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SLEEP OF HYPERKINETIC CHILDREN

by Linda Poitras

Thesis submitted to the School of Graduate Studies of the University of Ottawa in partial fulfillment of the requirements for the Master of Arts Degree in Psychology

Ottawa, Canada, 1981

Abstract

Although sleep recordings may provide unique information regarding state correlates of arousal, and despite the postulated involvement of arousal mechanisms in the Hyperkinetic (HK) syndrome, remarkably few studies of sleep have been reported for children with this disorder. In the present study, the sleep patterns of five unmedicated hyperkinetic children (9-12 yrs.) were compared to those of five normal controls matched for age and sex. All children slept at the laboratory for five consecutive nights at which time EEG, EOG, EMG, and SSPR measures were continuously monitored.

Analyses of sleep patterns revealed a significantly longer REM onset latency on the last two nights (night 4: $p < .05$; night 5: $p < .025$) and overall increased spindle activity for the HK group relative to controls ($p < .05$). Both of these differences may be related to decreased CNS excitability and are therefore most suggestive of CNS hypo-arousal in HK children.

There were no other major or consistent group-related variations in sleep parameters. Other more general features of sleep pattern variations which were noted corroborated previous observations regarding normative sleep in children of this age group. Based on variations in sleep patterns and associated events as observed in this investigation, the presence of a major arousal dysfunction in hyperkinetic children is not indicated.

CURRICULUM STUDIORUM

Linda Poitras was born October 26, 1954 in Grand Falls, New-Brunswick. She received the Bachelor of Arts degree from Moncton University (Edmunston, N. B. campus) in 1975 and the Bachelor of Arts (Honours) degree in Psychology from the University of Ottawa, Ottawa, Ontario, in 1976.

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Chapter 1

REVIEW OF THE LITERATURE

Hyperkinesis is a childhood disorder that has recently generated a great deal of research and controversy. The definition, etiology, and diagnosis of this disorder have suffered from global and ambiguous conceptualization which has been reflected in widely discrepant terminology, such as: minimal brain dysfunction (MBD), minimal brain damage, hyperkinesis, hyperactivity, learning disabilities, etc. The classical symptoms generally agreed upon to define and diagnose HK focus on behavioral, perceptual-cognitive, and social features of the disorder. The symptomatology includes excessive motor activity, short attention span, distractibility, impulsivity, excitability and poor scholastic performance despite normal performance on intelligence measures. Also, secondary signs such as aggressivity, low frustration tolerance and poor self-esteem resulting in emotional problems are often observed (Clements, 1966; Minde, Wiess, & Mendelson, 1972; Rosenthal, & Allen, 1978; Schmitt, Martin, Nellhaus, Cravens, Camp, & Jordan, 1973; Whalen, & Henker, 1976). Although some of these deficits are also present in children throughout normal developmental stages, it is the persistence, intensity, and clustering of symptoms which distinguishes HK behavior from normal behavior (Minde, Lewin, Weiss, Lavigueur, Douglas, & Sykes, 1971; Wender, 1975). This has prompted Kinsbourne (1973) and Rosenthal (1973) to hypothesize that these children may suffer from a neuro-develop-

mental lag. Prevalence estimates range from one to twenty percent for all school-age children (Chess, 1960; Huessy, 1967; Lambert, Sandoval, & Sassone, 1978; Stewart, Pitts, Craig, & Dieruf, 1966; Wender, 1971), and HK is the most common child psychiatric disability seen in referrals to mental health clinics. This disorder represents a major social and educational problem which may lead to poor adjustment later in life for these children (Safer, & Allen, 1976).

The etiology of behavior patterns resembling HK has been associated with neurological dysfunctions resulting from head trauma (Kasanin, 1929), premature birth and pre- or perinatal birth complications (Knobloch, & Pasamanick, 1966; Rogers, Lilenfeld, & Pasamanick, 1955), and lead poisoning (Thurston, Middelkamp, & Mason, 1955) but it became obvious that neurological dysfunction could not account for all the cases of HK behaviors. Congenital etiology has also been proposed and supported by observations of the presence of minor physical abnormalities (Firestone, Lewy, & Douglas, 1976; Rapoport, & Quinn, 1975: Waldrop, Bell, McLaughlin, & Halverson, 1978) and an increased occurrence of HK within families (Cantwell, 1975; Omenn, 1973; Safer, 1973; Safer, & Allen, 1976). Furthermore, the association of HK with brain damage was enhanced by clinical findings of "soft" neurological signs and borderline abnormalities in brain wave (electroencephalographic: EEG) activity (Clements, & Peters, 1962). However, Dubey (1976) suggested that only a small subgroup of HK children display obvious overt neurological abnormality. HK is still thought to be organically

determined, although the great volume of research done in this field has yet to delineate the brain structure(s) and system dysfunction(s) responsible for this disorder.

Recent studies have been increasingly oriented toward an explanation of HK based on difficulties in mechanisms of arousal and attention -- concepts which are themselves ambiguous and controversial (Ferguson, & Pappas, 1979). A variety of brain mechanisms have been proposed (neurochemical, anatomical, and physiological) to explain the brain dysfunction(s) responsible for the behaviors subsumed under the HK label. Most of the proposed mechanisms possess a common underlying feature: an abnormality in arousal levels or arousal functions of the central nervous system (CNS) (Ferguson, & Pappas, 1979). Therefore, HK is generally viewed as a dysfunction in the processing of incoming stimuli (Rosenthal, & Allen, 1978).

One theoretical approach to the attention-arousal problem in HK attributes the excessive motor activity to overstimulation resulting from an inability to filter irrelevant stimuli (Strauss, & Kephart, 1955; Strauss, & Lehtinen, 1947). Although this defective attentional mechanism may lie at a higher cognitive level (Douglas, 1972; Traver, & Hallahan, 1974), others (Laufer, Denhoff, & Solomans, 1957) have contended that the underlying physiological basis for this notion was hyperarousal of the various brain stem and subcortical structures. Cruickshank, Bentzen, Ratzeburg, and Tannhauser (1961) tried to confirm this hypothesis by placing HK children in a reduced stimulus environment over a one year period.

The HK group improved in their performance but not to a significant degree relative to a similarly treated control group. These latter results, together with other nonsupportive findings (Douglas, 1972; Zuk, 1963) have tended to disconfirm this hyperarousal hypothesis.

Another recently proposed explanation for HK behavior (Zentall, 1975; Zentall, 1977) also focuses on arousal mechanisms. This approach implies that the increased motoric activity observed in the HK child is secondary to a lowered level of arousal of the CNS and insufficient CNS inhibition (Satterfield, Cantwell, & Satterfield, 1974). Therefore, the excessive activity is viewed as "stimulus-seeking behavior" which serves to maintain CNS stimulation at some optimal level (homeostatic balance) by increasing the proprioceptive and exteroceptive sensory input. Consequently, this notion views the behavior of the HK child as emanating from a condition of physiological under- or hypoarousal. An extensive volume of research has been gathered to support the hypoarousal hypothesis in a variety of situations involving environmental stimulation (Douglas, 1972; Freedman, & Greenblatt, 1960; Heron, Doane, & Scott, 1956; Pope, 1970; Reardon, & Bell, 1970; Scott, Bexton, Heron, & Doane, 1959; Zentall, 1975; Zentall, & Zentall, 1976; Zubec, 1963).

The use and effects of stimulant drugs (primarily methylphenidate and dextroamphetamine) on HK behavior have also been explained within the hyperarousal/hypoarousal context. Generally, these drugs

have been reported to suppress restless and impulsive behavior and to facilitate attentional abilities in HK children (Campbell, Douglas, & Morgenstern, 1971; Conners, 1970; Knights, & Hinton, 1969). Within the hyperarousal (overstimulation) hypothesis, the CNS drug effects seem to act in a paradoxical manner in HK children because in normal adults administration of these compounds increases arousal and activity and enhances performance (Sroufe, & Stewart, 1973; Wender, 1971). According to the hypoarousal (understimulation) model, stimulant drugs act in an excitatory manner, which in HK children serves to increase level of arousal to an optimal level (Rosenthal, 1973; Satterfield, 1976) and result in decreased HK behavior (Zentall, 1975).

Psychophysiological Parameters

The behavior of the HK child does not provide definitive clues to the basis or origin of his physiological state of arousal. Questions related to the nature of a postulated underlying arousal dysfunction can be approached by using psychophysiological parameters. The search for these measures has gained increasing popularity in the past decade, and several different measures have been assessed to provide information regarding state of arousal, including EEG activity, evoked potentials, heart rate, finger pulse volume, blood pressure, electrodermal, and pupillometric activity. The following literature review will focus on two of the more intensely investigated measures -- EEG activity (central: cortical index) and electrodermal activity (peripheral: autonomic index).

Reports of abnormal spontaneous EEG activity in HK children have ranged from 18-47% of the populations studied. Excessive slow-wave activity, indicative of lower arousal levels, has been the major clinical EEG abnormality reported (Capute, Niedermeyer, & Richardson, 1968; Klinkerfuss, Lange, Weinberg, & O'Leary, 1965; Knights, & Hinton, 1969; Paine, Werry, & Quay, 1968; Satterfield, Cantwell, Saul, Lesser, & Podosin, 1973; Satterfield, Cantwell, Saul, & Yusin, 1974; Wikler, Dixon, & Parker, 1970). Grunewald - Zuberbier, Grunewald, and Rasche (1975), examining specific EEG frequency bands, found more alpha and less beta wave activity in HK compared to non-HK "maladjusted" children. Surwillo (1977) described the EEG patterns of his HK sample as being very similar to those exhibited by younger-aged normal children. While on stimulant medication, these patterns became normalized. It was again suggested that these HK children suffered from a development lag.

A variety of measures of changes in skin resistance or electrodermal responses have been studied in both medicated and unmedicated HK children. These measures have included basal skin conductance, galvanic skin response and spontaneous skin potential response. The results of these studies have been conflicting. For example, when basal skin conductance (BSC) was studied, some investigators have found no differences between groups (Boydston, Ackerman, Stevens, Clements, Peters, & Dykman, 1968; Cohen, & Douglas, 1972; Ferguson, Simpson,

& Trites, 1976; Firestone, & Douglas, 1975; Montagu, 1975; Satterfield, Cantwell, Lesser, & Podosin, 1972; Spring, Greenberg, Scott, & Hopwood, 1974; Zahn, Abate, Little, & Wender, 1975), whereas others have found lower BSC levels in HK subjects (Kløve, & Bu, 1976 cited in Ferguson, & Pappas, 1979; Satterfield, & Dawson, 1971; Shouse, & Lubar, 1978), and still others have noted higher BSC in HK children (Satterfield, Atonian, Brashears, Burleigh, & Dawson, 1974; Zahn, Little, & Wender, 1978). Conners (1976) also found higher BSC levels when HK subjects were asked to inhibit responding on a task.

The effects of stimulant medication on skin conductance levels have also been studied. Cohen, Douglas and Morgenstern (1971) found reductions, Satterfield, and Dawson (1971) reported increases, and Spring, Yellin, and Greenberg (1976) noted no change in the levels of skin conductance after drug administration.

Studies on spontaneous galvanic skin responses (GSR) have reported either no difference (Barkley, & Jackson, 1977; Spring et al., 1974; Zahn et al., 1975; Zahn et al., 1978) or fewer and smaller non-specific responses (Satterfield, & Dawson, 1971) in HK children compared to controls. Stimulant medication has been reported to increase the number of GSRs (Satterfield, & Dawson, 1971; Spring et al., 1974), but the finding is not unanimous (Cohen et al., 1971). The quality of GSRs has been noted to be a good predictor of vigilance performance (Surwillo, & Quilter, 1965). Since stimulant drugs have been reported to improve vigilance in HK children (Sykes, Douglas, Weiss, & Minde, 1971), it has been suggested by Hastings and Barkley (1978) and Rosenthal and Allen (1978) that a subgroup of HK children

may exist which is less autonomically reactive.

In the only study to date of spontaneous skin potential responses (SSPRs) during sleep comparing HK children to normal controls (Busby, 1980), no group differences were reported.

In general, the search for psychophysiological indexes specific to HK has not provided definitive conclusions regarding the state of arousal in these children. The studies of cortical activity suggest that some HK children may be underaroused or exhibit patterns of an immature nature. The studies on autonomic indexes of arousal have shown that the HK subjects generally do not differ from controls on resting-baseline measures. In studies which dealt with evoked responses (environmental stimulation), the results have been more consistently supportive of a underarousability hypothesis for some HK children (Conners, 1976; Conners, & Rothschild, 1973; Sroufe, Sonies, West, & Wright, 1973). It has therefore been suggested (Hastings and Barkley, 1978) that perhaps HK and normal children do not differ on arousal (resting-baseline) per se, but rather in the inability of HK children to react optimally to stimulation. The results of stimulant drug effects suggest that these drugs are generally arousal-producing and influence many cortical and autonomic measures in a normalizing manner. Hastings and Barkley (1978) have proposed that since similar effects have also been observed in normal children (Rapoport, Buchsbaum, Zahn, Weingartner, Ludlow, & Mikkelson, 1978), a mere organic differentiation between HK and normal children is unlikely.

Sleep and Hyperkinesis

The psychophysiological literature reviewed suggests that a common underlying feature in HK may be a dysfunction in the mechanisms producing arousal. Since both sleep and waking (arousal) mechanisms are intimately correlated, detailed studies of sleep in HK children might provide evidence on the postulated arousal dysfunction. Furthermore, these findings would be free from confounding variables usually encountered during wakefulness (e.g., stress, expectations, and undefined variations in arousal level).

Despite the potential of sleep studies to provide more complete understanding of physiological mechanisms involved in HK, there has been little research done in the field. In the following paragraphs, a detailed review of this limited literature is presented.

The first published study investigating sleep patterns in HK children was that by Luisada (1969) who postulated that since the overt behavior of adults deprived of rapid eye movement (REM) sleep clinically resembles that of children with HK, the latter should exhibit measurable variations in REM amounts. In his study, the sleep of 11 HK children (mean age = 9.2 years) was compared to that of a control group (n = 4; mean age = 9.5 years). Comparisons were made on only two dependent variables; -- REM percentage and percentage of REM awakenings -- and significant differences were found for both variables, i.e., HK children had less REM sleep

(20% versus 24%) and more REM periods disrupted by awakenings (21.5% versus 4.5%). In addition, a positive correlation was noted between the degree of qualitatively observed HK and percentage of REM awakenings.

In a second study (Small, Hibi, & Feinberg, 1971) the sleep of three markedly HK males with MBD, was compared to that of seven age-matched normal controls during baseline (5 consecutive nights) and medication periods (dextroamphetamine; 3 consecutive nights, 5 mg. each morning; 2 to 4 weeks of medication adjustment; 3 consecutive nights using optimal dosage of 20 mg./day; 3 consecutive nights on placebo after withdrawal of medication). Comparisons were made on a number of sleep variables, including total sleep time, amounts and cycles of sleep stages, and events during sleep stages (muscle activity during REM and NREM sleep and eye movements during REM). Only four measures revealed differences between groups during baseline. Three of these differences were related to high muscle activity (a presumptive indicator of restlessness) during the NREM sleep periods (HK had greater percentage of 20-sec epochs containing high muscle activity compared to controls), and the fourth was a shorter sleep onset latency in HK children. Medication administration was associated with an increased latency to sleep onset, a reduction in the number of sleep cycles, an increased latency to the first REM period and increased absolute and percentage amounts of slow-wave sleep (stages 3 and 4) as a proportion of total NREM sleep. Other sleep measures were not significantly modified.

Feinberg, Hibi, Cavness, Westerman, and Small (1974) further investigated the effects of amphetamines on the sleep of MBD children. The sleep of eight HK (MBD) males (three were from the study reported above) was studied during baseline (five nights), drug administration (three nights), and during withdrawal from medication (two or three nights). Six age-matched males served as the control group and slept in the laboratory four or five consecutive nights. No significant group differences were observed during baseline or medication periods for sleep measures. However, differences were observed within the MBD group across the different medication conditions. Eye movement density and eye movement burst indexes were significantly increased with the administration of stimulant drugs, and sleep latency was reduced and sleep time increased after stimulant withdrawal.

Haig, Schroeder, and Schroeder (1974) compared the sleep patterns of six HK boys (age 8.75 to 14.6 years) taking methylphenidate daily (10 to 61.5mg) with those of six normal boys from a previous study (age 6.5 to 12 years). In the first part of the study, the subjects slept five consecutive nights in the laboratory, of which the last three were recorded polygraphically. In the second part of the study, four of the six hyperactive subjects were recorded for three nights one week after drug withdrawal. Significant increases in latency to sleep onset and the first REM period were observed for the medicated hyperactive subjects relative to controls. Other sleep measures were not altered.

No significant differences were observed within the experimental group across the medication/non-medication paradigm with respect to sleep measures.

Another study (Nahas and Krynicki, 1977) investigated the effects of methylphenidate on the sleep patterns of four HK males (age 8 to 9.5 years) during an eight night paradigm; two adaptation nights, no recording; two baseline non-medication recording nights; two experimental-medication (20 mg. total) recording nights (1st and 21st (final) night of drug administration); and two withdrawal recording nights immediately following drug discontinuation. All patients improved behaviorally during drug administration. The standard sleep variables, e.g., sleep stages and sleep time, were not affected by methylphenidate (off-on-off conditions). Ultradian rhythms known to be present for REM and delta (stages 3 and 4) sleep (Naitoh, Johnson, Lubin, & Nute, 1973) were examined on three nights, one from each recording condition. Nocturnal ultradian rhythms were found to be present and an increase in the delta cycle length was reported.

The sleep patterns of five HK males (age 6 years 1 month to 8 years 5 months) were compared to those of seven normal children, five boys and two girls (age 6 years 7 months to 8 years 8 months) by Khan and Rechtschaffen (1978). Each subject was recorded three nights: one adaptation and two experimental. There were no significant group differences in percentage time asleep or percentage of time spent in any sleep stage. However, HK children had fewer

12-14 Hz cortical sleep spindles in stage 2 compared to controls. In an extended part of the study, three of the five young hyperkinetic children were treated with Ritalin and clinical improvement as well as doubled spindle frequency were observed. In addition, three older boys (12-13 years) previously diagnosed as "hyperkinetic" but showing minimal hyperactivity at the time of the recording, had "weighted" spindle scores similar to those of three age-matched controls: i.e., 15.1 and 13.4 respectively.

Stahl, Orr, and Griffiths (1979) compared the standard sleep variables and nocturnal levels of growth hormone of five unmedicated HK children of small stature to those of nine normal controls in a study involving four consecutive nights or recordings. The amounts and peaking of growth hormone occurred within the normal range and no significant group differences were observed for the sleep measures.

Busby (1980) studied sleep patterns and waking ultradian rhythms in performance and motility in eleven unmedicated HK males (age 8-12 years) and eleven age-and-sex-matched controls. The sleep recordings were monitored for five consecutive nights and the waking ultradian rhythms in performance (detection and false positive) and motility (global body movement and limb movement during task and off-task periods) were measured during the days following the third and fourth recording nights. A significantly longer REM onset latency was observed for the HK group as well as marginally significant greater amounts of movement time, body movements and NREM twitches for this group compared to controls. No significant group differences were observed for the other sleep parameters. On waking attention and motility

measures, HK subjects made significantly fewer detections and were more active during off-task periods on both days. Also, greater amounts of limb movement on task, were observed in the HK group compared to controls. Global body movements and false positive scores did not differentiate the groups.

The eight studies reviewed above constitute all studies published regarding sleep patterns and events in HK children. All totalled, the sleep patterns of fifty HK children have been examined. All studies except that by Luisada (1969) essentially agree that: a) baseline sleep patterns in HK children do not differ markedly from those of normal controls; and b) stimulants administered to HK children do not dramatically change ensuing sleep patterns. The predominantly observed normality of sleep patterns in HK children calls into question the hypothesized role of arousal mechanisms in this disorder, at least as determined by measurements of sleep. However, many interstudy inconsistencies present limitations as to the comparibility of results. Symptomatology, subject selection procedures, and the use of varying drugs and dosages of stimulant medication across studies make comparisons difficult. Also, the generalizability of these findings may be further limited by: a) small samples of both HK and controls in most studies; b) the use of limited number of measures in one study; c) the lack of control recordings in two studies; and, d) differing experimental designs (e.g. number of nights recorded on drug/off drug paradigms, etc.).

The purpose of the present investigation was to provide additional information pertaining to level of arousal in hyperkinesis by examining electrographic measures of arousal (EEG, autonomic, EOG & EMG indexes) during wakefulness and sleep in unmedicated HK and control groups matched for age and sex over a substantial baseline recording period. In addition to the standard sleep parameters, e.g. sleep onset latency, minutes and percentage of the different sleep stages, REM cycles, etc., the study will focus upon more transient phasic events, such as twitches and body movements. The present study will provide new information regarding: eye movement (EM) activity (horizontal, vertical and mixed) and cortical sleep spindles. Further elaboration is made below on three of these measures which are of special relevance to HK-normal comparisons (SSPR, EMs, and spindle activity).

Spontaneous Skin Potential Response: SSPRs have been found to be more frequent during stages 3 and 4 (slow-wave sleep, SW) compared to REM sleep in children and normal young adults (Broughton, Poire, & Tassinari, 1965; Johnson, & Lubin, 1966; Lester, Burch, & Dossett, 1967). Spontaneous electrodermal activity during SW, called "storming", seems to resemble that of wakefulness and has been related to some presleep variables such as stress (Lester et al., 1967; McDonald, Shallenberger, Kozesko, & Kinzy, 1976). Accordingly, if viewed within the hypo/hyperarousal context, the presence in HK children of lower electrodermal activity compared to normal children

would be consistent with the hypoarousal view. On the other hand, relatively greater electrodermal activity in HK children would be consistant with the hyperarousal model.

Eye Movements: EM density has been associated with low amplitude EEG of irregular frequency (5-30 μv; 15-20/sec and 5-8/sec predominating), higher respiration rate, and peaks of overt bodily activity in normal adults (Aserinsky, & Kleitman, 1953). It has been suggested that EM density measures during REM sleep provide an index of the overall amount of CNS activity in children and adults (Aserinsky, & Kleitman, 1955).

EM activity has been studied in small groups of young normals (mean age = 25.2 years) and normal adults (mean age = 75.0 years) (Feinberg, Braun, & Koresko, 1969). These investigators used two different methods concurrently for recording EMs. One method (H) recorded only horizontal EMs while the other method (VH) recorded both vertical and horizontal EMs. Method VH revealed significantly more EMs compared to method H in both groups, suggesting to the authors that horizontal EMs do not represent a random sample of all EMs occurring during sleep. Also, normal adults were found to have fewer vertical EMs compared to young normals. It has been hypothesized that the proportion of vertical EMs reflects the intensity of REM sleep processes (Feinberg et al., 1969).

Busby (1980) studied EM density in HK and normal children. Although no group differences were observed on this measure, significant increases in EM density from the first five-minute block

to the second five-minute block within a REM period (REMP) for all children were reported. Furthermore, a progressive increase in EM density across REMPs was obtained. Different EM categories (horizontal, vertical) were not examined in this study.

Spindle activity: Metcalf (1969) defined a spindle as a brief burst of rhythmic activity standing out from slower or non-rhythmic, lower amplitude activity. Spindles, usually fusiform in shape and ranging from 12 to 14 Hz in frequency, last not less than .4 second and may persist for 3-5 seconds. Longer spindle burst duration (4-6 sec) have been observed but are rare. Spindles develop maximally in prevertex regions, often shift between hemispheres and may be bilaterally symmetrical. Metcalf (1969) carried out a study to examine impact of experience upon innate maturational factors (EEG development) and observed that sleep spindles followed a regular time-dependent course. He reported that prematurely born infants show EEG development which is approximately four weeks in advance of term infants and suggested that this reflected an interaction of maturation and experience (in this case, extrauterine existence).

It has been found that during spindle activity, the nerve fibers conveying impulses away from the cerebral cortex were less excitable, suggesting a reduction in motoneuronal activity (Brookhart, & Zanchetti, 1956) -- a condition consistent with transient hypoarousal. Buchwald and Eldred (1961) reported that discharges of the gamma efferent system which provides background activity out of

which more major activity emerges, were reduced during sleep spindles.

Roth, Sterman, and Clement (1967) described a sensory motor rhythm (SMR) in awake cats, which had the same general cortical location (sensorimotor cortex) and characteristics (12-16 Hz EEG synchronization) as a spontaneous sleep spindle. Sterman and Wyrwicka (1967) showed that the SMR could be operantly conditioned and was associated behaviorally with an absence of phasic motor activity. After conditioning for SMR, a decrease of phasic movements together with an increase of spindle activity was observed (Sterman, Howe, & McDonald, 1970). Howe and Sterman (1972) proposed that the generators for the SMR and sleep spindle phenomena are probably located at the thalamic level and function as part of a mechanism concerned with the specific suppression of phasic motor activity.

Sleep spindles have also been studied in relation to body movements (Johnson, Hanson and Bickford, 1976) who examined the effects of Flurazepam (a central muscle relaxant) on sleep spindles and K-complexes of five subjects complaining of sleep problems. They found that Flurazepam significantly increased the spindle rate by the second night of drug administration compared to baseline. Also, Flurazepam was found to decrease the rate/min of K-complexes and the time spent in stage 4. However, no relation was found between the proximity of a spindle burst and the occurrence of movement. They have postulated that instead of motor activity inhibition, sleep spindles serve to raise the arousal threshold which, in turn, helps to maintain sleep by

reducing the probability of being awakened.

Therefore, if spindle activity is interpreted within the hypo-hyperarousal context, the HK child displaying more spindle activity would be viewed as being hypoaroused, whereas less spindle activity would be consistent with a hyperaroused condition.

Hypotheses

Tonic Sleep Measures. Based upon the results of past studies investigating sleep patterns in HK children, it is hypothesized that the amounts and distribution of the various sleep stages will not significantly differentiate the two groups. Specifically, amounts and percentage of standard sleep stages, REM cycles, latencies, etc., will not vary between groups.

Phasic Sleep Events. It is predicted that phasic motor activation, i.e., BM and twitches, will differentiate the HK group from the control group. The former group is expected to have increased activity for these indices. Correspondingly, it is hypothesized that cortical spindle frequency, a motor-related index, will be reduced in HK children relative to controls.

It is hypothesized that SSPR measures will not differentiate the HK group from the control group, and that the highest SSPR frequency will be associated with stage 4 sleep and the lowest rate with REM sleep.

EM density measures are not expected to differentiate the two groups, but will increase across REM periods for both groups.

Chapter II

METHODOLOGY

Subjects

Five unmedicated HK male children (9-12 years old, mean age = 10.6 years) and five normal male controls (9-12 years old, mean age = 10.7 years) served as subjects. Consensus among parents, teachers, and a psychiatrist was required for classification of children as hyperkinetic. Parents (mostly mothers) completed the Conners Parent's Questionnaire (1970; long form, see Appendix A) and scores of ≥ 15 on the hyperactivity scale of this questionnaire were required for a diagnosis of HK.[1] Teachers completed the Conners School Report (1969; see Appendix B) on which an index of ≥ 1.5 was required for HK classification. The psychiatrist utilized the criteria outlined in the Diagnostic and Statistical Manual of Mental Disorders (DSMII) to diagnose for "Hyperkinetic reaction of childhood" (308). The HK diagnosis was

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[1] A mother of one child included in the HK group rated her son below the score 15 on the Parent's Questionnaire. However, the child had been treated for HK since the age of 18 months, and was an inpatient at the time of data collection. The psychiatrist questioned the validity of the mother's responses on the questionnaire and concluded that the child was similar to the other children in the HK group. On the basis of these considerations, the child was included in the HK group, and in fact his responses on the variables measured were similar to children in the HK group.

based upon the following traits which are generally agreed upon as being persistently or recurrently present in children labelled HK: excessive motor activity, short attention span, distractibility, labile emotions, impulsiveness, and poor scholastic performance (Goyette, Conners, & Ulrich, 1978; Renshaw, 1974, Stewart et al., 1966; Wender, 1972). All HK subjects had a long-standing history of this symptomatology (at least 6 years). Four subjects had been treated with Ritalin and one with Mellaril. All HK children underwent a drug washout period of three weeks duration prior to data collection. Of the five HK subjects, two were inpatients and three were outpatients (2 in the day care center) at the Royal Ottawa Hospital (R.O.H.) under treatment for hyperkinesis. I.Q. measures were obtained for the HK children prior to acceptance into the study to screen for the presence of mental retardation. All HK subjects scored within the normal range on the three WISC-R scales. Subject group characteristics are summarized in Table 1.

Control subjects were recruited from the local school system and all lived at home with at least one parent. Although they were not tested for I.Q. measures and their teachers did not evaluate these children on the Conners School Report, all control subjects were in the appropriate grade for their age group and all were rated lower than 15 on the Parent's Questionnaire.

Children with the following symptoms or classifications were excluded from the study: over-anxious reactions, unsocialized aggressive reactions, peripheral sensory loss, epilepsy, mental

Table 1

Means (S.D.) Age, I.Q., and Behavioral Ratings

for Hyperkinetic Children

Group	Variable					
	Age	Verbal I. Q.	Performance I. Q.	Full Scale I. Q.	Conners Parent	Conners Teacher
HK	10.6 (0.75)	98.6 (8.85)	109.6 (13.24)	103.8 (12.11)	22.0 (9.06)	2.08 (0.62)

retardation, normal constitutional hyperkinesis, post-traumatic organic brain syndrome, encephalitis, toxic organic brain syndrome (drug), Sydenham's Chorea, or major sleep disturbances (e.g., somnambulism, enuresis, pavor nocturnus).

Prior to acceptance into the study all children underwent a clinical EEG evaluation to screen for gross abnormalities in brain wave patterns. This session also served to familiarize the children with the laboratory, procedures, and apparatus. The study was fully explained to the qualifying children and their parents, after which informed consent (see Appendix C) was obtained from the parents.

Experimental Design

Subjects reported to the sleep laboratory (R.O.H.) for electrode application one hour prior to their normal bedtime. Electroencephalographic (EEG: C_3/A_2), electro-oculographic (EOG: molopolar horizontal and bipolar vertical eye movements), electromyographic (EMG: orbicularis oris), and spontaneous skin potential response (SSPR: palm referenced to the forearm; silver-silver chloride electrodes) recordings were obtained using a Grass (Model 78D) polygraph. Waking measures of these variables were recorded prior to the child's bedtime (lights out). Sleep patterns and associated measures were recorded continuously on paper (15 mm/sec) for five consecutive nights. Data from nights 3 and 5 were also recorded on magnetic tape (Hewlett-Packard, Model 8868A instrument recorder). Total bedtime (TBT) was limited to 9 hours.

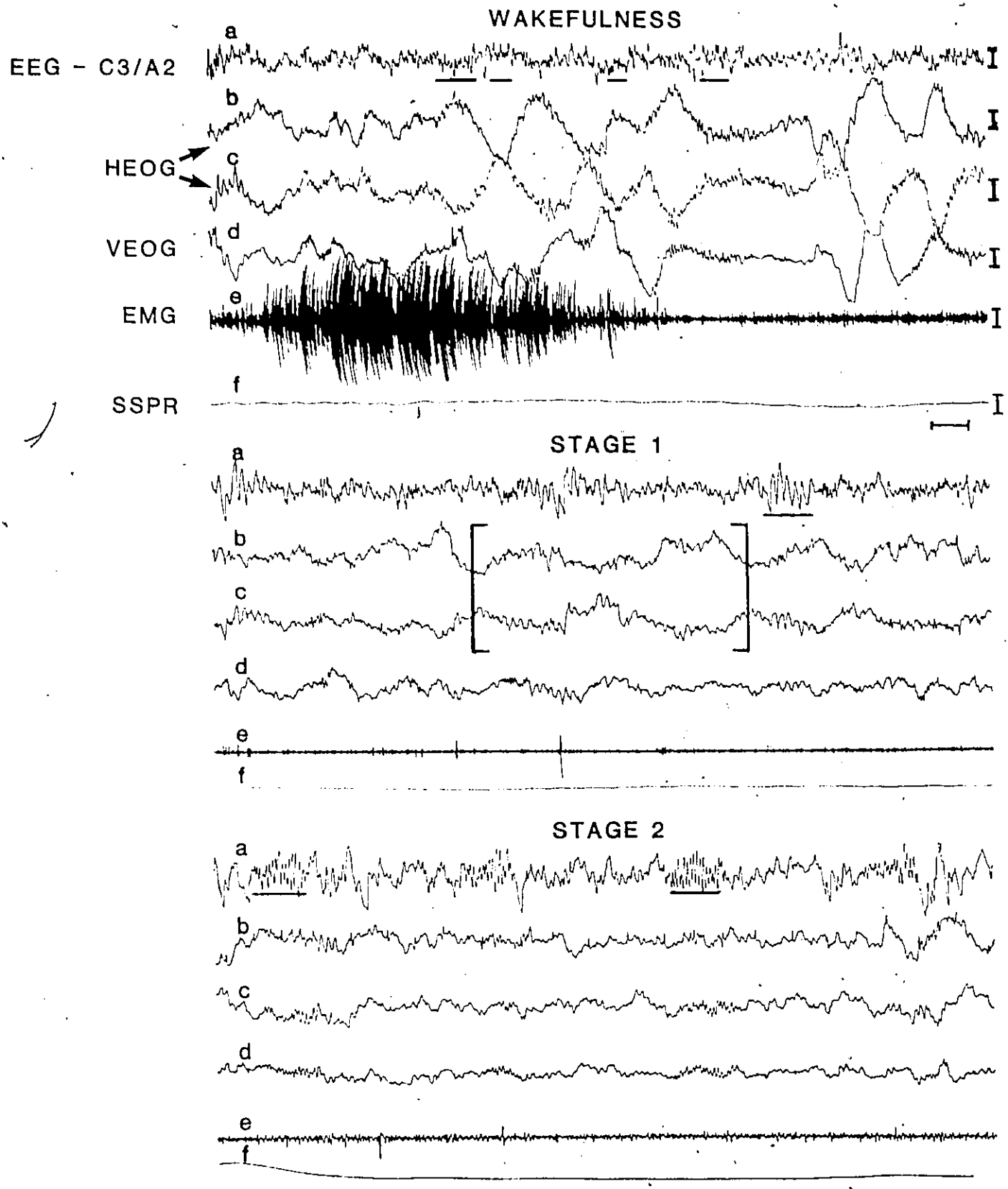
Data Analysis

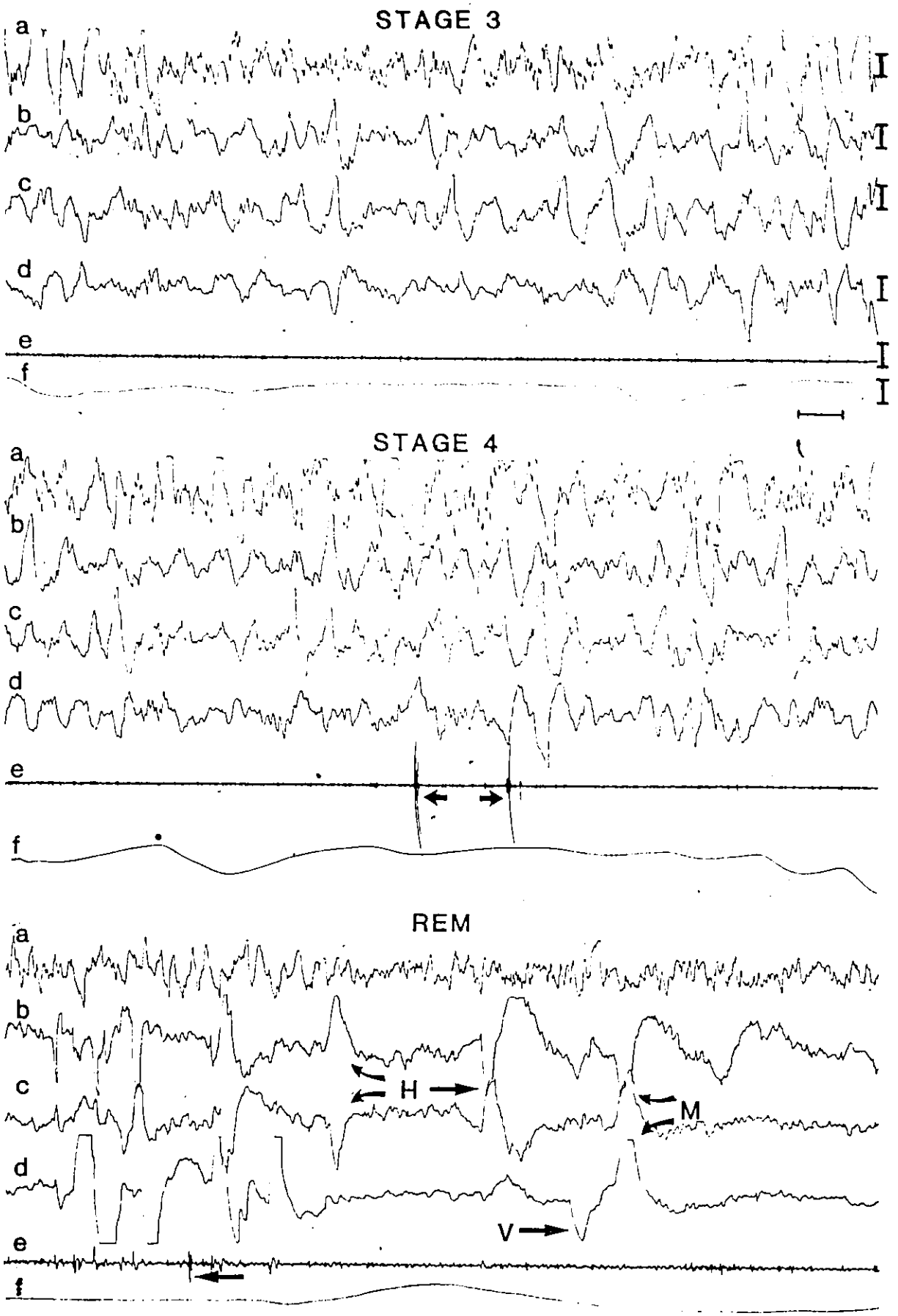
Sleep records were coded and scored blind using the standardized criteria of Rechtschaffen and Kales (1968). Two independent scorers achieved greater than 90% agreement on pilot data prior to experimental data scoring. Examples of the different behavioral categories examined are illustrated in Figures 1a and 1b and an explanation of scoring criteria will now be presented. Wakefulness (W) was scored when an epoch (20 seconds) was characterized by EEG containing 50% or more of alpha activity (8-12cps) and/or low voltage, mixed frequency activity, moderate to high levels of EMG activity, and occasional rapid eye movements and blinks. Stage 1 (S1) was scored when $\geq$ 50% of the epoch was characterized by slow rolling eye movements and EEG consisting of low voltage, mixed frequency (predominantly 2-7Hz range) activity with occasional vertex sharp waves. Tonic EMG levels in S1 were usually lower than those of relaxed W. Stage 2 (S2) was defined by the presence of sleep spindles (11-15 cps, 25μv, and lasting $\geq$ 0.5 sec) and/or K-complexes (negative sharp wave immediately followed by a positive component with a minimum total duration 0.5 sec) on a background of low voltage, mixed frequency EEG activity. An epoch was scored stage 3 (S3) when at least 20% but not more than 50% of the epoch consisted of delta waves (2 Hz or slower, 75μv from peak to peak). Stage 4 (S4) was defined by the presence of delta activity for more than 50% of the epoch. Stage REM was characterized by a relatively low voltage, mixed frequency EEG in conjunction with rapid eye movements and low level EMG.

Figure 1 (a) Recording samples of sleep stages.

Note: underscored alpha in EEG channel in W; underlined theta in EEG and slow-rolling eye movements (brackets, EOG) in S1; underscored spindles in trace a in S2.

(b) Note: Twitches (trace e, arrows) and SSPR (trace f, dot) in S4; horizontal (H), vertical (V), mixed (M) eye movements and twitch (trace e, arrow) in REM. All recording calibrations equal 50 μV and time marker is 1 second.


WAKEFULNESS
EEG - C3/A2
HEOG
VEOG
EMG
SSPR
a
b
c
d
e
f
STAGE 1
STAGE 2

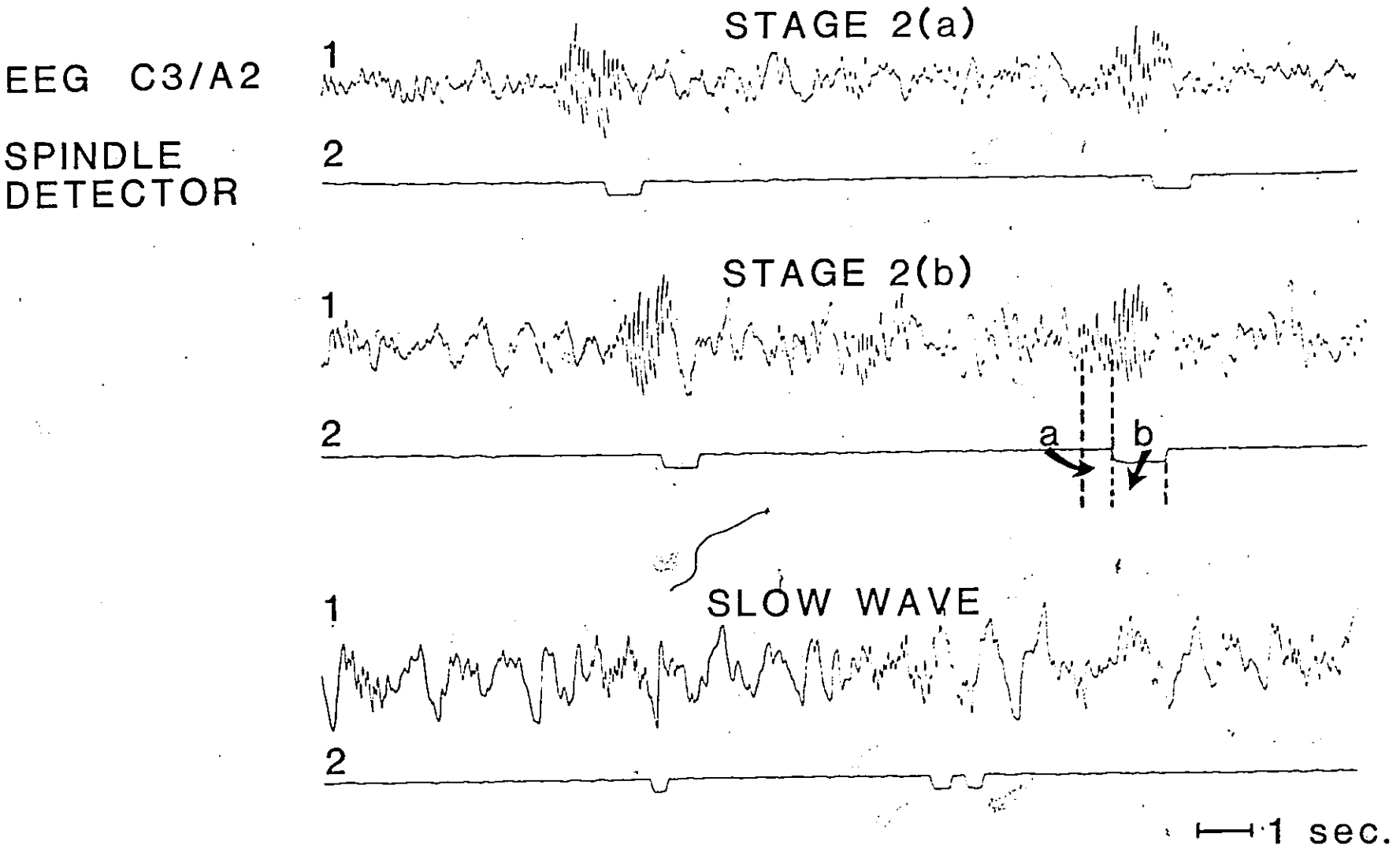

STAGE 3
a
b
c
d
e
f
I
STAGE 4
REM
H
M
V

SSPRs were scored according to the Johnson and Lubin (1966) criteria i.e., deflections of one millimeter representing a 100μv potential change and lasting ≥ 1 sec. The input impedance of the SPR amplifier was set at one megohm. Deflections occurring in conjunction with movements or within 5 sec after movement or in response to external stimuli (e.g. loud noise) were also excluded from analysis.

Eye movement density measures during REM sleep were scored using Aserinsky's (1969) criteria (number of two-second mini-epochs within a REM period containing at least one eye movement, divided by the total number of mini-epochs within that REM period). An eye movement was defined as a one millimeter (5° visual angle shift) pen deflection.

Analyses were also conducted on cortical sleep spindle activity. A phase locked loop (PLL) device (Broughton, Healey, Maru, Green, & Pagurek, 1978) was used to detect sleep spindles in tape recorded NREM sleep patterns. A spindle was defined as a sinusoidal 11.5-15 Hz activity, lasting ≥ 0.5 sec, and being ≥ 25μv in amplitude with an inter-spindle interval of ≥ 0.5 sec. The output of the detector (see Figure 2) was recorded on paper (Mingo Graph EEG 16) and on a magnetic tape (3 3/4ips), and these taped data were subsequently fed to a laboratory computer (PDP11). Computer analysis divided the data into consecutive two-minute bins, and determined the total number of spindles. Although spindle duration might provide important information regarding arousal levels, it was not analysed

Figure 2. Sample output of the spindle detector during sleep characterized by increasing amount of slow-wave activity. Note: .5 second delay (a) from spindle onset to spindle detector pulse (b).


STAGE 2(a)
EEG C3/A2
1
SPINDLE DETECTOR
2
STAGE 2(b)
1
2
a
b
SLOW WAVE
1
2
1 sec.

in the present study because the detector was designed to detect spindle incidence but not spindle duration.

Several tonic variables were scored from the polygraphic recordings including; movement time (MT), defined as an obscurring of the EEG and the EOG channels by superimposed EMG for at least 50% of the epoch; body movements (BM), defined as transient increases in EMG activity lasting > 1 sec but < 10 sec, with an amplitude at least twice that of the immediately preceding EMG tracing; REM and NREM twitches, defined as transient biphasic EMG deflections ($\geq$ 2 times the preceding baseline level) and $\leq$ 1 sec in duration.

The first two nights were considered laboratory adaptation nights and were excluded from group comparison analyses. However, trend analyses were conducted using data from all recording nights.

Group differences among the various sleep measures were statistically compared using individual analysis of variance with repeated measures (BMD program P2V with orthogonal component analysis to test for trends). Post hoc Newman-Keuls tests and t-tests were performed when indicated by significant F ratios. The $\leq .05$ level of significance was chosen for all statistical comparisons.

Chapter III

RESULTS

Tonic Sleep Measures

Analyses performed on data from all recording nights for latency to sleep onset or any sleep stages (Table 2), as well as for number of minutes and percentage amounts of conventional sleep stages (Tables 3 and 4) did not reveal any significant group or night effects. Since group means for latency to REM onset seemed markedly different on nights 3-5, t-tests were conducted comparing HK to controls on this variable. These tests revealed significant group differences on night 4 ($t = 2.05$, $df = 8$, $p < .05$) and night 5 ($t = 2.42$, $df = 8$, $p < .025$), with the HK group showing a longer latency (see Figure 3). No significant differences were observed between groups or across nights 1-3 and 5 for REM cycle frequency and length (Table 5).

Motor Activity Measures

Analyses conducted on data for nights 1, 3 and 5 for MT revealed a significant main group by night effect for minutes {$F (2,24) = 3.70$, $p < .03$} and a marginally significant main group by night effect for percentages {$F (2,24) = 2.97$, $p < .07$}. Post-hoc tests revealed that HK subjects exhibited greater amounts of MT on night one relative to controls ($p < .05$) for both absolute and percentage measurements. However, no significant group differences were present for this variable on nights 3 and 5.

Table 2

Means (S. D.) of Latencies (minutes)

Variable	Group	Night 1	2	3	4	5
Lights Out to Sleep Onset*	HK	7.64(6.72)	5.28(4.03)	10.82(10.76)	8.54(8.82)	8.08(5.10)
	C	9.26(10.05)	13.34(10.62)	10.52(14.78)	6.84(7.18)	9.06(10.43)
Lights Out to Stage 1	HK	6.90(5.83)	4.40(3.23)	10.22(10.82)	5.26(4.19)	6.52(2.91)
	C	8.40(10.26)	13.34(16.62)	7.98(12.62)	6.52(7.36)	6.26(6.84)
Sleep Onset to Stage 2	HK	2.64(1.50)	2.60(0.96)	4.40(2.87)	2.34(1.65)	8.44(13.23)
	C	3.20(1.32)	4.40(2.51)	3.38(2.12)	2.72(0.89)	3.76(1.67)
Sleep Onset to Stage 3	HK	12.20(3.87)	9.00(4.95)	9.54(3.83)	11.06(2.14)	11.14(4.42)
	C	8.78(3.66)	11.12(3.73)	33.80(51.60)	9.88(5.36)	10.88(2.67)
Sleep Onset to Stage 4	HK	19.48(10.05)	16.74(9.04)	19.08(4.40)	14.40(2.29)	18.19(7.22)
	C	18.46(4.30)	19.40(4.49)	53.20(54.88)	18.12(8.57)	18.82(8.70)
Sleep Onset to 1st REMP	HK	141.31(40.32)	137.49(31.03)	145.97(42.51)	124.80(31.35)	143.25(27.32)
	C	138.53(28.58)	133.99(51.64)	130.34(80.96)	88.17(25.49)	97.88(31.91)

* Sleep onset latency: time from "lights out" to 5 min. of continuous sleep (stage 1).

Table 3

Means (S. D.) of Sleep Measures (Minutes)

Variable*	Group	Night 1	2	3	4	5
TBT	HK	513.77(84.91)	541.99(60.48)	551.39(26.02)	532.53(18.04)	551.65(51.22)
	C	522.53(15.86)	550.65(22.87)	550.59(24.33)	551.27(21.37)	556.39(9.43)
SPT	HK	508.91(82.22)	537.31(62.73)	540.77(31.73)	524.39(25.69)	543.31(51.91)
	C	542.99(10.52)	537.39(19.14)	541.05(30.75)	545.13(24.00)	547.93(13.95)
TST	HK	496.25(78.52)	523.45(67.65)	526.37(25.00)	509.07(22.59)	531.67(56.82)
	C	522.65(4.46)	512.19(21.63)	515.71(49.71)	532.87(28.62)	531.37(17.28)
SEI(TST/TBT)	HK	0.96(0.01)	0.96(0.02)	0.95(0.02)	0.95(0.01)	0.95(0.01)
	C	0.94(0.02)	0.92(0.03)	0.93(0.07)	0.96(0.01)	0.95(0.02)
TWT	HK	6.94(7.23)	10.20(9.97)	16.06(13.04)	13.14(6.94)	12.44(8.19)
	C	27.66(13.19)	32.25(20.98)	26.40(35.76)	9.54(7.09)	15.62(13.30)
WASO	HK	2.08(2.50)	5.54(7.28)	5.46(9.82)	5.32(3.90)	4.12(6.45)
	C	16.80(13.16)	18.94(23.05)	16.88(21.98)	3.40(2.39)	7.16(5.01)
S1	HK	38.34(26.42)	28.99(9.51)	30.13(9.67)	30.91(13.67)	24.71(13.21)
	C	50.51(17.05)	51.08(37.17)	48.43(22.16)	32.51(10.77)	35.14(21.71)
S2	HK	269.45(51.06)	254.20(55.14)	271.61(33.80)	265.11(31.50)	284.91(67.86)
	C	264.27(47.71)	253.33(55.85)	272.17(52.14)	266.05(45.35)	273.60(53.89)
S3	HK	39.54(6.92)	44.85(12.32)	32.11(12.72)	36.39(10.34)	41.25(14.82)
	C	41.60(14.88)	37.40(17.33)	41.25(16.90)	46.34(21.40)	36.25(20.20)
S4	HK	51.97(10.25)	86.77(30.86)	79.54(15.81)	69.79(10.32)	70.11(15.76)
	C	52.68(24.90)	67.51(10.87)	56.74(44.36)	69.25(32.26)	70.99(33.03)
NREM	HK	399.33(58.39)	414.87(51.19)	413.37(27.39)	402.25(29.92)	421.05(58.98)
	C	409.07(27.20)	409.33(26.30)	418.65(29.37)	414.19(22.22)	415.99(20.86)
SW	HK	89.54(6.15)	131.65(35.87)	111.65(25.09)	106.17(13.96)	111.39(26.42)
	C	94.25(23.37)	104.89(14.41)	97.99(46.84)	115.59(44.62)	107.25(33.79)
REM	HK	96.91(28.86)	108.57(28.01)	112.98(13.93)	106.79(13.50)	110.57(24.35)
	C	113.57(30.25)	102.88(9.13)	97.08(28.67)	118.65(15.94)	115.39(25.45)

Table 3

Means (S.D.) of Sleep Measures (minutes)

*Variable abbreviations not explained in text:

SPT: sleep period time; TST: total sleep time;

SEI: sleep efficiency index; TWT: total wake time;

WASO: wake after sleep onset; SW: slow-wave.

Table 4

Means (S. D.) of Sleep Measures (percentage)

Variable	Group	Night 1	2	3	4	5
TWT	HK	0.66(0.60)	2.02(2.28)	2.86(2.31)	2.44(1.35)	2.36(1.81)
	C	4.92(2.23)	5.76(3.71)	4.82(6.57)	1.74(1.32)	2.80(2.35)
WASO	HK	0.28(0.46)	1.14(1.68)	0.94(1.65)	0.98(0.72)	0.82(1.40)
	C	3.02(2.35)	3.40(4.17)	3.18(4.32)	0.62(0.43)	1.30(0.90)
S1	HK	7.38(5.35)	5.42(1.46)	5.72(1.88)	6.06(2.77)	4.56(2.31)
	C	9.64(3.25)	10.12(7.76)	9.64(4.93)	6.04(2.22)	6.58(4.06)
S2	HK	54.00(2.71)	48.75(9.14)	52.48(5.79)	52.02(4.99)	53.09(8.53)
	C	50.57(9.50)	49.15(9.10)	52.44(6.27)	49.79(6.77)	51.53(10.87)
S3	HK	7.76(0.58)	8.44(1.74)	6.04(2.34)	7.12(2.02)	7.70(2.49)
	C	7.92(2.81)	7.34(3.59)	7.98(3.31)	8.62(3.92)	6.74(3.69)
S4	HK	10.80(4.50)	16.68(5.61)	15.10(3.19)	13.66(1.80)	13.56(4.29)
	C	10.00(4.70)	13.14(1.67)	11.20(8.89)	13.14(6.11)	13.36(6.46)
NREM	HK	80.43(3.66)	79.37(3.86)	78.45(2.73)	78.92(3.10)	78.98(4.70)
	C	78.25(5.68)	79.81(2.19)	81.35(4.07)	77.77(2.42)	78.29(4.39)
SW	HK	18.53(4.82)	25.15(6.06)	21.17(4.87)	20.82(2.47)	21.28(5.89)
	C	17.96(4.32)	20.50(2.96)	19.22(9.86)	21.78(8.46)	20.14(6.42)
REM	HK	19.30(3.67)	20.55(3.92)	21.47(2.72)	21.02(3.12)	20.96(4.69)
	C	21.65(5.65)	20.12(2.24)	18.55(4.10)	22.19(2.46)	21.65(4.42)

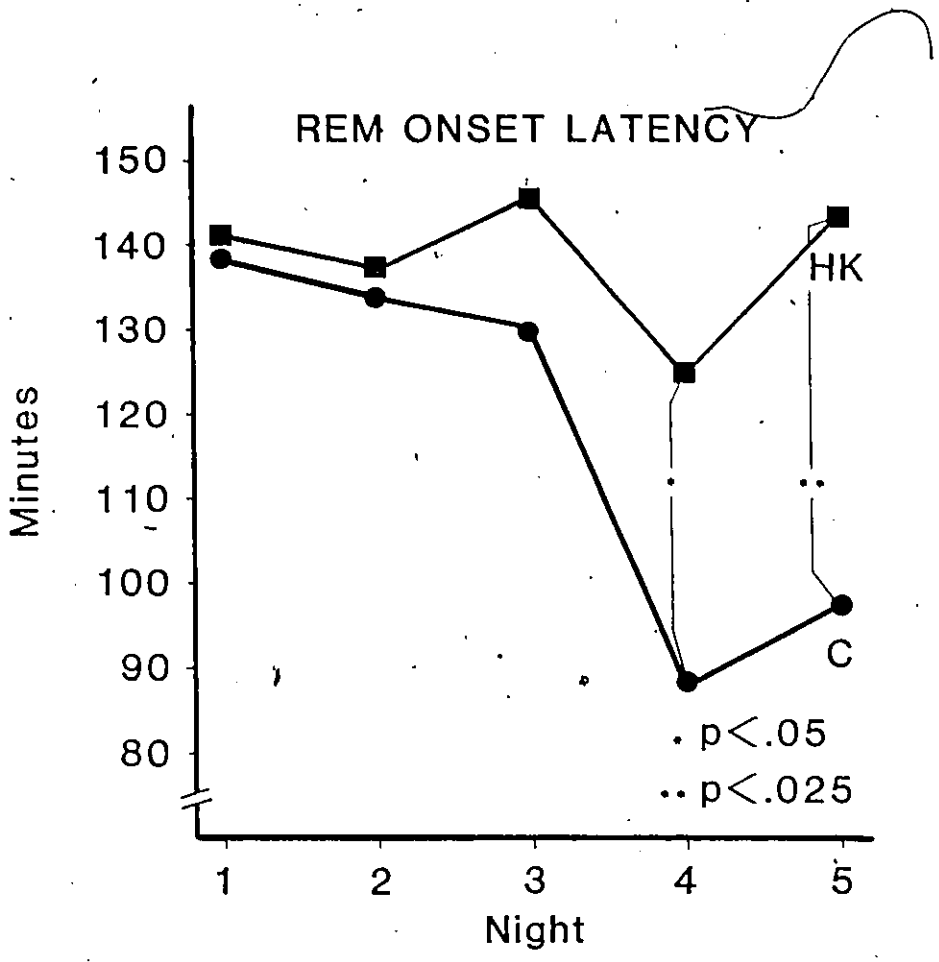


Figure 3. Mean REM onset latencies across nights.

Table 5

REM Cycle Analysis* (S. D.)

Variable	Group	Night 1	Night 3	Night 5
Frequency	HK	3.6 (1.14)	3.6 (0.89)	4.0 (0.70)
	C	3.6 (0.89)	3.6 (1.67)	4.4 (0.54)
Mean length (min.)	HK	87.57(10.45)	100.49(20.69)	86.57(16.46)
	C	98.73(17.04)	100.25(21.46)	92.43(8.23)

* REM Cycle: onset of first REMP to onset of second REMP.

Controls had significantly greater amounts of MT on nights 3 and 5 relative to night 1 for both absolute and percentage measurements ($p < .05$) whereas comparable changes were not present for the HK group (Table 6).

HK subjects did not differ significantly from control subjects on nights 3 and 5 when BMs (rates/min.) were analyzed. Twitches (rates/min.) during REM did not differ between groups or across nights. NREM twitches (rates/min.) differentiated the groups on night 3, with the HK subjects displaying significantly lower rates compared to controls ($p < .05$). Within group comparisons of REM versus NREM twitches revealed a significantly higher rate of twitches during REM compared to NREM on night 3 ($t = 4.55$, $df = 4$, $p < .005$) for the HK group, but no significant differences were present on night 5 for this group. Controls displayed significant higher rates of twitches in REM relative to NREM on night 3 ($t = 2.37$, $df = 4$, $p < .05$) and night 5 ($t = 2.4$, $df = 4$, $p < .05$; Table 7).

Cortical Spindles

Spindle activity analyses were conducted for tape recorded nights (3 & 5). Technical difficulties limited analyses to four subjects in each group. Spindle incidence analyses were performed for 18 minutes pre-post REMPs, stage 2 versus slow-wave, and spindle rate over a continuous six-hour period divided in four consecutive 90-minute blocks.

Table 6

Means (S.D.) of Movement Time

Variable	Group	Night 1	Night 3	Night 5
Minutes	HK	10.60(3.99)	8.92(2.81)	7.52(2.06)
	C	3.40(1.82)	8.46(5.74)	9.40(5.08)
Percentage	HK	1.40(0.55)	1.40(0.55)	1.0 (0.0)
	C	0.20(0.44)	1.20(1.30)	1.40(0.89)

Note: Significant differences among values reported in this table are discussed in the text.

Table 7

Means (S. D.) of REM-NREM Twitches (frequency/min) for Nights 3-5

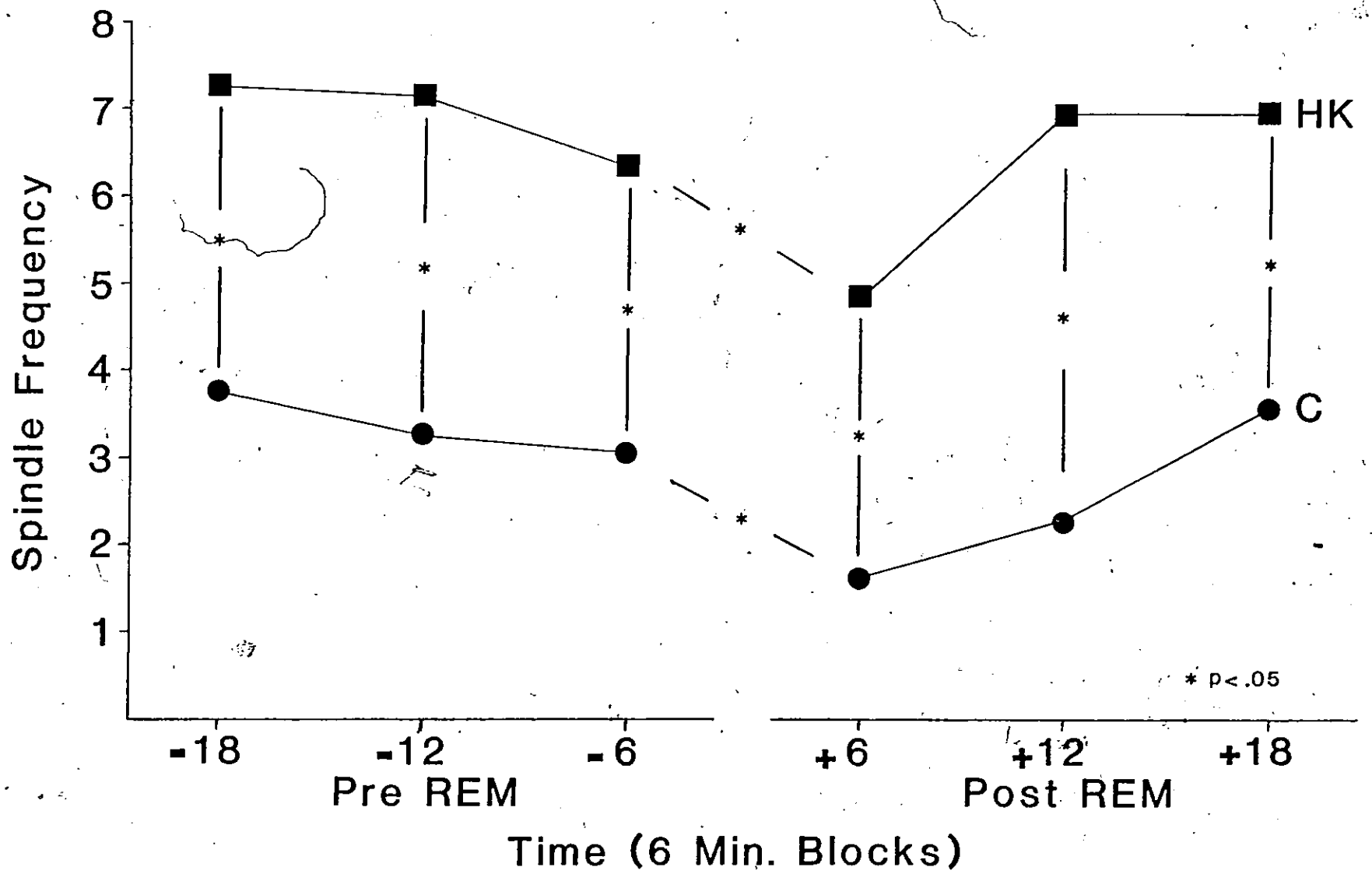
Group	Night			
	3		5	
	REM	NREM	REM	NREM
HK	1.88(.69)	.54(.18)	1.86(1.47)	.74(.51)
C	1.54(1.12)	1.12(.81)	.98(.64)	.54(.47)

Note: Significant differences among values reported in this table are discussed in the text.

Pre-Post REMPs. Spindle frequencies averaged for nights 3 and 5 were computed for three consecutive 6-minute blocks pre and post REMPs. Analyses were conducted for the first three REMPs only, since all eight subjects had at least this number of REMPs for each tape recorded night. A significant main effect for frequency was found {F (5,75) = 2.78, $p < .05$}. Post-hoc tests revealed that the HK group had significantly more spindles than controls both before and after REMPs in all 6-minute blocks ($p < .05$). When comparing pre and post REM spindle frequencies within groups, both groups were found to have significantly fewer spindles in the first post 6-minute block relative to that immediately preceding REM onset ($p < .05$). Also, a significant increase in spindle frequency from the first 6-minute block to the second 6-minute block in post REM was found for both groups ($p < .05$). These results are illustrated in Figure 4.

Stage 2 versus Slow-Wave. To equate for time within a sleep stage and time of night, analyses were conducted in time blocks of 16 minutes of stage 2 and slow-wave within the first third of nights 3 and 5. Results revealed a significant main effect for group {F (1,13) = 4.44, $p < .05$} and a significant main effect for stage {F (1,13) = 13.38, $p < .002$}, with post-hoc tests showing that the HK group had significantly more spindles/minute than controls in S2 on night 3 ($p < .01$).

Figure 4. Mean Pre-Post REM spindle frequency in 6-minute blocks pooled across REMPs for both nights 3 and 5.


Spindle Frequency
8
7
6
5
4
3
2
1
HK
C
* p< .05
-18
-12
-6
+6
+12
+18
Pre REM
Post REM
Time (6 Min. Blocks)

On night 5, no significant group differences were observed. Post-hoc analyses of the main stage effect revealed that the HK group had a higher spindle rate in S2 relative to SW on night 3 ($p < .01$). In addition, higher S2 spindle rates were found on night 3 relative to night 5 for the HK group ($p < .01$). Controls had a higher spindle rate in S2 on night 5 compared to night 3 ($p < .01$; see Figure 5).

Consecutive 90-Minute Blocks. Analyses conducted on spindle activity over 6-hour periods on nights 3 and 5 revealed a marginally significant main effect for frequency {F (3,39) = 2.35, $p < .08$} and for interaction of frequency by night {F (3,39) = 2.51, $p < .07$}, with the HK group showing a higher spindle frequency than controls on all four 90-minute blocks on night 3 ($p < .01$), but only on first 90-minute block on night 5 ($p < .01$). Within group post-hoc analyses revealed that HK subjects had significantly more spindles in the third and fourth 90-minute blocks of night 3 compared to those of night 5 ($p < .05$), whereas controls had significantly fewer spindles on night 3 relative to night 5 on all four 90-minute blocks ($p < .01$). Within night analyses across 90-minute blocks revealed no significant differences on night 3 for either group. On night 5, HKs had more spindles in the first block relative to the three later blocks ($p < .01$), whereas controls had significantly more spindles on the fourth block compared to the third block only

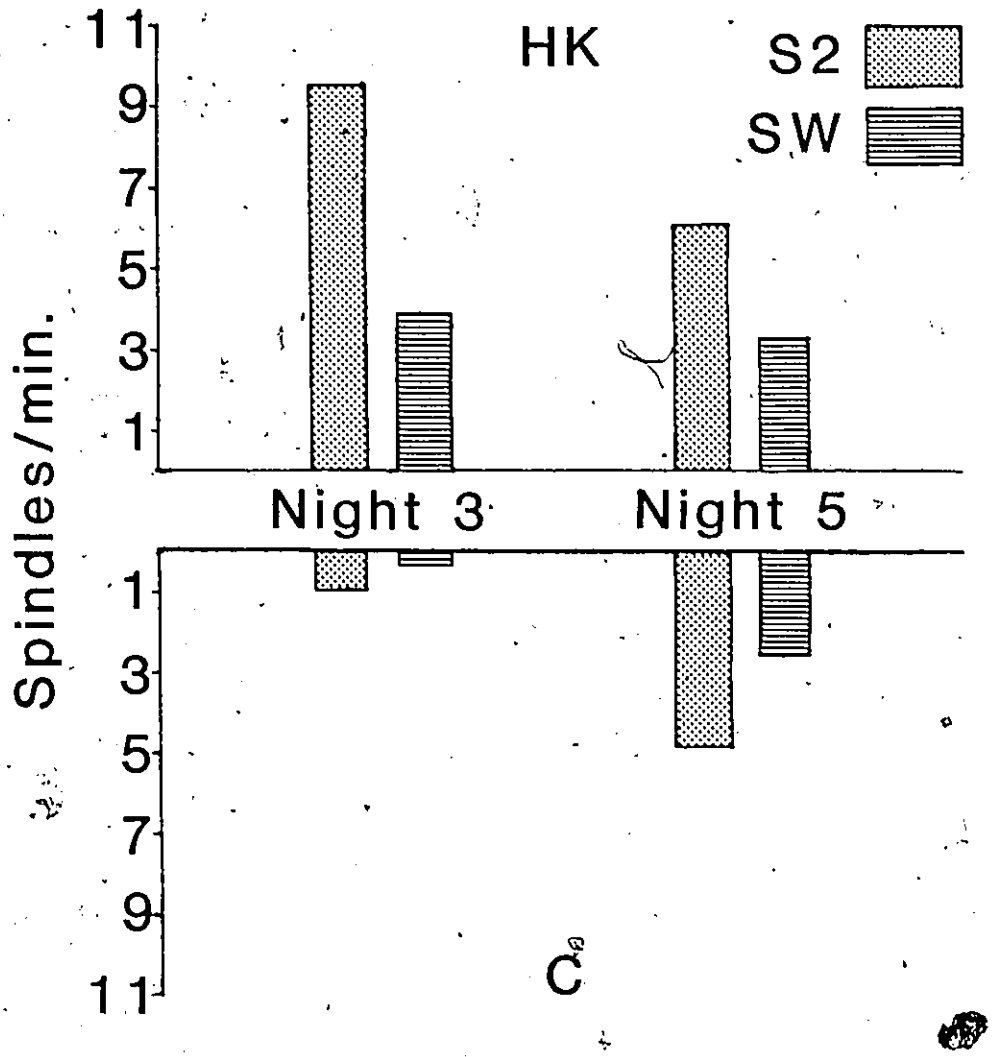


Figure 5. Mean spindle rate for 16 minutes of stage 2 and slow-wave within the first third of nights 3 and 5.

($p < .05$). Other comparisons did not reveal significant results (Figure 6). The previously noted inverse relationship between spindle activity and phasic motor activity was corroborated by the group data in the present study. For both groups, increases or decreases in spindle rate were parrallelled by decreases and increases in NREM twitches (Table 7 and Figure 6).

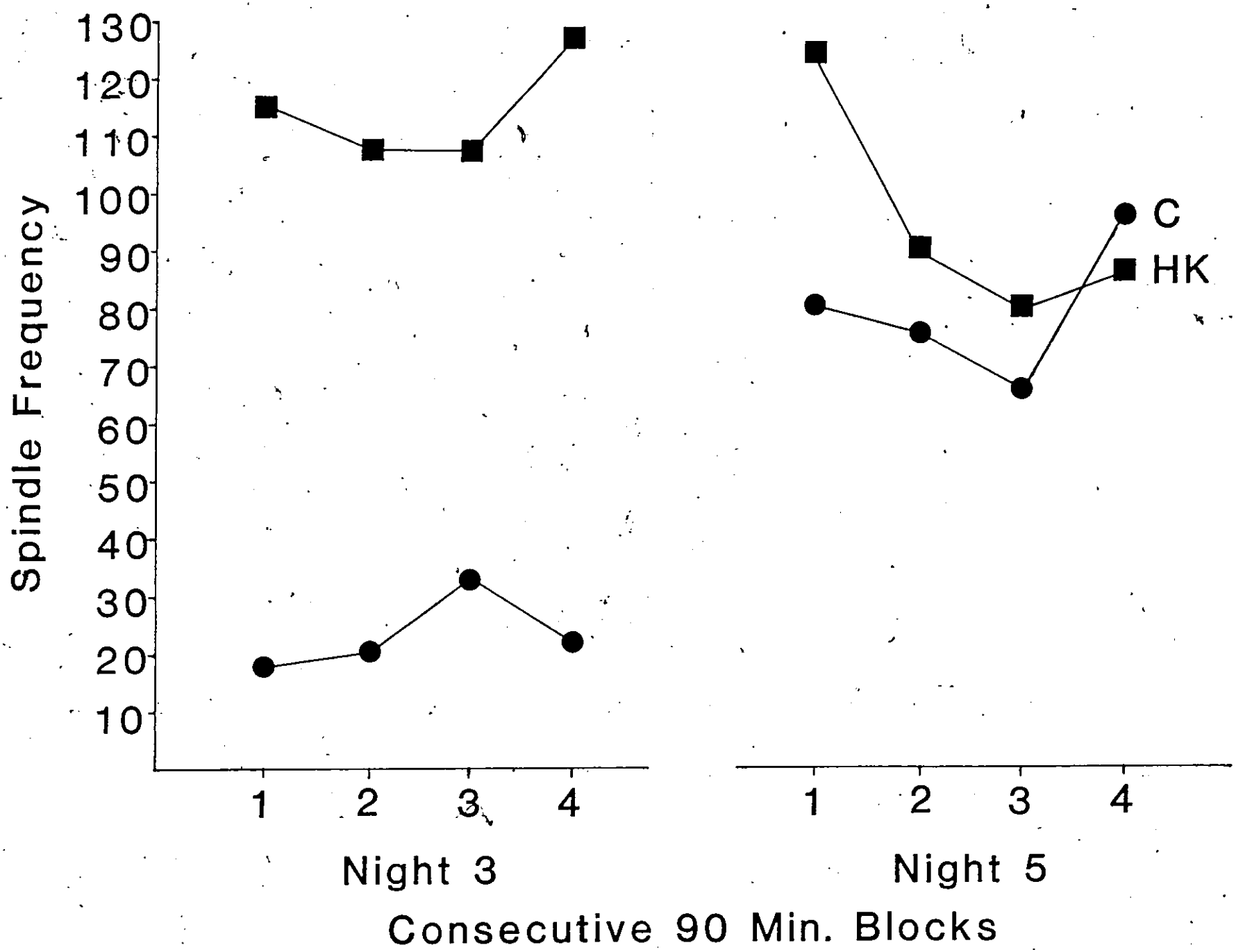
Autonomic Activity

SSPR rates on nights 3 and 5 were based on the total amounts of scorable responses divided by the number of minutes of the accompanying behavioral state, i.e., W, S4, NREM, and REM. No significant group or night differences were found. Comparisons of stage differences in mean SSPR rates (paired t-tests) revealed lower rates during REM than NREM for both groups (HK: $t = 2.38$, $df = 4$, $p < .05$; C: $t = 5.33$, $df = 4$, $p < .01$). Only the HK group showed higher rates in W compared to REM ($t = 2.65$, $df = 4$, $p < .05$) and in S4 compared to W ($t = 2.37$, $df = 4$, $p < .05$). When S4 was compared to the remaining NREM stages, both groups showed significantly higher SSPR rates in S4 (HK: $t = 2.51$, $df = 4$, $p < .05$; C: $t = 5.33$, $df = 4$, $p < .005$). Both groups also displayed higher rates in S4 relative to REM (HK: $t = 2.67$, $df = 4$, $p < .05$; C: $t = 6.41$, $df = 4$, $p < .005$).

Eye Movement Activity

REM density measures were computed for the first four REMPs on nights 3 and 5 for four categories of EMs (e.g., horizontal,

Figure 6. Mean spindle frequency on nights 3 and 5 for four consecutive 90-minute blocks.


Spindle Frequency
130
120
110
100
90
80
70
60
50
40
30
20
10
C
HK
1
2
3
4
Night 3
Night 5
Consecutive 90 Min. Blocks

vertical, mixed [horizontal & vertical EMs occurring simultaneously], and total). Repeated measures analysis of variance (ANOVA) was conducted on total EM density for the 4 REMPs to determine intra-night density changes. A significant main effect for REMP was present {F (3,24) = 9.67, $p < .05$}, and post-hoc tests showed that total EM density was significantly lower in the first REMP compared to the later REMPs for both groups ($p < .05$). Total EM density increased from the second REMP to the third for control subjects ($p < .05$). Furthermore, total EM density differentiated the two groups on the first and fourth REMPs ($p < .05$), with the HK group displaying higher densities on both occasions (see Figure 7).

Since REMP durations vary across the night and EM density measures may be influenced by longer REMPs, density measures were calculated over the first 15 minutes (5-minute blocks) of the four REMPs to control for effects of REM duration on EM densities. Individual analyses (ANOVA with repeated measures) were conducted on each 5-minute block for every EM category. These analyses limited comparisons to group differences and yielded only one difference, i. e., the HK group had more vertical EMs than controls in the second 5-minute block of the first REMP {F (1,8) = 22.56, $p < .01$; see Figure 8}. Total EM analyses performed on the first 5-minute block of each REMP, controlling for REM duration and time of night, revealed no significant group or night effects.

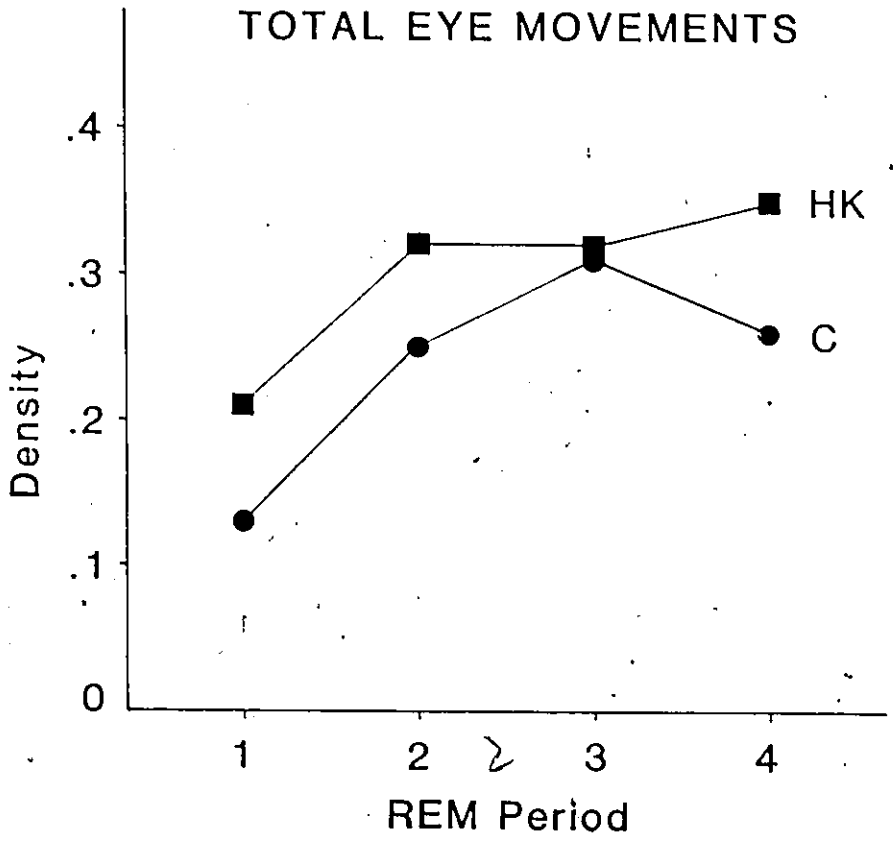
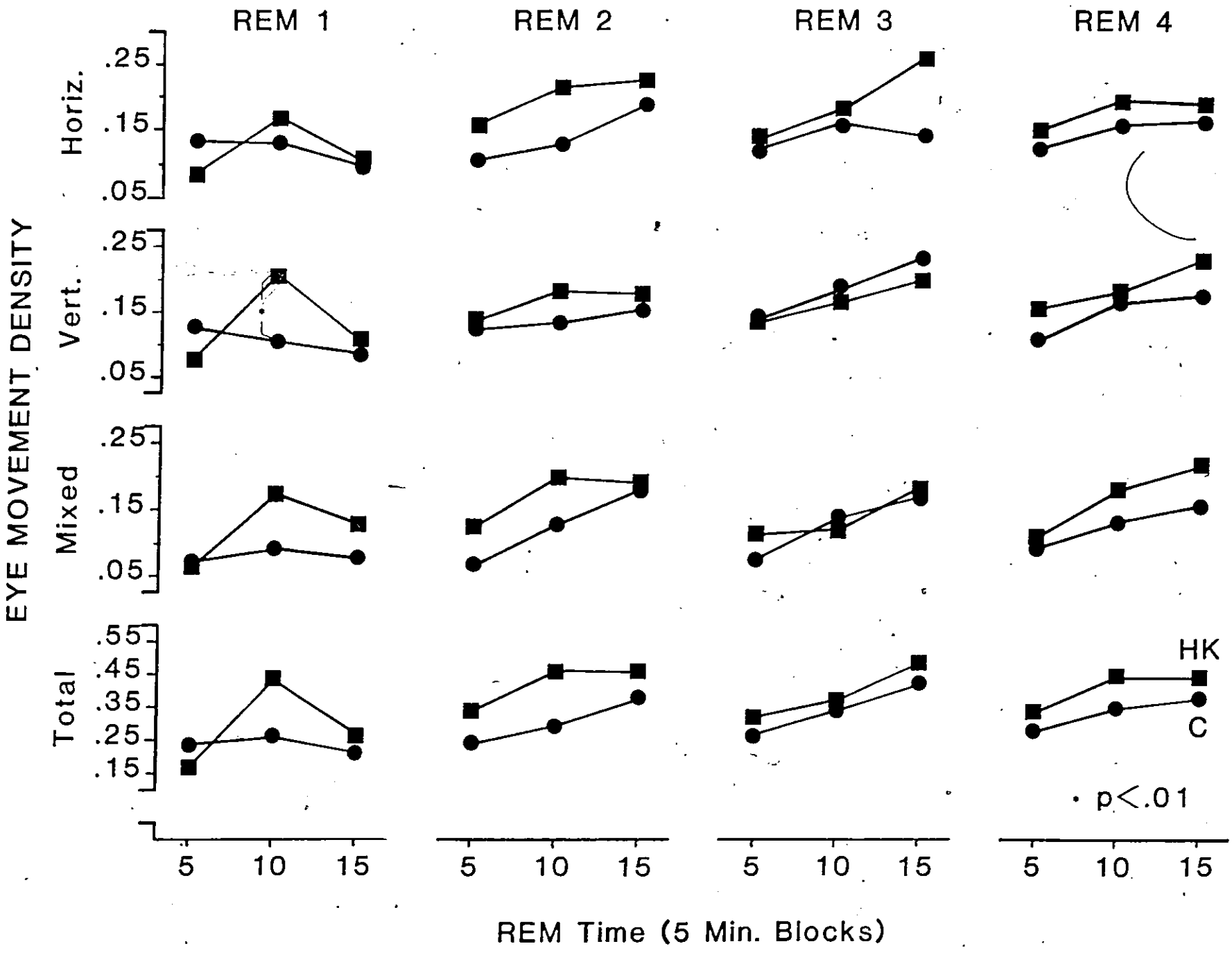


Figure 7. Mean total eye movement density across 4 REMPs.

Figure 8. Mean horizontal, vertical, mixed and total eye movement density on the first 15 minutes (5-minute blocks) across 4 REMPs.


REM 1
REM 2
REM 3
REM 4
EYE MOVEMENT DENSITY
Horiz.
Vert.
Mixed
Total
.25
.15
.05
.55
.45
.35
.25
.15
5
10
15
HK
C
• p<.01
REM Time (5 Min. Blocks)

Analyses were also conducted on 15-minute blocks comparing horizontal and vertical EM densities. A significant main interaction effect (category by group by REMP) was found {F (3,24) = 5.33, p < .005}, with HK subjects having significantly higher horizontal EM density on REMPs 2, 3, and 4 compared to controls (p < .05). HK subjects also had greater vertical EM density in the first and fourth REMPs relative to controls (p < .05). Within group analyses revealed a significant increase in horizontal and vertical EM densities from the first to the second REMP for the HK group (p < .05). Vertical EM density increased from REM1 to REM2 to REM3, and decreased from REM3 to REM4 (p < .05) for the control subjects. Within REMP analyses revealed higher horizontal than vertical EM density for the HK group during the second REMP (p < .05), and the control group was found to have higher vertical than horizontal EM density on the third REMP (p < .05).

Chapter IV

DISCUSSION

Tonic Sleep Measures

With the exception of the report by Luisada (1969), the results of the present study agree with others in finding that absolute (minutes) and percentage amounts of the various sleep stages, REM cycle durations, as well as latencies to sleep onset or NREM sleep stages, do not significantly differentiate unmedicated HK children from control subjects. In many respects, the sleep of HK children is unusually efficient. For example, these children generally fall asleep more quickly, waken less often during sleep, have less stage 1 sleep, and show a higher sleep efficiency index (95%) than control subjects. However, since the sleep of normal children is also quite efficient - - i. e., in the present study, on the average, 94% of the time in bed was spent asleep - - it would be difficult to demonstrate significant group differences in the direction of more efficient sleep, and indeed such differences were not observed. Nevertheless, the trend in this direction for HK subjects indicates exceptionally efficient sleep which could be considered consistent with central nervous system hypoarousal.

In corroboration of previous reports (Busby, 1980; Haig et al., 1974), HK children had longer REM onset latencies, but, in the present study, significantly so only on the fourth and

fifth nights. The enhancement of this effect on nights four and five could be related to adaptation effects in the control group. On these nights the values of several sleep parameters were indicative of adaptation effects, i. e., relative to the first three nights, TST was increased and TWT and WASO were decreased. The familiarity of the HK subjects with the hospital environment may have diminished or eliminated the commonly observed first night effect for this group (Agnew, Webb, & Williams, 1966).

The consistently longer latencies to REM onset in HK children cannot be attributed to sleep disruption or sleep deprivation since, as discussed above, sleep stage values and other indices of sleep efficiency are essentially the same across groups and are not indicative of disturbed sleep. Furthermore, although the initial REM period is often skipped in children of this age group (Roffwarg, Muzio, & Dement, 1966), the differential clustering of extended latencies in the hyperactive group and previous reports of this phenomenon in hyperactive children suggest that the basis for the extended REM latencies is probably not attributable to inconsistency in the appearance of the first REM period.

REM onset latency has been shown to vary in association with a variety of factors and conditions. Those associated with decreases in latency include: REM deprivation (Dement, 1960); maturational status. i.e., neonates and infants experience sleep

onset REM periods (Roffwarg, et al., 1966); clinical depression (Kupfer, 1976); and narcolepsy (Montplaisir, Billard, Takahashi, Bell, Guilleminault, & Dement, 1978). Increases in REM onset latency have been related to: maturation, i.e., 4-6 year old children have extended latencies relative to older children and adults (Feinberg, Koresko, & Heller, 1967); the administration of biochemical compounds, e.g., amphetamine (Rechtschaffen, & Maron, 1964); barbiturates (Kales, Malstrom, Scharf, & Rubin, 1969); and alcohol (Johnson, Burdick, & Smith, 1970); afternoon naps (Moses, Hord, Lubin, Johnson, & Naitoh, 1975); and exercise (Baekeland, 1970; Browman, & Tepas, 1976; Desjardins, Healey, & Broughton, 1974; Hobson, 1968; Matsumoto, Nishisho, Suto, Sadahiro, & Miyoshi, 1968).

The enhanced REM onset latency following exercise may be of potential relevance to the interpretation of results from the present study since the waking behavior of HK children is thought to be characterized by increased motor activity relative to that in same-age peers. Despite the logical appeal of this relationship, it must remain a tentative possibility since the association between enhanced latencies following exercise has been an inconsistent finding (Baekeland, & Lasky, 1966; Hauri, 1968; Walker, Floyd, Fein, Cavness, Lualahati, & Feinberg, 1978), and since no evaluation was made of pre-sleep activity levels in the present investigation.

Data regarding covariations in REM onset latency with maturation offer another possible basis for interpretation of the REM latency data in the present study. Feinberg et al. (1967) reported a mean REM onset latency of 123 minutes for a sample of 10 year old children and 140 minutes for sample of 6 year old children. In the present study, and in Busby's (1980) investigation, as well, REM onset latencies in the HK groups fell in the range of younger-aged children. These findings are suggestive of the presence of a neuro-developmental lag in HK children to which mechanisms responsible for the latency to onset of the first REM period might be especially sensitive. If such a neuro-developmental process is involved, it is not reflected in general sleep stage patterns since such measurements did not differentiate between groups in the present study.

Although increased day time motility and/or neuro-developmental (maturational) lag provide plausible explanations for the enhanced REM onset latencies observed in the present study, there may be additional factors, as yet unknown, which influence mechanisms involved in determining the time at which the initial REM period of the night occurs relative to sleep onset.

Motor Activity Measures

Although it might be expected that HK children would display more motor activity during sleep than control subjects, only two studies have reported such differences. Small et al. (1971)

reported that HK subjects had greater amounts of muscle activity during NREM sleep compared to controls and Busby (1980) reported greater amounts of MT, BMs and NREM twitches in HK children relative to control comparisons. In the present study, HK subjects had greater amount of MT (absolute and percentage value) compared to controls on the first night. Although no significant night effect was noted for this variable for HK subjects, decreasing amounts of MT were present across nights. Control subjects, however, showed an increase in MT on later nights relative to night one. The basis for this latter observation is unclear. Generally, a decrease in this parameter would be expected across nights as adaptation to the experimental environment and conditions occurred. As previously discussed, other sleep parameters do show tendencies in the direction of an adaptation effect. In view of these other indexes of post-sleep arousal in this group on night one, i.e., increased time awake after sleep onset and increased stage one sleep, one possibility is that the time these subjects spend asleep was relatively undisturbed, but that when any disturbance did occur, it resulted in a rapid awakening or was not sufficiently extensive to be scored as MT.

Expected group differences in phasic motor measures, i.e., BMs and twitches, were not present. Although more intense phasic motor activity was observed during REM relative to NREM (except for the HK group on night five where REM and NREM

twitches did not differ significantly), between group differences were present only on night three NREM twitch measures, with HK subjects exhibiting fewer twitches than controls. There is only one previous study examining phasic motor activity during sleep in HK children, and in that study (Busby, 1980) more phasic motor activity was found in both REM and NREM sleep in HK subjects relative to controls. The group difference in that study although significant ($p < .06$) was not marked. The absence of such an effect in the present study may derive from population differences or could be based in the smaller number of subjects (less than half as many) in the present investigation. Another factor of possible relevance to the issue of motor activity during sleep is that subjects in both the present study and that by Busby were not characterized by excessive amounts of waking motor activities. Small et al. (1971), who studied children with marked waking hyperactivity, found significantly more motor activity in both REM and NREM sleep. Taken together, these data suggest that the degree of motor involvement during waking is somewhat mirrored in levels of motor activity during sleep.

How can these data be applied to the hyperarousal - hypoarousal distinction? If the physiological systems of hyperkinetic children respond to changes in state, then, in hypoaroused children more phasic activity should be expressed -

during NREM sleep since a general reduction in CNS activation has occurred both because of the reduction in exogenous stimulation in the quiet sleeping environment, as well as the relative reduction of physiological activity attendant to NREM sleep. If the basis for hyperkinesis is CNS hyperarousal which become salient under conditions of increased exogenous stimulation, then phasic motor activity should be reduced during NREM sleep. In this regard the expectations regarding REM sleep are more problematical. The intense physiological activation generally present during REM sleep might be enhanced in the hyperaroused hyperkinetic child, but if hypoarousal were the basis for the hyperkinetic behavior, normal levels of phasic motor activity might be anticipated since the inherent physiological activation REM sleep could provide sufficient endogenous stimulation. In the present study and in that of Busby (1980), phasic skeletal muscle twitches during REM sleep did not differentiate HK and control subjects. However, the marginally significant increase in NREM twitches reported by Busby (1980) was not observed in the present study - - in fact on one night NREM twitch rate was significantly reduced in HK children relative to controls. Busby interpreted his data as most congruent with central hypoarousal, and the REM data from the present study could also be interpreted in this manner. However, results of NREM phasic motor activity analyses in the present study fail to clearly favor either

thedretical position.

Cortical Spindles

There is only one previous report dealing with cortical spindle activity during sleep in HK children (Khan and Rechtschaffen, 1978). In that study, analyses were done by visual scoring procedures and limited to stage 2 sleep and HK subjects were found to have fewer spindles compared to controls. In the present study, the analyses were automated and were not restricted to stage 2 sleep but encompassed all of NREM sleep containing spindle activity. In this study, HK children had more spindle activity relative to controls. Differences in methodology or subject characteristics may account for the difference in results of the two studies.

Cortical spindles have been related to reduced motor activity (Buchwald, & Eldred, 1961), specific depression of phasic motor activity (Howe, & Sterman, 1972) and increased arousal threshold (Johnson et al., 1976) - all of which suggest a transient depression of activity in association with spindles. Interpreted within the hyper-hypoarousal framework, these results may be most easily accommodated by the hypoarousal position.

The spindle analyses for frequency during stage 2 and slow-wave sleep (SWS) and pre- and post-REM periods provide new information regarding this activity under these conditions, but did not reveal additional group differences in these measures.

It is known that arousal threshold is generally higher during SWS than other NREM sleep stages, and it is therefore of interest to note that spindle frequency - - which is also postulated to increase with increased arousal threshold - - is higher during stage 2 than SWS. This may suggest the existence of different mechanisms underlying the determination of arousal, or the same mechanisms with varying EEG correlates, e.g., spindles during stage 2 sleep and delta activity during SWS. According to this interpretation, an arousal threshold study of sleep in HK children - - of which there are none in the literature - - should reveal higher thresholds to arousal during stage 2 sleep in HK children than in controls.

The pre-, post-REM analyses are relevant to an understanding of electrophysiological variations occurring during stage transitions. Furthermore, there are indications that the pre-REM period is a time ~~of increased~~ activation perhaps essential to the triggering of REM sleep. In the cat, phasic activity (PGO spikes) increases at this time (Jouvet, 1962), and in man, there is an increase in body movement and K-complexes preceding REM onset (Dement, 1967). In view of these increases in excitation and specifically motor activation, it is interesting to note that spindle activity does not increase at this time. Perhaps the triggering of REM mechanisms requires increased activation not offset by inhibitory processes. The

data suggest that spindle activity continues at a relatively consistent rate during the pre-REM period until the last moments prior to REM onset. By definition, this activity virtually ceases at REM onset. The pattern of the post-REM return of spindle activity is difficult to interpret. Spindle activity is depressed for several minutes post-REM, regaining pre-REM values within twelve minutes after the end of the REM period. The initial post-REM period is generally devoid of phasic motor activity and perhaps there is therefore no activation of spindle mechanisms. The soundness of these speculations regarding condition and group variations in cortical spindle activity will require studies specifically oriented towards conditions and group correlates of this activity. In view of the prevalence of spindling during sleep (i.e., stage 2 sleep, in which this activity is most profuse, accounts for more than half of total sleep time) such studies regarding the correlates and function of this activity are long overdue.

Autonomic Activity

The findings in this study on electrodermal activity in HK children during sleep and wakefulness are consistant with those of the only previous study examining this measure in HK children's sleep (Busby, 1980) i.e., SSPR rates did not differentiate between groups. Furthermore, these findings support those previously reported in sleep studies of children and normal young adults (Broughton et al., 1965; Johnson, & Lubin, 1966; Lester

et al., 1967) i.e., the highest SSPR rates were observed in stage 4 and the lowest in REM sleep.

This absence of state-related variations in spontaneous electrodermal activity suggests that this autonomic index, measured under passive conditions, does not provide discriminating information regarding the HK syndrome.

Eye Movement Activity

Although it has been proposed that EM density during REM sleep may serve as an index of the general level of CNS activation (Aserinsky, & Kleitman, 1955; Dement, Ferguson, Cohen, & Barchas, 1969), only three of the eight previous sleep studies involving HK children have performed eye movement density analyses (Busby, 1980; Feinberg et al., 1974; Small et al., 1977). Interestingly, none of these studies reported group differences in this measure. In the present study, although there was a trend toward relatively greater eye movement density in HK subjects which achieved significance in the first and fourth REM periods, the results generally corroborated previous reports in not finding significant group differences either in whole-night or in five minute REM period density measures. If the noted trend toward increased density in the HK group is meaningful, then it more likely emanates from a hypoaroused system since a much stronger synergistic effect would have been anticipated from a hyper-aroused system.

The total EM density results reported above failed to differentiate between eye movement on the basis of directionality, and this differentiation has been shown to be important, particularly for the horizontal-vertical dichotomy (Feinberg et al., 1969). None of the previous sleep studies of hyperkinetic children has reported EM analyses which distinguish among eye movements on the basis of directionality. Feinberg et al., (1969) reported a reduced proportion of vertical eye movements in normal aged adults (75 yrs.) compared to normal young adults (25.2 yrs.), and hypothesized that this reduced proportion of vertical EMs may reflect the reduced intensity of REM sleep accompanying old age. Although there were differential concentrations of horizontal and vertical eye movements observed in the present study, namely, HK children had significantly higher horizontal EM densities in the second, third, and fourth REM periods and higher vertical EM densities in the first and fourth REM periods relative to those of controls, differential EM densities pooled across REM periods did not differentiate the groups. Since a decrease in vertical EM density is thought to occur with age, the absence of a tendency toward higher vertical EM density in the HK group could be interpreted as evidence against a developmental lag explanation for the disorder in these children.

In summary, in many respects the sleep of HK children does not differ significantly from that of normal children. For example, there were no dramatic differences between groups in

the amounts of conventional sleep stages or intensities of motor, autonomic and eye movement activity. Effects that were prominent, namely increased REM onset latency and increased spindle activity in HK children, should be subjected to further study. The increased latency to the initial REM period has been observed in previous studies and may represent a significant deviation from normality in this clinical group. The dramatic difference in cortical spindle activity warrants replication and more detailed analyses in terms of motor and behavioral (arousal threshold) correlates of this activity.

An inherent problem in studies of sleep parameters is the statistical weakness which derives from the examination of many dependent variables in a limited number of subjects. Although such limitations apply to the analyses in the present study, it is important to note that the significant differences which were emphasized were either replications of previous observations or were of sufficient magnitude and consistency that the likelihood that such results would be spurious would be remote.

In general, the differences which were present between groups could be interpreted as CNS hypoarousal in these children, but the study of sleep patterns and associated with events does not indicate the presence of a major arousal dysfunction in these children.

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Appendix A

Parent's Questionnaire

PARENT'S QUESTIONNAIRE

Patient No. _____

Study No. _____

Name of Child _____ Date _____

Your Name _____ Relationship _____

1. Instructions: Listed below are items concerning children's behavior or the problems they sometimes have. Read each item carefully and decide how much you think your child has been bothered by this problem during the past month.

NOT AT ALL, JUST A LITTLE, PRETTY MUCH, or VERY MUCH

Indicate your choice by placing a check mark ( ) in the appropriate column to the right of each item.

ANSWER ALL ITEMS.

Observation	Not at all	Just a little	Pretty much	Very much
PROBLEMS OF EATING				
1. Picky and finicky				
2. Will not eat enough				
3. Overweight				
PROBLEMS OF SLEEP				
4. Restless				
5. Nightmares				
6. Awakens at night				
7. Cannot fall asleep				
FEAR AND WORRIES				
8. Afraid of new situations				
9. Afraid of people				
10. Afraid of being alone				
11. Worries about illness and death				
MUSCULAR TENSION				
12. Gets stiff and rigid				
13. Twitches, jerks, etc.				
14. Shakes				
SPEECH PROBLEMS				
15. Stuttering				
16. Hard to understand				
Wetting				
17. Bed wetting				
18. Runs to bathroom constantly				
BOWEL MOVEMENT				
19. Soiling self				
20. Holds back bowel movement				

ANSWER ALL ITEMS

Observation	Not at all	Just a little	Pretty much	Very much
COMPLAINS OF FOLLOWING SYMPTOMS EVEN THOUGH DOCTOR CAN FIND NOTHING WRONG				
21. Headaches				
22. Stomach aches				
23. Vomiting				
24. Aches and pains				
25. Loose bowels				
PROBLEMS OF SUCKING, CHEWING OR PICKING				
26. Sucks thumb				
27. Bites or picks nails				
28. Chews on clothes, blankets or other				
29. Picks at things such as hair, clothing, etc.				
CHILDISH OR IMMATURE				
30. Does not act his age				
31. Cries easily				
32. Wants help doing things he should do alone				
33. Clings to parents or other adults				
34. Baby talk				
TROUBLE WITH FEELINGS				
35. Keeps anger to himself				
36. Lets himself get pushed around by other children				
37. Unhappy				
38. Carries a chip on his shoulder				
OVER ASSERTS HIMSELF				
39. Bullying				
40. Bragging and boasting				
41. Sassy to grown-ups				
PROBLEMS MAKING FRIENDS				
42. Shy				
43. Afraid they do not like him				
44. Feelings easily hurt				
45. Has no friends				
PROBLEMS WITH BROTHERS AND SISTERS				
46. Feels cheated				
47. Mean				
48. Fights constantly				
PROBLEMS KEEPING FRIENDS				
49. Disturbs other children				
50. Wants to run things				
51. Picks on other children				
RESTLESS				
52. Restless or over active				
53. Excitable, impulsive				
54. Fails to finish things he starts - short attention span				

ANSWER ALL ITEMS Observation	Not at all	Just a little	Pretty much	Very much
TEMPER				
55. Temper outbursts, explosive and unpredictable behavior				
56. Throws himself around				
57. Throws and breaks things				
58. Pouts and sulks				
SEX				
59. Plays with own sex organs				
60. Involved in sex play with others				
61. Modest about his body				
PROBLEMS IN SCHOOL				
62. Is not learning				
63. Does not like to go to school				
64. Is afraid to go to school				
65. Daydreams				
66. Truancy				
67. Will not obey school rules				
LYING				
68. Denies having done wrong				
69. Blames others for his mistakes				
70. Tells stories which did not happen				
STEALING				
71. From parents				
72. At school				
73. From stores and other places				
FIRE SETTING				
74. Sets fires				
TROUBLE WITH POLICE				
75. Gets into trouble with police Why?				
PERFECTIONISM				
76. Everything must be just so				
77. Things must be done same way every time				
78. Sets goals too high				
ADDITIONAL PROBLEMS				
79. Inattentive, easily distracted				
80. Constantly fidgeting				
81. Cannot be left alone				
82. Always climbing				
83. A very early riser				
84. Will run around between mouthfuls at meals				
85. Demands must be met immediately - easily frustrated				
86. Cannot stand too much excitement				
87. Laces and zippers are always open				
88. Cries often and easily				
89. Unable to stop a repetitive activity				
90. Acts as if driven by a motor				
91. Mood changes quickly and drastically				
92. Poorly aware of surroundings or time of day				
93. Still cannot tie his shoelace				

II. Please add any other problems you have with your child.

III. How serious a problem do you think your child has at this time?

( ) No Problem ( ) Minor Problem ( ) Serious Problem

IV. Indicate the items you are most concerned about or those you think are the most important problems your child has by placing a circle around the number of those items.

Appendix B

School Report

SCHOOL REPORT *

Patient No.
Study No.

Name of Child _____ Date _____

School Attended _____ Grade _____

School Address _____
Number and Street City State

Name of Principal _____

I. How long have you known this child? _____ In your own words describe briefly this child's main problem. _____

_____

_____

_____

_____

II. STANDARDIZED TEST RESULTS

A. Intelligence Tests

Name of Test	Date	C.A.	M.A.	I.Q.

B. Most Recent Achievement Tests

Subject	Grade When Tested	Achievement Grade Level
Reading		
Spelling		
Arithmetic		

III. ACHIEVEMENT IN SCHOOL SUBJECTS

A. List subjects into the appropriate category

Very Good	Average	Barely Passing	Failing

B. Check special placement or help this child has received.

( ) Ungraded ( ) Sight-Saving ( ) Special Class ( ) Remedial Reading

( ) Speech Correction ( ) Tutoring, specify subjects _____

_____

( ) Other, specify _____

_____

* Report designed by C. Keith Conners, Ph. D.

IV. Listed below are descriptive terms of behavior. Place a check mark in the column which best describes this child. ANSWER ALL ITEMS

Observation	Degree of Activity			
	Not at all	Just a little	Pretty much	Very much
CLASSROOM BEHAVIOR				
1. Constantly fidgeting				
2. Hums and makes other odd noises				
3. Demands must be met immediately - easily frustrated				
4. Coordination poor				
5. Restless or overactive				
6. Excitable, impulsive				
7. Inattentive, easily distracted				
8. Fails to finish things he starts - short attention span				
9. Overly sensitive				
10. Overly serious or sad				
11. Daydreams				
12. Sullen or sulky				
13. Cries often and easily				
14. Disturbs other children				
15. Quarrelsome				
16. Mood changes quickly and drastically				
17. Acts "smart"				
18. Destructive				
19. Steals				
20. Lies				
21. Temper outbursts, explosive and unpredictable behavior				
GROUP PARTICIPATION				
22. Isolates himself from other children				
23. Appears to be unaccepted by group				
24. Appears to be easily led				
25. No sense of fair play				
26. Appears to lack leadership				
27. Does not get along with opposite sex				
28. Does not get along with same sex				
29. Teases other children or interferes with their activities				
ATTITUDE TOWARD AUTHORITY				
30. Submissive				
31. Defiant				
32. Impudent				
33. Shy				
34. Fearful				
35. Excessive demands for teacher's attention				
36. Stubborn				
37. Overly anxious to please				
38. Uncooperative				
39. Attendance problem				

V. FAMILY OF CHILD

A. Do other children in the family who attend your school, present any problems? If YES, please explain. _____

B. Please add any information concerning this child's home or family relationships which might have bearing on his attitudes and behavior, and include any suggestions for improvement of his behavior and ajustment. (Use reverse side if more space is required).

_____ _____ _____

Signature Title Date signed

Appendix C

Consent Form

ROYAL OTTAWA
HOSPITAL 1145 CARLING AVENUE, OTTAWA, ONTARIO K1Z 7K4 • TELEPHONE 613/722-6521

CONSENT FOR SLEEP STUDY

I, parent/guardian of

hereby agree that he/she has a few nights of sleep recorded by electroencephalogram (EEG), as part of a research study conducted at the Royal Ottawa Hospital by Dr. J. Simeon and R.T. Pivik, Ph.D.

The procedures, aims and method of the study have been fully explained to me. I was informed that any findings will be treated confidentially, and of my child's rights not to participate and to withdraw from the study at any time.

I understand that I will also have access to the results if I so desire at the completion of the study or at the discretion of the investigators.

Transportation to and from the Royal Ottawa Hospital and delivering my child to the charge of the sleep technicians who are volunteers working under the supervision of the investigators, will be my responsibility.

Signature of parent/guardian _____

Relationship _____

Address _____

Witness _____

I confirm that I have explained the nature of this study and consent to the parent.

Investigator _____

Date _____