The brain turns on, or turns off, the neural switches of all the sensory systems, so that they work in tandem to help people function in a smooth, synchronised way. Every minute human beings receive millions of sensations. Most of these are irrelevant to their current situations. Therefore, the brain inhibits some sensory information. **Inhibition** is the neurological process that reduces connections between sensory intake and behavioural output (Kranowitz 1998:43).

No one part of the nervous system works alone. Messages must go back and forth from one part to another, so that touch can aid vision, vision can aid balance, balance can aid body awareness, body awareness can aid movement, movement can aid learning, and so forth. When sensory information is received in an appropriate manner, appropriate motor messages can go out (Kranowitz 1998:45).

Judith Bluestone (1994, 2000; Bluestone & Sulitaneau 2001), director of the HANDLE Institute in Seattle, developed a diagrammatical representation of the integrated and interdependent subsystems responsible for effective functioning. The relative position of each subsystem on the Sensory-motor Interdependency and Interaction Chart is indicative of the hierarchical nature of the neurological system, and illustrates how higher level functions depend on those at a lower level.

The HANDLE (Holistic Approach to Neurodevelopment and Learning Efficiency) perspective defines neurodevelopment not as a given sequence of acquired skills but as an interactive hierarchy of brain functions, with a vestibular foundation for skills (Bluestone 1994). Bluestone (1994) states that other approaches isolate the different structures of the brain; however, when neurodevelopment is understood as interactive, no time frame limits brain function. Learning is thus the lifelong process of using sensory, motor, social and emotional input to realign output into effective behaviour (Kokot 2003a:14). The holistic nature of the approach also requires recognition of internal and external influences. This means acknowledging possible causal roles of chemicals, allergens, nutritional deficits (especially EFA), dehydration and toxins of any kind.

The Sensory-motor Interdependency and Interaction Chart shown below illustrates the dynamic interplay between the different subsystems. The chart depicts a hierarchical trajectory of human development, from bottom to top. The many arrows illustrate that the different subsystems support others while being supported by those beneath them. Systems higher up in the system could have an effect on systems below them. All the systems need to fulfil their function in the interaction, or they risk placing more strain on supporting functions. The chart should also be viewed in terms of the items right at the bottom of the chart. These items include intuition, ecosystem, time, maturation and nutrition. Bluestone proposes that the entire ecosystemic environment (both internal and external) is important when compiling a profile of the child. When trying to understand the reasons for a child's inability to pay attention or control motor activity, Bluestone emphasises both the pre- and postnatal periods in the child's ecosystem. Thus, environmental offenders such as cleaning agents, heavy metals, insecticides, and the like are considered. Allergies and their possible effect of cognitive functioning are explored. The birth and possible stressors in both the mother and foetus are taken into consideration.

The different subsystems depicted in the chart are discussed at length earlier in this chapter. The main point illustrated by this chart, however, is that none of these subsystems can function alone. They all work together to allow individuals to experience the world fully, and to allow people to react to this reality by assimilating incoming information through all the subsystems that permit the entry of information. Accurate assumptions and interpretations are only possible when information entering the organism compares accurately with the environment, and when the information presented is consistent. This allows people to react to incoming information in an appropriate fashion, with appropriate speed and intensity. Weakness in any of the subsystems places strain all the other subsystems due to their interconnectedness with the system as a whole. The chart thus represents the body's subsystems as a Gestalt, where the whole is more than the sum of the parts. The nett effect of a misfiring, weakness, or dysfunction in modulation could result in an unrealistic, inconsistent Gestalt of the perceived world. The long-term effect of this situation could result in emotional and physiological exhaustion that could in itself lead to frustration, misbehaviour and depression.

The Sensory-motor Interdependency and Interaction Chart (figure 43) does not only provide a framework for understanding the interplay of the subcortical subsystems, but also provides clues to a therapeutic intervention, starting at the root, and gradually developing and structuring the neural networks that support the cortical systems. A reflective, subjective report (Bluestone & Suliteanu 2001) indicates that 54 out of the referred 59 ADD clients showed long-lasting improvements after a HANDLE programme.



Figure 43

Sensory-Motor Interdependency and Interaction © 1992 Judith Bluestone

6. Disintegration / Disorganisation

At this point it is necessary to refer back to the section on information processing systems. Integration implies that each aspect of this process functions perfectly. Disintegration occurs when there is a glitch in any part of the system.

6.1 Information Processing System

Ineffective processing may occur in any part of the processing system. It may start at **input level**, when the brain receives too little or too much sensory information. The result is that the brain cannot react in a meaningful way. Taking in too much information is called hypersensitivity and taking in too little is called hyposensitivity. In essence, the process of modulation is faulty when too much or to little information is sent through to the brain. Difficulties during the processing stage may be termed neurological disrganisation. On an output level, ineffective motor, language and or emotional outputs may be observed. The brain may be inefficient at processing the sensory messages, thus depriving itself of the feedback it needs in order for people to behave in a purposeful way. These aspects are illustrated in figure 44.



Ineffective processing

Since the processing of information depends on the availability of information, the input system in the information processing system is of utmost importance. Deprivation of even one of the senses is likely to have a profound effect upon the individual, as will any alteration in the transmission of information via one of the

sensory channels. **Mode**, **intensity** and **duration** of sensation are also of prime significance, as the type and degree of sensory input will directly influence the level of response that is given. Too little sensation may result in lack of response. Too much may result in overreaction or vastly increased levels of stress as individuals attempt to maintain control over their response. Distortion or blurring of sensation may result in confusion and inappropriate response (Goddard 1996:63). Any of the mentioned faulty modulation systems could result in physiological stress.

6.2 Physiological Stress

Bluestone (1994:i) emphasises the role of physiological stress in the organism. The human function is intersystemic in nature, and neuro-scientific findings indicate that by performing a non-integrated activity for an extended length of time, the nervous system may become less functional. This is caused by two factors (Bluestone 1994:i):

- Prolonged stimulation of immature functions or systems can pattern an improperly integrated response; in other words, they can form a poor habit, which then needs to be broken.
- Even more significantly, over-stimulation of immature systems can cause the nervous system to become overwhelmed or stressed, which in turn leads to shutdown, a regression to earlier behaviours, and possibly lethargy (exhaustion and sleep).

The vestibular apparatus is also linked to the tenth cranial nerve, also called the vagus nerve (figure 18), at the level of the medulla (Blythe cited in Goddard 1996:117). The vagus nerve contains both sensory and motor fibres. The sensory fibres convey sensitivity to part of the external ear, and carry afferent impulses from the pharynx and the larynx and the internal organs of the thorax and abdomen. The motor fibres and accessory nerves serve the striated muscles of the palate, the larynx and the pharynx. If children are already overloaded by outside stimuli, their compensatory mechanisms are stretched to capacity, so that there is little energy available for expression. Far too much attention is concentrated upon making sense of conflicting perceptions. The confused vestibular system excites the vagus nerve

and its impulses to the organs of speech production. Over-action of the vestibular alerts earlier aberrant reflexes (Goddard 1996:104) or, when the overload is extreme, the Moro reflex. In the latter case, explosive reactions are possible (Goddard 1996:117). Physiological stress could also cause digestive problems as the sympathetic nervous system is activated.

Inappropriate sensory modulation may have an adverse effect on a child, especially if it occurs on an ongoing basis. Children who experience sensory modulation difficulty struggle to adapt to a classroom that is buzzing with noise, movement and activity, and may easily experience a physiological stress response. Such children may be unable to discriminate, categorise and occlude miscellaneous noise or movement immediately, due to a faulty automatic filtering mechanism for the auditory, visual or proprioceptive channels. People must either to attend to outside stimuli or dismiss them through a conscious and methodical route. Both the left and the right brain hemispheres have language centres, but the most efficient is on the left side for the majority of the population. It is the left side of the brain that is responsible for the execution of methodical, sequential tasks, while the right side is responsible for scanning and targeting. For example, if a person were seeking out an object amongst other objects, the left brain would go through every object in order, until it found the one it was looking for. The right brain would scan the objects until it hit on its target. For children without an adequate filtering mechanism, the left brain is greatly overworked, and they may occasionally need to switch to the right brain (subdominant) language centre, which they cannot utilise as fluently. An environment characterised by noise and disorganisation is likely to be a strange and frightening one for children (Goddard 1996:116-117).

Goddard (1996:117-118) explains the process of overdrive as follows: where there is arousal, internal excitation and muscle tension increase. Where there is prolonged muscle tension, eventually there is fatigue. Fatigue reduces performance, so that in order to maintain the same level of performance and make up for the loss of efficiency, there is a need for an increased level of arousal. Thus, a vicious circle is created, in which over-activity of body musculature becomes both a survival and a performance system. Tiredness becomes an enemy to be overcome, not by rest and restoration, but by increased movement and activity, much like changing one gear

up. A crisis occurs after top gear and overdrive have been reached: there are no further gears to engage, but somehow the same level of performance has to be maintained. This may be achieved temporarily by boosting adrenaline in the system, whether this be through drugs, more excitement, or by violent outbursts in the form of anger, extreme depression or states of elation.

6.3 Attentional Priorities

Considering the presented information (neurodevelopment, reflexes, all the subsystems and how they relate to one another, modulation of these senses, processing), it becomes clear that the human brain endures a flood of incoming information at a speed we cannot even comprehend.

Judith Bluestone coined the concept of **attentional priorities difficulties (APD)**. According to Bluestone (1994; 2000) individuals do not present with an attention deficit as the brain has been designed to pay attention to something all the time. However, the brain decides what to pay attention to. Clearly academic performance, social interaction and the like are less important than respiration, circulation, and the modulation of irregular sensory input that leads to general orientation problems due to vestibular overflow. The moment the brain struggles to attend to all its tasks, and the system starts to overflow, survival activities are initiated and secondary activities (such as academic skills) become unimportant.

Using as guidelines the Sensory-motor Interdependency and Interaction Chart (figure 43) and the above discussion on disorganisation and disintegration, professionals treating children diagnosed with AD/HD may ask the following questions:

- 1. Why does this child struggle to adjust attentional priorities flexibly to meet varying demands?
- 2. Why is this child struggling to pay attention and control motor activity?
- 3. Why does this particular child block certain types of stimulation and seek others?

Frequently, the answer is found in one or more of the interactive neurological subsystems, and APD can thus be treated at its root, yielding permanent changes in the nervous system (Kokot 2001:124). This perspective allows the professional not to focus on the symptom (inattention and inappropriate activity level), but rather on the aspects that the organism feels are more important, such as trying to make sense of incoming information and the processing of this information.

The HANDLE approach describes common patterns in individuals presenting with APD in figure 45.



Figure 45 Information Processing and APD

Conclusion

In chapter 3, the mechanisms (chemical, anatomical and physiological) underlying attention and movement were explored in depth. This exploration would have been worthless unless it led to knowledge that may have functional value in terms of application. At the beginning of chapter 4, a new approach was suggested that incorporates all these mechanisms and their interaction within an internal and external ecosystemic framework. Application of these mechanisms would be impossible without an in-depth understanding of each of these mechanisms and the underlying core of each mechanism. Information gained from research studies allows insight into possible ways to address difficulties in neurodevelopment. Such studies include Ito (1984, 1987), who demonstrated that changes in the cerebellum are



possible via the visual-occular reflex arc; Levi-Montalcini and Calissano (1979), who demonstrated the nerve growth factor; and Golgi (1906, cited in Bentivoglio 1998), who first described neural plasticity. Other research explored the developmental sequence of the brain and the brain structures, hierarchical mechanisms, and the developmental sequence of each neurological subsystem. The core of neuroanatomy was explored in the previous section, from the development of the neural tube, its specific developmental sequences, plasticity, information processing (neurophysiology) to the actual subsystems (tactile, olfaction, gestation, vestibular, proprioception, muscle tone, kinaesthesia, visual and auditory) as well as possible points of developmental delay. This knowledge has led to the design of various movement-based intervention programmes such as sensory integration (SI) (Ayres 1974, 1979, 1982, 1989), the HANDLE programme (Bluestone 1994, 2000, 2004), the Move to Learn programme (Phelong 1997, 2003) and Braingym (Hannaford 1995), to name but a few. Other programmes such as The Listening Programme (Madaule 1994), the Tomatis Method (Tomatis 1991) and Auditory Integration Training (Bérard 1993) have used this information to address a single area of the development, namely the auditory systems and its connections. These programmes have all been considered in the scope of this thesis as possible methods to address attentional difficulties and heightened motor activity.

7. Internal and External Biochemical Ecosystems

7.1 Introduction

Neuroanatomical, neurochemical and neurophysiological aspects of AD/HD were presented in chapter 3 as possible perspectives through which to view the symptoms of AD/HD. The following section focuses specifically on neurochemical aspects that could affect attention and motor activity. McCandless (2003:3-4) supports a biochemical approach over the traditional diagnostic and treatment models used for autistic spectrum disorders (ASD), bearing in mind that AD/HD falls within this spectrum. Since most children are treated with a single treatment (drugs), she states that children on the autistic spectrum have a multifaceted disorder that may affect any and sometimes all the major systems of the body. Inter-individual variation is the

most consistent characteristic of these children. In other words, each child is unique and should be viewed and treated as such. The notion of "one treatment fits all" does not pertain to these children. An ecosystemic approach to treating these children may well be the answer.

Other professionals, such as Holford (2006), Walker (1999), Bluestone (1994), Rapp (1996), Alecson (1999) and Shaw (1998) echo this statement. Walker (1999:155) writes in his book The hyperactive hoax, that the more you study "hyperactivity" or "attention deficit disorder," the less certain you are as to what it is, or whether it is a thousand different medical and non-medical situations all called by the same name. as if it were one thing. He poses the question: "Are these symptoms in search of a diagnosis?" He continues by stating that if a hundred different medical disorders can cause academic problems, difficulty in paying attention, overactivity, and sleep problems, then a single type of treatment (pharmacology) is inappropriate Walker (1999:10-12). Walker further believes that drug treatment may mask a child's symptoms, allowing the underlying disorders to continue, and in many cases, to worsen. He further states that one cannot treat that which you are not aware of. For this reason a full account of the child's internal and external environment needs to be considered. Judith Bluestone (1994) incorporates such a holistic examination into the HANDLE programme. Within this approach, both internal and external environments are considered before attempting to treat a neurodevelopment delay. This includes acknowledging the possible causal roles of chemicals, toxins, allergens, nutritional deficits, and dehydration. Bluestone's focus on these elements is evident in her placement of them at the bottom of the Sensory-motor Interdependency and Interaction Chart (see chart 43).

Alecson (1999:26) states that most paediatricians have limited knowledge about alternative treatments for children whose behaviours fall along the autistic spectrum. She believes that parents should seek a practitioner who knows how to determine if child has allergies, or is sensitive to food or environmental substances; and whether there may be an overgrowth of yeast or *candida albicans* in the child's system, as well as other metabolic abnormalities. Rapp (1996) adds her voice in support of such an approach.

The need for a holistic approach becomes further apparent when considering the array of possible underlying medical conditions that can cause hyperactivity, social problems, inattention and academic difficulties. A few such possible causes are the following (Holford 2006:5; Rapp 1996:45; Walker 1999:14-15):

- High lead levels, even in the absence of clinical lead poisoning, are associated with various medical and psychological conditions.
- High mercury levels can cause agitation and cognitive problems.
- Manganese toxicity is linked to aggression.
- An iron deficiency such as anaemia can lead to poor performance, despondency, fatigue, and often aggression and irritability.
- Vitamin B deficiencies, common in teens and young adults, can lead to symptoms of subclinical beriberi, including symptoms such as hostility and violent outbursts.
- Hyperthyroidism can cause fear, hostility, and demanding, hypercritical behaviour, all of which can lead to performance and social failure.
- Temporal lobe seizures, which sometimes occur almost continuously and which are often too subtle to be detected by the eye, can cause violent outbursts, restless movements, and bizarre behaviour.
- The fluctuating blood sugar levels seen in subclinical diabetes can cause fugue states, in which individuals commit unexplained and sometimes violent acts.
- Cardiac conditions can reduce the supply of blood, oxygen, and nutrients to the brain and, over time, can cause the death of brain cells. This results in impaired thinking and aberrant behaviour.
- Undetected, prolonged exposure to allergies could lead to inappropriate behaviour.

This list explains why Fred Pescatore, medical director of the Atkins Centre in New York City, conducts a full biochemical examination on all his patients. He conducts the following tests to his pediatric patients who are suspected of having learning and developmental delays (Alecson 1999:26):

- History and physical examination
- Chemistry profile

- Hematology profile
- Cytotoxic food sensitivity
- Candida antibody screening
- Glucose tolerance test

Currently, though, most professionals working with children presenting with AD/HD focus on the symptoms, and possible medical problems are often overlooked. Walker (1999:18) states that for centuries, doctors have obsessively sought the **roots of disease**, and this process of discovery continues mainly in the research setting. Learning the **cause of the symptoms** in these cases dramatically improves the practitioner's ability to help the patient. Walker calls this medical process "**splitting**", because it splits large, vague groups of symptoms (such as those listed for AD/HD) into ever more accurate diagnostic categories. He states that "when you know what you are treating, you know to treat it correctly" (Walker 1999:18). Walker goes on to lament the decline of such an approach in general medical practice, where many practitioners subscribe to a new and opposite trend in medicine, which he calls "lumping". This entails grouping many symptoms together into a handy but diagnostically worthless category. Hyperactivity and AD/HD, when used as diagnoses rather than symptoms, are classic examples of lumping.

Walker (1999:29) playfully refers to the **sick neuron**. He states that there are many ways to upset the brain's neurons. Some disorders such as anaemia cause reduced oxygen to the brain, which could disturb the functioning of neurons. Other disorders (such as metabolic disorders) reduce the brain's supply of glucose, the body's fuel. Some diseases rob the body of nutrients, some alter the levels of hormones or neurotransmitters, and yet others poison the brain with toxins. Some cause seizures, short-circuiting the brain's electrical systems. Tumours, cysts, and head injuries alter the brain's structure. Viruses, parasites, and immune disorders can make a child feel 'sick all over' (Walker 1999:29). Lyon and Laurell (2002:22) make an extremely valid point. They state that brain cells, unlike most other cells in the body, must endure and perform throughout an entire lifetime, as they are never replaced. Brain cells are the most sophisticated and demanding cells in the entire body. For this reason a responsible practitioner cannot practise without knowledge of these possible

underlying influences. A diagnosis of AD/HD will not identify any of the above as possible causes of aberrant behaviour.

McCandless (2003:38-39) echoes Walker's concern that the brain does not function in isolation. It is a team player; it needs correct, consistent informational input as well as vital nutrients. To fulfil these needs the brain depends heavily on complex interactions between the immune, endocrine, and gastrointestinal systems. McCandless supports a "broad-spectrum approach" that includes a biological diagnosis, followed by treatment to strengthen the immune system, heal the gut, and restore a healthful nutritional status. For this reason she proposes a **causation model**. McCandless also discourages treatment with Ritalin, together with all other attempts to control behaviour and other symptoms, and prefers rather to search for the root of the problem. She believes that the reason underlying the developmental disorders of almost all children (regardless of aetiology) is that proper nourishment does not reach their brain cells. Her main treatment protocol consists of the following steps:

- 1. Heal the inflamed digestive system.
- 2. Strengthen the immune system by providing needed supplemental vitamins, minerals, and other nutrients.
- 3. Remove toxins from the diet and heavy metals from the body (McCandless, 2003:34).

This chart illustrates aspects that may have an effect on the immune system, and the possible rollover effects of an impaired immune system. This chart is by no means complete as a vast number of scenarios are possible. The items in the chart are discussed individually in the following section.



Figure 46 The effects of an impaired immune system

7.2 Environmental Pollutants

Rapp (1996) believes that environmental pollutants include everything from pesticides, insecticides and cleaning agents, to heavy metals used in vaccinations, products such as sprays, and fuel vapours. Heavy metals alter cellular functioning and numerous metabolic processes in the body, including those related to the central and peripheral nervous systems (Klassen 1996; McCandless 2003:55). Much of the damage produced by heavy metals comes from the proliferation of oxidative free radicals. A free radical is an energetically unbalanced molecule, composed of an unpaired electron that "steals" an electron from another molecule (McCandless

2003:55). The immune system of genetically susceptible infants may be attacked by heavy metals such as lead and mercury. Walker (1999:85) calls these brain-altering pollutants, and he focuses particularly on solvents, pesticides, and heavy metals including lead, mercury, cadmium and manganese. Children are more vulnerable than adults to these toxins as standard levels have not been calculated for them. Children are also more at risk as they eat, drink and breathe far more for their body weight than adults do, so they inhale and ingest bigger proportional dosages. The younger the child, the more at risk he or she is. Babies and toddlers spend most of their time on the ground, which is a potentially toxic environment. A Scientific American study in 1998 pointed out that the average American carpet was one of the most dangerous spots in the country. Amongst the substances found in the house dust collected from carpets were cadmium, lead, PCBs, pesticides, and the highly toxic chemical benzo(a)pyrine. Walker (1999:85) states that children exposed to toxins present with overt physical symptoms, resulting from the effects to their brain and nervous system. He continues to say that these "silent poisonings" often go unnoticed, and that these children are often labeled as slow learners, with a low intelligence, and as having AD/HD. The effect of neurotoxins is demonstrated in figure 47 where a teacher and a student from the same classroom were evaluated against a control. The school was thought of as being an environmentally unsafe.

For the purpose of this study, only **lead** and **mercury** are discussed, although there are a variety of toxic heavy metals that may have an adverse effect on children.



Figure 47

Effect of neurotoxins

The brain structures show a definite variation in colour compared to that of the control. The large white outer areas in the images compared to the control indicate that the blood supply and brain function are not normal. The teacher's image also lacks the smoother outer-edge contour that is typical of the normal control (Rapp 1996:293)
7.2.1 Lead

Lead is known as a **neurotoxin** – a killer of brain cells. Excessive lead levels in children's bloodstreams have been linked to learning disabilities, to **attention deficit disorder**, **hyperactivity** and to reduced intellectual ability. The greatest risk for harm (even with minimal or short-term exposure), occurs among infants, young children and pregnant woman and their foetuses (McCandless 2003:55). After a decade of intense study, the harm caused by lead "can now be characterised with fair certainty" (Anderson & Breecher 1996:119). The Greater Boston Physician's Report (Greater Boston Physicians for Social Responsibility 2001) states that approximately one million American children have levels of lead in their bloodstreams above the threshold set by the Environmental Protection Agency (EPA) as adversely affecting behaviour and cognition (McCandless 2003:22). Walker (1999:85) states that measurements of heavy metals should not be taken on a once-off basis, as such a measurement does not indicate whether this level is rising or falling.

McCandless (2003:23) also mentions that the United States Centre for Disease Control (CDC) and the U.S. Public Health Service jointly agree that "lead poisoning remains the most common and socially devastating environmental disease of young children." These organisations further state that "long-term exposure to low levels of lead may cause a build-up in the brain and other tissues resulting in neurological damage to children even before they are born (cited in McCandless 2003:23).

Even mildly elevated levels of lead may result in a reduction on IQ scores, attention deficits and poor school performance. In one study by the EPA, Schwartz (cited in Walker 1999:88) reported that one out of nine children under the age of six had a high enough blood lead level to place them at risk. High lead levels have been found in everything from pottery to imported candy wrappers and beverages served in leaded crystal classes (Walker 1999:89). Walker notes that if lead is allowed to accumulate in a young developing brain, there is no way to repair the damage. Caught early, on the other hand, lead toxicity can be treated.

7.2.2 Mercury

Mercury poisoning induces cognitive and social deficits, including loss of speech or failure to develop it, memory impairment, sleep difficulties, poor concentration, self-injurious behaviour, word comprehension difficulties, and an assortment of autistic spectrum behaviour (McCandless 2003:57). An additional possible adverse connection with mercury is that it and other heavy metals destroy leukocytes and neutrophils, the specialised white blood cells that normally protect the body against fungi and bacteria (McCandless 2003:32). This may adversely affect the immune system.

Mercury, the substance found in old-fashioned thermometers, is ubiquitous in the environment. Fish are known to contain high levels of mercury. Another source of prenatal exposure might be the mercury contained in the amalgam dental fillings of their pregnant mothers (McCandless 2003:11). Evidence continues to mount that a relatively small but significant number of people are adversely affected by mercury poisoning.

Walker (1999:91) writes that children who grind their teeth release minute amounts of mercury into their systems, causing mercury sensitivity. Such sensitivity is associated with headaches, restless behaviour, and irritability. He notes that already in the 1970s and 1980s, researchers reported on the dangers of amalgam fillings, showing that mercury migrates from the teeth to nearly all the tissue, especially the brain, kidney and liver. They also presented research showing that mercury crosses over the placenta into the foetus. Mercury in vaccinations is another source of concern. This is discussed later in this section.

Toxins are dangerous and are a common cause of hyperactivity. They can lead to long-term damage, although if they are detected early, they can be eliminated. Even slightly elevated levels of heavy metals and other toxins place a burden on the brain of a young child (Walker 1999:91).

3.2.3 Phenols

Solvents can severely disrupt the function of brain cells and may even destroy neurons altogether. Many children are also chronically exposed to pesticides, which can cause nervousness, poor concentration, irritability, memory problems, and depression. Pesticides also lower their seizure thresholds. Walker (1999:91) states that low-grade carbon monoxide poisoning symptoms tend to resemble the flu, but long-term, low-grade exposure causes memory loss, hyperactivity, attention deficits, and personality changes.

Another substance that is implicated in hyperactivity and learning disabilities is cadmium, a dangerous heavy metal found in some batteries and older art supplies (Walker 1999:91).

7.2.4 Vaccinations

In infants and toddlers with increased susceptibility, vaccination with live viruses could contribute to and aggravate specific conditions. The current cohort of children diagnosed with ASD has had more exposure to mercury levels from vaccines than the EPA thinks is safe for adults. The reason for this is that children today have more vaccinations and have them closer together and earlier in life than ever before in history (McCandless 2003:25).

According to the CDC recommended immunisation schedule, infants who are fully vaccinated are exposed to 12,5 micrograms (mcg) of mercury at birth; 62,4 mcg at two months; 50 mcg at four months; 62,5 mcg at six months; and 50 mcg at approximately 18 months. According to McCandless (2003), the Hip B vaccination given at birth when the immune system and liver are immature acts as a trigger that sets into motion the cascade of events that results in neurological and gastrointestinal deficits (McCandless 2003:25).

7.3 Deficiencies of Essential Nutrients

To illustrate the importance of essential nutrients to the brain, one only needs to look at the consumption of these elements by the brain of a developing foetus. Holford



(2006:12) notes that during development in the womb, half of all the nutrition the foetus receives from its mother is directly channeled into feeding the growing brain. He adds that although it is a mere 450g in weight at birth, the child's brain consumes a vast quantity of nutrients, including protein, carbohydrate, vitamins, minerals and essential fats. Fats are probably the most important element here, as the brain is literally made out of it. In fact, if the brain were drained of all the water, 20 per cent of it would be fat (Holford 2006:12).

As it grows, a foetus builds thousands of brain cells, called neurons, every minute. By the age of two, a child's brain has approximately 100 billion neurons (Holford 2006:14). While the number of neurons does not increase in children beyond the age of two, the number of connections made between neurons increases dramatically. When a baby is born, every neuron in the cerebral cortex can connect with about 2 500 other neurons. By the time that child is three years old, that number has could be as high as 15 000 (Holford 2006:16). All these neurons are insulated with myelin (specifically, EFA).

Thought is represented by a 'ripple' of chemical activity across the network of neurons. With repeated thoughts and actions, be these speech or movement, the neuronal pathways are reinforced. Meanwhile, other, redundant connections are dismantled. Unlike other organs in the body, the brain is always restructuring itself (Holford 2006:16). Restructuring and remodeling consumes energy, and energy is derived from nutrition. Messages in the brain are sent from a sending station and received in a receiving station, called a receptor. These sending and receiving stations are built out of essential fats (derived from fish and seeds); phospholipids (found in eggs and organ meats); and **amino acids** (the raw material of protein). The message itself, a chemical known as a neurotransmitter, is in most cases made out of amino acids. Different amino acids make up the different neurotransmitters. Holford (2006:4) indicates, for example, that the neurotransmitter serotonin is made from the amino acid tryptophan. Adrenalin and dopamine are made from phenylalanine (see figure 6). Turning an amino acid into a neurotransmitter is by no means a simple process. Enzymes in the brain that depend on vitamins, minerals and special amino acids accomplish this task. These vitamins and minerals also control the steady supply of fuel (**blood sugar** or glucose) that powers each neuron.

This simplified explanation clearly illustrates the importance of a variety of nutrients in the general function of the brain and ultimately the body, as good brain nutrition leads to proper bodily regulation. Nutrition builds the very structure of the brain, from the neurons themselves to the messages that fire between them (Holford 2006:4). Holford further states that one of the most limiting concepts in the human sciences is that idea that mind and body are separated (2006:11), which promotes an ignorance of the effect of food on the mental aspects of living.

McCandless (2003) confirms this in her statement that the symptoms shared by most children on the autistic spectrum (including AD/HD) suggest that they have what she calls **starving brains**. Studies comparing autistic spectrum (AS) children with controls reveal that the AS children report higher incidences of:

- higher serum copper
- zinc deficiency
- magnesium deficiency
- iron deficiency
- higher copper/zinc ratios
- vitamin B12 deficiency
- lower vitamin B6
- below normal glutamine
- lower plasma sulphate
- lower amino acids specifically: tyrosine, carnosine, lysine, hydroxylysine
- lower methionine levels
- higher glutamine
- fatty acid deficiency
- calcium deficiency
- inadequate levels of vitamin D, E, and A (McCandless 2003:38)

7.3.1 Vitamins and minerals

Every one of the 50 known essential vitamins and minerals plays a major role in promoting mental health. In the table below, Holford (2006:69) lists the most vital

vitamins and minerals that could have an effect on the state of a child's brain, along with the symptoms that might manifest if the child is deficient, as well as the best natural food source. It should be considered that vitamins and minerals seldom work in isolation. The figure below illustrates the combined working of various nutrients that play a role in memory.



Figure 48 Combination of substances involved in memory (Holford 2003:95)

Table 6Key vitamins and minerals for brain health

Nutrient		Functioning and Symptoms of	Food Source
		Deficiency	
Thismiss (D1)			
Thiamine (BT)		Poor concentration and attention.	vvnolegrains, vegetables
		Helps turn glucose into energy so will affect	
		mental and physical tiredness.	
Niacin (B3)		Crucial in blood sugar balance and in	Wholegrains, vegetables
1		maintenance of serotonin and melatone.	
1		Depression, psychosis.	
Pantothenic	acid	Powerful memory booster and aids in	Wholegrains, vegetables

(B5)	boosting the neurotransmitter acetylcholine.	
	Poor memory, stress.	
B6	Irritability, poor memory, depression, stress.	Wholegrains, bananas
Folic acid	Anxiety, depression, psychosis.	Green leafy vegetables
B12	Confusion, poor memory, psychosis.	Meat, fish, dairy products, eggs
Vitamin C	Depression, psychosis.	Vegetables and fresh fruit
Magnesium	Irritability, insomnia, depression,	Green vegetables, nuts, seeds
	hyperactivity.	
Manganese	Dizziness, convulsions	Nuts, seeds, tropical fruit
Zinc	Confusion, blank mind, depression, loss of	Oysters, nuts seeds, fish
	appetite, lack of motivation and	
	concentration.	

(Holford 2006:70)

One of the main roles of **vitamins and minerals** is to help turn glucose into energy, amino acids into neurotransmitters, simple essential fats into more complex fats like GLA or DHA, and choline and serine into phospholipids. They are key to the task of building and rebuilding the brain and nervous system, and keeping everything running smoothly (Holford 2006:67).

Support for supplementation comes from a well-respected researcher in the autistic spectrum field. Dr Bernard Rimland from California compared 1,591 hyperactive children treated with drugs to 191 hyperactive children given nutritional supplements (specifically vitamin B). The nutritional approach was 18 times more effective in reducing hyperactivity. Yet, despite this, drug prescriptions for children nearly double every year (Holford 2006:4).

If a child suffers from a nutritional deficiency even though they are eating a healthy, balanced diet, the question should be asked as to **why** the child needs the supplements in the first place. McCandless (2003:34) states that in some cases good nutrition does not reach the child's brain. One reason for this is that intestinal parasites steal the nutrients. Children presenting with a nutritional deficiency can also suffer from iron deficiency, lead toxicity, or disorders in the intestines causing inefficient absorption (Walker 1999:66). Many children also present with nutritional deficiencies due to hypersensitivities to touch, smell and taste, and so prefer limited

diets without texture, strong tastes and odours. Many children also choose soft, smooth foods as opposed to foods that need chewing (this is due to low muscle tone in the oral area). A limited diet results in a limited variety of nutrients.

7.3.2 Essential fatty acids

Four specific kinds of fat (known as AA, DHA, EPA and DGLA) combined make up approximately 20 per cent of the brain. Deficiencies of these at any time, but especially during foetal or early development, can have huge repercussions on intelligence and behavior. So vital are these fats to the growing foetus that it will literally rob its mother's brain to fill its own quota. At birth, the level of essential fats in the umbilical cord of a newborn infant may correlate with the speed of their thinking at age eight (Holford 2006:13-14).

According to Holford (2006:41), the main functions of essential fats (**omega-3** and **omega-6**) are to

- 1. promote general physical health by reducing the risk of allergies, asthma, eczema and infections.
- promote mental health. A deficiency may result in depression, dyslexia, attention deficit disorder, autism, fatigue, memory and behaviour problems. Essential fats really are essential for maximising a child's intelligence.

Like vitamins, EFA cannot be produced or manufactured by the body. For this reason, and due to a diet generally low in these fats, it becomes vital to supplement these fats. The importance of EFA is illustrated when one looks at the role it plays in delivering messages in the brain. Delivering accurate messages in the brain is essential as it is responsible for every cell, tissue and organ in the human body. EFA are integral constituents of the anatomy and physiology of all cells in the body. Lack of fatty acids would therefore have an effect on the structure and function of all cells of the body (van der Merwe 2002:57). Omega-6 EFAs are distributed evenly in most tissues, but omega-3 EFAs are highly concentrated in only a few tissues, mainly the brain. More specifically, a substance termed 'docosahexaenoic acid' (DHA), converted from omega-3 EFAs, is the most abundant omega-3 EFA in the brain. The

brain has the greatest percentage of fat of any other organ in the body. We are, in fact, all "fat heads" (Stordy & Nicholl 2000:83).

Neurotransmitters deliver their messages across connection points called synapses into receptor sites. These receptor sites are contained within the myelin sheath, which surrounds every neuron in the brain. The myelin sheath could be compared to a layer of insulation around an electrical wire. Without this protective insulation the transmission of messages (and thus the working of the brain) would be impossible. The sheath is roughly 75 per cent fat, and it is here that the fatty acids play a vital role (Holford 2006:43).

The myelin sheath fat is made out of phospholipids, each with a saturated and unsaturated fatty acid attached. Both omega-3 and omega-6 are needed in a balance for the optimal working and restructuring of the brain (Holford 2006:44). There is some controversy surrounding this delicate balance between omega-3 and omega-6. Holford concludes that it may not just be the gross deficiency in omega-3 fats that has led to so many of the health problems found in children today, but also the gross imbalance between the two omegas. In addition, a high intake of saturated fats and damaged polyunsaturated fats (trans fats) inhibits the body from utilising the little essential fat that the average person does eat in a day (Holford 2006:47). A diagram illustrating the crucial periods of myelination is presented below.



Figure 49 Periods of myelination

(Goddard 1996:2)

The precursor to omega-3, (alpha-linolenic acid) as well as the metabolically active EPA (eicosapentaenoic acid) and DHA (docosahexaenoic acid) are more unsaturated and far more prone to damage by cooking, heating and food processing. Alpha-linolenic acid is abundant in cold climate seeds, such as flaxseeds, and in plankton. Our body can convert some of this alpha-linolenic acid into EPA and DHA, but a more effective way to increase supplies of these more 'active' omega-3 fats is to eat fish from areas low in mercury (Holford 2006:47).



Figure 50 Omega-3 fat family

The body is fairly inefficient in converting alpha-linolenic acid in seeds (e.g., linseeds and pumpkin seeds) to EPA and DHA. For this reason people who do not eat fish rarely have sufficient levels of EPA and DHA unless they eat significant quantities of flaxseeds, which are the richest source of alpha-linolenic acid. Consequently, during critical periods of development such as childhood, it is preferable to get a direct source of EPA and DHA from fish, backed up by an indirect supply from flaxseeds or flaxseed oil. This would certainly be recommended for a pregnant or breastfeeding woman to allow her to pass on sufficient EPA and DHA to her child. The World Health Organisation now recommends that infant formula feeds (milk supplements) include these oils. DHA is especially important during the foetal stage and in infancy because it is literally used to build the brain, and makes up a full quarter of the brain's dry weight. In many cases, children with learning and behavioural problems are even less efficient than the average at converting alpha-linolenic acid into EPA and DHA. Partly for this reason, a child with AD/HD or dyslexia may need double or triple their intake to correct their condition (Holford 2006:49; Stordy & Nicholl 2000:105).



The precursor of the omega-6 fat family is linoleic acid, which is found in seeds, especially hotclimate seeds such as sunflower and sesame seeds. Linoleic acid is converted by the body into gamma-linolenic acid (GLA), (the richest sources of which are found in evening primrose oil and borage or starflower oil). A derivative of GLA, known as DGLA, is found in high quantities in the brain (Holford 2006:49).

One omega-6 fat has something of a Jekyll and Hyde nature, however, and this is arachidonic acid (AA). AA is essential for brain function, but in excess it is associated with promoting inflammation. It can be derived either directly from meat or animal produce, or indirectly from linoleic acid or GLA. The latter may be a preferable source because GLA also produces anti-inflammatory substances that balance the inflammatory effects of arachidonic acid. For this and other reasons, Holford (2006:50) suggests a diet rich in omega-6 seeds and their oils to get enough of this essential fat family, rather that overdosing on meat and dairy produce.

Van der Merwe (2002:51) states that few practitioners and even paediatricians are aware that children with AD/HD present with EFA deficiencies that are due to an (inborn) error of metabolism. These children cannot absorb or metabolise EFA normally due to a problem with the enzyme delta-6-desatuarse, which is responsible for the first conversion of the natural fatty acid to gamma-linolenic acid. He continues that the social dysfunctional symptoms of AD/HD children, the obvious physical signs and symptoms of EFA deficiency, are often overlooked. Colquhoun and Bunday (1981) of the British Hyperactivity Support Group were the first to point to the physical symptoms of these children and suggest that a fatty acid deficiency was the cause. Two thirds of these children also have a zinc deficiency. This mineral is a co-factor in fatty acid metabolism (van der Merwe 2002, p 51).

In Colquhoun's and Bunday's (1981) initial study, the children investigated were usually boys who were overactive in utero, came from atopic families, were poor feeders, suffered from diarrhoea and catarrh, had recurrent otitis media requiring grommets in many instances, were prone to colic, and often had a wheezing chest or asthma, eczema, migraine, or hay fever. They had a striking hyperaccusis, avoided skin contact (cuddling), they had an unquenchable thirst (yet they did not always have polyuria) due to increased, imperceptible water loss through the skin. Many reported having head sweats; they were insensitive to pain, prone to auto-mutilation during frequent temper tantrums, prone to head banging, and cot rocking; they did not sleep, and tended to be clumsy and uncoordinated in gait. Many of these phenomena were seen in animals with EFA deficiencies (van der Merwe 2002:52).

Clinical studies confirmed this initial suspicion. Mitchell et al. (cited in van der Merwe 2002:52) found significantly lower levels of AA, ADGLA, and DHA in the blood of 44 hyperactive children compared to age-and gender-matched controls. The hyperactive group had significantly more auditory, visual, language, reading and learning difficulties and the birth weight of this group was significantly lower than that of controls. In addition, significantly more hyperactive children had more frequent coughs and colds, polydypsia, polyuria, and more often reported having a serious illness or accident in the past year than controls (van der Merwe 2002:52).

In another publication by the same researchers (Amen, Mitchell et al. cited in van der Merwe 2002:53), they reported on 31 children with marked ADD in a double-blind, placebo-controlled crossover study of essential fatty acids supplementation for only four weeks in each treatment modality. Supplementation caused higher concentrations in the blood of DHGLA than before. Supplementation was also associated with significant changes on two performance tasks and with significant improvement in parent rating on the Behaviour Problem Checklist.

More recently it was shown that omega-3 fatty acid deficiency was also implicated in hyperactivity. Stevens et al. (cited in van der Merwe 2002) found a greater frequency of physical symptoms indicative of EFA deficiency in boys with lower plasma phospholipid omega-3 and omega-6 levels than those with higher levels. These symptoms included thirst, frequent urination, dry skin and dry hair. The children with lower omega-3 levels were reported to suffer from more colds, to have used antibiotics more often since birth, showed more frequent and excessive temper tantrums and more sleeping difficulties. Teachers evaluated this group as scoring significantly lower in overall academic ability and particular in mathematics (van der Merwe 2002:53). These findings were confirmed with studies by Richardson and Montgomery (2005).



Figure 52 Production of enzymes for EFA conversion

(Schmidt 2001:66)



EFA does not function in isolation, nor can it be manufactured in isolation. The necessity of good nutrition is illustrated in the figure below. The ability to convert fats is dependent on minerals, vitamins and the absence of the so-called bad fats (found, among others, in deep-fried foods).

The body needs cofactor nutrients to manufacture the enzymes that assist in the conversion of own brain-fat DHA. Dietary fatty acids are converted by enzymes into their long-chain products that are ultimately used in forming the brain's structure. The enzymes can be assisted or blocked by many factors (Schmidt 2001:66).

Comment

Within an ecosystemic framework, it is important to note that EFA (neurochemical aspect) plays a vital role in the neuroanatomy. The EFA actually provides the strength for the neuron to maintain its position within the delicate neural networks in the brain. As stated by Rose (2005:146): The effectiveness of a synapse – independently of how much neurotransmitter it releases, depends on its address and geometry. Because neuronal geometry determines connectivity, the morphology of the dendrites themselves, with their many branches, adds to the complexity of the computations they make of the synaptic voices reaching them, even before their summation at the axon hillock. Dendrites need stability to maintain their position, and EFA provides that stability.

7.4 Hypoglycaemia

The brain's main source of energy is glucose. Unstable glucose levels will thus have an adverse effect on the ability of the brain to perform at optimal level. Too much glucose will result in the typical "wall-bouncing effect". Too little could cause a child to experience symptoms like fatigue, irritability, dizziness, insomnia, aggression, anxiety, sweating (especially at night), poor concentration, excessive thirst, depression, crying spells or blurred vision (Holford 2006:20). This sets in motion a vicious cycle that leads to more cravings, more extreme mood fluctuations and progressively poorer concentration and behaviour (Holford 2006:23). Holford (2006) further states that glucose is powerful, and can actually damage nerves and blood vessels. The body copes with this by enlisting the help of the hormone insulin, which is released from the pancreas when a burst of glucose hits the child's bloodstream (Holford 2006:22). Blood glucose is directly associated with poor memory, **poor attention** and aggressive behavior. Sugar has been implicated in aggressive behavior, hyperactivity and attention difficulties, eating disorders, anxiety, and depression, fatigue and learning difficulties. More dietary studies consistently reveal that hyperactive children eat more sugar than other children, and that reducing dietary sugar has been found to halve disciplinary action in young offenders (Holford 2006:24). The effect of sugar is well illustrated by Rapp in figures 53 and 54 below.



Figure 53 Effect of hypoglycemia

Gradual deterioration of handwriting caused by hypoglycemia (Rapp 1996:90)



Drawing before and after a high glucose breakfast (Rapp 1996:80).

A glucose tolerance test measures the body's ability to process carbohydrates, and is an indication of di abetes or hypoglycaemia. It is extremely important to recognise hypoglycaemia because unstable blood sugar, especially low blood sugar, causes symptoms that are easily confused with ADD and AD/HD (Alecson 1999:28).

Some doctors who use alternative treatments deal with hyperactivity by placing the child on a refined sugar and carbohydrate-controlled diet, or what is known as a low GI diet. Many children feel better on such a diet but again, hyperglycaemia is not a disease (Walker 1999:65); like hyperactivity, it is only a symptom. Hyperglycaemia can stem from thyroid disorders, liver or pancreatic problems, or adrenal gland disorders. More commonly, it can stem from high insulin levels occurring during the early stages of diabetes (Walker 1999:65). For this reason it remains important to investigate until the root cause of the condition has been identified.

7.5 Genetic Deficiency of Immune System

Walker (1999:98) states that the body is a remarkable system that comes with a set of instructions encoded in hundreds of thousands of genes. However, if even one of these genes is defective, the remarkable system breaks down. Sometimes the resulting genetic disorder is so mild that it is almost invisible. Other times, the disease is devastating (Walker 1999:98). Often these diseases make themselves initially known through subtle cognitive-behavioural changes, including hyperactivity, attentional disorders, or learning disabilities.

However, Gupta (cited in McCandless 2003:47) believes that this remarkable system does not work in isolation, but is affected by the environment. This author states that "genes load the gun, environment pulls the trigger".

7.6 General Impaired Immune System

Van der Merwe (2002:52) observes that a large number of children presenting with AD/HD also present with recurrent ear infections, asthma, colds, eczema and the like. McCandless (2003:6-7) noted the same pattern of recurrent ear infections in their first year of life, followed by a period of seemingly strong immunity. This may be due to a hyper-immune status in response to low-grade chronic infection, or the so-called adaptation stage on the Hans Selye scale, where the body is symptom-free but not disease-free (Heiniger & Randolph 1981:6.) Such a scenario may include the apparent healthy child who has nevertheless had chronic diarrhoea for years and has self-selected a daily diet limited to a few usually non-nutritious foods such as cola, french fries, and potato chips. In neuro-typical children these conditions may not be harmful; however, in susceptible children, intestinal treatment or changes in diet may not only be warranted but essential for them to improve. McCandless (2003:7) notes that chronic diarrhoea is very common in these children and a sure tip-off that their biochemistry is abnormal.

As figure 46 illustrates, the immune system may be effected by a number of factors such as emotional stress, dehydration, parasites, bacteria, and toxins, to name but a few. Typically, more than one factor is usually present, loading the overall system (Crook 2000:79).

7.7 Recurrent Infections

A low or impaired immune system could lead to recurrent infections of various natures. In chapter 2 the history of AD/HD was traced back to between 1917 and 1926, when a bizarre and terrifying epidemic of a brain inflammation called *encephalitis lethargica* swept the world (Sterman 2000:1). Those child victims that survived were left with symptoms of meanness, anger, and hostility. Physicians Sarah Cheyette and Jefferey Cummings (Walker 1999:82), who studied the epidemic, noted the following: "they became disobedient and quarrelsome, often leading to expulsion from school. Emotionally lability, irritability, and temper tantrums were common. These children were **hyperactive** and **impulsive**, and they appeared to lack empathy; they were often called 'moral imbeciles'". The *encephalitis lethargica* epidemic has passed, but the epidemic taught professionals a valuable lesson. Psychiatric symptoms, including **hyperactivity** and **impulsivity**, can stem from an **infection**.

Acute bacterial meningitis affects thousands of children each year, many of them under the age of three. Bluestone (1994) believes that meningitis can enter the middle ear and have an adverse effect on the vestibular system, cochlea and occicles, resulting in poor balance and auditory ability. Walker (1999:82) mentions a study of eighty paediatric bacterial meningitis cases that left five per cent of the victims with long-term hyperactivity afterwards and more than twelve per cent had learning difficulties. Encephalitis, a brain inflammation caused by a virus or bacteria, can cause long-term behavioural problems, as can prenatal infections such as cytomegalovirus (Walker 1999:82).

Walker continues by stating that one of the most commonly overlooked infections linked to hyperactivity is group called beta-hemolytic streptococcus, better known as "strep". Although these bacteria are most commonly thought as the cause of throat infections, left untreated, strep can lead to rheumatic fever as well as a movement disorder called Sydenham's chorea. Moreover, recurrent infections can lead to a group of symptoms collectively known as PANDAS (Paediatric Autoimmune Neuropsychiatry Disorders). Symptoms of PANDAS include obsessive-compulsive

behaviour, hyperactivity, Tourette's syndrome, and cognitive difficulties (Walker 1999:83).

Swedo, a physician at the National Institute of Mental Health who first identified the strep-PANDAS link in 1998, believed that PANDAS occurs when the immune system, reacting abnormally to a strep infection, begins attacking the basal ganglia cells in the brain. Symptoms usually subside after the initial infection, but can resurface after another strep infection, or even during an unrelated infection. Swedo and his team reported a dramatic change in abnormal movement after treatment for the infection (Walker 1999:83). Walker asks the question: "How many children are suffering from strep-linked psychiatric disorders?" Is it the same number of children diagnosed with AD/HD?

The recurrent bacterial infection ear infections that thousands of children suffer in infancy and early childhood also are strongly linked to hyperactivity, attention problems and learning difficulties. Streptococcus pneumoniae causes a large percentage of acute otitis media. Studies have indicated that increased otitis media early in life results in much higher incidents of hyperactivity. Robert and his colleagues (1994, cited in Shaw 1998:3) reported that recurrent otitis media during infancy was correlated with increased distractibility of students later in life. Other studies correlated recurrent otitis media in infancy with later low IQ scores, poorer performance on tests of reading, spelling, and math, increased retention in grade, increased attention deficits, and increased behavioural problems in school (Shaw 1998:3). Both autism and AD/HD researchers have assumed that the hearing impairment caused by the recurrent ear infections is what causes the abnormal behaviour. However, Shaw interprets this correlation differently, and hypothesises that the abnormal byproducts of yeast and drug-resistant bacteria that are absorbed into the body from the intestine following the excessive use of antibiotics is the cause of these epidemics (Shaw 1998:3).

The etiological significance of viral infections in susceptible children is well established by clinical data. It seems as though these children has a high viral titer count for herpes simplex virus, varicella, Epstein-Barr virus, and human herpes virus 6 (McCandless 2003:29). According to McCandless, various viruses are associated

with verbal impairment, demyelination, seizures, and other autism-spectrum traits. All this must be seen in the context of a child's overall medical portrait. Gut pathology must be minimised, nutrition status maximised, and heavy metals removed by physician-supervised chelating. A nett effect of these treatments is that the child's immune status may improve to the point that his or her subclinical infection can be more successfully immuno-suppressed (McCandless 2003:30).

Antibiotics were first produced on a commercial scale around the end of World War II. In 1949 approximately 80 tons were produced per year. This number has increased to 20 000 tons per year in 1990 (Shaw 1998:3). Many children who develop symptoms and behaviours that place them within the attentional difficulty spectrum have a history of ear and upper respiratory infections that were treated by rounds of antibiotics. However, antibiotics do not treat the underlying reasons why a child's immune system is so weak that it cannot fight the infection in the first place. Sometimes, it is just a matter of giving the child's natural defense system time to kick in. Most paediatricians, however, prescribe antibiotics for ear infections rather than alternative treatments that activate immune response.

7.8 Dysbiosis - Candida

As early as 460-370BC, Hippocrates suggested that "all diseases begin in the gut" (Campbell-McBride 2004:9). **Candidiasis** is an infection caused by a species of candida, especially **candida albicans** (Beers, Fletcher, Jones, Porter, Berkowits & Kaplan 2003:1150).

Candida albicans, a single-celled fungus, is always present in the genital and intestinal tracts. If it is present in disproportionate quantities, however, it can cause infection. Food allergies and environmental sensitivities are usually a comorbid condition in candida (Balch & Balch 2000:263).

Most specialists in the field of AD/HD mentioned so far in this chapter acknowledge that the gut plays an important role in the cascade of symptoms associated with hyperactivity and attentional difficulties. Walker (1999) confirms that candida growth may be caused by some drugs, including some antibiotics and birth control pills, as well as liver, thyroid, or adrenal gland abnormalities. Antibiotics kill both harmful and helpful bacteria that should coexist in the gut. When intestinal bacteria are unbalanced, a condition known as **dysbiosis** occurs. This is an imbalance of intestinal bacteria that results in an overgrowth of harmful bacteria and other microbes like candida albicans. A vicious cycle results: bacteria lead to ear infections, which lead to antibiotic use, which causes dysbiosis resulting in **immune dysfunction**, which leads to bacterial invasion, ear infections, and more antibiotics use until the intestinal lining is damaged (Alecson 1999:30). (See figure 46.)

The presence of dysbiosis is very important in the the digestive-immunologycalbehavioural connection. The gut contains some neurotransmitters and other chemicals that are identical to those found in the brain. In fact, the gust is considered to be a second brain by those who understand this connection. Gastrointestinal disturbances coincide with mental acuity and emotional stability. This has certainly been shown to be the case among children along the autistic spectrum. Immune dysfunction and gastrointestinal problems are interrelated, and the treatments that have been shown to be the greatest successes are those that address these two biological systems.

The body functions by properly utilising what it needs and eliminating what it does not need. Survival is dependent on the body's recognition of what is useful for growth and development and what is not. All the biological systems, down to the cellular level, rely on good nutrition for optimal functioning. Toxins that invade the body from the air we breathe and the water we drink should be eliminated in the same way. For even relatively healthy individuals, there are many ways in which assimilation and elimination can be compromised. Typical symptoms of an intestinal disruption are stomach upset, diarrhoea or constipation, and a general lack of energy, to name but a few. Children experience the same discomforts, but because they are still growing, their brains can be affected by nutritional deficits, food allergies and sensitivities (Rapp 1996:33), and by toxicity, resulting in cognitive and behavioural problems.

Some of the health problems associated with candida overgrowth include diarrhoea, stomach aches, gas pains, constipation, headaches, fatigue, and depression. Behavioural problems include concentration difficulties, **hyperactivity**, **short**

attention span, irritability and aggression (McCandless 2003:32). Although candida is often a clue pointing to the cause of hyperactivity, it is not a diagnosis in itself (Walker 1999:62).

Children along the autistic spectrum are at greater risk for developing candidiasis. There are several reasons why yeast overgrowth is found among these children. Antibiotic use is certainly one of the factors. Antibiotics kill bad bacteria as well as good bacteria – lactobacillus and bifidus – that are necessary to keep candida from overpopulating the gut. The affected children may lack something in their immune function that is needed to keep yeast from proliferating. Furthermore, according to lvker (1995), anything that weakens the immune system can contribute to yeast overgrowth. Included in this definition are environmental toxins and chemicals, such as pesticides, and exposure to lead and other heavy metals. It may be that children along the autistic spectrum are more sensitive to candida because they are allergic to the yeast itself. Finally, parasites aid in yeast production, and children may have intestinal parasites that their body has not been able to eliminate (Alecson 1999:31).

Everyone has yeast organisms in and on their bodies. The problem arises when yeast, fungi, or candida overpopulates the intestinal tract. Elizabeth Lipski, author of *Leaky Gut Syndrome* (1998:20), says that candida organisms are like bullies that push their way into the intestinal lining, destroying cells and brush borders. This damage allows macromolecules of partially digested food to pass through the lining. The macromolecules are the perfect size for antibodies to respond to. Your immune system then goes on alert for these specific foods so the next time you eat them, your antibodies will be waiting. The net result is increased sensitivity to foods and other food substances and the environment.

7.9 Candida Immune Toxins, Gliotoxin

One of these toxic byproducts is an enzyme that allows the yeast to burrow into the intestinal wall, which can contribute to what is termed the "leaky gut" syndrome. The yeast-generating toxins literally drill holes through the intestinal wall and seep into the child's bloodstream (D'Eufemia et al. 1996:1076; McCandless 2003:45). Ultimately, the toxic substances may inflame or cross the blood/brain barrier and interfere with

the flow of nutrients to the brain, impairing consciousness, cognition, speech, or behaviour (McCandless 2003:45).

As a result of the gliotoxins, the immune system will be negatively affected, which sets the whole process in motion again. Phospholipase protease that causes leaky gut results in malabsorption of nutrients and poorly digested foods.

7.10 Leaky Gut

It is extremely important to assess the digestive function of children along the autistic spectrum. Improper digestion leads to immune suppression, making the body more susceptible to infections and to immune dysfunction, which lead to adverse or allergic reactions to foods and the environment. One type of digestive disorder that is often found among children with autistic behaviours and learning problems is increased intestinal permeability, otherwise coined "the leaky gut syndrome" (Alecson 1999:29).

The gut and the brain are closely connected. This connection is so close that the gut is actually thought of as a second brain, which is called the **enteric nervous system**. This network of neurons, neurotransmitters and proteins lining the gut is in constant communication with the central nervous system in the brain and spinal cord (Holford 2006:223). Crook (2000:12) illustrates this connection in the figure below.



Yeast interplay



The gut, or small intestine, is the longest part of the digestive track (approximately seven metres). It is responsible for absorbing essential nutrients and preventing undesirable substances from entering the bloodstream through the intestinal lining. The intestinal lining can be damaged by a number of factors including chronic bacterial, viral, and fungal infections; and by the repeated use of antibiotics and nonsteroidal anti-inflammatory drugs such as aspirin. It can also be damaged by an incompetent "disposal" system for toxic waste that is accumulated daily by the body. This damage could result in openings in the lining, which enable otherwise impassable substances to reach the bloodstream, creating in turn an immune reaction.

During such an immune reaction the body responds as if being attacked by antigens. This immune response manifests in growing children as allergies to foods, which then take a behavioural form (Rapp 1996:69). A chronic allergic reaction taxes all the biological systems, keeps the immune system in alert status, creates bodily discomfort, and ultimately affects behaviour, sleep, mood, and ability to concentrate and learn. If left untreated, leaky gut syndrome overburdens the liver's ability to clean out toxins (Alecson 1999:30).

Candida albicans is one of many fungi that are normally present in most people's intestinal tracts and other places, where it is kept in check and causes no harm. If the intestinal balance is disrupted and healthy bacteria killed, candida albicans will flourish and release toxic chemicals into the bloodstream. Continual antibiotic use is not the only cause of candidiasis, or yeast overgrowth, in children. An infant born vaginally to a mother who has a vaginal yeast infection can become similarly infected.

An overgrowth of yeast causes leaky gut syndrome and allows food particles to enter the bloodstream. William Shaw (2002), a biochemist and the director of The Great Plains Laboratory, has done extensive research into yeast overgrowth in children along the autistic spectrum. He has found abnormal metabolites, organic compounds produced by metabolism, in the urine of these children. The nature of the metabolites suggests to him that yeast byproducts, which are toxic, are also passing through the intestinal wall.

lvker (1995:191) writes that one of the major toxins produced by yeast is acetaldehyde. Its multiple effects can be devastating. It is converted by the liver into alcohol, depleting the body of magnesium and potassium, reducing cell energy, and causing symptoms of intoxication – disorientation, dizziness, or mental confusion. The 'spaciness' or 'mental fog', as often described by patients, is one of the most frequent symptoms of candidas. Patients related a detached state of mind, **poor concentration**, faulty memory, and difficulty making decisions.

Adults are capable of describing their symptoms; children are not. Children along the autistic spectrum have behaviours that strongly resemble the symptoms listed above. They may also have physical signs of yeast overgrowth that include a distinct "yeasty" smell, and cravings for foods that yeast thrives on, such as sugars and carbohydrates, as well as a whitish coating of the tongue, nappy rash, and/or itching around the anus (Alecson 1999:32).

Walker (1999:60) mentions that an infection that is often linked to hyperactivity is an overgrowth of candida albicans. The treatment recommended by most doctors is an avoidance of yeast-promoting foods or yeast-containing foods together with an antifungal drug such as Nystatin. He confirms that there are some correlations between hyperactivity and candida, but insists that candida is not the cause of hyperactivity. There must be a reason that the person in constantly infested with this fungus. The possible cause is most that of the time an immune disorder, or a disorder affecting carbohydrate metabolism (thus altering sugar levels), makes the body a fertile environment for the growth of this fungus. He continues that the same disorder that causes candida is the same that causes hyperactivity – an undiagnosed immune disorder.

McCandless also notes that children with impaired immune systems and inflamed intestines are particularly vulnerable to invasions by fungi, especially yeast of the candida species. Faecal cultures and other laboratory tests often identify overgrowths with candida albicans. Many affected children's medical histories document recurrent otitis media and repeated use of antibiotics. As a result, the beneficial or probiotic flora in such children are likely to be destroyed, thereby setting the stage for adverse fungal as well as bacterial colonisations. Similarly, organic mercury compounds can adversely affect intestinal flora. Thus vaccinal ethylmercury - which exits the body primarily via the intestinal tract – may also injure intestinal flora. When vaccinal ethylmercury poisoning is accompanied by antibiotic overuse, probiotic flora may decrease, resulting in an adverse intestinal colonisation and the flourishing of intestinal yeast (McCandless 2003:31). As these species of yeast multiply they excrete toxins. Shaw (2002), who has conducted groundbreaking research on yeast and its effect on ASD, points out that these toxins are capable of impairing the central nervous system and immune systems (McCandless 2003:31).

7.11 Allergies

Food **sensitivities** are usually considered to be different from food **allergies**, though Rapp (1996), a paediatric allergist and author on the subject, includes sensitivities in the term "allergy." Allergies have been viewed as intense, sometimes life-threatening, reactions to foods and environmental substances. Sensitivities manifest as subtler, yet constant, irritations to bodily function and behaviour. However, sensitivities can cause reactions that become quite debilitating over the course of time. Sensitivity to gluten, a protein found in wheat, is not life threatening in the short term. However, if children who are sensitive to gluten continue to eat wheat, they will overburden their digestive and immune system, which will in turn affect their behaviour (Rapp 1996:33). She has published several signs of allergies in her book: *Is this your child's world?* Some of the signs are presented below (Rapp 1996:68-74):



Figure 56: Facial tic



Figure 57: Brilliant red ears Brilliant red earlobes are typical of many allergic children and adults. They provide a red flag at the outset of an allergic reaction. Red can affect part of whole of one or both ears.



Figure 58: Allergic eye wrinkles Eye wrinkles are typical of allergic children and adults, especially those who have eczema.



Figure 59: Typical nose-rubbing

The nose is rubbed upward or massaged in a circle because it is itching.



Figure 60: Eczema or atopic dermatitis on the cheeks

Eczema causes an itching skin, rashes on the cheeks, in the arm and legs creases, and around the wrists and ankles. Roundish patches scattered over the body are also common. The skin often becomes redder and itchier at the time of some adverse contact, such as while eating a problem food or when reacting to an allergy skin test.



Figure 61: Puffiness or dark areas under eyes

Puffy eye bags can develop directly below the eyes or somewhat more outward the outer edge of the face. Although the bags could have many causes, a dairy sensitivity could also cause this. Gluten found in wheat, rye, oats, barley, and other grains, and casein, a protein found in milk and dairy products, are the two most problematic substances for proper digestion for many children along the autistic spectrum. These children are either lacking the necessary enzymes to break down gluten and casein, or the enzymes, for whatever reason, are not doing their job. Gluten and casein are similar in molecular structure, and if they are not broken down into peptides, which then break down into amino acids, they can pass through the intestinal wall if it is not intact (leaky gut) and enter the bloodstream. Gluten and casein peptides in the bloodstream cause an autoimmune reaction.

Furthermore, it is believed that gluten and casein peptides affect the brain in ways similar to opiates, disrupting the normal function of the central nervous system. Like opium, gluten and casein appear to be addictive in nature. This explains the craving children have for foods like pizza, macaroni and cheese, cereal and milk, among others. Once gluten and casein are removed, children can suffer from withdrawal symptoms that affect their behaviour, sleep, and attentiveness. This may last for a week or two at the most. It may take a year before the child experiences the full benefits of a gluten- and casein-free diet. Many children along the autistic spectrum also have allergies to environmental substances such as pollen, molds, dust, mites, and animal dander, as well as to certain foods. Other common foods and ingredients that some children are sensitive or allergic to include peanuts, chocolate, soy, eggs, corn, additives, preservatives, colour dyes, citrus fruits, apples and apple products, nitrates, and sugar in varying forms. The list can be quite overwhelming.

Physical signs of allergy include rashes, hives, eczema, red ears, dark rings under the eyes, wrinkles under the eyes, headaches, and respiratory reactions such as sneezing, stuffed nose, and itchy eyes (Alecson 1999:38; Rapp 1996:68-78). Some of these signs are included in the figures below, as are the effects of allergies on cognitive performance.



Changes in Robert's handwriting during testing for allergy to oats.

affected by their diet. Hyperactive children were significantly more allergic (66%) than those who were not hyperactive (36%) and were also significantly more affected by their diet (48% versus 20%) (van der Merwe 2002:54).

Some specialists report success through following a specific diet that remove allergens – most commonly milk, wheat, eggs, citrus, chocolates and other foods. If an allergy is identified and eliminated, children generally feel better as symptoms of fatigue, migraine, hyperactivity and even bed-wetting disappear. Walker (1999:62) acknowledges that allergies can cause these symptoms, but warns against assuming that such symptoms are the result of allergies only. This is true even for children with the classic signs of allergies - wrinkles under the eyelid, puffy eyes and dark bags under the eyes, a wrinkle across the nose, dripping nose, sleeplessness, hyperactivity, headaches, and a pale, dull, apathetic look. Often allergy-orientated doctors will diagnose "allergy-induced hyperactivity", but this could be dangerous as some of these symptoms could also be caused by other medical disorders that need attention. A pale, dull, apathetic look may be due to an iron deficiency, a heart problem or even to the use of Ritalin. Exposure to pesticides can cause rashes, puffy and scratchy eyes, scratchy throats, indigestion and other symptoms commonly associated with allergies. A constantly dripping nose could stem from an allergy or could be the result of a tear in the dura mater (membrane surrounding the brain), which then leads to cerebral spinal fluid leaking through the nose. Headaches (a common allergy symptom) could be due to a calcium deficiency, tumours, sinus, blood vessel dilation or constrictions, collagen disease, vertebral problems, carbon monoxide poisoning or temporomandibular joint disorder. Clearly it is necessary to investigate such symptoms further and not stop looking when an allergy is suspected (Walker 1999:63).

7.12 Anaemia

Ronald Hoffman (1997) includes the following additional medical examinations in his guide, *The natural approach to Attention Deficit Disorder (ADD)*: serum ferritin screening to test for anaemia or iron deficiency, a thyroid profile to rule out hypo- or hyperthyroidism; and urinary amino acids and organic acids screening. Urine organic



acid testing detects nearly seventy different biochemical compounds according to The Plains Laboratory, which specialises in such testing (Alecson 1999:28).

7.13 Pests

Pinworms make children feel miserable on the outsideby causing anal itching. Other worms make children feel miserable on the inside and may result in hyperactivity, learning difficulties, or depression. These worms attach themselves to the walls on the intestinal track, and live parasitically on the iron supply and other nutrients (Walker 1999:82).

8. Conclusion

The information presented in this chapter discusses the entire central nervous system, imbalances in neurotransmitters, sensory-motor systems, their functioning and developmental delays. Evidence has been presented to support the notion that inattention and heightened motor activity may stem from a number of biological factors. Emphasis was placed on identifying the root cause of the disorder. The mechanisms involved in attention and motor activity (movement) are not as clear-cut as they seem. For this reason the symptoms associated with dysfunction in these systems may also not be as clear-cut.

A change in diet will not treat toxic-induced brain dysfunctions. Behaviour modification will not cure hyperthyroidism. Vitamins will not eliminate a parasytes, and neurofeedback will not correct anaemia. It seems that an effective intervention programme would not be as simple as applying one single solution. Multiple options may have to be incorporated in an intervention programme that addresses the multitude of possible underlying causes of hyperactivity and attention deficit disorder.

The following chapter presents an outline of possible interventions based on the information contained in the preceding chapters.

Chapter 5

Intervention Design

1. Introduction

It seems as though the DSM-IV and other diagnostic models do not provide an indepth solution to the true understanding of the root causes of the symptoms of inattention and heightened motor activity, and consequently would not be able to yield a truly comprehensive intervention model. A new model is therefore proposed in the next section. The previous chapter outlined a possible alternative approach towards understanding the symptoms of inattention and heightened motor activity. The information presented included aspects such as neurodevelopmental delays, toxic exposure, nutritional deficiencies, low immune system, blood sugar irregularities, allergies and sensory integration dysfunction, the role of nutrition in attention, the role of movement in neurodevelopmental delays and the effect of these on physiological aspects such as brainwave functions and audition. The fact that some of these factors can lead to inattention and heightened motor activity may indicate the need for a model that takes cognisance of these factors and address these factors at their root cause. In this chapter I give an outline of a proposed intervention programme as tested in the experimental research implemented in the study and reported in chapter 6.

The aim of the research, considering the literature study and the data presented thus far, would be to answer the hypotheses as set out in chapter 1. Within this chapter the intervention design is presented in order to test these hypotheses.

2. Ecosystemic Model

At the beginning of chapter 4, an ecosystemic model was proposed that incorporated neuroanatomy, neurochemical and neurophysiological aspects as well as the interrelatedness of these aspects. The three main aspects included in this approach form an interlocking system. One cannot function without the other. Chemical



working affects the anatomy (initial development of structures), and chemical activity produces physiological output. Physiological output in turn results in the release of neurotransmitters and firing of neurons at specific locations, resulting in structural changes in the brain. All three aspects (and the various combinations of these aspects) affect attention and movement (motor activity). For the purpose of clarity the model is repeated in this section. Various therapeutic intervention programmes that would support the said ecosystemic model on all the levels were considered. A combination of these intervention models was included in a single protocol.

Figure 65 Ecosystemic approach



A number of questions were posed in chapter 4 (section 2) in relation to this new theoretical model. These questions were:

• How can the information presented assist in a functional intervention programme?

Information was submitted that indicated that **neurodevelopmental blockages** could lead to sensory-motor disorganisation which would effect brain maturation, consequently influencing the differentiation of the different brain structures that facilitate and maintain attention. Neurodevelopment has a strong neuroanatomical component as it specifically implies the development and growth of specific areas in the brain in a specific sequence. Growth in itself encompasses neurophysiology and both these two concepts (anatomy and physiology) rely heavily on neurochemical activity to facilitate the communication and functioning between the neurons.

Further information was presented that indicated that **nutritional** aspects could interfere with blood sugar imbalances, allergies, malabsorption, toxicity, candida, malnutrition with subsequent nutritional deficiencies to name but a few. Nutrition essentially represents a strong neurochemical influence as all nutrients are fundamentally chemicals and produce chemical reactions. The health and structure of the digestive and transport systems will affect the absorption and transportation of these nutrients to all the cells in the body. Sensory systems were discussed, as was the information processing system when information is modulated incorrectly. Stimulation and strengthening of these systems may be a possible intervention approach.

These are just a few possible factors that could be included in an alternative approach to addres attentional difficulties and heightened motor activity. The two factors were specifically chosen as a possible explanation as both these aspects involve the internal and external ecosystem. • How can the chemical working of the brain be addressed naturally?

Science has demonstrated that certain neurotransmitters are released with physical activity of the body. All neurotransmitters are derived from amino acids and amino acids are derived from protein. Instead of supplying the neurotransmitter that is thought be chemically imbalanced, natural supplementation of the neurotransmitter may be a more acceptable way for the body to correct the neurotransmitter imbalance.

 How can the structural aspects of the brain be altered naturally to produce effective modulation of attention and motor activity?

Neural plasticity is a validated phenomenon in neuroscience. The use of certain parts of the brain in certain ways facilitates neural plasticity. Movement is one of the ways that this is facilitated. It has been observed that movement plays an essential role in structuring the brain. This is true in terms of cellular movement when chemicals move from one cell to the other through the cell membrane to facilitate homeostasis. This is also indicated when a baby performs certain activities (such as rolling and crawling) which facilitates the development of the brain. Movement involves the cerebellum, the vestibular system, RAS as well as the thalamus. These brain structures are also involved in the production and maintenance of attention and the ability to inhibit inappropriate movement.

 How can the overall physiological output (to be able to pay attention and to control motor activity) be appropriately modulated?

In essence, modulation could be described as coherence in the brain. This is possible only when the brain receives the correct information consistently at the correct intensity. This in turn is possible only when all the brain structures perform the way they were designed to at the appropriate level of maturation.
• Could the knowledge of basic neurodevelopment and effective nutrition be the answer, and if so, how can this be applied effectively?

There is no single answer to this question. Knowledge of the basic neurodevelopmental sequences and effective nutrition could certainly assist in understanding attentional difficulties and heightened motor activity on a deeper level. Knowledge and understanding could also possibly lead to identifying possible root causes to attentional difficulties and heightened motor activity. In the course of this research, these two components were tested as part of a combined protocol.

These questions, together with the formulated hypotheses, were instrumental in directing this research. In the section that follows I describe a combined, ecosystemic protocol to answer these questions.

The following section includes the therapeutic modalities that, combined, form the ecosystemic approach used in the experimental research study. The modalities, considered as intervention programmes, are in themselves ecosystemic as most of them function on all three levels (neuroanatomy, neurochemistry and neurophysiology). They comprise:

• Sound therapy

The important role of the vestibular system in balance, attention and movement in general was discussed in chapter 4. Allergies, chronic otitis media and lack of movement amoungst other concerns can affect the effective functioning of the vestibular system. Pioneering studies in field of sound therapy demonstrate a general improvement in vestibular functioning as well as modulation of other sensory systems resulting in a general improvement of attention and motor activity following sound therapy. This will be discussed in more detail in section 2.1

• A neurodevelopmental approach

Since optimal human functioning relies on optimal neurological functioning, this aspect is of profound importance in the ability to pay attention and modulate movement. The neurodevelopmental process and the outcome of neurodevelopmental delays were discussed in chapter 4. Appropriate movement is crucial to general neurodevelopment and a lack of appropriate movement (as seen in the presence of aberrant primitive reflexes) has been associated with delays in development. It is said that movement structures the brain, and is thus responsible for maturation of the brain. The neurodevelopmental intervention programme used in this research is discussed in section 2.2

Neurofeedback (EEG neurofeedback)

The literature study (chapter 3) indicates a possible link betweeen altered brainwave patterns and a lack of attention and heightened motor activity. Pioneers researching the field of EEG neurofeedback achieved significant results with this form of biofeedback specifically with children struggling with attention and controlled motor activity. A discussion on EEG neurofeedback is presented in section 2.3.

• Nutrition

The importance of the neurochemical processes in the brain was established in chapter 3. Since neurotransmitters are derived from amino acids and amino acids are found in a healthy diet, nutrition is included as part of the ecosystemic approach. In chapter 4, information was presented that indicates a possible correlation between attentional difficulties and heightened motor activity and allergies, intolerences and nutritional deficits such as a lack of EFA. More information on this is presented in section 2.4.

2.1 Sound Therapy

Independently of each other, Tomatis in France, and Christian Volf in Denmark, were pioneers in the field of sound therapy. Since then, various techniques (e.g., The Listening Programme, The Tomatis Method, Berard Auditory Integration Training) have been devised to assess and to retrain the listening skills of individuals with such diverse problems as autism, hyperactivity, dyslexia, depression and imperfect pitch discrimination amongst musicians (Goddard 1996:50). Sound as a construct and the

auditory mechanism will be discussed first, followed by psychoacoustics and then the specific sound therapy utilised in this research.

2.1.1 Sound as a construct and the auditory mechanism

Sound can be described as omnipresent. This is true if one considers that everything that moves vibrates. Though sound is invisible, every aspect of our material world is constantly in motion. Where there is motion, there is frequency. Though inaudible at times, all frequencies make a sound. All sounds resonate and affect one another. In the spectrum of sound – from the movement of atomic particles to the sensory phenomenon we call music – there is a chain of vibration:

- all anatomic matter vibrates
- frequency is the speed at which matter vibrates
- the frequency of vibration creates sound (sometimes inaudible)
- sound can be moulded into music (Leeds 2001:2-3).

Hearing is the last sense to "shut down" as humans lose consciousness and awareness, for instance when we are dropping off to sleep. This is also the first sense to return when consciousness returns (Stafford & Webb 2005:147).

To explain sound, one needs to explore the entire sound spectrum (audible and inaudible). The constant factor is vibration, a rapid rhythmic motion back and forth, and frequency, the periodic speed at which an object vibrates. Resonance refers to the frequency at which an object most naturally vibrates. These sonic identifiers are the basic building blocks of sound (Leeds 2001:2).

All sound are vibrations in air. Different amplitudes create different sound intensities (different frequencies of vibration create different pitches). Natural sounds are usually made up of overlaid vibrations that are occurring at a number of different frequencies. Our experience of pitch is based on the overall pattern of the vibrations. Pitch isn't, however, always a quality that is directly available in sound information. It has to be calculated by the brain (Stafford & Webb 2005:154).

The pitch is based on what is called the *fundamental* of the sound wave. This is the basic rate at which the vibration repeats. Normally a sound is made by something that vibrates. Depending on how and what is hit, the main vibration is established – this is the fundamental vibration– which will be accompanied by a second vibration – at a higher frequency, called harmonics. These harmonics vibrate at frequencies that are integer multiples of the fundamental frequency (so for a fundamental at 4 Hz, a harmonic might be a 8 Hz or 12 Hz) The pitch of the sound heard is based on the frequency of the fundamental alone, it doesn't matter how many harmonics there are, the pitch stays the same (Stafford & Webb 2005:154).

The region of the brain that deals with hearing are the first to complete the developmental process called **myelination**, in which the connecting "wires" of neurons are finished off with fatty sheaths that insulate the neurons, speeding up their electrical signals. In contrast, the visual system doesn't complete this step of myelination until a few months after birth (Stafford & Webb 2005:147). This myelination priority may indicate the relative priority of the developmental sequence.

Sound vibrations travel down the ear canal and are transmitted by the tiny ear bones (ossicles) to the snail-shaped cochlea, a piece of precision engineering in the inner ear. The cochlea performs a frequency analysis of incoming sound, not with neural circuitry, but mechanically. It contains a curled wedge, called the basilar membrane, which, due to its tapering thickness, vibrate to different frequencies at different points along its length. It is here, at the basilar membrane that sound information is converted into neural signals, and even that is done mechanical rather than chemically. Along the basilar membrane are receptors, called hair cells. These are converted into tiny hair, which are in turn linked by tiny filaments. When a motion of the basilar membrane pushes the hairs, the tiny filaments are stretched, and like ropes pulling open doors, the filaments open many minute channels on the hairs. Charged atoms in the surrounding fluid rush into the hair cells, and thus sound becomes electricity, which Stafford & Webb (2005:149) calls the native language of the brain. Movements, as small as those on the atomic scale, are enough to trigger a response.

Each cycle of a sound can trigger a separate group of electrical pulses for low frequency sounds (up to 1500 cycles per second). Individual cycles are not coded for higher frequencies. Only the average intensity of the cycle is coded. The cells that receive auditory timing input in the brain can fire at a faster rate than any other neurons, up to 500 times a second.

This arrangement means that the auditory system is finely attuned to frequency and timing information in sound waves. Sounds, as low as 20Hz and as high as 20,000Hz, can be represented. The timing sensitivity is exquisite; the human ear can detect periods of silence in sound of as little as 1 millisecond (thousandths of a second). Compare this with the visual system, which requires exposure to an image for around 30 milliseconds to report an input to consciousness. Furthermore, thanks to the specialised system in the ear and in the brain, timing between the ears is even more exquisite. If sound arrives at one ear as little as 20 *microseconds* (millions of a second) before arriving at the other, this tiny difference can be detected. To get a better perspective think of an eye blink which is in the order of 100,000 microseconds and is 5000 times slower (Stafford & Webb 2005:149) than the perception of sound by the ear and the brain.

2.1.2 Psychoacoustics

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In essence, sound therapy is based on the theory of psychoacoustics. Psychoacoustics is the study of the effect of music and sound on the nervous system (Leeds 2001:2), in other words it is the "study of the perception of sound." This includes how the human listens, the psychological responses, and the physiological impact of music and sound on the human nervous system. "The study of psychoacoustics dissects the listening experience" (Leeds 2001:3). Traditionally, psychoacoustics is broadly defined as "pertaining to the perception of sound and the production of speech." The abundant research that has been done in the field has focused primarily on the exploration of speech and of the psychological effects of music therapy. However, there is renewed interest in sound as vibration (Leeds 2001:3) and its therapeutic effects.

Leeds (2001:3) makes an important distinction is the difference between a psychological and a **neurological** perception. A song or melody associated with childhood creates a memory-based psychological reaction. There is however also a physiological response to sound. Tones that are slightly out of tune may, for instance, cause brainwaves to speed up or slow down. Additionally, soundtracks that are filtered and gated – a sophisticated engineering process – create a random sonic event. This event triggers and activates a listening response and thus tonifies the auditory mechanism, including the tiny muscles of the middle ear. Accordingly, sound is perceived more accurately, and so speech and communication skills improve. While a psychological response may occur with filtered and gated sounds, or detuned tones, the primary effect is physiological, or neurological, in nature.

Frequent ear, nose and throat infections in early childhood resulting in intermittent hearing loss over a period of time can prevent the development of such auditory discrimination skills (Goddard 1996:49). Goddard further states that lack of auditory stimulation, or even a constant cacophony of background noise in early life can discourage early "listening" and the child may learn to shut out and ignore sound from an early age.

Hearing too much or "auditory hypersensitivity" is just as much of a problem as a hearing deficit. The inability to filter or occlude miscellaneous sound, suggests poorly developed listening skills, and can have profound effect upon later learning, language, communication, and behaviour. Some children suffer from hyperacuity, but as this is not measured in standard hearing tests (audiogram), the child may be dismissed as not having a hearning problem. Hearing too much can result in enormous concentration difficulties, speech difficulties and problems with social interaction.

Tomatis (1991) described how high frequency sound is "energising" while low frequency sound tends to be relaxing or "enervating". Clinical tests on a number of hyperactive children at The Institute for Neuro-Physiological Psychology have revealed the children to be hypersensitive in the **high frequency range** of sound. In some cases they perceive sounds between 2 000 and 6 000 Hz at a volume of

(minus) – 10dB and beyond (Goddard 1996:49-50) which falls in the hyperacuity range.

Sound is vibration, motion and energy. In the absence of sound the human perceives danger, for the totally silent world is a dead world. Goddard (1996:55) notes that sound passes through all levels of the brain and body, affecting not just the ear and the vestibular system but also our bodies through bone conduction. The significance of sound for learning is immeasurable.

The result of auditory malfunction is more than a diminution of hearing. A weakened auditory system endangers auditory sequential processing. This function affects short-term memory also known as the critical ability to link pieces of auditory information. Auditory sequencing processing is critically affected by auditory tonal processing. The neurodevelopmental specialist Robert J. Doman Jr. believes that the inability to focus our listening (a symptom of auditory dysfunction) diminishes communication, language, and attentional skills in adults and children (Leeds 2001:4). Is it a coincidence that the explosive growth in learning disabilities (a sequential processing issue) is paralleled by an accelerating rate of food allergies that cause children's ear infection (Leeds 2001:4)? According to Tomatis (1991), Doman (1980), and other pioneers, sound is a vital stimulant, a "nutrient" for the nervous system. Consequently, weakened auditory function depletes a major fuel system for the brain (Leeds 2001:5).

Basically two main distinctions can be made in terms of a faulty auditory system:

 Noise-induced hearing loss (NIHL), which occurs when too many protracted loud sounds damage the inner ear. NIHL is physiological damage: the delicate cilia hair cells cannot be repaired

• Stress-induced auditory dysfunction (SIAD). This term was coined by Leeds who says that stress can impede the active absorption of sound. According to Billie Thompson, a leading Tomatis follower and expert in auditory impairment; "poor listening can begin at any age and for any reason. It can result from a health problem, an accident, trauma, and major lifestyle disruption and from stress (Thompson & Andrews 1999:92; Leeds 2001:5). Among the symptoms of a degraded auditory function, says neurodevelopmental expert Robert Doman Jr. are



disorganised neurological function – affecting the ability to perceive, assimilate, process, and retrieve data – and emotional over-reactivity (Doman cited in Leeds 2001:5).

In addition, when the person can no longer tolerate the specific sound they begin to "shut down" the mechanism of the middle ear. This has the effect of eliminating the vocal frequencies they reject (Leeds 2001:5). Such psychological muting becomes a reflexive and subconscious action. As they shut out sounds, however, they also decrease the audio frequency spectrum.

The nett effect of hearing loss, be it noise or stress induced, is that a vital energy source for the brain and nervous system is diminished. The degradation of auditory function can also result in muddled thinking and unstable emotions (Leeds 2001:5).

Stress-induced auditory dysfunction affects the muscles in the middle ear (the tensor tympani and stapedes muscles become flaccid; this causes the ossicles – the three tiny bones in the middle ear – to work less efficiently in protecting the eardrum from excessive sound and in transmitting a full spectrum of sound). This form of hearing and listening loss can be addressed, to differing degrees, with sound stimulation auditory retraining programmes. While inner-ear damage from noise cannot be repaired, auditory retraining of the middle ear allows for better use of what hearing remains (Leeds 2001:6).

2.1.3 Sound therapy applied

Sound therapy may be of help to most children who suffer from a learning disability. This includes attentional deficit disorder, poor concentration, hyperactive tendencies, poor organisational, difficulties with reading, writing, and spelling, as well as some difficulty with math and memory problems (Madaule 1994:31).

For the purpose of this study, the model presented by Berárd was utilised. This model proposes a ten-day consecutive sound intervention programme. At the outset, a listening profile is obtained by way of testing the individual threshold of sound perception in all eleven frequencies as presented on the audiogram (see section

chapter 4, section 4.19). An accurate listening profile is of extreme importance as the profile is utilised to determine the setting of the equipment used in Berárd Auditory Integration Training (AIT). The specific equipment used in Berárd AIT is known as an Earducator. This device receives musical input from a sound source, usually a compact disc player, which processes the music in one or both of two ways. One way, termed "modulation" involves using wide-band filters to attenuate frequencies within specific ranges for brief periods (250 msec. to 2 sec.). The AIT device determines the onset and duration of the modulated stimulus randomly. The second way, "filtering" involves using narrow-band filters to attenuate specific frequencies to which the individual is acutely sensitive. The music is presented for a period of 30 minutes, twice a day with at least three hours between the two sittings. In total, twenty hours of sound therapy is presented. Usually no follow-up sessions are needed after the initial programme.

2.1.4 Theories on the functionality of Berárd AIT

There are a number of theories as to why Berárd's AIT is successful. A short synopsis of some theories is presented here.

i) Cerebellar-vestibular system theory

A dysfunction of the cerebellar-vestibular system can produce an assortment of symptoms and behaviour. Some of these symptoms include; problems with balance, coordination, vision and hearing, motion sensitivity, problems with sense of time and direction, poor concentration and memory, hyperactivity, obsessive-compulsive disorder, difficulty with academic performance and anxiety. It has been reported that when there is impairment in the sensory channels, vestibular stimulation and rotation seem to open the door for further remediation, even when nothing else has worked. Ayres (1974, 1979, 1982), a sensory integration specialist supports this theory in her discussion of how sensory organisation dysfunction prevents the child from correctly processing the sensations from activities to organise the brain. When sensory input is properly provided, it can facilitate development of the nervous system and eliminate problem behaviour.

AIT may be one method of providing stimulation to the cerebellar-vestibular system to help recognise a dysfunctional system. AIT stimulates this system through specifically designed, vibrating sound waves, rather than rocking, or deep pressure massage often used to stimulate the cerebellar-vestibular system. Improvement in symptoms usually associated with the cerebellar-vestibular system is reported to improve after AIT (Veale 1993). This may occur since the vestibular system helps modulate neural activity and acts as a central reference point from which all other sensory information is composed. According to Ayres (1974, 1979, 1982), the reticular activation system (RAS) helps the brain focus on one sensory input or type of sensory input and inhibits other types. The unpredicted, modulated music used in AIT would stimulate a particular area in the reticular activation system, which is activated by novel sensory input. Therefore, the unique sounds used in AIT may be capable of producing changes not only in the auditory processing of sound, but also in posture, balance and spatial orientation. The reticular activating system also depends on the neurotransmitter norepinephrine, which plays a role in attention, motivation, emotion and arousal (Brockett 1993).

ii) Reduction in peaks through filtering

Another theory as to why AIT is successful is Berárd's theory, and the reduction in peaks due to a decrease in stimulation. During the AIT listening sessions, filters are used to dampen those frequencies, which the person hears too well, that is to which they are hypersensitive. He postulates that reduced sensitivity is due to the lack of stimulation to certain areas of the cochlea and/or the brain during the listening sessions. Furthermore, those areas(s) of the cochlea and/or brain, which are not filtered, receive intense stimulation; and this stimulation causes a slight improvement in the quality of hearing. Hence the peak frequencies are slightly reduced because of a lack of stimulation, and the non-peak frequencies are slightly improved because of stimulation. An extension to this theory is the theory of lateral inhibition.

One phenomenon that occurs throughout the sensory system is neural inhibition. Some neurons, when stimulated, inhibit the activation of other neurons. In addition, a phenomenon called "lateral inhibition" occurs when a stimulated area inhibits an adjacent area which is not receiving stimulation. Since the filtered frequencies are not stimulating certain portions of the cochlea and/or part(s) of the brain, stimulation of the adjacent, non-sensitive, (non-filtered) areas may be laterally inhibiting, and possibly conditioning, the sensitive filtered area(s). Consequently sensitive areas are inhibited by the adjacent, non-sensitive areas.

iii) Theories related to the middle ear

The middle ear contains the body's smallest three bones (malleus, incus and stapes – collectively called the ossicles), and two of the body's smallest muscles (tensor tympani muscle and stapedes muscle). When sound enters the outer ear and strikes the tympanic membrane, the vibration of the eardrum causes the malleus to move, which in turn, moves the incus and the stapes. The vibrating stapes then strikes the oval window of the inner ear (cochlea), which then causes fluid in the cochlea to bend the tiny hair cells, which the auditory nerve then interprets as various sounds.

The tensor tympani muscle and the stapedes muscle are responsible for providing the proper tension to the ossicles. Additionally, when exposed to loud sound, the two muscles react together, which in turn, restrict the movement of the ossicles. As a result, the stapes strikes against the inner ear with limited force. This is known as the acoustic reflex. This is important in protecting the person from loud and harmful sound.

Currently, there are two possible theories as to how AIT affects this system. One explanation is that muscle tone is not adequate for proper functioning of the middle ear. AIT listening sessions exercise and strengthen the muscles in the middle ear, which improves the muscle tension and enables efficient transmission.

Another theory is that the tensor tympani muscle and the stapedes muscle are no longer working together to form the acoustic reflex. This may result from a trauma to the middle ear, such as a middle ear infection. By listening to very stimulating, modulated music, the two muscles are given a 'good work-out' and as a result, they start to work together to form the acoustic reflex.

iv) Sensory re-organisation

In some individuals, the auditory system may be abnormally developed as a result of genetic problems in utero, and/or to early childhood experiences. Problems present more frequently in individuals with developmental disorders.

In the same way as the other senses in the body, the auditory system is very adaptable and can change given the appropriate stimuli and structure. During the AIT listening sessions, the auditory system is stimulated in such a way as to place it in a state of flux, disarray, or chaos. After the completion of the listening sessions, the auditory system reorganises itself, in a more natural, structured and functional manner.

2.1.5 AIT application in this research

Sound therapy, and specifically AIT, was presented after an initial period of neurodevelopment. A trained AIT practitioner presented AIT during the last 10 days of the eleven week programme.

2.2 Neurodevelopmental Protocol

The discussion on neurodevelopment with specific reference to specificity and plasticity (chapter 4 section 2.1.2) and knowledge of the developmental sequence and suppression of primitive reflexes (chapter 3 section 4.6.3.1) allows the professional to develop a neurodevelopmental programme to address neurodevelopmental delay. Once the rules and mechanisms of normal development are understood, a suitable programme of neurodevelopment can be designed. Specificity in development allows this understanding as certain patterns and sequences are almost always followed during normal neurodevelopment.

Since these patterns and sequences constitute specific movements, movement programmes that include these basic patterns were investigated for the purpose of this study. There are many excellent motor training programmes available, which can be used within the school system. Experts in the field such as Kephart (1975), Cratty (1972), Dennison and Dennison (1989), Goddard (1996), Hannaford (1995) and Pheloung (1997, 2003) support the importance of movement in general development. A recent study conducted by Fredericks, Kokot & Krog (2006:29) in a primary school indicated significant improvements in reading and math ages, lateralisation, coordination and gestalt after an eight-week motor programme. For the purpose of this study, the HANDLE (Holistic Approach to Neuro-development and Learning Efficiency) programme was incorporated as part of the ecosystemic approach. The following section includes information HANDLE's of on perception neurodevelopment, as well as results from previous research studies on the efficacy of the HANDLE approach.

2.2.1 Neurodevelopment as a construct (HANDLE's perspective)

Judith Bluestone (1994, 2000; Bluestone & Sulitaneau 2001), director of the HANDLE Institute in Seattle, developed this dynamic approach to address the root causes of, among other things, attentional difficulties and heightened motor activity. The HANDLE perspective defines neurodevelopment not as a given sequence of acquired skills but as an interactive hierarchy of brain functions, with a vestibular foundation for these skills (Bluestone, 1994). The interaction and interdependence of these functions is diagrammatically represented on the Sensory-motor Interdependency and Interaction Chart (figure 48). The relative position of these systems on the chart is an indication of the hierarchical nature of the neurological system, and illustrates how higher level functions depend on those at a lower level. Each of these systems is discussed in chapter 4. A comprehensive outline of neurodevelopment in general is presented in chapter 4 (section 2.1)

Bluestone (1994) states that other approaches isolate the different structures of the brain; however, when neurodevelopment is understood as interactive, no time frame limits brain function. Learning is thus the lifelong process of using sensory, motor, social and emotional input to realign output into effective behaviour (Kokot 2003a:14). The holistic nature of the approach also requires recognition of internal and external influences. This indicates a general acknowledgement of possible causal roles of chemicals, allergens, nutritional deficits (especially EFA), dehydration

and toxins of any kind (nutritional deficiencies are discussed in section 7.3 in chapter 4).

2.2.2 HANDLE's conceptualisation of attentional difficulties and heightened motor activity

According to the HANDLE perspective, the human being is incapable of suffering from a so-called attention deficit. The brain has been designed to pay attention to something all the time (eg: internal and external processes such as respiration, protecting the organism from allergens, maintaining equilibrium, cutting out disturbing noises from the environment, etc.) Following this perspective on the human's ability to pay attention, Bluestone (1994; 2000) coined the term Attentional Priority Disorder (APD) (chapter 4 section 6.3).

HANDLE clinicians incorporate information from many disciplines to uncover the reason why a specific individual blocks certain types of stimulation and seeks others, and why the person has difficulty adjusting attentional priorities flexibly to meet varying demands. According to this perspective, APD is not hereditary or irreversible, although tendencies may be inherited.

Information processing, a fundamental aspect of optimal neurological functioning is discussed in chapter 4, section 2.4 and illustrated in figure 26. According to the HANDLE perspective, there seems to be a common pattern among people presenting with APD. Most people who have difficulty sustaining their attention and/or adjusting readily to the demands of new situations demonstrate irregularities in specific developmental functions.

On an input level the following may be observed:

- hypersensitivity to at least one modality such as tactile, visual, or auditory input
- weakness in the vestibular system which supports and regulates such functions as listening, dynamic use of our eyes, balancing, feeling at ease with our bodies in space, having an appropriate state of readiness in our resting

muscles (chapter 4 section 4.4). It also integrates with systems that govern breathing, pulse, and so on

On an output level the following is commonly observed:

- difficulty processing various elements of a situation and acting upon those that require response in a self-regulated way due to problems such as
- · insufficient coordination between the two sides of the body and brain, and
- immature reflex inhibition

2.2.3 Treatment approach

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HANDLE relies on the fact that the nervous system is intended to adapt continually throughout the lifespan. Medical research indicates that individuals diagnosed with APD have thinner neural fibres than other people; and that thinner fibres process information more slowly than denser fibres, which have more network connections and myelination (Bluestone 1994; 2000). Through regular gentle enhancement of weak functions and providing proper nourishment to strengthen the nervous system, "weak" aspects of the nervous system can be strengthened. Thus, the individual gains the ability to focus, sustain attention, adjust to changes, and ultimately respond appropriately.

Human functioning is intersystemic in nature. HANDLE differs from other neurodevelopmental programmes as it recognises sound neuroscientific findings that indicate that by performing a non-integrated activity for an extended length of time, the nervous system may become less functional (Bluestone 1994:i) and a general shutdown may occur due to physiological stress. This is caused by two factors:

- Prolonged stimulation of immature functions/systems can pattern an improperly integrated response. In other words, a poor habit may be formed, which then needs to be broken.
- Even more significantly, overstimulation of immature systems can cause the nervous system to become overwhelmed or stressed, which in turn leads to shutdown, a regression to earlier behaviours, and possibly lethargy (Bluestone 1994:i).

2.2.4 Research on HANDLE

The HANDLE Institute conducted a retrospective outcome study to establish the time frame from the inception of the programme until progress was reported. The consistency of the progress was also measured. The study was conducted by Bluestone and Suliteanu (2001) who tracked 61 individuals in whom the referral concern was AD/HD. Clients included in the study ranged in age at first consultation from thirty months to over fifty years. Most of the clients were male (46), with only 15 females.

All the individuals were provided an individualised treatment programme of specific activities, based on the neurodevelopmental profiles that resulted from their assessment by certified HANDLE practitioners. Most clients were also advised to drink water and to supplement their diets with essential fatty acids.

To modify the programme in accordance with progress, practitioners encouraged follow-up sessions after approximately ten days and then at monthly intervals.

2.2.4.1 Summary of conclusions

The following conclusions were reported:

- Functional improvements were reported in 56 of the 61 individuals.
- Progress was reported by week 14 after the initial assessment and the onset of the therapeutic programme in all but 4 males and 4 females.
- The more significant patterns that emerged related to age and sex of the clients were that most males entered the programme between the age of 7.5 and 10 years. A comparable cluster occurred for ages 10.5 and 13.5 years of age. Therefore, nearly 58% of the male population entered the HANDLE programme between the second and sixth grade.
- Just as each client initially presented with individualised profiles, recorded improvements varied from client to client, despite all clients having had ADHD as a presenting concern. Areas of progress noted included but were not limited to:
 - better sleep
 - staying seated while working, eating, and while travelling by car

- improved homework
- more motivated
- improved mental state
- getting to work on time
- improvement in following instructions
- better attention
- reduced bedwetting
- improved impulse control
- better reading, maths, spelling
- more focused
- stopped medication
- makes friends more easily
- calmer
- no longer bumps into things and people in path
- school work is faster, with quicker follow-through
- diminished startle response
- greatly diminished tactile sensitivity
- reduced light sensitivity
- reduced auditory distractions
- less dawdling
- more organised
- increased appetite and range of foods
- accomplishes more around the house
- faster response time
- happier (Bluestone & Suliteanu 2001)

2.2.5 Treatment plan

The Sensory-motor Interdependency and Interaction Chart shown in chapter 4 (figure 43) illustrates the dynamic interplay between the different subsystems. The chart depicts a hierarchical trajectory of human development, from bottom to top. The many arrows illustrate that the different subsystems support others while being supported by those beneath them. Systems higher up in the system could have an



effect on systems below them. All the systems need to fulfil their function in the interaction, or they risk placing more strain on supporting functions. The chart should also be viewed in terms of the items right at the bottom of the chart. These items include intuition, ecosystem, time, maturation and nutrition. Bluestone (1994, 2004) proposes that the entire ecosystemic environment (both internal and external) is important when compiling a profile of the child. When trying to understand the reasons for a child's inability to pay attention or to control motor activity, Bluestone (1994, 2004) emphasises both the pre- and postnatal periods in the child's ecosystem. Thus, environmental offenders such as cleaning agents, heavy metals, insecticides, and the like are considered. Allergies and their possible effect of cognitive functioning are explored. The birth and possible stressors in both the mother and foetus are taken into consideration.

The different subsystems depicted in the chart are discussed at length earlier in this chapter. The main point illustrated by this chart, however, is that none of these subsystems can function alone. They all work together to allow individuals to experience the world fully, and to allow people to react to this reality by assimilating incoming information through all the subsystems that permit the entry of information. Accurate assumptions and interpretations are only possible when information entering the organism compares accurately with the environment, and when the information presented is consistent.

The Sensory-motor Interdependency and Interaction Chart not only provides a framework for understanding the interplay of the subcortical subsystems, but also provides clues to a therapeutic intervention that starts at the root, and gradually develops and structures the neural networks that support the cortical systems. Once assessment (clinical observation, questionnaires and specific tasks) is completed and a comprehensive understanding of the functionality of the underlying neurodevelopmental subsystems is obtained, the developmental programme is designed. Identification involves examination of possible dominant subcortical systems that may be interfering with cortical functioning, such as aberrant primitive reflexes, as well as dysfunction in visual and or auditory systems, vestibular system and proprioceptive system, to name but a few (Kokot 2001:124).

The therapeutic programme consists of a number of specific acivities presented on a daily basis. Supporting subsystems at the bottom of the chart are addressed first in order to assist the natural process of maturation. Concepts such as plasticity, specificity and developmental sequence were discussed in length in previous sections of this thesis. In general, this approach relies on certain essential aspects such as the fact the nervous system is designed to adapt continuously, and that the type of stimulation received influences the patterns in the brain. Other aspects are also considered, for instance that movement reflects our neural organisation and provides stimulation to our systems; that the nervous system, especially its weak parts, responds to overwhelming stimulation with a stress reaction; and that stressed systems shut down; and, finally, that there is a unity in the senses: irregularities in one area may resonate symptomatically in other areas. One of the fundamental aspects of the HANDLE perspective is gentle enhancement. Individuals are carefully monitored for signs of stress during therapeutic exercises and encouraged not to continue if any state changes are noted. This is particularly important as Volk (1997:12) emphasises that a stressed system is a system that shuts down.

Usually the intervention techniques are practised in the home or daycare/school setting. During this research, the activities were performed at school and facilitated by a trained HANDLE practitioner. In this way small, measured doses of specific activities were encouraged and incorporated into daily activities. This ensures that strengthened organisational patterns become functional for individuals in their environment.

The recommended activities are simple to perform, and require virtually no special equipment. Each exercise programme is specially designed to meet the individual client's specific needs. Some of the more frequently suggested activities involve:

- drinking from a crazy straw
- playing follow-the-leader with a flashlight, using only the eyes to follow the light
- rhythmic ball bouncing
- copying designs by feel alone
- catching a suspended ball
- stepping through a hula hoop "maze".

2.3 EEG Neurotherapy

In chapter 3 reference was made to the use of EEG as a possible indicator of attentional difficulties. In this section EEG neurotherapy will be addressed. Since evidence exists that there are actually EEG differences between clinical and nonclinical populations (see Clarke et al. 2002a:1036), this section investigates the possibility of employing base EEGs for the purpose of identifying markers in children diagnosed with AD/HD as well as the use of EEG neurofeedback as method of intervention.

2.3.1 Background

The first notion that human nerve impulses are somehow electrical in nature goes back to 1791, when Luigi Galvani, an Italian researcher, published a paper on the subject. An English physician named Richard Caton was the first to discover that the brain generated electricity. In the 1875 he used a device called a reflecting galvanometer to make the discovery. Caton detected the weak flow of current across an unopened skull and recorded the first account of what would become the brain's electrical signature, the electrical encephalogram (EEG) (Robbins 2000:16). Electroencephalogram literally means "electrical head picture" (Robbins 2000:55) and other have called it the "electrical fingerprint of the brain".

Hans Berger, a physiatrist, studied Caton's work and expanded on this. Even though the brain is a constant electrical storm, its electrical potential is only about fifty millionths of a volt, a tenth of the voltage that is measured from the heart. This makes it difficult to measure in comparison with the heart. In 1924 Berger made the first official EEG recordings. He was unsure exactly how precise his measurements were, resulting in a time laps of five years before publishing a paper in 1929 entitled "On the Electroencephalogram in Man." The first frequency he encountered was in the 10-hertz range (hertz – cycles per second – Hz), which at first was called the Berger rhythm (Robbins 2000:19). Berger later named this rhythm **alpha**, using the first letter of the Greek alphabet. He subsequently discovered and named the **beta** range (Thompson & Thompson 2003:7). Berger later observed that the EEG changes with mental activity when he hooked up his daughter and asked her to do mathematical divisions.

2.3.2 EEG terminology

The following terminology was extracted from Thompson and Thompson (2003:35-36):

Frequency

Frequency refers to the number of waves produced in a second. The EEG is merely a waveline. This line consists of many different morphologies (shapes). All the frequencies are mixed together in the many lines called an EEG



Figure 66 Illustration of different frequencies

Hertz

The unit of measurement for the frequency recorded is cycles per second, or Hertz (Hz) named after Heinrich Hertz.

Amplitude

Amplitude is usually measured in microvolts, or millionths of a volt, this will determine the actual strength of the specific brain rhythm measured (Thompson & Thompson 2003:17)

Bandwidth

Bandwidth refers to frequency ranges, e.g. alpha usually run in a frequency range between 8-12 cycles per second. But alpha can also run slower at 6 or 7Hz or faster

at 13Hz. It is thus not only the number of cycles per second that determines the waveform, but also the morphology of the wave (figure 66).

Spectral array is used to compare the amplitude of specific bandwidths with one another. In essence – this is a histogram showing the amplitude of each frequency usually between 2-32Hz.

Impedance

Impedance (electrical) may be defined as the resistance to alternating electrical current flow. Impedance shows the general "housekeeping" in terms of EEG. This assures that the recording being made is actually of brain frequencies and not other frequencies such as those emitted from alternate sources like the electrical outlet in the room. For the purpose of this research the impedance was kept at <5 kohms in all combinations of leads with <1 kohm difference between leads. This is according to international research standards (Thompson & Thompson 2003:65).

2.3.3 Generation of cortical potentials

i) Membrane potential

Brainwaves can be measured and recorded because electrical impulses are produced from the cortex. Electrical potentials exist across the membranes of essentially all cells of the body, and some cells, such as nerve and muscle cells are "excitable" – that is, capable of transmitting electrochemical impulses along their membranes (Guyton 1972:12). The fluids both inside and outside the cells are electrolytic solutions containing negative ions (anions) and the same concentration of positive ions (cations). Generally, an excess number of negative ions accumulate immediately inside the cell membrane along its inner surface, and an equal number of positive ions accumulate immediately outside the membrane. The result of this is the development of **membrane potential**. There are two basic means by which membrane potential can develop:

 active transport of ions through the membrane which creates an imbalance of negative and positive changes on the two sides of the membrane diffusion of ions through the membrane as a result of a concentration difference between the two sides of the membrane which also creates an imbalance of changes (Guyton 1972:12; Kalat 2001:42-43).

ii) Action potential

As long as the membrane of the nerve fibre remains completely undisturbed, the membrane potential remains unchanged which is called **resting potential**. Many factors can suddenly increase the permeability of the membrane to sodium. This is likely to elicit a **sequence of rapid changes** in membrane potential lasting a minute fraction of a second, followed immediately by the membrane potential returning to its resting value. This sequence of potential changes is called the **action potential**. Some of the factors that can elicit an action potential are **electrical stimulation** of the membrane, **application of chemicals** to the membrane to cause increased permeability to sodium, **mechanical damage** to the membrane, heat, cold, or almost any other factor that momentarily disturbs the normal resting state of the membrane (Guyton 1972:12; Kalat 2001:44-45).

iii) Propagation of the action potential

An action potential elicited at any point on an excitable membrane usually excites adjacent portions of the membrane, resulting in propagation of the action potential (Guyton 1972:12). Propagation usually occurs in one direction.

iv) Special aspects of impulse transmission in nerves

Figure 3 illustrates a typical neuron. The large fibres are myelinated and the small ones are unmyelinated. The average trunk contains about twice as many unmyelinated fibres as myelinated fibres. The central core of the fibre is the axon, and the membrane of the axon is the actual conductive membrane. The axon is filled in its centre with axoplast, which is a viscid intercellular fluid. Surrounding the axon is a myelin sheath that is approximately as thick as the axon itself, and about once every millimetre along the extent of the axon the myelin sheath is interrupted by a node of Ranvier. The myelin sheath is deposited around the axon by Schwann cells in this way: the membrane of the Schwann cell first envelops the axon, it then rotates around the axon several times and lays down multiple layers of cellular membrane containing the lipid substance sphingomyelin. This substance is an excellent insulator that prevents almost all flow of iron. However, at the juncture between each two successive Schwann cells along the axon, a small uninsulated area remains where ions can flow with ease between the extracellular fluid and the axon. This is the node of Ranvier (Guyton 1972:17; Kalat 2001:45-46). One can think of the myelinated axon as being super highways allowing fast speeds (Thompson & Thompson 2003:28).

Impulses are conducted from node to node by the myelinated nerve rather than, as in unmyelinated fibre, continuously along the entire fibre. This process is called *salutatory conduction*. That is, electrical current flows through the surrounding extracellular fluids and through the axoplasm from node to node, exciting successive nodes one after another. In this way the impulse jumps down the fibre, which is where the term "salutatory" comes from (Guyton 1972:17; Kalat 2001:45-46).

Salutatory conduction is believed to be of value for two reasons: firstly, by causing the depolarisation process to jump long intervals along the axis of the nerve fibre (it is reasonable to believe that this mechanism helps to explain the high velocity of nerve transmission in myelinated fibres); and secondly, salutatory conduction conserves energy for the axon, for only the nodes depolarise, allowing very little loss of ions and requiring little extra metabolism for retransporting the ions across the membrane (Guyton 1972:17; Kalat 2001:45; Marieb 2003:212).



Synaptic cleft

(http://www.txtwriter.com/Backgrounders/Drugaddiction/drugs1.html)

v) Synaptic cleft and neurotransmitters

Chemical conduction using neurotransmitters occurs at the synaptic junction between nerves (Thompson & Thompson 2003:17). The synaptic terminal releases neurotransmitters, which attach to specific receptor sites on the post-synaptic membrane causing the post-synaptic membrane to become temporarily permeable to the specific ions (Thompson & Thompson 2003:23).

Neurons are thus like microscopic batteries. Their membrane builds up a charge electrochemically and then releases it, over and over again. Cells fire in unison to create thought and movement, and information travels around the brain in networks. An EEG electrode (small metal disc) reads the activity of about one hundred thousand neurons at one time. Exactly how information (thought) is encoded in the electrochemical soup is still a deep mystery. There are as many as one hundred billion of these long and spidery neurons, or cells, in the human body, and each one may make from one hundred to hundreds of thousands of connections with other cells, which could calculate to a total of perhaps ten to a hundred trillion connections. These connections are at the heart of how well the brain functions (Robbins 2000:30). One of the key measures of the functional maturation of the brain is through the electrical activity, which is an index of trans-neuronal trafficking and communication. The sum of all this trafficking can be recorded as an EEG.

Although much is known about the brain, there still seems to be a great deal of uncertainty about the actual production of some of the brain rhythms. A general understanding of production of alpha (9-12Hz) is available (brain stem – thalamus – cortex axis). Alpha rhythms are under brain stem neuromodulatory control. For frequencies outside the alpha range, understanding is less complete. Sterman (1994) as sited in Abarbanel (1999:315) proposed that three systems of (integrative) brain activity influences the thalamic generation (also referred to as the pacemaker) of field potentials at scalp level:

 Vigilance (involves diffuse neural networks and specific centres in the brain stem and their ascending influence on thalamic, subcortical and cortical centres).

- 2. Sensorimotor integration (involves the ascending touch and proprioceptive pathways and their projections to the thalamus and on to sensor motor cortex, along with afferent pathways from this cortical area).
- 3. Cognitive integration (involves a wide range of neural centres that process and integrate sensory input and motor responses).

Sterman (1994) suggests that SMR, alpha and theta rhythms appear when specific input from the three systems are withdrawn from the thalamus. The prototypical theory is that an alpha generation stems from the thalamus by brain stem cholinergic activity. If sensorimotor inputs are withdrawn, SMR, across the sensorimotor strip, is affected. If cognitive processing is withdrawn (as in relaxed states without cognitive activity), alpha appears. If vigilance is withdrawn (as in states of inattentive drowsiness), theta appears. Consequently the presence of these rhythms on the EEG indicate the influence on the thalamus of underlying brain states related to vigilance (attention), cognitive processes (impulse control) and sensorimotor integration (motor activity). It is assumed that attentiveness intrinsically accompanies states of SMR-associated stillness (including frequencies between 15 and 20 Hz normal beta). Specifically, the combination of higher activity in beta and SMR range and lower activity in the theta range associates directly with states of increased stillness and attentiveness and with decreased drowsiness and other cognitive disturbances associated with theta activity (Abarbanel 1999:316-317). This theory forms the basis for most of the protocols used with patients diagnosed with AD/HD.

2.3.4 EEG neurofeedback application

One of the pioneers of neurofeedback was Barry Sterman. Sterman worked with cats in the late 1960s at the University of California Los Angeles, demonstrating that they could be trained using a method called operant conditioning to increase a specific spindle-like brainwave pattern which ran between 12 and 19 cycles per second. Sterman later coined the term SMR (sensorimotor rhythm as measured over the sensorimotor strip). Shortly after this discovery he observed that cats that had increased their SMR were resistant to seizures caused by a toxic chemical used in rocket fuel (this discovery occurred during a later research study funded by NASA that incidently used the same cats as those in the epilepsy research study). By teaching the cats to produce more SMR, Sterman strengthened their brain functioning at the sensory motor strip in the same way a person builds muscle mass by repeatedly lifting weights. In medical parlance, their "seizure threshold" had been increased; their brains were now functionally altered so as to resist the spread of slow theta waves across the motor cortex that caused seizures (Robbins 2000:42; Thompson & Thompson 2003:8).

Sternan's (1994, 2000) work demonstrated a clear connection between **mind** and **physiology**. He concluded that in the brain, cell firing patterns changed and cells in the motor pathway reduced their rate of firing. In conjunction to these, circuit patterns changed as well as changes in the body, namely the fact that respiration stabilised, heart rate went down and muscle tone in antigravity muscles decreased. It was also noted thet reflexes diminished.

These animal studies clearly demonstrate that the effect and changes of sensory motor rhythm training (SMR – Beta [12-15Hz] measured across the sensory motor strip) are physiological and not, as some critics say, a placebo. **Placebo** is Latin for "I shall please". Sterman goes on to say that there is no placebo effect in cats and monkeys (Robbins 2000:42).

What Sterman and his research team apparently accomplished, even though it was not fully appreciated at that time (or even today) was enormous. If brain cells are indeed becoming stronger, and grow new and permanent connections, as his model holds, he has gone a long way towards demonstrating a concept that is increasingly accepted by neuroscientists: **that the brain is a dynamic and extremely plastic organ.** He has also found a way to capitalise on that plasticity. He demonstrated that, by simply teaching a person how to nudge the brain in certain directions, the brain was capable of profound change. Sterman also provided tantalising evidence – under rigorous scientific conditions in the laboratory – that there was a way to harness the mind-body connections: simply guiding the way one thinks can change the structure of tissues in the brain and, subsequently, other key parts of the human physiology. Neurons, his research demonstrated, are where **mind meets body**. The concept was revolutionary: a whole new paradigm (Robbins 2000:51; Sears & Thompson 1998:205).





Joel Lubar continued the work of Sterman and discovered that measuring the theta/beta ratio showed up a key difference between non-AD/HD and AD/HD (Thompson & Thompson 2003:8, 117) at Cz, which showed a high sensitivity for distinguishing AD/HD from other individuals. Monastra et al. (1999) (cited in Thompson & Thompson 2003:116) published the most comprehensive work up to date on theta/beta ratios. They proposed the use of 4-8Hz/13-21Hz. Some EEG therapists also use other ratios, such as 4-8Hz/16-20Hz, as this usually correlates with the neurofeedback protocols used with AD/HD patients. A general guideline would be a ratio of >2.5 for children above seven years.

Six good reasons for using the EEG are:

- · mental state can be 'defined' by the EEG
- brainwave patterns correspond to correspond to common disorders or syndromes, for example the theta/beta ration corresponding to AD/HD
- animals and humans can learn to alter brainwave patterns with the use of operant conditioning
- animals and humans show changes in behaviour after they have learned to alter brainwave frequency patterns
- brainmaps using nineteen active electrodes can help distinguish psychiatric syndromes
- brainmaps using 19 active electrodes can demonstrate communication patterns between different areas of the brain (coherence and co-modulation) (Thompson & Thompson 2003:18-19)

2.3.5 Neuromodulator control

Biofeedback is the use of instrumentation to mirror physiological processes of which the person is normally unaware and which could be brought under voluntary control. In neurofeedback the physiological process brought to conscious awareness is the frequencies and amplitudes of various brainwaves which are being measured with electrodes. The electrodes comprise a positive (+ve), a negative (-ve) and a ground lead/electrode in EEG neurofeedback practices. The EEG electrodes measures the potential difference between the +ve and the –ve leads. The electrical activity being produced in the brain can be displayed on a computer screen almost immediately.

This is called a base EEG and differs from a clinical EEG as recorded by a neurologist using 19 active leads (fullcap).

The process of biofeedback implies more than a passive measurement. It implies an active involvement of the client. Biofeedback is performed in order to allow the client to become actively involved in controlling their own physiology; hence the term 'applied psychophysiology' (Thompson & Thompson 2003:2).

EEG neurofeedback is based on two basic tenets:

- brain electrical activity reflects mental states
- · electrical activity can be trained

As clients alter their own mental state, the amplitudes of various brainwave frequencies change. The client sees these changes on the computer screen and attempts to maintain these changes for longer periods of time by using the feedback from the computer. The end result is self-regulation. In this manner the client learns to self-regulate, and learns normalisation of EEG patterns (Thompson & Thompson 2003:3) or neuromodulater control.

It is well established that a positive reward for behaviour is followed by an increase in the probability of that behaviour recurring (Edward Thorndike's Law of Effect). The production of particular brainwave patterns is the behaviour that is rewarded in EEG neurofeedback. The reward is information about success using sound and visual displays provided by the computer (Thompson & Thompson 2003:3). Rewarding behaviour therefore 'shapes' the contributing component of the behavioural sequences in a way that results in an increased recurrence of that specific behaviour sequencing. Hence shaping is produced through the process known as operant conditioning. The evidence that EEG biofeedback can produce significant and sustained physiological changes was documented by Lubar after a ten-year follow-up study (Thompson & Thompson 2003:118).

One hypothesis about what might be going on in the brain during neurofeedback has to do with the way cells in the brain connect to one another. Since information travels along the branchlike dendrites, the denser and greater in number these connections are, the better the transfer of information. As frequencies increase (higher amplitude for the frequency being rewarded) during a neurotherapy session and the brain is activated, more blood than usual streams to that area of the brain. The nutrients in the blood may strengthen or reorganise existing connections, which increases the cell's ability to self regulate. This is what scientists think happens during any learning (brain scans show that the neurons in the area that governs their reading fingers become more robust for people who go blind and learn Braille.) The neurofeedback model holds that the brainwave training increases the stability of that area of the brain as well as its flexibility, or its ability to move between mental stages (from sleep to consciousness or arousal to relaxation for example.) An analogy often used is that of players (brainwaves) in the orchestra, and that neurotherapy allows the players to play their parts better, to find the correct tempo, to come in on time, and to stop playing when they are not needed. The thalamus is often referred to as the conductor of the orchestra. Since every aspect of a person is driven by an assembly of neurons, the healthier those neurons are, the healthier are the functions that they govern (Robbins 2000:45).

The goal of neurofeedback (NF) is to effect a lasting change in the system through learning. If NF is to make effective and lasting changes in the neural circuitry mediating attentional functions, those circuits must be adjustable by feedback control, and must also be able to maintain those adjustments over time. This implies that the system must be sufficiently **plastic** (Abarbanel 1999:316). Neurofeedback augments the brain's capacity to regulate itself, and that this self-regulation lies at the heart of its clinical efficacy. Schematically it could be plotted as shown in figure 68.



Figure 68 Neurofeedback augmentation

(Thompson & Thompson 2003:257)

For the purpose of measuring the brainwaves and for easy referral to the specific location of the brainwave pattern, the human skull has been divided into 19 different sites in an international system of measurement called the international 10-20 system (Robbins 2000:44).

The international 10-20 system:

According to the 10-20 system (Jasper 1958:374), electrode placement is the most widely used method to describe the location of scalp electrodes. The 10-20 system is based on the relationship between the location of an electrode and the underlying area of cerebral cortex - distance (10% or 20%) between nasion (anterior) and inion (posterior). Use of a percentage-based system allows for differences in skull size. Each site has a letter (to identify the lobe) and a number or another letter to identify the hemisphere location. Even numbers refer to the right hemisphere and odd numbers refer to the left hemisphere. "Z" refers to an electrode placed on the midline. Smaller numbers are closer to the midline. "Fp" stands for 'front polar'.



Figure 69 Diagram of the International 10-20 Electrode Placement System

(Adapted from Thompson & Thompson 2003:152)

It is at this point where a visible connection between neuroanatomy and neurochemistry occurs in neurophysiology. The site of the specific brainwave pattern measured results in a corresponding mind state. The same situation can also be

viewed from another angle. Purposefully producing a specific mind state results in a specific brainwave pattern (emphasising the body/mind inter-consecutiveness).

2.3.6 Neurotherapy protocol

The generally accepted neurotherapy protocol for attentional difficulties and heightened motor activity is based on the research presented by Sterman (1994, 2000) (observed excess theta and alpha and low SMR) and Lubar (1991, 1995, 1999) (theta/beta ratio).

It has been found that some people who have ADD may have a genetic alteration in the D4 dopamine receptors (Solanto et al. 2001:356). Frontal-striatal circuits that also involve the globus pallidus and the thalamic nuclei may be implicated in AD/HD. The frontal areas appear to be important in selection of appropriate stimuli, suppression of irrelevant stimuli and suppression of inappropriate actions. These are the three cardinal symptoms of ADD: inattention, distractibility and impulsivity. Theoretically, the same circuits may account for the observed increase in Theta seen in the EEG in children with ADD. As explained above, in ADD the striatum may exert too little inhibition on the globus pallidus. The globus pallidus then inhibits certain thalamic nuclei. These neurons become hyperpolarised and fire in a specific rhythm in the theta range. This activity is conveyed to the cortex and detected in the EEG (Thompson & Thompson 2003:117). Three groups are clear in Sterman's work:

- excess theta in the prefrontal cortex
- generalised excess of theta in frontal and central leads
- an excess of alpha activity at C3, C4 as well as in the frontal leads

Lubar identified EEG differences between children with AD/HD and those without AD/HD (the latter have higher theta/beta ratios) and a number of studies (Clarke et al. 2002a:1036-10044; Clarke, Barry, McCarthy & Selikowitz 2002b:276-285; Janzen, Graap, Stephanson, Marshall & Fitzsimmons 1995:65-82; Mann, Lubar, Zimmerman, Miller & Muenchen 1992:30-36; Monastra et al. 1999:424-233) have verified this. Interestingly, stimulants do not appear to significantly lower the theta/beta ratios but neurofeedback training has been shown to have this effect.

Attention resides mainly in the left hemisphere and motor activity in the right. Implications of this data are that neurofeedback training with patients presenting with attentional difficulties will be located at C3 (or Cz) and at C4 (or Cz) for those presenting with hyperactivity and impulsivity. Some studies show that training on T3 and T4 has the same effect (again left for attention and right for hyperactivity). It must be noted that all these sites are situated along the sensory motor strip and SMR (12-15Hz) will therefore be rewarded on the right side of the skull and normal beta (16-20Hz) will be rewarded on the left side.

Schematically the protocol could be plotted as shown in figure 70.



Figure 70 EEG neurofeedback protocol

Justification of the protocol is offered at three levels (Abarbanel 1999:312-313):

• Empirical: an elevated theta-beta (or theta-SMR) ratio correlates empirically with the presence of ADD symptoms, and a reduced theta-beta (or theta-SMR) ratio correlates empirically with the resolution of these symptoms.

• Theoretical: it can be suggested that in neurofeedback, the patient learns to exert neuromodulatory control over the circuitry system mediating the attentional process; long-term potentiation in the circuitry involved consolidates an optimisation of attentional processes. In terms of network theory it can be said that during NT the system is neuromodulated into an

attractor state, a stable point of equilibrium for the system. Other states functionally close to these will settle into their configurations.

 Neurophysiological: this involves the specific processes that are disordered in ADD and the processes mediating attentional phenomena. These include an understanding of oscillatory modes, event potentials, long-term potentiation (LTP), and neuromodulation (Abarbanel 1999:317).

Neurofeedback is based, for the most part, on two premises:

- increased theta activity does correspond to decreased attention to external stimuli (and less retention of material that the child is either reading or being taught).
- increasing SMR corresponds to a decrease in both hyperactivity and impulsivity.

Neurofeedback takes time to take effect but the effects appear to be long lasting. Often 40 or more sessions of at least 40 minutes are required. Lubar (1995) undertook a ten-year follow-up study, showing that results lasted (Thompson & Thompson 2003:118).

Neurofeedback sessions were offered as part of the ecosystemic approach on a daily basis for 20 minutes at at time.

2.4 Nutrition

The body and the brain are entirely made from molecules derived from food, air and water. The delicate balance between these elements is so fragile that a single molecule of toxin can fundamentally affect this balance. Any disturbance in this balance will have an effect on the brain. Holford (2003) believes that most of us do not reach our full mental health potential due to the fact that we do not achieve optimum nutrition for our mind. In this thesis, the notion that nutrition could radically affect the mental health of children and therefore their ability to learn efficiently and behave adaptively is discussed in chapter 4.

Professionals need to recognise that changes in nutrition and chemical imbalances possibly underlie the majority of mental health problems. As psychologists, we cannot use traditional psychotherapeutic approaches to address deficiencies in vitamins, essential fatty acids, minerals and other key brain nutrients. Chemistry directly affects the way the human organism feels, thinks and acts.

In a comprehensive nutritional approach, Holford (2003:8) outlines the following key tasks:

- balance glucose levels in the body
- supplement essential fatty acids
- supplement phospholipids
- add amino acids
- supplement vitamins and minerals

The chemical working of the brain is highly complex. A simple explanation of what happens would be that approximately 60 000 thoughts go through the mind daily. These thoughts are messages that are sent from a sending station and received at a receptor. The sending and receiving stations are constructed out of essential fats, phospholipids and amino acids. The neurotransmitter that carries the message is largely composed of amino acids. Turning an animo acid into a neurotransmitter is a delicate and complicated task. Enzymes in the brain that are dependent on vitamins and minerals are mainly responsible for the transition of amino acids into neurotransmitters.

The previous paragraph highlights the need that the brain has for raw materials in the form of good nutrition. While good nutrition is vital for healthy mental processes, the digestive system and its functioning is just as important. The digestive system contains 100 million neurons, and produces as many neurotransmitters as the brain. This system produces two-thirds of the body's serotonin. Parasites and dysbiosis in the digestive system will interfere with this delicate process.

Benson (2001:S21) proved that dips in blood sugar (hypoglycaemia), often brought about by diets high in sugars and simple carbohydrates, are directly associated with

poor attention, poor memory and aggressive behaviour. Another significant finding was the difference in IQ scores measured between children consuming complex carbohydrates and those that do not, with higher IQ scores linked to the intake of foods containing the complex carbohydrates (Benson 2001:S21), the so-called low glycemic index (GI) foods. Foods containing complex carbohydrates and which are higher in fibre are nowadays referred to as having a lower glycemic index (Iow GI) or glycemic load (GL) (Holford 2005:21).

These and other facts as described in chapter 4 need to be discussed with parents. Where possible, parents need to be encouraged to adhere to suggestions made about food containing a low glycemic index. Suggestions could be made concerning supplementation with vitamins, minerals, amino acids and essential fatty acids. Mostly, these should be taken in their natural foms by choosing healthy foods or alternatives when indicated and wherever possible.

3. Conclusion

This chapter outlined the ecosystemic approach used in the research design including modalities such as sound therapy, a neurodevelopmental programme, EEG neurofeedback and nutrition. They were selected to address possible causative aspects of attention and motor activity as revealed by the literature in the previous chapters. The modalities address attentional difficulties and heightened moter activity on the levels of neuroanatomy, neurochemistry and neurophysiology. Chapter 6 will describe the research methodology, and the results of the intervention will be discussed in chapter 7.
Chapter 6

Research Methodology

1. Introduction

The purpose of this chapter is to present the research methods used to conduct the empirical section of the research study. The discussion to follow outlines the basic design, the data collection method employed, intervention and the approach and techniques used to analyse the data.

2. Research Design

The design of the study is the plan or blueprint to be followed in order to answer the research objectives (McDaniel & Gates 2001:28). This section provides a discussion of the basic type of behavioural research used to obtain answers to the research questions or hypothesis. To select an appropriate design one should bear the following in mind: (a) the specific purpose that the design should accomplish; (b) criteria for deciding whether the design has a high probability of accomplishing that purpose; and (c) knowledge of the logic of the design so that the design can be modified to optimally accomplish the goal and avoid problems.

There are various designs to choose from. Rosnow and Rosenthal (1996:14) categorise them into three designs, namely descriptive, relational and experimental:

- Descriptive research refers to research where the goal of the research is to describe what is happening behaviourally (Rosnow & Rosenthal 1996:14)
- Relational research is when **two or more variables** or conditions are measured and related to one another (Rosnow & Rosenthal 1996:14)
- Experimental research seeks to obtain answers by manipulating a condition, that is by introducing changes into a situation; it seeks to manipulate the independent variable to establish conditions that will test the research hypothesis (Whitley 2002:184).

The current study can be classified as experimental research. It must be noted though, that the human organism can under no circumstances be viewed as an object, where all the variables can be manipulated and regulated to yield total experimental circumstances. Qualitative as well as quantitative methods were incorporated throughout the research. Other factors also influenced the decision to incorporate both research methodologies. Experimental research frequently strives to measure behavioural outputs (such as attention and performance) with standard evaluation tools that yield quantitative data. Other aspects, such as those derived from the lower brain areas (e.g., functioning of the vestibular system) are more difficult to measure guantitatively, and so qualitative measures were utilised.

3. Experimental Research

Two basic types of experimental research exist: true experiments and quasiexperiments. McBurney (1990:175) explains that in true experiments researchers have complete control over the experiment in terms of what, where, when and how. In a quasi-experiment the researcher lacks the degree of control and it is often necessary to select subjects from pre-existing groups.

3.1 True Experiments

True experimental design refers to a design where two groups are compared with one another and where one of the two groups received the intervention. A true experiment includes the random assignment of subjects to treatment conditions.

3.2 Quasi-experimental Design

The current research can be classified as quasi-experimental research study as the researcher used a pre-selected group in the form of a classroom setting in a private primary school. The specific primary school accommodates only children previously diagnosed with AD/HD by a medical professional (either a medical doctor, paediatrician or neurologist). The specific group of children were all in one classroom, which included both Grades, four and five. The total number of children included in the research equalled 12 (n = 12).

McBurney (1990:179) sets two requirements for a good experiment, 1) the existence of a control group or control conditions, 2) the randomisation of respondents to these groups. To answer to these requirements as set out by McBurney the respondents in the class were divided into two equal groups (n = 6) on a random basis providing that the two groups were equal in terms of age, gender and basic intellectual ability.

3.2.1 The one-group pretest-posttest design

Although the existence of a control group is a measure to reduce the threat to the validity of the results, no control group was available during the second part of the study. The second part of the study was conducted merely for ethical reasons as the experimental group received a valuable intervention, and the same was planned for the control group. However, the data were recorded as it was felt that valuable information could be obtained and possible long-term effects could be recorded. This is not an uncommon occurrence and McBurney (1990:179), refers to this type of design as "the one-group pretest-posttest design".

The one-group pretest-posttest design measures the behaviour of a single group of subjects both before and after treatment. Limitations exist in the form of threats to the validity of the experiment. **Validity** refers to the possibility that the effect seen in the results might be attributed to other reasons than the intervention (McBurney 1990:179).

These other reasons are referred to in research as the nonspecific treatment effects. The nonspecific treatment effects are changes brought about in respondents by all their experience in the research that do not include the treatment, such as contact with the examiner. (Whitley 2002:184). "Somebody showing interest in your behaviour may change it" (Breakwell, Hammond & Fife-Schaw 2000:86). This is called the Hawthorne effect.

The general research design is illustrated in figure 71.





4. Data Collection

As part of the discussion on the methodology, a description is given of the population and the measurement instrument used.

4.1 The Sample Population

Salkind (2000:86) describes a population as a group of potential respondents to whom you want to generalise the results of the study. One of the objectives of this study was to determine whether a neurodevelopment programme would be successful in school context. From the literature study presented, children of schoolgoing age represent the largest population diagnosed with AD/HD. For this reason, a private school specialising in children presenting with AD/HD was selected. A pre-selected group of children were included in the research (a specific classroom). The specific classroom had a combination of Grade four and Grade five pupils. The number of children in the classroom totalled twelve.

Table 7 Measurement tools

Point In Time	Stage A	Stage B	Stage C
	Pre-Test	Mid-Test	Post-Test
Qualitative			
Interview	1		✓
Parents Questionnaire	✓		✓
Teachers Questionnaire	✓		✓
HANDLE Screening	✓		✓
-			
Quantitative			
AIT	✓	1	✓
IVA	✓	1	✓
EEG	✓	✓	✓
Reading	✓	\checkmark	✓
Spelling	✓	✓	✓
Intellegence	✓		
Mathematics	✓	✓	✓
Visual Discrimination	~	1	✓
Cognitive Control Battery	~	✓	✓

4.2 The Measurement Instruments

In order to increase the scope of the research and the collectable data, both quantitative and qualitative measuring instruments were included at various stages in the research. An outline of these is presented in table 7. (If must be noted that none of these questionnaires are standardised for South African condions).

4.3 Interviews

Interviews were conducted with both the teacher and the parents of each respondent. During the interviews qualiatative data were collected to support the questionnaires. Information concerning the research was presented and permission to conduct the research was obtained.

Two sets of questionnaires were used in conjunction with the interviews and literature to triangulate the data. Breakwell et al. (2000:280) explains triangulation as referring to the credibility of the findings being enhanced by comparing data obtained from

different sources or from different investigators or different methods of collecting data. If such comparisons show that the findings hold, then one can have more confidence in the interpretations of the findings.

Point In Time	Stage A		Stage B		Stage C	
	Pre-Test		Mid-Test		Post-Test	
	Parent	Teacher	Parent	Teacher	Parent	Teacher
Biographic Information	✓					
Special Concern	✓	✓				
Speech and Language	✓	✓			✓	✓
Development	✓	✓				
Sensory Motor Development	✓	✓				
Emotional and Interpersonal	✓					
General Health	✓					
Candida Checklist	✓					
Biochemical Checklist	✓					
Round the Clock Observation	✓					
Temperature	✓	✓			✓	✓
Amen ADD Questionnaire	√	✓			✓	✓
Symptoms Check List	✓	✓				✓

Table 8

Questionnaires

Some of the questionnaires were obtained from the following sources, while the rest were compiled in order to abtain the necessary information:

Candida checklist (Crook 2000:71-73) Biochemical checklist (Holford 2003:158-168) The 24-Hour Day Observation (Walker 1999:186-190) Temperament (Thomas & Chess 1977) Amen ADD Questionnaire (Amen 2001:68-73)

An example of all the questionnaires combined could be viewed in appendix A (If must be noted that none of these questionnaires are standardised for South African condions).

4.4 HANDLE Screening

The HANDLE approach was briefly discussed in chapter 5 (section 2.2.1). A typical screening consists mostly of clinical observations. These observations focus on the underlying neurological subsystems discussed in Chapter 4, section 4 and include mainly the tactile, gestational and olfactory systems, as well as the vestibular system, muscle tone, proprioception, visual and auditory system, differentiation, lateralisation and interhemispheric integration. The interplay between all these systems is profiled as well as general health and nutritional aspects. The observations are used mainly to select activities for the individual neurodevelopmental programmes employed during the therapeutic stage of the research. The screening was conducted at the same time each day (early morning) by the same trained examiner during both the pre- and the posttests, and under the same circumstances.

4.5 Sound Therapy

Information on the auditory system was obtained from questions in the parent questionnaire pertaining medical history, including aspects such as the number of ear infections, grommets, allergies, age when speech was mastered, general speech and language development as well as a listening profile, according to the guidelines specifically for AIT as set out in Chapter 4 (section 4.10). The information obtained in the listening profile was generally used to determine the necessity for auditory integration training (AIT) as well as the filter settings to be used. The same examiner conducted the screening at the same time during the day at all three stages of testing.

4.6 The Integrated Visual and Auditory Continuous Performance Test (IVA)

The IVA is a comprehensive, computerised 13-minute continuous performance test (CPT) combining auditory and visual stimuli to measure objectively the triad of symptoms – inattention, impulsivity and hyperactivity usually associated with AD/HD. The IVA was primarily designed to assist in the diagnosis and quantification of the symptoms associated with AD/HD for clients aged five years to adults (Sandford & Turner 2000:ix-3). Continuous performance tests like the IVA have also been

extensively used in clinical research to evaluate the effects of treatments and interventions intended to improve attention or reaction time (Sandford & Turner 2000:ix-3). In this specific research study the IVA was utilised specifically for the latter purpose – to calculate the improvement after a period of intervention. The IVA was administered at the same time during the day, in the same room and by the same examiner during all three stages of testing.

Reliability refers to the consistency of test score performance for repeated testing by an individual under similar condition. A test-retest reliability study was conducted by the developers of the IVA (Sandford & Turner 2000) in conjunction with NOVA University. According to Sandford and Turner (2000:xv-4), the IVA was evaluated in terms of the stability of the test over time. The IVA was found to be a significantly stable measure of performance in many ways both globally and in terms of specific scales. Sensitivity of the IVA tested 92%. Specificity tested 90%, the positive prediction power tested at 89% and the negative prediction power tested at 93%.

Tinius (2002:452) however, raised questions about the IVA as neuropsychological measurement. He suggested that the effect of variables such as motivation and awareness of neuropsychological deficits on CPT performance needs to be investigated. The IVA was specifically selected as it links with the theory of Sternberg (see chapter 3 section 3.3.4). Sternberg focuses mainly on the mechanisms of attention such as signal detection, selective attention as well as divided attention. The IVA specifically measures these aspects of attention.

4.7 EEG Assessment

The literature study presented in Chapter 5 (section 2.3) points to the possible utilisation of EEG measurement to determine the presence of AD/HD as well as the employment of EEG technology in the form of neurofeedback as therapeutic intervention. Knowledge of a MINI-Q (mini quantitative EEG) was employed and sights across the skull, according to the international 10-20 system were used. The main sights used during interpretation were: Cz, C3, C4, T3, T4, Fz, F3, F4, P3 and P4. The data obtained were interpreted according to the information presented in

chapter 5 section 2.3.4 to determine the best location for neurofeedback and the specific protocol to be selected for each respondent. The MINI-Q assessment was conducted by the same examiner, in the same room at the same time of day during all three stages of testing. For the purpose of this research the impedance was kept <5 kohms in all combinations of leads with <1 kohm difference between leads (according to international research standards) and as explained in Thompson and Thompson (2003:65).

4.8 Academic Assessment

Basic scholastic assessments were used during all three stages of testing under the same conditions. The tests included:

- one minute sight reading (University of Pretoria)
- one minute mathematical calculation (plus, minus, multiplications and division) (University of Pretoria)
- a graded spelling test (University of Pretoria).

4.9 Intelligence

The Revised Senior South African Individual Scale (SSAIS-R) was used to conduct individual intellectual measurement at the outset of the research. The main purpose for the inclusion of such a measurement was to ensure that the two groups were equal in terms of intellectual ability.

4.10 Cognitive Control Battery (CCB)

The Cognitive Control Battery of Santostefano (1988) is intended for children and adolescents aged three to sixteen. The test can be used as a group assessment battery or individually, to serve a variety of clinical and research purposes. In this study only one test, the **Fruit Distraction Test** (FDT) was included as this test evaluates the ability of attention. The Fruit Distraction Test utilises one cognitive control, namely **field articulation**. Field articulation can be described as the manner in which an individual **attends selectively** to a particular stimulus while ignoring others (Santostefano 1988:3). Some individuals selectively withhold attention from



irrelevant information, thereby reducing interference when managing relevant information. Others attend to both relevant and irrelevant information and consequently, have difficulty managing the central task. The term "field articulation" is used to emphasise that the field of information is articulated into relevant and irrelevant parts and attended to selectively.

One method frequently used by psychologists to assess field articulation is the Stroop Word Interference Test (Stroop 1935:643-661). Santostefano (1988) found the original Stroop Test not applicable for some children as they became restless and often angry whilst taking the test. A task was therefore modelled after the Stroop Test that did not make use of printed words, but that nonetheless presented a task by means of which a child could reveal how compelling irrelevant information is characteristically managed. The result was the Fruit Distraction Test.

The Fruit Distraction Test consists of four cards, the first two being presented to obtain baseline information whereas the remaining two present increasing degrees of distraction or compelling irrelevant information that must be managed (Santostefano 1988:3). If children complete the last two cards as quickly as the first two cards and with few errors, they demonstrate the ability to selectively withhold attention from irrelevant information whilst directing attention to information relevant to the task at hand. The child who takes longer to name colours with Card 3 and Card 4 versus Card 1 and Card 2 and who recalls more peripheral figures after Card 3 is removed, tends not to withhold attention selectively from irrelevant information and is likely to be distracted from the central task. In addition to this broad interpretation, a distinction is made between the kinds of distraction represented by Card 3 versus Card 4. Card 3 has peripheral, irrelevant pictures provides a measure of the degree to which a child is vulnerable to the pull of external distractions (that is to irrelevant information in the immediate environment such as pictures on the wall, the sound of footsteps and so on). Card 4 displays contradictory colours and provides a measure of the degree to which a child is vulnerable to the pull of internal distractions (for instance private thoughts and fantasies as well as associated emotions) (Santostefano 1988:23).

The test interpretation includes six distractibility scores that are converted into tscores with a mean of 50. The scores are keyed so that higher t-scores represent developmentally more mature functioning; whereas the lower t-scores represent less mature functioning. The scores are graded as follows:

severe: t-scores of 35 and lower
moderately severe: t-scores between 36 and 40
borderline dysfunction: t-scores between 41 and 45
normal: t-scores between 46 and 55
above average functioning: t-scores between 56 and 65
hypermature functioning: t-scores of 66 and above

(Santostefano 1988:58).

The FDT was specifically selected in view of the data presented in chapter 2 and the theory of Levinson (1990) and Sternberg's (1999) conceptualisation of conscious attention, which includes selective attention. A further consideration for the inclusion of this test in the test battery was its association with the Stroop Test (1935) and the fact that the Stroop Test is a well-known applied test in the field. However, Santostefano's (1988) concern about the Stroop effects on younger children was taken into consideration and the FDT was selected to obtain qualitative data on selective attention.

5. The Intervention and Collection of the Pre- and Posttest

Results

The following section will focus on the procedures followed in order to prepare, conduct pretests, intervention provided and the final collection of data. Where possible, precise time frames are given.

5.1 Procedure

2004 Pre-reading and initial literature study.

February 2005:Initial contact with school.Presentation of proposed research to the selected school.



Identification of the classroom to be included in the research. Parent's evening to discuss proposed research. Information presented on the research design. Information presented on the intended intervention.

- March 2005: Group discussion with children included in the research. Pretest conducted (Stage A) at the school in individual settings. Parent and teacher questionnaires distributed. Initial short interview with parents and teacher.
- April 2005:Random selection to form two equal groups.Profiling according to the data obtained from the questionnaires
and measurement instruments.Protocol design for each respondent.
- April 2005: Intervention on daily basis with experimental group for eleven weeks with three therapists all of whom received previous training in the field of their specialty such as HANDLE, EEGneurofeedback and auditory integration training (AIT). The control group did not receive any intervention. Mostly children attended the therapy individually, where the rest of the class continued with the standard academic curriculum.
- June 2005: Data collection started (Stage B) with both the experimental group and the control group under similar conditions as in the pretest (Stage A).
- July 2005: Second leg of the research design started where both the experimental and the control group (now the second experimental group) received intervention. This section was included to provide the obligatory ethical intervention to the control group. The control group formed the second experimental group as data collected from their results could also provide valuable information. The first experimental group continued with

the intervention in order to obtain data on the possible long-term effect of the intervention. The intervention was conducted with both experimental groups daily for a period of eleven weeks with two therapists.

Nov 2005: Third and final test (C). Data collection with measurement instruments with both groups at school under similar conditions as in the pre- and midtests. Questionnaires to both parents and teacher.

January 2006: Data analysis began.

5.2 Intervention

A typical intervention programme is outlined here. It would however be impossible to provide a full account of the individual protocols presented to each child, as one of the main goals of this research is to establish an understanding that the symptoms presented could stem from various root causes. As far as possible, the intervention was designed with the specific child in mind. Most of the therapeutic techniques were provided on a daily. The neurodevelopmental programme was adjusted as development took place. Other therapies, such as sound therapy, were also conducted where the need was identified.

5.2.1 Nutritional suggestions

From the interview with the parents, information obtained from the biological background, medical history, candida questionnaire and biochemical checklist, some nutritional suggestions were made to parents. A general list is provided here, although each child received individual suggestions:

- If a child presented with past or present food or chemical allergies, the parents were advised to seek alternatives to the allergens (e.g. food and chemical substances such as soaps, cleaning fluids, etc.).
- If a child presented with recurrent otitis media, asthma, bronchitis, and any of the symptoms presented in chapter 4, section 7, parents were advised to

investigate the possibility of an allergy and to eliminate the allergen once detected.

- Children with a high candida score for the candida questionnaire, combined with some of the symptoms mentioned in chapter 4 section 7.8, were advised to include a good probiotic as part of their supplementation regime and to follow a low carbohydrate and low sugar diet.
- All children were encouraged to drink more water.
- Emphasis was placed on the value of good nutrition, a well-balanced breakfast with nutritional snacks for break and lunchtime. Protein was included in cases where glucose fluctuations were suspected and information on a low GI diet was presented to parents.
- Suggestions were made with regards to supplementations, specifically omega 3 and 6, zinc, calcium, magnesium and vitamins B 6 and 12.
- Information was drawn from Holford's biochemical questionnaire to advise parents.
- In all cases, parents were reminded that the therapists were not qualified in the field of nutrition and they were encouraged to consult with a dietician to ensure the best possible diet and health suggestions for their children.

5.2.2 Neurodevelopmental programme

From the data obtained in the developmental history of the respondents, interviews with both parents and the teacher, HANDLE screening, auditory screening, presenting symptoms and clinical observation, individual neurodevelopmental profiles were obtained. A neurodevelopmental programme for each child was compiled and presented daily to the experimental group; this lasted approximately fifteen to twenty minutes per session. The principles of a good neurodevelopmental programme were followed, including first strengthening the underlying, supportive subsystems, observing the natural developmental sequence and encouraging gentle enhancement so as to reduce any possible physiological stress. A typical programme included:

- · activities to suppress aberrant primitive reflexes if any were still present
- if physiological stress were detected, calming activities were included to allow the nervous system to return to the parasympathetic mode

- if sensitivities of the tactile, gustatory and olfactory systems were noted, these were addressed first
- emphasis was placed on the vestibular system. If vestibular irregularities were noted, stimulation was provided with slow movement on all three planes of the three semi-circular structures in the vestibular system. Particular attention was given to the avoidance of physiological stress whilst doing these activities
- if muscle tone and proprioception were noted, specific activities were included to address these
- in general, the lower systems were addressed first rather than the higher cognitive systems as illustrated in the chart (chapter 4, section 5, figure 43).

5.2.3 EEG neurofeedback

From the data obtained concerning the presenting symptoms, interviews and questionnaires (specifically the Amen ADD Checklist), protocols were designed for each child. A typical protocol included the following:

- children presenting with predominant inattention Cz or C3 were chosen for EEG neurofeedback; during these sessions (twenty minutes every day) theta (3-7Hz) and high beta (26-34Hz) were inhibited while beta (16-20Hz) was rewarded
- children presenting predominantly with hyperactivity and impulsivity Cz or C4 were chosen for EEG neurofeedback; during these sessions (twenty minutes every day) theta (3-7Hz) and high beta (26-34Hz) were inhibited while SMR (12-15Hz) was rewarded
- ProComp+ with Biograph Software from ThoughTechnology was the instrumentation used in this research
- the impedance was kept at <5 kohms in all combinations of leads with <1 kohm difference between leads. This was done according to international research standards as explained in Thompson and Thompson (2003:65).

5.2.4 Sound therapy

All those candidates who presented with auditory integration difficulties, a history of recurrent otitis media, hyperacuity, disorganisation of hearing between the two ears,

attentional difficulties and hyperactivity were given an AIT programme. The general protocol was that:

- AIT was presented towards the final stages of the intervention as vestibular strengthening was needed first, in order to support the auditory system
- AIT was presented towards the final stages as this would have ensured the elimination of possible allergens and thus produced a health, clear auditory channel for optimal benefit
- AIT is conducted over a ten-day period for 30 minutes twice a day, thus twenty sessions in total; during these sessions dynamic music is played through a specific instrument (in this case the Earducator) that covers the range from 125Hz to 8000Hz
- music is presented no louder than 80dB
- filter settings were chosen according to the listening profile obtained at the onset of AIT and after 10 sessions
- at midpoint, the sound in most cases was reduced in the left ear in order to stimulate the right ear and therefore the left hemisphere that contains the major language centres.

6. Analysis of the Results

The following discussion focuses on the different statistical techniques used during the analysis of the data. Both descriptive and inferential statistics are used to examine the research questions and hypotheses.

6.1 Descriptive Research

Descriptive statistics are called such because they describe the general characteristics of a set or distribution of scores (Salkind 2000:150). Simple descriptive statistics calculated for the study include frequencies, means and standard deviations.

- frequencies are the number of times a response has occurred (Salkind 2000:150)
- a mean is the sum of a set of scores divided by the number of scores
- a standard deviation measures variability around the mean (Salkind 2000:154).

6.2 Inferential Statistics

To confirm that the independent variables did have an effect on the dependent variable, the research used inferential statistics (Shaughnessy, Zechmeister & Zechmeister 2003:218). An integral part of the interpretation of any inferential statistic is the concept of statistical significance.

6.2.1 Statistical significance

Statistical significance tests begin with the supposition that the null hypothesis is correct.

The null hypothesis states that there is no difference between the improvement shown by the experimental group and the control groups. The alternative hypothesis states that the experimental groups improved more than the control group.

If it is assumed that the null hypothesis is valid, the results from the study seem unlikely (normally at the 0.05 p-value). The researcher then rejects the null hypothesis and concludes that there is support for the alternative; in other words, the treatment effect exists (Leong & Austin 1996:211).

Cohen (1994) mentions two common misconceptions about hypothesis testing:

- if the p-value is 0.05, it does not point to the probability that the null hypothesis is true, but rather to the probability of obtaining the results (from the study) given the fact that the null hypothesis is true. It is not proof of the truth or falsity of the null hypothesis or research (alternative) hypothesis
- if the results were significant at the 0.05 level in a study, this indicates that the results would be significant in most studies should the study be replicated. The p-value says nothing about the chances of replicating the results

The most frequently used levels of statistical significance are 0,05 and 0,01. For the purpose of this study, a level of 0,05 is considered adequate.

For this study the null hypotheses is stated as follows:

Hypothesis 1:

H0: Disturbances in attention will not improve after an ecosystemic intervention.

H1: Disturbances in attention will improve after an ecosystemic intervention.

Hypothesis 2:

H0: Disturbances in motor activity will not improve after an ecosystemic intervention.

H1: Disturbances in motor activity will improve after an ecosystemic intervention.

Hypothesis 3:

H0: Academic performance will not improve after an ecosystemic intervention.

H1: Academic performance will not improve after an ecosystemic intervention.

6.2.2 Effect size/ practical significance

Leong and Austin (1996:211) mention that Cohen (1994) and others have emphasised the importance of extending research beyond the significant/nonsignificant dichotomy. They feel that it is important not to limit all research to studies whose results hinge on a significant test. They add that a valuable contribution to theory should not be hindered by the pervading acceptance of the p <0.05 requirements.

Strict adherence to the 0.05 value provides an arbitrary cut-off that categorises potentially important findings as not significant and therefore as nonfindings. Rosnow and Rosenthal (1996:251) argue that statistical significance only provides half the picture. They claim that the next step should be to evaluate that size of the effect, interpreting the practical significance of the observed difference.

The effect size implies something very different from the p-value. A result that is statistically significant is not necessarily particularly important as judged by the magnitude of the effect; highly significant results should therefore not automatically be interpreted as reflecting large effects (Rosnow & Rosenthal 1996:276). One of the main methods of establishing effects sizes is to calculate **Cohen's d**. Effect size (Cohen's d) can be interpreted as:

0.2 = small effect size

0.5 = medium effect size

0.8 = large effect size (Cohen 1988:25)

7. Parametric vs. Non-parametric Tests

A parametric test is a test that requires a parametric assumption, such as normality. A non-parametric test does not rely on parametric assumptions like normality. Non-parametric tests are therefore used to analyse data that do not fit a normal distribution. They are based on the rank order of measurements rather than their values (Ward 1999; http://www.bamc.amedd.army.mil/DCl/nonpara/ppframe.htm).

Non-parametric tests may be, and often are, more powerful in detecting population differences when certain assumptions are not satisfied. All tests involving ranked data, that is data that can be put in order, are non-parametric.

Choosing between a parametric and non-parametric test for large samples is relatively easy: Tests exist to determine if assumptions of normality hold. As the sample in this particular investigation is relatively small (n=12), the researcher was presented with a challenge. It is difficult to tell if the data are normally distributed, and a further complication is that the non-parametric tests are not powerful and the parametric tests are not robust. Therefore both parametric tests and their non-parametric counterparts were conducted.

7.1 The Wilcoxon Signed Ranks Test

The Wilcoxon Signed Ranks test is designed to test a hypothesis about the location (median) of a population distribution. The Wilcoxon Signed Ranks test does not require the assumption that the population is normally distributed. It often involves the use of matched pairs, for example, before and after data, in which case it tests for a median difference of zero. This test's parametric counterpart is the t-test for dependent measures (http://www.cas.lancs.ac.uk/glossary_v1.1/nonparam.html).

7.2 Mann-Whitney U Test

The parametric version of the independent t-test is the Mann-Whitney U test. The Mann-Whitney U test also assumes homogeneity of variance with respect to the underlying population distributions, as does the parametric t-test for two independent samples. However Sheskin (2004:424) explains that there is some empirical evidence to suggest that the sampling distribution for the Mann-Whitney U test is not as affected by violations of the homogeneity of variance assumptions, as is the sample distribution of the t-test.

7.3 Spearman Correlation Analysis

Pearson correlation calculations are based on the assumption that both X and Y values are sampled from populations that follow a normal (Gaussian) distribution at least approximately; although with large samples, this assumption is not too important. Alternatively, the non-parametric Spearman correlation is based on ranking the two variables, and so makes no assumption about the distribution of the values (http://www-micro.msb.le.ac.uk/2060/2060-3.html).

8. Statistical Computer Package

All statistical analyses in the present study were computed using the Statistical Package for the Social Sciences (SPSS) statistical package for Windows version 11.1 (SPSS 2001).

9. Conclusion

The following chapter presents the research data and the analysis of the data according to the methodology explained in this chapter.

List of research project topics and materials

Chapter 7

Research Results

1. Introduction

The purpose of this chapter is to present the findings from the empirical research. The chapter blends qualitative and quantitative findings to present an overall picture and draw conclusions on the effectiveness of an ecosystemic intervention approach to addressing attentional difficulties and heightened motor activity.

The first section of the chapter presents a detailed description of the population used in the study. This description includes variables such as age and gender, a description of the family structure and history, development history, medical history, language and speech development, sensorimotor development, as well as presenting symptoms, and the like.

The basic research hypothesis, that children in the experimental group improved significantly more than the control group in terms of attention and hyperactivity, is tested in the second section. Better academic performance is the result. The long-term effect is also investigated when the control group also receives the intervention (the second experimental group). These results are then compared with the original experimental group, which received the intervention for twice as long as the second experimental group.

The last section of the chapter examines some additional general outcomes such as scholastic performance.

2. Sample Description

The sample consisted of twelve children in total (n=12): six in the experimental group and six in the control group. Qualitative results are presented as frequencies as percentages could become misleading due to the small sample numbers. Qualitative aspects are presented in terms of reference to individual respondents to be able to draw some qualitative conclusions. For this purpose the children are numbered 1 -

12. Children 1-6 represent the experimental group and children 7-12 represent the control group and second experimental group. All children retain their initial number allocation throughout the research. These quantitative aspects will be discussed under the heading "comment" and represent only general observations made.

2.1 Demographic Description

Both the experimental and control groups show similar profiles in terms of gender and age.





As is seen in figure 72, both groups consist mostly of boys (five per group), with only one girl per group. The average age of the children in both groups are given in table 9. The youngest children in both groups are 9.5 years old and the average ages are fairly close at 10.37 for the experimental group and 10.40 for the control group.

	Table 9						
Descriptive information on age (n = 12)							
non- N Minimum Maximum Mean							
netri	Experimental Group	6	9.5	11.58	10.37	_	

6

9.5

11.42

10.40

Std. Deviation

0.730

0.742

significant differences in mean age indicates that there is no significant difference between the two groups (z= -0.24; p = 0.809) in terms of age and therefore there is no need to control for age in the rest of the analysis. Both groups will be affected equally by age because the rule for significance (p=0.05) was not met.

2.2 Family Structure

Control Group

А

parametri

c test for

Information was obtained about aspects such as family structure, possible adoptions, foster care, number of children in the family and placement of child in family. The information that was obtained is presented in the section below.





(n = 11) Most families are still intact (4 in each group), or parents are remarried (2 in experimental group and 1 in control). All

children therefore have two parents at home. No data were presented for one of the children in the control group.

Further details about the family units include the following:

- There is one adopted child in the total sample (respondent 7 girl in control group).
- Family units tend to be small, with only one or two children per family; only one family had three children; five of the twelve families have only one child.
- Of the six families that have more than one child, half are firstborn children.
- Hereditary illnesses are seldom present; only one family mentioned a history of depression. This is however hearsay information provided by the families and possibly, therefore not a true reflection as hereditary illness might be a sensitive subject.

2.3 Historical Background

Information was obtained about the background aspects that could have an effect on general health.

2.3.1 Contact with potentially harmful substances

- Two fathers (respondent no 2 and 5) of children in the experimental group were in contact with heavy metals or chemicals before or during the pregnancy or in their children's early childhood.
- None of the families installed new carpets in their homes during pregnancy but renovations and painting did occur: two families in the experimental group (respondent 2 and 5) and one in the control group renovated and painted their homes (respondent 8).
- Comment: both respondents 2 and 5 were thus possibly in contact with heavy metals and or harmful chemicals.

2.3.2 Mother's pregnancy history

Most (four) of the children in the experimental group were planned while most (four) of the children in the control group were unplanned. (Respondents 2, 3, 7, 8, 9, and 12 were unplanned).



Figure 74 Planned pregnancies (n = 12)

Other factors concerning the pregnancy were:

- Most expectant mothers were generally healthy; one mother had pneumonia and three had water retention problems during pregnancy; one mother mentioned hypertension; none of the mothers were bedridden during pregnancy.
- While physical problems were rare, many of the mothers suffered emotional stress and depression, especially in the control group (five in the control group [7, 8, 9, 11 and 12], one in the experimental group [6]).
 Comment: interestingly enough, most of the mothers who had an unplanned pregnancy also presented with some signs of emotional stress or depression. These are the mothers of respondents 7, 8, 9 and 12.
- Generally, the mothers did not smoke or use alcohol; only one mother (number 10) from the control group smoked a little during the beginning of pregnancy and another consumed a little alcohol; exposure to passive smoke was common, however.

	Ν	Minimum	Maximum	Mean	Std. Deviation
Experimental Group	6	22	36	30.6	5.35
Control Group	6	19	29	25.8	3.6

Table 10 Age of mothers (n = 12)

Mothers in the experimental group were slightly older at the time of birth of their children than mothers from the control group, as is seen in table 10. The difference between the two groups is not statistically significant at the 95% level of confidence (p=0.096), however, this would be significant at the 90% level and could indicate that the mothers in the experimental groups were slightly older than those in the control group.

Comment: The nineteen-year-old mother was the biological mother of the adopted girl (respondent 7) in the control group. Because the girl was adopted, no prenatal information is available, such as general health of mother, alcohol and drug use, exposure to toxic substances, emotional tension, and so on. The mother of respondent 2 was 36 years old at the time of birth and the mothers of respondents 5 and 6 were 34 and 35 respectively.

2.3.3 Birth history

Pregnancies generally lasted between 38 and 40 weeks.

Table 11 Average birth weight (n = 11)

	N	Minimum	Maximum	Mean	Std. Deviation
Experimental Group	5	3	3.9	3.55	0.406
Control Group	6	2.2	4.10	3.26	0.672

The average birth weight of babies in the control group is slightly lower than that of babies in the experimental group (experimental group average 3.55 kg and control 3.26 kg) group. The two groups are not statistically significantly different in terms of birth weight as the p-value of 0.420 does not fall within the p > 0.05 rule.

Comment: The lower average birth weight of the control group may be due to the very low birth weight of 2.2kg for one respondent. This is the same baby that was adopted and had the very young mother at the time of the birth (respondent 7). Respondent 9 weighed 4,10kg at birth.

There were slightly more caesarean deliveries (4) than normal deliveries (2), in the control group.



Type of delivery (n = 12)

Three of the natural births (2 in experimental group and 1 in the control group) were longer deliveries and drugs were provided. Instruments were used in one of the longer births (respondent 3). The reported shorter deliveries did not involve any drugs or instruments.

Caesareans were mostly emergency (respondents 3, 8 and 9) procedures and were often performed after they had tried to induce labour. Some mothers received full anaesthesia while others only received epidurals, irrespective of whether it was an elective or emergency procedure.

Some problems experienced during birth include:

- One baby in the experimental group had anoxia (respondent 2).
- One baby experienced foetal distress in the control group (respondent 9) was reported.
- Two babies from the experimental group (respondents 4 and 5) were born with the umbilical cord around their neck.
- Three babies were in ICU (respondents 2, 8 and 12).
- One baby had jaundice (respondent 6 in the experimental group).

Comment: Respondent 2 experienced anoxia and was in ICU. Respondent 9 weighed 4,10kg, experienced foetal distress, and was delivered by emergency caesarean section.

2.3.4 Early feeding

Four of the six children in each of the groups were breastfed for a period of 1 to 7 months. One child in the experimental group (respondent 2) was breastfed for 36 months.

Solid foods were introduced as early as two months in one instance in the experimental group (respondent 4), as this baby was breastfed for only one month. On average most babies received solid foods at around four months old. There were no adverse reactions to the solid foods reported.

Three children were reported to have sucking problems. These included respondent 4 who was breastfed for one month, respondent 6 who was breastfed for 5 months and respondent 8.

2.3.5 Developmental history and general health

As young babies, all of the children in the control group were described as calm. However the experimental group had only three calm babies and one colicky baby (respondent 4), one bad sleeper (respondent 2) and one baby that was both restless and a bad sleeper (respondent 6).



Comment: these children are all in the experimental group. Respondents 4 and 6 could have presented with colic due to the difficulty with sucking. Two children experienced meningitis (respondents 1 and 9) and respondent 10 presented with rheumatic fever.

All babies in the control group received their recommended inoculations. Two of the children in the experimental group (respondents 2 and 3) did not receive all inoculations. None of the babies had any adverse reaction towards the inoculations.

2.3.6 Allergies

Three children (respondents 1, 2 and 6) from the experimental group and four from the control group (respondents 7, 8, 10, and 11) presented with allergies. Dairy, pollen and house dust were the most commonly reported allergens, but some were also allergic to gluten, egg, maize and animal dander.

Generally, most children received no treatment for allergies. When children are allergic to foods these are in some cases simply withheld from their diets. The one child who is allergic to bee stings receives regular injections for desensitisation.

2.3.7 Accidents and illnesses

The number of serious accidents, traumas, operations and so on that children experienced were also recorded to provide a full history of their health.

The most common problem reported was recurrent otitis media. Three children in the experimental group (respondents 2, 5 and 6) and four in control group (respondents 7, 8, 9 and 10) presented with recurrent otitis media. Some of these children had to have grommets inserted (respondents 6, 7, 8, 9, and 10). Respondent 6 received four sets of grommets and respondent 7 had three sets.

Comment: Four of the seven children (33%) that presented with allergies had recurrent otitis media and grommets. These are respondents 6, 7, 8 and 10. This information is significant in view of the information presented about the auditory system and the vestibular system and the role of these systems in movement and

sensorimotor development, as well as language development. This is significant when the connection between movement and general development is taken into consideration.

When asked specific questions about upper respiratory problems such as sinus and bronchitis, the parents indicated that four of the six children in both groups had some respiratory problem. This constitutes 67% of the children. Only two children (10 and 12), both in the control group, reported further physical injuries such as broken bones. One child, from the experimental group was treated for stress.

Medication and supplements

Only one child used medication during the research (respondent 6). Medication taken included Ritalin and Risperdal. This respondent was also the only child in the experimental group that took some supplementation. Two children in the control group were taking supplements, although they are not on any medication and supplements were given sporadically. While most of the children are currently generally healthy, half of the children did receive antibiotics regularly when they were younger. Probiotics were only given along with the antibiotics in three instances.

2.4 Developmental Background



2.4.1 Speech and language development

Figure 76 Rate of speech development



Speech development was normal for almost all children (5) in both groups. Orthodontic treatment was indicated for only one child from each group, both of whom were currently wearing braces (respondents 1 and 9).

Language development was less normal than speech development, especially in the control group (number 8, 9, 11 and 12). The language development of four of the six children in the experimental group was normal while only two (respondents 2 and 4) of the control group developed normally, the rest were slightly late. Respondent 2 of the experimental group, was a severely late developer.



Figure 77

Rate of language development

Comment: two of the children that developed late in terms of language also had grommets (respondent 8 and 9).

2.4.2 Motor and sensory development

Parents were asked to give their opinion of their children's rate of motor development on a scale ranging between early to severely delayed. The results are presented below.



Figure 78 Rate of motor development

Most parents felt that their children developed normally with regards to motor and sensory aspects. Two children from the experimental (respondents 4 and 6) and one (respondent 11) from the control group did however develop slightly late.

There are only two children (number 10 and 12) both from the control group who are reported to be clumsy and only one child had potty training problems (respondent 7).

While motor development was relatively normal, seven of the children (respondents 3, 4, 5, 7, 9, 11 and 12) have some kind of vision problems.

All the respondents (n=12) are right handed.

Problems with response to height and related factors are present amongst three (respondents 3, 6, 10,) of the children. Often when they have a problem with one of the aspects they also have problems with other height related factors. Merry-go-rounds seem to be the most common problem area.

Table 12

Vertigo-related problems

	Experimental Number "yes" have a problem	Control Number "yes" have a problem
Heights	2	1
Escalators	0	1
Elevators/Lifts	2	2
Climbing a tree	0	0
Flying	1	0
Swing	1	1
Slide	0	0
Merry-go-round	2	3

2.5 Intellectual Components

All the respondents were evaluated in terms of intellectual ability at the onset of the research. This was mainly done to ensure that the groups were equal in terms of intellectual ability. The intellectual abilities of the experimental group and the control group are presented in tables 13 and 14.

					Std.
	N	Minimum	Maximum	Mean	Deviation
Verbal	6	76	109	91.5	11.203
Nonverbal	6	85	124	107.7	13.619
Global	6	85	108	99.0	10.583

Table 13IQ measurements of experimental group

The experimental group as a whole presents with average global intellectual ability (99) as well as an average verbal ability (91). Nonverbal ability presents to fall within the above average scale (107.7). There seems to be a large difference between the

minimum (76 and 85) and maximum (109 and 124) scores of the individual respondents in the group on both verbal and nonverbal scale.

Table 14IQ measurements of control group

					Std.
	Ν	Minimum	Maximum	Mean	Deviation
Verbal	6	59	103	83.8	17.128
Nonverbal	6	94	136	112.7	16.046
Global	6	76	117	96.2	17.702

The control group as a whole presents within average global intellectual ability (96.2). The group's verbal scale falls within the below average (83.8) scale and the nonverbal scale falls within the above average scale (112.7). There seems to be a large difference between the minimum (59 and 94) and maximum (103 and 136) scores of the individual respondents in the group on both verbal and nonverbal scale.

On average the experimental group has a higher verbal and global intelligence while the control group have a slightly higher nonverbal intelligence. The difference between the groups is however not statistically significant, as can be seen from the table below.

Table 15

Non-parametric test for significant differences in intelligence between the control and experimental groups

	Wilcoxon W	z	Significance
Verbal	34.5	(0.722)	0.470
Nonverbal	37.5	(0.241)	0.810
Global	37	(0.321)	0.748

Table 15 shows where the non-parametric test for differences was calculated. No significant differences (no significant values of 0.05 or below) were found. As the two groups are on the whole, equal in terms of intelligence there is no need to control for this variable that could have possibly influenced the results of the tests.

2.6 General Observation

A 24-hour observation questionnaire was completed in order to identify possible significant information. The results are presented below:

Bedtime

Most children have no particular bedtime ritual.

All children sleep alone and none have problems with bed-wetting and frequent thirst during the night. Snoring and teeth grinding is not really a problem, as only two children (respondents 1 and 11) were reported to snore sometimes and three (respondents 2, 6 and 10) grind their teeth at night.

Children in the experimental group are generally quiet, restful sleepers who go to bed easily and wake up easily. Only one gets night terror (respondent 11) on a full bladder only. One child (respondent 6) is a restless sleeper and one is a heavy sleeper (respondent 4).

Three of the children in the control group (respondents 10, 11 and 12) are restless heavy sleepers, and one additional child, while a quiet sleeper, is also a heavy sleeper (respondent 9).

Morning

In the morning the children do not generally appear tired and sweaty (only one child in the control group). Two of the children in the experimental group (respondents 4 and 5) and one in the control (respondent 8) group appear to be affected by mood and energy levels in the mornings.
Afternoon

Half of the children, from both groups, get irritable just before lunchtime. These are respondents 1, 2, 5, 7, 11 and 12.

Late afternoon and evening

During the late afternoon children generally spend between one to three hours watching television. Children from the experimental groups spend slightly more time in front of the TV (50% watch three hours while the control group mostly watch only two hours of TV a day). The respondents that watched the most TV were number 1, 2, and 3.

Three children in the experimental group and two from the control group do not drink any caffeinated drinks during the afternoon. Respondent 2 drinks at least three caffeinated drinks, respondents 3, 7, 8, 10 drink only one, respondents 4 and 12 drink at least two. Most children have some water or juice in the evening. This is usually only one or two glasses, although two children from the experimental group and one from the control group do have three glasses a night.

Comment: The experimental groups seem to watch more TV and drink more caffeinated drinks.

A tantrum in the afternoon is rare, and only one child from the experimental group was reported to present with tantrums (respondent 1). This is the same child that watches almost three hours of television per day and consumes caffeinated drinks.

2.7 Symptoms and Previous Diagnosis

Parents were presented with a list of problems and conditions and asked to indicate those that their children have been diagnosed with or that they, as parents, suspect them to have. Since a professional has previously diagnosed all the children involved with AD/HD, this aspect was not included.

The list of problems and conditions indicated by the parents of children in the experimental and control groups are given below in table 16

-	Experimental	Control
Dyslexia	2	2
Dyspraxia	2	1
Depression	1	1
Low muscle tone	3	4
Visual perceptual problems	4	3
Auditory perceptual problems	2	1
Memory disorder	2	2
Epilepsy	2	1
Gifted	1	1
Learning disability	2	2
Conduct disorder	1	1
Tourette's syndrome		
Autism		
Asperger's syndrome		
Cerebral palsy		
Vision problems		
Hearing loss	2	3
Developmental delay	1	
Speech and articulation problems		
Problems with receptive and expressive language		
Brain injury	1	1
Obsessive compulsive behaviour		

Table 16Presenting symptoms and previous diagnoses

The most frequent aspects indicated by parents were low muscle tone and visual perceptual problems, followed by hearing loss, dyslexia memory difficulties and learning disabilities.

2.8 Biochemical Aspects

2.8.1 Candida checklist

A general questionnaire to investigate the possibility of candida (Crook 2000:71-73) was included. The questionnaire provides an indication of the possibility of candida according to the number obtained by calculating the questions ticked.

Table 17Average score on the Candida Checklist (n = 11)

	N	Minimum	Maximum	Mean	Std. Deviation
Experimental group	5	80	190	116	46.15
Control group	6	30	160	86.7	43.2

General interpretation of the candida checklist:

Yeast possibly plays a role in causing health problems in children with scores of 60 or more.

Yeast probably plays a role in causing health problems in children with scores of 100 or more.

Yeast almost certainly plays a role in causing health problems in children with scores of 140 or more.

Generally speaking, both groups mean scores (116 and 86.7) indicate that both groups may be affected by yeast.

Comments: four children that experienced allergies and recurrent otitis media (including three of them that had grommets inserted) also present with high candida scores. These are respondents 2, 6, 7 and 8.

The experimental group has a higher score on the candida checklist than the control. The difference is however not statistically significant as is seen from the nonparametric t-test for independent measures that shows a p-value of 0.232. Significance is indicated by a p-value of 0.05 or less as indicated in the table 18.



One respondent in the experimental group did not supply any information regarding this aspect.

Table 18

Mann-Whitney U test for differences in independent means

Mann-Whitney	Z	Significance
8.5	-1.19	0.232

2.8.2 Biochemical checklist

Another test used to determine the extent of problems that children face is the Biochemical Checklist (Holford 2002:158-168).

Firstly the scores on each section are given (based on the number of problems per section) and the scores of both groups are compared using a Mann-Whitney U test for differences in independent means. Thereafter the detailed view of significant sections is presented.

Table 19

Mean scores of numbers of problems per section and Mann-Whitney test for differences

		Experimental	Control	p-value
Niacin, pyridoxine, folic acid deficiency	1	1.33	1	0.658
EFA Deficiencies or imbalances	2	2.50	2	0.504
Heavy metal toxicity	3	2.67	1.5	0.515
Pyroluria and porphyria	4	0.83	0.75	0.908
Histamine imbalance	5	2.17	0.5	0.184
Detoxification overload and inflammation	6	1.33	1.5	0.738
Blood sugar problems	7	3.33	3.75	0.827
Adrenal imbalance	8	5.33	3.25	0.449
Food and chemical allergies and intolerances	9	1.50	2.5	0.228
Under-or over active Thyroid	10	1.33	2	0.661
Serotonin imbalance	11	1.00	1	0.645
Acetylcholine imbalance	12	1.17	1.5	0.376

Even though mean scores seem to differ in terms of section 5 (the experimental group obtained a score of 2.17 as opposed to the score of 0.5 for the control group), statistically, the two groups could be considered biochemically equal as none of the p-values are below 0.05. There is no significant difference between the two groups on this aspect.

Comment: Some of the sections were scored more frequently (by both the groups) than other sections. These include the section on blood sugar irregularities and adrenal imbalance. Respondent 2 indicated possible biochemical imbalances concerning heavy metals, histamine imbalance, toxin overload and inflammation, blood sugar problems and adrenal imbalance. Respondent 6 indicated possible biochemical imbalances, specifically an EFA deficiency or imbalance, heavy metal toxicity and adrenal imbalance. The scores that he obtained indicated a possible overactive adrenal system. Both of these children also obtained high candida scores on the candida checklist. Information worth mentioning is that three other respondents almost qualified for a biological imbalance because of a blood sugar imbalance (respondents 3, 7 and 8). Generally speaking, blood sugar imbalance and adrenal imbalance seem to be the overall biochemical indicators in both groups. For the purpose of possible significance, the subitems of the two sections are given below, together with the frequencies.

Table 20

Blood sugar imbalance

Section 7 Blood sugar imbalance	Experimental	Control
Crave sweet foods	2	4
Crave stimulant foods such as tea, coffee, cola and		
chocolates	1	2
Difficulty concentrating	6	4
Palpitations or blackouts		
Fainting or dizziness or trembling		
Excessive or night sweats		
Excessive thirst	1	



Frequent mood swings	2	
Forgetfulness and confusion		2
Tendency to depression	2	
Anxiety and irritability	2	
Feeling weak		
Aggressive outbursts and crying spells	1	1
Craving for sweets or stimulants	1	2
Drowsiness after meals		
Chronic fatigue	2	

Table 21

Adrenal imbalance

Section 8 Adrenal imbalance	Experimental	Control
Excessive levels of adrenalin or cortisol		
Irritability	2	1
Nervousness or anxiety	2	
Extreme fears		
Raised blood pressure		
Rapid or irregular heart beat	1	
Insomnia	1	
Cold hands and feet		
Excessive sweating		1
Teeth grinding	1	1
Headaches or migraines	2	
Muscle tension	1	
Restlessness	1	
Seeing or hearing things	1	
Adrenal Insufficiency		
Depression	2	
Difficulty concentrating	4	3
Short attention span	4	4
Lack of drive/motivation	4	1
Rarely initiates or completes tasks	4	2
Can't deal with stress	1	

Socially withdrawn		
Frequently tired	1	

The symptoms indicated in both sections are significant in the context of this research. In section 7 (blood sugar imbalance) ten children presented with concentration difficulties and in section 8 (adrenal imbalance) seven children struggled with concentration, eight reported a short attention span and six rarely initiated or completed tasks. This information links positively with the hypothesis that symptoms of AD/HD could be linked to biochemical factors and nutrition.

2.9 Conclusion

The following general information was obtained in terms of demographic description, background information, general health and development:

- All the children in both groups presented with attentional difficulties and/or heightened motor activity, as diagnosed with AD/HD by a medical professional (either a medical doctor, psychologist, psychiatrist or neurologist).
- The most frequent symptoms indicated by parents were low muscle tone and visual perceptual problems, followed by hearing loss, dyslexia memory difficulties and learning disabilities.
- All the children were in a combined Grade 4 Grade 5 classroom (n=12); the whole class formed part of the investigation and were either in the experimental or in the control group.
- A non-parametric test for significant differences in mean age indicates that there is no significant difference between the two groups (z= -0.24; p = 0.809) in terms of age and therefore there is no need to control for age in the rest of the analysis; both groups are affected equally by age because the rule for significance (p=0.05) was not met.
- Mothers in the experimental group were slightly older at the time of birth of their children than mothers from the control group; the difference between the two groups is not statistically significant at the 95% level of confidence (p=0.096). However this difference is significant at the 90% level and indicates

that the mothers in the experimental groups are slightly older than those in the control group.

- The average birth weight of babies in the control group is slightly lower than that of babies in the experimental group (experimental group average 3.55 kg and control 3.26 kg); the two groups are not statistically significantly different in terms of birth weight as the p-value of 0.420 does not fall within the p > 0.05 rule.
- On average the experimental group has a higher verbal and global intelligence while the control group has a slightly higher nonverbal intelligence; however, the difference between the groups is not statistically significant, as can be seen from the table below. No significant differences (no significant values of 0.05 or below) were found. As the two groups are basically equal in terms of intelligence there is no need to control for this variable.

The two groups (experimental and control group) are similar in terms of official diagnosis, presenting symptoms, age, age of mother at birth, birth weight and intellectual ability. It could therefore be said that the two groups were the same in terms of the tested aspects at the commencement of the research.

From the information obtained in the interviews, questionnaires and assessment it seems that the suggested possible alternative causes of attentional difficulties and heightened motor activity are warranted. Half of the children in the sample were unplanned pregnancies. More than half of the mothers with the unplanned pregnancies presented with signs of postnatal depression. Three children were born by emergency caesarean section (25%). Seven of the 12 children (58%) experienced some form of stress during and directly after birth. These range from anoxia, umbilical cord around neck, jaundice and placement in ICU. Three children (25%) experienced sucking difficulties. Eight (66%) children displayed difficult behaviour as infants. These range from sleeping difficulties, colic, restlessness, rheumatic fever and meningitis. Seven children (58%) presented with allergies and the same number of children (58%) experienced recurrent ear infections and five (42%) had grommets inserted. Six (50%) of the children displayed delayed language development and three (25%) delayed motor development. Seven children (58%) display some kind of visual difficulty. Seven (58%) children consume caffeinated products and 10 children

(83%) obtained significantly high scores on the candida checklist, indicating the possibility of yeast-related symptoms. Five children (42%) scored significant results on the biochemical checklist indicating that there may be some biochemical imbalances present. The most frequent biochemical imbalances mentioned were blood sugar and adrenal imbalances.

Some respondents scored high on more than one aspect. An example is respondent 2 (experimental group). His father was possibly in contact with heavy metals at the time of conception and they installed new carpets in the home during infancy. It was an unplanned pregnancy and his mother was 36 at the time of birth. He presented with anoxia and was placed in ICU directly after birth. He was breastfed for 36 months and was a bad sleeper. He did not receive all the recommended inoculations and suffers from numerous allergies as well as recurrent otitis media. His language development was described as very late. He has blood sugar irregularities, watches up to three hours of television per day and drinks up to three caffeinated beverages in the evening. He scored a count of 130 on the candida checklist, which most definitely could indicate yeast related difficulties. Biochemically he presents with imbalances in terms of heavy metal toxicity, histamine imbalance, toxic overload and inflammation, blood sugar and adrenal imbalance.

Another example is respondent 8 (control group). His family painted the house during his pregnancy. His was an unplanned pregnancy and his mother indicated signs of emotional stress and depression. He was delivered by emergency caesarean section and was placed in ICU directly after birth. He had sucking difficulties as well as numerous allergies and recurrent ear infections. Grommets were inserted a number of times. He experiences a general language developmental delay. This respondent also struggles with blood sugar irregularities, consumes caffeinated beverages and scored a candida count of 160. There are many cases like this, which indicates the necessity to look further for underlying causes.

3. Research Results

At the outset of the study, the respondents were tested on various aspects and through various different methods for attention, heightened motor activity and

academic performance. This constitutes the first measurement (pretest / point A) before either of the groups received any intervention treatment.

After 11 weeks, the children were tested again (midtest / point B). This constitutes the second measurement. At this stage the experimental group had received intervention treatment and the control group had received no treatment. Up to this point a true experimental design was followed.

Shortly after the second testing, the control group was also subjected to the intervention treatment for a period of 11 weeks. The control group is now effectively the **second experimental group**. The initial experimental group continued to receive the intervention treatment. After this period, the children were tested again; this is the third and final measurement (posttest / point C). For clarity, this is illustrated in figure 79.



Research design

3.1 Analysis Approach

The approach followed to investigate the effect of the intervention treatment was as follows:

 The mean score of the experimental and control groups were examined at pre (point A) and midtest (point B) stages. Where the experimental group had improved more than the control group (as the hypothesis states), the improvement was tested statistically.

- To ensure that any observed improvement in the experimental group was large enough relative to the control group, the difference in scores was then computed for both groups (by subtracting the pre-scores from the mid-scores), and the scores were compared using a **non-parametric independent t-test**. This gave an indication of statistically significant results.
- With regards to the tests that were conducted a third time (posttest / point C), after the control group had also received intervention treatment, a non-parametric pairwise t-test was conducted to determine if the control group (now the new experimental group) had improved from the second measurement to the third measurement (effectively their pre-two [point B] and post-two [point C] tests). There was no control group to measure their improvement against and thus no difference scores were calculated; rather a non-parametric pairwise t-test was used.
- A last comparison was possible, namely, short-term effect versus long-term effect, which was undertaken by testing whether the experimental group had improved significantly more than the control group at the third measurement. This was done by calculating difference scores from the first (A) to the third (B) measurement and then comparing the difference scores by means of an independent non-parametric t-test.
- All improvements were indicated in bold and red.

The results are discussed in terms of the three hypotheses posed at the outset of the research. These aspects are attention, motor activity and academic performance. It must be noted that motivation plays a role in determining the performance of most children and especially so in children presenting with AD/HD. Most of the tests were performed three times (pre, mid and post). By the third testing the respondents were generally not as excited to complete the tests as in the initial assessments. The placebo effect was only partly accounted for as respondents were drawn out of the classroom setting (to work on an individual basis) to participate in the intervention while the rest of the group continued with schoolwork.

3.1.1 Neurodevelopment

i) Assessment

A scale of none, slight, moderate and severe was used to assess children on a variety of neurological aspects through the HANDLE questionnaire. The scale is ordinal in nature (it orders people along some continuum, Howell 1995:16), yet it is interpreted as an interval scale for the purposes of statistical comparisons. An interval scale is a scale of measurement for which legitimately different scale points can be determined (Howell 1995:16). A non-parametric t-test for independent means was computed. Table 22 presents the average score (out of 4) for the pretest (point A) and posttests (point B). The base size of the sample is twelve as this measurement was conducted at the first (A) and last (B) testing phase.

Please note that an improvement is indicated by a decrease in the value, with the exception of binocularity.

		•	•	· ,	
	Mean	Mean	F	Significance	Significance –
	Pretest	Posttest	Z	– 2 tailed	1 tailed
Tactile System	2.8	2.1	(2.53)	0.011	0.006
Olfaction System	1.8	1.4	(1.89)	0.059	0.029
Gustation System	1	1	-	1.000	0.500
Vestibular System	3	2.3	(2.13)	0.033	0.017
Kinaesthesia	2.1	1.5	(2.24)	0.025	0.013
Muscle Tone	2.7	2.1	(2.12)	0.034	0.017
Proprioception number out of 5	3.1	2.4	(2.17)	0.030	0.015
Audition System	3.2	2.5	(2.53)	0.011	0.006
Oral Motor System	1.8	1.3	(1.89)	0.059	0.029
Auditory Sequencing	3.6	2.6	(2.76)	0.006	0.003
Auditory Linguistic	3.4	2.7	(2.83)	0.005	0.002
Vision	2.7	2.2	(1.32)	0.187	0.094
Binocularity	2.75	2.1	(2.07)	0.038	0.019
Suppression	2.6	1.4	(2.71)	0.007	0.003

Table 22

Measurement on Neurological Aspects (N = 12)

Attentional Priority	3.5	2.5	(2.97)	0.003	0.001
Differentiation	3.5	2.4	(3.28)	0.001	0.001
ATNR (Reflex)	1.7	1.4	(0.85)	0.395	0.198
Lateralisation	3.1	1.8	(3.02)	0.003	0.001
Interhemispheric Integration	3.5	2.6	(2.97)	0.003	0.001
Balance eyes open	10	10	0	1	0.50
Balance eyes closed	7.6	9.9	(2.388)	0.017	0.008
Binocularity	71.8%	84%	(2.014)	0.044	0.022
Ocular (R)	60.5	78.8%	(2.414)	0.016	0.008
Ocular (L)	58.3	77.5%	(2.229)	0.026	0.013

Nearly all the components on the neurodevelopmental aspects show **significant improvement**, with the exception of gustation, vision, the asymmetric tonic neck reflex (ATNR) and balance with eyes open. The components that show a **significant improvement** do so as all the p-values adhere to the >5 rule.

ii) Neurodevelopmental Outcome Rating

The HANDLE outcome questions were measured at three intervals, the pretest (point A), the midtest (with control group) and the posttest (C) test (without control group). Both the teacher and parents completed this questionnaire.

The average score per child, over all aspects listed and rated, were computed.

Table 23

Correlation between parents and teachers' ratings on HANDLE outcome

			Teachers			
				Post1	Post2	
			Pre A	в	С	
ş		Pearson Correlation	-0.36			
aren	Pre	Sig. (2-tailed)	0.383			
Pa		Pearson Correlation		0.46		
	Post1	Sig. (2-tailed)		0.133		
		Pearson Correlation			0.63	
	Post2	Sig. (2-tailed)			0.051	

While the correlation table only indicates nearly significant correlations between ratings by parents and teachers, the Pearson correlation value of 0.46 and 0.63 acts as an estimation of effect size and indicates **large effects**. However, the negative correlation found between the pretest ratings of parents and teachers indicate that they had conflicting views at the start of the study. Once again, context could have played a significant role.

The scores of the experimental group are interpreted relative to those of the control group for the measurements from pretest (A) to midtest (B). Both the parents and the teacher ratings were compared.

	Experimental			Control		
	Pretest (A)	Midtest (B)	Difference	Pretest (A)	Midtest (B)	Difference
Parents	5.62	2.56	3.06	6.4	3.6	2.8
Teachers	4.04	3.82	0.22	3.8	3.7	0.1

Table 24

Mean scores on the Neurodevelopmental Outcome Rating

Both the experimental and control showed improvement according to parent and teacher ratings. These results are explored further in the following table.

Table 25

Mann Whitney U test on difference scores of the experimental and control groups on Neurodevelopmental Outcome Rating

	Difference scores					
		Significance - Significance				
	Z	2 tailed	- 1 tailed	Effect size		
Parents	17	0.873	0.436	0.08		
Teachers	7	0.772	0.386	0.57		

Parents from both groups observed equal improvement, even though the difference is **not statistically** significant (0.426) and has a very small effect size (0.08). There is, however, a **medium effect** (0.57) observed from the teacher's point of view. The experimental group improved more than the control groups when rated by their teacher.

When considering improvement observed over the long term, namely, from the first testing (point A) to the third testing (point C), the table below indicates the means difference scores of the control and experimental groups at each of these phases, as observed by their parents and teacher.

Table 26

Mean difference scores from first to third measurement for Neurodevelopmental Outcome Rating

	Experimenta	II - Long term	Control - Short term		
	Mean	Std. Deviation	Mean	Std. Deviation	
Parents	-2.92	2.87	-4.65	2.20	
Teachers	-2.03	0.56	-1.18	0.47	

From first (A) to third (C) testing the control groups shows a greater improvement when rated by parents. However, when rated by teachers, the experimental groups improved more than the control group. The table below indicates that the different is significant (p = 0.038) with a **very large effect**.



Table 27

Mann Whitney U test on difference scores of the experimental and control groups (from first to third measurement) on the Neurodevelopmental Outcome Rating

	z	Significance - 2 tailed	Significance - 1 tailed	Cohen's D Effect size
Teachers	-1.7	0.076	0.038	1.6

The value as obtained by the teacher rating scale is **statistically significant** (0.038) as the value is less than 0.5 (p-value rule) with a **large size effect**.

3.1.2 Attention

3.1.2.1 Objective data analysis

i) Integrated Visual and Auditory Continuous Performance Test (IVA)

The IVA Continuous Performance Test (Sandford & Turner 2000) was used to measure both aspects of attention and aspects of heightened motor activity. In this section only the components of the IVA test that specifically measure attention are examined and discussed.

Scores on the IVA are read within a standard deviation graph, with 100 being the norm and a standard deviation of 15. Therefore an obtained value of between 85 and115 still falls within the normal range.

The Full Attention Quotient scores are based on equal measures of Visual and Auditory Vigilance, Focus and Speed.

- 1. **Vigilance** is a measure of inattention as evidence by two different types of errors of omission.
- 2. **Focus** reflects the total variability of mental processing speed for all correct responses.

3. **Speed** reflects the average reaction time for all correct responses throughout the test and helps identify attention-processing problems related to slow discriminatory mental processing.



A diagram illustrating the different subsections is given below.

Attention quotient

Table 28 includes the mean scores on the Attention dimension of the IVA test. Areas where significant results were obtained are indicated in **bold** and in **red**.

	Experimental		Control			
	First test	Second test	Difference	First test	Second test	Difference
		(Global Scales	5		
Attention	75.2	75.7	0.5	77.5	77.8	0.3
Auditory	81.0	80.2	-0.8	79.0	87.0	8.0
Visual	75.3	76.5	1.2	79.5	75.3	-4.2
			Auditory			
Vigilance	78.7	74.8	-3.8	77.7	84.5	6.8
Focus	82.8	82.5	-0.3	82.8	88.7	5.8
Speed	102.5	103.2	0.7	97.3	100.2	2.8

Table 28Mean scores on the Attention dimension of the IVA test

			Visual			
Vigilance	72.8	74.3	1.5	85.3	77.8	-7.5
Focus	83.2	86.3	3.2	89.5	91.0	1.5
Speed	92.7	91.5	-1.2	80.3	74.8	-5.5

In general there was **little improvement** in scores, and on some components the children's scores are worse at the second testing (B). The difference between the first (A) and second (B) measurements was calculated by subtracting the midtest (B) values from the pretest (A) values.

Positive scores indicate improvement from first (A) to second (B) measurement, while negative scores indicate a decrease in scores that could be interpreted as no improvement or regression.

On some aspects of Attention (Visual Attention Quotient, Auditory Speed, Visual Vigilance and Visual Focus), the experimental group showed more improvement than the control group, while on other aspects, the control group showed more improvement than the experimental group (Auditory Attention, Vigilance, Focus, Speed and Visual Focus).

Comment: The question arises of whether the intervention resulted in disorganisation, which would explain these results. Disorganisation before reorganisation is common in sensory integration interventions; and since only the experimental group received intervention this theory will only apply to them.

The hypothesis states that the experimental group will improve significantly more than the control group. Those aspects where the control group improved more than the experimental group were not tested statistically, as the alternative hypothesis is rejected.

These difference scores were compared using a non-parametric t-test for independent groups and the results are given in table 29.

Table 29

	Difference scores						
		Significance - Significance Cohen					
	Z	2 tailed	- 1 tailed	Effect size			
Attention Quotient	-0.160	0.873	0.218	0.01			
Visual Attention Q	-0.320	0.749	0.187	0.3			
	Visual A	Attention Quotient					
Vigilance	-0.964	0.335	0.084	0.50			
Auditory Attention Quotient							
Speed	-0.080	0.936	0.234	0.30			

Mann Whitney U test on difference scores of the experimental and control groups (from first to second measurement) on the Attention component of the IVA

The effect size implies something very different from the p-value. A result that is statistically significant is not necessarily particularly important as judged by the magnitude of the effect; highly significant results should therefore not automatically be interpreted as reflecting large effects (Rosnow & Rosenthal 1996:276). One of the main methods of establishing effects sizes is to calculate **Cohen's d** effect size (Cohen's d) and can be interpreted as:

0.2 = small effect size

0.5 = medium effect size

0.8 = large effect size

Effect size is important since the sample is small (n=12). The table above indicates **small to medium effect size** on the Visual Attention Quotient and Auditory Speed. A medium effect size is seen on the Visual Vigilance Scale (0.50).

In terms of the Attention component of IVA, the experimental group **did not improve significantly** relative to the control group on any of the subtests using the p-value - 0.05 rule. However, Visual Vigilance has a p-value of 0.084 and the effect size (Cohen's d) is medium (0.5) and warrants mention. This indicates the only possible

aspect where an improvement in the experiment group is greater than the control group; however, this was not a significant improvement.

After the second measurement (midtest / point B), the control group also received the intervention treatment and was then regarded as a **second experimental group**. There was therefore no control group this time. Table 30 indicates the test for significance improvement for this new experimental group.

Table 30

Non-parametric Wilcoxon Signed Ranks Test for significant differences between second (B) and third testing (C) on the Attention component of the IVA for the second experimental group (first control group)

	Mean	Mean		Significance -	Significance	Cohen's D
	Mid (B)	Post (C)	Z	2 tailed	- 1 tailed	Effect size
Attention	77.83	82.50	-0.631	0.528	0.264	0.18
Auditory	87.00	85.50	-0.314	0.753	0.377	0.08
Visual	75.33	83.17	-1.084	0.279	0.139	0.28
			Auditory			
Vigilance	84.50	87.67	-1.826	0.068	0.034	0.14
Focus	88.67	86.33	-0.210	0.833	0.417	0.19
Speed	100.17	97.83	-0.105	0.917	0.458	0.11
			Visual			
Vigilance	77.83	90.67	-2.023	0.043	0.022	0.45
Focus	91.00	89.67	-0.105	0.916	0.458	0.08
Speed	74.83	83.00	-0.946	0.344	0.172	0.34

Once again there is **little significant improvement** overall. **Significant improvement** is seen only on the Vigilance (auditory as well as visual) component. Visual Vigilance has a p-value of 0.022 (rule of 0.05 applies) and an effect size of 0.45 (medium).

Therefore it can be concluded that the IVA shows no significant improvement in terms of overall Attention Quotient among children who received the intervention treatment. It is only on the Visual Vigilance Scale that there is some support for the hypothesis, as both the first experimental group and the second experimental group showed improvement on this aspect.

While neither the experimental group nor the control group (after intervention) showed any significant improvement in terms of overall Attention Quotient, it may be that the experimental group improved on the Attention dimensions after a longer period of intervention, as they had received the intervention for a longer period of time.

The original experimental group continued to receive the intervention treatment. By examining scores at the third measurement (posttest / point C) relative to the first (A), the possible effects of a longer exposure to the treatment may be tested. To ensure that any possible effects are interpreted as long-term effects, the experimental group was evaluated relative to that of the control group. This is again achieved through the difference scores calculation.

An independent t-test calculation is then used to determine if the control group and experimental groups differ from one another. Table 25 indicates the mean difference between the first (pretest / point A) and the third (posttest / point C) measurements for both groups.

Table 31

Mean difference scores from first to third measurement for the experimental and control group

	Experimenta	al - Long term	Control – Short term		
	Mean	Std Deviation	Mean	Std Deviation	
Attention Quotient	8.33	11.66	5.00	21.32	
Auditory	3.50	10.77	6.50	18.58	
Visual	9.33	24.39	3.67	20.70	
		Auditory			
Vigilance	6.33	27.81	10.00	15.62	
Focus	(2.00)	15.56	3.50	25.33	
Speed	0.67	12.13	0.50	12.85	

Visual						
Vigilance	14.83	30.02	5.33	24.91		
Focus	2.67	19.59	0.17	15.20		
Speed	3.17	11.07	2.67	23.65		

In the cases where the experimental group showed greater improvement from the first (A) to the third (C) measurement, the significance test was performed. Table 32 indicates the significance test on the change in scores between the experimental and control groups.

Table 32

Mann Whitney U test on difference scores of the experimental and control groups (from first to third measurement) on the Attention component of the IVA

		Significance - Sign		Cohen's D
	Z	2 tailed	- 1 tailed	Effect size
Attention Quotient	-	1.000	0.500	0.19
Visual	(0.803)	0.422	0.211	0.25
		Auditory		
Speed	(0.080)	0.936	0.468	0.01
		Visual		
Vigilance	(0.641)	0.522	0.261	0.34
Focus	(0.160)	0.873	0.436	0.14
Speed	(0.160)	0.873	0.436	0.03

Using the p-value (0.05) rule, the experimental group did **not improve significantly** more than the control group over a longer period of time. However, Visual Vigilance did improve by a larger margin (14.83 vs. 5.33). Although it is not significant, it does show a small to medium size effect (0.34).

ii) Cognitive Control Battery (CCB)

Another test that measures attention is the Cognitive Control Battery (CCB). In line with the literature study conducted and the information obtained on attention, only

one component of the CCB was incorporated. This was the Fruit Distraction Test and relates to the Stroop effect. The scores of the experimental and control groups on the six dimensions of the Fruit Distraction Test are given in table 33.

Table 33

	Experimental			Control		
	First test	Second Test	Difference/ Improvement	First test	Second Test	Difference/ Improvement
Baseline – Time	40.7	44.5	3.83	43.3	48.2	4.83
Baseline – Errors	52.3	51.7	-0.67	49.5	56.3	6.83
External distractions						
-Time	49.8	39.8	-10.00	48.7	45.0	-3.67
External distraction						
– Errors	48.0	46.7	1.33	49.0	46.7	-2.33
Internal distraction -						
Time	46.3	47.8	1.50	36.7	46.5	9.83
Internal distraction -						
Errors	42.3	49.7	7.33	42.5	41.5	-1.00

Mean scores of the dimensions of the Cognitive Control Battery

Internal and external distraction errors were the only two aspects that showed greater **improvement** in the experimental group than in the control group. The improvements on these two aspects were examined relative to the control group by conducting an independent t-test on the mean scores of the difference scores (posttest – pretest scores).



Table 34

		•	•			
	Experimental group 2					
	z	Significance - 2 tailed	Significance - 1 tailed	Cohen's D Effect size		
External distraction error	-0.161	0.872	0.436	0.01		
Internal distraction error	-0.962	0.336	0.168	0.70		

Mann Whitney U Test for significant differences between the control and experimental groups on the Cognitive Control Battery

The experimental group showed a **large**, if not **statistically significant**, **improvement** relative to the control group on Internal Distraction Errors – with an effect size of 0.7, which is a large effect. This would indicate that the experimental group was able to control internal distracters better than the control group.

The improvement of the control group at the third testing (posttest / point C) was discussed here; now the second experimental group is given below.

Table 35

Mean scores of the dimensions of the Cognitive Control Battery of the control group after treatment (new experimental group)

	Ex	perimental Grou	ıp 2
	First test	Second Test	Difference/ Improvement
Baseline – Time	48.2	46.3	-1.8
Baseline – Errors	56.3	57.0	0.7
External distractions –Time	45.0	42.5	-2.5
External distraction – Errors	46.7	49.8	3.2
Internal distraction – Time	46.5	43.5	-3.0
Internal distraction – Errors	48.2	46.3	-1.8

The table shows that the second experimental group improved only on baseline errors and external distracters errors. Improvement on baseline errors was **not significant** (p=0.352). Improvement on External Distraction Errors shows a p-value of 0.139 (above the 0.05 cutoff) but had a **medium effect size** of 0.42 that warrants mentioning.

Table 36

Non-Parametric Wilcoxon Signed Ranks Test for significant differences between second (b) and third (c) testing on the Cognitive Control Battery for the control group (new experimental group)

	Experimental Group 2					
	z	Significance - 2 tailed	Significance - 1 tailed	Cohen's d Effect size		
Baseline – Errors	0.378	0.705	0.352	0.18		
Internal distraction – Time	1.08	0.278	0.139	0.42		

The table indicates the **medium size effect** of the improvement (0.42).

Again the long-term effect of treatment was tested by comparing the first experimental group with the second experimental group on the first (A) and third (C) scores to gain insight in the possible long-term effects of the intervention. Table 37 indicates the mean difference score (change from pretest to posttest) for both groups.



Table 37

Mean <u>difference</u> scores from first to third measurement for the experimental and control group on the Cognitive Control Battery

	Experimental - Long				
	term		Control - Short term		
		Std		Std	
	Mean	Deviation	Mean	Deviation	
Baseline – Time	(8.00)	7.13	(3.00)	8.90	
Baseline – Errors	(9.67)	12.42	(7.50)	7.84	
External distractions –Time	17.83	8.35	6.17	15.82	
External distraction – Errors	(1.83)	7.68	(0.83)	13.38	
Internal distraction – Time	1.33	9.83	(6.83)	5.71	
Internal distraction – Errors	(4.67)	12.29	1.33	16.55	

The long-term effect seems to apply to External Distraction Time and Internal Distraction Time. The experimental groups show an improvement of 17.83 relative to the 6.17 of the control group on External Distraction Time. In terms of Internal Distraction Time, the experimental group improved by 1.33 while the control group regressed by 6.83.

Table 38

Mann Whitney U Test on difference scores of the experimental and control groups (from first to third measurement) on the Cognitive Control Battery

	z	Significance - 2 tailed	Significance - 1 tailed	Cohen's D Effect size
External distractions –Time	-1.441	0.150	0.075	0.92
Internal distraction – Time	-1.878	0.060	0.030	1.02

Long-term effect on External Distraction Time shows an **almost significant improvement** value of p=0.075 (not within the p-value>0.050 rule), although it has a **very large size effect** (value of 0.92). Internal Distraction Time **improved significantly** (p=0.030; Cohen's d = 1.02). Therefore it can be concluded that on these two aspects the experimental group showed greater long-term improvement relative to the control group. This suggests that the experimental group showed

improvement after a prolonged period of time (24 weeks) in terms of processing speed whilst facing both internal and external distractions.

3.1.2.2 Subjective data analysis

i) Amen Rating Scales

The Amen Rating Scales consist of various sections. The first section, measured by questions 1-14, reflects "inattention". A score of six or more high scores (score of 3 or 4) is needed to make a diagnosis (Amen 2001:72).

The respondents' scores on all the items (1-14) were summed to obtain their inattention score; the higher the score the more they struggle with attention. Both parents and teachers were asked to rate children on this questionnaire. This questionnaire was conducted only at the beginning (pretest / point A) and at the very end (posttest / point C), after the control group had also received the intervention.

For this reason, these specific questions will be treated according to a "one-group" design and responses will be combined for both groups. While one can attempt to determine if there was any improvement, there is the limitation of not having a control group to measure improvement against. Only statistically significantly high scores were included in the interpretation.



Figure 81 Classification according to the Amen Rating Scale

Five of the six children (83%) in the experimental group, and four in the control group (66%), comply with the criteria for the Inattentive Type as rated by the parents. Because a new teacher started at the school during the research period, data obtained by the teacher on the same rating scale were very incomplete.

Table 39

					Std	
	N	Minimum	Maximum	Mean	Deviation	
		Parents				
Pretest (point A)	11	22	44	33.18	6.84	
Posttest (point C)	11	2	39	25.82	10.95	
Teachers						
Pretest (point A)	11	8	39	24.55	11.09	
Posttest (point C)	11	1	31	21.09	8.23	

Average scores on inattention on the Amen Rating Scale

At the pretest (point A), the overall score awarded by the parents for **inattention** was 33.18. At posttest (point C), this score changed to 25.83. The teacher also indicated an average difference from 24.55 to 21.09. Incomplete information was obtained for one child and was therefore not included in the data.

Comment: The difference between the parent and teacher ratings could be due to the difference in setting between the home and school environment. The different attentional requirements in each setting should also be considered.

Table 40

Wilcoxon Signed Ranks Test for significant improvement from pre (point a) to posttest (point c) on Inattention

	z	Significance - 2 tailed	Significance - 1 tailed	Cohen's d
Parents	-1.738	0.082	0.041	0.57
Teacher	-1.558	0.119	0.060	0.25

The one tailed p-value for the teachers is **nearly significant** (p=0.06), while the effect size is only **small to medium** (Cohen's d = 0.25). This indicates a positive move towards more attentive behaviour. The test for significant differences, namely, the Wilcoxon Signed Ranks Test, indicates that the parents report **significant improvement**. Parents thus noted a **significant improvement** in the attentive behaviour of the children (p=0.41) with a **large effect size** (Cohen's d = 0.57).

ii) Temperament Scale

The Temperament Scale (Thomas & Chess 1977) was used to obtain information on the different aspects of temperament. Of the nine aspects included on this bipolar scale, only Distractibility falls within the scope of this research. The bipolar scale varies from low (1) to high (9). The values below indicate the true values as obtained on the actual questionnaire.

Table 41Distractibility on the Temperament Scale

	Pare	ents	Teachers		
	Pre1	Post2	Pre1	Post2	
Distractibility	7.45	5.30	6.36	7.00	

On the Distractibility component on the Temperament Scale, parents showed a **large improvement** from 7.45 (close to highly distractible) to 5.30, indicating a more balanced profile. On the other hand, the teacher tended to report children being more distracted overall.

Table 42

Wilcoxon Signed Ranks Test for significant improvement from pre to posttest on Temperament - Parents

	z	Significance – 2 tailed	Significance – 1 tailed	Cohen's d Effect size
Distractibility	-2.095	0.036	0.018	1.40

The table above indicates that the improvement as noted by the parents is indeed **large** (1.40) and **significant** (p=0.018).

3.1.2.3 Conclusion

A summary of the statistically significant and noteworthy research results obtained are presented in the table below:

Stage A	Stage B	
Experimental Research	One Group Experiment	Short Term vs. Long Term
(Experimental Group)	(New Experimental Group)	Effect
Integrated Visual	and Auditory Continuous Perfor	mance Test (IVA)
Visual Vigilance has a p-value	Visual Vigilance did improve	Visual Vigilance did improve by
of 0.084 (not significant at 95%	significantly with a p-value of	a large margin (14.83 vs. 5.33),
level but significant at 90%)and	0.022 and medium effect size of	and although not significant,
a medium size effect (0.5) and	0.45	does show a small to medium
warrants mention		size effect (0.34)
	Auditory Vigilance did	
	improve significantly (0.034) but	
	has a small effect size	

Table 43Research Results for Attention

Cognitive Control Battery (CCB)					
Internal Distractibility	External Distractibility Error	External Distractibility Time			
Error Significantly large improvement with a size effect of 0.7 but not statistically significant	Shows a p-value of 0.139 (above the cutoff) but has a medium effect size of 0.42	Shows an almost significant improvement value of p=0.075 but a very large effect size of 0.92 Internal Distractibility Time Improved significantly with a p-value of 0.030 and large effect size of 1.02			
	Amen Rating Scale	1			
Parents noted significant impro	vement (p=0.41) in attentive beha	aviour in both groups with a large			
effect size of 0.57					
Temperament					
Parents indicated a significant in	nprovement (p=0.018) with a large	effect size (1.40)			

The following conclusions can be drawn from the table:

- Control and experimental group comparisons reveal improvements on some components of Attention in the experimental group with two standardised measuring instruments after a period of 11 weeks of intervention.
- Improvements are noted on components of Attention in the second experimental group (first control group) on two standardised measuring instruments after a period of 11 weeks of intervention.
- The results obtained by the second experimental group (control group) verifies to an extent the results obtained with the first experimental group.
- The long-term effect is illustrated with both the standardised measuring instruments.
- Parents in both groups noted improvements in Attention.

3.1.3 Motor activity

3.1.3.1 Objective data analysis

i) IVA – Response control

The dimension in the IVA that relates to Motor Activity is discussed in this section. The **Full Scale Response Control Quotient** is based on separate **Auditory** and **Visual Response Control Quotient** scores. These **Response Control Quotient** scores are derived from equal weights of visual and auditory **Prudence**, **Consistency** and **Stamina** scales, described below:

- 1. **Prudence** is a measure of impulsivity and response inhibition as evidenced by three different types of errors of commission.
- 2. **Consistency** measures the general reliability and variability of response times and is used to help measure the ability to stay with the task.
- 3. **Stamina** compares the mean reaction times of correct responses during the first 200 trials to the last 200 trials. This score is used to identify problems related to sustaining attention and effort over time.

The different components of the IVA overlap at times as noted in Stamina under Response Control. The information obtained here could thus also relate to attention in general.

Table 44 presents the actual scores of the experimental and control groups.

Table 44

Mean scores on the Hyperactivity dimension of the IVA Test (Response Control)

	Experimental			Control		
		Second			Second	
	First test	test	Difference	First test	test	Difference
Response control	76.00	84.17	8.17	74.50	83.00	8.50
Auditory	77.50	89.33	11.83	73.33	80.00	6.67
Visual	80.33	82.67	2.33	83.00	89.67	6.67

Auditory						
Prudence	76.83	77.50	0.67	72.67	69.83	-2.83
Consistency	81.00	85.50	4.50	77.17	87.50	10.33
Stamina	98.17	119.17	21.00	98.50	103.17	4.67
		V	isual			
Prudence	78.67	85.50	6.83	86.00	90.83	4.83
Consistency	78.17	87.50	9.33	83.67	85.67	2.00
Stamina	106.50	94.33	(12.17)	99.00	104.50	5.50

Differences are noted on Auditory Response Control Quotient in the experimental group as well as on Auditory Prudence, Stamina, Visual Prudence and Consistency. Only those scores relevant to the hypotheses were tested for significance. The difference scores (between first and second measurements) were compared by means of an independent non-parametric t-test. Results are given in table 45.

Table 45

Non-Parametric Mann-Whitney U Test for significant differences between difference scores of the experimental and control groups on the Hyperactivity component of the IVA

	Difference scores					
	z	Significance Significance		Cohen's D Effect size		
Auditory RQ	(0.56)	0.574	0.287	0.23		
Auditory						
Prudence	(0.48)	0.631	0.315	0.15		
Stamina	(1.29)	0.199	0.099	1.04		
Visual						
Prudence	(0.64)	0.522	0.261	0.14		
Consistency		1.000	0.500	0.39		
Stamina	(0.72)	0.470	0.235	0.72		

Although none of the improvements of the experimental group is statistically significant, a **large size effect** is seen on Auditory Stamina (1.04) and Visual Stamina (0.72). There is also a **small to medium effect** (0.39) for Visual Consistency.

The improvement of the control group at the third measurement (after intervention) is now examined. As no control group exist for this new experimental group, the scores are compared using the non-parametric test for dependent measures and the table below indicates the results of this test.

Table 46

Non-Parametric Wilcoxon Signed Ranks Test for significant differences between second and third testing on the Hyperactivity component of the IVA for the second experimental group

	Mean Pre	Mean Post	z	Significance - 2 tailed	Significance - 1 tailed	Cohen's D Effect size
Auditory	80.00	93.00	-1.99	0.046	0.023	0.72
Auditory						
Prudence	69.83	86.50	-1.34	0.180	0.090	0.67
Stamina	103.17	112.83	-0.94	0.345	0.173	0.93
Visual						
Prudence	Prudence	90.83	84.67	-1.57	0.116	0.058
Consistency	85.67	89.83	-1.26	0.206	0.103	0.18
Stamina	104.50	100.00	-0.94	0.345	0.173	0.20

Only values that are significant in terms of the hypotheses were discussed. The second experimental group showed improvement from the midtest (point B) to posttest (point C) when they received intervention in terms of overall Response Control, Auditory and Visual Prudence, Visual Stamina and Auditory Consistence. Scores on the Auditory component showed a **significant improvement** at 0.05 level (p=0.023) with a **large effect size** (0.72). Although not statistically significant,

Auditory Prudence and Stamina **have large effect** size values of 0.67 and 0.93 (indicating that the differences are really large and worth mentioning).

To test the long-term effect of the treatment, the improvement from first to third testing was calculated and the control and experimental groups compared.

Table 47

Mean difference scores from first to third measurement of Hyperactivity IVA for the experimental and control group

	Experimental - Long term		Control – Short term		
	Mean	Std Deviation	Mean	Std Deviation	
Response control	11.67	12.19	14.33	38.73	
Auditory	10.67	20.41	19.67	36.38	
Visual	8.83	13.63	3.83	32.58	
Prudence	16.33	26.49	13.83	34.97	
Consistency	(0.33)	20.95	7.50	31.29	
Stamina	6.67	19.54	14.33	8.59	
Prudence	10.33	18.63	(1.33)	10.01	
Consistency	10.67	12.16	6.17	23.40	
Stamina	(4.17)	16.68	1.00	38.51	

Table 48 shows the significance test used to determine whether the experimental group showed a statistically significant improvement over the control group.



Table 48

Mann Whitney U Test on difference scores of the experimental and control
groups (from first to third measurement) on the Hyperactivity component of the
IVA

	Z	Significance – 2 tailed	Significance – 1 tailed	Cohen's D Effect size		
Visual	-0.320	0.749	0.374	0.20		
Auditory						
Prudence	0.000	1.000	0.500	0.08		
Visual						
Prudence	-1.363	0.173	0.086	0.78		
Consistency	-0.241	0.810	0.405	0.24		

Although the experimental group shows a long-term improvement on the above elements, the only **large difference** worth mentioning is on Visual Prudence where the p-value of 0.086 is just above the cutoff (0.05) and a **large effect size** is seen.

3.1.3.2 Subjective data analysis

i) Rating scales (AMEN)

The component of the Amen Rating Scale that relates to motor activity is measured by questions 15-23 on this questionnaire. Only high values (3 and 4) qualify as significant according to the interpretation guidelines.



Figure 82

Motor Activity measured at the onset of the research
Three children in each group fall within this classification according to the Amen Rating Scale. The sum of scores obtained in the questions gives an indication of the intensity of the condition. The higher the scores, the more hyperactive the children were thought to be.

Table 49

					Std	
	Ν	Minimum	Maximum	Mean	Deviation	
Parents						
Pretest	11	4	32	16.82	7.55	
Posttest (3 rd)	10	4	35	14.80	8.87	
Teachers						
Pretest	11	0	35	13.45	10.01	
Posttest (3 rd)	11	1	21	7.73	6.92	

Average scores on Hyperactivity from Amen Rating Scale

Both teachers and parents awarded lower scores to children at the posttest (point C) stage. Table 50 tests if these improvements are statistically significant.

Table 50

Wilcoxon Signed Ranks Test for significant improvement from pre to posttest on Hyperactivity

	z	Significance - 2 tailed	Significance - 1 tailed	Cohen's d
Parents	-0.65	0.513	0.257	0.27
Teacher	-2.23	0.026	0.013	0.66

On Motor Activity, the parents did not report any significant improvement, although the teacher observed that the hyperactivity of students **decreased considerably** (p=0.013). A **medium to large effect** size was observed. Once again, the different setting could have played a role in the different viewpoints. Parents are more likely to observe children in an informal setting without a high value on motor and impulsivity control. However, teachers require children to be able to control movement during classroom sittings.

ii) Temperament

The Temperament Scale (Thomas & Chess 1977) was used to obtain information on the different aspects of temperament. Of the nine aspects included on this bipolar scale, only Activity Level falls within the scope of this research. The bipolar scale varies from low (1) to high (9). The values below indicate the true values as obtained on the actual questionnaire.

Table 51 Average scores on Temperament (N = 12)

	Pare	ents	Teachers		
	Pre1	Post2	Pre1	Post2	
Activity Level	5.73	5.70	6.27	6.55	

Overall values do not indicate any big movements on the bipolar scale. These values are further explored in the next table.

Table 52

Wilcoxon Signed Ranks Test for significant improvement from pre to posttest on Temperament - parents

		Significance	gnificance Significance	
	Z	- 2 tailed	– 1 tailed	Effect size
Activity Level	-0.171	0.864	0.432	0.02

None of these results are significant.

3.1.3.3 Conclusion

A summary of the statistically significant and noteworthy research results obtained are presented in table 53:

Table 53Research results for Motor Activity

Stage A	Stage B	
Experimental Research	One Group Experiment	Short Term vs Long Term
(Experimental Group)	(New Experimental Group)	Effect
Integrated Visual	and Auditory Continuous Perfor	rmance Test (IVA)
Auditory Stamina shows no	Auditory Response	Visual Prudence is not
significant improvement at the	Control shows a significant	significant (p=0.086) but shows
95% level but does show	improvement (p=0.023) with a	a large effect size of 0.78
significant improvement at 90%	large effect size (0.72)	
(p=0.099) but large effect size		
of 1.04	Auditory Prudence and	
Visual Stamina shows no significant improvement	Auditory Stamina do not show	
	a significant improvement but	
(p=0.235) but has large effect	nave a large effect size (0.67	
size (0.72)	anu 0.95)	
Visual Consistency shows no		
significant improvement		
(p=0.500) but a small to		
medium size effect (0.39)		
	Amen Rating Scale	
The teacher indicated a signific	ant difference (p=0.013) with a me	dium to large size effect of 0.66

The following conclusions can be drawn from the table:

- Improvements (not statistically significant) are noted in terms of components in motor activity in the experimental group.
- Significant improvements are noted in the second experimental group as well as general improvements with large effect sizes.
- Improvement was also detected over the long term, and although not statistically significant, a large effect size was noted.
- The teacher noted a significant improvement in hyperactive behaviour in both experimental groups.

3.1.4 Academic performance

Children's reading ability, mathematical and spelling skills were tested before the intervention (pretest / point A), after the initial intervention (midtest / point B), and again after a longer period of intervention (posttest / point C).

To evaluate the improvement of the experimental group relative to that of the control group, the difference scores were once again calculated. The table below provides the mean scores of the pretest (A) and the midtest (B), as well as the difference in scores between the two measurements. Scores on all the components with the exception of Visual Discrimination were expected to improve as a result of the intervention. Improvement on Visual Discrimination is indicated by a decrease in scores.

		Experime	ntal	Control		
	Pre	Post1	Difference	Pre	Post1	Difference
Reading ability	31.50	37.83	6.33	37.17	39.50	2.33
Visual discrimination	10.00	5.83	-4.17	7.33	3.50	-3.83
Maths: addition	8.21	8.72	0.51	7.94	7.71	-0.24
Maths: subtraction	8.55	8.96	0.40	8.24	8.25	0.01
Maths: multiplication	9.03	9.29	0.26	8.60	8.68	0.08
Maths: division	8.75	9.08	0.33	8.05	7.03	-1.03
Spelling	27.83	32.17	4.33	23.83	25.83	2.00

 Table 54

 Pretest and midtest scores on scholastic subjects

The experimental group improved more than the control group on all the academic components and these differences are tested significantly in the following table.

Table 55

	Mann-	7	Significance -	Significance -	Cohen's d
	winniey 0	2	z talleu	i taneu	Effect Size
Reading ability	8.5	-1.53	0.13	0.06	0.66
Visual discrimination	17	-0.16	0.87	0.44	0.09
Maths: addition	7	-1.77	0.08	0.04	1.30
Maths: subtraction	10	-1.29	0.20	0.10	0.88
Maths: multiplication	12	-0.97	0.33	0.17	0.40
Maths: division	10.5	-1.21	0.23	0.11	0.57
Spelling	13	-0.80	0.42	0.21	0.38

Comparisons of difference scores of the experimental and control groups: Mann Whitney U Test for differences in independent means

While the significance test struggled to indicate significant differences between the improvements to the control group relative to the experimental group, the effect size calculation, which is independent of the sample size, does show some **medium and large** effects.

Mathematical **addition** ability **improved significantly** among children in the experimental group (p = 0.04) with a large size effect of 1.30. In addition, **large size effects** (0.88) are seen for Maths subtraction and **medium effects** (0.57) for Maths division. Even the effect size of 0.4 is notable (multiplication). Reading ability also shows a **medium to large effect** size (0.66), and an **almost significant** p-value (0.06).

It can therefore be concluded that children in the experimental group did **improve significantly** more than children in the control group with regards to Reading and Maths.

The control group received the same intervention treatment between the second (point B) and third (point C) measurements. The scores of the control group (now the second experimental group) are compared below.

Table 56

Non-Parametric Wilcoxon Signed Ranks Test for significant differences
between first and third testing for the control group – "new experimental
group"

	Experimental						
	7	Significance	Significance	Cohen's d			
	Z	– 2 talled	- 1 talled	Effect size			
Reading ability	2.23)	0.03	0.013	0.34			
Visual discrimination	0.32)	0.75	0.376	0.00			
Maths: addition	0.13)	0.89	0.446	0.09			
Maths: subtraction	1.46)	0.14	0.072	0.25			
Maths: multiplication	1.75)	0.08	0.040	0.73			
Maths: division	1.83)	0.07	0.034	0.69			
Spelling	1.22)	0.22	0.111	0.86			

The results show **significant improvement** in terms of Reading (p=0.013) with a **medium size effect**, Maths multiplication (p=0.040) with a **large size effect** and Maths division (p=0.034) with a **medium to large size** effect. In addition, the **large effect size** shows that Spelling also improved (0.86). This supports the figures obtained from the first experimental group. These results are consistent with the improvements shown by the experimental group during the first phase.

To determine if the experimental group improved significantly more than the control group at the third testing (point C) after being exposed to the intervention for longer, the differences between their first and last scores were calculated, and these differences or improvement scores were compared by means of the Mann-Whitney Test for independent measures.

Table 57

Mean scores for experimental and control groups for testing at A and C

	Experimental			Control		
	Pretest	Posttest		Pretest	Posttest	
	A	C	Difference	A	C	Difference
Reading ability	31.5	41.1	9.67	37	45.7	8.5
Visual discrimination	10	1.3	-8.68	7	3.5	-3.8
Maths: addition	8.2	8.6	0.42	7.9	7.6	-0.30
Maths: subtraction	8.6	9.1	0.51	8	8	-0.21
Maths: multiplication	9.0	9.6	0.60	8.6	9	0.38
Maths: division	8.7	10.1	1.32	8	8.8	0.74
Spelling	28	34	6.5	24	31	7.33

Table 58

Non-Parametric Mann-Whitney Test for significant differences between improvements shown from first to third testing

		Exper	imental	
		Significance	Significance	Cohen's d
	Z	- 2 tailed	- 1 tailed	Effect size
Reading ability	(0.24)	0.809	0.404	0.30
Visual discrimination	(1.62)	0.106	0.053	1.14
Maths: addition	-	1.000	0.500	0.05
Maths: subtraction	(1.29)	0.197	0.099	0.67
Maths: multiplication	(0.16)	0.871	0.436	0.09
Maths: division	(0.65)	0.519	0.259	0.33

With regards to Visual Discrimination, the experimental group average scores came down from 10 to 1.33 from the first measurement (point A) to the third (point C). The control group only came down from 7.3 to 3.5 during the same time.



While both groups showed improvement in terms of Visual Discrimination, the experimental group showed a **particularly large difference** with a nearly significant p-value of 0.053 and a **very large effect** size of 1.14. Another component where a **large effect size** was found is in terms of Maths subtraction, with an effects size of 0.67.

The experimental group **improved significantly more** than the control groups between point A and B. After the second intervention the control group also showed **significant improvements** on the same components as the experimental group and the long-term effect of treatment resulted in **significant improvements** in Visual Discrimination and Maths subtraction.

These consistent results provide support for concluding that the intervention resulted in a statistically significant improved academic performance on Reading and Maths ability. Therefore there is support for rejecting the null hypothesis on the third stated hypothesis.

3.1.4.1 Conclusion

A summary of the statistically significant and noteworthy research results obtained are presented in the table below:

Stage A	Stage B	
Experimental Research	One Group Experiment	Short Term vs Long Term
(Experimental Group)	(New Experimental Group)	Effect
	Reading	
	Reading showed a significant	
	improvement (p=0.013) with a	
	medium effect size of 0.34	

Table 59Research results for academic performance

Mathematical Skills						
Maths addition showed a	Maths multiplication	Maths subtraction did not				
significant improvement	showed a significant	show a significant improvement				
(p=0.04) with a large effect size	improvement (p=0.40) with a	(p=0.099) but did show a large				
(1.30)	large effect size of 0.73	effect size of 0.67				
Maths subtraction did not	Maths division showed a					
show a significant improvement	significant improvement					
(p=0.10) but did show a large	(p=0.034) with a medium to					
effect size of 0.88	large effect size of 0.69					
Maths multiplication did not						
show a significant improvement						
(p=0.17) but did show a						
medium effect size of 0.40						
Maths division did not show a						
significant improvement						
(p=0.11) but did show a						
medium effect size of 0.57						
	Spelling					
	Spelling did not show a					
	significant improvement					
	(p=111) but did show a large					
	effect size of 0.86					
Visual Discrimination						
		Visual Discrimination				
		showed a nearly significant				
		improvement of p=053 with a				
		particularly large effect size of				
		1.14				

The following conclusions can be drawn from the table:

- Children in the first experimental group improved significantly more than children in the control group with regards to Maths skills.
- The second experimental group showed significant improvements with large effect sizes on Reading, Maths and Spelling.



- Results obtained by the second experimental group verify the results obtained in the first experimental group.
- A long-term effect is seen with significant results in Visual Discrimination and Maths subtraction.
- These consistent results provide support for the conclusion that the intervention resulted in a statistically significant improvement in academic performance with regards to Reading, Maths ability and Visual Discrimination. Therefore, there is support for rejecting the null hypothesis on the third stated hypothesis.

4. Conclusion

In this chapter, the results of the experimental research are presented. These results were obtained by calculating the **mean score** of the experimental and control groups at the pre- (point A) and midtest (point B) stages. Where the experimental group improved more than the control group (as the hypothesis states), the improvement was tested statistically. To ensure that any observed improvement in the experimental group was large enough relative to the control group, the difference scores were computed for both groups (by subtracting the pre scores from the mid scores), and the scores were compared using a non-parametric independent ttest. This gave an indication of statistically significant results. With regards to the tests that were conducted at the third point (posttest / point C), after the control group had also received the intervention treatment, a non-parametric pairwise t-test was used to determine if the control group (the new experimental group) had improved from the second measurement to the third measurement (effectively their pre 2 (point B) and post 2 (point C) tests). There was no control group during this stage of the research against which to measure their improvement, and no difference scores were calculated; rather a non-parametric pairwise t-test was used. The last comparison considered short-term effect versus long-term effect by testing whether the experimental group had improved significantly more than the control group at the third measurement. This was done by calculating difference scores from the first (A) to third (B) measurement and then comparing the difference scores by means of an independent non-parametric t-test.

The significant results that were obtained must be considered with caution. Usually significant results are obtained on either a 90% or 95% level (p-value). The results that were presented as significant were obtained at the 95% level. Since the sample size is small (n=12), another calculation was performed, namely Cohen's d. The effect size implies something very different from the p-value. A result that is statistically significant is not necessarily particularly important as judged by the magnitude of the effect; highly significant results should therefore not automatically be interpreted as reflecting large effects. For this reason, non-statistically significant values with large size effects were also mentioned. Another reason for using caution when interpreting the results is that attention and motor activity are terms that comprise a number of factors. During the course of the research, some but not all of these concepts were tested.

The difference between objective and subjective data should also be mentioned here. In a large number of studies pertaining to AD/HD, results rely solely on the subjective opinion of caregivers, teachers and parents. In this study both objective and subjective measurements were included.

The following general research results were obtained:

- Improvements were noted on some aspects of attention. These improvements included Visual Vigilance, Auditory Vigilance, and Internal Distractibility over a short period of time (11 weeks). Internal Distractibility also improved after a longer periods of time. These were standardised tests and objective in nature. Not all the aspects of attention showed significant improvement.
- Significant improvements were noted on the subjective questionnaires included in the research. These included the Attention components of the Amen and Temperament Questionnaires.
- A smaller improvement was noted on the subjective motor control aspects tested, although it should be remembered that hyperactivity is a difficult construct to evaluate using objective standardised measuring instruments. Auditory Response Control was the only aspect measured that showed significant improvement.
- Subjective teacher ratings of motor control showed significant improvement.

- Significant improvement was noted in the second control group in Reading and Maths multiplication. The first experimental group showed significant improvements in terms of Maths addition. Both groups also showed improvements in Visual Discrimination.
- There were non-statistical improvements that showed a large effect size in Mathematical subtraction and division, as well as Spelling.
- Consistent results were obtained for academic improvement in both the experimental and the control groups. These consistent results warrant the rejection of the null hypothesis on the third stated hypothesis.

Chapter 8

Conclusion and Recommendations

1. Introduction

This thesis addressed the need for experimental research into an alternative intervention protocol to aid learners with attentional difficulties and heightened motor activity. After an extensive literature study into the traditional sources and methods used to identify, diagnose and treat the symptoms of AD/HD, the non-traditional literature was presented to investigate the possible root causes of these symptoms. This chapter summarises the research study as well as the results obtained.

2. Outline of Thesis

Chapter 1

In this chapter, the basic aim of the research was stated, together with the reasons for the choice of topic and additional objectives for the research. The research question was posed and hypotheses generated. The research method was discussed in terms of the literature study, quantitative and qualitative data. Key terms were also presented and defined.

Chapter 2

Chapter 2 focused on the traditional conceptualisation of AD/HD. This included the historical background of the diagnosis of AD/HD from the early 1900s. Current clinical diagnostic procedures were explored, including the DSM-IV and the ICD-10. The prevalence of AD/HD was presented in terms of estimates, gender distribution and familial patterns. Suggested recording and diagnostic procedures were explored as well as associated features and comorbid disorders. Some possible causes according to the DSM-IV were included, together with the developmental course and early symptoms.

Traditional treatments methods, such as psychostimulants, behavioural modification, cognitive control and the Feingold Diet were explored.

The conclusion drawn from this chapter is that AD/HD is a condition that has received much attention in the literature and in research. Huge amounts of money have been invested in research on the subject, and thousands of articles and books have been published; yet it remains a heated topic with many unanswered questions. Many researchers believe that AD/HD is not curable, and many more have tried to prove that the symptoms are not conclusive evidence of a mental disorder. Medication and behavioural modification strategies have largely aimed at alleviating the symptoms of AD/HD rather than treating the underlying cause.

Chapter 3

Chapter 3 explored attention and heightened motor activity as constructs. These constructs were defined and different theories of attention and motor activity were presented from traditional literature. One main aim of this chapter was to explore the two constructs in terms of neuroanatomy, neurochemistry and neurophysiology. Exploration of the constructs in these terms permits insight into the possibility of different underlying causes of the symptoms of AD/HD as they present themselves in the lives of children.

The information presented in this chapter illustrated the need to view AD/HD within a broader framework. The undeniable connection between the body and the brain (body/brain) presented itself in the interaction in the effect of movement on the brain (releasing neurotransmitters, growth of pathways through movement, plasticity) and the effect of the brain on movement (orchestrating movement). The role of the chemical components (both in nutrition and supplementation) were highlighted, and ultimately, the interconnectedness of neuroanatomy, neurochemistry and neurophysiology were questioned.

Chapter 4

Chapter 3 allowed a broader view of attentional difficulties and heightened motor activity. This broader view resulted in broader questions being asked about the true nature of the symptoms of AD/HD, and the change in perspective from diagnosis or

labelling to the need to investigate further in a search for the true underlying cause of these symptoms.

An alternative way of looking at the presenting symptoms was explored. A possibly more responsible approach to AD/HD may be to view the relationship between the cause of disorders and symptoms. This new, broader frame of reference allowed the design of an ecosystemic model to view, understand and intervene in attentional difficulties and heightened motor activity.

The remainder of the chapter delved into aspects of neurodevelopment, including specificity and plasticity. Physiological stress and its effects on neurodevelopment were explored, as was information processing as a neurophysiological aspect of neurodevelopment. Neurodevelopmental delays were discussed in view of the presented material, together with some of the underlying sensory-motor aspects, their development and contributions to children's growth and development. These included the tactile, olfactory, gustation, vestibular, and kinaesthetic systems, muscle tone, proprioception, and the visual and auditory systems. The interactions between and modulations of these systems were also discussed.

Internal and external biochemical ecosystems were explored as possible underlying causes of attentional difficulties and heightened motor activity. These included environmental pollutants such as lead, mercury and phenols, and possibly dangerous metals found in vaccinations. The consequences of deficiencies in essential vitamins, fatty acids, minerals, and amino acids were discussed in terms of their implications for learning, behaviour and development. Finally, aspects such as allergies, hypoglycaemia, genetic immune disorder, recurrent infections and dysbiosis were examined.

The above constitute evidence that supports the notion that inattention and heightened motor activity may stem from a number of biological factors. In addition, this information provided clues to alternative treatment modalities.

Chapter 5

The previous chapter outlined an alternative, ecosystemic model of understanding the symptoms of inattention and heightened motor activity. Chapter 5 provided an outline of a proposed intervention programme based on such an holistic model.The intervention design included sound therapy, EEG neurotherapy, a neurodevelopmental programme and nutrition.

Chapter 6

This chapter detailed the research methodology, based on an experimental research design. A true experimental design was followed, using a quasi-experimental design including an experimental and a control group. In addition, a one-group pretest-posttest design was used as the control group also received the intervention after the experimental group, providing the opportunity for additional data collection. The sample population was described, and methods of data collection, measuring instruments, interviews, screenings and assessments employed were discussed. Finally, the analyses of the results and the types of tests run on the data were presented.

Chapter 7

This chapter discussed the research results. This included a description of the sample in terms of demographic factors, family structure and historical background. The data presented showed that the two groups (experimental and control) were equal in terms of age, gender and intelligence. Improvements on some aspects of attention were noted, while a smaller improvement was noted on motor activity. A consistent improvement in academic performance occurred, and so the the null hypothesis may be rejected.

3. Research Results

The following hypotheses were stated at the beginning of the research.

3.1 Attention

Hypothesis 1:

H0: Disturbances in **attention** will not improve after an ecosystemic intervention.

H1: Disturbances in **attention** will improve after an ecosystemic intervention.

The following results were obtained:

- Statistically significant improvements were noted on some aspects of attention over a short period of time (11 weeks), and based on standardised, objective measures. These improvements included Visual Vigilance, Auditory Vigilance, and Internal Distractibility. Internal Distractibility improved further after a longer period of time.
- Significant improvements were also noted on the subjective questionnaires administered to the respondents' parents. These included items on the Attention component of the Amen Questionnaire and on the Temperament Questionnaire.
- Attention is a multidimensional construct. Not all aspects of attention showed significant improvement following the intervention. For this reason the stated hypotheses can neither be accepted nor rejected.

3.2 Motor Activity

Hypothesis 2:

H0: Disturbances in **motor activity** will not improve after an ecosystemic intervention.

H1: Disturbances in **motor activity** will improve after an ecosystemic intervention.

The following results were obtained:

- Improvements which were not statistically significant were noted on components of motor activity in the experimental group.
- Significant improvements were noted in the second experimental group, as were general improvements with large effect sizes.



- Improvement was also noted following the longer term intervention, and although it was not statistically significant, a large effect size was noted.
- The teacher noted a significant improvement in hyperactive behaviour in both experimental groups.
- Once again, not all the aspects of motor activity improved. For this reason none of the hypotheses can be either accepted or rejected.

3.3 Academic Performance

Hypothesis 3:

H0: **Academic performance** will not improve after an ecosystemic intervention.

H1: Academic performance will improve after an ecosystemic intervention.

The following results were obtained:

- On measures of maths skills, children in the first experimental group improved significantly more than children in the control group.
- The second experimental group showed significant improvements with large effect sizes on reading, maths skills and spelling.
- Results obtained by the second experimental group verify the results obtained in the first experimental group.
- A long-term effect was seen with significantly improved results in Visual Discrimination and Maths subtraction.
- It can therefore be concluded that the intervention resulted in a statistically significant improvement in academic performance with regards to reading, maths ability and visual discrimination. Therefore, the null hypothesis on the third stated hypothesis may be rejected.

3.4. Conclusion

Some aspects of attention showed significant improvements following the intervention, including visual vigilance, auditory vigilance, and internal distractibility. Fewer aspects of motor activity showed improvement, although the teacher observation measure revealed some improvement in hyperactivity. Finally, significant

improvement was found following the intervention on academic skills, specifically reading (including visual discrimination), math skills and spelling.

4. Recommendations

This section discusses deficits in the study, and makes recommendations for future research.

4.1 Deficits in Study

4.1.1 Small sample size:

The small sample size (n=12) is a drawback in the study as the results are not necessarily generalisable. The fact that the class from which the sample was drawn consisted of only 12 children limited the ability to draw a larger sample.

4.1.2 Placebo effect

To counter a possible placebo effect, the control group needed some form of contact with the researcher. Due to the small sample size, this was not possible. To circumvent this difficulty, children were called out of the class individually while the rest of the experimental group and the whole control group remained in the classroom and participated in normal classroom activities.

4.1.3 Scope of the research

Although the literature study indicated a vast number of possible underlying causes of the symptoms of AD/HD, not all could be addressed in this study. Although indicated as being necessary in an ecosystemic approach, including a large number of intervention models in the study made it difficult to establish the contribution of each component of the ecosystemic model to the improvements.



4.1.4 Motivational aspect

All children find it easy to be actively involved in novel activities. However, there are few measuring instruments that test a single aspect of learning and behaviour in multiple different ways, and none were available for certain purposes of this study. The repeated use of the same measures may therefore have contributed to a lack of motivation among the respondents in the posttest (point C).

4.1.5 Change in teachers

After the initial orientation and presentation of the research design, the researcher was informed that the respondents' teacher was to be replaced. The emotional effect of this change on the respondents was not considered. The effects of this change, together with the simultaneous introduction of the research schedule and programme, may therefore have influenced the children's performance.

4.1.6 Parental involvement

In general, parents were not as involved as may have been the case if these children had received the intervention in in a private practice setting. Nutrition and dietary recommendations were made according to the individual profiles obtained, but the extent of adherence to these recommendations was difficult to ascertain.

4.1.7 Limited reference material

Due to the nature and scope of the study, it was difficult to find existing, relevant research. There is a dearth of experimental research dealing with alternative methods of interventions and only a limited number of references were available. Hopefully, this study will, to some extent, address this need for future research.

4.1.8 Non-standarised questionnairs

Currently, these are no South African standardised questionnaires pertaining to the assessment of AD/HD. For this reason, other questionnaires have been used. As

with all qualitative work it is possible that the results may have been influenced by the use of non standardised instruments. Additionally, it is uncertain as to weather the research method, data, findings are correct in all aspects. There is a possibility that data obtained may have been more positive than the reaclity. Further research needs to be conducted to validate this study.

4.2 Recommendations

Recommendations for subsequent research include the following:

- A larger sample size should be used, and measured over a longer period of time.
- The placebo effect should be fully accounted for.
- The different intervention techniques could be tested separately in order to obtain the most effective and possible combinations of the interventions used.
- Different methods of assessment could be investigated as children lose interest in tests that they have to perform more than once.
- Attention should be given to possible emotional components that may influence behaviour.
- Parents could be encouraged to be more involved to extend the benefits of the intervention.
- This research could be seen as exploratory in its consideration of alternative methods of addressing attentional difficulties and heightened motor activity. The literature study suggests that an ecosystemic approach may be more comprehensive and more useful than the traditional medical models, and therefore further research into alternative methods is encouraged.

5. Final Conclusion

The overreaching aim of this thesis was to create the awareness that attentional difficulties and heightened motor activity can and should be viewed from a number of different perspectives. This viewpoint is necessary as possible underlying causes of AD/HD symptoms could arise from various different sources. As more and more children are currently diagnosed and labelled with AD/HD, the need is becoming more urgent to investigate more and better solutions. In this thesis, an ecosystemic

approach was proposed and tested. Although this model is in its beginning stages and deserves further attention and research, it promises a better understanding of the symptoms of AD/HD. Hopefully this thesis provides a beginning point for dialogue and new thinking, to allow professionals to consider new ways of understanding children and their perplexing presenting symptoms.

Appendix A

Intake Questionnaire

Biographic Information:

Client's FULL name & surname:
Date of birth (day)(month)(year) ID No M/F
Home address: Street and suburb
Town/city Province Postcode
Postal address (must be completed)
Postcode
Parents or guardian (if applicable)
Client/parent telephone:
Day: ()
Evening: ()
Cell (mother) Cell (father)
Ask for:
Best time to call:
E-mail address:
Fax: ()
Medial Aid Society
FULL name of member:
I.D. NoMedical aid tel no:
Address of Med. Aid. Soc.
Code
Payment Cash □ Cheque □
Client's Current School or Employer: (Name):
Grade:or Occupation
Teacher's name and contact number:
Address: Street and suburb/Box
Town/cityProvincePostcode
<u>Referred by</u> (name):
Address: Street and suburb/Box

 Town/city......Province.....Postcode......

 <u>Current Physician(s)/Therapist(s)</u>:

 Name(s)......Speciality.....

 Name(s)......Speciality....

If medication is taken for ADD/ADHD, please *do not* take the medicine prior to the assessment. *Do* bring the medication with you so that it can be taken in the event that it is deemed necessary to complete the assessment. If the client has prescription lenses, please bring them to the assessment. If the client or caregiver suffers from hypoglycaemia (requires frequent feeding), please bring an appropriate snack to the assessment. Please be sure to dress comfortably. During the presentation and exercise check we will be performing a variety of movement forms.

Major areas of concern:

Auditory perception/processing dysfunction

- Attention Deficit Disorder (ADD)
- □ Autism/Pervasive Developmental Disorder (PDD)
- Bipolar Disorder
- $\hfill\square$ Closed head injury
- Conduct Disorder
- □ Depression
- Developmental delay
- Dyspraxia
- General learning disability
- □ Hyperactivity
- □ Memory Disorder
- Seizure Disorder
- □ Sensory Integration Disorder
- Specific language dysfunction
- Tourette's Disorder
- □ Visual perceptual motor dysfunction

□ Visual Processing Disorder / Scotopic Sensitivity

□ Other:....

Specific Concerns

Please mention any specific concerns:

<u>Enclosure</u>: Please attach a copy of particularly relevant reports, examples of schoolwork and other items, which demonstrate your concerns.

Family History

• Family structure (Please indicate)

ſ	Intact	Single	Divorced	Remarried	Other
		parent			

- Is the child an adopted child?
 If YES, at what age did the child join the family?
- Is the child a foster child?
 If YES, at what age did the child join the family?
- Number of children in family

Name	Gender	Age	Grade	Progress at school

- Other inhabitants in household
- Hereditary illness in the family

Birth History

- Where were the parents working before conception? Father Mother
- Were either of you in contact with any heavy metals or chemicals before conception, during pregnancy or in your child's early childhood?
- State any problems during pregnancy (e.g. serious illness, emotional tension, imminent abortion).

Describe.

- Was it a planned pregnancy?
- Did the mother experience any viral infection (e.g. influenza, measles) during the second trimester (3-6 months) of pregnancy?
- Name any difficulties with previous pregnancies.
- Did you put new carpets in your house while you were pregnant?
- Did you renovate your house while you were pregnant?
- Did you paint any part of you house/office while you were pregnant?
- Was the mother bedridden for any period of the pregnancy? When?

How long?

- Reason?
- Did the mother smoke during pregnancy?
- Did the mother consume any alcohol during pregnancy?
- Was the mother subjected to "passive smoke" during pregnancy?
- Was any other health or psychological problems experienced during pregnancy?
- Did the mother use any drugs during pregnancy?
- Age of mother at birth.
- Duration of pregnancy.
- Premature Time

• Birth weight

- APGAR score
- Birth process (please indicate X):

Normal	Caesarean Section
Without drugs	
With drugs	
Epidural	Epidural
Forceps	Full anaesthesia
Suction	
Short delivery	
Long delivery	

- Labour induced or spontaneous?
- Was it a breech or headfirst birth?
- Other aspects during and after the birth (please indicate X)
 - o Lack of oxygen
 - o Foetal distress
 - o Cord around neck
 - o Jaundice
 - o ICU Duration
 - o Other
- Was the baby breastfed?
 For how long?
- Age when solids were introduced?
- What type of food was introduced?
- Any reaction to any of the solid foods?
- If not breastfed, what substitute was used?

- Any reaction to any of the products used?
- How was the child's sucking?
- Has your child had measles, mumps, chicken pox, rheumatic fever, meningitis, or other significant diseases?

Reason



Developmental History

- Type of baby (please indicate X)
 - □ Cried a lot
 - □ Calm
 - □ Colic
 - Restless
 - □ Bad sleeper
 - \square Other
- Allergies

Past	Gluten	Dairy	Maize
	Eggs	Pollen	Animal dander
	House dust	Moulds	Oranges
	Other		
Present aller	gies		

- Type of treatment for allergies
- Did your child receive all the recommended innoculations?
- How did your child respond to inoculations?

Please indicate any accidents, injuries, traumas, hospitalisation (duration), operations, fever attacks and illness, head injuries or concussions.

Age	Incident	Duration

 Has your child had any serious illnesses resulting in long periods of bed rest (i.e. longer than one week)

Age	Reason	Duration

 Please supply information in respect of specialists your child has visited in view of the above

Specialist	Name	When	and	How	Reason	Success
		long				rate

- Is your child on any medication at present?
 If YES, state the reason, name of medication and dosage?
- Is your child on any supplementations at present?
 If YES, state the reason, name of supplementation and dosage?
- Is your child in contact with animals and or pets on a regular basis?

Motor Development

How would you describe your child's motor development?

Early	Normal	Slightly late	Severely
			delayed

At what age could your child do the following?

Activity	Age	How (if other than the norm)
Sit		
Crawled		
Pulled up against		
furniture		
Walk		
Eat on own		



Dress self	
Toilet training	
Riding a tricycle	
Sleep in own room	
Riding a bicycle	

- Does your child fall often?
- Did your child experience any problems with toilet training?

Speech and Language Development

- Did your child's speech develop normally?
- Is/was orthodontic treatment indicated?
- Was your baby very quiet, or did a variety of baby sounds occur?
- Indicate the rate of your child's language development

		Early	Normal	Slightly delayed	Severely delayed
--	--	-------	--------	------------------	------------------

- At what age was your child able to express him/herself?
- Please indicate if applicable (X)
 - $\hfill\square$ Does not pay attention (listen) to instruction 50% or more of the time
 - History of hearing loss
 - □ Is bothered by high pitched noises
 - Needs loud music to be able to concentrate on homework
 - □ Speaks in a loud voice
 - Hums or makes "white noises"
 - Does not learn well through use of the auditory channel
 - □ Demonstrates below average performance in one or more academic area(s)

□ Has difficulty following verbal directions – often necessary to repeat instructions

- □ Cannot always relate what is heard to what is seen
- □ Cannot attend to auditory stimuli for more than a few seconds
- □ Frequently misunderstands what is said
- □ Says "Huh?" and "What?" at least five or more times per day
- □ Forgets what is said in a few minutes

- □ Has a short attention span
- Daydreams attention drifts not with it at times
- Easily distracted by background noise
- □ Is considered to have Autism, Dyslexia, Pervasive Developmental Disorder,
- Central Auditory Processing Disorder, Asperger's Syndrome or Attention
- Deficit Hyperactivity Disorder (ADHD) or ADD.
- □ Experiences problems with sound discrimination
- □ Has "startle" response to sudden sound or movement
- Notices sounds before others do
- □ Gives unusual descriptions of sounds, auditory stimulation or sensation
- □ Constant humming or audible self-talk
- □ Needs frequent "quiet time" to regain mental energy and composure
- □ Does not comprehend many words, does not grasp verbal concepts appropriate for age/grade level
- □ Has a language problem (morphology, syntax, vocabulary, phonology)
- □ Has an articulation (phonology) problem
- Has a history of ear infection(s)

Information regarding ear infections:

Left ear

Right ear

- How many times Ages when it occurred Date of last infection Did the ears drain during infection? Grommets? How many times? Are the grommets still in the ear?
- Has the child's hearing been tested, and if YES, at what age and what were the results?
- Are there any voice problems and if YES, please indicate?
- Does your child experience any swallowing problems?

 Does the child suffer from chronic upper respiratory problems (e.g. sinus, bronchitis) or has he/she suffered from these in the past?

Sensory Motor Development

- Do you suspect vision problems?
- Were your child's eyes tested recently? If YES, what were the results?
- What hand does your child prefer?
- Reaction to the following
 - Heights
 - \square Escalators
 - Elevators
 - □ Climbing a tree
 - Flying
 - □ Swing
 - $\square \ Slide$
 - □ Merry go round

Emotional and Interpersonal Development

- Describe your child's interactional relationships with:
- Father
- Mother
- Siblings

- Peers
- Teacher
- Discipline: who is the figure of authority and how do you discipline your child?

Scholastic History

School attended	Age	Duration	Comments on progress

- Would you say that you child was ready for school?
- Were developmental or other concerns experienced during preschool years?
- If YES, indicate the nature of the concern at the preschool

□ Fine motor	Visual perception
Gross motor	Auditory perception
	Behaviours
Balance	□ Social
Emotional	□ Other

• Has your child received any form of therapy for the abovementioned?

Previous help	Reason	Duration	Degree of
			success
Physiotherapy			
Occupational therapy			
Psychotherapy/play			
therapy			
Speech therapy			
Remedial therapy			
Vision therapy			
Auditory therapy			
Other			

 Has the child experienced any problems since Grade one with any of the following (please indicate X and describe if need be):

Visual perceptual problems	Social problems
Auditory perceptual problems	Concentration/attention
□ Slow pace of work	Comprehension
Mathematics	□ Other
□ Reading	U Writing

 Has your child been diagnosed with or do you suspect any of the following (please indicate X and describe if need be):

	Conduct disorder/oppositional
	defiance
	Tourette's syndrome
Dyslexia	□ Autism
Dyspraxia	□ Asperger's syndrome
	Cerebral palsy
Low muscle tone	Vision problems (far-sightedness,
	astigmatism, strabismus)
Visual perceptual problems	□ Hearing loss
Auditory perceptual problems	Developmental delay
Memory disorders	Speech and articulation problems

Epilepsy	Problems with receptive and
	expressive language
□ Gifted	□ Brain injury
Learning disability	Obsessive compulsive behaviour
Other	

Has any member of the family experienced the same or similar issues?

Family member	Diagnosis

General Health

- Any other health problems?
- How frequently does your child use antibiotics?
 Past?
- Were any probiotics given with the antibiotics?
- Circle the appropriate point score for questions you answer YES

During the two years before your child was born, were			30
you bothered by recurrent vaginitis, menstrual			
irregularities, premenstrual tension, fatigue,			
headaches, depression, digestive disorders or "feeling			
bad all over"			
Was your child bothered by thrush? (Score 10 if mild	10	20	
and 20 if severe)			
Was your child bothered by frequent diaper rashes in	10	20	
infancy? (Score 10 if mild and 20 if severe)			
During infancy, was your child bothered by colic and	10	20	
irritability lasting more than 3 months? (Score 10 if			
mild and 20 if moderate to severe)			
Are his/her symptoms worse on damp days or in		20	

damp or mouldy places?			
Has your child been bothered by recurrent or			30
persistent "athlete's foot" or chronic infections of his			
skin or nails?			
Has your child been bothered by recurrent hives,	10		
eczema or other skin problems?			
a. 4 or more courses of antibiotic drugs during the			60
past year? Or has he received continuous			
"prophylactic" courses of antibiotic drugs?			
b. 8 or more courses of "broad-spectrum" antibiotic			40
(such as amoxicillin, Keflex, Septra, Bactrum, or			
Ceclor) during the past three years?			
Has your child experienced recurrent ear problems?		20	
Has your child had grommets inserted in his/her ears?	10		
Has your child been labelled "hyperactive"? (Score 10	10	20	
if mild and 20 if moderate or severe)			
Is your child bothered by learning problems (even	10		
though his/her early development history was			
normal)?			
Does your child have a short attention span?	10		
Is your child persistently irritable, unhappy and hard to	10		
please?			
Has your child been bothered by persistent or	10	20	30
recurrent digestive problems, including constipation,			
diarrhoea, bloating or excessive gas? (Score 10 if			
mild, 20 if moderate and 30 if severe)			
Has he/she had persistent nasal congestion, cough	10		
and/or wheezing?			
Is your child unusually tired or unhappy or	10	20	
depressed? (Score 10 if mild and 20 if severe)			
Has he been bothered by recurrent headaches,	10	20	
abdominal pain, or muscle aches? (Score 10 if mild			
and 20 if severe)			
Does your child crave sweets?	10		
--	----	----	----
Does exposure to perfume, insecticides, gas or other			30
chemicals provoke moderate to severe symptoms?			
Does tobacco smoke really bother your child?		20	
Do you feel that your child isn't well, yet diagnostic	10		
tests and studies haven't revealed the cause?			

Biochemical Information

 Please indicate with an X all that is applicable even if you have already indicated the item – as applicable for the patient

Section 1	Section 2		
Feeling "unreal"	Excessive thirst		
Hearing your own thoughts	Chronic fatigue		
Anxiety and inner tension	Dry or rough skin		
Inability to think straight	🗆 Dry hair		
Suspicious of people	Loss of hair or dandruff		
Good pain tolerance	Eczema, asthma or joint aches		
Seeing or hearing things	Dyslexia, or learning difficulties		
abnormally	□ Hyperactivity		
Having delusions or illusions	Depression or manic depression		
Loose bowel or skin problems at	Urinates a lot		
onset of mental health problems	Soft brittle nails		
Tendency to overweight	Night blindness/bad sight at night		
Frequent mood swings	Headaches/strain on eyes		
	Reading problems		
Section 3	Section 4		
Anxiety, extreme fears or paranoia	Nausea or constipation		
Phobias	White spots on fingernails		

Poor concentration and confusion	Pale skin that burns easily
Poor memory	Frequent colds and infections
Angry and aggressive behaviour	Stretch marks
□ Hyperactivity	Crowded upper teeth
Emotional instability	Irregular menstruation
Headaches and migraines	□ Impotency
□ Joint pain	Poor tolerance to alcohol
Nervousness	Poor dream recall
Section 5	Section 6
Headaches or migraines	Headaches and migraines
Sneezes in sunlight	$\hfill\square$ Watery, itchy eyes, red eyelids or
□ Cries, salivates or feels nauseated	dark rings under the eyes
easily	□ Itchy ears, frequent ear infections or
□ Abnormal fears, compulsions and	ringing in the ears
rituals	Excessive mucous, stuffy nose or
Light sleeper	sinus problems
Fast metabolism	 Excess sweating and strong body
Depression and suicidal thoughts	odour
Produces a lot of body heat	Indigestion or bloating
Little body hair and lean build	Constipation or diarrhoea
□ Large ears and long fingernails and	History of eczema, asthma
toes	$\hfill\square$ Joint or muscle aches or pains or
Inner tension	arthritis
Shy or over-sensitive as child	D Mental health symptoms are often
Seasonal allergies (e.g. hay fewer)	worse after eating
Obsessive or compulsive	
tendencies	

Section 7	Section 8
Craves sweet foods	□ Irritability
□ Craves stimulant foods such as	Nervousness or anxiety
tea, coffee, cola and chocolates	Extreme fears
Difficulty concentrating	Raised blood pressure
Palpitations or blackouts	Rapid or irregular heart beat
Fainting or dizziness or trembling	Insomnia
Excessive or night sweats	Cold hands and feet
Excessive thirst	Excessive sweating
Frequent mood swings	Teeth grinding
Forgetfulness and confusion	Headaches or migraines
□ Tendency to depression	Muscle tension
Anxiety and irritability	Restlessness
Feeling weak	Seeing or hearing things
Aggressive outbursts and crying	Depression
spells	Difficulty concentrating
Craving for sweets or stimulants	Short attention span
Drowsiness after meals	Lack of drive/motivation
Chronic fatigue	Rarely initiates or completes tasks
	Can't deal with stress
	Socially withdrawn
	Frequently tired
Section 9	Section 10
□ History of colic, eczema, asthma,	Physical or mental fatigue or
rashes or ear infections	lethargy
Daily mood swings	Depression or irritability
Deep depression for no particular	Dry skin and/or hair
reason	□ Intolerance to cold or cold hands
□ Frequent, rapid colds or blocked	and feet
nose	Constipation, gas, bloating or



Difficulty sleeping	indigestion		
□ Facial puffiness, circles or	Gains weight easily		
discolouring around eyes	Painful periods (if applicable)		
□ Hyperactivity	□ Muscle pain		
Dyslexia or learning difficulties	Poor memory		
Aggressive outbursts or crying	Sore throat, or nasal congestion		
spells			
Section 11	Section 12		
Depression	Poor dream recall		
Anxiety	Infrequent dreaming		
Aggressive or suicidal thoughts	Difficulty visualising		
Violent or impulsive behaviour	□ Dry mouth		
Mood swings	Poor memory or forgetfulness		
□ Obsessive or compulsive	Mental exhaustion		
tendencies	Poor concentration		
Sensitive to pain	Difficulty learning new things		
Craves sweet foods			
Sleeping problems			

Round the Clock Observation

Bedtime

- What time does your child go to bed?
- Is your child ready to go to bed or does he or she resist?
- Does your child have bedtime rituals (circling the bed, lining up shoes, etc.)?
- How impulsive is your child about these rituals?
- Does your child sleep alone or in the same room or bed as siblings or parents?
- Is there a gas or asbestos heater in your child's room? If so, is it an older heater? Is there any ventilation in the room?
- Does your child sleepwalk?

- Does your child grind his/her teeth during sleep?
- Is your child a quiet or restless sleeper?
- Is your child a heavy sleeper or easily awakened?
- Does your child get up frequently during the night to go to the bathroom?
- Does your child still wet his/her bed?
- Does your child frequently get up to drink water?
- Does your child get up to eat?
- Does your child snore heavily?
- Does your child have frequent nightmare or night "terrors"?
- Is your child's sleep pattern regular or irregular?
- Is it difficult to get your child to sleep?
- Is it difficult to wake your child when he/she is sleeping?

Morning

- What time does your child awaken?
- Does your child awaken refreshed or tired?
- Does your child eat breakfast? If so, what is it?
- Does it appear to affect your child's mood or energy level?
- Does he or she appear tired or sweaty, or regress behaviourally?
- What is your child's behaviour like in the morning?
- What is your child's energy like in the morning?
- Does your child eat a morning snack? If so, what is it? Does it appear to affect your child's mood or energy level?
- Does your child have tantrums in the morning? If so, at what time?

Afternoon

- At what time does your child eat lunch? What does he or she eat?
- Does your child take lunch, or buy something to eat from the tuck shop? Does your child trade lunch with other children?
- Does your child feel weak or irritable before lunch?
- Does your child feel good after lunch, or does your child appear tired or sweaty?
- What is your child's behaviour like in the afternoon?
- What is your child's energy level like in the afternoon?

- What is your child's mood like in the afternoon?
- Does your child eat an afternoon snack? If so, what is it? Does it appear to affect your child's mood or energy level?
- Does your child have tantrums in the afternoon? If so, at what time?

Late afternoon

- How does your child get home in the afternoon?
- What is your child's mood/energy level like in the afternoon?
- Does your child snack upon getting home? If so, what does he or she eat?
- How many hours does your child spend watching TV before dinner?
- What other activities does your child participate in before dinner (sport, computer games, video games, hobbies, etc.)
- How many caffeinated beverages (coffee, colas, tea, hot chocolate) does your child consume during the afternoon?
- What is your child's behaviour like immediately before dinner?
- Does your child have tantrums in the afternoon? If so, at what time?

Evening

- What does your child eat for dinner? How good is your child's appetite?
- What is your child's behaviour like immediately after dinner? Does your child feel good, or is your child fatigued or sweaty?
- How often does your child go to the bathroom in the evening?
- How much water, soda, or juice does your child drink during the evening?
- Does your child snack before going to bed? If so, what is it? Does it appear to affect your child's mood or energy level?
- How many hours does your child spend watching TV before going to bed?
- How many caffeinated beverages (coffee, colas, tea, hot chocolate) does you child consume during the evening?
- What is your child's behaviour like immediately before going to bed?
- Does your child have tantrums in the evening? If so, at what time?

Temperament

Plot each characteristic on the bipolar scale, either high or low or in between.										
				Act	tivity l	evel				
The amount of physical motion exhibited during the day										
Low	1	2	3	4	5	6	7	8	9	High
				Pe	rsiste	nce				
The	exter	nt of co	ntinuat	ion of I	behavi	our wi	th or w	vithout	interru	otion
Low	1	2	3	4	5	6	7	8	9	High
				Dis	tractik	oility				
The e	ase o	f being	interru	pted b	y sour	nd, ligh	it, etc i	unrelat	ed beh	aviour
Low	1	2	3	4	5	6	7	8	9	High
				Initi	al rea	ction				
Res	oonse	s to no	vel situ	ations	, whetl	her ap	proach	ing or	withdra	awing
Withdrawing	1	2	3	4	5	6	7	8	9	Approaching
				Ad	laptab	ility				
Т	he eas	se of ch	anging	g beha	viour i	n a soo	cial de	sirable	directi	on
Low	1	2	3	4	5	6	7	8	9	High
Mood										
-	The qu	uality of	emoti	onal e	xpress	ion, po	ositivel	y or ne	gative	у
Positive	1	2	3	4	5	6	7	8	9	Negative
				li	ntensi	ty				
	The	amoun	t of en	ergy ex	khibite	d in en	notiona	al expre	ession	
Low	1	2	3	4	5	6	7	8	9	High
Sensitivity										
The degree to which the person reacts to light, sound, etc.										
Low	1	2	3	4	5	6	7	8	9	High
Regularity										
The extent to which patterns of eating, sleeping, elimination, etc. are consistent or										
			inco	nsiste	nt from	n day to	o day			
Low	1	2	3	4	5	6	7	8	9	High

Sensory-Motor Aspects

Please indicate issues (past and present) in terms of severity, **1** being not an issue and **10** being a major issue.

	Present	Past	Comments
Clothing	Frequency / Severity	Issue	
1. Appears to be bothered by clothing in	1 2 3 4 5 6 7 8 9 10	yes/no	
general			
2. Is bothered by tags in shirts	1 2 3 4 5 6 7 8 9 10	yes/no	
3. Is bothered by seams in socks	1 2 3 4 5 6 7 8 9 10	yes/no	
4. Does not like stiff fabrics	1 2 3 4 5 6 7 8 9 10	yes/no	
5. Wears long sleeves or does not	1 2 3 4 5 6 7 8 9 10	yes/no	
remove jacket			
6. Refuses to wear synthetics	1 2 3 4 5 6 7 8 9 10	yes/no	
7. Other	1 2 3 4 5 6 7 8 9 10	yes/no	

	Present	Past	Comments
Motion	Frequency / Severity	Issue	
1. Suffers from motion sickness in	1 2 3 4 5 6 7 8 9 10	yes/no	
general			
2. Falls asleep in moving vehicle	1 2 3 4 5 6 7 8 9 10	yes/no	
3. Does not enjoy merry-go-round /	1 2 3 4 5 6 7 8 9 10	yes/no	
swings			
4. Wants to continue swinging, spinning	1 2 3 4 5 6 7 8 9 10	yes/no	
etc.			
5. Engages in a lot of jumping	1 2 3 4 5 6 7 8 9 10	yes/no	
6. Avoids most movement	1 2 3 4 5 6 7 8 9 10	yes/no	
7. Runs rather than walks	1 2 3 4 5 6 7 8 9 10	yes/no	
8. bumps into things / people in path	1 2 3 4 5 6 7 8 9 10	yes/no	
9. Exhibits balance problems	1 2 3 4 5 6 7 8 9 10	yes/no	
10. Engages in head banging, hand	1 2 3 4 5 6 7 8 9 10	yes/no	
flapping, etc.			
11. Other	1 2 3 4 5 6 7 8 9 10	yes/no	

Foods	Present Frequency / Severity	Past Issue	Comments
1. Is a fussy eater in general	1 2 3 4 5 6 7 8 9 10	yes/no	
2. Dislikes chewy foods	1 2 3 4 5 6 7 8 9 10	yes/no	

3. Dislikes foods or juices due to texture	1 2 3 4 5 6 7 8 9 10	yes/no	
4. Dislikes mixtures of textures in foods	1 2 3 4 5 6 7 8 9 10	yes/no	
5. Dislikes most vegetables	1 2 3 4 5 6 7 8 9 10	yes/no	
6. Eats a lot of sweets	1 2 3 4 5 6 7 8 9 10	yes/no	
7. Eats a lot of salt	1 2 3 4 5 6 7 8 9 10	yes/no	
8. Eats a lot of margarine, mayonnaise,	1 2 3 4 5 6 7 8 9 10	yes/no	
fried foods			
9. Eats a lot of chocolate	12345678910	yes/no	
10. Has a lot of milk, yogurt	12345678910	yes/no	
11. Drinks at least 4 cups of water daily	1 2 3 4 5 6 7 8 9 10	yes/no	
12. Has lots of citrus	1 2 3 4 5 6 7 8 9 10	yes/no	
13. Craves breads and pasta	12345678910	yes/no	
14. Other	1 2 3 4 5 6 7 8 9 10	yes/no	

	Present	Past	Comments
Lights	Frequency / Severity	Issue	
1. Is bothered by lights in general	1 2 3 4 5 6 7 8 9 10	yes/no	
2. Appears bothered by lights in supermarkets	1 2 3 4 5 6 7 8 9 10	yes/no	
3. Appears uncomfortable with fluorescent lights	1 2 3 4 5 6 7 8 9 10	yes/no	
4. Wants to wear a cap with a visor	1 2 3 4 5 6 7 8 9 10	yes/no	
5. Plays under tables, in "tents"	1 2 3 4 5 6 7 8 9 10	yes/no	
6. Prefers bright lights to dim lights	1 2 3 4 5 6 7 8 9 10	yes/no	
7. Sneezes upon entering bright sunlight	1 2 3 4 5 6 7 8 9 10	yes/no	
8. Other	1 2 3 4 5 6 7 8 9 10	yes/no	

	Present	Past	Comments
Grooming	Frequency / Severity	Issue	
1. Appears uncomfortable in water	1 2 3 4 5 6 7 8 9 10	yes/no	
2. Gets upset when needs to leave water	1 2 3 4 5 6 7 8 9 10	yes/no	
3. Gets hysterical when washing hair	1 2 3 4 5 6 7 8 9 10	yes/no	
4. Is extremely bothered by wet clothes	1 2 3 4 5 6 7 8 9 10	yes/no	
5.Seems uncomfortable when dirty,	1 2 3 4 5 6 7 8 9 10	yes/no	
sandy, etc.			
6. Does not appear to notice food on face	1 2 3 4 5 6 7 8 9 10	yes/no	
7. Complains about face washing	1 2 3 4 5 6 7 8 9 10	yes/no	
8. Complains about hair combing /	1 2 3 4 5 6 7 8 9 10	yes/no	
brushing			
9. Complains about nail cutting	1 2 3 4 5 6 7 8 9 10	yes/no	
10. Complains about haircuts	1 2 3 4 5 6 7 8 9 10	yes/no	

11. Complains about tooth brushing	1 2 3 4 5 6 7 8 9 10	yes/no	
12. Other	1 2 3 4 5 6 7 8 9 10	yes/no	

	Present	Past	Comments
Human touch	Frequency / Severity	Issue	
1. Does not like light touch	1 2 3 4 5 6 7 8 9 10	yes/no	
2. Seeks deep touch / hugs /	1 2 3 4 5 6 7 8 9 10	yes/no	
compression			
3. Is ticklish	1 2 3 4 5 6 7 8 9 10	yes/no	
4. Hits, pushes, kicks others	12345678910	yes/no	
5. Has difficulty quitting rough-house play	1 2 3 4 5 6 7 8 9 10	yes/no	
6. Needs to sit on, snuggle up to others	1 2 3 4 5 6 7 8 9 10	yes/no	
7. Complains about pain (falls, cuts, etc.)	1 2 3 4 5 6 7 8 9 10	yes/no	
8. Other	1 2 3 4 5 6 7 8 9 10	yes/no	

	Present	Past	Comments
Odours	Frequency / Severity	Issue	
1. Is bothered by strong odours	1 2 3 4 5 6 7 8 9 10	yes/no	
2. Brings food to nose to smell before	1 2 3 4 5 6 7 8 9 10	yes/no	
tasting			
3. Smells most objects	1 2 3 4 5 6 7 8 9 10	yes/no	
4. Smells people's hair and clothes	1 2 3 4 5 6 7 8 9 10	yes/no	
5. Is bothered by food cooking in	1 2 3 4 5 6 7 8 9 10	yes/no	
cafeteria			
6. Appears insensitive to odour in general	1 2 3 4 5 6 7 8 9 10	yes/no	
7. Has stuffed nose, is mouth breather	1 2 3 4 5 6 7 8 9 10	yes/no	
8. Other	1 2 3 4 5 6 7 8 9 10	yes/no	

	Present	Past	Comments
Sleep	Frequency / Severity	Issue	
1. Required being rocked to go to sleep	1 2 3 4 5 6 7 8 9 10	yes/no	
as baby			
2. Upon awakening, has difficulty	1 2 3 4 5 6 7 8 9 10	yes/no	
walking, eating			
3. Has trouble falling asleep in general	1 2 3 4 5 6 7 8 9 10	yes/no	
4. Has trouble sleeping with background	1 2 3 4 5 6 7 8 9 10	yes/no	
noise			
5. Has trouble sleeping with lights on	1 2 3 4 5 6 7 8 9 10	yes/no	
6. Needs music / tapes to fall asleep	1 2 3 4 5 6 7 8 9 10	yes/no	
7. Has trouble falling asleep in the dark	1 2 3 4 5 6 7 8 9 10	yes/no	

8. Awakens frequently in the night	1 2 3 4 5 6 7 8 9 10	yes/no	
(Reason?)			
9. Falls out of bed	1 2 3 4 5 6 7 8 9 10	yes/no	
10. Moves in bed quite a lot while	1 2 3 4 5 6 7 8 9 10	yes/no	
sleeping			
11. Sleepwalks	1 2 3 4 5 6 7 8 9 10	yes/no	
12. Needs other person in bed in order to	1 2 3 4 5 6 7 8 9 10	yes/no	
sleep			
13. Dislikes top sheet / cover on bed	1 2 3 4 5 6 7 8 9 10	yes/no	
14. Other	1 2 3 4 5 6 7 8 9 10	yes/no	

Name of Person Filling Form

.....

Relationship to Client

Date

Sample Permission Letter

Dear Parent

Thank you for your willingness to conduct this interview in order to explaine in more detail the research to follow as well as to obtain more information about your child. As discussed during our initial orientation session, Centurion Remedial Academy has agreed to allow me to conduct research on doctoral level under the Universaty of South Africa (UNISA) on their presesis with the Grade 4 and 5 children.

The class will be devided into two equal groups (gender, age, intelligence). Groups 1 will receive therapy while the second group will receive no intervention during the experimental phase. It would be appreciated if you could refrain from any additional intervention programmes during this period as we would like to be able to take all known aspects into consideration when we do our analysis of the data. The second group will receive the same therapy after the first experimental phase. In other words; all children will eventually receive the same therapy. The therapy will be discussed with parents and suggestions concering diet and supplementation will be given.

By signing this permission letter, you as parent of agree to the following:

- I understand that particioation in voluntary
- I understand that this research is part of a doctoral study (Unisa) and that the results of the research may be published. I also understand that, by giving permission I undertake to support the research until completed
- I undertake to follow recommendations made by the researcher as far as possible

I, parent of give permission to Beulah van der Westhuizen to include my child as part of the research study conducted at CRA.

.....

Signature

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