

## **CHAPTER ONE: INTRODUCTION**

### **1.1. GENERAL INTRODUCTION**

Attention Deficit Hyperactivity Disorder (ADHD) is one of the most commonly diagnosed disorders of childhood and accounts for the most referrals made to childhood psychiatric practitioners. The characteristic symptoms of this behaviour disorder are inattention, impulsivity and possibly hyperactivity (Lerner, 1993). Allopathic treatment of this disorder involves the use of central nervous system (CNS) stimulants, which may produce unwanted side effects and in some children may produce no response at all (Barkley, 1998).

Homoeopathic research studies concerning the treatment of ADHD have included the use of various Homoeopathic complex preparations such as Selenium Homaccord®, Cerbo® and Nerva 2® (Smith, 2001; Strauss, 1998).

*Phosphorus* is a homeopathic simplex remedy that is prescribed for symptoms that are diagnostic of ADHD, namely difficulty concentrating, hypersensitivity to external impressions, excitability, restlessness, fidgeting and an inability to sit or stand still for a moment (Vermeulen, 1997).

### **1.2. AIM OF THE STUDY**

The aim of this study is to determine the effect of *Phosphorus* 6CH in alleviating the symptoms of ADHD.

## **CHAPTER TWO: LITERATURE REVIEW**

### **2.1. ATTENTION DEFICIT HYPERACTIVITY DISORDER (ADHD)**

Early research on Attention Deficit Hyperactivity Disorder, (ADHD), was done by English paediatrician George Still. In 1902, a group of his patients, mostly boys, were noted to have difficult behaviours that had started before the age of eight. Most were inattentive, overactive and resistant to discipline. They had poor control of inhibition and were aggressive with “a lack of moral control”. Still considered the disorder to be inborn or due to a birth-related brain injury and not caused by poor parenting or an adverse environment (Diller, 1998; Green & Chee, 1997).

Today, ADHD is a persistent problem that may change with growth from pre-school through adulthood and interferes with many areas of typical development and functioning in a child’s life (Clarke *et al*, 2002). The disorder consists of difficulties sustaining attention, inhibiting activity levels, and maintaining impulse control. Other common symptoms include explosive tempers, moodiness, and tendency to be accident-prone (Gimpel & Kuhn, 1998).

The symptoms of ADHD continue into adolescence in seventy-five percent of sufferers and studies with North American sample groups suggest that behavioural problems persist for fifty to sixty-five percent of those ADHD children on reaching adulthood (Barlow & Durand, 1995).

### **2.2. PREVALENCE**

There has been an increase in interest in ADHD as public awareness and referral rates for suspected ADHD have increased (Hutchinson *et al*, 2000). In fact, it accounts for the most paediatric referrals made to psychiatric practitioners (Barkley, 1998).

Prevalence studies show that an average of 3 to 9% of all young children in the general population all over the world show the behavioural symptoms related to ADHD (Scholte *et al*, 2001). It is estimated that ADHD affects one in ten South African children (Peters, 2000).

The onset of symptoms may range from birth to eleven years, with peak presentation by seven years of age (Barkley, 1998). However, symptoms of ADHD are often first seen in the pre-school years, usually three to four years of age. At this stage parents will often describe their children as being different when compared to children of the same age, or describe their children as naughty and very lively (Barlow & Durand, 1995).

ADHD affects both boys and girls, with males believed to outnumber females by an average of 6:1 (Barkley, 1998; Santrock, 1997). Since girls are not usually as aggressive and hyperactive as their male counterparts their attention deficit behaviours are not brought as easily to parents' and teachers' attention. However, it is thought that ratio of boys: girls with ADHD may actually be closer to 1:1 (Faller, 2002).

ADHD children have difficulties in areas of cognitive, motor and behavioural development and require large amounts of energy to maintain concentration, control their movements and impulses and adjust their behaviour accordingly (Barkley, 1998).

## **2.3. PATHOGENESIS**

### **2.3.1. Genetics and ADHD**

There is sound research evidence that proves there is a genetic element to the cause of ADHD. Most children with ADHD seem to have a close relative with a similar problem (Green & Chee, 1997). Recent research shows that if a parent has ADHD, the risk to the offspring is 57% (Biederman *et al*, 1995). The same was not found in adoptive parents of hyperactive children (Szatmari, 1992). In identical twins (i.e., with the same genetic make-up) there is an almost 90% chance that if one twin suffers from ADHD the other twin will too (Goodman, 1989; Green & Chee, 1997). In siblings (including un-identical twins) the risk is lowered to between 30 and 40% (Gillis, 1992). This indicates a significant risk associated with inheritance considering that the general population only has about a 2 to 7% chance of suffering from ADHD symptoms (Green & Chee, 1997).

In a research study by La Hoste (1996), it was found that a variant of the dopamine receptor gene D4 is more common amongst children with ADHD than those without ADHD. The DRD4 gene produces a receptor that is “subsensitive” to dopamine. This in turn may produce underactivity in dopamine pathways, leading to a child having problems with sustaining attention (Swanson, 1998). The correlation between a child’s hyperactivity scores and their father’s DRD4 risk allele is significant. Such a significant correlation was not found between the mother and the child, suggesting that the gene is more often transmitted on the paternal Y chromosome than on the maternal X chromosome (La Hoste, 1996).

Although it is possible for children to inherit “Type A” or “brilliant, but absentminded” characteristics from their parents, it is also possible for a child to exhibit violence, tantrums and excessive restlessness that seems unrelated to their family’s personalities. Some homoeopaths believe that the reason some children and adults suffer from ADHD and others do not, lies in susceptibility. If you ask the parent of a child with ADHD when he first noticed problem behaviours or tendencies in his child, he will likely say from infancy or toddlerhood. Homeopaths frequently observe that the predisposition to ADHD-like behaviour depends on the constitution of the individual from birth and may even be affected by the state of the parents prior to conception and during pregnancy (Reichenburg-Ullman & Ullman, 1996).

### **2.3.2. Brain Structure and Function**

At present the main focus in finding the cause of ADHD is in brain research. Four main areas have been considered - frontal lobe function, areas of over and under function, levels of activity and study of neurotransmitters. It has been established that the frontal lobes of the brain and their close connections under-function in subjects with ADHD. There is also evidence to show that the areas of the brain that collect auditory and visual input seem overloaded in ADHD, suggesting that they are being bombarded by a lot of unnecessary, inappropriate information (Green & Chee, 1997).

The brain shows interesting specialisation with regard to attention. When attention is focused on one attribute of a stimulus (such as size, or colour), there is activation of areas on both sides of the frontal lobe. However, when humans perform tasks

involving more than one attribute, activation is limited to the right (representational) side of the brain. This indicates that sustained attention is a function of the representational hemisphere (used for visuospatial relations) (Ganong, 1993).

In Magnetic Resonance Imaging (MRI) studies of ADHD subjects it was shown that the width of the right cerebral hemisphere had decreased to become equal to or smaller than that of the left cerebral hemisphere. Normally, the right frontal lobe is slightly larger than the left lobe (Filipek *et al*, 1997; Hynd *et al*, 1990). According to Ganong (1993) this would suggest that these children with relatively smaller right lobes, would experience a lesser ability to sustain attention facing more than one stimulus than those with normally larger right lobes.

The corpus callosum is a structure in the brain that assists with the interhemispheric transfer of information (Barkley, 1998). In MRI scans of subjects with ADHD, it was found that the splenium (posterior portion of the corpus callosum) was significantly smaller than that found in subjects without ADHD, suggesting a dysfunction in the transfer of information between the hemispheres of the brain (Semrud-Clikeman *et al*, 1994).

Deep within the cerebral hemispheres, lies a group of grey matter called the subcortical nuclei. Predominant among them are the basal ganglia, which play an important role in the control of movement and posture, and in more complex aspects of behaviour (Ganong, 1993; Vander *et al*, 1994). In contrast to most other components of the motor control system, which transmit via excitatory synapses, the basal ganglia are characterised by their inhibitory influences. They seem to select appropriate components from the excitatory repertoire and suppress antagonistic or unwanted behaviours so that desired patterns of motor activity can be maintained (Vander *et al*, 1994).

The basal ganglia (caudate nucleus and putamen) are implicated in the development of ADHD (Guyton & Hall, 1996). Using isotope perfusion studies, Lou *et al* (1989) showed that ADHD subjects have regional hypoperfusion in the striatal regions of the basal ganglia (composed of the caudate nucleus, putamen and globus pallidus). Using functional Magnetic Resonance Imaging (fMRI), Teicher *et al* (2000) showed that

boys with ADHD had a diminished blood flow to the putamen.

The metabolism of the basal ganglia is unique in that the structures have high oxygen consumption. If blood flow to the region decreases, thus reducing the availability of oxygen, the region does not function as it should, and inhibitory influences of motor activity will be reduced (Ganong, 1993).

Also using MRI studies of boys with ADHD, Castellanos *et al* (1994) found a highly significant lack of normal asymmetry in caudate volume. They noted that although boys without ADHD had a thirteen percent decrease in caudate volume with age, no such age-related change occurred in boys with ADHD.

The basal ganglia are involved in the processes by which an abstract thought is converted into voluntary action. The caudate region is also involved in some cognitive functions. Diseases of the basal ganglia in humans produce marked and characteristic abnormalities of motor function. Both hyper- and hypokinetic disorders of movement can occur (Ganong, 1993).

Although there is evidence of neurological differences in children diagnosed with ADHD, no definitive mechanism has been found for these differences. It may be more accurate to view the syndrome as a cluster of various behavioural deficits, including attention, hyperactivity, and impulsivity. Even if neurotransmitters are found definitely to play a role in ADHD, homeopaths view such abnormalities as a result of a fundamental imbalance of the person as a whole rather than the cause of ADHD (Reichenburg-Ullman & Ullman, 1996).

### **2.3.3. Brain Chemicals in ADHD**

As far as brain chemicals are concerned, the basic difference between those who have ADHD and those who do not appears to lie in the balance of the neurotransmitter chemicals dopamine and noradrenaline found in the cerebrospinal fluid of the brain and spinal cord. In ADHD both these chemicals appear to be either produced in lower volumes by the pre-synaptic cell, or picked up less efficiently by the post-synaptic cell. This results in a relative reduction in dopamine and noradrenaline, an effect that

we presume is only found in certain areas of the brain, particularly the frontal lobes, and the basal ganglia circuits (Green & Chee, 1997).

Dopamine is a prolactin-inhibiting neurotransmitter that is secreted by the substantia nigra of the hypothalamus and travels in the blood to the basal ganglia (Ganong, 1993; Guyton & Hall, 1996). Dopamine is developmentally active which means that it changes in concentration at specific periods of the development and growth of the human. It's concentration in spinal fluid peaks at about two years old and rapidly declines over the next few years (Castellanos, 1997). This supports the theory that ADHD results from a form of delayed brain development. A dysfunctional dopamine pathway can be considered a major factor in the development of ADHD (Raskin *et al*, 1984).

In studies conducted by Castellanos (1997), it was found that boys with high degrees of hyperactivity had high concentrations of dopamine metabolites in their cerebrospinal fluid. Dopamine is probably the most important chemical in ADHD. It acts by sustaining readiness and not letting our own thoughts or outside activity distract us. Appropriate levels of dopamine allow us to inhibit what is unimportant and retain attention on a task. If dopamine levels are raised artificially high, a child may appear obsessively attentive. Noradrenaline provides the animal instinct that allows us to quickly focus on what is important and then act appropriately. It keeps us on a high level of alert, sensitising us to anything that is unusual, unexpected or of immediate significance. Too little noradrenaline causes us to be indifferent, disinterested and a bit withdrawn. Too much may produce a constant wish for a thrill and over-the-top excitement (Green & Chee, 1997).

A study conducted by Saltus (1999), involved injecting patients with a radioactively tagged chemical agent known as Altopane. It was seen to accumulate in dramatically higher levels in a particular part of the dopamine system in the brains of ADHD subjects as compared to normal controls. The chemical imbalance was viewed using single photon emission tomography (SPECT). Altopane is a synthetic analogue of cocaine, which binds to dopamine receptors that regulate the amount of dopamine in certain parts of the brain. This further supports the notion that ADHD is the result of impaired dopamine metabolism.

Shaywitz *et al* (1978) found that cerebrospinal fluid of ADHD subjects had lower levels of catecholamine metabolite, homovalinic acid, which suggests reduced catecholamine production. This theory was supported by Shekim *et al* (1979) when it was found that ADHD subjects had lower amounts of catecholamine metabolites in the urine compared to normal controls, suggesting that the body is either producing less catecholamine or metabolising less catecholamine than is produced.

A central premise in the “catecholamine hypothesis” of ADHD is that dopamine dysfunction leads to clinical symptoms. The hypothesis arises, in part, from the clinical efficacy of methylphenidate, as well as evidence from brain imaging studies that suggest reduced activity in frontal-striatal regions. The theory, however, overlooks the phenotypic complexity of the disorder and the possible interactions between the dopamine and serotonin neurotransmitter systems (Quist & Kennedy, 2001).

#### **2.3.4. Measure of Activity**

Over the years there has been a lot of preoccupation with the hyperactivity aspect of a sufferer of ADHD. Green and Chee (1997), for example, devised an instrument that could record the levels of activity throughout a day. The results showed that the problem in ADHD was not the level of activity, but the inability to adapt this to the expectations of the classroom and playground. All children were “hyped up” when they returned to the classroom from the playground, but those without ADHD settled on the teacher’s request, while the ADHD child took a longer time to calm down and concentrate. It was also found that during team sports the ADHD children were less active on the field due to an inability to follow the game. Thus, ADHD is not about general hyperactivity, it is about the inability on the part of the sufferer to focus activity in appropriate and acceptable ways (Green & Chee, 1997).

There is a clear correlation between the increasingly rapid pace of our highly technological society and the growing number of children diagnosed with ADHD. We live in an extremely over-stimulated society. Children spend hours playing computer games and sitting in front of the television rather than playing outside. Movies are speedier, scarier and more violent than ever before. There is a growing



atmosphere of hurriedness, intensity and urgency. We eat fast, play fast and channel-surf. People look for caffeine and drugs of all kinds to make them go faster and stay up longer. They use highly caffeinated amphetamine-like herbs, including guarana, which contains seven times as much caffeine as coffee. Our society places too little value on tranquillity, slowing down, quiet, solitude and the simple joy of being in nature. In such an environment there is little wonder that many children become overstimulated and unable to cope with all that comes their way (Reichenburg-Ullman & Ullman, 1996).

### **2.3.5. Diet**

Sometimes it's not so much what kids are eating as what they're not eating, and a large proportion of ADHD children are lacking in essential oils and vitamins (Taljaard, 2004). The body uses these to produce certain hormones in the brain, and symptoms of deficiencies include hyperactivity. It was allergy professor Dr Ben Feingold (1975) who first pinpointed certain foods as having a negative effect on hyperactive children. He claimed that more than half of all hyperactive children had developed their difficulties because of their diet. It is now known that these additives (mostly preservatives and colourants) will probably also lead to increased levels in activity in so-called "normal" children (Ridyard, 2001).

However, diet has a smaller part to play in the treatment of ADHD than popular mythology might suggest. Diet never causes ADHD, though in a minority of ADHD and non-ADHD children, certain foodstuffs may make their behaviour more active and possibly more irritable. There seems to be little evidence that diet directly affects attention, impulsivity or insatiability. Sugar, an often-implicated substance in hyperactivity, has not been shown to cause bad behaviour (Green & Chee, 1997).

## **2.4. DIAGNOSIS OF ADHD**

In order to diagnose ADHD, it is highly recommended that practitioners use the full diagnostic criteria laid down in the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) (1994) and use standardised behaviour rating scales from multiple informants and settings. In two studies, conducted by Gimpel and Kuhn

(1998) and Hutchinson *et al* (2000), it was found that relying solely on parental reports concerning isolated symptoms of ADHD might lead to incorrect diagnosis and over-identification of ADHD.

The DSM-IV outlines the characteristic symptoms and diagnostic criteria of ADHD. It stipulates that the symptoms must be present before age seven and must occur in two or more settings, for example at home and at school. It must be clear that clinically significant impairment in social, academic or occupational functioning is present and that the symptoms outlined in the DSM-IV be exclusive of any other developmental disorder and are not better accounted for by another mental disorder (American Psychiatric Association, 1994).

Two main symptom categories are detailed in the DSM-IV (refer to Table 2.1), namely inattention and hyperactivity/ impulsiveness. In each of these categories, at least six of the nine symptoms listed must have been present for at least six months to warrant a diagnosis of ADHD (American Psychiatric Association, 1994).

There are no specific physical features associated with ADHD. However, minor physical anomalies such as hypertelorism, a highly arched palate, and low-set ears occur at a higher rate in subjects with ADHD than in the general population (American Psychiatric Association, 1994).

Scholte *et al*, (2001) conducted a study in which the validity and reliability of the DSM-IV ADHD symptom ratings made by professional caretakers was determined. It was found that all DSM-IV models tested were consistent and reliable. It was also found that making use of professional caretakers in assessing symptoms of ADHD leads to more reliable diagnostic outcomes.

The diagnosis of ADHD is often difficult since the primary signs are behavioural, and vary with time (Berkow, 1992). Teicher *et al* (2000) suggest that a more objective method of diagnosing ADHD may be the use of functional Magnetic Resonance Imaging.

In a study using six healthy boys with no history of ADHD or psychiatric disorders, and eleven boys diagnosed with ADHD according to DSM IV criteria, Teicher *et al* (2000) gave all the boys a computer test that assessed activity, movement, and attention using infrared motion analysis. Of the eleven boys originally diagnosed as ADHD, only six were confirmed to be hyperactive by the computer test.

This suggests that since the DSM IV criteria are broad, they may include children in the ADHD diagnosis that could display similar behavioural problems caused by other reasons than an identifiable neurobiological abnormality.

Teicher *et al* (2000) was able to show a significant improvement in blood flow to the putamen (which has diminished blood flow in ADHD subjects) when treated with methylphenidate<sup>1</sup> in the children that tested objectively hyperactive. However, it was found that those five out of eleven boys that were diagnosed ADHD but not confirmed ADHD by the computer test actually showed a further decrease in blood flow to the putamen following the use of methylphenidate.

Ultimately, a better diagnostic tool would need to be researched comprehensively in order to prevent inconsistent and misdiagnosis of ADHD, since it is clear that the DSM IV criteria are not altogether sufficient.

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<sup>1</sup> Trade name: Ritalin®

**Table 2.1: DSM-IV Criteria for Attention Deficit Hyperactivity Disorder**

1. Inattention in at least six of the following instances:

- Often fails to give close attention to details or makes careless mistakes in schoolwork, work, or other activities
- Often has difficulty sustaining attention in tasks or play activities
- Often does not seem to listen when spoken to directly
- Often does not follow through on instructions and fails to finish schoolwork, chores, or duties in the workplace (not due to oppositional behaviour or failure to understand instructions)
- Often has difficulty organising tasks and activities
- Often avoids, dislikes, or is reluctant to engage in tasks that require sustained mental effort (such as schoolwork or homework)
- Often loses things necessary for tasks or activities (e.g. toys, school assignments, pencils, books, or tools)
- Is often easily distracted by extraneous stimuli
- Is often forgetful in daily activities

2. Hyperactivity/ impulsivity in at least six of the following instances:

Hyperactivity

- Often fidgets with hands or feet or squirms in seat
- Often leaves seat in classroom or in other situations in which remaining seated is expected
- Often runs about or climbs excessively in situations in which it is inappropriate (in adolescents or adults, may be limited to subjective feelings of restlessness)
- Often has difficulty playing or engaging in leisure activities quietly
- Is often “on the go” or often acts as if “driven by a motor”
- Often talks excessively

Impulsivity

- Often blurts out answers before questions have been completed
- Often has difficulty awaiting turn
- Often interrupts or intrudes on others (e.g. butts into conversations or games)

(American Psychiatric Association, 1994)

### **2.4.1. Subtypes of ADHD**

Subtypes have been designated to describe various combinations that occur in individuals. Most individuals display symptoms of both inattention and hyperactivity-impulsivity, but in some individuals one or the other pattern may dominate. The diagnosis of a specific subtype is based on the dominant symptom pattern for the previous six months (American Psychiatric Association, 1994).

- **ADHD Combined Type**  
This subtype is used when six or more symptoms of inattention and six or more symptoms of hyperactivity-impulsivity are present.
  - **ADHD Predominantly Inattentive Type**  
This subtype is used when six or more symptoms of inattention but fewer than six symptoms of hyperactivity-impulsivity are present.
  - **ADHD Predominantly Hyperactive-Impulsive Type**  
This subtype is used when six or more symptoms of hyperactivity-impulsivity are present but fewer than six symptoms of inattention occur.
- (American Psychiatric Association, 1994).

These subtypes may develop from one to the other, but the subtype is diagnosed according to the predominant pattern over six months. The diagnosis of: ‘ADHD In Partial Remission’ is made when criteria for any of the subtypes are no longer met, but significant clinical symptoms still remain. The diagnosis of: ‘ADHD Not Otherwise Specified’ is made when symptoms do not currently meet full criteria for the disorder but it is unclear whether criteria have been previously met (American Psychiatric Association, 1994).

### **2.5. CO-MORBID DISORDERS**

The presence of ADHD greatly increases the existence of associated or co-morbid disorders. ADHD does not cause these disorders, but merely exists alongside the other disorder (Green & Chee, 1997). Of the clinically diagnosed children with ADHD, up to two thirds have another Axis I disorder such as Learning Disability,

Conduct Disorder or Oppositional Defiant Disorder according to the DSM-IV criteria (American Psychiatric Association, 1994; Biederman *et al*, 1991).

It is important to identify co-morbidity during the diagnostic process due to its influence on prognosis, as some of the impairment in ADHD may be due to associated co-morbid conditions (Laurence & Greenhill, 1998).

### **2.5.1. Specific Learning Disabilities**

The prevalence of learning disorders with ADHD depends on the criteria applied. Barkley (1990) noted that when liberal criteria were applied, the prevalence was between forty and sixty percent, but using more rigid standards, twenty to thirty percent of ADHD children were identified as learning disabled. A specific learning disability implies that the child has a significant discrepancy between their tested intelligence and their performance in certain specific areas (Green & Chee, 1997). The most frequent discrepancies are in reading, spelling, writing, language, and mathematics (Semrud-Clikeman *et al*, 1992).

Longitudinal research appears inconclusive on the issue whether the presence of ADHD may lead to later learning difficulties or whether the learning difficulties may be associated with a rise in ADHD symptoms (Fergusson & Horwood, 1992; McGee & Share, 1988). However, it is still presumed that ADHD children are more likely than normal children to have co-morbid learning disabilities (Safer & Allen, 1976).

Specific learning disabilities are often distinguished from ADHD in that children with learning disabilities usually only express their frustration and loss of concentration when the work becomes too hard for them, whereas ADHD children express the inattention most of the time, even if the work is not troubling them (Green & Chee, 1997).

### **2.5.2. ADHD and Disruptive Behaviour Disorders**

Disruptive behaviour manifests as aggression, temper outbursts, and rule breaking such as fighting, stealing and swearing, and includes in its definition both Conduct

Disorder (CD) and Oppositional Defiant Disorder (ODD) (Steven & Pliszka, 1998).

Oppositional Defiant Disorder (ODD) is the most common co-morbid disorder associated with ADHD, and ranges from a mildly oppositional attitude to a constant state of hostile defiance (Green & Chee, 1997). Biederman *et al* (1991) found that more than fifty percent of children referred to clinics with ADHD have ODD as a co-morbid disorder, however, Green and Chee (1997) believe this figure to be lower.

A common misunderstanding is that the general unthinking behaviours of ADHD children is the same as the deliberately defiant feature of ODD. Thus, children on treatment for ADHD may have improvement of the ADHD symptoms but maintain the features of ODD. Parents need to begin a slow, behavioural management approach to treating ODD, and cannot expect conventional treatments for ADHD to improve the ODD symptoms (Green & Chee, 1997).

The behaviours of the more serious Conduct Disorder include lying, cheating, stealing, threatening, cruelty, violating the rights of others, destruction of property, fire setting, and inflicting pain (Green & Chee, 1997). Whereas an ADHD child may perform the occasional antisocial act, children exhibiting symptoms of CD are malicious, and show no remorse for their actions, unlike children with ADHD (Green & Chee, 1997).

ADHD children with co-morbid Conduct Disorder usually come from backgrounds with greater social adversity and have a higher prevalence of psychiatric disorders, particularly Antisocial Personality Disorder, substance dependence and abuse, major depression, and Conduct Disorder among their parents and relatives than ADHD children without significant conduct problems (Faraone *et al*, 1997; Jensen *et al*, 1997; McGee *et al*, 1984; Reeves *et al*, 1987; Szatmari *et al*, 1989).

The long-term outcomes of ADHD and ADHD with Conduct Disorder are very different. In a longitudinal study, August *et al* (1983) found that children with ADHD showed no abuse of drugs at the follow up, whereas over thirty percent of children with ADHD and Conduct Disorder had engaged in substance abuse by the age of fourteen years. It is believed that the child with ADHD that has not shown symptoms

of Conduct Disorder by the age of twelve years is unlikely to develop the problem later on (Green & Chee, 1997).

### **2.5.3. ADHD and Mood Disorders**

In 1993, Jensen *et al* found that nearly forty-nine percent of their sample of ADHD children had an anxiety disorder, depression, or both.

The co-morbidity of anxiety or mood disorder with ADHD is often associated with a history of greater family and personal stress, greater parental symptoms of anxiety and mood disturbance, and reduced responsiveness to stimulant medication (Jensen *et al*, 1997; Tannock, 1994).

The presence of anxiety with ADHD seems to significantly reduce the level of impulsiveness in these children below that seen in ADHD children without anxiety, though the latter remain more impulsive than children without ADHD (Barkley, 2002; Epstein *et al*, 1997; Gordon *et al*, 1990; Pliszka, 1992).

About six percent of ADHD children may have the co-morbidity for Bipolar Disorder (BPD) that is not the result of merely having more severe ADHD symptoms (Milberger *et al*, 1995). The diagnosis and treatment of Bipolar Disorder are strictly for qualified psychiatrists, and this is not a disorder to treat lightly (Green & Chee, 1997). Subjects with BPD are considerably more impaired in their functioning than those with ADHD alone. They experience a greater risk for hospitalisation and for additional forms of psychopathology (Wozniak *et al*, 1995).

Depressive symptoms generally have their onset after ADHD symptoms, which may imply that the symptoms of depression are consequent to ADHD (Biederman *et al*, 1995; Kovacs *et al*, 1994). It has also been noted that when stimulants are started in an excessive dose or the wrong preparation is prescribed, children may become teary, withdrawn, and “different” (Green & Chee, 1997).

However, if the ADHD child is in a chronic state of depression according to the DSM-IV criteria, the depression should be treated as the first priority rather than the



ADHD (Green & Chee, 1997). It is now common practice for selective serotonin reuptake inhibitor (SSRI) antidepressants to be prescribed in conjunction with stimulant medication (Green & Chee, 1997).

## **2.6. DIFFERENTIAL DIAGNOSIS**

ADHD shares some symptoms with other look-alike behaviours that are influenced by biological, psychological, and environmental factors. It is, however, important to rule out any other diseases before diagnosing a child with ADHD (American Psychiatric Association, 1994). Some possible diagnoses that may be made in place of ADHD include:

### **2.6.1. Biological factors**

- High levels of activity

Although high levels of activity may be difficult to distinguish from ADHD, since most children go through this stage of over-activity and lack of common sense, a good preschool and boundaries in discipline should determine whether this is just a normal, busy temperament that the child will grow out of, or whether it is a serious problem, like ADHD (Green & Chee, 1997). Research by Lapouse and Monk (1953) calculated that fifteen percent of boys meet criteria for motor hyperactivity without manifesting other signs of ADHD. Thus, over-activity is a common finding in children, particularly boys (Smith, 2001).

- Intellectual disability

It is possible that children with developmental delays are at increased risk of developing ADHD, and so it is necessary to diagnose each child carefully, ruling out any disabilities before diagnosing ADHD (Green & Chee, 1997). Children with low intelligence quotients (IQ) placed in mainstream education are frequently inattentive and sometimes disruptive. Placement in a class for slow learners is often all that is needed to ensure good concentration and compliance (Leary, 1994). A diagnosis of ADHD in children with mental retardation should be made only if the symptoms of inattention or hyperactivity are excessive for the child's mental age (American

Psychiatric Association, 1994).

- High intelligence

These children, if placed in academically under-stimulating environments, may become inattentive in the classroom (American Psychiatric Association, 1994). This is likely to be the result of boredom (Quin & Macauslan, 1986).

- Hearing impairment

These children usually present with behaviour that is more unresponsive and distant than that of an ADHD child (Green & Chee, 1997). The partially deaf child may have difficulty in concentrating or understanding what is asked of him (Quin & Macauslan, 1986). Inattentive children should be formally tested for any hearing impairment. Children frequently have middle ear infections that can lead to “glue ear” that can also possibly impair hearing (Green & Chee, 1997). This hearing loss may remain undetected, leading to classroom inattention and unacceptable behaviour (Leary, 1994).

- Loss of visual acuity

Sensory loss such as unrecognised significant myopia can cause inattentiveness and academic failure (Leary, 1994).

- Hyperthyroidism or hypothyroidism

Hyperthyroidism produces symptoms of restlessness and hyperactivity, whereas hypothyroidism causes slowing of cognitive functioning and memory loss (Laurence & Greenhill, 1998). Thus, either of these two dysfunctions of the thyroid gland could result in symptoms common in ADHD.

- Anaemia or hypoglycaemia

Both anaemia and hypoglycaemia may cause, impaired concentration and fatigue, symptoms that may be mistaken for symptoms of ADHD (Leary, 1994). The lack of circulating oxygen found in children with anaemia can result in lack of energy and possible reduction in learning ability, amongst other symptoms (Marotz *et al*, 2001). In hypoglycaemia the lack of sugar in the blood can cause sudden headaches, nausea,

faintness and shakiness, all of which reduce concentration and ability to participate in a classroom environment (Marotz *et al*, 2001).

- Chronic infections

Chronic infections may produce difficulty in sustaining attention (Leary, 1994). Undiagnosed and untreated chronic illnesses can interfere with a child's development of early learning skills. Children who have chronic allergies often experience long-term irritability and malaise (Marotz *et al*, 2001).

- Poorly controlled asthma

Asthmatics that are not under adequate management may suffer from shortened attention span and drowsiness due to overuse or underuse of medications (Leary, 1994). Overuse of asthma medications can lead to side effects including irritability, overactivity, excitement, agitation, and restlessness (Sifton, 2002). Underuse of medications results in asthma symptoms becoming more marked and resulting in distractibility and drowsiness (Sifton, 2002).

- Fatigue

Fatigue is a potent cause of inattention and fidgeting. Fatigue may be the result of poor bedtime discipline, a noisy domestic environment, or insufficient sleep due to sleep apnoea related to adenoidal hyperplasia (Leary, 1994).

- Autism or Asberger syndrome

Autistic children are aloof, have poor verbal and body language, and are somewhat obsessive. One subgroup of autistic children is extremely active (Green & Chee, 1997).

- Epilepsy

Children with epilepsy may have associated ADHD, due to brain structure problems (Green & Chee, 1997). Petit mal epilepsy may mimic ADHD, due to it causing brief, primarily generalised seizures manifested by a ten to thirty second loss of consciousness and associated eye/ muscle flutterings, with or without loss of muscle tone (Berkow *et al*, 1992). The child suddenly stops any activity in which he is

engaged, stares, may blink or roll up the eyes, fails to respond to commands, and then resumes their activity after the attack (Edwards *et al*, 1995). These symptoms may be confused with the inattentiveness of ADHD.

- Brain injury

The behaviour caused by brain injury can be hard to distinguish from that found in ADHD. Changes in behaviour and personality are usually much more sudden than in children with ADHD, which has a gradual onset (often from birth onwards) (Green & Chee, 1997). Brain damage following meningitis, encephalitis, or head injury may cause inattention and cognitive impairment (Leary, 1994).

- Fragile X Syndrome

This syndrome, inherited on the X chromosome, is characterised by intense motor over-activity (Laurence & Greenhill, 1998). It is caused when a person has a faulty gene (the fragile X mental retardation 1 gene, or FMR1) on the X chromosome that results in nonproduction of fragile X mental retardation protein (FMRP). Since it is a gene on the X chromosome, boys with this genetic abnormality tend to exhibit the physical, cognitive, behavioural, sensory, speech and language problems to a greater severity than girls with this gene, since boys only have the one X chromosome, while girls will have one faulty X chromosome and one normal one, the normal one able to produce enough FMRP to fill most of the body's needs, but not all (O'Donnell & Warren, 2002). The children with this syndrome do, however, display characteristic facies, such as large protuberant ears, prominent chin and forehead, and gaze avoidance, which should differentiate it from ADHD (Laurence & Greenhill, 1998).

- Side effects of drugs

Drugs such as theophylline (used to treat asthma and chronic bronchitis), clonazepam (used to treat panic attacks, and convulsive disorders such as epilepsy), and phenobarbital (used as a sleep aid, and to treat epilepsy) include hyperactivity and inattention or drowsiness in their list of common side effects (Leary, 1994; Sifton, 2002).

### **2.6.2. Psychological factors**

- Depression or Bipolar disorder

Depression in children results in the child being moody, preoccupied, sad, and withdrawn (Green & Chee, 1997). Depression may also manifest in children as over-activity, limited attention span, and poor classroom achievement (Leary, 1994). The drivenness and agitation of Bipolar disorder may resemble that of ADHD, although there is usually a family history of Bipolar disorder, and the course of the behaviour is cyclical rather than continuous (Laurence & Greenhill, 1998).

- Oppositional behaviour or Conduct disorder

Children who have oppositional behaviour may resist school tasks because of their unwillingness to conform to others' demands (American Psychiatric Association, 1994). This can be differentiated from the avoidance of school tasks seen in children with ADHD. However, some ADHD individuals develop oppositional attitudes to such tasks and devalue their importance as rationalisation for their failure (American Psychiatric Association, 1994). Conduct disorder manifests with more rule-breaking, aggressive and impulsive behaviour than ADHD (Laurence & Greenhill, 1998).

- Mental disorders

A mood disorder, anxiety disorder, dissociative disorder, or personality disorder must be excluded before diagnosis of ADHD can take place (American Psychiatric Association, 1994). Borderline personality disorder exhibits excitability, mood lability, and impulsivity. Seventy percent of adults with a diagnosis of Borderline Personality report a childhood history of ADHD (Laurence & Greenhill, 1998).

In all these psychological disorders, symptoms of inattention typically arise after the age of seven years, whereas symptoms of inattention in ADHD individuals typically occur before seven years of age. In addition, the childhood history of school adjustment of the other mental disorders is not characterised by disruptive behaviour or teacher complaints about inattentive, hyperactive and impulsive behaviour, as is the case with ADHD children (American Psychiatric Association, 1994).

### **2.6.3. Environmental factors influencing children's behaviour**

- Family dysfunction

The presence of family dysfunction can make children behave in a way that may resemble the behaviour of children with ADHD. Children become attention-seeking, defiant and unmanageable under stressful, inconsistent and emotionally difficult circumstances. Also, family dysfunction may be due to residual problems in ADHD parents. Since there is a genetic element to ADHD, these parents may have ADHD children and then it becomes a complicated problem (Green & Chee, 1997). Emotional disturbance relating to domestic tension or disorganisation is an important cause of distractibility and underachievement (Leary, 1994).

- Classroom anxiety

Some children may become anxious in the classroom due to the teacher's approach or methods, and this may cause inattentiveness and underachievement to occur (Leary, 1994).

## **2.7. TREATMENT**

The treatment of the child with ADHD usually involves three steps: firstly, careful and thorough evaluation; secondly, explanation of the problems to parents and child; and lastly, therapeutic intervention if necessary (Wender, 1987).

### **2.7.1. Evaluation**

As previously discussed, there appears to be no completely reliable test for ADHD. The methods currently available bring some objectivity into a very subjective area, but they are not foolproof and can be seen as nothing more than pointers towards a probable diagnosis of ADHD (Green & Chee, 1997).

The diagnostic process for determining ADHD in a given child depends firstly upon careful history taking and observation. Such procedures require several thorough visits to a clinician (Laurence & Greenhill, 1998). A major goal of such visits is the

determination of the presence or absence of ADHD as well as the differential diagnosis of ADHD from other childhood psychiatric disorders (Barkley, 1998).

Before the visit, the clinician usually clarifies the parents' chief concern, be it academic underachievement, difficulty in making friends, being disruptive in class, or family problems (Laurence & Greenhill, 1998).

A detailed history of the child's psychological development and current functioning, preferably with information from both parents and teachers, is needed for the evaluation. Often doctors find it desirable to obtain individual intelligence tests in order to ascertain any intellectual difficulties or specific learning disabilities, and a test of academic performance level (Wender, 1987). Practitioners also find it helpful to obtain school reports or results, examples of the child's work, achievement tests, IQ tests, and developmental screening tests before the visit, in order to become familiar with the child before the consultation (Barkley, 1998).

Wender suggests that the evaluator interview the child for a differential diagnosis, since inattentiveness and restlessness are often seen in children with other forms of psychiatric difficulties and many of these problems can be diagnosed only by talking to the child (Wender, 1987). He also suggests that, during the visit, the clinician observes the parent and child to evaluate for symptoms of ADHD or co-morbid conditions (Laurence & Greenhill, 1998). Barkley suggests that the DSM-IV should be reviewed for both categorical criteria (whether sufficient symptoms of ADHD are present) and dimensional criteria (whether the symptoms are severe enough to require medical intervention) (Barkley, 1998).

Few parents have the specialised parenting knowledge and skills necessary to bring children with behavioural problems under better control. As they become increasingly aware of their inability to resolve these child management problems, many parents begin to experience secondary personal difficulties, such as sadness, frustration, guilt, stress, and marital strain. Such conditions are often the impetus for seeking out professional advice regarding their ADHD child (Barkley, 1998).

With these sorts of circumstances in mind, the role that parent training and counselling plays in the overall clinical management of children with ADHD has become important (Barkley, 1997b).

Together with the parents, a viable treatment plan for the child should be suggested. In some cases, parents prefer not to use any form of medication in treating their child, and want to know about other treatment methods available to them (Green & Chee, 1997). If medication is to be used, it is suggested that it must be used in conjunction with concrete, specific, and flexible guidance techniques. Since it is symptomatic rather than curative, medication is usually utilised until the signs and symptoms of the disorder decrease with age, usually by adolescence (Wender, 1987).

## **2.7.2 Problems with treatment of ADHD**

In 1997, Breggin observed that children with symptoms of ADHD are being done a disservice by a society that resorts to medication as a first line of treatment. He noted that Ritalin® is the drug of choice for treatment of the symptoms of ADHD in most paediatric practices, but shows concerns that it is used too easily and without the necessary care.

### **2.7.2.1 A Way to Control the Classroom**

As early as 1987, Newsweek reported that experts on ADHD were concerned that an increasing number of non-ADHD children around the USA were given Ritalin®. Their concern was that if a child without ADHD was medicated for displaying reckless behaviour or having a drop in grades, the child may become more compliant, but the true cause of the behaviour would not be addressed (Saholz *et al*, 1997).

In South Africa, many classrooms have a high child to teacher ratio. Some of these children can be unruly, with an antipathy to learning, and for teachers that may prefer a rigidly controlled environment this might cause problems. Parents may be called in to discuss even mildly unruly children and pressured to put their children onto stimulant medications for the sake of the class as a whole. Even teachers that like a more relaxed and friendly environment for learning find that restless, disruptive



children disturb the classroom environment (Reichenberg-Ullman & Ullman, 1996).

In an observation of private primary schools in Cape Town's affluent suburbs, parents indicated that they felt excessively persuaded to seek the drug Ritalin® for their children. Parents reported that some children had been threatened with expulsion if they did not take the drug, and that at other schools it had been made a pre-requisite for admission. As one mother reported, "it is very 'in' to put children on Ritalin® if they show the slightest tendency to be noisy, active or disruptive." The issue is not one of whether Ritalin® works or not in cases where it is truly indicated, but why it has become the first resort for schools where large class sizes make it difficult for teachers to handle poorly behaved children (Nicodemus, 1999).

As Dr Peter Breggin, director of the centre for the Study of Psychiatry in Bethesda, Maryland, noted in an article dated 10 July 1997, "it is an incredible commentary on our society that instead of addressing the basic needs of our kids, we drug them".

In South Africa, Professor Mick Leary, neurology chief of Cape Town's Red Cross Children's Hospital, notes that there is a tendency, particularly amongst teachers of big classes, to start shouting Ritalin® when they spot a child who is not concentrating and is starting to fidget (Jordan & Jurgens, 1997). Gauteng's education MEC at the time, Mary Metcalfe, agreed that the drug could be prescribed too quickly (Jordan & Jurgens, 1997).

#### 2.7.2.2. Gifted Children

A question for debate put forward by Reichenberg-Ullman and Ullman (1997) is as follows: "If a child has an IQ of 150 and a photographic mind, how would he or she feel about being in a regular fourth-grade classroom?" They suggest the possibility of being bored unless the teacher found special activities and outlets for children with unusual intellectual capabilities. Children that are bored in class might tap their pencils on their desks, design paper skyscrapers, or invent a magical world of dinosaurs. Then when called on by the teacher, it is discovered that the child has not paid attention to what she has been saying. A gifted child's perceived inability to stay on a task might be related to boredom, curriculum, mismatched learning style or other

environmental factors. Gifted children may spend from one-fourth to one-half of their regular classroom time waiting for other children to catch up – even more if they are in a heterogenously grouped class (Reichenberg-Ullman & Ullman, 1997).

#### 2.7.2.3. Unclear Diagnosis

Problems arise when children present with only some of the common symptoms of ADHD and not all of the diagnostic symptoms. Parents are reluctant to medicate their child with a drug like Ritalin®, which may offer “miracles” but comes with possibilities of serious side effects, when they are not absolutely certain of the diagnosis of ADHD. Dr Harold Koplewicz, chief of child and adolescent psychiatry at Schneider’s Children’s Hospital in New York, is quoted by Green and Chee (1996) as saying that the litmus test should be if the child has distress and dysfunction. He suggests weighing up the cost and the benefits to the child of the medication being considered. ADHD in youngsters has for years been treated with a medication about which experts are now growing more sceptical. Some doctors are distressed about the way in which some parents are calling specifically for a diagnosis of ADHD and asking directly for medication (Green & Chee, 1996).

### 2.7.3. Therapeutic intervention

In 1937, Bradley investigated the use of amphetamine sulphate in the treatment of children with disruptive behaviours. Since then, many research studies have been done in an attempt to ameliorate the symptoms of ADHD. Allopathic medicines are currently used most frequently, and include stimulants, antidepressants, and neuroleptics (Barkley, 1998). Herbal, homoeopathic, diet, and behavioural management methods of treatment are also extensively researched and frequently used by those unwilling to place their children on extended courses of allopathic drug therapy (Reichenberg-Ullman & Ullman, 1996).

#### 2.7.3.1. Stimulants

The most commonly used drugs to treat the symptoms of ADHD are central nervous system (CNS) stimulants (Barkley, 1998). The stimulants methylphenidate (Ritalin®)

and dexamphetamine as well as pemoline (Cylert®) are the most commonly prescribed and most effective allopathic preparations for the treatment of ADHD. Second-line drugs are often used, like imipramine (Tofranil®), clonidine (Catapres®) and occasionally moclobemide (Aurorix®) (Green and Chee, 1997).

CNS stimulants are so named due to their ability to raise the level of activity, arousal, or alertness of the central nervous system. These drugs are structurally similar to brain catecholamines (i.e., dopamine and norepinephrine) and are called sympathomimetic compounds because they may mimic the actions of these brain neurotransmitters (Barkley, 1998).

It has been found that the stimulants enhance behavioural, academic and social functioning in about fifty to ninety-five percent of children treated (Barlow and Durand, 1995). Ironically, the stimulants used to treat the symptoms of ADHD produce activity and alertness when given to healthy individuals (Barkley, 1998).

The precise mechanism of the action of these stimulants is still poorly understood, but it is a subject under intense investigation (Barkley, 1998). Although significant improvement has been recorded concerning the use and dosage of stimulant medication, use of these drugs is noted to lead to potentially dangerous short-term and long-term side effects including loss of appetite, growth retardation, sleep disturbances, and seizures (du Plessis, 1999).

Despite evidence of the efficacy of stimulants at a group level of analysis, as many as 20% to 30% of children tried on stimulants may display no positive response to these medications, or may display worsening in behaviour in response to medication (Elia & Rapoport, 1991).

#### 2.7.3.1.1. Ritalin®

CNS stimulants are given orally, are quickly absorbed from the gastrointestinal tract, cross the blood-brain barrier rapidly and easily, and are eliminated from the body within 24 hours (Diener, 1991).

The recommended dose of Ritalin® is 0.25mg to 2.0mg per kilogram of body weight daily (Sprague & Sleater, 1977). To exert its effect on the neurotransmitters it must cross the blood-brain barrier, a process that varies greatly from child to child according to their individual rates of metabolism and blood flow. This variability in the amount of medication that moves from blood to brain shows the possible shortcoming of calculating dose by body weight alone (Green & Chee, 1997).

Several controlled studies have demonstrated immediate improvement in behaviour and scholastic performance when Ritalin® was given to hyperactive children (Douglas, 1988; Rapport, 1988; Kupietz, 1988; Richardson *et al.*, 1988). However, the long-term gains of using this drug have not been established (Jacobvitz *et al.*, 1990; McGee and Share, 1988).

Methylphenidate reaches peak plasma levels within 1.5 to 2.5 hours post-ingestion, and the drug is metabolised completely, with almost none of the drug appearing in the urine (Diener, 1991; Wargin, *et al.*, 1983). Behavioural effects occur within 30 to 60 minutes, peak within 1 to 3 hours, and are dissipated within 3 to 6 hours after oral ingestion, creating an effective span of clinical improvement ranging from about 4 to 6 hours in most children (Birmaher, *et al.*, 1989). Significant inter-individual variability exists with respect to these parameters, which means that clinical evaluation as to the frequency of dosage for individual children is necessary (Patrick, *et al.*, 1987).

Methylphenidate (Ritalin®) is very closely related to amphetamine in chemical structure, metabolism and clinical effects, and the close connection to this mood-altering and addictive drug is the chief reason why Ritalin® use raises concern amongst parents and others (Diller, 1998).

Boys who were objectively hyperactive according to infrared motion analysis showed enhanced blood flow to the putamen when given methylphenidate (Ritalin®), but when those that were not objectively hyperactive were given the same treatment, it further decreased the blood flow to the putamen. This suggests that methylphenidate may not be effective for all children diagnosed as ADHD according to DSM IV criteria alone (Teicher, *et al.*, 2000).

Methylphenidate produces acute growth hormone release in children, which could lead to alterations in prolactin, cortisol, and beta-endorphins (Reeve & Garfinkel, 1991). However, long-term effects have not been demonstrated as yet (Barkley, 1998).

Some of the short-term, general side effects include decreased appetite, insomnia, anxiety, palpitations, headaches, irritability or a tendency to cry. Other more serious side effects include tics and “behavioural rebound” which refers to a regression in behaviour occurring in the afternoon or evening following daytime medication administration (Barkley, 1998). Patients may also experience a sense of euphoria, making the use of the drug open to abuse and addiction. This euphoric feeling can be eliminated by alteration of doses. It is recommended that medication not be taken after 16h00 in order to prevent insomnia and allow appetite to return in time for the child to eat dinner (Diller, 1998).

It is also recommended that treatment for children with ADHD include psychotherapy sessions to enhance their lives and help in cases where the child may or may not respond to allopathic treatment (Santrock, 1997).

Although the stimulants are the most established treatment for ADHD, as many as 30% of affected individuals do not respond or may not tolerate such treatments (Spencer, *et al.*, 1996). Effective alternatives to stimulants have included medications traditionally considered antidepressants (Barkley, 1998).

#### 2.7.3.2. Antidepressants

Antidepressants are generally used in the treatment of ADHD when there is a contraindication to the use of stimulants, for example, in the case of a child with a tic disorder or where there is co-existing anxiety or depression. The antidepressants used most frequently include the tri-cyclic antidepressant, imipramine (Tofranil®) and fluoxetine (Prozac®), a serotonin reuptake inhibitor (Barkley, 1998).

The available literature seems to indicate that tri-cyclic antidepressants are as efficacious in controlling abnormal behaviours associated with ADHD, but are less

effective in improving cognitive impairments relative to the stimulants (Gualtieri & Evans, 1988; Quinn & Rapaport, 1975; Rapaport, *et al.*, 1973; Werry, 1980).

Side-effects and tolerance to imipramine treatment are commonly reported (Greenberg and Yellin, 1975; Waizer, *et al.*, 1974). Common short-term adverse effects of the tri-cyclic antidepressants include anticholinergic effects, such as dry mouth, blurred vision, and constipation (Barkley, 1998). Other commonly experienced side-effects include nausea, weight loss, insomnia, drowsiness and difficulty in micturition (Quinn & Rapaport, 1975).

A small open study suggested that fluoxetine may be beneficial in the treatment of ADHD children (Barrickman, *et al.*, 1991), but the consensus of ADHD experts at a National Institute of Health conference on the use of alternative agents for ADHD did not support the usefulness of these compounds in the treatment of core ADHD symptoms due to lack of conclusive research (National Institute of Mental Health, 1996).

Common adverse effects of fluoxetine include insomnia, irritability, gastrointestinal disturbances, and headaches (Grimsley & Jann, 1992). Nevertheless, because of the high rates of comorbidity in ADHD, these compounds are frequently combined with effective anti-ADHD agents (Barkley, 1998).

#### 2.7.3.3. Antipsychotic medication

Antipsychotic medication may be indicated in children that have ADHD comorbid with severe Tourette syndrome or tic disorder (Barkley, 1998). However, since the side-effects of Ritalin® include the worsening of tics and Tourette syndrome, doctors may be in fact treating side-effects rather than withdrawing or reducing the causative agent, i.e., Ritalin® (Barkley, 1998).

In studies reviewed by Green (1995), thioridazine, chlorpromazine, and haloperidol have been found effective in the treatment of children with severe developmental delay and mental retardation resulting in excessive hyperactivity, impulsivity and attentional deficits when compared with a placebo.

These drugs have anti-dopaminergic actions (Levy & Hobbs, 1988), and although children treated in this manner showed considerable improvement in their behaviour, no increased vigilance or test scores was noted (Gadow, 1992).

#### 2.7.3.4. Other allopathic medications

Antihypertensive agents have been used to treat ADHD since the early 1970's (Barkley, 1998). In child and adolescent psychiatry, clonidine was first used to treat tics in children with Tourette syndrome (Cohen, *et al.*, 1979). Clonidine appears partially effective in decreasing the frequency, intensity, and severity of impulsivity and hyperactivity and improving frustration tolerance in children with ADHD. Particularly the combined type or predominantly hyperactive-impulsive type as described by the American Psychiatric Association in 1994 (Conner & Fletcher, 1998). Clonidine is also useful for tic disorders and sleep disturbances, which often present clinically with ADHD (Barkley, 1998).

Side effects common to the use of clonidine include agitation, dizziness, drowsiness, fatigue, nervousness, mental depression, anxiety, loss of appetite, nausea, headache and behaviour changes, any of which may be detrimental to the health of a child being treated for ADHD (Sifton, 2002).

Anticonvulsant medications such as carbamazepine have been used to treat ADHD with some success (Silva, *et al.*, 1996). They are traditionally used in the management of epilepsy (Berkow et al, 1992) There appears to be significant benefit for overarousal, aggression, impulsivity, hyperactivity, restlessness, and excitability in children with ADHD and no neurological abnormalities, but further research is required before firm conclusions of its use can be drawn (Barkley, 1998).

The concurrent use of more than one medication in the treatment of ADHD is increasingly common in clinical practice (Barkley, 1998). This is an alarming practice when the many different adverse drug interactions are taken into account, but remains a poorly researched area.

## **2.7.4. Management without Allopathic Drugs**

### **2.7.4.1. Behavioural Management**

Effective behavioural management programmes directly target the areas in which change is desired (e.g., academic problems, social skills) (Barkley, 1998). If positive alternative behaviours are not taught and only problem behaviour is targeted for intervention, children may simply replace one problem behaviour with another (Barkley, 1998).

It is suggested that interventions in behaviour are managed in all settings in which they occur. In other words, parents and school personnel should collaborate in order to create an effective and well-monitored intervention programme (Burcham, et al, 1993; Kotkin, 1995). Changes in one setting rarely generalise without intervention in all other settings (Barkley, 1998). Behavioural modification strategies are usually applied with absolute consistency and parents and teachers need to adopt a uniform approach for there to be any success (Leary, 1994).

Behavioural management has the advantage of being able to target not only the child's primary ADHD symptomatology but also many of the comorbid features, including oppositional-defiant behaviour and conduct problems (Barkley, 1998). However, it has been found by Barkley (1998) that behavioural management has not added a great deal to treatment outcome above and beyond that accounted for by other treatments, such as medication.

### **2.7.4.2. Dietary Management**

Much publicity has focused on the role of foods and allergies in illness since Feingold (1975) first implicated certain foods as having negative effects on hyperactive children. The scientific evidence currently suggests that, in a few children who have problems with ADHD, dietary additives and preservatives may play some part in their problems. They may affect the child's level of activity and ability to concentrate (Baron-Cohen & Bolton, 1993).



A large proportion of ADHD children are lacking in essential oils and vitamins used by the body to produce certain hormones in the brain, and symptoms of these deficiencies include hyperactivity. Watching the diet and supplementing with “brain oils” will help, as well as ensuring adequate vitamins to help metabolise the fatty acids (Taljaard, 2003). Every year the Hyperactive and Attention Deficit Support Group of Southern Africa prints a chunky, 20-page list of foods (and specific brands) that can be eaten safely by hyperactive children (Ridyard, 2001).

The original Feingold diet did have some inconsistencies (Green & Chee, 1997). Brenner, (1977), Salzman (1976), and Rowe (1988) found improved behaviour in ADHD patients following the Feingold diet. Other controlled studies did not support the diet (Leary, 1994; Mattes and Tittkemam, 1981; Walker, 1983).

Today there are much more reliable diets. These diets start by excluding all potentially harmful foods, placing the child and their family on a diet of water, pear juice, preservative-free bread and unseasoned meats. This strict diet is kept going for a number of weeks and if there is no significant improvement it is stopped. If the diet helps, the dietician will gradually introduce other groups of food until those that are causing the harm have been clearly isolated. Finally, an individual diet is suggested which avoids the troublesome foods. It is suggested that all exclusion diets be implemented and supervised by a dietician or doctor specialising in this area (Green & Chee, 1997).

There is little evidence that diet significantly alters the inattention, impulsivity and insatiability behaviours that are so troublesome in ADHD. The current view is that diet does affect some children, but a change in diet makes little difference to the trio of behaviours that cause most of the bother in ADHD (Green & Chee, 1997).

Research investigations have been conducted since Feingold’s theories were publicised in 1975, the more rigorous of which found that substances implicated by Feingold (dyes, preservatives, salicylates) have little if any effect on children’s behaviour (Connors, 1980).

Unfortunately, however, dietary approaches only work for some children and not others. Medication, including use of homeopathic remedies, has been found to have a more consistent, profound and lasting impact on behavioural and learning problems (Reichenburg-Ullman & Ullman, 1996).

#### 2.7.4.3. Herbal Treatment

Due to its calmative action and its ability to improve concentration Valerian (extract of *Valeriana officinale*) has been used to treat ADHD (Eickestedt, 1969; Ortiz *et al*, 1999; Shaette, 1971). In a study of one hundred and twenty children with various behavioural disorders including hyperactivity, learning disorders, and anxiety, it was found that seventy-five percent of these children experienced significant progress or complete recovery over several weeks with Valerian (Klich & Gladbach, 1975). The use of Valerian in treating ADHD should be pursued in order to determine the optimum dosage and dosage period required for maximum effect (Smith, 2002).

Another herbal remedy that has been researched for ADHD is *Passiflora incarnata*, a herb found to have calming properties due to its action on neurotransmitter receptors (Lutamski, 1960). *Schizandra* berry is another herbal remedy used for ADHD. Its action as a tonic for the adrenal glands and lungs was found to enhance learning and memory performance (Landis & Khalsa, 1997; Nishiyama *et al*, 1996). *Gingko biloba* improves circulation to the small capillaries of the brain, and in combination with American ginseng extract was found to improve the symptoms of ADHD (Lyon *et al*, 2001).

#### 2.7.4.4. Homoeopathic treatment

In the Organon of Medicine, Hahnemann states that the highest ideal of cure is “a rapid, gentle and permanent restoration of health” where the disease is removed from the sufferer in its entirety without harming the patient (Hahnemann, 1998). The Hyperactive and Attention Deficit Support Group of Southern Africa advocates the use of homeopathic remedies for ADHD children (Ridyard, 2001).

Homoeopathic prescription is based on the principle of “like cures like” which states that if a weak dose of a crude substance is administered to a healthy individual, that person will present with similar symptoms to the ill patient and therefore this substance will be capable of curing these similar symptoms in the ill patient (Jouanny, 1994).

Homoeopathic medicines are known to have minimal to no side effects, and do not result in dependency (Lockie & Geddes, 1995). There is also no possibility of overdosage due to the fact that the medicines are diluted to the extent that they have no direct toxicity (Lockie & Geddes, 1995). Although there are little or no side effects, an aggravation, known as temporary worsening in symptoms preceding the alleviation of symptoms, may occur with homoeopathic treatment. Typically, aggravations are short-lived, and healing should follow rapidly (Lockie & Geddes, 1995). Homoeopathic medicines have also been found to be safe to use over long periods of time (Reichenberg-Ullman & Ullman, 1996).

Three studies have been performed concerning ADHD and non-allopathic treatment options: Strauss (1998) conducted a study to determine the efficacy of Selenium Homaccord®, a Homoeopathic complex preparation, in the management of ADHD. It was concluded that there was an overall improvement in the children’s symptoms, in both those who were taking Ritalin® and those who were not on any form of medication.

In a study conducted by Smith (2001) it was found that Cerbo® and Nerva 2®, both Homoeopathic complex preparations, reduced the symptoms pertaining to inattention and produced an improvement in the Conner’s Abbreviated Teacher Rating Scale (CATRS) results.

Meyer (2001) found that Melotone syrup, a nutritional supplement, produced an improvement in sustained attention, but with no significant difference between the experimental and control groups overall.

All the above studies involved complex preparations, i.e. all medication included more than one remedy, but Vithoukas (1998) believes that one of the fundamental

principles of homeopathy is that of prescribing only one remedy at a time. He explains that the most specific symptoms should be used to repertorise the case.

#### 2.7.4.4.1. Single remedy usage

Hahnemann advocated the single remedy on practical and theoretical grounds. He thought that the use of medical mixtures led to over-drugging of the patient, but, more specifically, he realised the impossibility of predicting the synergistic effect of several drugs administered simultaneously (Coulter, 1980).

Repertorisation is a method of cross-referencing compiled lists of remedies in which a specific symptom has been found. As advised by Vithoulkas (1998), this process will enable the researcher to review the many remedies that are known to have produced the symptoms being studied in a given case.

Through repertorisation of the symptoms used in DSM-IV diagnosis of ADHD (refer to Table 2.2.), it was discovered that *Phosphorus* most closely matches the symptom picture of this disorder (Schroyens, 1998).

In the *Organon of Medicine* (1982), Hahnemann wrote, in aphorism 104, “When the totality of the symptoms that specially mark and distinguish the cause of disease or, in other words, when the picture of the disease, whatever be its kind, is once accurately sketched, the most difficult part of the task is accomplished. The physician has then the picture of the disease, especially if it be a chronic one, always before him to guide him in his treatment; he can investigate it in all its parts and can pick out the characteristic symptoms, in order to oppose to these a homoeopathically chosen medicinal substance” (Hahnemann, 1982).

In the case of ADHD, the American Psychiatric Association (1994) has laid out the definitive symptoms of the disease, and it is this picture that can be used in the way outlined by Hahnemann above.

#### 2.7.4.4.2. *Phosphorus*

According to homoeopathic remedy pictures, *Phosphorus* can be used successfully in both children and adults (Lockie, 1989). As seen in Table 2.2., *Phosphorus* is the indicated remedy for the DSM-IV characteristic symptoms of ADHD as repertorised. A more detailed picture of patients requiring *Phosphorus* shows that these patients are easily vexed, anxious and fearful. They are oversensitive to external impressions and are restless and fidgety. They are disinclined to work and study and show a lack of willpower in these areas. They are inclined to cry and laugh inappropriately. Patients cannot sit or stand still for a moment and are very talkative, although it is possible to have a slow development of speech in the child. There is confusion, and the brain feels tired due to a great flow of thoughts that are difficult to arrange. Shamelessness is often present. Patients are noted for their bursts of intense and extreme energy that fizzles out leaving the patient feeling weak, tired and depressed (Boericke, 1999; Lockie, 1989; Vermeulen, 1997).

Paul Herscu wrote in 1991 that, “it is a *Phosphorus* child’s nature to be extroverted, open and very talkative, often straying from the topic at hand”. He goes on to explain that their expressiveness is attractive to others and can make these children very popular. Their desire to be the centre of attention at school, with friends, and at home is evident with their tendency to talk loud and long, and to be very active in public. If left out, the child often performs and cries until allowed into the group. *Phosphorus* children are generally self-centred, and do not like to be considered “average”.

Often, it is found that *Phosphorus* children are thin children, with fine skin, tall for their age, and with a tendency to stare. They are desperate for reassurance. Children requiring *Phosphorus* are generally intelligent, gregarious, sometimes artistic, and exhibit passions that tend to be short-lived and regretted afterwards (Lockie, 1989).

*Phosphorus* may be used to alleviate the following mental symptoms:

- nervous tension, especially from overwork
- loathing exams and homework
- bottling things up

- indifference to family and friends when ill
- want to be in the limelight
- fears: dark, thunder, being alone, death/dying, see catastrophe around every corner
- great sensitivity to atmosphere (Lockie, 1989).

Physical symptoms alleviated by *Phosphorus* include:

- difficulty picking out human voice from other noises
- perspiration during times of stress (mental or physical)
- palpitations from emotions (Lockie, 1989).

The above descriptions of symptoms exhibited by a child patient requiring *Phosphorus* treatment reiterates the idea that *Phosphorus* may be a well indicated remedy for use in the treatment of symptoms of ADHD. In most cases of children with ADHD, diagnostic symptoms of ADHD are usually coexistent with other symptoms such as those listed above (Barkley, 1998), thus suggesting a good match between the patients and *Phosphorus*.

**Table 2.2.: Repertorisation of DSM-IV Criteria**

List of rubrics used, as in DSM-IV diagnostic criteria for ADHD:

- (a) Generals – Restlessness
- (b) Mind – Concentration – Difficult
- (c) Mind – Heedless
- (d) Mind – Chaotic
- (e) Mind – Confusion
- (f) Mind – Absentminded
- (g) Mind – Mental exertion – aversion to
- (h) Mind – Forgetful
- (i) Mind – Loquacity
- (j) Mind – Impulsivity; Mind – Excitement
- (k) Mind – Impatience
- (l) Mind – Dictatorial
- (m) Generals – Sitting – aggravates
- (n) Generals – Activity – Increased; Mind – Physical exertion – desire
- (o) Mind – Sensitive

	(a)	(b)	(c)	(d)	(e)	(f)	(g)	(h)	(i)	(j)	(k)	(l)	(m)	(n)	(o)	<b>TOTAL</b>
Acon	3	2	-	1	2	2	3	2	1	3	2	-	1	2	2	26
Anac	1	3	2	1	2	2	1	2	1	2	1	-	1	-	3	22
Androc	1	2	-	1	1	2	-	2	-	-	2	2	-	-	-	13
Apis	2	2	1	-	1	3	-	1	1	-	2	1	2	-	1	17
Arg-n	1	1	-	-	2	-	1	2	-	3	2	-	-	-	3	15
Ars	3	2	-	2	2	1	1	1	1	2	2	1	3	1	2	24
Bamb-a	1	2	-	-	2	1	-	3	1	1	1	1	-	-	1	14
Bell	2	1	1	2	3	2	1	2	2	3	1	-	1	3	3	27
Calc	1	2	-	-	3	1	2	2	1	2	2	-	1	1	2	20
Camph	1	1	1	-	1	-	-	1	2	2	-	1	1	1	1	13
Cham	2	1	2	-	1	3	1	1	-	3	3	1	1	-	2	21
Coff	2	1	1	-	2	1	-	1	1	3	-	-	1	-	3	16
Cupr	1	2	1	-	2	2	-	1	2	2	-	1	1	-	1	16
Ferr	2	1	-	1	2	-	2	1	-	2	-	1	2	-	2	16
Hyos	3	2	2	-	2	2	1	1	3	3	2	-	1	2	2	26
Lyc	2	3	2	-	2	2	2	3	1	2	2	2	3	-	3	29
Merc	3	2	2	2	3	2	-	3	-	2	1	2	2	-	2	26
<b>Phos</b>	<b>2</b>	<b>3</b>	<b>2</b>	<b>2</b>	<b>2</b>	<b>2</b>	<b>3</b>	<b>3</b>	<b>2</b>	<b>3</b>	<b>-</b>	<b>1</b>	<b>3</b>	<b>2</b>	<b>3</b>	<b>33</b>
Puls	1	2	1	1	2	3	2	1	-	3	2	-	3	-	3	24
Rhus-t	3	1	1	1	3	2	2	2	1	1	2	-	3	-	1	23
Sep	3	3	1	-	3	3	2	1	-	2	3	-	3	-	2	26
Sil	1	3	1	-	3	2	2	1	-	2	3	-	1	-	3	22
Staph	1	1	1	1	2	1	2	1	1	3	1	-	1	-	3	19
Stram	1	2	1	1	2	1	-	1	3	2	-	-	1	2	1	18
Sulph	2	2	1	-	2	2	2	2	1	2	3	1	3	-	3	26
Tarent	3	-	-	-	-	1	-	1	1	2	1	-	-	-	1	10
Tub	-	1	1	-	1	1	1	2	-	-	-	-	1	-	1	9
Zinc	2	1	1	1	2	1	1	2	1	1	1	-	3	-	2	19

*Phosphorus* scores the highest overall score of 33. It scores in all but one of the rubrics used.