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**THE EFFECT OF SLEEP LOSS ON NIGHTTIME SLEEP, DAYTIME
SLEEPINESS, AND COGNITIVE FUNCTIONING IN CHILDREN WITH
ATTENTION DEFICIT HYPERACTIVITY DISORDER**

by

Stuart R. Cantor

A dissertation submitted to the Graduate Faculty in Psychology in
partial fulfillment of the requirements for the degree of Doctor of Philosophy,
The City University of New York.

1999

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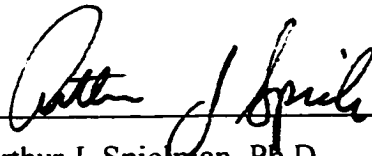
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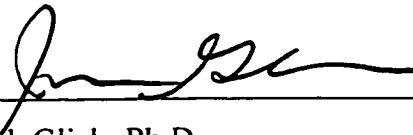
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Abstract

THE EFFECT OF SLEEP LOSS ON NIGHTTIME SLEEP, DAYTIME SLEEPINESS, AND COGNITIVE PERFORMANCE IN CHILDREN WITH ATTENTION DEFICIT HYPERACTIVITY DISORDER

by

Stuart R. Cantor

Advisor: Professor Arthur J. Spielman

The purpose of this investigation was to define groups of ADHD and Non-ADHD prepubertal children, and assess the impact of sleep loss on sleep, daytime sleepiness, and performance on a continuous performance task, the Test of Variables of Attention (T.O.V.A.TM). 20 children (11 ADHD and 9 non-ADHD) were evaluated in the CCNY Sleep Disorders Center. Participants who met inclusion criteria returned to begin a two night, two day study in which their sleep and levels of daytime alertness were electronically recorded. To partially sleep deprive participants, they went to bed two hours later on Night 2. Levels of daytime alertness/sleepiness were assessed with the Multiple Sleep Latency Test. Post sleep loss dependent variables included: 1) Night 2 of sleep, 2) Day 2 MSLTs, and 3) T.O.V.A.TM performance. One significant interaction effect was found for sleep. ADHD children had a significantly higher percentage of transitional, lighter Stage 1 sleep after sleep loss, in contrast to the non-ADHD group, who had lower Stage 1% on Night 2, a more expectable response. Parent reports on the questionnaire of children's sleep correlated somewhat with empirical recordings. Items seem to be useful in identifying children with sleep difficulties. ADHD children did not become sleepier than non-ADHD children from

mild sleep loss: there was no difference found between the groups on the MSLT. However, all participants, had statistically significantly lower Sleep Latency means on MSLT 2, suggesting that just two hours of sleep loss can have some impact on the high levels of alertness seen in most participants the first day. ADHD group T.O.V.A.TM means, before and after sleep loss, were not different. ADHD group's T.O.V.A.TM performance did not show more severe decrements than controls after the sleep loss condition. Preliminary findings showed non-ADHD children made more errors on the T.O.V.A.TM after mild sleep deprivation. Further research is indicated to follow up investigating the group differences and relationships suggested by this study between sleep loss and nocturnal sleep, decrements in diurnal alertness, and T.O.V.A.TM performance in ADHD and non-ADHD children.

Key Words: ADHD, children's sleep, sleep disorders, MSLT, sleepiness, T.O.V.A.TM

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CHAPTER ONE

INTRODUCTION

Background

Given the tremendous interest in ADHD among parents and educators, further inquiry into any possible factor which contributes to the understanding or control of the symptomatology associated with ADHD is of particular importance. Although sleep restlessness is no longer a DSM-IV diagnostic criterion as it was in the DSM-III (American Psychiatric Association, 1994; 1987), items about sleep difficulties are often included on checklists for ADHD or hyperactivity. Since a body of research exists describing the sleep problems in children with ADHD (Ball and Koloian, 1995; Mindell, 1994), further interest in the contributions of sleep differences in ADHD children seems warranted. Bergman (1976) found that one child's sleep disorders were misdiagnosed as hyperactivity. Salzarulo and Chevalier (1983) and Kaplan, McNichol, Conte and Moghadam (1987) both reported parents of ADHD children consider their children to have more sleep difficulties, both in falling asleep and staying asleep. Simonds and Parraga (1984) using the earlier diagnosis of Attention Deficit Disorder (ADD) found parents of ADD children at a clinic reported a number of sleep problems such as head banging, restless sleep and nighttime awakenings. Campbell, Schleifer, and Weiss (1978, 1979) reported interesting results of a longitudinal study that showed maternal reports of infant's sleep were related to maternal ratings of hyperactivity at both 4.5 and 6.5 years old. These studies of parental reports are intriguing because only a small percentage of parents of children in the age group from about 5 to 12 years old report their children have difficulty sleeping. Dahl, Pelham and Wierson (1991) present case study data of a 10 year old ADD child with sleep difficulties, in which a behavioral intervention focusing on increasing amount of sleep resulted in improvements in ADD symptoms. Sheldon, Spire & Levy (1992) suggest that "in some ADHD children...reducing sleepiness...and exogenously stimulating the cortex, may break the Cortex-Reticular Activating System-Cortex activation loop" that may be responsible for the hyperactivity seen in some of these

children and lead to the reduction in overactivity, restlessness and attentional deficits (p. 39).

While adolescents and adults exhibit familiar symptoms when they get sleepy, such as irritability, daytime sleepiness, napping, difficulty thinking, or mild depression, it has been suggested that it is more difficult to identify sleepy children. Sheldon, Spire & Levy, (1992) state that sleep loss in adults and adolescents is easier to identify in than in younger children, who have different symptoms, and may manifest attention deficits, cognitive, learning and reading disorders, and declining school performance. They present a hypothesis that it is possible some children diagnosed with ADHD may be experiencing impaired alertness due to some kind of sleep loss, which could in turn affect their ability to focus attention and/or inhibit impulsive behavior. In this age group (5-12) in which ADHD is first diagnosed, most children or their parents report few sleep difficulties or insomnia. These children are alert throughout the day and rarely take naps. Those who are not alert or sleepy may exhibit symptomology different than what we might expect in adults with similar sleep difficulties; symptoms similar to ADHD children, such as difficulty maintaining concentration, difficulty attending and orienting to a stimulus, and sustaining attention. This finding also provides an alternative explanation for the therapeutic effect of methylphenidate: impaired alertness and consequent daytime attentional, arousal and cognitive deficits the ADHD children are experiencing, is helped by amphetaminic drugs such as methylphenidate hydrochloride (trade name Ritalin), the way an insomniac is helped somewhat by drinking coffee.

The hypothesized impaired alertness may be due to sleep disturbances or other factors that deprive children of the required amount of sleep. Most of the data supporting the sleep impairment hypothesis comes from parents of ADHD children who consistently report greater sleep difficulties than the parents of non-ADHD children. Various other researchers, using polysomnograms to empirically investigate the sleep of both medicated and non-medicated ADHD children, have found some differences but the results do not seem to reveal any consistent pattern of differences from controls (Nahas and Krynicki, 1977; Busby, Firestone and Pivik, 1981; Khan, 1982). Other, and more recent research, has reported differences, but findings are still inconsistent. Ramos-Platon et al (1990) and

other researchers (Greenhill et al, 1983; DeLong, 1987) found differences in various sleep parameters including: shorter sleep onset latency, (the hyperactive group fell asleep sooner); an altered pattern of excess delta sleep; lower baseline REM activity.

It has been suggested of contradictory parental report data versus polysomnographic data that studies can not really be compared, since the “ADHD children” in the studies are a heterogeneous group due to the historical differences in diagnostic criteria and also due to the different definitions and measures that were used, both to define the groups and as outcome variables (Busby and Pivik, 1985). Samples may differ too much in behavioral and cognitive characteristics -- they may have had very different levels of impaired alertness, attention, impulsivity, and aggression. Another factor is that ADHD children are also a heterogeneous sleep group, of good and poor sleepers, differing in sleep characteristics and therefore levels of subsequent daytime impaired alertness. There may be a large subgroup of non-sleepy, and smaller subgroup of sleepy ADHD children. Also, as in the non-clinical population, they most likely reflect the natural variations in each individual’s need for nightly sleep, forming groups of long and short sleepers. Some ADHD children seem more likely than others to be impaired by sleepiness. Children designated as “Nonaggressive” (Halperin, et al, 1994) or, following the DSM-IV nosology, “Inattentive,” with their symptoms that look like sleepiness and higher levels of co-morbid reading and learning difficulties (areas of cognitive functioning also impacted by impaired alertness) may in fact be excessively sleepy, sleepier than Hyperactive-Impulsive ADHD children or than non-ADHD children.

The “cause” of this sleepiness is probably determined by a combination of a number of factors. Sleepiness may reflect the number of sleep disturbances often reported by parents to clinicians. Miller and Kraft (1992) suggest the reported polysomnographic studies that indicate differences in ADHD children’s sleep may be the result of sub-clinical differences brought out only episodically. Some type of chronic insomnia such as Delayed Sleep Phase Syndrome (Spielman, Nunes and Glovinsky, 1996) or periodic leg movements (PLMs) could be disturbing sleep at night (Picchietti and Walters, 1995). Sleep loss may reflect anxieties over poor social skills or poor classroom performance or from disturbances in family or peer relationships. It is important to learn to identify sleepy

ADHD children, because if there are “sub-clinical differences” as suggested by Miller and Kraft (1992), diurnal sleepiness could leave ADHD children more vulnerable to nocturnal sleep disturbance caused by stress or anxiety, resulting in decreased alertness the following day. To examine some of these concerns and add to the empirically derived knowledge in this area, it was necessary to assess and compare the effect of sleep loss on the sleep and levels of sleepiness between non-aggressive ADHD children and non-ADHD controls.

Significance of the study

This study was the first experiment of its kind to carefully investigate the effect of two hours of sleep deprivation on the subsequent sleep and levels of daytime sleepiness in ADHD children, compared to non-ADHD children of similar age, ethnicity, gender, SES, and levels of aggression. The children had their sleep and daytime nap opportunities electronically recorded for two nights and two days in the CCNY Sleep Disorders Center. The significance of the study is that, after carefully distinguishing the ADHD and non-ADHD groups, comparisons were made to determine if, as a result of the two hour sleep loss, there was a significantly lower level of daytime alertness in ADHD children than in non-ADHD controls the next day. This investigation was also the first determination of impairment in levels of attention and response inhibition on a continuous performance task (CPT), as a result of sleep loss. ADHD children were tested with the Test Of Variables of Attention™ (T.O.V.A.™) (Greenberg and Waldman, 1993; Greenberg, 1987), a normed CPT that utilizes visual stimuli. This determined the degree of attentional and impulse restraint impairment, both before and after mild sleep loss, as the ADHD children were compared, on the levels of impairment, to those of non-ADHD controls.

Better understanding of the relationship between sleep, the effect of sleep loss and impaired alertness leads to a better differential diagnosis, particularly by identifying children (both ADHD and non-ADHD) who are more vulnerable to sleep loss, especially if those who are impacted by sleep loss have subsequent decreased daytime alertness or sleepiness. Positive findings could lead to studies of how to best identify sleepy ADHD children as a basis for intervention studies using sleep schedules and other behavioral interventions to improve sleep, comparing their effectiveness to standard treatments such

as methylphenidate in reducing ADHD difficulties and other cognitive and neuropsychological outcome variables.

Study Hypotheses

This study had several underlying assumptions and hypotheses to be investigated. The major hypotheses concern the relationship of a complex set of variables: sleep, daytime sleepiness and degree of alertness, attention and impulsivity. The following research questions present the hypotheses the study tested.

- 1.) There may be distinct differences in the sleep of ADHD children. This question has not been settled definitively by the relevant research in the field, and further data could contribute to clarifying past findings. Strict adherence to DSM-IV criteria was maintained to clearly define the ADHD or non-ADHD groups according to known standards to allow this study to meaningfully test the hypothesis that the sleep of some ADHD children is different from non-ADHD children.
- 2.) Parents may be accurate as reporters of their children's sleep difficulties. This hypothesis concerns parents of ADHD children, who may or may not be reliable reporters of their children's sleep and sleep difficulties. While parents have often reported sleep difficulties, the polysomnographic recordings of sleep shows no particular pattern of differences. Ball and Koloian (1995) suggest the parent reports and polysomnography measure different variables. Are parents of more impulsive, overactive and inattentive children more sensitive recorders of their children's sleep difficulties than standard polysomnography, conducted by taping twenty electrodes and wires to a child's head, and then having the child sleep in an artificial environment for a relatively limited observation time? Such electronic recording affects what is observed in the short run, and may not be as reliable for detecting long term patterns of the child's customary sleep difficulties that parents are able to observe.
- 3.) Do ADHD children have somewhat different sleep architecture that leaves them more vulnerable to further impaired alertness and subsequent daytime attention difficulties when they lose sleep? If ADHD children have subclinical differences, as some researchers

suggest, it may be assumed that they could become sleepier from the same amount of sleep loss than non-ADHD children.

4.) Sleep loss (staying up late) may intensify ADHD behaviors and causes more difficulty focusing and sustaining attention for ADHD children than their non-ADHD peers was a further hypothesis of the study.

5.) Disturbed sleep may produce impaired alertness and epiphenomenal attentional and/or cognitive difficulties the next day. This final hypothesis suggested that some children may not sleep well, or have undiagnosed sleep disorders, and thus their symptoms could be mistaken for ADHD type behaviors. If sleep difficulties affect levels of alertness and ability to attend, and sustain attention, then sleepy children may show higher levels of daytime sleepiness, or lower levels of alertness that impacts attentiveness and impulse control..

Specific Aims

In order to test these hypotheses, this study was the first pediatric sleep study designed to carefully compare non-aggressive, ADHD children with non-aggressive, non-ADHD controls, both before and after the effect of staying “up late” at night. The study was looking at differences between the groups in sleep architecture, levels of daytime sleepiness, activity levels and cognitive functioning before and after the mild sleep loss of two hours past the child’s usual bedtime.

1.) Hypothesis one, that there may be distinct differences in the nighttime recordings of the sleep of ADHD children, was tested by comparing the sleep characteristics of non-ADHD children to children with non-aggressive, Inattentive, Hyperactive-Impulsive and Mixed types of ADHD behaviors, to investigate inconsistencies in previous findings that sleep characteristics of children with ADHD differ from non-ADHD children.

2.) Hypothesis two, that parents are accurate reporters of their children’s sleep difficulties, was tested by comparing subjective parental reports versus objective recordings of sleep to assess the degree of correlation, to determine whether subjective

reports of sleep by parents of ADHD children can reliably be used to differentiate sleep related difficulties.

3.) The third hypothesis, that ADHD children might get sleepier during the daytime than non-ADHD children from an equivalent amount of sleep loss, was tested by a comparison that was made of how sleepy both groups of children were before and after the sleep loss using the Multiple Sleep Latency Test (MSLT 1 & 2). The MSLT is a series of five nap opportunities during the day, lasting 25 minutes and scheduled every two hours, starting two hours after getting out of bed. After staying in the lab the first night, and staying “up late” for two hours on the second night, a comparison of the levels of daytime sleepiness/alertness in the different groups of children was made using the MSLT. It was proposed that the ADHD children would be made more sleepy if they have sleep differences that should leave them more vulnerable than age-matched controls, to further sleep loss.

4.) A fourth hypothesis was that the ADHD children should not do as well as the non-ADHD children on the T.O.V.A.™ assuming they have sleep differences leaving them more vulnerable to cognitive difficulties from further sleep loss. This was tested by comparing the effect of staying “up late” at night on the behavioral and cognitive functioning of the ADHD and non-ADHD children, as determined by their performance on this a visual stimulus computerized performance test (CPT). The T.O.V.A.™ presented a target and a non-target on the computer screen and children had to press a button in their hand at the target. Inattentiveness was operationally defined by the T.O.V.A.™ as errors of omission, (not pressing at the target). Impulsivity was operationally defined as errors of commission, (pressing at the non-target). How the sleep loss affects each group’s ability to attend and restrain impulsivity was measured before and after the sleep loss night using the Test of Variables of Attention (T.O.V.A.™ 1, 2 & 3).

5.) A fifth hypothesis, that sleep loss can produce epiphenomenal ADHD-like behaviors, was tested by comparisons of the non-ADHD children’s behavioral and cognitive performance before and after two hours of sleep loss, to determine if some sleep loss affects a non-ADHD child’s ability to focus and sustain attention, and restrain impulsivity, as measured by the Test of Variables of Attention (T.O.V.A.™ 1, 2 & 3).

CHAPTER TWO

REVIEW OF THE LITERATURE

Overview of ADHD

Attention Deficit Hyperactivity Disorder (ADHD) represents one of the most common referral complaints to mental health professionals (Ross and Ross, 1982). The fourth edition of the Diagnostic and Statistical Manual (DSM-IV) reports the prevalence to be estimated at three to five percent of school age children with the limited data for adolescents and adults not reported (American Psychiatric Association, 1994). Few childhood disorders have received as much theoretical and scientific scrutiny as ADHD in the past 25 years (Guevremont and Barkley, 1992). Still (1902) is attributed with being the first to identify ADHD as a cluster of aggressive, defiant behaviors in a group of clinical patients he saw, with symptoms including inattention and overactivity (Ball and Koloian, 1995). Since then, these difficulties have been referred to as minimal brain disorder, minimal brain dysfunction, hyperkinesis, hyperactive child syndrome and, as recently as 1987, Attention Deficit Disorder, with and without Hyperactivity (American Psychiatric Association, 1987). Although diagnostic concerns led the former Surgeon General, C. Everett Koop to write, that despite being widely researched, "there is no universal agreement...as to what constitutes hyperactivity" (Bain, 1991, p. 2), there is an official psychiatric definition or agreement: the DSM-IV.

The DSM-IV has redefined the disorder, reorganizing ADHD criteria as ADHD with three distinct subtypes: Inattentive, Hyperactive and Mixed (APA, 1994). The essential features of ADHD are persistent patterns of inattention and/or hyperactivity/impulsivity that is "more frequent and severe" than is age appropriate and began as a persistent pattern early in life, beginning before the child is seven years old (APA, 1994, p. 78). Two other criteria are also needed: six or more symptoms from a list of inattention and hyperactivity/impulsivity symptoms are required, and importantly, the impaired behavior must be observed in at least two contexts (home, school, work or social situations) before the diagnosis may be assigned. The difficulties must be pervasive across

time and situations and severe enough to interfere with relationships with caregivers, teachers, siblings and peers.

It might be useful to pause for a moment to define these major constructs and variables. The Random House Dictionary of the English Language (Stein and Urdang, 1979) provides some badly needed semantic precision. Inattention is lack of, or insufficient attention (Stein and Urdang, 1979). Attention is “the act or faculty of attending...concentration of the mind on single object or thought...a state of concentration characterized by concentration” (p. 96). Activity is “constantly engaged in action...in progress or motion...involving physical effort and action” (p. 15) and Overactivity is active to excess, too active” (p. 1025). Impulsivity is “activated or swayed by emotional or involuntary impulses” (p. 717). These definitions place an emphasis on the inability of the child to pay attention or inhibit impulsiveness and activity appropriately.

Diagnosing ADHD

The DSM-IV also acknowledges that both ADHD and non-ADHD children may display these “symptoms” as normal behaviors, and so, the revised diagnosis emphasizes the pervasive and damaging qualities of the difficulties of ADHD children. Accordingly, it is only made when there are long-standing behavioral patterns of “insufficient attention,” “being constantly engaged in action to excess” and being “swayed by emotional or involuntary impulses” both at home and at school that bring the child into grave conflict with caregivers, teachers and peers. There has to be “clear evidence of interference with developmentally appropriate social, academic, or occupational functioning” (APA, 1994, p. 78). The specific difficulties are listed as “symptoms” in the DSM-IV. Most ADHD children have symptoms of both inattention and hyperactivity/impulsivity, although in some children one of the subtype patterns predominates. “Inattentive Type” is used when six or more symptoms of inattentiveness are present, and less than six Hyperactive/Impulsive symptoms have persisted for six months. “Hyperactive/Impulsive” is used when six or more symptoms are present, and less than six Inattentive symptoms. “Combined Type” is used when six or more symptoms of both are present (p. 80). Some of the criteria behaviors Inattentive children show: they may fail to give close attention or sustain attention, shifting

from task to task, seem as if their mind is elsewhere, have difficulty organizing tasks and activities, fail to follow through on instructions. Hyperactive/Impulsive children are “on the go,” fidget in their seats and/or with objects, tap their hands, shake their feet, talk excessively, have difficulty delaying responses, blurt out answers, intrude on others, take and grab things, don’t wait their turn and so on.

The etiology of ADHD is, and always has been the subject of much debate and research. Several theoretical approaches underlie attempts to define the cause of inattention, yet “implicit in all theories is the notion of arousal or alertness” (Halperin et al., 1994, p. 5). Two contrasting theories about the broad physiological etiology of hyperactivity use the concept of an imbalance in level of arousal. Arousal is defined as how determined, or how motivated, a child is to act. Some theories postulate the source of increased motor activity is hypoarousal, so that a child becomes more active to increase arousal to an optimal level. Others suggest that hyperarousal is responsible (Hastings and Barkley, 1978; Busby, Firestone and Pivik, 1981). In this model, ADHD children are excessively active in order to “burn off” energy and decrease arousal to an optimal level. Both theories understand and explain ADHD symptoms as behavioral responses to attain a moderate, optimal level of arousal, which then facilitates optimal performance (Hebb, 1955).

Defining ADHD Constructs

Despite the lack of universal agreement about hyperactivity that Koop notes (Bain, 1991), the DSM-IV model serves as a standard for clinical and research purposes. There is now much clinical evidence and research in the field reflecting the general consensus of parents, teachers, psychiatrists and psychologists, that “hyperactive” children show pronounced deficiencies in focusing or sustaining attention and controlling impulsive responses as the primary impairment, with overactivity now seen as a separate impairment associated with impulsivity (Guevremont and Barkley, 1992). Factor analysis suggests that overactivity is a symptom distinct from inattention (Edelbrok, Costello and Kessler, 1984; Lahey et al, 1988). These distinct clusters of symptoms of hyperactive-impulsive and/or inattention have emerged out of both clinical and research work with ADHD

children, and have been reflected in the changing DSM-IV operational criteria for the diagnosis of ADHD (APA 1994).

Besides historically changing criteria (APA, 1980; 1987; 1994) there are other difficulties with the diagnosis of ADHD, such as concerns about the variability in the criteria: the first source of variability depends on who is making the diagnosis and applying the criteria. Who labels the child “hyperactive” often varies. Parents, teachers, pediatricians, psychiatrists and psychologists all do the initial identification of ADHD children, and all have different standards of appropriateness. Or, put another way, how much age-inappropriateness they will tolerate before they define the child as “too” inattentive or “excessively” impulsive. A subjective report bias, or Halo Effect exists, when children are overrated as more inattentive and more hyperactive/impulsive even when they are not. Different instruments and checklists are used, other than the evolving criteria of the DSM (Conners, 1989). It has long been noted that there is frequently a day-to-day variability in the number and severity of the symptoms (Weiss, 1975). There is also situational variability in the symptoms reported by clinicians and researchers, as these children are not always inattentive and impulsive, particularly in novel situations (Katz, Saraf, Gittelman-Klein and Klein, 1975). There is a maturational variability as well, as cognitive capacities are continually developing as young brains interact with their family and school environments and the child gains from experience and maturity. Then there is the long standing problem of considerable overlap with other psychiatric difficulties, especially Learning and Reading Disorders with the Inattentive group, and Conduct and Oppositional Defiant disorders with the Hyperactive-Impulsive groups. These concerns have been taken into consideration to some degree, in the most recent incarnation of the American Psychiatric Association criteria for ADHD, as reported above (APA, 1994).

Recently, researchers have begun to look at better understanding the relationship between the overlapping symptoms. Researchers have characterized subgroups of ADHD children on the basis of these common co-morbid diagnoses, Reading Disabilities (RD) and Learning Disabilities (LD) that together cluster with the Inattentive subtype versus Conduct Disorder (CD) and Oppositional Defiant Disorders (ODD) that cluster along with Hyperactive/Impulsive symptoms (Halperin, McKay, Matier and Sharma, 1994). This

reflects the cognitive versus behavioral domains of impairment that have long been recognized (Wender, 1987) but also reflects more recent understanding of an underlying contributing factor that differentiates the two ADHD subgroups, the dimension of aggression. It has also been long observed that children who are classified as hyperactive can also display aggressive, anti-social behaviors (Katz, Saraf, Gittelman-Klein and Klein, 1975; Halperin, McKay, Matier and Sharma, 1994). ADHD children are also frequently diagnosed with learning disabilities (Halperin, Gittelman-Klein, Rudel, 1984). RD and LD, or cognitive impairments are seen more often in non-aggressive ADHD children, whereas CD and ODD or behavioral deficiencies are more often seen in aggressive ADHD children (Halperin, et al., 1984; Halperin, et al., 1994). The Inattentive subtype seems to be less aggressive than the Hyperactive/Impulsive subtype, but they are by no means completely distinct, as there are many non-aggressive Hyperactive/Impulsive children. Halperin (1994) has suggested these related clusters of different symptoms reflect different underlying neuropsychological problems in the aggressive, versus non-aggressive groups. In fact, this recent line of research inquiry suggests that at least partially divergent neural substrates mediate each of these constructs. For example, Matier (1992) found, in two groups of aggressive and non-aggressive boys, differential effects of methylphenidate (trade name Ritalin) on objective measures of attention, impulsivity and overactivity.

Distinct long-term outcomes reinforce this notion of different underlying neural substrates. Impulsivity is most often associated with aggression (August and Garfinkel, 1989; Halperin, 1990b) and aggression has been found to be predictive of antisocial behavior and drug abuse (Loney, Kramer and Milich 1981). However, high levels of inattention are more closely related with LD (Halperin et al, 1990b; August and Garfinkel, 1989).

Measuring ADHD Constructs

Because the diagnosis emphasizes inappropriate levels of inattention, impulsivity and overactivity, these are the constructs most often examined in hyperactive children. There are separate sets of problems in operationally defining and then measuring each of these constructs (Halperin, Gittelman, Klein and Rudel, 1984; Halperin, et al., 1994).

While operationally defining constructs allows measurement, comparison and research, leading to improved clinical interventions, the difficulties noted in defining the constructs makes this process challenging. The first challenge is how to measure the constructs. Generally, the clinician or researcher can use subjective or objective measures to operationally define the constructs and quantify level of impairment. "Subjective" data refers to subjective report data from self report or more likely, observer reports from parents or teachers. The DSM-IV diagnostic interview is essentially a parent report rating scale (APA, 1994). The Conners Parent Rating Scale (CPRS) (Conners, 1989), and a variety of other parent and teacher instruments are also used, are reported in the literature. "Objective" data refers to data generated by testing the child with various kinds of instruments that rely mostly on the performance of the child, rather than subjective impressions of others. WISC-III subtests (Wechsler, 1994), various neuropsychological such as the Stroop Interference test have been used to distinguish between ADHD and controls (Stroop, 1935; for review, see Barkley et al, 1992). Different kinds of computerized performance tests (CPTs) such as the Tests of Variables Of Attention, or T.O.V.A.TM (Greenberg, 1987) have been used as well.

Specific issues concerning measurement of these constructs and the various measures used to operationally define them will be addressed in the next chapter on Methodology, while here focusing on the broader measurement issues. From how one is going to measure ADHD behaviors, clinicians and researchers then must decide exactly what to measure. Since each of the ADHD behavioral constructs have different cognitive and behavioral components to them, they must be measured in a number of different ways. Again, much more will be said about measuring these various elements in the next chapter. Inattention is composed of various cognitive processes: the orienting response, selective attention, sustained attention; there is also a capacity for attention. The construct of impulsivity may be the ability to withhold a goal directed response and delaying of gratification until an appropriate time (Halperin, et al., 1994). Activity is generally considered to be the amount of physical movement exhibited by an individual.

Each of these components of attention are typically assessed using different instruments, each of which pose difficulties. Level of motivation is also crucial -- if the

child doesn't want to try or try very hard, different results are obtained than when motivated to do well.

ADHD Interventions: Ritalin and other approaches

ADHD was originally conceptualized as a mild form of retardation in which lesions to specific cortical locations were thought to be the cause of the observed cognitive and behavioral deficiencies (Wender, 1974). As ADHD theory and research evolved, a new approach evolved based on the belief that neurotransmitter imbalances that have no direct underlying neurological lesions were responsible for the deficiencies in response inhibition and attention. Bassuk and Schoonover (1978) note that these behaviors were found to improve with central nervous system (CNS) stimulants, or neuroanaleptics such as dextroamphetamine (trade name Dexadrine) and later, methylphenidate hydrochloride (trade name Ritalin). Recent research in this area indicates that there may be separate neural determinants of each symptom cluster (Matier, 1992). Methylphenidate has its main effects on the catecholamine system, and has long been recognized as an effective tool in regulating impairments in both symptom-cluster domains, improving the behavior of 75-90% of children. This has reinforced the belief among child neuropsychologists that the central catecholamine systems are involved in regulating these constructs.

Medications remain the most common intervention for ADHD. Pelham, Bender, Caddell, Booth and Moorner (1985) and others report that most children with ADHD showed decreases in hyperactive behaviors when given stimulants. Although it has been associated with insomnia (Bassuk and Schoonover, 1978), or delayed sleep onset, methylphenidate seems to have little effect on sleep architecture or patterns (Feinberg, 1974; Haig, Schroeder and Schroeder, 1974; Nahas and Krynicki, 1977; Greenhill et al., 1984). Burd and Kerbeshian (1991) found methylphenidate and pemoline (trade name Cylert) improved sleep in the case of a 3 year old girl with pervasive hyperactivity and decreased sleep. Ferber (1986) states that medications like pemoline, methylphenidate, and the amphetamines work best in alleviating sleepiness in children who suffer from narcolepsy, children who are continually, excessively sleepy.

However, earlier researchers noted serious side effects, such as weight loss, insomnia and high blood pressure, reported when dextroamphetamine and methylphenidate are used by children (Safer and Allen, 1975). Although data has not established a causal relationship, some research on the long-term effects of stimulants on children's growth report suppression of growth (Huff, 1982). Due to the tremendous variability in the ranges of ages and severity of symptoms, for example, there is much difficulty finding the proper dosage (Sprague and Sleator, 1974; Sprague and Berger, 1980), resulting in gains that seem to be only short-term. Again, as with behavioral interventions, in the long run, ADHD children may not be any better off receiving these amphetamines (Whalen and Henker, 1976). Recent research indicates that other medications for ADHD are also problematic. Fluoxetine (trade name Prozac) given to adults with residual ADHD in one study resulted in excessive somnolence (Sabalesky, 1990) and exacerbated behavioral symptoms in children with ADHD in another (Riddle, King, Hardin and Scahill, 1990). Caffeine was suggested by Waksman (1983) as a possible, less-toxic substitute for CNS stimulants. However, Bernstein et al (1994) reviews and summarizes this literature as mixed results on the efficacy of caffeine. Some very good studies did not find effective improvement (Reichard and Elder, 1977) while others did (Schnackenberg, 1973). When floropipamide (trade name Dipiperone), a neuroleptic drug was given to six children, three children had reduced hyperkinetic symptoms and three did not, but all six had their REM sleep reduced.

Many other interventions for ADHD remain problematic. Feingold (1974) advanced a hypothesis involving carbohydrates and claimed to have developed a useful intervention based on the relationship between diet and behavior disorders. While this might be helpful in individual cases, and it caused a flurry of excited research, Waksman's (1983) review of the research up to that time found no controlled studies to support these claims of a link between hyperactivity and the pervasive, situational and persistent inattentiveness, impulsivity and overactivity seen in these children. A more recent review by Spring, Chiodo and Bowen (1987) has also concluded that a high-carbohydrate diet does not provoke hyperactivity. Operant conditioning programs have been somewhat effective in treating ADHD in the short run (Patterson, 1965; Ayllon and Rosenbaum,

1977). However, as promising as the case reports and short-term studies of such treatment seem to be, there have not been any studies of the long term effects. Furthermore, most of these studies did not include control groups (Rosenhan and Seligman, 1989). The strong emotional component of ADHD forces the recognition that emotional stress can cause increases in hyperactivity (Katz, Saraf, Gittelman-Klein, and Klein, 1975). Therapeutic approaches focusing not only on the child, but working with the whole family can often be highly effective in helping ADHD children and their families.

Overview of Children's Sleep

Before turning to a discussion of the sleep studies with ADHD children, it will be useful to review the normal development and features of sleep in children first, and then review typical sleep difficulties in children, focusing on the 6 to 12 year old age group, the age most children are first identified as having ADHD.

As any parent will tell you, newborns, infants and children spend a major portion of their lives asleep. Newborns spend approximately 70% of every 24 hours asleep (Sheldon, Spire and Levy, 1992). In contrast, adults spend 25 to 30% of their day sleeping. Sleep, therefore, represents a tremendously important area of each young child's experience, as sleep states are the predominant states of consciousness very early in life (Sheldon, Spire and Levy, 1992). Infants, children and adolescents not only show more need for sleep, their sleep has very different characteristics than those of adults (Hauri and Olmstead, 1980). Sleep continues to change and evolve from birth to adulthood, along with the physical maturation of the body. A fetus, by the seventh to eighth month, establishes a distinct cycle of activity and quiescence that can be seen in the fetus' behavior.

Babies, from birth to twelve months old have three distinct phases of sleep. *Active sleep*, (REM sleep) is approximately 30% of the total sleep time (TST) at six to twelve months old. Sucking movements are most common in active sleep. Twitches, grimaces, smiles, limb movements and tremors also occur in active sleep (Sheldon, Spire and Levy, 1992; Coons, 1987). Aserinsky and Kleitman (1953) first characterized Rapid Eye Movement (REM) sleep, which has more eye movements, body movements and irregular

breathing, and emerges as a regular cycle as early as seven months old. *Quiet sleep* (NREM) is the next distinct phase of infant's sleep. Quiet sleep continues to increase as a percentage of TST until it becomes the dominant form of sleep at about three months after birth (Sheldon, Spire and Levy, 1992). The final phase is undetermined, in which it seems neither REM nor Non-REM sleep. There is, however, some debate over what exactly is sleep or wakefulness in a neonate (since it is asleep more than awake) and over what criteria to use: observational, behavioral or polysomnography (Hoppenbrouwers, 1992; Sheldon, Spire, Levy, 1992).

Most of a person's existence is asleep very early in life - and most of sleep is active, perhaps REM sleep. Both sleep and REM sleep decrease throughout life, until stabilizing after adolescence at a little above young adult levels. By four months old, TST decreases from 16 to 17 hours per day to 14 to 15 hours per day. Sleep spindles, fast "bursts" of low amplitude waves, a distinct EEG waveform associated with Stage Two sleep, (Sheldon, Spire and Levy, 1992) emerge around four months old as well. Another distinct waveform, the delta wave, a slow, high amplitude wave associated with Stage 3 and Stage 4, emerges at about six to eight months. Now the baby is sleeping only about 13 to 14 hours per day. The child's sleep/wake cycle gradually becomes organized. By six months the child's longest sleep period is at night, and longest alert period is during the day; these patterns get "locked in" by nine months old (Sheldon, Spire and Levy, 1992). About 80% of infants in one study slept through the night by the age of six months, and after this consolidation of the longest sleep period coming after the longest wakeful period, the differentiation of NREM stages became more apparent (Coons, 1987).

By two years old, this pattern is often well established. Sleep has been consolidated into a long nocturnal period of approximately ten hours, while naps are gradually given up by 3-5 years old. At the end of their first year, children's REM periods continue to mature and develop. Gradually, the first REM period becomes quite short, and tends to become longer towards morning, establishing the beginning of their adult REM patterns. Between two and five years old, REM sleep as a percentage of TST decreases from 30% to the adult level of 20 to 25%. At this age, the child's sleep has stabilized and matured.

Sleep continues to evolve into adult patterns from five to ten years old, although TST is about two and a half hours longer. There is an orderly sequence of sleep stages by this time. Progressively longer REM periods have emerged. Body movements tend to decrease in frequency and naps become more rare. By early adolescence, electrophysiological variables of sleep approximate normal adult values (Sheldon, Spire and Levy, 1992), although adolescents require about an hour more TST than adults. REM sleep is now approximately 20 to 25% of TST, 2 to 5% of TST is spent in Stage 1, and about half of TST is in Stage 2.

Differences in school night versus non-school night sleep can be seen distinctly now, if not already apparent earlier. Children under ten usually have about the same TST on school versus non-school nights. Young adolescents sleep less on school nights (522 minutes) than non-school nights (560 minutes). There is often a continuous decrease in TST through mid and late teenage years of about two hours (Sheldon, Spire and Levy, 1992). This cumulative sleep restriction often results in the increased daytime sleepiness older adolescents often report (Carskadon and Dement, 1987b).

One theory of sleep centers on the role of sleep in the process of learning and memory (Dewan and Greenberg, 1968). Considerable brain activity occurs in REM sleep, oxygen consumption increases, cerebral blood flow increases, neurons are intensely active. Many studies in humans and animals have shown the beneficial effect of sleep on the retention of memories acquired during wakefulness. For example, there is evidence that RNA is more actively synthesized in sleep. RNA synthesis has been linked to memory process. Like infants, children and adults have also been found to have increased REM sleep following learning (Paul and Dittrichova, 1974). One of the purposes of REM sleep, then, may be a neurobiological expression of the process of long term memory consolidation. The learning theory may be supported by developmental sleep data. Infants, with their special task of organizing their sensations into coherent, stable perceptions, and also being continually preoccupied by another crucial task, that of making meaning of experiences with self and mother naturally needs much time processing and consolidating learned information.

Sleepiness is assessed using the Multiple Sleep Latency Test (MSLT) that enables researchers and clinicians to objectively measure sleepiness as a function of the speed of falling asleep during nap opportunities. (Carskadon and Dement, 1987). Using the MSLT, Carskadon and Dement (1987) found that, by late adolescence sleep debt can be significant as measured by the MSLT. A study by Carskadon and her co-workers on the sleep of adolescents, who were followed longitudinally for seven years, found that subjects would sleep ten hours when given the opportunity and usually slept for more than nine hours. One conclusion is that adolescents do not have fully adult sleep patterns yet. They still need 9 to 10 hours of sleep per night as they mature -- which is one reason why they get sleepy so often in the daytime. Before turning to the subject of the relationship between hyperactivity and sleep, it will be helpful to briefly review the kinds of difficulties sleeping younger children can experience.

Typical Disorders of Sleep in Childhood

The kinds of difficulties children experience sleeping are many and varied. Problems are often first classified on the basis of age, or whether they impact the night's sleep or daytime alertness. As with adults, this impact is considered to fall into one of two categories: Disorders of initiating or maintaining sleep (DIMS) or disorders of excessive somnolence, or (DOES) (Guilleminault, 1987; Schaefer, 1992). Sleep difficulties are then more precisely classified on the specific behavioral criteria exhibited (Spielman and Herrera, 1991). Studies attempting to link sleeping difficulties with specific causes in infants and young children often report contradictory results; leading Ferber (1987) to state that the individual differences seen in terms of susceptibility or precipitating events indicates that "multiple factors may have to exist simultaneously to cause a problem" (p. 143).

Problems which begin in childhood can have an impact on people their whole lives. Hauri and Olmstead (1980) showed that adult insomniacs whose insomnia started in childhood took significantly longer to fall asleep than adult-onset insomniacs. Interestingly, the patients with childhood-onset insomnia tended to show more evidence of hyperkinesis, dyslexia, attention deficit disorder and showed evidence of other "soft"

neurological impairment as well. Establishing fixed, regular sleep/wake schedules, is crucial for parents to do for their infants and children. Strict regulation of sleeping/waking and hunger/eating helps facilitate rather than impede circadian rhythmicity (Ferber, 1986). This "allows the child to have a sense of well-being during the day" (p. 33) that is grounded both in the physical satisfaction of "drive" needs, and also in the psychological satisfaction that comes from the kind of consistent, predictable caregiving that allows the infant to learn to trust the parent, and in turn, trust itself (Erikson, 1963).

Irregular sleeping/waking schedules, then, represent one of the earliest emergent disorders of children's sleep. This is a type of difficulty children have because their bedtimes, awakening and naps, if they take them, are all at varied times each day. Meals, too, often vary (Ferber, 1986). Lack of structured schedules may be preference, or time pressures at work that don't permit parents to more carefully regulate and enforce bedtimes. After a year, most children have consolidated their sleep into one long sleep period, at night, along with one or two naps in the morning and afternoon. This early type of sleep/wake cycle needs to be structured and supported by the child's parents in order to be firmly established. The kinds of things parents can do to avoid originating sleep difficulties are: avoiding play or other exciting stimulation at night so that night becomes associated with "sleeptime" and day becomes associated with play and "waketime." This conditioning process is also assisted by teaching the child that the bed is for sleeping, and discouraging sleeping on the sofa or in the parents' arms. Parents should also avoid making a fuss when the child wakes up and cries at night. Changing, feeding or comforting should be done in the crib or bed if possible (American Sleep Disorders Association, 1992).

From approximately ages one to three, children make tremendous advances toward self-regulation of physiological need states and towards autonomy, as sense of self continues to develop along with a developing sense of self and growing independence, competence and self-efficacy (Mahler, Pine and Bergman, 1975; Erikson, 1963). Of the types of problems reported to pediatricians at this age, crying and nocturnal awakenings are the most common. Often parents' efforts to soothe the child perpetuates the problem: taking the crying child out of bed to talk, sing, feed or read to them not only rewards

children with parental attention, it doesn't allow children to learn to soothe themselves (Ferber, 1986). To solve this exasperating difficulty, parents need to give the child a favorite toy, or a blanket when initially putting them to sleep, and to wait five minutes before returning if the child cries, staying for only a few minutes, and not to pick up the child. If the crying continues, waiting 10 minutes before returning, then 15 before returning once more, and so on, adding five minutes to each subsequent waiting period usually is effective, as it provides reassurance by the parent's presence, and also tacitly implies and promotes the child's confidence to fall asleep by him or herself (Ferber, 1987a).

During the ages from three to five, establishing a constant pattern can ease the transition from waking to sleeping. By avoiding scary or exciting television or bedtime stories, and resisting delaying requests for "one more story" or "a last drink of water" is crucial to teach children that these rules are consistent (American Sleep Disorders Association, 1992). Soon after this age, the child begins a time of robust sleep. From the ages of 4 to 12 years old, there is a considerable reduction in both problems initiating and maintaining sleep (Ferber, 1987). Most children this age, having established strong circadian cycles and independent waking and sleeping routines, sleep quite soundly, not yet affected by the stressors or sleep deprivation commonly experienced in adolescence. Children have by now, typically begun to manifest the kinds of lifelong trends of being either an early or late riser, ("larks" or "owls," respectively). These inherent factors need to be considered in identifying and treating a sleep disorder, and yet "there has been little formal study of the contribution of inherent biological differences to the establishment of normal circadian patterns and the likelihood of developing related sleep disturbances" (Ferber, 1987, p.166).

The most common problem reported at these ages most often is about bedtime, rather than sleep (American Sleep Disorders Association, 1992). Bedtimes may be pushed ahead to watch television or do homework. Sleepiness the next day may be the result of continual, insufficient sleep. Teachers may report cranky, irritable or sleepy children to their parents. Other, more serious sleep difficulties may also be interfering with a good

night's sleep. Narcolepsy, a neurological disorder, or a type of sleep apnea, a breathing disorder, can produce symptoms of excessive daytime somnolence.

Sleep apnea in children may occur in infancy and may be chronic or acute. It may accompany tonsillitis, for example (Hauri, 1982). Unless the apnea is particularly severe, however, many "of the less flagrant cases are not properly diagnosed and parent's complaints of a child's breathing difficulties is commonly ignored" (p. 59). Narcoleptic children may fall asleep while talking, for example, while children with apneas may snore or report headaches (American Sleep Disorders Association, 1992; Hauri, 1982). Narcoleptic teenagers show sleepiness far more excessive than that of their peers. Often overlooked because sleepiness in adolescents is quite common, it is often first noticed by teachers who report the excessive sleepiness to parents as a reason for poor classroom performance (Guilleminault, 1987). Behavioral treatment is essential, and regular naps are prescribed. Often, medication is also necessary, and pemoline, methylphenidate, amphetamines or tricyclic medications are used to treat narcoleptic adolescents (Guilleminault, 1987).

It has been reported that one in four children exhibit enuresis, or bedwetting. While a vexing problem for children and parents, and often the source of considerable worry and stress, it is typically not considered a disorder until it continues past the age of five, although one in ten six year olds and one in twenty ten year olds continue to have problems staying dry every night, bedwetting usually disappears over time by itself, and behavioral interventions can be quite effective (Guilleminault, 1987; Ferber, 1986).

Delayed Sleep Phase Syndrome can emerge as an area of major difficulty for adolescents. Late hours from after-school work schedules or socializing leaves teenagers dozing in class or sleeping later on weekend, disrupting their circadian regularity and causing concomitant sleeping problems. Adjusting the individual's sleep phases, either by staying up all night Friday and not napping Saturday, followed by rising at the proper time on Sunday, or by arising at the proper time on Saturday morning and going to sleep early on Saturday night are two ways to deal with the delayed phase difficulties (American Sleep Disorders Association, 1992).

Between the ages of twelve to twenty, the most rapid growth of the body takes place after infancy. Adolescents, studies show, tend to need about one hour or two more sleep each day than they tend to get. If permitted to sleep as long as they want, adolescents average about nine hours a night (American Sleep Disorders Association, 1992). Carskadon and Dement (1987) reported results of a longitudinal study that showed a significant sleep debt will build up in adolescents sleeping seven hours a night or less and they can be "excessively sleepy as a result" (p. 56).

Sleep Studies with ADHD children

As has been previously described, a growing body of research exists describing the sleep problems in patients diagnosed with ADHD (Mindell, 1993). Bergman (1976) cited evidence which sometimes suggests that significant sleep disorders can be misdiagnosed as "hyperactivity." Ferber (1986) reported retrospective studies on narcoleptic adults which indicated that "over ten percent of [adult narcoleptics] had been misdiagnosed as hyperactive as children...and were treated with stimulants--the right drug perhaps... but for the wrong reason" (p. 229). That these adults, with a sleep disorder associated with symptoms of excessive daytime sleepiness, were misdiagnosed as hyperactive is intriguing, and lends weight to the underlying hypothesis of the present study. Kaplan, McNicol, Conte and Moghadam (1987) found ADHD to be associated with sleep pathologies. In a survey of parents comparing children and adolescents evaluated at a clinic with a non-clinical sample, Simonds and Parraga, (1984) showed that a significantly greater number of children with ADD had problems with snoring, head-banging, restless sleep and nighttime awakenings (NA). A longitudinal study of maternal reports comparing behaviors in hyperactive and control children indicated that not only were the maternal reports of their infants' sleep problems related to maternal ratings of hyperactivity at 4 1/2 and 6 1/2 years, but that their ratings at 4 1/2 were predictive of 6 1/2 year hyperactivity (Campbell, Schleifer and Weiss, 1979). Porrino, Rapaport, Behar, and Bunney (1983) reported more body movements in ADHD children than in controls. Salzarulo and Chevalier (1983) stated that parents of children with ADHD reported sleep onset problems 16.5% of the time and nighttime awakenings 39% of the time.

While no definitive patterns have been confirmed, an emerging body of research indicates some REM and other sleep differences in ADHD children when compared to normal sleeping children. Several researchers have found certain REM sleep characteristics different from controls. When Greenhill, Puig-Antich, Goetz, Hanlon, and Davies (1983) compared nine ADHD boys to eleven normal controls before and after six months of medication with methylphenidate, four of the nine children were reported to have serious sleep disturbances. They found significant polysomnographic record differences in their sleep in lower REM density and REM activity. They also found that the methylphenidate treatment the ADHD children received was associated with longer TST, and increased number of REM periods. Greenhill, et al. (1984) found an ADHD group to have lower baseline REM activity than controls. One child in their sample of six had an unusually low REM sleep percentage of TST.

Comparing controls to ADHD children, Khan (1982) found no significant differences in total amount of REM sleep, but he did find the hyperactive group had significant shorter REM latencies. Ramos-Platon, Vela-Bueno, Espinar-Sierra, and Kales, (1990) found ADHD subjects, compare to controls had shorter sleep onset latency (SOL), along with greater total sleep time (TST), more nighttime awakenings and an altered sleep pattern in which delta sleep predominated. DeLong (1987) found that 51 of 57 children with 14 and 16 Hz positive spike phenomena were found to exhibit behavioral and sleep disorders and hyperactivity and attention deficits. Following the treatment of a ten year old girl for her sleep problems, her improvement in overall sleep contributed to clinically meaningful reductions of ADHD behaviors in school (Dahl, Pelham and Wierson, 1991).

Other research has shown that both unmedicated and medicated ADHD appear to produce few major effects on polysomnographic differences between ADHD children and normal controls (Stores, 1992). Sleep spindle activity in ADHD children was found by Kiesow and Surwillo, (1987) not to be significantly different from a control group. Similarly, Busby, Firestone, and Pivik (1981) found that for a small group of ADHD patients, clinically reported sleep problems could not be strongly supported by available polysomnographic evidence. However, these researchers also point out that there are serious difficulties pooling or comparing the data of these sleep studies with “hyperactive

children” because of widely different criteria, subject selection methods and experimental conditions.

There are indications from one recent study that ADHD children may be sleepier in daytime than controls. Palm, Persson, Bjerre, Elmquist and Blennow (1992) used modified MSLT's to assess sleepiness in a Swedish study of 10 children (two girls, eight boys) six to twelve years of age. They also used “deficits in attention, motor control and perception”, or “DAMP” as behavioral criteria, (p. 618.) They found somewhat shorter mean MSLT sleep latencies for the DAMP group (24.8 minutes) compared to controls (26.5 minutes), and found most DAMP children did not suffer from excessive sleepiness. However, three of the ten DAMP children were excessively sleepy, and had MSLT means of less than 20 minutes. Compared to the nighttime sleep of controls, the DAMP group had significantly longer sleep onset latency, longer awakenings and thus spent more time awake after the first sleep onset (p. 621). Reaction times on a CPT, stimulus identifying task were significantly slower in the DAMP group. The DAMP criteria used to define the group who had difficulty concentrating, short attention span, and clumsiness were somewhat different criteria than the DSM-IV. Palm, Persson, Bjerre, Elmquist and Blennow (1992). This study also used four 30 minute naps, a different protocol than the standard MSLT protocol commonly used in sleep research.

A related perspective for understanding the possible association of sleep and ADHD-like symptoms, comes from a neuropsychological perspective. Sleep is a vital and integral part of the catecholamine system, and in particular, the locus coeruleus, which is a major norepinephrine (NE) site crucial to the neurotransmitter "circuitry" of REM sleep (Ellman, 1985). NE and REM sleep have both long been implicated in alertness and arousal and their antithesis, sleepiness. There is evidence that suggests a disturbance in central NE mechanisms in some children diagnosed with ADHD (Shekim, Dekirmenjian, Chapel, Javoid and Davis, 1979). Palm et al (1992) suggests basal forebrain dysfunction in DAMP children, links the disorder to sleep. Yet, even without fully understanding the sleep-related neurological mechanisms underlying ADHD at the present time, it should prove useful to examine more fully the relationship between sleep, sleepiness and attention and impulsivity in ADHD children.

Daytime Sleepiness and its Measurement

Sleepiness is "a global condition that affects vigilance, attention, cognition, motivation, mood and performance" (Spielman and Herrera, 1991, p. 39). Carskadon and Dement (1987a, p. 307) describe how all daily activities are considerably influenced by the enormous range of daytime alertness/sleepiness, from "irresistible drowsiness...to optimal alertness with energy, clarity and optimism." The standard Multiple Sleep Latency Test (MSLT), is well-established as the primary tool sleep researchers utilize to quantify sleepiness (Carskadon and Dement, 1977). The MSLT is "a neurophysiological technique that quantifies drowsiness as the speed of falling asleep at intervals across a day [and] is used to identify patterns of sleepiness/alertness" (Carskadon and Dement, 1987a, p. 307).

The Multiple Sleep Latency Test (MSLT) is a gold standard for assessment of sleepiness in pre-adolescents (Carskadon & Dement, 1987b; Carskadon, Keenan & Dement, 1987). It is a clinical measure of daytime sleepiness, consisting of five nap opportunities of approximately 20 minutes each. The first nap is two hours after wake up time, and the naps continue every two hours throughout the day. In experienced hands, the recordings are a reliable tool for assessing and ruling out many sleep difficulties (Schaefer, 1992). In a recent report from the American Sleep Disorders Association (ASDA) on the clinical use of the MSLT, guidelines for use, scoring and indications list over seven uses for diagnosing sleep disorders from narcolepsy and insomnia to obstructive apneas (ASDA, 1992).

Summary

Sleep difficulties have been historically associated with ADHD clinically, and with research using parent reports. Although empirical NPSG results don't consistently support these subjective reports of sleep differences, the present study of ADHD children's sleep, will be the first to examine the impact of mild sleep loss on subsequent daytime sleepiness, or impaired alertness, and in turn, how it may affect attention and impulse inhibition.

CHAPTER THREE

METHODOLOGY

Subjects

Subjects were recruited in a number of ways. Most commonly, mothers and other relatives (aunts, cousins, etc.) of potential participants responded to flyers posted at City College of New York, and local parks. Also, through referrals from various sources such as friends and colleagues of the researcher. Potential participants were screened by initial phone contact with the parent. Those children who presented with many aggressive behaviors were excluded after this initial screening by phone (n=6). Next, parents and children came to the sleep lab and were fully briefed on the procedure. If they and their parents agreed to continue and the parent signed the subject consent form, participants and his parent were given an interview and various questionnaires to fill out, to determine ADHD behaviors and sleep histories for their child. If the child was determined by the DSM-IV criteria to be ADHD, or non-ADHD (control subjects) and if the child was low on the aggression and conduct scales, and if potential participants were able to lie down in the sleep lab for one Sleep Latency Test after having electrodes applied, and then complete an administration of the T.O.V.A.™, they were included in the study (n=20).

Most subjects were the children of students at City College of New York, or their relatives from the Washington Height and Harlem neighborhoods. Most were Latino or mixed ethnicity, and of middle and working class SES. Many of the children were bilingual, but all spoke fluent English. Many parents of the ADHD children sought evaluations for their children's behavioral or sleep difficulties. Many of the control children were age matched, non-symptomatic cousins and friends of the ADHD children. A \$50 fee for completing the study provided a powerful incentive to find participants, both ADHD and controls. This fee was funded by an 2 year NIMH grant awarded to the researcher by the City University of New York Health Psychology Concentration.

Potential participants were fully briefed on the procedure again after the evaluation day testing, and any questions were answered. All the boys also agreed to take ten naps

and the T.O.V.A.TM two more times. Parents and the children both agreed to the “up late” condition, staying up two hours past their average bedtime, as calculated by a sleep log they kept the week prior to sleeping in the lab.

All participants were between 8 and 12 years old. This age range was chosen because it represents the ages many children are initially diagnosed as having ADHD. Eleven non-aggressive ADHD children, and nine non-ADHD who met the selection criteria served as subjects. Ball and Koloian (1995) estimate that only about 100 ADHD children have participated in sleep studies. A group size of 11 represents a substantial number of ADHD children. With nine control subjects, the total sample size was 20. While the power of the experiment would be low with such a small sample size, it was hoped the effect size would be great enough to detect the hypothesized effects.

Determination of ADHD or Non-ADHD groups status was based on positive indications of pervasive difficulties with inattentiveness and/or hyperactivity, according to DSM-IV criteria (APA, 1994). All participants’ parents completed the DISC Disruptive Behavior Module structured interview. All participants were also rated as hyperactive or non-hyperactive as defined by minimum scores on the Conners Parent Rating Scale (CPRS-48) and the Child Behavior Checklist (CBC) Attention scale (Achenbach, 1991). They were part of a battery of measures administered to rule out the possibility of cognitive or neuropsychological functioning as sources of variation in the observed inattentive/impulsive behaviors (see next section for specific measures). This form of “blocking subjects” controls sources of error variance by reducing the possibility of sources other than the sleep loss intervention affected the results on the MSLT and the T.O.V.A.TM (Wiener, Brown & Michels, 1991, p.9).

Instruments

Specific Measurement Issues

The various constructs need to be examined carefully before choosing how to operationally define and measure them. The major difficulty measuring ADHD constructs is that they are complex, involving a number of neuropsychological, cognitive, emotional, and motivational factors that influence ADHD behaviors. Halperin, McKay, Matier and

Sharma (1994) have noted that Attention/Inattention, Activity/Hyperactivity and Impulsivity have several components that require separate neural tasks and capacities. Attention, for example, begins with noticing a stimulus. Cowan, (1988) calls the first cognitive element to come into play the orienting response. It is first an involuntary, then effortful and voluntary detection of a stimulus. This orienting response is a key component of attention. After becoming oriented to a stimulus, selective attention is the next constituent, and refers to the ability to focus on relevant information and ignore irrelevant stimuli (Cowan, 1988). Sustained attention is usually considered a separate component of attention and is more closely related to maintaining attention once something has been detected and selected (Halperin, et al., 1994).

Each of these components of attention are typically assessed using different instruments, all of which pose problems to the accurate measurement of attention. A variety of observational, or subjective techniques (usually parent or teacher rating instruments) can have problems such as poor inter-rater reliability and halo effects. More precisely, a negative halo effect occurs when a child is rated by parents or teachers as overactive or inattentive regardless of the actual levels of attention. (Abikoff, 1991; Schacher, et al., 1986; Halperin, et al., 1990b; van der Meere and Sergeant, 1988a; 1988b). This means that essentially subjective reports can be biased when parents or teachers see only expected, rather than actual behavior patterns. Observers rating research subjects should be "blind" to the child's ADHD status as much as possible. Yet despite the difficulties, parent ratings of their children's behavior, in either an interview or paper and pencil measure, represent many of the major instruments used to assess ADHD: the DSM Symptom Checklist (DISC); the Conners Parent/Teacher/Self Rating Scale-48 (CPRS-48) (Conners, 1989) and the Child Behavior Checklist (CBC) (Achenbach, 1991).

Objective techniques also have been used in an attempt to measure attention, focusing on various aspects of the construct. Attentional "capacity," it has been suggested, is assessed with the Wechsler Intelligence Scale for Children (WISC-III) subtests of Arithmetic, Coding and Digit Span (Wechsler, 1948). Neuropsychology tests can assess the ability to sustain attention with cognitive switching or vigilance tasks, also continuous performance tasks; or instruments such as the Stroop test (Stroop, 1935) are

also used to evaluate resistance to distractions (Halperin, 1994). Greenberg (1987) has developed the T.O.V.A.TM, a computerized vigilance task that measures inattention as errors of omission (not pressing when they see the target). Greenberg and Waldman (1993) have developed a database of normative data for the T.O.V.A.TM that shows age differences in attention, impulsivity, reaction time and variability, and errors that go down as the children get older, accurately reflecting normal developmental trends. The T.O.V.A.TM has also been shown to be sensitive to medication but CPT and clinical response is not correlated (Halperin et al, 1991).

Clearly more than a task of only selective and sustained attention, the T.O.V.A.TM draws upon cognitive processes as the children develop strategies to do better, or get bored. There are emotional processes as the child relates to, thinks about and interacts with, the tester who sits quietly behind, observing him. Also there are motivational qualities; the validity of the results depends to some degree on how willing the child is to try to do well, and keep going. They may be asked to take the test again for clinical purposes and so there can be fatigue effects, because the test is so boring. However level of compliance differs more or less with each child. Halperin, et al (1994) note that each of these tests and measures aspects of what can be typically considered "attention," but they also measure a number of cognitive processes as well. The DSM-IV notes that there are "no laboratory tests that have been established as diagnostic" for ADHD (APA, 1994, p. 81).

The construct of impulsivity, or, response inhibition, as with attention, has no universally accepted conceptual definition, and as a result there is no widely used operational definition. Factor analysis of behavioral rating scales used to assess impulsivity have revealed a two-factor solution, with items divided between inattention and hyperactivity factors (Healy, et al., 1987; Lahey, 1988). Impulsivity may be like attention, that is, a multi-faceted construct that cannot be measured with a single instrument or test (Olsen, 1989). Central to the construct, though, is the ability to withhold a goal directed response and delaying of gratification until an appropriate time (Halperin et al., 1994).

Besides the T.O.V.A.TM, instruments used to objectively measure impulsivity have included the Matching Familiar Figures Test (Kagan, 1966); Porteus Mazes (Porteus, 1965) and various delay tasks which attempt to index how long a child can inhibit a response. The construct validity of these tests has been called into question (Block, et al., 1986; Gjerde, Block and Block, 1985), that is, they have some degree of construct validity but often do not discriminate between impulsivity and closely related constructs. Halperin, Wolf, Greenblatt and Young (1991) and Halperin et al (1988) have attempted to establish a more direct and valid objective measure of ADHD constructs, through the use of a computerized continuous performance test (CPT). On the T.O.V.A.TM, impulsivity is measured as errors of commission (pressing when they see the non-target) (Greenberg, 1987).

Finally, defining and operationalizing activity is not as clear-cut as may seem at first. It is generally considered to be the amount of physical movement exhibited by an individual. High activity is often associated with aggressive or disruptive behaviors, but are considered to have distinct etiologies, as overactivity can be non-disruptive or non-aggressive, and children can be quite aggressive without being overactive. Hinshaw (1987) reviews studies which suggest overactivity is more strongly related to neurological based etiologies, whereas aggressive behaviors are more related to familial and environmental factors. Activity has also been found to be highly variable across situations (Porrino et al., 1983).

Activity is usually measured using subjective rating questionnaires (Conners, 1974) which may have ecological validity, but may not reflect actual levels of movement (Halperin et al., 1994). Direct coding (Abikoff et al, 1977) and playroom observation techniques (Milich et al., 1984; Routh et al., 1974) are known to be excellent rating systems, but can be prohibitively time consuming and require extensive training. Actigraphy has been used more recently, mechanical devices that record movement (Eaton, 1983; Porrino et al., 1983). Although becoming more accepted, these are sensitive to body placement and are very expensive.

Major Domains of Constructs and Measures Used

After considering these measurement issues, the following list of measures or instruments were used to collect data to both define the groups as similar on a number of important variables except for ADHD status, and to test the various hypotheses about group differences in sleep, sleepiness and performance on the T.O.V.A.TM. After listing them in major class sets, they will be described in detail.

1.) Measurement of ADHD behavior clusters: Inattention, Hyperactivity and Impulsivity

DSM-IV / DISC Structured Interview -- Disruptive Behaviors Module

Others instruments used to confirm the ADHD and Non-ADDH status:

Conners Parent and Teacher Rating Scales (Conners, 1989)

Test Of Variables of AttentionTM (Greenberg, 1987)

T.O.V.A.TM Observer Ratings (Greenberg, 1987)

2.) Measures of Sleep Variables

Nocturnal Polysomnography (NPSG)

Child Sleep Questionnaire (CSQ)

Child Sleep Log

3.) Measuring Daytime Sleepiness

Multiple Sleep Latency Test (MSLTs)

4.) Measures of Cognitive, School, and Neurological Functioning

Wechsler Intelligence Scale for Children-III (Wechsler, 1991)

Wide Range Achievement Test-III (Wilkinson, 1993)

Beery Test of Visual Motor Integration (Beery, 1982)

Stroop Test (Stroop, 1935)

Trail Making Tests (Parts A and B)

5.) Emotional and Behavioral Functioning

DISC structured Interview for DSM-IV Oppositional Defiant Disorder and Conduct Disorder Scales (APA, 1994)
Child Behavior Checklist (Achenbach, 1991)

Detailed Descriptions of Measures

1) ADHD constructs: Inattention, Hyperactivity and Impulsivity

DSM-IV Symptom Checklist (DISC)

Parents completed the Disruptive Behavior Module of the Diagnostic Interview Schedule for Children (DISC) for the DSM-IV. This allows diagnosis of ADHD, Conduct Disorders (CD) and Oppositional Defiant Disorders (ODD) according to DSM-IV criteria and standards (APA, 1995). If a child met ADHD criteria according to the DSM-IV checklist they were further distinguished into one of the subtypes of ADHD: Inattentive or Hyperactive-Impulsive. Aggressive children, (meeting CD or ODD criteria) were excluded. Excluding potential participants on the basis of aggressive behaviors increased subject homogeneity and reduced error variance.

Child Behavior Checklist (CBC)

This behavioral adjustment measure is becoming known as a “gold standard” for assessing child psychopathology because of its empirically derived categories, scales and standardization (Achenbach, 1991). One or both of the child’s parents, complete this 138 items behavioral report form. The “Attention Problems” subscale is of particular interest here, as is the “Behavioral Problems” subscale. Besides screening for emotional functioning in broader terms, this instrument has scales of attentional and aggressive difficulties to further support the DSM-IV diagnosis. It was also used as a broader measure of overall emotional functioning.

Conners Parent (CPRS-48) and Teacher Rating Scales (CTRS)

The 48 item version of the Conners CPRS-48 (Conners, 1989) characterizes the typical areas of difficulties or patterns of behavior in ADHD children, and in addition to an

Impulsivity- Hyperactivity subscale, it includes a 10-item Hyperactivity Index, which comprise the items most sensitive to medication. Other subscales include: Conduct Problem, Learning Problem, Psychosomatic, Impulsive-Hyperactive and Anxiety scales. The Teacher Ratings (CTRS) were given to parents and were intended to be used to add information about the child from another adult's perspective. Unfortunately, only 4 of 20 CTRS forms were returned so teacher data was not available and was not used. The CPRS-48 normative data is based on children 3 to 17 years old, separated by gender (Conners, 1989).

Continuous Performance Task-- The T.O.V.A.™ (Greenberg, 1987)

The T.O.V.A.™ is a 22.5 minute auditory, computer version of a continuous performance test that has minimal language demands and no left/right hand discrimination (Greenberg & Waldman, 1993). A target or a non-target is presented, in the first half, the target was presented on 22.5% of the trials, and on 77.5% in the second half. Normative data allows the T.O.V.A.™ to have standardized indices of omission and commission errors, response time means and standard deviations, as well as indices of anticipatory responses. The T.O.V.A.™ was used as a dependent measure intended to reflect several areas of cognitive functioning: sustaining attention, selective attention, impulsivity, reaction times, and variability of responses.

T.O.V.A.™ Observer Ratings

Included at the suggestion of the T.O.V.A.™ administration manual, as an explicit acknowledgment that the empirical data derived from the computer task must be seen and understood clinically, in conjunction with, rather than instead of, subjective interpretation of the child's behaviors. One observer was used, and often the observer knew the child's ADHD or Control group status because he administered the DISC interview to assess ADHD behaviors. Although not blind, these were administered to provide a behavioral assessment of T.O.V.A.™ performance, as suggested in the manual.

WISC Freedom from Distractibility factor Subtests

The Freedom from Distractibility IQ (FDIQ) subtests of the WISC-III were administered as part of the WISC, and used as a convergent measure to strengthen the validity of the definition as ADHD or non-ADHD. The subtests are: Digit Span and Arithmetic. These three subtests are useful for this purpose because they assess the ability to pay attention in several different ways. The child must listen to the tester carefully, remembering and manipulating both numerical and verbal information, and also must work on his or her own, scanning, and remembering numbers and symbols. Cautious use of FDIQ as a measure of attention is advised by Kaufman, (1979), however factor analytic studies of the two subtests, show they form a set of tasks that seem to depend heavily on the abilities to concentrate, and sustain attention while screening out distractions.

It is typical for children to score badly because of more distractible behavior, while they can't score really well just because they are less distractible (Kaufman, 1979), meaning the FDIQ reflects both behavioral and cognitive attributes. Digit Span, according to Kaufman (1979) reflects the abilities of short-term auditory memory, facility with numbers, and mental alertness. Arithmetic reflects the ability for computational skill, as well as school opportunities and learning, and ability to work under pressure.

2.) Measures of Sleep Variables

Nocturnal Polysomnography (NPSG)

Standard NPSG obtained numerous sleep recording variables to characterize sleep and make a formal sleep assessment of variables of sleep architecture, total sleep time and sleep continuity. The standard criteria of Rechtschaffen and Kales (1968) was the basis for electrode placement, recording and scoring. It is as follows: two central electroencephalogram (EEG) channels (C3 / C4 and O1 / O2), two horizontal electrooculogram channels(left and right outer canthus, LOC/ROC, each referred to A2). Two electromyogram (EMG) channels were used, three placed submentally, and four were placed on the left and right anterior tibialis of each leg. All electrophysiological parameters were recorded using silver chloride disk electrodes filled with electrode gel or creme and affixed with either tape or creme-soaked gauze (for EEG placements). Heart

rate (ECG) and oxygen saturation (O₂) in the blood were recorded. The clinical screening night (the first night) included air-flow measures to detect respiratory events.

Child Sleep Questionnaire (CSQ)

The Child Sleep Questionnaire is a parent interview and observer rating measure developed for this study, designed to get an initial qualitative assessment of important sleep variables such as the quantity, quality, and regularity of a child's sleep. It uses a structured parent interview format, as well as visual analog self report scales for the parent to complete, to assess a number of sleep qualities such as: sleep onset latency, total sleep time, sleep-onset difficulties, sleep-maintenance difficulties, and daytime levels of sleepiness and alertness. Post-hoc analysis was expected to reveal which items or questions are most predictive of poor sleep and excessive daytime sleepiness.

Child Sleep Log

This is a self report measure designed specifically to assess the daily cycle, quantity, quality, and regularity of a child's sleep. Hopefully, the sleep log is a more objective, quantified measure than the more subjective sleep questionnaire, of sleep onset latency, total sleep time, sleep-onset disturbances, sleep-maintenance disturbances, and daytime levels of sleepiness and alertness.

3.) Measuring Daytime and Nocturnal Sleepiness

Multiple Sleep Latency Tests (MSLT)

The usefulness, construct validity and reliability of the MSLT has long been established for measuring sleepiness in both adults, adolescents and preadolescents, as determined as sleep propensity, or the number of minutes it takes to fall asleep during a nap opportunity (ASDA, 1992; Carskadon and Dement, 1987; Carskadon, Keenen and Dement, 1987).

The following description represents the study's MSLT protocol. The subject is given five 25 minute long nap opportunities to go to sleep on both Friday and Saturday (before and after the sleep loss). Standard protocol for MSLTs are 20 minutes in length,

however for this study, with children who should be quite alert during the day, the nap length was lengthened to 25 minutes to avoid a ceiling effect and increase any likelihood the children would fall asleep. For each trial, called a "nap" (whether or not the subject has a sleep episode, and falls asleep), the subject lies down in a dark bed chamber, and is asked to "go to sleep" until the research technician returns. The subject is not given a specific time period for the nap, but if necessary, will be reassured that there will be substantial time to get comfortable and fall asleep. Before each nap, electrode impedance's are checked to ensure that the PSG recording will be adequate. After a brief series of movements (look straight ahead, look left, right, etc.) that serve for a final calibration of the polygram, the lights are turned off. The child is told to try to fall asleep and PSG recording begins. The "nap" is terminated after one of the following conditions is met: Once the subject has clearly fallen asleep (Stage 1) and has accumulated 1.5 minutes of sleep; (a Sleep Episode) or, if the subject has half of a minute of any other stage of sleep (Stage 2, 3, 4 or REM.) If the child didn't fall asleep, Sleep Latency is considered 25 minutes for that nap. After answering a few questions, the subject is allowed to move around freely, eat, read, play, etc. between naps.

4.) Measures of Cognitive, School, and Neurological Functioning

The Wechsler Intelligence Scale for Children-III (WISC-III)

The WISC-III (Wechsler, 1991) is a widely used, valid and reliable clinical and research instrument used to assess cognitive functioning. Children participating in the study must be capable of understanding and then following fairly complex instructions, so any child scoring below 70 FSIQ was excluded from the study. No participant was excluded for this reason. As previously described, certain subtests of the WISC-III will also be used to assess the factor of Freedom from Distractibility, revealed in factor analysis (Kaufman, 1979).

The Wide Range Achievement Test 3 (WRAT-III)

The WRAT-III (Wilkinson, 1993) is a measure of the basic skill levels of reading, writing and arithmetic. Three scales, Absolute Scores, Standard Scores, and Grade

Scores are provided to compare children and adults achievement levels on these recently restandardized tests (Wilkinson, 1993). In addition to restandardization, the scales were designed in 2 alternate forms (Blue and Tan). The WRAT 3 was used to look at the resiliency of school learned material and long term memory function.

Beery Test of Visual Motor Integration

The Beery Test of Visual Motor Integration (Beery, 1982) is a better normed instrument of VMI than the Benton, and was used as a measure to determine gross level of integrated visual and motor abilities, planning ability, pen and pencil skills, and similar cognitive tasks. The Beery is standardized, easy to administer, and relatively fast (about 15-20 minutes.) It has age normed visual scoring criteria that allows for very specific interpretation of the drawing's components.

Trail Making Test

Trails Tests is a popular neuropsychological screening tool, originally part of the Army individual Test Battery, and later included by Retina in the Halted Battery. The intermediate form is usually administered to 9 to 14 year old children. The test is composed of two timed subtests. Part A requires the child to connect 25 numbers in circles, randomly placed on a page, in the correct numerical sequence. Part B requires connecting 25 circles with both letters and numbers, in alternating order. This requires switching cognitive sets, back and forth from numbers to letters in parallel sequences: #1, then letter "A", then #2 and then the letter "B" and so on. It was included and used as a measure intended to reflect several areas of neurological and cognitive functioning. In Part A, children's ability to plan, anticipate and solve problems is reflected. In Part B, they must do the same, but it also requires cognitive flexibility to switch "sets" (from numbers to letters). It has also been used as a measure of development of appreciation for the symbolic importance of numbers and letters (Erickson, 1992), as well as planning ability while sustaining attention, visual scanning ability, and pencil and paper skill.

The Stroop Test (Stroop, 1935)

The Stroop Test is a task that again demands the cognitive ability of the subject in ignoring distractions and sustaining concentration on a task that involves some cognitive dissonance. The subject has to read words, name colors and name colors in an interference condition. It was used as measure intended to reflect several areas of cognitive functioning: sustaining attention, selective attention, impulsivity, processing speed, visual scanning. Barkley, Grodzinsky and Dupaul (1992) in a review of ADHD neuropsychology testing literature found that in five of six studies, the ADHD groups were more impaired than normal children, especially in the interference condition. The authors suggest this indicates the Stroop “may be sensitive to frontal lobe impairments hypothesized to exist in ADD/+H” (p.169).

5.) Measures of Emotional and Behavioral Functioning

DSM-IV (DISC)

Parents reported aggressive behaviors in the DISC interview as well. DSM-IV Oppositional and Conduct criteria behaviors were assessed.

Child Behavior Checklist (CBC)

Besides having an Attention and Disruptive Behavior Problem Scales, the CBC (Achenbach, 1991) can be useful for the assessment of general emotional functioning. Social competency, school and activity subscales, as well as scales for mood, anxiety and other problems allow the researcher a normed estimate of a wide range of the child’s psychological and interpersonal strengths and weaknesses.

Procedure

The study proceeded in three phases. The first phase was an evaluation of potential participants to determine ADHD group status. Phase two was a screening and adaptation night, and the third phase is the sleep loss intervention.

Phase One: Evaluation

After a brief phone screening, the screening process continued when the child and at least one parent came to the Sleep Disorders Center a week or two before sleeping there (n=22). After expressing an interest in participation, an appointment was scheduled, and potential participants came to City College and completed the following instruments:

- Parent:** DISC (Disruptive Behaviors Module)
CBC (Achenbach, 1991)
CPRS-48 (Conners, 1989)
Child Sleep Questionnaire
- Child:** WISC-III (Wechsler, 1991)
(1) Sleep Latency Test (Evaluation Day Nap)

These were used to determine the characteristics of the subjects and to assign them to the ADHD or non-ADHD groups on the basis of the parent's responses to the DISC, and the assignment of the DSM-IV diagnosis or not. The other measures were used to assess the validity of the diagnosis. Subjective reports of behaviors indicating attention, impulsivity and activity were evaluated with the Child Behavior Checklist parent, (and teacher reports, if possible). The parents completed the CPRS-48. Besides these parent report scales, a Children's Sleep Questionnaire was developed for this study to assess the sleep characteristics and level of sleep disturbance. Children's levels of cognitive functioning were assessed with the WISC-III to ensure that low IQ did not confound understanding instructions or completing the various psychomotor tasks. Sleep Logs were then explained to the participants and their parents and were kept for at least one week, to assess daily patterns of sleep before coming back to the center. After the evaluation phase, children were placed in one of two groups. 1) Inattentive, Hyperactive-Impulsive and Mixed type of ADHD children, who all had clinically significant levels of ADHD behaviors and were non-aggressive (that is, had a low number of ODD and CD behaviors) were designated to this group. 2) Non-ADHD, with fewer than clinically significant numbers of behaviors. Overall, the control children were non-symptomatic, with 7 of 9 parents reporting fewer than three behaviors. If subjects had aggressive difficulties

reported they would have been excluded from the study, but no one evaluated after initial phone screening had to be excluded for this reason

This evaluation day also included one Sleep Latency Test. It was important to facilitate adaptation to the laboratory conditions, so the child had some electrodes attached in standard placements, and experienced one twenty-five minute Sleep Latency Test. Two subject did not want to go through this process and were excluded from the study ($n = 2$).

Phase Two: Night One and Day 1 in the Sleep Lab

Having the child and parent return to CCNY Sleep Lab for the first night served several purposes: Night 1's nocturnal polysomnography (NPSG 1), was used to determine if potential subjects had any sleep disorder, and also to help them adapt to the lab's research equipment, instrumentation, procedures and bedrooms; also to provide some basis for comparison to post sleep loss sleep. Friday and Saturday night bedtimes were calculated from the sleep logs that participants kept from the previous week. Participants went to bed at their average bedtime for the week and got up Saturday morning at their average weekly wake-up time. Children, as with non-excessively sleepy adults, usually experience only slight difficulty sleeping in the lab on the first night, and seem to adapt after one night.

During the first day, on Saturday, children were tested in the domain of daytime sleepiness using the MSLT, and for levels of attention, impulsivity and cognitive functioning, were assessed with the T.O.V.A.TM. Studies have shown normal preadolescent children respond to the first night in the lab, with some altered sleep patterns but children seem to adapt after one night (Carskadon, Keenan & Dement, 1987), and their sleep is robust enough that they don't seem to have serious subsequent decrements in alertness.

Phase Three: Night Two and Day 2: The "Up Late" / Sleep Loss Condition

On Saturday night, all participants stayed up two hours past their weekly average bedtime, but were awakened in the morning at the same time as they woke up the previous

morning, resulting in a sleep loss of two hours. The impact of the two hour sleep loss intervention on the domains of subsequent daytime sleepiness and alertness, and its consequent impact in attention and cognitive functions was assessed using the MSLT and the T.O.V.A.TM.

Apparatus

The experiment was performed in the City College of New York Sleep Disorders Center. The sleep rooms are about ten feet by twelve feet, electrically shielded and sound attenuated. They are equipped with a twin bed, and a night table and a night light. Two model 78 D, 14-channel Grass polysomnographs were used to record all electrophysiological parameters simultaneously.

Two SSM-920 Sony TV low illumination video cameras were used for the continuous monitoring of the subject during sleep sessions. A Resptrace monitor, and a custom-built communication system connected to the sleep room were used to monitor and communicate with the subject. Time was recorded with the same VCR digital clock, used as a standard.

CHAPTER FOUR

RESULTS

Describing the Groups

The participants in the study who met the selection criteria were twenty pre-adolescent boys between eight and twelve years of age ($n = 20$, mean age 9.73 years). The ADHD group was about seven and a half months older, but Table 1 shows this age difference was not statistically significant (ADHD group mean 10.01 years old; non-ADHD group mean 9.38 years old, NS). All were attending school as follows: Third Grade ($n = 7$); Fourth Grade ($n = 8$); Fifth Grade ($n = 4$); or Sixth Grade ($n = 1$). Seventy percent of the sample were Dominican-American boys ($n = 14$), reflecting the Washington Heights and Harlem communities of New York City where subjects were recruited. The other 30% were of other ethnic heritages: White ($n = 1$); African-American ($n = 2$) and Mixed Ethnicity ($n = 3$). (Recruitment is described in the Method section.) All children spoke fluent English, some of the boys were Spanish-dominant bilingual speakers as well. In total, 32 parents contacted researchers. Six potential participants were excluded by initial phone contact and 26 potential participants came to the sleep lab for the evaluation day measures. Those that were excluded from the study were as follows: Four potential participants ($n = 4$) were excluded for having more than five total ODD and CD (aggressive) behaviors. Two potential participants ($n = 2$) decided not to participate after the preliminary Sleep Latency Test, leaving 20 total number of participants in the study. Of the 20 participants, all completed the study.

On the basis of parental responses to a DSM-IV structured interview, participants were classified into two groups, **ADHD** ($n = 11$) or **Non-ADHD** ($n = 9$). Potential participants were first designated as ADHD if their parents reported six or more of the nine Inattentive behaviors, and/or at least six of the nine Hyperactive and Impulsive behaviors. Criteria behaviors had to be seen at home and at school, and the children also had to meet all other DSM-IV criteria (ADHD criteria behaviors are listed in the DISC Summary Sheet in Appendix B).

All participants were further described as **Non-aggressive** or **Aggressive**. “Aggressive” behaviors were defined as the total number of Oppositional Defiant Disorder (ODD) and Conduct Disorder (CD) criteria parents reported. Potential volunteers who presented more than five of the eight DSM-IV criteria for ODD or more than three of the fifteen CD criteria were excluded from the study. For this study, a decision was made to limit the number of ODD and CD behaviors, for both practical considerations and also to focus on non-aggressive ADHD boys to limit the number of independent variables under investigation.

Table 1 shows the results of comparing the groups using a DSM-IV structured interview to assess ADHD and “aggressive” behaviors. While the two groups differed significantly in mean number of ADHD behaviors, with the ADHD group exhibiting, on average, clinically significant levels of criteria behaviors. T-tests comparing the scores indicated that the parents of the ADHD children reported significantly more Inattentive behaviors (ADHD group mean = 6.2 behaviors; non-ADHD mean = 1.0 behaviors, $t(18) = -4.55, p < .001$). Means were also higher for Hyperactive-Impulsive behaviors (ADHD group mean = 6.1 behaviors; non-ADHD mean = 1.3 behaviors, $t(18) = -5.33, p < .001$) than the parents of the non-ADHD children. The total possible number of behavioral criteria listed in the DSM-IV for each disorder. The groups were clearly distinct, but the ADHD group had moderate difficulties, due to the study’s focus on non-aggressive type ADHD. By excluding potential participants with over five aggressive symptoms many more severe ADHD children were excluded from the study.

Table 1 shows the ADHD group had a larger mean number of ODD and CD behaviors (ADHD group mean: 3.45; non-ADHD group .67, $p < .05$). Because subjects with more than five total ODD and CD behaviors were excluded from the study, between group differences were not expected to be significant. Even given this restriction, the ADHD children were reported by parents to have significantly more aggressive behaviors ($t(18) = -6.08, p < .001$).

Table 1

Mean DSM-IV behaviors reported by parents on structured interview.

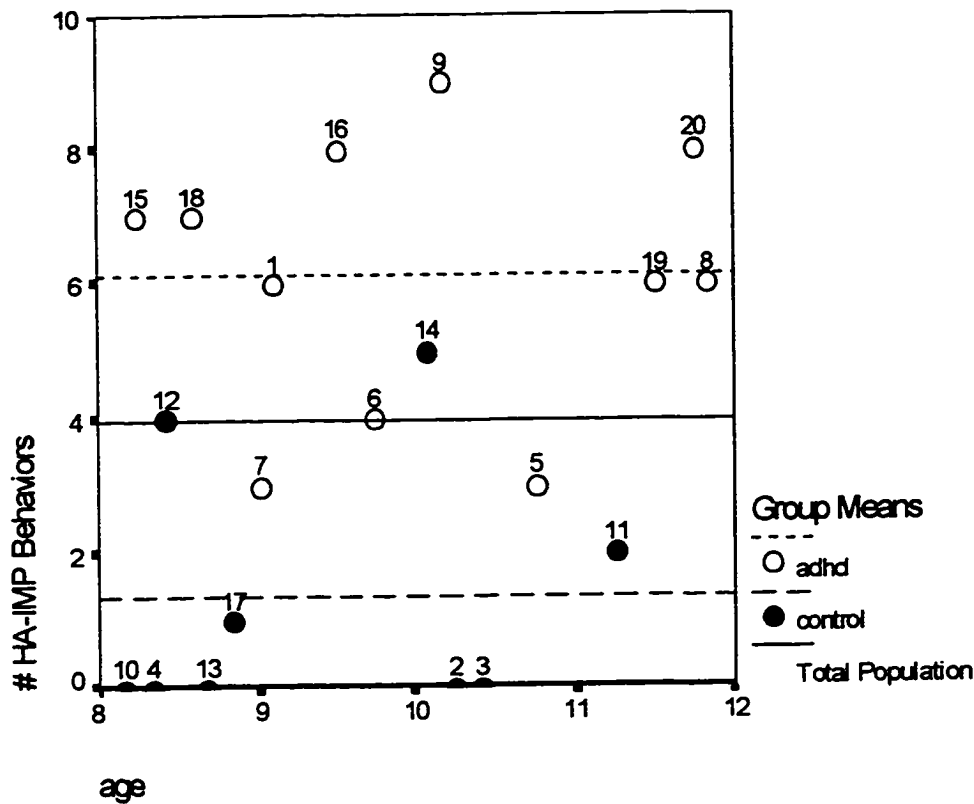
Variable	ADHD (n=11)		Non-ADHD (n=9)		t	df	Sig. of t (2 tailed)
	Mean	SD	Mean	SD			
Age in yrs: (n = 20) Mean 9.73 SD 1.23	10.01	1.3	9.38	1.1	-1.17	18	.26 NS
<u>DSM-IV Behaviors</u> (number of behaviors)							
Inattentive (9)	6.2	3.1	1.0	1.7	-4.55	18	.0001
Hyperactive/Impulsive (9)	6.1	2.0	1.3	1.9	-5.33	18	.0001
Total ADHD behaviors (18)	12.3	3.2	2.3	3.3	-6.80	18	.0001
Oppositional Defiant (8)	2.5	0.7	0.4	0.7	6.34	18	.0001
Conduct Disorder (15)	1.0	0.8	0.2	0.4	2.67	18	.016
Total "Aggressive" Behaviors (23)	3.5	0.9	0.7	1.1	-6.08	18	.0001

To examine these important differences in behaviors more closely, Figures 1 through 4 show scatterplots of individual participants by age and DSM-IV behaviors. These figures describe the differences between the groups, with most of the non-ADHD children having low numbers of these behaviors reported by parents, and most of ADHD having a high number of difficulties.

Figures 1 through 4 also show each child's status as ADHD or non-ADHD, and each individual is labeled by his case number.

Figure 1

Number of DSM-IV Hyperactive-Impulsive Behaviors (HA-IMP)



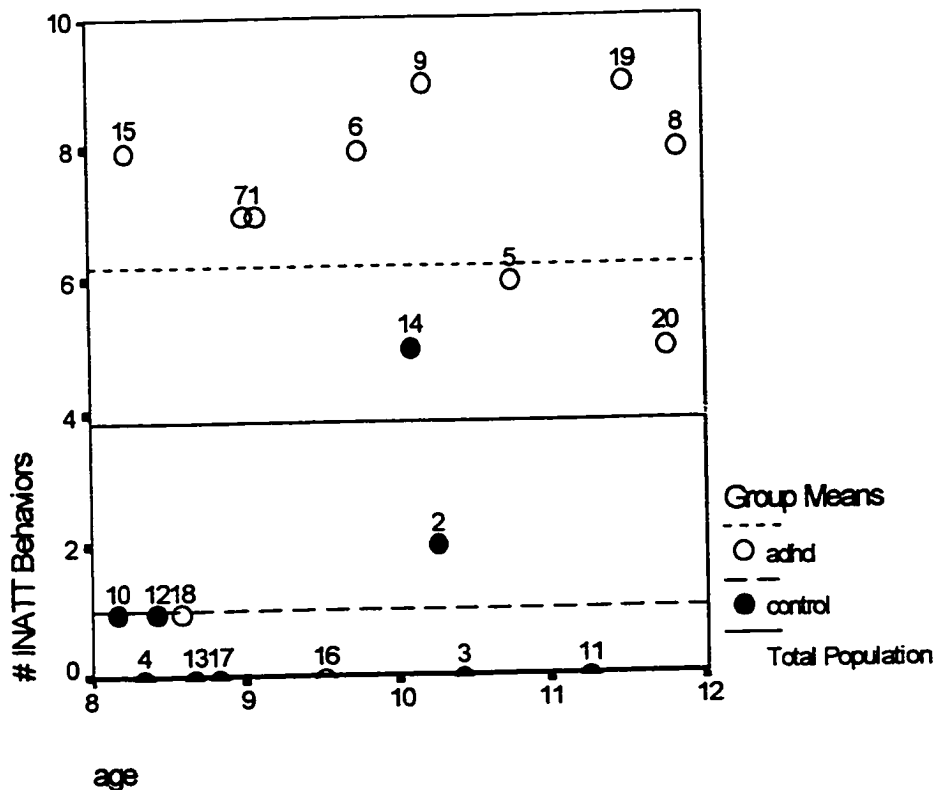
In Figure 1, the x-axis shows the distribution by age, and the y-axis shows the number of Hyperactive-Impulsive behaviors from the DISC structured interview with a parent. Here, the highest total possible score was nine, for the nine behaviors that could be seen at home or at school. We see that the parents of the non-ADHD group clearly reported many fewer hyperactive behaviors. Eight of the eleven ADHD children had six or more Hyperactive/Impulsive behaviors reported, while seven of nine non-ADHD children had two or less. We can see that there was about equal variance within each group (ADHD range = 6; non-ADHD = 5). The ADHD group is mostly clustered in a higher

range of Hyperactive-Impulsive behaviors, around their significantly higher mean of about six behaviors. The controls are mostly clustered below their mean of about two behaviors. We can also see that the ADHD group averaged just enough behaviors to be classified as ADHD, representing mild to moderate degree of clinical impairment.

In Figure 2, Inattentive Type behaviors are similarly presented, and the groups are also clearly differentiated. The highest possible number of inattentive criteria parents could report was also nine, for the nine behaviors seen at home and/or at school. Here we see eight out nine controls had two or fewer behaviors, and eight of eleven ADHD children had six or more. As with the Hyperactive/Impulsive behaviors, there were clinical levels of mean ADHD behaviors, but they indicate a moderately impaired ADHD group.

Figure 2

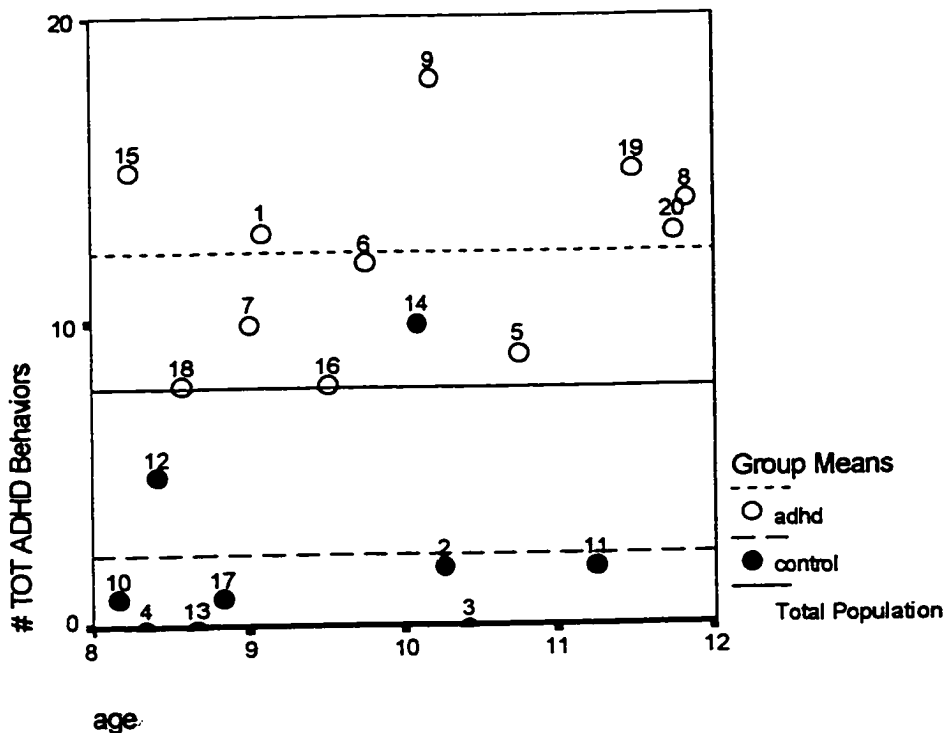
Number of DSM-IV Inattentive Behaviors (INATT)



The combined number of all ADHD behaviors reported, out of a total score of 18 are presented in Figure 3. Here, we see the groups are clearly different. Although one non-ADHD participant (#14), had ten total ADHD behaviors, they were not significant in either domain of ADHD. Additionally, his mother reported him having difficulties only with one teacher, and she saw no developmentally inappropriate difficulties impairing social or cognitive functioning at home. Therefore, he did not meet the DSM-IV ADHD criteria of having pervasive behavioral difficulties both at school and at home and was assigned to the control group.

Figure 3

Total Number of DSM-IV ADHD Behaviors (TOTSX)



In Figure 4, the total number of “aggressive” (ODD and CD behaviors) behaviors parents reported in the interview are shown. Here, again we see the clear distinction between the groups on this dimension of behavior. Six of the nine children of the control group had no aggressive behaviors at all. In contrast, parents of 10 of 11 ADHD children reported more than three or more ODD and CD criteria.

Figure 4

Total of DSM-IV Aggressive (ODD+CD) Behaviors (AGGRSX)

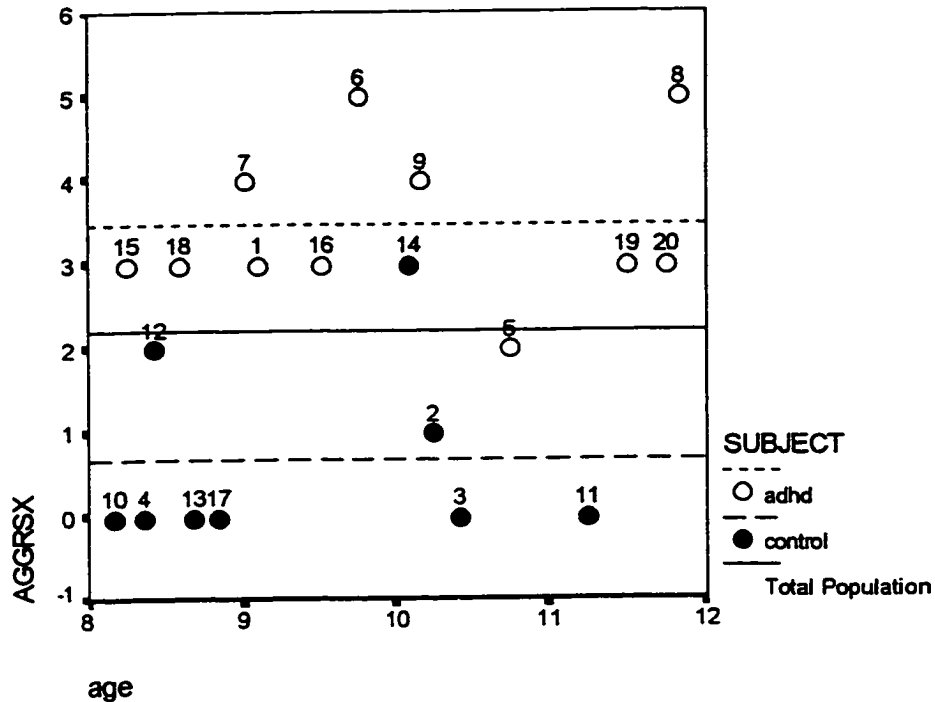


Table 2 shows the results of comparisons of two widely used questionnaires parents completed, the 48 item Conners Parent Rating Scale (CPRS-48) and the Child Behavior Checklist (CBC). Both are commonly used for child assessment in research and describe a broader range of difficulties than the structured interview and other measures used. Scores for both instruments presented are \bar{X} scores, with a mean of 50. A score of 70 on both measures is significantly deviant from the standardized mean. Group means, standard deviations (SD), degrees of freedom (df) and the significance of t are shown.

Table 2 shows that on the CPRS-48, the parents of the ADHD group reported significantly more difficulties. The results of the independent groups t -tests are presented here as well. While the ADHD means are below “clinically significant” means (i.e. >70) the ADHD group has significantly higher numbers of symptomatic behaviors.

Table 2

Mean Behavior Difficulties Reported By Parents On Questionnaires

Variable	ADHD (n=11)		Non-ADHD (n=9)		t	df	Sig. of t
	Mean	SD	Mean	SD			
Conners PRS-48							
Scale A: Conduct	53.6	8.4	41.6	7.1	3.47	18	.003
Scale B: Learning	62.6	10.8	39.8	5.4	6.11	18	.0001
Scale C: Psychosomatic	62.0	18.5	47.1	7.8	2.25	18	.037
Scale D: Impulsive/Hyp.	58.9	10.3	38.7	3.0	6.22	18	.0001
Scale E: Anxiety	54.0	7.5	49.4	7.9	1.32	18	.20 NS
Scale F: HA Index	63.7	8.3	39.3	6.4	7.41	18	.0001
Child Behavior Checklist							
Scale 4: Social Problems	63.7	11.2	54.0	6.2	2.46	18	.026
Scale 5: Thought	59.5	6.3	53.1	6.2	2.26	18	.037
Scale 6: Attention	68.1	8.6	53.1	6.5	4.41	18	.0001
Scale 8: Aggressive	59.0	6.4	52.4	3.3	2.97	18	.01
Internalizing T Scores	60.6	17.2	51.9	12.7	1.29	18	.21 NS
Externalizing T Scores	60.3	7.3	43.4	13.9	3.28	18	.007
Total Items	45.6	21.9	20.0	18.8	2.81	18	.013
Total Score	59.7	18.3	48.3	14.6	2.48	18	.024

As expected, the two groups differed significantly on a number of these Conners factors. In fact, they were not different on only one scale, for Anxiety. The ADHD group was rated as having significantly more difficulties on the scales for Conduct ($t(18) = 3.47, p < .01$), Learning ($t(18) = 6.11, p < .001$), Psychosomatic ($t(18) = 2.42, p < .05$) and Impulsive-Hyperactive problems ($t(18) = -6.22, p < .001$). The group means for Scale F, the Conners “Hyperactivity Index”, was also significantly higher in the ADHD group ($t(18) = 6.22, p < .001$).

On the Child Behavior Checklist (CBC) parent questionnaire, the ADHD group again had significantly higher scores from non-ADHD on a number of scales. Table 2 shows the T-scores and their significance levels for comparisons of the CBC are

presented here. CBC Scale VI, Attention, as well as for, Social problems (IV), Thought problems (V), and Externalizing I scores were all significantly higher in the ADHD group. There was no difference on scales for Withdrawn (I); Somatic complaints (II), Anxious/Depressed (III), Delinquent behavior (VII), and Internalizing I .score.

Table 3 shows the means, standard deviations, and results of the t-tests comparing the groups on the measures administered to determine levels of cognitive and neuropsychological functioning, and academic achievement. The ADHD group scored significantly lower, at the $p < .05$ level of certainty, for WSIC-III Full Scale IQ, Verbal IQ, and Verbal Comprehension IQ. Interestingly, there was no difference between group means for the Freedom from Distractibility IQ factor (ADHD group mean, 98.60; non-ADHD group, 108.56, NS). There were no differences between the groups on performance tasks, for either the Performance IQ, or the Perceptual Organizational IQ. The results of the WISC-III "ACID" sub-scale described in the Method section on measures shows the ADHD group had a significantly lower mean on these WISC-III subtests than the non-ADHD group.

The Beery data presented in Table 3 shows the ADHD group performed much worse than the non-ADHD group on this visual-motor integration task of copying geometric forms. Table 3 shows the ADHD group mean was significantly lower, in fact was less than half the non-ADHD mean score. The ADHD group had significantly lower raw and their percentile rank in the general population was just under the 13th percentile compared to about the 56th percentile for the non-ADHD group.

Table 3 shows the group means differed significantly on a number of WRAT III factors as well. On this task of school achievement, the ADHD group scores reflected a consistently lower level of school performance means for Reading, Spelling, and Arithmetic. As Table 3 also shows, the non-ADHD boys were performing about a half grade above their present grade level in school, while the ADHD group was almost 3½ grades below level.

Table 3
Means, Standard Deviations and Univariate Analysis Results of Demographic Variables

Variable	ADHD Mean	(n=11) SD	Non-ADHD(n=9) Mean	SD	t	df	Sig. of t (2-tailed)
<u>WISC-III</u>							
FSIQ	98.6	11.0	113.8	10.7	-3.11	18	.006
VIQ	101.8	15.5	116.6	7.7	-2.77	18	.014
PIQ	95.5	11.7	107.0	13.7	-2.01	18	.06 NS
VCIQ	94.7	12.6	106.8	7.7	-2.72	18	.015
POIQ	98.6	10.8	108.6	15.3	-1.74	18	.10 NS
FDIQ	90.9	15.7	98.0	10.6	-1.21	18	.24 NS
ACID Profile	8.1	2.2	10.0	1.43	-2.34	18	.031
<u>WRAT-III</u>							
Reading	91.0	22.4	106.4	8.3	2.12	18	.05 NS
Spelling	89.7	19.4	105.8	27.2	2.29	18	.035
Arithmetic	82.6	27.3	105.9	9.1	2.63	18	.021
<u>VMI / Beery</u>							
Scaled Scores	4.9	2.3	10.3	2.3	-5.26	18	.001
Percentile	12.5	10.4	55.8	27.2	-4.52	18	.001
Age difference	-3.4	1.8	0.5	1.9	-4.76	18	.0001
<u>Stroop (Raw scores)</u>							
Word Reading	62.1	12.8	69.4	14.2	-6.31	18	.53 NS
Color Naming	41.6	7.8	55.8	27.2	-1.20	18	.24 NS
Interference	22.7	5.0	23.8	6.4	-.401	18	.69 NS
<u>Trails (Raw Scores)</u>							
Part A	54.7	7.3	54.8	20.4	-.025	18	.98 NS
Part B	189.1	61.5	131.7	64.3	2.03	18	.06 NS

Although the ADHD group had significantly lower scores on the Beery visual motor integration task, they did not differ in terms of the other neuropsychological measures administered. The Stroop interference condition reflects skills in a task of selective attention in word reading and color naming; and the Trailmaking task reflects planning, anticipation and problem solving skills. There were no differences in tasks reflecting neuropsychological functioning of participants. Table 3 shows the raw scores

for the Stroop scores for Word Reading, Color Naming, or for the Interference condition, and also, for Trailmaking Part B. The results showed that mean group times to complete the task were not significantly different.

Statistical Analysis of Sleep Measures

Table 4 presents the means, standard deviations (SD), F values, degrees of freedom and p values for two nights of sleep variables using a Repeated Measures MANOVA procedure to compare the groups across two nights of NPSG recordings. Nocturnal polysomnography (NPSG) variables were analyzed for the between groups comparison "Subjects" main effect (ADHD $n=11$; non-ADHD $n=9$), for the "Time" main effect within groups comparison all participants ($n=20$) across the two nights of NPSG recordings and for the "Subject by Time" interaction effect.

There were no differences between the ADHD and non-ADHD on any of the four sleep variables (Subject main effect). In contrast, there were several significant differences between Night 1 and Night 2 (Time main effect). Table 4 shows that Time In Bed (TIB), Total Sleep Time (TST) and Total Wake Time (TWT) were all significantly lower on the second night as expected. The experimental intervention essentially deprived participants of two hours of sleep by keeping them up two hours later on the second night.

Table 4 also shows that there was one significant interaction effect. There was a significant Subject by Time interaction [$F(1, 18) = 7.41, p < .01$]. Univariate results reveal that for non-ADHD children, percentage of Stage 1 sleep decreased on Night 2, while the ADHD group showed a significant increase. Here, the ADHD group's response to two hours of sleep loss is the opposite of what was expected.

Although none of these subject differences were significant, in contrast, most of the time differences were significant or close to significant (see Table 4). Figures 5 through 7 graphically present sleep data on Night 1 and Night 2.

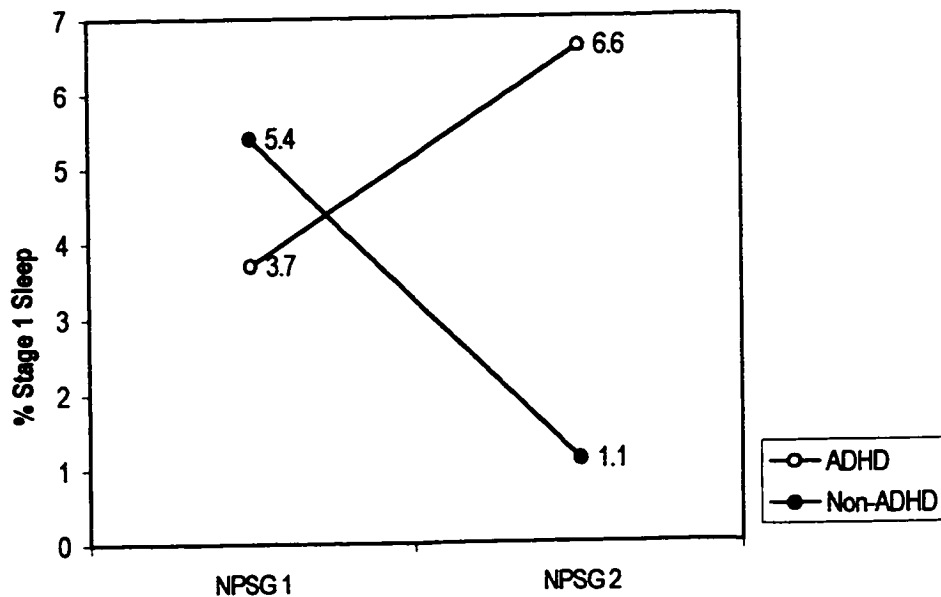
Table 4
Means, Standard Deviations and Multivariate Analysis for Night 1 and 2 Sleep Variables

Source Variable	ADHD (n = 11)		Non-ADHD (n = 9)		F_{Subject}	df	Sig. of $F_{\text{Subj.}}$
	Mean	SD	Mean	SD			
Subject Effect					All comparisons were NS		
Time Effect (Values below represent % of TST)					F_{Time}	df	Sig. of F_{Time}
TIB Night 1	575.3	37.1	592.5	49.1	139.64	1,18	.0001
TIB Night 2	469.1	52.3	466.3	57.0			
TST N1	522.8	50.0	514.3	41.9	26.75	1,18	.0001
TST N2	450.3	45.7	455.9	52.9			
TWT N1	58.1	47.5	77.6	52.6	22.52	1,18	.0001
TWT N2	18.6	14.4	10.9	9.0			
SOL N1	9.7	8.4	13.0	11.5	5.52	1,18	.03
SOL N2	3.8	3.0	7.8	7.0			
(Values below represent % of TST)							
% SE N1	90.0	8.1	87.2	8.0	19.8	1,18	.0001
% SE N2	96.1	2.9	97.7	1.7			
% Wake N1	9.5	9.7	12.7	10.6	10.62	1,18	.004
% Wake N2	4.1	3.2	2.4	1.9			
% Stage 1 N1	3.7	2.7	5.4	1.1	.30	1,18	.59 NS
% Stage 1 N2	6.6	7.6	1.1	0.8			
% Stage 2 N1	50.3	10.0	45.0	9.6	6.90	1,18	.07 NS
% Stage 2 N2	39.4	10.3	42.3	10.4			
% Stage 3 N1	10.3	4.0	11.6	6.7	.78	1,18	.39 NS
% Stage 3 N2	14.2	5.4	10.1	2.4			
% Stage 4 N1	16.0	5.9	21.3	6.7	16.28	1,18	.001
% Stage 4 N2	23.0	10.4	27.0	9.3			
% SWS N1	27.7	8.2	32.8	10.9	8.69	1,18	.09 NS
% SWS N2	37.6	11.0	37.4	11.0			
% REM N1	20.7	5.6	20.3	4.8	3.48	1,18	.08 NS
% REM N2	16.5	6.8	18.7	8.3			
Subject x Time Effect					F_{SxT}	df	Sig. of F_{SxT}
% Stage 1					7.41	1,18	.01
% Wake					1.06	1,18	.32

The significant interaction effect found for Stage 1 % on Night 2 is shown in Figure 5. Percentages of wakefulness and Stage 1 sleep, a transitional stage from waking to sleeping reflects how well, or poorly participants are falling and staying asleep. The Subject by Time interaction effect found for Percentage of Stage 1 sleep was caused by the ADHD group's higher percentage than the non-ADHD group on the second night.

Figure 5

Percentage of Stage 1 Sleep During Night 1 and Night 2 for Both Groups



In the non-ADHD group, Stage 1 percentage decreased on Night 2. A decrease in percentage of Stage 1 was anticipated as a result of the sleep loss condition. It was expected to make the children sleepier, and therefore fall asleep faster, and stay asleep more of the time they were in bed. The increase in ADHD percentage Stage 1 on Night 2, along with the higher mean percentage of wakefulness relative to the non-ADHD control group, suggests a less adaptive response to sleeping in the sleep lab.

Figure 6

Percentage of wakefulness during Night 1 (WP1)

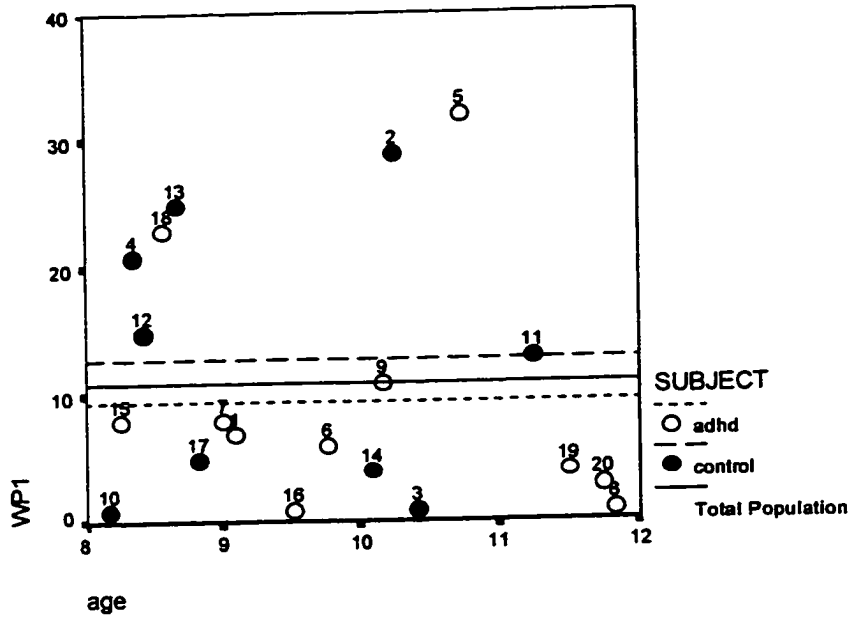
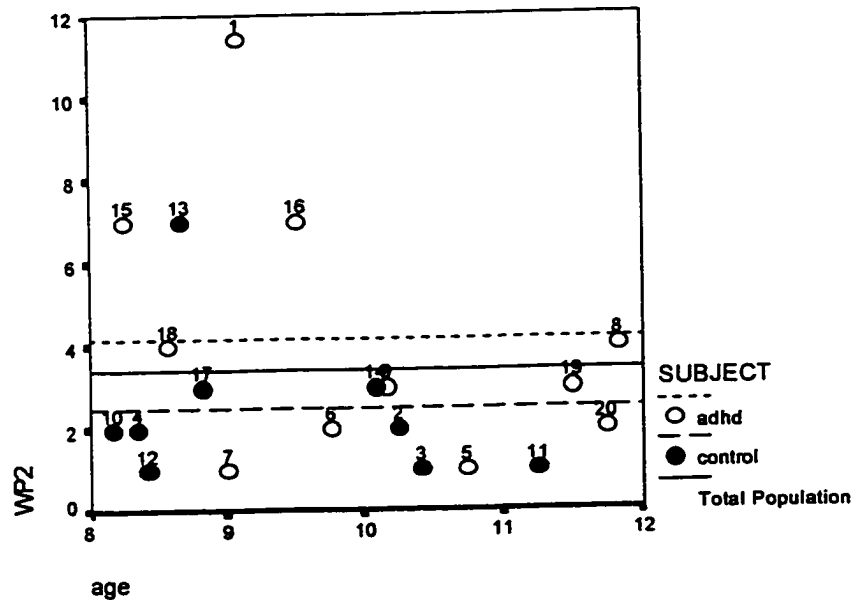


Figure 7

Percentage of wakefulness during Night 2 (WP2)



Figures 6 and 7 shows the pattern of differences in wakefulness on Nights 1& 2. On Night 2, three of four young ADHD children had difficulty sleeping, but only one non-ADHD child under 10 was awake more than 6% of Night 2. Instead of less wakefulness than non-ADHD children, the ADHD group was awake more, on average, twice as much during their time in bed than the non-ADHD group (ADHD Wake % Night 2 = 4.1; non-ADHD group Wake % Night 2 = 2.4). In contrast to five ADHD children, only one non-ADHD child was awake more than 4% of the night after sleep loss.

Power Analysis of the Sample

The findings of no differences have been presented as resulting from the research hypothesis being false. However, due to the small number of subjects ($n = 20$) in the sample, failing to find significant results is actually inconclusive. Not finding differences between the ADHD and control groups does not mean there are no differences. To determine the power of the study to reject the null hypothesis when it is really false, a power analysis was done for testing the significance between the groups' means for sleep, sleepiness and T.O.V.A.TM performance variables. The harmonic mean for the unequal groups (9.9) was calculated and an effect size of .40 (between small and medium) was estimated. For a two-tailed alpha level set at .05, with a sample size of about 10 per group, the resulting power is .15. That is, with 20 participants the study had only a 15% chance of finding differences between groups for these measures.

To determine the sample size needed to increase the experiment's power enough to find differences if they really exist, another power analysis was done. With a conventional, modest power specified (.80), and a moderate effect size estimated (.50), it was found that an $n = 63$ is necessary for each group to have an 80% chance to reject the null if it were really false. Setting the power to a more modest level (.70) would still require $n = 49$ to find differences, if they exist.

Leg movements

Leg movements were recorded for the first night of sleep only, as part of standard electrode placement on screening nights in the lab. Leg movement data was calculated to determine the level of sleep disturbance. Following the Atlas Task Force of the American

Sleep Disorder Association (ASDA) scoring criteria (Bonnet et al, 1995) standardizing scoring for leg movement, several types of leg movements were defined and compared. Movements were classified by how long the movement lasted and by the presence or absence of elevated submental (chin) electrode activity at the same time as the leg activity. Because the participants slept for different lengths of time, indexes of these movements per hour of sleep were calculated for comparing the groups. Table 5 presents the distinguishing features of these movement variables, while Table 6 shows the univariate analysis of the group means, standard deviations (SD), F values and significance of F .

Table 5

Types of Movement Indexes and SPSS Variable Labels

Length of Time	No Chin Activity	With Elevated Chin Activity	With Arousals	Summaries of Movements
<u>Short Movements</u>				
0.5 to 5 seconds	L	LC		Short = L+LC
<u>Medium Movements</u>				
5 to 15 seconds with Regular Wave Envelope	M	MC		M_Medium = M + MC
5 to 15 seconds with Irregular Wave Envelope	R	RC		R_Medium = R + RC
				All_Medium = M + MC + R + RC
<u>Long Movements</u>				
Over 15 seconds	MB		MBA	Long = MB + MBA
Total Movements = Short + All Medium + Long				

Short movements (L) were defined by duration and other features according to ASDA Atlas Task Force criteria, as a movement greater than 0.5 seconds and less than five seconds in duration, with an amplitude 25% greater than baseline recording levels. Two types of medium length movements were defined as movements that lasted from 5 to 15 seconds. Two distinct types of medium length movements were identified, based on the waveform envelope of leg activity. Uniform activity was scored (M), and irregular waveforms with at least three distinct pulses were classified as (R) movements. Long movements were scored

when elevated muscle activity lasted longer than 15 seconds. Long movements (MB) were scored with or without the presence of arousals (MBA). The three types of short and medium movements (L, M and R) were also scored according to the presence or absence of elevated chin muscle activity, as "LC," "MC" or "RC."

Periodic Leg Movements (PLM) were also scored. PLM's were defined according to the Atlas Task Force criteria, as a minimum of four small leg movements occurring within 90 seconds. Leg movement data were then converted to indexes, describing number of movements per hour of sleep time. Summaries, totaling the types of movements, were also calculated and compared.

Table 6

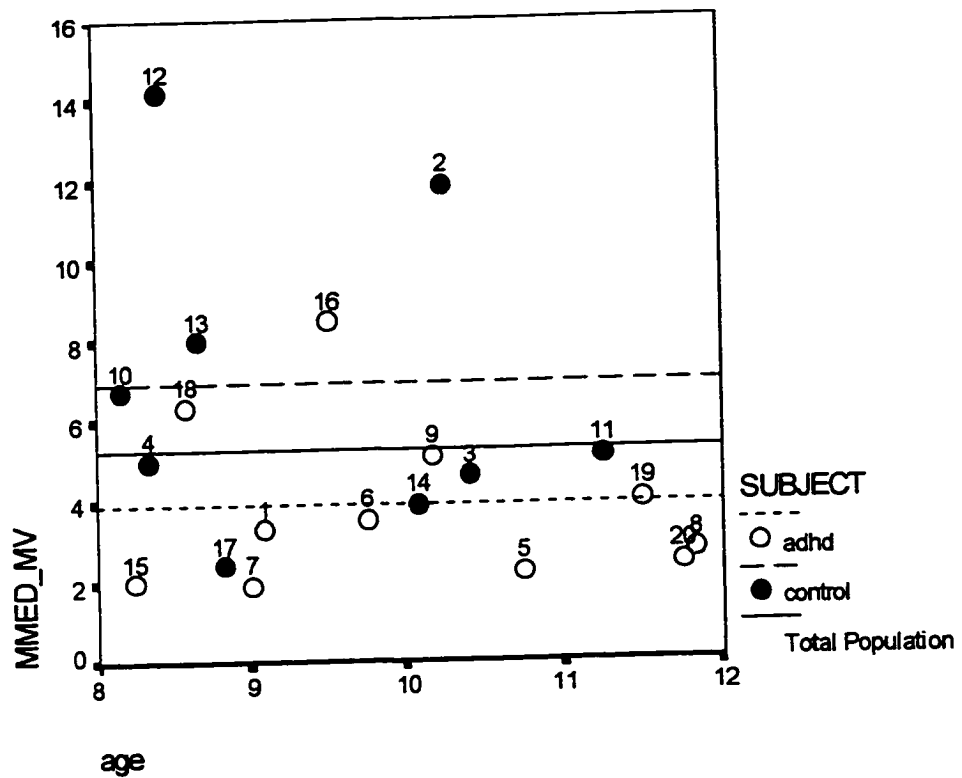
Movement Variables: Means, Standard Deviations and Univariate Analysis Results

Source Variable	ADHD (n = 11)		Non-ADHD (n = 9)		F	(1, 18) df	Sig. of F
	Mean	SD	Mean	SD			
All Indexes are # of movements / hr							
<u>Short Movements</u>							
L Index	4.9	4.3	8.3	5.1	1.66	1, 18	.11
LC Index	4.3	4.0	3.9	1.3	-.250	1, 18	.81
PLM Index	0.2	0.2	0.4	0.5	1.12	1, 18	.28
<u>Medium Movements</u>							
M Index	0.5	0.6	1.7	1.8	2.0	1, 18	.07
MC Index	3.3	2.1	5.3	3.8	1.44	1, 18	.17
R Index	1.8	1.4	3.6	4.0	1.47	1, 18	.16
RC Index	4.4	2.4	3.1	1.7	-1.43	1, 18	.17
<u>Long Movements</u>							
MB Index	1.5	0.9	3.5	4.8	1.38	1, 18	.19
MB with Arousal	0.2	0.2	0.4	0.4	1.63	1, 18	.12
<u>Summary Indexes</u>							
Short	9.1	6.6	12.2	5.3	1.15	1, 18	.27
M Medium	3.9	2.1	6.9	3.9	2.25	1, 18	.04 *
R Medium	5.9	3.0	7.4	4.4	.93	1, 18	.36
All Medium	9.8	5.0	13.6	5.7	1.63	1, 18	.12
Long	1.6	.9	2.6	3.1	.97	1, 18	.35
Total Movements	27.8	22.6	28.5	11.9	.08	1, 18	.93

Results of a One Way ANOVA procedure comparing the groups for movement indexes are presented in Table 6. Although the non-ADHD group exhibited a larger mean number of all types of movements on all but two measures (LC Index, RC Index), there was only one significant difference found comparing the groups. The non-ADHD group had significantly more "M Medium" movements than the ADHD group (ADHD group 3.9 per hour; non-ADHD group 6.9 / hr., $p < .05$), see Figure 8.

Figure 8

Scatterplot of M Medium Index Means by Individual (MMED MV)



In addition to the higher levels of the M_Medium Index in the control group, there was much within group variance, as four of the nine non-ADHD children were above the sample mean, compared to only two ADHD children.

Figure 9

Scatterplot of RC Index Means by Individual

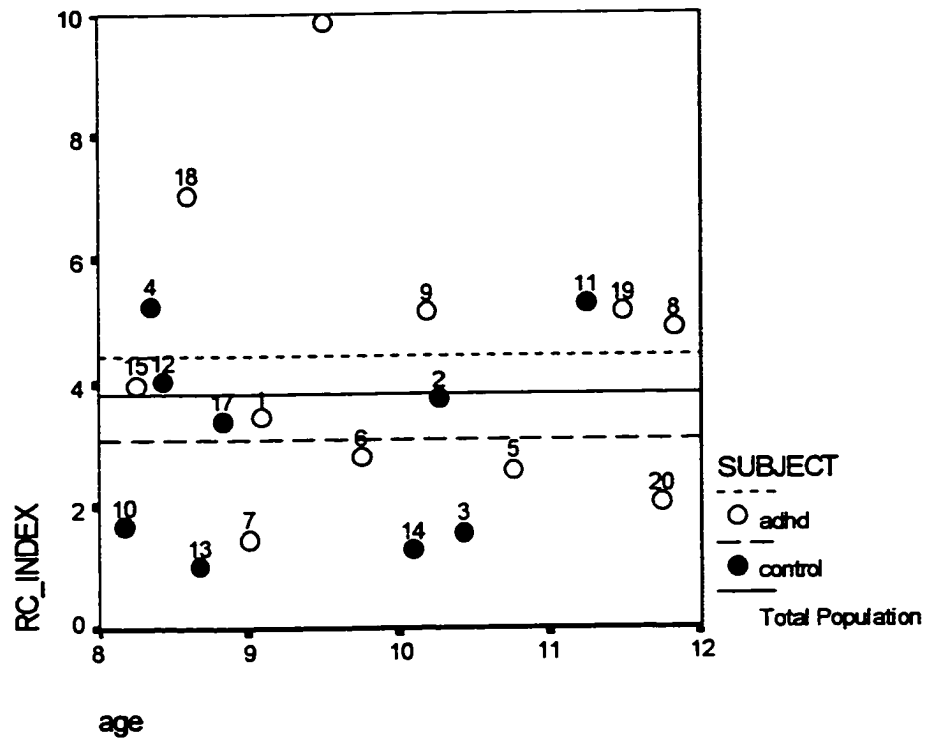


Figure 9 graphically presents the RC Index, showing the distribution of individuals for this type of movement.

Parent Reports of Sleep

Parental reports of their child's sleep on the Child Sleep Questionnaire also provided a different kind of data used to compare the groups' sleep. The Questionnaire is presented with a scoring sheet in Appendix E. Interview questions were asked about clusters of sleep behaviors at different times of the day: Bedtime, Nighttime, Morning, Daytime. All questions were negative behaviors; the higher the score, the more sleep related difficulties.

In addition, Likert scales, with a lowest score of 1 to a highest rating of 7, were also presented to parents to rate their children's daytime and sleep behaviors.

Table 7 shows the name of each scale, along with the total possible score for each scale in parentheses. A "Grand Average" was also calculated as the overall mean of the four Likert rating scales. As parents also reported both weekday and weekend bedtimes, and "uptimes," and differences were calculated, in hours between weekend and weekday sleep schedules.

Parents reported no differences for Time in Bed (TIB) differences in weekday and weekend bedtime and wakeup sleep schedules (ADHD group mean, 0.9 hours less time in bed; non-ADHD mean, 0.3 hours, $p = .98$, NS). But, parents of non-ADHD children reported bedtimes were about 2.4 hours later on the weekend, while the ADHD children went to bed, on average 1.6 hours later ($p = .06$, NS). Non-ADHD children were in bed, on average, 1.5 hours later on the weekend, in contrast to the ADHD group who went to bed 1.2 hours later ($p = .63$, NS). The experimental manipulation may have replicated, to some degree, the "real world" experiences of the participants staying up later, but did not allow them to "make up" their sleep debt in the morning.

Table 7 also presents the means and standard deviations and results of the univariate analysis performed on the Child Sleep Questionnaire (CSQ) variables. In general, parents reported significantly more sleep related problems for ADHD children than for non-ADHD children on the "Grand Average" ($t(18) = -2.12$, $p < .05$). The Grand Average was calculated as the overall mean of the four Likert rating scales, with the lowest score of 1 to the highest rating of 7. On other CSQ scales describing Bedtime, Nighttime, Sleep Disorders, Morning, and Daytime difficulties, parents reported a higher number of problems for the ADHD group, and although these differences were not statistically significant, several were very close to significance at the $p < .05$ level. Total possible scores for each scale are in parentheses.

Table 8 shows the results of an analysis of the relationships between parent's reports of sleep at home (CSQ) with recordings in the sleep lab. Pearson Product Moment Correlations results show that there were strong relationships that are conceptually meaningful, between some of the scales for sleep related difficulties reported by parents and empirical recordings.

Table 7

Means, Standard Deviations and Univariate Results for Child Sleep Questionnaire Variables Reported by Parents

Variable	ADHD (n=11)		Non-ADHD (n=9)		t	df	Sig. of t (2-tailed)
	Mean	SD	Mean	SD			
<u>Interview Questions</u>							
(Number of difficulties)							
Bedtime (6)	2.8	2.1	1.9	2.2	-0.98	18	.34
Daytime (8)	2.5	2.2	1.6	1.6	-1.06	18	.32
Disorders (9)	1.8	1.4	1.4	1.7	-0.54	18	.60
Morning (7)	3.6	2.5	2.3	2.5	-1.18	18	.25
Nighttime (12)	2.6	2.1	1.9	1.3	-0.87	18	.40
<u>Likert Scales</u>							
(mean values range from 1 to 7)							
Morning	4.8	1.9	3.6	3.0	-1.06	18	.30
Daytime	3.1	1.5	1.6	1.9	-1.98	18	.06
Bedtime	3.8	2.4	1.9	2.0	-1.92	18	.07
Nighttime	2.6	2.1	1.7	1.6	-1.40	18	.18
"Grand Average"	3.7	1.3	2.2	1.7	-2.01	18	.05*
(mean of 4 Likert scales)							
<u>Weekday/Weekend Sleep</u>							
<u>Schedule Differences</u> (in hours)							
Bedtime Difference	1.6	0.9	2.4	1.1	1.97	18	.06
Uptime Difference	1.2	1.4	1.5	1.1	0.47	18	.63
TIB Difference	-0.3	0.3	-0.9	0.6	-0.02	18	.98

In general, there were a number of moderately strong relationships between CSQ variables and some post sleep loss (NPSG 2) variables. Although the CSQ was not related to Night One (NPSG 1) variables, higher parent ratings correlated with five post sleep loss NPSG 2 variables in meaningful ways.

Table 8

Intercorrelations Between CSQ and Post-Sleep Loss NPSG Recordings

<u>CSQ Interview</u>	<u>Bed Qx</u>	<u>Day Qx</u>	<u>Morn. Qx</u>	<u>Night Qx</u>	<u>Morn. Likert</u>	<u>Bed Likert</u>	<u>Grand Avg.</u>	<u>Uptime Difference</u>
NPSG								
Variables								
SOL N2	.33	-.07	.38	.14	.52*	.18	.20	.12
SE % N2	-.07	-.08	.38	.14	-.25	-.23	.20	.31
% W N2	.06	-.25	.10	-.14	.28	.23	.22	-.30
%Stg1 N2	-.16	.23	.04	.07	.11	-.12	.07	.09
%Stg2 N2	-.28	-.24	-.44	-.15	-.54*	-.20	-.34	-.49
%Stg 3 N2	.65**	.21	.27	.33	.16	.64**	-.50*	-.16
%Stg 4 N2	.03	.02	.19	-.13	.30	-.09	.05	.31
% REM N2	.11	.26	.17	.29	.11	-.01	-.01	.46*

* p < .05

** p < .01

Taking longer to fall asleep after being kept up late (SOL Night 2) was positively and moderately associated with higher parental ratings of their children's morning difficulties ($r = .52, p < .05$). Parents rate children who take longer to fall asleep at night as having more problems in the morning. Higher ratings of problems going to bed were strongly and positively associated with increased percentage of Stage 3 on Night 2 ($r = .65, p < .01$).

Parents rate children having increased Stage 3 rebound as having more difficulties going to bed. The children having difficulty going to bed and settling down experienced Stage 3 rebound, an increased percentage often seen in response to either previous sleep deprivation or physical exertion.

The overall mean of all Likert scales (Grand Average) was inversely related to percentage of Stage 3 sleep ($r = -.50, p < .05$). As the overall average of sleep-related difficulties increased, post sleep loss levels of Stage 3 % decreased. Here, parents rate children who had lower Stage 3% on Night 2 having overall, lower global quality of sleep ratings.

Lower percentage of Stage 2 sleep was related to an increase in number of morning difficulties parents reported. For children whose parents reported problems such as going to

sleep or morning difficulties, those children showed larger Slow Wave Sleep rebound after sleep deprivation, and less Stage 2 sleep.

Taking a closer look at the relationships between sleep and parent reports of sleep related behaviors, Pearson Product Moment correlations were calculated for CSQ and movement variables. In Table 9, we see the correlations between the parents' reports of sleep difficulties at home and the lab recordings of movement indexes. A number of significant inverse relationships were found between two types of movements, the L Index and R Index, and daytime problems on the CSQ. Also moderately inversely related was the mean of all small movements and Night Questions. As the number of these movements increased, parent ratings decreased. This may reflect the fact that these three movement indexes are not particularly disruptive to children's sleep.

Table 9

Intercorrelations Between CSQ and Movement variables

CSQ Interview	Bed Q's	Day Q's	Morn. Q's	Night Q's	Morn. Likert	Bed Likert	Grand Avg.	Up Diff.
L Index	-.44	-.47*	.03	-.44	.05	-.23	-.28	.21
LC Index	.05	-.05	.31	-.28	.01	-.18	-.17	.03
PLM Index	-.20	-.33	.10	-.15	.25	-.08	-.10	.18
M Index	-.14	-.29	.26	-.11	.34	-.09	-.09	.06
MC Index	-.27	.32	.15	-.30	-.03	-.27	-.17	-.03
R Index	-.26	-.45*	-.14	-.24	-.09	-.14	-.25	.11
RC Index	-.13	.91	.17	-.14	.03	-.08	-.02	.04
MB Index	-.07	.03	-.17	-.43	-.17	-.20	-.20	-.31
MB with Arousal	-.17	.21	.06	-.13	.06	.06	.12	-.06
Short	-.31	-.40	.18	-.49*	.04	-.27	-.31	.18
M Medium	-.30	.18	.24	-.32	.12	-.28	-.19	-.01
R Medium	-.15	-.29	.01	-.15	.03	-.07	-.11	.24
All Medium	-.36	-.08	.18	-.36	.06	-.26	-.25	.10
Long	-.19	.38	.03	-.28	.01	-.04	.05	-.16
Total	.33	.24	.39	.33	.22	.28	.32	.01

p < .05

Power Analysis for Intercorrelations between CSO and NPSG Sleep Recording

Again, power was determined to determine the extent to which the study could find relationships between the groups. Given an assumption of a large effect size (.50) and a sample size of 20, the power is .26. That is, there is a 26% chance of finding any significant relationships between parental reports and empirical recordings of sleep. A one in four chance of finding significance with this sample size reflects the weak power of the study to reject the null hypotheses (that there are no differences).

The sample size necessary to find differences was calculated: Sixty-nine (69) participants for each group would be required, assuming a moderate effect size (.30), a power of .70, and using a two-tailed test with alpha set at .05.

Statistical Analysis of Daytime Sleepiness

Table 10 shows the means, standard deviations and multivariate analysis results for measures of daytime sleepiness, before and after sleep loss. There were no differences between the groups (Subject main effect) for either of the two variables reflecting levels of daytime sleepiness, the average time it took to fall asleep (Stage 1 sleep onset latency) and the average number of times the children actually fell asleep (number of sleep episodes). There were no differences found or between the groups from Day 1 to Day 2 (Subject By Time interaction effect).

In contrast, there were significant differences found between Day 1 and Day 2 recordings (Time main effect) found for both measures of sleepiness, Stage 1 Sleep Latency [$F(1,18) = 25.94, p < .001$], and Number of Sleep Episodes [$F(1,18) = 16.94, p < .001$]. After some sleep loss, all participants were significantly sleepier: that is, they took less time to fall asleep, and actually fell asleep more often on the second day of nap opportunities. Post-hoc univariate analysis shows that both the non-ADHD and ADHD groups had significantly decreased sleep latency to Stage 1 (ADHD mean decreased 3.1 min.; non-ADHD group mean decreased 5.2 min. [$F(1, 18) = 25.94, p < .001$]). Both groups also showed an increased number of sleep episodes on Day 2 (ADHD mean = 1.1 more sleep episodes; non-ADHD group mean = 1.3 more sleep episodes [$F(1, 18) = 25.94, p < .001$]). The whole sample was made more sleepy from staying up two hours later, but there was no differential effect on the groups.

Table 10 also shows differences in means for the other measure of sleepiness, the number of times participants actually fell asleep during MSLT 1. On the first set of nap opportunities, the ADHD group had shorter Sleep Latency, indicating somewhat more sleepiness overall, but they fell asleep less often. The non-ADHD group fell asleep more often (ADHD group mean: 1.6 naps; non-ADHD mean: 2.1 naps, NS). On the second day, all participants fell asleep significantly more [$F(1,18) = 16.94, p < .0001$], but the non-ADHD group again fell asleep more often than the ADHD group (ADHD group mean: 2.7 naps; non-ADHD mean 3.4 naps, NS).

Table 10

MANOVA results for MSLT variables

Source of F	ADHD (n=11)		Non-ADHD (n=9)		F	df	Sig. of F
	Mean	SD	Mean	SD			
Subject Main Effect (minutes)					$F_{Subject}$	df	Sig. of $F_{Subject}$
Sleep Latency Day 1	21.7	3.4	22.5	1.6	.03	1, 18	.88 NS
Sleep Latency Day 2	18.6	5.1	17.3	4.0			
Number of Sleep Episodes – Day 1	1.6	1.6	2.1	1.3	.90	1,18	.36 NS
Number of Sleep Episodes - Day 2	2.7	1.7	3.4	1.4			
Time Main Effect (n=20)					F_{Time}	df	Sig. of F_{Time}
Sleep Latency					25.94	1.18	.0001
Number of Sleep Episodes					16.94	1.18	.0001
Subject by Time Interaction					$F_{Subject \times Time}$	df	Sig. of F_{SxT}
Sleep Latency					1.69	1,18	.21 NS
Number of Sleep Episodes					.17	1,18	.69 NS

While the ADHD group showed an unusual response to mild sleep loss, with somewhat more light, Stage 1 sleep than controls, their alertness was not more impaired, as determined by the second day of nap opportunities.

To investigate further the relationship between daytime sleepiness and nighttime sleep, the relationships between NPSG and MSLT variables were assessed using Pearson

Product Moment correlations (Table 11). Several variables showed moderately strong significant relationships. The average number of sleep episodes on nap opportunities was associated with total wake time (TWT N2) and sleep efficiency on the second night of sleep in the lab (SE % N2).

The fewer times participants fell asleep on the first day of nap opportunities, the longer participants were awake on the second night. TWT on Night 2 was inversely and moderately associated with the number of times the participants fell asleep on the first day of nap opportunities ($r = -.45, p < .05$). Hyperarousal during the first day predicted increased TWT later that night.

Table 11

Intercorrelations between NPSG and MSLT Variables

Scale	1. Sleep Latency Day 1	2. Sleep Latency Day 2	3. Sleep Episodes Day 1	4. Sleep Episodes Day 2
<u>MSLT</u>				
1. SL MSLT 1	---			
2. SL MSLT 2	.59**	---		
3. No. Sleep Episodes Day 1	-.81**	-.68**	---	
4 No. Sleep Episodes Day 2	-.53*	-.87**	-.68**	---
<u>NPSG</u>				
5. TWT N 1	.16	.04	-.11	-.13
6. TWT N 2	.39	.33	-.45*	-.37
7. SE % N 1	-.13	-.04	.11	.13
8. SE % N 2.	-.40	-.32	.47*	.36
9. REM % N 2	-.07	-.39	.12	.50*

* $p < .05$ ** $p < .01$

Also, the number of sleep episodes during Day 1 were moderately and positively correlated to Sleep Efficiency percentage on Night 2 ($r = .47, p < .05$). The more times the children fell asleep during the first day, the more efficiently they slept later that night. More

daytime sleep episodes signifies higher levels of sleepiness, which then translated into sleeping for more of the time they were in bed later that night. The number of sleep episodes on Day Two (Number of Naps 2) was positively and moderately related to percentage of Stage REM sleep on Night 2 ($r = .50, p < .05$). As the number of post sleep loss sleep episodes increased, Stage REM % increased that night. These are interesting relationships, given the study's findings that both groups were more sleepy during Day 2. The ADHD group's tendency toward less sleep episodes while experiencing increased levels of sleepiness is in contrast to the non-ADHD children, who fell asleep more often as they became sleepier on Day 2.

Table 12

Intercorrelations Between MSLT Variables and Leg Movements.

Scale					
MSLT:	1. Sleep Latency Day 1	2. Sleep Latency Day 2	3. Sleep Episodes Day 1	4. Sleep Episodes Day 2	
Movements					
5. L Index	.41	-.03	-.24		-.01
6. LC Index	.15	.29	-.10		-.46*
7. PLM Index	.10	-.18	-.10		.08
8. M Index	.11	-.36	-.04		.18
9. MC Index	.37	.26	-.26		-.33
10. R Index	.23	.07	-.13		-.03
11. RC Index	.29	.45*	-.20		-.55
12. MB Index	.07	.14	-.16		-.14
13. MB w/Arousal	.38	.23	-.41		-.22
14. Short	.40	.12	-.24		-.24
15. M Medium	.39	.10	-.26		-.23
16. R Medium	.29	.23	-.10		-.19
17. All Medium	.47	.24	-.31		-.33
18. Long	.31	.24	-.35		-.24
19. Total	-.14	-.02	-.03		-.07
Movements					

* $p < .05$

In Table 12, we can see the correlations exploring the relationships between the MSLT measures of sleepiness and nocturnal recordings of movements. Increases in the LC and RC Indexes, in particular, correlated with increases in daytime sleepiness and less number of naps.

Results of an analysis of the relationships between the CSQ and MSLT variables are presented in Table 13. Three of the parent report scales were significantly related to measures of daytime sleepiness. Increased nighttime sleep problems that parents reported at home were inversely and moderately related to shorter Sleep Latency times during Day 1 recordings in the lab ($r = -.47, p < .05$).

Table 13
Intercorrelations Between CSQ and MSLT Variables

Scale	1	2	3	4	5	6
MSLT						
1. Sleep Latency Day 1	---					
2. Sleep Latency Day 2	.59**	---				
3. Sleep Episodes Day 1	-.81	.68**	---			
4. Sleep Episodes Day 2	-.52	-.87**	.65*	---		
CSQ						
5. Night Questions	-.47*	-.31	.31	.24	---	
6. Bedtime Difference	-.03	-.45*	.33	.54*	.27	---
7. Uptime Difference	-.10	-.46*	.36	.35	.27	.58**

* $p < .05$ ** $p < .01$

Parent reported differences between sleep schedules for weekdays versus weekends were found to be significantly related to increases in post sleep loss sleep latency during Day 2 recordings. As time differences in bedtimes and uptimes increased, sleep latency decreased significantly. Table 13 shows that as the difference between weekday and weekend bedtimes parents reported (Bedtime Difference) increased, the average number of actual times participants fell asleep on Day 2 increased as well ($r = -.46, p < .05$). Sleep latency on Day 2 was positively related to the difference between weekday and weekend wake times (Uptime Difference).

Statistical Analysis of T.O.V.A.™ Performance

Table 14 shows there were significant differences found across the three trials of the T.O.V.A.™ (Time main effects) for two scaled score variables: Commission Errors [$F(2, 32) = 4.17, p < .05$] and Variability [$F(2, 32) = 4.26, p < .05$]. There were no differences across the three trials, between the groups (Subject main effect) or between groups across the three trials (Subject by Time interactions).

Post hoc univariate analysis of significant commission error differences shows that after the sleep loss, all participants made a higher number of these “impulsive” errors on Trial 3, ($t(19) = -2.30, p < .05$) compared to Trial 2, however, there were no differences between the first two, pre-sleep loss trials ($t(19) = -1.94, NS$). For the Variability scaled scores, Table 14 shows there were significant differences between Trials 1 and 2 ($t(18) = -2.14, p < .05$) and between Trials 2 and 3 ($t(18) = -2.45, p < .05$).

Raw scores were used in the analysis for scales that were not available as scaled or T -scores. Using a Repeated Measure MANOVA procedure, there were significant differences found across the three trials (Time main effect) for percentage of correct responses [$F(2, 36) = 9.29, p < .01$]. No other differences were found for raw score variables. Univariate tests showed performance continually declined on this measure of accuracy between the 2 pre-sleep loss Trials 1 and 2 ($t(19) = 2.84, p < .01$) and between the Trial 1 and the post sleep loss Trial 3 ($t(19) = 3.25, p < .01$).

One non-blind observer watching the participants rated the ADHD group as more distractible in general, and more easily distracted by noises and sounds than the non-ADHD group. Table 14 shows some of the results of a Repeated Measures MANOVA for the T.O.V.A.™ Observer Rating Scale data. The Distractibility scale [$F(1, 18) = 4.43, p = .05$] and Auditory Distractibility [$F(1, 18) = 9.13, p < .01$] scales were significantly different between the groups (Subject main effect). Post hoc univariate analysis revealed that means for distractibility, as rated by observers, was significantly higher between Trials 1 and 2 ($t(19) = 2.18, p < .05$).

Table 14

Means, standard deviations and multivariate analysis results of T.O.V.A.™ performance.

	ADHD (n=11)			Non-ADHD (n=9)			F	df	Sig of F
	Time 1	Time 2	Time 3	Time 1	Time 2	Time 3			
Scaled Scores							F_{Time}	df	Sig of F_{Time}
Omissions Errors							0.42	2,36	.66 NS
Mean	79.1	79.3	75.9	81.6	88.2	85.6			
SD	27.5	26.6	18.5	22.9	19.0	23.2			
Commission Error							4.17	2,36	.02*
Mean	75.8	85.5	86.0	74.4	91.2	101.2			
SD	30.5	32.5	35.5	26.5	32.4	24.1			
Variability							4.26	2,36	.02
Mean	48.5	57.6	56.8	48.8	64.8	72.3			
SD	31.3	25.7	29.4	24.3	29.6	34.1			
Reaction Time (in milliseconds)							.54	2,36	.59 NS
Mean	78.2	77.6	69.2	81.8	87.7	86.8			
SD	24.9	22.6	25.6	16.0	11.4	10.8			
Raw Scores									
Multiple Responses							2.98	2,36	.06 NS
Mean	200.6	222.4	207.6	217.8	227.4	209.8			
SD	68.5	75.5	83.9	64.8	75.6	74.5			
Percentage Correct							9.29	2,36	.001
Mean	47.2	36.7	36.4	49.6	33.0	24.6			
SD	23.5	26.8	28.8	19.4	24.9	22.3			
Observer Ratings (from 1=least to 7=most)							F_{Subj.}	df	Sig of F_{Subject}
Distractibility							4.43	1,18	.05
Mean	1.4	1.9	1.6	1.1	1.3	1.0			
SD	0.5	0.9	0.8	0.3	0.7	.00			
Auditory Distraction							9.13	1,18	.007
Mean	1.5	1.6	1.2	1.0	1.0	1.0			
SD	0.7	0.5	0.6	.00	.00	.00			
Prompts needed							.350	1,18	.08 NS
Mean	1.7	2.4	1.9	1.0	1.2	1.3	F_{Time}	Sig of F_{Time}	
SD	1.3	1.4	1.5	.00	0.4	0.5	3.94	.28 NS	

The number of prompts not to talk while the participants took the T.O.V.A.™ was recorded by observers. Here, the ADHD group needed more prompts not to talk during all three trials [$F(2,36) = 3.94, p < .05$]. Post hoc analysis shows that prompts were significantly higher for the ADHD group than the non-ADHD group on Trial 2 and Trial 1 ($t(19) = -3.94, p < .01$). There were no differences between the other trials.

Examining the pattern of significant errors more closely, Table 14 shows that on the two pre-sleep loss administrations, the average amount of errors went up on the second trial, suggesting a fatigue effect.

After the sleep loss condition, the number of omission errors for both groups continued to decrease somewhat. In contrast, the number of commission errors for both groups increased after the sleep loss night. Overall, they made fewer omission errors and increased commission errors as they got bored by the task, and began pressing faster and more often.

That the sleep loss had some effects on T.O.V.A.™ errors, although there were no differences found between the ADHD and non-ADHD groups (Subject main effect), suggests a relationship between the variables, rather than a causal effect. Pearson Product Moment correlations were done to determine the extent of these relationships.

Table 15 shows the significant relationships found between MSLT and T.O.V.A.™ data. It can be seen that some T.O.V.A.™ errors were positively related to some measures of sleepiness. Errors of impulsivity, scaled and raw score commission errors, went up as time to fall asleep went down. Sleep Latency on Day 1 was inversely related to scaled score Commission Errors on Trial 3. Those children who fell asleep more often on the first set of nap opportunities made more post sleep loss “impulsivity” errors. Sleep Latency on MSLT 1 was also inversely related to Percentage of Correct Responses on Trial 3.

Table 15

Intercorrelations Between MSLT and T.O.V.A.™ Variables

Variables MSLT:	SL MSLT 1	SL MSLT 2	No. of Naps 1	No. of Naps 2
<u>T.O.V.A.™ Scaled Scores</u>				
Commission 1	.05	.17	.06	-.36
Commission 2	-.14	.08	.31	-.21
Commission 3	-.41*	-.21	.47	-.02
Omission 1	.34	.21	-.09	-.38
Omission 2	.37	.18	-.16	-.41
Omission 3	.12	-.09	.03	-.22
Reaction Time 1	-.01	.12	.09	-.23
Reaction Time 2	.19	.21	-.06	-.17
Reaction Time 3	.03	.14	.02	-.11
Variability 1	-.10	.13	.26	-.39
Variability 2	-.09	-.05	.17	-.24
Variability 3	-.19	-.10	.18	-.16
<u>Raw Data</u>				
Anticipatory 1	.07	-.23	-.09	.39
Anticipatory 2	.22	-.16	-.16	.30
Anticipatory 3	.40	.10	-.35	.13
Commission 1	.04	-.06	-.15	.21
Commission 2	.25	-.02	-.39	.11
Commission 3	-.45*	.22	-.50*	-.03
% Correct 1	.15	-.11	-.15	.34
% Correct 2	.31	.02	-.43	.11
% Correct 3	.47*	.23	-.52*	-.05

* p < .05

**p < .01

Movements were correlated to determine the extent of relationships with T.O.V.A.™ variables. Table 16 presents these data, and shows that the study found several significant correlations between these sets of variables. Scaled scores for Variability on trial 2 and 3 were both moderately and positively related to M Medium movements. Raw scores and T

scores for Omission Errors were significantly related to R Medium Index. Interestingly, we can see that one of the only two types of movements, the RC Index, was correlated with eight out of the 36 the T.O.V.A.TM variables, more than any other movement on across three trials.

Table 16

Intercorrelations Between T.O.V.A.TM and Movements

Movement Indexes:	M Med.	Total N./hr.	R Med.	All Med.	LC	MC	RC	MB
<u>Scaled Scores</u>								
Commission 1	.35	.48*	.29	.31	.35	.37	.49*	-.32
Commission 2	.31	.39	.06	.32	.38	.34	.47*	.11
Commission 3	.33	.29	-.25	.19	.25	.27	.14	.19
Omission 1	.23	.33	.29	.30	.27	.20	.42	-.24
Omission 2	.37	.24	.06	.27	.12	.35	.28	.28
Omission 3	.39	.13	-.25	.12	-.06	.32	.01	.34
Reaction Time 1	-.01	.19	-.12	-.06	-.48	-.26	-.05	-.16
Reaction Time 2	-.33	.28	-.46*	-.48	-.28	.02	-.22	.16
Reaction Time 3	-.29	.29	-.09	-.28	.23	-.23	.10	-.02
Variability 1	.35	-.01	.04	.23	.34	.45*	.51*	-.11
Variability 2	.54*	.20	.02	.34	.16	.53	.33	.42
Variability 3	.46	.09	-.18	.16	-.26	.40	-.01	.29
<u>Raw Data</u>								
Anticipatory 1	-.31	-.31	-.16	-.26	-.29-	-.34	-.46	.37
Anticipatory 2	-.21	-.25	-.11	-.18	-.30	-.25	-.37	-.16
Anticipatory 3	-.27	-.21	-.02	-.14	-.25	-.25	-.23	-.16
Commission 1	-.33	-.41	-.16	-.28	-.30	-.34	-.48*	.45*
Commission 2	-.30	-.34	-.24	-.31	-.35	-.32	-.43	-.12
Commission 3	-.31	-.25	-.04	-.19	-.23	-.26	-.13	-.15
% Correct 1	-.30	-.46*	-.01	-.17	-.34	-.34	-.41	.24
% Correct 2	-.30	-.37	-.14	-.25	-.38	-.33	-.38	-.07
% Correct 3	-.31	-.27	-.03	-.14	-.25	-.28	-.11	-.21

* p < .05

** p < .01

CHAPTER FIVE

DISCUSSION

The purpose of this study was to compare the effects of sleep loss between two well defined groups of twenty ADHD and Non-ADHD prepubertal children (ADHD $n = 11$, Non-ADHD $n = 9$). Assessments were made in a number of domains of social, academic and neuropsychological functioning to carefully define the similarities and differences between the groups, prior to beginning the 2 night, 2 day design in the CCNY Sleep Disorders Center, where their sleep and levels of daytime sleepiness were electronically recorded. Determination of ADHD or Non-ADHD group status was based on parental reports of the inattentive and/or hyperactive behaviors listed as the DSM-IV criteria (APA, 1994). All parents completed the DISC structured interview for DSM-IV behaviors.

The ADHD group had significantly more behaviors than the non-ADHD group and they averaged just enough behaviors to be classified clinically as ADHD. This represents a mild to moderate degree of clinical impairment. Restriction of aggressive behaviors for methodological and practical considerations excluded severe difficulties associated with more aggressive problems. Aggressive behaviors were also reported as significantly higher, on average for the ADHD group (ADHD group mean ODD/CD behaviors = 3.5, non-ADHD group = 0.7, $p < .001$). However, none of the children in the study exhibited developmentally inappropriate levels of aggression.

The non-significant deviation from the CPRS-48 and CBC means may reflect the moderate ADHD symptomatology exhibited by the study's mildly impaired ADHD group, or these measures may have difficulty identifying a moderately, but still clinically impaired ADHD children.

There was no difference in average age of the groups (ADHD group mean 10.0 years old; non-ADHD group mean 9.4 years old, NS). On the Wechsler Intelligence Scale for Children, (WISC-III), the ADHD group had significantly lower FSIQ, VIQ,

VCIQ scores, but there were no differences for PIQ, POIQ, FDIQ scales. On the measure of academic functioning, (WRAT III) the ADHD group scored significantly lower on the Spelling and Arithmetic scales, but there were no differences on the Reading scale. Overall, the non-ADHD boys were performing about a half grade above their present grade level in school, while ADHD group was almost 3½ grades below level in Spelling and Arithmetic. The ADHD children in our sample were having significant difficulties in school. In grades 3, 4 and 5, the impact of their attention/motivation/ability on school work can not be covered up, and indicates the serious academic risk ADHD children can face. Learning Disabilities, often present with non-aggressive ADHD, may have impacted these scores as well.

On the VMI/Beery, a visual-motor integration task of copying geometric forms that become progressively more difficult, the ADHD group scored significantly lower. They performed significantly worse than the non-ADHD group; just under the 13th percentile compared to about the 56th percentile for the non-ADHD group. Many of the ADHD boys were not careful and approached this task casually. Many worked too quickly and lost points for careless errors. On two basic neuropsychological measures, the Stroop and Trailmaking tasks, there were no differences between the groups. The ADHD children had no difficulty, relative to the other children, in switching cognitive sets on Trails tasks, or concentrating during the interference condition of the Stroop task. Generally, most of the children found these novel tasks interesting.

Recent research with adults with ADHD ($n = 489$) with mean age of about 35, that shows differences in neuropsychological functioning (Taylor & Miller, 1997). ADHD adults were slower than controls completing the Trails task and had higher interference times on the Stroop. Considering that executive frontal lobe functioning has been implicated in ADHD behaviors by Palm (1992) no differences between our sample groups might reflect the moderate difficulties seen in the ADHD children, as opposed to many other ADHD children with more severe symptoms.

However, children may not respond similarly to adults on these tasks. Miller, Kavacic and Leslie (1996) found no differences in baseline performance between 8 to 10 year old ADHD and control children ($n = 32$). Also, mixed results in previous research have been reported by Barkley, Gordzinsky & Paul (1992). There may be limited

usefulness of this measure, in contrast to adults, to distinguish ADHD groups, not just the present sample with moderate numbers of behaviors. Alternatively, mild ADHD children may not be very different in frontal lobe functioning from controls.

NPSG Sleep Measures

A number of different types of assessments were made in the area of sleep and waking behaviors. Both objective and subjective measures of sleep and daytime alertness were administered to the children and their parents to test several hypotheses about nighttime sleep and daytime behaviors. Subjective ratings by parents of their children's sleep were obtained with a Child Sleep Questionnaire (CSQ). Objective measurement using nighttime polygraph recordings began on Friday night, as participants went to bed in the CCNY Sleep Disorders Center. Bedtimes were calculated as their average bedtime for previous week, according to their sleep logs. Then their sleep was electronically recorded (NPSG). After staying in the lab all day, on Saturday, all children went to bed two hours later than Friday night bedtime and their sleep was recorded again. During Saturday and Sunday, levels of daytime sleepiness were assessed with the Multiple Sleep Latency Test (MSLT) and compared between groups, over time, and the interaction effect. Dependent variables included: 1) nighttime NPSG sleep variables, 2) parent reports of sleep and sleep-related difficulties. Excluding outliers was considered, but subsequent data analysis was beyond the scope of this study.

On Night 1, there were no differences between groups (Subject Main Effect) for any of the NPSG sleep variables. There was much variance within both groups – a few individuals from each group didn't sleep well, and this variance obscured differences between the groups.

On Night 2, the ADHD group had a higher percentage of Stage 1 sleep the second night, responded in the opposite direction compared to controls. They had a somewhat elevated percentage of this transitional phase of sleep. This is contrary to what we would expect following sleep loss. Furthermore, they had a harder time falling asleep after some sleep loss, indicating a less adaptive response to the sleep loss.

There were also significant differences in the whole sample across the two nights of sleep (Time Main Effect) on Night Two: Time In Bed (TIB), and percentages of Total

Sleep Time (TST), Total Wake Time (TWT), were all significantly lower. This reflects the experimental intervention, keeping the children up two hours past their previous night's bedtime. Both groups also fell asleep faster, on average, and stayed asleep more of the time they were in bed, and had more Stage 4 (Slow Wave) Sleep.

Leg Movements

The non-ADHD group exhibited somewhat higher means for most types of leg movements, on all but two indexes (LC Index, RC Index). There was only one significant difference found, however, comparing the groups. The controls had significantly more "M Medium" movements than the ADHD group (ADHD group = 3.9/hour; non-ADHD group = 6.9/hr., [F (1, 18) = 2.25, $p < .05$]. That the non-ADHD children's sleep was somewhat more restless as recorded in the sleep lab on the first night when compared to the more overactive ADHD children was not expected. The ADHD children had less small and large movements, had half as many arousals and PLMs. This is inconsistent with the literature that generally supports a general higher movements and restlessness in ADHD groups. Here, the non-aggressive, ADHD children, with less difficulty restraining overactivity and impulsivity looked like controls.

Hypothesis 1: The first research question the study asked was this: Are there differences in the sleep of ADHD children? Overall, no differences were found, except for an interesting interaction effect involving ADHD over Time. One distinct difference found in how the ADHD children responded to the sleep loss night in contrast to the non-ADHD children. Percentage of the lighter, poor-quality, transitional Stage 1 sleep decreased in the non-ADHD group, as in healthy control children (Coble, Kupfer, Reynolds & Houck, 1987), after some sleep loss. In contrast, Stage 1 sleep increased in the ADHD group and the interaction of group status over time was significant. This suggests differences in ADHD children's sleep manifest only with some cumulative sleep loss, beyond a first night effect or staying up late one night.

This finding of the study somewhat supports the first hypothesis. This data also relates to the common clinical complaints of trouble getting ADHD children to bed or

stay in bed and fall sleep. Even for moderate ADHD children, these type of problems may escalate after a night of mild sleep loss.

The findings here support Palm (1992) that DAMP are not sleepier than controls before a sleep loss condition. The findings may also support Miller & Kraft's (1992) theory ADHD children's sleep may have sub-clinical levels of differences, that can be brought out at times, when under stress, sleep loss or other factors exacerbate those differences.

Findings support Randazzo's (1998), use of five hours of restricted sleep condition. Given that the parents in a the study reported large differences in weekday and weekend bedtimes, future studies need to use a greater degree of sleep loss to investigate its effect on this age group of ADHD children's sleep.

Another limitation of the study was not counterbalancing the sleep loss condition, introducing the possibility of confounding the results with an order effect. However, our data support findings of a minimal first night effect in this age group (Carskadon, Keenan & Dement, 1987). The influence of the first night effect on daytime alertness to confound results by adding additional, uncontrolled amounts of sleep loss, was considered minimal, as there were no main effect for ADHD found, as Stage 1 and Wakefulness percentages were not different. Follow up studies on the effect of sleep loss should also include Actigraphy, for example, to help assess behavioral functioning in other areas impacted by ADHD.

Power Analysis for NPSG, MSLT and T.O.V.A.™ Groups Differences

An important statistical limitation of this study is the small sample size. Although for an empirical ADHD sleep study, an $n = 20$, with 11 ADHD boys is a substantial number, statistically speaking, it means low power and the increased likelihood of making a Type II error, where no differences are found when, in fact, there are differences.

As stated in the results section, there was only a 15% chance of finding differences. It was also shown that it is necessary to have an $n = 63$ for each group, or a total sample size of $n = 126$ children, to determine with 80% certainty if there were differences between the groups. Although sleep research is technically complicated, time-consuming and labor intensive work, further ADHD sleep studies need to overcome these

difficulties and have larger samples, to obtain more conclusive results than are reflected generally in the literature.

Parent Reports of Sleep

On the questionnaire of their children's sleep, parents of ADHD children reported one scale was higher for the ADHD group. A trend was found in the difference between weekday and weekend bedtimes. The ADHD group went to bed 1.6 hours later on weekends, and the non-ADHD group averaged 2.4 hours later bedtimes ($p < .06$), according to parent's responses on the CSQ. The experimental manipulation, keeping the children up two hours past their bedtime, seemed to replicate, to some degree, the real life experiences of our entire sample. A further limitation of the study was that the amount of sleep deprivation was not sufficient to produce decrements in alertness and, in turn, performance.

Parent reports were correlated significantly with polygraph recordings of sleep. For example, the longer it took to fall asleep after being kept up late (SOL Night 2) was positively, moderately associated with higher parental ratings of morning difficulties ($r = .52, p < .01$), accounting for a substantial 27% of the variance. Unlike with adult and adolescent controls (Carskadon & Dement, 1977; 1987), SOL was not a good indicator of sleep need and prior sleep. This suggests that, for some children, despite a growing need for sleep, difficulties going to sleep persists and have an impact on parental ratings of morning behaviors. Morning ratings were also moderately, inversely associated to percentage of Stage 2 sleep. Less sleep meant more morning difficulties that were reported by parents ($r = -.54, p < .05$). Findings of the study show that parent ratings corresponded to objective variables in meaningful ways.

In Table 9, we see the correlations between the parents' reports of sleep difficulties and the movement indexes. Taking a closer look at the relationships between sleeping activity levels and parent reports of sleep-related behaviors, several significant relationships were found. Two types of movements, the L Index and R Index, were moderately associated with daytime problems on the CSQ. As the number of these movements increased, parent ratings decreased. These data suggest the children slept

through these L and R type movements. Since the children slept well, they were rated as functioning well. Their sleep was not disturbed by these movements.

The difference in weekday and weekend sleep schedules is moderately positively related to REM % following partial sleep deprivation. The later parents reported children slept on the morning, the higher percentage of REM sleep they had on the second night. This suggests parents can predict Delayed Sleep Phase Syndrome (DSPS), a sleep disorder involving sleep difficulties due to differences in weekday and weekend sleep schedules. Greater sleep need caused by DSPS can lead to the sleep-related difficulties listed in the CSQ.

Parents reports of their children's bedtime problems were positively, moderately associated with percentage of Stage 3 after partial sleep deprivation ($r = .64, p < .01$). Only when sleepiness reaches a threshold following additional sleep loss, is the result better sleep and increased sleepiness. The chronic sleep debt that some children had built up was discharged with further sleep deprivation. At home, later wake up times may compensate for the effects of some sleep loss.

Results of the Child Sleep Questionnaire support some findings of overall higher levels of restlessness reported by parents (Rapaport, Behar & Bunney, 1981; Porrino et al, 1983) and others. After factor analysis to reduce the CSQ to its most highly predictive questions, the CSQ should be normed with a large standardization sample, and used to reliably identify children at risk or having sleep-related difficulties.

Hypothesis 2: This research question asked about the relationship between parental reports of their children's sleep to nightly recordings of sleep. Here, the study compared parent's "subjective" perceptions of their child's sleep to the "objective" assessment of sleep by NPSG recordings. The study findings showed some significant relationships between parent reports found between some NPSG variables, and a logical correspondence between them.

Overall, parents of ADHD children in our sample did not report much different levels of sleep related disturbances than parents of controls on the questionnaire, but for the entire sample, parents ratings of bedtime and morning behaviors were related to some sleep stage percentages. A recommendation to parents of an ADHD child having trouble

with sleep would be to first make sure to restrict weekend bedtimes to no more than an hour past weekday bedtimes.

Power Analysis for CSQ and NPSG and MSLT Relationships

Power was also determined to find relationships between the subjective and objective sleep variables. That there is a one in four chance of finding significance with this sample size reflects the weak power of the study to reject the null hypotheses. A 26% chance of finding any significant relationships between parental reports and empirical recordings of sleep signifies the inconclusive nature of the findings of no differences. Sixty-nine (69) participants for each group, or a total sample of 138 participants would be required to have a 70% chance of finding between significant relationships. Follow-up research with a larger sample could develop and refine the Child Sleep Questionnaire to quickly and accurately reflect sleep problems.

Daytime Sleepiness

This findings of this study supports previous findings that preadolescent children and “DAMP” children are not sleepy during the day (Carskadon, Keenan & Dement, 1987; Palm, 1992). Both groups were quite alert on the first day, as seen in their means for SOL, on MSLT 1 (ADHD group mean = 22 minutes; non-ADHD mean = 23 minutes, NS). Means greater than twenty minutes to fall asleep indicates a high level of alertness throughout the day. Table 10 shows the Time main effects found; that both groups had significantly lower Sleep Latency and increased Number of times they fell asleep during MSLT 2. Overall, after the sleep loss condition, the participants were not impaired and did not seem sleepy during the day, but did show statistically significantly less alertness on the MSLT.

No differences between ADHD and non-ADHD groups on MSLT sleepiness variables of sleep onset latency and number of actual naps for either day of nap opportunities.

In Tables 11 and 12, we can see that only a few significant correlations out of 60 were found between NPSG and MSLT variables, including leg movements. These correspondences are interesting but represent very preliminary findings that need to be

assessed and followed up on. However, the patterns of those who are sleepy on MSLT 1, the baseline measure, were correspondingly sleepier on the post-sleep loss day' measures ($r = .59, p < .01$ for MSLT 1 and MSLT 2). Those who have a tendency towards sleepiness the first day, continue to experience difficulties after staying up late at night.

Hypothesis 3

Do ADHD children become sleepier than non-ADHD from sleep loss? It was proposed that there may be differences in the sleep of ADHD children that leaves them vulnerable to further sleep loss. Here, the data do not support this hypothesis, there was no difference found between the groups.

However, overall, participants did have significantly lower means on MSLT 2, suggesting that two hours of sleep loss has some impact on their high levels of alertness seen during Day 1.

Performance on the T.O.V.A.™

During Saturday and Sunday, levels of daytime cognitive performance was also assessed with two administrations of a computerized visual performance task, the Test of Variables of Attention (T.O.V.A.™). After the first 25 minute nap opportunity of the day, all children took the approximately 23 minute long computer task. A boring, redundant and slow test, the children generally disliked it, although none refused to take it. Observers sat in the room, behind the children and rated them using a series of Likert scales. Here, all the children were overall, quite alert, and not sleepy, as seen in their high means for Sleep Onset Latency on the MSLTs, as discussed previously. So their performance on the T.O.V.A.™ needs to be considered understanding that this was a time chosen for peak performance, as if they were in school after settling in for a while, but well before lunchtime.

The study found no differences between the groups or an interaction effect, but in general, all participants had significant decrements in performance over time, after partial sleep deprivation, as indicated by significantly higher commission errors and variability means. Post hoc analysis shows a higher number of "impulsive" errors and variability between Trial 2 and the post sleep loss Trial 3. There were no differences over time for

errors of omission. One raw score variable, percentage of correct responses, showed significant differences. Post hoc tests show percentage of correct responses continually declined for all participants, between Trial 1 and Trial 2, as well as between Trial 2 and Trial 3. The study also found that observers rated the ADHD group as more easily distracted by noises and sounds than the non-ADHD group, but these differences were not related to the sleep loss.

The findings of the study show a small number of significant relationships between leg movement variables and T.O.V.A.TM variables. These findings merely suggestive findings that need replication, but interestingly, the study found the RC Index was significantly related with eight out of thirty-six T.O.V.A.TM variables, more than any other movement variable. RC movements were moderately, positively associated with commission errors and variability on the first trial. These errors predicted, to some degree, which children would have high levels of this movements.

Hypothesis 4

Do ADHD criteria behaviors worsen or increase as a result of sleep loss? The study asked this research question about the impact of sleep loss and if increased sleepiness might result in further increases in ADHD—criteria behaviors. To test this hypothesis ADHD group's performance on the T.O.V.A.TM before and after the sleep loss condition was compared. The findings of the study do not support this hypothesis, as the ADHD group means from pre- to post sleep loss trials were not different than the non-ADHD children.

Hypothesis 5

Does the effect of sleep loss in controls resemble ADHD behaviors? The study asked this final and interesting research question about the impact of sleep loss: Do decrements in alertness result in ADHD-like behaviors in the control group? The non-ADHD group's performance on the T.O.V.A.TM before and after the sleep loss condition were compared. Some of the findings of the study speculatively support this hypothesis. In our sample, the controls made more post sleep loss commission errors on the T.O.V.A.TM than ADHD children, indicating greater levels of impairment in attention

(ADHD group mean commission errors = 86; non- ADHD group mean = 101 errors, $p < .03$)

The percentage of correct responses on the T.O.V.A.TM also showed impairment, the non-ADHD group mean decreased from 33% correct responses on Trial 2 to 25% on the post sleep loss Trial 3. The ADHD group stayed virtually the same (Trial 1 = 37%, Trial 2 = 36%). Considering that the children were, overall, quite alert, but they did experience decreases in time to fall asleep after the sleep loss suggests that two hours of sleep loss has some effect on level of daytime alertness.

The data suggest there may be an impact of two hours partial sleep deprivation on the T.O.V.A.TM for the non-ADHD group but not the ADHD group, who may be more accustomed to coping with tasks they find boring. ADHD children usually do worse on CPT tasks, so the preliminary findings here suggest an interesting direction for future research, looking at the impact of sleep loss on cognitive performance in preadolescent children.

After sleep loss omission errors went down and commission errors went up. This pattern of errors was made as the children got bored and began to quickly and continually pressed the button, regardless of the target. The moderate impairment in this ADHD group may have enabled them to perform better than a more typical clinical ADHD group. Also, the moderate impairment is reflected in the findings. Certainly some children with higher levels of symptomatic behaviors would have refused to complete the T.O.V.A.TM. The T.O.V.A.TM also may not, like the CBC or the CPRS-48, discriminate between mild ADHD and controls as well as it does with other ADHD children.

Summary Of Findings

There were clear differences between the groups in the mean number of ADHD behaviors the participants were reported by parents. The children differed according to group status on number of cognitive, academic and behavioral scales, they showed no differences on neuropsychological scales and the Performance domain IQ scales. All the children were boys, between eight and twelve years old, almost all were Latino.

One significant sleep difference was found for the Subject x Time Interaction effect. ADHD children had significantly higher percentages of transitional, lighter Stage

1 sleep on the post sleep loss night of recordings, counter to the expectable response of the non-ADHD group, who had lower Stage 1%.

Parents reports did correlate somewhat with empirical recordings. The study findings showed some significant relationships found between some CSQ scales and NPSG variables. For the entire sample, parents ratings of bedtime and morning behaviors were related to some sleep stage percentages.

The data do not support the hypothesis that the ADHD children would become sleepier than non-ADHD from sleep loss. There was no difference found between the groups. However, all participants, did have significantly lower Sleep Latency means on MSLT 2, suggesting that two hours of sleep loss can have some impact on the high levels of alertness seen during the first day.

The ADHD group means on the T.O.V.A.[™] from the pre to post sleep loss trials were actually impacted less than the non-ADHD means. The hypothesis that the ADHD group's performance on the T.O.V.A.[™] after the sleep loss condition would be impacted more severely than the non-ADHD group was not supported by the data. In fact, the non-ADHD children made more errors on the T.O.V.A.[™] than ADHD children after partial sleep deprivation.

The major drawback to this, or any pediatric ADHD sleep study, is the use of a small sample size. The power analysis indicates there was only a 15% of finding differences between the groups, and only a 26% chance to find relationships. The weak power of the study to find differences and to determine relationships means there was a small chance of finding differences in the sleep, or alertness levels of ADHD children that may really exist.

This presents an alternative explanation of the differences between inconsistent NPSG research findings and parent reports of ADHD sleep. Rather than postulating sub-clinical differences that emerge only episodically, the serious limitations inherent in the power of the small samples typically used in children's sleep research, produces mostly inconclusive results. The preliminary findings here suggest that further research is indicated to follow up investigating the interesting relationships between sleep loss and alertness in non-ADHD children.

APPENDICES

APPENDIX A Subject Consent Form

**PARENT AGREEMENT TO PARTICIPATE IN A STUDY TITLED:
Daytime Sleepiness and Attention-Deficit Hyperactivity Disorder.**

**Study Director: Stuart R. Cantor, MA
City University of New York
Clinical Psychology Doctoral Program**

PLEASE READ THE FOLLOWING CAREFULLY BEFORE SIGNING

I agree to allow my child to participate in a research study in which s/he will sleep for two nights in The City College Sleep Disorders Center.

- 1) I have been told that this agreement does not mean s/he is eligible for this study. In order to participate my child must meet a number of requirements and I am willing to have my child evaluated to determine if s/he qualifies.**
- 2) I have been told that the evaluation involves the following:**
 - a) my filling out sleep and activity level logs for my child.**
 - b) an interview with Dr. Arthur J. Spielman, the Director of the Sleep Disorders Center.**
 - c) an interview by a physician.**
 - d) an interview by a psychologist or a doctoral level clinical psychology student and a number of personality tests.**
 - e) one week before coming into the sleep lab, you will be asked to discontinue giving your child Ritalin or other medication s/he may be receiving.**
 - f) you will be asked to have your child's physician sign a consent form approving the safety of discontinuing this medication for one week.**
 - g). I have been told that myself, the child's other parent or some other familiar adult will come to the sleep laboratory of The City College of New York Sleep Disorders Center with my child for the evaluation tests. I have been told that on that this evaluation includes the tests mentioned above, and my child may have a few electrode sensors applied to his / her scalp, face and legs to record brain waves, eye movements, and muscle activity. S/he may be asked to lay down in the laboratory bedroom and have the door closed. Then my child and I will go home.**
 - h) I have been told that s/he may be disqualified from participation at this point in the evaluation process. If disqualified, no further evaluation will be performed.**
- 3) If your child qualifies:**
 - a) The study begins with you returning with him / her within two weeks and spending two consecutive nights and days in the Sleep Disorders Center. These nights and days include having electrode sensors applied to his / her scalp, face and legs to record brain waves, eye movements, and muscle activity. There may also be sensors placed on his / her chest to measure his / her**

heart rate. On the first night only, there may also be a loosely fitting device around his / her waist, and sensors around his / her nose and mouth to measure airflow. These sensors will be removed after the first day and reapplied before sleeping the second night in the lab.

b) In addition, s/he will spend two days in the sleep laboratory following each of the two nights. S/he will be asked to take some tests and be asked to take a series of brief naps. Also, you will be asked to allow your child to stay up about two hours later than their usual bedtime before the second night in the lab, watching videos or doing arts and craft activities. During these two days, you will be asked to rate your child's activity level.

c) If my child is accepted into the study after the first night, and stays a second night and two days in the lab, I have been told that s/he will receive either \$50 in cash or as a \$50 gift certificate. S/he will have a choice of gift certificates from either Barnes and Noble Bookstore or Herman's Sports.

4) I have been told that either my child or I may not get as much sleep in the laboratory as we are used to. This may result in some minor daytime fatigue or sleepiness. I have been told that if I become too sleepy I should not operate a motor vehicle or heavy equipment.

b) I have been told that Arthur J. Spielman, PhD, Director of The City College of the City University of New York Sleep Disorders Center is supervising the Study Director: Stuart R. Cantor, MA, Adjunct Faculty, Department of Psychology and Psychotherapist, The Psychological Center of The City College of New York. Dr. Spielman is available at the following telephone numbers: 212) 650-5397, or 212) 832-1544.

5) I have been told that my child's participation in this study is completely voluntary and that refusal to participate will not prejudice me or my child's further treatment or future relations with the University, the Sleep Disorders Center, or its doctors. I have been told that I may discontinue my child's participation at any time and that this will involve no penalty or loss of benefits.

6) **BENEFITS AND RISKS:** I have been told that s/he will, after acceptance into the study and upon completion of the second night, receive a small monetary payment in the form of \$50 in cash or a gift certificate. A gift certificate from either Barnes and Noble Bookstore or Herman's Sports may be chosen.

b) **Transportation to and from the lab will be paid for.**

c) My child and I will receive 3 meals during each of the two days we stay in the laboratory. Other than these above mentioned benefits, I can expect no benefit directly to me or my child. The study is being conducted to further our understanding of the sleep process and its relationship to daytime sleepiness in ADHD children and may have some application in the future. At the end of the procedure any and all questions will be answered in complete detail in a debriefing sessions for both parents and children.

d) I have been told that occasionally skin abrasions occur where the electrodes (used to monitor sleep) are applied. The technician on duty will have ointment available if this occurs. As mentioned, sleeping in the lab may reduce the amount of sleep parents or children receive. This may cause sleepiness. If this occurs, returning to the habitual sleep schedule should quickly reverse the fatigue and sleepiness.

7) I understand that in the unlikely event of physical injury resulting from the research procedures used in this study, only immediate essential treatment as determined by the technician on duty or Dr. Spielman will be available for the injury without charge to me personally. There will be no monetary compensation.

8) I have been told that the results of all these evaluation and sleep studies will be kept strictly confidential. Any publications of the results of this study will not include any information that could identify me or my child. Most results will be combined in group form.

Signature of Parent

(Parent)

Print Name

Date

Signature of Witness

Print Name

Date

APPENDIX B DISC Interview Summary Sheet

**PARENT DISC (3.0) INTERVIEW
DSM-IV SYMPTOM CHECKLIST OF DISRUPTIVE BEHAVIOR
DISORDERS**

Name: _____ Subject # _____ D.O.B. _____ Age: _____ Date: _____
Interviewer: _____ Grade: _____ Respondent: _____

ATTENTION DEFICIT HYPERACTIVITY DISORDER (ADHD)

HYPERACTIVE/IMPULSIVE SYMPTOMS (NEED 6 OF 9)			INATTENTIVE SYMPTOMS (NEED 6 OF 9)		
SYMPTOM	HOME	SCHOOL	SYMPTOM	HOME	SCHOOL
Fidgets/Squirms in seat	(3) _____	(4) _____	Careless mistakes(36B)	_____	(37A) _____
Can't stay in seat	(5) _____	(6) _____	Disorganized	(38B) _____	(39B) _____
Climbs/runs around	(7) _____	(8) _____	Easily distracted	(40A) _____	(41A) _____
Overly noisy	(11) _____	(12) _____	Can't concentrate(42/4A)	_____	(43/5A) _____
Very talkative	(13C) _____	(14A) _____	Doesn't listen	(46B) _____	(47A) _____
Interrupts/butts in	(15/17A) _____	(16/18A) _____	Needs reminding	(48C) _____	(49A) _____
Blurts out responses	(19A) _____	(20A) _____	Loses things	(50B) _____	(51A) _____
Trouble waiting turn	(21/23A) _____	(22/24A) _____	Forgetful	(52A) _____	(53A) _____
Always moving	(25A) _____	(26A) _____	Can't pay attention(54A)	_____	(55A) _____

+++++

HYPERACTIVE SYMPTOMS: TOTAL NUMBER _____

27	Symptoms impair social functioning?	YES	NO
28/29	Impair relationship with caregiver?	YES	NO
30/31	Impair functioning at school?	YES	NO
32	Global age of onset < 7 at home?	YES	NO
33	Global age of onset < 7 at school?	YES	NO

INATTENTIVE SYMPTOMS: TOTAL NUMBER _____

56	Symptoms impair social functioning?	YES	NO
57/58	Impair relationship with caregiver?	YES	NO
59/60	Impair functioning at school?	YES	NO
61	Global age of onset < 7 at home?	YES	NO
62	Global age of onset < 7 at school?	YES	NO

WAS AGE OF ONSET PRIOR TO 7 YEARS OLD? YES NO

IS THERE IMPAIRMENT AT HOME AND AT SCHOOL? YES NO

(YES must have positive response to at least one of questions 27, 28, 57, 58 AND positive response to at least one of questions 30, 31, 59 or 60)

DOES CHILD MEET CRITERIA FOR NON-ADHD? YES NO

(i.e., < 9 ADHD symptoms with < 6 symptoms in either domain, inattention or hyperactivity)

OPPOSITIONAL DEFIANT DISORDER (ODD) (MUST HAVE 4 OF 8)

<u>SYMPTOM</u>		<u>SYMPTOM</u>	
Loses temper (67-C)_____	Blames others for own mistakes (72-C)_____		
Argues with adults or talks back (68-C)_____	Is grouchy/easily annoyed (73-C)_____		
Does things not supposed to/refuses (69/70-C)_____	Angry or resentful(74-C)_____		
Deliberately annoys others/messing things(71-C)_____	Gets even by telling lies or hurting (75-C)_____		
78 Interfere with relationship with caregiver?		YES	NO
79 Symptoms interfere with peer relationships?		YES	NO
80/81 Symptoms cause problems at school?		YES	NO
82 Global age of onset for symptoms _____ years old			
TOTAL NUMBER SX? _____	DOES CHILD MEET CRITERIA FOR ODD?	YES	NO

CONDUCT DISORDER (CD) (MUST HAVE 3 OF 15 SYMPTOMS)

<u>SYMPTOM</u>		<u>SYMPTOM</u>	
Stolen with confrontation (90)_____	Threatens/Intimidates others (109)_____		
Age of onset _____	Physical fights frequently (110-A)_____		
Shoplifted/Stole no one saw(95)_____	Threatened with weapon (111-A)_____		
Ran away overnight more (97-A)_____			
more than once: NO YES	Hurt other with weapon (112-A)_____		
more than two weeks NO YES	Physically cruel to other (113-C)_____		
Lied to get something/or (100)_____	School suspensio (119/119-A)_____		
to get out of something	School expulsion (120) _____		
Started Fires(102-C)_____	Belongs to gang (121) _____		
Skipped Class/Played Hookey(103-A)_____			
Stayed out > 2 Hrs. Late (104-A)_____			

SERIOUS RULE VIOLATION:

Often abuses curfew before 13 y. o. (104-A)_____	Broke into House/Bldg./car(105-A)_____
Runaway overnight at least (97-A) _____	twice or once/extended period of time
Broke things purposefully (106-A)_____	Is truant frequently before 13 y. o. (103-A)_____
Cruel to animals (107-A)_____	

DESTRUCTION OF PROPERTY:

Intentional fire setting (102-C)_____	Deliberate destruction of property (106-A)_____
---------------------------------------	---

AGGRESSION AGAINST PEOPLE/ANIMALS:

Bullies/threatens/intimidates (109)_____
Physical fights often (110-A)_____

DECEITFULNESS OR THEFT:

Threatened with weapon (110-A)_____	Broke into house/car(105)_____
Hurt other with weapon (110-A)_____	Lies to get things/get out of things(100)_____
Physically cruel to animals (110-A)_____	Stolen/without confrontation(95)_____
Physically cruel to people (110-A)_____	
Stolen with confrontation (110-A)_____	

TOTAL NUMBER SX? _____ DOES CHILD MEET CRITERIA FOR CD? YES NO

DOES CHILD MEET CRITERIA FOR ADHD INATTENTIVE TYPE?	YES	NO
DOES CHILD MEET CRITERIA FOR ADHD HYP./IMPULSIVE TYPE?	YES	NO
DOES CHILD MEET CRITERIA FOR ADHD COMBINED TYPE?	YES	NO

DOES CHILD MEET CRITERIA FOR ODD (i.e., 4/8 symptoms)?	YES	NO
DOES CHILD MEET CRITERIA FOR CD? (i.e., 3/15 symptoms)?	YES	NO

APPENDIX C Conners Parent Rating Scale-48

Parent's Questionnaire

Name of Child _____ Date _____

Please answer all questions. Beside each item below, indicate the degree of the problem by a check mark (✓)

	Not at all	Just a little	Pretty much	Very much
1. Picks at things (nails, fingers, hair, clothing).				
2. Sassy to grown-ups.				
3. Problems with making or keeping friends.				
4. Excitable, impulsive.				
5. Wants to run things.				
6. Sucks or chews (thumb; clothing; blankets).				
7. Cries easily or often.				
8. Carries a chip on his shoulder.				
9. Daydreams.				
10. Difficulty in learning.				
11. Restless in the "squirmy" sense.				
12. Fearful (of new situations; new people or places; going to school).				
13. Restless, always up and on the go.				
14. Destructive.				
15. Tells lies or stories that aren't true.				
16. Shy.				
17. Gets into more trouble than others same age.				
18. Speaks differently from others same age (baby talk; stuttering; hard to understand).				
19. Denies mistakes or blames others.				
20. Quarrelsome.				
21. Pouts and sulks.				
22. Steals.				
23. Disobedient or obeys but resentfully.				
24. Worries more than others (about being alone; illness or death).				
25. Fails to finish things.				
26. Feelings easily hurt.				
27. Bullies others.				
28. Unable to stop a repetitive activity.				
29. Cruel.				
30. Childish or immature (wants help he shouldn't need; clings; needs constant reassurance).				
31. Distractibility or attention span a problem.				
32. Headaches.				
33. Mood changes quickly and drastically.				
34. Doesn't like or doesn't follow rules or restrictions.				
35. Fights constantly.				
36. Doesn't get along well with brothers or sisters.				
37. Easily frustrated in efforts.				
38. Disturbs other children.				
39. Basically an unhappy child.				
40. Problems with eating (poor appetite; up between bites).				
41. Stomach aches.				
42. Problems with sleep (can't fall asleep; up too early; up in the night).				
43. Other aches and pains.				
44. Vomiting or nausea.				
45. Feels cheated in family circle.				
46. Boasts and brags.				
47. Lets self be pushed around.				
48. Bowel problems (frequently loose; irregular habits; constipation).				

Conners Parent Rating Scale (CPRS-48) Summary Sheet

T Scores	Name:		Age		Grade		Subject #		Rater:		Interpretation for T-scores:		
	A. Conduct Problem		B. Learning Problem		C. Psycho-somatic		D. Impulsive-Hyperactive		E. Anxiety			F. Hyper-activity Index	
	6/8	9/11	6/8	9/11	6/8	9/11	6/8	9/11	6/8	9/11		6/8	9/11
94		16							12		20		
93	13										21		
92													
91		15	8	8								19	
90									11		20		
89	12				5	5							
88		14										18	
87							12	12	12		19		
85				7						10	18	17	
84	11	13	7										
83								11	11				
82											17	6	
81		12					11			9			
80	10				4	4					16	15	
79								10	10				
78			6	6									
77		11											
76	9						10			8	15	4	
75													
74		10											
73													
72													
71													
70		9						8			13	2	
69													
68							8		7	6			
67	7	8									12	11	
66								7				Much Above Average	
65				4							11		
64							7		6			10	
63		7								5		Above Average	
62	6					2		2		6	10		
61												9	
60		6							5				
59							6			4	9	Slightly Above Average	
58	5		3	3				5				8	
57									4				
56		5									8		
55							5			3		7	
54								4					
53	4	4				1		1		3	7		
52												6	
51			2	2			4						
50		3							2	2	6	Mean	
49	3							3				5	
48										1		Average	
47		2					3				5		
46												4	
45			1	1				2	1		4		
44	2					0		0				3	
43													
42		1					2				3	Slightly Below Average	
41								1	0	0		2	
40	1												
39		0		0							2		
38			0				1					1	
36								0			1	Below Average	
35	0											0	
34							0				0	Much Below Average	

Scale Scores:

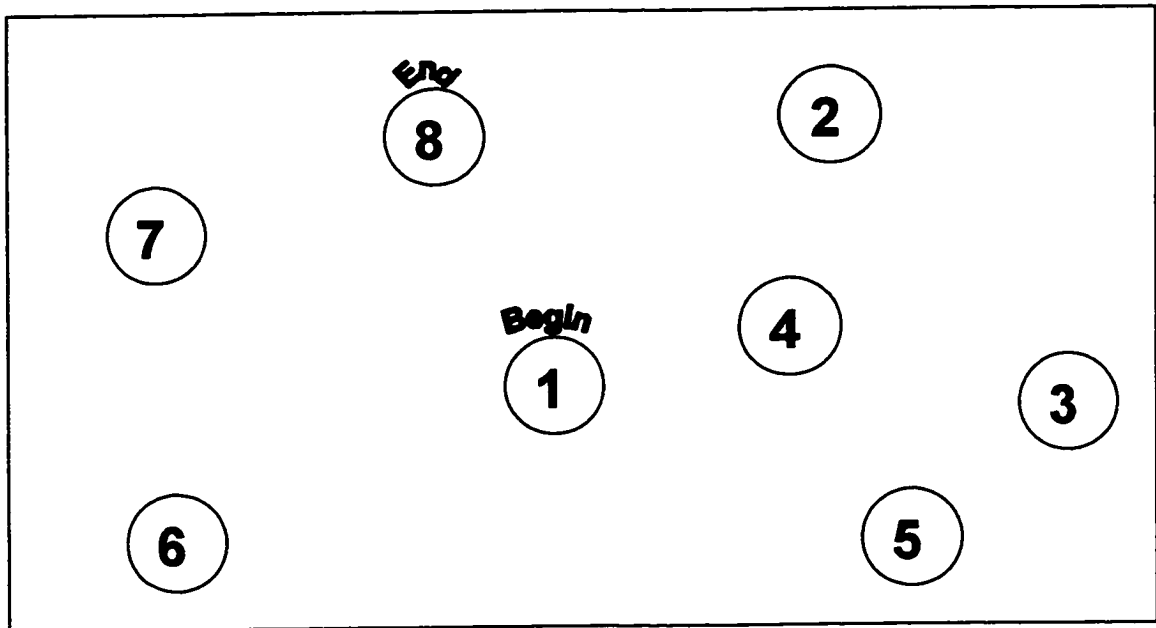
T-scores:

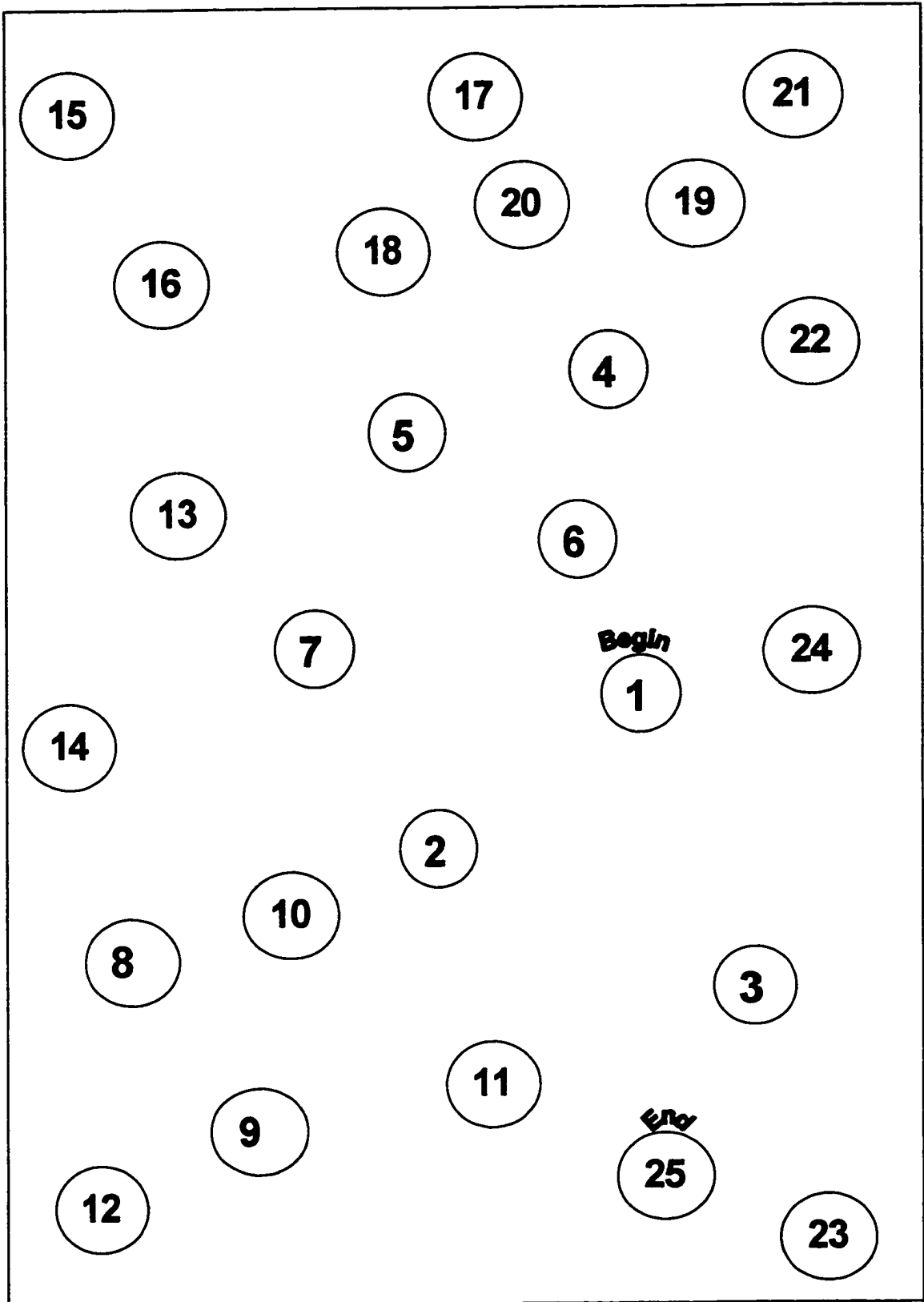
APPENDIX D Trail Making, Stroop Tests and Summary Sheet

TRAIL MAKING

Part A

SAMPLE

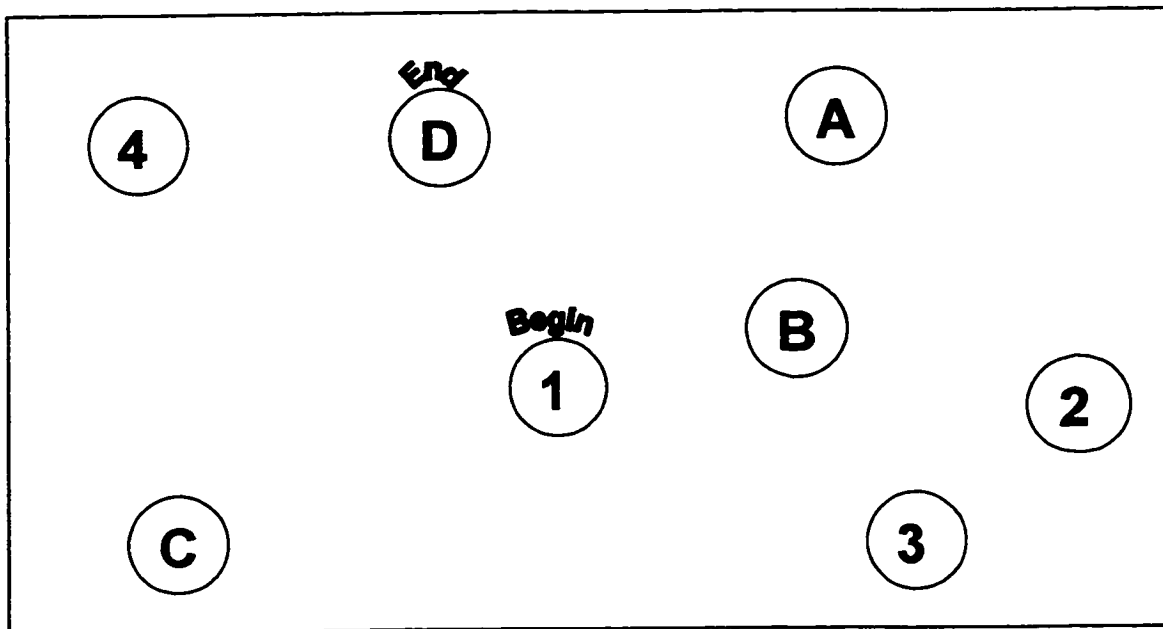


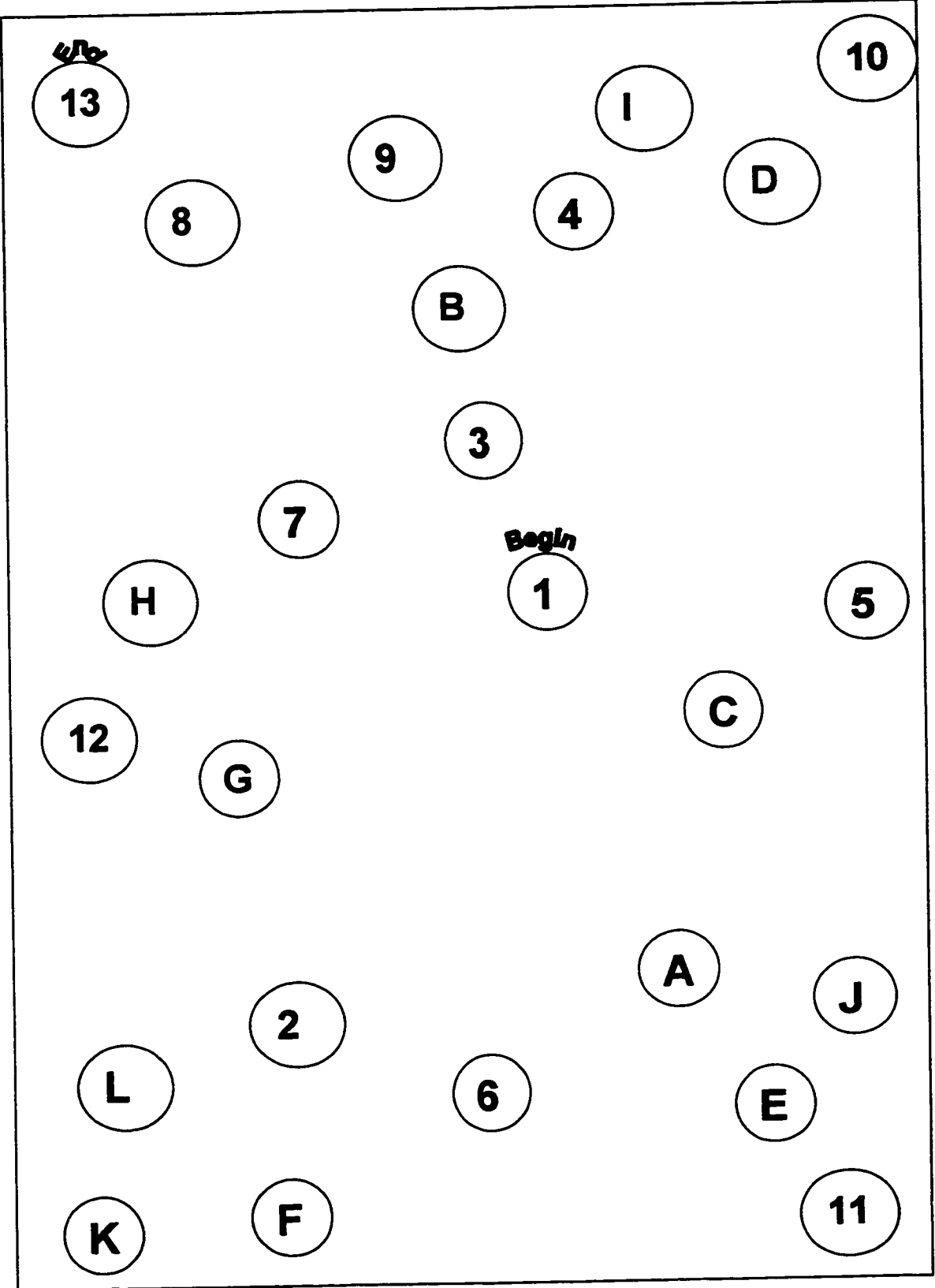


TRAIL MAKING

Part B

SAMPLE





Neuropsychological Tests Scoring Sheet
ADHD Study
The Sleep Disorder Center, CCNY

NAME: _____ TESTER: _____ DATE: _____

I. Stroop Color and Word Test

1. Word Reading:

R B G R B
G G R B G
B R B G R
G B R R B
R R G B G
B G B G R
R B G B G
B G R G R
G R B R B
B G G B G
G R B R R
R B R G B
G R B R G
B B R G R
R G G B B
B B R G R
R G B R G
G R G B B
R B R G R
G R G B G

2. Color Naming (Xs):

B R B G R
R B G R B
G G R B G
R B G B G
G G R R B
R B G B G
G G R G R
R R B R B
B B G B G
R R R G B
B B G B G
G G B R R
R B R B B
G G G R G
B R B G R
G G G B B
B R R G R
R B B R G
G R G B B
B G B R R

3. Color Naming (Words)

B R B G R
R B G R B
G G R B G
R B G B G
G G R R B
R B G B G
G G R G R
R R B R B
B B G B G
R R R G B
B B G B G
G G B R R
R B R B B
G G G R G
B R B G R
G G G B B
B R R G R
R B B R G
G R G B B
B G B R R

Word Score = _____

Color Score = _____

Color-Word Score = _____

II. Trail Making Test

1. Part A: Time: _____ Error: _____
2. Part B: Time: _____ Error: _____

APPENDIX E

Your Child's Sleep Questionnaire

Your Name: _____ Child's Name _____
 Address: _____
 Ages: _____ Ethnicity _____

Bedtime Questions

In general, does your child have difficulty falling asleep? Yes No
 How often does it take a long time for your child to go to sleep? _____ # of times/month

In general, is it hard for your child to settle down to go to sleep? Yes No
 How often does he/she have trouble falling asleep? _____ # of times/month

In general, is it hard to put your child to bed? Yes No
 How often is your child unwilling to go to bed? _____ # of times/month
 How often does your child stay up later than you want? _____ # of times/month

In general, is your child cranky at bedtime? Yes No
 How would you describe their bedtime behavior?

Please rate how difficult it is for your child to fall asleep:

 very difficult not difficult at all

Please rate how difficult it is for your child to settle down at bedtime:

 very difficult not difficult at all

Please rate how difficult it is for you to put your child to bed:

 very difficult not difficult at all

Please rate how unwilling your child is to go to bed:

 very unwilling not unwilling at all

Please rate how cranky your child is:

 very cranky not cranky at all

Nighttime Sleep Questions

In general, is your child a sound sleeper? Yes No
 In general, does your child sleep through the night? Yes No

In general, is your child a restless sleeper? Yes No
 If yes, how often does your child wake up at night? _____ # of times/month
 In general, will your child fall back asleep quickly? Yes No

In general, is your child a deep or light sleeper? Light Deep
 In general is your child easily disturbed or awakened? Yes No

In general, how long does it take for your child to fall asleep? _____ # minutes/night
 In general, about how long does your child sleep each night? _____ # of hours/night

In general, what time does your child go to sleep at night? _____ o'clock
 In general, what time does your child get up in the morning? _____ o'clock

In general, does your child sleep later on the weekends/vacations? Yes No
 If yes, how much later than usual? _____ # of hours/night

Does your child have (or ever had) any of the following sleep difficulties:

Does anyone in your family have (or ever had) any of the following sleep difficulties? Name(s):
Your child Other children Other family members How often?

- Wets the bed?
- Sleepwalks?
- Talks Sleep?
- Snores loudly?
- Night terrors?
- Nightmares?
- Insomnia?
- How would you describe your child's and/or family member's insomnia?

How would you describe your child's sleep at night

Please rate how deep or light your child sleeps:

 very light very deep

Please rate how easily disturbed your child is:

 very easily disturbed not easily disturbed at all

Please rate how restless he/she is:

 very restless not restless at all

Please rate how long it takes for your child to fall asleep:

 very long time very quickly

Morning Questions

In general how difficult is it for your child to get out of bed in the morning?
 How often is it hard for your child to get out of bed? _____ # times/month

In general, how difficult is it for your child to "get going" in the morning?
 In general, how is your child's appetite in the morning?
 In general, would you describe your child as a "morning lark" or a "night owl"?

In general, does your child seem alert or sleepy in the morning?
How often does your child complain of being very sleepy in the morning? _____ # times/month

Please rate how difficult it is for your child to get out of bed:
|_____|
very difficult not difficult at all

Please rate how difficult it is for your child to "get going" in the morning
|_____|
very difficult not difficult at all

Please rate your child's morning appetite:
|_____|
very poor very good

Please rate how alert or sleepy your child seems on most mornings::
|_____|
very sleepy very alert

Daytime Questions

In general, does your child seem rested during the day?

In general, does your child complain of being sleepy in the day?
If yes, how often? _____ # times per month

Does your child nap in the afternoon?
If yes, _____ # times/week
If yes, _____ # minutes/nap

Does your child seem to slow down after lunch and pick up later?
If yes, how often? _____ # times per month

Does your child yawn a lot during the day? Yes No

In general does your child seem drowsy during the day? Yes No

Please rate how rested your child seems during the day:
|_____|
very tired very rested

How sleepy during the day does your child seem to you?
|_____|
very sleepy not sleepy at all

Please rate your child's daytime appetite:
|_____|
very poor very good

APPENDIX F Child Sleep Log

Daytime Sleepiness & ADHD Sleep Log
Name _____

Day and Date										
SLEEP LAST NIGHT: Were you in bed before going to sleep last night (eg, watching, TV, reading)? If yes: the time you got into bed last night.										
The time you turned the lights out or began trying to fall asleep?										
How long it took you to fall asleep.										
The time you finally got out of bed this morning?										
Your estimate of the total amount of time you were asleep last night?										
Number of times you woke up?										
Time you last woke up and didn't go back to sleep?										
Total time you were physically out of bed and awake during the night?										
List the type and dosage of medicine you took last night? What time?										
Rate how difficult it was waking up this morning: 1.....2.....3.....4.....5 not difficult very difficult										
YESTERDAY: What time did you first go outdoors? How long were you outside in the morning?										
Number of naps or number of brief episodes of dozing yesterday?										
Total amount of sleep while napping or dozing yesterday?										
Total number of cups of coffee and or cans of caffeinated beverages yesterday?										
Did you exercise yesterday? If YES the time of day was the: Mom/Midday/Eve										
Rate your alertness yesterday 1.....2.....3.....4.....5 least alert most alert										
Rate your fatigue yesterday 1.....2.....3.....4.....5 least fatigued most fatigued										

APPENDIX G MSLT Summary Sheet

/ S.#	MSLT 1	Sat.	MSLT 2	Sun.
NAP 1 TIME:			NAP 1 TIME:	
L/OUT		Sleep?	L/OUT	Sleep?
L/ON		Duration?	L/ON	Duration?
Bed Time(B/T)		Latency Est.?	(B/T)	Latency Est.?
Sleep Time (S/T)		Dream?	(S/T)	Dream?
Sleep Latency (SOL)		Refreshed?	(SOL)	Refreshed?
NAP 2 TIME:			NAP 2 TIME:	
L/OUT		Sleep?	L/OUT	Sleep?
L/ON		Duration?	L/ON	Duration?
(B/T)		Latency Est.?	(B/T)	Latency Est.?
(S/T)		Dream?	(S/T)	Dream?
(SOL)		Refreshed?	(SOL)	Refreshed?
NAP 3 TIME:			NAP 3 TIME:	
L/OUT		Sleep?	L/OUT	Sleep?
L/ON		Duration?	L/ON	Duration?
(B/T)		Latency Est.?	(B/T)	Latency Est.?
(S/T)		Dream?	(S/T)	Dream?
(SOL)		Refreshed?	(SOL)	Refreshed?
NAP 4 TIME:			NAP 4 TIME:	
L/OUT		Sleep?	L/OUT	Sleep?
L/ON		Duration?	L/ON	Duration?
(B/T)		Latency Est.?	(B/T)	Latency Est.?
(S/T)		Dream?	(S/T)	Dream?
(SOL)		Refreshed?	(SOL)	Refreshed?
NAP 5 TIME:			NAP 5 TIME:	
L/OUT		Sleep?	L/OUT	Sleep?
L/ON		Duration?	L/ON	Duration?
(B/T)		Latency Est.?	(B/T)	Latency Est.?
(S/T)		Dream?	(S/T)	Dream?
(SOL)		Refreshed?	(SOL)	Refreshed?

Sleep Latency M_1 = # Sleep Episodes =	Sleep Latency M_2 = # Sleep Episodes =
---	---

APPENDIX H

Examples of Sleep Stages (Rechtschaffen & Kales, 1968)

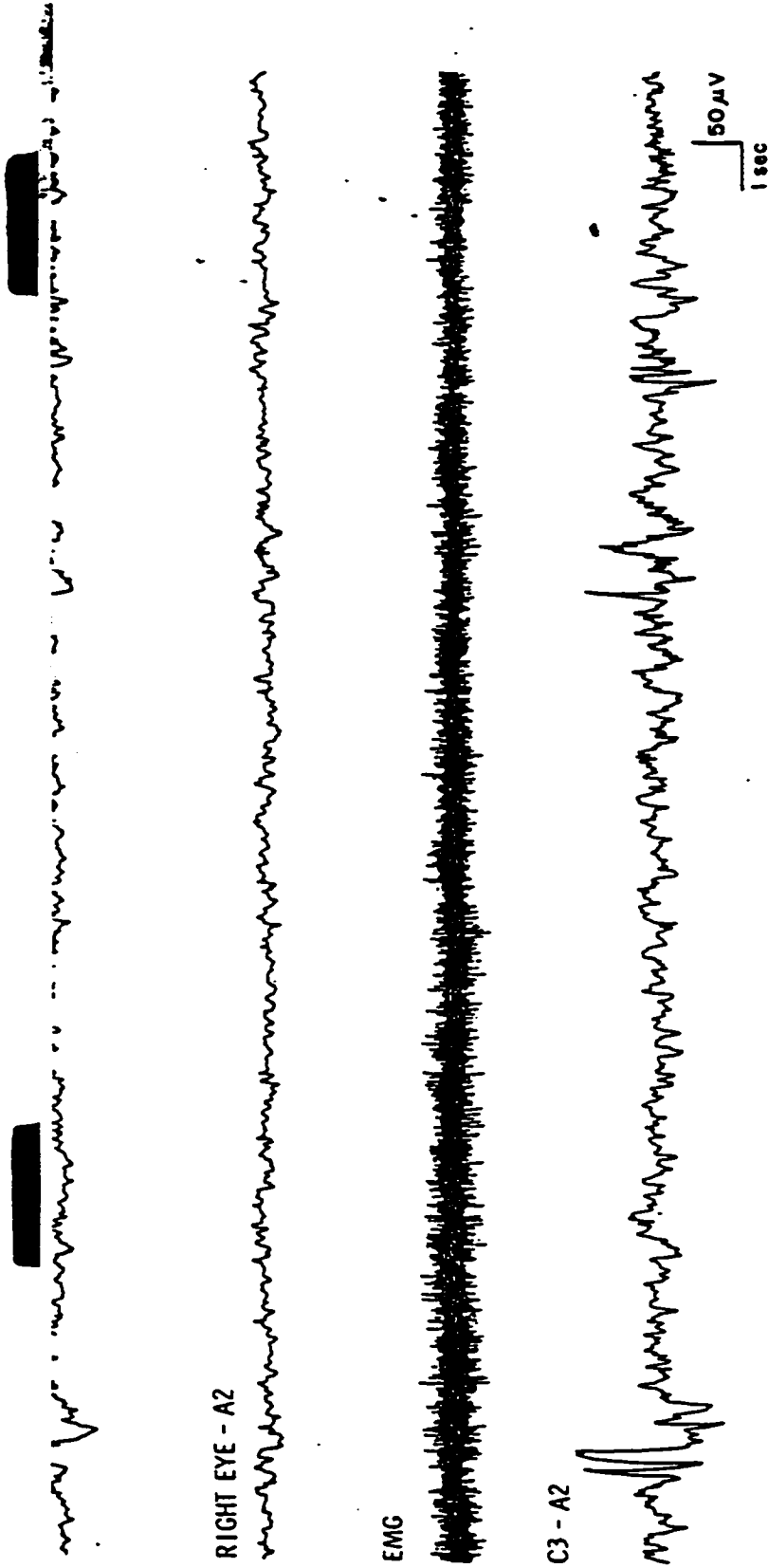


FIGURE 14

Stage 2. This illustrates Stage 2 with relatively elevated tonic EMG. The presence of sleep spindles is unambiguous.

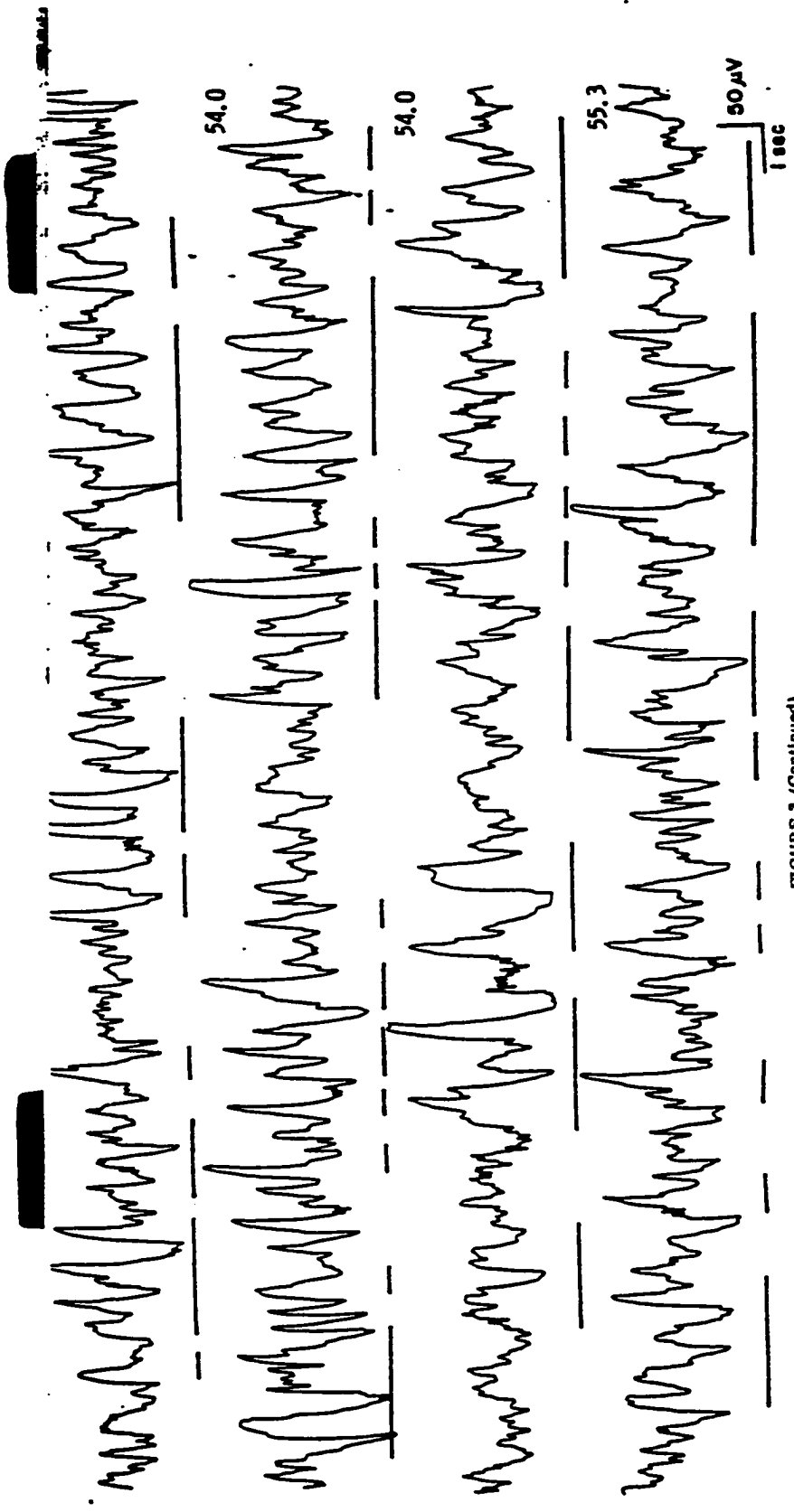


FIGURE 3 (Continued)

"acceptable" high amplitude, slow wave activity, i.e., 2 eps or slower and greater than 75 μ V peak to peak. These illustrations depict 30 sec epochs recorded on a Beckman Type R Dymograph with a paper speed of 10 mm/sec, a time constant of 0.3 sec and a calibration of 50 μ V/cm.

The four tracings on this page were selected because they show just enough high amplitude slow wave activity to qualify for Stage 4. In borderline instances where there is a question of whether to score Stage 3 or Stage 4, a comparison of the record in question with these tracings may facilitate a decision. The underlined portions of each tracing were considered

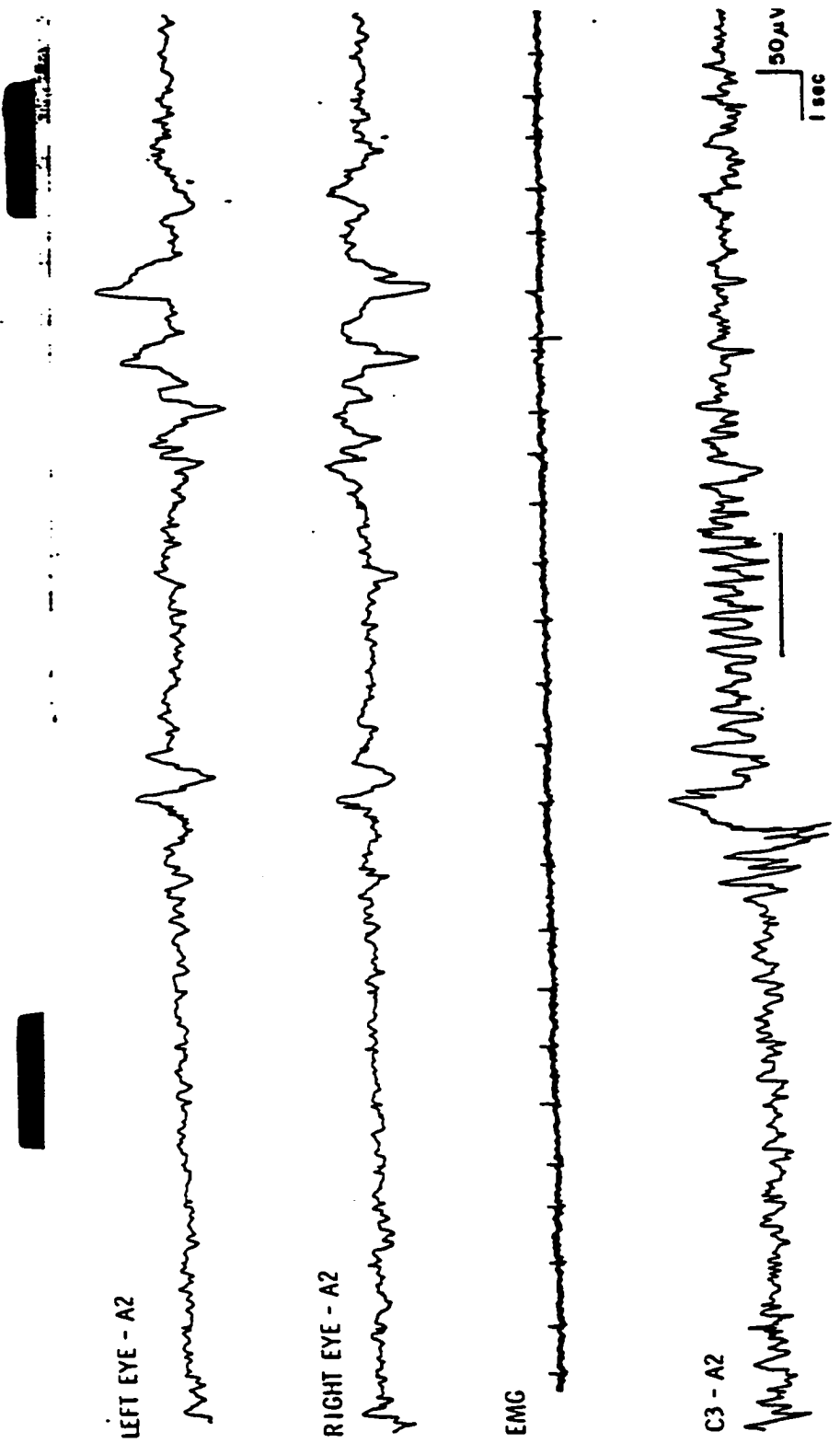


FIGURE 19

Stage REM. This epoch illustrates a transition between Stage 2 and Stage REM. The record had been Stage 2 for some time prior to this epoch. Just before the midpoint of the epoch, there is a clear sleep spindle followed by a K complex. Following the K complex are clear saw-tooth waves (underlined). that not all the saw-tooth waves have the distinctive notched appearance.) Stage REM is considered to have begun immediately after the end of the K complex, and to continue for the remainder of the epoch. The interval of Stage REM occupies just over 50% of the epoch; hence the entire epoch is

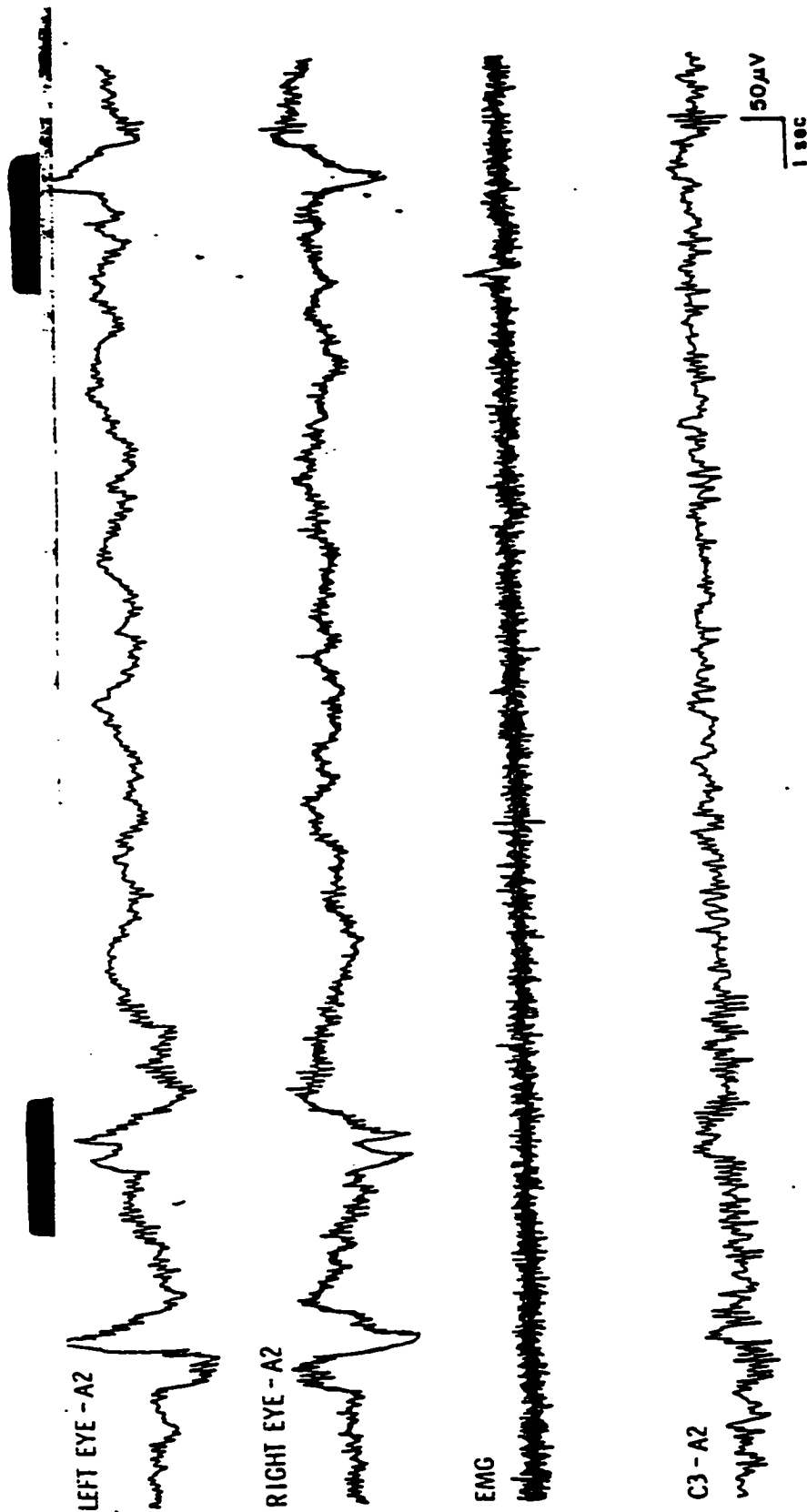


FIGURE 10

end of the epoch, a REM and a burst of alpha activity signal a return to Stage W, but slightly more than half the epoch is Stage 1, and the epoch is scored accordingly. The relatively elevated EMG is maintained in the transition from Stage W to Stage 1.

Stage 1. This illustrates the transition from Stage W to Stage 1 within a single epoch; REMs and alpha activity at the start of the epoch are followed by slow eye movements and the typical relatively low voltage, mixed frequency EEG of Stage 1 (much activity at 3-4 cps) later in the epoch. There are no vertex sharp waves, which is typical of the early minutes of Stage 1. At the

RECORDING AND SCORING LEG MOVEMENTS

749

Recording and Scoring Leg Movements*

The Atlas Task Force:

Michael Bonnet, David Carley, Mary Carskadon (Consultant), Paul Easton (Consultant), Christian Guilleminault (Chairman), Ronald Harper, Boyd Hayes, Max Hirshkowitz, Periklis Ktonas, Sharon Keenan, Mark Pressman (Consultant), Timothy Roehrs, Jack Smith, Jim Walsh, Steven Weber, Philip Westbrook. ASDA Administrative Support: Bruce Jordan.

RECORDING AND SCORING LEG MOVEMENTS

The scoring rules that follow were written specifically for leg movements, which constitute the vast majority of limb movements during sleep. Noninvasive surface recording of anterior tibialis activity provides the basic data from which leg movements are scored using polysomnography. Surface electrodes placed over the belly of a limb muscle provide information related to gross motor activity and are therefore suitable for detecting partial flexion of the big toe, ankle, knee or hip. This technique, however, does not provide information concerning specific motor unit activity. If quantitative analyses of leg movements beyond those described here are desired, more sophisticated recording and calibration techniques will be required (1).

Placement of electrodes

To record leg movements (LMs), electrodes are placed over the anterior tibialis muscles. It is important to record activity from both legs because movements may occur in one leg only or switch from one leg to the other throughout the night. Therefore, single leg recording provides insufficient information to score LMs. Two electrode sites are selected on the belly of the anterior tibialis muscle from each leg. It is insufficient to record from only one leg. The electrodes are placed 2-4 cm apart, to avoid salt bridges, on the long axis of the muscle (see Fig. 1). To distinguish some abnormal movement patterns involving agonists and antagonists, electrodes may also be placed over the corresponding gastrocnemius muscles. For clinical recording, noninvasive surface electrodes with an impedance of less than 30,000 ohms are adequate. Impedances less than 10,000 ohms are preferable, but this may require significant scrubbing.

* This section of the Atlas was also presented to the membership at the 1992 APSS national meeting. Feedback from the membership is welcome.

Recording calibration and verification

Use of separate channels for the electromyographic activity recorded from each pair of surface electrodes (henceforth referred to as the EMG) allows a comparison of left and right leg movements. When the number of recording channels available is limited, one can link left and right leg electrodes to provide a single channel tracing (see Fig. 2). However, this procedure limits interpretation of the recording to event detection and classification. Quantitative analysis of LM periodicity requires separate recording channels for each leg. The amplifier settings for recording LMs are selected with a preference for normal EMG activity, which typically ranges in frequency from 20 to 200 Hz (2). For example, band-pass filter settings of 10 Hz and 90 Hz with a time constant of 0.003 second may be used.

Recording verifications should be performed both before and after sleep. While lying awake in bed, patients are instructed to slowly dorsiflex and plantarflex the great toe of each foot to approximately 30° without resistance. Comparison of the EMG pattern during periods of flexion and relaxation ensures adequate detection of leg movements (see Fig. 2). Recording of muscle tone during a segment of relaxed wakefulness may be helpful. It should demonstrate a low EMG activity.

Respiratory monitoring allows differentiation of primary LM activity from LMs associated with breathing impairment arousals. Without respiratory monitoring, LM activity associated with respiratory disturbance cannot be determined. Video recording can also be helpful for the analysis and interpretation of leg movements.

Scoring rules for leg movements

In the field of sleep and sleep pathology, attention has been focused on the periodic limb movement disorder. The basic polysomnographic event defined here as the "leg movement" is related to this syndrome. The following rules acknowledge that other leg movements exist and describe scoring rules for the detection, classification and analysis of "leg movements". Leg movements considered here are not part of a more

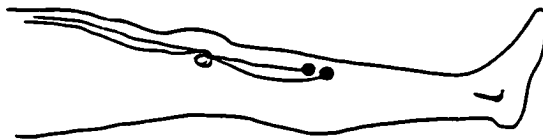


FIG 1. Application of surface (gold or silver-silver chloride) electrodes for recording anterior tibialis electromyogram. The electrode application site is prepared by gently rubbing the surface of the skin with an alcohol wipe followed by rubbing briskly with Omniprep or some other slightly abrasive substance. The condition of the patient's skin must be kept in mind. The possibility of poor circulation due to diabetes, edema, varicose veins or other conditions may precipitate the formation of decubitus ulcers as a result of too much abrasion. An electroconductive material is placed either directly onto the skin or inside the cup of the electrode. Tape should be used to reinforce the electrode placement and to create a strain relief loop in the electrode wires at the knee level.

EMG electrodes should be applied to ensure a) that the two electrodes are applied to the surface of the skin above and directly superficial to the anterior tibialis muscle with a separation of 2-4 cm. Location is determined by palpating the muscle while the patient dorsiflexes the great toe; b) that the electrodes are aligned in a vertical plane perpendicular to the axis of the heart to decrease ECG artifact; and c) that electrode impedances are less than 30,000 ohms.

generalized neurological disorder also seen during wakefulness, such as tremor, choreoathetosis, intention-action-myoclonus et cetera. These guidelines were developed using a general approach to the polysomnographic assessment of leg movements. A variety of LM classifications and analyses are presented that broadly reflect the literature regarding LMs. Those scoring procedures that should be uniformly employed for clinical polysomnography are marked with a double asterisk (**). Other procedures are optional but may be useful for research studies; many of these procedures are not amenable to manual scoring.

* *

Event detection

**** Leg movement.** A burst of anterior tibialis muscle activity with a duration between onset and resolution of 0.5-5 seconds and with an amplitude of at least 25% of the bursts recorded during calibration is defined and scored as a "leg movement". Onset and resolution are identified by comparison of the burst envelope to the tonic background as illustrated in Figs. 3 and 4. Amplitude is measured in μV using the EMG envelope. Leg movements as defined here must be distinguished from several other events including:

Sleep starts. Often involving generalized hypnic myoclonus, these jerks occur during the transition from wakefulness to sleep and are not periodic (3, 4). They may be either brief (20-100 msec in duration) and associated with upper and or lower extremity movement, or longer (> 1 second in duration), involving other muscle groups. Agonists and antagonists contract synchronously, as in a normal "startle" response.

Rapid eye movement (REM) extremity movements. Brief movements of the upper and lower extremities that are associated with REM sleep (5) are defined as "REM extremity movements". These movements follow neither a periodic nor a stereotypic pattern and occur in most REM periods (6). The movement durations exhibit more variability than LMs. These may be abnormally enhanced, as in REM sleep behavior disorder.

Phasic EMG activity. This activity may occur in the anterior tibialis in the absence of a tonic back-

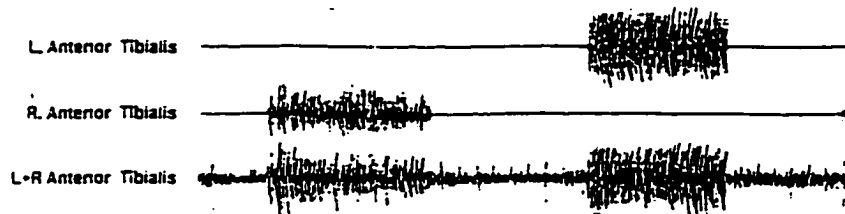


FIG 2. Calibration procedures.

Equipment calibration. An AC amplifier is used to record EMG activity, which typically ranges in frequency from 20 to 200 Hz (2). Low-frequency filters are adjusted to the maximum available setting (usually 5 or 10 Hz). This corresponds to a short time constant (0.035 seconds, low-frequency filter setting of 5 Hz). The high-frequency filter should be adjusted to the upper limits (typically > 70 Hz). Sensitivity should be initially adjusted to 50 μV cm. This setting should allow for adequate display of the EMG activity but may require adjustment during behavioral maneuvers.

Behavioral maneuvers (Bicoali). To verify and optimize recordings of anterior tibialis EMG activity, the subject is instructed to first dorsiflex and plantarflex the right great toe without resistance (see middle tracing). The subject is then instructed to repeat this maneuver with the left great toe (see top tracing). The EMG bursts associated with these movements should have an amplitude of approximately 1-cm pen deflection. If a single recording channel is used that links right and left anterior tibialis activity, left versus right leg activity cannot be distinguished (see bottom tracing).

RECORDING AND SCORING LEG MOVEMENTS

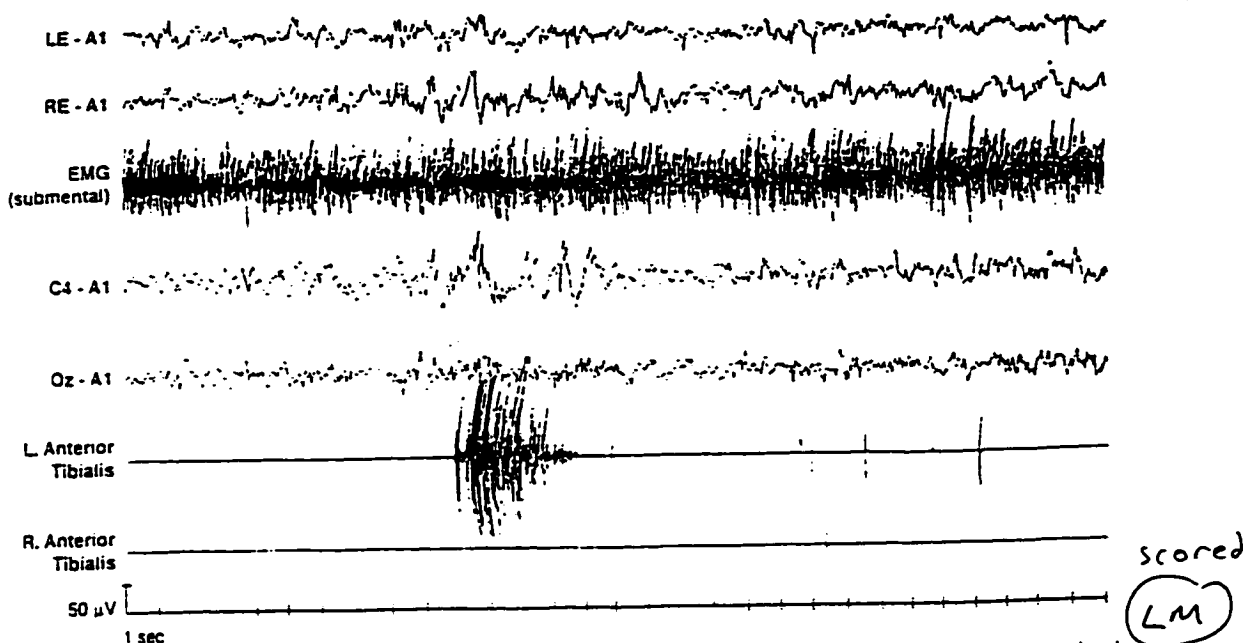


FIG. 3. A burst of activity is present in the left anterior tibialis muscle. Because this burst has a duration greater than 0.5 second and less than 5 seconds and an amplitude greater than 25% of calibration movements, it is scored as an LM. LM onset and termination are recognized by comparison of the burst activity with the tonic activity.

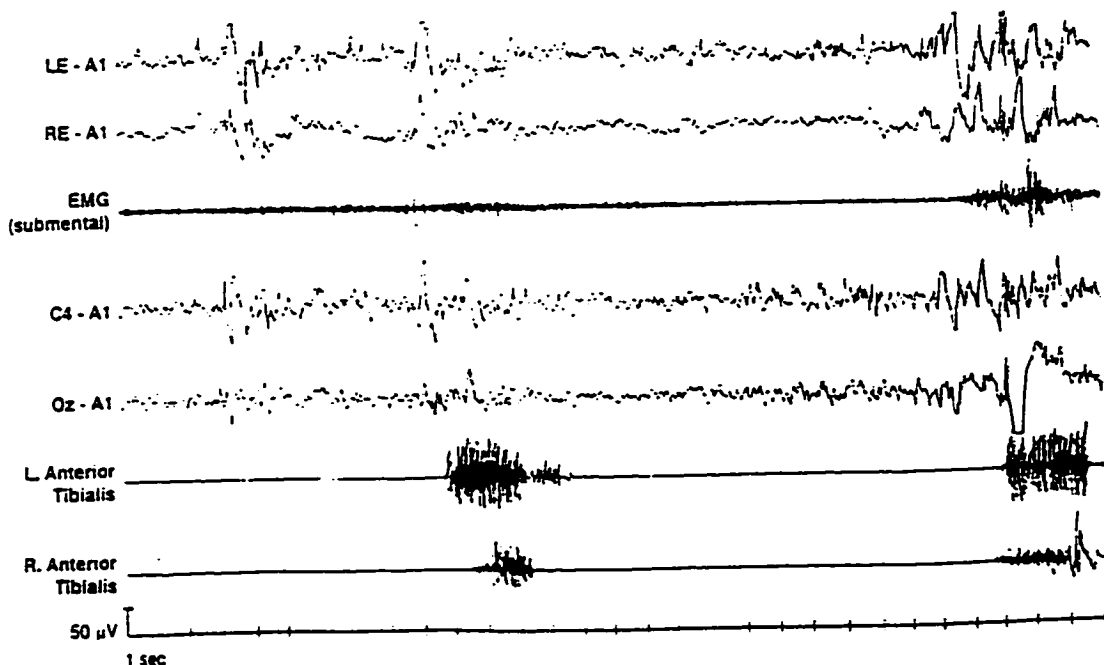


FIG. 4. EMG bursts are apparent on the left and right anterior tibialis EMG waveforms. Because the right and left bursts are separated by less than 5 seconds, a single leg movement event is scored for calculation of event statistics.

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ATLAS TASK FORCE

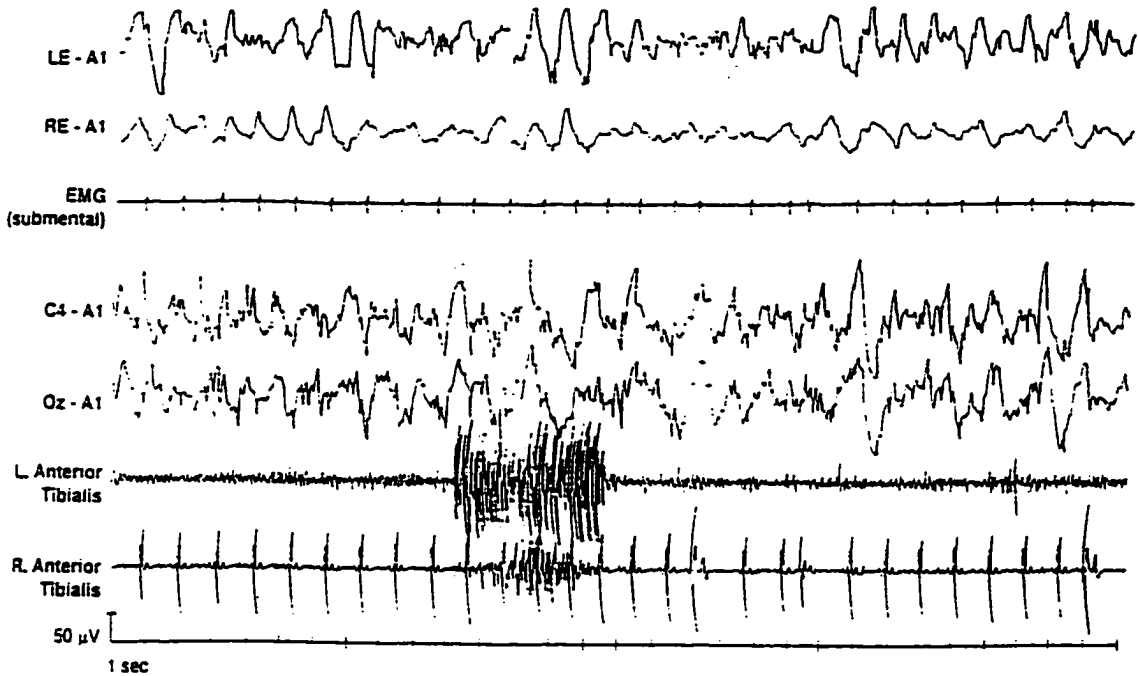


FIG. 5. Example of an LM during stage 4 sleep. LMs can occur and should be scored during all stages of sleep. Also note the prominent ECG artifact on the right anterior tibialis channel.

Score
in
all
stages

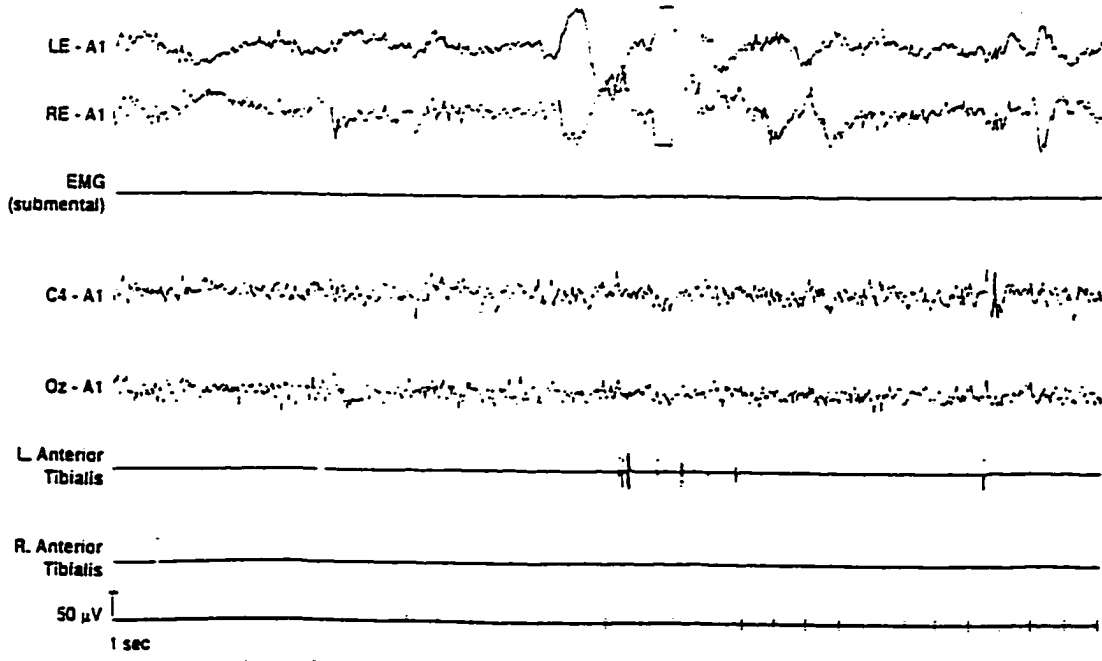


FIG. 6. Brief (0.5 second) bursts of anterior tibialis activity occur during this epoch of REM sleep. These bursts are of lower amplitude than LM bursts and are not associated with observable leg movements. However, phasic EMG activity may precede or indicate the emergence of periodic LMs.

REM
bursts

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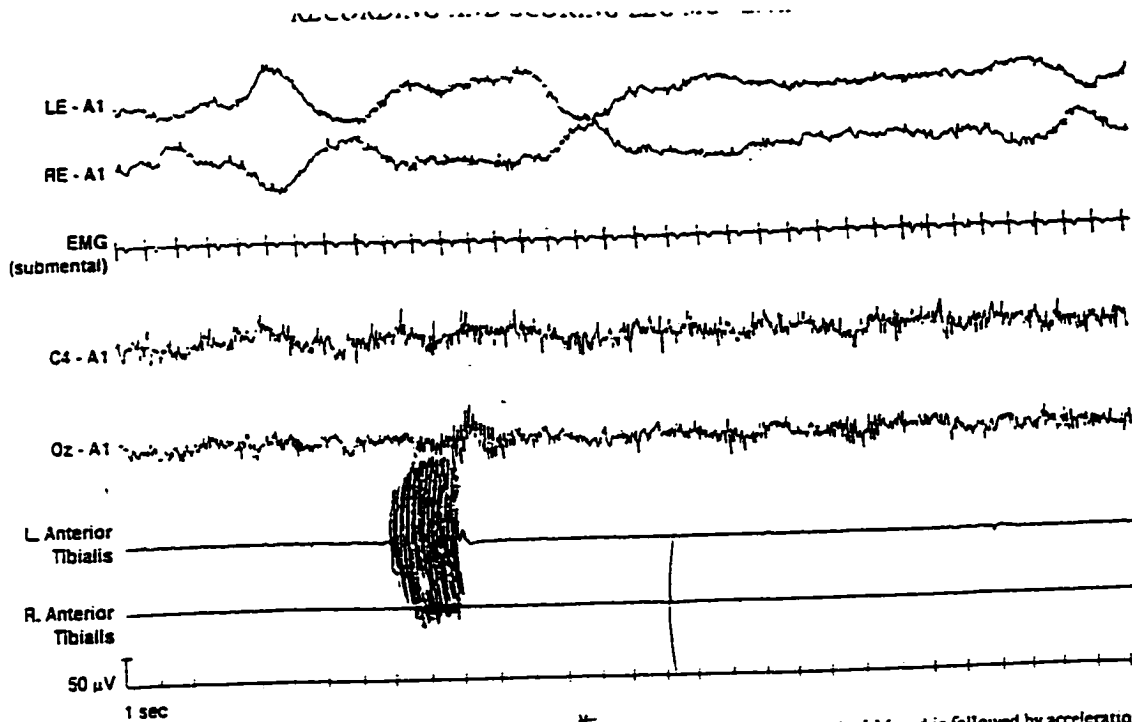


FIG. 7. The LM in this epoch is associated with arousal. Emergence of alpha activity occurs during the LM and is followed by acceleration and desynchronization of the EEG (most notable in the occipital lead). These EEG changes last for more than 3 seconds and fit the definition of arousal. The LM of this epoch is therefore classified as associated with arousal.

LM with arousal
* $\alpha > 3 \text{ sec}$

ground during wakefulness and sleep (7). During sleep, many patients with periodic LMs have periodic phasic EMG discharges from the anterior tibialis, even when periodic LMs subside. This activity is characterized by brief (100–500 mseconds), low-amplitude discharges (see Fig. 6). Observable leg movements do not accompany this activity. However, such phasic EMG activity may precede and indicate the emergence of periodic LMs (8).

Fragmentary myoclonus. These consist of brief (<200 msec) phasic potentials with an amplitude of up to 250 μV . The largest potentials may be associated with twitching movements of various body regions. They may be observed during wakefulness and all stages of sleep (9).

Restless legs activity. This activity is characterized by EMG activity in the anterior tibialis during wakefulness, at sleep onset and during sleep. The activity typically has a duration of greater than 5 seconds and recordings reveal an irregularly shaped activity envelope. Muscle activity with this profile can accompany unusual, uncomfortable, sometimes described as "crawling" sensations in the legs and provide a diagnostic indicator for restless legs syndrome.

Restless legs syndrome rarely occurs in the absence of periodic limb movement disorder.

Movements associated with painful legs and moving toes. The syndrome is characterized by pain in one or both feet, with or without a sensation of burning, and involuntary movements of the toes (10). However, these movements are irregular and are not related to the sleep-wake cycle (11).

Event classification

Leg movement events should be classified for at least two purposes: 1) to assess potential sleep disturbance and 2) in an attempt to identify the etiology of LM genesis. Leg movements should be classified and tabulated according to the following criteria. Left and right LMs should be tabulated and analyzed separately.

According to sleep stage. LMs should be classified according to the sleep stage within which they occurred and should be scored during wakefulness. LMs may be observed during wakefulness and may make sleep onset difficult to identify. LMs should not be scored during epochs scored as movement time or during time out of bed.

According to presence of arousal. LMs may be as-

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ATLAS TASK FORCE

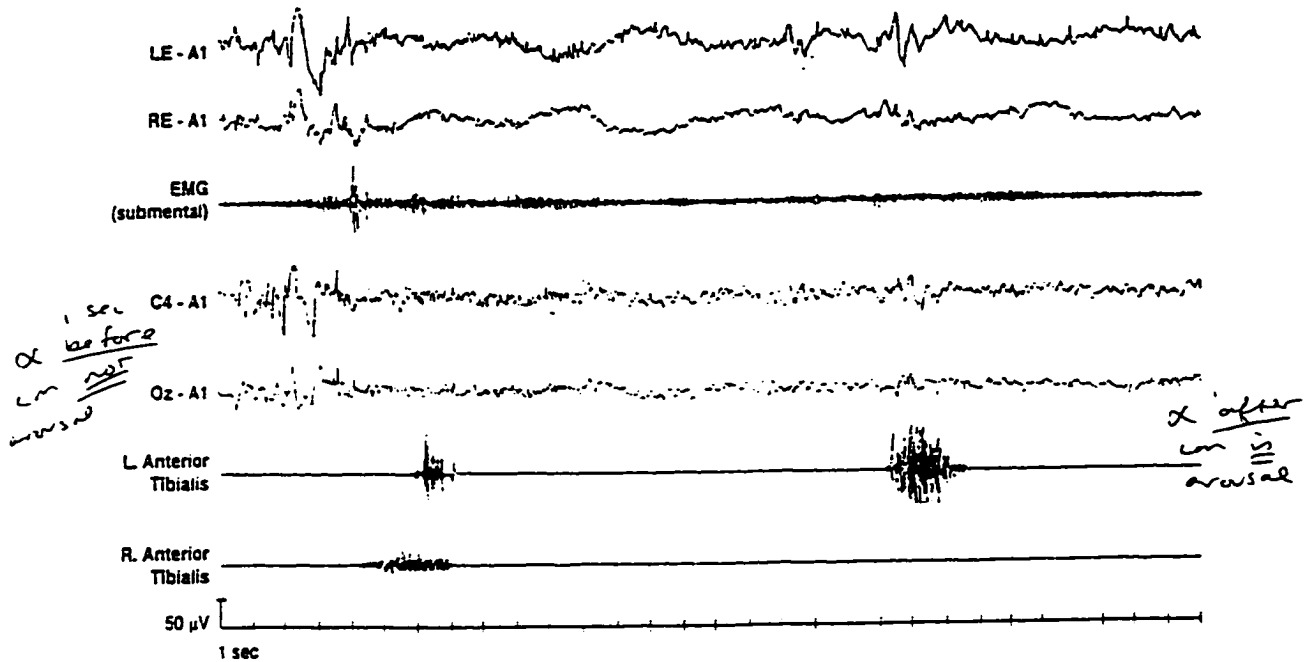


FIG. 8. The first LM occurs in both left and right anterior tibialis activity. This LM is not associated with arousal because the arousal precedes the LM by approximately 1 second. The second LM is associated with arousal, seen most clearly in the C4-A1 tracing. The K-complex that immediately follows the LM onset is not included in the arousal, but more than 3 seconds of clear EEG acceleration and desynchronization occurs.

sociated with arousal, awakening or both (see Figs. 7, 8, 11, 13 and 14). In this case, the onset of an electroencephalogram (EEG) arousal occurs concurrent with or after the onset of, and is closely related to the leg movement. (It is the consensus of the committee that the arousal should follow the LM termination by not more than 1-2 seconds.)

- According to the presence of respiratory disturbance. LMs frequently occur in conjunction with apneas and hypopneas. Because these respiratory events may also initiate EEG arousal, LMs that occur at the resolution of an apnea, hypopnea or other obstructive event should be so classified (see Figs. 9 and 15). If significant numbers of EEG arousals are associated with concurrent respiratory events and LMs in a symptomatic patient, a judgment regarding the primary cause of arousals must be made and clinical follow-up conducted. LMs that occur during regular breathing or begin and end prior to resolution of an apnea or hypopnea should not be classified as associated with apnea or hypopnea. In patients with significant sleep-related breathing disorder and LMs, it may be necessary to treat the breathing disorder before evaluating the importance of the leg movements.

According to the presence of other limb movements. LMs may be associated with movements of the arms or head. Because the mechanisms underlying various body movements remain unclear, separate classification and tabulation is appropriate. Additional monitoring is required for detection of arm and head movements.

Event analysis

LM event statistics. The following descriptive analyses of LM events can be performed. All event statistics should include a differentiation of LMs associated with arousal versus those not associated with arousal.

- 1. Number of leg movements (NLM).
- 2a. Number of leg movements with arousal but not awakening.
- 2b. Number of leg movements with awakening for one or more epochs.
- 3. Number of leg movements associated with respiratory events.
- 4. Leg movement index (LMI; scored separately with awakening, with arousal, without arousal and with respiratory events). The number of LMs per hour of total sleep time (TST).

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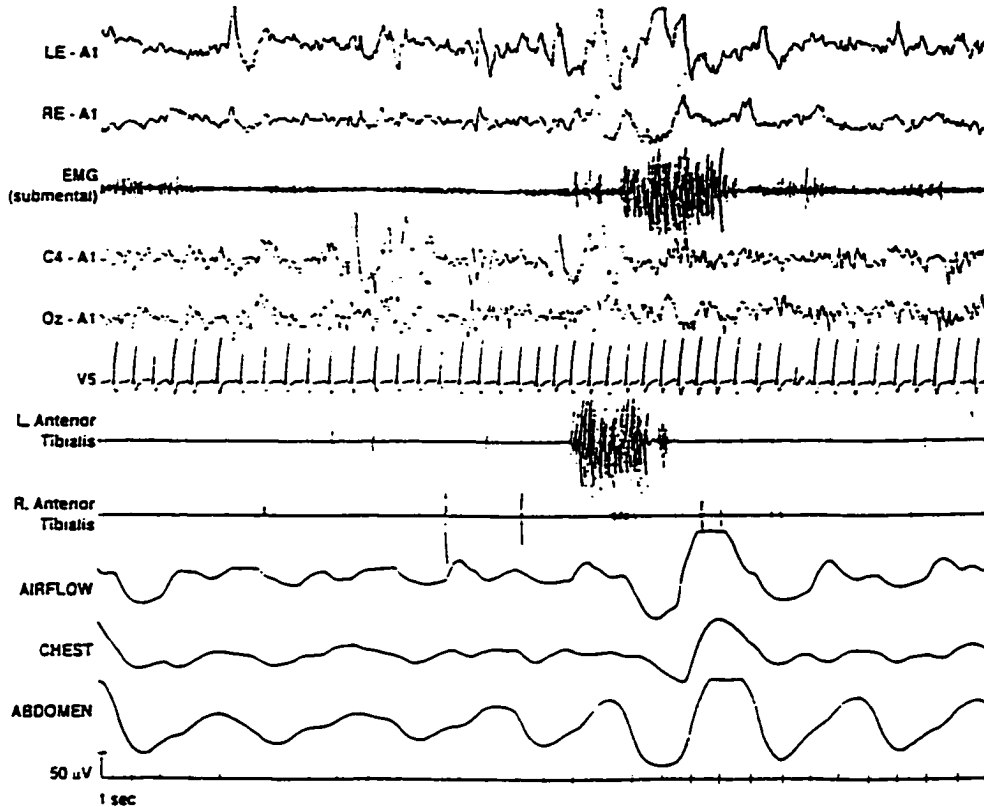


FIG. 9. As demonstrated by the thermistor airflow channel, this LM also occurs near the resolution of a sleep disordered breathing event and should be so classified. Although a clear burst in submental EMG follows the LM, clear EEG acceleration is not present and this LM is not associated with arousal.

Temporal pattern analysis. Diagnosis of the periodic limb movement disorder requires identification of periodic leg movements (PLMs). Only LM activity that occurs during sleep which does not follow arousal should be considered for PLM analysis. These analyses should be performed for left and right legs separately.

PLMs may be identified manually or with computer-assisted methods by scoring periodic sequences of LMs (PLM sequences).

PLM sequence. A sequence of four or more LMs, separated by at least 5 and not more than 90 seconds (see Figs. 10-15). This separation is measured from LM onset to LM onset and is different from inter-LM interval, which is measured from LM offset to LM onset.

4 x's at least 5 but not > 90 sec

PLM sequences may be further characterized by computation of the following:

1. Percentage of LMs occurring as part of PLM sequences (scored separately with and without arousal).
2. Percentage of LMs within PLM sequences for each sleep stage or state.
3. Average LM period (LM onset to LM onset) for PLM sequences. This parameter, measured in seconds, characterizes the periodicity of the muscle activity related to movement sequences.
4. Average inter-LM interval.
5. Average sequence duration during TST, nonrapid eye movement (NREM) and REM sleep.

Several additional sequence statistics have been described:

1. Total duration of all PLM sequences.
2. Percentage of total sleep time associated with PLM sequences.
3. Percentage of each sleep stage associated with PLM sequences.

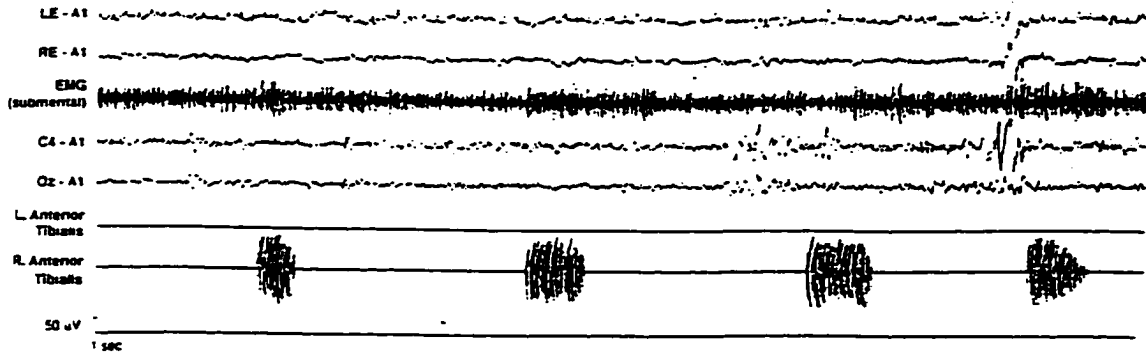


FIG. 10. LMs generally occur concomitantly in both legs, but may occur in one leg alone. These two epochs demonstrate an excerpt from a PLM sequence with EMG bursts occurring only in the right anterior tibiais. Four individual LMs are present. Each successive LM is separated from the last by more than 5 and less than 90 seconds, thus conforming to the definition of a PLM sequence. These four movements were extracted from a sequence containing more than 20 leg movements. None of the movements illustrated are associated with arousal.

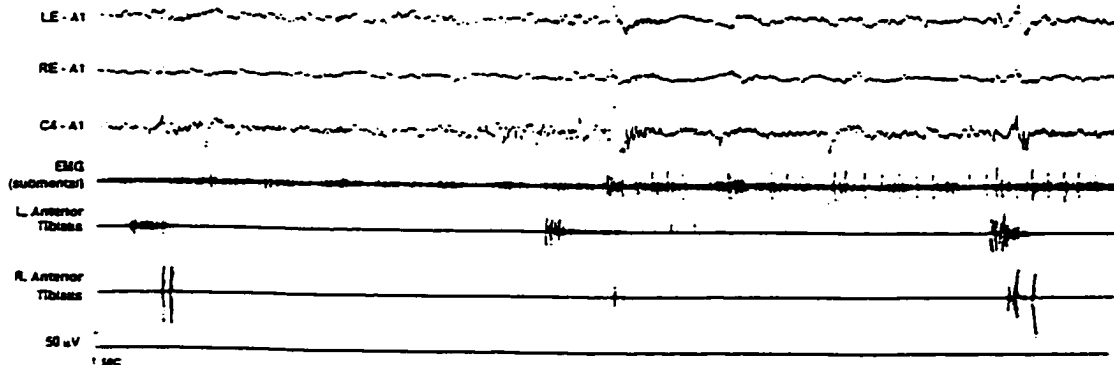


FIG. 11. Excerpt from a PLM sequence. Each of the three LMs depicted is associated with arousal. Although an occipital EEG derivation is not illustrated, arousals should be scored using at least one occipital lead. Scoring arousals from only a single central lead may cause underestimation of the number of arousals.

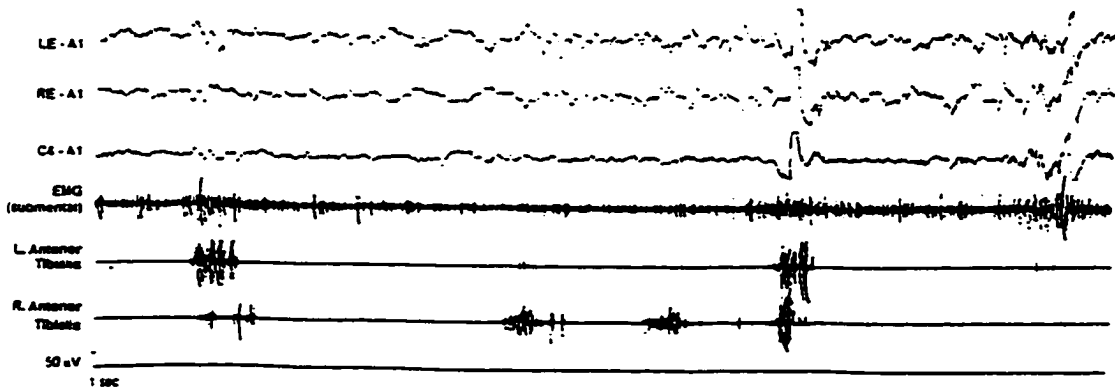


FIG. 12. Periodic leg movement sequences can occur during wakefulness or can continue from sleep into wakefulness. The four LMs depicted constitute a PLM sequence during two epochs of stage wake.

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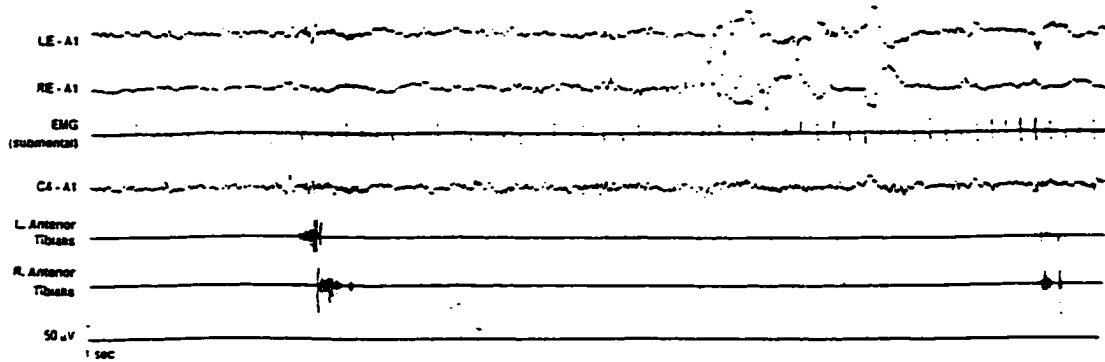


FIG. 13. Excerpt from a PLM sequence during REM sleep. Both EEG acceleration and increased submental EMG follow the first LM, which is therefore associated with arousal. Phasic EMG activity is also present in these REM epochs and is not scored as an LM.

Additional information regarding the temporal pattern of LMs should be obtained if computer-based analysis is available. These techniques allow a more complete and accurate assessment of the periodicity and variability of LM events and may include:

1. Computation of the inter-LM interval histogram and descriptive statistics. A narrow histogram peak and low coefficient of variation of these intervals may indicate the presence of a consistently periodic sequence of LMs. These histograms and statistics may be computed for each PLM sequence, for each sleep stage and sleep state, for each NREM-REM cycle or by hour of recording time.
2. Computation of the LM duration histogram and descriptive statistics with stratification similar to that described for inter-LM interval statistics (if desired).
3. Trend analysis of inter-LM intervals over time.
4. Trend analysis of LM durations over time.

Artifacts

1. Respiratory artifact introduces a slow rolling background in the EMG(s), which is synchronous with respiration on the polysomnograph. With appropriate filter settings (10 Hz), a respiratory artifact may appear as intermittent high-frequency bursts timed with respiration. Usually this results from long leg-electrode wires passing over the abdomen or chest. Repositioning the electrode wires away from the abdomen or chest may eliminate this artifact.
2. Other body movements, such as head turning, may be reflected in the leg channels.
3. Snoring can produce artifact in the leg channels.
4. Electrocardiogram (ECG) artifact can appear on a leg channel but rarely interferes with interpretation of the EMG activity.
5. Appearance of 60-Hz artifacts indicates electrode problems. Faulty electrodes, poorly applied elec-

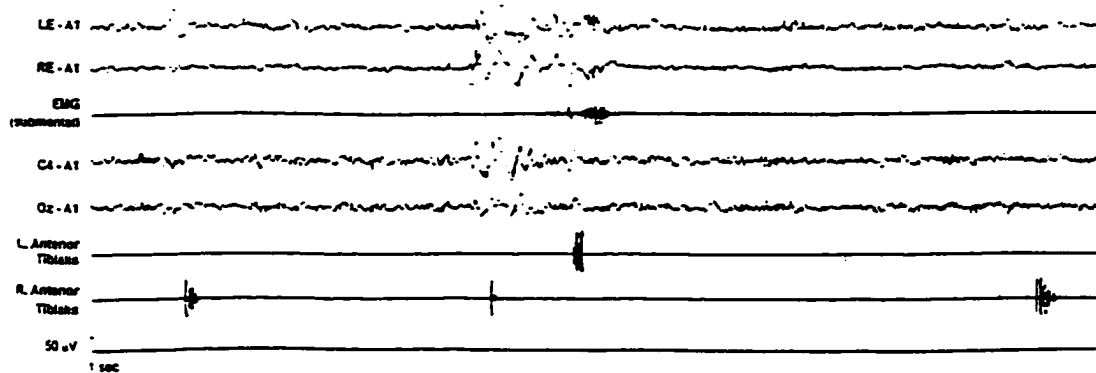


FIG. 14. Excerpt from a PLM sequence during REM sleep. Three LMs are illustrated. Because the EMG burst observed on the LAT tracing is separated from the immediately preceding RAT burst by less than 5 seconds (at middle of page), only a single LM is scored. This LM is followed by increased submental EMG and EEG acceleration and is therefore associated with arousal.

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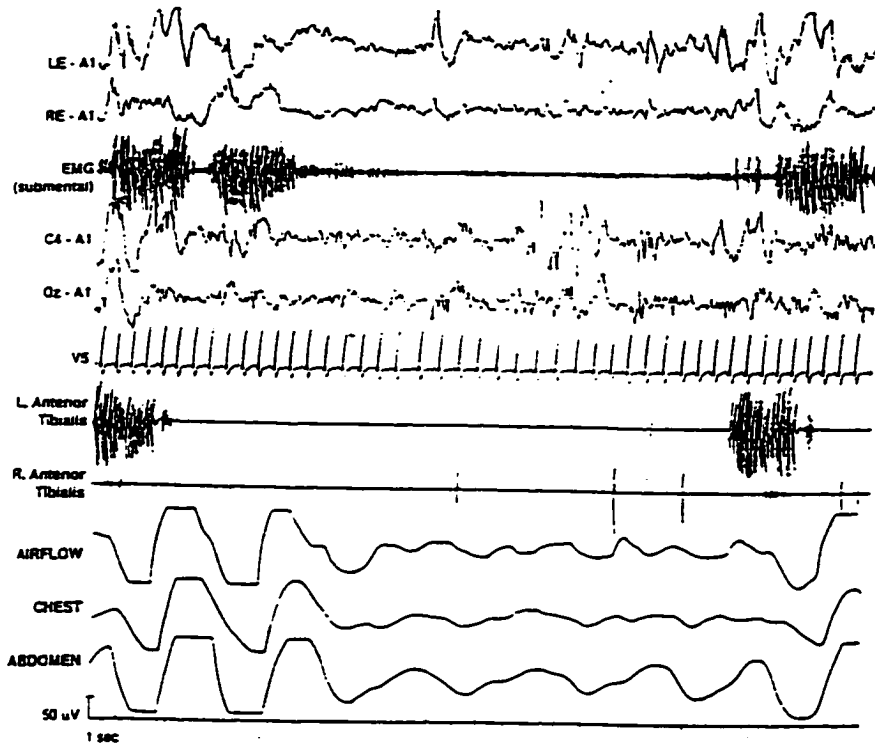


FIG. 15. Excerpt from a PLM sequence. Each LM is associated with apnea resolution. Although the apneas and the LMs are periodic, this pattern does not provide the basis for diagnosing periodic limb movement disorder unless periodic LMs also occur independent of respiratory events.

trodes and electrodes with significant increases in resistance require reapplication.

RECORDING AND SCORING LEG MOVEMENTS: SUMMARY

Greater detail regarding these methods is found in the text. Many other calculations have been described and some of these are noted in the text. This summary reflects a consensus of scoring and calculation that should be performed routinely for clinical purposes.

Scoring

1. *Leg movement (LM)*. A 0.5 to 5 second burst of anterior tibialis activity with an amplitude >25% of calibration movements (Fig. 2).
2. *Periodic leg movement sequence*. Four or more LMs separated by more than 5 and less than 90 seconds. To score periodic LM sequences all LMs must be scored during all stages of sleep and wakefulness.
3. To assess sleep disturbance, LMs associated with arousal or awakening must be counted. To score

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LMs as associated with arousal, the arousal onset must follow LM onset by not more than 3 seconds.

4. LMs associated with respiratory events are classified and counted as such.

Calculations

1. Total number of leg movements (NLM) should be tabulated. If appropriate to the interpretation of an individual study, these leg movements should then be subclassified as follows:

Number of LMs associated with arousal (including arousal to wakefulness: NLMAr).

Number of LMs with arousal to wakefulness for at least one epoch (NLMW).

Number of LMs associated with apnea or other respiratory event (NLMAp).

2. To control for variable recording time and sleep efficiency, each of the above LM counts may be expressed as indexes, or counts per hour of sleep:

$$LMI = (NLM/TST) \cdot 60$$

$$LMArI = (NLMAr/TST) \cdot 60$$

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$$\text{LMapi} = (\text{NLMapi}/\text{TST}) \cdot 60$$

$$\text{LMWI} = (\text{NLMW}/\text{TST}) \cdot 60$$

(TST = total sleep time in minutes.)

3. LMs that occur as part of periodic leg movement sequences are referred to the total number of leg movements as:

%NLM occurring within PLM sequences.

%NLMA occurring within PLM sequences.

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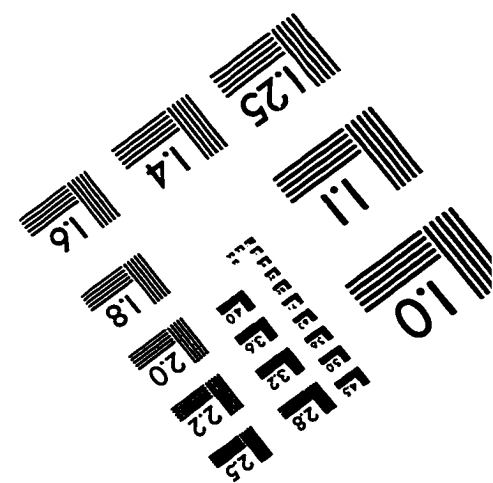
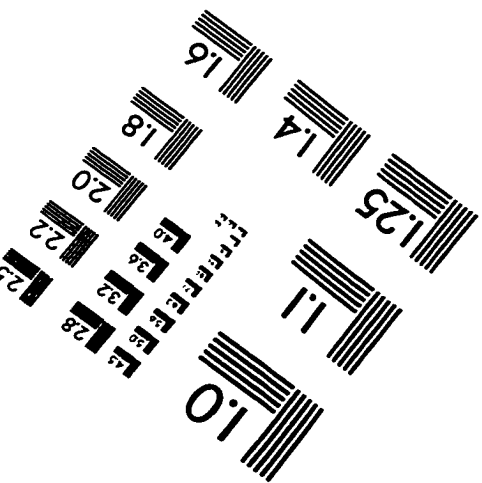
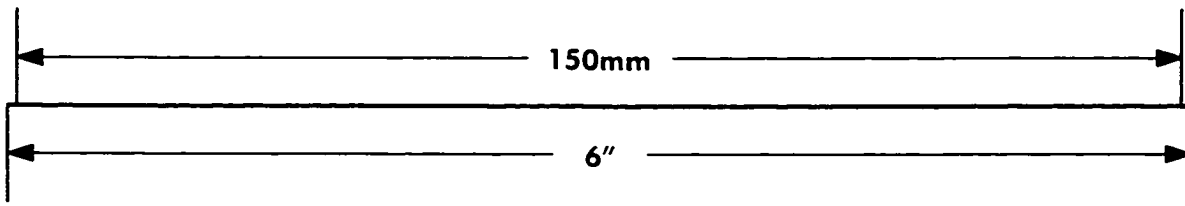
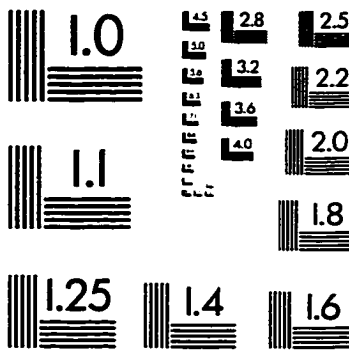
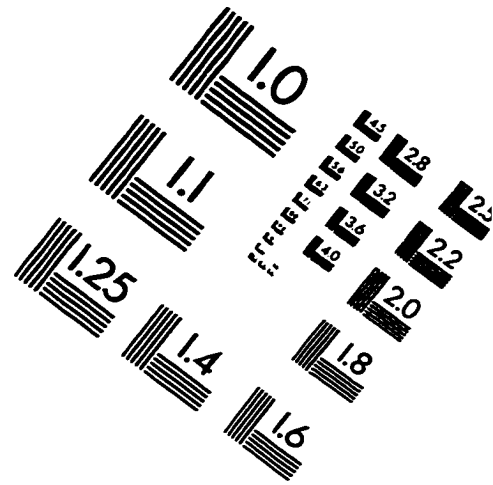
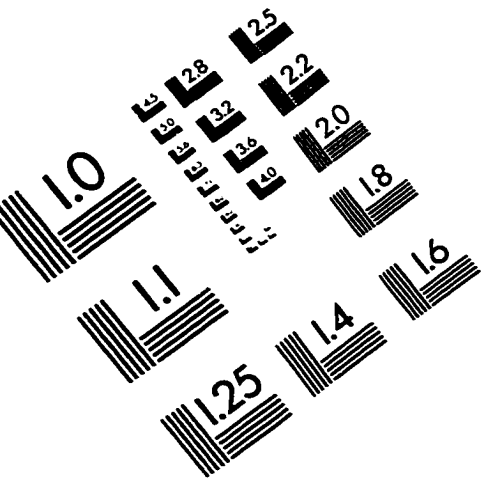
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