NEUROCOGNITION AND NEUROLOGICAL SOFT SIGNS IN CHILDREN WITH ATTENTION DEFICIT HYPERACTIVITY DISORDER

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CERTIFICATE

This is to certify that the dissertation titled, **"Neurocognition and neurological soft signs in children with attention deficit hyperactivity disorder"**, submitted by **Dr.KOMAL. J,** in partial fulfillment for the award of the **MD degree in Psychiatry** by the Tamil Nadu Dr. M. G. R. Medical University, Chennai, is a bonafide record of the work done by him in the Institute of Mental Health, Madras Medical College during the academic year 2012 – 2015.

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DECLARATION

I, Dr. KOMAL.J, solemnly declare that the dissertation titled, **“Neurocognition and neurological soft signs in children with attention deficit hyperactivity disorder”** is a bonafide work done by me at the Madras Medical College, Chennai, during the period from Aug 2014 - Oct 2014 under the guidance and supervision of Dr. JEYAPRAKASH R. MD, DPM, Professor of Psychiatry, Madras Medical College.

The dissertation is submitted to The Tamilnadu Dr. M. G. R. Medical University towards part fulfilment for M.D. Branch XVIII (Psychiatry) examination.

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CONTENTS

ABSTRACT

Context: Attention deficit hyperactivity disorder (ADHD) is a common neurodevelopmental disorder with wide repercussions. Since it is etiologically related to delayed maturation, neurocognition and neurological soft signs (NSS) could be a tool to assess this. Further the correlation of NSS and neurocognition with severity of ADHD and presence of Specific Learning Disability (SLD) would give further insight into it.

Aims: To study neurological soft signs and neurocognition in children with ADHD and to correlate NSS and neurocognition with severity of ADHD and with co morbid Specific Learning Disability.

Settings and Design: The study was carried out in Institute of child health, Department of Child Psychiatry, Madras Medical College. It was a cross-sectional single interview study.

Materials and Methods: 40 consecutive children diagnosed as having ADHD in each group were assessed for the presence of neurological soft signs using Revised Physical and Neurological Examination soft Signs scale (PANESS) and neurocognition using Trail Making Test, Stroop Test, Verbal Fluency, Verbal N Back Test and Continuous Performance Test. The ADHD was assesses by SNAP IV scale.

Statistical Analysis: The data was analyzed using the t-test, chi-squared test and Pearson's co-relational analysis.

Results and Conclusions: Neurological soft signs were more in ADHD with SLD, especially timed movements. As the severity of ADHD increased, neurological soft signs increased in numbers. ADHD with SLD performed poorly in attention, speed of processing and working memory and the performance worsened with increasing ADHD severity, especially inattention severity.

Key words: Attention deficit hyperactivity disorder, neurological soft signs, specific learning disability

INTRODUCTION

Attention deficit hyperactivity disorder (ADHD) is neuro-developmental disorder affecting around 5.2% of school-age population globally(1). ADHD may affect certain areas of the brain that allow problem solving, planning ahead, understanding others' actions, and impulse control(2)(3). It is a characterized by inattention, hyperactivity/impulsivity, or combined, some symptoms of which must be present before the age of 7 years and impairment must be observable in at least two settings. It is two to four times more common in boys(4).

Inattention probably reflects problems with the executive functions of memory(5). However, delay in the development (by 3 yrs.), specially of the frontal and parietal cortex was believed to be responsible for the ability to control and focus thinking(6). In contrast, the motor cortex in the ADHD patients was seen to mature faster than normal, suggesting that both slower development of behavioral control and advanced motor development might be required for the fidgetiness or hyperactivity that characterizes ADHD(7).

Regarding etiology, there's a growing consensus that the condition has neurobiological underpinnings. A general reduction of brain volume was found with the core ADHD features of inattention, hyperactivity, and

impulsivity reflecting frontal lobe dysfunction but other brain regions particularly the cerebellum had also been implicated(6).

Neurological Soft Signs (NSS) are non-normative performance on a neurological examination of motor and sensory functioning in the absence of a focal lesion. They are poor coordination, speed or accuracy of limb or axial movements, including those required to keep the balance, dysrhythmias, and overflow are often found during the clinical examination of young children. They are studied in terms of timed and untimed motor movements(8).

They are considered developmentally normal in young children (under 6) in whom they persist over time when cortical inhibitory functions fail to develop in order to stop the radiation of motoric impulses to body parts other than the target body part. They are indicators of delayed development of motor inhibition(8)

NSS in young adults have been associated with a number of neuropsychiatric and behavioral disorders, such as psychosis, obsessive-compulsive disorder, and also in conditions of atypical development, like autism and learning disability and ADHD(9).

Most cognitive research on ADHD has involved school-age children. Several studies have demonstrated that a number of cognitive problems are associated with ADHD(10). For example, there is strong

evidence that children with ADHD have deficits in executive functions, especially an inhibitory control deficit. A deficient inhibitory control can be described as a failure to withhold or suppress inappropriate responding. This would lead to an impaired ability to use control strategies in order to optimize behavior.

Some investigators believe that the deficient inhibitory control can be explained by non-optimal motivation or by an aversion of delay(11). Other executive processes, such as organizing and planning behavior, goal-directed behavior, working memory, verbal fluency, or the ability to shift response sets during tasks, are also impaired in ADHD children(12)

Cognitive study in young children of ADHD is relevant for several reasons. First, cognitive tests may contribute to the accuracy of the early identification of children at risk of ADHD and provide information about what difficulties exist. Second, this research yields information about the cognitive mechanisms that underlie the symptoms of ADHD(13).

Our review of literature failed to find Indian studies on this subject. Thus, the study was planned with the aim of studying neurocognition, neurological soft signs in children with ADHD and the correlation of neurocognition, neurological soft signs to severity of ADHD and with the co-morbidity of specific learning disability.

REVIEW

OF

LITERATURE

ATTENTION-DEFICIT/HYPERACTIVITY DISORDER

Attention-Deficit/Hyperactivity Disorder (ADHD) is the term devised to label children, adolescents, and some adults, who are absent-minded, easily side tracked, abnormally over excited, and impetuous in their behavior. ADHD is a neurodevelopmental disorder with no definite cause. Various factors have been proposed as a causative factor for ADHD. Management of ADHD involves various disciplines like neuropsychological, medical, parental and educational disciplines(1).

ADHD has been documented for more than a century under diverse terms. It is not a novel diagnosis or a thinking of this twentieth century. Heinrich Hoffman (l809–l874), a German physician and poet, wrote about Fidgety, Philip, l955 who could not be seated still. The poet describes the activities of a kid similar to ADHD. Kids during those times were subject to discipline less liberal than at present-day.

DIAGNOSTIC CRITERIA FOR ADHD SUBTYPES (DSM IV TR)

(l) ADHD Inattentive subtype, without hyperactivity (Code 3l4.00).

At least six of the following nine symptoms have been noted for at least six months and are often present during school or play activities:

1. Makes careless mistakes;

2. Cannot maintain attention;

3. Does not listen when spoken to;

4. Fails to finish tasks;

5. Seems disorganized;

6. Avoids tasks;

7. Loses things;

8. Easily distracted;

9. Forgetful.

(2) ADHD Hyperactive-Impulsive subtype (Code 3l4.0l). Six (or more) of the following symptoms have been present for at least six months:

Hyperactivity:

l. Fidgety;

2. Leaves seat in classroom or at dinner table;

3. Runs or climbs excessively;

4. Cannot play quietly;

5. Always "on the go";

6. Talks a lot.

Impulsivity:

7. Blurts out answers to questions;

8. Cannot wait in line or take turn;

9. Often interrupts.

(3) ADHD Combined type.

Criteria for both the Inattentive and the Hyperactive-Impulsive types have been present for at least 6 months. The term In Partial Remission is applied to older children and adolescents whose symptoms have lessened with age or treatment and no longer add up to the required number for diagnosis(4).

Further Diagnostic Criteria

1. The typical diagnostic symptoms of ADHD are a persistent pattern of inattention and/or hyperactivity and impulsiveness of an abnormal severity and frequency.
2. Symptoms should have been present before the age of 7 years.
3. Symptoms should be observed in at least two settings (school, home, workplace, or doctor's or psychologist's office).
4. Symptoms are sufficient to impair academic, social, or occupational functions.
5. Symptoms cannot be explained by a mental illness such as depression, anxiety, or personality disorder(4).

A neurological examination may uncover soft neurological signs and psychological assessment may show deficits in perception and learning ability. They can be additional supportive evidence, but not essential for the diagnosis of ADHD. A specific chemical or laboratory test is not available. Rarely abnormal lead levels in the blood, thyroid dysfunction, or certain chromosomal anomalies (fragile X disease) may offer a clarification for the symptoms(14).

ADHD IN DSM 5

There are some changes in the DSM-5 for the diagnosis of ADHD. Symptoms can now occur by age l2 rather than by age 7; several symptoms now need to be present in more than one setting rather than just some impairment in more than one setting; new descriptions were added to show what symptoms might look like at older ages; and for adults and adolescents age l7 or older, only 5 symptoms are needed instead of the 6 needed for younger children(15).

PREVALENCE OF ADHD AND GENDER FACTOR

Approximately 5% of children and adolescents are affected, or at least one in every classroom. Boys are affected three to six times more commonly than girls. Some authorities have estimated the prevalence as high as l0%, and even 20%, in school children between 5 and l2 years of age(16).

One report claimed a total of 3 million children with ADHD in the United States. ADHD is recognized worldwide, but the reported prevalence varies in different countries, with less than 1 in a l000 in a study of l0- and 11-year-old children in the Isle of Wight, UK (17).

The accuracy and comparison of these statistics are affected by the age of the study population, the variability of the patient selection, and the lack of agreement on the definition of diagnostic criteria. In a study at Vanderbilt University, Nashville, TN, involving 8000 children in a Tennessee county, with ratings completed by 400 teachers, the estimates of prevalence of ADHD was higher when using the new diagnostic criteria listed in DSM-IV, as compared to DSM-III-R criteria(18).

Prevalence rates were 7% for ADHD using DSM-III-R, and 11% with DSM-IV criteria, an increase of 57%. The inattentive (AD) subtype of ADHD occurred in 5%, the hyperactive-impulsive (H-I) type in 2.5%, and the combined type in 3.5%. Boys outnumbered girls with a 4:l ratio for the ADHD-HI and 2:l for ADHD-AD(18).

AGE OF ONSET OF ADHD

According to the DSM-IV criteria for diagnosis of ADHD, some symptoms should be present before the age of seven years. Hyperactivity is recognized most commonly at about four or five years of age, when the child starts school, although many parents complain about excessive

motor restlessness in infancy. In fact, some mothers have predicted the birth of a hyperactive child because of excessive fetal movements during pregnancy(19).

The environment will often influence the time of onset of symptoms. A child who is mildly restless at home or in the doctor's office may become hyperactive and distractible when entering a structured situation, such as a school classroom. On a one-to-one, student-teacher ratio, as in private tutoring, the child may function reasonably well, whereas in a large class of students, the symptoms of ADHD will immediately become apparent. Parents are sometimes dismayed at the reports from school, because in the home environment symptoms can be less obvious(20).

CAUSATIVE FACTORS

ADHD is a highly heritable disorder but in addition to genetic causes, acquired and environmental factors are sometimes uncovered that may be amenable to prevention or specific treatment. The causes of ADHD may be characterized as idiopathic, arising spontaneously from an unknown cause, symptomatic and secondary to a brain structural abnormality, or familial and presumed genetic. A majority of cases of ADHD are idiopathic or of uncertain cause. A delay in development or maturation of the nervous system is sometimes proposed as an

explanation for ADHD, especially in children with mild or "soft" neurological deficits(21).

ETIOLOGICAL CLASSIFICATION

The etiologies of ADHD are sometimes classified by the time of their occurrence:

Etiologic Classification of ADHD		
Group	*Timing*	*Etiologic Factors*
Genetic		Dopamine deficit, idiopathic
Acquired	Prenatal	Developmental cerebral abnormality, chromosome anomaly, viral exanthema, alcohol, nicotine, lead, cocaine, anemia, hypothyroidism, iodine lack
	Perinatal	Prematurity, low birth weight, anoxic-ischemic encephalopathy, meningitis, encephalitis
	Postnatal	Viral meningitis, encephalitis, cerebral trauma, iron deficiency, fatty acid deficiency, thyroid dysfunction, otitis media

The syndrome may be genetic and familial, or acquired and environmental. Rarely, a chromosomal anomaly is the underlying cause of ADHD.

Prenatal causes include developmental cerebral abnormality, maternal anemia, toxemia of pregnancy, alcohol and cocaine abuse, and tobacco smoke(22). Other environmental factors sometimes suspected are

exposure to lead, PCBs and pesticides in the water and diet, lack of iodine and hypothyroidism(23).

The season of birth may be a risk factor, and exposure to viral infections, especially influenza and viral exanthema, in the first trimester of pregnancy or at the time of birth has been correlated with the diagnosis of ADHD(24).

Perinatal etiological factors include the following: premature birth, breech delivery, anoxic-ischemic-encephalopathy, cerebral hemorrhage, meningitis, and encephalitis(25).

Postnatally, the infant may have suffered a head injury, meningitis, encephalitis, frequent attacks of otitis media, or low blood sugar(26).

Drugs used to treat childhood illnesses, asthma and epilepsy, frequently cause or exacerbate hyperactive behavior and result in attention and learning deficits. The role of diet in the cause of ADHD is controversial, but the ingestion of food additives and sucrose, lack of omega 3 fatty acids, and allergies to certain foods are occasionally significant(27).

A lack of iron in the diet and anemia are documented potential causes and rarely, thyroid hormone dysfunction is associated with ADHD (28).

An abnormality in sensory input alleviated by oral potassium is proposed as a novel mechanism of ADHD in a 9-year-old boy with symptoms of sensory overstimulation and potassium sensitivity (29).

ROLE OF GENETIC FACTORS IN ETIOLOGY OF ADHD

Parents will often admit that fathers and, less often, the mothers were hyperactive or had a learning problem during childhood. Occasionally, they will deny any childhood behavior or attention problem, despite their inability to sit quietly during the consultation. A history of siblings and cousins who have been diagnosed with ADHD and who have had a favorable response to stimulant medications is not uncommon.

The clear distinction between the effects of nature and nurture in the cause of ADHD is difficult to prove, and both genetic and acquired factors are important. In some patients, the cause may be purely inherited, in others, mainly acquired and environmental, and in many, a combination of both(30)(31)(32).

CHROMOSOMAL ANOMALIES ASSOCIATED WITH ADHD

Chromosomal syndromes are rare among ADHD-clinic patients. They include fragile X, velocardiofacial (22Q.ll.2 deletion), Williams, Turner and Prader-Willi syndromes, and neurofibromatosis type l (33).

In the absence of clinical signs or family history, routine chromosome analysis in children with ADHD is not generally recommended(33).

MOLECULAR GENETIC STUDIES

The above reports point to the role of genetic factors in the cause of ADHD. The identification of a specific metabolic or enzyme marker is also required to prove an inherited predisposition. Studies focused on catecholaminergic candidates support the involvement of the dopamine receptor and dopamine transporter genes (DAT1)(34).

Deficits in dopamine-modulated frontal-striatal circuits are correlated with subtypes of ADHD. The relation of dopamine deficits to fetal and perinatal stresses may explain the mechanism of environmental etiologies of ADHD (35).

Preterm birth complicated by susceptibility to cerebral ischemia may contribute to increased dopamine receptor availability, deficient dopaminergic transmission, and subsequent development of ADHD(25).

Evidence of environmental mediators in ADHD has been demonstrated in twin studies. Affected twins have greater exposure to risk factors such as maternal smoking, lower birth weights, and delayed growth and development compared with unaffected co-twins (36).

Gene-environment interaction is increasingly recognized as an important mechanism in the etiology and development of ADHD, with some genes (e.g. DATl) affecting the individual sensitivity to environmental etiological factors (37).

NEUROLOGICAL BASIS FOR ADHD

The neurologic or anatomic theory of hyperactivity and ADHD is based on numerous experimental studies in animals, neurological and electroencephalographic (EEG) examinations, and magnetic resonance imaging (MRI) of the brain(38). Positron emission tomography (PET) studies, showing changes in glucose metabolism in the frontal lobes of the brain, point to a localized cerebral abnormality in adults who were hyperactive since childhood(38).

Neurological "soft" signs, including motor impersistence (an inability to maintain postures or movements), distractibility (an inability to maintain attention), and attentional control and response inhibition, are indicative of right-sided frontal cerebral lesions.

Frontal cerebral lesions and their connections with the basal ganglia or striate cortex produce the greatest number and degree of hyperactive behavioral responses. The right prefrontal cortex has a role in attentional control and inhibiting responses, whereas the basal ganglia are involved in motor control and the execution of behavioral responses.

Distractibility and impulsivity in ADHD children reflect deficits in response inhibition(39).

Injury or abnormal development of areas of the brain other than the frontal lobes may also be associated with the syndrome of ADHD and impairment of language and social skills. Cognitive dysfunction and ADHD are reported in children with temporal lobe lesions, and a connection with the fronto-striatal circuitry is possible in these cases.(38)

Localized cerebral hemisphere and cerebellar anomalies of development in ADHD are correlated with abnormal fronto-striatal-cerebellar function and sometimes with response to stimulant medication. (40).

The different anatomical sites of injury or lesion in the brain, sometimes detected in children with ADHD, can account for the varying symptoms and complications of the syndrome. (21).

A frontal-motor cortex disconnection syndrome, or "lazy" frontal lobe, in ADHD is hypothesized on the basis of cerebral blood flow, EEG studies, and MRI volumetric analyses (41). This concept is developed from the function of the frontal lobe as an inhibitor of excessive motor activity, children with ADHD having disinhibited motor activity. The calming effect of methylphenidate may stem from a stimulatory effect on the frontal lobe causing motor inhibition(42).

BIOCHEMICAL BASIS FOR ADHD

Evidence is accumulating that changes in the brain chemistry – the catecholamine neurotransmitters (dopamine, norepinephrine, and serotonin) – might account for hyperactivity, inattentiveness, and other symptoms of ADHD. The central nervous system stimulants, dextroamphetamine and methylphenidate (Ritalin R), benefit ADHD by increasing catecholamine concentrations in the brain. Catecholamine metabolism and levels of norepinephrine are related to arousal, attention span, and motor activity.

The biochemical studies in children with ADHD are experimental. Measurements of metabolites, or breakdown products, of dopamine and norepinephrine in the urine or of enzymes in the blood are not of practical significance in the diagnosis and treatment of ADHD, but they increase our understanding of the neurobiology of ADHD (33,43).

SYMPTOMS AND SIGNS OF ADHD

The symptoms of ADHD are outlined in the DSM-IV diagnostic criteria in two main subtypes or groups: (1) symptoms of inattentiveness and (2) hyperactivity & impulsivity.

Signs of brain dysfunction and associated perceptual and learning disabilities are omitted from the current definition, as outlined by the

American Psychiatric Association. The recognition of both symptoms and signs of ADHD is important, however, particularly in terms of defining the cause and treatment.

ADHD as defined by the DSM-IV rarely occurs alone. Certain neuropsychiatric disorders frequently complicate the diagnosis of ADHD and often modify the treatment. Many of these disorders are neurological, including headache, seizures, tics or Tourette syndrome, and speech and language and motor coordination problems(40).

Others are psychiatric or neuropsychological in nature, principally oppositional defiance disorder (ODD); conduct disorder (CD), and learning disorders. The differential diagnosis, or conditions that may present with some of the symptoms of ADHD, includes bipolar disorders (depression, dysthymia), pervasive developmental disorders (autism, Asperger's syndrome), personality disorders (obsessive compulsive disorder (OCD)), and mental retardation syndromes(44).

OCCASIONAL INATTENTIVENESS OR AN ATTENTION DEFICIT DISORDER?

Most children have periods of "day dreaming" in school when attention wanders transiently, but not sufficiently to impair learning. Inattentiveness becomes an attention deficit disorder (ADD) when the child is unable to sustain attention and is frequently distracted by outside

stimuli. In order to attend, the child must ignore or tune out irrelevant distracting stimuli. The child with ADD fails to inhibit the background "noise" in the classroom environment(9). Symptoms of ADD also include a listening problem, forgetfulness, weakness in organization, and inability to complete a task.

If the inattentiveness is episodic and the child appears confused, the possibility of absence or partial complex seizures is considered and an electroencephalogram (EEG) is recommended. The distinction between a sustained inattentiveness, characteristic of ADD, and seizures is important in determining the medical management. Stimulant medication prescribed for ADD may worsen the episodes of inattention related to a seizure disorder. A non-stimulant medication is often preferred for children with ADD and an abnormal EEG(45).

WHEN IS HYPERACTIVITY ABNORMAL?

Children normally have an excessive degree of motor restlessness at times, particularly in emotionally charged environments. Hyperactive behavior is abnormal when accompanied by short attention span and distractibility, and when it is purposeless, inappropriate and undirected toward a specific, meaningful goal. The inability to focus and perform structured tasks is the hallmark of the hyperactive school-age child. The quality and direction of the hyperactivity are abnormal, not necessarily

the total daily activity. Hyperactivity is frequently accompanied by impulsivity, a tendency to interrupt others and inability to wait in line.(45)

The child with ADHD is often restless in infancy. As a toddler, "he is into everything," and has to be watched constantly for his own protection and that of household breakables. In later childhood, he is constantly fidgeting, always "on the go," and is unable to sit still at the dinner table. At school, the teacher also reports an inability to sit still; he gets up and walks around in the classroom, he talks excessively, interrupts, and tends to distract and disturb others. The motor hyperactivity is often accompanied by "verbal hyperactivity," and sometimes a flight of ideas, without focus on the topic of conversation.

In anatomical studies of the origin of hyperactivity, two types are distinguished: (1) over activity caused by frontal lobe injury and a response to external environmental stimulation; and (2) essential over activity caused by striatal lesions and a release of motor activity normally inhibited by frontal-striatal connections in the brain (21). We may infer that some children with ADHD are overactive only when stimulated by a noisy environment, whereas others exhibit a constant uninhibited motor activity unrelated to the environment. The hyperactivity may appear

normal in the playground but abnormal and inappropriate in the classroom.

COMORBID DISOREDERS

Psychiatric disorders sometimes associated with ADHD include learning disability, oppositional defiant disorder, conduct disorder, mood disorders, and anxiety disorders, including obsessive compulsive disorder. These are referred to as comorbid disorders. They complicate the management of ADHD and, if severe enough to impair school and social functioning, will require psychiatric or psychological intervention(44).

PHYSICAL AND NEUROLOGICAL EXAMINATIONS

The general physical examination includes measurements of head circumference, height and weight, vision and hearing, heart sounds and blood pressure, birthmarks and congenital developmental anomalies or dysmorphisms. The neurological examination should test for subtle neurological abnormalities or soft signs, including dyspraxia of gait and incoordination, dysdiadochokinesia, mirror movements, motor impersistence, graphaesthesia, handwriting problems, handedness, right–left disorientation, finger agnosia, dyscalculia, reading disorder, and speech and language delay. The recognition of subtle neurological abnormalities is an indication of delayed maturation or complicating

visual perception and learning disorders. These findings may impact the response to treatment and outcome of ADHD(46).

"SUBTLE" OR "SOFT" NEUROLOGICAL SIGNS

Mild or subtle neurologic abnormalities are sometimes referred to as "soft" signs. Many children with ADHD are described as clumsy or uncoordinated. They may be poor at sports, especially basketball and activities requiring a quick reaction and facile movements(47). Soft signs are usually indicative of immaturity or delayed development of the nervous system. Sometimes, the clumsiness and signs will persist into adult life. Unlike cerebral palsy, the incoordination of movement is not associated with obvious muscle weakness or spasticity. Movements (synkinetic or mirror) that are normally inhibited by 5–7 years of age persist into older age groups, and coordination abilities (hopping and tandem gait) usually accomplished by five years are delayed(8). In eliciting motor function, the speed, overflow, and rhythm of movement are important characteristics of subtle neurological abnormalities. Timed repetitive finger tapping, foot-to-hand overflow in tandem gait, and failure to maintain a steady rhythm of motor movement are examples used in quantitative tests of neurological assessment of subtle signs (9).

Attention deficit/hyperactivity disorder (ADHD) is the behavioral disorder most commonly diagnosed in childhood. In addition to the main

symptoms of inattention, impulsiveness and hyperactivity, neurological soft signs (NSS) are often associated with ADHD. NSS are discrete motor and sensory disorders that cannot be linked to specific cerebral lesions. Review of literatures support the presence of an alteration in the neural networks for motor control inhibition, at the base of the pathophysiology of NSS in children with ADHD, as well as a possible central role of dopamine in these neural circuits.

Motor impersistence is a characteristic neurological soft sign of ADHD. The term was first coined to describe the difficulty in maintaining a motor behavior experienced by brain injured adults, even though they had no problem in initiating or performing the movements. The injury was located in the right hemisphere of the brain, especially the frontal lobe, an area often involved in ADHD. Motor impersistence is manifested by an inability to maintain movements such as the following on request: "close your eyes," "look at my nose or finger," "put out your tongue," or "hold your arms outstretched"(48)

In addition to motor impersistence, the child with ADHD demonstrates an inability to inhibit responses. A child will be unable to look away from a stimulus when requested. When asked to hold out the arms and stand still, he will frequently initiate other movements such as walking. The teacher will complain, "he can't keep his hands to himself."

If he sees a pencil or a pair of scissors, he is unable to look at the object without putting it to its intended use, a sign of "utilization behavior." Deficits in response inhibition are a reflection of inattentiveness, the tendency to respond to distracting stimuli, and of impulsivity(48).

Dyspraxia or apraxia is another soft neurologic sign commonly recognized in association with ADHD. Dyspraxia is a loss or delayed development of dexterity in purposeful movements, as in hopping, tandem walking, or the use of scissors, despite normal muscle strength. The term dyspraxia is also applied to an inability to protrude the tongue on command, yet the movement is carried out involuntarily. A delay in speech may be a form of apraxia. A constructional apraxia is an inability to build blocks or copy simple designs. Dyspraxias are caused by dysfunction or damage to the frontal lobes of the brain.

Dysdiadochokinesia is the neurological term for clumsiness in the performance of rapidly alternating movements (pronation and supination) of the forearms. Children aged 5–7 years should be able to imitate the examiner's movements without mirroring the movement in the opposite forearm. Involuntary mirror movements are referred to as synkinesia, and are a common sign of minimal brain dysfunction in ADHD. When the child is older he is usually able to inhibit these mirror movements. Ataxia and incoordination, also described as clumsiness, may represent an

immaturity or damage to the cerebellum and its connections. An attempt to walk a straight line is performed unsteadily, and finger-to-nose movements of the upper limbs bring out a tremor. Choreiform movements are involuntary jerky movements, usually demonstrated by asking the child to stretch out his arms. Choreiform movements are considered as a sign of minimal brain dysfunction, an earlier term for ADHD(49).

Graphaesthesia, an inability to recognize numerals traced on the skin of the palms or the back, is a common finding with ADHD. It is indicative of dysfunction of the parietal lobes of the brain.

Other neurological signs sometimes elicited include a tendency to walk on the toes due to tight heel cords or contractures of the Achilles tendons, and Babinski signs, an extension of the great toe and fanning of the second to the fifth toes when the plantar surface of the foot is stroked with a blunt object. These signs are not included under the term "soft" neurological abnormalities, since they commonly persist and may be evidence of permanent dysfunction of the pyramidal tracts and cerebrospinal motor system(39).

Soft neurological signs were of predictive value for learning disabilities in preschool children, aged 3–5 years, in a study at the Wyler Children's Hospital, University of Chicago, IL (50). A poor neurological

test score at age five correlated with a lower Full-scale IQ at age seven. Neurologic soft signs accurately identified nearly all the children who needed special educational help. Abnormal neurologic signs identical to those included in the above test battery were previously correlated with hyperactive behavior, ADHD, and a beneficial response to stimulant medication, in a study at Northwestern University Medical School, Chicago (42).

A careful examination and recording of neurological abnormalities help in our understanding of the causes and anatomical lesions in the brain that may explain the mechanism of ADHD. The need for special diagnostic laboratory investigations such as EEG and MRI is determined by the neurological history and examination findings(41).

LEARNING AND LANGUAGE DISORDERS IN ADHD

Learning and language disorders frequently complicate attention deficit disorder, and their recognition and remediation are essential for the successful management of ADHD. Specific learning disorders that involve the "three Rs" are termed dyslexia, dysgraphia, and dyscalculia. Dyslexia or reading disability is the prototype of learning disabilities. Speech and language disorders include dysarthrias, or disorders of articulation, and aphasias or dysphasias, inabilities to comprehend and use language despite normal hearing and intellect(51).

Of 119 children, ages 8–16 years, evaluated in a child diagnostic clinic at Department of Psychiatry, Penn State University College of Medicine, a learning disability (LD) was present in 70% of children with ADHD. LD in written expression was two times more common (65%) than an LD in reading, math, or spelling. Children with LD and ADHD had more severe learning problems than children who had LD but no ADHD. LD and attention problems are on a continuum, are interrelated, and usually coexist (52). Comorbidity with LD is a modifying factor in the health-related quality of life of children with ADHD.

At the University of British Columbia, Canada, a questionnaire survey of 131 families of children diagnosed with ADHD found 51(39%) with comorbid learning disorder, and 45(34%) having oppositional defiant disorder or conduct disorder (53).

Estimates of the prevalence of ADHD and learning disability (LD) in US children 6–17 years of age, 2004–2006, at the National Center for Health Statistics, Hyattsville, MD, found 5% of children had ADHD without LD, 5% had LD without ADHD, and 4% had both ADHD and LD. Boys were more likely than girls to have each of the diagnoses (14). Neurological assessment is recommended in children with learning disabilities who fail to show academic progress despite appropriate educational intervention.

At the Neuropediatric Unit, Shaare Zedek Medical Center, Jerusalem, Israel, 7 third-grade children with developmental dyscalculia had neurological disorders with direct bearing on the cognitive disabilities and remedial therapy (54). Four had ADHD, one had absence seizures, one had dyslexia for numbers, and another had Gerstmann syndrome, a parietal lobe disorder resulting in LD affecting handwriting and arithmetic.

A sample of 235 families with ADHD was assessed for familial association with LD at the University of California, Los Angeles. The prevalence rates were highest for reading disability, followed by writing (dysgraphia), then math disability (dyscalculia). Strong familial association was demonstrated for reading disability, with weaker association for writing disability. Independent familial factors appeared to underlie ADHD and LD (55).

THE GENETIC FACTOR IN READING DISABILITY

The strong predilection for boys is well-documented but the method of genetic transmission of dyslexia is less well defined. Nancy Millichap, in her thesis on dyslexia (1986), reviewed studies of twins and found evidence in support of a genetic influence. Of a total of 96 twin pairs reported in the literature, 36 (88%) monozygotic twins were concordant for dyslexia compared to only 16 (29%) dizygotic twins.

Between 25 and 50% of children with reading disability show a hereditary influence, with autosomal dominant, sex-linked recessive, and polygenetic transmission. The genetic pattern depends in part on the definition and the association with other learning disabilities in the families studied(45).

A subsequent twin study at the Hospital for Sick Children, London, UK, showed that genetic factors play a moderate role in reading retardation and a stronger influence in spelling disability (37). A twin study at the University of New South Wales, Australia, involved children with ADHD complicated by reading and speech problems. Male twins were affected more frequently than female, and the reading disability in male twins became more severe in adolescence while that in female twins showed improvement (36).

ADHD and reading disability tend to co-occur, and molecular genetic studies support the hypothesis that the alpha 2A adrenergic receptor (ADRAZA) gene is contributing to this association. Linkage and association studies in dyslexia suggest that a susceptibility locus exists on chromosome 15q15- q21. Further systematic studies are required to identify the true dyslexia susceptibility gene(s)(37).

EVIDENCE FOR A NEUROANATOMICAL BASIS FOR DYSLEXIA

Neuroanatomical and related studies in dyslexics have led to a so-called neural signature of dyslexia (56). Since the original Harvard University study showing cerebral developmental anomalies in CT scans of four dyslexic male subjects(51), the findings have been confirmed at autopsy in three women with dyslexia. Microscopic scars in the brains were linked to lupus erythematosus in the mother, and an immune mechanism for dyslexia was proposed (57). Left-handedness was more important than immune disorders in a study of associated factors in dyslexic children at the Center for Reading Research, Stavanger, Norway(58).

A reappraisal of the anatomical basis for dyslexia using MRI studies at Yale University School of Medicine, New Haven, CT, concluded that differences in sex, age, handedness, and the definition of dyslexia could explain discrepancies in brain region volumes in children with dyslexia and other learning disabilities (59). A small corpus callosum was a further cerebral anomaly discovered in MRI studies of dyslexic children at the University of Georgia, Augusta (60). Familial left-handedness and ADHD distinguished the dyslexic children from control children in the study. Measurements of the corpus callosum in

genetic studies of twin pairs at Dartmouth Medical School (1989) showed greater conformity and might be more reliable in anatomical dyslexic studies(21).

MRI and CT measurements of brain regions in dyslexic subjects have provided evidence of anomalies suggesting interruptions in brain development, possibly related to immune mechanisms. These studies are investigational and the anomalies are insufficiently documented for use in diagnosis.

In addition to studies of the neuroanatomy of developmental dyslexia in subjects born with the disorder, occasional cases are described in adults with acquired dyslexia who have undergone surgery for cerebral tumor or other discretely localized brain lesions. A right-handed woman, a patient at the University of Iowa College of Medicine, developed severe dyslexia and dysgraphia following the surgical removal of a small tumor located in the left premotor frontal lobe. By contrast, she was able to write numbers and perform written calculations without difficulty. The isolated simultaneous occurrence of dyslexia and dysgraphia, without dyscalculia, is rare. This case report suggests that the frontal lobe of the brain may be involved in some cases of dyslexia(61).

NEUROPSYCHOLOGICAL PROFILE OF SLD

Studies of individuals with and without reading difficulties suggested that phonological decoding, defined as the ability to translate sequences of printed letters into the corresponding sounds, has a central role in both normal and abnormal reading development(62). The unique contribution of phonological decoding (PD) to most cases of RD has been suggested by the presence of significant group deficits in PD when older children with RD are compared with younger normal readers at the same reading level(63). Moreover, twin studies showed that there are strong genetic influences on the group deficit in PD and that these genetic influences overlap largely with genetic influences on the group deficit in word reading (64).

Deficits in PD and word reading are, in turn, linked to genetic influences on deficits in the oral language skill of phoneme awareness (PA), defined as the ability to recognize and manipulate the phonemic constituents of speech. Many regard problems with PA as the most proximal cause of most cases of RD(65).

Finally, whereas some studies suggest that individuals with RD may also exhibit mild deficits in additional areas such as visual tracking(63), deficits on measures of PA or other measures of

phonological processing consistently account for a much larger proportion of the reading deficit(56).

Along with phonological deficits executive functions are also impaired related to receptive linguistic skills. A group of 43 heavily-affected young dyslexics, divided into two groups based on the results of a receptive language test, and 20 non-dyslexic controls, were tested with a Dichotic Listening Test, the Stroop Color Word Test and the Wisconsin Card Sorting Test. The dyslexic subjects demonstrated significant impairment on all tasks, but with different patterns of impairment according to the subgrouping. The subgroups were equally impaired on the Dichotic Listening Test, but differed on the Stroop and the Wisconsin Tests. The data support a hypothesis suggesting executive problems in dyslexia, depending on receptive language skills(66).

Neuroimaging studies mention different brain activation in working memory tasks compared to controls. In a study, dyslexic readers were compared with the control group. Compared with the dyslexic readers, controls showed increased fMRI activation in the left superior parietal lobule and the right inferior prefrontal gyrus. Unlike controls, dyslexics did not show a significant increase in activation in WM areas with increased memory load. These findings provide support for a specific working memory deficit in dyslexic individuals(67).

NEUROPSYCHOLOGICAL PROFILE OF ADHD

A growing body of research suggests that ADHD is associated with a core deficit in executive functions(12), defined as cognitive functions that serve to maintain an appropriate problem-solving set in order to attain a future goal(68)

These EF deficits are particularly pronounced in the domain of behavioral inhibition, as indexed by measures such as commission errors on a continuous performance test(69) and speed of response inhibition after an auditory stop signal(70)

Some studies found that children with ADHD exhibit deficits in additional EF domains such as cognitive flexibility and verbal working memory as well as slower and more variable response speed across cognitive tasks(71).

Attention deficit hyperactivity disorder (ADHD) comprises a deficit in behavioral inhibition. A theoretical model is constructed that links inhibition to 4 executive neuropsychological functions that appear to depend on it for their effective execution: (a) working memory, (b) self-regulation of affect-motivation-arousal, (c) internalization of speech, and (d) reconstitution (behavioral analysis and synthesis). Extended to ADHD, the model predicts that ADHD should be associated with

secondary impairments in these 4 executive abilities and the motor control they afford(12).

Motivation and organization aspects of executive function appear particularly important for functioning in real life. A prospective longitudinal study of 59 college students evaluated whether parent-ratings and self-ratings of executive function (EF) predicted the academic and overall functioning of college students with ADHD and whether EF deficits mediated the relationship between ADHD symptoms and functioning. Student-rated motivation and parent-rated emotion regulation significantly predicted overall impairment above and beyond symptoms of ADHD. Student-rated EF motivation mediated the relationship between ADHD symptoms and overall impairment. Student-rated EF organization mediated the relationship between ADHD symptoms and end of the year grades(72).

Verbal fluency tasks are commonly used in cognitive and developmental neuropsychology in assessing executive functions, language skills as well as divergent thinking. Twenty-two typically developing children and 22 children with ADHD between the ages of 8 and 12 years were examined using verbal fluency tasks, prepotent response inhibition, and working memory tests. The clinical group showed impaired inhibitory and spatial working memory processes.

Qualitative analysis of verbal fluency tasks was done to explore the lexical and executive strategies (word clustering and switching), and the temporal properties of the responses. Children with ADHD had a leeway in applying relevant lexical or executive strategies related to difficulties in strategy using. The reduced efficiency of children with ADHD in semantic fluency task is based on suboptimal shifting between word clusters and is related to the lack of ability of producing new clusters of items. The group difference appeared at the level of accessing and/or activating common words; however, the executive process of searching the lexicon extensively is intact(71).

Meta-analyses indicate that the gene coding for the dopamine transporter (DAT1 or SLC6A3) is associated with an increased risk for ADHD. The mechanisms of this gene for ADHD are unclear. Studies linking the VNTR in the 3' UTR of the DAT1 to neurophysiological and neuropsychological measures were reviewed. In addition, a broad set of executive/cognitive and motor tests was administered to 350 children (5-11 years) and adolescents (11-19 years) with ADHD and 195 non-affected siblings. Two VNTRs (in intron 8 and the 3' UTR) and four SNPs (two 5' and two 3') in DAT1 were genotyped. The effect of the polymorphisms on neuropsychological functioning was studied. The review indicated that the majority of studies did not find a relation

between DAT1 and neurophysiological or neuropsychological measures. In our sample, several of the polymorphisms of DAT1 were associated with ADHD and ADHD was associated with impaired neuropsychological functioning. However, none of the DAT1 polymorphisms was convincingly associated with neuropsychological dysfunctioning. This suggests that the effect of DAT1 on ADHD was not mediated by neuropsychological performance. However, since DAT1 is mainly expressed in the striatum and not the prefrontal cortex, it may influence striatum-related functions (such as delay aversion) more heavily than prefrontal related functions (such as executive functions). Associations of DAT1 with ADHD were only found in adolescents, which may suggest that DAT1 mainly exerts its effect in adolescence, and/or that having a more persistent form of ADHD may mark a more severe or homogeneous genetic form of the disorder(73)

The consideration of age of onset of impairment as part of the ADHD diagnosis is controversial and has been a revisited issue with the emergence of the new classifications in Psychiatry. A study was done to compare patients with early and late onset of ADHD impairment in terms of neuropsychological and personality characteristics. Adult patients with ADHD (n = 415) were evaluated in the ADHD outpatient program at Hospital de Clínicas de Porto Alegre, Brazil. The diagnostic process for

ADHD and comorbidities was based on DSM-IV criteria. The comparison between the two ages of onset groups (before 7; n = 209 or from 7 to 12 years; n = 206) was performed with ANOVA, followed by Stepwise forward regression analyses to restrict the number of comparisons and access the possible effect of multiple confounders. Patients with early onset ADHD present higher scores in novelty seeking in both analyses (respectively P = 0.016 and P = 0.002), but similar cognitive and attention features as compared with the late onset group. These data add to previous evidence that despite a more externalizing profile of early onset ADHD, the overall performance is similar reinforcing the need for awareness and inclusion of the late onset group in DSM-V diagnostic criteria(74).

ADHD is associated with impaired performance on measures of response inhibition, working memory, and other aspects of executive functions, yet data also suggest significant neuropsychological variability within and across ADHD samples(13).

Other findings, however, suggested that individuals with ADHD are not significantly impaired on these measures(75,76)

Moreover, many of these studies did not assess important covariates such as IQ and academic achievement, leaving open the possibility that many of the EF deficits associated with ADHD could, in

fact, be attributable to differences in IQ or to the association between ADHD and RD(77).

NEUROPSYCHOLOGICAL PROFILE OF ADHD WITH SLD

Neuropsychological profile of ADHD with SLD have shown deficits in both phonological fluency and executive functioning compared to ADHD alone or SLD alone

		MEASURES		
STUDY	GROUPS	EXECUTIVE FUNCTIONS	PHONOLOGICAL PROCESSING	MAIN FINDING
Ackerman & Dykman (1993)	CC,AD,RD		Bradley Oddball Rhyme Ident.	RD < AD, CC
August & Garfinkel (1990)	NC, AD, AD + RD	PMT, SOT		AD, AD + RD < NC
Barkley et al. (l992)	NC, LD, AD	Stroop		CPT omissions: AD > LD
		CPT, TMT WCST		Stroop: LD, AD < NC
				WCST, TMT: no differences
Dykman & Ackerman	NC, AD, AD + RD	TMT		AD, AD + RD > NC on DRL

(1991)				
		Gordon DRL		TMT: no differences
Javorsky (1996)"	NC, AD, LD, LD + AD		LAC	LD + AD < NC
Klorman et al. (1999)	NC, AD, RD, RD + AD	WCST, TOH		WCST: no differences
				TOH: ADHD, RD main effect
Korkman & Pesonen (1994)"	LD, AD, LD + AD	NEPSY	NEPSY	Inhibition: AD < LD
		Inhibition	AAS	AAS: LD < AD
Kupietz (1990)	NC, RD, RD + AD	CPT		RD, RD + AD > NC
McGee et al. (1989)	NC, RD, AD, RD + AD	ROCFT, TMT		ROCFT: RD + AD < RD, AD
		WCST		TM, WCST: no differences
Narhi & Ahonen (1995)	CC, RD, AD, RD +	WCST, TMT		No differences on EF

	AD			composite
Nigg et al. (1998)	NC, RD, AD, RD + AD	ROCFT, TTD-20		ADHD, ADHD + RD impaired
		Porteus mazes		on EF composite
		Underlining		
Pennington et al. (1993)	NC, RD, AD, RD + AD	TOH, CPT	Pig Latin	PP: RD, RD + AD < NC, AD
		WCST, MFFT	Word Attack	EF: AD < NC, RD, RD + AD
Reader et al. (1994)	AD, RD + AD	WCST, TOVA		EF composite: AD = AD + RD
		ROCFT, Word F		
Robins (1992)	LD, AD, LD + AD	TMT, ROCFT		Discriminant function with
		MFFT, CPT MFFT,		GDS discriminates groups
B. A. Shaywitz et al. (1995)	NC, RD, AD, RD +	Underlining	Aud, An. Test	PP: RD, RD + AD < NC, AD

	AD		
		Emb. Phonemes	Underlining task: AD < RD, RD + AD, NC
Weyandt et al. (1998)	NC, RD, AD adults	TOH, WCST	WCST: RD < NC; no other significant differences
		TOVA	

Note. CC = clinic control; NC = normal control; RD = reading disability or reading disorder; AD or ADHD = attention-deficit disorder with hyperactivity or attention-deficit hyperactivity disorder; LD = learning disability; Rhyme Ident. = rhyme identification task; PMT = Progressive Maze Test: SOT = Sequential Organization Test; Stroop = Stroop Color and Word Test; CPT = Continuous Performance Test; TM or TMT = Trailmaking Test; WCST = Wisconsin Card Sorting Test; LAC = Lindamood Auditory Conceptualization; ROCFT = Rey-Osterreith Complex Figure Test; TTD-20 = Time-to-Do 20; TOH = Tower of Hanoi; MFFT = Matching Familiar Figures Test; Word Attack = Woodcock-Johnson Revised Word Attack subtest; TOVA = Test of Variables of Attention; Word Fl. = Word Fluency; Aud. An. Test = Auditory Analysis Test; Emb. Phonemes = Embedded Phonemes; DRL = differential reinforcements of low response rates; AAS = Auditory Analysis of Speech; EF = executive functioning; PP = phonological processing; GDS = Gordon Diagnostic System.

Though there are individual differences in cognitive dysfunction, speed of processing was consistently impaired in ADHD with dyslexia in many studies. 457 twin pairs from the Colorado Learning Disabilities Research Center (CLDRC) twin study, an ongoing study of the etiology of RD, ADHD, and related disorders was taken for a study. Phenotypic analyses compared groups with and without RD and ADHD on composite measures of six cognitive domains. Twin analyses were then used to test the etiology of the relations between the disorders and any cognitive weaknesses. Phenotypic analyses supported the hypothesis that both RD and ADHD arise from multiple cognitive deficits rather than a single primary cognitive deficit. RD was associated independently with weaknesses on measures of phoneme awareness, verbal reasoning, and working memory, whereas ADHD was independently associated with a heritable weakness in inhibitory control. RD and ADHD share a common cognitive deficit in processing speed, and twin analyses indicated that this shared weakness is primarily due to common genetic influences that increase susceptibility to both disorders.

In previous studies, children with both Attention-Deficit Hyperactivity Disorder (ADHD) and a Reading Disorder were found to have more difficulties with processing speed, working memory, and

timed as opposed to non-timed executive functioning (EF) measures when compared with those with either disorder alone.

A recent study found that older adolescents and adults with both disorders also had more difficulties on processing speed and working memory measures than individuals who only had ADHD. There were no differences among non-timed EF scores. These results add support to the premise that common underlying features may be contributing to the high co-morbidity between these disorders and associated cognitive weaknesses(78).

Children with both ADHD and SLD were found to be performing poorly in academic activities. In a study of 50 children with ADHD and SLD, clinical profile and soft neurological sign were assessed. All children had poor school performance, 15 (30%) had already experienced class retention and 20 (40%) had developed aggressive or withdrawn behavior. Children with SLD and co-occurring ADHD need to be identified at an early age to prevent poor school performance and behavioral problems(50).

SOFT NEUROLOGICAL SIGNS IN ADHD WITH COMORBID SLD

The prevalence of soft neurological signs is greater in ADHD children compared to SLD. In fact, inclusion of soft neurological signs

may improve the sensitivity and specificity of the diagnosis. Similarly soft neurological signs are greater in SLD compared to normal controls.

A study was done to evaluate the reliability and validity of soft neurological signs in ADHD. The examination of neurological soft-signs had a sensitivity of 0.80 and a specificity of 0.76 in predicting motor problems as evaluated by the physical education teacher. It was recommended for routine evaluation in ADHD children(79).

A study was done with the aim to examine whether neurological soft signs identified in children with attention deficit hyperactivity disorder (ADHD), learning disorders (LD), comorbid ADHD-LD and children with no known disorders could be grouped and whether these groups of soft signs would differentiate between the clinical groups and the non-clinical group.

A total of 148 children (114 boys, 34 girls) participated in the study, with a mean age of 8.84. The exploratory factor analysis for Neurological Examination for Subtle Signs (NESS) items revealed five factors, explaining 81.7% of the variation. Multivariate analysis of variance showed that these factors of NESS were significantly different between the clinical groups and the non-clinical group.

The discriminant functional analysis also yielded significant canonical discriminant functions, correctly classifying 85% of the clinical

and non-clinical groups of children. Certain factors of NESS such as speed of movement, dysrhythmia and overflow with timed movements, provide important information that may enhance our understanding of the neurobiological bases of ADHD and LD and the clinical implications of neurological soft signs(80).

Studies have found that certain factors of PANESS such as speed of movement, dysrhythmia and overflow with timed movements are greater in ADHD with comorbid SLD(49).

Our review of literature failed to find Indian studies on this subject. Thus, the study was planned with the aim of studying neurocognition, neurological soft signs in children with ADHD and the correlation of neurocognition, neurological soft signs to severity of ADHD and with the co-morbidity of specific learning disability.

AIM

AIM

To study the neurocognition, neurological soft signs in children with ADHD and the correlation of neurocognition, neurological soft signs to type and severity of ADHD and with the co-morbidity of specific learning disability.

OBJECTIVES

OBJECTIVES

1. To assess the neurocognition and neurological soft signs in ADHD children with and without SLD.
2. To find the correlation between neurocognition and severity of ADHD with and without SLD
3. To find the correlation between neurological soft signs and severity of ADHD with and without SLD

NULL HYPOTHESIS

1. There is no difference between the neurocognition and neurological soft signs in ADHD children with and without SLD.
2. There is no the correlation between neurocognition and type, severity of ADHD with and without SLD
3. There is no the correlation between neurological soft signs and type, severity of ADHD with and without SLD

METHODOLOGY

METHODOLOGY

Sample Selection

The study is a cross sectional observational study to be conducted in Department of Child Psychiatry, Institute of child health, Chennai. Ethical committee approval was obtained from Madras Medical College ethics committee. 40 consecutive children with diagnosis of ADHD with SLD and 40 consecutive children without SLD attending Child Psychiatry outpatient department are taken up for further evaluation. Diagnosis of ADHD and SLD is made according to MINI KID. All the children, taken up for study are examined after obtaining due consent from parent and assent from children.

METHODOLOGY

Inclusion criteria

1. Age 6-12 yrs.
2. IQ > 70 percentile

Exclusion Criteria

1. Diagnosis of Mental Retardation, Seizure Disorder and other psychiatric or physical illness with obvious neuropathology.
2. Not consenting for study

METHODOLOGY

Materials:

1. Semi Structured Proforma for socio demographic profile and risk factors
2. MINI KID
3. Ravens Coloured Progressive Matrices
4. ADHD SNAP IV
5. NIMHANS LD battery
6. PANESS (Physical & Neurological Evaluation for Soft Signs)
7. Neuropsychological Assessment
    - Executive Function – Stroop Test, MT A & B
    - Attention: category fluency, continuous performance test
    - Working Memory – Verbal N Back Test

MINI KID

The Mini-International Neuropsychiatric Interview (M.I.N.1.) is a short structured diagnostic interview developed jointly by psychiatrists and clinicians in the United States and Europe. for DSM-IV and ICD-IO psychiatric disorders. With an administration time of approximately 15 minutes it was designed to meet the need for a short but accurate structured psychiatric interview for multicenter clinical trials and epidemiology studies and to be used as a first step in outcome tracking in nonrescarch clinical settings. The MINI-KID generates reliable and valid psychiatric diagnoses for children and adolescents and does so in a third of the time as the K-SADS-PL.

SNAP IV

The SNAP-IV Rating Scale is a revision of the Swanson, Nolan and Pelham (SNAP) Questionnaire (Swanson et al, 1983). The items from the DSM-IV (1994) criteria for Attention-Deficit/Hyperactivity Disorder (ADHD) are included for the two subsets of symptoms: inattention (items #1-#9) and hyperactivity/ impulsivity (items #11-#19). Also, items are included from the DSM-IV criteria for Oppositional Defiant Disorder (items #21-#28) since it often is present in children with ADHD. Items have been added to summarize the Inattention domain (#10) and the Hyperactivity/Impulsivity domain (#20) of ADHD. Two other items

were added: an item from DSM-III-R (#29) that was not included in the DSM-IV list for ODD, and an item to summarize the ODD domain (#30).

The SNAP-IV is based on a 0 to 3 rating scale: Not at All = 0, Just A Little = 1, Quite A Bit = 2 and Very Much = 3. Subscale scores on the SNAP-IV are calculated by summing the scores on the items in the subset and dividing by the number of items in the subset. The score for any subset is expressed as the Average Rating-Per-Item.

PANESS

PANESS is a Revised Physical and Neurological Examination for soft Signs scale by Martha Denckla. It is used for physical and neurological soft signs. It can be used for children and adolescents. It is an observational scale having 21 questions covering gait, stance, laterality, quality of rapid movements, impersistence score, involuntary movement score, repetitive speed of movement score, and sequenced speed of movement score, asymmetrical movement score. It assesses in terms of laterality, timed and untimed motor movements. It has been found to have adequate test retest reliability, inter-rater reliability, and internal consistency. The PANESS is particularly useful for assessment of motor speed in children because it is brief, minimizes the need for equipment, provides lateralized data, and is applicable to children as young as 5 years.

STATISTICAL ANALYSIS

STATISTICAL ANALYSIS

1. Comparison of sociodemographic details by chi-square test and t-test.
2. Comparison of risk factors by chi-square and t-test, if appropriate.
3. Comparison of ADHD severity by t-test
4. Comparison of neurological soft signs using t-test
5. Comparison of neuropsychological functioning by t-test
6. Correlation between ADHD severity and neuropsychological performance by Pearson correlation
7. Correlation between ADHD severity and neurological soft signs by Pearson correlation
8. Comparison of two correlation factor by fisher r to z transformation

All statistical analysis was done using SPSS version 20 statistical software. Level of significance was kept at $p<0.05$ and highly significant if $p<0.01$

RESULTS

RESULTS

The present study compares the risk factors, soft neurological signs and neuropsychological functioning among two groups of ADHD children. One group is with comorbid SLD and other group is without SLD.

SOCIODEMOGRAPHIC DETAILS

Table 1

	ADHD without SLD			ADHD with SLD			p-value
	n	Mean	SD	n	Mean	SD	
Child Age	40	8.5	1.76	40	8.65	1.45	0.678
IQ	40	85.25	5.18	40	83.25	6.74	0.141
Father Age	40	34.5	3.21	40	35.7	4.56	0.196
Mother Age	40	30.6	2.76	40	31.45	1.92	0.114

Table 1 shows sociodemographic profile of the study group.

The mean age of the groups are around 8 years and their mean IQ is above 80 as per the study inclusion criteria. There is no significant difference between the groups in age and IQ of the children. There is no significant difference between the mean ages of their parents.

Table 2

		ADHD without SLD		ADHD with SLD		Chi Square test
		n	%	n	%	
Sex	Male	28	70	25	62.5	0.478
	Female	12	30	15	37.5	
Socioeconomic Status	Low	13	32.5	16	40	0.485
	Middle	27	67.5	24	60	
Religion	Hindu	36	90	38	95	0.36
	Christian	2	5	1	2.5	
	Muslim	2	5	1	2.5	
Mother Tongue	Tamil	37	92.5	38	95	0.644
	Telugu	2	5	2	5	
	Urdu	1	2.5	0	0	

Table 2 shows the sociodemographic profile of the two groups.

Though the males outnumber the females, there is no significant difference between the groups. The upper middle and lower middle socioeconomic status of the children are grouped together as middle socioeconomic status. The upper lower and lower socioeconomic status of the children are grouped together as low socioeconomic status. There is no significant difference between the groups in socio economic status, religion and mother tongue.

Table 3

	ADHD without SLD		ADHD with SLD	
	n	Median	n	Median
Birth Order	40	2	40	2
Child Education Level	40	3	40	3

Table 3 shows the basic profile of the children in terms of birth order and child education level.

The median birth order of both the groups belongs to 2nd birth order. The median child education level in terms class or standard in school of both the groups belongs to 3rd standard. All of them are studying in normal school.

Medication status

All the children included in the study are either drug naïve or under treatment. If under treatment, they are all on tablet atomoxetine 10mg or less per day. On the day of interview and assessment, they are asked to skip the drug. This is done to reduce the effect of drug on assessment.

Table 4

SLD	ADHD WITH SLD	
	n	%
Reading	22	55
Writing	3	7.5
Arithmetic	4	10
Mixed	11	27.5

Table 4 shows the specific learning disability profile the ADHD with SLD group.

Specific learning disability was identified by NIMHANS learning disability battery and the predominant deficit was categorized as above.

Nearly 55 percent of the group comprised of reading disorder. 27.5 percent of children were suffering from mixed or nos type. 7.5 percent of the children were suffering from writing type and 10 percent were suffering from arithmetic type.

Table 5

Risk Factors	ADHD without SLD		ADHD with SLD		Chi Square Test
	n	%	n	%	
Genetic					
Family H/O SLD	2	5	3	7.5	0.644
Family H/O ADHD	1	2.5	4	10	0.166
Prenatal					
Maternal Smoking	0	0	0	0	NA
Maternal Alcohol	0	0	0	0	NA
Maternal cocaine	0	0	0	0	NA
Perinatal					
Nature of Delivery					
Normal	32	80	28	70	0.301
LSCS/OTHER	8	20	12	30	
Preterm Delivery	10	25	11	27.5	0.799
Low birth weight	3	7.5	4	10	0.692
Post Natal					
Developmental delay	3	7.5	4	10	0.692

Table 5 shows the risk factors for ADHD and SLD.

There is no significant difference between the groups.

Table 6

	ADHD WITHOUT SLD		ADHD WITH SLD		p-Value
	MEAN	SD	MEAN	SD	
Inattention	2.11	0.42	2.56	0.47	0.0001
Hyperactivity/impulsivity	2.67	0.31	2.78	0.35	0.1408

Table 6 shows the mean SNAP IV ADHD severity rating scale score.

The mean ADHD inattention score for ADHD with SLD is slightly higher than the ADHD without SLD group and the difference of mean is found to significant.

The mean ADHD Hyperactivity/Impulsivity score for ADHD with SLD group is higher than the ADHD without SLD group. But the difference of mean between the groups is not found to be significant.

ADHD with comorbid SLD is found to be significantly more inattentive than pure ADHD alone.

Table 7

Stroop Test	ADHD WITHOUT SLD		ADHD WITH SLD		p-Value
	MEAN	SD	MEAN	SD	
Reading	174.35	26.15	276.45	77.65	0.001
Naming color	422.25	84.55	545.65	98.55	0.001
Stroop Effect Score	247.9	58.4	269.2	20.9	0.001

Table 7 shows the neuropsychological performance of the two groups in Stroop Test.

The mean duration taken for reading the words and naming the color of the word in stroop card is greater in ADHD with SLD group. The difference between the two groups is found to be significant.

Stroop effect score, the difference of duration in reading and naming is higher in ADHD with SLD group. The difference of mean between groups is also found to be significant.

Table 8

Trail Making Test	ADHD WITHOUT SLD		ADHD WITH SLD		p-Value
	MEAN	SD	MEAN	SD	
PART A	63.02	4.85	68.12	9.54	0.003
PART B	92.35	7.43	163.65	23.12	0.001

Table 8 shows the neuropsychological performance of the two groups in Trail making Test

The mean duration for Part A of Trail Making test in ADHD with SLD group is higher than the ADHD without SLD group. The difference between the mean is also found to be significant.

The mean duration for Part B of Trail Making test in ADHD with SLD group is higher than the ADHD without SLD group. The difference between the mean is also found to be significant.

Table 9

Category fluency	ADHD WITHOUT SLD		ADHD WITH SLD		p-Value
	MEAN	SD	MEAN	SD	
Animal naming	10.05	1.89	9.45	2.05	0.177

Table 9 shows the neuropsychological performance of the two groups in category fluency test.

Animal naming is used for category fluency. The mean number of words said by ADHD without SLD is higher than the ADHD with SLD. But there is no statistical difference between the groups.

Table 10

N-back test	ADHD WITHOUT SLD		ADHD WITH SLD		p-Value
	MEAN	SD	MEAN	SD	
1-back hits	8.12	2.2	7.25	1.98	0.067
1-back errors	1.08	0.24	2.08	0.42	0.001
2-back hits	4.18	1.25	3.08	1.42	0.001
2-back errors	5.18	1.89	6.33	2.34	0.018

Table 10 shows the neuropsychological performance of the two groups in Verbal N-Back test.

In both 1 Back and 2 Back verbal testing, the mean scores are greater for ADHD without SLD group. The difference of mean is found to be significant in all except 1-Back hits. In both tests, children committed commission errors only. There were no omission errors. Omission errors were more for ADHD with SLD group and it is found to be significant.

Table 11

CPT	ADHD WITHOUT SLD		ADHD WITH SLD		p-Value
	MEAN	SD	MEAN	SD	
Time taken	426.13	30.76	440.13	50.22	0.137
Errors	30.3	5.34	52.35	9.45	0.001

Table 11 shows the neuropsychological performance of the two groups in Continuous Performance test.

Though the mean time taken for the test is slightly higher in ADHD with SLD group, there is no significant difference between the groups.

The mean number of errors is slightly greater in ADHD with SLD group and the difference is found to be significant. The errors committed by children in both the groups are of omission type only.

Table 12

Untimed movements	ADHD WITHOUT SLD		ADHD WITH SLD		p-Value
Gait & stations	MEAN	SD	MEAN	SD	
Right axial	1.08	0.41	1.15	0.32	0.397
Left axial	1.03	0.32	1.18	0.41	0.072
Total axial	2.11	0.39	2.33	0.38	0.187

Table 12 shows the untimed movements in gaits and station scores of soft neurological signs in PANESS scale.

There is slight higher preponderance of soft neurological signs in ADHD with SLD group. But the difference is not found to be statistically significant.

Table 13

Untimed movements	ADHD WITHOUT SLD		ADHD WITH SLD		p-Value
Overflow gaits	MEAN	SD	MEAN	SD	
Right overflow	1.13	0.52	1.15	0.41	0.849
Left overflow	1.05	0.22	1.23	0.56	0.622
Total overflow	2.18	0.35	2.38	0.46	0.306

Table 13 shows the scores of soft neurological signs in PANESS scale

This table shows the untimed overflow gait scores in heel, toes and sides.

The mean scores for both sides are greater in ADHD with SLD. The difference of mean between the groups is not statistically significant.

Table 14

Untimed movements	ADHD WITHOUT SLD		ADHD WITH SLD		p-Value
Misc. & involuntary	MEAN	SD	MEAN	SD	
Right	0	0	0	0	0
Left	0	0	0.08	0.27	0.607
Total	0	0	0.08	0.27	0.607

Table 14 shows the untimed miscellaneous and involuntary movement scores of soft neurological signs in PANESS scale.

There is no significant difference between the groups.

Table 15

Untimed movements	ADHD WITHOUT SLD		ADHD WITH SLD		p-Value
	MEAN	SD	MEAN	SD	
Total gait and stations	4.29	0.36	4.79	0.41	0.214

Table 15 shows the total gait and stations in untimed movements.

Though the mean scores of soft neurological signs in untimed category is greater in ADHD with SLD group, there is no statistically significant difference between the groups.

Table 16

Timed movements	ADHD WITHOUT SLD		ADHD WITH SLD		p-Value
Overflow	MEAN	SD	MEAN	SD	
Right	0.13	0.33	2.08	0.26	0.001
Left	1.06	0.73	2.18	0.42	0.001
Total	1.19	0.50	4.26	0.32	0.001

Table 16 shows the timed overflow movements in PANESS scale.

The mean score for ADHD with SLD is greater than the ADHD without SLD and the difference between the groups is statistically significant.

Table 17

Timed movements	ADHD WITHOUT SLD		ADHD WITH SLD		p-Value
Dysrhythmia	MEAN	SD	MEAN	SD	
Right	1.05	0.32	2.13	0.42	0.002
Left	2.1	0.52	3.18	0.86	0.001
Total	3.15	0.4	5.31	0.64	0.001

Table 17 shows the timed dysrhythmia scores in PANESS scale.

The mean score for ADHD with SLD is greater than the ADHD without SLD and the difference between the groups is statistically significant.

Table 18

Timed movements	ADHD WITHOUT SLD		ADHD WITH SLD		p-Value
Misc.	MEAN	SD	MEAN	SD	
Right	0	0	0	0	-
Left	0	0	0.15	0.43	0.164
Total	0	0	0.15	0.43	0.164

Table 18 shows the timed miscellaneous scores in PANESS scale.

The mean score for ADHD with SLD is greater than the ADHD without SLD but the difference between the groups is not statistically significant.

Table 19

Timed movements	ADHD WITHOUT SLD		ADHD WITH SLD		p-Value
SFA scores	MEAN	SD	MEAN	SD	
Right	1.15	0.21	1.3	0.32	0.015
Left	3.1	0.32	3.98	0.22	0.001
Total	4.25	0.25	5.28	0.26	0.001

Table 19 shows the timed SFS scores in PANESS scale.

The mean score for ADHD with SLD is greater than the ADHD without SLD and the difference between the groups is statistically significant.

Table 20

PANESS	ADHD WITHOUT SLD		ADHD WITH SLD		p-Value
	MEAN	SD	MEAN	SD	
Total gaits & stations	4.29	0.44	4.79	0.65	0.001
Total right overflow	1.26	0.40	3.31	0.32	0.001
Total left overflow	2.11	0.45	3.41	0.49	0.001
Total overflow	3.37	0.41	6.72	0.39	0.001
Total timed	8.59	0.39	15.00	0.55	0.001
Total PANESS	12.88	0.43	19.79	0.63	0.001

Table 20 shows the final scores of PANESS scale.

The mean score for ADHD with SLD is greater than the ADHD without SLD and the difference between the groups is statistically significant.

Table 21

		INATTENTION		r to z transformation
Test	Measure	ADHD WITHOUT SLD	ADHD WITH SLD	
		r	r	p value
Stroop test	Effect score	0.34	0.45	0.576
TMT	Part a	0.41	0.44	0.875
	Part b	0.48	0.62	0.384
Category fluency	Animal naming	-0.2	-0.21	0.968
N back test	1-hits	-0.07	-0.11	0.865
	1-error	0.32	0.44	0.549
	2-hits	-0.12	-0.17	0.826
	2-errors	0.33	0.42	0.653
CPT	Time	0.22	0.21	0.96
	Errors	0.52	0.67	0.316

Table 21 shows the correlation between the inattention severity in both the groups and their neuropsychological performance.

There is a positive correlation between inattention scores and stroop test, TMT, CPT and N-Back test error scores. There is a negative correlation between the inattention scores and category fluency and N-Back test hit scores.

The scores imply that severe the inattention component, poorer the performance in neuropsychological testing.

The trend is similar in both the groups with slightly greater coefficient ratio in ADHD with SLD group. But the difference in correlation coefficient is not statistically significant.

The correlation coefficient value in each group is also not high enough to mention for clinical significance, except for TMT and Stroop effect score.

Table 22

		HYPERACTIVITY/IMPULSIVITY		r to z transformation
TEST	MEASURE	ADHD WITHOUT SLD	ADHD WITH SLD	
		r	r	p value
Stroop test	Effect score	0.22	0.27	0.818
TMT	Part A	0.37	0.32	0.811
	Part B	0.36	0.35	0.961
Category fluency	Animal naming	-0.08	-0.16	0.726
N back test	1-hits	-0.11	-0.12	0.968
	1-error	0.27	0.37	0.631
	2-hits	-0.09	-0.14	0.826
	2-errors	0.27	0.41	0.497
CPT	Time	0.2	0.22	0.928
	Errors	0.47	0.51	0.818

Table 22 shows the correlation between the hyperactivity/impulsivity (HY/IMP) severity in both the groups and their neuropsychological performance.

There is a positive correlation between hyperactivity/impulsivity scores and stroop test, TMT, CPT and N-Back test error scores. There is a negative correlation between the inattention scores and category fluency and N-Back test hit scores.

The scores imply that severe the hyperactivity/impulsivity component, poorer the performance in neuropsychological testing.

The trend is similar in both the groups with slightly greater coefficient ratio in ADHD with SLD group. But the difference in correlation coefficient is not statistically significant.

The correlation coefficient value in each group is also not high enough to mention for clinical significance, except for TMT and CPT error score.

Table 23

PANESS	INATTENTION		p value
	ADHD WITHOUT SLD	ADHD WITH SLD	
	r	r	
Total gaits & stations	0.42	0.38	0.898
Total right overflow	0.34	0.35	0.967
Total left overflow	0.42	0.38	0.823
Total overflow	0.36	0.35	0.985
Total timed	0.54	0.46	0.765
Total PANESS	0.36	0.32	0.824

Table 23 shows the correlation between inattention scores and PANESS scores.

There is a positive correlation between the inattention severity and PANESS scores. But there is no statistical significant difference between the groups.

Table 24

PANESS	HYPERACTIVITY/IMPULSIVITY		p value
	ADHD WITHOUT SLD	ADHD WITH SLD	
	r	r	
Total gaits & stations	0.38	0.35	0.798
Total right overflow	0.36	0.40	0.767
Total left overflow	0.42	0.39	0.812
Total overflow	0.40	0.39	0.985
Total timed	0.52	0.49	0.765
Total PANESS	0.34	0.36	0.824

Table 24 shows the correlation between inattention scores and PANESS scores.

There is a positive correlation between the hyperactivity/impulsivity severity and PANESS scores. But there is no statistical significant difference between the groups.

DISCUSSION

DISCUSSION

STUDY DESIGN

The study was a cross sectional observational study and the evaluation was done after giving an appointment of their convenient time. It helped in keeping all the subjects included in the study for detailed assessment. Children cooperated well for assessment in single session.

SOCIODEMOGRAPHIC DETAILS

The sociodemographic details of the study group show no significant difference between the groups. The mean age of children is around 8 years and most of them were studying in 3rd standard in both the groups. This shows the common age group and standard where ADHD is being identified or being treated. The IQ of all children in the study is around 84. This is in accordance with inclusion criteria, where child with IQ<70 is not included. Though boys are more than the girls in both groups, the groups are comparable. The prevalence of ADHD in boys is greater than females and accordingly the boys are more in our study(81).

The family`s socioeconomic status and religion had no difference between the groups. Mother tongue also showed no difference. Only one child`s mother tongue was Urdu and she was comfortable in Tamil and English.

SPECIFIC LEARNING DISABILITY

ADHD children with specific learning disability were analyzed using NIMHANS learning disability battery and grouped based on their major disability.

Majority of the children belonged to reading disorder. Followed by mixed or learning disorder nos type, arithmetic and written expression. The prevalence is similar to other studies in ADHD with comorbid SLD(51).

RISK FACTORS

There are numerous factors proposed as risk or causative factor for ADHD and SLD. We have taken few risk factors for analysis in this study. Most of the data are historical and few have recorded evidence. The factors have been grouped as genetic and environmental factors (Prenatal, Perinatal and Postnatal). Prematurity and Low birth weight are associated with ADHD and SLD in numerous studies(22,82).

In our study, though few cases have risk factors for ADHD and SLD, the difference between the groups is not significant. Some of the factors seem to be irrelevant in Indian population. No cases are found to have history of smoking and alcohol abuse in mother. Similarly lead exposure and diet theory of ADHD cannot be evaluated.

Family history of ADHD or SLD is found to be positive in 10 out of 80 children in total. All the positive family history children had ADHD or SLD in either father or mother. History is inconclusive for other relatives of proband. So they were neglected.

ADHD SEVERITY

The results are similar to previous studies(12,83,84). There is statistically significant difference between the groups in inattention category. Inattention score for ADHD with SLD is greater than the ADHD without SLD. SLD without ADHD are found to have inattention, though not up to the level of classifying it as ADHD. So ADHD with SLD are found to have more inattention. The hyperactivity scores show no difference between the groups.

NEUROPSYCHOLOGICAL PERFORMANCE

STROOP TEST

Stroop test measures attention and set shifting process. The inattention scores for ADHD with SLD are significantly greater than ADHD without SLD. Hence in stroop test, ADHD with SLD children performed poorly and there is significant difference between the groups. Stroop Test has been extensively studied in ADHD alone and along with

SLD. It was found that ADHD with SLD performed poorly than ADHD alone(41,81,85). Our study confirms the previously stated findings.

TRAIL MAKING TEST

Trail Making Test part A measures the attention span, speed of processing, motor control. Part B measures the set shifting ability along with the above functions. Few studies have measured executive function by subtracting Part b score from Part A.

Here there is no difference between groups in Part A of TMT. But there is a significant difference between the groups in Part B. Part B requires frequent shift in norm and attention. The results are similar to previous studies(86).

CATEGORY FLUENCY

Category fluency measures the verbal fluency. Here animal naming category is used to assess the verbal fluency. Children with ADHD had a leeway in applying relevant lexical or executive strategies related to difficulties in strategy using. The reduced efficiency of children with ADHD in semantic fluency task is based on suboptimal shifting between word clusters and is related to the lack of ability of producing new clusters of items(65,71,87). But in our study, no difference was found between the groups.

VERBAL N BACK TEST

Verbal N Back test measures short term memory / working memory. Verbal working memory is impaired in specific learning disability, especially for serial working memory. Compounded by inattention, verbal memory is impaired in ADHD with SLD. There is significant difference between the groups in all categories, except 1-Back Hit. Similar to other studies, ADHD with SLD performed poorly (78,88,89).

CONTINUOUS PERFORMANCE TEST

Continuous performance test measures the attention, impulsivity / response inhibition and speed of processing. ADHD with SLD made significant omission errors. There is no difference between the groups for total time taken. Our finding is similar to previous studies done before(42,70,90,91).

CORRELATION BETWEEN ADHD SEVERITY AND NEUROPSYCHOLOGICAL PERFORMANCE

Deficits in working memory and inhibitory control arising from dysfunction in the prefrontal cortex are stated in many studies(67,78,88,92). Attention-deficit/hyperactivity disorder has been associated with a prominent disturbance of executive functions. There is

no pathognomic neuropsychological profile for the disorder, however. Nonetheless, results of neuropsychological testing, in concert with other clinical information, provide a more comprehensive and detailed picture of the individual patient's cognitive and emotional strengths and weaknesses than a psychiatric diagnostic interview alone. This approach to the evaluation of ADHD therefore can provide a strong objective basis from which to make patient-specific recommendations for compensatory strategies and treatment.

It should be noted, however, that although executive dysfunction in the form of impaired response inhibition remains the most prominent cognitive theory of ADHD, other theories have been put forth that also deserve further investigation. These include a disturbance in delay aversion (referring to intolerance for waiting) and impaired temporal processing, among others.

The neural substrates of executive dysfunction in ADHD have begun to be revealed by a growing body of structural and functional neuroimaging research(11,83,93,94). Although still in its infancy, neuroimaging of ADHD is pointing toward disruption of FSTC circuitry and the cerebellum as being central to the cognitive and motor abnormalities seen in the disorder. Further research using cognitive tasks

assessing executive functions in combination with functional imaging techniques will provide further insight into the etiology of the disorder.

It is expected that advances in structural and functional neuroimaging will yield valuable information that will facilitate the differential diagnosis of ADHD. Evidence cited suggests that psychostimulant medication can improve executive functions and their underlying FSTC circuitry(5,32,35,95).

Furthermore, a recent study of adults with ADHD found significant improvements in organization skills and other symptoms of ADHD following cognitive remediation targeting several executive and emotional aspects of the disorder(11). Additional studies investigating the effects of treatment on executive dysfunction and brain integrity in ADHD will be necessary to determine the degree to which the structural and functional brain abnormalities observed are mutable.

Finally, because the myriad cognitive, behavioral and emotional symptoms in ADHD likely reflect the interplay of multiple cognitive and psychosocial factors, development of treatments for ADHD likely will require a multi-modal approach.

Studies have mentioned the strong correlation between the severity of disease with neurocognition by direct correlation and studies have

mentioned improvement in cognitive domains after reduction of symptoms with treatment(32,42).

NEUROPSYCHOLOGICAL PERFORMANCE OF ADHD WITH SLD

Children with both Attention-Deficit Hyperactivity Disorder (ADHD) and SLD were found to have more difficulties with attention span, processing speed and working memory(67,88,92). In our study, though the correlation coefficient factor supports the above statement, there is no significant statistical difference between ADHD with SLD and ADHD without SLD.

NEUROLOGICAL SOFT SIGNS

NSS in young adults have been associated with a number of neuropsychiatric and behavioral disorders, such as psychosis, obsessive-compulsive disorder, and also in conditions of atypical development, like autism and learning disability and ADHD(8).

NSS are known to be able to discriminate between abnormal and normal group of children. Children with ADHD had significant overflow movements and motor incoordination when compared to SLD alone. Children with problems such as ADHD, SLD or academic under achievement had soft neurological signs at varying severity and type.

ADHD children had more overflow movements. This reflects cortical immaturity. Timed overflow, speed of movement and dysrhythmia are significant factors in differentiating ADHD with SLD from ADHD without SLD(96).

Our study shows significant difference between the groups in timed overflow and dysrhythmia scores. The final component outcome scores of PANESS scale also shows significant difference between the groups.

CORRELATION BETWEEN ADHD SEVERITY AND NEUROLOGICAL SOFT SIGNS

Few studies have found correlation to be positive between ADHD severity and soft neurological signs(96). Our study also finds it to be positive, though not significant clinically. There is no significant difference between the groups, when correlation factors are compared.

CONCLUSION

CONCLUSION

1. Inattention is significantly higher in ADHD with SLD than ADHD without SLD as measured by neurocognitive assessment.
2. Timed Overflow and timed Dysrhythmia are significantly higher in ADHD with SLD than ADHD without SLD
3. Soft neurological signs are significantly higher in ADHD with SLD than ADHD without SLD.
4. SLD and ADHD sharing common neural substrates with more brain dysfunction if SLD is a comorbidity in ADHD
5. Severe the ADHD score, poorer the cognitive performance.
6. Severe the ADHD score, more the soft neurological signs
7. Soft neurological signs and cognitive performance can predict the severity of illness.

LIMITATIONS

1. Study Design

   Inclusion of controls would have made the study more valid.

   Instead of naturalistic cross sectional design, drug naïve children and a prospective analysis with treatment would make the study more reliable.

2. Sample Selection

   ADHD with other comorbid disorders were included in the study, which might have confounded the results.

   Effect of medication would have confounded the results

3. The present study was limited with a relatively small sample size, which precluded comparisons according to sex and age of children.
4. The normative data for the PANESS was not of Indian children and could not be used for analysis.

FUTURE DIRECTIONS

Further research with large sample size and avoiding the said limitations is needed to understand the implications of soft neurological signs and cognitive performance of ADHD children in clinical setting.

Prospective study of severity of soft neurological signs in ADHD to predict its influence on fine motor performance in future is needed.

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ANNEXURES

Proforma for Sociodemographic Details

Sociodemographic Data	
Name	
Age	
Sex	
Father Age	
Mothers Age	
Socioeconomic Status	
Child`s Education	
Religion	
Mother Tongue	
Birth order of the child	
Risk Factors	
Maternal Smoking	
Lead Exposure	
Maternal Anaemia	
Alcohol/Illicit drug abuse	
Labour Problem	
Nature of Delivery	
Delivery Complications	
Prematurity	
Birth Weight	
Milestones	

The SNAP-IV Teacher and Parent Rating Scale

James M. Swanson, Ph.D., University of California, Irvine, CA 92715

Name:_____ Gender:_____ Age:_____ Grade:_____

Ethnicity (circle one which best applies): African-American Asian Caucasian Hispanic Other_____

Completed by:_____ Type of Class:_____ Class size:_____

For each item, check the column which best describes this child:	Not At All	Just A Little	Quite A Bit	Very Much
1. Often fails to give close attention to details or makes careless mistakes in schoolwork or tasks				
2. Often has difficulty sustaining attention in tasks or play activities				
3. Often does not seem to listen when spoken to directly				
4. Often does not follow through on instructions and fails to finish schoolwork, chores, or duties				
5. Often has difficulty organizing tasks and activities				
6. Often avoids, dislikes, or reluctantly engages in tasks requiring sustained mental effort				
7. Often loses things necessary for activities (e.g., toys, school assignments, pencils, or books)				
8. Often is distracted by extraneous stimuli				
9. Often is forgetful in daily activities				
10. Often has difficulty maintaining alertness, orienting to requests, or executing directions				
11. Often fidgets with hands or feet or squirms in seat				
12. Often leaves seat in classroom or in other situations in which remaining seated is expected				
13. Often runs about or climbs excessively in situations in which it is inappropriate				
14. Often has difficulty playing or engaging in leisure activities quietly				
15. Often is "on the go" or often acts as if "driven by a motor"				
16. Often talks excessively				
17. Often blurts out answers before questions have been completed				
18. Often has difficulty awaiting turn				
19. Often interrupts or intrudes on others (e.g., butts into conversations/games)				
20. Often has difficulty sitting still, being quiet, or inhibiting impulses in the classroom or at home				
21. Often loses temper				
22. Often argues with adults				
23. Often actively defies or refuses adult requests or rules				
24. Often deliberately does things that annoy other people				
25. Often blames others for his or her mistakes or misbehavior				
26. Often touchy or easily annoyed by others				
27. Often is angry and resentful				
28. Often is spiteful or vindictive				
29. Often is quarrelsome				
30. Often is negative, defiant, disobedient, or hostile toward authority figures				
31. Often makes noises (e.g., humming or odd sounds)				
32. Often is excitable, impulsive				
33. Often cries easily				
34. Often is uncooperative				
35. Often acts "smart"				
36. Often is restless or overactive				
37. Often disturbs other children				
38. Often changes mood quickly and drastically				
39. Often easily frustrated if demand are not met immediately				
40. Often teases other children and interferes with their activities				

Check the column which best describes this child:	Not At All	Just A Little	Quite A Bit	Very Much
41. Often is aggressive to other children (e.g., picks fights or bullies)				
42. Often is destructive with property of others (e.g., vandalism)				
43. Often is deceitful (e.g., steals, lies, forges, copies the work of others, or "cons" others)				
44. Often and seriously violates rules (e.g., is truant, runs away, or completely ignores class rules)				
45. Has persistent pattern of violating the basic rights of others or major societal norms				
46. Has episodes of failure to resist aggressive impulses (to assault others or to destroy property)				
47. Has motor or verbal tics (sudden, rapid, recurrent, nonrhythmic motor or verbal activity)				
48. Has repetitive motor behavior (e.g., hand waving, body rocking, or picking at skin)				
49. Has obsessions (persistent and intrusive inappropriate ideas, thoughts, or impulses)				
50. Has compulsions (repetitive behaviors or mental acts to reduce anxiety or distress)				
51. Often is restless or seems keyed up or on edge				
52. Often is easily fatigued				
53. Often has difficulty concentrating (mind goes blank)				
54. Often is irritable				
55. Often has muscle tension				
56. Often has excessive anxiety and worry (e.g., apprehensive expectation)				
57. Often has daytime sleepiness (unintended sleeping in inappropriate situations)				
58. Often has excessive emotionality and attention-seeking behavior				
59. Often has need for undue admiration, grandiose behavior, or lack of empathy				
60. Often has instability in relationships with others, reactive mood, and impulsivity				
61 Sometimes for at least a week has inflated self esteem or grandiosity				
62. Sometimes for at least a week is more talkative than usual or seems pressured to keep talking				
63. Sometimes for at least a week has flight of ideas or says that thoughts are racing				
64. Sometimes for at least a week has elevated, expansive or euphoric mood				
65. Sometimes for at least a week is excessively involved in pleasurable but risky activities				
66. Sometimes for at least 2 weeks has depressed mood (sad, hopeless, discouraged)				
67. Sometimes for at least 2 weeks has irritable or cranky mood (not just when frustrated)				
68. Sometimes for at least 2 weeks has markedly diminished interest or pleasure in most activities				
69. Sometimes for at least 2 weeks has psychomotor agitation (even more active than usual)				
70. Sometimes for at least 2 weeks has psychomotor retardation (slowed down in most activities)				
71. Sometimes for at least 2 weeks is fatigued or has loss of energy				
72. Sometimes for at least 2 weeks has feelings of worthlessness or excessive, inappropriate guilt				
73. Sometimes for at least 2 weeks has diminished ability to think or concentrate				
74. Chronic low self-esteem most of the time for at least a year				
75. Chronic poor concentration or difficulty making decisions most of the time for at least a year				
76. Chronic feelings of hopelessness most of the time for at least a year				
77. Currently is hypervigilant (overly watchful or alert) or has exaggerated startle response				
78. Currently is irritable, has anger outbursts, or has difficulty concentrating				
79. Currently has an emotional (e.g., nervous, worried, hopeless, tearful) response to stress				
80. Currently has a behavioral (e.g., fighting, vandalism, truancy) response to stress				
81. Has difficulty getting started on classroom assignments				
82. Has difficulty staying on task for an entire classroom period				
83. Has problems in completion of work on classroom assignments				
84. Has problems in accuracy or neatness of written work in the classroom				
85. Has difficulty attending to a group classroom activity or discussion				
86. Has difficulty making transitions to the next topic or classroom period				
87. Has problems in interactions with peers in the classroom				
88. Has problems in interactions with staff (teacher or aide)				
89. Has difficulty remaining quiet according to classroom rules				
90. Has difficulty staying seated according to classroom rules				

PANESS CODING SHEET

Name/Subject #: DoE: Age:
Gender: DoB:

Lateral Preference: EYE: R L FOOT: R L Mixed HAND: R L *Mixed

*Code as Mixed if nondominant hand is preferred for 3 or more items.

- Code PANESS scores below. Note that some movements are coded differently depending upon age.
- If "CD" is circled on the PANESS, code as a "2" if movement is expected to be WNL for age group.
- Tandems, Stand, and Tongue have unilateral scores only.

GAITS	R			L		
1. Heels	0	1	2	0	1	2
2. Toes	0	1	2	0	1	2
3. Sides (Code errors only if age ≥9 yo; if age ≤ 8, code as 0 regardless of errors)	0	1	2	0	1	2
4. Forward Tandem			0 1 2			
5. Backward Tandem (Code errors only if age ≥10 yo; if age ≤ 9, code as 0 regardless of errors)			0 1 2			

STATIONS	R			L		
6. Tandem (Code errors only if age ≥10 yo)			0 1 2			
7. Stand with Two Feet			0 1 2			
10. Stand on one foot	0	1	2	0	1	2
11. Hop (Unilateral)	0	1	2	0	1	2

Right Axial = _____ (Range 0-10) (R side #1-3, 10, 11)

Left Axial = _____ (Range 0-10) (L side #1-3, 10, 11)

Total Axial = _____ (Range 0-28) (R + L + 4,5, 6, 7)

OVERFLOW GAITS	R		L		
1. Heels (Code errors only if age ≥6 yo)	0	1	0	1	
2. Toes (Code errors only if age ≥6 yo)	0	1	0	1	
3. Sides (Code errors only if age ≥9 yo)	0	1	0	1	

***Right Overflow** = _____ (Range 0-3)

***Left Overflow** = _____ (Range 0-3)

***Total Overflow** = _____ (Range 0-6)

Note: Age Appropriate PANESS Score:** ***Only sum scores here if abnormal for age – Do not include score here if normal for the child's age group.

INVOLUNTARY MOVEMENTS	R			L		
7. Choreiform (Abnormal arm/finger movements)	0	1		0	1	
8. Tremor (Finger to nose)	0	1	2	0	1	2
9. Choreiform (Reptile tongue)			0 1			

MISC. OBSERVATIONS	R		L		
Posture Hemiparetic	0	1	0	1	
Posture Dystonic	0	1	0	1	
Nystagmus	0	1	0	1	
Strabismus	0	1	0	1	

Right Misc. _____ (Range 0-7) **Left Misc.** _____ (Range 0-7)

Miscellaneous and Involuntary Totals

Right = _____ (Range 0-7) (R Invol. 7 + R Invol. 8 + R Misc.)

Left = _____ (Range 0-7) (L Invol. 7 + L Invol. 8 + L Misc.)

Total = _____ (Range 0-15) (R Misc. & Invol. + L Misc.& Invol + Invol. 9)

Total Gaits and Stations = _____ (0-48) (Total Axial + *Total Overflow + Total Miscellaneous & Involuntary) (0-28) (0-6) (0-15)

Revised 09/04/2008

PANESS Timed Movements

OVERFLOW – TIMED MOVEMENTS	**R**	**PANESS Score**	**L**	**PANESS Score**
Foot Tap (FT)	0 1 2	_____	0 1 2	_____
Heel/toe tap (HT)	0 1 2	_____	0 1 2	_____
Hand Pat (HP)	0 1 2	_____	0 1 2	_____
Hand Pronate/Supinate (HPS) (For Mirror, Code errors only if age ≥9 yo)	0 1 2	*_____	0 1 2	*_____
Finger Tap (FR)	0 1 2	_____	0 1 2	_____
Finger Apposition (FS) (For Mirror, Code errors only if age ≥13 yo)	0 1 2	*_____	0 1 2	*_____
	***Timed Right Overflow**	_____ (Range 0-12)	***Timed Left Overflow**	_____ (Range 0-12)

Under **R** and **L**, transfer scores directly from PANESS. Code as a
Score of 0 if no overflow is present regardless of age appropriateness.
Score of 1 if only Proximal **or** Oro-Facial **or** Mirror are present *or* if both Proximal ***AND*** Oro-facial
Score of 2 if Both Proximal ***AND*** Mirror *or* if both Oro-facial ***AND*** Mirror *or* Proximal ***AND*** Oro-facial ***AND*** Mirror

Under **PANESS Score**, directly transfer scores from answers recorded under Right and Left, except when asterisk is present. If *, copy the score to the **PANESS Score** only if score is abnormal for age; otherwise, if age appropriate, change score to 0 in this column.

Tongue (jaw synkinesis) 0 1

***Total Timed Overflow** _____ (Range 0-25) (Sum R Overflow + L Overflow + Tongue)

DYSRHYTHMIA – TIMED MOVEMENTS	**R**	**L**
Foot Tap (FT)	0 1	0 1
Heel/toe tap (HT)	0 1	0 1
Hand Pat (HP)	0 1	0 1
Hand Pronate/Supinate (HPS)	0 1	0 1
Finger Tap (FR)	0 1	0 1
Finger Apposition (FS)	0 1	0 1
Right Dysrhythmia	_____ (Range 0-6)	**Left Dysrhythmia** _____ (Range 0-6)
Tongue	0 1	

Total Dysrhythmia _____ (Range 0-13) (Sum R Dysrhythmia + L Dysrhythmia + Tongue)

MISC. TIMED OBSERVATIONS	**R**	**L**
Choreoathetoid (Extended arm/elbow turned outward)	0 1	0 1
Hemiparetic (Flexed elbow)	0 1	0 1
Other (_____)	0 1	0 1
Right Timed Misc.	_____ (Range 0-3)	**Left Timed Misc.** _____ (Range 0-3)

Total Timed Misc. _____ (Range 0-6)

Revised 09/04/2008

TIMED MOVEMENTS (SFA Scores)

	Right					Left				
	Seconds	z-Score	SFA Score			Seconds	Z-Score	SFA Score		
FT	_____	_____	0	1	2	_____	_____	0	1	2
HT	_____	_____	0	1	2	_____	_____	0	1	2
HP	_____	_____	0	1	2	_____	_____	0	1	2
HPS	_____	_____	0	1	2	_____	_____	0	1	2
FR	_____	_____	0	1	2	_____	_____	0	1	2
FS	_____	_____	0	1	2	_____	_____	0	1	2

Right SFA _____ (Range 0-12) **Left SFA** _____ (Range 0-12)

Under **Seconds**, copy times in seconds to decimal points for each Right and Left sided movement.
Under **z-Score**, calculate z-values using Mean and Standard Deviation from the PANESS Timed Motor Movements Norms.
Normative data are stratified by child's **age**, **gender**, and **handedness**.
Use right-handed norms for left-handed children 11 years or older.
Calculate reverse scored z-values [(Normative score - Child's Score) / Standard Deviation (SD)] so that positive scores indicate better performance.
To determine **SFA score**:
If **z-score** is greater than -1 SD below the mean (i.e., Child is WNL or Child is more than 1 SD above the mean, thus faster), SFA = 0
If **z-score** is between -1 SD and -2 SD below the mean, SFA = 1
If **z-score** is less than -2 SD below the mean (i.e., indicating very poor performance), SFA = 2

Tongue (jaw synkinesis) _____ 0 2

Circle 0 or 2 for the tongue. If the child is:
For children age 5-9, the mean is **< 6** seconds:
If Time is ≤ 6 seconds, score as 0. If time > 6 seconds, score as 2.

For children Age 10 and above, the mean is <3 seconds:
If Time is ≤ 3 seconds, score as 0. If time > 3 seconds, score as 2.

***Total SFA** _____ **(Range 0-26)** (Sum R SFA + L SFA + Tongue SFA)

TOTALS

Total Right Overflow _____ (Range 0-15) (*Right Overflow [pg 1] + *Timed Right Overflow [pg 2]) (Range 0-3) (Range 0-12)

Total Left Overflow _____ (Range 0-15) (*Left Overflow [pg 1] + *Timed Left Overflow [pg 2]) (Range 0-3) (Range 0-12)

Total Overflow _____ (Range 0-31) (*Total Overflow [pg 1] + * Total Timed Overflow [pg 2]) (Range 0-6) (Range 0-25)

Total Gaits & Stations _____ (Range 0-49) (Total Axial [pg 1] + *Total Overflow [pg 1] + Total Miscellaneous & Involuntary [pg 1]) (0-28) (0-6) (0-15)

Total Timed _____ (Range 0-70) (Total Timed Overflow [pg 2] + Total Dysrhythmia [pg 2] + Total Timed Misc. [pg 2] + Total SFA [pg 3]) (Range 0-25) (Range 0-13) (Range 0-15) (Range 0-26)

Total PANESS _____ (Range 0-119) (Total Gaits and Stations + Total Timed) (Range 0-49) (Range 0-70)

* Indicate totals in which only abnormal scores for age group are included.

Revised 09/04/2008

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI-3

EC Reg No.ECR/270/Inst./TN/2013
Telephone No. 044 25305301
Fax : 011 25363970

CERTIFICATE OF APPROVAL

To
Dr. J. Komal,
Postgraduate M.D. (Psychiatry),
Madras Medical College,
Chennai – 600 003.

Dear Dr. J.Komal,

The Institutional Ethics Committee has considered your request and approved your study titled **"Neurocognition and neurological soft signs in children with attention deficit hyperactivity disorder." No.23082014.**

The following members of Ethics Committee were present in the meeting held on 05.08.2014 conducted at Madras Medical College, Chennai-3.

1.	Dr.C.Rajendran, M.D.,	: Chairperson
2.	Dr.R.Vimala, M.D., Dean, MMC, Ch-3	: Deputy Chairperson
3.	Prof.B.Kalaiselvi, M.D., Vice-Principal, MMC, Ch-3	: Member Secretary
4.	Prof.R.Nandhini, M.D., Inst.of Pharmacology, MMC	: Member
5.	Dr.G.Muralidharan, Director Incharge, Inst.of Surgery	: Member
6.	Prof.K.Ramadevi, Director i/c, Inst.of Biochemistry, MMC	: Member
7.	Prof.Saraswathy, M.D., Director, Pathology, MMC, Ch-3	: Member
8.	Prof.Tito, M.D., Director i/c, Inst.of Internal Medicine, MMC	: Member
9.	Thiru S.Rameshkumar, Administrative Officer	: Lay Person
10.	Thiru S.Govindasamy, B.A., B.L.,	: Lawyer
11.	Tmt.Arnold Saulina, M.A., MSW.,	: Social Scientist

We approve the proposal to be conducted in its presented form.

The Institutional Ethics Committee expects to be informed about the progress of the study and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.

28/8/14

Member Secretary, Ethics Committee

MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE
CHENNAI-600 003

Information to Parent/Guardian

Title: "Neurocognition and neurological soft signs in children with attention deficit hyperactivity disorder"

Principal Investigator: Dr.J.KOMAL
Third Year, MD Psychiatry Post Graduate,
Madras Medical College

Co-Investigator(if any):

Name of Participant:

Site : Institute of Child Health, Dept of Child Psychiatry, MMC, Chennai

You are invited to take part in this research. The information in this document is meant to help you decide whether or not to take part. Please feel free to ask if you have any queries or concerns.

PURPOSE OF STUDY:

ADHD is the most common childhood behavioural disorder in children (5.2% of school-age population globally). It is a neurodevelopment disorder with frontal lobe dysfunction, cerebellum has also been implicated. Neurological Soft Signs are non-normative performance on a neurological examination of motor and sensory functioning in the absence of a focal lesion. They are indicators of delayed development of motor inhibition.NSS have been associated with a number of disorders, such as psychosis, OCD, and also in conditions of atypical development, like autism and learning disability.

Cognitive study in young children of ADHD is relevant for several reasons. First, cognitive tests may contribute to the accuracy of the early identification of children at risk of ADHD and provide information about what difficulties exist. Second, this research yields information about the cognitive mechanisms that underlie the symptoms of ADHD.

Thus, the study was planned with the aim of studying neurocognition, neurological soft signs in children with ADHD and the correlation of neurocognition, neurological soft signs to type and severity of ADHD and with the co-morbidity of specific learning disability.

The study design

Your child will be interviewed at ICH, Department of Child Psychiatry OPD.

Study Procedures

The study involves evaluation of neurocognition, neurological soft signs in children with ADHD and study the correlation of neurocognition, neurological soft signs to type and severity of ADHD and with the co-morbidity of specific learning disability for which we will be interviewing you

and your child with various questionnaires. You will be required to spare roughly an hour for a one-time interview during your visit to opd in the hospital.

Possible benefits to you –
Your child is assessed for type and severity of ADHD, neurological soft signs and neurocognition that will help in treatment and management.

Possible benefits to other people
The results of the research may provide benefits to the society in terms of advancement of medical knowledge and/or therapeutic benefit to future patients.

Confidentiality of the information obtained from you
You have the right to confidentiality regarding the privacy of your medical information (personal details, results of physical examinations, investigations, and your medical history). By signing this document, you will be allowing the research team investigators, other study personnel and the Institutional Ethics Committee, to view your data, if required. The information from this study, if published in scientific journals or presented at scientific meetings, will not reveal your identity.

How will your decision to not participate in the study affect you?
Your decision not to participate in this research study will not affect your medical care or your relationship with the investigator or the institution. You will be taken care of and you will not lose any benefits to which you are entitled.

Can you decide to stop participating in the study once you start?
The participation in this research is purely voluntary and you have the right to withdraw from this study at any time during the course of the study without giving any reasons. However, it is advisable that you talk to the research team prior to stopping the treatment/discontinuing of procedures etc.

Signature of Investigator

Signature of Participant (parent)

Date

Date

ஆராய்ச்சி தகவல் தாள்

தலைப்பு: **கவனக்குறைபாடு மற்றும் அதிக செயல் ஆற்றல் உள்ள குழந்தைகளிடம் மெல் நரம்பியல் அறிகுறிக ் மற்று ் முலையான செயல்பாடுகள் குறித்த ஆய ்**

ஆராய்ச்சி செயல்பாளரின் பெயர்: **மரு . ஷகிலா ஜெ**

பங்குபெறுபவரின் பெயர்:

மருத்துவ நிலைமை: **குழந்தைகள் நல மருத்துவம்**, **மனநலம்**

கவனக்குறைபாடு மற்று ் அதிக செயல் ஆற்றல் உள்ள குழந்தைகளிடம் மெல் நரம்பியல் அறிகுறிக ் மற்று ் முலையான செயல்பாடுக ் குறித்த ஆய ் நடைபெறுகிறது . நீங்கள் இந்த ஆராய்ச்சியில் பங்கேற்க வருமாறு கேட்கப்படுகிறீர்க ். முடிவுகளை அல்லது கருத்துக்களை வெளியிடும்போதோ அல்லது ஆராய்ச்சியாள் போதோ தங்களின் குழந்தையை ் பெயரையோ அல்லது அடையாளங்களையோ வெளியிடமாட்டோம் என்பதை ் தெரிவித்துக் கொள்கிறோம்.

இந்த ஆய்வின் முடிவுகள் ஆராய்ச்சியாள்போது அல்லது ஆராய்ச்சியின் முடிவ ் போது தங்களுக்கு அறிவிக்கப்படும் என்பதை ் தெரிவித்துக் கொள்கிறோம்.

இந்த ஆராய்ச்சியில் பங்கேற்ப ் தங்களுடை ் வருவதற்கு போர்ல தான் இருக்கிற ் . மேலும் நீங்கள் எந்நேரமும் இந்த ஆராய்ச்சியிலிருந்து பின்வாங்கலாம் என்பதை ் தெரிவித்துக் கொள்கிறோம்.

பங்கேற்பாள் கையொப்பம்

நாள் :_____

ஆராய்ச்சியாள் கையொப்பம்

நாள் :_____

பாதுகாவல் கையொப்பம்

நாள் :_____

Informed consent form

Title of the study – **"Neurocognition and neurological soft signs in children with attention deficit hyperactivity disorder"**

Name of the participant: _____

Name of the Principal/Co-Investigator: DR. J.KOMAL

Name of the Institution: Institute of Child Health, Dept of Child Psychiatry, MMC

Name and address of the sponsor / agency (ies), if any: _____

I,_____(parent/guardian of participant), have read the information in this form (or it has been read to me). I was free to ask any questions and they have been answered. I am over 18 years of age and, exercising my free power of choice, hereby give my consent that my child/ward be included as a participant in the study about the – Neurocognition and neurological soft signs in children with attention deficit hyperactivity disorder.

(1) I have read and understood this consent form and the information provided to me.
(2) I have had the consent document explained to me.
(3) I have been explained about the nature of the study.
(4) I have been explained about my rights and responsibilities by the investigator.
(5) I have informed the investigator of all the treatments I am taking or have taken in the past, including any native (alternative) treatments.
(6) I am aware of the fact that I can opt out of the study at any time without having to give any reason and this will not affect my future treatment in the hospital.
(7) I hereby give permission to the investigators to release the information obtained from me and my child as result of participation in this study to the regulatory authorities, Government agencies, and ethics committee. I understand that they may inspect my original records.
(8) I understand that my identity will be kept confidential if my data are publicly presented.
(9) I have had my questions answered to my satisfaction.
(10) I consent voluntarily to participate as a participant in the research study.
I am aware, that if I have any questions during this study, I should contact the investigators. By signing this consent from, I attest that the information given in this document has been clearly explained to me and understood by me. I will be given a copy of this consent document.

Name and signature / thumb impression of the parent/guardian
(Name of parent) _____(Signature)_____ Date: _____

Name and signature of impartial witness (required for illiterate patients):
(Name) _____ (Signature) _____ Date _____
Address and contact number of the impartial witness: _____

Name and signature of the Investigator or his representative obtaining consent:

(Name) _____ (Signature) _____ (Date) _____

ஆராய்ச்சி ஒப்புதல் படிவம்

ஆராய்ச்சியின் தலைப்பு - **கவனக்குறைபாடு மற்று அதிக செயல் ஆற்றல் உள்ள குழந்தைகளிடம் மன நம்பியல் அறிகுறிக மற்று முளையின செயல்பாடுகள் குறித்த ஆய்**

பங்குபெறுபவர் பெயர்:

ஆராய்ச் செய்பவ பெயர்: **மு . கோமல ஜெ**

மருத்துவ நிலையம்: **குழந்தை நல மருத்துவமனை, சென்**

_____ வரும் நா வழங்கு கொடுக்கப்பட்ட தகவல் தெளிவ படித்த புரிந்தது கொண்டே . நா 18 வயதை கடந்திருப்பதா வ்வாறு சுய நிலையுட , மற்று ப சுதந்திரத்துட இந்த ஆராய்ச்சியில் வ குழந்தைய சேர்த்துக்கொள்ள சம்மதிக்கிறே .

நா வழங்கு கொடுக்கப்பட்ட தகவல் தெளிவ படித்த புரிந்தது கொண்டே .

வழங்கு இந்த ஆராய்ச்சியால் ஒப்புதல் படிவம் வழங்கப்பட்ட .

வழங்கு இந்த ஆராய்ச்சியால் மறுக்க , வ்வாறான வழங்கப்பட்ட .

வழங்கு வ்வாறு உரிமைக்க பற்ற வழங்கப்பட்ட .

நா இதுவர வ டுத்துக்கொண்ட அனைத்த மருத்துவ முறைக தெரிவித்திருக்கிறே .

இந்த ஆராய்ச்சியால் இருந் வ குழந்தைக்க வாழ்நா பல வாய்ப்ப வ்வாறு அதன்ம வந்த பாதி ஏற்பட வ்வாறு நா புரிந்துகொண்டே .

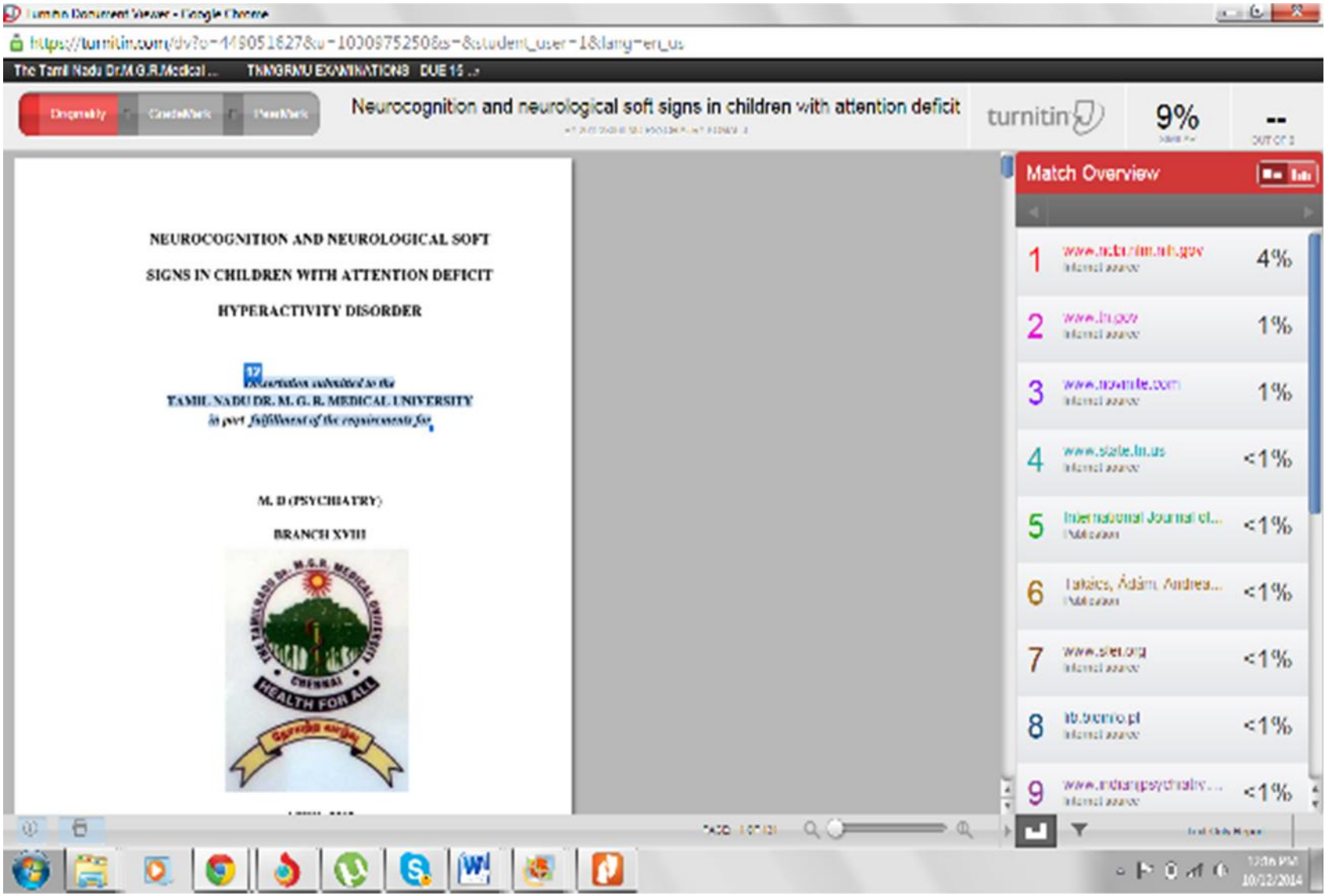
வ குழந்தைக்க பற்றான வந்த தகவல அனைத்தும் வெளியிடப்ப மாட்ட வ்வாறு நா புரிந்துகொண்டே .

வ்வாறு சுதந்திரத்துட இந்த ஆராய்ச்சியில் வ குழந்தைக்க சேர்ப்பதற்கு சம்மதிக்கிறே .

பெற்றோரின் பெயர் மற்றும் கையொப்பம்: _____ & _____ நா : _____

பாதுகாவல பெயர் மற்று கையொப்பம்: _____ & _____ நா : _____

ஆராய்ச்சியாளரி பெயர் மற்றும் கையொப்பம்: _____ & _____ நா : _____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SIGNS IN CHILDREN WITH ATTENTION DEFICIT
HYPERACTIVITY DISORDER
TAMIL NADU DR. M. G. R. MEDICAL UNIVERSITY
M. D (PSYCHIATRY)
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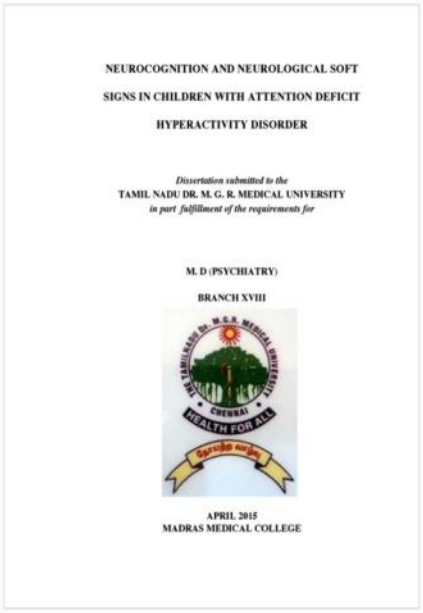

NEUROCOGNITION AND NEUROLOGICAL SOFT

SIGNS IN CHILDREN WITH ATTENTION DEFICIT

HYPERACTIVITY DISORDER

Dissertation submitted to the
TAMIL NADU DR. M. G. R. MEDICAL UNIVERSITY
in part fulfillment of the requirements for

M. D (PSYCHIATRY)

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APRIL 2015
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